

대한신경과학회

KOREAN NEUROLOGICAL ASSOCIATION

2015년도
제34차 추계학술대회

2015 34th Annual Meeting of the Korean Neurological Association

- 초 록 집 -

일시: 2015. 11. 13.(금) - 11. 14.(토)

장소: 그랜드힐튼호텔 컨벤션센터



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준비위원회

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이 사 장	윤병우	서울의대	법제이사	박건우	고려의대
부 회 장	신현길	두 신경과	홍보이사	석승한	원광의대
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총무이사	성정준	서울의대	무임소이사	이일근	서울브레인신경과
학술이사	서대원	성균관의대	진료지침이사	홍승봉	성균관의대
수련이사	김재문	충남의대	정도관리이사	고임석	국립중앙의료원
고시이사	김승현	한양의대	정책이사	김원주	연세의대
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편집이사	오건세	을지의대	교육이사	이동국	대구가톨릭의대
재무이사	이용석	서울의대			

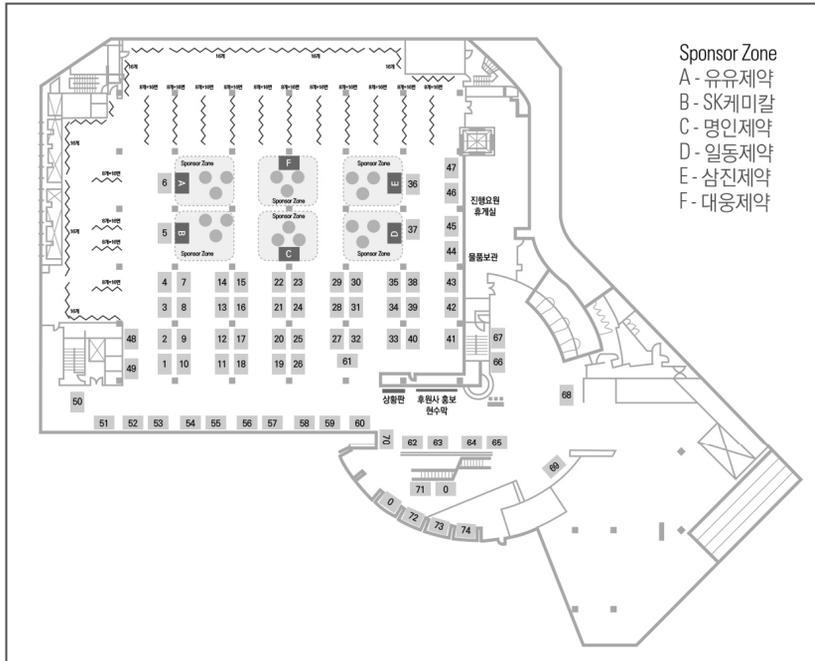
학술위원회

직책	성명	소속병원	직책	성명	소속병원
위원장	서대원	성균관의대	위 원	박민수	영남의대
간 사	윤진영	성균관의대	위 원	박종무	을지의대
위 원	고상배	서울의대	위 원	성정준	서울의대
위 원	김병건	을지의대	위 원	손영민	가톨릭의대
위 원	김병곤	아주의대	위 원	안태범	경희의대
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위 원	김성민	서울의대	위 원	이일근	서울브레인신경과
위 원	김지현	단국의대	위 원	정슬기	전북의대
위 원	박경원	동아의대	위 원	조양제	연세의대
위 원	박광열	중앙의대	위 원	홍근식	인제의대
위 원	박만석	전남의대			

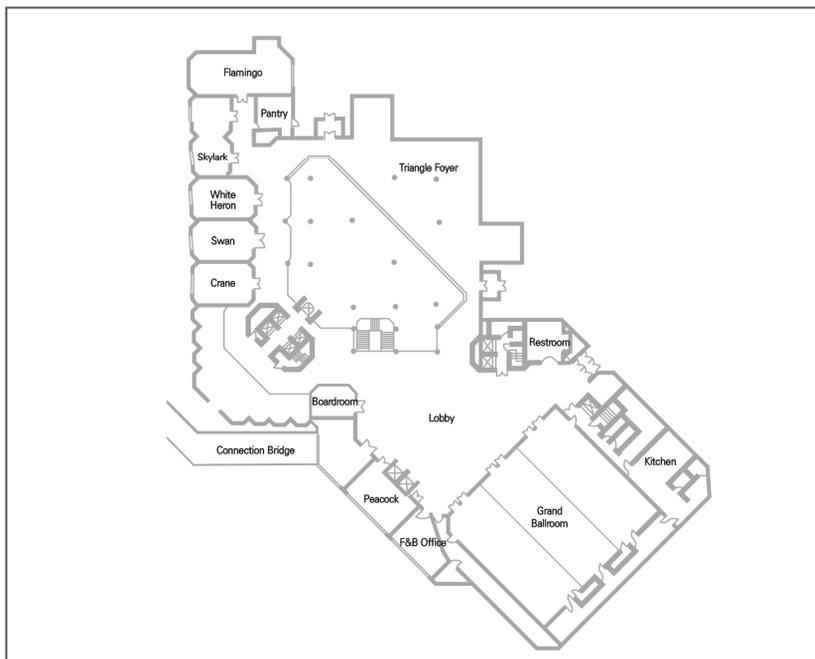
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학술대회장 안내

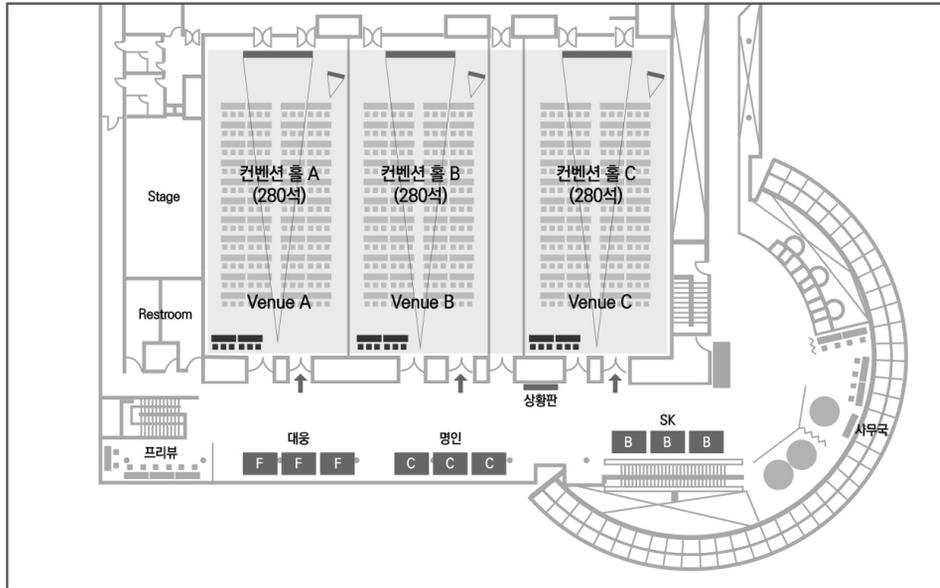
1F Convention Center



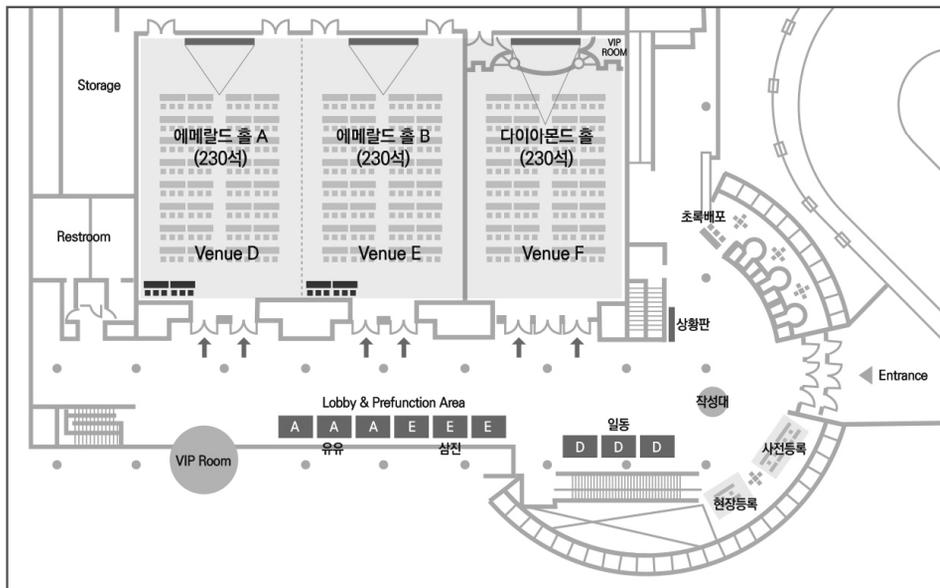
2F Main Hotel



4F Convention Center



3F Convention Center



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일정안내

1일차, 11월 13일 금요일

Time	Convention Hall A (4F)	Convention Hall B (4F)	Convention Hall C (4F)	Emerald Hall A (3F)	Emerald Hall B (3F)	Diamond Hall (3F)	Flamingo (Main Hotel 2F)
8:40-9:40	Neurology Update I			Video Session I: Movement Disorders	Video Session II: Epilepsy	Video Session III: Neuro-otology	
9:40-10:00	Coffee Break						
10:00-10:05	Opening Remark						
10:05-11:05	Myung's Memorial Lecture (Eng)						
11:05-11:50	Plenary Session (Eng)						
11:50-12:50	Luncheon Symposium and Awards (Eng)						
12:50-13:00	Break						
13:00-14:10	Poster Presentation 1 (Convention Center, 1st Floor)					신경과 정책포럼 12:40-14:10	
14:10-14:20	Break						
14:20-15:20	PS1: Dementia I (Eng)	SS1: Stroke I (Eng)	SS2: Neuroscience	SS3: Movement Disorders I	SS4: Muscle and Nerve I	SS5: Epilepsy I	편집위원회 Workshop 14:20~16:40
15:20-15:40	Coffee Break						
15:40-16:40	PS2: Neuroscience	SS6: Stroke II	SS7: Demyelinating Disorders	SS8: Sleep Disorders	SS9: Dementia I (Eng)	SS10: Headache	
16:40-16:50	Break						
16:50-17:50	PS3: Stroke I	PS4: Muscle and Nerve	PS5: Headache	PS6: Epilepsy I	PS7: Movement Disorders	PS8: Neuro-critical Care	
17:50-18:00	Break						
18:00-19:00	이사장 초청만찬 (Hotel 2F Grand ballroom)						

* Eng: English

일정안내

2일차, 11월 14일 토요일

Time	Convention Hall A (4F)	Convention Hall B (4F)	Convention Hall C (4F)	Emerald Hall A (3F)	Emerald Hall B (3F)	Diamond Hall (3F)
7:00-8:00						평의원회
8:00-9:30	Neurology Update II	Case-based Learning		Workshop I: Neurology Video Round	Workshop II: Interpretation of Neurological Tests	
9:30-9:50	Coffee Break					
9:50-10:50	PS9: Sleep Disorders	SS11: Muscle and Nerve II (Eng)	SS12: Dementia II	SS13: General Neurology I	SS14: Stroke III	SS15: Neuro-otology
10:50-11:00	Break					
11:00-11:50	Presidential Lecture (Eng)					신경계질환 우울 및 행동장애연구회 12:40-14:10
11:50-12:50	Luncheon Symposium and Awards (Eng)					
12:50-13:00	Break					
13:00-14:10	Poster Presentation 2 (Convention Center, 1st Floor)					
14:10-14:20	Break					
14:20-15:20	PS10: Demyelinating Disorders	PS11: Neuro-intervention	SS16: General Neurology II	SS17: Movement Disorders II (Eng)	SS18: Epilepsy II	SS19: Stroke IV
15:20-15:40	Coffee Break					
15:40-16:40	PS12: Neuro-otology	PS13: Dementia II	PS14: Epilepsy II	PS15: Stroke II	PS16: INM	Special Lecture: 성공적인 신경과 개원 방향
16:40-17:00			Closing Remark and Awards			
17:00-18:00					치매특별등급 교육	수련병원과장회의

* Eng: English, INM: Intra-operative neurophysiologic monitoring

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세부일정

1일차, 11월 13일 금요일

Session	Hall name	Time	Title	Chairs / Speakers
Update in Neurology/ Video Session	Convention Hall A,B,C (4F)	8:40-9:40	Update I Neurology Update I	Chairs: 정진상(성균관대의대) 박건우(고려의대)
		8:40-8:55	Dementia: Biomarkers in Alzheimer disease	정은주(인제의대)
		8:55-9:10	Epilepsy	이은미(울산의대)
		9:10-9:25	Neuroscience	김병곤(아주의대)
		9:25-9:40	Headache	조수진(한림의대)
	Emerald Hall A (3F)	8:40~9:40	Video Session I Movement Disorders: Clinical Approach to Movement	Chairs: 김희태(한양의대) 김윤중(한림의대)
		8:40-9:00	Hypokinetic movement disorders	박정호(순천향의대)
		9:00-9:20	Hyperkinetic movement disorders	김현숙(CHA의대)
		9:20-9:40	Secondary movement disorders	이승환(강원의대)
	Emerald Hall B (3F)	8:40~09:40	Video Session II Epilepsy: Differential Diagnosis of Paroxysmal Spells	Chairs: 이상암(울산의대) 김지연(대구가톨릭의대)
		8:40-9:00	Typical seizures	이가현(부산의대)
		9:00-9:20	Atypical, uncommon seizures	문혜진(계명대의대)
		9:20-9:40	Seizure-mimicking nonepileptic spells	구대림(서울의대)
	Diamond Hall	8:40~9:40	Video Session III Neuro-otology: Ocular Motility Disorders	Chairs: 김재일(단국의대) 이태경(순천향의대)
		8:40-9:00	Supranuclear-internuclear ocular motility disorders	박지윤(예수병원)
		9:00-9:20	Nuclear & infranuclear ocular motility disorders	박재한(대구가톨릭의대)
9:20-9:40		Nystagmus and related disorders	최광동(부산의대)	
Myung's Memorial Lecture	Convention Hall A,B,C (4F)	10:05-11:05	Myung's Memorial Lecture	Chair: 이광우(서울의대)
		10:05-11:05	Gene silencing therapy in ALS, spinal muscular atrophy, Huntington's disease and beyond	Don Cleveland (UCSD, USA)
Plenary Session	Convention Hall A,B,C (4F)	11:05-11:50	Plenary Session	Chair: 이병철(한림의대)
		11:05-11:50	Cluster headache	Alan M. Rapoport (UCLA, USA)

1일차, 11월 13일 금요일

Session	Hall name	Time	Title	Chairs / Speakers
Luncheon Symposium and Award	Convention Hall A,B,C (4F)	11:50~12:50	Luncheon Symposium and Awards Honorable Awards	Chairs: 윤병우(서울의대) 안무영(순천향의대)
		11:50~12:50	향설학술연구비상	이순태(서울의대)
			향설젊은연구자상	김범준(울산의대)
			SK 젊은연구자상	김치경(서울의대)
			SK 젊은연구자상	박정아(NIH)
		12:05-12:50	Luncheon Symposium	Chair: 윤병우(서울의대)
12:05-12:50	The treatment of sleep-disordered breathing in TIA/Stroke: a novel therapeutic target	Henry Klar Yaggi (Yale University, USA)		
신경과 정책 포럼	Diamond Hall (3F)	12:40-14:10	신경과 정책포럼 의료수가 결정에 대한 정책	Chair: 이병철(한림의대) Panel: 이준홍(보험공단일산병원) Panel: 오동호(미래신경과)
		12:40-13:05	의료수가 개선의 향후 방침	손영래(보건복지부 보험급여과)
		13:05-13:30	상대가치 개정연구 현황과 도입방안	공진선(건강보험심사평가원 상대가치개발부)
		13:30-13:55	수가급여결정과정에 대한 이해	지점분(건강보험심사평가원 수가관리부)
		13:55-14:10	Q & A	
Parallel Symposium/ Oral Presentation	Convention Hall A (4F)	14:20-15:20	Parallel Symposium-1 Dementia I : Amyloid & Vascular Burden in Vascular Cognitive Impairment (VCI)	Chairs: 이재홍(울산의대) 박경원(동아의대)
		14:20-14:40	Amyloid imaging in VCI	Vincent Mok (Prince of Wales Hospital, Hong Kong)
		14:40-15:00	Clinical impact of amyloid and cerebrovascular disease in SVCI patients: Korean studies	서상원(성균관의대)
		15:00-15:20	Cerebral amyloid angiopathy and VCI	Hidekazu Tomimoto (Mie University, Japan)
	Convention Hall B (4F)	14:20-15:20	Oral Presentation-1 Stroke I	Chairs: 이광호(성균관의대) 장대일(경희의대)
		14:20-14:32	Association between meteorological variables and acute stroke incidence in South Korea	권형민(서울의대)
		14:32-14:44	Risk factors for the development of depressed mood after stroke	김예림(가톨릭의대)
		14:44-14:56	Baseline characteristics and clinical outcomes of acute symptomatic internal carotid artery stumps	이성은(아주의대)
		14:56-15:08	Changes in the common carotid artery after radiotherapy: Wall, calcification and atherosclerosis	김범준(울산의대)
		15:08-15:20	One-year effect of medical treatment in atherosclerotic middle cerebral artery stenosis measured by Transcranial Doppler	김민(아주의대)

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1일차, 11월 13일 금요일

Session	Hall name	Time	Title	Chairs / Speakers
Parallel Symposium/ Oral Presentation	Convention Hall C (4F)	14:20-15:20	Oral Presentation-2 Neuroscience	Chairs: 고재영(울산의대) 김병곤(아주의대)
		14:20-14:32	Increased glucose uptake in the hemisphere with more severe ischemia among mice with right common carotid occlusion: luxury metabolism, not luxury perfusion	이진수(아주의대)
		14:32-14:44	FUS abnormalities in ALS patient-derived induced neurons	임수민(한양의대)
		14:44-14:56	The emergence of an abnormal form of long-term potentiation in aged Alzheimer's disease mouse model	최성민(전남의대)
		14:56-15:08	Characterization of white matter injury in a rat model of chronic cerebral hypoperfusion	김한영(건국의대)
		15:08-15:20	Neuroprotective effects of acetyl-L-carnitine against oxygen-glucose deprivation-induced neural stem cells death via the PI3K pathway	최호진(한양의대)
	Emerald Hall A (3F)	14:20-15:20	Oral Presentation-3 Movement Disorders I	Chairs: 이명식(연세의대) 김진호(조선의대)
		14:20-14:32	Neuropsychological tests and MCI subtypes more related to dementia in non-demented patients with Parkinson's disease	이수윤(동아의대)
		14:32-14:44	The earliest findings of comfortable gait in de novo Parkinson's disease: gait hypokinesia and effect of levodopa	권겸일(순천향의대)
		14:44-14:56	Neuropsychologic and radiologic comparisons in patients with parkinson's disease dementia in terms of onset age	김영광(연세의대)
		14:56-15:08	Retinal thinning correlates with clinical severity in multiple system atrophy	이지영(서울의대)
		15:08-15:20	The origin of T2* contrast in human Substantia Nigra: A Postmortem Validation Study at 7T MRI	이재혁(부산의대)
	Emerald Hall B (3F)	14:20-15:20	Oral Presentation-4 Muscle and Nerve I	Chairs: 임정근(계명의대) 최영철(연세의대)
		14:20-14:32	Pharmacokinetic parameters and the outcome of Guillain-Barré syndrome after intravenous immunoglobulin treatment. : Preliminary report of DEMIAN (DElta seruM IgG As a Novel biomarker for treatment outcome of Guillain-Barré syndrome) project	배종석(한림의대)
		14:32-14:44	Long-term clinical course and outcome of myasthenia gravis associated with thymoma	선우일남(선우&조 신경과)
		14:44-14:56	Human SCN4A N440K Zebrafish model of nondystrophic myotonia	남태승(전남의대)
		14:56-15:08	Prevalence and incidence of myasthenia gravis in Korea: Nationwide population-based epidemiological study	이형석(연세의대)
		15:08-15:20	Identification of de novo variants by trio-based whole exome sequencing and functional analysis of candidate genes in Korean patients with sporadic ALS	김영은(성균관대의대)

1일차, 11월 13일 금요일

Session	Hall name	Time	Title	Chairs / Speakers
Parallel Symposium/ Oral Presentation	Diamond Hall (3F)	14:20-15:20	Oral Presentation-5 Epilepsy I: Clinical Epileptology	Chairs: 박성파(경북의대) 권오영(경상의대)
		14:20-14:32	Short and long-term mortality in status epilepticus	이유진(울산의대)
		14:32-14:44	Clinical efficacy and safety of levetiracetam in epilepsy	고상준(전남의대)
		14:44-14:56	Prognostic Factors of Status Epilepticus to Predict Outcomes including Seizure Control and Future Development of Epilepsy	김태경(이화대의대)
		14:56-15:08	Risk factors and clinical outcomes associated with seizures following open heart surgery	서지혜(성균관대의대)
		15:08-15:20	The HLA-A*2402/Cw*0102 haplotype is associated with lamotrigine-induced maculopapular eruption in the Korean population	문장섭(서울의대)
	Convention Hall A (4F)	15:40-16:40	Parallel Symposium-2 Neuroscience : Tissue-clearing techniques for visualization of neural connections: basic concepts and clinical implication	Chairs: 정 옹(카이스트) 선 응(고려의대 해부학교실)
		15:40-16:00	Rapid and efficient clearing of brain tissues for 3D imaging	선 응(고려의대 해부학교실)
		16:00-16:20	High throughput whole brain optical imaging	정웅규(울산과학기술대)
		16:20-16:40	Clinical translations of 3D brain imaging and connectomics	이향운(이화대의대)
	Convention Hall B (4F)	15:40-16:40	Oral Presentation-6 Stroke II	Chairs: 유경호(한림의대) 정슬기(전북의대)
		15:40-15:52	Morphology of susceptibility vessel sign (SVS) predicts clot fragility and recanalization	강동완(서울의대)
		15:52-16:04	Different features of anterior circulation and posterior circulation dissections	김민경(영남의대)
		16:04-16:16	Ischemic stroke in critically ill patients with malignancy	유정암(성균관대의대)
		16:16-16:28	Asymmetrical cerebral white matter hyperintensities and predilection of lacunar infarct	류위선(동국의대)
		16:28-16:40	Impact of the academic year-end changeover on stroke outcomes	원혜연(서울의료원)

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세부일정

1일차, 11월 13일 금요일

Session	Hall name	Time	Title	Chairs / Speakers
Parallel Symposium/ Oral Presentation	Convention Hall C (4F)	15:40-16:40	Oral Presentation-7 Demyelinating Disorders	Chairs: 김광국(울산의대) 김호진(국립암센터)
		15:40-15:52	Differences in the clinical and laboratory features according to the lesion length in non-infectious myelitis	좌승주(제주대의대)
		15:52-16:04	Autoantibodies and autoimmune co-morbidities in seropositive neuromyelitis optica spectrum disorder	이주경(울산의대)
		16:04-16:16	Enhanced immunosuppressive effect of regulatory B cells in neuromyelitis optica and multiple sclerosis; an illustrative report	조혜진(성균관대의대)
		16:16-16:28	Glycyrrhizic acid might reduce the NMO-IgG induced cell death by inhibition of the complement activation	천소영(서울의대)
		16:28-16:40	Neutralizing antibodies in Korean multiple sclerosis patients treated with Interferon-beta	현재원(국립암센터)
	Emerald Hall A (3F)	15:40-16:40	Oral Presentation-8 Sleep	Chairs: 윤창호(서울의대) 주은연(성균관대의대)
		15:40-15:52	Drug related alternation of the resting-state brain connectivity in restless legs syndrome	조용원(계명대의대)
		15:52-16:04	Clinical and polysomnographic characteristics of REM sleep dependent obstructive sleep apnea in Korean adults	김도의(순천향대의대)
		16:04-16:16	Quantitative test for primary restless legs syndrome using the current perception threshold test	강민성(계명대의대)
		16:16-16:28	Blood pressure improvement with continuous positive airway pressure is associated with evening-to-morning blood pressure variations in obstructive sleep apnea syndrome	한수현(울산의대)
		16:28-16:40	Assessment of cardiovascular risk burden in subjects with obstructive sleep apnea syndrome	황경진(경희대의대)
	Emerald Hall B (3F)	15:40-16:40	Oral Presentation-9 Dementia I	Chairs: 최경규(이화대의대) 박건우(고려대의대)
		15:40-15:52	A new blood based biomarker in Alzheimer's Disease: Self-standard measurement of monomeric forms of A β	노지훈(울산의대)
		15:52-16:04	Resting-state fMRI in amnesic mild cognitive impairment with and without Parkinson's disease	예병석(연세의대)
		16:04-16:16	Multilevel hierarchical mixed model for the prediction of longitudinal cognitive changes after ischemic stroke	임재성(서울의대)
		16:16-16:28	Progression of Korean mini-mental status examination score in Alzheimer's disease	한지영(서울의대)
		16:28-16:40	Changes of cognitive function in patients with mild to moderate Alzheimer's disease associated with and without white matter lesions after rivastigmine patch therapy: a multi-center, 24-week, prospective, open-label study (CAREER study)	박경원(동아대의대)

1일차, 11월 13일 금요일

Session	Hall name	Time	Title	Chairs / Speakers
Parallel Symposium/ Oral Presentation	Diamond Hall (3F)	15:40-16:40	Oral Presentation-10 Headache	Chairs: 정재면(인제의대) 김병건(울지의대)
		15:40-15:52	Serial assessment of cerebral blood flow correlates with disease course in migraineurs: a longitudinal follow-up study	이미지(성균관의대)
		15:52-16:04	Thalamocortical dysconnectivity in migraine without aura: a combined fMRI and DTI study	강성훈(고려의대)
		16:04-16:16	Relationship between physical activity and status of headache in episodic migraineurs using smartphone Applications-based electronic headache diary	조수진(한림의대)
		16:16-16:28	Insomnia in migraineurs is closely associated with anxiety and depression: a population-based study	주민경(한림의대)
		16:28-16:40	Headache attributed to acute pyelonephritis	이준원(인제의대)
편집위원회 Workshop	Flamingo	14:20-16:40	편집위원회 Workshop	Chair: 이상암(울산의대)
		14:20-15:20	Meta-analysis in a systematic review for interventional studies (중재 연구에 관한 체계적 고찰 관점에서의 메타분석) 1	이준영(고려의대 의학통계학교실)
		15:20-15:40	Coffee Break	
		15:40-16:40	Meta-analysis in a systematic review for interventional studies (중재 연구에 관한 체계적 고찰 관점에서의 메타분석) 2	이준영(고려의대 의학통계학교실)
Parallel Symposium	Convention Hall A (4F)	16:50-17:50	Parallel Symposium-3 Stroke I : Endovascular Therapy in Acute Ischemic Stroke	Chairs: 정진상(성균관의대) 허지희(연세의대)
		16:50-17:10	Endovascular trials in acute ischemic stroke since 1998: What we have achieved	홍근식(인제의대)
		17:10-17:30	How to select patients for endovascular therapy: Time-based and imaging-based selection	손성일(계명대의대)
		17:30-17:50	Treatment faster and treatment more: System implementation for endovascular therapy	차재관(동아대의대)
	Convention Hall B (4F)	16:50-17:50	Parallel Symposium-4 Muscle and Nerve : Small Fiber Neuropathy	Chairs: 이동국(대구가톨릭의대) 김승현(한양의대)
		16:50-17:10	Introduction of small fiber neuropathy	배종석(한림의대)
		17:10-17:30	Common and uncommon small fiber neuropathy	오지영(건국의대)
		17:30-17:50	Skin biopsy and small fiber neuropathy	손은혜(충남의대)

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1일차, 11월 13일 금요일

Session	Hall name	Time	Title	Chairs / Speakers
Parallel Symposium	Convention Hall C (4F)	16:50-17:50	Parallel Symposium-5 Headache : Recent Advances in Headache	Chairs: 오건세(을지의대) 주민경(한림의대)
		16:50-17:10	Antibodies against CGRP in migraine treatment	Alan M. Rapoport (UCLA, USA)
		17:10-17:30	Vestibular migraine	손종희(한림의대)
		17:30-17:50	Procedural approach in migraine treatment	문희수(성균관대의대)
	Emerald Hall A (3F)	16:50-17:50	Parallel Symposium-6 Epilepsy I : Principles of Anti-epileptic Drug (AED) Selection	Chairs: 이병인(인제의대) 홍승봉(성균관대의대)
		16:50-17:10	Pharmacology of AED	김성은(인제의대)
		17:10-17:30	Adverse effects of AED	김동욱(건국대의대)
		17:30-17:50	AED selection in newly diagnosed epilepsies	권오영(경상대의대)
	Emerald Hall B (3F)	16:50-17:50	Parallel Symposium-7 Movement Disorders : Miscellaneous Movement Disorders	Chairs: 하충건(인하의대) 박미영(영남의대)
		16:50-17:10	Paroxysmal dyskinesias	이지영(서울의대)
		17:10-17:30	Psychogenic movement disorders	백종삼(인제의대)
		17:30-17:50	Neurodegeneration with brain iron accumulation	이재혁(부산의대)
	Diamond Hall (3F)	16:50-17:50	Parallel Symposium-8 Neurocritical Care : Interesting Cases in Intensive Care Unit	Chairs: 이광수(가톨릭의대) 이준홍(국민건강보험 공단 일산병원)
		16:50-17:10	Severe stroke	홍지만(아주의대)
		17:10-17:30	Refractory seizure	강중구(울산의대)
		17:30-17:50	Reversible coma	유정암(성균관대의대 중환자의학과)

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2일차, 11월 14일 토요일

Session	Hall name	Time	Title	Chairs / Speakers
Update in Neurology/ Case-Based Learning/ Workshop	Convention Hall A (4F)	8:00-9:30	Update II Neurology Update II	Chairs: 임병훈(경상의대) 하정상(영남의대)
		8:00-8:15	Muscle and nerve	신하영(연세의대)
		8:15-8:30	Movement disorders	최성민(전남의대)
		8:30-8:45	Neurocritical care	고상배(서울의대)
		8:45-9:00	Neuro-otology	최정윤(고려의대)
		9:00-9:15	Demyelinating disorders	조종양(인제의대)
		9:15-9:30	Stroke	이용석(서울의대)
	Convention Hall B (4F)	8:00-9:30	Case-Based Learning	Chairs: 최진영(건국의대) 송홍기(한림의대)
		8:00-8:15	Sudden respiratory difficulty with hypoxemia in a patient with right pontine infarction	최재철(제주대의대)
		8:15-8:30	Genetic muscle disease	신진홍(부산의대)
		8:30-8:45	Seizures in the medical ICU	조양제(연세의대)
		8:45-9:00	Mild cognitive impairment due to Alzheimer's disease	심용수(가톨릭의대)
		9:00-9:15	Movement disorders	고성범(고려의대)
		9:15-9:30	Rare causes of dizziness	김현아(계명대의대)
	Emerald Hall A (3F)	8:00-9:30	Workshop I Neurology Video Round	Chairs: 김지수(서울의대) 안태범(경희의대) 김지현(고려의대)
		8:00-8:30	Movement disorders	권도영(고려의대)
		8:30-9:00	Neuro-otology	정성해(충남의대)
		9:00-9:30	Epilepsy and sleep disorders	송파멜라(인제의대)
	Emerald Hall B (3F)	8:00-9:30	Workshop II Interpretation of Neurological Tests	Chairs: 주인수(아주의대) 오지영(건국의대)
		8:00-8:15	Nerve conduction study/Electromyography	석정임(대구가톨릭의대)
		8:15-8:30	Evoked potentials	윤병남(인하의대)
8:30-8:45		Video nystagmography	이학승(원광의대)	
8:45-9:00		Transcraial doppler/Duplex scan	한상원(인제의대)	
9:00-9:15		Electroencephalography/Polysomnography	구대림(서울의대)	
9:15-9:30	Neuropsychological tests	문소영(아주의대)		

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구분	Hall name	Time	Title	Chairs
Parallel Symposium/ Oral Presentation	Convention Hall A (4F)	9:50-10:50	Parallel Symposium-9 Sleep : Under-recognized but important sleep issues in neurology	Chairs: 홍승봉(성균관대의대) 남현우(서울의대)
		9:50-10:10	Sleep disorders in patients with neuromuscular disorder	양광익(순천향의대)
		10:10-10:30	Sleep problems in patients with traumatic brain injury	김지현(단국대의대)
		10:30-10:50	Sleep and pain	조용원(계명대의대)
	Convention Hall B (4F)	9:50-10:50	Oral Presentation-11 Muscle and Nerve II	Chairs: 최병욱(성균관대의대) 성정준(서울의대)
		9:50-10:02	Clinical, pathologic, genetic features of collagen VI-related myopathy in Korea	이정환(연세의대)
		10:02-10:14	The STIM1 mutations without tubular aggregate and its pathogenicity	이종목(일본국립정신신경센터)
		10:14-10:26	Development of myasthenia gravis after thymectomy	이혜림(성균관대의대)
		10:26-10:38	Predictor for secondary generalization in late onset myasthenia gravis	김지선(고려의대)
		10:38-10:50	Identification of pathogenic/likely pathogenic variants in inherited muscular disorders by targeted next-generation sequencing	박형준(이화대의대)
	Convention Hall C (4F)	9:50-10:50	Oral Presentation-12 Dementia II	Chairs: 김병채(전남의대) 최성혜(인하의대)
		9:50-10:02	The synergistic effects of amyloid and subcortical cerebral small vessel disease on the progression of lobar microbleeds: three-year longitudinal study in patients with subcortical vascular mild cognitive impairment	김여진(성균관대의대)
		10:02-10:14	Regional comparison of PET imaging biomarkers in the striatum between early versus late-onset Alzheimer's disease	김지은(울산의대)
		10:14-10:26	Elevation of the plasma A β 40/A β 42 ratio as a diagnostic marker of sporadic early-onset Alzheimer's disease	신호식(순천향의대)
		10:26-10:38	Analysis of brain imaging and fluid biomarkers for Alzheimer's disease in Korean population	왕민정(서울의대)
		10:38-10:50	Relation between postural instability and subcortical volume loss in Alzheimer's disease	이현아(계명대의대)

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구분	Hall name	Time	Title	
Parallel Symposium/ Oral Presentation	Emerald Hall A (3F)	9:50-10:50	Oral Presentation-13 General neurology or other issue I	Chairs: 서만욱(전북의대) 최영철(연세의대)
		9:50-10:02	Paraneoplastic neurologic syndrome: associated antibodies and underlying neoplasm	변정익(서울의대)
		10:02-10:14	Oral intake of anti-hangover substance increases aldehyde dehydrogenase activity: new preventive and therapeutic potentials for oxidative neuronal injury?	강보승(한양대의대)
		10:14-10:26	Differences in intraoperative neurophysiological monitoring between spinal intramedullary ependymoma and hemangioblastoma	김동건(서울의대)
		10:26-10:38	Pattern of inpatient neurology consultation in Korean tertiary care hospital	박하늘(가천의대)
		10:38-10:50	Usefulness of intraoperative transcranial motor evoked potential for predicting acute postoperative neurological complication in patients with Aneurysmal subarachnoid hemorrhage	주병역(성균관대의대)
	Emerald Hall B (3F)	9:50-10:50	Oral Presentation-14 Stroke III	Chairs: 황성희(한림의대) 이승훈(서울의대)
		9:50-10:02	Association between grades of right-to-left shunt and TOAST subtypes of the ischemic stroke	이찬혁(전북의대)
		10:02-10:14	Trends in Risk factor prevalences among Young adults with ischemic stroke	하현욱(전남의대)
		10:14-10:26	The effect of high-intensity statin use in the acute phase after thrombolysis	김태경(울지의대)
		10:26-10:38	Preliminary evaluation of the brain saver: A novel pre-hospital stroke notification system using mobile application platform	오미선(한림의대)
		10:38-10:50	Longitudinal study on the associations between cognitive status and transcranial doppler parameters in patients with mild to moderate dementia	임재성(서울의대)
	Diamond Hall (3F)	9:50-10:50	Oral Presentation-15 Neuro-otology	Chairs: 성기범(순천향의대) 이형(계명대의대)
		09:50-10:00	Randomized trial on short-term efficacy of vibration and Gufoni maneuver for apogeotropic horizontal benign paroxysmal positional vertigo	김현아(계명대의대)
		10:00-10:10	The dizziness practice in South Korea: a National survey	정성해(충남의대)
		10:10-10:20	Acute transient vestibular syndrome due to cerebellar ischemia	최재환(부산의대)
		10:20-10:30	Canalith repositioning in apogeotropic horizontal canal benign paroxysmal positional vertigo: Do we need faster maneuvering?	김상훈(전남의대)
		10:30-10:40	Chasing dizzy chimera: Diagnosis of combined peripheral and central vestibulopathy	최서영(울지의대)
		10:40-10:50	Simultaneous recordings of cervical and ocular vestibular-evoked myogenic potentials	이찬혁(전북의대)

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구분	Hall name	Time	Title	
Presidential Lecture	Convention Hall A,B,C (4F)	11:00-11:50	Presidential Lecture	Chair: 윤병우(서울의대)
		11:00-11:50	Intracranial atherosclerosis: current status	Ka Sing Lawrence Wong (Chinese University of Hong Kong, Hong Kong)
Luncheon Symposium and Award	Convention Hall A,B,C (4F)	11:50-12:00	Luncheon Symposium and Awards Honorable Awards	Chairs: 서만욱(전북의대) 윤병우(서울의대)
		11:50-12:00	송파의학상	
			송파의학상	김주한(한양의대)
			명인학술상	서상원(성균관의대)
		12:00-12:50	JCN 논문상	윤성상(경희의대)
12:00-12:50	Luncheon Symposium	Chair: 박성호(서울의대)		
		12:00-12:50	Alzheimer's disease: an update on pathophysiology and outlook for treatment	Edward Koo (UCSD, USA)
신경계질환 우울 및 행동장애 연구회	Diamond Hall (3F)	12:40-14:10	신경계질환 우울 및 행동장애 연구회	Chairs: 홍승봉(성균관의대) 박건우(고려의대)
		12:40-13:20	신경계 질환 환자의 우울증 약물치료	한일우(대전시립제일노인전문병원)
		13:20-13:45	신경계 질환 환자의 피로 증상	김종성(울산의대)
		13:45-14:10	두통환자의 우울증과 불안증	박성패(경북의대)
Parallel Symposium/ Oral Presentation	Convention Hall A (4F)	14:20-15:20	Parallel Symposium-10 CNS Demyelinating Disorders : Pathogenesis of Multiple Sclerosis and Neuromyelitis Optica	Chairs: 김병준(성균관의대) 김병조(고려의대)
		14:20-14:40	The pathogenesis of multiple sclerosis: old and new players	권오현(을지의대)
		14:40-15:00	Mechanism of drugs for multiple sclerosis	박민수(영남의대)
		15:00-15:20	The relevance between neuromyelitis optica and AQP4-Ab	김성민(서울의대)
	Convention Hall B (4F)	14:20-15:20	Parallel Symposium-11 Neuro-intervention: Special Topics in Interventional Neurology	Chairs: 나정호(인하의대) 김응규(인제의대)
		14:20-14:40	Endovascular revascularization for basilar artery occlusion	이 준(영남의대)
		14:40-15:00	Rescue treatment option for intracranial artery occlusion	박희권(인하의대)
		15:00-15:20	Prevention of Ischemic complication in neurointervention	이진수(아주의대)

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구분	Hall name	Time	Title	
Parallel Symposium/ Oral Presentation	Convention Hall C (4F)	14:20-15:20	Oral Presentation-16 General neurology or other issue II	Chairs: 권기한(한림의대) 박기종(경상의대)
		14:20-14:32	Serum and CSF cytokine profiles in autoimmune encephalitis	변정익(서울의대)
		14:32-14:44	Cerebral and cardiovascular autoregulation during head-up tilt test in posturally related syncope patients	최지현(이화대의대)
		14:44-14:56	Autonomic parameters in postural orthostatic tachycardia syndrome with different onset time	김현아(계명대의대)
		14:56-15:08	Is the Valsalva maneuver can predict the response to head-up tilt table test?	이장준(대구파티마병원)
		15:08-15:20	Quantitative analysis of human cutaneous neurovascular system	손은희(충남의대)
	Emerald Hall A (3F)	14:20-15:20	Oral Presentation-17 Movement disorders II	Chairs: 김재우(동아의대) 전범석(서울의대)
		14:20-14:32	Causes of poor outcome of STN DBS in patients with Parkinson's disease	이종식(울산의대)
		14:32-14:44	Role of methylmalonic acid and peripheral neuropathy in idiopathic Parkinson's disease	박진성(경북의대)
		14:44-14:56	Alpha-synuclein in gastric and colonic mucosal tissue in Parkinson's disease: limited role as a biomarker	유호성(울산의대)
		14:56-15:08	Public knowledge and awareness about Parkinson's disease: A National Population based Survey in Korea	김지선(순천향의대)
		15:08-15:20	Postural sensory deficits correlates with gait freezing in Parkinson's disease	허영은(성균관의대)
	Emerald Hall B (3F)	14:20-15:20	Oral Presentation-18 Epilepsy II	Chairs: 신동진(가천의대) 손영민(가톨릭의대)
		14:20-14:32	Relationship between EEG findings and prognosis of post-hypothermia therapy	함주연(충남의대)
		14:32-14:44	Significance of thalamus in temporal lobe epilepsy with hippocampal sclerosis: Effective connectivity analysis	박강민(인제의대)
		14:44-14:56	Cortical and subcortical structural abnormalities in juvenile myoclonic epilepsy	임성철(가톨릭의대)
		14:56-15:08	Temporal lobe epilepsy with amygdalar enlargement: Its unique clinical characteristics and prognosis	이현조(가톨릭의대)
		15:08-15:20	Altered thalamocortical functional connectivity in juvenile myoclonic epilepsy: an fMRI study	김정빈(고려의대)

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구분	Hall name	Time	Title	Chairs
Parallel Symposium/ Oral Presentation	Diamond Hall (3F)	14:20-15:20	Oral Presentation-19 Stroke IV	Chairs: 김경문(성균관의대) 홍근식(인제의대)
		14:20-14:32	Degree of early recanalization and functional outcome in acute ischemic stroke with intravenous	장준영(서울의대)
		14:32-14:44	Use, time delay, and outcomes of drip-and-ship thrombolysis paradigm for patients with acute ischemic stroke; an analysis of the Clinical Research Center for Stroke-5th division registry	최재철(제주대의대)
		14:44-14:56	Is it safe to administer antithrombotic medications within 24 hours after recanalization treatments for acute ischemic stroke patients?	정한길(서울의대)
		14:56-15:08	Intra-arterial thrombolysis in acute ischemic stroke: Updated meta-analysis of randomized controlled trials	홍근식(인제대의대)
		15:08-15:20	Retinal artery occlusion is at an increased risk of subsequent ischemic stroke	홍정호(계명대의대)
Parallel Symposium	Convention Hall A (4F)	15:40-16:40	Parallel Symposium-12 Neuro-otology: International Classification of Vestibular Disorders (ICVD)	Chairs: 김지수(서울의대) 김병건(울지의대)
		15:40-15:55	Classification of vestibular symptoms	김지수(서울의대)
		15:55-16:10	Diagnostic criteria of BPPV	이승환(전남의대)
		16:10-16:25	Diagnostic criteria of Meniere's disease	최재환(부산의대)
		16:25-16:40	Diagnostic criteria of vestibular migraine	김병건(울지의대)
	Convention Hall B (4F)	15:40-16:40	Parallel Symposium-13 Dementia II: Neuroimaging in Alzheimer's Disease	Chairs: 한설희(건국대의대) 김상윤(서울의대)
		15:40-16:00	Amyloid imaging in Alzheimer's disease	노지훈(울산의대)
		16:00-16:20	Tau imaging in Alzheimer's disease	김병채(전남의대)
		16:20-16:40	Diffusion tensor imaging in Alzheimer's disease	양동원(가톨릭의대)
	Convention Hall C (4F)	15:40-16:40	Parallel Symposium-14 Epilepsy II: Morbidity and Mortality in People with Epilepsy	Chairs: 김재문(충남의대) 박수철(연세의대)
		15:40-16:00	Psychiatric comorbidities	박성파(경북의대)
		16:00-16:20	Medical comorbidities	김지연(대구가톨릭의대)
		16:20-16:40	Mortality in epilepsy	이상암(울산의대)

2일차, 11월 14일 토요일

구분	Hall name	Time	Title	
Parallel Symposium	Emerald Hall A (3F)	15:40-16:40	Parallel Symposium-15 Stroke II : Secondary Stroke Prevention in Atrial Fibrillation	Chairs: 김중성(울산의대) 이병철(한림의대)
		15:40-16:00	Pharmacology of anticoagulation	정슬기(전북의대)
		16:00-16:20	Clinical issues with warfarin use	송희정(충남의대)
		16:20-16:40	Practical use of non-vitamin K antagonist oral anticoagulants	구자성(가톨릭의대)
	Emerald Hall B (3F)	15:40-16:40	Parallel Symposium-16 INM: An Expansion to a New Horizon	Chairs: 김주한(한양의대) 강중구(울산의대)
		15:40-16:00	INM in scoliosis	류한욱(전북의대)
		16:00-16:20	INM in spastic cerebral palsy & tethered cord syndrome	이유빈(서울의대 재활의학과)
		16:20-16:40	Special issues : Evidence-based INM	구용서(고려의대)
	Diamond Hall (3F)	15:40-16:40	성공적인 신경과 개원방향	Chairs: 신현길(두신경과) 이태규(이태규신경과)
		15:40-16:00	노인병원	윤웅용(맑은수병원)
		16:00-16:20	신경통증 클리닉	한범기(연세신경과의원)
		16:20-16:40	신경과 클리닉	이일근(서울브레인신경과)
	치매특별 등급 교육	Emerald Hall B (3F)	17:00-18:00	치매특별등급 교육

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Stroke 1 (Eng)

001	Association between meteorological variables and acute stroke incidence in South Korea [2014년도 명인학술상 수상-연구 주제 발표]	서울의대 권형민
002	Risk factors for the development of depressed mood after stroke	가톨릭의대 김예림
003	Baseline characteristics and clinical outcomes of acute symptomatic internal carotid artery stumps	아주대의대 이성은
004	Changes in the common carotid artery after radiotherapy: Wall, Calcification and Atherosclerosis	울산의대 김범준
005	One-year effect of medical treatment in atherosclerotic middle cerebral artery stenosis measured by Transcranial Doppler	아주대의대 김 민

Neuroscience

001	Increased glucose uptake in the hemisphere with more severe ischemia among mice with right common carotid occlusion: luxury metabolism, not luxury perfusion	아주대의대 이진수
002	FUS abnormalities in ALS patient-derived induced neurons	한양의대 임수민
003	The emergence of an abnormal form of long-term potentiation in aged Alzheimer's disease mouse model	전남대의대 최성민
004	Characterization of white matter injury in a rat model of chronic cerebral hypoperfusion	건국대의대 김한영
005	Neuroprotective effects of acetyl-L-carnitine against oxygen-glucose deprivation-induced neural stem cells death via the PI3K pathway	한양의대 최호진

Movement Disorder 1

001	Neuropsychological tests and MCI subtypes more related to dementia in non-demented patients with Parkinson's disease	동아대의대 이수윤
002	The earliest findings of comfortable gait in de novo Parkinson's disease: gait hypokinesia and effect of levodopa	순천향대의대 권겸일
003	Neuropsychologic and radiologic comparisons in patients with parkinson's disease dementia in terms of onset age	연세의대 김영광
004	Retinal thinning correlates with clinical severity in multiple system atrophy	서울의대 이지영
005	The origin of T2* contrast in human Substantia Nigra: A Postmortem Validation Study at 7T MRI	부산의대 이재혁

Muscle and Nerve I

001	Pharmacokinetic parameters and the outcome of Guillain-Barré syndrome after intravenous immunoglobulin treatment : Preliminary report of DEMIAN (DElta seruM IgG As a Novel biomarker for treatment outcome of Guillain-Barré syndrome)	한림의대 배종석
002	Long-term clinical course and outcome of myasthenia gravis associated project with thymoma	선우엔조신경과 선우일남
003	Human SCN4A N440K zebrafish model of nondystrophic myotonia	전남의대 남태승
004	Prevalence and incidence of myasthenia gravis in Korea: nationwide population-based epidemiological study	연세의대 이형석
005	Identification of de novo variants by trio-based whole exome sequencing and functional analysis of candidate genes in Korean patients with sporadic ALS	성균관의대 김영은

Epilepsy I: Clinical Epileptology

001	Short and long-term mortality in status epilepticus	울산의대 이유진
002	Clinical efficacy and safety of levetiracetam in epilepsy	전남의대 고상준
003	Prognostic factors of status epilepticus to predict outcomes including seizure control and future development of epilepsy	이화대의대 김태경
004	Risk factors and clinical outcomes associated with seizures following open heart surgery	성균관대의대 서지혜
005	The HLA-A*2402/Cw*0102 haplotype is associated with lamotrigine-induced maculopapular eruption in the Korean population	서울의대 문장섭

Stroke II

001	Morphology of Susceptibility Vessel Sign (SVS) Predicts Clot Fragility and Recanalization	서울의대 강동완
002	Different features of anterior circulation and posterior circulation dissections	영남의대 김민경
003	Ischemic Stroke in Critically Ill Patients with malignancy	성균관대의대 유정암
004	Asymmetrical cerebral white matter hyperintensities and predilection of lacunar infarct	동국의대 류위선
005	Impact of the academic year-end changeover on stroke outcomes	서울의료원 원혜연

Demyelinating Disorders

001	Differences in the clinical and laboratory features according to the lesion length in non-infectious myelitis	제주대 좌승주
002	Autoantibodies and Autoimmune Co-morbidities in Seropositive Neuromyelitis Optica Spectrum Disorder	울산의대 이주경
003	Enhanced immunosuppressive effect of regulatory B cells in neuromyelitis optica and multiple sclerosis; an illustrative report	성균관의대 조혜진
004	Glycyrrhizic acid might reduce the NMO-IgG induced cell death by inhibition of the complement activation	서울의대 천소영
005	Neutralizing antibodies in Korean multiple sclerosis patients treated with Interferon-beta	국립암센터 현재원

Sleep

001	Drug related alternation of the resting-state brain connectivity in restless Legs Syndrome	계명대의대 조용원
002	Clinical and polysomnographic characteristics of REM sleep dependent obstructive sleep apnea in Korean adults	순천향의대 김도의
003	Quantitative test for primary restless legs syndrome using the current perception threshold test	계명대의대 강민성
004	Blood pressure improvement with continuous positive airway pressure is associated with evening-to-morning blood pressure variations in obstructive sleep apnea syndrome	울산의대 한수현
005	Assessment of cardiovascular risk burden in subjects with obstructive sleep apnea syndrome	경희의대 황경진

Dementia 1 (Eng)

001	A new blood based biomarker in Alzheimer's Disease: Self-standard measurement of monomeric forms of A β	울산의대 노지훈
002	Resting-state fMRI in amnesic mild cognitive impairment with and without Parkinson's disease	연세의대 예병석
003	Multilevel hierarchical mixed model for the prediction of longitudinal cognitive changes after ischemic stroke	서울의대 임재성
004	Progression of Korean Mini-Mental Status Examination (K-MMSE) score in Alzheimer's disease	서울의대 한지영
005	Changes of cognitive function in patients with mild to moderate Alzheimer's disease associated with and without white matter lesions after rivastigmine patch therapy: a multi-center, 24-week, prospective, open-label study (CAREER study)	동아대의대 박경원

Headache

001	Serial assessment of cerebral blood flow correlates with disease course in migraineurs: a longitudinal follow-up study	성균관대의대 이미지
002	Thalamocortical dysconnectivity in migraine without aura: a combined fMRI and DTI study	고려의대 강성훈
003	Relationship between physical activity and status of headache in episodic migraineurs using smartphone Applications-based electronic headache diary	한림의대 조수진
004	Insomnia in migraineurs is closely associated with anxiety and depression: a population-based study	한림의대 주민경
005	Headache attributed to acute pyelonephritis	인제의대 이준원

Muscle and Nerve II (Eng)

001	Clinical, pathologic, genetic features of collagen VI-related myopathy in Korea	연세의대 이정환
002	The STIM1 mutations without tubular aggregate and its pathogenicity	일본국립정신신경센터 이종목
003	Development of myasthenia gravis after thymomectomy	성균관대의대 이해림
004	Predictor for secondary generalization in late onset myasthenia gravis	고려의대 김지선
005	Identification of pathogenic/likely pathogenic variants in inherited muscular disorders by targeted next- generation sequencing	이화의대 박형준

Dementia II

001	The synergistic effects of amyloid and subcortical cerebral small vessel disease on the progression of lobar microbleeds: three-year longitudinal study in patients with subcortical vascular mild cognitive impairment	성균관대의대 김여진
002	Regional comparison of PET imaging biomarkers in the striatum between early versus late-onset Alzheimer's disease	울산의대 김지은
003	Elevation of the plasma A β 40/A β 42 ratio as a diagnostic marker of sporadic early-onset Alzheimer's disease	순천향의대 신호식
004	Analysis of brain imaging and fluid biomarkers for Alzheimer's disease in Korean population	서울의대 왕민정
005	Relation between postural instability and subcortical volume loss in Alzheimer's disease	계명대의대 이현아

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General Neurology or other issue I

001	Paraneoplastic neurologic syndrome: associated antibodies and underlying neoplasm	서울의대 변정익
002	Oral intake of anti-hangover substance increases aldehyde dehydrogenase activity: new preventive and therapeutic potentials for oxidative neuronal injury?	한양의대 강보승
003	Differences in intraoperative neurophysiological monitoring between spinal intramedullary ependymoma and hemangioblastoma	서울의대 김동건
004	Pattern of inpatient neurology consultation in Korean Tertiary Care Hospital	가천의대 박하늘
005	Usefulness of intraoperative transcranial motor evoked potential for predicting acute postoperative neurological complication in patients with Aneurysmal subarachnoid hemorrhage	성균관대의대 주병익

Stroke III

001	Association between grades of right-to-left shunt and TOAST subtypes of the ischemic stroke	전북의대 이찬혁
002	Trends in risk factor prevalences among young adults with ischemic stroke	전남의대 하현욱
003	The effect of high-intensity statin use in the acute phase after thrombolysis	을지의대 김태경
004	Preliminary evaluation of the brain saver: a novel pre-hospital stroke notification system using mobile application platform	한림의대 오미선
005	Longitudinal study on the associations between cognitive status and transcranial doppler parameters in patients with patients with mild to moderate dementia	서울의대 임재성

Neuro-otology

001	Randomized trial on short-term efficacy of vibration and Gufoni maneuver for apogeotropic horizontal benign paroxysmal positional vertigo	계명대의대 김현아
002	The dizziness practice in South Korea: a National Survey	충남의대 정성해
003	Acute transient vestibular syndrome due to cerebellar ischemia	부산의대 최재환
004	Canalith repositioning in apogeotropic horizontal canal benign paroxysmal positional vertigo: do we need faster maneuvering?	전남의대 김상훈
005	Chasing dizzy chimera: diagnosis of combined peripheral and central vestibulopathy	을지의대 최서영
006	Simultaneous recordings of cervical and ocular vestibular-evoked myogenic potentials	전북의대 이찬혁

General Neurology or other issue II

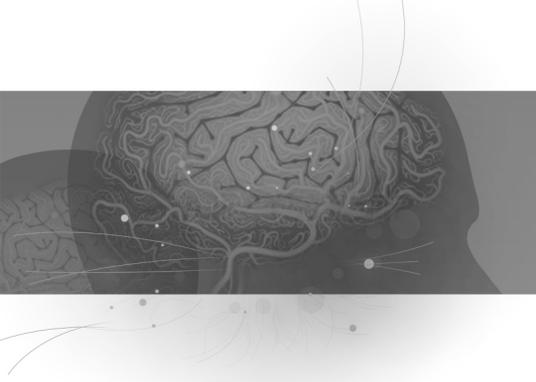
001	Serum and CSF cytokine profiles in autoimmune encephalitis	서울의대 변정익
002	Cerebral and cardiovascular autoregulation during head-up tilt test in posturally related syncope patients	이화의대 최지현
003	Autonomic parameters in postural orthostatic tachycardia syndrome with different onset time	계명대의대 김현아
004	Is the valsalva maneuver can predict the response to head-up tilt table test?	대구파티마병원 이장준
005	Quantitative analysis of human cutaneous neurovascular system	충남의대 손은희

Movement Disorder II (Eng)

001	Causes of poor outcome of STN DBS in patients with Parkinson's disease	울산의대 이종식
002	Role of methylmalonic acid and peripheral neuropathy in Idiopathic Parkinson's disease	경북의대 박진성
003	Alpha-synuclein in gastric and colonic mucosal tissue in Parkinson's disease: limited role as a biomarker	울산의대 유호성
004	Public knowledge and awareness about Parkinson's disease: a national population based survey in Korea	순천향의대 김지선
005	Postural sensory deficits correlates with gait freezing in Parkinson's Disease	성균관대의대 허영은

Epilepsy II

001	Relationship between EEG findings and prognosis of post-hypothermia therapy	충남의대 함주연
002	Significance of thalamus in temporal lobe epilepsy with hippocampal sclerosis: effective connectivity analysis	인제의대 박강민
003	Cortical and subcortical structural abnormalities in juvenile myoclonic epilepsy	가톨릭의대 임성철
004	Temporal lobe epilepsy with amygdalar enlargement: its unique clinical characteristics and prognosis	가톨릭의대 이현조
005	Altered thalamocortical functional connectivity in juvenile myoclonic epilepsy: an fMRI study	고려의대 김정빈



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Stroke IV

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|-----|--|-----------|
| 001 | Degree of early recanalization and functional outcome in acute ischemic stroke with intravenous thrombolysis | 서울의대 장준영 |
| 002 | Use, time delay, and outcomes of drip-and-ship thrombolysis paradigm for patients with acute ischemic stroke; an analysis of the Clinical Research Center for Stroke-5th division registry | 제주대의대 최재철 |
| 003 | Is it safe to administer antithrombotic medications within 24 hours after recanalization treatments for acute ischemic stroke patients? | 서울의대 정한길 |
| 004 | Intra-arterial thrombolysis in acute ischemic stroke: updated meta-analysis of randomized controlled trials | 인제의대 홍근식 |
| 005 | Retinal artery occlusion is at an increased risk of subsequent ischemic stroke | 계명대의대 홍정호 |

001	Comparison of normal value between transcranial color-coded Doppler sonography and transcranial Doppler sonography in Korean healthy population	고려의대 표선중
003	The correlation of SOD1 (rs1041740), SOD2 (rs4880), GPx1 (rs1050450), GPx4 (713041) SNPs with Ischemic stroke	전북대학교 의생명연구소 야다브 비노드 쿠마
004	Clinical and radiological characteristics in acute cerebral infarction with and without cerebral aneurysm	한림의대 이채영
005	The association between brachial-ankle pulse wave velocity and perivascular space topography in stroke patients	중앙의대 박무석
006	Tremor due to left middle cerebral artery occlusion	울산의대 김현진
007	A case of posterior spinal cord infarction presenting pure sensory disturbance	대구파티마병원 이상래
008	A case of spontaneous middle cerebral artery dissection with distal internal carotid artery involvement confirmed by high-resolution MRI	경북의대 전지수
009	Intracranial venous reflux caused by occlusion of brachiocephalic vein mimicking intracranial dural arteriovenous fistula	서남의대 명지병원 이수빈
010	Bilateral pontine infarction with branch atheromatous plaque	인제의대 이준원
011	Sinking skin flap syndrome after decompressive craniotomy	제주대의대 좌승주
012	Comparative outcome and pathomechanism in acute stroke model after hypoxia and exercise	서울의대 김태준
013	Associations among disruption of cholinergic pathways, cholinergic-innervated cortical/subcortical volumes, and cognitive function in Alzheimer's disease with vascular factors: Methodologic aspects and preliminary results	서울의대 임재성
014	Post-stroke delirium in acute stroke care unit	아주의대 임태성
015	Tachycardia burden in stroke unit is associated with functional outcome after ischemic stroke	서울의대 정한길
016	Impact of cardiac function on outcome after ischemic stroke in patients with atrial fibrillation	성균관대의대 신종화
017	Bilateral substantia nigra lesions on MRI in a patient who presented with abulia	계명대의대 유수연
018	Isolated proximal lower extremity weakness in a patient with small cortical hemorrhage	고신대의대 채희운
019	A case of anterior spinal cord infarction after bronchial artery embolization	단국대의대 류세열
020	A case of hemichorea following parieto-temporal infarction sparing basal ganglia	인하대의대 오단아
022	Dural arteriovenous fistula presenting as a seizure mimicking transient ischaemic attack: Advantages of susceptibility-weighted imaging	경희대의대 이지훈
023	A case of posterior circulation infarction associated with spinal cord infarction	가톨릭대의대 강일웅
024	The comparison of characteristics between Solitaire stent and Trevo stent in Mechanical thrombectomy	인제의대 김정민
025	Intravenous thrombolysis with recombinant tissue plasminogen activator for ischemic stroke patients over 80 years old	CHA의대 김종욱
026	Predicting prognosis of mechanical thrombectomy by using the modified DRAGON score	성균관대의대 김근현

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027	Prehospital notification using emergency medical service is an optimal strategy to reduce the time to thrombolytic therapy after acute ischemic stroke	동아의대 심동현
028	Efficacy of proximal aspiration thrombectomy for using balloon-tipped guide catheter in acute intracranial internal carotid artery occlusion	경북의대 최동호
029	Usage of rt-PA for the acute ischemic stroke patients with aortic dissection	고려의대 권예지
030	Stent edge occlusion with shortening of the previously implanted carotid stent in a patient with hyperacute ischemic stroke	영남의대 김민경
031	A case of carotid dissection in hyperacute ischemic stroke	울지의대 김재국
032	Post coil-embolization related ischemic stroke: how to prevent it?	고려의대 권예지
033	Multiple embolic infarctions due to a primary aortic intimal sarcoma	서울의대 권형민
034	Lateral medullary infarction with headache and autonomic dysfunction	고신대의대 이지훈
035	Unilateral medial thalamic infarction without midbrain involvement presenting as vertical gaze palsy	고신대의대 이지훈
036	Clinical characteristics according to histologically-confirmed thrombus composition in hyperacute ischemic stroke	아주의대 이규선
037	Endovascular thrombectomy in acute ischemic stroke patients with early neurologic deterioration	인제의대 서정화
038	Uric acid consumption in the patients after intraarterial thrombolysis	서울의대 김도연
039	Futile Recanalization and predicted Therapeutic gain by Initial Stroke Severity after Endovascular treatment	서울의대 이상화
040	The incidence and mechanism of neurological deterioration after endovascular thrombectomy	중앙의대 김정민
041	Initial factors affecting the clinical outcome after successful recanalization via MR based mechanical thrombectomy in patients with acute ischemic stroke due to basilar artery occlusion	경상의대 김민정
042	Acute ischemic patient with the hyperintense acute reperfusion marker (HARM) after spontaneous recanalization	경상의대 홍지호
043	Retrieved thrombus in two patients with embolic stroke underlying atrial fibrillation with a proper INR level: platelet- dominant white clot?	아주의대 김민승
044	A case of cerebral venous thrombosis related to iron deficiency anemia and increased factor VIII activity	고려의대 우연선
045	Possible stress cardiomyopathy after carotid artery stenting	울지의대 조남주
046	Cerebral venous infarction in mount everest	건국대의대 김정희
047	A case report of fibromuscular dysplasia in the posterior cerebral artery	충북의대 여민주
048	Prevention Strategies for Stroke in Korean Adults: the prevention of stroke and dementia survey 2007-2009	조선의대 강현구
049	Carotid artery calcification predicts carotid bulb atherostenosis	대구파티마병원 이상래
050	The role of signal intensity ratio on FLAIR MRI in acute ischemic stroke patients with endovascular treatment	서울의대 김태훈
051	The correlation of aphasia with cortical lesion in diffusion-weighted image	울산의대 류은화

052	Dabigatran effect on left ventricular thrombus in a patient with acute ischemic stroke and atrial fibrillation	인제의대 정규윤
053	Use of new oral anticoagulants in a patient with recurrent cerebral venous thrombosis and spontaneous intracranial hemorrhage	충남의대 정혜선
054	Anticoagulant therapy can eliminate fresh thrombus in the internal carotid artery: a case report	원광의대 황윤수
055	A case of splenic infarction associated with positive anticardiolipin antibody	한림의대 최취철
056	Primary local tirofiban infusion and interventional treatment in internal carotid artery stump causing an acute ischemic stroke and early progression	아주의대 이규선
057	A case of cerebral infarction due to artery to artery embolization from calcified plaque of ipsilateral internal carotid artery	전북의대 나성인
058	A case of right parietal infarction presenting as Gerstmann's syndrome	국립중앙의료원 박혜성
059	A Case of bilateral thalamic infarction with Pulmonary Arteriovenous Malformation	전북의대 왕수정
060	D-dimer as a risk factor for END in cryptogenic stroke with cancer	서울의대 남기웅
061	Low plasma proportion of omega 3-polyunsaturated fatty acids determine cerebral small vessel disease in acute ischemic stroke patients	이화의대 정아름
062	Polycythemia Vera as a risk factor of borderzone infarction	한양의대 이종민
063	Relation between glycoalbumin level and acute ischemic stroke	가천의대 박하늘
064	Risk factors of carotid artery calcification	대구파티마병원 박종완
065	Cardiac myxoma: another cause of transient global amnesia? – heart and mind	가톨릭의대 이시백
066	Subarachnoid hemorrhage in patients with systemic lupus erythematosus	한양의대 김용성
067	Brainstem cavernous malformation with ipsilateral abducens nerve palsy with good outcome after conservative treatment	한양의대 김승재
068	A 60-year-old woman with recurrent stroke-like episodes : what is the standard range of diagnostic work-up for cryptogenic strokes?	서울의대 유달라
069	An unusual case of lateral medullary infarction initially presenting as isolated vertigo and magnetic gait preceding change of diffusion weighted MR imaging	성균관의대 남궁동욱
070	A case of midbrain infarction causing isolated fourth nerve palsy	한양의대 한석길
071	A woman with transient left side motor weakness with systemic lupus erythematosus	가천의대 강민주
072	Decreased emotion recognition is associated with impaired activity of daily living in the patient with early Alzheimer's disease	경북의대 고판우
073	The combined influence of vascular risk factors on cognitive decline among community-dwelling elderly in Seoul	이화의대 김건하
074	2 cases of adult-onset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP) diagnosed by CSF1R gene mutation	울산의대 박계원
075	Predictive factors for objective cognitive impairment in a screening population with subjective cognitive impairment	서울의대 김민정
076	A comparison of the performances on the K-VCiHS-NP according to the CDR-SB in vascular MCI, amnesic MCI, vascular dementia, and Alzheimer's disease	한림의대 김상순
077	Quantification of perivascular drainage in mouse cerebral cortex and its role in Alzheimer disease	한국과학기술원 김신흔

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078	A Case of Creutzfeldt-Jakob disease as progressive nonfluent aphasia	대구파티마병원 박종완
079	Effects of apolipoprotein E4 on progression of amyloid and cortical thinning in amnesic mild cognitive impairment patients: a three-year longitudinal study	성균관의대 김여진
080	A study of questionnaires about the dementia awareness and relative factors in community dwelling healthy adults	원광의대 김영서
081	The factors related with acquisition of grades in the long-term care service in demented patients	국민건강보험공단 일산병원 김종현
082	A case of reversible neurologic manifestations and Parkinsonism caused by vitamin B12 deficiency	연세의대 백민렬
083	Survival in patients with dementia who are in the institutions	국민건강보험공단 일산병원 김종현
084	Clinical and neuropsychological differences in patients with cognitive decline of Alzheimer's type stratified by positive vs. negative amyloid PET status	울산의대 김지은
085	Serial magnetic resonance imaging changes in a patient with Gerstmann-Sträussler-Scheinker Syndrome (P102L)	경희의대 송종민
086	Amyloid beta-weighted cortical thickness : five distinctive relationships between Amyloid beta and cortical thinning in Alzheimer's disease	서울아산병원 김찬미
087	Visual rating of posterior atrophy as a marker of progression to dementia in mild cognitive impairment patients	서울의대 김항래
088	Distinctive cognitive trajectories related to amyloid and cerebrovascular disease in patients with mild cognitive impairment	성균관의대 김희진
089	Dementia progression in anatomical subtypes of Alzheimer's Disease: malignant progression in parietal dominant atrophy subtype regardless of onset age	가천의대 노 영
090	Atypical Wernicke's encephalopathy involving frontal lobe during parenteral nutrition	한양의대 성원재
091	The pattern of brain iron accumulation of vascular dementia and Alzheimer's dementia using quantitative susceptibility mapping imaging	건국의대 문연실
092	In vivo evaluation of neurofibrillary tangles in Alzheimer's disease using [18F]THK5351 PET	가천의대 노 영
093	Memory complaints in subjective cognitive impairment, mild cognitive impairment and Alzheimer's disease	가톨릭의대 류선영
094	Hashimoto's encephalopathy presenting with dementia and relapsing mental change	성균관의대 손우현
095	The steadiness of neuronal integrity in the recurrent attack of transient global amnesia	건국의대 문연실
096	Predictors of Institutionalization in longitudinal follow-up of Patients with Alzheimer Disease	아주의대 박동규
097	Spontaneous ventriculostomy in a patient with normal pressure hydrocephalus	가천의대 김갑수
098	Different normal EEG variation between TGA patients and normal healthy group	중앙의대 박무석
099	Internal structure of Computer Assessment of Memory and Cognitive Impairment (CAMCI)	고려의대 박윤아
100	Supernumerary phantom limb in patient with right basal ganglia hemorrhage	서울의대 김항래
101	Factors associated with depression and anxiety in caregivers for demented patients	대구가톨릭의대 박정아

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102	Diagnostic accuracy of entorhinal cortical volume, hippocampal volume, and fractional anisotropic value of hippocampus in mild cognitive impairment	가톨릭의대 양동원
103	Neuroimaging prognostic factors in patients with delayed post-hypoxic leukoencephalopathy after high dose corticosteroid therapy	동아의대 손상욱
104	The association between body mass index and cognitive decline in patients with small vessel disease - preliminary study	가톨릭의대 신혜은
105	The influence of the subcortical ischemia and cognitive patterns and their changes in normal hospital visited elderly	서울의대 안상준
106	Entorhinal cortex, earlier structural change than the hippocampus in subjective memory impairment	가톨릭의대 양동원
107	Marchiafava-Bignami disease with callosal and extracallosal lesions documented by diffusion tensor imaging	전남의대 김지민
108	Practical age difference cut-offs for T1-axial medial temporal atrophy visual rating scales in clinical diagnosis of Alzheimer's disease: CREDOS Data	이화대의대 최경선
109	Sleep influences the cognitive functions in mild to moderate dementia with Alzheimer's disease	서남의대 명지병원 한현정
110	Prediction of AD pathophysiology based on cortical thickness patterns	울산의대 황지혜
111	Systolic blood pressure variability relates to microstructural changes in white matters and cortical atrophy in cerebral small vessel disease	인하의대 최성혜
112	The differences of caregiver burden between the young old and the oldest old Alzheimer's disease patients	건국대의대 한설희
113	Autopsy confirmed case of frontotemporal dementia with motor neuron disease	부산의대 오은혜
114	The effect of group musical therapy on cognitive function in patients with probable Alzheimer's disease	서남의대 명지병원 한현정
115	Preoperative cerebrospinal fluid biomarkers and MRI findings in patients with idiopathic normal pressure hydrocephalus showing favorable surgical outcome	울산의대 홍윤정
116	Neuropathologic changes in two patients with advanced dementia	이화대의대 최경선
117	Clinical implication of A β accumulation in occipital lobes using [18F]-Florbetaben PET	울산의대 황지혜
118	Neurotoxoplasmosis presented with gait disturbance and cognitive impairment	한림의대 오학주
119	Protective effects of choline alfoscerate (L-alpha-glycerolphosphorylcholine, α -GPC) on seizure-induced neuronal death and cognitive impairment	한림의대 최휘철
120	Safety and efficacy of zonisamide in patients with epilepsy: a result of post-marketing surveillance study	건국대의대 이혜진
121	Uric acid change is a valuable marker to reflect refractory status epilepticus	아주대의대 최준영
122	Emboic stroke related to bradyarrhythmia after intravenous infusion of fosphenytoin	계명대의대 강민성
123	Transient Blood-Brain Barrier disruption after cerebral concussion which showed good prognosis	원광의대 황윤수
124	Posterior Leukoencephalopathy with Reversible Cerebral Vasoconstriction Syndrome after Blood transfusion; a case report	부산의대 채송화
125	Intracranial cortical calcifications in a epilepsy patient with pseudohypoparathyroidism	성균관대의대 김예슬
126	Diffuse hemispheric edema in an epilepsy patient	경상대의대 남원식

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127	Reversible splenial lesion syndrome following sudden withdrawal of antiepileptic drugs	건양외대 김재운
128	Myoclonic status epilepticus in adult patients without a previous history of epilepsy: a report of three cases	국립중앙의료원 김현경
129	Seizure disorder concurrent with acute ischemic stroke: single hospital experience	CHA외대 윤승재
130	Correlation of EEG and MRI in Sporadic Creutzfeldt-Jakob disease	CHA외대 임병수
131	Efficacy, tolerability, and pharmacokinetics of fosphenytoin loading in patients with subarachnoid hemorrhage	건국외대 김정희
132	A case of wernicke encephalopathy presenting subclinical seizure	대구파티마병원 박종완
133	A case of recurrent postictal bilateral facial petechiae without subconjunctival hemorrhage	대구파티마병원 박종완
134	Focal non-convulsive status epilepticus manifested as an antegrade amnesia	충남외대 임영기
135	Positive sharp waves in the Electroencephalography of Adult patient with Posterior Reversible Encephalopathy Syndrome	CHA외대 신정원
136	A case of status epilepticus after locoregional anesthesia techniques with lidocaine	영남외대 박재건
137	Intermittent lorazepam injection with magnesium infusion as alternative therapeutic strategy in refractory status epilepticus: a case report	아주외대 최준영
138	Usefulness of perfusion MRI to monitor encephalopathy of presumed autoimmune etiology: a case report	아주외대 최준영
139	Cerebral endothelial dysfunction in posterior circulation is related with migraine chronification	성균관외대 이미지
140	Subtypes and comorbidity of chronic daily headache in the outpatient department of a tertiary hospital	충북외대 김인하
141	Botulinum toxin a for chronic migraineurs: a single center experience	성균관외대 남궁동욱
142	Validation of the Patient Health Questionnaire-9 (PHQ-9) and PHQ-2 in patients with migraine	경북외대 이건희
143	A case of transient global amnesia during a migraine without aura attack	한림외대 임선영
144	Closure of patent foramen ovale in the patient with stroke and chronic intractable migraine	전북외대 이찬혁
145	A case of status migrainosus with thyroiditis	대구가톨릭외대 백종규
146	Reversible splenial lesion of the corpus callosum in migraine with aura	대구파티마병원 이효민
147	A case hemicrania continua related to an underlying lung neoplasm	제주외대 고근혁
148	Prolonged hemiparesis in sporadic hemiplegic migraine; resolution after steroid	원광외대 김지웅
149	A case of typical aura without headache presenting repetitive transient visual symptom	연세의대 김한결
150	Zonisamide-Responsive SUNCT syndrome	원광외대 임선재
151	Markers predicting treatment outcome of epidural blood patch in patients with spontaneous intracranial hypotension: a clinico-radiological study	성균관외대 최한나
152	Differences of central facilitation between episodic and chronic migraine in nociceptive-specific trigeminal pathways	한림외대 손종희
153	A case of cerebrospinal fluid volume depletion with spine MRI	을지의대 윤수진

154	Idiopathic trigeminal sensory neuropathies showing improvement of Brain MRI abnormalities after steroid treatment	대구가톨릭의대 김민석
155	Posterior Reversible Encephalopathy Syndrome (PRES) probably due to Leuprolide acetate	한림의대 이채영
156	A case of secondary headache attributed to retropharyngeal lymphadenopathy: an uncommon condition mimicking meningitis	분당제생병원 김병수
157	A case of intracranial hypotension following acupuncture	원광의대 황윤수
158	A case of trigeminal neuralgia caused by arteriovenous malformation in cerebellopontine angle	순천향의대 최나리
159	IgG4-related sclerosing disease presenting as intractable unilateral trigeminal neuralgia	단국의대 류세열
160	CNS involvement of granulomatosis of polyangiitis presenting as SIH mimic headache	성균관의대 정영희
161	Giant-cell temporal arteritis in 80-year-old female presenting visual manifestation	가천의대 강민주
162	Painful neuralgia of C2-3 followed by herpes zoster infection at trigeminal nerve distribution: two case reports	한림의대 이민우
163	Olfactory dysfunction is related to postoperative delirium in Parkinson Disease	아주의대 윤정환
164	Overview of the Parkinson's disease smell and taste study	순천향의대 백지훈
165	Olfactory deficits in the cognitive impaired de novo patients with Parkinson's disease	인제의대 임진희
166	Does depression in parkinson's disease contribute the pattern of striatal dopamine depletion?	연세의대 이윤주
167	Olfactory dysfunction in Parkinson's disease may be associated with the central cholinergic system	충남의대 오응석
168	Therapeutic singing activities to the vocal quality and the depression in Parkinson's disease: Case series	이화대의대 한은영
169	Cranio-cervical myoclonus with reversible bilateral dentate nucleus lesion by metronidazole toxicity	한림의대 이현창
170	Chorea and parkinsonism in a patient with systemic lupus erythematosus	아주의대 김 민
171	Hereditary geniospasm in a Korean family	서남의대 명지병원 엄관희
172	Primary lingual dystonia induced by speech	가톨릭의대 이종윤
173	PSP-like Syndrome without prominent ocular motor abnormality after aortic aneurysm repair	경희의대 이지훈
174	The usefulness of quantitative autonomic function test for the differentiation of multiple system atrophy from idiopathic Parkinson's disease	순천향의대 김새로미
175	Gait analysis of PD patients with or without FOG – comparison with healthy control	동아의대 이수윤
176	An application of smartphone tapper for assessment of bradykinesia in Parkinson's disease: A pilot study	한림의대 이채영
177	Central cholinergic dysfunction could be associated with oropharyngeal dysphagia in early Parkinson's Disease	울산의대 장우영
178	Association of body mass index and the depletion of nigrostriatal dopamine in Parkinson's disease	연세의대 이재정

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179	Validation of the Seoul-instrumental activity daily living in the detection of dementia in Parkinson disease	인제의대 정은주
180	Two cases of Parkinson's disease accompanied by retinitis pigmentosa	대구가톨릭의대 김신엽
181	A rare complication of rasagiline, acute cholecystitis, a case report	가톨릭의대 이양현
182	Clebopride-induced parkinsonism	한림의대 강석운
183	Delayed parkinsonism after treatment of dural arteriovenous fistula	아주의대 이선민
184	Normal dopamine transporter imaging in a spinocerebellar ataxia type 17 with parkinsonism	서울의대 김경준
185	Imaging of nigrosome 1 at 3T MRI for distinguishing drug-induced Parkinsonism from Idiopathic Parkinson's Disease	가천의대 성영희
186	Does the pattern of striatal dopamine depletion contribute to apathy in Parkinson's disease?	연세의대 정수진
187	The impact of prolonged temporal discrimination threshold on kinematic parameters of finger tapping and dexterous finger movements of Parkinson's disease	부산의대 이명준
188	Gender difference in depletion of presynaptic nigrostriatal dopamine in de novo Parkinson's disease	연세의대 이재정
189	HMPAO SPECT study of cerebral perfusion in Parkinson's disease with depression and Major depression disorder	가톨릭의대 김영도
190	Analysis of White Matter integrity in Parkinson's disease	강원의대 이승환
191	Incomplete recovery and minimal dopamine transporter decrease in drug-induced parkinsonism	연세의대 홍진용
192	Creutzfeldt-Jacob disease with gait disturbance as an initial manifestation mimicking progressive supranuclear palsy	고려의대 김진희
193	A case of cerebellar dysfunctions in MSA-C and PSP-C	한양의대 류창현
194	Freezing of gait in extrapontine myelinolysis	경희의대 이도경
195	Coexistence of ocular neuromyotonia and hemifacial spasm	서울의대 김경준
196	Primary central nervous system lymphoma: clinical experience of a single institution	서울의대 손성연
197	Analysis of normal appearing white matter in multiple sclerosis using myelin water imaging	국립암센터 정인혜
198	Treatment outcomes with rituximab in 100 patients with neuromyelitis optica: influence of FCGR3A polymorphism on the therapeutic response to rituximab	국립암센터 김수현
199	Clinical characteristics of disabling attack at onset in patients with neuromyelitis optica spectrum disorder	성균관의대 석진명
200	Clinical and radiologic characteristics of Alexander disease: A single center experience	전남의대 남태승
201	A case of Chronic Lymphocytic Inflammation with Pontine Perivascular Enhancement Responsive to Steroids (CLIPPERS)	성균관의대 김주현
202	Cerebral Salt Wasting Syndrome in a Patient with Neuromyelitis Optica Spectrum Disorder	인제의대 백영민
203	A case of Sjögren syndrome involving central nervous system with phonic tic	대구가톨릭의대 김신엽
204	Multiple Sclerosis diagnosed by pain and paresthesia shortly after Cervical trauma	중앙보훈병원 한상우

205	Non-traumatic spinal cord infarction after surfing – a case of surfer’s myelopathy	울산의대 박계원
206	A case of acute disseminated encephalomyelitis following pneumococcus vaccination in adult	한양의대 곽현승
207	Neuromyelitis optica spectrum disorder associated with syphilis: case study	충남의대 이주헌
208	Etiology of spontaneous downbeat nystagmus	서울의대 김기태
209	Diurnal variation of upbeat nystagmus: Is this the gravity effect?	부산의대 김승주
210	Assessment of vestibular recovery using video head impulse test after vestibular neuritis	순천향의대 김준현
211	A case of inferior vestibular neuritis shown at video head impulse test	순천향의대 김준현
212	Persistent otolith dysfunction even after successful repositioning in benign paroxysmal positional vertigo	전북의대 나성인
213	Reduced resting-state functional connectivity in the vestibular cortical network in normal aging	전북의대 나성인
214	Positional nystagmus in lateral semicircular canal fistula	대구가톨릭의대 박재한
215	Perverted head-shaking nystagmus; a few examples of peripheral vestibular disorder	전북의대 양태호
216	Changes in resting-state functional magnetic resonance imaging in vestibular neuritis	전북의대 양태호
217	Analysis of vestibulo-ocular reflex in patients with cerebellar ataxia using video head impulse tests	전남의대 김성식
218	Modulation of nystagmus by vision, proprioception, and efference copy signals: a systematic study	고려의대 최정윤
219	X-linked adrenoleukodystrophy presenting with positional downbeat nystagmus	전남의대 조관열
220	Variants of windmill nystagmus	부산의대 최광동
221	Perverted Head-shaking nystagmus in central lesions: characteristics and mechanism	고려의대 최정윤
222	Vitamin D status in patients with myasthenia gravis: a pilot study	제주의대 강사윤
223	Clinical and pathologic features of point mutations in Dystrophinopathy	연세의대 황희원
224	Ultrasound helps discriminating true carpal tunnel syndrome in diabetic polyneuropathy	동국의대 이민오
225	High serum B-cell activating factor levels in myasthenia gravis	제주의대 강사윤
226	The overlap of Arnold-Chiari malformation type 1 in a patient with Charcot-Marie-Tooth disease type 2D	전남의대 강경욱
227	Facial spasm associated with neuromyelitis optica spectrum disorder	제주의대 고근혁
228	A case of ocular myasthenia gravis initially presenting with sudden onset complete third nerve palsy	한양의대 곽현승
229	Hoffmann’s Syndrome: a rare form of hypothyroid myopathy	고려의대 권예지
230	A case of lumbosacral plexopathy and avascular necrosis in carbon monoxide(CO) poisoning	서울의대 김봉제
231	A case of late-onset mitochondrial myopathy	강원의대 김성근
232	Subacute sensory-dominant thoracic polyradiculopathy associated with anti-GM1 IgM antibody	가톨릭의대 김용방

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233	Guillain-Barré syndrome is not always presented with ascending and symmetric quadriparesis: A finger drop pattern of acute motor axonal neuropathy as a prominent variant form	동아의대 윤별아
234	Is decrement response at limb muscles on repetitive nerve stimulation the risk factor for generalization of ocular myasthenia gravis?	연세의대 김기훈
235	Steroid sparing effect of Tacrolimus in Myasthenia Gravis	연세의대 김윤희
236	Diabetes mellitus exacerbates the clinical and electrophysiological features of Guillain-Barré syndrome	한림의대 유진혁
237	Accelerated neuropathy of renal failure	충북의대 김인하
238	Meralgia paresthetica caused by obturator hernia	건양외대 김재윤
239	Ataxic form of Guillain-Barre syndrome associated with anti GD1b antibody-a case report of subacute stage	CHA의대 김종욱
240	A case of paraneoplastic myopathy related to very rapidly progressive malignant lymphoma	서울의대 김준순
241	Acute motor axonal neuropathy in association with Graves' disease	건양외대 김재윤
242	Respiratory failure as an initial symptom of amyotrophic lateral sclerosis	강원의대 김진수
243	A case of Guillain-Barre Syndrome and myositis associated with preceding hepatitis E virus infection	울산의대 김효재
244	Discrepancy between the electrodiagnostic study and ultrasonography in clinically suspected carpal tunnel syndrome	아주의대 엄영인
245	Clinical and pathological characteristics of genetically confirmed autosomal dominant Emery-Dreifuss muscular dystrophy patients	연세의대 이영건
246	Expiration to inspiration ratio as a prognostic factor in systemic AL amyloidosis	성균관외대 정우교
247	Mutations in the muscle adenylosuccinate synthetase-like 1 gene cause an autosomal recessive distal myopathy	성균관외대 최병욱
248	A case of antiphospholipid syndrome with transverse myelitis mimicking Guillain-Barré Syndrome	경희외대 나부석
249	NTRK1 mutations in Korean patients with congenital Insensitivity to pain with anhidrosis	전남외대 남태승
250	Respiratory failure due to restrictive chest wall deformity in muscular dystrophy	단국외대 류세열
251	A case of acute inflammatory demyelinating polyradiculoneuropathy in chronic diabetic patient	경희외대 문주선
252	Hypokalemic paralysis caused by renal tubular acidosis type I: a case report	울산외대 류은화
253	Polymyositis in a patient with Charcot-Marie-Tooth disease type 1A; diagnostic delay in unusual combination	성균관외대 문준규
254	A case of carpal tunnel syndrome with bifid median nerve and concurrent persistent median artery confirmed by ultrasonography	경희외대 문주선
255	Ulnar nerve conduction study using the first dorsal interosseous muscle recording in healthy Korean subjects	인제의대 정규운
256	Mutation in heat shock 27 kDa protein causes axonal neuropathy in mouse model	성균관외대 김은자
257	Neuroprotective approach of uric acid in ALS; clinical and therapeutic role	인제의대 오성일

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258	X-linked dominant Charcot-Marie-Tooth disease type 6 (CMTX6) with mutation in the pyruvate dehydrogenase kinase isoenzyme 3	성균관의대 김은자
259	A case of Isaac's syndrome with unknown origin	서남의대 명지병원 박두용
260	A case of autosomal dominant Emery-Dreifuss muscular dystrophy associated with a heterozygous LMNA mutation	부산의대 박영은
261	Presenilin 1(PSEN1) mutation in patient with primary lateral sclerosis	한양의대 박진석
262	Spinal xanthomatosis, a rare phenotype of Cerebrotendinous xanthomatosis : a case report	경북의대 박진성
263	Effective treatment with tacrolimus in corticosteroid-resistant polymyositis	서울의대 백설희
264	Treatment-induced neuropathy of diabetes at newly diagnosed diabetes mellitus	순천향의대 백지훈
265	A case of generalized Myasthenia Gravis with spasmodic dysphonia	경북의대 박진성
266	Exploration of modifier genes in Korean patients with facioscapulohumeral muscular dystrophy	이화의대 박형준
267	A novel mutation in DNAJB6, p.(Phe91Leu), in childhood-onset LGMD1D with a severe phenotype	전남의대 남태승
268	Late-onset myasthenia gravis in Korea: comparison with early-onset and very late-onset myasthenia gravis	성균관의대 조은빈
269	Experience of myasthenia gravis in single tertiary center for 5 years: epidemiological and clinical characteristics of elderly MG	가천의대 양지원
270	Vitamin B12 (cobalamin) deficiency with extreme hyperhomocysteinemia presenting as subacute combined degeneration, pancytopenia and splenic MRI lesion	아주의대 서홍일
271	Optic neuritis in patient with overlapping Bickerstaff's Brainstem Encephalitis and Guillain-Barre' Syndrome	영남의대 신규식
272	A case of recurrent Miller-Fisher syndrome	가천의대 양지원
273	Churg-Strauss syndrome can also mimic chronic inflammatory demyelinating polyneuropathy	서울의료원 원혜연
274	Delayed facial diplegia in a case with serologically confirmed acute motor axonal neuropathy form of Guillain-Barré syndrome	동아의대 윤별아
275	2 cases of delayed cranial nerve palsy in Miller-Fisher syndrome	고신의대 이대승
276	Brachial plexopathy after robotic thyroidectomy using Da Vinci robotic system	아주의대 엄영인
277	Changes of cortical excitability in obstructive sleep apnea syndrome	가톨릭의대 김지연
278	Relationship between obstructive sleep apnea syndrome and cerebral microbleeds	서울의대 구대림
279	Long-term Adherence of positive airway pressure therapy in Patients with Obstructive Sleep Apnea Syndrome; What are the main determining factors?	삼성창원병원 이정화
280	Acute response of heart rate variability to continuous positive airway pressure treatment in obstructive sleep apnea	고려의대 김정빈
281	Significance of snoring for predicting obstructive sleep apnea severity	인제의대 이준원
282	The quadratic relationship between apnea severity and depressive symptoms in patients with obstructive sleep apnea	울산의대 윤혁준
283	Comorbid Insomnia in Korean patients with obstructive sleep apnea	계명대의대 임상훈

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284	Electrophysiological characterization of subjects with obstructive sleep apnea syndrome presenting as insomnia	성균관의대 박혜리
285	The relationship between sleep and biophysiological measures in subjects with psychophysiological Insomnia	삼성서울병원 최수정
286	White matter hyperintensity in RLS; a comparison of subtypes of RLS	계명대의대 조용원
287	Transient global amnesia impairs small-world topology	서울의대 박영호
288	Brain areas involved in performing TTCT (Torrance Tests of Creative Thinking)	동국대의대 김광기
289	Annotating system for PhosphoVariant	국민건강보험공단 일산병원 김종현
290	Ubiquitination-dependent and - independent tau clearance via SQSTM1/ p62	순천향의대 임지영
291	A mini-gene for measuring alternative splicing of tau exon 10	순천향의대 안상일
292	The differential caspase activity in cortex and striatum by 3-nitropropionic acid chronic infusion	연세의대 조경주
293	DGAT2 mutation relevant with an autosomal dominant early-onset axonal Charcot-Marie-Tooth Disease	성균관의대 김은자
294	Patient fibroblasts-derived induced neurons demonstrates neuropathology of Krabbe Disease	한양의대 임수민
295	Novel mutations in MKS3/TMEM67 genes in COACH syndrome	전남의대 남태승
296	Ketamine increases excitability of hippocampal neurons through KCNQ Currents	강원의대 김성훈
297	A case of baclofen-induced encephalopathy presenting reversible structural brain lesions	전남의대 오윤창
298	Spinal tuberculosis	한양의대 박성호
299	A case of ventriculitis associated with extended spectrum beta-lactamase producing Klebsiella pneumonia after acupuncture	동국대의대 박수현
300	Infection related cerebral venous thrombosis after phlebotomy and dermopuncture on cervical muscle	원광의대 황윤수
301	A case of Lemierre's Syndrome with chronic otitis media	강원의대 김성근
302	Mumps-virus-associated mild encephalopathy with a reversible splenial lesion in an adult patient	고신대의대 채희운
303	Fulminant bacterial meningitis with intracisternal and intraocular abscess	대구파티마병원 박상원
304	A case of isolated spinal neurocysticercosis	경희대의대 신유용
305	Septic cavernous sinus thrombosis as a complications of an dental infection	한양의대 곽현승
306	Multiple cranial neuropathy possibly due to invasive aspergillosis	울지대의대 최서영
307	Rhino-orbito-cerebral mucormycosis presented with painful ophthalmoplegia	원광의대 이학승
308	Unusual manifestation of tuberculous cerebral vasculitis on vessel wall MRI	분당제생병원 장현순
309	Prognostic factors of acute encephalitis	충남의대 김용수
310	A case of mild encephalopathy with a reversible splenial lesion after mumps infection	중앙의대 배재한
311	A case of Meningitis-retention syndrome : a rare complication of aseptic meningitis	예수병원 이형수
312	Neurosyphilis presenting with committing arson	울지대의대 이승아

313	Serial MRI findings of slowly progressing probable Creutzfeldt–Jakob disease: case report	중앙의대 박무석
314	A case of disseminated Group B streptococcal infection with Nicolau syndrome	서남의대 명지병원 박두용
315	Cryptococcal meningoenkephalitis associated with systemic lupus erythematosus	건양의대 홍순호
316	A case of the fulminant cerebellitis in adult	가천의대 김갑수
317	Eosinophilic meningitis associated with toxocariasis	연세의대 최진교
318	Bilateral optic neuropathy after influenza vaccination in Korean middle aged female	강원의대 김예신
319	Syndrome of inappropriate antidiuretic hormone associated with herpes simplex encephalitis	부산의대 박순운
320	Rhabdomyolysis in anti-NMDA receptor encephalitis	서울의대 임정아
321	Invasive pituitary macroadenoma	한양의대 박성호
322	Survey of clinical practice of north Korean defectors in neurology department: a university hospital experience	충남의대 임영기
323	NUDT15 R139C causes Azathioprine-induced early leukopenia in Korean patients with neurological diseases	울산의대 김선영
324	lipotrophy after local steroid injection, two cases	충북의대 김인하
325	Unilateral isolated hypoglossal nerve palsy caused by arachnoid cyst	건양의대 김재환
326	Developmental abnormalities of the craniocervical junction resulting in Collet-Sicard syndrome	을지의대 김건우
327	Leukoencephalomyelopathy in chlorfenapyr intoxication	고려의대 강성훈
328	γ -aminobutyric acid B receptor (GABABR) antibody encephalitis misdiagnosed as post traumatic psychotic disorder after traffic accident	울산의대 이종민
329	Can we diagnose recurrent attack of acute intermittent porphyria (AIP) only by measuring delta-aminolevulinic acid (ALA) and porphobilinogen (PBG)?	한림의대 음시원
330	A case of Brainstem astrocytoma presenting with persistent hiccups	한양의대 한석길
331	Gliomatosis cerebri mimicking Herpes Simplex Encephalitis	부산의대 박순원
332	Primary diffuse large B-cell lymphoma of the choroid plexus	연세의대 라윤경
333	Parkinson’s disease in a patient with Klinefelter’s syndrome	가톨릭의대 이지은

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003	Different risk factors between intracranial and extracranial arterial stenosis in ischemic stroke	국군서울지구병원 김준엽
004	Does sleep apnea cause ischemic stroke during sleep?	강원의대 김진수
005	Two cases of endovascular treatment in stenosis of cavernous- petrous segment of ICA	대구파티마병원 박종완
006	A case of recurrent limb-shaking transient ischemic attack improved after contralateral carotid artery stenting	충남의대 이민영
007	Cortical subarachnoid hemorrhage and acute ischemic stroke associated with intracranial atherosclerosis	충북의대 장혜연
008	A case report: the effect of valproate on dystonic movement of women who suffer from moyamoya disease	충북의대 장혜연
009	Brainstem infarction caused by dissecting aneurysm of a basilar artery in a child	충남의대 김지영
010	A case of bilateral intracranial ICA stenoses causing ischemic stroke in the patient with chronic myeloid leukemia treated with nilotinib(Tasigna [®])	순천향의대 최나리
011	Huge giant cervical carotid artery aneurysm with ipsilateral cerebral infarction: ultrasonographic findings of two cases	고려의대 김한준
012	Multiple intracranial aneurysms in a patient with systemic sclerosis	고신대의대 이대승
013	The influence of minimal daily temperature upon the incidence of stroke in Seoul	순천향의대 신동원
014	Clinical characteristics of ischemic stroke in the 80 year-old or older compared with younger patients	조선의대 김만영
015	Effect of lesion location on the development of dementia after acute ischemic stroke	한림의대 장민욱
016	Association between polymorphisms in microRNA machinery genes (DICER1, DROSHA, RAN, and XPO5) and ischemic stroke	CHA의대 김옥준
017	Bilateral paramedian thalamic infarction: a case report of artery of percheron occlusion	울산의대 이유진
018	Malignant middle cerebral artery infarction with thyrotoxic atrial fibrillation	충북의대 양세진
019	A case of Takayasu's arteritis with assessment of response to treatment using high-resolution dark blood MRI	부산의대 김기태
020	Transient ischemic attack in hereditary hemorrhagic telangiectasia(HHT) patient	울산의대 김한아
021	A case of forced conjugate eye deviation in small pontine infarction	부산의대 김민경
022	Cerebral infarction with massive intra-aortic mass in young female	울산의대 김홍재
023	Acute intracerebral hemorrhage with a spot sign during computed tomographic angiography	조선의대 김광훈
024	Endovascular therapy for acute stroke in patients with current malignancy	전남의대 강승호
025	Transient global amnesia-like tiny hippocampal lesion in patients without transient global amnesia	건국의대 정문영

027	The ischemic lesion volume of diffusion weighted images is much more important to determined the prognosis after using thrombolysis in acute MCA occlusion than that of perfusion weighted images on MRI	동아의대 손상욱
028	Atypical bilateral medial medullary infarction due to unilateral vertebral arterial dissection	원광의대 김동희
029	Small vessel TIA or stroke detected with perfusion-weighted MRI	동아의대 나현욱
030	Two cases with only abnormal perfusion MRI findings in the transient ischemic attack	인제의대 정은주
031	Predictive ability of focal perfusion abnormality on perfusion weighted MRI in diffusion negative transient ischemic attack	울산의대 이상헌
032	Increased plasma homocysteine levels not MTHFR variant are associated with cerebral microbleeds	한양의대 류창환
033	Susceptibility vessel sign with bright vessel appearance that differentiates clot composition and its association with stroke etiology	서울의대 정한길
034	Venous infarction related to dural arteriovenous fistula directly drained into cortical veins	을지의대 김재국
035	Lateralized MRI findings of cerebral hypoxia in patients with carotid artery stenosis	원광의대 유인섭
036	Extensive cerebral microhemorrhage after extracorporeal mechanical oxygenation	한림의대 이은주
037	Transient ischemic attack in patient with an anruptured anuerym	충북의대 김진현
038	Factors associated with Do-Not-Resuscitate order in acute ischemic stroke patient	울산의대 김옥주
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040	A case of a negative DWI MRI within 12 hours of acute ischemic stroke symptoms	원광의대 황윤수
041	Cavernous malformation in pineal gland	한림의대 이채영
042	Are the anomalous vertebral arteries more hypoplastic? CT and MR angiographic analysis	한림의대 김철호
043	Acute ischemic stroke caused by IgA nephropathy without nephrotic syndrome	건양의대 김재윤
044	Clinical outcomes associated GI bleeding following ischemic stroke	CHA의대 이한빈
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046	Rt. Pontine tegmental infarction with contralateral ataxia	충북의대 차민주
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049	Prognostic impact of the collateral status in the vertebrobasilar occlusive stroke	경북의대 최동호
050	Cerebral small vessel disease score determines short and long-term prognosis in acute ischemic stroke patients	이화의대 송태진
051	Paradoxical role of vascular stiffness in lacunar stroke in progression	가천의대 신동훈
052	Higher pulsatility index of the middle cerebral artery is associated with lacunar stroke progression	질병관리본부 이건주
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054	Influence of the interval between serum levels of admission and fasting glucose on the functional outcome in hyperglycemic patients with acute ischemic stroke	부산의대 조기용
055	A case of contrast leakage mimicking intraventricular hemorrhage in patient with intravenous thrombolysis	부산의대 김백균
056	Successful repeated mechanical thrombectomy in patients with recurrent basilar artery occlusion	연세의대 박민철
057	A case of sudden hearing loss in PICA infarction	대구가톨릭의대 박정아
058	Middle cerebral artery infarction caused by large-sized artery vasculitis in systemic lupus erythematosus	연세의대 박민철
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060	Delayed reversible cerebral vasoconstriction syndrome following traumatic brain injury: a case report	서울의대 전진선
061	Modified dysphagia screening test in stroke patients also reduce the incidence of in-hospital pneumonia	아주의대 이성은
062	Stabilizing course of blood pressure at acute stage of ischemic stroke and 3-month functional outcome	성균관의대 강지훈
063	Initial glucose fluctuation increased poststroke cardiovascular events in diabetic patients	순천향의대 윤지은
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068	Comparison of gastrointestinal bleeding risk among different statin exposures with warfarin: An electronic health record-based retrospective cohort study	아주의대 이진수
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071	Detection of aorta and common carotid artery dissection in acute stage of stroke	인제의대 김준엽
072	A case of cryptococcal meningitis presenting as acute ischemic stroke	경북의대 이건희
073	Effectiveness of intravenous magnesium on the progressive lacunar infarction	순천향의대 이동현
074	Lipoic acid use and functional outcome after tissue plasminogen activator treatment in patients with acute ischemic stroke and diabetic polyneuropathy	전남의대 최강호
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076	Exome sequencing nominates the ACOX3 gene as a cause of recurrent extracranial internal carotid artery vasospasm	전남의대 강경욱
077	The correlation of SNPs of rs17501010, rs893051 and rs9290927 with small vessel disease	전북의대 아다브 비노드 쿠마

078	Association study of between Plasminogen activator inhibitor-1 (PAI-1) polymorphisms and ischemic stroke risk in a Korean population	CHA의대 김옥준
079	The early recognition of cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is important: two cases review	원광의대 김영서
080	CADASIL initially presented with isolated internuclear ophthalmoplegia	제주의대 김홍전
081	Primary angiitis of central nervous system in a patient with CADASIL	경희의대 이도경
082	A case report of cerebral infarction in a patient with a large atrial septal defect and pulmonary hypertension	CHA의대 김 윤
083	Catastrophic venous thrombosis refractory to conventional treatment	울산의대 이문규
084	Stroke-like manifestation of High-dose MTX toxicity	울산의대 정 수
085	Posterior ventricular enlargement sign differentiating dementia with lewy bodies from Alzheimer's disease	연세의대 예병석
086	Dissociation between β -amyloid burden and neurodegenerative changes in Alzheimer's disease: report of two cases	부산의대 최유진
087	Transient global amnesia: a study of 97 cases	이화의대 윤민정
088	Does serum uric acid act as a modulator of CSF AD biomarker-related cognitive decline?	연세의대 예병석
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090	Peri-operative rivastigmine patch reduces the delirium occurrence in the elderly at risk of dementia	중앙의대 윤영철
092	Investigation of neural substrate of transient global amnesia using positron emission tomography	서울의대 이상학
093	The association between cognitive status and abnormal findings on MR spectroscopy in the patients with hypoxic brain damage	인하의대 이응석
094	Differences in hippocampal surface and white matter structure in early and late MCI: ADNI Study	한국과학기술원 이재현
095	Unusual idiopathic normal pressure hydrocephalus patient with marked asymmetric and upper body parkinsonism	경북의대 최동호
096	Heterogeneous etiology of SNAP in MCI Stage: longitudinal changes of cortical thinning	연세의대 예병석
097	Slower progression of subcortical vascular dementia patients who met the Seoul criteria compared to those who did not	성균관의대 이주연
098	Donepezil induced Rhabdomyolysis	고신의대 채희운
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100	Association of sleep qualities and cortical thickness in Subjective Memory Impairment patients	성균관의대 이주연
101	Diffuse type of frontal lobe atrophy shows a poorer prognosis than focal type in the behavioral variant frontotemporal dementia: A CREDOS-FTD Study	성균관의대 이진산
102	Paraneoplastic limbic encephalitis presenting with persistent anterograde amnesia	영남의대 조진혁

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103	Comparisons of hippocampal subfields volumes between mild cognitive impairment and Alzheimer's disease	계명대의대 이현아
104	The relationship between neuropsychiatric symptoms and hippocampal subfields volumes in Alzheimer's disease	계명대의대 임상훈
105	Relative cognitive impairment in neuropsychological tests as a predictor for future cognitive decline	아주의대 임태성
106	Independent effects of physical exercise and education on age-related cortical thinning in cognitively normal individuals	성균관대의대 이진산
107	An autopsy confirmed case of nonfluent/agrammatic variant primary progressive aphasia combined with generalized chorea with corticobasal degeneration pathology	부산의대 이명준
108	Clinical impacts of lobar microbleeds in patients with clinically probable cerebral amyloid angiopathy: a pilot study	성균관대의대 장영경
109	Functional connectivity of the posterior insular in Wernicke's encephalopathy with impaired vestibular ocular reflexes	전북의대 장혁수
110	Anatomical subtypes of subjective memory impairment individuals	부산의대 정나연
111	A case of rapid progressive cognitive impairment caused by human immunodeficiency virus	동아의대 이형진
112	The distribution and clinical impact of Apolipoprotein E4 among subjective memory impairment and early mild cognitive impairment	연세의대 조한나
113	Tract-specific correlates of neuropsychological deficits in patients with subcortical vascular cognitive impairment	부산의대 정나연
114	Novel PSEN1 (G209A) mutation in a case of early onset Alzheimer dementia	중앙의대 정다운
115	Screening of cognitive dysfunction in chronic hemodialysis patients - comparison of the mini mental state exam to the Montreal cognitive assessment	한림의대 이선화
116	Glucose metabolism in early onset versus late onset behavioral variant frontotemporal dementia	부산의대 정나연
117	The correlation between CSF biomarkers, regional brain atrophy, cognitive performance in early onset neurodegenerative dementia	이화대의대 정아름
118	A episode like transient global amnesia associated with intraventricular tumor adjacent to the fornix	연세의대 정승호
119	Neuropsychiatric characteristics of PiB-negative subcortical vascular dementia versus behavioral variant frontotemporal dementia	부산의대 정나연
120	Identification of PSEN1 variants in Korean patients with early-onset Alzheimer's Disease	연세의대 조한나
121	Decreased blood pressure after rapid intravenous fosphenytoin infusion	국립중앙의료원 김현경
122	Seizure disorder presenting as stroke-like symptoms	CHA의대 신정원
123	Prolonged loss of consciousness in patients with reflex syncope: diagnostic challenge between epilepsy and syncope	인제의대 박강민
124	Alcohol-related seizures presenting with nonconvulsive status epilepticus and a thalamic lesion: an atypical presentation of subacute encephalopathy with Seizures in Chronic Alcoholism (SESA) Syndrome	을지의대 김태경

125	Different EEG pattern between waking and sleeping status during hypoglycemia in type 1 DM patient	전북의대 이설원
126	Reversible MRI abnormalities in a patient with complex partial status epilepticus	울산의대 구준모
127	Lesional focal seizures triggered by the the hyperglycemia	울산의대 이문규
128	Anti-GAD antibody associated encephalitis	연세의대 이승하
129	Sleep deprivation electroencephalographic findings in the patient with sporadic hemiplegic migraine	전북의대 이찬혁
130	Selective serotonin reuptake inhibitor induced posterior reversible encephalopathy in a normotensive patient	전남의대 이세영
131	A case of sick sinus syndrome as seizure-like manifestation	아주의대 김현재
132	Can pursuit eye movements reflect the efficacy of antiepileptic drugs?	인제의대 이준원
133	Difference in heart rate change between temporal and frontal lobe seizures	성균관의대 황우섭
134	Epileptic nystagmus in a patient with occipital lobe epilepsy: a case report	계명대의대 임상훈
135	Somatosensory evoked potential (SEP) induced myoclonic status epilepticus in hypoxic ischemic encephalopathy	강원의대 김예신
136	Posterior reversible encephalopathy syndrome(PRES) involving deep brain structures as the presentation of eclampsia	성균관의대 김홍직
137	Human herpesvirus-6 encephalitis presenting non-convulsive status epilepticus	단국의대 강봉희
138	Creutzfeldt-Jakob disease presenting as frontal lobe epilepsy with migraine	대구기톨릭의대 김민석
139	Convulsive movement as an initial manifestation of sporadic Creutzfeldt-Jakob disease	원광의대 김동희
140	Neurosyphilis presenting with non-convulsive status epilepticus	서울의대 안선재
141	Aphasic status epilepticus associated with uremia	을지의대 조수현
142	Field testing the criteria for primary stabbing headache according to the third beta edition of the international classification of headache disorders	한림의대 이민우
143	Headache as an aura of focal seizures; video-EEG monitoring study	건국대의대 진재현
144	Cluster headache associated with intracranial carotid artery dissection	을지의대 이은경
145	Management of diffuse idiopathic skeletal hyperostosis presenting with neck pain	을지의대 이은경
146	Reversible cerebral vasoconstriction syndrome with vasogenic edema after blood transfusion	울산의대 윤혁준
147	Acromegaly caused by pituitary adenoma in patient with normal hormone level	가톨릭의대 성빈센트병원 노상미
148	Intracranial hypertension after massive blood transfusion	고려의대 반광현
149	A case of overdose chemotherapy-associated intracranial hypotension mimicking dural metastasis in breast cancer	서남의대 명지병원 박두용
150	Secondary stabbing headache caused by skull metastasis: a case report	성균관대의대 최한나
151	Headache caused by chronic carbon monoxide exposure	부산의대 김기태
152	Cerebral paragonimiasis presenting as chronic headache	경상대의대 양태원
153	The prevalence of Parkinson`s disease in south Korea: a 11-year nationwide, population-based study	국민건강보험공단 일산병원 이지은

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154	Comparison of dysphagia between patients with Parkinson's disease and progressive supranuclear palsy	울산의대 권미선
155	Cognitive and motor aspects of Parkinson's disease associated with dysphagia	순천향의대 김지선
156	Diabetes and risk of Parkinson's disease: an updated meta-analysis of observational studies	CHA의대 김원찬
158	Stridor occurred in a patient with SCA17	서울의대 김경준
159	Parkinsonism in spinocerebellar ataxia type 7	경희의대 이도경
160	Escitalopram induced extrapyramidal symptoms: A case report	충북의대 민경현
161	Facial diplegia masqueraded as an aggravating parkinsonism	충북의대 민경현
162	Apraxia of lid opening caused by ropinirole withdrawal	건국대의대 김희진
163	Mirror movements in a patient with pontine infarction	중앙의대 박무석
164	Subthalamic electrode Insertion with deep brain stimulation for impulse control disorder	울산의대 최유진
165	Effect of unilateral subthalamic deep brain stimulation in highly asymmetric Parkinson disease: 7-year follow-up	서남의대 명지병원 엄관희
166	Risk factors for surgical site infections after deep brain stimulation of patients with Parkinson's disease in a 13-years period	울산의대 김미선
167	Bilateral deep brain stimulation of the subthalamic nucleus under sedation with propofol and fentanyl	엘지의대 이웅우
168	Electrode reposition cases in subthalamic nucleus deep-brain stimulation for Parkinson's disease	울산의대 박성철
169	Mortality after deep brain stimulation surgery for patients with advanced Parkinson's disease	울산의대 유호성
170	Incongruent hemiatrophy and hemiparkinsonism in a patient with schizencephaly	경희의대 권영남
171	Selective fascicle injection of botulinum toxin at the flexor digitorum superficialis and flexor digitorum profundus in patient with focal dystonia affecting fingers	경북의대 고평우
172	Unilateral limb asterixis related to hypoperfusion of middle cerebral artery territory	조선의대 정지연
173	Dystypia without aphasia in a patient with Parkinson's disease after deep brain stimulation	가톨릭의대 전기평
174	Parkinsonism in EPM and CPM without striatal dopamine binding dysfunction: a case report	가톨릭의대 김성훈
175	Glutamic acid decarboxylase antibody associated paraneoplastic cerebellar syndrome in thymoma	한림의대 이현창
176	The quality of life in patients with hemifacial spasm	제주의대 강미경
177	Clinical implication of initially affected side in typist's cramp	한림의대 강석운
178	Respiratory dysfunction in patients with Parkinson's disease and multiple system atrophy	고려의대 이혜미
179	Restless legs syndrome in Parkinson's disease patients: a comparative study on prevalence, clinical features, motor and non-motor symptoms	계명대의대 유수연
180	Association between urine protein/creatinine ratio and cognitive dysfunction in Parkinson's disease	가톨릭의대 오윤상

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181	Improvement of standing stability after weight-shifting training in spinocerebellar staxia type 6: a case report	순천향의대 강여정
182	Corticobasal syndrome-like sporadic Creutzfeldt-Jakob disease	가톨릭의대 오주희
183	Inherited cerebellar ataxia type 17 (SCA17) associated with 41 trinucleotide repeats of TATA-box binding protein gene(TBP) in a family	서울의대 김아련
184	Aspergillus abscess presenting as chronic progressive cerebellar ataxia	한림의대 김민기
185	A case of acute focal myelitis presented with painful tonic spasm involving both legs	순천향의대 이상우
186	The first case report of adult onset Niemann-Pick disease type C in Korea	동아의대 이수윤
187	Inhibitory effect of apocynin on proteasome inhibition-induced apoptosis in differentiated PC12 cells	보훈공단 중앙보훈병원 강진호
188	Biochemical protective effect of 1,25-dyhydroxyvitamin D3 through autophagy induction in the MPTP mice model of Parkinson's disease	울산의대 장우영
189	The neuroprotective effect of erythropoietin on rotenone-induced neurotoxicity in SH-SY5Y cells through the induction of autophagy	울산의대 장우영
190	Novel compound heterozygous mutations of PLA2G6 in a Korean pedigree of young-onset Parkinson's disease: a study of whole genome sequencing	울산의대 최유진
191	Impaired vascular endothelial function in patients with idiopathic restless leg syndrome: a new aspect of vascular pathophysiology	아주의대 윤정환
192	Ataxia with oculomotor apraxia type 1 without oculomotor apraxia: A case report	한림의대 이민우
193	Correlation between presynaptic dysfunction on F-18 FP-CIT PET and trinucleotide CAG repeat in Huntington's disease	대구가톨릭의대 김민석
194	CSF1R mutations presenting with atypical Parkinsonism	부산의대 김민경
195	Dentatorubropallidolusian atrophy (DRPLA) with recurrent seizure and esotropia	한림의대 오학주
196	Whole-exome sequencing identifies the first Asian patient with ovarioleukodystrophy related to AARS2 mutation	울산의대 양희준
197	Korean patients with spinocerebellar ataxia type 6 presenting with unusual manifestations	제주대의대 김홍전
198	Comparison of myelin water fraction values in white matter lesions between multiple sclerosis and neuromyelitis optica spectrum disorder	국립암센터 정인혜
199	MRI characteristics of short segment myelitis in NMO-IgG-positive neuromyelitis optica spectrum disorders	울산의대 진주애
200	Anti-Ro/La antibody may be associated with time to next relapse in neuromyelitis optica spectrum disorder	성균관의대 이혜림
201	Antibody to myelin oligodendrocyte glycoprotein in adults with inflammatory demyelinating disease of the CNS	서울의대 김성민
202	Visual evoked potentials and optic coherence tomography in neuromyelitis optica spectrum disorders: which is more sensitive?	성균관의대 장혜민
203	A case of neuromyelitis optica spectrum disorder presenting as area postrema syndrome with posterior reversible encephalopathy syndrome	성균관의대 김동엽

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204	Diffuse large B cell lymphoma with open ring enhancement mimicking tumefactive multiple sclerosis	서울의료원 이정현
205	Complete tongue paralysis as a rare presentation of seropositive neuromyelitis optica spectrum disorder affecting dorsal medulla oblongata	충남의대 김용수
206	Magnetic resonance imaging of trigeminal neuritis after influenza vaccination	한림의대 김보희
207	Spinal dural arteriovenous fistula as a potential mimic of transverse myelitis	전남의대 강승호
208	A case of acute combined central and peripheral demyelination	연세의대 최진교
209	Reversible corpus callosal lesions associated with a use of Adalimumab in patient with ulcerative colitis	고려의대 박진우
210	Listeria monocytogenes central nervous system infection mimicking Neuromyelitis optica spectrum disorder	조선의대 추인성
211	Unilateral ptosis with partial third nerve paresis in midbrain infarction	가천의대 강민주
212	A case of multiple orbital myositis accompanied by herpes zoster ophthalmicus	아주의대 길영은
213	Unilateral hemorrhage restricted to the middle cerebellar peduncle	서울의대 김성희
214	Isolated anterior cerebellar vermian infarction	서울의대 김성희
215	Recurrent nonpupil-sparing isolated complete third nerve palsy without compressive neuropathy	건양의대 김재윤
216	Wall-eyed bilateral internuclear ophthalmoplegia in unilateral midbrain infarction	건양의대 김재환
217	A case of ophthalmoplegia and blepharoptosis mimicking incomplete 3rd nerve palsy following injection of filler into the nasal bridge	인제의대 김정민
218	Clinical characteristics and prognosis of patients presenting with binocular diplopia	CHA의대 김종욱
219	Vertical diplopia with unilateral ptosis as an initial manifestation of pleomorphic adenoma of the in a lacrimal gland	전남의대 김종훈
220	A case of midbrain infarction causing isolated fourth nerve palsy	한양의대 한석길
221	Idiopathic hypertrophic cranial pachymeningitis presenting as Tolosa-Hunt syndrome	원광의대 황용수
222	A case of spontaneously resolved isolated oculomotor nerve palsy due to cavernous sinus fistula	CHA의대 윤승재
223	Recurrent acute isolated third cranial nerve palsy in the anti-GQ1b antibody syndrome	전북의대 양태호
224	Optic chiasm involvement in ethambutol-induced optic neuropathy	고려의대 김재겸
225	Immunoglobulin G 4-related disease presenting headache and diplopia	계명대의대 강민성
226	A case of dural arteriovenous fistula misconceived as carotid cavernous fistula	한림의대 김철호
227	Isolated horner syndrome in infective cervical myelitis	대구가톨릭의대 박재한
228	Neural networks related to Opsoclonus-myoclonus syndrome revealed by PET study	전북의대 양태호
229	A case of normal pressure glaucoma presenting as monocular nasal visual field defect	전북의대 이설원
230	Topographic lesion analysis of cerebellar tumor related positional nystagmus	전남의대 이세영
231	A case of opsoclonus myoclonus ataxia syndrome: transition of coulomotor findings from ocular flutter to opsoclonus	삼성창원병원 이정화
232	Delayed loss of visual acuity after closed head trauma: a case with indirect traumatic optic neuropathy	한림의대 이주영

233	Optic tract syndrome due to posterior communicating artery aneurysm	서울의대 지요다
234	Periodic pupillary change in Cheyne-Stokes respiration	부산의대 채송화
235	Miller-Fisher syndrome antecedent acute hepatitis A in a patient with chronic hepatitis B	충북의대 김진현
236	Prognostic value of brain MRI, blink reflex in Bell's palsy	대구가톨릭의대 배창범
237	Early prediction of poor outcome in Guillain-Barré syndrome	동국의대 박수현
238	High risk group screening of later onset Pompe disease in unspecified myopathy patients	연세의대 이정환
239	Pontine venous malformation mimicking ocular myasthenia gravis	한림의대 이민우
240	Multifocal motor neuropathy with conduction block presented with calf muscle rippling	고려의대 김지선
241	Neurofibromatosis presenting as chronic inflammatory demyelinating disease	연세의대 이원우
242	A case of anti-GQ1b antibody syndrome presenting with acute unilateral ophthalmoplegia without ataxia and areflexia	경희의대 이재홍
243	Guillain-Barré Syndrome with persistent and prominent asymmetric weakness	한림의대 이민우
244	A case of Pompe disease having a diagnostic difficulty based on genetic study	연세의대 이정환
245	Chronic relapsing axonal neuropathy	한림의대 이민우
246	Isolated compressive temporal branch palsy of facial nerve after drug intoxication	고려의대 김지선
247	Acid alpha-glucosidase pseudodeficiency allele in normal Korean populations	부산의대 이종목
248	Genetic characteristics of Korean patients with amyotrophic lateral sclerosis using multi-gene panel testing	한양의대 오기욱
249	Genetic profiles in patient with nemaline myopathy	부산의대 이종목
250	Proteomic analysis of the alteration of nuclear cytoplasmic distribution of intracellular proteins in motoneuron cell lines expressing mutant SOD1 G93A	서울의료원 김지은
251	A case of arterial type Thoracic outlet syndrome (TOS) presenting with an axillary pain	울산의대 이종민
252	Serial ultrasonographic findings of herpes zoster neuralgia with motor weakness	경희의대 이지훈
253	Myasthenia gravis accompanied by adrenal gland tumor	한림의대 이채영
254	Spontaneous temporal meningocele, a rare cause of facial synkinesis	가톨릭의대 이현조
255	Alternating recurrent painful ophthalmoplegia as an idiopathic hypertrophic tentorial pachymeningitis	동아의대 이형진
256	Stiff-person Syndrome in a patient with myasthenia gravis after thymomectomy : a case report	성균관의대 이혜림
257	Facial palsy after dental procedure	한림의대 이채영
258	Clinical characteristics of MuSK-MG in Korea: comparison with double seronegative MG	서울의대 박기홍
259	Charcot-Marie-Tooth (CMT) with Acute Demyelinating Disease	대구파티마병원 최종환
260	Diffusion-weighted MRI: useful diagnostic tool for early detection of spinal cord infarction	울산의대 진주예

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261	Dynamic electromyography and gait analysis in patients with Charcot-Marie-Tooth neuropathy	성균관의대 황선희
262	Anti-GD1b antibody-associated acute motor conduction block neuropathy with reversible conduction failure	부산의대 장지영
263	GNE myopathy showing diffuse lumbosacral root thickening	건국대의대 정문영
264	Multifocal motor neuropathy in patient with rheumatoid arthritis receiving infliximab treatment	울산의대 정 수
265	Frontotemporal dementia with motor neuron disease in a patient with antiphospholipid syndrome :cases report	한양대의대 정윤철
266	Anterior interosseous nerve palsy in neuralgic amyotrophy due to fascicular involvement of the median nerve proper	을지의대 정의성
267	Colchicine induced myoneuropathy manifested by polyradiculoneuropathy	건국대의대 최교민
268	Surfer's myelopathy- An unusual case of nontraumatic myelopathy	고려대의대 최종석
269	Phenotypic and genetic profiles in 121 Korean patients of X-linked dominant Charcot-Marie-Tooth disease type 1	성균관대의대 김은자
270	Clinical efficacy of pulsed radiofrequency neuromodulation for intractable meralgia paresthetica	한림의대 손종희
271	A case of Guillain-Barre syndrome with pain dominant initial symptoms	대구파티마병원 조용국
272	Phenotypic characteristics of Charcot-Marie-Tooth type 1E (CMT1E) patients from cohort study	성균관대의대 김은자
273	Ocular myasthenia gravis in patients with sarcoidosis	건양의대 홍순호
274	Exome sequencing reveals compound heterozygous DYSF mutations in a myopathy family with decreased acid-alpha glucosidase activity	이화대의대 최지현
275	Clinical Heterogeneities in NEFL Mutations from 24 Korean Patients with Charcot-Marie-Tooth disease: CMT type 1F, CMT type 2E, and intermediate type CMT	성균관대의대 김은자
276	A case of recurrent Tolosa-Hunt Syndrome	대구파티마병원 조용국
277	Overlap case of pharyngeal-cervical-brachial variant of Gullain-Barre Syndrome and Miller-Fisher Syndrome	건양의대 홍순호
278	Co-occurred case of Guillian-Barre syndrome and acute transverse myelitis	한림의대 최취철
279	A case of Polymyalgia rheumatica with extremities weakness dominant symptoms	대구파티마병원 조용국
280	Late use of electronic media and its impact on insomnia, depression and suicidality among Korean adolescents	성균관대의대 서지혜
281	The anti-obesity effect of weekend catch-up sleep among adults in Korean population	서울의대 임희진
282	Different cutoff value of the Korean Version of the REM sleep behavior disorder screening questionnaire between patients with obstructive sleep apnea and healthy people	울산의대 백준현
283	Preliminary study for REM sleep behavior disorder (RBD) in Parkinson's disease using RBD screening questionnaire	가천의대 강민주
284	Quality of life in idiopathic REM sleep behavior disorder in Korea	계명대의대 김근태

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285	Comparing the quality of life between patients with narcolepsy only and comorbid narcolepsy patients	계명의대 조용원
286	Health-related quality of life in patients with narcolepsy type1 and type2	부산의대 김백균
287	Anti-Ma2-associated encephalitis presented with hypersomnia	부산의대 장지영
288	Sleep and related symptoms in shift workers and day workers in a single enterprise corporation	인제의대 송파멜라
289	Regional grey matter changes in shift workers: a voxel-based morphometry study	고려의대 김정빈
334	The sleep pattern of Korean woman professional basket players	경희의대 조수현
290	Mesenchymal stem cells modulate the functional properties of microglia via TGF- β secretion	CHA의대 권민수
291	Neuroprotective effects of uric acid against sodium arsenite-induced motor neuronal cell death	한양의대 백수정
292	Abi, an Abl tyrosine kinase-interacting protein, regulates synaptic development and neuronal survival via inhibition of BMP signaling	한양의대 남민엽
293	Neuroprotective effects of atorvastatin against oxygen-glucose deprivation-induced neural stem cell death through the activation of the PI3K pathway	한양의대 오경필
294	Effects of aspirin and clopidogrel on neural stem cells	한양의대 최호진
295	Interaction between sublethal dose of amyloid beta and hypoxia in neural stem cells	한양의대 최호진
296	Apolipoprotein E deficiency worsens small vessel pathology and cognitive dysfunction in a mouse model of subcortical vascular dementia	한국과학기술원 이익성
297	Paracrine action of High Mobility Group Box 1 (HMGB1) via Toll-like receptor 2 prevents ischemia-induced oligodendrocyte death	아주의대 최준영
298	Amelioration of abnormal genomic alteration as a molecular therapeutic mechanism of intravenous administration of human mesenchymal stem cells in rodent stroke model: transcriptome analysis	CHA의대 오승현
299	Cilostazol reduces huntingtin accumulation in cultured astrocytes	울산의대 김현진
300	The Distribution of Cerebral Microbleeds Determines Their Association with Vascular Resistance in Noncardioembolic Stroke Patients	이화의대 장윤경
301	Sustained attention is linked to the spectral content of sleep EEG activity	서울의대 백신혜
302	Dysphagia in familial dysautonomia assessed by surface electromyography	국민건강보험공단 일산병원 조정희
303	Different alarm criteria in muscle MEP between cervical and thoracic OPLL surgery	서울의대 조성래
304	Association of BDNF Val66Met with memory dysfunction and cortical thickness changes in Parkinson's disease: an imaging genetics study	한림의대 김윤중
305	Cerebral-perfusion reserve after carotid-artery stenting: relationship with power spectrum of electroencephalography	경상의대 정석원
306	Evaluation of extensor digitorum brevis thickness in healthy subjects: a comparative analysis of nerve conduction studies and ultrasound scans	대구가톨릭의대 석정임
307	Difference in the responses to head-up tilting test depending on the age	대구파티마병원 이장준
308	A case of Ross syndrome with segmental anhidrosis and anisocoria: application of finger wrinkle test	부산의대 채송화

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309	Ventricular tachycardia imitating epileptic seizures	서울의료원 이정현
310	Potential risk factors for developing herpes zoster-associated aseptic meningitis in patients with herpes zoster	전남의대 김상훈
311	Unique color perception recovery following optic neuritis after acute Hepatitis A	조선의대 봉정빈
312	A case of ramsay Hunt Syndrome that initiated from the palate	조선의대 김만영
313	Acute myeloradiculitis associated with herpes simplex virus type2	고신의대 이대승
314	Acute respiratory paralysis as an unusual presenting symptom of Japanese encephalitis	경상의대 정다혜
315	Acinetobacter baumannii meningoencephalitis related to Incidental durotomy	울지의대 이혜민
316	A case of influenza-associated acute necrotizing encephalopathy	서울의대 강동완
317	The case of intramedullary spinal abscess	경희의대 나부석
318	Atypical mitochondrial encephalopathy, lactic acidosis and stroke-like episodes with false positive 14-3-3 protein mimicking Creutzfeldt-Jakob disease	울산의대 류재찬
319	A case of neurosyphilis presenting as unilateral oculomotor nerve palsy	이화의대 김태경
320	Trigeminal neuropathy associated with aggressive NK-cell Leukemia	고려의대 권예지
321	Anti-Ma2 antibody paraneoplastic encephalitis patient diagnosed with peripheral T cell type lymphoma	울산의대 구준모
322	Recurrent oculomotor neuritis related to autoimmune hypothyroidism	경희의대 신유용
323	Gamma amino butyric acid B receptor(GABABR) Encephalitis that developed after viral encephalitis: a case report	가톨릭의대 김성훈
324	Accompanied myelitis in a patient with mild encephalitis with a reversible splenial lesion(MERS)	한림의대 이주영
325	Central nervous system involvement in the necrotizing vasculitis presenting bilateral facial palsy	울산의대 이문규
326	MPNST presenting only unilateral leg pain in a patient with neurofibromatosis type I	연세의대 오여진
327	Green tobacco sickness in elderly patient who had never been exposed to nicotine	순천향의대 이승근
328	Severe hypothermia caused by organophosphate poisoning	순천향의대 백지훈
329	Pseudotumor cerebri in a patient undergoing sexual reassignment therapy	중앙의대 배재한
330	Stroke-like manifestation in metastatic pancreatic cancer	경희의대 나부석
331	Relapsing polychondritis with encephalitis: a case report	계명대의대 임상훈
332	Anti-NMDA receptor encephalitis presenting as an acute psychotic symptom in a man	가톨릭의대 임성철
333	Cerebral syncope versus neurally mediated syncope	울산의대 남효정



2015년

대한신경과학회 제34차 추계학술대회

– Oral Presentation –

【Oral Presentation 좌장명】

SS1: Stroke I (Eng)

이광호(성균관의대) · 장대일(경희의대)

SS2: Neuroscience

고재영(울산의대) · 김병곤(아주의대)

SS3: Movement Disorder I

이명식(연세의대) · 김진호(조선의대)

SS4: Muscle and Nerve I

임정근(계명의대) · 최영철(연세의대)

SS5: Epilepsy I: Clinical Epileptology

박성파(경북의대) · 권오영(경상의대)

SS6: Stroke II

유경호(한림의대) · 정슬기(전북의대)

SS7: Demyelinating Disorders

김광국(울산의대) · 김호진(국립암센터)

SS8: Sleep

윤창호(서울의대) · 주은연(성균관대의대)

SS9: Dementia I (Eng)

최경규(이화의대) · 박건우(고려의대)

SS10: Headache

정재면(인제의대) · 김병건(울지의대)

SS11: Muscle and Nerve II (Eng)

최병욱(성균관대의대) · 김정준(서울의대)

SS12: Dementia II

김병채(전남의대) · 최성혜(인하의대)

SS13: General Neurology or other issue I

서만옥(전북의대) · 최영철(연세의대)

SS14: Stroke III

황성희(한림의대) · 이승훈(서울의대)

SS15: Neuro-otology

성기범(순천향의대) · 이 형(계명의대)

SS16: General Neurology or other issue II

권기한(한림의대) · 박기종(경상의대)

SS17: Movement Disorder II (Eng)

김재우(동아의대) · 전범석(서울의대)

SS18: Epilepsy II

신동진(가천의대) · 손영민(가톨릭의대)

SS19: Stroke IV

김경문(성균관대의대) · 홍근식(인제의대)

Oral Presentation

Scientific Session 1

Stroke I (Eng)

• 시간: 14:20~15:20 • 장소: Convention Hall B (4F)

1 [2014년도 명인학술상 수상-연구 주제 발표]

Association between meteorological variables and acute stroke incidence in South Korea

Hyung-Min KWON, Jae-Sung LIM, Yong-Seok LEE, Byung-Woon YOON, Junyoung LEE, Seung-Eun KIM

Department of Neurology, Boramae Medical Center, Seoul National University Hospital

Background & Objectives: Over a few decades many environmental studies have evaluated the association between weather and stroke, with varying conclusions. The purpose of the present study was to determine whether seasonal variations in stroke incidence exist and whether they are related to meteorological parameters under similar weather condition in South Korea. **Method:** From January 1, 2011, to December 31, 2011, 2,894 acute stroke patients were selected from standard patient sampling of Korean health insurance review and assessment service. We used simple correlation and multiple regression analysis between stroke incidence and variable meteorological parameters [temperature (min, max, mean), temperature difference between day before, temperature change of day, atmosphere, humidity, wind speed, physiological equivalent temperature] and calculated the relative risk of stroke incidence associated with meteorological parameters. **Results:** Among all stroke patients, the mean age was 67.3 years (male, 64.5; female, 70.3). Of a total 2,894 strokes, 2,176 were ischemic stroke and 718 were hemorrhagic stroke. There was no seasonal variation of incidence of ischemic and hemorrhagic stroke. The temperature change of day was positively correlated with ischemic stroke in male patient [relative risk (RR)=1.027; 95% CI=1.006-1.05, P<0.05] and older age group (≥ 65 years) (RR=1.031, 95% CI=1.011-1.052, P<0.05). The temperature difference between day before showed negative correlation with all stroke incidence (RR=0.968, 95% CI=0.941-0.996, P<0.05), especially among the older female group. **Conclusion:** We firstly demonstrated seasonal variation in the nationwide incidence of stroke and its subtypes through consideration of the meteorological parameters. We therefore expect that these findings may enhance our understanding of the relationships between stroke and weather.

2

Risk factors for the development of depressed mood after stroke

Yerim KIM, Tae Jung KIM, Han-Gil JUNG, Sang-Bae KO, Seung-Hoon LEE, Byung-Woo YOON

Department of Neurology, Seoul National University Hospital, Department of Neurology, The Catholic University of Korea Bucheon St. Mary's Hospital

Background & Objectives: Although poststroke depression (PSD) is common and may affect outcome, it is often overlooked. The reported prevalence of PSD is about 30%. Although the mood profile of primary depression is similar to PSD, the standardized diagnostic criteria of the DSM-IV for depression is not appropriate for stroke. The aim of this study was to evaluate the risk factors for the development of depressed mood after stroke. **Method:** A total of 775

patients with acute ischemic stroke were enrolled in this study from March 2010 to May 2013. Patients who had depressed mood or markedly diminished interest were prescribed the antidepressants. **Results:** Among the 775 subjects, 39 patients (5.0%) showed depressed mood and were prescribed the antidepressants during admission. Those were older, more likely to have dysphagia, tube feeding, cardioembolic etiology, long hospitalization, ICU stay history, longer duration of ICU stay, mechanical ventilation, and infection history. The initial neurological severity did not differ between two groups. After adjusting for multiple covariates, hospital duration (OR 1.07, 95% CI: 1.04-1.10, p<0.001) and mechanical ventilation (OR 5.15, 95% CI: 1.53-17.40, p<0.01) were significantly related to depressed mood. **Conclusion:** Depressed mood after stroke might be under-detected in our clinical practice. However, considering higher mortality in stroke patients with initial depression, we should pay attention to detect and manage the depressed mood more aggressively. More systemized and standardized scale for PSD might be needed. Furthermore, in patients with long hospitalization or mechanical ventilation, we might consider to use of the antidepressants.

3

Baseline characteristics and clinical outcomes of acute symptomatic internal carotid artery stumps

Kyu Sun LEE, Sung Eun LEE, Ji Man HONG, Jin Soo LEE

Department of Neurology, Ajou University School of Medicine, Ajou University Hospital

Background & Objectives: A proximal remnant of internal carotid artery (ICA) following complete occlusion of the ICA is referred as a "stump". Theoretically, recurrent cerebrovascular symptom caused by severe ICA stenosis might cease when ICA stenosis progresses to complete occlusion. However, there are some cases in which recurrence or progression of ischemic stroke symptom occur due to occluded ICA stump, so called 'carotid stump syndrome'. It has not been rigorously studied for this disease entity, and no universally accepted treatment exists up to now. Therefore, we aimed to investigate clinical characteristics, treatment results and clinical outcomes of ICA stump cases. **Method:** The study population was recruited from stroke registry in a university hospital between February 2005 and January 2015. The eligibility criteria was 1) demonstration of extracranial ICA occlusion on computed tomographic angiography (CTA) as a stump morphology and 2) acute ischemic stroke or transient ischemic attack in corresponding arterial territory within 7 days from symptom onset. The exclusion criteria was 1) presumed mechanism as cardiac embolism or other miscellaneous causes such as dissection, moyamoya syndrome and vasculitis or 2) concomitant carotid T type occlusion on CTA. A total of 114 included patients were dichotomized as stable group vs. unstable group. Unstable group indicated cases of stroke recurrence within 3 months or progression of ischemic stroke in hospitalization period. Progression of stroke was defined as 1) change of National Institutes of Health Stroke Scale (NIHSS) ≥ 2 or 2) change of NIHSS ≥ 1 with malignant change or increase of infarct extent. Intensive statin treatment accounted for 10 or more mg of rosuvastatin and 20 or more mg of atorvastatin. By logistic regression analyses, prognostic factors were evaluated after age, sex, diabetes, previous cerebrovascular accidents and initial NIHSS score were adjusted. **Results:** There were no statistical differences of age, sex, and mode of acute thrombolytic treatment in unstable group (n=24) and stable group (n=90). Although other vascular risk factors did not show significant differences, metabolic syndrome was more frequent in unstable group. (83.3% vs. 46.7%,

p=0.001) Initial heparinization was more frequent in unstable group as well (41.7% vs. 10.0%, p<0.001). Frequency of intensive statin treatment was less in unstable group comparing to stable group (41.7% vs. 65.6%, p=0.033). The metabolic syndrome, initial heparinization and no use of high intensive statin were independent risk factors for progression or recurrence in multiple logistic regression analyses. **Conclusion:** The present study suggested that metabolic syndrome could be main risk factor for progression or recurrence of stroke caused by ICA stump. Intensive statin treatment seems to be a good strategy in this disease entity for acute phase treatment or secondary prevention. On the contrary, initial heparinization is questionable for the treatment strategy.

4

Changes in the common carotid artery after radiotherapy: Wall, Calcification and Atherosclerosis

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¹Department of Neurology, Asan Medical Center, ²Department of Neurology, Chosun University Hospital, ³Department of radio-oncology, Asan Medical Center

Background & Objectives: As the long-term survival rate has improved in laryngeal cancer patients who receive radiotherapy, concerns about post-radiation complications, including carotid atherosclerosis and stroke, have increased. We followed the pathological changes of the common carotid artery (CCA) after radiotherapy and identified the associated risk factors. **Method:** Consecutive patients with laryngeal cancer who underwent radiotherapy between January 1999 and December 2009 with before and after computed tomography (CT) were enrolled. Changes in the wall-thickness, vessel and lumen areas, and the presence of calcification or atherosclerosis were compared between the initial and follow-up CT scans. Demographics, risk factors, and medication use were compared between patients with and without atherosclerosis at follow-up CT scan. **Results:** A total of 125 patients were enrolled. Several months after radiotherapy, the wall-thickness increased and lumen area decreased. These changes were not associated with vascular risk factors and were not progressive. Calcification and atherosclerosis were observed in 37 (29.6%) and 71 (56.8%) patients, respectively. Hyperlipidemia was more prevalent in patients with atherosclerosis (28.2% vs. 11.1%; p=0.02) and for a longer period post-radiation (1,887 ± 949 vs. 1,238 ± 686 days; p<0.001). Baseline cholesterol levels robustly correlated with the degree of atherosclerotic stenosis. Positive remodeling was also observed but less so in patients with calcification (p=0.02). **Conclusion:** Various types of post-radiation changes occur in the CCA. However, only post-radiation atherosclerosis is progressive and closely related to baseline cholesterol levels. Lipid-lowering agents may have potential benefits in preventing carotid artery disease progression following radiotherapy.

5

One-year effect of medical treatment in atherosclerotic middle cerebral artery stenosis measured by Transcranial Doppler

Min KIM, Kyu Sun LEE, Ji Man HONG, Jin Soo LEE

Department of Neurology, Ajou University School of Medicine

Background & Objectives: Atherosclerotic intracranial artery stenosis (ICAS) is one of the common causes of ischemic stroke. Appropriate treatment is important for preventing recurrent stroke but intracranial stenting had failed in a recent clinical trial. We evaluated the efficacy of medical treatment for atherosclerotic middle cerebral artery stenosis (MCAS) by using Transcranial Doppler (TCD). **Method:** Patients who were diagnosed as MCAS from June

2001 to April 2014 were recruited in this retrospective study. Among them, patients whose initial TCD and repeat TCD between 180 and 730 days after initial one were obtained were included. Patients who were unable to evaluate TCD due to poor temporal window and ones diagnosed with moyamoya disease or dissection were excluded in this study. Patients were divided into stenosis regression group and stenosis non-regression group by using transcranial Doppler (TCD). Regression was defined as 10% or more decrease of mean velocity of repeat TCD compared to initial one. On the other hand, progression was defined as 10% or more increase of mean velocity of follow up TCD. History of taking antiplatelet and statin medication, and various clinical factors were acquired and analyzed. Obesity was defined by waist circumference as male >90cm and female >80cm. **Results:** Among included 60 patients, 26 patients were classified into regression group and 34 were into non-regression group. Age, sex and initial NIHSS score did not differ between groups. Initial mean velocity on TCD was 164±68 cm/s in regression group and 110±64 cm/s in non-regression group (p=0.004). Follow up mean velocity was 94±40 cm/s in regression group and 147±73 cm/s in non-regression group (p=0.001). As for treatment modalities, a combination of three antiplatelets, including aspirin, clopidogrel and cilostazol, was more frequently prescribed for secondary prevention in the regression group (7 [26.9%] vs. 2 [5.9%], p=0.024). Rosuvastatin was also more frequently prescribed in the regression group (12 [46.2%] vs. 6 [17.6%], p=0.017). Twenty one patients were classified into progression group and 39 were into non-progression group. There was no significant difference in age, sex and initial NIHSS score. Initial mean velocity on TCD was 102±47 cm/s in progression group and 151±75 cm/s in non-progression group (p=0.004). Follow up mean velocity was 164±63 cm/s in progression group and 102±57 cm/s in non-progression group (p=0.001). Obese patients were more frequent in progression group than in non-progression group (16 [76.2%] vs. 17 [43.6%], p=0.015). Regarding treatment modalities, both groups had similar tendency as regression and non-regression groups but statistical significance somewhat lacked. **Conclusion:** In this study, regression was more observed in atherosclerotic MCAS patients taking triple antiplatelet therapy and rosuvastatin. Therefore, aggressive medical therapy such as triple antiplatelet and high-intensity statin therapy is thought to be beneficial in ICAS patient. On the other hand, obese patients appeared to more frequently have progression. Life style modification should be also important to treat ICAS.

Scientific Session 2

Neuroscience

• 시간: 14:20~15:20 • 장소: Convention Hall C (4F)

1

Increased glucose uptake in the hemisphere with more severe ischemia among mice with right common carotid occlusion: luxury metabolism, not luxury perfusion

Jin Soo LEE

Department of Neurology, Ajou University School of Medicine

Background & Objectives: In the right common carotid artery occlusion (rCCAO) mouse model, 18F-fluorodeoxyglucose positron emission tomography (FDG PET) revealed a defect in glucose uptake in the relevant ipsilateral brain hemisphere at 8 weeks after the operation, as observed in a previous study. However, some of those mice showed high uptake in the entire ipsilateral hemisphere on FDG PET at 1 week from our preliminary study. We aimed to evaluate whether this phenomenon is associated with luxury perfu-

sion and which cellular factors influenced this PET finding. **Method:** Immediately after C57BL/6 mice underwent FDG PET 1 week after rCCAO operation, their cerebral blood flow was examined using laser Doppler flowmetry. Subsequently, they were sacrificed, and histological analyses and western blot were performed. Mice were grouped into those with high uptake on FDG PET (Group 1) and those without (Group 2). For histological analyses, the same number of mice was randomly selected from both groups. **Results:** Overall, the frequency of the presence of high uptake in the entire ipsilateral hemisphere on FDG PET was 12.3% among 65 operated mice. Among them, 33 mice underwent laser Doppler flowmetry (6 in Group 1 and 27 in Group 2). Cerebral blood flow assessed by flowmetry tended to be lower in Group 1 than in Group 2 (right to left ratio [%], 36.4 ± 21.8 vs. 58.0 ± 24.8 , $p=0.059$). Neuronal death was observed in 3 of 6 mice in Group 1 on cresyl violet staining, whereas it was not observed in any mouse in Group 2. Glial fibrillary acidic protein expression on western blot, indicating astrocyte activation, was higher in the right hemisphere in Group 1 than in Group 2 ($180.0 \pm 21.0\%$ vs $100.0 \pm 21.7\%$, $p<0.01$). The densities of glucose transporter 1 on immunohistochemistry ($177.2 \pm 84.5\%$ vs $100.0 \pm 21.8\%$, $p=0.039$) were higher in the right hemisphere in Group 1 than in Group 2. **Conclusion:** The high uptake in the right hemisphere on FDG PET was not related to luxury perfusion but rather to low cerebral blood flow. This more severe ischemic condition increased the glucose uptake, and astrocyte activation appeared to be involved in this phenomenon. Glucose transporter 1 was more activated, and it helped to increase the uptake. This phenomenon can be termed "luxury metabolism."

2

FUS abnormalities in ALS patient-derived induced neurons

Su Min LIM¹, Young-Eun KIM², Ji Young CHOI³, Sung Hoon KIM³, Ki-Wook OH³, Chang-Seok KI², Seung Hyun KIM³

¹Department of Translational Medicine, Hanyang University, ²Department of Laboratory Medicine and Genetics, Samsung Medical Center, ³Department of Neurology, Hanyang University

Background & Objectives: Mutations in the DNA/RNA-binding protein Fused in Sarcoma (FUS) are a cause of amyotrophic lateral sclerosis (ALS). In ALS patients, FUS mutations lead to a neuronal cytosolic mislocalization and abnormal accumulation of the protein. ALS patient fibroblasts or induced pluripotent stem cell (iPSC)-derived neurons are used as potential reliable model of ALS-associated FUS (ALS-FUS). However, neuronal pathological signature is not fully identified in patient fibroblasts and generating iPSC-derived neurons of ALS patients require relatively intricate procedures. **Method:** Here we report the generation of disease-specific induced neurons (iNeurons) by repressing a single RNA binding polypyrimidine-tract-binding (PTB) protein from the patient fibroblasts with FUS mutations. Among the seven different FUS mutations found in Korean ALS patients, three of them located at the C-terminal nuclear localization signal (NLS) region of the protein (Q519E, G504WfsX12, R495X) were processed for the generation of iNeurons. **Results:** Aberrant cytoplasmic mislocalization of FUS were detected in these patient-derived iNeurons and accumulation of cytoplasmic FUS granules were formed upon stress induction. Notably, iNeurons showed a loss of nuclear FUS and accumulation of cytoplasmic FUS which recapitulated neuronal pathological features shown in the patient autopsy case. **Conclusion:** Easy and rapid production of iNeurons recapitulating the FUS pathology enables more proficient disease-relevant modeling for understanding the pathophysiology of ALS.

3

The emergence of an abnormal form of long-term potentiation in aged Alzheimer's disease mouse model

Seong-Min CHOI¹, Seonghoo HUH², Soo-Ji BAEK², Kyung-Hwa LEE³, Daniel J WITCOMB⁴, Jihoon JO¹, Dong Hyun KIM⁵, Man-Seok PARK¹, Byeong C KIM¹

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Background & Objectives: Mouse models of Alzheimer's disease (AD) have been developed to study the pathophysiology of A β toxicity that causes severe clinical symptoms such as memory impairment in AD patients. However, there are inconsistencies between studies using these animal models, in terms of the effects on synaptic plasticity, a major cellular model of learning and memory; whereas some studies find impairments in plasticity, others do not. **Method:** Here, we showed that long-term potentiation (LTP) in the AD mouse model Tg2576 is impaired at its adult age around 6 - 7 months old, but appears again at aged 14 - 19 months old. **Results:** In the aged Tg2576, we found that density of parvalbumin (PV)-expressing interneurons is significantly decreased in hippocampal CA1-3 region, and LTP inhibition or reversal mediated by NRG1/ErbB signaling, which requires ErbB4 receptors in PV interneuron, is impaired. Inhibition of ErbB receptor kinase in adult Tg2576 rescues LTP but impairs depotentiation as shown in aged Tg2576. **Conclusion:** Our study suggests that hippocampal LTP reemerges in aged Tg2576, however in an insuppressible form due to impaired NRG1/ErbB signaling, possibly by the loss of PV interneuron.

4

Characterization of white matter injury in a rat model of chronic cerebral hypoperfusion

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¹Department of Neurology, Konkuk University School of Medicine, ²Department of Radiology, Konkuk University School of Medicine, ³Department of Rehabilitation Medicine, Konkuk University School of Medicine

Background & Objectives: Chronic cerebral hypoperfusion can lead to ischemic white matter injury resulting in vascular dementia. To characterize white matter injury in vascular dementia, we investigated disintegration of diverse white matter components using a rat model of chronic cerebral hypoperfusion. **Method:** Chronic cerebral hypoperfusion was modeled in Wistar rats by permanent occlusion of the bilateral common carotid arteries. We performed cognitive behavioral tests, including the water maze task, odor discrimination task, and novel object test; histological investigation of neuroinflammation, oligodendrocytes, myelin basic protein, and nodal or paranodal proteins at the nodes of Ranvier; and serial diffusion tensor imaging. Cilostazol was administered to protect against white matter injury. **Results:** Diverse cognitive impairments were induced by chronic cerebral hypoperfusion. Disintegration of the white matter was characterized by neuroinflammation, loss of oligodendrocytes, attenuation of myelin density, structural derangement at the nodes of Ranvier, and disintegration of white matter tracts. Cilostazol protected against cognitive impairments and white matter disintegration. **Conclusion:** White matter injury induced by chronic cerebral hypoperfusion can be characterized by disintegration of diverse white matter components. Cilostazol might

be a therapeutic strategy against white matter disintegration in patients with vascular dementia.

5

Neuroprotective effects of acetyl-L-carnitine against oxygen-glucose deprivation-induced neural stem cells death via the PI3K pathway

Hojin CHOI, Hyun-Hee PARK, Seong Wan PARK, Kyung-Pil OH, Kyu-Yong LEE, Young Joo LEE, Seong-Ho KOH

Department of Neurology, Department of Neurology, Hanyang University Guri Hospital

Background & Objectives: Oxidative stress is the main cause of neuronal cell death during cerebral infarction, which can lead to severe morbidity and mortality. In general, the neuroprotective therapies applied after ischemic stroke have been unsuccessful, despite many investigations. Acetyl-L-carnitine (ALCAR) plays an important role in mitochondrial metabolism and in modulating the coenzyme A (CoA)/acyl-CoA ratio. We investigated the protective effects of ALCAR against oxygen-glucose deprivation (OGD) in neural stem cells (NSCs). **Method:** After treatment with several concentrations of ALCAR under OGD, we measured cell viability by MTT (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide) assay, trypan blue staining. To evaluate the effects of ALCAR on proliferation of OGD-injured NSCs, we performed BrdU labeling and colony formation assays. And, western blotting for the evaluation of effect on intracellular signaling proteins was achieved. **Results:** ALCAR protected NSCs against OGD by reducing apoptosis and restoring the proliferation rate. It did this by increasing the expression of a number of survival-related proteins such as phosphorylated Akt (pAkt), phosphorylated glycogen synthase kinase 3b (pGSK3b), B-cell lymphoma 2 (Bcl-2) and Ki-67 in NSCs that had been injured by OGD. It also reduced the expression of death-related proteins such as Bax, cytosolic cytochrome C, cleaved caspase-9 and cleaved caspase-3. **Conclusion:** We conclude that ALCAR exhibits neuroprotective effects against OGD-induced damage to NSCs by enhancing expression of survival signals and decreasing that of death signals.

Patients with less than 3 years of follow-up duration were excluded to rule out atypical or secondary parkinsonism. PDD was defined according to the DSM-4 and PD-MCI by impaired performance (>1.5 SD) on at least one of five cognitive domains. Five scorable tests (forward digit span, Boston Naming Test, Rey Complex Figure, Seoul Verbal Learning Test and phonemic word test) were chosen as a baseline assessment to represent five cognitive domains including attention, language, visuospatial, memory and frontal/executive functions, respectively. Those tests were evaluated between PDD converters and non-converters in non-demented and MCI patients with PD. **Results:** Among total 790 patients who underwent a comprehensive neuropsychological test, 476 (60.3%, 164males) patients with PD were included. Among them, 41 patients had become to PDD converters. There were no statistically significant differences of interval from symptom onset to first visit and follow-up period between PDD converters and non-converters. PDD converters showed older age at onset (66.1±7.5 vs 60.4±10.1), lower education level (5.4±4.6 vs 7.9±5.0) and MMSE score (23.8±3.7 vs 26.4±3.2), and higher initial MCI frequency (65.9% vs 40.9%). Patients that converted to dementia showed more deficits on measures of language, visuospatial and memory at baseline than non-converters. Among 205 PD-MCI patients, 27 converted to PDD. Patients that converted to PDD showed more deficits on only visuospatial function than non-converters. **Conclusion:** Visuospatial function was found to be a domain most related to PDD conversion in non-demented patients with PD. It was suggested that the performance of test with posterior cortical basis be more related to conversion to dementia in patients with PD. Prospective longitudinal studies will be needed to confirm that.

2

The earliest findings of comfortable gait in de novo Parkinson's disease: gait hypokinesia and effect of levodopa

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¹Department of Neurology, Soonchunhyang University Gumi Hospital, Soonchunhyang University School of Medicine, ²Department of Neurology, Korea University Guro Hospital, Korea University College of Medicine

Background & Objectives: Gait dynamics in de novo Parkinson's disease (PD) have not been fully elucidated yet, although those in advanced PD have been relatively well investigated. We designed to study the characteristic features for spatiotemporal parameters of gait in de novo PD patients under drug naïve as well as levodopa trial condition. In addition, we aimed to analyze gait parameters according to the subtypes including tremor-dominant type (TDT) and non-TDT group. **Method:** We prospectively recruited de novo PD patients and healthy subjects. Spatiotemporal parameters were measured using computerized analysis with GAITrite system during comfortable gait. In PD group, the Unified Parkinson's Disease Rating Scale (UPDRS) and gait parameters were examined in both drug naïve and levodopa trial (100mg) condition. **Results:** Twenty four patients with de novo PD and 27 healthy controls (matched for age, sex, and height) were finally recruited for the study. Among PD patients, TDT type was 14, intermediate type was 2, and postural instability gait difficulty (PIGD) type was 8, respectively. Compared to the controls, patients with de novo PD showed the decrease of stride length in med-on as well as med-off condition. Furthermore the stride length, adjusted by height, correlated with PIGD score, but not with tremor, rigidity, bradykinesia, or total motor scores. Compared to the controls, drug naïve PD demonstrated decreased walking speed, reduced swing phase, and increased double support time. Levodopa trial resulted in shortening of stride time and increase of cadence to compensate walking speed. Besides, swing phase of de novo PD was more variable than that of the controls in both med-off and med-on state. However, gait asymmetry was not different between the groups. Notably,

Scientific Session 3

Movement Disorder I

• 시간: 14:20~15:20 • 장소: Emerald Hall A (3F)

1

Neuropsychological tests and MCI subtypes more related to dementia in non-demented patients with Parkinson's disease

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Department of Neurology, Dong-A University College of Medicine

Background & Objectives: Dementia is one of the most disabling symptoms in patients with Parkinson's disease (PD). Although several factors such as older age, longer disease duration are regarded as predictors of dementia in Parkinson's disease (PDD), there is still a variety of opinions about vulnerable neuropsychological characteristics and subtypes of mild cognitive impairment (MCI) for PDD in non-demented patients with PD. The aims of this study were to investigate the neuropsychological tests and MCI subtype more related to PDD conversion in non-demented PD and PD-MCI patients. **Method:** This is a retrospective longitudinal follow-up study comparing initial neuropsychological tests between the PDD converters and non-converters. PD patients who had been available to comprehensive neuropsychological test at their early visiting were included from the Dong-A Alzheimer and Parkinson Registry.

we found that those observations were more prominent in non-TDT type, compared to TDT type. **Conclusion:** We found that gait hypokinesia (decrease of stride length) was related with PIGD, but not with tremor, rigidity, or bradykinesia in de novo PD patients. As compensatory mechanism for the decreased walking velocity, gait with levodopa showed the increase of cadence by shortening stride time in drug naive PD. Moreover, those phenomena were more remarkable in non-TDT type rather than TDT type. Taken together, our observations may indicate that gait dynamics in PD is related with axial symptom-associated mechanism.

3

Neuropsychologic and radiologic comparisons in patients with parkinson's disease dementia in terms of onset age

Younggwang KIM¹, Dongkyun LEE², Jae Jung LEE¹, Jee Hyun HAM¹,
Byoung Seok YE¹, Jong-Min LEE², Young Ho SOHN¹, Phil Hyu LEE¹

¹Department of Neurology, Yonsei University College of Medicine, ²Department of Biomedical Engineering, Hanyang University

Background & Objectives: Cognitive dysfunction in Parkinson's disease (PD) is significant non-motor symptom affecting patients' performances of daily living and caregiver burden. Clinically, early-onset PD patients are known to show slower disease progression and lower rates of dementia. However, there are few studies concerning about timing of cognitive dysfunctions in Parkinson's disease dementia (PDD). In the present study, we analyzed whether the neuropsychological profiles and patterns of cortical atrophy differ depending on onset of cognitive decline in PD patients. **Method:** We assessed 116 patients with Parkinson's disease dementia from the Movement Disorders and Dementia outpatient clinic at Yonsei University Severance Hospital from March 2007 to December 2014. Depending on diagnosed age of PDD, the patients were divided into two groups with cutoff value of 70 years. Of the 116 patients with PDD, 39 patients were under 70 years old (Young onset PDD, YOPDD) and 77 patients were over 70 years old (Late onset PDD, LOPDD). Each patient underwent history taking, laboratory exams, physical, neurologic examination, neuropsychologic test, and conventional brain Magnetic Resonance Image (MRI) scans. Duration of disease and memory complaints was based on the interview with patients and caregivers living with patients. When analyzing neuropsychologic test, the scores of these quantifiable cognitive tests were classified as abnormal when they were below the 16th percentiles of the norms for the age-, sex-, and education-matched normal subjects. 71 patients underwent three-dimensional T1-weighted MRI scans. Among these patient, 25 patients were under 70 years old. We recruited 121 healthy age- and sex-matched normal controls who had no history of neurologic diseases and no abnormalities on neurologic examinations. This group of patients exhibited no objective cognitive dysfunctions on the mini-mental state examination (MMSE) and neuropsychological tests (SNSB). These patients were divided into two groups with cutoff value of 70 years to compare with their age-matched PDD groups. 49 were allotted to the young controls and 72 were allotted to the old controls. We excluded patients with focal neurologic deficits, evidences of focal brain lesions, diffuse white matter intensities, multiple lacunes in the basal ganglia by MRI, or other past medical comorbidities which could contribute to cognitive decline. Possible medical comorbidities were excluded by laboratory tests, including thyroid function test, vitamin B12 and folic acid levels, and VDRL test. The χ^2 and Mann-Whitney U tests were used for categorical and continuous variables, respectively. The effect of diagnosis (PDD versus control) and age (<70 versus \geq 70), and their interaction on neuropsychological tests and cortical thickness was assessed with analysis of covariance (ANCOVA), controlling for educational years, HTN, DM, dyslipidemia. **Results:** Age at onset in the YOPDD was 64.6 ± 4.3 years and in the LOPDD was 75.5 ± 4.2 years. No significant differences between two groups in gender, MMSE scores, education

duration, PD duration, levodopa equivalent dose and duration of memory complaints. LOPDD patients showed poorer UPDRS motor score ($p=0.005$). In neuropsychologic test, no significant interaction effects between EOPDD and LOPDD in language, visuospatial function, frontal-executive functions. However, YOPDD group exhibited poorer performance in backward span ($p=0.011$) and visual recognition tests ($p=0.002$) when adjusting the effect of aging. In cortical thickness analysis, YOPDD group exhibited more cortical thinning involving anterior cingulate gyrus with a lower threshold of uncorrected $p < 0.001$. However, no area remained significant after FDR correlation ($p < 0.05$). **Conclusion:** Our findings show that earlier cognitive decline in PDD may be predominant in attentional deficit and related frontal dysfunction, which are correlated with cortical thinning in anterior cingulate gyrus.

4

Retinal thinning correlates with clinical severity in multiple system atrophy

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Background & Objectives: Current management of multiple system atrophy (MSA) is merely symptomatic and biomarkers for early detection of the disease and monitoring progression are still in unmet need. Optical coherence tomography (OCT) is a non-invasive imaging technique capable of imaging the retina with high resolution. To investigate retinal thickness changes in MSA patients according to the clinical severity, and also to the 2 subtypes of MSA. **Method:** A total of 36 MSA (27 MSA-p and 9 MSA-c) patients and 71 control subjects underwent general ophthalmologic examination and optical coherence tomography (OCT) scans. Peripapillary retinal nerve fiber layer (RNFL) thickness and perifoveal retinal thickness were separately analyzed. Both eyes were examined and within-subject inter-eye correlations were accounted by using generalized estimating equation model. Clinical severity of MSA patients were evaluated by using unified MSA rating scale (UMSARS) and global disability score (GDS). **Results:** MSA patients showed significantly decreased superior, inferior, superotemporal and inferotemporal RNFL thickness and showed significant perifoveal thinning in the superior and inferior outer sectors compared to control. Both the RNFL and perifoveal thinning were more marked and widespread in MSA-P than MSA-C patients. The UMSARS scores and the GDS showed a consistent and significant negative correlation with perifoveal and peripapillary thickness. **Conclusion:** RNFL and perifoveal retinal thinning was observed in MSA patients, which was significantly correlated with the clinical severity. Further studies are warranted to investigate the clinical and functional consequence of retinal thinning in MSA and if such retinal changes can act as a biomarker to monitor the progression of the disease.

5

The origin of T2* contrast in human Substantia Nigra: A Postmortem Validation Study at 7T MRI

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Background & Objectives: High field magnetic resonance imaging (MRI)-based delineation of substructures of the substantia nigra (the pars reticulata, SNr; the pars compacta, SNc) and visualization of its inner organization are promising in the evaluation of structural changes associated with neurodegenerative diseases but still necessitate rigorous ex vivo validations with corre-

sponding histological information. **Method:** Multiple slices from two post-mortem substantia nigra (SN) tissues were imaged at 7T MRI with T1 and T2* imaging protocols and subsequently stained with Perl's Prussian blue (ferric iron), Klüver-Barrera (myelin and cell bodies), tyrosine hydroxylase (dopaminergic neurons), and calbindin immunohistochemistry serially. With precise co-registration of MRI and histology, we investigated the association between T2* values and quantitative histology. **Results:** We found that the paramagnetic hypointense lesions with decreased T2* values within dorsolateral SN corresponded well to the clusters of neuromelanin-pigmented neurons, although not accompanied by sufficient amounts of stainable iron. These neuromelanin-rich zones were distinct from the hypointense ventromedial SNr regions with high iron pigments. We also identified the ventral hypointense layers between the SN and the crus cerebri, which were not coextensive with the SN, but extended partially into the posterior part of the crus cerebri overlapping with densely myelinated fibers, while the boundaries between the SNr and the SNc were difficult to draw with T2* imaging. **Conclusion:** It is observed that nigral T2* imaging at 7T can reflect the density of neuromelanin-containing neurons as the metal-bound neuromelanin macromolecules may decrease T2* values and cause hypointense signal in T2* imaging.

Scientific Session 4

Muscle and Nerve I

• 시간: 14:20~15:20 • 장소: Emerald Hall B (3F)

1

Pharmacokinetic parameters and the outcome of Guillain-Barré syndrome after intravenous immunoglobulin treatment : Preliminary report of DEMIAN (DELta seruM IgG As a Novel biomarker for treatment outcome of Guillain-Barré syndrome) project

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Background & Objectives: Intravenous immunoglobulin (IVIg) has been accepted as a standard treatment of Guillain-Barré syndrome (GBS). However, until recently, the most effective regimen for GBS treatment remains to be unknown. This study aimed to investigate the overall pharmacokinetics of IVIg in GBS treatment and identify useful parameters for prediction of treatment outcome in GBS. **Method:** From September 2014 to July 2015, we enrolled GBS patients who undertaken IVIg treatment from 7 university-based hospitals in Korea. GBS was diagnosed by experienced neurologist at each hospital. Only cases of IVIg infusion within initial 2 weeks were enrolled in this study. To calculate pharmacokinetic parameters, serum IgG (sIgG) level was measured 4 times across the disease course from each enrolled patient. By utilizing special software (PKSolver), we calculated two ratio of delta serum IgG level [delta sIgG 1 (%) which means concentration between initiation of IVIg and termination of IVIg; delta sIgG 2 (%) which means concentration between termination of IVIg and 2 weeks after IVIg initiation], maximal serum IgG level, effective half-life during the treatment (eT1/2), and area under the

curve. We compared and correlate each parameter with various clinical scores for GBS severity. **Results:** Total 26 GBS patients were enrolled in this study. Median age was 47 years old (22 - 81). Proportion of GBS subtypes were following in order of decreasing frequency: AMAN (42%), AIDP (31%) atypical regional variants (27%). Of pharmacokinetic parameters, eT1/2 was significantly longer in GBS showing mild disability of locomotion. Thus, patients capable of "unaided walk" (GBS disability score ≤ 2) at nadir, as well as the initial presentation, showed significantly longer eT1/2 than patients not capable of "unaided walk" [eT1/2 at initial (days) 18.6 ± 14.6 vs 9.1 ± 4.4, P = 0.03; eT1/2 at nadir (days), 22.0 ± 16.1 vs 8.5 ± 3.1, P = 0.02]. In bivariate correlation between each pharmacokinetic parameters and various clinical scores disclosed no significance association. However, there was some tendency of inversely linear relationship between delta sIgG 1 (%) and GBS disability score at convalescent period of disease. **Conclusion:** This preliminary data showed that pharmacokinetic parameters may reflect either bioavailability of IVIg or disease activity of GBS. Of these, eT1/2 was associated with the disease severity during both initial and nadir stage. These findings suggest that ideal regimen of IVIg would be different among each patient with GBS and should be individualized according to disease severity. It is inferred that IVIg booster therapy can be considered as an additional therapy for severe neurological impairment or disability in GBS predicting poor prognosis.

2

Long-term clinical course and outcome of myasthenia gravis associated with thymoma

Il-Nam SUNWOO

Department of Neurology, Sunwoo & Cho Neurology Clinic, Professor Emeritus Yonsei University College of Medicine

Background & Objectives: There are many clinical reports about post-surgical prognosis on MG with and without thymoma but very rare about the post-surgical clinical course. It is very hard to see the long term post-surgical clinical course if many doctors with different philosophy are involved on post-surgical medical therapy. My personal MG database, I think, would be appropriate to know the long term clinical course and prognosis, and followings are results of analyzing this database. **Method:** There were 434 cases (21.8%) of thymoma in my database of 1,991 MG patients and can be divided to 3 groups; 386 of thymectomy after the onset of MG, 25 of MG onset later thymomectomy and 23 of non-thymectomy. The mean total duration of follow-up after the diagnosis of MG was 7.7 years and 7.3 years post-thymectomy. The last outcome of MG was defined using MGFA postintervention status and the long term clinical course of each patient was analyzed retrospectively. **Results:** The long-time prognosis could be defined in 9 of 23 non-thymectomy patients. Other 14 cases were not treated in this hospital (NT 10)) or recently operated (4) to know the prognosis or clinical course. One patient who visited ER in the respiratory failure expired before appropriate treatment. Four patients were died due to progression of thymoma in 6-10 years later. Until now 4 patients were surviving for 4-14 years after the diagnosis. The final outcome of MG at the last visit was PR in 3, improved in 4 and unchanged 1. The thymomectomy was performed 411 cases: 237 in YUMC and 174 in other hospitals. Mean duration of medical treatment after the thymectomy in other hospital was 1.6 year (53 for less than 1 year, 45 for 1-3 years and 76 for more than 3 up-to 24 years). In these 411 thymectomy patients, 45 cases (10.9%) revealed metastatic lesions on the follow-up radiological study at 0.5-15 years (mean; 6 years) after thymectomy. The clinical status of MG associated with thymoma tended to be worse than MG without thymoma except the pre-thymectomy MG group. The last outcome was determined in 359 patients of the followed-up duration for more than 2 year and results were followings : CSR 76 (21.2%), PR 113 (31.5%) and MM 53 (14.8%),

Imp 88 (24.5 %) and Death; 34 (9.2%). Post-surgical death (actually vegetable state) occurred in one due to massive hemorrhage during the operation. Causes of death were MG crisis in 4, thymoma progression and/or complications of treatment of thymoma in 12, worsening of both MG and thymoma in 2, and other diseases not related to MG or thymoma in 10. The final outcome of MG would be changed depending the follow-up duration because the clinical course of MG was different in each case. **Conclusion:** Analyzing my MG database, the long term prognosis would be changed by the clinical course depending on the follow-up duration. The long term clinical course of MG associated with thymoma was variable and un-expectable. Physicians had to remember that the remission (CSR or PR) for more than 1 year did not mean the permanent remission in some patients.

3

Human SCN4A N440K zebrafish model of nondystrophic myotonia

Tai-Seung NAM¹, Se-Young LEE¹, Kyung-Wook KANG¹, Seung-Ho KANG¹, Seung-Han LEE¹, Seong-Min CHOI¹, Ki-Hyun CHO¹, Seok-Yong CHOI², Myeong-Kyu KIM¹

¹Department of Neurology, Chonnam National University Medical School, ²Department of Biomedical Sciences, Chonnam National University Medical School

Background & Objectives: Mutations in SCN4A, coding for the skeletal muscle sodium channel Nav1.4, cause altered channel behavior that lead to a heterogeneous group of heritable, nondystrophic disorders including paramyotonia congenita characterized by exercise- and cold-induced myotonia. **Method:** To gain insight into the pathophysiological basis for the observed phenotypic heterogeneity of human SCN4A N440K mutation, we developed a novel transgenic zebrafish model of human nondystrophic myotonia. Compulsory exercise test (CET) was designed under the assumption that a strong enough water current renders the fish being exercised obligatory, like in treadmill test, and vulnerable to the disease phenotypes which can be differentiated from normal behavior of zebrafish. **Results:** The behavior of the fish at the water temperature of 24°C or 18°C (for cold-induced myotonia) was recorded and the entire video film was analyzed frame by frame. The SCN4A N440K transgenic zebrafish showed abnormal behaviors in CET that mimic exercise- and cold-induced myotonia, which is consistent with the universally accepted phenotype of paramyotonia congenita in human. **Conclusion:** This result suggests that SCN4A N440K transgenic zebrafish model can be used to understanding the pathophysiological basis of human nondystrophic myotonia and new therapeutic avenues.

4

Prevalence and incidence of myasthenia gravis in Korea: nationwide population-based epidemiological study

Hyung Seok LEE, Ha Young SHIN, Seung Min KIM

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Background & Objectives: Myasthenia gravis (MG) is the most common neuromuscular junction disorder with a prevalence of 5.3-14.0 per 100000 inhabitants in Asian countries. The incidence of these regions is well known for 0.4-2.1 per 100000. Yet the epidemiologic data of MG in Korea is not well investigated. Korean Health Insurance Review and Assessment Service (HIRA) compiled by the Korean government covers the claim data of 97.0% of the Korean population. We have previously established the population-based epidemiological data of diabetes mellitus, tuberculosis, Huntington's disease, multiple sclerosis, interstitial lung disease, idiopathic pulmonary fibrosis and venous thromboembolism from the HIRA database. The purpose of this study is to investigate the prevalence and incidence of MG in Korea using data from

the HIRA. **Method:** We have filtered the HIRA data to be used for the determination of the total MG population. Among all the payment requests between 2010 and 2014, we extracted all claims data, whose main or secondary cause of the visit was G70 or G70.0 (MG) according to the International Classification of Diseases, 10th revision. Claimed data from the neurologist, pediatrician or ophthalmologist were selected in the study. An individual was defined as a case if he or she had at least 2 or more hospitalizations or physician visits for MG between 2010 and 2014. Among them, newly diagnosed patients were operationally defined as the cases whose claims data for MG had never been made before, and also who were treated with pyridostigmine or prednisolone. For cases that occurred between 2011 and 2014, we presented the annual incidence for each year and the average incidence during 2011-2014. The annual prevalence was calculated as the number of cases of MG divided by population of each year. The population in Korea during 2010-2014 was obtained from the Korean Statistical Information Service (<http://kosis.kr/>). The institutional review board from Severance Hospital, Yonsei University Health System, waived the informed consent requirement. **Results:** Prevalence There were 5001 cases (male: 1920, female: 3081) of MG in Korea on 2010 when Population and Housing Census was conducted (total population: 47990761, male: 23840896, female: 24149865). Annual total number of MG cases steadily increased from 2010 to 2014. The male to female ratio of MG was stable (1.51-1.60) during study period. The prevalence on 2010 was 10.42 per 100000 and increased constantly in both sexes to 2014. The prevalence was higher for female than for male in all investigated years. It was higher than male in the majority of age groups except more than 85 years (Female > Male, RR 1.584, 95% CI, 1.496-1.677, P < 0.001). Both sexes showed peak prevalence at roughly 50-69 years. Incidence During period of 2011 to 2014 MG was diagnosed in 1316 patients (546 male, 752 female). The annual incidence ranged from 0.55 to 0.71 (male: 0.43-0.69, female: 0.67-0.87) during study period. The average incidence during 2010-2014 was 0.69 per 100000 per year. In most age specific groups, the incidence for female was higher than for male (Male: 0.59, Female: 0.78; RR 1.316, 95% CI, 1.180-1.468, P < 0.001). The incidence of MG steadily increased with age except more than 85 years of age group. For both sexes combined, the highest incidence was seen in the group older than 55 years. Incidence of infantile group MG (0-4 years) seemed higher than age group of 5-14 years. **Conclusion:** The prevalence of MG is 10.42 per 100000 and the incidence is 0.69 per 100000 per year in Korea. We found the characteristics of prevalence and incidence of MG that is consistent with study from neighboring Asian countries.

5

Identification of de novo variants by trio-based whole exome sequencing and functional analysis of candidate genes in Korean patients with sporadic ALS

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Background & Objectives: Amyotrophic lateral sclerosis (ALS) is an adult-onset neurodegenerative disorder characterized by progressive loss of cortical, bulbar, and spinal motor neurons over a time course of approximately 3 to 5 years from symptom onset. Approximately 10% of patients are considered as familial ALS (fALS) and often show autosomal dominant pattern of inheritance, while the remaining are apparently sporadic (sALS) cases. To date, a number of genes have been identified in patients with ALS including SOD1,

TARDBP, FUS, C9orf72 and so on. However, the genetic backgrounds have been elucidated in only two third of fALS cases and ~10% of sALS cases. Recently, de novo variants have been identified in various neurological and developmental disorders including autism spectrum disorder, schizophrenia, and ALS. This study aims to identify de novo variants by trio-based whole-exome sequencing and functional analysis of candidate genes in Korean patients with sALS. **Method:** Fourteen sALS and their unaffected parents trios were subjected to whole exome sequencing (n=42). First of all, pathogenic variants were screened for known ALS-related genes. Then, we tried to find de novo variants that both parents were homozygous for the wild type allele while the proband was heterozygous for the variant allele. After identification of the genes with de novo variants, functional analyses were performed for those genes with high priority scores by ToppGene software or other reasons. **Results:** No pathogenic variants in known ALS-related genes were found in any of the 14 sALS trios. Interestingly, 9 de novo variants of unknown significance (VUS) were found in 8 trios including 6 missense, 1 in-frame deletion and 1 nonsense variant. Allele frequency of all de novo VUSs were lower than 0.1% in dbSNP141, 1000 Genome Project, and Exome Aggregation Consortium. All variants were not detected in 100 ethnically-matched normal control and in-house disease control subject. Prioritization analysis by ToppGene software revealed that one of the eight genes showed significant priority scores with previously known ALS genes (overall p-value < 0.05). When we screened this gene in 184 unrelated sALS patients, two more missense variants were identified in two patients, respectively. Functional analysis of two genes showed that de novo variants in these genes might disturb normal function and show abnormal protein aggregations in diverse ALS model systems including directed converted induced neuronal cell model from skin fibroblast, patient's own plasmacytoid dendritic cells and a Drosophila ALS model. **Conclusion:** We successfully applied trio-based whole exome sequencing to identify novel ALS genes in Korean sALS and could found that at least one or more genes might be related to sALS.

Scientific Session 5

Epilepsy I: Clinical Epileptology

• 시간: 14:20~15:20 • 장소: Diamond Hall (3F)

1

Short and long-term mortality in status epilepticus

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Background & Objectives: Status epilepticus(SE) is an urgent disease in neurology. It is defined that continuous or intermittent seizures lasting more than 5 minutes, without full recovery of consciousness between seizures. Over the decades, there are many studies that analyze acute mortality and cause for mortality in SE, but there are not sufficient studies including long-term mortality in SE. Therefore, we analyzed both short and long-term mortality, causes and predictors of death in patients with SE. **Method:** We retrospectively identified 152 patients who were treated due to convulsive or/and non-convulsive SE in Asan medical center from January 1989 to December 2010. All patients were over 15 years old. We excluded 23 patients who had underlying diseases which may cause SE or death itself and who was lost to follow up. Finally, total 129 patients were included in this analysis. Medical records of patients were reviewed in detail and information on the cause of death was collected from the National Statistics Office database. **Results:** Among 129 patients, CNS in-

fection was the most common cause of SE (70 cases, 54.2%), followed by known seizures (40 cases, 31.0%). Long-term and short-term mortality were significantly higher in patients with an etiology of encephalitis (42.9%, P=0.003). Additionally, patients with de novo seizure showed higher mortality compared with those who had a pre-exist seizure disorder (41.5%, P=0.002). Other predictors were patients initially presented with generalized tonic-clonic seizure(38.5%, p=0.005), patients who were not alert at initial neurologic exam(40.4%, p=0.000) and patients who were treated with midazolam (44.2%, P=0.032), with pentobarbital (52.3%, P=0.000) and with mechanical ventilator (44.5%, P=0.000). 41 patients of 129 were died (31.8%) because of acute complication of SE (32 cases, 78.0%), underlying disease (5 cases, 12.2%), suicide (2 cases, 4.9%), accident (1 case, 2.4%) and unknown cause (1 case, 2.4%). **Conclusion:** It is the first study about long-term mortality in SE in South Korea. Our results indicate that encephalitis, de novo seizure patients, initial generalized tonic-clonic seizure, patients who were not alert at initial exam, treatment with coma therapy and treatment with mechanical ventilator are the predictors of death in SE. Also, mortality in acute period is higher than mortality in long-term period.

2

Clinical efficacy and safety of levetiracetam in epilepsy

Sang Jun KO, Sae Young LEE, Sang Hun KIM, Kang Ho CHOI, Tai Seung NAM, Sung Min CHOI, Man Seok PARK, Seung Han LEE, Byeong C KIM, Myeong Kyu KIM, Ki Hyun CHO

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Background & Objectives: An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity. Epilepsy is a disease characterized by an enduring predisposition to generate epileptic seizures and by neurobiological, cognitive, psychological condition. The treatment of epilepsy depends on appropriate classification of seizure type and the epileptic syndrome. The mechanism of Levetiracetam (LEV) is that it binds to synaptic vesicle protein 2A(SV2A) and thereby modulates one or more of its actions, affecting neural excitability. In contrast to traditional therapy, LEV has no drug interaction, lower proportion of binding to serum protein, and wide safety margin without requirement for serum drug monitoring. LEV has efficacy and favorable tolerability as adjunctive for partial seizures in adult and pediatric patients. Recently, It is used to treat generalized seizure with mono or adjunctive therapy. The aim of this study is to analyze the efficacy of LEV in each seizure types and epileptic syndromes **Method:** Four-hundred and six epilepsy patients who were treated with levetiracetam from 2007 and 2011 were initially included in this retrospective study, and 266 patients were eligible for analysis. To analyze the efficacy, we compared the seizure frequency before and after the introduction of levetiracetam to the antiepileptic regimen of each patients and also collected data of adverse effects based on our electronic medical records. **Results:** The mean age of patients was 32 years, and males accounted for 45% of the total patients. In patients with GTC, secondary generalization, and CPS, the seizure frequencies were lowered with the introduction of levetiracetam but it was not statistically significant in the CPS group (GTC : 1.53/month vs 0.57/month, p <0.05), secondary generalization : 2.19/months vs 1.58/month, p = 0.014, CPS : 2.19 vs 1.83, p = 0.06). When the comparison was made between the epileptic syndromes, the analysis revealed significant decrease of seizure frequencies in JME (1.3/month vs 0.45/month, p = 0.04), LRE (2.07/month vs 1.66/month, p = 0.017), stroke (1.509/month vs 0.36/month, p <0.05), and undiagnosed seizures (1.77/month vs 0.95/month, p <0.05). In patients with initial monotherapy with levetiracetam, 84.2% of patients showed decrease of seizure frequency. The proportion of patients with decreased frequency was lower in patients under levetiracetam with combined antiepileptic regimens; it was 48.5% in patients tak-

ing 2 anti-epileptic drugs(AED), 69.8% in patients with 3 AEDs, and 51.4% in patients under 4 AEDs. We also studied side effect of levetiracetam. 60 patients of 266 have side effect. The most side effect is dizziness(21 of 66), next is somnolence(17 of 66). 17 of 66 patients have Neuropsychiatric side effect such as psychosis, aggression or depression. **Conclusion:** In patients with undiagnosed GTC, IGE, or GTC with stroke, levetiracetam monotherapy may be a reasonable and effective choice. Levetiracetam was also effective in TLE.

3

Prognostic factors of status epilepticus to predict outcomes including seizure control and future development of epilepsy

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Background & Objectives: Status epilepticus (SE) is a neurologic emergency with various mortality rates of 7-39% and frequently poor neurologic outcomes even in survivors. It is very important for neurologists to predict prognosis in the early stage of treatment, but clinical factors to predict outcomes still controversial. This study was aimed to identify various prognostic factors of SE, especially related to the short-term seizure control and future development of epilepsy. **Method:** Patients were recruited based on the discharge database of the patients who had been hospitalized as SE and performed EEG monitoring at intensive care unit in Ewha Womans University Mokdong hospital from July 2007 and June 2015. Total 117 patients who met the criteria were enrolled so far, and their medical records for the age onset, etiology, comorbidity, consciousness level before treatment, overall duration of SE, brain imaging, visual interpretation of EEG and quantitative EEG indices including band powers in different frequency bands were reviewed and analyzed for score development of seizure outcome. Clinical outcomes were classified in four categories based on short-term seizure control and future development of epilepsy: poor outcomes without control of SE including death, severe neurologic deficit with future development of intractable epilepsy, good seizure outcomes with seizure-free and/or minimal neurologic sequelae. **Results:** In 117 patients, 65 were male (55%) and 53 were female (45%), age ranged between 9 and 93 (mean 60.1 ± 20.7) years old. Overall 48.3% of patients (57/117) showed poor control of SE including death in 13% (16/117). Older age and unconsciousness before treatment were strong predictive factors of short-term poor control of SE and long-term development of intractable epilepsy. Outcomes based on seizure control were poor control of SE including death (13.5%), severe neurologic deficit with future development of intractable epilepsy (21.9%), mild to moderate disability with good seizure control (33.8%), and seizure-free with or without minimal neurologic sequelae (16.9%). Important predictors of seizure outcome were CNS infection as an etiology, initial lower consciousness level, and EEG findings of burst suppression or epileptiform discharge. Etiologies predicting poor outcomes were CNS infection (15.2%, 18/117) or hypoxic brain damage (5%, 6/117) compared with other etiologies such as sepsis (0.8%, 1/117), metabolic encephalopathy (7.6%, 9/117), and acute cerebrovascular attack (0.8%, 1/117). In comparison, low antiepileptic drug level (12.7%, 15/117) or alcohol withdrawal (5.9%, 7/117) were related with good seizure outcomes. **Conclusion:** Our study characterized the high risk factors for short-term poor seizure control and long-term development of intractable epilepsy in patients presenting SE. Age, initial consciousness level and underlying etiology were important clinical factors for determining prognosis of SE. Quantitative EEG might be useful as an additional predictor of future development of intractable epilepsy in patients with SE.

4

Risk factors and clinical outcomes associated with seizures following open heart surgery

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Background & Objectives: The aim of this study is to investigate the incidence and outcome of postoperative seizures manifesting a variety of semiology, and to determine their risk factors in the perspectives of before, during, and after surgery in patients undergoing open heart surgery with tranexamic acid (TXA). **Method:** We enrolled consecutive adult patients, older than 15 years, undergoing open heart surgery with cardiopulmonary bypass at Samsung Medical Center from January 1, 2012 to December 31, 2013. The medical and surgical records of patients following open heart surgery were reviewed to collect demographic and clinical information. Records for consultation with the departments of neurosurgery and neurology during hospitalization were also reviewed in all patients enrolled to identify the incidence of symptomatic stroke and seizures following open heart surgery. In the group of postoperative seizures, all hospitalization, outpatient, and emergency room records of patients, where existing after the hospitalization for surgery, were reviewed through May 31, 2015 for analysis of seizure recurrence. **Results:** A total of 799 patients who had no history of seizures underwent open heart surgery with cardiopulmonary bypass during the period. New onset seizures developed in 22 patients (2.8%) and symptomatic stroke occurred in 55 patients (6.9%) among the total patients enrolled. We analyzed 213 randomly selected patients without postoperative seizures and 22 patients with postoperative seizures. In the preoperative variables, significantly higher blood levels of PT, blood urea nitrogen (BUN), and creatinine, as well as lower blood levels of hemoglobin, hematocrit, eGFR, and albumin were found in patients with seizures than in those without seizures. Furthermore, the seizure group was more likely to have a history of previous cardiac surgery, and comorbid renal and hepatic diseases than the control group. After the surgery, the postoperative seizure group was revealed to have more cerebrovascular events with no significant differences in types of stroke. Length of hospital stay after operation was significantly longer in patients with seizures than in those without seizures. The result of multiple logistic regression analysis showed that newly developed acute stroke and comorbid hepatic diseases were identified as risk factors of seizures following open heart surgery after controlling for the other factors. Patients in the seizure group had their first seizure on a median of 1.5 days postoperatively, and over 70% of patients (77.3%) had recurrent seizures after the first seizure. Almost half of them (10 patients, 45.5%) had generalized seizures as their first semiology, and four patients (18.2%) with complex partial seizures had dialeptic seizures without prominent movement of the body. After the initial seizure event, the rate of seizure recurrence was 9.1%, 18.2%, and 27.4% at 1, 3, and 15 days, respectively. Median time of seizure recurrence was revealed to be 27 days, within which is the part of the curve showing the greatest increases in the recurrence rate of seizures. **Conclusion:** Our study shows that 2.8% of patients had postoperative seizures following open heart surgery with TXA treatment. Most of the postoperative seizures were found to be recurrent, and hospitalization was found to be a quantitative and qualitative burden on patients with postoperative seizures. We also found that patients with comorbid hepatic disease, and newly developed stroke after surgery are likely to have seizures postoperatively. Close observation of not only abnormal movements, but also conscious changes and neurological examinations with neuroimaging studies, are necessary to detect postoperative seizures and elucidate their etiology in patients following cardiac surgery. After the first postoperative seizure, continuous neurologic intervention might be required in the early, high risk stage of seizure recurrence.

5

The HLA-A*2402/Cw*0102 haplotype is associated with lamotrigine-induced maculopapular eruption in the Korean population

Jangsup MOON¹, Jun-Sang SUNWOO¹, Jung-Ick BYUN¹, Jung-Ah LIM¹, Tae-Joon KIM¹, Jung-Won SHIN², Soon-Tae LEE¹, Keun-Hwa JUNG¹, Ki-Young JUNG¹, Kon CHU¹, Sang Kun LEE¹

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Background & Objectives: The use of lamotrigine (LTG) can be limited by the occurrence of cutaneous adverse drug reaction (cADR) that range from maculopapular eruption (MPE) to the more severe Steven-Johnson syndrome and toxic epidermal necrolysis. A few HLA-related genetic risk factors for carbamazepine-induced cADR have been identified. However, the HLA-related genetic risk factors associated with LTG-induced cADR are not well known yet. **Method:** We performed HLA-genotyping in 50 Korean epileptic patients, including 21 patients presenting LTG-induced MPE and 29 LTG-tolerant patients. Upon the clinical identification of MPE, LTG was immediately discontinued to prevent the patients from experiencing severe cutaneous adverse reactions (SCARs), such as SJS/TEN. The LTG-tolerant group was composed of patients who had received ≥ 300 mg/day of LTG and had a documented medical history of LTG serum levels >10 mcg/ml without any cADR during minimum 1 year of LTG treatment. **Results:** A significant association between the HLA-A*2402 allele and LTG-induced MPE was identified, in comparison with the LTG-tolerant group (OR 4.09, $P=0.025$) and the general Korean population (OR 3.949, $P=0.005$). The frequencies of the Cw*0102 or Cw*0702 alleles were significantly higher in the LTG-MPE group than in the Korean population, whereas the frequency of the A*3303 allele was lower. The coexistence of the A*2402 and Cw*0102 alleles was significantly associated with the LTG-MPE group than the LTG-tolerant group (OR 7.88, $P=0.007$). In addition, Cw*0701 allele was more frequent in the LTG-tolerant group than in the Korean population. **Conclusion:** These findings suggest the presence of HLA-related genetic risk factors for LTG-induced MPE in the Korean population.

Scientific Session 6

Stroke II

• 시간: 15:40~16:40 • 장소: Convention Hall B (4F)

1

Morphology of Susceptibility Vessel Sign (SVS) Predicts Clot Fragility and Recanalization

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Background & Objectives: Susceptibility vessel sign (SVS) is seen in cardioembolic stroke on T2*-MRI, which reflects RBC-rich red clot. Due to development of imaging technique, recent studies showed that SVS can be seen in noncardioembolic stroke as well. Recent studies showed that more proximal and longer clots are difficult to treat, leading to a worse outcome as compared with shorter and distal clots. However, there is lack information about correlation between SVS and clot composition. We hypothesized that there are distinct morphological difference between RBC-rich red clots and platelet-rich white clots on SWI image, and by which outcome of recanalization can be pre-

dicted more precisely than before. **Method:** We collected consecutive patients who was performed endovascular treatment between February 2010 and June 2015 in Seoul national university hospital. We retrospectively analyzed pre-treatment susceptibility weighted images (SWI) for evaluation of clot diameter, length, and location, and analyzed conventional angiography for evaluation of puncture-to-begin-to-recanalization time, puncture-to-full-recanalization time, and thrombolysis in cerebral infarction (TICI) score. Patients with severe artifacts on SWI were excluded. **Results:** We analyzed 76 consecutive acute stroke patients (mean age 67 years, 61.8% of male) treated with endovascular treatment, with median puncture-to-begin-to-recanalization time and puncture-to-full-recanalization time of 45.8 (5-205) and 78.1 (10-205) minutes, respectively. The SVS was present in 61 (80.3%) patients with median clot diameter and length of 5.0 (2.3-9.0) mm and 12.0 (2.6-30.8) mm, respectively. Puncture-to-begin-to-recanalization time was shorter in SVS+ patients, but had no statistical significance (48.2 min vs. 33.0 min, p -value 0.07). There was significant difference in puncture-to-full-recanalization (102.5 min vs. 72.3 min, p -value 0.03) between SVS+ and SVS- patients. Among SVS+ patients, time spent in intervention is decreased with clot diameter (35 min in diameter ≥ 6 mm vs. 17 min in diameter < 6 mm, p -value < 0.05). 13 (86.6%) of 15 patients had TICI score $\geq 2b$ in clot diameter ≥ 6 mm and 33 (71.7%) of 46 patients in clot diameter < 6 mm. Among SVS- patients, 9 (60.0%) patients had TICI score $\geq 2b$. **Conclusion:** Our data showed that there are invisible clots in SWI image which is more difficult to recanalize by endovascular treatment. Among SVS+ patients, clot diameter is associated with clot fragility and outcome of recanalization in acute stroke.

2

Different features of anterior circulation and posterior circulation dissections

Min-Gyeong KIM, Jung-Im GWON, Jun LEE

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Background & Objectives: Cervicocerebral arterial dissections are an important cause of stroke in young and middle-aged patients. The features of vascular images and risk factors might be different according to the location of cervicocerebral artery dissection. We investigate whether the risk factors, neuroimaging features, and imaging outcomes are differ according to the dissection site (anterior versus posterior circulation). **Method:** Consecutive patients who presented with ischemic symptoms and underwent brain vascular imaging within 7 days after the onset of symptoms were enrolled. Demographic characteristics, putative risk factors, imaging findings, and type of antithrombotic medication (antiplatelet versus anticoagulation) were assessed between two groups according to the site of dissection (anterior circulation versus posterior circulation). The baseline and follow-up vascular images (6 months or 1 year after the onset of symptoms) were compared to determine the prognostic difference in the reverse of the lesion. **Results:** A total of 117 patients ($n=32$ with anterior circulation dissection and $n=85$ with posterior circulation dissection) were eligible for these analysis. The median delay from symptom onset to baseline vascular imaging was in a day (IQR 1, 2). Patients with posterior circulation dissection were older (OR = 1.53 by increasing 10 years old [1.02-2.03], $p=0.038$) and more frequently had a dissection associated with exercise or neck manipulation (OR = 4.83 [1.2-18.1], $p=0.019$) compared to patients with anterior circulation dissection. Arterial stenosis or occlusion was the most common imaging features (94% in anterior circulation dissection versus 86% in posterior circulation dissection, $p=0.345$). Aneurysm or pseudoaneurysm was identified in 9.4% of anterior circulation dissection and 24.7% of posterior circulation dissection ($p=0.067$). Among patients with posterior circulation dissection vertebral artery was the most common lesion site (64.7% [53-74%]). Abnormal high signals on T1-weight-

ed MR by the fat suppression technique was more common (OR = 3.36 [1.272-8.884], $p=0.014$) and double lumen on the vascular images was more frequently identified in patients with posterior circulation dissection (OR = 4.01 [1.54-10.78], $p=0.005$) compare to patients with anterior circulation dissection. Complete reverse on the follow-up images at the 6 months or 1 year was identified in 9 patients with anterior circulation dissection and 11 patients with posterior circulation dissection (45% versus 22%, $p=0.054$). Neither anticoagulation nor antiplatelet demonstrated significant differences between the frequency of the complete reverse on the follow-up vascular images in patients with anterior circulation and posterior circulation dissection (33.3% versus 21.4%, $p=0.613$ for anticoagulation; 44.4% versus 22.7%, $p=0.087$ for antiplatelet). **Conclusion:** These results substantiate the difference in the risk factors and radiologic features according to the dissection site. There was no difference in efficacy of antiplatelet and anticoagulant drug at complete reverse of the lesion after cervicocerebral artery dissection. Our data suggest that a prospective study of prognostic factors for outcomes in clinical events and imaging finding in patients with cervicocerebral artery dissection.

3

Ischemic Stroke in Critically Ill Patients with malignancy

Jeong-Am RYU¹, Oh Young BANG², Daesang LEE¹, Jinkyong PARK¹, Jeong Hoon YANG¹, Gee Young SUH¹, Joongbum CHO¹, Chi Ryang CHUNG¹, Chi-Min PARK¹, Kyeongman JEON¹

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Background & Objectives: Cerebrovascular disease may occur commonly in cancer patients. Systemic cancer is related to ischemic stroke (IS) via various mechanisms. Among them, cancer-related mechanism such as coagulopathy and tumour occlusion is a common cause of IS in cancer patients. In addition, treatment modalities including chemotherapy, radiotherapy and hormone therapy may also increase the risk of IS. Little is known, however, about the cause and outcome of stroke in critically ill cancer patients. Therefore, we conducted a retrospective observational study with critically ill cancer patients admitted to medical intensive care unit (ICU) to evaluate the clinical characteristics of IS developed during their ICU stays and to identify predictors of IS when underwent brain magnetic resonance imaging (MRI) for acute neurologic symptoms or signs. **Method:** All consecutive patients were retrospectively evaluated who underwent brain MRI for suspicion of IS with acute abnormal neurologic symptoms or signs developed in the oncology medical ICU of Samsung Medical Center from March 2010 to February 2014. A multiple logistic regression analysis was used to identify independent predictors of IS. **Results:** Over the study period, a total of the 88 patients who underwent brain MRI for suspicion of IS, 43 (49%) patients had a final diagnosis of IS. Multiple lesions were more common (41%) than single lesion (8%). Conventional stroke mechanism (CSM) were shown in 16 (37%) patients, including cardioembolism ($n=6$), large-vessel atherosclerosis ($n=2$), small-vessel occlusion ($n=2$), and others ($n=6$). However, IS without CSM ($n=27$, 63%) was more common than IS with CSM. In addition, brain metastases were newly diagnosed in 7 (8%) patients. There was no significant difference in gender, type of malignancy, recent chemotherapy, vascular risk factors, and serum D-dimer level at the time of suspicion of IS. Thrombotic events were more common in IS group than in non-IS group ($P=0.028$). However, patients finally diagnosed with IS had more hemiparesis symptom at the time of suspicion of IS ($P=0.001$). Non-IS group had more seizure ($P=0.001$). After adjusting for potential confounding factors, hemiparesis (adjusted OR 5.339; 95% CI, 1.521-19.163) was one of associative factors of IS in patients who underwent brain MRI for suspicion of IS in the oncology medical ICU. In contrast, seizure

was inversely associated with IS (adjusted OR 0.141; 95% CI 0.027-0.736). There were no significant differences in length of stay in the ICU ($P=0.299$), ICU mortality ($P=0.114$), or in-hospital mortality ($P=0.085$) between patients with or without IS. **Conclusion:** Approximately half of critically ill cancer patients diagnosed with IS when underwent brain MRI scanning for suspicion of IS during their ICU stay. Multiple territorial lesions were commonly observed in stroke patients. Acute altered mentality was most common cause to scan brain MRI. Half of patients who showed acute altered mentality were revealed as IS and newly diagnosed brain metastasis was relatively rare. Although, it is generally difficult to differentiate between IS and non-IS based on symptom or sign alone, caution and careful examination are required when hemiparesis symptom is observed in critically ill cancer patients.

4

Asymmetrical cerebral white matter hyperintensities and predilection of lacunar infarct

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Background & Objectives: Pathogenesis of white matter hyperintensities (WMHs) is largely unknown, although accrued pathological studies have shown that arteriolosclerosis, lipohyalinosis, and myelin pallor are major features of WMHs that are mainly resulted from chronic ischemia. Recent studies using serial MRI scans have indicated that the distinction between WMHs and lacunar infarcts seems to be less clear than previously thought. In addition, these studies have suggested that WMHs increase the susceptibility of brain to developing of lacunar infarct. Asymmetrical distribution of WMHs between cerebral hemispheres is not uncommon in patients with acute ischemic stroke. If WMHs increase the susceptibility of lacunar infarct and lacunar infarct turns into WMHs, symptomatic lacunar infarct may preferentially develop in the hemisphere with more severe WMHs. In the present study, we tested this hypothesis in the patients with acute supratentorial lacunar infarct confirmed by diffusion weighted MRI. **Method:** 315 patients with supratentorial acute lacunar infarct were included. WMHs on MRI were graded by a neurologist blinded to the study hypothesis using modified Scheltens Scale (0-30 for each hemisphere). Because there is no consensus on the definition of WMHs asymmetry, we used diverse cut points of the difference of modified Scheltens Scale between hemispheres ($\geq 2, \geq 3, \geq 4, \text{ or } \geq 5$). Chi-square test was used to examine the relationship between asymmetrical WMHs and the location of lacunar infarct in each definition of asymmetrical WMHs. **Results:** Mean age was 63.7 (standard deviation 13.0) and 190 (60.3%) were men. For all patients, the location of lacunar infarct was left in 175 patients (55.5%) and right in 140 (44.4%), suggesting the preference of left-sided stroke as expected. When the difference of modified Scheltens Scale ≥ 2 was used as the definition of asymmetrical WMHs, 71 patients had right-dominance WMHs, 70 left-dominance and 174 symmetric. In patient with right-dominance WMHs, 42 (59%) had lacunar infarct in right hemisphere whereas, in those with left-dominance WMHs, 49 (70%) had lacunar infarct in left hemisphere ($p=0.002$). When the difference of modified Scheltens Scale ≥ 3 was used as the definition, the preference of lacunar infarct was stronger. Among 34 patients having right-dominance WMHs, 27 (79%) had lacunar infarct in right hemisphere, whereas among 43 patients having left-dominance WMHs, 29 (67%) had lacunar infarct in left hemisphere ($p<0.001$). When the difference of modified Scheltens Scale ≥ 5 was used as the definition, 8 patients had right-dominance of WMH, 6 left-dominance and 301 symmetric. In patients with right-dominance WMHs, 7 (88%) had lacunar infarct in right hemisphere, whereas, in those with left-dominance WMH, 5 (83%) had lacunar infarct in left hemisphere ($p=0.019$). **Conclusion:** We found that lacunar infarct preferentially develops in the side with more severe WMH. Our findings corroborated the notion that

small lacunar infarcts turn into WMHs. Further studies are warranted to decipher a temporal relationship between lacunar infarct and WMH.

5

Impact of the academic year-end changeover on stroke outcomes

Hye Yeon WON¹, Tai Hwan PARK¹, Sang-Soon PARK¹, Youngchai KO², Soo Joo LEE², Kyung Bok LEE³, Jun LEE⁴, Moon-Ku HAN⁵, Kyu Sik KANG⁶, Jong-Moo PARK⁶, Yong-Jin CHO⁷, Keun-Sik HONG⁷, Jay Chol CHOI⁸, Dong-Eog KIM⁹, Dae-Hyun KIM¹⁰, Jae-Kwan CHA¹⁰, Joon-Tae KIM¹¹, Byung-Woo YOON¹², Ji Sung LEE¹³, Juneyoung LEE¹⁴, Hee-Joon BAE⁵

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Background & Objectives: There are concerns about lower quality of care and poorer outcomes when new trainees (e.g. residents) start at the beginning of the academic year in teaching hospitals. We evaluated outcomes among patients with an ischemic stroke (IS) or transient ischemic attack (TIA) admitted in March versus other eleven months of the year. **Method:** We evaluated 12,761 ischemic stroke and 1,317 TIA patients who admitted to 11 nation-wide hospitals in Korea between August 1, 2011, and July 31, 2014, identified from the Clinical Research Center for Stroke-5 (CRCS-5) registry. The main outcomes were poor functional outcome defined as modified Rankin Scale 3 to 6 at 3 months and death at 3 months after stroke. Multilevel linear regression analyses with hospital specified as a random effect were conducted. **Results:** Of 14,078 eligible patients, 1,250 (8.9%) were admitted in March and 12,828 during the remaining months. There was no difference in demographics or stroke severity between the two groups. Thrombolysis performed in March was comparable to that in other months (15.4% vs. 16.6%; Fisher's, p=0.318). March admission was not associated with either of poor functional outcome at 3 months (adjusted OR, 95% CI; 1.08, 0.93-1.26) or death (0.94, 0.71-1.24). However, poor functional outcome was likely to increase (1.13, 1.00-1.27) after expanding to include both February and March as effect of academic year-end changeover. **Conclusion:** Patients with IS or TIA admitted to teaching hospitals during March had similar outcomes. However, the rate of stroke outcome might be influenced by the predefined period of the beginning of the academic year.

Scientific Session 7

Demyelinating Disorders

• 시간: 15:40~16:40 • 장소: Convention Hall C (4F)

1

Differences in the clinical and laboratory features according to the lesion length in non-infectious myelitis

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Background & Objectives: Non-infectious myelitis consists of post infectious

myelopathy, multiple sclerosis (MS), neuromyelitis optica (NMO), myelopathy related with connective tissue or anti-phospholipid antibody and paraneoplastic syndrome. There is no specific radiological finding to the diagnosis of each disease. We investigated to determine if any difference in the clinical characteristics of the patients of the non-infectious myelitis as compared with the lesion length. **Method:** This study is a retrospective cross-sectional study. We included patients who had more than 18 years old and diagnosed of non-infectious myelitis. We excluded patients who had traumatic spinal cord lesion, compressive myelopathy, infectious myelopathy, spinal cord tumor and vascular disease of spinal cord. Clinical data of the patient are collected retrospectively using electronic medical record. Each patient was divided into long and short segments group according to the lesion length by MRI findings. Long segments group was defined as having lesion length more than three vertebral segments and short segments group had less than three vertebral segments. We compared the clinical and laboratory features between both groups. For comparison of clinical information among each group, we used independent T test and Wilcoxon rank-sum test for continuous variables, and X2 test and Fisher's exact test for nominal variables. All statistical significance level was as p<0.05. **Results:** Of a total of 38 non-infectious myelitis patients, long and short segments group were 23 and 15 patients, respectively. Demographic features including onset age, median time from first symptoms to diagnosis had no significant difference between two groups. The CSF oligoclonal band was positive in 7 patients (30%) among long segments group, but none of patients in short segments group showed oligoclonal band. The CSF oligoclonal band positive rate had significant difference statistically between two groups (p<0.05). Mean values of CSF protein of long and short segment group were 47.05±26.80 mg/dL (mean± SD) and 29.77 ±12.48 mg/dL, respectively. CSF protein is much higher in long segments group for average 17.28 mg/dL [95%CI(1.02-33.54 mg/dL)] than short segment group. Anti-aquaporin-4 (anti-AQP4) antibodies were detected in 5 patients. Of 5 patients with anti-AQP4, three patients belonged to long segments group. The etiology of 20 patients (long segments; 12 patients, short segments; 8) with non-infectious myelitis could not be determined. Nine patients were diagnosed with atopic myelitis (long segments; 5 patients, short segments; 4). The etiology of myelitis between two groups had no significant difference. **Conclusion:** Our study showed significant differences in CSF oligoclonal band positive rate and CSF protein value between long and short segments group. As both CSF oligoclonal band and protein reflect the inflammatory condition, we assumed that more severe inflammatory responses were developed in non-infectious myelitis patients with long segments lesion. However the difference in lesion length did not give help to predict the etiology of non-infectious myelitis.

2

Autoantibodies and Autoimmune Co-morbidities in Seropositive Neuromyelitis Optica Spectrum Disorder

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Background & Objectives: Neuromyelitis optica spectrum disorder (NMOSD) is a heterogeneous disease group, which includes optic neuritis or longitudinally extensive myelitis associated with systemic autoimmune disease. The disease-specific autoantibody NMO-IgG found at 2004, but strong association of neuromyelitis optica (NMO) and systemic autoimmune disease has reported before the discovery of NMO-IgG. And also, various organ-specific or non-organ specific autoantibodies are found in the sera of NMO patients. The aim of this study was to investigate autoantibodies and autoimmune co-morbidities

in seropositive NMOSD, compared to the multiple sclerosis (MS) and seronegative NMOSD. **Method:** The medical records of NMO, NMOSD and MS patients were retrospectively reviewed. Demographic feature and laboratory findings were reviewed. All tested autoantibodies, including antinuclear antibody (ANA), anti-SSA/Ro antibody, anti-SSB/La antibody, anti-phospholipid antibody (APL), rheumatoid factor (RF), anti-double strand DNA (dsDNA) antibody, anti-neutrophil cytoplasmic antibody (ANCA) and thyroid autoantibody were analyzed. In seropositive NMOSD, we reviewed the titer of NMO-IgG, if tested. If specific autoimmune disease was diagnosed by neurologist, rheumatologist or endocrinologist, we described it. We also analyzed annualized relapse rate (ARR) and Expanded Disability Status Scale (EDSS) for compare the prognosis of NMOSD. **Results:** From January 2007 to June 2015, 94 NMOSD and 207 MS patients were included. Among 94 NMOSD patients, 75 patients were seropositive for NMO-IgG and 19 patients were seronegative. Among 75 seropositive NMOSD patients, 66 patients were female and 9 patients were male. Mean age of onset was 38.04 ± 13.25 years. Twenty-nine (38.7%) of patients showed positive ANA and 26.7/14.7% of patients showed positive anti-SSA/SSB antibody, respectively. Five (6.7%) patients had positive anti-dsDNA antibody and thyroid autoantibody. APL, RF and ANCA were positive in four patients (5.3%). Eighteen (24%) patients had co-morbid autoimmune disorder. Sjogren syndrome (SS) was the most common, followed by systemic lupus erythematosus (SLE) and autoimmune thyroid disease (Graves's disease or Hashimoto's thyroiditis). Other co-morbid diseases were autoimmune hepatitis, myasthenia gravis and idiopathic thrombocytopenic purpura. We compared seropositive NMOSD patients with MS and seronegative NMOSD patients. There were no significant differences between seropositive and seronegative NMOSD in autoantibodies or autoimmune disease. But when compared with MS, seropositive NMOSD has a significantly higher proportion of positive ANA or anti-SSA/SSB antibodies. MS also showed variable co-morbid autoimmune disease, but the proportion was significantly lower than NMOSD (17 out of 207 MS patients, 8.5%). The type of autoimmune disease in MS also differed from NMOSD. In seropositive NMOSD, almost 2/3 of autoimmune disorders were SS or SLE. But MS showed more variable autoimmune diseases with fewer SS or SLE. Seropositive NMOSD patients were classified as two groups depending on the presence of co-morbid autoimmune disease. We compared EDSS and ARR, but there was no significant difference between two groups. The titer of NMO-IgG was also compared in these groups, but high-NMO IgG titer does not necessarily mean higher risk of combined autoimmune disorder. **Conclusion:** Autoantibodies and autoimmune co-morbidities are common in NMOSD regardless of positivity of NMO-IgG. Even compared to the MS patients, NMOSD has significantly higher co-morbid autoimmune diseases, especially SS and SLE. But even if NMOSD patients have co-morbid autoimmune disorder, it does not appear to be related higher relapse rate or worse prognosis.

3

Enhanced immunosuppressive effect of regulatory B cells in neuromyelitis optica and multiple sclerosis; an illustrative report

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Background & Objectives: Regulatory B cells (CD24+CD38+ among CD19+ B, Breg) are generally considered to be negative regulators of the immune response by producing regulatory cytokines. However, the function of Breg in the patients with CNS autoimmune disease in comparison to healthy in-

dividuals remains understudied. In this study, we investigated the differences of B cell subsets, and the function of Breg in patients with CNS autoimmune disease as well as healthy controls (HC). **Method:** Two neuromyelitis optica patients and one multiple sclerosis patient in remission state were enrolled, three healthy volunteers were recruited as healthy control (HC). To analyze B cell subsets, PBMCs were surface stained and detected by flow cytometry. CD24+CD38+ among CD19+ B cells were co-cultured 1:1:1 (0.5 x 10⁵ cells each) with autologous CD3+ T cells and CD3+CD4+ T cells and multiple cytokines were analyzed with cytometric bead array in culture supernatant for the functional study. **Results:** Compared with patient group (PG) and HC, PG had a higher proportion of peripheral Breg. Interestingly as the T lymphocytes were co-cultured with Breg, cytokines such as IL-6 (ratio=Breg+T/T x100 (%), 250% to 50.42%), TNF-alpha (102.33% to 2.12%) and IL-2 (101.94% to 27.17%) were decreased in PG compared to HC. **Conclusion:** The co-culture of Breg with T cell were induced the decrease in some pro-inflammatory cytokines in patient with CNS autoimmune diseases compared to HC. The higher proportion of Breg and the potentiation of Breg function may play an important role in maintaining remission state of CNS autoimmune disease.

4

Glycyrrhizic acid might reduce the NMO-IgG induced cell death by inhibition of the complement activation

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Background & Objectives: Neuromyelitis optica (NMO) is an inflammatory demyelinating disease in central nervous system. The pathogenesis of NMO involves binding of aquaporin4 autoantibody (AQP4-Ab) to the astrocyte, activation of the complement pathway, and subsequent breakdown of the blood brain barrier. The glycyrrhizic acid (GL) is a triterpene that is obtained from the roots and rhizomes of licorice. **Method:** Here, we report that GL can decrease the complement dependent cytotoxicity in vitro model of NMO, by inhibiting the binding of C1q complex. **Results:** Treatment of NMO patient sera (0.5, 1, 2.5, 5, and 10%) dose-dependently increased LDH release in both AQP4 overexpressed U87 cells and primary culture of mice astrocyte. Treatment of GL (0.1, 0.5, 1, 1.5, and 2 mM) reduced the complement mediated cytotoxicity of NMO IgG in a dose dependent manner. Though GL did not block the binding of NMO IgG to AQP4 overexpressed U87 cells, it reduced binding of C1q to NMO-IgG. **Conclusion:** Our result implies that GL might play a protective role in the pathogenesis of NMO, through the inhibition of the complement activation.

5

Neutralizing antibodies in Korean multiple sclerosis patients treated with Interferon-beta

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Background & Objectives: Interferon beta (IFN β) was the first disease-modifying treatment (DMT) approved for multiple sclerosis (MS) and is still one of the most commonly prescribed first-line DMTs. Patients treated with IFN β can develop neutralizing antibodies (NAbs) against IFN β , which negatively affect its therapeutic responses. We aimed to investigate prevalence of NAbs and to evaluate the impact of NAbs on the treatment response of IFN β in Korean

patients with MS. **Method:** This was a real-world setting, single center, retrospective study of MS patients treated with subcutaneous IFN β -1a and IFN β -1b in Korea. Patients with MS who were treated with IFN β at least for 6 months were enrolled. Sera, which were not influenced by acute treatment for MS, were tested for NABs using a test involving the use of division-arrested IFN β -sensitive cells in a bioassay capable of measuring IFN β bioactivity. **Results:** A total of 96 MS patients were enrolled. NABs were found in 27 (28%) patients at the single test; 22 of 66 (33%) patients treated with subcutaneous IFN β -1b and 5 of 30 (17%) patients treated with subcutaneous IFN β -1a. The positive rate of NABs was highest (39%, 18/46) in patients treated with IFN β for 12 to 24 months (range 11-104 months). Up to now, 18 of 27 patients with NABs were tested at two different time points to confirm the persistent positivity of NABs. Of 18 patients with NABs, 10 patients showed persistent positivity of NABs and 8 of them (80%) revealed disease activity (clinical relapses and/or active lesions on MRI) before and after 6 months of NABs positivity. In contrast, 2 of 8 patients (25%) who showed transient positivity of NABs showed disease activity. The probability of developing clinical relapse and/or active lesions on MRI was associated with persistent positivity of NABs. **Conclusion:** The current study is ongoing to confirm persistent positivity and titers of NABs. We will clarify whether the presence of NABs is associated with clinical and MRI disease activity, which helps early identification of patients who would benefit from a switch to alternative treatment.

such as reduced thalamic connectivity with the right postcentral gyrus and bilateral lingual gyri, and enhanced areas such as the right inferior temporal gyrus, right precuneus, and left cingulate gyrus. Among these, the connectivities in the areas such as the cingulate gyrus, precuneus, and postcentral gyrus of the treated RLS subjects, either were enhanced or reduced, toward both the strength of the controls' connectivities. **Conclusion:** The results showed that there is a drug related alteration of the resting state brain connectivity in RLS. These alterations are more related with the drug itself activated by the dopamine circuit, and there was also a tendency of change toward the patterns of the control group. We need further study within these groups to figure out the symptom improvement related findings in the brain.

2

Clinical and polysomnographic characteristics of REM sleep dependent obstructive sleep apnea in Korean adults

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Background & Objectives: Rapid eye movement (REM) sleep dependent obstructive sleep apnea (OSA) has been suggested as a subcategory of OSA in which respiratory events occur predominantly during REM sleep. REM dependent OSA has been found to be more prevalent in women than men, and more prevalent in younger patients than older patients of both sexes in Western country. They showed that the prevalence accounts for 10 to 36% of OSA and is more common in mild and moderate OSA. There were a few studies regarding REM dependent OSA in Korea, which were small sample size and had some different finding. We investigated the relationship of obstructive sleep apnea with REM dependency and to identify its clinical and polysomnographic characteristics in Korean adults. **Method:** A total of 616 adults (429 men and 187 women) with OSA were included from June 19, 2012 to November 12, 2014 in tertiary university hospital. Demographic, clinical, and polysomnographic characteristics of 616 patients undergoing polysomnography (PSG) for OSA were examined. The apnea-hypopnea index (AHI) during total sleep time, the AHI during REM (AHI-REM), and the AHI during non-REM sleep (AHI-NREM) were calculated. We defined REM dependent OSA patients if an AHI-REM was at least two times higher than AHI-NREM and REM sleep period required at least 5% of total sleep time. Patients were stratified according to the severity of disease in mild ($5 \leq \text{AHI} < 15$), moderate ($15 \leq \text{AHI} < 30$), and severe ($30 \leq \text{AHI}$) cases. The Korean version-Epworth Sleepiness Scale (KESS) Korean version-Pittsburgh Sleep Quality Index (KPSQI), Korean version-Insomnia Severity Scale (KISS), the Korean version-Beck Depression Inventory-2 (KBDI2), and Body Mass Index (BMI) were evaluated. **Results:** Of those of 616 patients with OSA, 28.2% (n = 174) of cases fulfilled the REM dependent OSA criteria. The REM dependent OSA was presented in 47.9% (n = 105) of patients with mild OSA (n = 219), in 32.3% (n = 54) of those with moderate OSA (n = 167), and in 6.5% (n = 15) of those with severe OSA (n = 230). The REM dependent OSA prevalence was 48.7% (91/187) in women and 19.3% (83/429) in men. The mean age of the REM dependent OSA was younger than no-REM dependent OSA in both sexes (men, 45.3 ± 12.4 vs. 50.7 ± 14.31 , $p = 0.002$; women, 56.1 ± 9.4 vs. 59.5 ± 12.4 , $p = 0.04$). The PSG data indicated that the overall AHI was significantly lower in the REM dependent OSA group than the no-REM dependent OSA group (overall AHI, 15.39 ± 9.68 vs. 34.67 ± 23.95 , $p < 0.001$). REM dependent OSA showed increased total sleep time (352.66 ± 65.87 vs. 336.43 ± 67.70 ,

Scientific Session 8

Sleep

- 시간: 15:40~16:40
- 장소: Emerald Hall A (3F)

1

Drug related alternation of the resting-state brain connectivity in restless Legs Syndrome

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Background & Objectives: There are few resting-state connectivity studies of RLS. In our previous study, we found some connectivity changes between the thalamus and the other brain areas in RLS patients during the asymptomatic periods. The aim of our study is to examine drug related alteration in the resting-state connectivity in RLS patients. **Method:** Resting state fMRIs were obtained from 32 idiopathic, age and sex matched RLS patients (16 drug naïve; 16 on dopamine agonists) and 16 controls, in the morning when RLS symptoms are absent. The resting state connectivity was measured by a seed based method using AFNI software. The bilateral thalami (ventral posterolateral nucleus) were selected as seeds, as they have previously been shown to be differentially activated during RLS symptoms. The independent t-test was used for comparing the connectivity strength between the thalamus and other brain regions, among three groups: drug naïve, drug treatment, healthy controls (FWE corrected p -value=0.05). And the connectivity strength changes of the treated RLS subjects (in regions where there were differences between the drug naïve RLS subjects and controls) were investigated in order to find the drug related effect on the RLS aberrant changes. **Results:** In RLS patients after medication compared to the controls, the connectivity between the thalamus and the bilateral frontal gyrus was enhanced, while the connectivity between the thalamus and the bilateral precentral gyrus and left postcentral gyrus were reduced. There were different areas between the drug naïve RLS and controls,

$p = 0.007$) and increased percent of REM sleep time (18.96 ± 5.81 vs. 17.01 ± 6.79 , $p < 0.001$), decreased REM sleep latency (109.0 ± 72.42 vs. 126.57 ± 107.74 , $p = 0.020$), better sleep efficiency (81.78 ± 13.90 vs. 78.84 ± 13.80 , $p = 0.018$), lower total arousal index (24.58 ± 12.81 vs. 41.39 ± 39.94 , $p < 0.001$), and higher average SaO₂ (95.25 ± 2.71 vs. 94.64 ± 2.74 , $p = 0.013$). The KPSQI of REM dependent OSA was significantly higher than that of no-REM dependent OSA (9.26 ± 4.33 vs. 8.43 ± 4.25 , $p = 0.030$). No significant differences were found in KESS, KISI, KBDI2, and BMI. **Conclusion:** This study demonstrates that the prevalence of REM dependent OSA among Korean is 28.2%. The Patients with REM dependent OSA has different clinical and polysomnographic features comparing with the patients with no-REM dependent OSA, which are similar with Western studies. The REM dependent OSA group is more common in women and younger than the no-REM dependent OSA group. The REM dependent OSA is more common in mild to moderate OSA, but sleep quality was worse than that of no-REM dependent OSA.

3

Quantitative test for primary restless legs syndrome using the current perception threshold test

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Background & Objectives: Restless legs syndrome (RLS) is a sensorimotor neurological disorder, causing an urge to move, and is usually associated with abnormal sensations in the leg, which is especially aggravated at night. The purpose of this study was to investigate the sensory dysfunction of primary restless legs syndrome patients compared with a normal group and any differences between the diurnal variation using the current perception threshold (CPT) test, which is widely used to assess peripheral sensory perception. **Method:** Thirty four primary RLS subjects and 34 healthy controls were enrolled in this study. Secondary RLS was excluded through face to face interviews and laboratory tests, if needed. Peripheral polyneuropathy was also excluded through an nerve conduction study of all subjects. We used a Neurometer[®] machine for the CPT and assessed patients at three levels: at 2000, 250 and 5 Hz stimulating both big toes. We also evaluated the severity of RLS and sleep problems of all subjects. The CPT was given two times, once during the daytime asymptomatic period and again in the evening when they had symptoms. **Results:** The mean ages of the RLS group and controls were 48.7 ± 11.8 and 46.6 ± 11.2 . The RLS group had 25 (73.5%) females and controls had 26 (76.5%) females, which showed no difference between the two groups. The mean IRLS score was 28.5 ± 4.8 . There was no significant difference in the current perception thresholds between the RLS patients and controls in both daytime and nighttime. However, in the RLS patients, the mean CPT measurements of all three Hertz levels in the evening time were lower than those of the daytime (2000Hz; 413.5 ± 80.4 vs 437.3 ± 82.0 , 250Hz; 182.1 ± 47.2 vs 199.2 ± 41.6 , 5Hz 106.6 ± 31.1 vs 124.1 ± 25.4), while there were no differences in the control group. **Conclusion:** RLS patients showed a lower current perception threshold in the evening. This finding suggested hyperalgesia of sensory perception and nocturnal predominance symptoms. The diurnal variation of hyperalgesia in RLS patients indicates a central sensory processing disturbance rather than a peripheral sensory disturbance. Therefore our findings suggest further evidence of a central pain modulation abnormality in RLS patients.

4

Blood pressure improvement with continuous positive airway pressure is associated with evening-to-morning blood pressure

variations in obstructive sleep apnea syndrome

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Background & Objectives: Obstructive sleep apnea syndrome (OSAS) is associated with hypertension (HT). OSAS patients also had a high prevalence of an unfavorable circadian pattern, compared with subjects without OSAS. Evidence to date has established that the gold-standard treatment for OSAS, positive airway pressure, has beneficial effects on the cardiovascular sequelae of OSAS. Several meta-analyses have demonstrated CPAP significantly reduced both systolic BP and diastolic BP. Certain variables, such as CPAP compliance, somnolence status and baseline BP, may modulate the strength of the effect of CPAP treatment on BP. However, it is not well known that abnormal circadian pattern of BP affect the strength of CPAP treatment independently in patients with OSAS. Therefore, this study aimed to determine that the BP improvement with CPAP is associated with Evening-to-Morning blood pressure variations in OSAS, independently. **Method:** Patients aged over 18 years referred to a sleep clinic underwent polysomnography (PSG) with measurement of baseline BP at two time points (evening: just before lights out on the night of the sleep PSG study, morning: just after waking following the PSG study) and morning BP after CPAP titration. Circadian variation was assessed using morning to evening BP ratios at PSG (RM/E). They were classified into two groups (RM/E ≥ 1 , RM/E < 1), and RM/E ≥ 1 means abnormal diurnal BP variation. BP reduction was defined as the morning BP after CPAP titration subtracted from baseline morning BP (at PSG). Student T test was used for comparisons between two groups, and paired T test was used for comparison morning BP before and after CPAP titration. Multiple linear regression analyses were performed for the association between the BP reduction with CPAP titration and diurnal BP variation (RM/E) in OSAS, independently. **Results:** SBP or DBP at any time points and SBP or DBP M/E ratios in patients with SBP or DBP M/E ratios ≥ 1 were significantly higher than in SBP M/E ratios < 1 . OSA patients with abnormal circadian pattern of BP are likely to have higher BP than others. The change in morning SBP and DBP after CPAP titration in patients with SBP or DBP M/E ratios ≥ 1 was significantly larger than in SBP or DBP M/E ratios < 1 . OSA patients with abnormal circadian pattern of BP are likely to gain the largest benefit from PAP. Lastly, the reduction of both SBP and DBP increased with SBP M/E ratios and DBP M/E ratios, respectively. Abnormal circadian pattern of BP was predicted to the change in morning SBP and DBP with CPAP titration. **Conclusion:** CPAP significantly reduced BP in patients with OSAS. OSA patients with abnormal circadian pattern of BP are likely to gain the largest benefit from CPAP in terms of a substantial reduction in BP, even after controlling for disease severity.

5

Assessment of cardiovascular risk burden in subjects with obstructive sleep apnea syndrome

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Background & Objectives: Despite large numbers of researches describing associations between obstructive sleep apnea (OSA) and cardiovascular disease (CVD), there was a paucity of studies exploring the cardiovascular risk factors regarding to sleep quality of OSA. To investigate the cardiovascular risk burden in polysomnography-confirmed OSA subjects, **Method:** We enrolled 543 OSA subjects (apnea-hypopnea index, AHI ≥ 5 /h) who participated in the medical checkup program. In addition to anthropometric measurement and

laboratory analyses, cardiovascular risk burden was assessed with carotid intima-media thickness (cIMT), coronary artery calcification (CAC), arterial stiffness as brachial-ankle pulse wave velocity (baPWV), and ankle-brachial index (ABI). One-way Analysis of variance for continuous variables and the chi-square-test for categorical variables were performed. Partial correlation analysis was applied to make analysis of correlations with PSG parameters after adjustment of age. **Results:** Participants (mean age 55.69 ± 7.65 y) were grouped by AHI severity into mild (AHI 5-15/h, N=178), moderate (AHI 15-30/h, N=179) and severe (AHI ≥ 30 /h, N=184). No significant differences were found in age, smoking (%), systolic blood pressure (SBP), intraocular pressure, cIMT, ABI and baPWV. Participants in severe group showed the greatest scores in abdominal girth (87.7 ± 6.34 in mild vs. 90.9 ± 6.59 in moderate vs. 93.0 ± 7.62 in severe, $p < 0.001$), body mass index (BMI, 24.8 ± 3.32 vs. 25.6 ± 2.36 vs. 26.6 ± 3.09 , $p < 0.001$), diastolic blood pressure (DBP, 74.7 ± 9.44 vs. 76.7 ± 9.68 vs. 79.0 ± 11.04 , $p < 0.001$), triglyceride (TG, 130.1 ± 62.52 vs. 147.4 ± 87.89 vs. 165.4 ± 99.18 , $p < 0.001$) and C-peptide (2.13 ± 0.84 vs. 2.21 ± 0.97 vs. 2.62 ± 1.27 , $p < 0.001$) level than other groups. Smoking (%) was not different among groups, however, alcohol consumption rate was the highest in severe group (73.6 vs. 77.3 vs. 85.9% , $p = 0.012$). Markers for cardiovascular risk burden (cIMT, CAC, baPWV, ABI) did not show significant differences among groups. AHI was positively correlated with BMI, abdominal girth, DBP, TG, HbA1c, and C-peptide. CAC scores were significantly correlated with sleep efficiency, sleep/REM latency and PLM index. Arterial stiffness (baPWV) was associated with arousal index, sleep efficiency, and sleep/REM latency. **Conclusion:** This cross-sectional study showed that OSA severity (AHI) did not show significant association with markers for cardiovascular risk burden that was contrary to previous studies. However, we found that poor sleep quality caused by OSA definitely affected on cardiovascular health status.

Scientific Session 9

Dementia I (Eng)

• 시간: 15:40~16:40 • 장소: Emerald Hall B (3F)

1

A new blood based biomarker in Alzheimer's Disease: Self-standard measurement of monomeric forms of A β

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Background & Objectives: Blood-based biomarkers in AD have drawn relatively less attention due to its high variation within groups and even within subjects compared to amyloid PET and cerebrospinal fluid biomarkers. However, due to high cost for PET scans and invasiveness of CSF studies, having a sensitive blood based biomarker will be very helpful for clinic and for research. We investigated whether untangling aggregated forms of amyloid beta (A β) in the blood can increase monomeric forms of A β which can be detected and enhance the diagnostic accuracy of the blood-based test. **Method:** After completion of detailed neuropsychological tests and 3T-MRI for clinical diagnosis of AD and normal cognition status, [18F]-Florbetaben amyloid PET was performed to assess in vivo amyloid deposition status of each participant. Finally, a total of 24 AD patients and 20 normal control subjects were enrolled

for the blood biomarker study. The institutional review board of Asan Medical Center approved the study protocol and a consent to participate in the study was obtained from each participant. Plasma from each subject was sampled and processed using conventional methods. After measurement of A β in untreated blood samples, KMSB600 was spiked into each aliquot of plasma to untangle aggregated forms of A β . To ultimately develop a self-standard measurement system of A β in the blood, we calculated a ratio of KMSB600 treated plasma A β concentration over untreated plasma A β concentration. The interdigitated microelectrodes (IMEs) sensor was used to detect A β in the blood by monitoring the impedance change of surface functionalized IMEs. **Results:** Baseline levels of A β was not statistically different between AD and normal control subjects. Untangling of aggregated forms of A β in AD increased levels of A β monomers to be detected by the IME sensor. The ratio of KMSB600 treated plasma A β concentration over untreated plasma A β concentration was increased up to 4-7 folds in the AD group compared to ~2 folds in a normal control group. Updated demographic information of participants and clinical implication of the dissolved monomeric forms of A β with regard to clinical and imaging biomarkers will additionally be presented. **Conclusion:** Self-standard measurement of monomeric forms of A β in the blood can be a new blood based biomarker of AD. Given the easy accessibility and clinical implication of the dissolved forms of A β in blood, current findings will potentially move the field forward.

2

Resting-state fMRI in amnesic mild cognitive impairment with and without Parkinson's disease

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Background & Objectives: To find functional correlates of amnesic mild cognitive impairment (aMCI) in patients with Parkinson's disease (PD; aMCI-PD+) and those without (aMCI-PD-) with the hypothesis that functional connectivity might differ depending on underlying pathology. **Method:** The institutional ethics committee approved this study, and informed consent was obtained. Functional connectivity was compared in 66 patients with aMCI (34 aMCI-PD- and 32 aMCI-PD+) and 29 subjects with normal cognition using resting-state functional MRI. Region of interest (ROI)-based analyses were performed with ROIs in the posterior cingulate cortex (PCC), posterior putamen, and substantia innominata (SI). **Results:** The aMCI-PD+ group exhibited decreased connectivity in the bilateral temporal gyri, and increased connectivity in the left middle frontal gyrus with the PCC ROI and decreased connectivity in the widespread posterior cortical regions with the posterior putamen ROI compared with the aMCI-PD-. No significant difference in connectivity with the SI ROI was observed between two groups. In the aMCI-PD+ group, functional connectivity between the PCC ROI and right superior temporal/left postcentral gyri positively correlated with verbal memory performance. In the aMCI-PD- group, functional connectivity between the PCC ROI and middle frontal gyrus positively correlated with general cognitive performance. In both aMCI groups, increased connectivity with the SI ROI in several regions correlated with better cognition. **Conclusion:** Different patterns of functional connectivity with the PCC and posterior putamen in aMCI-PD- and aMCI-PD+ patients may reflect different neuropathological bases of cognitive dysfunction, whereas increased connectivity with SI in both aMCI groups may reflect common compensatory brain changes.

3

Multilevel hierarchical mixed model for the prediction of longitudinal cognitive changes after ischemic stroke

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Mi-Sun OH⁶, Kyung-Ho YU⁶, Byung-Chul LEE⁶, SangYun KIM², Hee-Joon BAE²

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Background & Objectives: About 10 to 30 percent of post-stroke patients are suffered from post-stroke cognitive impairment. In the previous studies, post-stroke cognitive functions showed various trajectories during follow-up. However, these studies have not considered time-dependent variables such as stroke recurrence or various follow-up numbers and intervals. Furthermore, the detailed information of both acute and chronic neuroimaging characteristics including lesion characteristics were not simultaneously considered. Thus, we aimed to construct the prediction models for the domain-specific longitudinal cognitive changes including baseline clinical and neuroimaging variables as well as time-dependent clinical events such as stroke recurrence. **Method:** We enrolled patients who were undergone the Korean-Vascular Cognitive Impairment Harmonization Standards- Neuropsychology Protocol (K-VCIHS-NP) at least three times after acute ischemic stroke from March 2007 to September 2014 in a university hospital. Patients who had severe aphasia or hemiparesis that preclude the neuropsychological tests were excluded. Baseline demographic, cardiovascular risk factors, and index-stroke characteristics were investigated as well as the stroke recurrence during follow-up. The acute and chronic neuroimaging variables were also included as follows: lesion laterality, multiplicity, cortical involvement; white matter hyperintensities, microbleeds, lacunes, and hippocampal atrophy. Multilevel hierarchical mixed model was used for the prediction of longitudinal domain-specific cognitive changes considering the various intervals between follow-ups. For the analysis, missing values in K-VCIHS-NP were imputed using the lowest values of each corresponding test in case of poor performances. Baseline characteristics including vascular risk factors and neuroimaging parameters were set as level-one hierarchy, while time-dependent variables such as stroke recurrence and time intervals between K-VCIHS-NP were included as level-two hierarchy. **Results:** Total 187 post-stroke patients with at least three times of K-VCIHS-NP were enrolled (mean age \pm standard deviation 68.9 \pm 9.0 years, NIHSS median 4, baseline MMSE median 24). During follow-up (range 10-71 months, mean 30 months), a total of 14 (7.5%) patients had recurred ischemic strokes. For the general cognition, age, initial stroke severity, baseline MMSE scores, lesion multiplicity, and cortical involvement in acute neuroimaging were the significant predictors for longitudinal cognitive changes. As for the memory domain, age, baseline memory test scores were associated with cognitive changes. For the frontal functions, sex, diabetes mellitus, previous stroke history, baseline depression score were the significant predictors. Time-dependent event, stroke recurrence during follow-up, has not reached statistical significances in all domains. **Conclusion:** Predictors for the longitudinal cognitive changes of post-stroke patients were varied according to the cognitive domains. Stroke recurrence itself did not reach statistical significance in our analysis. Currently, we are expanding our database with patients who were undergone the K-VCIHS-NP at least twice after index-stroke.

4

Progression of Korean Mini-Mental Status Examination (K-MMSE) score in Alzheimer's disease

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Background & Objectives: Progression of Alzheimer's Disease (AD) is well known from studies regarding the natural courses, while only few studies have shown in patients on medications. Previous clinical trials addressed that cholinesterase inhibitors (ChEIs) and N-methyl-D-aspartate (NMDA) receptor blockers in Alzheimer's disease(AD) alter the rate of cognitive decline, but their long-term effects need further investigation. In this study, we aimed to demonstrate the long-term cognitive changes on Korean AD patients in clinical settings receiving pharmacological treatments and identify the associated variables affecting the rate of cognitive decline. **Method:** This was a retrospective cohort study from patients whom visited Seoul National University Bundang Hospital, between 2003 to 2013. From medical records of AD patients using ChEIs and/or NMDA receptor blockers, we determined the progression of Korean Mini-Mental Status Exam (K-MMSE) by calculating the rate of change in years from each pairs of consecutive K-MMSE assessments. The mixed random/fixed coefficient method was used for modeling predictions of K-MMSE progressions and verifying the rate modifying risk factors. **Results:** Total number of 366 patients and their 1337 assessments were included in analysis. Mean rate of K-MMSE change calculated from 971 pairs of K-MMSE per year was 1.31 points (95% CI -1.47 to -1.14) in all score ranges. From the mixed model analyses, earlier-onset disease, presence of APOE ϵ 4 allele, higher level of education, and lower initial K-MMSE scores were associated with faster cognitive decline rates. **Conclusion:** This was the first study in Korean AD patients on pharmacological treatments demonstrating the long-term progression course in clinical settings. The model acquired from our study may be of use to clinicians who encounter AD patients in long-term follow-up, by giving information on progression rate and aiding clinical decisions.

5

Changes of cognitive function in patients with mild to moderate Alzheimer's disease associated with and without white matter lesions after rivastigmine patch therapy: a multi-center, 24-week, prospective, open-label study (CAREER study)

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Background & Objectives: We aimed to evaluate and compare the changes of cognitive function as measured by ADAS-Cog in the two groups of patients with Alzheimer's disease (AD) associated with and without white matter lesions after rivastigmine patch therapy. **Method:** Patient subjects were allocated to the group of AD without white matter lesions (group 1) and the group of AD with white matter lesions (group 2) at the baseline. Periventricular and deep white matter lesions were defined by the CREDOS protocol. Periventricular white matter (PWM) lesions are classified into P1 (caps or rim <5 mm), P2 (5 mm). **Results:** Three hundred thirty-two patients were screened and 300 patients were included in this study from July 2011 to December 2014. Of these 300 patients, 206 patients were allocated to the group 1 and 94 were allocated to group 2. Intention-to-treat (ITT) group were 198 patients and they were divided to the group 1 (n=136) and group 2 (n=46) during 24-week study period. Demographic findings between the group 1 and group 2 were not different. There were no significant change difference between patients with mild to moderate Alzheimer's disease associated with (group 2, -0.23±5.98) and without (group 1, -0.62±5.70) white matter lesions after rivastigmine patch therapy on primary outcome measure of ADAS-cog (p=0.378). Group 1 showed a 0.63-point improvement from the baseline score of 10.2 on the FAB at the end of study, while those group 2 exhibited a 0.16-point worsening from the baseline score of 10.1 (p=0.037). There were no significant change difference from the baseline scores of K-MMSE, CDR-SB, FAB, CGA-NPI, ADCS-ADL, and Mini-Zarit at the end of study. The incidence of adverse events (AEs) (42.6 vs. 40.3%) and discontinuation due to AEs (vs. %) were not different in the patients on group 1 and 2. The most frequently reported AEs were skin irritation (itching, rash, eruption) in both groups (30.9 vs. 32.3%, p=0.34). **Conclusion:** There was no significant change difference between patients with mild to moderate Alzheimer's disease associated with and without white matter lesions after rivastigmine patch therapy on cognitive function. However, there is a tendency of cognitive improvement on frontal executive function in patients with AD without or minimal white matter lesions after rivastigmine patch therapy.

Scientific Session 10

Headache

- 시간: 15:40~16:40
- 장소: Diamond Hall (3F)

1

Serial assessment of cerebral blood flow correlates with disease course in migraineurs: a longitudinal follow-up study

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Background & Objectives: Migraine is conceptualized as a chronic disorder with episodic attacks. Clinical course of migraine is variable, but three representative patterns have been recognized: remission, persistence, and progression. However, it is unclear that which physiologic changes are associated with alteration of disease status. Basal cerebrovascular flow velocities have been reported to correlate with cortical activity via neurovascular coupling. We hypothesized serial assessment of cerebral flow velocities by transcranial Doppler (TCD) is associated with temporal changes of disease status in migraineurs. **Method:** Using a prospectively maintained headache registry, we identified patients who diagnosed with migraine and underwent

serial TCD tests from March 2012 to June 2015. Patients with intracranial vessel stenosis were excluded. Qualitative assessment was made at each visit: improvement, progression, and persistent course. Mean flow velocities (MFVs) of middle cerebral artery (MCA), posterior cerebral artery (PCA), vertebral artery (VA), and basilar artery (BA) and Lindegaard index (LI; ratio of ipsilateral MCA/ICA MFVs) were serially measured and compared within groups. For bilateral arteries, any side with a higher value than the contralateral artery was chosen for the analysis. Paired t-test and ANOVA were performed to compare serial MFVs within groups and between groups. **Results:** Among 130 migraineurs who completed serial TCD assessment, 119 patients were included in the analysis. With a median 16.9 (interquartile range, 13.3 - 21.9) months of follow up, 66 patients showed remission; 37, persistent course; 16, progression. Patients with remission showed serial decrement of MCA, VA, and BA MFVs (p=0.009, p=0.004, and p<0.001). In patients with clinical progression, PCA MFVs were significantly increased (p=0.022), and a similar trend was observed in MCA (p=0.065). There were no significant serial difference of all the MFVs in patients with persistent course. Between groups, changes of MCA MFVs were significantly difference (mean difference = 6.43 in patients with progression; 1.93 in patients with persistent course; -3.23 in patients with remission) **Conclusion:** Our data provide evidences that serial TCD might be a noninvasive surrogate marker of disease activity in migraineurs. Neurovascular activation caused by cortical hyperactivity might correlate with increasing susceptibility to migraine attack.

2

Thalamocortical dysconnectivity in migraine without aura: a combined fMRI and DTI study

Sung Hoon KANG, Ji Hyun KIM

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Background & Objectives: Recent advances in multimodal neuroimaging techniques have offered an opportunity to explore brain regions or networks that are likely to be involved in generation and mediation of migraine headache. In view of previous findings of thalamic and cortical involvement in migraine pathophysiology, it is surprising that there is no currently available study investigating connectivity changes between the thalamus and its connections with cortical areas in migraine. We therefore used multimodal MRIs to examine changes of the thalamic structure and thalamocortical connectivity in migraineurs. We predicted that specific thalamocortical connectivity would be preferentially dysfunctional in migraineurs, and that these abnormalities would be related to disease severity. **Method:** Consecutive 55 patients with migraine without aura (all females, mean age = 35.8 years) and 34 matched controls (all females, mean age = 34.4 years) were scanned on a 3T MR scanner by acquiring 3D volumetric images and diffusion tensor imaging (DTI) as well as resting-state functional MRI (rs-fMRI). Volumetric and shape analyses were conducted to identify morphological changes of the thalamus between the groups. DTI probabilistic tractography was employed to examine changes of structural connectivity between thalamus and 5 cortical seeds (prefrontal, motor-premotor, somatosensory, parietal-occipital, and temporal) in migraineurs compared to controls. DTI-derived metrics, fractional anisotropy (FA) and mean diffusivity (MD), were retrieved from the 5 thalamic parcels and compared between the groups. Seed-based analysis was conducted to evaluate functional connectivity changes between thalamus and 5 cortical seeds in migraineurs relative to controls. Anatomical data were then correlated with disease duration and migraine attack frequency in migraineurs. **Results:** Migraineurs and controls did not differ in thalamic volume and shape. Connectivity-based thalamic parcellation using a 'winner-take-all' approach was largely consistent with previous DTI studies. Between-group comparisons

showed reduced connection probabilities between motor-premotor cortex and bilateral ventrolateral thalamus (corrected $P < 0.001$), and between somatosensory cortex and bilateral ventral posterior-lateral thalamus (corrected $P < 0.003$) in migraineurs relative to controls. Compared to controls, migraineurs had significant FA decreases in the thalamic parcels connected with motor-premotor cortex and somatosensory cortex (corrected $P < 0.05$). In intrinsic connectivity analysis using rs-fMRI, compared to controls, migraineurs exhibited significant reductions in functional connectivity between motor-premotor cortex and bilateral anterior ventrolateral thalamus (corrected $P < 0.004$) and between somatosensory cortex and bilateral posterior ventrolateral thalamus (corrected $P < 0.002$). Both structural connectivity and functional connectivity between thalamus and prefrontal, parieto-occipital, and temporal cortices did not differ between controls and migraineurs. No significant correlations were found between MRI data and clinical data in migraineurs. **Conclusion:** Our results indicate that migraine without aura is associated with reduced functional connectivity as well as structural connectivity between somatosensory cortex and ventral posterolateral thalamus and between motor-premotor cortex and ventrolateral thalamus, implicating thalamo-sensorimotor network abnormality in the pathophysiology underlying migraine without aura. In addition, our finding of reduced FA in the thalamic subregions connecting with somatosensory and motor-premotor cortices further points to microstructural abnormality of the sensorimotor thalamus in migraine without aura. Lack of correlation between anatomical data and disease duration or attack frequency suggests that abnormal thalamo-sensorimotor network may not be a consequence of repeated attacks but a reflection of an intrinsic pathophysiologic change underlying migraine.

3

Relationship between physical activity and status of headache in episodic migraineurs using smartphone Applications-based electronic headache diary

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Background & Objectives: Exercise or physical activity is not only a trigger of headache but also a recommended behavioral therapy in migraine. We studied the status of physical activity and its association with severity of headache among migraineurs using smartphone application-based electronic (SA-E) headache diary. **Method:** Migraine patients with average 2 or more headache days per month and having personal smartphone were recruited. The patients were provided with a Headache diary app for android phone and detailed instructions and were recommending keeping the diary for 3 months. The patients were asked to write the occurrence, severity, duration, triggers of headache, and intensity and duration of exercise in the App. Disability of headache by HIT-6 was assessed at the baseline and 3 months after initial visit. We defined good physical activity (GPA) as ≥ 150 min per week of moderate or vigorous physical activity. **Results:** A total of patients were enrolled in this study and 62 participants were accomplished 3 months trial. Exercise as a risk factor for headache was listed in 3 patients on baseline survey and in 7 patients upon SA-E diary. GPA was performed by 11 patients at the baseline survey and 15 patients upon SE-E diary. Moderate or vigorous exercise was recorded on 15.8 ± 18.2 days (0-79 days) during study period. HIT-6 was 62.8 ± 6.5 at baseline and 56.8 ± 6.8 at final survey and final HIT-6 scores were not different according to the presence of GPA. Worsening of HIT-6 score more than 1 point was present in 45.2% (8/42). Proportion of worsening of HIT-6 was less frequent among patient with GPA than those without GPA (14% vs. 20%,

$p < 0.001$). **Conclusion:** GPA was not well performed among episodic migraineurs. GPA may decrease patients suffering from headache.

4

Insomnia in migraineurs is closely associated with anxiety and depression: a population-based study

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Background & Objectives: Insomnia, anxiety and depression are common complaints among migraineurs. Insomnia had been reported to be associated with anxiety and migraine. However, the association among insomnia, anxiety and depression in migraineurs has not been reported. This study is conducted to investigate the association of insomnia, anxiety and depression among migraineurs. **Method:** We selected a stratified random population sample of Koreans aged 19-69 and evaluated them by door-to-door visit and face-to-face interview using questionnaire. The questionnaire was designed to identify headache type, insomnia, anxiety and depression. We used Insomnia Severity Index (ISI) (ISI score ≥ 8), Goldberg Anxiety Scale (positive ≥ 2 at 4 screening questions and ≥ 4 at all questions) and Patient Health Questionnaire-9 (PHQ-9) (PHQ-9 score ≥ 10) for assessing insomnia, anxiety and depression, respectively. **Results:** Of 2,695 participants, 143 (5.3%), 442 (16.5%), 268 (10.0%) and 116 (4.3%) participants were classified as having migraine, insomnia, anxiety and depression, respectively. Fifty four (37.5%), 43 (29.5%) and 24 (16.7%) participants have insomnia, anxiety and depression, respectively in migraineurs. Among migraineurs with insomnia, 49.0% of them had anxiety and 29.6% had depression. Multiple logistic regression analysis adjusting sociodemographic variables including age, gender and size of residential area revealed that migraineurs with insomnia showed increased odds ratios (OR) for anxiety (OR = 5.4, 95% confidence interval [CI] = 2.3-12.6, $p < 0.001$) and depression (OR = 4.6, 95% CI = 1.7-12.2, $p = 0.002$) compared to migraineurs without insomnia. **Conclusion:** Insomnia was a common complaint among migraineurs. More than 2/3 of migraineurs reporting insomnia had anxiety or depression.

5

Headache attributed to acute pyelonephritis

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Background & Objectives: This study identified the incidence and risk factors for headache attributed to acute pyelonephritis. **Method:** The inclusion criteria were patients who were admitted with acute pyelonephritis at our hospital and ≥ 18 years of age. The following exclusion criteria were used: 1) patients who could not express their headache because of mental deterioration, 2) the presence of meningitis or meningoencephalitis, or 3) structural lesions on brain computed tomography or magnetic resonance images that could cause headache. The primary endpoint for this study was headache attributed to acute pyelonephritis as a dependent variable. The differences were analyzed using demographic and laboratory profiles as independent variables. **Results:**

A total of 479 patients met the inclusion criteria for this study, and 97 patients (20.2%) developed headache attributed to acute pyelonephritis. Patients with headache attributed to acute pyelonephritis were younger, female, and had a lower incidence of diabetes. However, laboratory profiles that reflected the severity of acute pyelonephritis were not predictive factors for headache attributed to acute pyelonephritis. Multiple logistic regression analysis demonstrated that young age and non-diabetes were independently significant variables for the prediction of headache attributed to acute pyelonephritis. **Conclusion:** We determined that headache attributed to acute pyelonephritis was relatively common, and it was related to demographic characteristics but not acute pyelonephritis severity.

Scientific Session 11

Muscle and Nerve II (Eng)

• 시간: 09:40~10:50 • 장소: Convention Hall B (4F)

1

Clinical, pathologic, genetic features of collagen VI-related myopathy in Korea

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Background & Objectives: Mutations in the collagen VI (COL6A1, COL6A2, COL6A3) cause Bethlem myopathy (BM) and Ullrich congenital muscular dystrophy (UCMD), which were believed previously in separate disease entities but now known as collagen VI-related myopathy, having broad clinical spectrum. We aimed to analyze the clinical, pathological and genetic characteristics of patients with collagen VI-related myopathy in Korea. **Method:** We studied 17 patients of 11 families, who were suspected collagen VI related myopathy or unspecified myopathy. 6 patients in 1 family were confirmed by previous report of our group and 11 patients in 10 families underwent 69 gene-targeted next generation sequencing, including collagen VI related genes. And other information of clinical manifestation, electrodiagnostic study, and muscle biopsy finding were reviewed in electrical medical records. **Results:** Of total 17 patients, mean age at the first symptoms and diagnosis was 3.6 years old and 18.4 years old. Joint contractures are found in mostly ankle joint, and patients didn't show any distal hyperlaxity. 11 patients in 6 families presented autosomal dominant inheritance. According to clinical manifestation, we divided to 2 groups, which are intermediate collagen VI-related myopathy (IM) and Bethlem myopathy (BM). 4 patients in 4 families are IM and 13 patients in 7 families are BM. Except 2 patients in 2 families, all patients can walk alone without aid. Muscle biopsy, which is performed with 10 patients of 9 families, showed increased muscle fiber size variation, degenerating fibers with endomysial fibrosis in nine muscle specimen. And muscle specimen of 1 patient showed sarcolemma specific collagen VI α deficiency in immunohistochemistry. In genetic study, according to clinical phenotype, all 4 IM (4 families) had De novo missense mutations of triple helix domain in COL6A1 gene without family history, and 11 BM (4 families) showed exon 14 splicing site mutations. And we detected two novel mutations, c.1056+1insT in COL6A1 gene and c.856-1G>C in COL6A2 gene, which is a first reported gene associated with collagen VI related myopathy in Korea. **Conclusion:** Clinically IM patients have De novo missense mutations of triple helix domain in COL6A1 gene and tendency of early onset and relatively severe progression rather than BM patients. Our findings would be helpful for understanding and finding colla-

gen VI-related myopathy.

2

The STIM1 mutations without tubular aggregate and its pathogenicity

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Background & Objectives: Ultrastructural presence of tubular aggregate is a diagnostic hall mark of tubular aggregate myopathy (TAM). Up to 10 genes have been identified in TAM, STIM1 and ORAI1 are well known as causative genes. The protein encoded by these genes are concerned with store-operated calcium entry. These autosomal dominant mutations allow constitutive calcium entry from extracellular space through calcium release activating channel (CRAC), which is a beginning of tubular aggregate. Recently, we experienced novel STIM1 (p.E249K) mutation without tubular aggregate in addition to two known mutations showing tubular aggregate and investigated its pathomechanism. **Method:** Three families with presence of STIM1 mutation were included via whole exome sequencing. C2C12 cells expressing mutated STIM1 and myotube induced from fibroblast of patient were used to measure cytosolic calcium concentration utilizing Fura-2-AM. CRAC blocker and manganese quenching image were performed to prove calcium influx from extracellular space. The Fura-2-AM intensity was measured by single-cell calcium imaging. **Results:** In light microscopic finding, tubular aggregate was not identified in one family harboring p.E249K. Patients of the other two families revealed tubular aggregate in light and electro microscope. The intensity of Fura-2-AM in C2C12 cells with mutated STIM1 and myotube from affected patient was increased in the situation of extracellular solution containing calcium. In manganese quenching image, fluorescence of Fura-2-AM was quenched in Fura-2-AM loaded C2C12 cells and myotube. Cytosolic Fura-2-AM intensity was not increased in myotube from affected individual add CRAC blocker. **Conclusion:** We confirmed altered calcium homeostasis is induced by the dominant novel STIM1 (c.745G>A) and two known mutations through activation of CRAC channels. Linkage of calcium influx with formation of tubular aggregate is another topic worth investigation.

3

Development of myasthenia gravis after thymectomy

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Background & Objectives: Myasthenia gravis (MG) may occur in patients with thymoma. Removal of thymoma may be helpful to control myasthenic symptoms. MG after thymectomy is a very rare condition. This study aimed to analyze the incidence and clinical characteristics of post-thymectomy MG. **Method:** We retrospectively reviewed the medical records of patients with MG and thymoma who underwent thymectomy at Samsung medical center. The analysis was focused on the patients with MG developed after thymectomy. **Results:** From Nov, 1995 to May, 2015, thymectomy was performed for 107 patients with MG and thymoma. Ninety-six patients were diagnosed with MG before thymectomy (pre-thymectomy MG). Eleven patients were diagnosed with MG after thymectomy. The symptoms of MG developed within 0 to 101 months after thymectomy (median; 21 months). All patients were seropositive

MG. The mean antibody titer at diagnosis of MG was 6.3nmol/L for post-thymectomy MG, and 9.0nmol/L for pre-thymectomy MG ($p=0.018$). Post-thymectomy MG patients initially presented ocular symptoms with the exception of 1 patient who started dysphagia and dyspnea. Only 2 patients used immunosuppressive agents, and 1 patient suffered from an MG crisis, which was not a post-operative crisis. **Conclusion:** About 10 percent of thymomatous MG developed myasthenic symptoms after thymectomy in our hospital series. Clinical symptoms of MG tended to be milder in post-thymectomy MG patients comparing with those in pre-thymectomy MG.

4

Predictor for secondary generalization in late onset myasthenia gravis

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Background & Objectives: Secondary generalization is one of most important clinical event for prognosis of myasthenia gravis (MG). Several factors are reported as predictor for secondary generalization such as presence of thymoma. Nowadays, concern about late onset MG (LOMG) has increased, and several different clinical characteristics between late onset MG and early onset MG have been reported. Likewise, predictors for secondary generalization may be different between late onset group and early onset group. **Method:** The study was a multi-center, retrospective study. Medical records of MG patients with onset age ≥ 20 , diagnosed between 2000 and 2014, were reviewed. Basic demographics, medical history, laboratory and imaging results of the patients were collected. The late onset MG was defined as onset after the age ≥ 50 . Clinical characteristics between the late onset and early onset groups were compared, using independent t-test for continuous variables and chi-square test for categorical variables. Logistic multivariate regression analysis was used for analyzing significant clinical predictors for secondary generalization. **Results:** Medical records of total 644 patients were reviewed. Among them, records of 632 patients with onset age ≥ 20 were finally selected. The number of the late onset MG group was 276, 43.7% of total selected patients. Compared to early onset group, late onset group showed shorter disease duration before presentation, more ocular type at onset, higher acetylcholine receptor antibody titer, and lower thymic hyperplasia rate. In late onset group, positive acetylcholine receptor antibody and steroid use before secondary generalization was significant predictor for secondary generalization. Addition to significant predictors identified in late onset group, positive in RNS test and presence of thymoma are additional predictors in early onset group. **Conclusion:** Late onset MG showed difference about predictor for secondary generalization compared to early onset MG. The comprehension about these differences would be crucial for adequate treatment of late onset MG patients.

5

Identification of pathogenic/likely pathogenic variants in inherited muscular disorders by targeted next-generation sequencing

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Background & Objectives: Inherited muscular disorders are a clinically and genetically heterogeneous group of muscle diseases caused by defects in muscle proteins. This complexity of these disorders frequently makes it difficult to

identify the causative gene accurately. In this study, we investigated the efficiency of a targeted next-generation sequencing approach for molecular diagnosis of inherited muscular disorders. **Method:** We retrospectively selected DNA samples of 209 Korean patients who with a diagnosis of muscular dystrophy, congenital myopathy, distal myopathy, metabolic myopathy and congenital muscular dystrophy. They were tested by targeted next-generation sequencing for 69 myopathy-related genes. All variants were classified as by according to the American college of medical genetics and genomics (ACMG) and the association for molecular pathology (AMP) guidelines. **Results:** The present study revealed 106 different pathogenic/likely pathogenic variants were identified in 97 (46%) patients. They consisted of 51 missense variants, 22 splicing variants, 17 frameshift variants and 16 nonsense variants. Among them, 48 variants were novel. Among 106 variants, 94 variants were classified as pathogenic/likely pathogenic variant without family study, but 11 variants were classified as likely pathogenic variants after segregation studies. Ninety-seven patients had pathogenic or likely pathogenic variants. Among them, 78 (37%) patients were clinically and genetically confirmed cases and 19 (9%) patients had only one pathogenic/likely pathogenic variant of recessive myopathy. Confirmed cases included 21 patients with DMD mutations, 9 patients with DYSF mutations, 9 patients with COL6A1 mutations, 7 patients with GNE mutations, 7 patients with LMNA mutations, 6 patients with CAPN3 mutations, 5 patients with RYR1 mutations, and 3 patients with GAA mutations. Mutations in COL6A2, DNAJB6, DNEM2, FKRP, MTM1, PYGM, SGCA, SGCG, TPM3, TTN and VCP gene were identified in each one patient, respectively. Among 19 patients with only one mutation of recessive myopathy, 14 (7%) patients had clinical phenotype compatible with identified genotype. However, two patients had two mutations of two different recessive myopathies, and three patients with one mutation of recessive myopathy had different phenotype compared with known phenotype of identified genotype. **Conclusion:** The present study confirmed not only the usefulness but also limitations of ACMG/AMP guidelines and targeted next-generation sequencing. These findings suggested that clinical and pathological analysis was as important as genetic analysis in the diagnosis of inherited muscular disorder.

Scientific Session 12

Dementia II

• 시간: 09:40~10:50 • 장소: Convention Hall C (4F)

1

The synergistic effects of amyloid and subcortical cerebral small vessel disease on the progression of lobar microbleeds: three-year longitudinal study in patients with subcortical vascular mild cognitive impairment

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Background & Objectives: Cerebral microbleeds have generated intense interest, since they may result from two key age-related small vessel pathologies: amyloid angiopathy and subcortical cerebral small vessel disease (cCSVD). It is hypothesized that amyloid burden and cCSVD changes synergistically affect the progression of lobar microbleeds. We tested this hypothesis longitudinally

over three years using structural MRI markers of sCSVD and [11C] Pittsburgh compound-B (PiB) PET imaging in patients with subcortical vascular mild cognitive impairment (svMCI). **Method:** Among 72 patients with svMCI who underwent amyloid imaging with PiB-PET and MRI to detect baseline microbleeds and sCSVD markers, 52 (72.2%) completed the third year follow-up. These patients were evaluated by annual brain MRI and a follow-up PiB PET. **Results:** Both baseline and longitudinal changes in lacune numbers were associated with the increased numbers of lobar and deep microbleeds, while baseline and longitudinal changes in PiB retention were associated only with the progression of lobar microbleeds, especially in the temporal, parietal, and occipital areas. There were interactive effects between the baseline and longitudinal lacune and PiB retention changes on lobar microbleed progression. **Conclusion:** Our findings suggest that amyloid-related pathology and sCSVD have synergistic effects on the progression of lobar microbleeds, providing new clinical insight into the interaction between amyloid burden and sCSVD with implications for developing effective prevention strategies.

2

Regional comparison of PET imaging biomarkers in the striatum between early versus late-onset Alzheimer's disease

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Background & Objectives: Striatum appears to play a key role in complex cognitive functions via its delicate and compact connections with cerebral cortices. Neuropathologic studies suggest that the striatum is also a vulnerable region to tangle and plaque pathology in Alzheimer's disease (AD). Sufficient evidences have demonstrated that the striatum is affected even in the pre-symptomatic stage of autosomal dominant AD by amyloid imaging. However, the pathophysiology underlying early accumulation of AD pathologies in the striatum is not fully understood, yet. Up to present, several studies reported striatal morphology, volumetric change and amyloid deposition pattern using structural MRI or 11C-Pittsburgh compound B (PiB)-PET in early-onset (EOAD) compared to late-onset AD (LOAD). In this study, we compared regional differences of β -amyloid accumulation as well as glucose metabolism in the striatum between EOAD and LOAD and investigated whether the changes in glucose metabolism is associated with amyloid pathology in the striatum.

Method: We analyzed clinical, neuropsychological profiles and neuroimaging biomarkers of 77 probable AD patients from the Alzheimer's Disease Neuroimaging Initiative (ADNI)-2 dataset who completed 3-T MRI, 18F-FDG-PET and 18F-AV-45 (Florbetapir) amyloid PET. Patients were dichotomized into two groups based on onset age of clinical symptoms: EOAD < 65yrs vs. LOAD \geq 65yrs after carefully reviewing clinical dataset. A standardized uptake value ratio (SUVR) of the striatum were obtained and subsequently normalized to the cerebellar cortex in both amyloid and FDG-PET scans. **Results:** A total of 18 EOAD patients and 59 LOAD patients were compared. There were no statistically significant differences in demographic factors including gender, disease duration, education, handedness, vascular risk factors, APOE genotyping and family history of dementia between the two groups. Clinical assessment using K-MMSE (Korean mini-mental state examination), FAQ (Functional activities questionnaire), NPI (Neuropsychiatric inventory), CDR (Clinical dementia rating), CDR-SOB (sum of boxes) and GDS (Geriatric depression scale) were also similar between groups. Detailed neuropsychological test results did not reveal statistically significant difference between groups. Bilateral caudate nucleus and bilateral putamen showed prominent glucose hypermetabolism in EOAD (SUVR: Lt.caudate; 0.96 ± 0.09 , Rt.caudate; 0.94 ± 0.09 , Lt.putamen; 1.16 ± 0.11 , Rt.putamen; 1.18 ± 0.11) compared to LOAD (SUVR: Lt.caudate; 0.88 ± 0.12 , Rt.caudate; 0.86 ± 0.12 , Lt.putamen; 1.08 ± 0.10 , Rt.putamen; 1.10 ± 0.10). 18F-AV-45 amyloid PET showed somewhat inconsistent

results in each hemisphere. Marked β -amyloid accumulation was observed in the right caudate nucleus and left pallidum in EOAD group, whereas left caudate nucleus showed more prominent β -amyloid accumulation in LOAD group. **Conclusion:** There are increasing interests about the role of striatum in AD. Emerging evidences suggest there exist differences of pathophysiology in the striatum compared to other cortices. Given the fact that glucose hypermetabolic phases were noted before final hypometabolic phases in cortical regions of autosomal dominant AD, findings of this study suggest that hypermetabolism in caudate nucleus and putamen in EOAD is potentially indicative of increased production or compensatory mechanisms in early phases of the disease. Patterns and changes of metabolism in the striatum will potentially be a noteworthy marker of age of onset in AD.

3

Elevation of the plasma A β 40/A β 42 ratio as a diagnostic marker of sporadic early-onset Alzheimer's disease

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Background & Objectives: Background: Although plasma A β levels have been evaluated as a possible diagnostic marker of Alzheimer's disease (AD), the findings are inconsistent. Objective: The present study aimed to validate plasma levels of A β 40, A β 42 and the A β 40/A β 42 ratio as biomarkers of AD in subjects with early-onset AD (EOAD) without familial AD genetic mutations. **Method:** Methods: Patients with sporadic EOAD (sEOAD) were prospectively recruited by nine neurology clinics. Plasma levels of A β 40 and A β 42 were measured using a sandwich enzyme-linked immunosorbent assay (ELISA) in 100 sEOAD (50-69 year-old) and 46 age-matched normal control subjects (50-72 year-old). Cerebrospinal fluid (CSF) was obtained from 32 sEOAD subjects and 25 controls. The integrity of the blood-brain barrier was assessed using the CSF/plasma albumin ratio. **Results:** Results: The plasma levels of A β 42 were significantly lower, while the A β 40/A β 42 ratio was significantly higher in sEOAD patients than in controls. The levels of A β 40, A β 42 and the A β 40/A β 42 ratio did not differ in relation to the APOE ϵ 4 allele. The CSF/plasma albumin ratio was comparable between the two groups, and the plasma parameters of A β proteins were not significantly associated. A multivariate analysis revealed that an increased A β 40/A β 42 ratio is valuable for the discrimination of sEOAD from controls ($\beta = 0.344$, $p = 0.000$). The area under the ROC curve for the A β 40/A β 42 ratio was 0.76, and a cut-off ratio of 5.87 was suggested to have 70% sensitivity and 68% specificity. **Conclusion:** Conclusion: The plasma A β 40/A β 42 ratio had moderate validity for the discrimination of sEOAD patients from age-matched controls.

4

Analysis of brain imaging and fluid biomarkers for Alzheimer's disease in Korean population

Min Jeong WANG¹, So Young PARK², In Kook CHUN³, Young Jun LEE⁴,

Young Ho PARK¹, SeongSoo AN⁵, Young Chul YOUN⁶, Sang Eun KIM⁷, SangYun KIM¹

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Background & Objectives: The recent diagnostic criteria of AD provided the probabilities of AD pathology according to the biomarker state. The verification of the current biomarker state is beneficial to detect the patient with prodromal AD. However, there is a considerable variability in measured biomarker values between laboratories by process protocols or analytical methodology. To overcome this limitation, the Korea consensus protocol of CSF AD biomarkers was suggested on 2015 to achieve the qualified and valid AD biomarker in CSF. In this study, we report the comprehensive analysis of well-known biomarkers of AD in Korean population and suggest the cut-off value to discriminate between AD patients and cognitively normal. **Method:** We designed the "Alzheimer's Disease All Markers (ADAM)" study to identify biomarkers in patients with AD. Twenty seven AD patients(AD) and 30 cognitively normal controls(CN) were enrolled. The CSF biomarkers are acquired following the Korea consensus regarding the preanalytical processing of CSF AD biomarkers. We measured cerebrospinal fluid amyloid beta protein 1-42(A β 42), total tau(t-Tau), and phosphorylated tau(p-Tau) protein as fluid biomarkers using multiplex immunoassay. Pittsburgh compound B(PIB) and 18fluorodeoxyglucose positron emission tomography(PET) scans were performed as imaging biomarker. A global cortical PIB PET retention ratio was calculated using mean standardized uptake value (SUV) of region of interest. To identifying the cutoff values of fluid and imaging biomarkers, we used receiver operating characteristics(ROC) analysis. **Results:** For fluid biomarkers, we confirmed trends reported in previous studies, which were decreases amyloid beta and increases t-Tau, p-Tau, t-Tau/A β 42, and p-Tau/A β 42 ratio in AD patients when comparing normal controls. the greatest ROC area under curve(AUC) value was obtained for CSF t-Tau/A β 42 ratio (0.9542), the sensitivity and specificity values were 100% and 86.21%. For the CSF p-Tau/ A β 42 ratio, the AUC value is 0.9478, comparable to CSF t-Tau/A β 42 ratio, with sensitivity of 88.46% and specificity of 92.86%. The cutoff values of CSF t-Tau/A β 42 and p-Tau/A β 42 ratio were 0.210 and 0.1350. The cutoff values of CSF A β 42, t-Tau, and p-Tau were 357.1 pg/ml, 83.35 pg/ml, and 38.00 pg/ml. For the differentiation AD from normal controls, CSF t-Tau/A β 42 or p-Tau/A β 42 ratios were superior to p-Tau, t-Tau or A β 42 alone. For imaging biomarker, the cutoff value of PIB PET was mean cortical SUV ratio 1.275. The ROC AUC value is 0.8959 with sensitivity of 84.62% and specificity of 89.66%. **Conclusion:** This is the first comprehensive data about the biomarkers of AD in Korean population. The cutoff values of fluid biomarkers in this study have been obtained abiding by this Korea consensus protocol of CSF AD biomarkers. Our data can be applied to clinical practice for diagnosis of the prodromal AD patients.

5

Relation between postural instability and subcortical volume loss in Alzheimer's disease

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Background & Objectives: To assess whether there are differences of the ability of balance control between Alzheimer's disease (AD) and controls, and to investigate the association between subcortical gray matter volumes and postural

instability in AD. **Method:** During the study period, 107 consecutive patients with AD and 37 controls were enrolled. All of the participants underwent detailed neuropsychological evaluations, 3D T1-weighted MRI at 3 T and tests for balance control using computerized dynamic posturography (CDP). We segmented the volumes of six subcortical structures of the amygdala, thalamus, caudate nucleus, putamen, globus pallidus and nucleus accumbens, and of hippocampus, using FMRIBs Integrated registration and segmentation tool. **Results:** Analyses of variances, adjusted for age and gender, showed that all structures, except for the globus pallidus, were smaller in AD than in controls. Falling frequency with eyes-open in the unilateral stance test (UST), composite score in sensory organization test (SOT) were worse in AD than in controls. The motor control test did not show any differences between groups. Falling frequency and composite score were correlated with cognitive function ($p < 0.05$). On subgroup analyses in AD, the groups with poor performance in UST or SOT showed significantly smaller volumes of the nucleus accumbens and putamen for UST, and nucleus accumbens for SOT. The smaller volume of the nucleus accumbens was associated with the postural instability in AD (OR (95% CI) 17.847 (2.594-122.804) for UST, 42.827(6.064- 302.470) for SOT, $p < 0.05$). **Conclusion:** AD patients had worse ability to control balance than controls and this postural instability was associated with the nucleus accumbens volume loss. Furthermore, postural instability was correlated with severity of cognitive impairment.

Scientific Session 13

General Neurology or other issue I

• 시간: 09:40~10:50 • 장소: Emerald Hall A (3F)

1

Paraneoplastic neurologic syndrome: associated antibodies and underlying neoplasm

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Background & Objectives: Paraneoplastic neurologic syndrome (PNS) occur by aberrant immune responses on the central or peripheral nervous systems associated with tumor. Several onconeural antibodies have been identified and have been proposed to associate with the syndrome, such as Hu, Ma2/Ta, Amphiphysin, Ri, CV2/CRMP5. Individual antibodies are often associated with specific neurological syndrome or tumors. Clinical and immunological characteristics of patients with PNS have been reported previously by few study groups, usually in western populations. However, the literature concerning PNS in Asian populations still remains scarce. We aimed to evaluate the frequency of onconeural antibodies and underlying malignancies, along with their epidemiological and clinical characteristics. **Method:** From July 2012, consecutive patients with possible PNS were screened for onconeural antibodies. Onconeural antibodies (anti-Hu, -Yo, -Ri, -Ma2, Amphiphysin, -CV2/CRMP5, SOX1, Recoverin, and Titin antibody) were tested by indirect immunofluorescence test on serum or CSF immunoblotting method. PNS was diagnosed according to the criteria described by Graus. For each patient, following information was reviewed: autoantibody type, clinical syndromes and symptoms, result of brain MRI, CSF study, and type of underlying neoplasm. **Results:** Between Jul 2012, and Jun 2015, 2159 consecutive patients with possible PNS was screened for onconeural antibodies, and the antibody was de-

ected in 123 (5.7%). Median age of the patients was 59 and 49.2% were male. Nine patients tested positive for two onconeural antibodies. Most commonly detected onconeural antibody was anti-Ma2/Ta (n=38, 30.9%), Amphiphysin (n=29, 23.6%), -Yo (n=27, 21.9%), and -Hu (n=13, 10.6%). Frequent presenting syndromes were dysautonomia (n=46, 34.8%), subacute cerebellar degeneration (n=26, 19.7%), limbic encephalitis (n=19, 14.4%), encephalomyelitis (n=18, 13.6%), and peripheral neuropathy (n=17, 12.9%). Underlying malignancy was screened in 110 patients and was detected in 44 (40.0%). Most frequent type of tumor was small cell lung cancer (n=11), followed by gastric cancer (n=7), cervical, ovary and breast (n=2). **Conclusion:** Seroprevalence of onconeural antibody was low among patients with possible PNS. However, detection of the antibody is helpful in defining PNS and searching for underlying malignancy.

2

Oral intake of anti-hangover substance increases aldehyde dehydrogenase activity: new preventive and therapeutic potentials for oxidative neuronal injury?

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Background & Objectives: Acetaldehyde is a key cause of hangover headache or associated CNS symptoms, which is oxidatively metabolized by mitochondrial aldehyde dehydrogenase (ALDH2), a well-known enzyme of ethanol metabolism. This enzyme also detoxifies endogenous toxic aldehydes, such as 4-hydroxy-2-nonenal, a major compound derived from reactive oxygen species-dependent peroxidation of cellular membrane lipid, which appears to be critically implicated in ischemic stroke, neuropathic pain, and neurodegenerative disease. Recently, a variety of anti-hangover products are commercially available in the market, however, almost none of them has been proven to show enhanced activity of ALDH2 in a live subject. Therefore, we aimed to investigate a specific product of interest. **Method:** The enzyme activities of the anti-hangover substance were examined by in vitro & in vivo experiments to measure the amount of NADH formation which is generated through catalytic conversion of alcohol and acetaldehyde, by using a spectrophotometer at 340 nm. Powder sample of a commercial anti-hangover product (KISLip®, Pico Entech, South Korea) was used as the experimental substance. In vivo examination tested the ethanol and acetaldehyde concentration in blood of rats with oral infusion of experimental substance before or after ethanol intake. In first test, twenty four SD male rats were randomly assigned into one of four groups: group1 received only saline, group2 was subjected to ethanol only, group 3 received ethanol with substance (73 mg/kg), and group 4 ethanol with substance (220mg/kg). Oral dosing of 50% ethanol (3g/kg body weight) was given 30 minutes after substance gavages, followed by time-dependent collection of rat's blood when zero, 1, 3, 5, and 8 hours after dosing of ethanol. In second test, similar examination was repeated with two groups including ethanol only (n=6) and ethanol with substance (220mg/kg) (n=6). The differentiator of second in-vivo test was that experimental substance be given 1hr after ethanol gavage, approximately near maximum level of blood acetaldehyde. **Results:** In vitro measurements of the activities of alcohol dehydrogenase & aldehyde dehydrogenase within the anti-hangover substance were 1.84 unit/g and 0.28 unit/g, respectively. The enzyme activities in rats' blood under the substance that was given 30 minutes before ethanol intake are as follows: after 1 hour of ingestion of ethanol, the concentration of ethanol in blood showed maximum values for all testing group, but decreases by 15.5% (p<0.246) and 28.3% (p<0.011) were observed for testing groups of dosing substance 73 and 220 mg/kg, respectively. In the case of a group with dosing 220 mg/kg, meaningful decrease in the concentration of ethanol through all measurement times

was observed compared to a group of ingestion of ethanol only. With acetaldehyde level in blood, the maximum values for all testing groups were measured 1hr after ethanol ingestion, demonstrating no significant differences among testing groups. However, the concentration of acetaldehyde in blood for ethanol only group started to decrease after 3 hours, in contrast, those of groups with anti-hangover substance have shown concentration-dependent reduction after one hour. As for a group of dosing substance 220 mg/kg, meaningful level of decreases were observed after 3 hours (p<0.01) and 5 hours (p<0.05). Finally, the cases with oral intake of substance 220 mg/kg after 1hr of ethanol intake have shown more significant and obvious decreases in blood acetaldehyde concentration through the period of all measurement times. **Conclusion:** Oral intake of anti-hangover substance (KISLip®) has significantly enhanced enzyme activities of alcohol metabolism in rat model, particularly mitochondrial aldehyde dehydrogenase activity in blood. Using this substance, further research on animal model of disease and detoxification of other toxic aldehydes is recommended to conduct.

3

Differences in intraoperative neurophysiological monitoring between spinal intramedullary ependymoma and hemangioblastoma

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Background & Objectives: Intraoperative neurophysiological monitoring (INM) using transcranial muscle motor evoked potentials (TC-mMEPs) and somatosensory evoked potentials (SSEP) is an established method for detecting perioperative neural damage in intramedullary spinal cord tumor (IMSCCT) surgery. Ependymomas and hemangioblastomas arise in different anatomical locations, and require different surgical techniques. However, previous studies on INM during IMSCCT surgery have not taken into account tumor pathology. Therefore, the aim of our study was to assess differences in INM findings between ependymoma and hemangioblastoma. **Method:** We selected for our study pathologically confirmed cases of intramedullary ependymoma and hemangioblastoma from patients who underwent surgery with INM between August 2009 and July 2014. Final samples selected for analysis included 56 limbs from 16 patients diagnosed with ependymoma, and 18 limbs from six hemangioblastoma patients. The alarm criterion for motor evoked potentials (MEP) was a 50% decrease in amplitude, while for SSEP it was a 50% decrease in amplitude, and/or a 10% delay in latency. We compared changes in MEP/SSEP and INM data between ependymoma and hemangioblastoma. **Results:** We found that 14 out of the 56 ependymoma limbs (25.9%), and 8 out of the 18 hemangioblastoma limbs (44.4%) included in the study met the alarm criteria for MEP during surgery. Eight limbs of ependymoma patients (57.1%), and one limb of a hemangioblastoma patient (12.5%) did not recover MEP at the end of surgery. Among those that recovered MEP, six ependymoma (10.7%), and six hemangioblastoma (33.3%) limbs did not show postoperative motor deficits (p = 0.04). In addition, 18 out of 55 ependymoma (32.7%), and eight out of 15 hemangioblastoma (53.3%) limbs met the alarm criteria for SSEP during surgery (p = 0.12). After surgery, 14 limbs of ependymoma patients (77.8%), and five limbs of patients with hemangioblastoma (62.5%) did not recover SSEP (p = 0.36). Finally, 11 limbs of ependymoma patients, and one limb of a hemangioblastoma patient showed postoperative weakness. In these limbs, neurological deficits were potentially attributed to white matter long tract injury in nine limbs of ependymoma; while in other two limbs of ependymoma, and one of hemangioblastoma, the neurological deficits were potentially attributed to focal injury in the grey matter. **Conclusion:** In our study, the incidence of transient changes in MEP

was higher in hemangioblastoma than in ependymoma. This difference may be related to specific features and location of these tumors, as well as to the use of different surgical techniques. On the other hand, there were no differences in SSEP between hemangioblastoma and ependymoma. Our data suggest that it may be necessary to consider tumor features and the type of surgical technique used, particularly when interpreting INM profiles of IMSCT such as ependymoma and hemangioblastoma.

4

Pattern of inpatient neurology consultation in Korean Tertiary Care Hospital

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Background & Objectives: Neurologic disorders are not uncommon at inpatient departments of different hospitals. We have conducted the study to see the pattern and burden of neurologic disorders at different inpatient departments of a tertiary care center. **Method:** This prospective observational study was carried out from the evaluation by the neurology specialist of neurology department of Gachon University Gil Medical Center from April 2015 to July 2015. The consultations were recorded, categorised and analysed. Consultation by the neurological service led to a significant contribution in the management of clinical cases in one of three ways: establishing a de novo diagnosis in patients admitted with active neurological symptoms where no working diagnosis exists, significant alteration in diagnosis where the referring service have already established a specific working diagnosis, or offering advice in the ongoing management of active neurological symptoms when the diagnosis is historically established and secure. **Results:** A total 440 patients were evaluated by consultant neurologist during this period. 209 of the 440 patients were female (47.5%), with a mean age of 64.9 ± 15.6 years. Department of medicine was the largest consultation seeker (280, 63.6%). And more consultations were referred from general ward (389, 88.4%) than intensive care unit (ICU). The diagnostic categories and relative numbers are detailed. Cardiovascular disorder was the most common condition (119, 30.5%), followed by toxic/metabolic encephalopathy (64, 14.5%), epilepsy (49, 11.1%), and peripheral neuropathy (41, 9.3%). The reason for referral categories and relative numbers of patents are also detailed. In order of frequency the most common reason for referral was operability (79, 18.0%), mental change (71, 16.1%), minor problem (59, 13.4%), motor weakness (46, 10.5%), seizure (40, 9.1%), medication adjustment (36, 8.2%). These patterns are slightly different in accordance with the request department. Mental change (13, 25.5%), seizure (12, 23.5%) were common reasons for referral from ICU different from general ward. **Conclusion:** We first report the pattern of inpatient neurology consultation in Korea. Neurological ward consultations are therefore clearly beneficial in a multidisciplinary tertiary referral setting, but the cost of the activity is unclear. The consultation process was time consuming, however, both in respect of the initial review, but also with follow-up visits. It is necessary to make the structured consultation guidelines to be resourced appropriately.

5

Usefulness of intraoperative transcranial motor evoked potential for predicting acute postoperative neurological complication in patients with Aneurysmal subarachnoid hemorrhage

Byung-Euk JOO, Dong-Jun KIM, Sang-Ku PARK, Dae-Won SEO

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Background & Objectives: The occurrence of hydrocephalus and spasm of cerebral vessels producing cerebral infarction during the acute phase after surgical

clipping could lead to poor outcome in patients with aneurysmal subarachnoid hemorrhage(A-SAH). Recently, Intraoperative transcranial motor evoked potential have been used to prevent the injury of corticospinal tract during surgery in patients with A-SAH. The changes on intraoperative TcMEPs during surgery is suggestive of the injury of motor pathway in brain, which may result in generation of hydrocephalus and spasm of cerebral vessels producing cerebral infarction after surgery. There was few reports about usefulness of intraoperative TcMEPs for predicting acute postoperative complication in patients with A-SAH. To define usefulness of intraoperative TcMEPs for predicting acute postoperative complication in patients with A-SAH. **Method:** From January 2010 to June 2014, 74 patients with A-SAH that underwent emergency surgery were enrolled. The anesthesia was induced with total intravenous anesthesia. The recording electrode were placed on the abductor pollicis brevis and the abductor digiti minimi muscle of upper extremity function and on the tibialis anterior and the abductor hallucis brevis of lower extremity. When the amplitude of MEP on unilateral extremity was smaller by more than 50% when compared with the opposite side, it was defined that there was a significant difference, and loss of MEP on unilateral extremity was also defined. The severity of SAH was also evaluated by the using the Fisher grade, Hunt-Hess grade and World Federation of Neurosurgical Societies (WFNS) on initial brain CT findings. To define the usefulness those findings for predicting the occurrence of acute postoperative complication, Spearman correlation was used. **Results:** On Fisher grade, 2 patients were in grade I, and grade II was 7, and respectively 36 and 13 patients were in grade III and IV. 17(22.97%) patients showed a significant change on the baseline intraoperative TcMEP. 10 of 17 patients presented a significant decrease, and other 7 patients showed wave loss on the baseline intraoperative TcMEP. 10 patients had reoperation due to hydrocephalus after surgical clipping. Also cerebral infarction due to spasm of cerebral vessels was occurred in 9 patients. When analyzing the association between the change of intraoperative TcMEPs and the occurrence of acute postoperative complication, significantly high correlation was observed. ($\rho=0.536, p<0.001$) The correlation of Fisher grade for acute postoperative complication was observed relatively low($\rho=0.317, p=0.006$). **Conclusion:** The changes of intraoperative TcMEPs was useful for predicting the occurrence acute postoperative complication in patients with A-SAH. We should pay more attention in caring those patients showing significant change on intraoperative TcMEPs after surgery.

Scientific Session 14

Stroke III

• 시간: 09:40~10:50

• 장소: Emerald Hall B (3F)

1

Association between grades of right-to-left shunt and TOAST subtypes of the ischemic stroke

Chan-Hyuk LEE, Man-Wook SEO, Byoung-Soo SHIN, Sun-Young OH, Tae-Ho YANG, Han Uk RYU, Seul-Ki JEONG

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Background & Objectives: Patent foramen ovale (PFO) is a remnant of incomplete septum primum and secundum closure after birth. PFO is considered one of the cause for ischemic stroke. However, the hole's position as a cause of cerebral infarction is quite unstable. If another conclusive reason is detected, PFO can't be contributable to TOAST classification. The objective of the study is to reveal weather right-to-left shunt, such as PFO is related to is-

chemic stroke or not, in the aspects of right-to-left shunt with transcranial doppler (RLS with TCD). **Method:** This study population was composed of 816 subjects (ischemic stroke patients: 520, 63.7%) who examined the RLS with TCD, which was conducted in Chonbuk National University Hospital between November 2013 and February 2015. Grade of RLS with TCD is divided into 4 types, as recommended. Ischemic stroke was categorized according to the criteria of Trial of Org 10172 in Acute Stroke Treatment (TOAST). TOAST classification consists of five subtypes depending on causes leading to a ischemic stroke, which are large artery atherosclerosis, small vessel occlusion, cardioembolism, stroke of undetermined etiology, and stroke of other etiology. The relationship between various grades of RLS and TOAST subtypes is examined by chi-square test and multivariate association was performed with logistic regression analysis. **Results:** The number of patients who possessed PFO confirmed by echocardiography among target patients was higher in the ischemic stroke group than the non-stroke group. Additionally, when moderate-to-severe or only-severe cases were placed in more risky group, the odds ratios (ORs) were higher in the SUE subtype exclusively. The outcome indicated that 'stroke of undetermined etiology' (SUE) were associated with higher grade (Grade 3 and 4) of RLS with TCD (OR, 2.151; 95% confidence interval [CI], 1.079 to 4.289 for Grade 3, 4 (moderate to severe), Table 4). On the other hand, the odds ratios (ORs) indicated that 'small vessel occlusion' (SVO) had negative correlation with higher grade (Grade 3 and 4) of RLS with TCD (OR, 0.364; 95% confidence interval [CI], 0.189 to 0.699 for Grade 3, 4 (moderate to severe), Table 4). **Conclusion:** SUE subtype of TOAST classification would be associated with the degree of right-to-left shunt through specific routes, such as PFO. In other words, it could be another evidence that PFO is contributable factor for ischemic stroke. On top of that, the fact that the number of patients who possessed PFO confirmed by echocardiography were higher in the ischemic group could be another supporting reason. Subsequent researches are needed to unveil the role of PFO on ischemic stroke.

2

Trends in risk factor prevalences among young adults with ischemic stroke

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Background & Objectives: Ischemic stroke in young adults is rare but can be devastating for the affected individuals and their families. Recently, the increasing ischemic stroke rates in young adults aroused many researchers to study on this topic. However, the risk factors of ischemic stroke in the young adults are not thoroughly identified. In this study, we aimed to investigate temporal trends of vascular risk factors in young adults with ischemic stroke. **Method:** This study is based on the prospectively collected stroke registry of CNUH between October 2008 and December 2014. The diagnosis of ischemic stroke was based on focal neurological deficit with corresponding ischemic lesions on brain CT/MRI and MR angiography. Risk factors were based on their history and laboratory findings for admission. The data were compared with that of other studies to analyze the temporal trends of vascular risk factors. **Results:** This study included 528 young adults from among 6396 patients with ischemic stroke in CNUH between October 2008 and December 2014. Thus the study population consisted of 528 patients (355 men, 173 women) aged below 50 years (mean 42.8). Cigarette smoking was the most prevalent factor in young adults with ischemic stroke, was accounted for 49% (260 of 528) of risk factors. Dyslipidemia as the second most prevalent factor was accounted for 45.5%. Hypertension was accounted for 33% (178 of 529). **Conclusion:** This trend study of risk factors for young adults with ischemic stroke indicate that

smoking is still one of the most prevalent risk factors. Further it is noteworthy that Dyslipidemia has emerged as a new prevalent risk factor. It may possibly be attributed to the westernized eating habits.

3

The effect of high-intensity statin use in the acute phase after thrombolysis

Tae Kyoung KIM, Eui Sung JUNG, Kyusik KANG, Woung-Woo LEE, Jungju LEE, Ohyun KWON, Byung-Kun KIM, Jong-Moo PARK

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Background & Objectives: The impact on stroke outcome and safety of high-intensity statin use in the acute phase after thrombolysis is uncertain. This study investigate the safety and efficacy of high-intensity statin initiated in the early time window for the acute ischemic stroke patients after IV or IA thrombolysis. **Method:** Acute stroke patients who received IV or IA thrombolysis within 8 hours after index stroke were included. Patients with pre-morbid mRS over 3 were excluded. High-intensity statin use in early window was defined as the administration of atorvastatin 40-80mg or rosuvastatin 20-40mg within 24 hours after thrombolysis. Primary efficacy outcome is neurologic improvement (NIHSS \leq 4 points from baseline or NIHSS=0), major neurologic improvement (NIHSS \leq 8 points from baseline or NIHSS=0) at 7 days and favorable functional outcome at 3 months (mRS \leq 2), while primary safety outcome is symptomatic hemorrhagic transformation (PH type 2) with NIHSS \geq 4 points from baseline or death within 36 hours and any intracranial hemorrhage during admission and neurologic deterioration (NIHSS \leq 4 points from baseline or death) at 7 days. **Results:** Between April 2007 and March 2015, 323 patients received IV or IA thrombolysis in our hospital. Of the 323 patients entered into the study, 229 (70.8%) were treated with statins. The statin intensity were high in 97 (42.4%), moderate in 127 (55.5%), low in 3 patients. Statin administration was started within 24 hours after thrombolysis in 58 (25.3%), between 24 and 48 hours in 112 (48.9%), after 48 hours in 59 (25.8%). Among all patients who received thrombolysis, neurologic improvement was noted in 102 (31.6%), major neurologic improvement in 71 (22.0%), and neurologic deterioration in 48 (14.9%), while sICH were observed in 36 (11.1%) and any hemorrhage during admission in 52 (16.1%) patients. On multivariable analysis with adjustment, statin use was associated with more neurologic improvement (OR 2.158, 95% CI 1.148-4.075, p=0.017), smaller risk of neurologic deterioration (OR 0.105, 95% CI 0.043-0.260, p=0.000), and favorable functional outcome at 3 months (OR 8.564, 95% CI 1.077-68.109, p=0.042). Statin use was associated with a reduced risk of sICH occurrence (OR 0.097, 95% CI 0.035-0.273, p<0.001). High-intensity statin use in early window after thrombolysis was noted in 27 (17.8%) patients of all statin group. Risk of sICH was similar between high-intensity statin use in early window and others. **Conclusion:** Our study shows that, in patients treated with thrombolysis, statin use was associated with good outcome of safety and efficacy, while the risk of any hemorrhage during admission time tended to be increased but not statistically significant means. The patient administered high intensity statin within 24hr did not increased the risk of symptomatic hemorrhage, any hemorrhage compared to other statin regimen groups.

4

Preliminary evaluation of the brain saver: a novel pre-hospital stroke notification system using mobile application platform

Mi Sun OH, Kyung-Ho YU, Byung-Chul LEE

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Background & Objectives: Prehospital notification of patients with suspected acute stroke by emergency medical services (EMS) personnel is recommended to reduce delays in time-dependent management such as thrombolytic therapy. We developed “the Brain Saver”, which is a smartphone-assisted prenotification system using mobile application platform, and evaluated preliminarily the effects of this system on improving quality indicators for thrombolysis therapy in acute ischemic stroke. **Method:** Through the Brain Saver, the EMS personnel sent the clinical information, such as age, sex, time of symptom onset, expected arrival time, neurological symptom of the patient with suspected acute stroke in the field, so that the stroke team prepared to prioritize the hospital resources such as computerized tomography (CT) and various laboratory test for suspected stroke patients. On arrival at emergency room, patients were transferred directly from triage onto the CT room on the ambulance stretcher, and the stroke team met patient. Between September 2014 and July 2015, we compared quality indicators including door-to-needle time (DNT), door-to-imaging time (DIT) of patients with intravenous tissue plasminogen activator (tPA) with prenotification to those without prenotification. **Results:** During study period, time of onset to arrival in patients with prenotification using the Brain Saver was similar in those without. However, compared with tPA patients who arrived without prenotification, the median (interquartile range) DNT decreased from 55 (42-92) to 36 (32-42) minutes ($p=0.003$) and DIT was reduced from 11 (7-15) to 3 (2-6) minutes ($p<0.0001$). **Conclusion:** The Brain Saver was the efficient tool to communicate between EMS personnel and the stroke team for the prenotification of patients with acute stroke. The prenotification improved quality indicators of in-hospital stroke management by reducing time delay in thrombolytic therapy.

5

Longitudinal study on the associations between cognitive status and transcranial doppler parameters in patients with patients with mild to moderate dementia

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Background & Objectives: Transcranial doppler (TCD) parameters were associated with cognitive functions in the previous cross-sectional studies. However, these associations were seldom evaluated through the longitudinal studies. We aimed to investigate the associations between longitudinal changes of cognitive status and transcranial doppler parameters. **Method:** Patients with mild to moderate dementia (Alzheimer’s disease and vascular dementia) who aged 60 to 79 years old were enrolled. Those who had poor temporal windows or significant stenosis of intracranial arteries ($\geq 50\%$) were excluded. Mean flow velocity (MFV), pulsatility index (PI), vasomotor reactivity (VMR) was evaluated through TCD. Baseline cognitive functions were assessed using Alzheimer’s Disease Assessment Scale (ADAS-cog), Frontal assessment battery (FAB), MMSE, Clinical Dementia Rating (CDR), CDR sum of boxes (SOB), and geriatric depression scale (GDepS). TCD and cognitive assessment was followed up 1 year later, and the absolute differences between the baseline and follow-up evaluations were evaluated (Δ). Demographic characteristics and vascular risk factors were also investigated. Correlation analysis was conducted between the changes of TCD parameters and cognitive assessment scores. **Results:** A total of 75 patients were enrolled, and 43 patients (54.4%) were currently completed the follow-up evaluations. For those who have completed the follow-up, mean age was 71.4 ± 5.0 years, baseline MMSE score 21.3 ± 3.6 , and GDepS 17.0 ± 7.1 . In the baseline evaluations, ADAS-cog scores was positively associated with both ACA and MCA PI values, and right PCA PI. Right PCA MFV was negatively associated with CDR SOB ($r=-0.26$, $p=0.04$). Left ACA PI was negatively associated with baseline MMSE scores

($r=-0.33$, $p=0.01$). However, MFV and VMR was not associated with any cognitive scores. As for the follow-up data, the Δ left PCA PI were positively associated with Δ FAB scores ($r=0.35$, $p=0.04$). Δ right PCA PI was positively associated with Δ GDepS, and Δ right ACA PI with Δ CDR SOB ($r=0.50$, $p=0.01$; $r=0.33$, $p=0.06$; respectively). Δ dVMR ratio was marginally associated with Δ MMSE score ($r=-0.73$, $p=0.10$). Δ MFV was not associated with any cognitive changes in the follow-up. **Conclusion:** Our results revealed that longitudinal PI changes were significantly associated with the longitudinal changes of frontal function, general cognition as well as depression scores. Further large clinical studies are needed to confirm our results with adjustments for various confounders.

Scientific Session 15

Neuro-otology

• 시간: 09:40~10:50 • 장소: Diamond Hall (3F)

1

Randomized trial on short-term efficacy of vibration and Gufoni maneuver for apogeotropic horizontal benign paroxysmal positional vertigo

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Background & Objectives: The apogeotropic type of benign paroxysmal positional vertigo involving the horizontal canal (HC-BPPV) is likely due to otolith debris in the anterior arm of the canal (canalolithiasis) or the debris attached to the cupula (cupulolithiasis). It is important to seek the method to convert treatment-resistant apogeotropic form to the more treatment-responsive geotropic form in HC-BPPV. To determine the short-term therapeutic efficacy of vibration and Gufoni maneuvers in apogeotropic type of HC-BPPV, we designed a randomized, prospective, sham-controlled study. **Method:** In 8 nationwide dizziness clinics in Korea, 209 consecutive patients (76 men, age range: 21-86 years, Mean age: 61.9 ± 12.7) with apogeotropic HC-BPPV were enrolled. Patients were randomly assigned to only one treatment by Gufoni ($n=70$), vibration ($n=67$), or sham maneuver ($n=72$). During vibration maneuver, patients were taking mastoid oscillation with heads turned to the lesion side 135 degree and healthy side 90 degree. Immediate and short-term responses were determined within 1 hour after one trial of each maneuver and in the following day, respectively. Successful treatment was defined as resolution of positional nystagmus, or as transition into geotropic horizontal nystagmus. **Results:** Immediate response of 30 minutes after one maneuver, Gufoni (33/70, 47.1%) and vibration (32/67, 47.8%) maneuvers showed better responses than the sham maneuver (14/72, 19.4%) ($p=0.00$). The second day results did not differ between three groups ($p=0.53$). The short-term outcome determined on the next day were also better with Gufoni (52/68, 76.5%) and vibration (47/65, 72.3%) maneuvers compared with the sham maneuver (38/71, 53.5%) ($p=0.00$). However, therapeutic efficacies did not differ between the Gufoni and vibration groups in terms of both immediate and short-term outcomes ($p=0.69$). **Conclusion:** Both the Gufoni and vibration maneuver are valid methods for treating apogeotropic horizon-

tal canal BPPV with a success rate of approximately 70% at one maneuver during short-term follow up period.

2

The dizziness practice in South Korea: a National Survey

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Background & Objectives: Since the dizziness and vertigo could be a presenting symptom of various medical conditions, both patients and doctors have experienced confusion in management of dizziness. The aim of the present study is to assess the change of dizziness practice in South Korea for recent 5 years. **Method:** We analyzed the data on Health Insurance Review & Assessment Service for 2010 to 2014. The Korean standard classification of disease (KCD) was used to select diagnosis related to dizziness. **Results:** The diagnoses of dizziness and vestibular dysfunction (KCD R42 and H81) have been ranked as those of top 100 common diseases for 2010 to 2014. The incidence of dizziness and medical expenses are increasing for recent 5 years. The most common department for dizziness (KCD R42) is internal medicine (31.2%). And vestibular dysfunction (KCD H81) has been cared most commonly in otolaryngology (40.2%), which is followed by internal medicine (23.8%), neurology (17.8%), general practitioner (13.8%) and emergency medicine (5%). Benign paroxysmal positional vertigo (KCD H811) was managed by otolaryngology (44.4%), neurology (21.6%), internal medicine (15.5%), general practitioner (8.2%), and emergency medicine (7.2%). And the 7-13% of dizzy patients was managed by more than 2 departments. **Conclusion:** Our data suggest the need to develop adequate care system for common dizziness patients.

3

Acute transient vestibular syndrome due to cerebellar ischemia

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Background & Objectives: To elucidate the frequency of posterior circulation stroke and transient ischemic attack in acute transient vestibular syndrome. **Method:** We prospectively recruited 290 patients with first onset of vertigo/dizziness at the emergency department (ED) of Pusan National University Yangsan Hospital from January to December 2014. After excluding patients with BPPV, we divided 182 patients into two groups according to symptoms duration: acute transient vestibular syndrome (duration < 24 hours, ATVS) and acute vestibular syndrome (duration > 24 hours, AVS). We assessed the etiology, clinical manifestations and brain MRI including perfusion weighted image (PWI) in patients with ATVS. **Results:** Of 182 patients, 86 (47%) and 96 (53%) presented with ATVS and AVS, respectively. Of 86 with ATVS, 63 (73%) have visited to ED with improvement of vertigo/dizziness, and 23 still had symptoms during ED visit. ATVS were classified into 15 pATVS (peripheral, 17%), 23 cATVS (central, 27%), and 48 uATVS, (unclassified, 56%). Only 3 of 23 with cATVS still had symptoms during ED visit, and showed positive HINTS for a stroke. Seven developed transient neurological symptoms with vertigo/dizziness although their symptoms and signs disappeared during ED visit. The other 13 presented with isolated vertigo/dizziness without focal neurologic symptoms. cATVS was more likely in patients with male, headache, focal neurologic signs, and VA stenosis or hypoplasia. Isolated vertigo/dizziness was more common in patients with cATVS than cAVS. More patients (10/23, 43%) with cATVS were confirmed as having cerebellar hypoperfusion by PWI only than those with cAVS (3/50, 6%). The cerebellum (83%) was the most

common responsible lesion for cATVS, whereas either cerebellum or lateral medulla was the common site for cAVS. **Conclusion:** Our study demonstrated that ATVS has a distinctive etiology from that of AVS. Isolated vertigo/dizziness was more common in patients with cATVS than cAVS. PWI can be useful to detect cerebrovascular cause in ATVS.

4

Canalith repositioning in apogeotropic horizontal canal benign paroxysmal positional vertigo: do we need faster maneuvering?

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Background & Objectives: A correct diagnosis and a proper treatment may yield a rapid and simple cure for benign paroxysmal positional vertigo (BPPV). Although the Gufoni maneuver is widely used to treat apogeotropic horizontal-canal BPPV (HC-BPPV), few studies have clarified the relation between the speed and intensity of maneuver execution and successful canalith reposition. Therefore, to evaluate the effect of accelerated execution of the Gufoni maneuver, a prospective randomized controlled study was conducted with HC-BPPV patients in a single dizziness clinic for a certain period. **Method:** The study was performed among patients diagnosed with apogeotropic HC-BPPV at the dizziness clinic of a tertiary university hospital from January, 2013 to August, 2014. We attempted to compare the resolution rate between two groups with different speed of maneuver execution, following a prospective randomized controlled design. The accelerated maneuver group was subjected to faster position changing -within 1 second-during the reposition maneuver, while the non-accelerated maneuver group underwent slower maneuvers. The therapeutic effect was determined as the relief of dizziness or the resolution of nystagmus within 1 hour after the treatment. **Results:** Fifty patients with apogeotropic HC-BPPV were enrolled and treated with the Gufoni maneuver in two groups (accelerated group, n = 25, vs. non-accelerated group, n = 25). The overall resolution rate was 48% (24 of 50) regardless of acceleration. There was no significant difference in the resolution rate of the apogeotropic HC-BPPV treated with the Gufoni maneuver (48% in each group) between both the accelerated and non-accelerated groups (p = 1.00). **Conclusion:** Our results suggest that faster and more intense execution may give little effect in treating apogeotropic HC-BPPV with Gufoni maneuver. The detachment of otolith itself from the cupula, or the gravitational force-in cases with the otolith is in the anterior arm of HC-may be more important contributing factors.

5

Chasing dizzy chimera: diagnosis of combined peripheral and central vestibulopathy

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Background & Objectives: Diagnosis of combined peripheral and central vestibulopathy remains a challenge since the findings from peripheral vestibular involvements may overshadow those from central vestibular disorders or vice versa. The aim of this study was to enhance detection of these intriguing disorders by characterizing the clinical features and underlying etiologies.

Method: We had recruited 55 patients with combined peripheral and central vestibulopathy at the Dizziness Clinic of Seoul National University Bundang Hospital from 2003 to 2013. Peripheral vestibular involvement was determined by decreased caloric responses in either ears, and central vestibulopathy was diagnosed with obvious central vestibular signs or the lesions documented on MRIs to involve the central vestibular structures. **Results:** Combined peripheral and central vestibulopathy could be classified into four types according to the patterns of vestibular presentation. Infarctions in the territory of anterior inferior cerebellar artery were the most common cause of acute unilateral cases while cerebellopontine angle tumors were mostly found in chronic unilateral ones. Wernicke encephalopathy and degenerative disorders were common in acute and chronic bilateral disorders. Isolated audio-vestibulopathy was found in 25 (45.5%) patients, but association with gaze-evoked nystagmus (GEN), impaired smooth pursuit (SP) or central types of head shaking nystagmus (HSN) indicated a central vestibular involvement in most of them (23/25, 92.0%). HINTS (negative head impulse test, direction changing nystagmus, and skew deviation) was negative in five (17.2%) of the 29 patients with acute combined vestibulopathy. The abnormalities of caloric and head impulse tests were dissociated in eight (8/55, 14.5%) patients. **Conclusion:** Given the requirements for urgent treatments and potentially grave prognosis of combined vestibulopathy, central signs should be sought even in patients with obvious clinical or laboratory features of peripheral vestibulopathy. Scrutinized bedside evaluation, however, secured the diagnosis in almost all the patients with combined vestibulopathies.

6

Simultaneous recordings of cervical and ocular vestibular-evoked myogenic potentials

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Background & Objectives: Cervical (cVEMP) and ocular (oVEMP) vestibular-evoked myogenic potentials are short latency manifestations of vestibulo-ocular and vestibulocollic reflexes that originate from the utricle and saccule. The aim of the present study was to determine whether simultaneous recordings of cVEMP and oVEMP can be used to measure reliable responses compare to separate recordings of those reflexes. **Method:** In 39 healthy subjects, both simultaneous recordings and conventional separate recordings of cVEMP and oVEMP responses to air-conducted tone bursts were obtained. p13 and n10 latencies, p13-n23 and n10-p16 amplitudes of cVEMP and oVEMP waveforms were measured, respectively. The asymmetry ratios (AR) of amplitudes were calculated. **Results:** In cVEMP testing, p13 latencies and p13-n23 amplitudes produced no significant difference between simultaneous and conventional separate recordings. In oVEMP testing, n10 latencies measured from simultaneous recordings showed a little but significant prolongation compare to the results of separate recording. And, the n10-p16 amplitudes are significantly higher during simultaneous recordings. AR of simultaneous recordings of cVEMP and oVEMP did not show difference between the two recording methods. **Conclusion:** Simultaneous recordings of cVEMP and oVEMP are reliable tests and could be a clinically useful and simplified diagnostic tool for evaluating the dizzy patients.

1

Serum and CSF cytokine profiles in autoimmune encephalitis

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Background & Objectives: Autoimmune encephalitis occurs by aberrant cellular or humoral immune responses that attack neurons in the brain. Cytokines are important in development, differentiation and regulation of immune cells. Abnormal cytokine production can play a central role in the development of autoimmune disease. However, little is known about the significance of cytokines in antibody-mediated autoimmune encephalitis. This study aimed to evaluate serum and CSF cytokines and chemokine in anti-NMDA receptor and LGI1 encephalitis patients. **Method:** We measured 14 cytokines (IL-1b, 2, 4, 5, 6, 10, 12, 13, 17A, 23, GM-CSF, IFN-gamma, TNF-alpha) and chemokine (CXCL13) in serum and CSF from 14 patients with anti-NMDA receptor encephalitis and 10 with anti-LGI1 encephalitis by multiplexed fluorescent bead-based immunoassay or ELISA. Serum and CSF from 10 patients with non-inflammatory neurological diseases was used as a control. **Results:** Compared with the controls, patients with NMDA receptor encephalitis had increased serum IL-1b and IL-2 but not in CSF samples. CSF IL-17A and CXCL13 were significantly elevated in anti-NMDAR encephalitis compared with controls, but not in serum samples. Patients with LGI1 encephalitis had higher serum GM-CSF and CSF IL-17A level compared with controls. There was no difference in cytokine levels between anti-NMDA receptor and LGI1 encephalitis, except for serum IL-1b, which was higher in anti-NMDA receptor encephalitis. **Conclusion:** Our finding suggests distinct cytokine and chemokine changes in serum and CSF among anti-NMDA receptor and LGI1 encephalitis patients. Interleukin 17A may be an important therapeutic target in treating autoimmune encephalitis.

2

Cerebral and cardiovascular autoregulation during head-up tilt test in posturally related syncope patients

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Background & Objectives: Syncope is a transient loss of consciousness due to cerebral hypoperfusion, associated with inability to maintain postural tone. However, the detailed mechanism related to the cerebral hypoperfusion, whether it contributes to syncope or reflects simply a normal cerebral autoregulatory response to systemic arterial hypotension is not clearly understood yet. This study aims to assess dynamic cerebral autoregulation associated with complex interactions between sympathetic and parasympathetic systems by measuring cerebral arterial blood flow velocity and heart rate variability by orthostatic stress during head-up tilt (HUT) test. **Method:** Total 252 patients were reviewed from the clinical database of patients who manifested as one or more episodes of syncope and visited Neurology department in Ewha Womans University Hospital from June 2005 to May 2015. Among them, 46 patients were performed HUT test with continuous recordings of cerebral blood flow velocity (CBFV), electrocardiogram (ECG), instantaneous arterial blood pressure (ABP), and electroencephalogram (EEG) at baseline, 70° head-up, and supine position. CBFV was assessed by transcranial doppler (TCD) study using pulsatile index, mean CBFV from right or left middle cerebral artery, and

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General Neurology or other issue II

• 시간: 14:20~15:20 • 장소: Convention Hall C (4F)

heart rate variability (HRV) were calculated linear spectral and nonlinear HRV indices. In addition, we analyzed autonomic function tests including HRV during valsalva maneuver and response to deep breathing, orthostatic blood pressure test, sympathetic skin reflex, and Q-sweat test. **Results:** The TCD study during HUT test showed positive responses in 16/46 (35%) patients; symptoms of presyncope in 2/46 (4%) vasovagal syncope, 3/46 (7%) orthostatic hypotension, 8/46 (17%) postural orthostatic tachycardia syndrome, and 3/46 (7%) vasovagal syncope and postural orthostatic tachycardia syndrome. In addition, significant decreases of diastolic CBFV and an increase of pulsatile index were observed in 5/46 (11%) patients, which suggests a possibility of cerebral syncope. Autonomic function test revealed positive results in 13/46 (28%) patients; sudomotor dysfunction in 11/46 (23%), orthostatic hypotension in 2/46 (4%), and HRV during valsalva maneuver and deep breathing in 1/46 (2%). Linear and nonlinear indices of HRV showed alternations in sympathetic and parasympathetic interactions, and spectral EEG indices revealed changes in cerebral function in major portion of patients. **Conclusion:** We analyzed dynamic regulations between cerebral and cardiovascular systems in posturally mediated syncope patients using cerebral blood flow velocity and arterial blood pressure, pulse rates, HRV as an index of sympathetic and parasympathetic cardiovascular regulation, and electroencephalogram as an index cerebral perfusion. Continuous TCD, ECG and EEG monitorings during HUT test can be useful to diagnose vasovagal or neutrally mediated syncope effectively and also to investigate the underlying mechanisms related to dynamic interactions of cerebral and cardiovascular regulations.

3

Autonomic parameters in postural orthostatic tachycardia syndrome with different onset time

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Background & Objectives: The postural tachycardia syndrome (POTS) is characterized by a sustained heart rate (HR) increment of more than 30 beats/minute without orthostatic hypotension within 10 minutes of standing or head-up tilt (HUT) test. Sudden HR increase within 3 minutes or slowly increased HR response after 3 minutes of tilting can be observed during HUT test in patients with POTS. Moreover, HR increase after 10 minutes of tilting is also frequently found during HUT test. It is unclear if there are different pathophysiological mechanisms in various pattern of POTS in terms of onset time. We investigated autonomic parameters in patients with POTS with different onset time. **Method:** We retrospectively reviewed autonomic data from patients presenting as orthostatic intolerance including orthostatic dizziness from February 2011 to July 2015. POTS was defined as a sustained HR increment of more than 30 beats/minute during HUT test or the standing HR was more 120 beats/minute. A standardized battery of autonomic tests, including the HUT test, Valsalva maneuver, heart rate deep breathing test, and quantitative sudomotor axon reflex test using Finometer devices to record the beat-to-beat blood pressure (BP) and heart rate (HR) response was performed. The beat-to-beat derived hemodynamic parameters, including systolic, mean and diastolic BP, HR, cardiac output, stroke volume (SV), and total peripheral resistance (TPR), were also collected during HUT test. According to onset time of POTS, we divided into three groups; 1) HR increase within 3 minutes (immediate POTS), 2) HR increase between 3 and 10 minutes (early POTS), 3) HR increase after 10 minutes (late POTS). **Results:** We identified 210 patients showed POTS during HUT test. The increase in mean HR during the tilt was 31.2 ± 7.0 bpm. Forty three percent of patients (90/210) were included in immediate POTS, 28% (59/210) were early POTS, and 29% (61/210) were late POTS. Patients with immediate POTS showed higher E:I ratio and HR response, higher baseline HR and increase in HR during tilting, prominent SV

decrease, and smaller baseline TPR and lesser TPR decrease compare than immediate POTS and early POTS groups. Late POTS group were older and decreased cardiovagal autonomic parameters, higher systolic BP and mean BP during supine and tilting, lower HR during tilting, and smaller HR increase compared than immediate POTS and early POTS group. **Conclusion:** Patients with earlier appearance of HR increment after tilting tended to have younger in age, active cardiovagal function, prominent SV decrease, and lower BP during tilting than the patients without.

4

Is the valsalva maneuver can predict the response to head-up tilt table test?

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Background & Objectives: Autonomic function tests (AFTs) have three domains which test for sudomotor function, cardiovagal function and adrenergic function. In adrenergic function tests, this domain consists with the Valsalva maneuver (VM) and head-up tilt table test (HUTT) and is a method for adrenergic function, especially baroreflex function. The patient with sympathetic lesion shows that response of phase II-late and IV are decreased or absent in the VM, and so positive response to HUTT may be predicted. Therefore, responses to both the VM and HUTT are formed by same underlying mechanism and then the response of the VM may be represented response to HUTT in the patient with sympathetic lesion, according to the degree of adrenergic failure of the VM. So we analyzed correlation of responses between the VM and HUTT in the same patients for estimating predictive value of VM to HUTT. **Method:** We included 299 patients, which have done AFTs due to experienced orthostatic dizziness or loss of consciousness from January 2013 to December 2014. We excluded 53 patients, which have not performed the VM because of various other reasons. Total 246 patients were included in this analysis. First, we divided patients by the response of HUTT into both negative (HNRG) and positive (HPRG) groups. Second, we divided patients by the response of the VM into negative (VNRG) and positive (VPRG) groups on both HUTT response groups. Third, we analyzed that an association between responses of HUTT and both responses of VM, an association between severity of VM and response of HUTT, prediction for HUTT positive by VM at score 0 and 1, and prediction for HUTT positive by VM at score from 0 to 3. We statistically analyzed data by chi-square test, binary logistic regression in statistically significant with $P < 0.05$. **Results:** The chi-square test was used to test for association between responding to the VM and response to HUTT, association between severity of VM and positive response to HUTT. Binary logistic regression was used to test for predictions for HUTT positive by the VM and predictions for HUTT positive by the VM according to severity. HNRG included 21 cases and HPRG included 225 cases. VNRG included 56 cases and VPRG included 190 cases. These were subclassified in both HPRG and HNRG. VNRG included 10 cases and VPRG included 11 cases in HNRG, VNRG included 46 cases and VPRG included 179 cases in HPRG. The association between response to the VM and response to HUTT was analyzed, HPRG in VPRG was 179 (94.21%) cases and HNRG in VNRG was 10 (17.86%) cases, so that the positive rate of HUTT by the VM was 94.21% ($P = 0.005$). For analysis of association between severity of the VM and positive response to HUTT, HPRG cases were subclassified according to severity of VM score from 0 to 3 points, each subgroup included 46 (82.14%) cases, 108 (93.1%) cases, 26 (96.3%) cases, 45 (95.74%) cases ($P = 0.036$). Therefore, the association between severity of VM and positive response to HUTT was present. In predictions for HUTT positive by the VM, odds ratio (OR) was 3.538 (95% CI=1.416-8.838, $P = 0.007$). In predictions for HUTT positive by the VM according to severity from 1 to 3, OR is each 2.935 (95% CI=1.089-7.911,

P=0.033*), 5.652 (95% CI=0.684-46.673, P=0.108), 4.891 (95% CI=1.015-23.577, P=0.048*). **Conclusion:** The association between response to the VM and response to HUTT was presented by positive rate was 94.21% (P=0.005). And the association between severity of VM and positive response to HUTT was present. OR of predictions for HUTT positive by the VM is 3.538 (95% CI=1.416-8.838, P=0.007), so we can predict HUTT positive by the VM and may be depended on VM score.

5

Quantitative analysis of human cutaneous neurovascular system

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Background & Objectives: Skin biopsy is well-established technique for quantifying epidermal sensory nerve fiber density. Several studies have established the utility of the skin biopsy in quantifying the autonomic innervation of arrector pili muscles and sweat glands, however, quantification of the cutaneous vasomotor innervation is not established yet. We conducted this study to establish a reliable method for the assessment of cutaneous vasomotor innervation. **Method:** Total 8 healthy controls (mean age, 30.4±6.1; Women/Men = 3) who didn't have any medical conditions that might cause micro-circulation abnormality or neuropathy were enrolled. Skin biopsy samples were obtained from the distal thigh and the distal leg. Immunohistochemical staining underwent with the pan-axonal marker PGP 9.5, and with the endothelial marker CD 31. Confocal Z-stack images were used to analyze the whole thickness of the tissues. Unbiased stereological method was used to quantify the superficial dermal neurovascular system. All the nerve fibers and the blood vessels within 500 µm from the epidermal surface were counted and they were normalized by the total area. Neural density, vascular density, and neurovascular density were calculated. The area of interest was divided into the superficial layer (SL) and the deep layer (DL) to find the relationship between capillaries and subepidermal neural plexuses. SL is as shallow as possible in shallower SL method and SL contains capillaries and subepidermal neural plexuses in deeper SL method. Intraclass correlation coefficient (ICC) was calculated to compare the reliability of the shallower and deeper SL methods. **Results:** Neural densities on the distal leg/distal thigh were 0.22±0.03/0.25±0.05 in shallower SL method and 0.19±0.04/0.20±0.05 in deeper SL method. Vascular densities on the distal leg/distal thigh were 0.32±0.04/0.33±0.04 in shallower SL method and 0.25±0.04/0.25±0.03 in deeper SL method. Neurovascular densities on the distal leg/distal thigh were 0.13±0.02/ 0.15±0.03 in shallower SL method and 0.10±0.02/0.10±0.02 in deeper SL method. ICC values of shallower SL method were a little bit higher than those of deeper SL method. **Conclusion:** We report the successful application of an unbiased stereological method to quantify the cutaneous neurovascular density. This method is a reliable way to investigate the dermal neurovascular system and define the relationship between the vascular and the vasomotor nervous system in health and disease will provide greater understanding of the pathogenic mechanisms of disease that affect peripheral vascular function.

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Movement Disorder II (Eng)

• 시간: 14:20~15:20 • 장소: Emerald Hall A (3F)

1

Causes of poor outcome of STN DBS in patients with Parkinson's disease

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Background & Objectives: While most patients with Parkinson's disease (PD) show remarkable improvement of off parkinsonism following deep brain stimulation in the subthalamic nucleus (STN DBS), some patients do not. We reviewed the outcome of STN DBS for PD patients and investigated the causes of poor outcome. **Method:** We studied 73 patients with PD who underwent STN DBS from March 2013 to July 2015 in Asan Medical Center by one neurosurgeon and one neurologist (J.K. Lee and C.S. Lee). Motoric, cognitive and behavioral aspects of clinical features were evaluated before surgery using the clinical test battery. The outcome of DBS was assessed after the surgery by the patient's global impression scale (PGI, ranging from -3 to +3). The poor outcome was defined as PGI score 0 or less. The location of DBS leads was measured in the postop MRI using Framelink®(Medtronic, USA). **Results:** Among 73 patients with PD who received STN DBS surgery, 11 patients showed poor outcomes (15.1%). PGI score was -1.91 ±1.04 (mean ±SD) for the poor outcome group (n=11), and 1.95 ± 0.73 for the good outcome group (n=62, p<0.01). As for the cause of poor outcome, one case was turned out to be of inappropriate selection for DBS, six cases were due to surgical causes (two case with symptomatic intracranial hemorrhage; four cases with sub-optimal location of the DBS leads), and the rest four cases, all of whom showed optimal location of the DBS leads in the subthalamic nucleus, were due to nonsurgical causes (psychogenic gait disorder, persistent anxiety, persistent psychosis, and severe depression, respectively). Four cases with sub-optimal location of the DBS leads received the reoperation for relocation of DBS leads in the STN with successful outcomes. **Conclusion:** In our data, the suboptimal outcome following STN DBS for PD patients amounted to about 15% of cases. We showed that lead localization using Framelink® could identify cases of poor outcomes that were suitable for reoperation to relocate DBS leads in the STN. Furthermore, our study suggests that a substantial portion of PD patients showed the poor outcome after STN DBS by non-surgical causes, mostly by psychiatric symptomatology.

2

Role of methylmalonic acid and peripheral neuropathy in Idiopathic Parkinson's disease

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Background & Objectives: Peripheral neuropathy has recently described in significantly higher proportions in patients with Idiopathic Parkinson's disease (IPD) than in normal controls (37.8% vs 8.1%). The underlying hypothesis is that persistent levodopa therapy leads to vitamin B12 deficiency, eventually causing peripheral neuropathy in IPD. The aim of our study was to find the role of vitamin B12, methylmalonic acid (MMA) and homocysteine in peripheral neuropathy of IPD and to find the most important surrogate marker that plays an important role in causing peripheral neuropathy in Parkinson's disease. **Method:** We performed a cross-sectional study of 47 clinically and Dopamine transporter scan (DaT scan) positive patients using Total neuropathy scale, revised (TNSr), Korean Neuropathy Questionnaire (KNQ). The prevalence of peripheral neuropathy was determined by the clinical symptom, neurological examination and/or abnormality in their nerve conduction study.

We excluded patients who had diabetes, lumbosacral radiculopathy and autoimmune related disorders including thyroid disease. The IPD group was divided into 2 groups (IPD with and without peripheral neuropathy). We also determined the correlation between age, IPD duration, levodopa equivalent dose (LED), UPDRS III, vitamin B12, methylmalonic acid and homocysteine levels in all patients. We also determined the most important factor that influences neuropathy in IPD. **Results:** 17 of 47 IPD patients (36%) had clinically and electro-physiologically confirmed peripheral neuropathy and this correlates with the recent study that showed a similar prevalence rate of peripheral neuropathy in IPD patients. There was no statistical difference between the 2 groups concerning sex, LED, UPDRS III, vitamin B12, methylmalonic acid and homocysteine. However methylmalonic acid showed a strongly positive correlation to TNSr and KNQ in the IPD patients who had peripheral neuropathy (TNSr : $r=0.882$, $p<0.001$, KNQ $r=0.710$, $p=0.004$), while Vitamin B12 and homocysteine showed no statistically significant correlation to any of the neuropathic scales. **Conclusion:** Vitamin B12 deficiency caused by prolonged intake of levodopa is known to cause peripheral neuropathy in IPD patients. Our study showed a similar prevalence rate of 36% but they had no vitamin B12 deficiency. The level of Vitamin B12 can be normal and MMA is known to be a more sensitive biomarker of vitamin B12 deficiency especially in the early stage. Our study showed no statistical significant difference of vitamin B12, MMA and homocysteine but showed that MMA strongly correlates with the severity of neuropathic pain in IPD patients with peripheral neuropathy. Therefore early screening of MMA is recommended in the clinical assessment of IPD and appropriate supplementation can eventually lead to amelioration of peripheral neuropathy in IPD.

3

Alpha-synuclein in gastric and colonic mucosal tissue in Parkinson's disease: limited role as a biomarker

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Background & Objectives: Gastric and colonic alpha-synuclein (α -SYN) immunoreactivity has been reported in Parkinson's disease (PD) patients. However, enteric α -SYN has also been reported in healthy individuals. We aimed to investigate the utility of α -SYN immunoreactivity from gastric and colonic mucosal tissues obtained by routine endoscopy to detect PD, and to correlate the pathological burden of α -SYN with motor and nonmotor features of PD. **Method:** We recruited 104 study subjects, consisting of 38 PD patients, 13 probable multiple system atrophy (MSA) patients, and 53 controls. Gastric and colonic mucosal tissues obtained by endoscopic gastroduodenoscopy and colonoscopy were assessed using α -SYN immunohistochemistry. Detailed motor and nonmotor features of PD were correlated with enteric α -SYN pathology. **Results:** There was no difference in the enteric α -SYN immunoreactivity among PD patients (31.6% for stomach and 10.4% for colon), MSA patients (40.0% for stomach and 8.0% for colon), and controls (33.3% for stomach and 18.5% for colon). The frequency of positive α -SYN immunoreactivity was higher in gastric biopsy tissues than in colonic biopsy tissues in all of study groups ($P < 0.05$). There was no significant correlation between the presence of α -SYN immunoreactivity and the motor and nonmotor

features of PD. **Conclusion:** The presence of α -SYN immunoreactivity in gastric and colonic mucosa was detected in a similar manner in PD patients, MSA patients, and controls, and thus suggesting the limited role of enteric mucosal α -SYN as a diagnostic biomarker for PD. Future studies are warranted in order to detect pathological α -SYN strains from gastric and colonic biopsy tissues in PD patients.

4

Public knowledge and awareness about Parkinson's disease: a national population based survey in Korea

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Background & Objectives: Parkinson's disease (PD) is a second common neurodegenerative diseases and the prevalence is likely to accelerate as the population of community ages. A variety of motor and non motor symptoms of PD are adversely associated with quality of life and socio-economic burden. Therefore, early diagnosis and intervention would be mandatory for maintaining or enhancing quality of life, social relationship but lacking knowledge and awareness of PD could be hazard for recognition of symptom and seeking expertise advisory. In this study, we aimed to ascertain baseline awareness and knowledge regarding PD and to suggest clue which point should educate the general public about PD in South Korea. **Method:** 22-item structured open-ended and close-ended questionnaire regarding to PD was developed for the study. A cross-sectional survey was performed from April to May 2015. A total 1000 residents in South Korea were randomly sampled and were interviewed by well-trained interviewer. Multivariate logistic regression model was applied to identify factors associated with awareness and knowledge of PD. **Results:** Among PD motor symptoms, tremor (63.5%), rigidity (51.2%), and postural instability (47.6%) were well identified in all respondents. Bradykinesia was only regarded as PD symptoms in 44.4% of respondents. Subjects with aged 20 to 39 years-old and with above 60 years old tended to recognize fewer PD motor symptoms than subjects with 40 to 59 years old. Low education years group also revealed less knowledge about PD symptoms. Cognitive deficit was most well recognized non motor symptoms (55.6%) and 90.9% and 89.1% in respondents reported at least 1 PD motor and non motor symptoms, respectively. In multivariate logistic regression model, age, household income, and education level were independently associated with awareness of PD. Subjects with aged 40 to 59 years and those with completion of 12 year-education were more knowledgeable to awareness of PD (Adjusted OR: 2.40, CI: 1.31-4.52, $p < 0.01$; Adjusted OR: 2.18, CI: 1.24-3.87, $p < 0.01$). Regarding to knowledge of PD definition, younger subjects revealed the least recognition of PD definition compared to 40 to 59 and above 60 years old age group ($p < 0.05$, $p < 0.05$). Low education level and household income tend to be associated with knowledge of PD definition. **Conclusion:** In this study, we demonstrated that awareness and knowledge of PD need to be optimized, especially group with young age, less education, and low income. Lack of awareness and knowledge about PD could lead to uncovered PD patients population and evoked harmful effect of public health care system. To improve appropriate intervention, pertinent educational strategies targeting specific subgroups are necessary to improve public's awareness and knowledge about PD.

5

Postural sensory deficits correlates with gait freezing in Parkinson's Disease

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Background & Objectives: To elucidate the unique patterns of postural sensory deficits contributing to freezing of gait (FOG) in Parkinson's disease (PD) and identify postural sensory determinants that correlate to the severity of FOG. **Method:** Twenty five PD patients with FOG (PD-FOG), 22 without (PD-noFOG), and 26 age-matched controls underwent sensory organization tests. Equilibrium scores and sensory ratios were analyzed to investigate contributions of visual, vestibular, somatosensory inputs to maintain balance. We correlated FOG severity, as measured using a New Freezing of Gait Questionnaire, with posturographic and clinical measures, including the Unified Parkinson's Disease Rating Scale motor score (UPDRS-III), Montreal Cognitive Assessment, frontal assessment battery (FAB), Activities-specific Balance Confidence (ABC), and Berg Balance Scale (BBS). **Results:** In PD-FOG, postural sensory integration was deteriorated significantly during balance demanding conditions, compared to PD-noFOG. Especially, inability to use the vestibular ($p=0.007$, $OR=1.443$) information and control over the perturbed somatosensory inputs ($p=0.044$, $OR=2.904$) contributed significantly to the generation of FOG. FOG severity was more profound in PD-FOG with the higher reliance on visual information ($R=-0.432$, $p=0.039$). It was also correlated with FAB ($R=-0.471$, $p=0.017$), ABC ($R=-0.637$, $p=0.001$), and BBS ($R=-0.484$, $p=0.014$) scores. **Conclusion:** Postural sensory integration deficits are strongly associated with FOG. Quantitative measurements of postural sensory integration in PD patients with FOG may provide a better understanding for pathomechanism of FOG and be used to develop tailored rehabilitation strategies based on specific postural sensory loss.

Scientific Session 18

Epilepsy II

• 시간: 14:20~15:20 • 장소: Emerald Hall B (3F)

1

Relationship between EEG findings and prognosis of post-hypothermia therapy

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Background & Objectives: Therapeutic hypothermia for patients who underwent cardiopulmonary resuscitation (CPR), is widely used with the purpose of reducing neurological sequela. The main objective of this study is to find out the relationship between the EEG findings after the hypothermia and the neurological prognosis. **Method:** From January 2012 to June 2015, 110 patients took EEG after CPR at Chungnam National University hospital. Among them, patients who underwent hypothermia were 52 patients. Twenty-two out of 52 patients took EEG between 8-72 hours after the end of hypothermia, were subjected to this study. The prognosis was classified into four groups (independent-living, partly independent, vegetative state, expired). The relationship between EEG findings and neurological prognosis were analyzed. We also compared the relationship between the clinical factors of age, sex, duration of cardiac arrest, CPR duration, underlying causes of cardiac arrest, and presence of

post-hypoxic myoclonus and prognosis. **Results:** The mean age of 22 patients (17 men and 5 women) was 56 ± 16.5 years. The EEG findings were as follows; severe background suppression (4 patients), suppression-burst patterns (2 patients), cyclic alteration patterns (2 patients), generalized periodic epileptiform discharges (3 patients), irregular mixed delta and theta or posteriorly depressed with frontal delta patterns (11 patients). The patients with burst-suppression and cyclic alteration in the EEG finding were all expired and prognosis of the patients with severe background suppression and generalized epileptiform discharges were mostly remained vegetative state (62.5%) or expired (37.5%). On the other hand, in patients with irregular mixed delta or theta waves, the prognosis was fairly favorable (independent 27.3%, independent 18%). Background activity with generalized slow-wave activity were well correlated with better prognosis compared with other EEG patterns ($P = 0.001$). Other factor, did not have a major impact and to predict the prognosis. **Conclusion:** The majority of patients (17 of 22) had poor outcomes despite of therapeutic hypothermia. EEG with generalized slow-wave activity was correlated with higher survival rate and better prognosis.

2

Significance of thalamus in temporal lobe epilepsy with hippocampal sclerosis: effective connectivity analysis

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Background & Objectives: We hypothesized that limbic network could be different between temporal lobe epilepsy patients with and without hippocampal sclerosis. This study was aimed to evaluate the role of thalamus in temporal lobe epilepsy patients with hippocampal sclerosis. **Method:** Twenty-nine patients with temporal lobe epilepsy with hippocampal sclerosis and 30 normal controls were enrolled in this study. In addition, we included eight patients with temporal lobe epilepsy without hippocampal sclerosis as a disease control group. Using whole-brain T1-weighted MRIs with FreeSurfer 5.1, we analyzed the volumes of the limbic structures including hippocampus, thalamus, and total cortex, and the effective connectivity of limbic network with SPSS Amos 21. Moreover, we quantified correlations between duration of epilepsy and the volumes of these structures. **Results:** There was a statistically positive effective connectivity from the hippocampus to the cortex through thalamus in temporal lobe epilepsy patients with hippocampal sclerosis. Moreover, the volumes of the left and right thalamus were negatively correlated with duration of epilepsy ($r=-0.42$, $p=0.0315$ and $r=-0.52$, $p=0.0062$, respectively). However, there were no differences of effective connectivity between temporal lobe epilepsy patients without hippocampal sclerosis and normal controls. **Conclusion:** The limbic network could be different between temporal lobe epilepsy patients with and without hippocampal sclerosis, and the thalamus might play a critical role in temporal lobe epilepsy patients with hippocampal sclerosis.

3

Cortical and subcortical structural abnormalities in juvenile myoclonic epilepsySung Chul LIM¹, Seong Hoon KIM³, Jiyeon KIM², Woojun KIM⁴, Young-Min SHON²

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Background & Objectives: Some previous neuroimaging studies reported the evidence that the pathophysiology of juvenile myoclonic epilepsy (JME) is associated with structural abnormalities of thalamo-frontal circuit. But recent

studies suggest that structural abnormalities exist extend beyond frontal area. In this study, we studied to investigate the anatomical and pathophysiological nature of JME using multimodal neuroimaging method. **Method:** High-resolution T1 and diffusion tensor images (DTI) were acquired on a 1.5T MRI in 18 patients of JME and 22 normal controls. Between The group comparisons of white matter (WM) water diffusivity alteration and cortical thickness were analyzed by tract-based spatial statistics (TBSS) and Constrained Laplacian-based Anatomic Segmentation with Proximity (CLASP) algorithm, respectively. Volume of bilateral thalamus and hippocampus were also obtained by manual volumetry. **Results:** In patients with JME, compare to normal control, structural abnormalities were detected by multimodal neuroimaging analysis. TBSS revealed that patients with JME had white matter alterations in both anterior superior corona radiate, genu and body of corpus callosum, multiple frontal white matter, left temporal white matter and posterior part of corpus callosum. CLASP demonstrated that patients with JME had the thickness reduction in both medial frontal, parietal, dorsolateral parietal, left medial temporal, right superior temporal and both inferior temporal area. Also, we could observe the definitely significant volume reduction in the thalamus and hippocampus by using manual volumetry in JME patients. The volume reduction of left hippocampus is associated with changes in cortical thickness of the ipsilateral superior temporal lobe, orbitofrontal cortex, inferior bilateral temporal lobe, and supplementary motor area. And the volume reduction of right hippocampus is associated with the inferior temporal lobe, contralateral orbitofrontal cortex, and contralateral supplementary motor area. But, the cortical thickness changes associated with a volume reduction of both thalamus is not observed. The cortical volume change associated with the seizure frequency in JME patients is the parietal (paracentral) lobe. However, the volume reduction associated with seizure duration and age of onset was not observed. **Conclusion:** These findings of our study suggest that structural abnormalities in JME extend beyond frontal lobe, especially temporal lobe. Also, it may suggest that the JME pathophysiology is associated to not only frontal connection but also temporal or extra-frontal connectivity.

4

Temporal lobe epilepsy with amygdalar enlargement: its unique clinical characteristics and prognosis

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Background & Objectives: Amygdala enlargement (AE) can be observed in a certain temporal lobe epilepsy (TLE) patients without MRI signs of hippocampal sclerosis. Some recent studies suggest that TLE with AE probably represent a distinct subgroup which has clinically different to other group of TLE. However, it has remained poorly understood a role for the amygdala enlargement as a focus of epilepsy, unlike hippocampal epilepsy. We aimed to study the impact of AE on clinical significance in patients with TLE. **Method:** We investigated the histories, clinical course, seizure semiology, EEG findings and MRI features in 42 patients who had mesial TLE without hippocampal sclerosis. We compared the clinical variables from 17 mTLE patients with unilateral amygdala enlargement (AE+ group) with those from 25 patients with mesial TLE without AE (AE- group). **Results:** Average age was 41.0 years (SD 13.3) in the MRI negative group and 48.9 years (SD 16.9) in the AE+ group. Average age at onset of epilepsy was significantly higher in the AE+ group 44.7 years (SD 18.8) than in the AE- group 27.9 years (SD 15.1) ($t = 3.20, p = 0.003$). The frequency of CPSs was more often in the AE+ group than in the MRI negative group (15 per month versus 1.8 per month) ($t = 3.91, p < 0.001$). However, no statistically significant difference in the prevalence of febrile seizure between the two groups. With regard to aura constellation, there was no

significant difference between the AE+ and AE- group. Remarkably, AE+ group showed more favorable clinical outcome (higher remission rate) than AE- group ($P = 0.014$). **Conclusion:** TLE with AE probably represents a distinct nosological entity which is most likely a subtype of TLE without ipsilateral HS. The chronic and long lasting inflammatory processes or focal cortical dysplasia could lead to amygdala enlargement possibly. Even if TLE with AE can be a subpopulation of TLE without HS, further larger and long-term studies which cover these aspects should be warranted.

5

Altered thalamocortical functional connectivity in juvenile myoclonic epilepsy: an fMRI study

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Background & Objectives: Juvenile myoclonic epilepsy (JME) represents a common subsyndrome of idiopathic generalized epilepsy, characterized by generalized spike-wave discharges on EEG and generalized seizures. Cumulative evidence over the decades has suggested a critical role of abnormal thalamocortical connection in the fundamental pathogenesis underlying JME. Recently, computational analysis of multimodal neuroimaging data has provided several lines of evidence that patients with JME have structural and functional abnormalities of the thalamus and cortex, especially the frontal lobe. Intrinsic functional connectivity analysis using resting-state fMRI is now a standard technique to disclose brain regions that are functionally connected, and has been shown to successfully identify distinct thalamocortical functional networks in healthy subjects and in patients with brain disorders. In the present study, we used resting-state fMRI analysis to investigate functional connectivity changes of the thalamocortical networks in JME. We hypothesized that specific thalamocortical connectivity (e.g., thalamo-frontal network) would be preferentially affected in patients with JME, and that these alterations would be related to disease severity. **Method:** Consecutive 52 patients with JME (25 females, mean age = 26.0 years) and 52 controls matched for age and gender (26 females, mean age = 27.1 years) were scanned on a 3T MR scanner by acquiring conventional MRI and resting-state fMRI. Seed-based functional connectivity analysis was conducted using SPM12. Mean BOLD signal time course was extracted from each of 5 cortical seeds (prefrontal, motor-premotor, somatosensory, parietal-occipital, and temporal cortices) and was then correlated with the time course of the thalamic voxels, allowing for the calculation of a correlation coefficient for each thalamocortical connectivity using partial correlations. The resulting 5 thalamocortical functional connectivity maps were entered into the second-level random-effects analysis to create within-group functional connectivity maps and to compare maps between groups (corrected $P < 0.05$ with familywise error correction). Functional connectivity strength was retrieved from each thalamocortical map and correlated with disease duration and frequency of generalized tonic-clonic seizures ($P < 0.05$). **Results:** In controls, 5 cortical seeds were connected to distinct, largely non-overlapping regions within the thalamus, according well previous studies. The patterns of functional connectivity between the cortical seeds and thalamus in patients were overall similar to those observed in controls. In between-group comparisons, JME patients showed significant reductions in functional connectivity between prefrontal cortex and left anteromedial thalamus (corrected $P < 0.004$) and right anteromedial thalamus (corrected $P < 0.001$) as compared to controls. The other thalamocortical maps did not differ between patients and controls. Functional connectivity strength between prefrontal seed and anteromedial thalamus was negatively correlated with disease duration (partial correlation, $r = -0.378, P = 0.006$), as with that of motor-premotor seed and ventrolateral thalamus ($r = -0.329, P = 0.018$). **Conclusion:** Our re-

sults indicate that JME is associated with decreased thalamocortical functional connectivity between prefrontal cortex and anteromedial thalamus, further corroborating recent concept of thalamo-prefrontal network abnormality in JME patients. Our finding of greater reduction in functional connectivity strength between thalamus and prefrontal and motor cortices in relation to increasing disease duration suggests that thalamo-frontal network dysconnectivity, the proposed pathophysiologic mechanism underlying JME, may be the consequence of repeated seizure attacks and cumulative epileptiform discharges.

Scientific Session 19

Stroke IV

• 시간: 14:20~15:20 • 장소: Diamond Hall (3F)

1

Degree of early recanalization and functional outcome in acute ischemic stroke with intravenous thrombolysis

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Background & Objectives: The aim of this study is to evaluate the association between timely recanalization during or right after intravenous thrombolysis and functional outcome. We assessed whether degree of early recanalization is associated with reperfusion injury and outcome. **Method:** We prospectively enrolled the patients of acute ischemic stroke with major vessel occlusion who underwent intravenous thrombolysis in our institute between January 1, 2009 and December 31, 2013. Demographics (age, sex, body weight) and baseline characteristics of the patients including vascular risk factors (history of stroke, hypertension, diabetes, dyslipidemia, smoke, atrial fibrillation, and coronary artery disease), initial laboratory findings (total cholesterol, triglyceride, HDL, LDL, glucose, HbA1c, platelet) and initial systolic and diastolic blood pressure, dosage of rtPA used, occluded vessel, stroke mechanism, initial NIHSS, time from symptom onset to intravenous thrombolysis, recanalization status after intravenous thrombolysis, and clinical outcome (MRS at 3 month, occurrence of any or symptomatic hemorrhage) were retrospectively reviewed. Major vessel occlusion included middle cerebral artery (M1, M2), posterior cerebral artery (P1, P2), anterior cerebral artery (A1, A2), internal cerebral artery, basilar artery, and vertebral artery. Recanalization status was confirmed by subsequent MRA or conventional angiography in case of performing CTA first, and by conventional angiography in case of performing MRA first. Partial recanalization (PR) was defined as modified Mori grade 2 (partial recanalization in <50% of the branches in the occluded arterial territory) on MRA, 10 or TIC1 grade 2 on conventional angiography. Complete recanalization (CR) was defined as modified Mori grade 3 on MRA, or TIC1 grade 3 on conventional angiography. Favorable outcome was defined as modified Rankin Scale (MRS) equal or less than 2. **Results:** Among consecutive 3692 patients with acute ischemic stroke, 406 (11.0%) patients underwent intravenous thrombolysis. Major vessel occlusion was documented in 279 (68.7%) patients. 18 patients with premorbid MRS more or equal than 3 were excluded and 261 (93.5%) patients were eligible for the final analysis. Vessel occlusion sites were as follows in order of frequency; M1 (88, 33.7%), M2 (48, 18.4%), distal ICA (47, 18.0%), proximal ICA (38, 14.6%), basilar (21, 8.0%), vertebral (5, 1.9%), P2 (6, 2.3%), P1 (5, 1.9%), and A2 (3, 1.1%). Recanalization was achieved in 41 (15.7%) of the 261 patients during or right after intravenous thrombolysis. PR was achieved in 33 (12.6%), and CR in 8 (3.1%) patients. Any hemorrhage was detected in 6 of 33 (18.2%) among the patients with PR compared with 3 of 5

(62.5%) patients with CR ($P=0.01$). More proportion of patients with PR showed a trend of favorable functional outcome compared with patients with CR (26/33, 78.8% vs. 4/8, 50.0%, $P=0.10$). Univariate analysis associated with favorable outcome in patients received intravenous thrombolysis was as follows: age, male sex, initial NIHSS <18, Smoking, Triglyceride (TG), glucose, HbA1c, diastolic BP, occurrence of any hemorrhage, symptomatic hemorrhage, and degree of recanalization after IV thrombolysis. In multivariable analysis, male, initial NIHSS <18, TG level, diastolic BP, absence of any intracranial hemorrhage or sICH, lower dosage of rtPA, partial recanalization were significantly associated with favorable 90-day outcome. **Conclusion:** Partial recanalization during or right after intravenous thrombolysis could be an indicator of favorable outcome by minimizing reperfusion injury.

2

Use, time delay, and outcomes of drip-and-ship thrombolysis paradigm for patients with acute ischemic stroke; an analysis of the Clinical Research Center for Stroke-5th division registry

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Background & Objectives: Drip-and-ship thrombolysis paradigm is one way to increase the recipient of tPA treatment among acute stroke patients at regional level. The utilization of drip-and-ship thrombolysis paradigm, clinical characteristics and outcomes of patients treated with the paradigm could vary with regional stroke care system. The aim of this study was to describe utilization, clinical characteristics, time delay and to analyze outcomes of drip-and-ship thrombolysis paradigm for patients with acute ischemic stroke using prospective multicenter stroke registry. **Method:** The registry data of Clinical Research Center for Stroke-5 (CRCS-5) which is a web-based prospective, multicenter, nationwide registry of patients with acute ischemic stroke admitted to 14 academic centers in Korea, was used in this study. From the registry, we identified acute ischemic stroke patients treated with tPA who arrived hospital within 6 hours from the symptom onset. Using multivariable analysis, we compared the modified Rankin scale (mRS) score at 3 months and symptomatic intracranial hemorrhages (sICH) between patients treated with drip-and-ship paradigm and those treated via direct visit. **Results:** Among 1,843 patients who met the eligible criteria, 244 patients (13.2%; 95% CI, 11.7-14.9) were treated using drip-and-ship paradigm. Patients treated with the paradigm were more likely to have atrial fibrillation and symptomatic stenosis or occlusion of cerebral artery and less likely to have small vessel occlusion subtype. Patients treated with drip-and-ship paradigm had shorter onset to needle time compared with patients treated via direct visit (median [IQR], 110 minutes [79-150] vs 126 minutes [90-173], $p<0.001$). After multivariable analysis, patients treated with drip-and-ship paradigm had significantly greater risk of unfavorable functional outcome (mRS 2-6) at 3 months after the stroke compared with patients treated via direct visit (OR 2.15; 95%

CI, 1.50-3.08; $p < 0.001$). SICH also occurred more frequently in patients treated with drip-and-ship paradigm (OR 1.78; 95% CI, 1.02-3.12; $p = 0.04$). Of 1,843 patients, 509 patients (27.6%, 71 patients with drip-and-ship paradigm and 438 patients via direct visit) received subsequent endovascular recanalization therapy. The onset to groin puncture time was significantly longer for patients treated with the paradigm compared with those treated via direct visit (305 minutes[260-345] vs 200 minutes[155-245], $p < 0.001$). However, the associations between unfavorable outcome or SICH and drip-and-ship paradigm were not found for patients treated with subsequent endovascular recanalization therapy. **Conclusion:** Drip-and-ship thrombolysis paradigm was used in less than 15% of patients treated with tPA. Approximately one third of the patients treated with drip-and-ship paradigm received subsequent endovascular recanalization therapy with additional treatment delay more than 1½ hour compared with those treated via direct visit. At a regional level, strategies to reduce time delays in interfacility transfer are desperately needed to improve patient outcome.

3

Is it safe to administer antithrombotic medications within 24 hours after recanalization treatments for acute ischemic stroke patients?

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Background & Objectives: Data on the safety of oral antithrombotic agents when given within 24 hours of intravenous thrombolysis and/or endovascular treatment are lacking. Majority of hemorrhagic transformation (HTf) occurs within 24-hour period, and earlier antithrombotics, when HTf was excluded, may be useful in preventing early reocclusion of recanalized cerebral arteries and ischemic neurologic deteriorations in hyperacute period. In this context, the authors hypothesized that the initiation of antithrombotics earlier than 24 hours after recanalization treatment would not increase hemorrhagic transformation of ischemic tissues. **Method:** From a total of 6777 stroke cases who admitted to Seoul National University Bundang Hospital between July 2007 and March 2015, the authors collected eligible cases with the following inclusion criteria; (1) Lesion-documented ischemic strokes (N=5451); (2) received recanalization treatments (N=792). We excluded cases with (1) missing in the time of recanalization treatment initiation or antithrombotics administration (N=19), (2) ultraearly bleeding complications within 24 hours (N=41), (3) limitation in overall aggressiveness of care due to extremely grave prognosis (N=5), and (4) surgical treatment (N=7). Finally, 720 cases were included in the analysis dataset. We systemically gathered the exact timing of antithrombotics use from a database of electronic bar-code medication administration system. The outcome variables were (1) any HTf assessed by follow-up imaging (CT or MRI) at 5 to 7 days after recanalization treatment, (2) symptomatic HTf (associated with increase of NIHSS score ≥ 4 -point), and (3) modified mRS score 0 - 1 at 3 months after stroke. **Results:** Of the 720 analyzable cases, male was 57% (n=407), mean age was 68.9 ± 12.8 , and median NIHSS score was 12 [7 - 19] point. Recanalization treatment was consisted of 34% (n=243) of intravenous-only, 32% (n=231) of endovascular-only, and 34% (n=246) of combined IV-endovascular strategies. Outcomes after stroke was as following; 218 (30%) any HTf, 31 (4%) symptomatic HTf, and 266 (37%) good functional recoveries. In this population, antithrombotics were initiated within 24 hours after recanalization treatment in 64% (n=458) of cases and within 12 hours in 24% (n=170) of patients. In multivariable logistic regression models adjusting for relevant clinical covariates and variables with bivariate $P \leq 0.10$, early initiation of antithrombotics within 24 hours after recanalization treatment was significantly associated with lower odds of having any HTf (adjusted OR, 0.69; 95% CI, 0.48 - 0.98). Early initiation was not sig-

nificantly associated with symptomatic HTf (adjusted OR, 0.71; 95% CI, 0.34 - 1.46) and good functional recovery at 3 months after stroke (adjusted OR, 1.41; 95% CI, 0.97-2.06). **Conclusion:** We documented that earlier initiation of antithrombotics within 24 hours after recanalization treatment did not significantly increase hemorrhagic complications after stroke. Further clinical research is warranted to clarify which subgroup of stroke patients will benefit of earlier antithrombotics.

4

Intra-arterial thrombolysis in acute ischemic stroke: updated meta-analysis of randomized controlled trials

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Background & Objectives: In contrast to the negative results of earlier randomized clinical trials (RCTs), recent RCTs using stent-retriever thrombectomy consecutively or simultaneously demonstrated the benefit of intra-arterial thrombolysis (IAT). We aimed to estimate the IAT benefit in acute ischemic stroke (AIS). **Method:** We systematically searched Pubmed and EMBASE until 31 Apr 2015, using search terms of ischemic stroke AND intra-arterial AND thrombolysis or thrombectomy. Inclusion criteria for our study selection were 1) RCT, 2) the active arm receiving IAT, 3) the control arm receiving standard therapy including IV-TPA, but not treated with IAT, and 4) mRS score reported at 90 days or at the end of the trial. Using a random-effect model, we generated a pooled estimate as an odds ratio (OR) with 95% CI for the effect of IAT on efficacy and safety outcomes for all trials, stent-retriever trials, and RCTs comparing IAT and intravenous TPA. **Results:** We identified 15 relevant RCTs involving 2,899 patients: 1575 patients were randomized to IAT arms and 1324 to control arms. For all trials, IAT compared to control was associated with increased good outcome (OR [95% CI], 1.79 [1.34, 2.40]; $P < 0.0001$). IAT also increased outcomes of no or minimal disability, good neurological recovery, good activity of daily living, and recanalization. IAT was not associated with increased risks of symptomatic intracranial hemorrhage (1.19 [0.83, 1.69]; $P = 0.3453$) and death (0.83 [0.68, 1.01]; $P = 0.0666$). On the contrary, IAT significantly reduced extreme disability or death (0.77 [0.61, 0.97]; $P = 0.0246$). Analysis restricting RCTs comparing IAT and intravenous TPA showed similar efficacy and safety findings. In stent-retriever trials, the benefit was even greater: 2.39 [1.88, 3.04], $P < 0.0001$ for good outcome; 0.57 [0.41, 0.78], $P = 0.0006$ for extreme disability or death. **Conclusion:** This updated meta-analysis shows that the IAT on top of the current standard therapy substantially improve clinical outcome and reduce extreme disability or death without increasing SICH. Now, the time has come to reorganize our acute stroke care system that enables to provide IAT for eligible patients more and faster.

5

Retinal artery occlusion is at an increased risk of subsequent ischemic stroke

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Background & Objectives: Retinal artery occlusion (RAO) is a visually disabling, ocular vascular occlusive disorder resulting in sudden visual loss and

shares risk factors with cerebrovascular and cardiovascular diseases. We investigated systemic vascular risk factors, etiologies and their clinical event during 1 year in RAO patients. **Method:** We retrospectively reviewed 151 consecutive patients with acute non-arteritic RAO from 2003 to 2013 in a single tertiary hospital. Clinical event was a composite of vascular events defined as stroke, myocardial infarction or vascular death and non-vascular event. The Kaplan-Meier survival analysis was used to compute the clinical event rate. **Results:** The mean age was 61.5 year (65.5% males). Hypertension (57.6%) was the most common risk factor for RAO and large artery atherosclerosis (LAA) was main etiology. Among patients received transfemoral cerebral angiography or intra-arterial thrombolysis, steno-occlusion of pure ophthalmic artery and combined ophthalmic and carotid artery was 8.8% (7/80) and 11.3% (9/80), respectively. One year clinical event, vascular event and ischemic stroke rate was 10.6%, 9.3% and 8.6% respectively. More than a half of clinical event occurred within 1 month and over three fourth of patients with ischemic stroke occurred ipsilateral to side of RAO. Independent factor associated with clinical event was LAA as etiology. **Conclusion:** RAO shared same underlying vascular risk factors with ischemic stroke and the most common etiology was LAA. Vascular event rate during 1 year was similar with ischemic stroke. Aggressive medical management for preventing subsequent ischemic stroke after RAO could be recommended.



2015년 대한신경과학회 제34차 추계학술대회

- Poster Presentation I -

【Poster Presentation 좌장명】

Stroke I	P-1-1~P-1-11	이 준(영남의대)
Stroke II	P-1-12~P-1-23	박경필(부산의대)
Stroke III	P-1-24~P-1-35	박종무(을지의대)
Stroke IV	P-1-36~P-1-47	송희정(충남의대)
Stroke V	P-1-48~P-1-59	안성환(조선의대)
Stroke VI	P-1-60~P-1-71	조용진(인제의대)
Dementia I	P-1-72~P-1-83	문소영(아주의대)
Dementia II	P-1-84~P-1-95	윤보라(건양의대)
Dementia III	P-1-96~P-1-107	이현아(계명의대)
Dementia IV	P-1-108~P-1-118	박기형(가천의대)
Epilepsy I	P-1-119~P-1-128	김혜윤(관동의대)
Epilepsy II	P-1-129~P-1-138	김성훈(가톨릭의대)
Headache I	P-1-139~P-1-150	박광열(중앙의대)
Headache II	P-1-151~P-1-162	김수경(경상의대)
Movement I	P-1-163~P-1-173	정선주(울산의대)
Movement II	P-1-174~P-1-184	김중석(가톨릭의대)
Movement III	P-1-185~P-1-195	이지영(서울의대)
MS/NMO I	P-1-196~P-1-207	민주홍(성균관의대)
NO I	P-1-208~P-1-221	이승한(전남의대)
Muscle and Nerve I	P-1-222~P-1-232	김남희(동국의대)
Muscle and Nerve II	P-1-233~P-1-243	배종석(한림의대)
Muscle and Nerve III	P-1-244~P-1-254	안석원(중앙의대)
Muscle and Nerve IV	P-1-255~P-1-265	박민수(영남의대)
Muscle and Nerve V	P-1-266~P-1-276	홍윤호(서울의대)
Sleep I	P-1-277~P-1-286	양광익(순천향의대)
Neuroscience I	P-1-287~P-1-296	김한영(건국의대)
Infection I	P-1-298~P-1-308	이순태(서울의대)
Infection II	P-1-309~P-1-319	김동욱(건국의대)
Miscellaneous	P-1-320~P-1-332	이근호(단국의대)

Poster Presentation I

• 시간: 13:00~14:10

• 장소: 컨벤션센터 1st Floor

P-1-1

Comparison of normal value between transcranial color-coded Doppler sonography and transcranial Doppler sonography in Korean healthy population

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Background & Objectives: Transcranial Doppler (TCD) and transcranial color-coded Doppler (TCCD) have come into general use for the noninvasive study for cerebral hemodynamic status. Several comparative studies between TCD and TCCD by which normal values in TCCD could be estimated, were performed in USA and Europe. However, in Korea, normal value of TCCD could be beyond description. Therefore, we obtained the normal range of TCD/TCCD parameters and compared them between TCCD and TCD among healthy Korean population. **Method:** This study is a prospective observational study among healthy Korean people. The subjects were recruited among those who were visited Korea University Guro Hospital Health Promotion Center with normal brain MR angiography. Those who had previous history of stroke, coronary artery disease, or medication that could influence on the cerebral blood flow velocity were excluded. TCD examination was performed after TCCD examination in the same session. For each segment, peak systolic velocity (PSV), end-diastolic velocity (EDV), mean velocity (MnV), and pulsatility index (PI) were obtained. If there was no color Doppler signal for any vessels and no background echogenicity, we defined it as poor temporal window. **Results:** Finally, 130 subjects (age 51.32 ± 10.24 years, female 44.6%) were included. Poor temporal window were found in 14 for TCD and 19 for TCCD without significant difference. In overall, PSV, EDV, and MnV were higher in female and younger subjects than male and elderly subjects in both TCD and TCCD, respectively. In terms of arterial segments, the velocity was highest in middle cerebral artery followed by anterior cerebral artery, basilar artery, vertebral artery, and posterior cerebral artery. When the subjects were classified into 3 groups with age, most of the velocity parameters (PSV, EDV, MnV) were similar between TCD and TCCD. However, PIs were significantly higher in TCCD than TCD on each arterial segment at different age group. **Conclusion:** In this study, we can provide normal values for TCD and TCCD among health Korean population. Although parameters for velocity were not significantly different between TCD and TCCD, PIs were higher in TCCD than TCD.

P-1-3

The correlation of SOD1 (rs1041740), SOD2 (rs4880), GPx1 (rs1050450), GPx4 (713041) SNPs with Ischemic stroke

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Background & Objectives: Reactive oxygen species (ROS) are small molecules which are unstable, highly reactive and short-lived. ROS play an important

role in the development of vascular disease, including hypertension, atherosclerosis, diabetes, cardiac hypertrophy, heart failure, ischemia-reperfusion injury, and stroke. ROS are usually produced at very low concentration which is controlled by endogenous antioxidant system that include superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase (CAT) and antioxidant vitamins (E and C). Defect in these antioxidant systems may develop oxidative stress and different studies have shown the profound effect of oxidative stress in the stroke pathogenesis because of high susceptibility of the brain to ROS-induced damage. ROS can accelerate oxidation of LDL, forming ox-LDL, which is not recognized by the LDL receptor leading to foam cell formation, a major cause of atherosclerosis which ultimately may cause stroke. Superoxide is dismutated to H₂O₂ by SOD which is further converted to H₂O and O₂ by glutathione peroxidase or catalase in the mitochondria and the lysosomes. The present study was aimed to investigate the association of SOD and GPx polymorphism in the development of stroke. **Method:** A total of 674 patients and 308 controls subjects were recruited in this study. Genotyping of SOD1 (rs1041740), SOD2 (rs4880), GPx1 (rs1050450) were performed by LightCyclerreal-time PCR (Roche, Germany) using Light SNI Preagents (coupled primer and probe, TIBMOBIO, Germany) and Fast Start DNA Master HybProbe (Roche Diagnostics) while genotyping of GPx4 (713041) were done by PCR-RFLP were done by PCR-RFLP. All biochemical parameter were measured in automated clinical chemistry analyzer, at department of laboratory medicine, CBNU hospital. **Results:** A total of 674 patients and 308 controls subjects were recruited in this study. Genotyping of SOD1 (rs1041740), SOD2 (rs4880), GPx1 (rs1050450) were performed by LightCyclerreal-time PCR (Roche, Germany) using LightSNI Preagents (coupled primer and probe, TIBMOBIO, Germany) and Fast Start DNA Master HybProbe (Roche Diagnostics) while genotyping of GPx4 (713041) were done by PCR-RFLP were done by PCR-RFLP. All biochemical parameter were measured in automated clinical chemistry analyzer, at department of laboratory medicine, CBNU hospital. **Conclusion:** Analysis of genetic polymorphism in oxidative stress genes (SOD/GPx) in case-control study suggests a possible role for oxidative stress in the risk of stroke and clearly states that genetic component of stroke is polygenic as altered SOD and GPx genes interaction showed increased risk for stroke development.

P-1-4

Clinical and radiological characteristics in acute cerebral infarction with and without cerebral aneurysm

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Background & Objectives: Asymptomatic intracranial aneurysms are frequently encountered among patients with acute cerebral infarction. Since rupture of asymptomatic aneurysm brings catastrophic consequences, identifying clinical and radiological characteristics of patients who have acute cerebral infarction and asymptomatic intracranial aneurysm (UIA) is important. The aim of this study is to analyze the clinical and radiological characteristics of patients diagnosed with UIA and cerebral infarction in order to wisely select patients who need imaging work up for aneurysm more strongly than others. **Method:** Consecutive adult (≥ 18 years old) patients diagnosed with cerebral infarction admitted to our clinic during 17-month period from Jan 2013 through Jun 2014 were included. Based on MR angiography, we divided and

analyzed patients into those with and without UIAs. **Results:** Of 321 patients, there were significant differences in female sex ($P=0.004$), presence of concurrent cerebral stenosis ($P=0.036$) between groups with (20; 6.2%) and without (301; 93.8%) UIAs. Factors associated with aneurysm in cerebral infarction patients according to logistic regression analysis were female gender (OR 4.37, 95% CI 1.537 - 12.394, $P=0.006$), and cerebral arterial stenosis (OR 2.87, 95% CI 1.004 - 8.18, $P=0.049$). **Conclusion:** Incidental unruptured intracranial aneurysms found in acute cerebral infarction patients show several peculiar characteristics or tendencies including female predominance and cerebral stenosis. When treating female patients diagnosed with acute cerebral infarction and cerebral arterial stenosis, more cautions need to be paid to look for coexisting intracranial aneurysm.

P-1-5

The association between brachial-ankle pulse wave velocity and perivascular space topography in stroke patients

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Background & Objectives: Arterial stiffness is associated with cerebral small vessel disease. We investigated the association between brachial-ankle pulse wave velocity (baPWV) and the topography of MR imaging (MRI) visible perivascular space in acute stroke population. **Method:** We analyzed clinical and laboratory data of acute ischemic stroke or transient ischemic attack patients who had been admitted to Chung-Ang University Hospital within 7 days after symptom onset between January 1st 2014 and May 31st 2015. We included those patients who underwent both brain MRI including T2 weighted sequence and baPWV. The topography of perivascular space was examined in basal ganglia and centrum semiovale level with validating scale (score 0-4), and dichotomized as low (score<3) and high (score \geq 3). **Results:** A total of 481 patients were included (mean age 68.2 ± 12.3 , 44.7% male). When the distribution of baPWV were divided into quartile, the proportions of high PVS in basal ganglia was 10%, 20%, 40% and 40%, respectively. The proportion of high PVS in centrum semiovale was 20%, 40%, 36%, and 49%, respectively. Multivariate logistic regression analysis revealed that the high degree of basal ganglia PVS was associated with $baPWV > 2255\text{cm/s}$ (odds ratio 1.7, confidence interval 1.0-2.7, $p=0.048$) after adjusting age>70 years, hypertension, current smoking status and previous stroke history. The high degree of centrum semiovale PVS showed marginal association with $baPWV > 2255\text{cm/s}$ (odds ratio=1.4, confidence interval 0.9-2.3, $p=0.137$) after adjusting age>70 years and hypertension. **Conclusion:** Our pilot study suggests potential pathophysiological association between arterial stiffness and PVS topography.

P-1-6

Tremor due to left middle cerebral artery occlusion

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Background & Significance: Various types of tremor are observed after stroke. Here, we report a patient presenting with tremor which has a resting, postural and intentional component. The symptom was caused by hypoperfusion due to left middle cerebral artery (MCA) occlusion probably associated with Moyamoya disease. The tremor was fully recovered after revascularization surgery. **Case:** A 33-year-old male smoker was admitted to our center due to left-side weakness and right-side shaking movements which developed 2 weeks ago. The symptoms were intermittent before the visit, but became per-

sist after admission. Neurologic examination showed mild left hemiparesis (Medical Research Council grade IV+) and tremoric movements in the right arm and leg, which were observed at rest, at posture, and with intention. Laboratory tests including complete blood count, chemistry, electrolytes with serum and urine copper and thyroid function tests were within normal limits. Brain MRI showed no acute and chronic ischemic lesions in diffusion weighted image (DWI) and fluid-attenuated inversion recovery (FLAIR). MR angiography(MRA) revealed left MCA occlusion and right MCA stenosis. Transfemoral cerebral angiogram(TFCA) showed complete left proximal MCA occlusion with leptomeningeal collateral flows from anterior cerebral arteries and posterior cerebral arteries. Leptomeningeal collateral vessels filled the cortical territory of the left MCA. However, the basal ganglia was underperfused. Brain Diamox SPECT showed decreased cerebral perfusion and perfusion reserve at the left MCA territory. Because the patient was young and did not have risk factors aside from smokings, the underlying etiology was considered Moyamoya disease (MMD). His baseline systolic blood pressure was 120 mmHg. Continuous intra-venous (IV) phenylephrine was used to increase systolic blood pressure above 150 mmHg, which significantly improved his left limb weakness and right limb tremor. His weakness and tremor re-occurred whenever IV phenylephrine was discontinued. Clonazepam 1.5mg/day was also ineffective. As the patient's neurological findings fluctuated according to his blood pressure, we performed extracranial to intracranial arterial bypass (EIAB) surgery 7 weeks after symptom onset. Left superficial temporal artery was attached to left MCA M4 branch by end-to-side fashion. After this surgery, the weakness and tremor considerably improved. The symptoms completely resolved at 2 months of follow-up. Follow-up of TFCA and brain perfusion SPECT showed improved perfusion in the left MCA territory. **Conclusions or Comments:** Limb shaking is the best-known transient involuntary movements symptom associated with hypoperfusion in the anterior circulation. In this case, patient's symptom was persistent tremor but not transient limb shaking. Our case highlights that tremor may be a manifestation of vascular occlusion associated with hypoperfusion in the basal ganglia.

P-1-7

A case of posterior spinal cord infarction presenting pure sensory disturbance

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Background & Significance: Spinal cord infarctions are a rare but often devastating disorder caused by a wide array of pathologic states. These are uncommon lesions that can occur in the territories of the anterior or posterior spinal arteries, or both. Infarcts in the territories of posterior spinal arteries are rare due to the pial collateral network and the dual posterior spinal artery. The diagnosis is generally made clinically, with neuroimaging to confirm the diagnosis and exclude other conditions. We describe a case of acute posterior spinal cord infarction. **Case:** A 31-year-old right-handed man came to the emergency room because of numbness on the right arm and leg. At 7:00 A.M. on the day of admission, he suddenly felt numbness on right arm and leg. He also complained that he could not feel things with right arm and that it felt tingling. Furthermore, he had some difficulties in using right hand. On neurologic examination, the power of both arms and legs are intact. Sensory examinations showed intact in light touch and pinprick sense. But, It were dominantly decreased position and vibration sense in right arm and leg. There were hyperreflexia on both patellar and ankle. And He suffered from upper posterior cervical pain without any trauma or neck extension history. Spinal cord magnetic resonance image (MRI) showed that hyperintense signal zone with T2WI in the right dorsolateral portion of upper cervical level. Also high signal change in diffusion WI (DWI) with decreased signal in apparent diffusion co-

efficient(ADC) value were seen. Other laboratory results such as antithrombin III, antinuclear antibody(ANA), anti-neutrophil cytoplasmic antibody(ANCA), anti ds DNA antibody, protein C, protein S, C3, C4 were normal. Antiplatelet agent was started for treatment. Symptoms improved after 2 weeks. **Conclusions or Comments:** We present a case of posterior spinal cord infarction in the young with no cardiovascular risk factors. The cause of arterial infarction remained unknown, but neurologists should be aware of the fact that posterior cord infarction can occur in the young with no cardiovascular risk factors.

P-1-8

A case of spontaneous middle cerebral artery dissection with distal internal carotid artery involvement confirmed by high-resolution MRI

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Background & Significance: Dissection of the isolated middle cerebral artery (MCA) is an uncommon cause of stroke and is less frequent compared with dissection of vessels in the vertebrobasilar. Recently, high-resolution cross-sectional MRI (HRMRI) has emerged as a potentially useful technique for atherosclerotic plaque imaging in the MCA. Here we report cases of spontaneous middle cerebral artery dissection with internal carotid artery involvement confirmed by high-resolution MRI which could not find in routine MRA and conventional angiography. **Case:** A 49-year-old man visit the emergency department with speech disturbance. He had no history of head trauma, but experienced aortic dissection, Stanford type B in 2 years ago. On neurological examination, he had mild fluency impairment with dysarthria. He did not show sign of motor weakness and other cranial nerve sign were normoactive bilaterally. Brain computed tomography(CT) showed no sign of hemorrhage or ischemia. Diffusion Weighted Image(DWI) showed multifocal small ischemic change in left MCA territory. MR angiography and trans-femoral cerebral angiography showed focal stenosis on left MCA with intraluminal irregularity without other vessel involvement. Additional lab was done for rule out other vasculitic disease but all result showed negative. Transthoracic echocardiogram for finding cardiac source was within normal range. HRMRI was done for evaluate pathophysiology of MCA stenosis and it revealed hyperintense sign on T1-weighted images (WI) visualized intramural crescentic enhancement in patient's left MCA and ICA. He was treated with aspirin 100mg and statin 40mg daily for treatment of acute ischemic stroke with intracranial artery dissection. **Conclusions or Comments:** MCA dissections are very low incidence rate, approximately 4% of cervicocephalic arterial dissection. MCA dissection with ICAD were much more than isolated MCA dissection. Some authors have suggested that the impact of the distal ICA-MCA against the sphenoid ridge causes an intimal tear and even rupture, which resulted in a posttraumatic dissection. However, typical cerebral angiography findings are uncommon and it can't find intramural pathophysiology. Our patient done serial imaging of MCA for finding pathophysiology of isolated MCA stenosis, and there were no definite evidence of dissection by clinical history and imaging finding. But in HRMRI, slight hyperintense on T1 and T2WI were seen which were characteristics of subacute hematomas in distal ICA and MCA. Recently, HRMRI has emerged as a potentially useful technique for atherosclerotic plaque imaging in the MCA. We could detect distal ICA dissection which propagate to proximal MCA dissection by using HRMRI which could not find routine brain MRA and conventional angiography. Because precise diagnosis and treatment is important to prevent deterioration of neurologic symptom by hemorrhage or ischemia, HR MRI in isolated MCA dissection or stenosis can make early differential diagnosis of pathophysiology and helpful patient's treatment.

P-1-9

Intracranial venous reflux caused by occlusion of brachiocephalic vein mimicking intracranial dural arteriovenous fistula

Subin LEE, Dooyong PARK, Doyoung YOON, Gwanhee EHM, Jee Young KIM, Jong-Ho PARK, Hyun Jeoung HAN, Chang Hun KIM

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Background & Significance: High signal intensity of cerebral venous sinuses on magnetic resonance angiography (MRA) indicates high flow of shunt from arterial blood suggesting of dural arteriovenous fistula (AVF) or arteriovenous malformation. We report a patient with intracranial venous reflux caused by occlusion of brachiocephalic vein (BCV) mimicking dural AVF on MRA. **Case:** A 71-year-old women receiving AVF surgery for hemodialysis presented with headache with nausea for several months. Left jugular vein, inferior petrosal sinus and ophthalmic vein showed high signal intensity on MRA suggesting intracranial dural AVF. However, cerebral angiography revealed left BCV occlusion and intracranial venous reflux through jugular vein from left subclavian vein on ipsilateral AVF site. After balloon angioplasty with stenting for BCV occlusion was performed, normal venous drainage was restored, and her symptoms were also resolved. **Conclusions or Comments:** Central venous occlusion is common in patients with AVF for dialysis. Intracranial venous reflux resulting from occlusion of BCV induces various neurological disorders including intracranial hypertension or venous cerebral infarction. Prompt recognition and endovascular treatment can be helpful for the treatment of symptomatic central venous occlusion.

P-1-10

Bilateral pontine infarction with branch atheromatous plaque

Joon Won LEE, Si Eun KIM, Hyung Chan KIM, Soo Young BAE, Sung Eun KIM, Kyong Jin SHIN, JinSe PARK, Kang Min PARK, Sam Yeol HA

Department of Neurology, Haeundae Paik Hospital, Inje University College of Medicine

Background & Significance: Bilateral anteromedial pontine infarction with "heart appearance sign" on MRI is a very rare case. Pathophysiology of bilateral pontine infarction is suggested by atherosclerotic change of basilar artery involving the bilateral paramedian and short circumferential branches and sparing the long circumferential branches. The purpose of the article is to reveal the atherosclerotic change using high-resolution vessel wall MRI. **Case:** A 79-year-old woman presented with right leg weakness and bulbar symptoms of dysarthria and dysphagia followed by progressive weakness of all four limbs. A repeated MRI performed two days after admission disclosed bilateral pontine infarction with branch atheromatous plaque. EKG, echocardiography and Holter monitoring for 24 hours revealed no cardiac source of embolism. **Conclusions or Comments:** Our case showed the atheromatous plaque involving bilateral paramedian and antero-medial arterial branches in the basilar artery with enhancement in high-resolution vessel wall MRI suggesting cause of the bilateral pontine infarction.

P-1-11

Sinking skin flap syndrome after decompressive craniotomy

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Background & Significance: Sinking skin flap syndrome (SSFS) is a rare complication of skull defect that after craniotomy. SSFS is consists of sinking skin flap with neurologic symptoms. When these changes are recovering, the neu-

rological symptoms and sunken skin flap may also be resolved. We present a case suggesting that SSFS is able to be developed by the skull defect after the decompressive craniotomy in the patient with cerebral infarction. **Case:** A 69-year-old woman visited us because of stupor mentality. She had a history of hypertension, diabetes mellitus, congestive heart failure, complete atrioventricular block and atrial fibrillation. She had a pacemaker inserted 13 years ago due to complete atrioventricular block. In neurological examination, the mentality was stuporous state, and had global aphasia and right side hemiplegia. She was diagnosed with the left middle cerebral artery infarction with the occlusion of the left distal internal carotid artery by the brain imaging studies and underwent the intra-arterial mechanical thrombo-embolectomy. By the intervention the occluded artery was recanalized, and then she regained consciousness but still had aphasia and hemiplegia. On one day hospitalization, cerebral herniation was developed, so the decompressive craniotomy was performed. After the craniotomy, she recovered her consciousness but continued hospitalization due to pneumonia and diarrhea. On 23th days hospitalization, she underwent lumbar puncture for CSF laboratory test in order to search for the cause of fever. On the second days after lumbar puncture, her mentality was decreased again. At the examination her left pupil was fixed and dilated and the skin of the craniotomy site sunk. Brain CT scans showed the concave feature of craniotomy site with transtentorial herniation. We were supplying adequate fluids to her, and lowered her head to craniotomy site down. After then, her awareness and light reflex of pupil were recovered. Also the recovery of sinking skin flap and transtentorial herniation were observed on follow up CT. After a month, she had the cranioplasty by autograft skull and then was transferred to hospital care without neurological deterioration. **Conclusions or Comments:** Sinking skin flap syndrome (SSFS) is a rare complication after craniotomy and consists of sinking skin flap with neurologic symptoms such as severe headache, mental change, focal neurologic deficit and seizure. SSFS may be progressed to paradoxical herniation when the intracranial pressure (ICP) falls below the atmospheric pressure. This also can lead to coma or death. Although the obvious mechanism did not revealed, direct cortical compression, hydrodynamic disturbance of CSF parameters, hemodynamical reduction of cerebral blood flow (CBF), cerebrovascular reserve capacity, venous return and cerebral metabolism disturbance are supposed to the mechanism of SSFS. Risk factors of SSFS are thought to be lowering ICP treatment (such as mannitol, CSF drainage, hyperventilation), upright posture, CSF leakage and dehydration. The treatment is proposed to reduce the pressure gradient between ICP and atmospheric pressure such as head down posture (Trendelenburg position), adequate hydration, cranioplasty. SSFS is a rare complication but maybe fatal, so watchful care is needed to patients had a skull defect. In particular to the patient with craniotomy, the extreme caution is required about dehydration and lumbar puncture study as well as adequate timing for cranioplasty.

P-1-12

Comparative outcome and pathomechanism in acute stroke model after hypoxia and exercise

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Background & Objectives: Obstructive sleep apnea and exercise and have conflicting effect on the ischemic stroke. Obstructive sleep apnea has negative effect on ischemic stroke, but exercise prevents and protects deficit from ischemic stroke. In this study, we show that chronic hypoxia and exercise result differently in mice ischemia model. **Method:** Male C57BL6 mice were randomly assigned to three experimental groups: hypoxia (n = 15), exercise (n = 12), and normal control (n = 8). Chronic hypoxia was exposed to 15 mice using automated hypoxic chamber. 12 mice in exercise group were exposed to voluntary chronic aerobic exercise for total two weeks. Cerebral ischemia was induced by

performing intraluminal filamentous occlusion of the MCA permanently. The Y-maze test and neurological function scoring were conducted by 2 individuals blinded to the mouse treatment status. At 72 h after the ischemic insults, ischemic volume was evaluated in the ischemia model. To examine the carotid body status of exercise and chronic hypoxia, total RNA was isolated from each carotid bifurcation and common carotid artery and we conducted RT-PCR. **Results:** Chronic hypoxia resulted in high mortality in acute period after carotid occlusion compared with the control group ($p < 0.05$), and showed worse neurological outcome compared with the control group in hyper-acute stroke period (24 h after ischemia) in neurological scoring ($P < 0.05$). Exercise group showed better neurological outcome compared with the control group in acute stroke period (up to 3 days after ischemia) in neurological scoring and Y-maze tests ($P < 0.05$), but the infarction size was not definitely smaller in exercise group ($P = 0.051$, mean 57% reduction), although the tendency was positive. The amount of exercise was correlated with the neurological outcome in Y-maze test ($P < 0.05$). RNA results are being analyzed and will be added to these results. **Conclusion:** These results show that chronic hypoxia might lead to poor outcome after ischemic stroke, whereas exercise can have importance role in reducing the neurological deficit by acute ischemic stroke.

P-1-13

Associations among disruption of cholinergic pathways, cholinergic-innervated cortical/subcortical volumes, and cognitive function in Alzheimer's disease with vascular factors: Methodologic aspects and preliminary results

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Background & Objectives: Interactions between small vessel diseases (SVD) and cognitive functions were reported in the previous studies. Furthermore, these effects were varied according to SVD burden and distributions. However, the exact mechanisms how SVD contributes cognitive impairment is not yet conclusive. Cholinergic pathway has gained attention for its pivotal role to maintain normal cognitive function. Thus, we aimed to investigate the associations between small vessel diseases burden and distributions of the cholinergic pathways and structural changes of cholinergic innervated cortical/subcortical regions. **Method:** We have enrolled patients who were diagnosed as mild to moderate Alzheimer's disease and those who were undergone volumetric MRI. Among them, subjects who had at least one of SVD markers were included: WMH and/or old lacunar infarction and/or cerebral microbleeds. Cholinergic Pathway HyperIntensities Scale scores were modified to quantify the cholinergic pathway disruptions by WMH, lacunes, and microbleeds. Cortical thickness and subcortical volumes of cholinergic innervated structures - prefrontal cortex, hippocampus, amygdala, ventral striatum - were analyzed using Freesurfer software. Correlation analyses were done among total scores of modified CHIPS scores, volumetric MRI biomarkers, general cognitive scores. Lastly, distribution of WMH, lacunes, and microbleeds were visualized using cumulative mapping in standard templates. **Results:** The results of preliminary analysis will be presented. **Conclusion:** Our study would reveal the underlying mechanisms how small vessel diseases might contribute to the cognitive impairment in the network perspectives. Small vessel diseases in the cholinergic pathway might contribute to the degeneration of innervated cortical/subcortical structures, which result in the corresponding cognitive impairment. This study has implications for identifying good candidates who would respond to cholinesterase inhibitors treatment.

P-1-14

Post-stroke delirium in acute stroke care unit

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Background & Objectives: Post-stroke delirium is a common problem in care of stroke patients and is known to be associated with longer hospitalization, high short term mortality and increased need for long-term care. It usually occurs in about 10~30% of patients but little has been known about the risk factors of post-stroke delirium in acute stroke care unit. **Method:** A total of 576 consecutive patients with stroke (mean age, 65.2 years; range, 23-93 years) were screened for delirium from 2012/8/1 through 2014/7/31 in acute stroke care unit of a tertiary referral hospital. We screened delirium with the Confusion Assessment Method (CAM). Once delirium was suspected, we evaluated the symptoms with Korean Version of the Delirium Rating Scale-Revised-98 (K-DRS-R-98). Neurologic deficits were assessed with the NIH Stroke Scale at admission and discharge and functional ability with the Barthel Index (BI) and modified Rankin Scale (mRS) at discharge and 3 month after discharge. **Results:** Thirty-eight patients with stroke (6.7%) developed delirium during admission in acute stroke care unit. Patients with delirium were significantly older (70.6 vs 64.9, $p=0.001$) and smoking cigarette more frequently (40% vs 24%, $p=0.050$) compared to no delirium group. In terms of clinical features, delirium group had significantly higher rate of major hemispheric stroke (55% vs 26%, $p<0.001$) and showed poor functional performance at discharge and 3 months after discharge and significantly longer hospitalization period than no delirium group. Independent risk factors for delirium were older age, cigarette smoking and major hemispheric stroke. **Conclusion:** Post-stroke delirium occurs frequently in patients with advanced age and substance abuse history such as smoking or alcohol and major hemispheric stroke. The occurrence of delirium after stroke is associated with longer hospitalization period and worse outcomes and should be monitored and managed carefully.

P-1-15

Tachycardia burden in stroke unit is associated with functional outcome after ischemic stroke

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Background & Objectives: Stroke unit care is associated with decrease in mortality and improvement in neurological outcome in patients with acute stroke. Heart rate is a commonly monitored variable in the stroke unit. However, little is known about tachycardia burden in the stroke unit and its association with outcome. We investigated the effects of tachycardia burden in the stroke unit on functional outcome in patients with acute ischemic stroke. **Method:** We collected data from 251 patients with acute ischemic stroke admitted to our stroke unit between July 2013 and June 2014. Tachycardia burden was defined as duration of heart rate over 95 per minute divided by the total monitoring time, using the heart rate data sampled every 1 minute. We divided the study population into quartiles of tachycardia burden and analyzed their association with poor 3-month functional outcome (modified Rankin Scale score of ≥ 3). **Results:** Among included patients (age, 67.5 ± 12.8 ; male, 53.8%), tachycardia burden was 0.7% (median, interquartile range [0.1%-5.8%]). The patients with higher tachycardia burdens were older, more likely to have higher stroke severity, cardioembolic etiology, atrial fibrillation, fever, pneumonia, higher initial glucose level and higher white blood cell count. As compared with the lowest quartile ($<0.1\%$), the highest quartile of tachycardia burden ($\geq 6.0\%$)

was significantly associated with poor outcome (adjusted odds ratio, 5.10; 95% confidence interval, 1.38-18.90; $P=0.01$) after adjustment for covariates. **Conclusion:** Patients with increased tachycardia burden during stroke unit stay have poor functional outcome. Countermeasures against worsening factors might be utilized for patients with increased tachycardia burden.

P-1-16

Impact of cardiac function on outcome after ischemic stroke in patients with atrial fibrillation

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Background & Objectives: Atrial fibrillation (AF) adversely affects cardiac hemodynamics and consequently leads to an increased risk of intra-cardiac thrombus formation and embolism. Previous studies suggested that stroke patients with AF have a worse prognosis than those without AF. However, there is paucity of data on predictors of outcome among stroke patients with AF. In this study, we hypothesized that the cardiac dysfunction could predict stroke outcome in patients with AF. **Method:** This retrospective study was performed on consecutive patients with ischemic stroke within 7 days of symptom onset and with AF. Neuroimaging and transthoracic echocardiographic (TTE) findings were evaluated. LV dysfunction was defined as presence of a severe RWMA or systolic/diastolic dysfunctions which were defined as $EF \leq 40\%$ and $E/E' > 15$, respectively. Functional and radiologic outcomes were analyzed. **Results:** Of 453 patients, 153(33.8%) were classified as Left ventricular (LV) dysfunction group, and 300(66.2%) as normal LV function group. NIHSS score at admission and mortality rate were lower in normal LV function group ($p=0.015$ and $p=0.0175$). Two groups had distinct distribution of mRS at 1month after stroke onset. Normal LV function group had a higher good-prognosis ($mRS \leq 2$) ($p=0.029$) For the radiologic outcome, there was no difference in DWI volume between two groups. But the ratio of large vessel occlusion was higher in LV dysfunction group (22.0% vs 13.4%; $p=0.02$). In a multivariable model adjusted for other covariates, LV dysfunction were independently associated with large vessel occlusion (OR, 1.89; 95% CI, 1.098-3.255, $p=0.022$). **Conclusion:** Among ischemic stroke patients with AF, LV dysfunction evaluated by TTE-derived parameters influenced risk of large vessel occlusion regardless of pre-admission medication.

P-1-17

Bilateral substantia nigra lesions on MRI in a patient who presented with abulia

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Background & Significance: Abulia is defined as impairment of ability to perform voluntary actions and show initiative along with decreased movement and emotional reaction. This is the behavioral disturbance commonly caused by bilateral central core brain lesions which can reduce dopaminergic activity. We report a case diagnosed with abulia after cerebrovascular attack that exhibited lesions in bilateral substantia nigra on MRI. **Case:** A-71-year-old female developed decreasing of speech, movement, and emotional reactions for 2 days. Physical examination findings were unremarkable. Her mental status was alert. Neurological examination revealed mild dysarthria, expressionless face, diminished blinking, a monotonous voice, cogwheel rigidity, decreased response to outer stimulations, and dysphagia. Brain MRI diffusion imaging performed on the first hospital day showed high-intensity lesions in the bilateral substantia nigra. There were also found old infarction lesions in right

pons and cerebellum on T2-weighted and FLAIR image. Brain FDG PET performed on 8 day after admission revealed decreased metabolism of left frontal cortex. A treatment with levodopa/benserazide and methylphenidate was started on day 10. Dose of dopaminergic medication increased gradually until 200mg levodopa/50mg benserazide thrice daily. The patient partially recovered 1 month later, she showed spontaneous communication and walked with assistant device. **Conclusions or Comments:** There was no report about abulia after bilateral substantia nigra infarction. This case suggests that bilateral substantia nigra lesions may damage to mesencephalic dopaminergic pathways and cause abulia.

P-1-18

Isolated proximal lower extremity weakness in a patient with small cortical hemorrhage

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Background & Significance: In general, pure leg monoparesis thought to be caused by a spinal cord, plexus or peripheral nerve lesions. Cerebral lesion also could lead to pure monoparesis, but it is very rare. We report an isolated proximal lower extremity weakness in a patient with small cortical hemorrhage. **Case:** An 80-year-old woman complained sudden onset of right leg weakness. She noticed the weakness of her right leg while walking. She had past medical history of diabetes. General physical examination was normal. On neurologic examination, she was alert and oriented. There was no aphasia, agnosia, or apraxia. Her cranial nerves were all intact. Motor examination revealed weakness of the right hip flexor (Medical Research Council grade, 3/5) and right hip extensor (Medical Research Council grade, 2/5). The rest of the motor examination findings were normal. There was no muscular atrophy in both proximal and distal muscles bilaterally. Deep tendon reflexes were symmetric, and no pathological reflexes were observed. Her coordination and sensory systems were normal. Electrocardiography revealed normal sinus rhythm. Non-contrast CT images showed focal high density in the left fronto-temporal cortex. Diffusion weighted images and fluid-attenuated inversion recovery images revealed hypointensity lesion in identical location. CT angiogram demonstrated no evidence of significant arterial stenosis, aneurysm or dissection. Two days after the onset of the symptom, T2-weighted gradient-echo MRI revealed focal hypointense lesion in the medial region of the right precentral gyrus. She had received physical rehabilitation therapy. Two weeks later, her right proximal leg weakness improved to motor grade 4. **Conclusions or Comments:** Small lesion was located in the precentral gyrus, more medially than the precentral knob, which is a reliable landmark of the motor hand area and more laterally than the topmost part of the precentral gyrus, which is distal leg motor area. Previous studies suggest that proximal leg motor area located between hand knob and distal leg motor area. We report an isolated proximal lower extremity weakness in a patient with small cortical hemorrhage.

P-1-19

A case of anterior spinal cord infarction after bronchial artery embolization

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Background & Significance: Bronchial artery embolization (BAE) remains the primary and most effective method in controlling massive hemoptysis. However, several complications of BAE have been reported. Anterior spinal cord infarction is a rare but serious complication that can occur during BAE. The embryonic regression of blood supply to the anterior spinal cord and the

relatively large area supplied by each anterior spinal artery makes it quite vulnerable to disastrous ischemia during intervention. Early recognition of anterior spinal cord infarction after BAE by clinicians is crucial. There are few reports of anterior spinal artery syndrome associated with BAE in Korea. **Case:** A 58-year-old man with a 3-year history of pulmonary tuberculosis presented with severe hemoptysis (200ml). In an effort to control his hemoptysis, the patient was taken to the interventional radiology suite and underwent angiography, which demonstrated hypertrophy and tortuosity of both bronchial and multiple right intercostal arteries. Both bronchial and right 1st to 8th intercostal arteries were superselected with a microcatheter system. Embolization was performed via the microcatheter using 350 to 500 μ m polyvinyl alcohol and 500 to 700 μ m gelatin sponge particles. The day following procedure, the patient demonstrated acute paraparesis with voiding difficulty. Sensory examination revealed sensory levels below T8 on the right side and T9 on the left side. Magnetic resonance imaging (MRI) of the thoracic spine revealed short segmental linear T2 hyperintensity in anterior spinal cord at the T7 level with subtle enhancement. The patient was treated with steroids and physical therapy. His muscle strength of lower extremities was rapidly improved over the course of 2 weeks. **Conclusions or Comments:** We report a case of anterior spinal cord infarction associated with BAE. Early detection of anterior spinal cord infarction using MRI is vital for proper management and favorable prognosis.

P-1-20

A case of hemichorea following parieto-temporal infarction sparing basal ganglia

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Background & Significance: Vascular chorea is typically associated with ischemic or hemorrhagic lesions involving basal ganglia. Hemichorea after cerebral infarction sparing basal ganglia is extremely rare. We report a case which developed hemichorea of the right extremities following left parieto-temporal infarction without basal ganglia involvement. **Case:** A 77-year-old right-handed woman was admitted to our emergency room with sensory aphasia. A diffusion-weighted image showed acute infarction in the territory of the inferior division of the left middle cerebral artery. The basal ganglia, thalamus, and brainstem were normal. A few hours after admission, she developed hemichorea of her right arm and leg. Follow up computed tomography scan on the 2nd hospital day showed a low density only in the same areas of previously seen diffusion restriction. Her choreic movement gradually improved with haloperidol. Hemichorea lasted for 4 days and disappeared. **Conclusions or Comments:** This case demonstrates that hemichorea can occur in acute stroke patient with parieto-temporal lesion sparing basal ganglia.

P-1-22

Dural arteriovenous fistula presenting as a seizure mimicking transient ischaemic attack: Advantages of susceptibility-weighted imaging

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Background & Significance: A dural arteriovenous fistula (DAVF) can occur anywhere within the intracranial dura mater. Patients with DAVF may be completely asymptomatic. When symptoms are present they may range from mild (e.g. tinnitus, bruit or headache) to more fatal ones (e.g. intracerebral, subarachnoid or subdural haemorrhage). In addition, there are previous reports of patients with DAVF mimicking transient ischaemic attack or seizure disorder, similar to our patient. In addition to its non-specific manifestations, it is

generally less conspicuous on conventional imaging modalities. For these reasons, the diagnosis of DAVF is always challenging. Here in, we introduce a case of middle aged woman, who presented with ambiguous symptom and efficiency of susceptibility weighted imaging (SWI) in diagnosis of such obscure vascular malformation, compared with other conventional magnetic resonance image (MRI) sequences. **Case:** A 56-year-old woman with known hypertension, diabetes and dyslipidemia presented with transient dysarthria and right hemifacial numbness. On brain MRI scanning, diffusion-weighted imaging (DWI) showed no acute lesion and fluid attenuated inversion recovery (FLAIR) imaging showed abnormal high-signal intensity lesions at the left fronto-parietal juxtacortical area. Gradient echo sequence (GRE) and angiography did not reveal abnormalities suggesting vascular malformation. Three weeks later, she again presented to the emergency department with a secondary generalized seizure. In a second brain MRI that included SWI, the FLAIR findings were nearly identical with those found 3 weeks prior. However, in contrast to the prior GRE imaging, the SWI showed distinctly abnormal findings that strongly suggested vascular malformation. Digital subtraction angiography (DSA) confirmed a dural arteriovenous fistula at the left cavernous sinus. **Conclusions or Comments:** In our case, SWI has higher sensitivity for detecting this vascular abnormality than other conventional sequences, enough to depict lesions that are invisible on T2-GRE images. Prominence of the venous vasculature caused by DAVF can be identified through SWI, because of its high sensitivity to detect and delineate intravascular deoxygenated blood and venous structures. Besides DAVF, several other types of vascular malformations can also be visualized with SWI, including developmental venous anomalies, cerebral cavernous malformations, telangiectasia and Sturge-Weber syndrome. We suggest that SWI can be useful in diagnosing many other ambiguous cerebral vascular malformations.

P-1-23

A case of posterior circulation infarction associated with spinal cord infarction

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Background & Significance: Posterior circulation infarction may coexist with cervical spinal cord infarction because higher cervical spinal cord is supplied by anterior spinal artery from vertebral artery. This is the first report of top of basilar syndrome which simultaneously occurred with cervical spinal cord infarction. **Case:** A 74-year-old woman with alleged history of hypertension and diabetes was presented with drowsiness and generalized weakness which developed 2 hours ago. On neurological examination, bilateral upper and lower extremities weakness (MRC grade II on right side, grade III on left side) and decreased sensation of pain, temperature and light touch on four extremities and trunk were observed. Deep tendon reflexes were all normo-active and Babinski reflex was observed on right side. Her brain CT looked normal and intravenous tissue plasminogen activator was injected. Diffusion weighted brain MRI revealed acute ischemic lesion on right cerebellum, right occipital lobe, and right thalamus, all of which are not responsible for her four-limbs weakness. On CT angiography, right proximal vertebral artery was severely narrowed and the whole left vertebral artery was not visualized. Thereafter, spinal cord MRI revealed high signal intensity with edematous change on anterior aspect of upper cervical spinal cord which suggests acute ischemic infarction. **Conclusions or Comments:** This case shows the co-occurrence of posterior circulation infarction and cervical spinal cord infarction caused by both vertebral artery atherosclerosis.

P-1-24

The comparison of characteristics between Solitaire stent and Trevo stent in Mechanical thrombectomy

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Background & Objectives: Recent reports have showed that mechanical thrombectomy (MT) has been demonstrated to improve functional outcome in patients with acute ischemic stroke (AIS). Their main device for MT was Solitaire stent. Trevo stent is a novel embolectomy device specifically designed to remove the thrombus in AIS. These two retrievable stent are known as effective devices for successful recanalization. We report our experiences to compare the safety and effectiveness of two retrievable stent systems during MT. **Method:** From January 2014 through April 2015, all seventy patients underwent MT for AIS with anterior circulation stroke. Among them, fifty two patients underwent MT using Trevo or Solitaire stent. Patients were treated either with Trevo stent or Solitaire Stent according to the neurointerventionist preference. Recanalization was classified by TICI grade. Efficacy and safety during MT was analyzed first recanalization TICI grade after puncture, clot retrieve rate, final recanalization grade, pass number of stent, necessity of rescue method, hemorrhagic complication and thromboembolic complication. **Results:** Twenty nine were treated with Solitaire stent and 23 patients with the Trevo stent. Overall good recanalization (TICI 2b and 3) was achieved in 18 patients (62%) in the solitaire group and in 20 (87%) of the Trevo group (P = 0.043). First recanalization TICI grade after puncture, pass number of stent, necessity of rescue method were not significant between two groups. However, clot retrieve rate was 100% in Trevo group and 79% in solitaire group (P = 0.023). Rate of symptomatic ICH was 14.2% for Trevo versus 11.5% for Solitaire. Rate of thromboembolism was 14.2% and 19.2% for Trevo and Solitaire. **Conclusion:** Our study showed superiority of Trevo stent to achieve successful recanalization and to retrieve the clot from the vessel. Higher recanalization rates of Trevo stent may be caused by higher clot retrieve rate.

P-1-25

Intravenous thrombolysis with recombinant tissue plasminogen activator for ischemic stroke patients over 80 years old

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Background & Objectives: Intravenous recombinant tissue plasminogen activator (rtPA) given within time window is established treatment for acute ischemic stroke. However, the safety and efficacy of intravenous rtPA therapy for very elderly patients remains controversial. Among patients treated with rtPA, old age is a predictor for poor outcomes and complications. We reviewed clinical outcomes of patients aged over 80 years treated with rtPA for acute ischemic stroke. **Method:** We retrospectively reviewed the treatment cases with IV rtPA in patients aged over 80 with acute ischemic stroke from January 2005 to May 2015. All patients satisfied the indication and exclusion criteria of National Institute of Neurological Disorders and Stroke (NINDS) rtPA stroke trial. We obtained data for development of symptomatic intracranial hemorrhage (ICH), functional outcome at discharge, and in-hospital mortality. ICH was investigated by brain CT or gradient echo MR imaging (GRE). Poor functional outcome was defined as mRS >2. **Results:** A total of 27 patients were included. Male were 9 patients (33.3 %) and mean age were 83.9. Occluded vessel was mostly middle cerebral artery (n=18, 66.7%) and internal carotid

artery (n=4, 14.8%). Basilar artery occlusion was present in 2 patients and other territories in 3 patients. Onset to door time was 69 +/- 40 minutes. Median initial National Institute of Health Stroke Scale (NIHSS) was 12 (mean : 14.4 +/- 5.5). Eight patients were further treated with mechanical thrombectomy due to persistent large vessel occlusion after IV tPA treatment. At discharge, there were 11 patients with good functional outcome (40.7%) and 16 patients with poor outcome. In-hospital mortality was 29.6% (8 patients). Symptomatic ICH was present in three patients (11.1 %), all of them were expired. The factors associated with poor functional outcome were high initial NIHSS score, diabetes mellitus and previous stroke history. Predictors of in-hospital mortality were diabetes and occlusion of basilar artery. Among 8 patients underwent adjuvant mechanical thrombectomy, two patients showed good functional outcome and 3 patients died at discharge. **Conclusion:** The overall mortality of very elderly patients treated with IV rtPA was not negligible. However, patients with good functional outcome reach to forty percent. So then, we suggest that, although patient is over 80 years old, IV tPA could be considerable after seriously comparing with potential benefit and risk of rtPA.

P-1-26

Predicting prognosis of mechanical thrombectomy by using the modified DRAGON score

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Background & Objectives: The DRAGON score, a composite of six clinical variables in acute ischemic stroke (AIS), was reported to predict the outcome of intravenous thrombolysis (IVT). The aim of this study is to test whether can be used to predict modified DRAGON (mDRAGON) score, in which the onset-to-treatment time was extended, with the outcome in AIS patients treated with mechanical thrombectomy (MT). **Method:** We assessed 40 AIS patients who were treated by MT alone or combined with IV tissue plasminogen activator (tPA). The mDRAGON score is: HyperDense sign on CT (both = 2, either = 1, none = 0), prestroke modified Rankin scale (mRS) > 1 (yes = 1), Age ($\geq 80 = 2$, 65-79 = 1, < 65 = 0), Glucose level ($> 144\text{mg/dL} = 1$), Onset-to-treatment time (> 230 minutes = 1) and baseline National Institutes of Health Stroke Scale (NIHSS) ($> 15 = 3$, 10-15 = 2, 5-9 = 1, 0-4 = 0). The 90d-mRS was used for the outcome measure. The predictive ability of the mDRAGON score was calculated using C-statistics **Results:** Among 40 AIS patients treated with MT, the proportions of good outcome (mRS 0-2) for the 2-3, 4-5, 6-7, 8-10 mDRAGON score group were 75%, 20%, 0%, 0%, while the those of poor outcome (mRS 3-6) for 2-3, 4-5, 6-7, 8-10 mDRAGON score group were 25%, 80%, 100%, 100%, respectively. AUC-ROC was 0.87 (0.80-0.87) for mDRAGON score. **Conclusion:** The mDRAGON score can be used to predict the clinical outcome of AIS patients following endovascular treatment.

P-1-27

Prehospital notification using emergency medical service is an optimal strategy to reduce the time to thrombolytic therapy after acute ischemic stroke

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Background & Objectives: The use of emergency medical services (EMS) is an

effective way for stroke patients to reduce time from symptom onset to hospital arrival. A prehospital notification can facilitate earlier intrahospital processes due to activation of a stroke team at the receiving hospital before patient arrival. The aim of this study is to investigate whether EMS use and prehospital notification can shorten the time to thrombolytic therapy after ischemic stroke, in a government-initiated comprehensive stroke center (CSC) of the Metropolitan area. **Method:** Since 2011, all paramedics of 119 EMS unit were annually trained using the Cincinnati Pre-hospital Stroke Screen for early detection and transportation of stroke patients. Stroke experts in our CSC received direct calls from paramedics pre-notifying the transportation of the patients with suspected stroke. We analyzed time intervals from symptom onset to various points, of 237 patients with intravenous thrombolytic therapy in our CSC from January 2012 to December 2014. **Results:** Of 237 patients, 173 patients (72.9%) were transported to the hospital via EMS and 64 patients (27.1%) were admitted with private modes. Among those with EMS use, only 15 patients (8.7%) were prenotified by EMS paramedics. The patients with EMS transport had shorter onset-to-arrival times (67 minutes versus 112 minutes, $P < 0.001$). In the patients admitted via EMS, EMS prenotification was associated with shorter door-to-imaging times, (8 minutes versus 11 minutes, $P = 0.02$) and door-to-needle times (19 minutes versus 28 minutes, $P = 0.003$). There was no difference of symptom onset-to-arrival times (85 minutes versus 96 minutes, $P = 0.259$) between the two groups. **Conclusion:** We confirmed that hospital prenotification using EMS was an optimal strategy to reduce the time to thrombolytic therapy after onset of ischemic stroke (onset-to-arrival and door-to-needle time) in our CSC. These data support the need for targeted initiatives to improve the rate of EMS prenotification.

P-1-28

Efficacy of proximal aspiration thrombectomy for using balloon-tipped guide catheter in acute intracranial internal carotid artery occlusion

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Background & Objectives: Mechanical thrombectomy (MT) for acute intracranial ICA occlusion is often complicated by difficult revascularization and non-involved territory embolization possibly related with larger clot-burden. This study aims to evaluate the efficacy of proximal aspiration thrombectomy (PAT) using a balloon-tipped guide catheter for clot-burden reduction in such cases with period-to-period analysis (period 1: standard MT without PAT; period 2: PAT first, then standard MT for the remaining occlusion). **Method:** Eighty-six patients who underwent MT for acute intracranial ICA occlusion were included in this analysis from the prospectively maintained stroke registry (33 patients in period 1 and 53 in period 2). In period 2, 'responder' was defined as a case where some amount of clot was retrieved by PAT and the following angiography showed partial or full recanalization. **Results:** Fifteen of fifty-three patients in period 2 (28.3%) were 'responders' to PAT. There was a significantly higher incidence of atrial fibrillation in the 'responder' subgroup. Period 2 showed a significantly shorter puncture-to-reperfusion time (94.5 minutes versus 56.0 minutes; $P = 0.002$), a significantly higher TICI of 2b-3 reperfusion (45.5% versus 73.6%; $P = 0.009$), but only a trend for better 3-month favorable outcome (mRS 0-2; 36.4% versus 54.7%; $P = 0.097$). There was no increase in the incidence of procedure-related complications or intracranial hemorrhage in period 2. **Conclusion:** A strategy of PAT before standard MT may result in shorter puncture-to-reperfusion time and better angiographic outcome than a strategy of standard MT for acute intracranial ICA occlusion.

P-1-29**Usage of rt-PA for the acute ischemic stroke patients with aortic dissection**

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Background & Significance: Aortic dissection is a rare, life-threatening emergency which has typical symptoms of radiating chest or back pain and sudden hypotension. Interestingly, 10-55% of the patients do not experience any relevant pain, but neurological symptoms attributed to comorbid neurological diseases such as brain ischemic stroke are reported instead in 18-30% of cases. Painless aortic dissection with neurological symptoms dominant is challenging for neurologists because it can mask the underlying condition, which results to delaying diagnosis and proper management. Also, there have been concerns regarding an increased risk of hemorrhage due to thrombolytic agent in the treatment of hyperacute stroke due to aortic dissection. Here we describe a case of a hyperacute stroke patient who was treated by intravenous rt-PA, and was diagnosed with painless type B aortic dissection in time with computed tomographic (CT) angiography. **Case:** A 73-year-old male was transferred to our emergency department. He complained left side weakness, which was abruptly occurred thirty minutes ago. He denied any other symptoms, including chest pain, nausea, vomiting or transient loss of consciousness. On the neurological examination, we found that he had central type facial palsy, left hemiparesis, and tactile and visual neglect, which resulted to the National Institute of Health Stroke Scale (NIHSS) of 7. Although his blood pressure (BP) was elevated at 168/92mmHg, other vital signs and physical examinations, laboratory evaluations, electrocardiography, and chest x-ray were unremarkable. His computed tomography (CT) of the brain showed neither acute hemorrhage nor low density including sulcal effacement, but hyperdense arterial sign in left middle cerebral artery (MCA) was noticeable. On detailed his past history, he previously had inserted pacemaker for sick sinus syndrome. Now he was only under aspirin medication. As there were no contraindications, intravenous tissue-plasminogen activator (rt-PA) was administered within 1 hour from the onset of stroke symptoms. Thirty minutes after the start of the infusion, his neurologic status improved (NIHSS 2). Under our hospital protocol, brain CT angiography was performed while his rt-PA continued. When we found that he had a Stanford type B aortic dissection from his brain CT angiography, rt-PA infusion was already finished. Because medical, rather than surgical, therapy is recommended for uncomplicated type B dissection, he was managed with medical therapy, including strict BP controlling. On the next day, we followed-up the brain image, which revealed a lobal intracranial hemorrhage in the right frontal area, while his NIHSS showed no interval change. However, his mental status and motor grade became worse from the evening of second day. On the third day, his mental status progressed worse, and we found newly developed anisocoria. On the follow-up brain CT, the size of previous ICH was increased with subtle midline shifting, and there was newly developed subdural hemorrhage (SDH) in the right fronto-parietal area. He underwent emergency decompressive craniectomy and hematoma removal. After surgery, his mentality as well as motor grade improved gradually, and he had hospitalized for three months with physical therapy, and was discharged with final modified Rankin Scale (mRS) 3. **Conclusions or Comments:** In our case, it was nearly impossible to diagnose the painless aortic dissection if further cerebral CT angiography was not taken. Even though we could detect it from the angiography after IV thrombolysis, aortic dissection did not result in rupture or hemorrhage. In several previously reported cases, type A aortic dissection was considered as a contraindication of intravenous thrombolysis. Our case showed that thrombolysis in acute ischemic stroke and type B aortic dissection was safe, suggesting it could depend on dissection type.

P-1-30**Stent edge occlusion with shortening of the previously implanted carotid stent in a patient with hyperacute ischemic stroke**

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Background & Significance: Carotid artery stenting (CAS) is an alternative treatment of the carotid endarterectomy to revascularize the extracranial stenosis of the internal carotid artery (ICA). Shortening the length and changing the location of the carotid stent in a patient who underwent CAS with closed-cell stent can evoke the distal edge stenosis of the stent. We report a case of hyperacute ischemic stroke due to the occlusion at the stent-edge of the Carotid WALLSTENT after successful CAS. **Case:** A 77-year-old woman was brought to the emergency room because of a sudden disturbance in speech and a weakness in the right upper and lower limbs (NIHSS score; 9). The patient was revascularized using a 10 X 40 mm Carotid WALLSTENT due to symptomatic severe stenosis of the left proximal ICA eight years ago. However, a follow-up examination by a neurologist was not performed after the carotid stenting because the patient only visited a cardiologist after the coronary stenting due to chronic stable angina pectoris. Intravenous thrombolysis with tissue plasminogen activator (tPA) after obtaining a CT examination was begun two hours after the symptoms onset. MRI and MR angiography which were performed during continuous infusion of the tPA revealed an acute infarction in the territory of the left middle cerebral artery (MCA) and an occlusion of the left proximal ICA. Emergent digital subtraction angiography showed that the distal edge occlusion of the previously implanted carotid stent demonstrated a shortening of the length of about 50% in the ICA when compared with the stent scan taken immediately after CAS. After aspiration with guiding catheter a visualization of the flow in the extracranial and intracranial artery beyond the severe stenotic ICA (NASCET criteria: 85%) at the distal edge of the previously implanted Carotid WALLSTENT. CAS using a 7 X 10 X 40 mm Protégé Rx Carotid Stent after ballooning was performed. Immediate post-stent angiography after post-stent ballooning showed successful stent implantation with a still present occlusion at the proximal portion of the inferior division of the left MCA. Neurologic examination on the day after carotid revascularization revealed improved neurologic deficit with dysarthria and a weakness of the right limbs (NIHSS score; 4). **Conclusions or Comments:** Shortening of Carotid WALLSTENT seems to have been caused by a marked mismatch of the diameter between the ICA and common carotid artery. Mechanical injury at the edge segment of the vessel with pulsation may contribute to the development of edge stenosis. Short term follow-up evaluation is important to detect unexpected complications such as stent shortening and stent-edge stenosis.

P-1-31**A case of carotid dissection in hyperacute ischemic stroke**Jae Guk KIM¹, Eun Kyung LEE¹, Seong-A LEE¹, In Kyu YU², Soo Joo LEE¹¹Department of Neurology, Eulji University Hospital, Eulji University, School of Medicine,²Department of Radiology, Eulji University Hospital, Eulji University, School of Medicine

Background & Significance: Most cervical carotid dissection can safely be conservatively management, but treatment in patient with hyperacute ischemic stroke has not been studied. We report a case of cervical carotid dissection in hyperacute stroke. **Case:** 40 year old male with a history of hypothyroidism presented with acute onset of left-sided weakness and dysarthria. He denied trauma, fall or injury to his head or neck. Neurological examination revealed left hemiplegia (MRC grade 1/5) with hypesthesia, and left central type facial palsy. Brain imaging with CT angiography and diffusion MRI showed right

MCA territory infarction with right proximal ICA occlusion. He had thrombolysis with IV tPA and underwent digital subtraction angiography (DSA) for considering mechanical thrombectomy. DSA demonstrated a tapered, flame-like narrowing and occlusion in the proximal portion of the right ICA, suggesting dissection. Although long and multisegment carotid dissection, we decided medical therapy with dual antiplatelet and high-dose statin without endovascular stent placement because the patency of ICA was kept in delayed internal carotid angiography. On 5th hospital day, follow up MRA revealed restoration of right ICA flow with segmental ectasia of ICA. He discharged on 10th hospital day with remained left hand clumsiness and central type facial palsy. **Conclusions or Comments:** This case suggests the early identification of luminal patency in carotid dissection may be important to select treatment option such as endovascular stent placement or conservative management with anticoagulation or antiplatelet therapy.

P-1-32

Post coil-embolization related ischemic stroke: how to prevent it?

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Background & Significance: For the management of intracranial aneurysms, endovascular coiling is a well known alternative procedure to surgical clip placement. There are, however, some post-procedure potential complications because of the risk of coil protrusion into the parent vessel. These complications are thromboembolic events, vasospasm, perforator artery occlusion, dissection, and ruptures of the parent artery or aneurismal sac, and etc. Among these complications, most frequent complications are thromboembolic events and secondary ischemic lesions. We describe a patient who had suffered ischemic stroke as a post-procedure complication of intracranial coil embolization. **Case:** A 65-year-old woman was admitted with a chief complain of transient left side weakness. She had history of well-controlled diabetes mellitus for ten years and smoked 0.5-pack a day for thirty years. She had two intracranial artery aneurysms which were found incidentally a month ago. She had managed by coil embolization on her intracranial artery aneurysms: right distal internal carotid artery (diameter 3mm, saccular type) and left posterior communicating artery (diameter 5.3mm, saccular type). Her procedure was successful without any post-procedure complications, and she was discharged two days after procedure without any anti-platelet agent continued. A day before the admission of our institution—a month from her coil embolization—sudden weakness of her left arm was developed for approximately two seconds. On her initial neurological examination, there were no abnormal findings. Transient cerebral ischemic attack (TIA) was suspected according to her transient symptom. On her brain diffusion-weighted image, however, acute infarction was found in right inferior frontal and precentral gyrus, and right basal ganglia (figure). MR angiography showed moderate stenosis in the supraclinoid segment of right distal ICA. We further evaluated with simple skull x-ray and CT angiography, and a long linear metallic foreign body protruded into the lumen ranging from right middle cerebral artery (MCA) M1 segment to M2 segment. She was managed with hydration and medical therapy: antiplatelet agents along with high dose statin. Because her symptom was not progressed, she was discharged on the seventh day of the admission, and she was transferred to the hospital where she had received the coiling procedure, in order to have repairing procedure. **Conclusions or Comments:** According to recent studies, the rates of thromboembolism in coiling of unruptured aneurysms and the rate of actual infarction is likely higher due to undetected silent infarction. Despite tight coil packing, migration of coil segment into the parent artery from the aneurysm sack still occurs. Wide-neck aneurysms with unstable and loose neck framing are especially more vulnerable to coil protrusion. Our case re-

veals that thromboembolic stroke associated with coil protrusion into parent artery could develop as a delayed complication of aneurysm embolization. Further investigation is warranted for the efficacy of using pre and post-procedure anticoagulation and/or antiplatelet therapy.

P-1-33

Multiple embolic infarctions due to a primary aortic intimal sarcoma

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Background & Significance: Intimal sarcoma of the aorta is extremely rare and aggressive tumor. The most common initial presentation is claudication or peripheral vascular disease, but embolic infarction to the brain is very rare manifestation of the tumor. Here we describe a patient with embolic stroke due to aortic intimal sarcoma. **Case:** A 41-year-old woman visited the emergency room for sudden-onset vertigo. She was previously healthy, current smoker, and habitual drinker. Brain MRI revealed multiple tiny infarctions in the right cerebellar hemisphere and left parietal cortex. MR angiography and carotid duplex sonography did not show any abnormalities. In the contrast-enhanced CT angiography, showed a mass-like lesion at the ascending aorta suspected of a mural thrombus. Initial serum D-dimer was 1.30 mg/L. One day later, she experienced severe abdominal pain. From follow-up CT, we found splenic infarction and left subclavian thrombus. She was immediately given anticoagulant therapy to prevent further attack. However, recurrent embolism developed despite anticoagulation. Transthoracic echocardiography showed a large hypermobile mass attached at ascending aorta. MRI of the heart showed elongated lobulating mass in the ascending thoracic aorta, attached to the left lateral wall of aorta, suggesting angiosarcoma. Because of the recurrent embolic event, she was transferred to the department of thoracic surgery for the replacement of aorta with graft. Histopathological examination was consistent with myxosarcoma that stained positive for vimentin. The patient started palliative chemotherapy with Doxorubicin followed by Pazopanib because of vertebral body metastases. **Conclusions or Comments:** Primary intimal sarcoma of the aortic arch and ischemic stroke is rare. Since our patient was a current heavy smoker and chronic heavy drinker, and the CT angiography finding was suggestive of a mural thrombus of aorta, index stroke might be explained by embolism due to aortic thrombus initially. However, the patient experienced multiple embolism throughout the whole body during anticoagulation and the isolated location of thrombus in the ascending aorta was very unusual without evidence of hypercoagulability. Those clinical findings strongly suggested embolic mechanism other than conventional etiology. Clinicians should be aware of intimal aortic sarcoma in patients with recurrent embolism and unusual location of thrombus in aortic arch.

P-1-34

Lateral medullary infarction with headache and autonomic dysfunction

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Background & Significance: Few patients with posteroinferior cerebellar artery (PICA) infarcts develop headache which can be associated with sympathetic dysfunction in the form of Horner's syndrome. Parasympathetic activity, such as lacrimation and eye injection has rarely been described in lateral medullary infarction. We report a case of lateral medullary infarction accompanied by headache and autonomic dysfunction. **Case:** A 43-year-old male presented with sudden onset vertigo, headache, and numbness in his left limbs. The pa-

patient complained of an orbital, retro-orbital and temporal continuous pain which was accompanied by ipsilateral conjunctival injection, lacrimation and nostril blockage. He had no past history of hypertension or diabetes. He was not a smoker but a social drinker. On neurological examination, cranial nerve exams revealed a right Horner's syndrome, right facial hyposthenia and hypoesthesia. Impaired sensation over the left limbs was also present. Muscular tone and strength were conserved; deep tendon reflexes were normal and symmetrical. Axial diffusion-weighted MRI revealed an acute right lateral infarction. **Conclusions or Comments:** Central nervous system lesions can rarely present with cluster-like or SUNCT-like symptomatology. The hypothalamus is a regulatory center for integration of sympathetic and parasympathetic systems via the hypothalamospinal tract which lies in the lateral medulla. Damage to the hypothalamospinal tract may lead to an imbalance between the two autonomic systems leading to an increase in parasympathetic activity.

P-1-35

Unilateral medial thalamic infarction without midbrain involvement presenting as vertical gaze palsy

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Background & Significance: Vertical gaze palsies are usually associated with lesions of the rostral interstitial medial longitudinal fasciculus, the interstitial nucleus of Cajal, the posterior commissure and peri-aqueductal gray matter. **Case:** A 73-year-old female presented with sudden onset dizziness. She had severely limited upward and downward gaze while horizontal gaze was intact. She had been diagnosed with hypertension one month prior to admission. She was not a drinker or a smoker. Neurological examination revealed bilateral vertical gaze palsy. Other cranial nerve exams were intact. Muscular tone and strength were conserved; deep tendon reflexes were normal and symmetrical. Axial diffusion-weighted MRI done showed a right medial thalamic infarction. **Conclusions or Comments:** Previous vertical gaze palsies with medial infarction have been attributed to coexisting lesions of the rostral midbrain. Vertical palsies as a manifestation of isolated medial thalamic infarction are rare. Interruption of supranuclear fibers as they traverse the medial thalamus en route to the pretectal and prerubral areas could possibly lead to vertical paresis. We report a case of isolated medial thalamic infarct without midbrain involvement presenting as vertical gaze palsy.

P-1-36

Clinical characteristics according to histologically-confirmed thrombus composition in hyperacute ischemic stroke

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Background & Objectives: With the advance of endovascular retrieval device for the cerebral thrombosis, it enables us to obtain a fresh thrombus of cerebral occlusion-site even in stroke survivors. Since thrombus components can reflect a fundamental stroke mechanism, it might be important to thoroughly investigate clinical, radiological, and histological findings of thrombi. Therefore, we investigated the histological composition of retrieved thrombus and compared clinical and radiological characteristics according to fibrin predominance (at least 60%) of the thrombus. **Method:** We prospectively enrolled acute ischemic stroke patients who underwent computed tomography (CT)

and magnetic resonance image (MRI) prior to endovascular thrombectomy at a stroke referral center from July 2014 to June 2015. Hyperdense middle cerebral artery sign (HMCAS) on non-contrast CT and susceptibility vessel sign (SVS) on multi-planar gradient-echo (MPGR) MR images were evaluated. National Institutes of Health Stroke Scale (NIHSS) was investigated as an index of clinical severity. The proportion of red blood cells (RBC) and fibrin of the thrombus was qualitatively and quantitatively analyzed by a commercial system, Aperio Scanscope XT digital camera (Aperio, Vista, CA). The color of gross thrombi specimen was graded into pink, scarlet, red, and dark-red. **Results:** The mean age was 63.0 ± 11.3 years and 50.0% female. All (n=16) had a successful recanalization and intravenous tissue Plasminogen Activator (t-PA) was used in 11 patients. According to the component of thrombus, 4 had RBC dominant (25.0%), 8 fibrin dominant (50.0%), and 4 mixed thrombi (25.0%). We dichotomized them into two groups: fibrin dominant (n=8) vs. RBC dominant or mixed groups (n=8). As compared with fibrin dominant group, RBC dominant or mixed group had significantly higher prevalence of HMCAS (100.0% vs. 50.0%, p=0.021) and SVS (100.0% vs. 37.3%, p=0.007). Grossly, fibrin-dominant group was shown as pink to scarlet (62.5%), whereas RBC-dominant or mixed group was shown as red to dark-red (100%). RBC-dominant or mixed group had more severity with a higher baseline NIHSS score (18.5 vs. 12.0, p=0.001). **Conclusion:** Our results suggest that the histological composition of the thrombus might lead to the clinical differences of early vessel signs, color of gross specimen, and initial severity.

P-1-37

Endovascular thrombectomy in acute ischemic stroke patients with early neurologic deterioration

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Background & Objectives: Even though recanalization is strongly associated with improved functional outcomes and reduced mortality, clinical benefit from thrombolysis is reduced as stroke onset to treatment time increases. In recent study, Endovascular thrombectomy (ET) has been demonstrated to improve functional outcome in patients with acute ischemic stroke (AIS) within time window of onset to 6 or 8 hours. However, beyond usual thrombolysis time window, early neurologic deterioration (END) related with proximal artery occlusion is not uncommon in AIS. Hereby, we report AIS case series treated with ET over usual thrombolytic time window because of END related proximal artery occlusion. **Method:** From January 2012 through October 2014, all 173 patients underwent ET for AIS with anterior circulation stroke. Among them, forty one patients underwent ET beyond 8 hours after stroke onset. In eighteen of 41, ET was applied due to END. At admission, all eighteen patients showed near to complete occlusion of proximal artery and had diffusion-perfusion mismatch. **Results:** Four patients were applied ET beyond 8 hours to last seen normal time. Fourteen patients were applied ET beyond 24 hours to last seen normal time. Mean age was 65. Initial mean initial National Institutes of Health Stroke Scale (NIHSS) was 4.7 and NIHSS after END was 10.6. All patients had diffusion-perfusion mismatch over 200%. Seven patients treated with IV-tPA before ET. Good recanalization (TICI 2b/3) was achieved in 94.4%. Hemorrhagic complication was seen in the follow-up computed tomography scan in 3 of 18 cases: two were hemorrhagic transformation, another was subarachnoid hemorrhage. Thromboembolic complication was occurred 1. Ten patients had modified Rankin Scale ≤ 2 after 3 months. There was no mortality case. **Conclusion:** In our report, ET beyond usual 8 hours time window achieved safe and successful recanalization. And

successful recanalization was associated with good clinical outcome. We think ET could be another treatment option in case of END in AIS patients with proximal artery near to complete occlusion, even beyond usual 8 hours time window of thrombolytic therapy.

P-1-38

Uric acid consumption in the patients after intraarterial thrombolysis

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Background & Objectives: Precedent experimental studies implicit a neuroprotective role of uric acid, however the controversial in clinical studies lasts whether serum uric acid correlates with the better clinical outcome indicating a role of neuroprotection or the worse outcome indicating a role of pro-apoptotic effect. If serum uric acid level takes a role of neuroprotection or pro-apoptosis, uric acid will be consumed during the acute stroke for its role, and correlated with ischemic-reperfusion burden of the acute stroke. We defined the decrements of uric acid level in acute phase of acute ischemic stroke without matter of neuroprotection or pro-apoptosis, as "Uric acid consumption". Ischemia-reperfusion injury burden in acute stroke will be correlated with the time duration from the time point of arterial occlusion to the time point of recanalization. We could know the time point of recanalization in patients who undergone intraarterial thrombolysis exactly, and therefore, could measure the ischemic time duration. Therefore, we analyzed the duration of ischemia with uric acid consumption, and furthermore, how the neurologic outcome is correlated with the consumption of uric acid. **Method:** With 86 patients undergone recanalization and with serum uric acid follow up results (within 1week), we analyzed the relationship between the amount of uric acid consumption and neurologic outcome index e.g. discharge mRS, difference of discharge NIHSS from admission NIHSS, and the difference of discharge mRS from admission mRS. Furthermore, the relationship between the duration of ischemia and uric acid consumption was analyzed as well. The logistic regression using R 3.2.1 version was done. **Results:** Among acute ischemic patients who undergone intraarterial thrombolysis, the patients with the better discharge mRS group ($p=0.002$), the group of higher difference of discharge mRS from admission mRS ($p<0.0001$), and the group of higher difference of discharge NIHSS from admission NIHSS ($p=0.0137$) in quantiles accounts for the log value of uric acid consumption as suggestion of the association, and implicit that the higher uric acid consumption indicates the better neurologic outcome. Furthermore, The duration of ischemia has a linear association with the log value of uric acid consumption in the group of patients with better outcome after intraarterial thrombolysis (the difference of mRS from discharge and admission ≥ 2) in regression model ($p=0.02$), and not meaningful association with the group of patients of poorer clinical improvement. (the difference of mRS from discharge and admission < 2). According to the result, uric acid consumption is correlated with the duration of ischemia and therefore ischemic burden as well in better clinical improvement group. In additional research done with the patients failed to recanalized (TICI=0), uric acid consumption was higher than the other of recanalized. We can assume that if uric acid is fully consumed by brain tissue of ischemia during ischemic time waiting for perfusion, but ischemic brain tissue of failed recanalization or late recanalization would use up all uric acid available, therefore associates with poorer neurologic improvement. Therefore, the lower association of ischemic duration and uric acid consumption in the patient group of poorer clinical improvement can be interpreted as the brain tissue has used up available portion of uric acid. **Conclusion:** In conclusion, the result of this study indicates that the higher uric acid consumption is associated with the better neurologic outcome and improvement. Furthermore, the result of study implicit that uric acid consumption is associated with the duration of brain is-

chemia, and therefore uric acid consumption is correlated with the ischemic burden of acute ischemic stroke..

P-1-39

Futile Recanalization and predicted Therapeutic gain by Initial Stroke Severity after Endovascular treatment

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Background & Objectives: The rate of futile recanalization, defined by 3-month modified Rankin scale (mRS) 3 to 6 despite complete recanalization (Thrombolysis in Cerebral Infarction grade 2b-3), ranged from 29 to 67% according to recent endovascular treatment (EVT) trials. The earlier studies showed that initial stroke severity, measured by National Institutes of Health stroke scale (NIHSS) score, was a strong predictor of futile recanalization. The higher is the NIHSS, the more frequent is the futile recanalization. However, the two EVT trials (SWIFT-PRIME and REVASCAT) data showed that the higher is the NIHSS (>17), the greater is the benefit of EVT. This study aimed to investigate the patterns of futile recanalization and therapeutic gain by EVT across the whole range of initial NIHSS. **Method:** Using a prospective multicenter stroke registry (Clinical Research Center for Stroke-5) database, we identified acute ischemic stroke patients whose pre-stroke mRS was 0 to 1 and who were confirmed anterior circulation large artery (middle cerebral artery and internal carotid artery) occlusion and were treated with EVT within 12h of onset between November 2009 and July 2014. Information on clinical characteristics, acute management and outcomes was obtained directly from the registry database or by review of medical records. We calculated the rates of futile recanalization across the whole range of NIHSS score, categorized as ≤ 5 , 6-10, 11-20 and >20 . To estimate therapeutic gain, which was defined as a difference in the proportions of mRS 3-6 between those recanalized completely with EVT and those not treated, the proportions of mRS 3-6 in patients not treated but hospitalized within 12 h of onset were obtained with age-specific direct standardization. **Results:** Among 21,591 patients with acute ischemic stroke, 4.5% ($n=972$) received EVT within 12 h of onset. Of those 972 EVT-treated patients, 54.8% ($n=533$) were completely recanalized. Among those 533 patients, 440 with anterior circulation larger artery occlusion were enrolled for this study (male 58%, age 67.3 ± 12.3 years, onset to EVT time 4.19 ± 1.96 hours). Seventy percent of patients were treated with intravenous alteplase prior to EVT. The rate of futile recanalization was 51.4% ($n=226$). Compared with patients without futile recanalization, those with futile recanalization were more likely to be female and hypertensive, and have higher creatinine level and NIHSS score; and less likely to be current smoker ($p < 0.05$). Those with futile recanalization tended to be older than those without futile recanalization ($p=0.08$). The onset to EVT starting time was not different between two groups. The distribution of initial stroke severity as follows: NIHSS

≤ 5 was 9.8% (n=86); NIHSS 6~10 18.4% (n=162), NIHSS 11~20, 58.6% (n=516); and NIHSS >20 13.2% (n=116). The rates of futile recanalization increased with the increase of stroke severity; 20.9% in NIHSS ≤ 5; 34.6% in NIHSS 6~10; 58.9% in NIHSS 11~20; 63.8% in NIHSS >20 (p for trends <0.001). The therapeutic gain of EVT significantly differed by initial stroke severity (p for interaction=0.009); -1.7% in NIHSS ≤ 5; 20.2% in NIHSS 6~10; 25.9% in NIHSS 11~20; 32.4% in NIHSS >20. **Conclusion:** This study emphasized the impact of initial stroke severity on futile recanalization and therapeutic gain in patients receiving EVT for acute ischemic stroke caused by anterior circulation large artery occlusion. These results could be helpful to make the therapeutic decision to clinicians at emergent practice.

P-1-40

The incidence and mechanism of neurological deterioration after endovascular thrombectomy

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Background & Objectives: Recent clinical trials have shown that endovascular thrombectomy is an effective and safe recanalization modality for acute ischemic stroke patients. We investigated the incidence and pathomechanism of neurological deterioration after endovascular thrombectomy. **Method:** Between January 1st 2011 and July 31st 2014, the acute ischemic stroke patients who had been treated by endovascular thrombectomy in Chung-Ang University Hospital were included. We reviewed clinical record and laboratory data, and neurological deterioration is defined as 2 or more National Institute of Health Stroke Scale increase compared to the best neurological status within 7 days after endovascular treatment. The mechanism of neurological deterioration was categorized into ischemia progression, hemorrhagic transformation and brain edema. **Results:** Total of 49 acute ischemic stroke patients received endovascular treatment, including 10 patients with basilar artery occlusion. Neurological deterioration was detected in 20 patients (40.8%) and 17 cases occurred within 72 hours after endovascular treatment. The mechanism of neurological deterioration included 8 ischemia progression, 9 brain edema, and 3 hemorrhagic transformation cases. Brain edema and hemorrhage were concentrated within 72 hours after treatment, whereas most neurological progression after 72 hours was due to ischemia progression. The patients with neurological deterioration was more frequently associated with poor functional outcome of modified Rankin scale 5 or 6 at discharge, than neurologically stable patients (10/20 vs. 6/29, p=0.032 by Chi square test). **Conclusion:** Neurological deterioration frequently occurs after endovascular thrombectomy, which was concentrated with 72 hours after treatment.

P-1-41

Initial factors affecting the clinical outcome after successful recanalization via MR based mechanical thrombectomy in patients with acute ischemic stroke due to basilar artery occlusion

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Background & Objectives: To determine initial factors, including patient characteristics, stroke etiology and severity, time factors, and imaging findings, that could affect the clinical outcome of patients with acute ischemic stroke (AIS)

caused by basilar artery occlusion (BAO) where successful recanalization was achieved via mechanical thrombectomy. **Method:** Between March 2011 and December 2014, 35 patients with AIS caused by BAO received MRI/MRA-based mechanical thrombectomies, and recanalization was achieved with a TICI score of more than 2b. We divided the patients into a 'good-outcome' group (n = 19), defined as a mRS score of 0-2 at 3 months after stroke onset, and a 'poor-outcome' group (n = 16), defined as a score of 3-6. We analyzed differences between the groups. **Results:** Initial NIHSS score (good vs. poor: 17.9 ± 8.9 vs. 27.6 ± 8.5, p = 0.003), pc ASPECTS based on initial DWI (good vs. poor: 7.8 ± 1.6 vs. 5.4 ± 1.8, p = 0.001), pc ASPECTS based on contrast staining on the post-thrombectomy control CT (good vs. poor: 9.2 ± 1.5 vs. 6.3 ± 2.2, p < 0.001), and presence of contrast staining in the brainstem on that CT (good vs. poor: 15.8% vs. 81.6, p < 0.001) were significantly different between the groups. **Conclusion:** Patients with AIS caused by BAO with a lower initial NIHSS score, fewer lesions on initial DWI, and less contrast staining on the post-thrombectomy control CT have higher probabilities of a good clinical outcome after successful recanalization via a mechanical thrombectomy.

P-1-42

Acute ischemic patient with the hyperintense acute reperfusion marker (HARM) after spontaneous recanalization

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Background & Significance: The hyper-intense acute reperfusion marker (HARM) on T2 fluid attenuation inversion recovery (FLAIR) image is considered in consequence of blood brain barrier (BBB) disruption. In generally HARM has been observed after recanalization of large intra or extra cranial artery such as carotid artery or M1 portion of middle cerebral artery. We report a case of acute ischemic stroke patient with HARM after infusion of intravenous recombinant tissue plasminogen activator (r-TPA), who did not have definite steno-occlusive lesion on the large arteries. **Case:** A 69-year-old man who took a sauna during 3 hours just before symptom onset visited our emergency room complaining of difficulty of speech and right upper extremity weakness. The initial National Institutes of Health stroke scale (NIHSS) scores at admission were 5 and his difficulty of speech and weakness were improving since symptom onset. Brain magnetic resonance image (MRI) and magnetic resonance angiography (MRA) showed multiple acute infarctions on the left hemisphere with focal narrowing of left proximal internal carotid artery (Figure). The patient was administered intravenous (IV) r-TPA. 3 hours after IV r-TPA infusion, his motor weakness, motor aphasia, disorientation and mental status had progressed and NIHSS scores were 18. Brain computed tomography (CT) revealed no significant abnormalities including mass effect or intracerebral hemorrhage within 5 hours after symptom onset. But brain MRI which were performed 8 hours after symptom onset showed newly appeared high signal intensities on left cortical area on diffuse weighted image (DWI) and hyper-intense signals within the whole sulci and cisterns of the left hemisphere on T1, T2, T2 FLAIR and susceptibility weighted imaging (SWI). We diagnosed him with HARM and initiated supportive management including increased intra cranial pressure (IICP) treatment and strict blood-pressure control. Four days after admission, the patient's mental status improved and motor aphasia had recovered. He walked home at discharge without assistance. **Conclusions or Comments:** In generally HARM occurred after recanalization of large artery. Interestingly our patient did not show initial steno-occlusive lesion on the large arteries, but the HARM was occurred hyper-acute course after IV r-TPA infusion. Our case suggests that r-TPA infusion after spontaneously recanalization of intracranial arteries could accelerate BBB disruption and induced

HARM with clinical worsening.

P-1-43

Retrieved thrombus in two patients with embolic stroke underlying atrial fibrillation with a proper INR level: platelet- dominant white clot?

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Background & Significance: background& Significance Acute occlusion of the cerebral arteries can be explained by various thrombus from different origins, such as mural cardiac thrombi, cardiac mass, and atherosclerotic plaque. This thrombus can be pathologically categorized as “red” or “white” clot according to its cellular components. With the advance of endovascular thrombo-retrieval devices, some studies have been reported on the characteristics of retrieved-thrombus’ components. A post-mortem study reported that “red thrombi” was closely associated with cardioembolic stroke. To our best of knowledge, there has been no report on the pathological characteristics of clot in cardio-embolic stroke patients under the well-controlled oral anticoagulation therapy. We report two patients with embolic stroke underlying atrial fibrillation with a proper INR level, presenting a “white clot” from mechanical thrombectomy. **Case:** Case 1 A 69-year-old male had a right middle cerebral artery (MCA) territory infarct. Due to his old stroke history of left thalamic infarct with paroxysmal atrial fibrillation, he has taken oral anticoagulation as warfarin. He presented with the left hemiplegia with the characteristic cortical manifestations including 12 points of the NIH stroke scale. He arrived hospital after event 1 hour later. The patient’s brain CT showed right distal ICA occlusion and diffusion image of MRI showed right MCA territory infarct. Gradient echo image of MRI didn’t show blooming artifact on occluded artery, and CT scan also showed no dense MCA sign on right distal ICA. The patient’s INR was 1.93, which was contraindication for IV t-PA, so we tried intra-arterial mechanical thrombectomy and the clot was successfully retrieved. The clot was fixed by formalin, embedded by paraffin. Finally we made a histologic section of clot and took a microscopic picture. Case 1 A 40-year-old woman had been diagnosed atrial fibrillation with mitral valve disease 20 years ago and she had been performed mitral valve replacement with prosthetic valve. She has taken oral anticoagulant therapy as warfarin due to previous embolic stroke. She presented left hemiplegia, left homonymous hemianopsia, left facial palsy and extinction, total NIHSS was 13 points. She arrived hospital 73 minutes after event and her brain CT showed right distal M1 occlusion. Her INR was 2.62, and we decided to try intra-arterial thrombectomy procedure without IV t-PA injection. The clot was retrieved by solitare stent and her right MCA was successfully recanalized. The patient’s initial brain CT showed right MCA occlusion and MRI showed MCA territorial infarct on diffusion image. There was no HMCAS on CT and no BA on gradient echo image of MRI. We performed same procedure for clot and we took clot section and microscopic image. **Conclusions or Comments:** Both two cases showed fibrin dominant white clots, contrast to traditional concept that clots from embolic stroke was red thrombi. Red thrombi is generated by activation of coagulation pathway. Warfarin inhibits factor 2,7,9 and 10, so those anticoagulation effect inhibits the generation of red thrombi. In this situation, some triggers such as underlying endothelial damage or valvular injury could generate white thrombi instead of red thrombi.

P-1-44

A case of cerebral venous thrombosis related to iron deficiency anemia and increased factor VIII activity

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Background & Significance: Cerebral venous thrombosis (CVT) is caused by various conditions such as hematological disorder, pregnancy, oral contraceptives use, cancer, dehydration and other uncommon causes. The association between cerebral venous thrombosis (CVT) and iron deficiency anemia has been well understood for a long time. Also elevated factor VIII activity is thought to be an independent risk factor for venous thrombosis. We present an adult case of CVT related to iron deficiency anemia and increased factor VIII activity. **Case:** A 46-year-old woman presented with acute right hemiparesis. She denied a history of oral contraceptive use, or regular ingestion of alcohol or drugs. Brain diffusion-weighted MRI showed cytotoxic edema combined with vasogenic edema in the left frontal area and the right parieto-occipital area which was not compatible with cerebral arterial territories. Gradient-echo MRI revealed a low signal intensity in superior sagittal sinus and right transverse sinus suggesting venous thrombosis. Cerebral MR venography demonstrated a defect in superior sagittal, right transverse, right sigmoid sinus and also right jugular vein. She had complained hypermenorrhea for several years and the laboratory data revealed iron deficiency anemia (Hb 8.1 g/dL) with slight thrombocytosis (platelet count 442,000/ μ L, normal range : 150,000~400,000). The cause of hypermenorrhea was uterine myoma which needed to be surgically removed. All results of coagulation tests and vasculitis tests were negative except factor VIII activity. Serum level of factor VIII was 225% (normal range : 60 ~ 150). Following a lumbar puncture, a septic condition was ruled out. The patient was treated by blood transfusion and anticoagulation with intravenous heparin, followed by warfarin. The patient’s right hemiparesis gradually improved to normal strength. Four months later, she had an elective total hysterectomy to prevent anemia. **Conclusions or Comments:** Despite extensive investigations, the origin remains unknown in up to 35% of CVT cases. Iron deficiency anemia is relatively common incidental finding in young to middle aged women. In a recent prospective study of 121 adult patients with iron deficiency anemia and cerebral venous thrombosis, the authors suggest a significant association of severe anemia (Hb < 9 g/dL) and CVT. The proposed mechanism is that the low iron levels disinhibit megakaryocyte activity, which provokes secondary thrombocytosis, thus leading to a hypercoagulable state. Therefore it is important to consider the anemia as a possible cause of CVT and correct the curable condition such as gastrointestinal bleeding, internal hemorrhoid or uterine myoma. Besides the anemia, our patient showed the increased factor VIII activity. Levels above 150% have been shown to be associated with a fivefold increased risk of venous thrombosis. In one study, patients with a factor VIII level above the 90th percentile showed a 37% likelihood of recurrence within 2 years. Therefore, it seems to be reasonable to use warfarin as long-term prophylaxis in patients with elevated factor VIII activity.

P-1-45

Possible stress cardiomyopathy after carotid artery stenting

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Background & Significance: Stress cardiomyopathy, also called Takotsubo cardiomyopathy, is characterized by regional left ventricular dysfunction that cannot be explained by an occlusive lesion in a coronary artery. Catecholamines are implicated in the pathogenesis of stress cardiomyopathy but the mechanisms involved are unknown. This case provides the first description of possible stress cardiomyopathy after carotid artery stenting. **Case:** A 76-year-old man was admitted for evaluation of acute left upper extremity weakness. He had

been treated for hypertension and chronic kidney disease for 10 years. Magnetic resonance imaging(MRI) revealed right middle cerebral artery territory acute ischemic infarction. MR angiography revealed high grade stenosis of the right proximal internal carotid artery(ICA) with the left proximal ICA occlusion. Echocardiography performed on admission showed no abnormality. The patient was pretreated with aspirin and clopidogrel for 5 days before undergoing stenting at the right proximal ICA via a transfemoral approach under local anesthesia. Post-stenting cerebral angiography showed the widened right ICA stenosis. After the stent insertion, there was no evidence of aggravation of the patient's neurologic status as compared with his neurologic status at the time of admission. However, there was systolic blood pressure lowering (70mmHg) without bradycardia during the periprocedural period. The patient was treated with a fluid bolus of normal saline and intravenous administration of dopamine. However, the patient required increasing doses of dopamine to maintain his blood pressure. One day after carotid artery stenting(CAS), he complained about dyspnea and chest tightness. Laboratory measures showed an elevated troponin(0.372 ng/ml) and creatinine (2.29 mg/dl). Electrocardiogram(ECG) showed no evidence of myocardial infarction. Chest x-ray demonstrated pulmonary congestion. The patient required additional administration of norepinephrine because of hemodynamic instability. However, despite of adequate inotropic therapy, there was aggravation of the patient's symptoms. Taking into account his clinical presentation and laboratory characteristics, diagnosis of possible stress cardiomyopathy was done. As there was no response to medical therapy and volume resuscitation, we used of an intra aortic balloon pump(IABP). Despite of IABP, hypotension was not corrected adequately. Resuscitation efforts were unsuccessful and he expired one day later. **Conclusions or Comments:** Hemodynamic instability is common after CAS, especially postprocedural hypotension. However, stress cardiomyopathy is a rare complication associated with hypotension after CAS. Therefore, recognition and understanding of stress cardiomyopathy by clinicians is essential for proper patient treatment. Furthermore, applying the appropriate prophylactic measures and strictly monitoring blood pressure during the periprocedure period should be encouraged for early recognition and correction of these hemodynamic disturbances.

P-1-46

Cerebral venous infarction in mount everest

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Background & Significance: Cerebral venous infarction (CVI) caused by a hypercoagulable state is relatively rare compared to arterial ischemic stroke; it accounts for only approximately 1% of all strokes. Known risk factors for CVI include the use of oral contraceptives, pregnancy, the period of puerperium, and other prothrombotic conditions such as dehydration, thyrotoxicosis, infection, trauma, neoplasm, vasculitis, and polycythemia, etc. CVI caused by a high altitude state has very rarely been documented. We report an interesting case of CVI as a consequence of a hypercoagulable state, secondary to adapting to a high altitude **Case:** A 46-year-old, healthy man with no remarkable medical history, planned to climb a highest mountain in the Everest. During his previous climbing to Island Peak, he experienced mild acute mountain syndrome (AMS) including headache, nausea, and facial swelling, which was completely recovered without any neurological sequela. However, his final challenge to the highest mountain in the Everest fell in trouble. He arrived in Lukla (2800 m) in Nepal and climbed 300 to 400 m per day. When he arrived at Namche Bazar (3440 m), he experienced headache without other neurological symptoms. He continued to climb and arrived at Base Camp (5365 m) in 10 days after leaving Lukla. After rest for 2 days at the base camp, he reached Camp I (5970 m) and returned to Base Camp to accommodate to very high altitude. Next day he was

scheduled to climb up to Camp II (6500 m), however, when washing his hair in the morning, he experienced weakness in his right arm with an accompanying headache. He was carried down the mountain and admitted to a hospital in Kathmandu, Nepal. Brain MR performed in this hospital showed that localized edematous lesions in the left frontal cortex. The following day, he returned to Korea and was admitted to our hospital. Follow-up MR showed edematous lesion with petechial hemorrhages in the left pre- and post-central gyri with predominant vasogenic edema indicating venous infarction. No thrombosis was observed in the venous sinus or cortical veins. Because his hemoglobin titer was high as 18.2 g/dl, genetic test including BCR/ABL, JAK-2 mutation was performed to rule out polycythemia vera and the results were negative. Further diagnostic work-up for the hypercoagulability including tests for protein C, protein S, anti-thrombin 3, lupus anticoagulant, anticardiolipin antibodies, factor V Leiden, and anti-b2 GPI Ab were unremarkable. Five days later, he was discharged with subtle weakness in his right hand grip. **Conclusions or Comments:** Though cerebral venous infarction is uncommon and has rarely been reported in the setting of high altitude, however, we should be careful to approach AMS due to mimicking of venous infarction known as the non-specific symptoms of AMS; altered consciousness, severe lassitude. In our patient, known risk factors for cerebral infarction were excluded by stroke work up. At the time cerebral infarction occurs, initial hemoglobin and hematocrit level are not verified because of closing of that institution in Nepal. However, after transferred to our hospital, increased hemoglobin (18.2 g/dL) be measured, after discharge, hemoglobin levels in ambulatory care have been identified to decline to 17.1g/dL. The conjecturable etiology of stroke in this case was a combination of dehydration due to restriction of fluid of fluid intake and vomiting induced by AMS, high altitude hypoxia, hematologic factor change on exposure to high altitude (eg. Secondary polycythemia, thrombocytopenia), endothelial dysfunction, vulnerability of existing genetic factor. The increasing popularity of trekking in alpine regions has drawn attention to high altitude-associated health concerns. In the highlands, it is likely to be hypercoagulable state causing venous thromboembolism. It is difficult to distinguish cerebral venous infarction having focal neurologic deficit from AMS, when we have to be doubtful examining a patient.

P-1-47

A case report of fibromuscular dysplasia in the posterior cerebral artery

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Background & Significance: Fibromuscular dysplasia (FMD) is a non-atherosclerotic, non-inflammatory arterial disease that affects cerebral vasculature. The carotid and vertebral arteries are most commonly involved and intracranial arteries are seldom affected. We report a patient with FMD involving the posterior cerebral artery (PCA). **Case:** A 24-year-old woman visited to the neurology clinic with a 4-day history of right side visual field defect with left side throbbing headache. Her past medical and family history was of no significance. She did not have any medication including oral contraceptives. In physical examination she showed homonymous right side hemianopsia. The rest of the physical examination was unremarkable. A diffuse-weighted image (DWI) of the brain revealed high signal intensity in the left PCA territory. Magnetic resonance angiography (MRA) revealed stenotic abnormality in the PCA. Cerebral angiogram revealed a 'string of beads' appearance, pathognomonic for FMD. The laboratory work up including CBC, electrolytes, calcium, PT, PTT, INR, ESR, homocystein, anticardiolipin, RPR, rheumatoid factor and antinuclear antibody was normal. The transcranial Doppler and the carotid duplex were normal. Transthoracic echocardiography showed no remarkable finding. The hospital course was stable, and the patient was dis-

charged with aspirin. Headache was regressed but, right visual field defect persisted. **Conclusions or Comments:** FMD of the ICA is commonly extracranial. Most often, intracranial FMD corresponds to an intracranial extension of extracranial FMD. Isolated FMD is reported a few case and FMD in PCA is reported only one case. This is second report of the FMD involved in the PCA. The finding of MRA may be non-specific luminal irregularity that that is not corresponded with 'string beads appearance' in the patient of intracranial FMD. Intracranial FMD must be considered as possible etiology of young age stroke, though the finding of MRA is non-specific.

P-1-48

Prevention Strategies for Stroke in Korean Adults: the prevention of stroke and dementia survey 2007-2009

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Background & Objectives: The proportion of ischemic stroke among all strokes has been steadily increasing. Therefore, recent stroke prevention strategies have targeted the risk factors for ischemic stroke. Community-based data that quantify the prevalence of these risk factors are needed to develop effective stroke prevention strategies. **Method:** The Prevention of Stroke and Dementia (PRESENT) survey collected data associated with stroke risk factors between 2007 and 2009 (358 men, 422 women, aged 50 years and older). **Results:** Men had more risk factors for stroke, and higher rates of diabetes mellitus and current smoking. However, women had higher cholesterol and obesity rates and lower physical activity. The proportion of individuals with >1 risk factor for stroke increased with age. Hypertension, diabetes mellitus, and cardiovascular diseases also increased with age, but low-density lipoprotein levels and current smoking decreased. **Conclusion:** Together these findings suggest a need for customized strategies for primary and secondary stroke prevention that consider the prevalence of individual risk factors and population attributable risk

P-1-49

Carotid artery calcification predicts carotid bulb atherostenosis

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Background & Objectives: Previous study noted that severe atherosclerosis in the internal carotid artery (ICA) in the neck was associated with calcification of the artery, more distally in the head. The purpose of this study is to evaluate the correlation between calcification of intracranial ICA and carotid bulb stenosis. **Method:** We analyzed consecutive patients who had checked computerized tomography angiography (CTA) scans of head and neck in Boston university medical center (BMC) since January, 2006 to June 2009. We reviewed for two characteristics: the amount of calcification around ICA and presence and severity of atherosclerosis in the carotid bulb. Total amount of calcification was calculated from area of calcification obtained from every slices of CTA source image with 5mm thickness from petrous portion to supraclinoid portion of ICA. We divided patients by 5 groups according to estimated total volume of ICA calcification (absence, minimal $\leq 10\text{mm}^3$, mild 11-40mm³, moderate 41-80 mm³, severe $\geq 81\text{mm}^3$). **Results:** A total of 201 patients were included and 402 arteries were examined in the study. Mean age was 64.13(40-88, SD;12.93). The amount of calcification of intracranial ICA was significantly correlated with the degree of carotid bulb stenosis. ($P < 0.0001$, Pearson's correlation coefficient; 0.378). The probability of presence of significant (mo-

derate and severe group) carotid calcification was more than 90% in case of 60% \geq sum of bilateral carotid bulb stenosis. **Conclusion:** Calcification of ICA had significant association with atherosclerosis in the ICA in the carotid bulb.

P-1-50

The role of signal intensity ratio on FLAIR MRI in acute ischemic stroke patients with endovascular treatment

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Background & Objectives: Fluid-attenuated inversion recovery (FLAIR) imaging is accepted as a tissue clock in acute ischemic stroke. This study aimed to investigate whether FLAIR hyperintensity can be a surrogate marker for the severity of ischemic insult, therefore, can predict the lesion growth. **Method:** Based on a prospective stroke registry database, we identified ischemic stroke patients who received endovascular treatment (EVT) within 8 hours of onset, achieved successful recanalization (modified Thrombolysis In Cerebral Infarction (mTICI) $\geq 2\text{B}$), and underwent magnetic resonance imaging (MRI) before EVT. Information on demographics, clinical and laboratory findings, stroke characteristics, modified Rankin scale (mRS) score at 3 months, and symptomatic hemorrhagic transformation (sHT) was directly obtained from the registry database. FLAIR hyperintensity was measured using the signal intensity ratio (SIR), defined as the ratio of mean signal intensity of the diffusion-restricted lesion to the signal intensity of the contralateral hemisphere on FLAIR. Lesion growth was calculated as the ratio of final infarct volume on follow-up FLAIR to initial infarct volume on diffusion-weighted imaging (DWI) at presentation. **Results:** Of the 69 patients, who met the eligibility criteria, mean FLAIR SIR was 1.17 (SD, 0.11) and mean lesion growth, 3.25 (SD, 5.42). FLAIR SIR was not correlated significantly with the lesion growth (Pearson coefficient = -0.146, $p = 0.23$). In multiple linear regression models including initial infarction volume, various time parameters, and other clinical covariates, FLAIR SIR was not significantly correlated with the lesion growth. Interestingly, time interval from initial MRI to successful recanalization was independently correlated with the lesion growth. When FLAIR hyperintensity was dichotomized at its known cutoff (SIR 1.15), it was associated with neither favorable outcome (modified Rankin scale 0-2) (odds ratio, 0.27; 95% confidence interval, 0.06 to 1.21; $p = 0.09$), nor symptomatic hemorrhagic transformation (4.02; 0.39 to 41.04; $p = 0.24$) significantly with adjustments for age, initial stroke severity and time interval from initial MRI to recanalization. **Conclusion:** Contrary to our hypothesis, FLAIR SIR on initial MRI before EVT was not associated with the lesion growth in patients who were recanalized successfully with EVT. FLAIR SIR might not be used as a surrogate marker for tissue injury in acute ischemic stroke. Furthermore, this study suggests that time interval from initial MRI to successful recanalization is independently correlated with the lesion growth.

P-1-51

The correlation of aphasia with cortical lesion in diffusion-weighted image

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Background & Objectives: In the most of the cases, aphasia occur when the left perisylvian cortical areas are damaged, which are also found in the subcortical or right cortical regions in some cases. It has been reported that each type of aphasia has the correlation of specific brain lesion. But still, many studies are on searching which specific lesion manifests specific type of aphasia. In the previous study, age, aphasia type, and infarct size were associated with long-term improvement of aphasia. Thus, it is worth to determine aphasia type and severity just depending on MR imaging. In this study, we aimed to find the correlation of left cortical lesion with aphasia type with severity in diffusion-weighted image(DWI). **Method:** Patients with aphasia due to ischemic stroke were examined after stroke who admitted to Asan Medical Center (AMC) from January 2011 to December 2013 were enrolled. Patients with aphasia caused by ischemic lesions at right middle cerebral artery (MCA) or with aphasia as a sequela of previous stroke were excluded. The language performance was evaluated using Korean version of the Western Aphasia Battery (K-WAB). K-WAB test was performed within 2 weeks of stroke onset. The severity of aphasia was measured using the AQ index, a summation of multiplying each language components by 10, ranging from 0 to 100. The ischemic lesion was measured by manual drawing of the margin of DWI lesion by an investigator. Infarct location was quantified as regional extent of ischemic lesion (rEIL). The rEIL in each individual cortex was calculated as; (the volume of lesion within the individual gyrus / the total volume of the individual gyrus) on the automated anatomical labeling (AAL) atlas to specify the regional damages quantitatively. **Results:** During the study period, 231 patients admitted to the stroke center of AMC, among them 189 (81.8%) were enrolled to this study according to the inclusion criteria. The mean age of the included patients were 65.9 ± 12 and 126 patients (66.7%) was male. The mean AQ index of all types of aphasia was 41.2 ± 30.8 . In severity order, aphasia types were; global, Broca's, Wernicke's, mixed transcortical, transcortical sensory, conduction, transcortical motor, and anomia, respectively. After bonferroni correction, insula, rolandic operculum, putamen, and heschl gyrus were significantly related to AQ index. In global aphasia, the AQ index was positively associated with rEILs of insular ($p < 0.001$), rolandic operculum ($p < 0.001$) and frontal inferior operculum ($p < 0.001$). In Wernicke's aphasia, the AQ index was positively associated with rEILs of temporal superior cortex ($p < 0.0000$), angular ($p = 0.001$) and rolandic operculum ($p = 0.04$). In Broca's aphasia, the AQ index was positively associated with rEILs of precentral ($p = 0.004$) and frontal middle cortex ($p = 0.023$). In conduction aphasia, the AQ index was positively associated with rEILs of supramarginal ($p < 0.001$) and parietal inferior cortex ($p = 0.022$). **Conclusion:** In the lesion overlay, interestingly, insula was correlated with AQ index. Therefore, insula can be a predicting factor in aphasia severity and poor prognosis. The lesion of transcortical motor aphasia was more overlapped in putamen and caudate of subcortical area. So, basal ganglia could be probably crucial in speech production which is in line with recent studies which suggested that speech motor functions are affected by neural connections between the motor cortex and basal ganglia. Conduction aphasia has correlation with supramarginal and parietal inferior cortex, and this is in accord with the previous studies. This study described the correlation between cerebral infarct lesion location and aphasia severity with type. Moreover, we suggested that rEIL and voxel-wise infarct location are useful to evaluate aphasia. Further research should be needed to elucidate the correlation of lesion location with prognosis of aphasia.

P-1-52

Dabigatran effect on left ventricular thrombus in a patient with acute ischemic stroke and atrial fibrillation

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Background & Significance: The prevalence of coronary heart disease (CAD) in patients with atrial fibrillation (AF) is high, ranging in 18-47%. However, the incidence of left ventricular (LV) thrombus is rare in patients who have stable CAD and are taking antithrombotics. Accordingly, for patients who have acute ischemic stroke, atrial fibrillation, and LV thrombus associated with stable CAD, specific recommendations based on sufficient data are not available, particularly for the use of non-vitamin K antagonist (NOAC). Here we report a patient who had acute ischemic stroke, LV thrombus, and atrial fibrillation and was treated with dabigatran. **Case:** A 57 year-old man had sudden left hemiparesis, left hemihypesthesia, and anosognosia that occurred 20 minutes before the ER arrival. He had a history of stent insertion in the proximal left anterior descending artery due to ST-elevation myocardial infarction and atrial flutter 7 months ago. He had been taking aspirin and clopidogrel since then. At admission, the initial National Institute of Health Stroke Scale (NIHSS) score was 10, and CT angiography revealed right distal M1 occlusion. Intravenous alteplase and stent-retriever thrombectomy successfully recanalized the right M1 occlusion after 2.5 hours from the onset. Transthoracic echocardiography (TTE) performed at the day of admission revealed a thrombus (size: 1.1 x 0.89cm) at LV apex associated with apical inferior wall akinesia. After 24 hours, his neurological symptom substantially improved, and his NIHSS score was 0. Diffusion-weighted and gradient-echo images taken 24 hours after symptom onset showed multifocal ischemic changes in both MCA territory without hemorrhagic transformation. Taking into account both the necessity of and the bleeding risk with immediate anticoagulation, we decided to start dabigatran 110 mg twice daily. On follow-up TEE conducted after 7 days of dabigatran treatment, LV thrombus disappeared and there was no thromboembolic episode during the period. After discharge, the dose of dabigatran was increased from 110 mg twice daily to 150 mg twice daily, and he has had neither recurrent stroke nor bleeding complication. **Conclusions or Comments:** This is the first case report of dabigatran use in patients who had LV thrombus, atrial fibrillation, and acute ischemic stroke. In this clinical scenario that urgently necessitates effective and safe anticoagulation, dabigatran might be a useful option.

P-1-53

Use of new oral anticoagulants in a patient with recurrent cerebral venous thrombosis and spontaneous intracranial hemorrhage

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Background & Significance: The new oral anticoagulants (NOACs) are already certified for the treatment of deep vein thrombosis (DVT) in previous large trials. Cerebral venous thrombosis (CVT) is a kind of venous thrombosis which occurs under the hypercoagulable condition such as infectious or inflammatory disease and hematologic disorder. Despite pathophysiology and risk factors are similar in DVT and CVT, NOAC treatment for the CVT has not been established. We report a case of patient who presented recurrent CVT and intracerebral hemorrhage (ICH) and treated with oral factor Xa inhibitor, one of the NOACs. **Case:** A 67-year-old female presented emergency room (ER) with recurrent episode of facial numbness and dysarthria lasting 10 to 30 minutes. In ER, she was alert, cooperative and did not complain any neurologic symptom. She denied any history of hypertension, diabetes, and cerebrovascular disease. Brain MRI showed sulcal effacement in left fronto-temporal area, and dark signal intensity in left postcentral sulcus and left temporal lobe sulci on gradient echo image. MR venography showed decreased venous drainage from left temporal lobe toward the internal jugular vein. Laboratory study for the inherited and acquired coagulopathy showed no abnormality.

Under the diagnosis of CVT, oral anticoagulant, warfarin was prescribed for the patient. One month later, she revisited ER with transient right facial palsy and tingling sense on her right face. On follow up MRI, sulci blurring was more increased since last visit. Electroencephalography (EEG) was normal. Because INR was below therapeutic range, she was discharged after adjusting the warfarin dose. For one and a half year, there was no recurrence of any symptoms with taking warfarin before revisiting ER with stuporous mental state due to intracerebral hemorrhage (ICH). Despite INR level was within the therapeutic range at presenting ER, warfarin was discontinued for 4 months until recurrence of small ICH. There was no evidence of vasculopathy or aneurysmal change on transfemoral cerebral angiography. We considered CVT was a cause of recurrent ICH, oral factor Xa inhibitor was started. She discharged with mild cognitive impairment and no further event occurred more than 1-year follow-up period. **Conclusions or Comments:** We report a patient who suffered recurrent CVT and ICH under warfarin medication. In such patients, NOACs, which has been known to be stable in therapeutic efficacy and lower at risk of ICH to compare with warfarin, will be suitable.

P-1-54

Anticoagulant therapy can eliminate fresh thrombus in the internal carotid artery: a case report

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Background & Significance: In large atherosclerotic stroke patients, we generally manage to use anti-platelet agent, statins and angiotensin receptor antagonist as plaque-stabilizing agents. Nevertheless, it is not rare for them to have recurrent stroke. When it occur specifically artery-to-artery embolic stroke which originated in plaque rupture without severe stenosis in the internal carotid artery (ICA), fresh thrombi may be included in this plaque in the majority of this cases. This fresh thrombus has much higher risk of recurrent stroke than old one. Fresh thrombus contains much of red blood cells and can be dissolved with anticoagulant therapy. The optimal management of patients with this condition is unclear. **Case:** We report a patient with a recurrent ischemic stroke caused by thromboembolism from unstable plaque with combined fresh thrombus in the internal carotid artery. **Conclusions or Comments:** Plaque morphology may be important in causing stroke more than severity of stenosis. As a result, we recommend systemic anticoagulation as the first line treatment for fresh thrombus in addition to plaque-stabilizing agents. A duplex ultrasound is a useful tool to evaluate plaque vulnerability and detect fresh thrombus. Serial carotid ultrasound can definitely show propagation and dissolution of plaque and thrombus in spite of short-term follow up.

P-1-55

A case of splenic infarction associated with positive anticardiolipin antibody

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Background & Significance: Infarction of the corpus callosum is a relatively rare occurrence, as only 3-8%. The most susceptible location of ischemic corpus callosum lesion was the splenium. Splenium infarctions were often associated with bilateral cerebral hemisphere involvement (46.2%). The genu and/or body infarctions were associated with atherosclerosis. The most likely

vascular etiology has been variably reported as small-vessel disease, large-vessel atherosclerosis, and cardioembolism. **Case:** A 55-year-old male patient was admitted with motor weakness of left upper extremity. He had 14 days history of left upper arm weakness transiently. Two days ago, left upper arm weakness was progressive and persistent. He had a history of spontaneous intracranial hemorrhage with 5 years ago. He had no medications and no trauma history, recently. He had no stroke risk factor, such as hypertension, diabetes, dyslipidemia and smoking. On neurological examination, manual motor examination revealed left hemiparesis. Sensory modality was preserved, there was no babinski sign. He had no symptoms of disconnection syndrome. There was no dysarthria, and the initial NIHSS score was 0. Brain MRI revealed diffusion restrictions in right internal capsule posterior limb and splenium. There were multiple old lacunar infarctions in both basal ganglia and thalami. Also, there were multiple old small infarctions in both cerebral deep and subcortical white matters. Focal small subacute hemorrhage was found in left frontal deep white matter. There were no grossly abnormal findings on MR angiography and MR venography. Laboratory examinations including blood count, renal, and liver function analyses, C-reactive protein, and erythrocyte sedimentation rate revealed no abnormality. Lupus anticoagulant, FANA, ANCA, anti-Ro, La and aPTT was within normal range. But, anti-cardiolipin IgM titer was positive. He was very young, no risk factor of stroke, and there was no abnormality on MR angiogram and venogram. This condition may be antiphospholipid syndrome, although, lupus anticoagulant was negative. Unfortunately, the patient was follow-up loss and we could not repeat the anti-cardiolipin antibody and lupus anticoagulant. **Conclusions or Comments:** In conclusion, typical disconnection symptoms and signs are rare in the splenic infarction. Although the main pathogenesis of splenic infarction was unknown, it is important to more investigate for risk factors and etiology because splenic infarction has other specific or rare stroke etiologies.

P-1-56

Primary local tirofiban infusion and interventional treatment in internal carotid artery stump causing an acute ischemic stroke and early progression

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Background & Significance: Carotid stump syndrome is known to be associated with aggravation or recurrence of cerebrovascular events in the ipsilateral carotid territory. Although the role of patent proximal remnant of the occluded internal carotid artery (ICA) was not fully established, hemodynamic or embolic mechanism are presumed for cerebrovascular symptom. There have been several reports related with endovascular treatment for ICA stump, however, a universally accepted treatment does not exist up to now. We report a patient with acute ischemic stroke in which carotid stump was treated successfully with intra-arterial tirofiban infusion and following angioplasty and stenting. **Case:** A 64-year-old woman visited emergency room with dysarthria, left facial palsy and left lower limb weakness. Initial National Institutes of Health Stroke Scale (NIHSS) score was nine. From onset to door time was eight hours. She had a history of hypertension without treatment. Multifocal low density lesion at right middle cerebral artery (MCA) territory was seen on non-contrast computed tomography (NCCT). CT angiography (CTA) showed total occlusion of right proximal ICA, looking like a stump, with dense calcified plaques at the right carotid bulb, segmental occlusion of right MCA distal M1 and focal stenosis of right ACA proximal A1. Perfusion magnetic resonance image (MRI) showed significant delay in the whole right MCA territory. As an initial antithrombotic treatment, intravenous heparin was applied. After twenty one hours from initial presentation, aggravation of neurologic symptom with shift to NIHSS fifteen was noted. Repeat CTA showed

an extension of occlusion into distal right ICA to MCA distal M1 and ACA mid A1. The patient was transferred to angiographic suite. On transfemoral cerebral angiography, the total occlusion of right proximal ICA was observed. For revascularization treatment, tirofiban was administered through the microcatheter by hand injection in the proximal occlusion site. After injection of 0.5mg of tirofiban over 10 minutes, the occluded ICA was partially recanalized with remaining severe stenosis. Then, percutaneous angioplasty by 5×20 mm Sterling Monorail angioplasty balloon (Boston Scientific Corporation, Maple Grove, MN, USA) across the stenotic site was performed. The stenotic lesion improved with mild residual stenosis after angioplasty but the occlusion of right MCA distal M1 was still noted. Mechanical thrombectomy with a 4×20 mm of Solitaire FR (Medtronic-Covidien, Minneapolis, USA) was tried and the occlusion of right distal M1 was successfully revascularized. There was no reocclusion of right proximal ICA or right MCA on final common carotid artery angiogram. Repeat CTAs performed after 1 day and 9 day after endovascular treatment showed no evidence of reocclusion on corresponding arteries but twenty percent remnant stenosis of right proximal artery. Clinically, she did not present either recurrence or aggravation of neurologic symptom after the neurointervention. Other possible embolic sources were not found through further diagnostic investigation. At discharge, the patient's NIHSS had improved to two. **Conclusions or Comments:** Our case could suggest that intra-arterial tirofiban might be a feasible primary endovascular treatment option for ICA stump causing acute ischemic stroke and early progression. From our case and other literature evidences, approval and reimbursement of tirofiban as an endovascular treatment modality in acute phase of ischemic stroke should be necessary.

P-1-57

A case of cerebral infarction due to artery to artery embolization from calcified plaque of ipsilateral internal carotid artery

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Background & Significance: Artery to artery embolization is known to be the major cause of cerebral infarction. It has origin that can be traced back to many locations and has diverse histological structure. Previously, origin of calcified emboli with cerebral infarction could be presumed only based on brain imaging test. In this case, we present a case of cerebral infarction in which calcified cerebral emboli was detected in brain computed tomogram (CT). The calcified emboli was supposed to be from the severe proximal carotid stenosis, which supported the evidence of artery to artery embolization. **Case:** A 77-yr old men came to the emergency room of the Chonbuk National University Hospital with a complaint of sudden onset slurred speech and weakness of the right upper and lower limbs. The onset to door time was 30 minutes, and the initial NIHSS score was 4. The unenhanced brain CT showed no evidence of hemorrhage, but calcified spot was observed in the left middle cerebral artery. For the hyperacute stage of ischemic stroke, IV thrombolysis with rt-PA was performed. Magnetic resonance angiography of the carotid artery showed the severe stenosis of left proximal internal carotid artery, and carotid duplex ultrasonography of the artery showed the peak systolic velocity was 94.9 cm/sec. A left carotid endarterectomy was performed and the histologic examination showed surface ulcer and intraplaque hemorrhage. In addition, micro-CT was performed and showed the ulcerative calcified plaque. **Conclusions or Comments:** The present case report was an ischemic stroke which was due to artery-to-artery embolization. The etiology was evidenced by calcified spot of the relevant distal artery in brain CT and the ulcerative calcified lesion in the proximal ICA.

P-1-58

A case of right parietal infarction presenting as Gerstmann's syndrome

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Background & Significance: Gerstmann's syndrome is a neurologic disorder consisted of the clinical tetrad; finger agnosia, agraphia, acalculia, and right-left disorientation. The exact localization of the syndrome is remains uncertain, but most probable lesion is the dominant parietal lobe; left angular gyrus with a subcortical extension. However, there are few reports describing patients with right cerebral hemispheric lesion showing Gerstmann's syndrome. Herein, we reported a case study of right parietal infarction presenting as Gerstmann's syndrome. **Case:** A 52-year-old right-handed male patient was referred to our emergency room with chief complaint of aphasia. He was a high school-graduated salesman and had a history of HIV infection and hypertension. On neurologic examination, the patient was alert, oriented but not cooperative. Attention was impaired. Language testing revealed occasional hesitations in spontaneous speech. Naming and repetition was also impaired. Fluency was partially impaired. Mild weakness of right arm (grade IV+), mild dysarthria was shown. Diffusion weighted image showed high intensity of right parietal lobe and brain MRA showed focal narrowing of left A1 and poorly visualization of both MCA peripherals. Echocardiography and 24 hour holter monitoring were normal. 1 week after admission, Seoul Neuropsychological Screening Battery (SNSB) was done. SNSB revealed impaired function in fluency, comprehension, finger agnosia and periodic right-left confusion. **Conclusions or Comments:** It is recognized that the four components of Gerstmann's syndrome in an individual patient may manifest to a variable degree. Gerstmann's syndrome usually occurs with lesions of the left parietal lobe, and it is unusual for this syndrome to occur with lesions of the right parietal lobe. We have experienced and reported that Gerstmann's syndrome occurred with right parietal infarction.

P-1-59

A Case of bilateral thalamic infarction with Pulmonary Arteriovenous Malformation

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Background & Significance: Bilateral thalamic infarcts are rare presentations of stroke. They are the result of a complex combination of risk factors and a predisposing vessel distribution. The artery of Percheron, characterized by a single arterial trunk (type 2b) that irrigates both paramedian thalamic regions, can be occluded as a result of embolic diseases leading to bilateral paramedian thalamic infarcts. Pulmonary arteriovenous malformation (AVM) have been considered as the potential source of embolism through the right to left shunt in stroke patients. In one study, more than Grade 3 shunts were associated with a 10.4-fold increase in stroke/abscess. We report clinical and image findings of this uncommon form of posterior circulation infarct presented along with their anatomic and pathophysiologic correlates. **Case:** A 35-year-old woman was diagnosed as ischemic stroke in her bilateral thalamic infarction. The posterior circulation was patent including the tip of the basilar artery and both posterior cerebral arteries, making the case compatible with occlusion of the artery of Percheron. Transcranial Doppler sonographic examination with agitated-saline contrast revealed curtain-type microembolic signals. Microembolic signals last for 6 minutes from the infusion of agitated-saline (Grade 3 shunt). Further evaluation with an aim to define the etiology (spi-

ral chest CT) revealed a patent with pulmonary AV malformation as the cause of embolism. **Conclusions or Comments:** Bilateral thalamic infarcts are unusual presentations of posterior circulation stroke; once they are diagnosed by an adequate neuroimaging protocol, a further evaluation to define the cause is necessary. Cardioembolism should always be considered in relatively young patients. TCD findings as a screening test can be helpful to decision evaluation to pulmonary AVM or PFO. A complete evaluation should be conducted by an interdisciplinary team including neurologists, cardiologists and neurosurgeons.

P-1-60

D-dimer as a risk factor for END in cryptogenic stroke with cancer

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Background & Objectives: Early neurologic deterioration (END) is a common event in acute ischemic stroke up to 40%, and it makes poorer outcomes. But END risk factors and rate of END events in stroke with cancer have not been addressed. In previous study, cryptogenic stroke with cancer patients were thought to be more related to coagulopathy, representing higher d-dimer levels, more embolic signal in TCD monitoring. In this study, we evaluate the possibility of D-dimer as a predictor of END in cryptogenic stroke with cancer. **Method:** Between March 2001 and June 2015, we enrolled 70 patients with cryptogenic stroke with active cancer. Active cancer was defined as a diagnosis of cancer, within 6 months before enrollment, or recurrent or metastatic cancer, as described previously. We assessed the neurological severity on admission by NIHSS and baseline demographic data, including age, sex, hypertension, diabetes, dyslipidemia, familial history of stroke, cigarette smoking, history of venous thrombosis. Data relating to cancer, including type and stage of cancer and diagnosis or metastasis or progression of cancer to stroke were also recorded. END was defined as an increase of 1 point or more on the NIHSS total score after 72 hours from baseline assessment. All patients underwent MRI, MRA, routine blood test, coagulation study, CRP, D-dimer, ECG, transthoracic echocardiography. **Results:** END was determined in 24 patients (34.3%) within 1st 72 hrs. The mean d-dimer level was 10.92 ± 10.84 mg/mL in the END(+) and 6.22 ± 8.92 mg/mL in the END(-). Initial NIHSS score, D-dimer levels, MRI multiple vascular territory pattern, DM were associated with END by univariate analysis ($P < 0.05$). After logistic regression, only DM and D-dimer remained as independent predictors of END. The odds ratio for END increased with increasing quartile levels of D-dimer with the lowest quartile used as the reference value. The fourth highest quartile of D-dimer level was identified as independent predictors of END. **Conclusion:** In this retrospective study, we found that D-dimer level is an independent marker of END in cryptogenic stroke patients with cancer, regarding hypercoagulability related stroke.

P-1-61

Low plasma proportion of omega 3-polyunsaturated fatty acids determine cerebral small vessel disease in acute ischemic stroke patients

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Background & Objectives: Cerebral small vessel diseases (SVDs) are related with stroke or cognitive dysfunction. Meanwhile, ω 3-polyunsaturated fatty acids (FAs), such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are highlighted as disease modifying factors for cardiovascular disease

or dementia. In this study, the association between composition of plasma FAs and cerebral SVDs were investigated in acute ischemic stroke patients. **Method:** We prospectively enrolled 220 patients with first episode cerebral infarction within 7 days after symptom onset. The composition of FAs was analyzed by gas chromatography methods. The presence and burden of cerebral microbleeds (CMBs), high grade white matter changes (HWCs), high grade perivascular spaces (HPVSs) and asymptomatic lacunar infarctions (ALIs) were investigated. **Results:** The mean proportion of EPA was 2.0 ± 0.7 , DHA was 8.9 ± 1.5 and that of $\Sigma \omega$ 3-polyunsaturated fatty acids (PUFAs) was 12.0 ± 2.1 . In total patients, 46 (20.9%) patients had CMBs, 64 (29.1%) had HWCs, 57 (25.9%) had HPVSs and 65 (29.5%) had ALIs. The burden of CMBs, HWCs and HPVSs were negatively correlated with proportion of EPA, DHA and $\Sigma \omega$ 3-PUFAs. In multivariate analysis, lower proportion of EPA, DHA and $\Sigma \omega$ 3-PUFAs were associated with presence of CMBs, HWCs and HPVSs, but not with ALIs. Total SVDs score was significantly and inversely correlated with proportion of EPA, DHA and $\Sigma \omega$ 3-PUFAs. **Conclusion:** Our results demonstrate low proportions of ω 3-PUFAs are associated with SVDs pathologies. Regulation of ω 3-PUFAs may protect against progression of cerebral SVDs.

P-1-62

Polycythemia Vera as a risk factor of borderzone infarction

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Background & Objectives: Polycythemia Vera (PV) is a bone marrow disease that leads to an abnormal increase in the number of red blood cells. Increased blood viscosity can result in vascular complications such as ischemic stroke. Here we report a case of patient with recurrent cerebral infarction in borderzone area with only minimal intracranial and extracranial vasculature abnormalities. **Method:** A 82-year-old man presented left arm weakness over 3 days. The only risk factor was hypertension for 20 years and he did not have diabetes, dyslipidemia, smoking history and alcohol consumption history. One month before admission, he visited nephrology department due to both lower extremity pitting edema. It was spontaneously improved after discontinuation of calcium channel blocker. However, laboratory finding showed increased hemoglobin up to 18.3 g/dL and he was transferred to hematology department. Before evaluating bone marrow and genetic testing, left arm weakness was suddenly developed. Brain MRI showed right MCA-ACA borderzone infarction without angiographic abnormalities. He did not show any cardiac abnormalities on EKG, Echocardiography and Holter monitoring. After acute treatment for ischemic stroke, his symptoms were improved and secondary prevention was started with clopidogrel 75mg/day. He referred to hematology department, and bone marrow biopsy with genetic testing was performed. Five days after discharge, he complained sudden onset quadriplegia with confusion before diagnosis of polycythemia was made. Brain MRI showed newly developed both MCA-ACA borderzone infarction without angiographic abnormalities. After second admission, diagnosis of polycythemia vera was made based on hypercellularity of bone marrow and JAK2 V617F mutation. For treatment of polycythemia vera, hydroxyurea 500mg twice was started and phlebotomy was performed. He discharged with relative improvement of the initial symptoms without recurrent event for 2 months. **Conclusion:** Polycythemia vera increases blood viscosity which is well known risk factor for thrombotic event and also for ischemic stroke. This case highlights the occurrence of multiple and recurrent ischemic strokes as an initial presentation of PV with normal vasculature. Further research and screening about PV as a risk factor for ischemic stroke seems to be critical for prevention of ischemic stroke.

P-1-63

Relation between glycoalbumin level and acute ischemic stroke

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Background & Objectives: Hyperglycemia has been observed after acute stroke, and is associated with a poor prognosis. Glycated hemoglobin (HbA1c) level is widely recognized as the independent risk factor of the AIS. Albumin is the most abundant serum protein and all of which can be glycated. Just like HbA1c, glycoalbumin(GA) measurements serve as an index of the mean concentration of glucose in the blood during the preceding several weeks. We aim to investigate the role of GA in the risk prediction of ischemic stroke. **Method:** We retrospectively studied 256 AIS patients admitted to our University Hospital between January 2015 and June 2015. Type of stroke, level of GA, HbA1c, NIH stroke scale(NIHSS) score measurements were done at the baseline, and NIHSS score and modified Rankin scale(mRS) score follow up at the discharge day. **Results:** On admission, AIS patients had GA values collected ; mean age, 65.1 [SD, 12.4] years; GA levels showed a statistically significant correlation with HbA1c level ($P<0.01$) in study group. However there were no significant differences neither in the GA level nor HbA1c level and prognosis of AIS. **Conclusion:** Diabetes are commonly found in acute ischemic stroke patients. GA provides advantages over measuring HbA1c as they are not affected by RBC life span, and the results of GA levels can be received faster than HbA1c. However, our data cannot be shown the significant relation between GA level and the prognosis of stroke. Further studies are needed to confirm our findings in larger populations.

P-1-64

Risk factors of carotid artery calcification

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Background & Objectives: Previous study noted that ICA calcification was related to several vascular risk factors. The purpose of this study is to assess variables associated with carotid artery calcification. **Method:** We analyzed consecutive patients who had checked computerized tomography angiography(CTA) scans of head and neck in Boston university medical center(BMC) since January, 2006 to June, 2009. Total amount of calcification was calculated from area of calcification obtained from every slices of CTA source image with 5mm thickness from petrous portion to supraclinoid portion of ICA. We divided patients by 5 groups according to estimated total volume of ICA calcification (absence, minimal $\leq 10\text{mm}^3$, mild 11-40 mm^3 , moderate 41-80 mm^3 , severe $\geq 81 \text{mm}^3$). Demographic information and basic medical information available in the electronic chart were recorded retrospectively. **Results:** A total of 201 patients were included and 402 arteries were examined in the study. Among several variables, age, hypertension, previous ischemic stroke and coronary heart disease were associated with carotid calcification in univariate analysis. Age ($P<0.0001$) and old ischemic stroke (OR, 4.327; 95%CI, 1.72-10.88; $P=0.002$) were only 2 factors associated with significant carotid calcification in multivariate logistic regression analysis. **Conclusion:** Age and previous ischemic stroke were independent predictors of ICA calcification.

P-1-65

Cardiac myxoma: another cause of transient global amnesia? - heart and mind

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Background & Significance: We report a patient who presented with transient global amnesia (TGA) with right parietal cortical infarction caused by cardioembolism due to cardiac myxoma. **Case:** A 38 year-old right-handed male with twelve year of education came to ER with acute onset amnesia. His wife said he did not remember anything he did during the day and repeatedly asked her "What did I say?" any time saying something to her. He did not complain about dyspnea, faintness or dizziness. Previously he had dyslipidemia but not treated with medication. On examination, he was well oriented except to time and was unable to retain new information. His general and neurological examinations were normal. He had a brain MRI with diffusion, MRA and MR perfusion. For further evaluation, he underwent neuropsychological test, Holter monitoring, transcranial Doppler with saline agitation test, routine blood tests, transthoracic and transesophageal echocardiography. On MRI, there were multiple small nodular and irregular diffusion restrictions at the right posterior frontal, parietal subcortical white matters and insular lobe. And MRA and MR perfusion showed no abnormal stenotic lesions and perfusion delay. His amnesic episode resolved after 12 hours of onset. But neuropsychological test performed on the 3rd hospital day revealed memory impairment especially delayed recall in verbal memory without overcome by recognition while immediate and delayed recall impaired but overcome by recognition in visual memory. Also it showed impairment in frontal executive function but sparing of attention, language function and praxis. Holter monitor was normal. Transcranial Doppler showed normal intracranial flow pattern and no microembolic signal was found during saline agitation test. On transthoracic echocardiography there showed 10 X 6 mm sized mass-like lesion on the anterior leaflet of mitral valve in the left atrium without any other valvular or wall motion abnormality. During transesophageal echocardiography this lesion revealed as 15 x 7 mm sized fluctuating lobulating mass-like lesion suggestive of myxoma. Emergent surgery was decided and performed on the 4th hospital day. Irregular shaped pale brown soft tissue was resected and the diagnosis of cardiac myxoma was confirmed by pathology report. No perioperative complication was occurred. And he discharged home without neurologic deficit on the 14th hospital day. **Conclusions or Comments:** In young stroke patient with unknown etiology, thorough cardiac evaluation is important to find intracardiac abnormalities such as cardiac myxoma or infective endocarditis as a cause of cardioembolism.

P-1-66

Subarachnoid hemorrhage in patients with systemic lupus erythematosus

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Background & Significance: Systemic lupus erythematosus (SLE) is an autoimmune disorder of multifactorial etiology with a broad range of clinical manifestations. Psychosis, seizures and cerebrovascular accidents such as SAH are commonly described in SLE patients. Subarachnoid hemorrhage (SAH) has been shown a higher incidence in patients with SLE than in the general population. Although saccular aneurysms are the most frequent cause of SAH, uncommon forms of SAH has been rarely reported in SLE patients. We report two cases, which we outline these uncommon patterns of SAH in patient with SLE whose detailed description may help to improve diagnostic and management strategies. **Case:** Case 1 : A 18-year-old woman complained of sudden severe headache and vomiting. She had been diagnosed with SLE 8 years ago, and was being treated with prednisolone, mycophenolate mofetil. On neurologic examination, she had severe headache with neck stiffness and Kernig sign. She did not showed any motor and sensory deficit. Brain CT revealed high attenuated lesion in premedullary cistern without aneurysmal dilatation of vertebralbasilar arteries. However, vertebralbasilar arteries were stenotic

compared with previous angiography which was performed 1 month ago. Immediate TCD showed high flow velocities on bilateral MCAs (120-160cm/s), and basilar artery (77-81cm/s) which was compatible with reversible cerebral vasoconstriction syndrome. Intravenous injection of calcium channel blocker with nimodipine was administered for eight days and her headache and SAH were resolved. Case 2 : A 30-year-old woman was transferred to our hospital with dyspnea. She has been diagnosed with SLE 8 years ago and interstitial lung disease 6 years ago. On hospital day 4, she complained of sudden severe headache and brain CT revealed cortical SAH located in the right frontal lobe. Subsequent CT angiography showed no significant steno-occlusive lesions or aneurysmal dilatation. Transcranial doppler (TCD) ultrasonography showed decreased flow velocities on bilateral MCAs (range 30-34 cm/s) and basilar artery (22 cm/s) which could be attributed to increased intracranial pressure. High resolution MRI vessel wall imaging revealed concentric enhancement on post-contrast T1 enhancement scans. According to the diagnosis of flare up SLE and systemic vasculitis including intracranial arteries, high dose steroid (1000mg) and intravenous-immunoglobulin (15g) therapy was administered and headache was improved. **Conclusions or Comments:** Nonaneurysmal SAH in patients with SLE is rare and has clinical significance affecting the mortality and morbidity. The cause of SAH could be related to the vasculitis in high disease activity. However, clinicians should be aware of RCVS which could show non-vasculitic SAH because the treatment is different from each other. Imaging studies including the MR angiography and vessel wall imaging as well as TCD may be helpful to make the differential diagnosis.

P-1-67

Brainstem cavernous malformation with ipsilateral abducens nerve palsy with good outcome after conservative treatment

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Background & Significance: The management of the cerebral cavernous malformation (CCM) remains controversy over whether surgical removal makes better outcome. Prior studies suggested that selected symptomatic patients should undergo surgery, but recent studies demonstrated that surgical removal of CCM was related with worse outcomes over 5 years compared to conservative management. But, in some cases of BSCMs, if the lesions are left without removal, progressive brainstem dysfunction could be inevitable, so some clinicians recommend surgical removal of BSCMs in surgically accessible lesions. We report a case of patient with isolated unilateral sixth nerve palsy with brainstem cavernous malformation, whose symptoms was improved by conservative treatment. **Case:** A 21-year young male was admitted to our hospital via outpatient clinic. He had diplopia symptom which suddenly developed 5 days ago. Before 1 week, he suffered for headache without nausea but it improved after taking analgesics. And one day after, binocular horizontal diplopia was developed. On neurological examination, limited abduction on left eye was detected by clinician. Horizontal diplopia developed only during the left-sided gaze and his diplopia symptom was non-fluctuating. Otherwise, he had normal visual acuity and found no significant ptosis. Orbital pain was not exist. We didn't observe any other neurological deficit in neurologic examinations. He didn't have any other underlying disease, medical history, congenital malformation, family history and trauma history. In laboratory examination including spinal tapping, We didn't finding any specific information. But his brain magnetic resonance imaging, there was focal lesion at left anterior ponto-medullary junction, which consistent with cavernous malformation. In our case, we considered the radiosurgery by Novalis treatment if the symptom aggravated or newly developed. But prior to do the invasive method, the conservative treatment was done, and his diplopia was almost improved 3 months later. **Conclusions or Comments:** Even if the recurrence of cavernous

malformation's bleeding risk is relatively high, cavernous malformations do not involve internal neural tissue, so hemorrhage of these lesions can be asymptomatic or result in only minor neurologic dysfunction. But hemorrhage from a BSCM is more likely to be symptomatic than hemorrhage from a CCM. Otherwise, the surgical risk of BSCMs is also very high. So the priority of the management whether surgical removal or conservative treatment is still controversy and still discussed. Because the bleeding risk and adjacent symptoms of cavernous malformation are variant, and the complications of CCM's bleeding progress slowly in most cases, close observation and conservative treatment should be considered first. And if the recurrence occur frequently, or the complication are obvious, surgical treatment should be considered. But in some cases, acute massive hemorrhage with severe complications can occur, further studies for estimating the bleeding risk and complications will be needed. We report a case with good outcome after conservative treatment and natural course of the brainstem cavernous malformation by follow-up MR image.

P-1-68

A 60-year-old woman with recurrent stroke-like episodes : what is the standard range of diagnostic work-up for cryptogenic strokes?

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Background & Significance: Cryptogenic strokes could be divided into embolic strokes of undetermined source (ESUS) and non-ESUS. In contrast to the fact that ESUS is explored through extensive and meticulous heart work-up, non-ESUS is still considered apologetic. We define non-ESUS of cryptogenic stroke for the first time and suggest the standard range of diagnostic evaluation. **Case:** We presented a case of 60-year-old woman with recurrent stroke-like episodes, who is initially diagnosed as cryptogenic stroke, but finally intravascular lymphoma (IVL). On her first visit, she complained of sudden language disturbance, mild weakness, and dull sense on the right side from 2 months ago. Neurologic examinations showed transcortical motor aphasia, dysarthria, right hand grip weakness, and hypoesthesia on the right side to pain and touch modalities. The lesions were thought to be involved in the left middle cerebral artery territory and the most plausible etiologic diagnosis would be vascular cause because the clinical presentation was sudden and recurrent. Magnetic resonance imaging (MRI) showed multistage and multifocal diffusion restriction in the left parietotemporal subcortical areas and intracranial and extracranial arteries were normal. We investigated the embolic source via transthoracic and transesophageal echocardiography and 24-hour Holter monitoring. All the results showed no evidence of cardiac emboli, but patent foramen ovale (PFO) was documented on the transcranial doppler (TCD) ultrasonography. She was diagnosed as cryptogenic stroke and treated with aspirin and clopidogrel. She spontaneously recovered except mild dysarthria during hospital stay. It was 3 weeks later that she revisited hospital complaining of aggravated dysarthria, gait difficulty, and abnormal behavior from 2 days ago. Neurologic examination showed altered mentality, motor-dominant global aphasia, dysarthria, and weakness in 4-extremities. Suspecting global encephalopathy, we performed brain imaging and electroencephalography. The results showed newly appeared multistage multifocal infarctions in both cerebral hemispheres and left temporal lobe epilepsy. We took steps toward assessment through chest, abdomen, and pelvis computed tomography (CT) resulting in no evidence of malignancy. Changing medication from antiplatelet agent to anticoagulant based on the follow-up TCD revealing 2 spontaneous embolic signals, she experienced neurologic deterioration once more. Unexpectedly, she started to show sustained inflammatory signs of fever and elevated C-reactive protein (CRP), but no infection focus was identified. Pursuing fever of unknown origin as a clue, we

suspect central nervous system (CNS) involvement of vasculitis or malignancy. Steroid response was remarkable for subsiding both fever and CRP. The cerebrospinal fluid (CSF) analysis supported malignancy because T-cell receptor-gamma (TCR-gamma) monoclonality was positive in CSF. Finally, the brain tissue biopsy was done and the pathologic diagnosis was intravascular lymphoma. It was after the lapse of 8 weeks from her first visit that she started to undergo chemo- radiotherapy. **Conclusions or Comments:** As IVL requires earlier treatment to better prognosis, we suggest that the diagnostic evaluation for non-ESUS jump over a fence of established cardiac work-up and expand to the decisive brain biopsy.

P-1-69

An unusual case of lateral medullary infarction initially presenting as isolated vertigo and magnetic gait preceding change of diffusion weighted MR imaging

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Background & Significance: Lateral medullary infarction is clinically identified by presenting the triad of Horner's syndrome, ipsilateral ataxia, and contralateral hypalgesia. Also, other variable symptoms of lateral medullary infarction reported including dysphagia, hoarseness, hiccup, ataxia of limb and gait. Isolated vertigo and magnetic gait symptoms like psychogenic movement disorders are not known as initial symptoms in lateral medullary infarction. Our aim is to report a case of unusual phenotype of lateral medullary infarction which shows isolated vertigo and magnetic gait as initial neurological symptoms preceding change of diffusion MR imaging without other typical symptoms **Case:** A 61 year-old Korean women referred to us due to isolated vertigo and magnetic gait. She had visited at emergency room because the same symptoms two days ago, but at that time she had not presented other neurological symptoms and signs and diffusion-weighted brain magnetic resonance imaging had showed no signal changes. However, further studies and examinations revealed small lesions of lateral medullary infarction and progression of hemisensory symptoms and gait ataxia. She diagnosed as lateral medullary infarction associated dissection of vertebral artery and her symptoms were improved by treatment of anticoagulation therapy. **Conclusions or Comments:** Based on review of the literature, initial neurological symptoms of lateral medullary infarction are vertigo and hemisensory symptoms or Horner's syndrome accompanied change of diffusion weighted MR imaging, but we have reported an unusual case of lateral medullary infarction initially presenting as isolated vertigo and magnetic gait preceding change of diffusion MR imaging and other typical neurological symptoms.

P-1-70

A case of midbrain infarction causing isolated fourth nerve palsy

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Background & Significance: Trochlear nerve innervates the superior oblique muscle, which adducts and intorts the eye. Trochlear nerve palsy affects eye to be hypertropic and extorted, resulting in vertical diplopia. Traumatic, congenital, and microvascular disorders are the most common etiologies of trochlear palsy. Trochlear palsy mostly accompanies other neurological deficits such as Horner's syndrome, sensory change, internuclear ophthalmoplegia (INO), nystagmus and ataxia. However, isolated trochlear palsy due to midbrain in-

farction is uncommon. Few cases of isolated trochlear nerve palsy with mid-brain infarction has been reported. We report a patient with isolated trochlear nerve palsy due to midbrain infarction. **Case:** A 78-year-old woman with hypertension and type 2 diabetes for seven years presented to neurology department. She complained of sudden vertical diplopia which suddenly developed one day ago. She was taking medication for hypertension and diabetes. Examination revealed left eso-deviation and left hypertropia at left eye in the neutral position. Vertical diplopia aggravated in downward and right gaze and on left head tilting. She has no history of head trauma. Laboratory evaluations were all normal. Brain diffusion weighted image showed a small infarction restricted to right trochlear nucleus. And magnetic resonance angiography showed vascular irregularity at both vertebral arteries. She was started anti-platelet therapy. And appropriate fluid therapy was applied. And then, she take absolute bed rest. After 5 days later, she has no diplopia and her symptom has been completely resolved. And she discharged with medication. **Conclusions or Comments:** The etiological spectrum of acquired trochlear palsy is diverse. Trauma and microvascular diseases are most common etiologies. But other etiologies include tumors of the pineal region, fourth ventricle or cisterns of the great cerebral veins, aneurysms, Fisher syndrome, connective tissue diseases and infections such as meningitis, herpes zoster, and syphilis. Furthermore, ocular myasthenia gravis, thyroid ophthalmopathy and skew deviation may mimic trochlear palsy. Focal brain stem infarction occasionally causes trochlear palsy by involving trochlear nuclei or fascicles. However, since the trochlear nucleus and fascicles are surrounded by the ascending trigeminothalamic and spinothalamic tracts, medial longitudinal fasciculus (MLF), brachium conjunctivum, descending sympathetic tract and superior cerebellum, trochlear palsy due to brain stem stroke usually accompanies other symptoms such as sensory impairments, internuclear ophthalmoplegia, upbeat nystagmus, Horner's syndrome or ataxia In this chase, we suggest that focal midbrain infarction should be considered as a rare cause of trochlear palsy even though there are no associated neurological deficits.

P-1-71

A woman with transient left side motor weakness with systemic lupus erythematosus

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Background & Significance: Systemic lupus erythematosus (SLE) is closely associated with cerebrovascular disease, and several etiologic factors such as vasculitis, cardiac diseases coagulopathies and atherosclerosis have been suggested. Here, we describe a case of SLE patient with rapidly progressive large vessel occlusion within 3 months. **Case:** A 29-year-old woman was admitted with a chief complaint of transient left side motor weakness and sensory change for 3 days. She had diagnosed with SLE nine years ago, continued taking corticosteroid per oral. Three months before admission, brain Magnetic resonance imaging (MRI) at that time revealed mild stenosis of both M1 and right M2 division, but no parenchymal lesion. On admission to neurologic department, she described that the transient left motor weakness occurred more than 5 times during less than 2 minutes. In laboratory study, she had elevated triglyceride, hyperhomocysteinemia and leukocytosis. Mild inflammatory syndrome, tests for anti-double stain DNS (dsDNA), IgG anti-cardiolipin antibody, IgG anti-phospholipid antibody and lupus anticoagulant (LA) was positive. On brain MRI, focal diffusion restriction in right frontal lobe was seen in the diffusion restriction image and occlusion of right MCA and stenosis of left M1 division. Right middle cerebral artery stenosis was much progressed compared to previous vessel image which was taken three months ago. Diagnostic cerebral vessel angiography was done to investigate the evidence of vasculitis, but only severe atherosclerosis at right middle cerebral artery was

noted. Intravenous heparin infusion started immediately and the outcome was favorable. On day of the discharge, no neurologic deficit was noted. **Conclusions or Comments:** In our patients, cerebral artery stenosis was aggravated within short period without evidence of vasculitis. Previous studies revealed that patients with SLE were more vulnerable to cerebral vascular accidents and pathogenesis of ischemic stroke is described in many different mechanisms. Both coagulopathy and mechanism of atherosclerosis may play role in progression. In autoimmune disease patient, progression of atherosclerosis can occur rapidly; tight regulation of risk factor, autoantibody study, and early anti-coagulation may be helpful for prevention of ischemic stroke and TIA.

P-1-72

Decreased emotion recognition is associated with impaired activity of daily living in the patient with early Alzheimer's disease

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Background & Objectives: The accurate recognition of facial expression is an important for interpersonal communication and social behavior. This study was to determine characteristics of facial emotion recognition in patients with early Alzheimer's Disease (AD) and was to investigate the effect of facial emotion recognition on early AD. **Method:** 21 early AD patients and 19 normal controls were enrolled. All patients and normal controls conducted the comprehensive neuropsychological tests including facial recognition task and emotion recognition task. The measurement index of facial recognition task and emotion recognition task was proportion of correct responses. **Results:** Performance on facial recognition task and emotion recognition task was significantly different between early AD group and control group. Performance on emotion recognition task was also significantly different between two groups after the effect of facial perception was controlled. Among the emotion, it significantly impaired to recognize happiness, sadness, anger and surprise in early AD group than control group. In addition, there was significant negative correlation between emotion tasks and instrumental assessment of daily living in early AD group. **Conclusion:** Emotion recognition deficit were independent of face recognition deficit and was associated with impaired activity of daily living in early AD. It should be noted that emotion recognition is also valued as well as cognitive impairment.

P-1-73

The combined influence of vascular risk factors on cognitive decline among community-dwelling elderly in Seoul

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Background & Objectives: The effects of vascular risk factors on the incidence of dementia have been well researched. However, the combined influence of these vascular risk factors on cognitive decline in the elderly with normal cognition is not so clear. **Method:** This three-year longitudinal observational study (2010-2013) includes 58,852 community-dwelling healthy elderly with normal cognition at the baseline. This study is part of the Seoul Dementia Managing Project in Seoul, Korea. Cognitive impairment was diagnosed if participants scored below 1.5 standard deviation on the mini-mental status examination (MMSE) compared to norms. All baseline information related to participants was self-reported using a structured questionnaire. For the preliminary analy-

sis, all participants were further divided into the four groups according to presence of hypertension or diabetes; none, hypertension only, diabetes only, and both hypertension and diabetes group. The Kaplan-Meier method was used to estimate the cumulative incidence rate. Differences in incidence of cognitive impairment according to the four groups were analyzed with the log-rank test. The Cox proportional hazard model was used to explore the hazard ratio (HR) for the development of cognitive impairment according to those four groups. **Results:** Among a total of 58,852 participants, 24,900 (42.3%) were included in the Group 1 (neither hypertension nor diabetes), 2,737 (4.6%) for Group 2 (diabetes only), 23,245 (39.5%) for Group 3 (hypertension only) and 7,970 (13.5%) for Group 4 (both of hypertension and diabetes). People in the Group 3 and Group 4 were significantly older and less educated compared to others, while the scores of MMSE at the baseline was lower only in the Group 4 compared to other groups. The average decline of MMSE scores over 3 years in the Group 1 was -0.6 ± 2.8 , whereas those in the Group 2,3,4 were -1.0 ± 2.9 , -0.7 ± 2.8 and -0.8 ± 3.0 respectively. The cumulative incidence rate (CIR) of cognitive impairment in Group 1 was 3.24 % (95% CI, 3.21-3.26), whereas the CIR of cognitive impairment in Group 2,3,4 was 3.00 % (95% CI, 2.95-3.04), 3.25% (95% CI, 3.21-3.26), and 4.81 % (95% CI, 4.87-4.74). **Conclusion:** Our study showed that there were no significant additive interactive effects between hypertension and diabetes on the development of cognitive impairment. Interestingly, people with diabetes only (Group 2) showed greater decline of MMSE scores compared to those with both risk factors (Group 4). Further studies should be warranted to investigate the combined relationship of vascular risk factors on cognitive decline in the elderly

P-1-74

2 cases of adult-onset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP) diagnosed by CSF1R gene mutation

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Background & Significance: Adult-onset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP) is an emerging differential diagnosis of middle age onset dementia. It is a rare autosomal-dominantly inherited neurodegenerative white matter disease composed of 2 disease entity - hereditary diffuse leukoencephalopathy with axonal spheroids (HDLS) and pigmentary orthochromic leukodystrophy (POLD). These two diseases were recently found to be derived from a mutation of the same gene, colony-stimulating factor 1 receptor (CSF1R). CSF1R is a cell surface receptor expressed highly on myeloid lineage cell lines, including microglia. Its mutation is known to cause leukodystrophy by impairing microglial survival, proliferation and differentiation. We described 2 patients diagnosed as ALSP with CSF1R gene mutation. Each patient was formerly diagnosed as late onset cerebellar ataxia and undetermined leukodystrophy. **Case:** Patient 1. A 55 year old woman visited a memory clinic complaining of a 5-year history of progressive gait disturbance and cognitive impairment. There was no family history of neurodegenerative diseases. Neurological examination revealed cerebellar ataxia with pyramidal tract signs. Executive dysfunction was noted with relative sparing of memory function. Gene tests performed under the tentative diagnosis of late onset cerebellar ataxia, including Spinocerebellar ataxia (SCA), Dentatorubral-pallidoluysian atrophy (DRPLA), and Huntington's disease were negative. Brain MR images at 5-year after symptom onset demonstrated thinning of corpus callosum and progressive white matter signal changes on FLAIR sequence with relative sparing of cerebellum. A CSF1R gene test revealed a missense mutation (c.1946T>G), a novel mutation, which is likely to be pathogenic rather than a mere polymorphism, according to simulation program. Patient 2. A 47 year old man visited a movement disorder clinic with a 2-year history of pro-

gressive gait disturbance and mental slowing. Because the patient was the only child of a deceased single mother, the family history was not obtainable. Neurological examination revealed left dominant bilateral parkinsonism with severe cognitive impairment. Brain MRI showed thinning of corpus callosum and high signal intensities in white matter similar to the patient 1. Gene tests, targeting leukodystrophy with cognitive impairment, including Adrenoleukodystrophy, Metachromatic leukodystrophy, DRPLA, and cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) were negative. While being followed up with a tentative diagnosis of leukodystrophy with behavioral symptoms, the patient developed recurrent seizures. A CSF1R gene test revealed a pathologic missense mutation (c2381T>C). **Conclusions or Comments:** ALSP is a middle-age onset, autosomal-dominantly inherited, rapidly progressive white matter disease with wide range of clinical symptoms, including dementia, behavioral change, seizure and various movement symptoms. The MR findings are known to be relatively consistent among patients, which is characterized by thinning of corpus callosum and white matter signal changes with frontal predominance on T2-weighted image. ALSP was formerly identifiable only by brain biopsy or autopsy findings of axonal spheroids or pigmented macrophages, until CSF1R gene mutation was discovered. Impairment of CSF1R-mediated microglial repair of axonal degeneration might contribute to the white matter changes of ALSP. There have been only a few pathologically or genetically diagnosed ALSP patients in South Korea. Since gene test is a more approachable and less invasive diagnostic tool than a biopsy, it is promising to have the CSF1R gene test for diagnosing ALSP. In summary, the CSF1R gene test can be considered in patients with early onset dementia with gait disturbance who do not fit into typical courses of known dementia or movement syndromes, particularly, if the brain MR images show thinning of corpus callosum and white matter signal changes.

P-1-75

Predictive factors for objective cognitive impairment in a screening population with subjective cognitive impairment

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Background & Objectives: Although individuals with subjective cognitive impairment tend to be at increased risk for significant cognitive impairment including various types of dementia, it is still challenging to differentiate those having objective cognitive impairment from those who are cognitively normal but having only subjective impression for their cognitive decline in health screening programs. Based on this practical necessity, we aimed to investigate the characteristics of specific individuals showing objective cognitive impairment on neuropsychological tests among visitors with subjective cognitive complaints in a health screening center. **Method:** Individuals who underwent a health checkup between March 2013 and January 2015 and who did not have a history of major stroke or dementia were enrolled in the study. Subjects were screened with the Subjective Memory Complaints Questionnaire (SMCQ) for their complaints of subjective cognitive impairment, and also underwent detailed neuropsychological tests, brain MRI, and apolipoprotein E genotyping as well as medical screening tests for routine checkup. Logistic regression analyses were conducted with objective cognitive impairment and diagnosis of amnesic mild cognitive impairment (MCI) or Alzheimer's disease (AD) as dependent variables. **Results:** Among 265 subjects enrolled (118 males), 174 subjects (65.7%) were objectively normal in cognition while 91 subjects (34.3%) showed objective as well as subjective cognitive impairment. 62 out of 91 subjects with objective cognitive impairment (68.1%) were diagnosed as either amnesic MCI or AD. When logistic regression analyses were conducted,

previous history of ischemic heart disease, hippocampal atrophy, and positive answer for SMCQ 4 (Do you feel that your everyday life is difficult due to memory decline?) and 12 (Do you have difficulty in remembering 2 or 3 items to buy when shopping?) were significantly associated with existence of objective cognitive impairment while only hippocampal atrophy and positive answer for SMCQ 4 and 12 were associated with diagnosis of amnesic MCI or AD. **Conclusion:** On the basis of the results, visitors with past history of ischemic heart disease, those feeling difficulty in their everyday life due to memory decline or having difficulty in remembering items to buy when shopping, and those showing hippocampal atrophy on their MRI could have higher risk for showing significant objective cognitive impairment on further neuropsychological tests in health screening programs.

P-1-76

A comparison of the performances on the K-VCIHS-NP according to the CDR-SB in vascular MCI, amnesic MCI, vascular dementia, and Alzheimer's disease

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Background & Objectives: The present study was conducted to examine whether the subgroups classified by CDR-SB scores show the corresponding group differences on the K-VCIHS-NP. **Method:** The subjects were 41 normal elderly (NE), 89 VaMCI, 42 VaD (CDR-SB 4.5~7.0), 86 aMCI and 41 AD (CDR-SB 4.5~6.5). The MCI groups were classified into "Early MCI (E-MCI, CDR-SB 0.5~2.0)" and "Late MCI (L-MCI, CDR-SB 2.5~4.0)" subgroups based on the CDR-SB. All the subjects were given the K-VCIHS 60-minute Neuropsychology Protocol. The MANCOVA was conducted with a Bonferroni correction for multiple comparisons based on z-scores of each test. **Results:** In the executive function tests, all the MCI and dementia groups showed significantly lower performances than NE. Significant differences were found between the E-VaMCI and L-VaMCI, and between the E-VaMCI and VaD, but not between the L-VaMCI and VaD, whereas there were no significant differences among the E-aMCI, L-aMCI, and AD. Also, there were no significant differences between the E-VaMCI and the E-aMCI, although the L-VaMCI and VaD showed significantly lower performances than the L-aMCI and AD, respectively. In the RCFT copy test, there was no group difference among the NE, E-aMCI and L-aMCI, although significant group differences were found between the NE and E-VaMCI and between the E-VaMCI and VaD. The L-VaMCI and VaD showed significantly lower performances than the L-aMCI and AD, respectively. In the S-K-BNT, the L-VaMCI and VaD showed significantly lower performance than the E-VaMCI, whereas there was not a significant difference among the E-aMCI, L-aMCI, and AD. There were no group differences between the E-VaMCI and E-aMCI, between the L-VaMCI and L-aMCI, and between the VaD and AD in the S-K-BNT. In the memory tests, all patient groups showed lower performances than the NE. The AD showed significantly lower performance than E-aMCI in the delayed recall, whereas there was no difference between the L-aMCI and AD. Although there was no difference among the E-VaMCI, L-VaMCI, and VaD in the delayed recall, the L-VaMCI showed lower performances than the E-VaMCI in the recognition. **Conclusion:** On the executive, visuospatial, language, and memory (recognition) tests of K-VCIHS-NP, the significant and consistent group differences were found in the three subgroups of VCI, not in the aMCI and AD. It suggests that the K-VCIHS-NP measures the progression of cognitive deterioration well enough for VCI. Thus, we can conclude that K-VCIHS-NP is a sensitive and optimized neuropsychological protocol for VCI patients.

P-1-77**Quantification of perivascular drainage in mouse cerebral cortex and its role in Alzheimer disease**

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Background & Objectives: The lymphatic system in the central nervous system has not been identified yet, however, researchers have investigated the presence of lymphatic system in the brain. Perivascular drainage (PVD) is the phenomenon that interstitial fluid and solutes in parenchyma are drained along vessel walls and finally into cervical lymph nodes. PVD has been assumed as a candidate of lymphatic system in the brain. Small molecules such as amyloid beta peptide are also cleared through this pathway. There is no, however, consensual method to quantify PVD or examine its direction whether toward artery or vein. Little is known about the contribution of PVD on neurodegenerative diseases related to accumulation of abnormal protein such as Alzheimer's disease. Here we propose a novel method and two parameters, i) uniformity index and ii) delta area above curve (Δ AAC), to quantify PVD. We also investigate the role of PVD in Alzheimer's disease mouse model (AD mouse). **Method:** FITC conjugated dextran (4kDa) was directly injected into mouse (C57BL/N), young (8~12 weeks) and aged (21~24 months), and AD mouse (APPswe/PS1 Δ E9, 21~24 months) cerebral cortex. FITC signals through cranial window were recorded with wide-field CCD camera (Optical imaging Ltd, Photonfocus MV1-D1312-160CL). For control 0.5 % of agarose gel as diffusion medium was used. The images were analyzed within 2.6 mm diameter of circle mask. The circle mask was divided into eight equivalent fan-shaped pieces. Each piece was classified into artery-dominant, vein-dominant, and mixed piece based on vessel distribution inside of it. Injected FITC- dextran mainly moves radially from injected site by diffusion, however, PVD changes this uniformed radial transport. Therefore, non- uniformed radial distribution within the mask would reflect the flow direction and degree of PVD. We calculated 'uniformity index' that reflects the non-uniform diffusion of the fluorescent signal. As PVD is the additional force affecting fluorescent flow, the change of FITC intensity in artery-dominant piece is decreased faster compared with vein-dominant piece. Thus, difference of area above curve (Δ AAC) of fluorescence signal between artery-dominant and vein-dominant area also represents PVD function thus used as a parameter for quantifying PVD. **Results:** In these experiments, we observed that injected FITC-dextran signal moved more preferentially into artery-dominant piece than vein- dominant piece. The fluorescent signal moved faster toward the artery-dominant piece. Uniformity index decayed exponentially during the imaging session and the averaged uniformity index was significantly higher than agarose (simple diffusion) case. In aged and Alzheimer mice, the uniformity indices were relatively steady. The Δ AAC value in artery-dominant piece was significantly higher than vein-dominant piece that would reflect the degree of PVD. In AD mouse, the Δ AAC is significantly decreased compared with normal mouse. **Conclusion:** We observed non-uniformed radial movement of FITC-dextran in mouse cerebral cortex and the movement was preferentially more and faster to arterial side. This result indicates that PVD influences on the movement of the FITC-dextran and the PVD direction is mainly toward artery. We also observed accumulation of the dye along the arterial wall but not venous wall. The uniformity index reflecting the contribution of PVD thus can be used as a quantitative parameter of PVD. Δ AAC calculated from the intensity change curve between artery-dominant and vein dominant piece also reflects the amount of drainage. We found that uniformity index and Δ AAC were decreased in AD mouse and aged mouse indicating decreased PVD. These parameters can be used in other brain diseases or states that may affecting PVD.

P-1-78**A Case of Creutzfeldt-Jakob disease as progressive nonfluent aphasia**

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Background & Significance: Creutzfeldt-Jakob disease (CJD) is rare, fatal neuro-degenerative disease and the most frequent of the human prion diseases, with various clinical presentations including dementia, cerebellar syndromes, visuospatial disturbance, and akinetic mutism. The most frequently observed symptoms in CJD are cognitive and behavioral. Of the cognitive symptoms, memory loss is most frequent, followed by aphasia and frontal, executive dysfunction. We report a patient who presented with a circumscribed language disturbance consistent with progressive nonfluent aphasia, in whom rapid progression and subsequent radiologic and cerebrospinal fluid(CSF) study confirmed a diagnosis of CJD. **Case:** A 72-year-old right handed woman presented with 1 year history of word finding difficulties. She reported a progression, with increasingly halting speech and marked word-finding difficulties, although she understanding appeared entirely intact. She had no difficulty in performing social functions. On neurologic examination, she was alert, and did not show dysarthria, cerebellar dysfunction, motor and sensory deficits or myoclonic features. Magnetic resonance imaging (MRI) findings revealed hyperintensity of both cerebral hemisphere in diffusion-weighted imaging, T2 Weighted sequences and Fluid-attenuated inversion-recovery images. CSF examination revealed no pleocytosis but showed positive 14-3-3 protein by Western blot method. The patient attended a review 2 months after the initial assessment. Her condition had deteriorated markedly to the extent that she could produce no intelligible speech. She developed balance problems and was unable to walk unaided. And he was observed myoclonus. Based on clinical manifestations and para-clinic findings, she was diagnosed with probable CJD. Her condition continued to deteriorate, and she was lost to follow up after 5months. **Conclusions or Comments:** Progressive nonfluent aphasia is diagnosed one of three clinical syndromes associated with frontotemporal lobar degeneration. Our patient showed frontotemporal cortical signal changes on MRI scans, which could explain her progressive nonfluent aphasia. It is the case that a patient who has symptom of progressive nonfluent aphasia is finally diagnosed ad probable CJD.

P-1-79**Effects of apolipoprotein E4 on progression of amyloid and cortical thinning in amnesic mild cognitive impairment patients: a three-year longitudinal study**Yeo Jin KIM¹, Jin Ju YANG², Jin San LEE¹, Juyoun LEE¹, Young Kyoung JANG¹, Sung Tae KIM³, Jong Min LEE², Jae-Hong LEE⁴, Jae Seung KIM⁵, Duk L. NA¹, Sang Won SEO¹, Hee Jin KIM¹

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Background & Objectives: Apolipoprotein E4 (APOE4) is a genetic risk factor for developing Alzheimer's disease (AD). However, the effects of APOE4 on mild cognitive impairment were less clear. We investigated whether APOE4 affects the progression of amyloid burden and cortical thinning using [11C] Pittsburgh compound-B (PiB) PET and structural MRI in patients with amnesic vascular mild cognitive impairment (aMCI). **Method:** We prospectively recruited 45 aMCI patients (19 APOE4 carriers, 26 non-carriers) who under-

went PiB-PET and brain MRI at baseline. They were annually followed up with brain MRI for 3 years and underwent second PiB-PET with a mean interval of 31.2 months. Amyloid burden was measured by PiB-PET and cortical thickness was measured by brain MRI. To evaluate whether APOE4 affect the progression of amyloid burden and cortical thinning, linear mixed model was performed after controlling for age and gender. **Results:** At baseline, amyloid was more accumulated in APOE4 carrier aMCI ($p < 0.001$). However, during 3 years of follow-up, there was no difference in progression of amyloid burden between APOE4 carrier and non-carrier in aMCI patients ($p = 0.952$). At baseline, there was no significant difference in cortical thickness between APOE4 carrier and non-carrier in aMCI patients. However, over the course of 3 years, APOE4 carriers showed rapid cortical thinning in the bilateral dorso-lateral frontal, lateral temporo-parietal, medial temporal and right precuneus areas, compared to APOE4 non-carriers. **Conclusion:** Our findings suggest that in aMCI patients, APOE4 affects the rate of cortical thinning but not on the rate of amyloid accumulation over the course of 3 years.

P-1-80

A study of questionnaires about the dementia awareness and relative factors in community dwelling healthy adults

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Background & Objectives: Republic of Korea is expected to enter a super-aged society, and increase proportion of the elderly and dementia patients. As a consequence, early detection, diagnosis and treatment of dementia are on the rise as significant medical issues. But the general population have insufficient knowledge on dementia, that leads to delay the early detection, diagnosis and treatment. The aim of this study is to investigate the knowledge of dementia and factors related to the disease knowledge in general population. **Method:** We conducted a survey for 716 people in a local community. Survey was composed of questions for the level of interest, comprehension and knowledge of dementia. And then we analyzed the results by their characters such as gender, age, education level, presence of employment and spouses. **Results:** The ratio of at least interested people in dementia (93.3%, 668 people) and at least comprehensive people (91.1%, 652 people) is similar. But the ratio of very interest people (39.0%, 279 people) and good comprehensive people (10.1%, 72 people) is low. Average scores of correct answer is 8.77 (0-13 scores). They have relatively accurate knowledge of basic concepts and symptoms of the dementia but inaccurate knowledge of treatment and management after the diagnosis. Analyzed the results and their characteristics, they have significantly high knowledge level that they have more years of education, spouses and more interested in dementia ($p < 0.01$). **Conclusion:** The present study performed general population aged between 50 to 75 years old and investigated the individual's knowledge of dementia and factors related to the disease knowledge. In general, increasing knowledge of the specific diseases is promoted the disease-related behaviors such as comprehension, prevention, management and help-seeking. In other words, increasing knowledge of dementia, we will improve the early detection and treatment rate and reduce the socio-economic cost of dementia.

P-1-81

The factors related with acquisition of grades in the long-term care service in demented patients

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Background & Objectives: In Korea, the long-term care service was started in 2007 for improvement of life quality in the older and their caregivers. The long-term care service costs large amount of health insurance budget. Therefore, we need to check whether it is used in proper situations. In addition, the equality of the distribution of the service is important. We studied the factors related with acquisition of grades in the long-term care service in demented patients. **Method:** The study included patients with 2072 Alzheimer's disease (AD), 1274 Alzheimer's disease with small vessel (AD with) and 620 subcortical vascular dementia (SVaD) selected from the database of the CREDOS study, a nationwide multicenter cohort study of cognitive disorders, between March 2006 and December 2013. Patients with AD met the probable criteria of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA). SVaD patients met vascular dementia of DSM-IV criteria and Erkinjuntti's criteria for SVaD. We defined "AD with" as AD with moderate subcortical ischemic change which is not sufficed to diagnosis of SVaD. We merged the CRCOD database with the data of the National Health Service. **Results:** The acquisition rates of the long-term care grades were 70.8% in SVaD patients and 57.4% in AD patients ($P < 0.0001$). In "AD with" patients, the rate was 64.6% and there was statistical significance with acquisition rate in AD ($P = 0.0017$). The times from diagnosis of dementia to acquisition of the grades were the shortest in SVaD patients and the longest in AD patients. There were statistical difference in the acquisition rate between cities and other areas ($P = 0.0054$). The education years was another factor related with the acquisition of the long-term service grades ($P = 0.0278$). **Conclusion:** SVaD patients had the higher acquisition rate in the long-term service than AD and "AD with" patients. It may reflect more severe frontal dysfunction and neurological deficit in SVaD patients. In addition, there can be inequality in accessibility for applying the long-term service care between cities and other areas. Lastly, the complexity in the process of application of the long-term service care may cause differences of the acquisition rate according to education years.

P-1-82

A case of reversible neurologic manifestations and Parkinsonism caused by vitamin B12 deficiency

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Background & Significance: An association of vitamin B12 deficiency and degenerative disorders including Parkinson's disease has been suggested. Although explanations including impaired production of monoamine or neurotoxicity from increased serum homocysteine level have been suggested, the underlying mechanism has not been elucidated. **Case:** Methods: A sixty-three year-old man was admitted to a university hospital due to an involuntary movement of limbs, gait disturbance, and cognitive impairment. Detailed history taking, neurologic examination, laboratory tests, neuropsychological evaluation, and brain imaging were performed. Results: History taking revealed that the patient's first symptom was unsteady gait that has begun 1.5 years before the admission. One year before the admission, he started to show intermittent drowsiness and confusion, and his cognitive function deteriorated slowly thereafter. Six months before the admission, he had hand tremor in both hands, and slow movements. He visited the dementia outpatient clinic of Severance hospital due to the involuntary writhing movements of both hands and body, which began a week ago. He had a history of radical total gastrectomy due to the gastric cancer 20 years ago and was a heavy alcoholic. On

neurologic examination, rigidity of both arms and bradykinesia were noticed, which were more severe in the right side. He also showed a decreased position/vibration sensation of fingers and toes, and pseudoathetosis of both hands. On walking, he could not walk by himself and showed a wide based gait with very short steps and absent arm swings. He could barely stand himself due to the sensory ataxia. His initial Mini-Mental State Examination (MMSE) score was 12. Laboratory tests revealed that his serum vitamin B12 level was lower than 50 pg/ml (normal range, 180~947), and he had anemia of megaloblastic pattern. Other laboratory tests including serum folic acid and homocysteine were within normal ranges. Brain magnetic resonance imaging (MRI) was not remarkable, but spine MRI revealed an inverted V-shaped, T2 hyper-intense lesion in the dorsal column at the C1-C6 level. Nerve conduction studies showed sensory dominant polyneuropathy. Brain 18F-FP-CIT positron emission tomography exhibited decreased dopamine transporter uptake in the left posterior putamen. We diagnosed him as a dementia, megaloblastic anemia, and subacute combined degeneration due to vitamin B12 deficiency and initiated the treatment with intramuscular injection of actinamide (6mg q.o.d for 6 times, then 1mg q.o.d for 8 times). He was also treated with levodopa. One month after the treatment, his serum vitamin B12 level was more than 1500pg/ml. He could walk independently and his MMSE score increased to 22, but neuropsychological tests performed still showed impairments in all cognitive domains. **Conclusions or Comments:** Parkinsonism in this case could be caused by the depletion of dopamine based on the result of dopamine transporter imaging. Considering that the level of homocysteine was normal, our case supports the possibility that vitamin B12 deficiency could lead to Parkinsonism by the depletion of dopamine, which is independent of serum homocysteine.

P-1-83

Survival in patients with dementia who are in the institutions

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Background & Objectives: From adoption of the long-term service care, the institutionalization of the demented patients was dramatically increased. In this study, we study the survival and related factors in patients with dementia in the institutions. **Method:** The study included patients with 2072 Alzheimer's disease (AD), 1274 Alzheimer's disease with small vessel (AD with) and 620 subcortical vascular dementia (SVaD) selected from the database of the CREDOS study, a nationwide multicenter cohort study of cognitive disorders, between March 2006 and December 2013. Patients with AD met the probable criteria of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA). The Institutional Review Board of Samsung Medical Center approved this study. SVaD patients met vascular dementia of DSM-IV criteria and Erkinjuntti's criteria for SVaD. We defined "AD with" as AD with moderate subcortical ischemic change which is not sufficed to diagnosis of SVaD. We found out the date and state of institutionalization from the data of the long-term service. **Results:** The poor survival factors in the demented patients living in the institution were late age onset ($P < 0.0001$), men ($P < 0.0001$), other areas than cities ($P = 0.0246$), and the presence of DM ($P = 0.0155$). **Conclusion:** The known poor survival factors in demented patients were subcortical vascular dementia, old age, men, advanced dementia stages. In our study, the types of dementias and the dementia stages were not factors related with survival in demented patients. Interestingly, there was difference between cities and other areas in the survival. This may mean that the

institutions in other areas need to improve their services.

P-1-84

Clinical and neuropsychological differences in patients with cognitive decline of Alzheimer's type stratified by positive vs. negative amyloid PET status

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Background & Objectives: Amyloid PET imaging allows in-vivo detection of amyloid beta ($A\beta$) fibrillar plaques, a core neuropathological feature of Alzheimer's disease (AD). According to US-ADNI data, around 15% of people diagnosed with clinically probable AD represented negative PiB-PET imaging. There have been emerging efforts to define these AD phenocopies. The purpose of this study was to determine whether there are clinical and neuropsychological differences between positive vs. negative [18F]-Florbetaben amyloid PET imaging in Korean patients with AD spectrum disorders. **Method:** From February to July 2015, patients who visited the Memory and Dementia Clinic of Asan Medical Center were evaluated with structural brain MRI, detailed neuropsychological test and 18F-florbetaben amyloid PET. PET images were acquired after obtaining a consent for study participation from each subject. [18F]-Florbetaben amyloid PET were performed 90 to 110 min after intravenous injection of 300MBq florbetaben. Two neurologists (J.L. and J.H.R.) and a nuclear medicine physician (J.S.K.) reviewed PET scans according to the predefined regional cortical tracer binding (RCTB) and brain amyloid plaque load (BAPL) scoring system and final scoring was rated after consensus was reached. Standardized uptake value ratios (SUVRs) were calculated using cerebellar cortex as a reference region. Among all patients who underwent [18F]-florbetaben PET, a total of 43 patients were analyzed whose clinical diagnosis was an AD spectrum disorder: single or multiple domain amnesic mild cognitive impairment (MCI) and AD. **Results:** Of 43 patients, 28 showed a positive amyloid PET scan and 15 revealed a negative scan. The amyloid positive group consisted of 5 single-domain amnesic MCI (17.9%), 6 multiple-domain amnesic MCI (21.4%) and 17 AD patients (60.7%). On the other hand, the amyloid negative group comprised of 3 single-domain amnesic MCI (20.0%), 8 multiple-domain amnesic MCI (53.3%) and 4 AD patients (26.7%). All demographic features, including age, gender, education level, onset age, disease duration, body mass index (BMI), and vascular risk factors, were not significantly different between the two groups. ApoE4 carrier status was not different between the groups. Severity of hippocampal atrophy assessed by Scheltens' visual rating scale was not different, either. However, K-MMSE, CDR-SOB (clinical dementia rating, sum of boxes) and K-DSQ (Korean-Dementia Screening Questionnaire) represented lower scores in the amyloid positive group compared to the amyloid negative group ($p = 0.027$, 0.002 , and 0.021 , respectively). CDR global score, GDS (Global Deterioration Scale) score, NPI (Neuropsychiatric inventory) total score and GDepS (Geriatric Depression Scale) score did not show statistically significant difference between the groups. The amyloid positive group showed more severe impairment in RCFT (Rey Complex Figure Test) copy ($p = 0.006$) and recall tests ($p < 0.001$). **Conclusion:** Amyloid PET scan enables assumptive diagnosis of preclinical AD and probable AD dementia by in vivo assessment of amyloid pathology in the brain. Our findings suggest that amyloid-positive AD spectrum disorders have more impairment in overall cognitive impairment as well as non-verbal memory and visuospatial function. Detailed neuropsychological test results from additional participants and discriminative features reflecting amyloid-negative AD will be presented.

P-1-85**Serial magnetic resonance imaging changes in a patient with Gerstmann-Sträussler-Scheinker Syndrome (P102L)**

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Background & Significance: Gerstmann-Sträussler-Scheinker Syndrome (GSS) is a rare inherited prion disease characterized by midlife onset and slowly progression of cerebellar ataxia and dementia. We describe serial MRI findings of a patient with GSS with a mutation in the prion protein gene at codon 102 (Proline to Leucine) in a Korean family. **Case:** A 31-year-old woman who was born and lived in Korea began to experience gait unsteadiness 5 months before admission. Neurological examination revealed slow saccadic eye movement and bilateral limb ataxia. Brain MRI including DWI showed no abnormality. Her family members (mother, aunt, grandfather, and great grandfather) had a history of progressive gait disturbance and becoming bedridden that had been left undiagnosed. Over a period of next 3 months, the patient developed insomnia, voiding difficulty, and cognitive impairment. DWI and FLAIR imaging of the brain showed hyper-intense signal changes in the cerebral cortex, predominantly in the parieto-temporal regions, caudate head, and ventral region of putamen. CSF 14-3-3 protein was found to be weakly positive. EEG showed diffuse delta waves without periodic synchronous discharges. Analysis of the PRNP gene showed a proline-to-leucine substitution at codon 102. The neurological status had worsened progressively and she had reached akinetic mutism 9 months after onset. Follow-up brain MRI with an interval of 10 months showed areas of spreading diffusion restriction in entire cerebral cortex and marked aggravation of atrophy of cerebral and cerebellar hemispheres, and brainstem. **Conclusions or Comments:** It has been reported that MRI scans may be normal during the early stage of GSS. In this case, serial MRIs show the progressive extension of the high signal intensity on DWI in accordance with the neurological status, which has been attributed to the severity of spongiform degeneration and to gliosis, and marked brain atrophy following the DWI changes relatively for a short period of time.

P-1-86**Amyloid beta-weighted cortical thickness : five distinctive relationships between Amyloid beta and cortical thinning in Alzheimer's disease**Chan-Mi KIM¹, Ji Hye HWANG¹, Jong-Min LEE², Jae-Hong LEE¹, Jee Hoon ROH¹¹Department of Neurology, Asan Medical Center, University of Ulsan College of Medicine,²Department of Biomedical Engineering, Hanyang University

Background & Objectives: Alzheimer's disease (AD) is the most common neurodegenerative disorder pathologically characterized by amyloid beta (A β) plaques and neurofibrillary tangles. Although A β has been known as primary factor in AD progression, the relationships between A β deposition and cortical thinning remained somewhat controversial. In this study, we proposed a novel approach to investigate whether cortical thinning patterns can be differentiated in AD based on existence of amyloid plaque deposition. We hypothesized that there are cortical areas that have prominent changes associated with A β deposition and areas that are relatively reluctant to A β pathology where pathologies other than A β (such as tau) are predominant. **Method:** We investigated a total of 21 AD, 56 MCI (mild cognitive impairment), and 18 NC (normal control) subjects from the ADNI database with six AD subjects from Asan Medical Center who completed MRI and Pittsburgh Compound B (PiB) PET. All MRI data sets were processed using a standard Montreal Neurological Institute CIVET pipeline to measure cortical thickness. Each individual PiB-PET image was co-registered into the corresponding native MR image

and then scaled using a mean value in the cerebellar cortex to create a standardized uptake value ratio (SUVR) image. Surface-based PiB-SUVR values were calculated using the mean of interpolated values along the column line between the outer GM/CSF and inner GM/WM vertices. We applied partial volume correction by GM probability maps derived from corresponding native MR images. Then, we obtain A β -weighted cortical thickness values, each normalized z-scores of cortical thinning value was multiplied by corresponding A β -weight value. **Results:** A β -weighted cortical thickness patterns were highlighted in some cortical regions, such as posterior cingulate cortex and inferior parietal gyri, which were known to be A β deposition-associated cortical regions. In addition, the A β -weighted cortical thickness patterns were de-emphasized in some medial temporal lobes which were known as relatively independent from A β deposition. We observed more significant group difference by A β -weighted cortical thickness values than by cortical thickness analysis only. We identified three types of relationships between cortical thinning and A β deposition, such as A β -associated cortical thinning, A β -independent cortical thinning, and A β deposition without cortical thinning. A β -associated cortical thinning patterns were further divided into three types: A β deposition before cortical thinning, A β deposition after cortical thinning, and concurrent appearance of both. **Conclusion:** In this study, we incorporated information of the amount of A β in measurement of cortical thickness by developing A β weighted cortical thickness analysis. We classified patterns of A β -weighted cortical thickness based on relationships among cortical thinning, A β uptake, and A β -weighted cortical thickness by comparing groups of AD, MCI, and NC. The A β -weighted cortical thickness patterns were prominent in cortical regions with well-known A β accumulation, such as posterior cingulate cortex and inferior parietal gyri. On the other hand, brain regions known to harbor pathologies other than A β , such as medial temporal lobes, represented less A β -weighted cortical thickness. Taken together, these findings suggest that A β -weighted cortical thickness can be used as an objective biomarker of MRI based cortical structural changes driven by A β deposition.

P-1-87**Visual rating of posterior atrophy as a marker of progression to dementia in mild cognitive impairment patients**Hang-Rai KIM¹, Young Ho PARK², Jae-Won JANG³, So Young PARK², Min Jeong WANG², Min Jae BAEK², Beom Joon KIM², Soyeon AHN⁴, SangYun KIM²¹Department of Neurology, Seoul National University Hospital, Seoul National University College of Medicine, Seoul, Korea, ²Department of Neurology, Seoul National University Bundang Hospital, Seongnam-si, Korea, ³Department of Neurology, Kangwon National University Hospital, Chuncheon-si, Korea, ⁴Medical Research Collaborating Center, Medical Research Collaborating Center, Seoul National University Bundang Hospital, Seongnam-si, Korea

Background & Objectives: Alzheimer's disease (AD), which is the main cause of dementia in elderly has a symptomatic pre-dementia phases known as a mild cognitive impairment (MCI). It is known that MCI patients with a medial temporal lobe atrophy (MTA) have a higher risk of progression to dementia. However, substantial MCI patients without MTA still progress to dementia. Previous studies have reported that beside the medial temporal lobe, the parietal lobes are also involved in AD which is known as a posterior atrophy (PA). We evaluated the predictive value of PA for MCI progression to dementia and assessed whether PA provides independent value in addition to MTA. **Method:** This was a retrospective cohort study of the patients who visited Clinical Neuroscience Center of Seoul National University Bundang Hospital. A total of 148 MCI patients were studied and they all had brain MRI performed within 1 year before the diagnosis. They were followed up for up to 3 years for detecting their progression to dementia. MTA and PA were assessed using a visual rating scale, developed by Schelten et al and Koedam et al in

which higher scale indicates more severe atrophy. Visual rating of the parietal lobe included the assessment of widening of the posterior cingulate and parieto-occipital sulcus as well as the atrophy of the precuneus. We performed the cox regression analysis to examine the hazard ratio (HR) of MTA and PA for the MCI progression to dementia. Multivariate model were performed with adjustment for covariates which were clinically and statistically related. In order to examine and compare the discriminative abilities of MTA and PA, we used the Harrell's c-index of the multivariate model using PA and the model using MTA. **Results:** During the follow-up period, 47 patients had progressed to dementia. In the univariate cox regression analysis, MTA and PA were associated with the hazard of progression to dementia but no other variables were statistically significant. (MTA; 5.515 (95% confidence interval, CI 2.330-13.057), PA; 2.781 (95% CI 1.497-5.167)) Multivariate Cox regression analysis showed that no covariates changed the significance of the HR of MTA and PA. The HR and the 95% CI of MTA, PA, Age, APOE ε4, prior stroke were 4.446 (1.763-11.208), 2.131 (1.086-4.183), 0.984 (0.919-1.054), 1.125 (0.596-2.124) and 0.996 (0.234-4.237), respectively. The Harrell's c-index and 95% CI for PA model and MTA model were 0.662 (0.562 - 0.761) and 0.703 (0.626 - 0.780), respectively and difference between two models were statistically insignificant (p value = 0.385). **Conclusion:** In this study, we demonstrated that PA is an independent marker for predicting MCI progression to dementia. Assessing PA can be as helpful as MTA in predicting the progression to dementia in patients with MCI.

P-1-88

Distinctive cognitive trajectories related to amyloid and cerebrovascular disease in patients with mild cognitive impairment

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Background & Objectives: Amyloid and cerebral small vessel disease are the two major causes of cognitive impairment in the elderly. It is well known that amyloid has great impact on further cortical thinning or cognitive decline in AD. However, longitudinal changes of amyloid burden related to cerebral small vessel disease changes or their clinical impacts remains unknown. We investigated the relationships between longitudinal measurements of amyloid, lacune, and cortical thickness, and their effects on cognitive decline, over 3 years in mild cognitive impairment (MCI) patients. **Method:** We prospectively recruited 117 MCI patients (45 amnesic MCI (aMCI) and 72 subcortical vascular MCI (svMCI)) who underwent neuropsychological tests, brain MRI, and PiB-PET at baseline. They were annually followed up with neuropsychological test and brain MRI for 3 years. 83 (70.9%) patients (33 aMCI and 50 svMCI) completed second PiB PET with mean interval of 32.1 months. We used linear mixed effects models to evaluate the associations between longitudinal measurements. **Results:** Longitudinal measurements of PiB SUVR was negatively associated with longitudinal measurements of lacune number (beta=-1.74, p=0.034). Increase in PiB SUVR contributed to cortical atrophy in the medial temporal and precuneus regions, whereas increase in lacune number contributed to cortical atrophy in the medial frontal and anterior cingulate regions. Increase in PiB SUVR predicted cognitive decline in visual/ver-

bal memory, and general cognitive function whereas increase in lacune number predicted cognitive decline in attention and frontal-executive function. In addition, PiB SUVR and lacune predicted cognitive decline in memory and frontal executive function, respectively, regardless of cortical atrophy. **Conclusion:** We found that progression of amyloid and lacune each independently contributed to cortical atrophy in specific regions which further predicted cognitive decline in corresponding domains. We suggest that trajectories of cognitive decline related to amyloid and lacune have different paths and that they are independent to one another.

P-1-89

Dementia progression in anatomical subtypes of Alzheimer's Disease: malignant progression in parietal dominant atrophy subtype regardless of onset age

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Background & Objectives: Recently, we reported that the earlier stages of Alzheimer's disease dementia (AD) can be categorized into three anatomical subtypes, using cluster analyses based on cortical atrophy patterns: medial temporal-dominant (MT) subtype, parietal-dominant (P) subtype and diffuse atrophy (D) subtype. This study aimed to compare the rate of longitudinal cognitive decline in each subtype. **Method:** Among the 152 patients in the previous study, 100 AD patients who underwent follow-up neuropsychological tests were evaluated retrospectively (MT, n=36; P, n=20; D, n=44). Linear mixed model or generalized estimation equation analysis was performed to compare the changes of neuropsychological test scores over four years. **Results:** Compared with other two subtypes, the P subtype exhibited the greatest longitudinal decrease in mini-mental status examination (MMSE), clinical dementia rating (CDR), attention, language, visuospatial, and frontal-executive function. Among the six memory subtests (free recall, delayed recall, and recognition parts of verbal and visual memory tests), cognitive decline in delayed recall in the visual memory and immediate recall in verbal memory were significantly rapid in P subtype, while the remaining four subtests were similar to D subtype. MT and D subtype did not differ in changes of all cognitive tests. When MT and P subtype were compared in early onset AD patients, the P subtype patients still showed a remarkable disparity in their disease progression profile. **Conclusion:** P subtype patients showed the most rapid cognitive decline, while leaving the memory function relatively spared when considering the aggressive progression profile of P subtype. The subtyping proposed by our study may play a crucial role in distinguishing EOAD with more rapid (P subtype) or slower cognitive decline (MT subtype).

P-1-90

Atypical Wernicke's encephalopathy involving frontal lobe during parenteral nutrition

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Background & Significance: Wernicke's encephalopathy is an acute neurologic deterioration due to reversible brain lesion caused by thiamine deficiency. Most of the affected patients are thiamine-depleted alcoholics usually involving medial thalamic, mammillary bodies, periaqueductal area. However, atypically involved lesions in Wernicke's encephalopathy, such as cerebellum, cere-

bral cortex, are reported especially in non-alcoholic patient. We report a case of atypical Wernicke's encephalopathy involving bilateral frontal lobes in patient treated in intensive care unit with prolonged parenteral feeding. **Case:** A 54-year-old female was admitted to urology department due to septic emphysematous pyelonephritis. On her medical history, she had advanced stage of cervical cancer. Due to severity of the disease, she was admitted in intensive care unit having parenteral nutrition. After two months of parenteral nutrition, she was consulted to neurology department about altered mental status which was continued for several days. Patient was alert but abulic and neurologic examination revealed no other neurologic deficit. Brain MRI showed increased signal intensity in both frontal cortex, medial thalamic, periamygdaloid body, periaqueductal gray matter, dorsal medulla on FLAIR image. Taken together with MRI findings and patient's septic condition demanding higher thiamine requirement and prolonged parenteral nutrition, we diagnosed patient with Wernicke's encephalopathy with cortical involvement. Although massive thiamine was supplied, she was expired due to aggravation of general condition. **Conclusions or Comments:** This is the rare case of showing atypical involvement of bilateral frontal lobes in Wernicke's encephalopathy. Wernicke's encephalopathy associated with cortical involvement is known to have worse prognosis than the others due to potentially irreversible brain damage. Clinicians should be aware of atypical pattern of Wernicke's encephalopathy which may involve bilateral cortical involvement to evaluate the disease and prognosis.

P-1-91

The pattern of brain iron accumulation of vascular dementia and Alzheimer's dementia using quantitative susceptibility mapping imaging

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Background & Objectives: Emerging evidence suggests that the excessive accumulation of iron in subcortical and deep gray matter has been related with dementia. However, the presence and pattern of iron accumulation in vascular dementia (VaD) and Alzheimer's disease (AD) are rarely investigated. We examine and compare the pattern and presence of brain iron accumulation of VaD and AD using quantitative susceptibility mapping (QSM). **Method:** Twelve patients with VaD, twenty-seven patients with AD and eighteen control subjects were included in the study. Four regions of interest were drawn manually on the QSM images for each case: globus pallidus, putamen, caudate nucleus, and pulvinar nucleus of thalamus. Data were assessed and reconstructed by using the MEDIA, and analyzed using MIPAV. Comparisons of patient demographics, and iron concentration among the VaD, AD and control subjects were assessed by analysis of variance with post hoc Bonferroni correction. The relation of age and cognitive state with susceptibility values were analyzed using simple and partial correlation analysis, respectively. **Results:** In VaD and AD, overall susceptibility value was higher than control subjects. A significant difference of susceptibility values were found in the putamen and caudate nucleus. ($p < 0.001$ and $p = 0.002$, respectively) However, susceptibility value was not different between VaD and AD. Age and cognitive severity were not related with susceptibility values in both VaD and AD groups. **Conclusion:** We found out the increased iron deposition at putamen and caudate nucleus in VaD and AD patients, and iron accumulation was not related with age or cognitive severity.

P-1-92

In vivo evaluation of neurofibrillary tangles in Alzheimer's disease using [18F]THK5351 PET

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Background & Objectives: Neurofibrillary tangles are known as the pathologic hallmark of Alzheimer's disease (AD) along with amyloid plaques and are composed of paired helical filaments that result from the abnormal aggregation of tau protein. Recently, a novel tau PET tracer, [18F]THK5351 was developed. The aim of this study was to evaluate the clinical usefulness of [18F]THK5351 PET tracer. **Method:** We included the 12 patients with AD dementia, 5 patients with amnesic mild cognitive impairment (aMCI) and 7 age-matched control with normal cognition (NC). All participants underwent [18F]THK5351 PET, [18F]FDG PET, 3D magnetic resonance imaging and detailed neuropsychological tests. Standard uptake value ratios at 50-70 minutes and 40-60 minutes post-injection were calculated for [18F]THK5351 PET and [18F]FDG PET respectively. Reference region was cerebellum crus 1 and 2 in [18F]THK5351 PET and pons in [18F]FDG PET respectively. **Results:** Global tau retention was significantly greater in AD compared to NC. The regions with higher tau uptake in AD compared to NC were frontal, lateral temporal, mesial temporal posterior cingulate, and striatum. The patients with amnesic MCI showed higher uptake in the lateral temporal lobe compared to NC. The global tau retentions were correlated with the glucose hypometabolism. Tau uptake was correlated with cognitive functions, as well. **Conclusion:** [18F]THK5351 PET is the useful technique for in vivo evaluation of neurofibrillary tangles of Alzheimer's disease and would be a valuable imaging biomarker for disease stages.

P-1-93

Memory complaints in subjective cognitive impairment, mild cognitive impairment and Alzheimer's disease

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Background & Objectives: Memory complaints are a frequent phenomenon in elderly people, which can lead to opportunistic help-seeking behaviour. The aim of this study was to compare whether different aspects of memory complaints (i.e., prospective versus retrospective complaints) differed between individuals with subjective cognitive impairment (SCI), amnesic mild cognitive impairment (aMCI), and mild Alzheimer's disease (AD). **Method:** The study included a total of 115 participants (mean age: 68.82 ± 8.83 years) with SCI ($n = 34$), aMCI ($n = 46$), and mild AD ($n = 35$). Memory complaints were assessed using the Prospective and Retrospective Memory Questionnaire (PRMQ) consisting of 16 items which describe everyday memory failure of both prospective memory (PM) and retrospective memory (RM). Participants with aMCI and mild AD also completed informant-rated version of the PRMQ. All participants completed detailed neuropsychological tests. **Results:** Memory complaints (measured as PRMQ total score) were equivalent across the three groups. Prospective complaints (by PRMQ-PM subscore) of memory complaints also showed similar results among the three groups. In regard to retrospective memory complaints (by PRMQ-RM subscore), the subjects with aMCI showed higher complaints than those with SCI, while being no differences between aMCI and mild AD. The individuals with mild AD had higher informant-reported memory complaints (by informant-rated PRMQ total score) than those with aMCI. **Conclusion:** The retrospective complaints of subjective memory complaints may be helpful in discriminating between SCI and aMCI. Both self-reported prospective and retrospective memory complaints is of limited value in differentiating aMCI from mild AD, while informant-reported memory complaints being helpful.

P-1-94**Hashimoto's encephalopathy presenting with dementia and relapsing mental change**

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Background & Significance: Hashimoto's encephalopathy (HE) is a rare, still not well understood, autoimmune disease with neurological and psychiatric manifestations. And elevated titers of antithyroid antibodies in serum and cerebrospinal fluid (CSF) as a hallmark of the disease. Disease course can be acute, subacute, chronic or relapsing-remitting. Clinical importance of the disease is high because diagnosis is difficult, and the disease is treatable if diagnosed successfully. We present a case of hashimoto's encephalopathy presenting with dementia and relapsing mental change. **Case:** A 80-year-old man with slowly progressive memory loss during last 6 months, disorientation in space and time, lack of concentration and agitation was admitted to our hospital after relapsing altered mentality. 5 months ago, he diagnosed dementia at other hospital due to memory loss. 3 months ago, he visited ER due to altered mentality and admitted to that hospital. After 3days he could talk but general weakness was remained but didn't find the cause. During 3months, he had 3 similar episodes and admitted to department of nephrology of our hospital due to acute kidney injury with altered mentality. Past medical history revealed only atrial fibrillation. He had no focal motor, sensory, cranial nerve, or cerebellar abnormalities. Routine blood test shows elevated serum BUN, Cr, blood ammonia. CBC shows no abnormality. Thyroid status showed hypothyroidism with high titers of thyroglobulin antibody and microsomal antibody in serum (>3000 IU/ml; normal<60 IU/ml and 1224 IU/ml, normal<60 IU/ml; respectively). Markers of autoimmune diseases were negative, including normal antinuclear antibody, rheumatoid factor level, anti-double stranded DNA antibody, anti-SS-A antibody, anti-SS-B antibody, anticardiolipin antibody and myeloperoxidase anti-neutrophil cytoplasmic antibody. No clotting abnormalities were detected. There was no CSF evidence of bacterial or viral infection, as determined by culture or molecular analysis. The EEG showed diffuse slowing without triphasic potentials and cranial MRI showed no abnormality. He was treated conservatively and thyroid replacement was done. But he was drowsy. Do he transferred to neurologic department and was treated with oral glucocorticoid. In the Mini Mental Test he reached 7 of 30 points. After treatment he reached 11of 30points. **Conclusions or Comments:** Hashimoto's encephalopathy is rare and has various neurologic symptoms. It is still debatable whether this is a specific syndrome seen in patients with Hashimoto's thyroiditis or the coincidence of a rare neurologic condition with a common endocrinologic disease. When neurologists are faced with a patient with a possible diagnosis of acute or subacute encephalopathy and no other specific cause is found, an autoimmune process is considered in the differential diagnosis.

P-1-95**The steadiness of neuronal integrity in the recurrent attack of transient global amnesia**

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Background & Objectives: The imaging study of patients with multiple episodes of transient global amnesia (TGA) to infer the long-term outcome is

rare. We compared the neuronal integrity of the memory network in patients with repeated episodes of TGA with those who experienced a single attack. **Method:** Seven patients who were precisely defined as having recurrent TGA and 14 age- and sex-matched control subjects who had only a single episode of TGA participated in the study. Diffusion tensor images from both groups were assessed and analyzed using tract-based spatial statistics with a nonlinear registration algorithm. **Results:** No significant differences were found in vascular factors, precipitating factors, or the mean duration of amnesic episodes between the two groups. The fractional anisotropy and mean diffusivity values did not differ for any lesions in the memory pathway. **Conclusion:** No disruptions in the neuronal integrity of the memory pathway were observed in patients with recurrent TGA attacks, refuting the hypothesis that TGA patients present predisposing weakness of the memory network and suggesting that repeated hippocampal lesions associated with TGA do not affect the microstructure of the brain. The stability of neuronal integrity suggests that recurrent TGA is a relatively benign entity.

P-1-96**Predictors of Institutionalization in longitudinal follow-up of Patients with Alzheimer Disease**

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Background & Objectives: Dementia, of which Alzheimer disease is the most prevalent type, is the most frequent reason for the institutionalization of the elderly. Knowing the estimated time until institutionalization is important for decision making of patient or caregivers, and also it allows clinicians to offer appropriate care in the course of the disease progression. Many studies have reported predictors of institutionalization in patients with Alzheimer disease or dementia, but only few studies have evaluated predictors that measured long-term change. In this study, we evaluated predictors of institutionalization in patients with Alzheimer disease, focusing on variables regarding the rate of change in cognition and neuropsychiatric symptoms. **Method:** Among the patient with Alzheimer disease enrolled in CREDOS study between November 2005 and December 2013, we selected only those who underwent clinical evaluation (CDR-SB, K-MMSE, NPI) multiple times to incorporate variables regarding the rate of change in cognition and neuropsychiatric symptoms. Because public long-term care insurance (LTCI) has been implemented since July 2008 in South Korea, to utilize data from this program we excluded the patients who were enrolled in CREDOS study before July 2008. Those who have already been institutionalized at the time of enrollment and those who lacked enough information were also excluded. Then we separated these patients into institutionalized group and not-institutionalized group, and examined demographic and clinical variables that predict institutionalization. **Results:** Finally 816 patients were examined. Among these patients, 130 (16%) patients were institutionalized at the time of investigation. Patients who were institutionalized had significantly higher CDR-SB difference from baseline (1.9 vs 1.6, $p<0.0001$), higher K-MMSE difference from baseline (-1.8 vs -1.6, $p=0.0024$), had higher usage of antipsychotics (40.8% vs 25.2%, $p=0.0003$) and Memantine (33.9% vs 25.4%, $p=0.0451$). Institutionalized group had significantly higher medication possession ratio (MPR) of antipsychotics than not-institutionalized group (0.4 vs 0.2, $p=0.0013$). Sociodemographic factors such as age, gender, education, and comorbidities (diabetes mellitus, hypertension, cardiovascular disease, stroke) showed no difference between groups. NPI difference from baseline and benzodiazepine use also showed no sig-

nificant difference. In Cox proportional hazard model, higher CDR-SB difference from baseline (HR 1.15, $p=0.0003$) and higher MPR of antipsychotics (HR 1.89, $p=0.0054$) predicted a shorter time until institutionalization. **Conclusion:** Aggravation in dementia severity (higher CDR-SB difference from baseline) and more frequent use of antipsychotics predicts earlier institutionalization in patients with Alzheimer disease. Longitudinal assessment of clinical symptoms may provide the best way of predicting which patients will be institutionalized.

P-1-97

Spontaneous ventriculostomy in a patient with normal pressure hydrocephalus

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Background & Significance: Spontaneous ventriculostomy (SV) is a rare condition that results from spontaneous rupture of the ventricular wall and pia mater connecting the ventricular system directly to the subarachnoid space. Previously reported cases were noted to have occurred secondary to obstructive hydrocephalus caused by neoplastic disease or by benign stenosis of the aqueduct, and occasionally congenital stenosis of the foramen of Monro. We first report a case of SV that were discovered by magnetic resonance imaging (MRI) in a patient with non-obstructive hydrocephalus. **Case:** A 48-year-old man with a known normal pressure hydrocephalus (NPH) was admitted to evaluate for worsening headache. Two years earlier, he had developed lower limb weakness, and gait disturbance and admitted to the department of neurology for evaluation. The neurologic examination had been normal. Brain MRI had shown non-obstructive hydrocephalus. cerebrospinal fluid (CSF) drainage had been done and his gait had improved. He could had walked independently. Then he had been diagnosed NPH. During follow-up, he was hospitalized once more for CSF drainage due to progressively sustained headache. The patient complained of worsening headache, therefore a new evaluation of the hydrocephalus was considered. Brain MRI performed demonstrated an improved ventriculomegaly, cerebral aqueductal dilation and interstitial edema. Flow-sensitive phase-contrast cine MR images revealed CSF flow through the floor of the third ventricle between the tuber cinereum and the mammillary bodies connecting the ventricular system with the prepontine cistern. Because of the fact that symptoms had improved and the spontaneous ventriculostomy was patent, no shunting procedure was deemed necessary. **Conclusions or Comments:** In this report, we describe a case involving the patient with normal pressure hydrocephalus who developed a spontaneous third ventriculostomy. Although this condition is rare, clinicians should be aware of the phenomenon because it may, as in this particular case, obviate the need for a CSF diversion procedure.

P-1-98

Different normal EEG variation between TGA patients and normal healthy group

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Background & Objectives: Transient global amnesia(TGA) is a sudden onset of anterograde amnesia represented by repetitive questioning, impaired to register new memories, lasting up to 24hours. It is syndrome caused by several etiologies like seizure, stroke and even unknown etiology. Previous reports showed electroencephalographic(EEG) abnormalities in TGA. However, many TGA patients have normal EEG. We analyzed these EEG in patients with TGA. **Method:** Between January 2010 and March 2014, those TGA patients

who came to Chung-Ang university hospital were included the research. Patients with abnormal MRI and EEG were excluded. Finally, 35 patients who suspected TGA had been analyzed their EEG with normal healthy control. Normal healthy control was recruited from community, who had no past medical history and matched with age and sex. Alpha waves are extracted from EEG and analyzed using sLORETA, which is software for a digitalized EEG analyzing tool. **Results:** The EEGs of 33 TGA patients were analyzed by sLORETA with normal control. There is no specific age- related differentiation in both TGA patients and normal groups. However, according to sLORETA brain mapping result, EEGs of the patients showed more left dominance comparing to control. **Conclusion:** Computer based EEG analysis discloses that the TGA patients, even if showing normal EEG by conventional interpretation, have a left dominance in alpha waves.

P-1-99

Internal structure of Computer Assessment of Memory and Cognitive Impairment (CAMCI)

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Background & Objectives: The Computer Assessment of Mild Cognitive Impairment (CAMCI) is a computer-based tool on a tablet designed to evaluate cognitive function by assessing six neuropsychological domains with twenty-one measurements of accuracy and speed. This study evaluated the internal structure of the CAMCI in an older African American population. **Method:** Ninety-six African Americans aged 60 years or older from a primary clinic in an academic medical center were recruited and evaluated using the CAMCI. The factor structure of the CAMCI was evaluated using exploratory factor analysis. **Results:** Factor analysis revealed a two-factor solution. Four measurements from executive and verbal memory loaded on one factor. One measurement from attention and two reaction times from executive and non-verbal memory loaded onto the other factor. **Conclusion:** This study contributes understanding to the psychometric properties of the CAMCI in an older African American population.

P-1-100

Supernumerary phantom limb in patient with right basal ganglia hemorrhage

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Background & Significance: The phantom limb phenomenon is described by the amputee who experiences a vivid illusion of persistent sensation of the limb which is physically lost. However this phenomenon is also found in the patient without amputated limb, who perceives the extra limb in addition to the regular set of limbs. This phenomenon is called a supernumerary phantom limb (SPL). The SPL has been reported in various neurologic disorders but its mechanism is still unknown. Herein we report a case of SPL in the patient with the right basal ganglia hemorrhage and review the previous literatures about this peculiar neurologic phenomenon. **Case:** A 78-year-old male was admitted to our hospital with sudden onset headache. He had a left side mild sensory impairment and a dense motor impairment. The brain computed tomography showed right basal ganglia hemorrhage extending medially to the thalamus with an approximate volume of 50 cc. On the second day of hospitalization, the patient started to report that besides his left paretic arm, he had another

arm protruding from the left shoulder. He could feel but could not see nor touch this phantom arm. The phantom arm persisted even with applying sensory stimulation to it. He could intentionally move his phantom arm without difficulty but the passive movement of his paretic arm did not elicit any movement of his phantom arm. His mini mental state examination showed 30 out of 30. He had no sign of seizure. We diagnosed the SPL as a complication from the right basal ganglia hemorrhage. His phantom arm persisted until 3 weeks and it gradually faded away. **Conclusions or Comments:** Through the literature search, including our case, we could find 20 well described cases of the SPL. The basal ganglia was the most prevalent lesion (9 out of 20 cases) and the most of the lesions were at right (16 out of 20 cases). The causative event was mainly the hemorrhage (13 out of 20 cases). The time to the onset of the phantom limb after the stroke ranged from the day to months. Pain was associated in 3 cases of the SPL. Although the reason why the SPL is mostly associated with right rather than left is still unclear, it is suggested that because that the right hemisphere is mainly involved in maintaining the internal representation of the body state by monitoring somatic states. The time onset varies from the day to months thus the time interval could be too short to develop a neuronal plasticity for some cases suggesting that the SPL may be caused by an acute disorder that damages pre-existing neuronal networks. There are some hypothesis about the mechanism underlying the SPL. It is suggested that the body schema is assumed to be genetically determined and it is continually modified by the integration of multiple sensory inputs as well as motor outputs. Deafferentation by cerebral lesions may cause a perceptual mismatch between the internal representation of the body and sensory inputs from ascending tracts. In our patient, the basal ganglia hemorrhage extending to the thalamus might had interrupted the ascending sensory pathway as well as the integration of their inputs. Our patient could control movement of his phantom arm but the passive movements or any sensory stimulations did not illicit any changes on his phantom arm. The possible explanation for these phenomenon is that in voluntary movement, the brain programs the set of muscle movements to adjust the resulting motor action and ongoing perception. In absence of sensory feedback due to the cerebral lesion, the perception of a movement may be based on its predicted outcome rather than on its actual consequences leading to a false perception of moving body. According to the review, we speculated that impairment of the sensory and motor system as well as system maintaining the internal representation causes the SPL. Although exact mechanism underlying the SPL still remains unknown, it provides some aspects of understanding the brain's body perception.

P-1-101

Factors associated with depression and anxiety in caregivers for demented patients

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Background & Objectives: The aging of the population has rapidly progressed in Korea, and the number of demented patients is increasing these days. Many studies have found that caregiving is extremely stressful and results in adverse physiologic and psychologic outcomes, not only for caregivers themselves but also for recipients. This study investigated the factors associated with the depression and anxiety of caregiver. Furthermore we aimed to arouse attention to the psychiatric burden of caregiver. **Method:** Caregivers for demented patients were recruited from 9 medical care centers in the metropolitan city of Daegu and Gyeongsanbuk-do province. 220 participants were included and they were all non-family group. During face-to-face interviews with a psychologist, they completed the Burden Interview, Beck Depression Inventory(BDI), Beck Anxiety Inventory(BAI), their health status, the severity of dementia in their patients, and the length of care time were evaluated. The depression and

anxiety experienced by care workers and the factors affecting it were assessed using statistical analyses. **Results:** The caregivers were mainly women having a mean age of 46 years. The mean caregiver's BDI was 8.68 and BAI 7.33. The level of caregiver's depression was affected by health status($p=0.009$) and residing form($p=0.004$). The level of caregiver's anxiety was associated with age($p=0.000$), total time for care($p=0.003$), BMI ($p=0.000$), educational status($p=0.000$), health status($p=0.000$) and residing form($p=0.013$). **Conclusion:** Our study investigated factors affecting caregivers' depression and anxiety in Korea. The health status was most closely related to depression. For the anxiety of caregiver, total time for care was the most closely related.

P-1-102

Diagnostic accuracy of entorhinal cortical volume, hippocampal volume, and fractional anisotropic value of hippocampus in mild cognitive impairment

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Background & Objectives: Mild cognitive impairment (MCI) is a clinical state that has high risk of conversion to Alzheimer's disease (AD). With developing of disease modifying medications to reduce amyloid or tau protein accumulation in the brain, early diagnosis of MCI with reliable biomarker is very important for the early intervention in preclinical state of AD. We compare diagnostic accuracy of entorhinal cortical volume, hippocampal volume, and fractional anisotropy (FA) value of hippocampus in MCI patients that has been known as early imaging biomarker of AD. **Method:** We recruited 23 control and 23 MCI patients. They underwent clinical interview, neuropsychological tests, and MRI scanning. 3D T1 SPGR image and diffusion tensor image were obtained for the image analysis. Entorhinal cortical volume and hippocampal volume were measured by manual segmentation with Analyzer program. FA value was measured with ROI method at the head of hippocampus with Volume One program. **Results:** Hippocampal volume, entorhinal cortical volume and FA value of hippocampus were lower in MCI group ($p<0.001$). Age, gender, and education were not different between two groups. Diagnostic accuracy of each of markers was evaluated with receiver operating characteristic curve. Area under the curve (AUC) value was highest in entorhinal cortical volume (AUC=0.989). AUC of the FA value of hippocampus were lower (AUC=0.944) and hippocampal volume had the lowest AUC value (AUC=0.796). Diagnostic accuracy of Mini-Mental State Examination was higher than the hippocampal volume (AUC=0.832). **Conclusion:** These results revealed that entorhinal cortex involved in the earlier stage of MCI and the hippocampus affected later on. Microstructural change of hippocampus that measured by DTI analysis preceded by macrostructural volume loss. Entorhinal cortical volumetry is a very sensitive imaging biomarker for the diagnosis of MCI. We need to develop effective method to evaluate entorhinal cortical volume with less time consumption

P-1-103

Neuroimaging prognostic factors in patients with delayed post-hypoxic leukoencephalopathy after high dose corticosteroid therapy

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Background & Objectives: Delayed post-hypoxic leukoencephalopathy (DPHL)

is a rare disease condition in which patients appear to make a complete clinical recovery after an acute episode of anoxia or hypoxia but then develop several neurologic deficits characterized by apathy, confusion, agitation, extrapyramidal signs and/or progressive cognitive impairment. We analyzed clinical findings, neuroimaging characteristics and prognostic factors of patients with DPHL after high dose steroid therapy. **Method:** We recruited consecutively 9 patients who were diagnosed to DPHL at a university hospital from January 2013 to March 2015. We also evaluated baseline characteristics, etiology, duration from hypoxic event to symptoms onset, characteristics of Magnetic resonance imaging (MRI) and prognosis after 3-6 months follow-up in patients with DPHL. **Results:** Mean age of patients was 54.78 years and 77.8% were males. Several causes of DPHL were detected in these patients as follows: CO intoxication (n=6); post-sudden cardiac arrest (n=1); post-general anesthesia (n=1); opioid analgesics intoxication (n=1). In MRI, all included patients had diffused high signal intensity lesions on bilateral cortical and subcortical areas and periventricular areas in diffusion weighted image (DWI), T2 and Fluid-attenuated inversion recovery (FLAIR) MR images. Eight patients out of 9 subjects received high dose corticosteroid therapy. In these patients who received steroid therapy, 7 patients showed significant improvement on cognitive functions and other neurologic deficits after 3-6 months follow-up periods. However, there were differences in degree of recovery in each patient that showed good prognosis. Patients who showed better response to treatment had a few characteristics as follows: relatively young age; no lesion involvement to basal ganglia and thalamic areas in DWI, T2 and FLAIR MR Images; mild low or iso-signal intensity lesions in apparent diffusion coefficient (ADC) MR images. **Conclusion:** The exact pathogenesis of DPHL remains unknown. Also, there is no standardized treatment that proven its effectiveness, except for eliminating causes and symptomatic management. For proper treatment and prognosis assessment, furthermore large sample size and appropriate detailed neuroimaging evaluations should be performed.

P-1-104

The association between body mass index and cognitive decline in patients with small vessel disease - preliminary study

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Background & Objectives: Obesity in middle age might increase the risk of dementia in old age. The potential underlying mechanisms linking higher body mass index (BMI) to dementia may include direct effect of adiposity as well as indirect effects such as vascular risk factors and cerebrovascular disease. We investigated the association of BMI with cognitive decline according to the severity of the small vessel disease. **Method:** We recruited 92 individuals who visited complain of the cognitive decline. All subjects were assessed with Seoul Neuropsychological Screening Battery (SNSB) to evaluate their cognitive functions and checked their BMI. Severity of the small vessel disease was categorized in three groups by clinical white matter rating scale using their FLAIR images of brain MRI. We calculated the correlation coefficient of BMI on the cognition in each small vessel disease groups. **Results:** Mean age of the individuals were 73.05 ± 7.19 and mean BMI was 24.39 ± 3.13 . According to BMI, overweight ($25-29.9 \text{ Kg/m}^2$) and obese ($\geq 30 \text{ Kg/m}^2$) individuals tend to low p-value of the MMSE score, but it was not significant. Total 77 individuals were performed brain MRI and the number of patients in each small vessel disease scale was follows: 44 had a minimal, 18 had a moderate and 15 had a severe. In each subgroup of small vessel disease, we did not find the correlation between BMI and their cognition. **Conclusion:** This study did not demonstrate the association between BMI and cognitive decline under the severity of small vessel disease. These findings suggest that the possible another mechanism of the influence of BMI to dementia as well as vascular risk factors is exist.

However, more caution is needed because there is a need for more research with larger sample and further study is needed to evaluate by dementia subtypes.

P-1-105

The influence of the subcortical ischemia and cognitive patterns and their changes in normal hospital visited elderly

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Background & Objectives: Recent studies on the cognitive changes of normal elderly showed diverse results due to the variety of the methodology and the participants. The cognitive changes during normal aging showed two distinct patterns such as generalized cognitive decline and inhibitory dysfunction. The domestic longitudinal studies of the normal aging, however have been performed rarely and limited. We performed a short term longitudinal study related to the normal cognitive changes who had visited university affiliated hospital for the cognitive evaluation. **Method:** Participants were consecutively recruited from Myongji hospital in Korea from January 2008 to June 2012. On magnetic resonance imaging (MRI), deep white matter (DWM) ischemic lesions were classified into D1 (the longest diameter of DWM lesion $< 10 \text{ mm}$), D2 ($10 \text{ mm} \leq \text{DWM} \leq 24 \text{ mm}$), and D3 ($25 \text{ mm} \leq \text{DWM} < 30 \text{ mm}$). **Results:** A total of 100 Participants were recruited (76 in minimal, 15 in moderate and 4 in severe). Mean age were 62.0 ± 11.7 and female was predominant. Mean score of Korean version of mini-mental status examination (K-MMSE) was 27.1 ± 2.7 and that of clinical dementia rating some of boxes (CDR-SOB) was 0.9 ± 0.8 . In clinical profiles, hypertension, hyperlipidemia, cardiac disease, stroke were common. Depending on the subcortical ischemia focal neurological sign ($p=0.015$) and Hachinski ischemic score ($p=0.004$), hypertension ($p=0.015$), cardiac disease ($p=0.027$) and stroke ($p=0.016$) were increased. By the increasing of subcortical ischemia, complex cognitive abilities such as multiplication and division of calculation were affected. Rey-Osterreith complex figure test (RCFT) recognition test of true positive ($p = 0.014$) and Korean color word stroop test (KCWST) word reading correct ($p = 0.012$) were also affected according to the ischemic burden. In the follow-up cognitive function test practice effects was prominent in the K-MMSE place orientation. CDR-SOB score suggest the possibilities of the useful clinical tool in the longitudinal study of the normal cognitive function ($p = 0.009$). **Conclusion:** These results suggest that in the frontal and some parietal areas are sensitive cognitive domains in normal elderly. By the increasing of the subcortical ischemia the frontal inhibitory function and some parietal functions required complex cognitive abilities are more likely affected. During the course of normal aging, CDR-SOB score has the possibility of useful clinical tools of the longitudinal study of the normal cognition. The ischemia sensitive cognitive domains during normal cognition are frontal inhibitory dysfunction and visual memory recognition tests.

P-1-106

Entorhinal cortex, earlier structural change than the hippocampus in subjective memory impairment

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Background & Objectives: Subjective memory impairment (SMI) is common in the elderly. SMI has been considered as a risk state with rapid cognitive decline or convert dementia in the longitudinal follow up. The aim of this study was to investigate the structural changes of entorhinal cortex and hippocampus with volumetry and diffusion tensor imaging analysis. **Method:** We recruited 23 control and 21 SMI patients. They underwent clinical interview, neuropsychological tests, and MRI scanning. 3D T1 SPGR image and diffusion tensor image were obtained for the image analysis. Entorhinal cortical volume and hippocampal volume were measured by manual segmentation with Analyzer program. FA value was measured with ROI method at the head of hippocampus with Volume One program. **Results:** Age, gender, education were not different between groups ($p > 0.05$). Mini-Mental State Examination score was lower in SMI group (28.7 VS 26.6, $p < 0.05$). Entorhinal cortical volume and FA value of hippocampus were lower in SMI group ($p < 0.001$). Hippocampal volume ratio was not different between groups (0.153% VS 0.149%, $p = 0.43$). Diagnostic accuracy of each of markers was evaluated with receiver operating characteristic curve. Area under the curve (AUC) value was highest in entorhinal cortical volume (AUC=0.943). AUC of the FA value of hippocampus were lower (AUC=0.877) and hippocampal volume had the lowest AUC value (AUC=0.531). Diagnostic accuracy of Mini-Mental State Examination was higher than the hippocampal volume (AUC=0.769). **Conclusion:** These results revealed that pathologic changes of SMI involves in the entorhinal cortex earlier than the hippocampal involvement. Entorhinal cortical volumetry may be a sensitive imaging biomarker for the early detection of SMI. Longitudinal study should be needed to find out the entorhinal cortical volume as a progression biomarker in SMI.

P-1-107

Marchiafava-Bignami disease with callosal and extracallosal lesions documented by diffusion tensor imaging

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Background & Significance: Marchiafava-Bignami disease (MBD) is rare and a typically alcohol associated disorder characterized by demyelination and necrosis of the corpus callosum. There are no pathognomonic clinical presentations of MBD, however, radiologically, MBD is characterized by hyperintense on T2-weighted image (T2WI) and FLAIR, and hypointense on T1-weighted images in the corpus callosum. The involvement of cortex at the acute stage of MBD is associated with an unfavorable outcome. We report a case of MBD who showed functional recovery after treatment, with the involvement of cerebral cortexes which was identified by diffusion tensor imaging (DTI). **Case:** A 44-year-old man visited our hospital with disorientation and progressive disturbance of consciousness. He had a long history of severe alcoholism without other underlying diseases. He complained of dysarthria and gait disturbance. Neurologic examination revealed no lateralizing signs, however, cognitive functions were impaired with Korean Mini-Mental state Examination (K-MMSE) score of 3. Laboratory studies showed only a mild anemia with low level of thiamine. Brain MRI showed high signal intensities on entire corpus callosum in both T2WI and FLAIR. And DTI revealed significantly diminished fiber density throughout corpus callosum. Once he was diagnosed with MBD, treatment with intravenous thiamine and steroid were started. After 1 month, his neurologic status was more improved (K-MMSE =17). Follow-up DTI study 9 months later showed more increased density of association fibers. **Conclusions or Comments:** In this case, we report a typical MBD patient even with extracallosal lesions who showed a favorable prognosis confirmed by clinical findings and a compatible radiological recovery using

DTI.

P-1-108

Practical age difference cut-offs for T1-axial medial temporal atrophy visual rating scales in clinical diagnosis of Alzheimer's disease: CREDOS Data

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Background & Objectives: To increase certainty of diagnosing Alzheimer's disease (AD), recently revised research criteria included neuroimaging and CSF biomarkers presenting amyloid β deposition and neuronal injury. The widely used tool for evaluating extent of medial temporal atrophy (MTA) as a biomarker of neuronal injury is the visual rating scale (VRS) with brain MRI. Visual assessment of MTA is quick and easy to use. However, no cut off values for the VRS of MTA have been specified for age. Our study aimed to generate age-adjusted cut-off scores for axial MTA VRS, for practical usage in differential diagnosis of AD from age related MTA but cognitively normal elderly population. **Method:** From the CREDOS study data (November 2005 to March 2013), total of 3432 subjects (1429 with no cognitive impairment (NC), and 2003 AD patients with first diagnosis, minimal ischemia) were recruited and divided into age ranges of 50-59, 60-69, 70-79 and 80-89 years. Using "Sample Size Tables for Clinical Studies" software, 57 individuals were chosen randomly within the age group box except for the 47 subjects in 80-90 NC group. T1-axial MTA VRS was graded by a neurologist who was blinded to the subject's age and diagnosis. The right and left MTA were rated separately and the more severe MTA was selected for each subject. The cut-off values were evaluated from the area under the curve (AUC) that was obtained from the receiver operating characteristic curve analysis. The intra-rater agreement of cut offs for the T1W-axial VRS used kappa value. All statistical analyses were performed using the Statistical Package for the Social Sciences 21.0 (SPSS Inc., Chicago, IL, USA). $P < 0.05$ was considered statistically significant. **Results:** In subjects who were between 50-59 years of age, the appropriate cut-off point used to discriminate AD from NC was ≥ 1 (sensitivity 88%, specificity 91%, $p < 0.001$) with AUC value of 0.900 and 95% CI of 0.838-0.963. Also, for subjects who were sixties, the optimal cut-off score was ≥ 1 (sensitivity 94%, specificity 84%, $p < 0.001$) with AUC value of 0.901 and 95% CI of 0.838-0.963. In subjects aged 70 to 79, the optimal cut-off was ≥ 2 (sensitivity 79%, specificity 86%, $p < 0.001$) with the AUC value of 0.852 and 95% CI of 0.781-0.923. For the oldest age group (80-89-year olds), the cut-off ≥ 3 (sensitivity 70%, specificity 100%, $p < 0.001$) was optimal with the AUC value of 0.904 and 95% CI of 0.846-0.961. The intra-rater reliability of cut-offs for the T1W-axial VRS-MTA was 0.782 ($p < 0.001$). **Conclusion:** The Cut-offs of T1-axial MTA VRS for the diagnosis of AD was different based on age range in decades. The proposed age-decades-specific cut scores could be useful in practical clinical setting in differential diagnosis of AD from age related MTA but cognitively normal elderly population.

P-1-109

Sleep influences the cognitive functions in mild to moderate dementia with Alzheimer's disease

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Background & Objectives: Sleep is important for cognitive function including memory consolidation in elderly persons. Sleep disturbances are very common in Alzheimer's disease (AD), but whether they contribute to cognitive function is unknown. Our primary objective was to estimate differences in sleep architecture and variable parameters between patients with probable AD and healthy elderly persons. Secondary objective was to determine whether sleep disturbances were associated with cognitive decline of AD. **Method:** We have analyzed sleep parameters and architectures in 16 healthy elderly subjects and 25 patients with probable AD using overnight polysomnography (PSG) for this study. The sleep variables included proportions of rapid eye movement (REM) sleep, non-REM sleep such as N1, N2 and N3, total sleep time (TST), sleep efficiency (SE), wake after sleep onset (WASO), Periodic limbs movement during sleep (PLMS) index, respiratory disturbance index (RDI), latency to sleep onset were analyzed. Cognitive impairment and dementia severity were assessed with detailed structured neuropsychological tests. **Results:** RDI in AD group was significantly higher than healthy elderly controls ($p < 0.05$). In addition, TST, the proportion of N2/N3 sleep and SE in AD group were decreased. On the other hand, the proportion of N1 sleep, latency to sleep onset, WASO, PLMS index were increased, compared with control group. The RDI and TST were statically associated cognitive decline in probable AD ($p < 0.05$). **Conclusion:** The patients with AD had significantly lower sleep quality than healthy elderly in overnight PSG. Our results provide an additional data supporting the hypothesis that obstructive sleep apnea is associated with AD pathology. We suggest that the sleep study might be a useful clinical marker to predict AD progression.

P-1-110

Prediction of AD pathophysiology based on cortical thickness patterns

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Background & Objectives: Pathologically defined subtypes of Alzheimer's Disease (AD) have represented distinctive clinical characteristics of the patients, but it cannot be applied to most of AD subjects. If the new clustering analyses based on cortical thickness patterns can be potentially comparable to the classification of AD subjects according to autopsy findings, it would enable classification and prediction of prognosis of AD patients. **Method:** This cross-sectional study included subjects with AD ($n=77$) from the Alzheimer's Disease Neuroimaging Initiative (ADNI) who underwent structural 3T magnetic resonance imaging (MRI), [18F]Fludeoxyglucose (FDG)-positron emission tomography (PET), florbetapir-PET, and cerebrospinal fluid (CSF) study. The MRI images were processed to measure cortical thickness, and hierarchical agglomerative cluster analysis was performed using Ward's clustering linkage. We analyzed each PET data using region of interest based analysis according to group divided by clustering analysis. We compared CSF results and neuropsychological test for each group. **Results:** Subgroups of AD using clustering based on cortical thickness patterns -19.5% of medial temporal (MT) subtype, 55.8% of diffuse atrophy (D) subtype, 24.7% of parietal dominant (P) subtype - was well replicated in ADNI2 data. Among the 3 subtypes, patients in the P subtype were younger than the other 2 subtypes ($p=0.0002$). Patients in the P subtype represented glucose hypometabolism in the left inferior parietal, right superior parietal, left middle occipital cortex matched well with regions with cortical atrophy, measured by FDG-PET, compared to with other subtypes. Patients in the MT subtype revealed glucose hypometabolism in left

hippocampus and bilateral frontal cortices compared to the other 2 subtypes. Patients in the P subtype represented marked A β accumulation in most of brain regions, including superior, middle, inferior frontal cortices, superior, inferior parietal cortices, and precuneus measured by florbetapir-PET, compared to the other 2 subtypes. Among the 3 subtypes, CSF results and neuropsychological test results revealed no statistically significant differences. **Conclusion:** Cortical thickness patterns can reflect the pathophysiological status of AD assessed by FDG-PET and florbetapir PET.

P-1-111

Systolic blood pressure variability relates to microstructural changes in white matters and cortical atrophy in cerebral small vessel disease

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Background & Objectives: Neuroimaging markers of cerebral small vessel disease (SVD) are lacunes, white matter hyperintensities (WMH), microbleeds, and brain atrophy. Both blood pressure (BP) and its variability (BPV) are established as risk factors for development of atherosclerotic disease. We investigated the correlation between BP and BPV, and neuroimaging markers of cerebral SVD in patients with cerebral SVD. **Method:** In this study, 172 patients with cerebral SVD were recruited. The average BP and BPV, as determined by the standard deviation of the systolic and diastolic BP, were recorded during a mean 5.1 ± 2.1 outpatient clinic visits for 45.2 weeks. They underwent multi-modal MRIs including fluid-attenuated inversion recovery lesion load, fractional anisotropy (FA) and mean diffusivity (MD) from diffusion tensor imaging (DTI), Gradient Echo imaging, and T1 Spoiled Gradient Recalled Echo imaging. **Results:** Systolic BPV was correlated with the WMH volume of left occipital lobe, average MD ($r=0.18$, $p=0.04$) and MD at peak ($r=0.18$, $p=0.03$) of MD histograms within normal-appearing white matter (NAWM). Systolic BPV was negatively correlated with cortical thickness of bilateral parietal and occipital lobes. The number of cerebral microbleeds was correlated with average SBP ($r=0.26$, $p=0.04$). Diastolic BPV, and average SBP and DBP were not correlated with WMH volume, MD and FA values, and cortical thickness. **Conclusion:** Systolic BPV was associated with microstructural changes in white matters and cortical atrophy in cerebral SVD.

P-1-112

The differences of caregiver burden between the young old and the oldest old Alzheimer's disease patients

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Background & Objectives: The aim of this study was to investigate the pattern of changes in caregiver burden between the young old Alzheimer's disease (AD) and oldest old AD to improve the care counselling and management plan. **Method:** This study included 138 pairs of child caregivers who attend to AD dementia patients. Dementia patients were from the 12 participating cen-

ters of the CARE (Caregivers of Alzheimer's disease Research) study. The patients were divided to two groups by the age of 85; 108 of the oldest old (OO) AD group and 30 of the young old (YO) AD group. We investigated baseline and follow up data of sociodemographic and caregiver burden, including Caregiver Burden Inventory (CBI), Beck Depression Inventory (BDI), SF-36. Comparisons of baseline characteristics between two groups and of changes from baseline to follow up data after 12 months in each group were made using Mann-Whitney U test and Wilcoxon signed rank test, respectively. Values of $p < 0.05$ were considered statistically significant. **Results:** Patients of the OO group were in more severe state and functionally disabled state (CDR; 1.1 ± 0.6 , 1.7 ± 0.9 , p -value=0.003, SIADL; 25.6 ± 13.0 , 34.6 ± 12.5 , p -value=0.001) than patients of the YO group. Caregivers of the OO group were significantly older (50.3 ± 8.6 , 56.2 ± 5.4 , p -value < 0.001) and decreased physical functioning (83.1 ± 22.2 , 74.4 ± 21.1 , p -value=0.014) than those of the YO group. After 12 months, five subscale items (physical functioning, role limitation due to physical health, role limitation due to emotional problems, social functioning and pain with p -value of 0.035, 0.002, < 0.001 , 0.004 and 0.049, respectively) and BDI (13.1 ± 10.8 , 10.4 ± 9.5 , p -value=0.001) were improved in caregivers of the YO group. However, only BDI was improved in caregivers of the OO group (14.4 ± 10.4 , 9.0 ± 9.8 , p -value < 0.019). **Conclusion:** The physical burden is attenuated though the time in caregivers of young old patients, however, did not decrease in caregivers of oldest old patients. The depressive mood of caregivers is improving as time goes by. The policy to alleviate physical suffering of caregivers to take care the oldest old dementia patients are need.

P-1-113

Autopsy confirmed case of frontotemporal dementia with motor neuron disease

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Background & Significance: Approximately 15% of patients with frontotemporal dementia (FTD) can co-occur with motor neuron disease (MND). The FTD-MND cases have frontotemporal lobar degeneration (FTLD) - transactive response DNA binding protein (TDP) pathology which is divided as four subtypes (type A, B, C, D) based on the morphological appearance, cellular location and distribution of the inclusions. Here, we report a patient with FTD-MND whose pathological diagnosis was FTLD-TDP type B. **Case:** A 65-years-old woman presented with progressive cognitive dysfunctions and motor deficits. Her cognitive dysfunctions started after traumatic brain injury due to car accident at the age of 61, but detailed history about cognitive dysfunctions including behavioral changes could not be obtained because she had lived alone until she was 64 years old when dysarthria and right hand weakness developed. Past medical history was significant for high blood pressure. There were no specific family and social histories. She lost 20 kg during 1 year before the admission. Neurological examination revealed generalized motor weakness (Right: 3/5, Left 4/5), aphonia, tongue atrophy and fibrillation, fasciculation on bilateral forearm and thigh, hyperactive deep tendon reflexes, and positive pathological reflexes. Needle EMG showed diffuse denervation and reinnervation processes indicating an underlying MND. Her K-MMSE

score was 5/30, CDR 4 and GDS 6. The scores of neuropsychiatric inventory and frontal behavioral inventory were 41/144 and 34/72. Brain MRI demonstrated diffuse cortical atrophy and FDG-PET showed decreased glucose metabolism in bilateral frontal areas (worse on the left). Based on clinical and neuroimaging findings, her clinical diagnosis was behavioral variant FTD (bvFTD) with MND. She continued to decline and died at the age of 67 years, 6 years after the onset of disease. The brain weight was 994g. Grossly, there were definite atrophy in dorsolateral frontal cortices and hippocampus, and mild atrophy in thalamus, basal ganglia, and amygdala. Microscopically, many TDP-43 immunoreactive neuronal cytoplasmic inclusions (NCIs) with few dystrophic neurites (DN) were seen in affected cortices and anterior horn of spinal cord. The morphology and distribution of TDP-43 inclusions was consistent with FTLD-type B. Tau-immunoreactive neurofibrillary tangle pathology was incidentally found only in the entorhinal cortex (Braak stage I). No beta-amyloid and alpha-synuclein-immunoreactive pathology was observed. **Conclusions or Comments:** This is the first documented autopsy-confirmed case with FTD-MND in Korea.

P-1-114

The effect of group musical therapy on cognitive function in patients with probable Alzheimer's disease

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Background & Objectives: Beside pharmacological treatment, non-pharmacological interventions are a great deal of interest resides on ways that allow modulation of brain plasticity in the elderly. Music therapy is a potential non-pharmacological treatment for the behavioral and psychological symptoms of dementia, but a few studies reported it to be helpful. The aim of this study was to evaluate the effect of structured musical intervention therapy in patient with probable AD. **Method:** The subjects of the study were a total of sixty patients with probable AD (K-MMSE: 16 ± 3.04 , CDR: 1.20 ± 0.38). The musical therapy was applied to the group twice a week, fifty minutes per session for eight weeks. The data were analyzed by using chi-square and paired t-test before and after musical intervention. **Results:** The study showed a significant improvement in cognitive function after musical therapy measured with K-MMSE ($p < 0.05$). Activities daily living (ADL) markedly improved after the all session of musical interventions ($p < 0.001$). **Conclusion:** Group music therapy is a safe and effective method for cognitive dysfunction, and also improving ADL in patients with probable AD.

P-1-115

Preoperative cerebrospinal fluid biomarkers and MRI findings in patients with idiopathic normal pressure hydrocephalus showing favorable surgical outcome

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Background & Objectives: Idiopathic normal pressure hydrocephalus (INPH) is known to be a potentially treatable condition causing dementia. However, the neurocognitive outcomes after shunt surgery have been variable. It is im-

portant to define preoperative characteristics of INPH patients who showed favorable surgical outcomes. Our study aimed to compare baseline differences between surgery-responsive patients and unresponsive patients after 1 year from shunt surgery which can serve as predictive factors. **Method:** All patients were recruited from the memory clinic in Asan Medical Center and Samsung Medical Center between December 2012 and May 2015. After recruiting 69 INPH patients, 38 were excluded due to screen failure (n=5), refusal of shunt surgery (n=28), follow up loss (n=4) or death (n=1). Finally, 31 patients with INPH who underwent shunt surgery and 1 year follow-up examinations were analysed. Patients were divided into two groups according to the surgical outcomes using the INPH grading scale and the modified Rankin Scale (mRS): patients with improvement ≥ 3 on INPH total score or ≥ 2 on mRS for the responsive group (n=17), and patients with progression or no change or ≤ 2 on INPH total score and ≤ 1 on mRS for the unresponsive group (n=14). Preoperative cerebrospinal (CSF) amyloid beta, total tau and phosphorylated tau levels, MRI findings, patient's characteristics and clinical findings were compared between the two groups. Correlations between shunt outcomes and preoperative characteristics were also made. **Results:** Fifty-five percent (17/31) of INPH patients showed favorable outcomes after 1 year of shunt surgery and gait problem was most likely to improve. Shunt-responsive INPH patients showed lower CSF phosphorylated tau/amyloid beta, fewer lacunes and more disproportionately enlarged subarachnoid space hydrocephalus (DESH) sign compared to those with shunt-unresponsiveness. Preoperative severity of symptoms, symptom durations, comorbidities, presenting symptoms, valve difference, and other neuroimaging/ CSF parameters were not different between the groups. Favorable shunt outcome was related to positive baseline DESH sign and lesser number of lacunes. **Conclusion:** Preoperative CSF biomarkers and neuroimaging findings were different between shunt-responsive and unresponsive patients. Our results suggest that concomitant pathology to INPH might impact adversely on the shunting outcome.

P-1-116

Neuropathologic changes in two patients with advanced dementia

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Background & Significance: Although the neuropathologic examination is very important for the definite diagnosis of dementia etiology, it has been rarely performed in Korea. We analyzed neuropathology findings in two patients with history of dementia, of which Alzheimer's disease (AD) neuropathologic changes were evaluated according to NIA-AA guidelines. **Case:** Patient 1 was a 77-year-old man with 4-year history of progressive cognitive decline including episodic memory loss. Visuospatial dysfunction developed 2 year after the onset. For one year, he had been dependent in all activities of daily living. His brain MRI showed diffuse cerebral atrophy including marked medial temporal atrophy and diffuse severe subcortical small vessel disease and old cerebellar hemorrhage. Based on the patient's history, examination and neuroimaging, the tentative clinical diagnosis was probable AD combined with vascular dementia. The gross examination of the brain (1,100gram) revealed symmetrical cerebral hemispheres with no softening, atrophy, or masses. Multifocal variable sized lacunar infarctions were noted involving basal ganglia and other deep gray matter. The right hemisphere of cerebellum showed a large old cystic infarction (4.5 x 4cm). Microscopic examination confirmed the presence of microvascular lesions with vascular brain injury. In addition, changes of Braak stage II with neurofibrillary tangles (NFTs) predominantly in

entorhinal cortex and closely related areas were also noted. Patient 2 was a 94-year-old woman with a history of progressive cognitive impairment for 13 years. She first visited the outpatient clinic due to episodic memory loss (at 83 years old). Mood fluctuation and visual hallucination developed 4 and 6 years after the onset, respectively. Although we could not check the brain imaging, the tentative clinical diagnosis was the possible AD. The gross examination of the brain (1,071gram) revealed symmetrical cerebral hemispheres with a focal old infarction involving base of left occipital lobe (2 x 2cm) and an irregular defect (1.5 x 0.5cm) at upper surface of left cerebellum. Bilateral hippocampi were markedly atrophic. Degenerated left parahippocampal gyrus was connected with old infarction involving left occipital lobe. Microscopic examination confirmed the presence of old cystic infarctions involving left occipital lobe and left cerebellum. There were changes of isocortical tangle stage with NFTs in all sectors of hippocampus and subiculum and tangles in thalamus and amygdala. Ghost tangles with neuronal loss and astrocytic gliosis were distributed throughout the CA1. **Conclusions or Comments:** We report two autopsy-defined dementia patients with ischemic lesions and AD neuropathologic changes.

P-1-117

Clinical implication of A β accumulation in occipital lobes using [18F]-Florbetaben PET

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Background & Objectives: Accumulation of beta-amyloid (A β) plaques in the occipital lobes have not been relatively neglected in neuroimaging and neuropathology studies. However, substantial uptake of A β plaques in occipital cortices can be noted in many amyloid PET imaging studies. In this study, we investigated the prevalence of A β uptake in the occipital lobes and its clinical implication. **Method:** A total of 520 patients were clinically diagnosed to have AD-spectrum disorders after completion of 3T MRI and detailed neuropsychology tests from May 2013 to July 2015 in Dementia clinic at Asan medical center. After obtaining a consent for study participation in each individual, [18F]-fFlorbetaben amyloid PET were performed in 79 patients. After visual rating of positivity of global A β accumulation using Regional Cortical Tracer Uptake (RCTU) and Brain Amyloid-beta Plaque Load (BAPL) scoring system, we divided AD subjects into A β positive and negative group based on accumulation of A β plaques in occipital lobes. Demographics and neuropsychology test results between the two groups, occipital positive group vs. occipital negative group, were compared. **Results:** Among the 79 patients, 43 patients (43/79, 54.4%) represented increased global amyloid uptake by [18F]-Florbetaben-PET scan. Among these 43 patients, 21 patients (21/43, 48.8%) had increased amyloid uptake in occipital lobes. Occipital positive (OCC+) group was younger (mean \pm SD=66.67 \pm 9.91, years) than occipital negative (OCC-) group (73.14 \pm 6.39, p=0.016). OCC+ group had lower score in global cognitive score such as CDR (p=0.011), CDR-SB (4.47 \pm 1.93 vs 2.55 \pm 2.38, p=0.008), and GDS (4.21 \pm 0.79 vs. 3.10 \pm 1.09, p=0.001). Detailed neuropsychological tests revealed that OCC+ group had lower z-score on RCFT copy (-4.44 \pm 5.96 vs -1.03 \pm 2.74, p=0.047), RCFT immediate recall (-2.42 \pm 0.75 vs -1.22 \pm 0.72, p<0.001), RCFT delayed recall (-2.45 \pm 0.61 vs -1.37 \pm 0.76, p<0.001), RCFT recognition (-2.98 \pm 3.22 vs -1.23 \pm 1.61, p=0.037), SVLT immediate recall (-1.83 \pm 1.40 vs -0.79 \pm 1.35, p=0.028), SVLT delayed recall (-2.84 \pm 1.41 vs -1.1 \pm 1.64 p=0.022), Stroop color reading (-2.82 \pm 1.88 vs -1.16 \pm 1.38 p=0.004), and COWAT phonemic test (-1.30 \pm 1.00 vs -0.53 \pm 1.10 p=0.034) than OCC- group. **Conclusion:** AD patients in the OCC+ repre-

sented features noted in early onset AD: they were younger and have more severe cognitive dysfunction in neuropsychological tests. OCC+ in amyloid PET scans need to be emphasized and it would potentially be a marker of early onset AD dementia. Cortical thickness patterns and additional features reflecting OCC+ and OCC- AD will be presented.

P-1-118

Neurotoxoplasmosis presented with gait disturbance and cognitive impairment

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Background & Significance: Neurotoxoplasmosis, also known as cerebral toxoplasmosis, is an opportunistic infection, caused by the parasite *Toxoplasma gondii*, which typically affects patients with HIV/AIDS, and is the most common cause of cerebral abscess. *Toxoplasma gondii* is found ubiquitously and antibodies to the organism can be identified in 30% of all humans. The rate varies greatly from population to population and has a wide reported prevalence: from 6-90%. In most cases, the infection is asymptomatic. However, in immunocompromised patients (especially those with HIV/AIDS), infection can become established. In immunocompetent patients, acute encephalitis is extremely rare. Even in the immunocompromised symptoms are typically vague and indolent. Here, we present a case of neurotoxoplasmosis with gait disturbance and cognitive impairment. **Case:** The 55-year-old male admitted because of gait disturbance and cognitive impairment occurred the admission day. He was working as a day laborer on construction site. About three months ago, he had admitted for hypersensitive pneumonitis. At that time, he was received antibiotics, steroid therapy and discharged without any sequelae. After one month, he complained mild dysarthria but there was no problem in daily life. Dysarthria progressed slowly. On admission day, he noticed gait disturbance and cognitive impairment when he woke up. Mental status was alert and MMSE score was 15. He complained mild dysarthria and gait disturbance, pattern of bilateral swaying. Initial vital sign was stable and CBC, chemistry test and urinalysis were normal. We checked brain MRI and CSF analysis. CSF study revealed normal range. On brain MRI, there were multiple rim like enhanced small nodules on bilateral cerebral hemisphere, pons, medulla and midbrain. The nodules presented low signal intensity in T1 weighted image. In T2 weighted image, nodules were central iso-intensity or slightly hypointensity, inner iso, outer high signal intensity. Initially we thought neurocysticercosis and empirically started intravenous steroid, antiparasite agent, albendazole and praziquantel. He discharged with improving symptoms. About two weeks later, he re-admitted due to headache and confusion. We performed brain MRI again and found more edema around nodule. At this time, we performed brain biopsy and additional parasites laboratory study. In parasite study, we found toxoplasma IgG(+) and IgM(-) in serum and AFB(-), Tb PCR(+) in brain tissue biopsy. So we started pyrimethamine, leucovorin, clindamycin and intravenous steroid. Until now his symptoms are not significant change. We are now planning to start TB medication if his symptoms are worsening. **Conclusions or Comments:** *Toxoplasma gondii* is an intracellular parasite that infects birds and mammals. Its definitive host is the cat and other Felidae species. Excretion of oocytes in its faecal content followed by human contaminated uncooked consumption can lead to human infection. In immunocompetent individuals, it primarily causes a subclinical or asymptomatic infection. In immunocompromised individuals (e.g. AIDS patients), toxoplasmosis is the most common cause of a brain abscess. In our case, the patients lived in a house with mold a lot through last winter. We guessed maybe this circumstances made him immunocompromised state. Furthermore, he was staying alone so we don't know exactly what he ate in the past. In con-

clusion, when we saw a patient with sudden cognitive impairment and confusion, we must also think about parasite mediated disease such as toxoplasmosis.

P-1-119

Protective effects of choline alfoscerate (L-alpha-glycerylphosphorylcholine, α -GPC) on seizure-induced neuronal death and cognitive impairment

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Background & Objectives: An epileptic seizure is a brief episode of abnormal electrical activity in the brain that manifests as behavioral or sensory changes. While the episodes are transient, patients with epilepsy are at an increased risk for developing lasting deficits to cognitive function and/ or behavioral abnormalities. Choline alfoscerate (α -GPC) is a natural choline compound found in the brain. It is also a parasympathomimetic acetylcholine precursor that has been shown to be effective in the treatment of Alzheimer's disease and dementia. α -GPC is used to enhance memory and cognition for stroke and Alzheimer's patients but currently remains untested in patients suffering from epilepsy. This study aimed to evaluate whether α -GPC treatment after seizure can ameliorate seizure-induced cognitive impairment and neural injury. **Method:** The potential therapeutic effects of α -GPC on seizure-induced cognitive impairment were tested in an animal model of pilocarpine-induced epilepsy. Seizure was induced by intraperitoneal (i.p) injection of pilocarpine (25 mg/kg) in adult male rats. α -GPC (250 mg/kg) was injected into the intramuscular (i.m) space three weeks after seizure onset for three weeks once-daily administration. To evaluate if treatment with α -GPC provides protection to hippocampal dependent cognitive abilities following seizure we analyzed subject performance using a standard water maze test protocol and brain NeuN immunohistochemistry to determine hippocampal neuronal survival. All groups were sacrificed at 6 weeks post-seizure. **Results:** Standard water maze test after injection α -GPC showed decreased time compared with pilocarpine-induced seizure rats injected with saline. Seizure-induced blood brain barrier disruption was reduced in the pilocarpine-induced seizure rat after injection α -GPC. Seizure-induced neuronal death was also reduced in the pilocarpine-induced seizure rat after injection α -GPC (brain NeuN immunohistochemistry). Neuroblast production was increased by injection α -GPC in the subgranular zone of pilocarpine-induced seizure rats. **Conclusion:** In the present study, we observed enhanced survival of hippocampal neurons and improved cognitive function in animals receiving α -GPC injection after pilocarpine-induced seizure. Therefore, choline alfoscerate (α -GPC) injection may serve as a beneficial treatment for improvement of cognitive function in epilepsy patients.

P-1-120

Safety and efficacy of zonisamide in patients with epilepsy: a result of post-marketing surveillance study

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Background & Objectives: Background: Zonisamide is one of new anti-epileptic drug, and is known to inhibit seizure by its mechanism of action involved in the signal transduction of nerve cells by blocking the influx of sodium ions into the axon membranes, blocking T-type calcium channels on the axon terminal, blocking the secretion of glutamates from the presynaptic neurons, and exhibiting similar effect as Gamma-Amino-Butyric acid (GABA). In

Korea, zonisamide was approved as an antiepileptic drug in 1992 and has been used for epilepsy patients with partial and generalized seizure. The objective of this study was to investigate the efficacy and tolerability of zonisamide in patients with epilepsy and to identify the incidence of adverse events in the real clinical setting. **Method:** Method: This study was carried out in patients treated with zonisamide for the indication of epilepsy including partial seizure, generalized seizure and unclassified seizure. Patients were observed for at least 12 weeks upon treatment with zonisamide and had safety information obtained through post-dose visits, which were acceptable as evaluable subjects. Status and type of adverse events occurring during the medication were obtained regardless of causal relationship to zonisamide. The information of efficacy was assessed by the study physicians and patients at week 12 post-dose of zonisamide. **Results:** Results: A total of 1,948 subjects were included in the study, and 1,744 of these were analyzed for efficacy. Zonisamide was used as a monotherapy in 1,095 patients and as an adjunctive drug in 853 patients. 1,345 (69.1%) patients had partial seizure, 563 patients had generalized seizure and 40 patients were classified as undermined. The mean dosage of zonisamide was 186.72 ± 98.68 mg. Adverse events were reported in 65 patients (3.34%). The most common adverse effect was dizziness (12 patients) followed by drowsiness (6 patients) and skin rash (4 patients). 755 patients (43.29%) became seizure free with the zonisamide treatment, and additional 322 patients (18.41%) experienced marked improvement with the zonisamide treatment. **Conclusion:** Conclusion: Our study showed the safety and tolerability of zonisamide treatment in 'real-life patients with epilepsy'. Our study also demonstrated that zonisamide appears to be an effective option as a monotherapy or adjunctive treatment in patients with epilepsy.

P-1-121

Uric acid change is a valuable marker to reflect refractory status epilepticus

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Background & Objectives: Refractory status epilepticus (RSE) is a devastating neurologic condition. Recent studies suggested etiology, level of consciousness and levels of acute phase proteins in status epilepticus patients could predict patient's outcome and identify RSE. Uric acid is one of well-known natural anti-oxidants, ROS/RNS scavengers. Because prolonged seizures can result in increasing ROS/RNS production in neuronal cells, we hypothesized uric acid would be consumptive and uric acid level was lower in persistent on-going electrical hyper-excitability patient than in subsided patient after up to second line treatment. We also evaluated predictable value of readily established parameters. **Method:** We prospectively collected status epilepticus patients who admitted to the Ajou university medical center. Blood sampling for CRP, albumin and uric acid (UA) was performed when patients were arrived at ER and 24 hours after admission and STESS was measured. All patients were treated based on SE management guideline. After second line AED treatment, continuous EEG was performed at least 24 hours. Expert epileptologist interpreted cEEG according to 2012 ACNS guideline without any clinical information. On-going group was defined as unequivocal electrographical seizure or periodic discharges on cEEG and subsided group was defined as none of above features on cEEG. Δ UA was calculated as follows: $((\text{UA at 24h} - \text{initial UA}) / \text{initial UA}) * 100$ **Results:** There were 26 patients during enrollment period. Seven patients were female, mean age was 51-year-old. Median STESS was 3, initial mean values of CRP, albumin and UA were as follows; CRP: 1.68 ± 3.36 , albumin: 4.14 ± 0.58 and UA: 5.64 ± 3.01 . There were 15 patients in on-going group (10 patients showed electrographical seizure and 5 patient showed PD). There was no significant differences of age, etiology, CRP and al-

bumin levels between on-going and subsided group. On-going group showed higher STESS (2 ± 0.894 vs 3.67 ± 1.67 $p=0.004$), lower UA at 24h (6.25 ± 2.50 vs 2.34 ± 2.09 , $p<0.0001$) and increased Δ UA ($+2.63 \text{ mg/dl} \pm 37.01$ vs $-52.27 \text{ mg/dl} \pm 28.09$, $p<0.0001$) compared to subsided group. Furthermore, STESS, UA at 24 hours and Δ UA significantly predicted electrographical seizure or PD (STESS; AUC: 0.824, $p=0.005$, UA at 24 hours; AUC: 0.927, $p<0.001$ and UA level change; AUC: 0.939, $p<0.001$) but not CRP and albumin. Cut off point of 3 in STESS showed sensitivity 80%, specificity 81.8% and OR 5.25 (95% CI: 1.39-19.73), cut off point of 3.0 mg/dl in UA at 24h showed sensitivity 86.7%, specificity 90.9% and OR 13.09 (95% CI 2.03-94.41). Cut off point of 32% decrease in Δ UA showed sensitivity 93.3%, specificity 90.9% and OR 13.66 (95% CI: 2.03-91.42). **Conclusion:** This study confirmed predictable value of STESS in SE and showed value of UA level to reflect on-going electrical hyperexcitability after second line AED treatment in status epilepticus patient. Future larger scale study is warranted to confirm and validate the predictive value of UA level in status epilepticus.

P-1-122

Emboic stroke related to bradyarrhythmia after intravenous infusion of fosphenytoin

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Background & Significance: Fosphenytoin was introduced as an alternative to phenytoin because of safety from cardiac toxicity. But there were some reports of cardiac toxicity such as bradyarrhythmia and cardiac arrest. We experienced a case of embolic stroke related to bradyarrhythmia after fosphenytoin infusion. **Case:** A 53-year-old man presented with repetitive seizures and stuporous mental status. He had history of recurrent epileptic seizures but had no regular medication. He had any other medical disease such as cardiac arrhythmia except untreated epilepsy. Electroencephalogram (EEG) was consistent with right temporal status epilepticus. Initial electrocardiogram (ECG) and brain magnetic resonance image (MRI) were normal. We infused lorazepam 4 mg intravenously but seizure relapsed, so we prescribed 800mg phenytoin equivalent of fosphenytoin to be administered at 30 PE/min via intravenous pump. After fosphenytoin infusion there were no more seizure clinically and electroencephalographically. After 12 hours intravenous fosphenytoin infusion, he developed sudden sinus bradycardia dropped to 30/min and hypotension dropped to 50/30 mmHg. After norepinephrine infusion, his blood pressure and heart rate were recovered in 3 hours. There was no causes of sudden hemodynamically unstable bradycardia such as electrolyte imbalance, proarrhythmic drugs, and structural disease of heart confirmed by echocardiography. In spite of seizure termination, his mentality was not recovered. EEG revealed consistent generalized theta slow activity. MRI taken 5 days after admission showed multiple diffusion restriction lesions over both cerebral hemisphere (Fig. 1). Follow up ECG taken after recovery showed no more bradycardia and any other abnormal findings such as ST elevation. 10 days after admission, his mentality was improved. **Conclusions or Comments:** This case indicates fosphenytoin may cause hemodynamically unstable bradyarrhythmia and subsequent embolic cerebral infarction even 12 hours after fosphenytoin infusion with recommended dose and infusion rate in patient without any premorbid cardiac disease. We recommend that clinicians consider more intensive and prolonged cardiac monitoring when infusing intravenous fosphenytoin.

P-1-123

Transient Blood-Brain Barrier disruption after cerebral concussion

which showed good prognosis

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Background & Significance: Traumatic brain injury(TBI) can present variety of neurologic deficits, such as mental deterioration, seizure, hemiparesis, dysarthria and other many cortical dysfunction. the severity of patient's neurologic symptoms rely on the extents and site of brain lesion, which can be visualized in radiologic brain imaging such as brain CT and MRI. We reported one case of a patient who showed only transient Blood-Brain Barrier(BBB) disruption after cerebral concussion which showed good prognosis. **Case:** A 63 years old female arrived to the emergency room, showing transcortical mixed aphasia and right sided hemiparesis(MRC grade IV) after falling down with cerebral concussion. Brain CT was immediately done, but no hemorrhage was revealed. In series, brain MRI was performed. In diffusion weighted image(DWI)-MRI, several tiny restricted diffusion lesions were found in left hemisphere. In enhanced T1 images, diffuse parenchymal enhancement was visible in left hemisphere, which suggests early BBB disruption. She was hospitalized in diagnosis of TBI on basis of history of cerebral concussion and BBB disruption. After admission, her neurologic symptoms(aphasia, hemiparesis) were gradually improved. At hospital day 5, she didn't show any neurologic deficit. Follow-up MRI was performed, which diffuse BBB disruption and several tiny diffusion restriction lesions found in the previous study were normalized. After observation for more days, she did not show any other symptoms, so was discharged. **Conclusions or Comments:** BBB disruption or breakdown has been used as prognostic factors for hemorrhagic transformation or massive hemorrhage in acute ischemic stroke, progression of hemorrhagic lesion and risk of seizure or cortical dysfunction in TBI. But in this case, which reveals no hemorrhage and only BBB disruption, showed good prognosis and complete remission of symptoms and radiologic findings. We reported this case for good example of transient BBB disruption showing good outcome.

P-1-124**Posterior Leukoencephalopathy with Reversible Cerebral Vasoconstriction Syndrome after Blood transfusion; a case report**

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Background & Significance: Posterior leukoencephalopathy (PLE) is a clinicoradiologic disease entity and caused by altered cerebral vasculature such as hypertensive encephalopathy, reversible cerebral vasculature syndrome (RCVS), posterior reversible encephalopathy syndrome (PRES), and migraine. Sometimes, PLE is regarded as the radiologic presentation of RCVS. Clinical feature of PLE is characterized by headache, altered mental status, seizure, visual loss, and motor deficit. PLE is associated with the systemic condition such as hypertension, eclampsia, cytotoxic agent application such as cyclosporine and rarely, blood transfusion. We report a patient with repetitive seizure and headache after blood transfusion, finally she was diagnosed as PLE with RCVS. **Case:** A 50 years old woman was transferred to hospital because of repetitive generalized seizures. Recently, she got massive blood transfusion for 2 days (1200cc per day) for correction of severe anemia (hemoglobin 2.8 g/dL) caused by menorrhagia from uterine adenomyolysis. At the time of discharge, hemoglobin level elevated up to 10.9 g/dL and her general condition became stable except for mild headache, which initiated during blood transfusion. 7 days after discharge, she had her first seizure attack during sleep. After twenty minutes, she had several seizure attacks more. On admission, blood pressure was 90/60 mmHg, pulse rate 105/min, respiration rate 20/min, and body tem-

perature was 36°C. With injection of lorazepam and infusion of phenytoin intravenously, seizure stopped and mentality recovered to normal. However, she started to complain of periodically repetitive headache. Headache lasted 2-3 hours and disappeared without medication. Magnetic resonance T2 weighted images showed high signal intensity in bilateral parietal, temporal and partial frontal lobe involvement without diffusion weighted image restriction. Magnetic resonance angiography (MRA) during asymptomatic period revealed no steno-occlusive lesion, on the other hand, brain computed tomography angiography (CTA) during symptomatic period showed multifocal stenosis in bilateral middle cerebral arteries. Based on the clinical presentation and angiography findings, we diagnosed her as PLE with RCVS. She was treated with verapamil, and headache rapidly improved. She had no headache any more after taking verapamil for 2 months. **Conclusions or Comments:** This case represent PLE with RCVS caused by massive blood transfusion in a short time. PLES is relatively common in asian, middle-aged, women and 87% of patient accompanied cerebral vasoconstriction or focal angiopathy. The pathophysiology of PLE caused by blood transfusion still unclear but failure of cerebrovascular autoregulation and endothelial dysfunction, and hypermagnesemia induced calcium reflux that cause cerebral vasospasm could contribute to. Rapid increase of hemoglobin and hematocrit may have affected the reactivity of cerebral vasculature despite the normal blood pressure. Disturbed cerebral blood flow autoregulation cause vasoconstriction or vasodilation and that cause mixture of vasogenic and cytotoxic edema predominant in posterior regions. PLES also implicates endothelial dysfunction especially in case without hypertension, leading to blood brain barrier destruction that leads brain capillary leakage which may trigger vasogenic edema. MRA findings of reversible, irregular narrowing of large and medium-sized arteries consistent with vasospasm frequently observed. Neuroimaging maybe the first clues to the diagnosis of PLE. Middle-aged women with blood transfusion should be observed well due to the occurrence of PLES at least 7-10 days after transfusion. It is also important to consider the possibility of PLES when the patients suffer from recurrent seizure, headache, and altered mentality.

P-1-125**Intracranial cortical calcifications in a epilepsy patient with pseudo-hypoparathyroidism**Ye Seol KIM¹, Ji Hyung PARK¹, Kyung Jin HWANG², Dae Lim KOO³, Dae Young KIM⁴, Dae Won SEO¹*¹Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, ²Department of Neurology, Kyung Hee University School of Medicine, ³Department of Neurology, Seoul National University Boramae Medical Center, ⁴Department of Neurology, Chungnam National University*

Background & Significance: In chronic hypocalcemia, intracranial calcification is often accompanied. The distribution of intracranial calcification is usually limited to subcortical area like basal ganglia (BG), thalami and white matter (WM). **Case:** We report a case of a newly diagnosed epilepsy patient with pseudohypothyroidism who had intracranial calcification in cerebral cortices in addition to the usual distribution. He was recently diagnosed with hypocalcemia and his blood lab at admission shows high levels of PTH and phosphorous and low level of calcium and 25-hydroxyvitamin D. Brain MR showed calcification in left temporal and bilateral occipital cortices in addition to the usual distribution of BG and subcortical WM. Video EEG monitoring revealed that he had a partial seizure with the ictal EEG mainly consist of background attenuation and muscle artifacts. The interictal EEG showed multiple focal spike-wave discharges. After the monitoring, he was given calcium and calcitriol supplement besides the levetiracetam he had been taking previously. His seizure quickly discontinued and he had become seizure free one month after starting the supplements. His calcium level rose from 5.2 to 6.8 mg/dl and

phosphorous decreased from 6.6 to 6.2 mg/dL at month 1, 7.8 and 4.5 mg/dL at month 3. At month 3, 25 hydroxyvitamin D level increased from 12.5 to 15.8 ng/mL and PTH level also increased from 149.0 to 187.4 pg/mL. **Conclusions or Comments:** This was first case to show cortical deposition of calcium in chronic hypocalcemia due to hypoparathyroidism and its probable relationship to the cause of hypocalcemic seizure. No lateralization of Ictal EEG and the multiple distributions of the interictal epileptiform spikes suggest multifocal cortical disruption by calcification some of which might be too microscopic to be seen in current brain imaging modalities. With more advanced protocols like SWI MRI, it may be possible to visualize cortical calcification in hypocalcemic patients for evaluation of seizure burden.

P-1-126

Diffuse hemispheric edema in an epilepsy patient

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Background & Significance: Seizure-induced cerebral edemata are characteristically localized on the cortical and subcortical areas. In spite of the characteristics, it is difficult to distinguish them from other conditions such as ischemic strokes and encephalitis by brain imagings. We observed a diffuse hemispheric edema in an epilepsy patient who was found out in a state of loss of consciousness (LOC). When her mental state was recovered, we noticed that she had left hemiparesis and global aphasia. **Case:** A 66 years old female with epilepsy was found out in a state of LOC. When she was delivered to the emergency room, her mental state was recovered but she was in the state of right hemiparesis and global aphasia. On a brain computerized tomography (CT) study, diffuse edema was observed in the left hemisphere but there was no signal change. The clinical manifestation and CT finding made us have impression of an early stage of diffuse cerebral infarction involving multiple arteries of the left hemisphere. We did an emergency magnetic resonance imaging (MRI) study for the preparation of an intervention therapy for acute strokes. The brain MRI revealed multiple high-signal intensities of cortical areas on the diffusion-weighted study and those of cortical-subcortical areas on the fluid-attenuated inversion recovery study. The edemata were widely distributed within the left hemisphere. Despite the diffuse distribution of hemispheric edemata, the hemiparesis and aphasia were recovered after the MRI study. An electroencephalography taken after the MRI did not show any epileptiform discharges and just disclosed polymorphic slow waves on the left hemisphere. Because of the subcortical and cortical distribution of the edemata and the early recovery of hemiparesis and aphasia, we assumed the lesions as seizure-induced cerebral edemata. **Conclusions or Comments:** When epilepsy patients are found in a state of LOC. It is impossible to know the exact cause of LOC at initial examination. In spite of that the state can be a postictal phenomenon; other conditions including strokes should be considered as possible causes of the state. In this patient, the diffuse hemispheric edema on the CT, and postictal hemiparesis and aphasia lead us to make misunderstanding as diffuse cerebral infarction of the left hemisphere initially. We have to be more careful to discern between seizure-induced cerebral edemata and acute ischemic strokes in epilepsy patients with postictal neurologic deficits.

P-1-127

Reversible splenial lesion syndrome following sudden withdrawal of antiepileptic drugs

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Background & Significance: Reversible splenial lesion of corpus callosum are demonstrated in the several situation including viral encephalitis, seizure, malnutrition, antiepileptic drug withdrawal and metabolic disturbances such as hypoglycemia or hypernatremia. The typical magnetic resonance imaging features are focal hyperintensity on T2 and FLAIR images in the splenium of corpus callosum. Disconnection syndrome is not most common feature of these patients. Its clinical symptom was reported earlier not only confusion and psychiatric symptom is common clinical findings, but also ataxia, increased muscle tone and hallucination would accompanied. Here in, we present a reversible splenial lesion of patient due to suddenly discontinued antiepileptic treatment. **Case:** A 31-year old female patient with a 15-year history of auditory hallucination was referred to our outpatient clinic. In prior to illness, she was so good at social life that she attended a college. However, at the first time to admission she presented mutism and no responsiveness even though she was alert. The neurological examination was not easily obtained because conversation was practically impossible and she have been generalized tremor after she encountered traffic accident 20-years ago. There was no history of diabetes, hypertension, malnutrition and systemic infection. She only takes anti-epileptic drug (carbamazepine 400mg) for a long time. Carbamazepine was discontinued 2 weeks ago before admission because of her general weakness. Mutism was occurred at 4 days after the discontinuation of antiepileptic drugs. Brain MRI was performed 10 days after disease onset and revealed lesion involving the splenium of corpus callosum, hyperintensity on T2, and FLAIR sequences, and hypointensity on T1 weighted image. The lesion was hyperintense on diffusion weighted image, and hypointense on the ADC map. Serum laboratory findings including glucose, sodium and vitamin B12 was normal. CSF profile was revealed no specific findings and free of influenza and herpes virus antigen by CSF polymerase chain reaction (PCR). EEG was observed excessive fast activity which means taking benzodiazepine. We maintain discontinuation of carbamazepine for 2 weeks after she admission, mutism was slowly in remission. A follow-up MRI was performed 1 month after disease onset and showed complete resolution of the lesions. **Conclusions or Comments:** Different causes associated splenial injury including vitamin deficiency, metabolic disturbance, viral infection and pathophysiology of reversible focal splenial lesion have not been clarified the mechanism of cytotoxic edema. Although several hypothesis was suggested but, it was not consistent in all the cases. Anti-epileptic drug (AED) related reversible splenial lesion syndrome is occurred with both classic and new AED, however, direct toxicity of AED seems to be not related and most often observed after withdrawal of AED. It is also occurred in non-epileptic patients receiving AED therapy and not seem to be related type and duration of seizure. In other reports, most frequently associated AED was carbamazepine, phenytoin and lamotrigine, in order of frequency. Splenial lesion is observed between as early as 24 hours and 1 week after AED withdrawal and disappeared usually within 1-2 weeks. Clinically complete recovery is definite and take place a few weeks after MRI normalized.

P-1-128

Myoclonic status epilepticus in adult patients without a previous history of epilepsy: a report of three cases

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Background & Significance: Myoclonic status epilepticus (MSE) has been described in generalized epilepsy syndromes, neurodegenerative diseases, toxic-metabolic state, and following anoxic brain injury. We described clinical features of 3 adult patients with non-postanoxic MSE who had no definite history

of seizures. **Case:** Patient 1) A 77-year-old woman visited ER presenting with continuous irregular myoclonic movements in her face, neck and upper extremity for 2 days. She had been taken medicines benzotropine and alprazolam for resting hand tremor seven years ago and some drugs including formoterol (bronchodilator, β_2 agonist) ofloxacin for URI symptoms ten days ago. Neurological examination revealed the patient to be alert and no focal neurological deficit. Laboratory tests and diffusion weighted imaging MRI showed unremarkable. The EEG showed bilateral synchronous spikes or polyspike accompanying generalized jerking movements. We stopped formoterol and ofloxacin and treated her with intravenous valproic acid (1000mg/day). The next day, her myoclonic seizures completely disappeared and follow-up EEG revealed neither epileptiform discharges nor abnormal slow waves. Patient 2) A 76-year-old woman presented with irregular myoclonic twitching in face, shoulder and arm without impairment of consciousness one day ago. She denied history of previous seizures and morning jerks. She had a history of ischemic heart disease and had taken medications including nicorandil, aspirin, clopidogrel and pravastatin. Her brain MRI showed cerebral atrophy and chronic ischemic lesions, but no acute abnormality. The laboratory test were unremarkable. The EEG revealed very frequent bursts of high voltage polyspikes accompanying generalized jerk movements. She was started with IV valproate (800mg) and switched to oral valproate (900mg per day). After IV valproate loading, her myoclonic seizures nearly disappeared. Patient 3) A 36-year-old man visited ER presented with continuous generalized myoclonic movements one day ago. He did not have a prior diagnosis of epilepsy, but had suspicious history of left arm jerking movements in late adolescence. His brain MRI and laboratory test showed no abnormality. The EEG revealed very frequent generalized spikes and polyspikes, which were associated with generalized myoclonia. Photoc stimuli aggravated epileptiform discharges and seizures. He received with IV lorazepam (2mg), and then he received with oral valproate (1000mg per day) and levetiracetam (1000mg per day). Two day later, his myoclonic seizures were nearly diminished. **Conclusions or Comments:** MSE in patients without epilepsy is a rare event and usually related with drugs. In these cases, etiology of MSE was unknown (cryptogenic, patient 2) or MSE may have occurred in the setting of remitted juvenile myoclonic epilepsy (patient 3).

P-1-129

Seizure disorder concurrent with acute ischemic stroke: single hospital experience

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Background & Objectives: The incidence of early-onset seizures was found to be between 2.4% and 6%. However, the exact frequency of seizure at stroke onset is not known, except that one study showed a frequency of 5.7% with single or recurrent seizures at stroke onset. The aim of this study was to investigate the clinicoradiological characteristics of cases with seizure disorder concurrent with acute ischemic stroke. **Method:** A total of 8 seizure patients had been retrospectively analyzed who were simultaneously diagnosed with acute ischemic stroke in CHA Bundang Medical Center from 2005 to 2015. Acute ischemic stroke was confirmed by diffusion-weighted image (DWI) of brain magnetic resonance image. Case with previous history of seizure and those who had other etiologies including metabolic and toxic causes without the presence of DWI-positive lesion was excluded in this study. **Results:** The average age of total eight patients was 71 years old and five patients were men. Six of patients had an old CVA history and the type of CVA was all ischemic strokes. Four cases had anterior, two cases posterior and two cases anterior/posterior circulation territory acute ischemic lesions. Only three cases, including one cerebral venous thrombosis, had one lesion site and the other five

cases had multifocal lesion sites. On the evaluation of ictal semiology and EEG, generalized tonic-conic seizure (GTC) was found in 6 patients and the remaining two cases showed simple partial seizure. All patients were well controlled with antiepileptic drugs and discharged. **Conclusion:** The clinicoradiological characteristics of seizure disorder concurrent with acute ischemic stroke were not different with previously reported early-onset seizure, but, we presumed that these cases have better clinical outcome than others. However, this study has a small sample size, so then, additional studies are required to clarify the seizure disorder concurrent with acute ischemic stroke.

P-1-130

Correlation of EEG and MRI in Sporadic Creutzfeldt-Jakob disease

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Background & Objectives: Creutzfeldt-Jakob disease is a rapidly progressive, fatal, transmissible neurodegenerative disorder caused by prion protein. Electroencephalography (EEG) revealed typical periodic synchronous discharge (PSD) and the brain magnetic resonance imaging (MRI) showed high signal intensities in cerebral cortex and basal ganglia on diffusion weighted images (DWI). We investigated the correlation of EEG and DWI on brain MRI in patients with sCJD. **Method:** We collected 6 cases with the diagnosis of sCJD admitted to our neurology department between July 2005 and July 2015. The EEG and DWI patterns in brain MRI in sCJD were compared in all patients. **Results:** The mean age of patients was 66 years (range: 38-83) and there was female predominance (four patients). All patients showed cognitive disturbance upon admission. The other symptoms were ataxia (5/6), myoclonus (1/6), and extra-pyramidal symptoms (1/6). One patient had history of psychotic symptoms manifesting as hallucination and delusion. All patients were examined the EEG and DWI at 3 to 8 weeks from onset. In four patients, unilateral or bilateral maximal amplitude site of typical PSD in EEG and lesion site on DWI were compatible. In these cases, all four patients have definite unilateral or bilateral basal ganglia lesion on DWI. However, in the two remaining patients, EEG and MRI findings were not compatible. Although one patient have bilateral hemispheric cortical hyperintense signal change on DWI, EEG did not showed typical PSD. In this patient, there was no basal ganglia lesion on DWI. Although the other patient widespread left frontally dominant cortical lesion with left basal ganglia, EEG showed bilaterally typical PSD in EEG. In this patient, left midline frontal lesion adjacent corpus callosum might induce bilaterally synchronized frontally dominant PSD in EEG. **Conclusion:** To construct typical PSD in EEG in sCJD, it is necessary to involve basal ganglia on MRI. This finding is in good accordance with recent hypothesis that the functional integrity of the subcortico-cortical network is a prerequisite for the generation of PSD. Further study is needed to ascertain this finding by reviewing more patients in sCJD.

P-1-131

Efficacy, tolerability, and pharmacokinetics of fosphenytoin loading in patients with subarachnoid hemorrhage

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Background & Objectives: Prophylactic use of antiepileptic drugs is common in subarachnoid hemorrhage (SAH), and fosphenytoin is frequently used for the rapid delivery of phenytoin in SAH patients. The present study was performed to investigate the safety, tolerability, and pharmacokinetic profiles of rapid intravenous loading of fosphenytoin in SAH patients. **Method:**

Fosphenytoin was administered intravenously at a single loading dose of 20mg phenytoin-equivalent (PE)/kg with an infusion rate of 150mgPE/min to 30 adult patients with SAH, who experienced seizures or had a clinical suspicion of nonconvulsive seizure. Plasma concentrations of total phenytoin and free phenytoin were determined, and adverse events were assessed at 0, 10, 20 minutes and 24 hours after the infusion of fosphenytoin. **Results:** Four patients experienced transient lowering of blood pressure, but other adverse events were not observed. All patients reached the therapeutic level of free phenytoin (1-2mg/L) at the end of infusion, but most patients (28/30) reached markedly supratherapeutic range with mean plasma concentration was 17.7 ± 8.13 mg/L. The higher plasma concentration maintained to 20 minutes after infusion (mean concentration; 3.46 ± 3.75 mg/L). At 24 hours after loading, a majority of patients (25/30) maintained within therapeutic range of free phenytoin. **Conclusion:** Rapid intravenous loading of fosphenytoin is well tolerated and effective in prompt achieving the therapeutic level of free phenytoin, but most patients experienced overshoot of free phenytoin at the end of infusion. Because increased plasma concentrations may increase the risk for cardiovascular complication, additional studies would be needed to find out the optimal dose and infusion rate of fosphenytoin in SAH patients.

P-1-132

A case of wernicke encephalopathy presenting subclinical seizure

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Background & Significance: Wernicke's encephalopathy (WE) may be present in the general population with a prevalence of around 2%, and is considered underdiagnosed. Because many cases are in patients who do not have symptoms presumed to be associated with WE. WE is characterized by the triad ophthalmoplegia, ataxia, and confusion. However, only 10% of patients exhibit all three features, and other symptoms, including seizure may also be present. Furthermore nonconvulsive seizure is extremely rare in WE. Here, we report a case of WE presenting subclinical nonconvulsive seizure. **Case:** A 72-year-old-woman who was previously diagnosed gastric cancer with pancreatitis admitted because of she complained poor oral intake and nausea, vomiting for a month. She underwent gastric endoscopy at a local medical center, and transfer to our hospital due to failing to supply a scope is passed through a stenosis gastric outlet obstruction. At 3 days after admission, she was confusion mentality and slow verbal response to answer. But the other neurologic examinations were unremarkable. We have done electroencephalogram (EEG), brain enhanced magnetic resonance imaging for differential diagnosis. The MRI showed that bilateral symmetric diffusion high signal intensity with T2 signal change in both medial thalamus without abnormal enhancing lesion in the brain. The EEG showed continuously, regularly irregular sharp waves were appeared on T3, C3 foci with spreading to the right side, and symmetric medium to high voltage 5-6Hz theta wave background. Gastric endoscopic findings were recurrence of gastric cancer with gastric outlet obstruction on stomach, previously operated state (subtotal gastrectomy with Billroth II & Braun anastomosis state). She was treated with thiamine and valproic acid intravenously and then change to levetiracetam oral. Clinical symptom and EEG finding were improved after treatment. **Conclusions or Comments:** Typical symptoms were not common in PW, especially non-convulsive seizure is very rare. So we consider this finding in WE with alteration of mentality.

P-1-133

A case of recurrent postictal bilateral facial petechiae without subconjunctival hemorrhage

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Background & Significance: The postictal state is characterized by drowsiness, confusion, nausea, hypertension, headache or migraine. The other symptoms associated such as amnesia or other memory defects, Todd's paresis, postictal psychosis are less common. Furthermore, skin or ophthalmic change is very rare. And such cases were rarely reported in the world widely. In general, skin petechial hemorrhages over face, neck and chest, as well as conjunctival hemorrhage, are due to capillary bleeding. We reported the case of a patient with recurrent postictal skin change. **Case:** A 18-year-old man who patient with complex partial seizure with 2ndary generalization visited in our hospital because left temporal lobe epilepsy since childhood. He has taken with levetiracetam 2,000mg/day with oxcarbazepine 750mg/day. But he experienced a seizure attack each 1 to 2 per month to 1per 2 month. He complained stereotyped, both facial, cheek and periorbital purpuric eruptions without subconjunctival hemorrhage that occurred after secondarily generalized tonic-clonic seizures, which resolved in 24 to 48 hours after a seizure attack. **Conclusions or Comments:** We report a rare case of recurrent postictal bilateral facial petechiae without subconjunctival hemorrhage.

P-1-134

Focal non-convulsive status epilepticus manifested as an antegrade amnesia

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Background & Significance: Non-convulsive status epilepticus is defined as seizure activity seen on electroencephalogram (EEG) without clinical findings associated with generalized convulsive status epilepticus. Usual clinical feature includes confusion or severely impaired mental status. Limbic encephalitis predominantly involves hippocampus and parahippocampal structures which have crucial role in memory registration. Memory impairment is common sequelae of limbic encephalitis. We report a woman with persistent memory impairment after recovery from limbic encephalitis and status epilepticus **Case:** A previously healthy 38-year-old right-handed woman presented with a first-ever generalized seizure. At admission, she was alert and well-oriented. Subsequently she had repeated generalized seizures and her consciousness had worsened gradually to stuporous state. Electroencephalography (EEG) demonstrated repetitive non-convulsive seizures originating from the right anterior temporal area (Figure 1). Brain magnetic resonance imaging (MRI) showed subtle signal increment in bilateral mesial temporal lobes (Figure 2). Cerebrospinal fluid (CSF) analysis showed mild lymphocytic pleocytosis. She was diagnosed as a status epilepticus with probable limbic encephalitis. Antiepileptic treatment including anesthetic dose of midazolam continuous infusion were undertaken under continuous EEG monitoring. Empirical antiviral treatment and immunomodulating therapy including high-dose corticosteroid were also undertaken. Her electrographic seizures were terminated 62 hours after the onset. Midazolam continuous infusion was continued for 12 more days until periodic discharges in the right anterior temporal area disappeared. She recovered from the coma 18 days after the initiation of antiepileptic treatment, 4 days after discontinuation of midazolam coma therapy. After 24 days of treatment, her neurologic deficits were completely recovered except one. She suffered from persistent modest antegrade amnesia. Neurocognitive testing indicated significant impairment in the memory domain (0.12 %ile) while other domains were relatively preserved (Table 1). A follow up EEG was unremarkable. A follow up MRI showed slightly high signal intensity in bilateral mesial temporal lobes which were not different from

the initial MRI. Positron emission tomography (PET) using Tc99m-fluorodeoxyglucose (FDG-PET) revealed focal hypermetabolism in the body of the right hippocampus (Figure 3). We concluded that the antegrade amnesia was a manifestation of persistent focal seizure activity and antiepileptic drugs were adjusted to control the focal seizure activity **Conclusions or Comments:** Memory impairment and antegrade amnesia is one of most common neurologic sequelae of the limbic encephalitis. However, uncontrolled focal seizure activity can present as a memory impairment and PET or single photon emission computed tomography (SPECT) could be helpful in discriminating focal non-convulsive status epilepticus from sequelae of hippocampal injury after the limbic encephalitis

P-1-135

Positive sharp waves in the Electroencephalography of Adult patient with Posterior Reversible Encephalopathy Syndrome

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Background & Significance: Positive sharp waves (PSWs) have been extensively studied in the electroencephalography (EEG) of the neonate. However, there is a paucity of data regarding the significance of this EEG activity in adults. We reported a case that exhibited epileptic discharges with positive polarity in EEG of an adult patient with posterior reversible encephalopathy syndrome. **Case:** A 60-year-old woman with ovarian adenocarcinoma had a treatment plan with three cycles of 'bevacizumab + doxorubicin' regimen. The patient had no history of hypertension. After the second cycle of chemotherapy, the patient presented with both eye blinking with confusion and developed stupor followed by generalized tonic-clonic seizure. Diffusion-weighted image on brain MRI showed hyperintensities in the cortex and subcortical white matter of bilateral parieto-occipital lobes. A first EEG was performed showing continuous spike and wave complexes with negative polarity in left occipital areas, consistent with the diagnosis of status epilepticus. 2000mg loading dose of intravenous levetiracetam was administered but without complete resolution of EEG abnormalities. On hospital day 4, periodic discharges with PSWs were seen in the continuous EEG monitoring and the patient was still drowsy. After more appropriate treatment with antiepileptic drugs (topiramate 400mg bid, levetiracetam 1000mg tid, lacosamide 50mg bid, gabapentin 800mg tid), PSWs disappeared and the mental status also recovered compared to the previous status. **Conclusions or Comments:** In our case, PSW may be generated by the product of excitatory post-synaptic potentials (EPSP's) in the deep cortical areas or horizontal dipoles from gyri located in the medial occipital areas. PSW is a rare and probably under-reported electroencephalography (EEG) abnormality which, similar to negative epileptiform discharges, signifies focal epileptogenicity in adult patients.

P-1-136

A case of status epilepticus after locoregional anesthesia techniques with lidocaine

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Background & Significance: Lidocaine, a widely used local anesthetic, crosses the blood brain barrier rapidly and has been recognized as a dose-dependent proconvulsant drug. Seizures caused by lidocaine intoxication have been reported in various situations, such as intravenous, topical or regional injection for control of pain and arrhythmia. Locoregional anesthesia techniques are increasingly used for cataract surgery. We present a patient who developed a gen-

eralized clonic seizure following retrobulbar injection of lidocaine and bupivacaine for phacovitrectomy to treat macular hole. **Case:** A 64-year-old woman with macular hole was scheduled for phacovitrectomy. She had mild tricuspid valve regurgitation but no history of epilepsy, allergy, and psychiatric problems. Twenty minutes after retrobulbar injection of lidocaine and bupivacaine, she developed a generalized clonic seizure with loss of consciousness, tachycardia and cyanosis. The seizure lasted for a minute and ceased spontaneously. After awakening, she had confusion, drowsiness, and amnesia. A neurological examination noted no deficits. Electroencephalogram showed no epileptiform discharges and brain MRI revealed no abnormal lesions to provoke a seizure. She developed no further seizures. **Conclusions or Comments:** Various complications can occur during locoregional anesthesia techniques with lidocaine. As seizures or status epilepticus may occur after locoregional injection of usual dosage of lidocaine, physician have to pay attention to the condition of patient after injection of lidocaine and start appropriate treatment immediately if seizure occurs.

P-1-137

Intermittent lorazepam injection with magnesium infusion as alternative therapeutic strategy in refractory status epilepticus: a case report

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Background & Significance: Refractory status epilepticus (RSE) defined as continuous clinical and/or electrical seizure after second line AED treatment is one of main causes for neurological mortality and morbidity. Treatment of RSE is still challenging and evidence-free zone to establish solid treatment strategy despite recent guideline was published. Main therapeutic modality of RSE is continuous anesthetic infusion to stop seizure or to achieve burst-suppression pattern on EEG. There has been obstacles to start continuous anesthetic infusion in RSE patient because frequent cardiopulmonary depression that needs endotracheal intubation, mechanical ventilation and inotropic agent. Here we report that treatment of RSE with intermittent benzodiazepine injection and intravenous magnesium infusion rather than continuous anesthetic infusion **Case:** A 83-year-old female patient was admitted for acute onset involuntary movement of left extremities with decreased mental status. At emergency room, she was stuporous mental state with continuous left arm clonic movement and left facial twitching. She was injected with intravenous benzodiazepine immediately after arrival. Her clonic movement was stopped after intravenous benzodiazepine in ER. Unfortunately after several minutes, clonic movements reappeared. Therefore, on-call neurologist decided to load fosphenytoin. Her clonic movement re-stopped after intravenous fosphenytoin loading. After stopping clonic movements, electroencephalogram (EEG) was taken. There was no clinical overt seizure, although, EEG showed frequent electrographical seizures from right posterior quadrant head region during tracing. Thereafter she was treated with another intravenous antiepileptic drugs, levetiracetam. Just after intravenous levetiracetam loading, EEG seizures were disappeared. After disappearance of EEG seizure, MRI of her brain were taken, and showing cortical diffusion restriction over right parietal cortex. Continuous EEG was taken after MRI acquisitions and cEEG showed reappearance of EEG seizure from right posterior quadrant head region. Therefore, at this time point, she could be diagnosed with refractory status epilepticus. Establishing refractory status epilepticus diagnosis, we decided to induce coma using continuous midazolam infusion. Despite severity of her refractoriness of status epilepticus, her family didn't accept medication induced coma with endotracheal intubation because of her old age and poor pre-morbid state. After discussion with her family, we decided to treat refractory

status epilepticus without continuous anesthetic infusion and endotracheal intubation. Thereafter we treated her with intermittent scheduled lorazepam injection with gradual tapering and intravenous magnesium sulfate infusion followed by twenty-four hours maintenance of magnesium infusion according to FAST- MAG trial protocol. Fortunately, her EEG seizures were disappeared just after magnesium sulfate infusion with lorazepam injection. Despite magnesium infusion, there was no cardiopulmonary depression. Next-day, she was able to follow simple command and 2 days later, she recover to premonitory state. **Conclusions or Comments:** This case shows efficacy of intermittent lorazepam injection with magnesium infusion to treat RSE and suggests this treatment method as an alternative therapeutic strategy to manage RSE patient

P-1-138

Usefulness of perfusion MRI to monitor encephalopathy of presumed autoimmune etiology: a case report

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Background & Significance: Recent advances of neuroimmunology have found many autoantibodies to neuronal surface antigens or onconeural antibodies in encephalopathy patient. Despite diagnosing encephalopathy of autoimmune etiology, there has been no definite bio- or neuroimaging- markers to reflect disease state or severity except titration of autoantibodies from cerebrospinal fluid and/or serum. Here we report encephalopathy patient caused by presumed autoimmune etiology with dynamic changes of perfusion MRI in response to immunotherapies. **Case:** A 69-year-old male patient was admitted to Ajou university medical center due to decreased mental status. Three weeks before admission to our hospital, he admitted other tertiary hospital because of acute mental change. During previous admission, he was undertook brain MRI, CSF analysis and other etiology work up for acute mental change. Despite thorough evaluation, there was no definite etiology to explain his mental change except diffuse cortical atrophy and his neurological status didn't change until admission to our hospital. At admission in our hospital, he was drowsy and showed partial global aphasia. During admission he showed intermittent right beating nystagmus with right side lip twitching. Thereafter we checked EEG and found frequent electrographic seizures from left occipital area and re-checked brain MRI. Brain MRI revealed high signal intensity area over left hemisphere especially posterior head region. Because there was no other definite cause to explain his symptoms, he could be diagnosed with presumed autoimmune encephalopathy with partial seizures. We injected intravenous levetiracetam to treat partial seizure, intravenous methylprednisolone and immunoglobulin to treat presumed autoimmune encephalopathy. Despite five days treatment with intravenous immunoglobulin and steroid, his symptoms didn't changed. Furthermore he developed different seizures manifesting as left side automotor seizures. Assuming progression of underlying encephalopathy, we re-evaluated him with brain MRI. Follow up brain MRI showed similar extent of high signal area, except increased perfusion on left hemisphere. He was undertook continuous EEG after MRI and cEEG showed frequent seizures from left central area. Confirming increased perfusion on lesion side and electrical seizure, we started continuous midazolam infusion and second line immunotherapy with rituximab. After continuous midazolam infusion, his seizures were disappeared and 72 hours after infusion, we slowly tapered midazolam infusion. After second cycle of rituximab, his mental state was improved. He opened his eyes spontaneously and appropriately responded to verbal command. To check his brain lesions, follow up brain MRI was taken and showed normalized perfusion on lesion side, left hemisphere. **Conclusions or Comments:** This case showed perfusion MRI could reflect disease state of presumed autoimmune encephalopathy and suggests perfusion

MRI as a neuroimaging monitoring marker of immunotherapy and anti-seizure medications in presumed autoimmune encephalopathy.

P-1-139

Cerebral endothelial dysfunction in posterior circulation is related with migraine chronification

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Background & Objectives: Migraine is a neurovascular disorder, which is related with increased incidence of major cardiovascular events and cerebrovascular disorders. Endothelial dysfunction has been suggested as a possible mechanism. Using cerebral vasomotor reactivity tests, there are growing evidences of impaired cerebral endothelial dysfunction in migraineurs. However, there is a paucity of study regarding the clinical implication of endothelial dysfunction. The aim of this study is to evaluate the association between endothelial dysfunction and clinical characteristics in migraineurs. **Method:** We prospectively included patients with migraine who first visited Samsung Medical Center headache clinic from Apr 2015. Inclusion criteria was as the following: 1) age between 18 and 50 years, 2) primary headache disorder was diagnosed as migraine without aura (MO) or migraine with aura (MA) by headache experts using ICHD-3 beta criteria, 3) subjects who completed structured headache questionnaire. We excluded patients with probable migraine. Cerebral endothelial function was assessed by Breath Holding Index (BHI) on transcranial Doppler in bilateral middle cerebral arteries, posterior cerebral arteries, and basilar artery. **Results:** Although the study is still ongoing, we provide preliminary results of 25 patients who completed the evaluation. BHIs in the left and right posterior cerebral arteries were negatively associated with headache days per a month (Spearman's rho = -0.519 and -0.490, P = 0.016 and 0.024, respectively). HIT-6 scores were negatively associated with the left posterior cerebral artery BHI (Spearman's rho = -0.462, P = 0.023), while total MIDAS scores also showed a similar trend (Spearman's rho = -0.358, P = 0.085). Demographic factors and comorbidities including presence of allodynia were not associated with chronic daily headache and/or chronic migraine, except for higher HADS score (p=0.011). **Conclusion:** Cerebral endothelial dysfunction, especially in posterior circulation, is associated with increased number of headache days and headache-related disabilities in migraine sufferers. Whether the reversal of endothelial dysfunction has a therapeutic implication should be evaluated in a larger prospective study.

P-1-140

Subtypes and comorbidity of chronic daily headache in the outpatient department of a tertiary hospital

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Background & Objectives: Depression, anxiety, insomnia, chronic fatigue, fibromyalgia, stress, and analgesic overuse etc are common in patients with chronic daily headache. In order to manage the patients with chronic daily headache effectively, neurologists consider the subtype of headache, comorbidities, analgesic overuse, and maladaptive illness behavior, etc. **Method:** We recruited consecutive patients with chronic daily headache seen in the department of neurology of Chungbuk National University Hospital from October 2013 to June 2015. The subtypes of chronic daily headache were classified according to the ICHD-II criteria. We investigated the comorbidities such as depression, anxiety, insomnia, chronic fatigue syndrome, fibromyalgia,

and stress by questionnaire and interview. We also investigated medication overuse and maladaptive illness behavior. **Results:** Forty patients with chronic daily headache were recruited. The mean age was 47.2 years, and 75% were women. Chronic tension-type headache (CTTH) was diagnosed in 26 patients (65%) and chronic migraine (CM) in 14 patients (35%). Probable medication overuse headache (MOH) was diagnosed in 17 patients (42.5%): 9 out of 26 patients with CTTH; 8 out of 14 patients with CM. The mean number of comorbidities was 2.75 (1-5). The most frequent entity was a persisting stress (72.5%). Seventeen patients (47.5%) showed maladaptive illness behavior. **Conclusion:** These results suggest that chronic daily headache are frequently accompanied by multiple comorbidities, medication overuse, and maladaptive illness behavior.

P-1-141

Botulinum toxin a for chronic migraineurs: a single center experience

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Background & Objectives: Botulinum toxin type A (BoNT-A) for the treatment of patients with chronic migraine (CM) has been studied, but there is a paucity of data in Korean CM patients. Botulinum toxin type A (BoNT-A) for the treatment of patients with chronic migraine (CM) has been studied, but there is a paucity of data in Korean CM patients. **Method:** We retrospectively analyzed 58 patients who underwent BoNT-A treatment for CM at Kangbuk Samsung Hospital from Jan 2014 to Mar 2015. All patients were administered 155 unit for 31 sites according with the protocol proposed by the PREEMPT study. The efficacy analyses were based on the change from baseline in headache days/month, number of medication intake/month and Headache Impact Test (HIT)-6 score at week 12. **Results:** Total 58 patients (mean age: 44.2±13.0, female/male ratio, 4.8:1) received BoNT-A injection for CM and 38 patients (65.6%) of them had medication overuse headache (MOH). Patients reported a significant decrease in headache days/month (pre 22.4± 5.7, post 12.2±7.0, P<0.001) and the number of medication intake (pre 21.4±5.8, post 11.7±7.1 P<0.001) after BoNT-A injections. Total HIT-6 score was decreased significantly after BoNT-A treatment (63.9± 10.4 vs 53.7±11.7 P<0.001). Thirty-two patients (55.1%) were 50% responder that at least a 50% decrease from baseline in the headache frequency. No difference were observed in a reduction in headache days/month and total HIT-6 score between patients with MOH and without MOH (p=0.647 and p= 0.999). **Conclusion:** These results demonstrated that BoNT-A may be an effective prophylactic treatment for CM in Korean patients. We can also expect the efficacy of BoNT-A treatment for patients with MOH.

P-1-142

Validation of the Patient Health Questionnaire-9 (PHQ-9) and PHQ-2 in patients with migraine

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Background & Objectives: Psychiatric problems have been commonly reported in patients with migraine. This study investigated the reliability and validity of the Patient Health Questionnaire-9 (PHQ-9) and Patient Health Questionnaire-9 (PHQ-2) in patients with migraine. **Method:** Subjects were recruited from a headache clinic and completed several instruments, including the Mini International Neuropsychiatric Interview-Plus Version 5.0.0 (MINI),

the PHQ-9, the Beck Depression Inventory-II (BDI-II), the Migraine Disability Assessment (MIDAS), the Headache Impact Test-6 (HIT-6), and the Migraine-Specific Quality of Life (MSQoL). **Results:** Among 132 participants, 39 patients (29.5%) had a major depressive disorder (MDD) as determined by the MINI. Cronbach's α coefficients for the PHQ-9 and PHQ-2 were 0.894 and 0.747, respectively. At a cutoff score of 7, the PHQ-9 had a sensitivity of 79.5%, a specificity of 81.7%, a positive predictive value (PPV) of 64.6%, and a negative predictive value (NPV) of 90.5%. At a cutoff score of 2, the PHQ-2 had a sensitivity of 66.7%, a specificity of 90.3%, a PPV of 74.3%, and a NPV of 86.6%. The scores of the PHQ-9 and PHQ-2 well correlated with the BDI-II score, the MIDAS score, the HIT-6 score, and the MSQoL score. **Conclusion:** The PHQ-9 and PHQ-2 are both reliable and valid screening instruments for MDD in patients with migraine.

P-1-143

A case of transient global amnesia during a migraine without aura attack

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Background & Significance: Transient global amnesia (TGA) is pure loss of fixation memory, no impairment of alertness, self-identity, or other focal neurological involvement, ceasing within 12 hours. The etiology of TGA remains unclear, and the hypothesis that transient retrograde venous congestion and venous ischemia of temporo-basal structures cause TGA has been suggested. The occurrence of TGA during migraine attack is a rare condition. We described a patient presenting TGA after migraine without aura attack. **Case:** A 44-year-old women visited outpatient clinic for recurrent headache since 5 years ago. Her usual headache was exacerbated, presenting severe intensity and accompanying with nausea at outpatient clinic visit day. She did not report visual aura before and during migraine attack. Her brain computed tomography revealed normal findings. Based on clinical history, normal neurological examinations and computed tomography findings, she was diagnosed as having migraine without aura. Zolmitriptan (2.5mg per attack) was prescribed for her acute migraine treatment. After 5 days, she revisited outpatient clinic for an episode of anterograde and retrograde amnesia lasting 12 hours, a day before her visit. Her amnesia started during the migraine without aura attack, approximately several hours after the onset of migraine attack. Her amnesia improved after 12 hours but she was persistently amnesic for 2 hours after the onset of TGA. She was diagnosed as having TGA occurring during a migraine attack and was admitted for evaluation of her amnesic event. She did not report any aura symptoms. Based on clinical history, there was no triggering event such as emotional stress, sudden contact with cold or Valsalva-associated maneuvers. No existence of vomiting efforts was systematically described. She was healthy other than migraine and did not take any medication for her migraine attack at the onset of TGA. Brain magnetic resonance imaging showed several nonspecific subependymal nodules in frontal horns of both lateral ventricles. Electroencephalography and routine blood test were normal. **Conclusions or Comments:** The underlying pathomechanism of TGA is still under discussion. Current hypothesis about etiology of TGA includes hippocampal transient ischemic attacks, epilepsy and migraine. Pathophysiological mechanisms linking migraine and TGA have been widely discussed. TGA was classically associated with a past history of migraine with or without aura. However, the occurrence of TGA during a migraine attack is a rare condition. A patient with low frequency migraine reported a TGA episode occurring during a severe migraine attack, with vomiting effort. Here, we reported a case of TGA patient during migraine attack without prominent vomiting.

P-1-144

Closure of patent foramen ovale in the patient with stroke and chronic intractable migraine

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Background & Significance: A patent foramen ovale (PFO) can be observed incidentally in about 25% of adults. However, PFO can be associated with clinical conditions like cryptogenic stroke or migraine. Until now, 3 randomized clinical studies have reported the equivocal effect of percutaneous closure of PFO, in comparison with medical therapy. **Case:** We present a 38-year old man with right-sided hypesthesia and paresthesia. He had no traditional risk factor except smoking and chronic intractable migraine. Transcranial Doppler-(TCD) examination for right-to-left shunt(RLS) revealed grade4 degrees of micro-bubble signals. Transesophageal echocardiography showed 18mm-sized patent foramen ovale (PFO) and functional RLS. After the PFO closure, the follow-up TCD presented no evidence of RLS. After the procedure, his migraine was disappeared **Conclusions or Comments:** Patients with cryptogenic stroke and chronic intractable migraine could be thought carefully for the PFO closure.

P-1-145

A case of status migrainosus with thyroiditis

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Background & Significance: Status migrainosus is a debilitating migraine headache lasting for more than 72hours. Association of thyroiditis and status migrainosus has rarely been reported. We experienced a patient of status migrainosus with thyrotoxicosis due to thyroiditis. **Case:** A 37-year-old woman with a history of migraine admitted for severe headache and vomiting. The headache was located in left temporal area and throbbing in nature. She tried conventional therapy including analgesics and beta blockers. But, the headache persist for 5 days. She was very obese, and neurological examinations were normal. Brain MRI, cerebral CT angiography and laboratory findings were almost normal, but thyroid function tests suggested hyperthyroidism; TSH less than 0.1 uIU/mL (normal value ; 0.4-4.7), free T4 of 7.77 ng/dL (normal value; 0.80-1.90), T3 of 3.25 ng/mL (normal value 0.6~1.7). Thyroid scan and USG showed thyroid parenchymal disease, but thyroid autoantibodies were all normal. We diagnosed her as status migrainosus and started IV methylprednisolone for 3 days. After 2 days of treatment the headache dramatically improved. **Conclusions or Comments:** We report a case of status migrainosus with non-autoimmune thyroiditis, which showed dramatical improvement after steroid therapy.

P-1-146

Reversible splenic lesion of the corpus callosum in migraine with aura

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Background & Significance: Lesion in the splenium of corpus callosum(SCC) occurs in the patients of seizure or antiepileptic drug history, even in chronic alcoholics, who have Marchiafava-Bignami disease. But migraine with a reversible lesion in the SCC is not common. Migraine with aura has long been be-

lieved to be related to the phenomenon of cortical spreading depression. Clinically, there are no consistent neuroimaging findings corresponding to an attack of migraine with aura. **Case:** A 24-year-old man with a history of migraines presented to our hospital due to an episode of pins and needles sensation on the left side of his body. The paresthesia lasted for 30 minutes, and was followed by a severe throbbing headache with nausea and vomiting. His headache lasted for about 1 day. He had similar episodes within 1 week before the current episode. His history was significant for migraines without aura for approximately 5 years. The frequency of attacks was 3 or 4-times a month. The patient denied any family history of seizures or epilepsy. The brain MRI findings of this patient showed a transient focal lesion in the SCC. The characteristic MRI feature included an oval high signal lesion on the T2 and diffusion-weighted images(DWI) in the central part of the splenium and a low signal lesion on the apparent diffusion coefficient(ADC) map. Follow-up MRI was performed 1 month later. It showed complete resolution of high signals in the SCC observed on initial DWI with normalized ADC reductions. **Conclusions or Comments:** There have been reports of various etiologies associated with a temporary focal lesion in the SCC manifested as obviously restricted diffusion. These findings may be the consequence of a high vulnerability of the splenium of the corpus callosum to cytotoxic damage. Migraine with aura was considered to be the cause of the reversible focal injury of the splenium.

P-1-147

A case hemicrania continua related to an underlying lung neoplasm

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Background & Significance: Hemicrania continua(HC) is an indomethacin responsive primary headache disorder characterized by strictly unilateral, mild to moderate continuous headache with ipsilateral cranial autonomic features during periodic exacerbations of headache. Secondary or symptomatic HC is associated with another neurological or non-neurological disease. primary lung neoplasm have been reported to cause ipsilateral facial pain(HC like headache) before the underlying malignancy is diagnosed. we describe a case hemicrania continua related to an underlying lung neoplasm **Case:** A 74-year old female smoker presented with a 3-month history of right sided headaches without any prior history of significant headaches. the pain exacerbations, usually of throbbing type with an intensity of 10/10. A few of the exacerbations were associated with nausea, vomiting, conjunctival injection but she denied photophobia, phonophobia. The headache never occurred on the left side. the neurological and general examinations were normal. Computerized Tomography (CT) and CSF study were normal. Magnetic Resonance Imaging(MRI) of brain showed mild enlarged & tortuous right superficial temporal artery without demonstrable vessel wall enhancement. With the exception of a mildly elevated sedimentation rate(78mm/hr), serum studies were unremarkable. Initially it was thought of as a temporal arteritis. So steroids were administered & temporal artery biopsy was performed. Right temporal artery biopsy results were arteriosclerosis(medial calcification) and headache persisted. Repeat MRI of brain and CSF study were unremarkable. but, Four months later after the headache occurs, patient began to complain general weakness, cold sweat and anorexia. so we conducted a test for cancer. Although initial chest x-ray didn't reveal a definite mass, a CT scan of chest demonstrated right lung cancer with metastatic lymphadenopathy. there was lymphadenopathy along the right side of the pericardium, obstructive atelectasis in the right middle lobe, bilateral pleural effusions. A Abdominal & Pelvis CT showed liver and spleen metastasis and multiple lymph node enlargement. she transferred to internal medicine and got supportive care. but she died 2 weeks later from diagnosis of lung cancer **Conclusions or Comments:** When older patients, especially smok-

ers, present with undiagnosed unilateral headache or facial pain with normal brain imaging and an elevated ESR, undiagnosed lung cancer should be considered as well as other possibilities such as temporal arteritis. If the chest x-ray is negative, a CT scan of the chest may lead to the diagnosis.

P-1-148

Prolonged hemiparesis in sporadic hemiplegic migraine; resolution after steroid

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Background & Significance: Sporadic hemiplegic migraine (SHM) is a rare form of migraine with aura, consists of fully reversible motor weakness and at least 1 other aura symptom and has no 1st degree relative who has those symptoms. But severe attacks could include decreased consciousness, seizures, agitation, and complex delusions and all these aural symptoms could last several days to several months until full recovery achieved. The management of HM is not established yet, depending on experience and similar to principle of management of the common types of migraine. We report a patient with severe attack including prolonged motor weakness, memory impairment, and ataxia whose aural symptoms are diminished with steroid therapy. **Case:** A 49-year-old female visited emergency room with sudden onset of dizziness and left side hemiplegia on awakening. Her family history was unremarkable. The 1st attack including headache and hemiparesis came 3 years ago, but no abnormality was shown in CT, MR, or TCD during her first hospitalization. Treatment for MR negative stroke was applied and the motor weakness recovered fully in 15 days. During her 2nd hospitalization in our department, no abnormality was found in MR or EEG and antiplatelet was started as it was 3 years ago but the symptom had not been resolved over a month. This persisted motor weakness was suspected as a severe aural symptom of hemiplegic migraine, we started methylprednisolone therapy for 2 weeks, then she felt her gait partially recovered and regained motor power objectively. The memory impairment has also improved and this raised MMSE score from 23 to 30. A month later from methylprednisolone received, the motor symptoms have been fully resolved. **Conclusions or Comments:** This case is possible to be included in rare SHM cases of which the onset is later than mean onset age and hemiplegic aura has been prolonged more than a week. Although the mechanism of persistent aura of SHM has not clarified yet, implication of augmented vasogenic leakage at cortical area corresponding to prolonged aura has been reported, thus we speculate that steroid could be an optional therapy for prolonged neurologic deficit which may be affected by this neurogenic inflammation with trigeminovascular activation.

P-1-149

A case of typical aura without headache presenting repetitive transient visual symptom

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Background & Significance: Typical aura without headache (TAWH) is rare type of migraine, and incidence of migraine is 3% in women and 1% in men of migraine patients. The prevalence of TAWH present biphasic distribution. TAWH can be presented not only positive neurological symptom, but also negative symptom. Therefore, it is easy to confuse to other neurological conditions, such as transient ischemic attack (TIA) or epilepsy. **Case:** A 58-year-old man presented with chief complaint of intermittent reversible visual symptom which lasts 10 to 40 minutes. This symptom started 2 years before visiting

the outpatient clinic. The patient described that tiny and shiny saw-toothed wheel came gradually into the one side of sight, became bigger and bigger, and faded out. It became more three times a month, and appeared by each side, predominantly on right side. The patient had no following headache. During the attack, he had any other focal neurologic deficit. Therefore, the patient was performed brain MRI, MRA, and EEG for rule out TIA and epilepsy. Brain MRI, MRA and EEG were unremarkable. We started valproate and added on topiramate, for prevention of visual symptom. After 2 years, the symptom had been decreased, however, it rarely came up due to poor compliance. **Conclusions or Comments:** In this patient, the symptom was dynamic and slow changed rather than static and abrupt onset or offset. Duration was longer than that of nonconvulsive epilepsy with visual symptom. The patient had only positive visual symptom, and besides had no cardiovascular risk factors and normal MRA. Consequently, we considered that the symptom was migraine aura rather than TIA or epilepsy. TAWH has benign course, therefore, TAWH should be considered in the patient who shows recurrent transient neurologic symptoms with no evidence of TIA or epilepsy. Delicate medical history taking may be helpful to distinguish TAWH from other neurologic diseases.

P-1-150

Zonisamide-Responsive SUNCT syndrome

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Background & Significance: SUNCT syndrome is characterized by short lasting, unilateral, neuralgiform headache attacks with conjunctival injection, tearing, nasal stuffiness and rhinorrhea, all on the symptomatic side. SUNCT syndrome is considered an intractable headache, several drugs had been tried with insufficient effect. **Case:** A 51-years-old man had been suffering from left hemi-facial pain attacks that was triggered by simple stimuli such as touching the face or scalp, eating, washing, coughing for 2 months. The pain was burning character with moderate intensity and attacks lasted 60-120 seconds each. The pain accompanied by ipsilateral eyelid edema, conjunctival injection and lacrimation. Neurological examination was normal. He was treated with Zonisamide, reaching the dose of 200mg per day. Beneficial effect started at the dose of 100mg daily and at the 200mg daily, painful attacks markedly decreased. After 1 months of treatment, Zonisamide was gradually tapered without headache recurrence. **Conclusions or Comments:** We report a 51-year-old man diagnosed with SUNCT syndrome, whose symptoms were successfully relieved by Zonisamide.

P-1-151

Markers predicting treatment outcome of epidural blood patch in patients with spontaneous intracranial hypotension: a clinico-radiological study

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Background & Objectives: The epidural blood patch (EBP) is a well-established treatment of spontaneous intracranial hypotension (SIH). However, the success rate of the 1st EBP is not excellent. Failed EBP may lead to longer hospital days, re-admission, and consequent socioeconomic loss. However, there is no radiologic marker to predict the treatment failure. In this study, we aimed to identify clinical and radiological predictors of treatment failure in patients with SIH. **Method:** By retrospective chart review, we identified patients who diagnosed with SIH and received EBP in Samsung Medical Center from January 2005 through March 2015. We defined a poor response as a persistent symptom or sign prompting a repeat EBP, and a good response as complete re-

covery or minimal symptoms requiring only medication after the 1st EBP. We measured radiologic markers including subdural fluid collections, pachymeningeal enhancement, engorgement of venous structures, pituitary hyperemia, downward displacement of the brainstem and cerebellar tonsil, angle between the vein of Galen and the straight sinus (vG/SS angle), pontomesencephalic angle, mammillopontine distance, and lateral ventricle, with other clinical factors. **Results:** One hundred patients met the inclusion criteria, and 32% were classified as poor responders. Among the radiologic markers, vG/SS angle was significantly narrower in poor responders (47.54 ± 21.29 vs. 60.70 ± 27.40 , $p=0.014$). Multiple sites of CSF leakage were also associated with treatment failure (37.5% vs. 19.1%, $p=0.048$). Among the clinical factors, pre-morbid headache was more frequently reported in poor responders (37.5% vs. 19.1%, $p=0.048$). **Conclusion:** Narrower vG/SS angle is predictive of treatment failure of the 1st EBP.

P-1-152

Differences of central facilitation between episodic and chronic migraine in nociceptive-specific trigeminal pathways

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Background & Objectives: The trigeminal nociceptive system plays a pivotal role in the pathophysiology of migraine. To investigate whether difference of trigeminal pain processing at brainstem as well as supraspinal level (thalamus, cortical) using nociceptive blink reflex (nBR) and pain-related evoked potentials (PREP) between episodic and chronic migraine (EM and CM, respectively) **Method:** 68 female patients with migraine (30 CM, 38 EM) according to the International Classification of Headache Disorders-3 beta version as well as 40 age-matched controls, were investigated using simultaneous recordings of the nBR and PREP during inter-ictal period. **Results:** EM patients displayed significantly decreased latency, larger amplitude and AUC values of both R2 component in nBR, whereas decreased both N1 and P1 latency in PREP compared with those displayed by controls ($p < 0.05$). But, CM patients showed significantly prolonged latencies, smaller amplitude and AUC values of the R2 component in nBR, whereas decreased left N1 and both P1 latency compared with controls ($p < 0.05$). In comparison between CM and EM, significant R2 suppression in CM and R2 facilitation in EM were displayed, but no significant difference in parameters of PREP. Moreover, the amplitude and AUC of the R2 component was negatively correlated, whereas the latency of the R2 component for the nBR was positively correlated with the frequency of headaches in the migraineurs ($p < 0.01$). **Conclusion:** We found facilitation of trigeminal nociceptive processing on both nBR and PREP in EM, but not of nBR in CM. R2 suppression at brainstem level and additional central facilitation changes at supraspinal level in CM may related to impaired anti-nociceptive descending pain modulation. These findings may be expression of adaptive or maladaptive mechanisms due to chronification of migraine.

P-1-153

A case of cerebrospinal fluid volume depletion with spine MRI

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Background & Significance: Spontaneous intracranial hypotension typically results from a spontaneous cerebrospinal fluid (CSF) leak, mainly at the spine level and leads to a loss of volume. Sinking of the brain, and the resultant traction on pain-sensitive suspending structures, is thought to be the main cause

of orthostatic headaches. Spine MRI is able to show spinal dural enhancement, meningeal diverticular and dilated nerve root sleeves in T2-weighted images, engorgement of epidural venous plexus, and extra-arachnoid fluid collections. We report the changes of spine MRIs after epidural blood patch in a case of CSF volume depletion. **Case:** A 51 year old woman visited our hospital with an orthostatic headache. Three days prior, she lifted and moved 12 heavy kimchi boxes. She developed a posterior nuchal headache, which worsened shortly after standing, but was alleviated after lying down. A nausea sensation and tinnitus accompanied her throbbing headache. Brain MRI revealed crowding of the posterior fossa and an increased in the anteroposterior diameter brainstem resulting from distortion. Spinal MRI of the whole spinal cord showed a multifocal leak along the bilateral paravertebral plexuses of the T12-L4 root sleeves in T2-weighted sagittal images. The CSF opening pressure was only 3cmH₂O. We performed an epidural blood patch, which dramatically improved her headache. A follow-up spine MRI disclosed an improvement in the previous multilevel active CSF leak. **Conclusions or Comments:** Evaluating CSF pressure requires a dural sac puncture, which may cause a secondary CSF leak. A spine MRI can detect the presence of epidural CSF in patients with an orthostatic headache, which may be sufficient to plan the therapeutic approach, avoiding invasive investigations. Additionally, we showed the changes of spine MRI after epidural blood patch in a patient of CSF volume depletion.

P-1-154

Idiopathic trigeminal sensory neuropathies showing improvement of Brain MRI abnormalities after steroid treatment

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Background & Significance: Trigeminal sensory neuropathy present a chronic pain, anesthesia or paresthesia that affects the trigeminal nerve. Because the clinical findings do not permit accurate localization, brain MRI must be used to visualize the entire course of the trigeminal nerve. We report 2 cases of idiopathic trigeminal sensory neuropathy showing improvement of symptoms and Brain MRI abnormalities after steroid treatment. **Case:** 1) Case 1 A 44-years-old man presented with acute sensory change of V2 and V3 area of left face for 3 days. He had complained of hypoesthesia and abnormal sensation on left face. The facial nerve conduction study (NCS) and blink reflex study showed left trigeminal neuropathy. Brain MRI showed enhancement of the porus of the Meckel's cave of the left trigeminal nerve, suggesting trigeminal perineuritis. After steroid treatment, there are improvement of symptoms and MRI findings. 2) Case 2 A 57-years-old man presented subacute sensory change of V2 and V3 area of left face for 2 weeks. He complained of progressive hypoesthesia and pain on left face. The facial NCS and blink reflex study showed left trigeminal neuropathy. Brain MRI showed linear enhancement of pontine segment in left trigeminal nerve. After steroid treatment, there are improvements of symptoms and MRI findings. **Conclusions or Comments:** We report 2 cases of idiopathic trigeminal sensory neuropathy showing improvement of Brain MRI abnormalities after steroid treatment. Early diagnosis by NCS and Brain MRI and prompt steroid treatment is important for idiopathic trigeminal sensory neuropathy.

P-1-155

Posterior Reversible Encephalopathy Syndrome (PRES) probably due to Leuprolide acetate

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Background & Significance: Leuprolide is GnRH agonist desensitizing GnRH receptor, thereby decreasing or inhibiting the release of LH and FSH, and subsequently suppresses gonadal sex hormone production. It has been used to treat of prostate cancer, uterine myoma, endometriosis, breast cancer or central precocious puberty and in vitro fertilization techniques. Leuprolide, however, has some adverse effects. Some cases are reported that patients treated with leuprolide underwent angina or ischemic heart disease. There has been no case report about PRES associated with using leuprolide. However, in some health report, some cases of complaint of occurring PRES after using leuprolide. We describe a case of PRES in a woman with a using leuprolide due to adenomyosis. **Case:** 41-year-old woman visited our hospital suffering from sudden onset headache. She has undergone mild headache intermittently, but this is the worst headache she had ever before (VAS 9). At 1 AM, while sleeping, sudden blunting headache was started at left occipital area and spread to whole head. She had no underlying disease and a nonsmoker. But only medical history she had done hysterectomy due to adenomyosis before 3 months ago. And she had been treated with leuprolide acetate intramuscularly, once every 4 weeks at a dose of 3.75 mg. Her vital sign was stable and no abnormalities on laboratory test. Brain CT performed at ER revealed hypodensity at left occipital lobe. And She needed to evaluate about new onset headache and brain lesion detected at CT. Brain MRI was performed and there were multiple high signal lesions in both occipital, left frontal, parietal lobe, mainly involving cortex and subcortical white matter on T2WI and FLAIR. And these lesions appeared as high signal on DWI and ADC. It was most likely posterior reversible encephalopathy syndrome (PRES) (Figure). Her headache was getting better with medication and she was discharged. **Conclusions or Comments:** This case was diagnosed as PRES based on the positive radiological findings and sudden new onset headache. And, this case is the first report of leuprolide causing PRES. Perfusion abnormality of cerebral blood is due to BBB dysfunction with cerebral vasogenic edema. After exposure to toxic agent, endothelial cell activation and upregulation of endothelial surface antigens and the release of endothelin affect the local vascular tone. All these changes result in vascular instability with vasoconstriction and downstream hypoperfusion. Blood-brain barrier dysfunction occurs, leading to vasogenic cerebral edema. Decreased serum estrogen levels associated with causing intracranial hypoperfusion, so it would make worsening of ischemia and decreased intracranial vasodilator function. Therefore, in this case, decreased levels of estrogens due to the use of leuprolide acetate, causing the dysfunction of endothelium-dependent intracranial vasodilatation, might have resulted in hypoperfusion and ischemia of brain. We should be careful about PRES in premenopausal women with using leuprolide making decreased serum estrogen level.

P-1-156

A case of secondary headache attributed to retropharyngeal lymphadenopathy: an uncommon condition mimicking meningitis

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Background & Significance: Retropharyngeal space could be potential anatomical site for secondary headache. Herein, we report a patient with secondary headache probably attributed to retropharyngeal lymphadenopathy. **Case:** A 17-year old girl visited our outpatient headache clinic due to newly developed headache. She had no history of migraine and other headache disorder. Her headache started the day before, and was accompanied by fever and chilling. She felt continuous squeezing pain in right posterior head. Her associated symptoms included nausea and vomiting. Neck stiffness was not prominent; she underwent cerebrospinal fluid (CSF) analysis to diagnose viral meningitis or encephalitis, preferentially. However, results of CSF study

showed no evidence of infection of central nervous system. In magnetic resonance imaging of the brain and neck computed tomography (CT), there was a 2-centimeter necrotic lymph node in the right retropharyngeal space and multiple enlarged lymph nodes in the both sides of neck and retropharyngeal space. A biopsy obtained from the enlarged cervical lymph nodes revealed benign reactive hyperplastic findings. The patient took symptomatic treatment for head pain, nausea/vomiting, and fever. Those symptoms have been gradually improved during 2-weeks period. A follow-up neck CT revealed that the necrotic retropharyngeal lymph node was disappeared and the size of the multiple enlarged lymph nodes was slightly decreased compared to the initial imaging work-up. **Conclusions or Comments:** This may be an uncommon case manifested with meningitis-like headache due to the retropharyngeal lymphadenopathy, although we could not confirm the pathogen for cervical lymphadenopathy.

P-1-157

A case of intracranial hypotension following acupuncture

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Background & Significance: Intracranial hypotension is clinically characterized by orthostatic headache that worsen in vertical position and is relieved by a recumbent position. The diagnosis of intracranial hypotension can be done through clinical history, clinical exam and various imaging techniques such as CT or MRI. In some patients, intracranial hypotension may be preceded by minor trauma. We describe a patient with intracranial hypotension after receiving an acupuncture for lower back pain. **Case:** A 55-years-old woman presented with orthostatic headache. The patient had received acupuncture treatment at an oriental medicine clinic for her lower back pain 5 days prior to admission to our hospital. There was no prior history of headaches. Physical and neurological examination were normal except for orthostatic headache. A lumbar puncture showed an opening pressure of 6cm H₂O with normal cell count, glucose, and protein. An initial brain MRI showed multifocal small subdural fluid collections, small ventricle and diffuse dural gadolinium enhancement. After epidural blood patches two times with bed rest, her headaches had improved. **Conclusions or Comments:** We reported a case of intracranial hypotension, suspected postdural puncture headache following acupuncture for lower back pain in which symptomatic relief was obtained after treatment with a epidural blood patch.

P-1-158

A case of trigeminal neuralgia caused by arteriovenous malformation in cerebellopontine angle

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Background & Significance: Arteriovenous malformation (AVM) is characterized by hemorrhage, seizure, focal deficits and rarely chronic headache. According to a recent study, 1.3% of AVMs are presented with trigeminal neuralgia. We report a patient who was relieved trigeminal neuralgia after embolization of AVM with cyberknife surgery. **Case:** A 49-year-old man admitted our hospital for the left facial pain characterized by brief electric shock-like pain limited in maxillary division of trigeminal nerve. On neurologic examination, there was no focal neurological deficit, but a pain-triggered zone in the left oral cavity by eating and tooth brushing. The pain was not relieved by carbamazepine treatment (200mg x 3/day). The brain magnetic resonance imaging revealed high signal intensity in left trigeminal nerve and numerous vessels

near cisternal portion of the left trigeminal nerve. The main feeder comes from the left posterior inferior cerebellar artery with early venous drainage through petrosal vein on the vertebral angiogram. He underwent cyberknife surgery, and the pain had disappeared. **Conclusions or Comments:** Cyberknife surgery can be a successful treatment for trigeminal neuralgia by AVM in cerebellar pontine angle in case it is refractory to medical therapy.

P-1-159

IgG4-related sclerosing disease presenting as intractable unilateral trigeminal neuralgia

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Background & Significance: IgG4-related sclerosing disease (IgG4-RD) is an immune-mediated systemic condition affecting diverse organs which lately has been increasingly recognized. Pancreas, liver, kidneys, biliary trees, thyroid gland, lungs and aorta are commonly involved major organs. Recently, idiopathic orbital and ocular inflammation has been reported as IgG4-RD with histopathologic evaluation. Herein, we report a case of IgG4-RD with a manifestation of intractable unilateral trigeminal neuralgia. **Case:** A 40-year-old male presented with tingling sensation, pinpricking, lancinating pain and dysesthesia of left mid-facial area compatible with maxillary branch distribution of trigeminal nerve. It started 3 months ago and the symptom had been aggravated. Initial ENT examination and brain MRI with enhancement were unremarkable. Neurologic examination was unremarkable other than dysesthesia of V2 area of left face. Due to the constant and aggravating facial pain he had to take many medication including gabapentin, carbamazepine, amitriptyline, NSAID and opioids. Because his symptom lasted almost a year with a need of pain medication on a regular basis, the second evaluation including oromaxillary unit CT scan and follow-up brain MRI was performed. They revealed ill-defined infiltrative enhancing lesion involving left pterygopalatine fossa and left medial pterygoid muscle with bone destruction at posterior wall of left maxillary sinus. The pathology of the lesion showed marked sclerosis with extension to bony tissue and diffuse lymphoplasmacytic infiltration with positive IgG4. Further medical workup did not show any other organ involvement. Under the diagnosis of IgG4-RD, oral prednisolone was prescribed. His pain was dramatically reduced not to require any pain medication. **Conclusions or Comments:** Our case showed IgG4-RD can manifest with the involvement of focal intracranial structure without other systemic signs. Intractable or prolonged trigeminal neuralgia requires repeated evaluations to find secondary etiology.

P-1-160

CNS involvement of granulomatosis of polyangiitis presenting as SIH mimic headache

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Background & Significance: To report about a secondary hypertrophic pachymeningitis presenting as SIH mimic headache and cerebral hygroma, which was caused by granulomatosis polyangiitis. We performed serial clinical and radiologic assessments. We also present a literature review about recent similar cases. Our observations add further evidence for atypical and misleading presentations of central nervous system involvement in GPA. These might help clinicians have clinical suspicion and lead to early diagnosis and treatment of

secondary hypertrophic pachymeningitis without systemic symptom **Case:** headache which started two weeks ago. The headache was so severe that he could not perform daily activities, but substantially relieved promptly by lying down. Mild degree of nausea, horizontal diplopia, and photophobia were also developed. Neurologic examination revealed bilateral abducens nerve palsies. Papilledema or meningeal irritation signs were not present. Post-contrast brain MRI demonstrated diffuse continuous pachymeningeal enhancement and subdural hygroma. The clinical manifestation was compatible with spontaneous intracranial hypotension. CT myelography was performed to seek a possible leakage site but was negative for leakage. Autologous epidural blood patch was done at L4-5 interspace. However, the headache progressed and did not respond to repeated epidural blood patch. Follow up MRI demonstrated increased extent of pachymeningeal enhancement which extended to cavernous sinuses and bilateral orbital apices. Evaluations for hypertrophic pachymeningitis were performed. Markers of acute phase reaction including ESR and CRP increased, as well as white blood cells. Lumbar puncture showed an increased CSF protein level (55mg/dL) with an opening pressure of 22cmH₂O. Blood and CSF screening were negative for infectious agents. There was no cytological evidence of dural carcinomatosis. Blood testing for autoimmune disorders revealed a weakly positive ANA. P-type anti-neutrophil cytoplasmic antibody (p-ANCA) was positive by serum immunofluorescence test. Angiotensin converting enzyme, CSF oligoclonal band, and other markers for immunologic disease were all negative. With a suspicion of immune-mediated or idiopathic hypertrophic pachymeningitis, high dose steroid injections and azathioprine were tried. An excellent response to steroid therapy was observed. During 7 months of follow up, the patient did not continue the steroid medication while maintaining oral azathioprine 100 mg (2 mg/kg). The headache became intractable. Gait disturbances and left-sided visual field defect were also developed. Brain MRI was followed up, which showed aggravated pachymeningeal enhancement in bilateral cerebral convexities and left tentorium, and leptomeningeal enhancement. Hydrocephalus also newly developed. Open biopsy and ventriculoperitoneal shunt were performed. The tissue diagnosis was compatible with granulomatosis with polyangiitis (formerly Wegener's granulomatosis). Cyclophosphamide and steroid treatment were started and effective for the remission of headache. Serum CRP and ESR levels were normalized. The patient has been followed without recurrence. **Conclusions or Comments:** We suggest that, 1) GPA should be included in the differential diagnosis of spontaneous intracranial 2) Extensive systemic evaluations are required because systemic symptoms and signs are often lacking in GPA with pachymeningitis, making it difficult to be distinguished from idiopathic hypertrophic pachymeningitis, 3) Atypical features including SDH, hydrocephalus, leptomeningeal involvement might be clues of GPA.

P-1-161

Giant-cell temporal arteritis in 80-year-old female presenting visual manifestation

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Background & Significance: Giant cell arteritis or temporal arteritis is a vasculitis of medium-to-large sized arteries that is confirmed with temporal artery biopsy. Ophthalmic involvement in temporal arteritis is one of the ocular emergent cases since permanent visual loss can be resulted in more than 50% of cases, and treatment should be initiated shortly. There have only been few reported cases of silent GCA-biopsy proven patients in Asia, and most cases resulted blindness. Here, we report a case of silent TA in an elderly woman, as confirmed by temporal artery biopsy, and improved with intravenous glucocorticoid therapy. **Case:** An 80-year-old woman was referred from oph-

thalmology clinic to neurology department with gradual visual dimness that had developed in both eye 5 days ago. She was diagnosed hypertension 10 days ago, and she was 20 pack-year smoker. She complained mild headache on temporal area, scalp tenderness and jaw claudication. Her visual acuities included perception of light in the right eye (OD) and finger counting in the left eye (OS). Cerebrospinal fluid tapping revealed normal intracranial pressure and cell counts. Brain magnetic resonance imaging and angiography results showed diffuse bilateral stenosis of vertebral arteries and external carotid arteries without significant intracranial vessels stenosis. The CR level, ESR and platelet count were elevated and measured to be 5mg/dL, 55mm/h, and 510K/uL, respectively. Following a presumptive diagnosis of GCA associated AAION; the patient was hospitalized and treated with intravenous 1g methylprednisolone qd for 5 days. Biopsy of the left temporal artery was performed, and 3cm of the temporal artery was acquired. Lymphocytes and multinucleated giant cells had diffusely infiltrated on vessel wall. After 7 days, the patients' visual acuity OS improved from 0.6 to 0.2 and OD from blindness to light perception. Disc swelling OU had decreased and the level of CRP and ESR decreased from to 1.07mg/dL and 30mm/h respectively. Oral prednisolone was maintained for 1 month. **Conclusions or Comments:** Clinical feature of GCA-Associated AAION may present either with typical headache or no specific symptom other than visual loss. In our patients, intravenous glucocorticoid therapy was done shortly, and her vision was improved. Considering raising number of incidence of GCA in Asia, physicians should suspect GCA-associated AAION, even in the absence of typical symptoms.

P-1-162

Painful neuralgia of C2-3 followed by herpes zoster infection at trigeminal nerve distribution: two case reports

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Background & Significance: Painful neuralgia of C2-3 is mostly paroxysmal stabbing or sharp pain of severe intensity within cervical nerve dermatomes. This pain syndrome could be diagnosed as either 4.7 Primary stabbing headache (PSH) or 13.4 Occipital neuralgia (ON) depending on the associated symptoms and disease courses if secondary causes are not identified. Here, we report two patients with neuralgia at C2-3 dermatome. **Case:** Case 1. A 56-year-old male experienced acute onset stabbing headache with severe intensity at right great auricular nerve (GAN) dermatome. The pain was not reproduced with palpation of GAN. He was initially diagnosed as 4.7 primary stabbing headache (PSH) according to ICHD-3 Beta. Three days after initial visit, he developed multiple blisters at right mandibular nerve branch (V3) of trigeminal nerve which was later diagnosed as herpes zoster infection. However, his neuralgia was confined to the GAN dermatome and did not extend to the trigeminal nerve. An anti-viral agent, valacyclovir was administered and his skin lesion and neuralgia gradually improved. Case 2. A 28-year-old male presented with sudden onset sharp and stabbing pain in the occipital area. The pain was localized at the left lesser occipital nerve (LON) dermatome. He was initially diagnosed as 4.7 PSH according to ICHD-3 Beta since he did not complain of dysesthesia, allodynia or tenderness. However, 1 day after first visit, vesicles and skin redness developed in his left maxillary nerve branch (V2) of trigeminal nerve which was later diagnosed as herpes zoster infection. He was also treated with anti-viral agents and his neuralgic pain gradually improved. **Conclusions or Comments:** Our cases had preceding stabbing pain of severe intensity before the development of skin lesion which is of usual findings. However, their pain was localized at cervical dermatome while herpes zoster was activated as skin lesion at the trigeminal nerve branch. This referred pain, from trigeminal herpetic infection to cervical neuralgia, may be explained by the anatomical convergence of C1 to C3 spinal nerve afferents and trigeminal

nerve afferents. In conclusion, possibility of herpes zoster infection outside the pain dermatome is also to be considered, even when the stabbing headache fulfills either 4.7 PSH or 13.4 ON at initial presentation.

P-1-163

Olfactory dysfunction is related to postoperative delirium in Parkinson Disease

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Background & Objectives: Olfactory dysfunction is evident in patients with Parkinson's disease (PD) and has been proven to be a useful tool for diagnosis of PD. Further, severe olfactory dysfunction is reported to be associated with poor cognitive decline and predict conversion to dementia. However, the prognostic validity of the olfaction concerning the development of postoperative delirium in PD is unclear. The goal of this study was to determine whether olfaction is associated with postoperative delirium in PD. **Method:** Patients with or without postoperative delirium were selected among patients with PD undergoing surgery under general anesthesia from 2006-2012 (each=17). Baseline olfactory function as measured with the Cross-Cultural Smell Identification (CCSI) test. Multiple logistic regression was performed to predict independent factor for postoperative delirium. **Results:** Baseline clinical features were not different between PD with postoperative delirium and without. PD with postoperative delirium (4.4 ± 1.5) showed decreased CCSI score than did those without (6.8 ± 2.4 , $p < 0.005$). Multivariate logistic regression revealed that olfaction (odds ratio, 0.32; 95% confidence interval, 0.12-0.80; $P = 0.016$ and operation time odds ratio, 1.01; 95% confidence interval, 1.00-1.02; $P = 0.047$) were significant predictors of the development of postoperative delirium. **Conclusion:** Impaired olfaction is associated with postoperative delirium in PD. Olfactory function test is useful at identifying PD patients vulnerable to postoperative delirium.

P-1-164

Overview of the Parkinson's disease smell and taste study

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Background & Objectives: Parkinson's disease (PD) age group of 60 and over has an incidence of about 1%, which is likely to go up in an aging society. The hallmark symptoms of PD include resting tremor, rigidity, bradykinesia, and postural instability. Differential diagnosis is particularly tricky in the early disease stage marked by mild clinical symptoms. Olfactory dysfunction in PD has been identified in multiple studies, and an attempt was made to use it for differentiation from other neurological disorders. Olfactory impairment in PD is independent of cognitive or motor symptoms, and it was used for early diagnosis of PD based on its early symptomatic expression. This study is investigating changes in the smell and taste senses in patients with PD and how the changes or degree of changes relate to the duration and/or clinical manifestation of PD. **Method:** The 12-item cross-cultural smell identification test (CC-SIT) developed by the smell and taste center at the hospital of the university of Pennsylvania is used. The subject is asked to smell the micro-encapsulated crystals in the scratch and sniff form and to choose one right answer out of 4. Selection has to be made even if the subject is unable to determine. Record the answer on the test sheet and score according to how many right answers are made. In order to avoid olfactory adaptation, each test is performed

at an at least 30 second interval. Five gradient solutions (No.1 ~ No. 5) of NaCl (saltiness), tartaric acid (sourness), sucrose (sweetness), and quinine HCl (bitterness) are prepared. The tongue is mapped into 6 zones, and the test starts from the tongue tip to the lateral border to the posterior one third of the tongue. Sweetness is tested on the tip, sourness and saltiness on the lateral sides, and bitterness on the back of the tongue. Saltiness, sourness, sweetness are tested in random order. **Results:** We interviewed 37 consecutive PD patients without nasal problem according to the clinical diagnosis criteria suggested by the United Kingdom Parkinson's Disease Society Brain Bank. Control subjects comprised 47 healthy spouses of the PD patients. The mean age of the patients was 68.5 ± 4.51 year and the control group was 66.8 ± 8.70 year. We used non-parametric statistical Mann-Whitney test (SPSS 17.0 version) Odor identification test (7.81 ± 1.62 versus 4.97 ± 2.72 , $P < 0.01$) was significantly lower in the PD patients compared with the control group. Taste detection threshold of tartaric acid (2.55 ± 0.68 versus 3.03 ± 0.95 , $P < 0.01$) was higher in the PD patients, and taste identification threshold of quinine HCl (4.23 ± 0.57 versus 3.84 ± 1.11 , $P < 0.02$) was lower in the PD patients. Taste detection threshold of tartaric acid and taste identification threshold of quinine HCl were statistically significant, but that was meaningless, because of the result did not match. Olfactory threshold test, taste detection threshold and taste identification of NaCl and sucrose, taste identification threshold of tartaric acid and taste detection threshold of tartaric acid did not differ between the PD patients and controls. **Conclusion:** We investigated the change of smell and taste by a clinical interview and test of PD patients and healthy controls. The results indicate that the smell of PD patients was significant lower than healthy control group. But, the responses rate to pleasant or unpleasant taste stimuli did not show difference in both group.

P-1-165

Olfactory deficits in the cognitive impaired de novo patients with Parkinson's disease

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Background & Objectives: To determine whether olfactory deficits may be related to the cognitive impairment in de novo Parkinson's disease patients. **Method:** The study participants included 86 de novo Parkinson's disease patients, and we divided two groups with cognitive not impaired (≥ 26) and impaired (< 26) patients by Montreal Cognitive Assessments. We compared the olfactory deficits between two groups using Korean version of Sniffin Sticks test. **Results:** Thirty-one (36.9%) of the 84 de novo patients had olfactory deficits. There are no differences among demographic factors including depression, sleep, and body mass index. However, except of odor-threshold score, odor-discrimination (7.45 vs. 6.56 , $p = 0.75$), odor-identification (7.54 vs. 6.37 , $p = 0.84$), and odor-total score (27.27 vs. 25.92 , $p = 0.35$) were lower in the cognitive impaired Parkinson's disease patients, compared with not impaired patients, although there were no significant differences. **Conclusion:** Our study showed that olfactory deficits, especially odor identification and discrimination are more in cognitive impaired patients than not impaired Parkinson's disease patients, although further studies with large sample size are required to validate our findings.

P-1-166

Does depression in parkinson's disease contribute the pattern of striatal dopamine depletion?

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Background & Objectives: Depression is common in patients with Parkinson's disease (PD) and can influence on the patient's quality of life. We performed this study to investigate whether the pattern of striatal dopamine depletion contributes depression in PD, we hypothesized that PD patients with depression might have a different pattern of striatal dopamine depletion compared to those without depression. **Method:** We analyzed the data of 199 de novo PD patients (mean age, 64.6 ± 9.8 years; 90 men, 45%) who had undergone both generic depression rating scale, Beck depression inventory (cut off value; 21), and dopamine transporter (DAT) scans. Quantitative analysis of DAT activities were performed based on volumes of interests, and each unilateral striatum was divided into 6 subregions: anterior and posterior caudate nucleus, ventral striatum, and anterior, posterior, and ventral putamen. **Results:** The higher frequency of depressed PD patients were found in female compared to non-depressed PD patients ($p = 0.036$). The age, levodopa equivalent dose, initial part III of the Unified Parkinson Disease rating scale, and disease duration were comparable between the two groups. A general linear model showed that depressed PD patients showed significantly less DAT activities after adjusting the patient's age and gender in the posterior putamen ($p = 0.004$), ventral putamen ($p = 0.004$), anterior putamen ($p = 0.021$), and ventral striatum ($p = 0.019$). **Conclusion:** We found that the depressed PD is more pathological involvement in the striatal dopaminergic neurons, especially in the putamen area. These results suggest that non-depressed PD may contribute the benign process compared to depressed PD.

P-1-167

Olfactory dysfunction in Parkinson's disease may be associated with the central cholinergic system

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Background & Objectives: Although the relationship between early Parkinson's disease (PD) and olfactory dysfunction is well recognized, the mechanism of olfactory dysfunction remains unclear. Recently, some studies have shown that cholinergic deficits may be associated with olfactory dysfunction. **Method:** A total of 56 PD patients and 14 control subjects were enrolled. Olfaction was evaluated with the Korean version of the Sniffin' stick (KVSS) test in PD patients. According to the KVSS test, 31 were classified into the PD with hyposmia group and 25 were classified into the PD with anosmia group. Short-latency afferent inhibition (SAI) was assessed at the affected hand during a medication 'on' state. **Results:** The SAI (%) values were significantly different among the PD with hyposmia, PD with anosmia and control groups (PD with hyposmia versus PD with anosmia, $p < 0.01$; PD with hyposmia versus control, $p < 0.01$; and PD with anosmia versus control, $p < 0.01$). In the PD patients, there was also a significant negative correlation between the severity of olfactory dysfunction and SAI (%). **Conclusion:** We observed that the SAI (%) may be strongly associated with olfactory dysfunction in PD patients. This finding indicates that central cholinergic dysfunction may contribute to the pathogenesis of olfactory dysfunction in PD.

P-1-168

Therapeutic singing activities to the vocal quality and the depression in Parkinson's disease: Case series

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Background & Objectives: Music acts as a stimulus to obtain motor and emotional responses and has been applied to variable neurologic disorders. We tried to examine the use of the therapeutic singing activities to improve the vocal quality and alleviate the depression in the Parkinson's disease (PD) patients. **Method:** We studied three PD patients whose clinical findings were compatible to PD following a published diagnostic criterion. We collected their clinical information, including age, disease duration, Mini-Mental State Examination (MMSE), Hoehn & Yahr (H&Y) stage, Unified Parkinson Disease Rating Scale part 3 (mUPDRS) and levodopa equivalent dose (LEDD). Each patient received 40 minutes of the therapeutic singing activities in a total of 6 sessions for 2 weeks. The activities included vocalizing with the simple melody and the vowels, the vocal improvisation, and the music making with the favorite songs. In the pre- and post-test, the Maximum Phonation Time (MPT) via the Praat test, the Voice Handicap Index (VHI), the Voice-Related Quality of Life (V-RQOL), and the Geriatric Depression Scale (GDS) were measured. Especially, the client's self-report with the 5 Likert scale and MPT were measured before and after the sessions. **Results:** The vocal quality of the clients, including the acoustic and subjective vocal evaluations, was improved and the depression symptoms of the clients were also alleviated through the therapeutic singing activities. In detail, the results demonstrated that the patients showed the acoustic vocal improvements in the MPT data. Secondly, the data of the VHI for the patient A and C were decreased and the V-RQOL for the patients A and C were increased. These data showed that clients tend to improve their acoustic and subjective vocal quality. Thirdly, the GDS of all the clients were remarkably lowered. Fourthly, according to the self-report for using one's own voice and feeling every session, all the clients were kept with their positive emotions without the vocal conditions through the therapeutic singing activities. Lastly, all the subjects also showed not only the changed singing attitudes with the sustained breathing patterns, increased the phonation time, the flexibility of the intonations and the dynamics with their own voices, but also positive and active participated in the vocal improvisations and the music making with their favorite songs. Therefore, the expressions of the verbal and the emotional with regard to all the clients were increased in the sessions incrementally. **Conclusion:** This study, as one of the initial attempts at applying the therapeutic singing activities to the PD patients, has confirmed that the potential effectiveness of the integration of the vocalizing with the simple melodies and the vowels, the vocal improvisations, and the music making of the favorite songs not only improves the vocal quality but also reduces the depression. Moreover, the therapeutic singing activities program was demonstrated to effectively facilitate and relax the muscles of the larynx, the vocal cord as well as the abdominal muscles for the PD patients so that these improved the phonation functions with their own voices, which tended to help the participation in the vocal improvisations and the music making positively. Further studies are warranted to develop the protocol required to determine and adjust the intensity and the level of the tasks in these therapeutic singing activities in consideration of the differing vocal functions and the depression states of the individual PD patients.

P-1-169

Craniocervical myoclonus with reversible bilateral dentate nucleus lesion by metronidazole toxicity

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Background & Significance: Metronidazole is a widely prescribed drug for amoebic and anaerobic germ infections, but metronidazole use rarely causes neurotoxicity. We reported a patient presenting myoclonus only with bilateral dentate nucleus lesion following metronidazole use, which was completely resolved after cessation of metronidazole. **Case:** 82-year-old man with hypertension and Alzheimer's disease (AD), presented with involuntary movement for 3 days. He had been taking metronidazole for acute cholecystitis at nursing home for 12 days before presentation (estimated cumulative dose of about 30g). Myoclonus on eyebrow, neck and upper arm was observed with 1-2 Hz frequency. The other neurological examination except general weakness was unremarkable. Evaluation of cerebrospinal fluid was normal. Repeated electroencephalography did not show cortical activity associated with the myoclonus. Glomerular filtration rate was 35ml/min and liver function was normal. Magnetic resonance imaging (MRI) showed high signal intensity on the bilateral dentate nuclei of cerebellum on the T2 fluid-attenuated inversion recovery (FLAIR) scans. After metronidazole was discontinued, myoclonus did not appear again. After 16 days of initial MRI scan, MRI was performed again which showed complete resolution of signal changes on dentate nucleus. **Conclusions or Comments:** Case presented only myoclonus in metronidazole induced neurotoxicity was very rarely reported. Clinical manifestation in this case was myoclonus at eyebrow, neck and upper arm (Frequency, 1-2 Hz) with similar frequency with palatal myoclonus, not presented at both lower extremities. This case showed bilateral cerebellar dentate nucleus lesion, which located on a functional circuit, guillain-Mollaret triangle which commonly causes palatal myoclonus. Withdrawal of metronidazole commonly reverses the neurologic symptom and radiological manifestations. Therefore early suspicion and detection of metronidazole neurotoxicity and cessation of drug can make better prognosis.

P-1-170

Chorea and parkinsonism in a patient with systemic lupus erythematosus

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Background & Significance: Movement disorders as a manifestation of central nervous system (CNS) lupus is extremely rare. Further, concomitant chorea and parkinsonism associated with SLE has never been reported in patients with SLE. The nature and pathophysiology of the basal ganglia lesion responsible for chorea and parkinsonism remains unclear. **Case:** A 36-year-old man with SLE developed generalized chorea and parkinsonism. Brain MRI was normal but hyperperfusion in the bilateral basal ganglia was observed with more affected on left. After steroid treatment, chorea much improved with normalization of brain SPECT but parkinsonism still persisted. **Conclusions or Comments:** It is plausible that microischemia due to the immune-mediated vasculopathy in the basal ganglia may contribute to the pathogenesis of irreversible parkinsonism.

P-1-171

Hereditary geniospasm in a Korean family

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Background & Significance: Hereditary geniospasm (OMIM190100), also known as familial chin trembling, is a rare paroxysmal disorder characterized by recurrent episodes of involuntary rhythmic movements of the mentalis muscle. It is transmitted as an autosomal dominant trait with high penetrance,

although sporadic cases have been described. There are approximately forty reported families, most of which are from North America and Europe. Two separate families have been described in Latin America and India, respectively. To date, this condition has not been reported in East Asia. In this report, we describe the first Korean family with hereditary geniospasm. **Case:** A 40-year-old woman presented with chin trembling since early childhood. This movement selectively involved the bilateral mentalis muscle and was reminiscent of a prelude to crying. The involuntary movement occurred spontaneously and was easily triggered by emotional disturbance such as agitation and excitement, and it was not initiated or suppressed at will. An episode lasted a few seconds or minutes, and then disappeared spontaneously. The chin trembling did not interfere with speech or eating. It occurred up to several times a day. She denied its occurrence during sleep. The neurological examination was otherwise normal. There was no facial weakness or previous history of facial palsy. Interestingly, the occurrence and duration of the symptom had increased from the initial onset into to her twenties and then, remained static from her early thirties. Clonazepam 0.5 mg or carbamazepine 100 mg three times a day only partially ameliorated her symptoms, and she refused botulinum toxin injections. Her family history revealed that six more members of her family have the same chin trembling as that of the patient. Chin trembling started in early childhood in all affected members. All affected members did not have any other neurological disorders except for chin trembling. **Conclusions or Comments:** Herein, we present the first Korean family with autosomal dominant hereditary geniospasm. Although it is a neurologically benign condition, patients may feel social embarrassment and seek medical treatment. According to the literature, the frequency and severity of most of the affected people peaked in early adulthood and then gradually decreased or virtually disappeared in some individuals by the fourth or fifth decade. This observation holds true for two of the affected members in our case family. However, interestingly, the geniospasm of the index patient has been aggravated throughout her adulthood. Jarman and colleagues reported that hereditary geniospasm was linked to the chromosome 9q13-q21 locus in one family, but this linkage was not found in other family they analyzed and also in other studies. Thus, the locus for hereditary geniospasm still needs to be identified. Unfortunately we were not able to perform genetic tests for the family because our patient refused them. The pathogenesis of and highly selective involvement of the bilateral mentalis among the facial muscles in hereditary geniospasm still needs further investigation.

P-1-172

Primary lingual dystonia induced by speech

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Background & Significance: Primary lingual dystonia is a rare, disabling form of focal dystonia that impacts on daily activities including speaking, chewing, and swallowing, and causes social and vocational disabilities. The movements vary, from repetitive and/or episodic to sustained tongue protrusion, and can also be action induced with speaking or eating. Primary lingual dystonia induced by speaking is a rare type of focal dystonia that is usually idiopathic in origin and is characterized by increased tonus of the tongue, which causes protrusion only during speaking. This report describes a rare condition of isolated lingual dystonia only induced by speaking. **Case:** A 26-year-old woman presented to our outpatient clinic with a speech disturbance complaint that had begun 2 months ago. She was able to eat and drink normally. She had no family history of neurological disease and no history of neuroleptic drug use. She also showed neither previous infection history nor psychological problem. Her neurological examination was normal except for uncontrolled protrusion of her tongue during prolonged speech which was causing dysarthria. This ab-

normal movement was leading to unpredictable sounds when the tongue touched her front teeth. She felt more comfortable when she had something in her mouth, such as gum or candy. Brain magnetic resonance imaging and electroencephalography were normal. Standard blood tests, including tests for thyroid, parathyroid, and Wilson's disease were within normal limits. The condition was diagnosed as a focal dystonia specifically induced by speaking. We started treatment with trihexyphenidyl and she presented no problem with speaking. We have a plan to taper the dosage gradually and discontinue. **Conclusions or Comments:** Focal lingual dystonia is a rare condition that can be misdiagnosed as a psychogenic problem because it may interfere with chewing, swallowing, and speaking. Our case had a lingual dystonia which was relieved by sensory tricks such as, when something was in her mouth. Our case had a unique features showing focal lingual dystonia only induced by speaking. Some previous reports about speech induced focal dystonias described clinical signs extending to the orofacial area, not only the lingual area. We suggest that trihexyphenidyl might be considered a possible drug of choice in minor focal lingual dystonia induced only by speaking. There has been a report of second option of treatment performing EMG-guided botulinum toxin injection into the dystonic muscle. This case report has a limitation that a psychogenic cause cannot be absolutely excluded.

P-1-173

PSP-like Syndrome without prominent ocular motor abnormality after aortic aneurysm repair

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Background & Significance: The syndrome resembling progressive supranuclear palsy (PSP) is known as a rare complication of aortic aneurysm repair surgery. We demonstrate a case of PSP-like syndrome without ocular motor abnormality after aortic aneurysm repair. Ischemic insult is a possible explanation as a mechanism of injury. **Case:** A 74-year-old woman presented to the emergency room for the sudden onset of severe pain in the left chest and upper back area. She was generally healthy with the exception of a history of hypertension. Computed tomography (CT) angiography of the aorta showed a type A aortic dissection. She developed transient left-sided weakness immediately after the CT scan and emergency surgery to repair the dissected aorta was performed. No specific event occurred during the operation but the patient developed clustered tonic-clonic seizures after the surgery which showed repetitive spikes in the left frontal area on electroencephalography (EEG). The seizures were successfully controlled by a medication with antiepileptic drugs and the level of consciousness improved gradually. Magnetic resonance images (MRI) of the brain demonstrated 8 mm-sized nodular lesion with rim enhancement in the left frontal cortex which was relevant to the EEG finding. Two months after the 1st operation, she underwent the 2nd operation for the descending aorta which was repaired by artificial graft. After two months after the operation, her daughter noticed that her gait had become slow and unsteady. This progressively worsened and the resulted in frequent loss of balance and falls. Over the next two years, she developed aggravated resting tremor of the both hands, rigidity of the limbs, bradykinesia, gait disturbance, and postural instability resulting in retropulsion which was not responsive to the medication with levodopa. Neurological examination revealed preserved range of movement of the eyes in both vertical and horizontal directions but the latency of saccadic eye movement increased with reduced amplitude. Neuropsychological tests for the reduced spontaneity showed impaired frontal executive function and memory. The Mini Mental Status Examination score was 9. Follow-up MRI of the brain with an interval of two years showed no ab-

normality which showed up on the previous MRI scan. **Conclusions or Comments:** The syndrome of saccadic gaze palsy with parkinsonism resembling PSP has been known as one of the complications of ascending aortic aneurysm repair. Also there have been several case reports about loss of volitional saccades without parkinsonian features. Our patient developed bilateral resting tremor, bradykinesia, changes in muscle tone, gait disturbance, postural instability, and cognitive impairment without prominent ocular motor abnormality. It is possible that the neurological manifestation develops as a part of clinical spectrum of PSP ranging from pure ocular abnormality to full-blown PSP phenotype. There is no definite imaging evidence for the ischemic insult on previous reports that could be correlated to the symptom complex but focal abnormality on MRI of the brain, which disappeared on follow-up imaging, and global cognitive dysfunction in our case partly suggest thromboembolic event with diffuse cerebral hypoxemia as a possible mechanism. This type of surgery is fairly common while this complication is very rare. It is speculated that there is individual susceptibility or preexisting PSP pathology as a participating factor.

P-1-174

The usefulness of quantitative autonomic function test for the differentiation of multiple system atrophy from idiopathic Parkinson's disease

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Background & Objectives: Differential diagnosis of idiopathic Parkinson's disease (IPD) and multiple system atrophy (MSA) is challenging since they share clinical features with various combination of parkinsonism and autonomic dysfunction. Early and prominent manifestation of autonomic failure, with the combination of parkinsonism that is poorly response to levodopa treatment, abnormal cerebellar symptom and corticospinal dysfunction are main characteristics to differentiate MSA from IPD. However, some patients with IPD present prominent autonomic dysfunction and some MSA patients show good levodopa responsiveness especially in their early stage of diseases. Investigators have reported the diagnostic implication of autonomic function test to improve those diagnostic challenges. With the advent of the equipment for the non-invasive and quantitative autonomic function test (AFT), there have been efforts to improve the diagnostic accuracy using more fractionized autonomic indexes. Several autonomic indexes have been validated to represent the degree of autonomic dysfunction, and the selective damage of the sympathetic or parasympathetic dysfunction in early IPD. However the results are not consistent and failed to demonstrate the selectivity of the autonomic damage in both IPD and MSA. We aimed to determine the usefulness of quantitative autonomic test and the correlations of autonomic functional status and subjective symptomatic scales with more fractionized autonomic indexes to distinguish MSA from IPD. **Method:** 40 patients with parkinsonism of similar severity were enrolled prospectively. Four parasympathetic and seven sympathetic indexes during autonomic function tests by non-invasive beat-to-beat blood pressure and heart rate monitoring were compared between patients with IPD (n=20, age=72.75±9.24, Hoen&Yahr stage=1.53±0.66) and MSA (n=20, age=65.60±9.24, Hoen&Yahr stage=1.93±0.67). Parasympathetic indexes include expiration/ inspiration (E/I) ratio during deep breathing, valsalva ratio, 30:15 ratio, and BRSv (regression slope of systolic BP in early phase II during valsalva maneuver). Sympathetic indexes were pressure recovery time (PRT), sympathetic index1 (BP fall during phase 2, SI1), SI3 (the difference in BP between baseline and the end of phase 2) and BRSa (BP decrement associated with phase 3 divided by the PRT), early phase II mean BP drop, pulse pressure reduction rate (PPR) during valsalva maneuver and QSART test

(quantitative sudomotor axon reflex test). To demonstrate the correlation of disease severity and the autonomic abnormality, we also compared the UPDRS, UMSARS, and H&Y stage to the abnormalities of those autonomic indexes. **Results:** PRT (p value <0.002) and BRSa (p value <0.004) were significantly different between IPD and MSA groups with similar severity of diseases. **Conclusion:** Compare to IPD, MSA patients show significantly increased PRT and decreased BRSa. It means that the sympathetic dysfunction is predominantly decreased in MSA and these indexes are the significant autonomic indexes in differentiating MSA from IPD

P-1-175

Gait analysis of PD patients with or without FOG - comparison with healthy control

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Background & Objectives: Freezing of gait (FOG) in Parkinson's disease (PD) is one of main causes of falls. Characteristics of FOG are still not fully understood. To understand the FOG in patients with PD, motion analyses were done in this study. **Method:** PD patients were consecutively recruited from outpatient clinic. FOG group was defined as patients experienced FOG episodes during past month based on new FOG questionnaire. Nine freezers and 11 non-freezers were enrolled in this study. Also, 13 healthy volunteers were constituted as control group. Motion analysis using the VICON 3D motion analysis system was done at defined 'off' state. They performed "timed up and go test"; stand up from an arm chair, walk straight 3 meters, and return to the chair and at their preferred walking speed. Tasks was repeated three times. We analyzed basic parameters of gait and range of motions (ROM) in hip, knee, and ankle joints, comparing more affected and less affected footsteps. Demographic and clinical characteristics of patients were also evaluated. **Results:** There were no statistically significant difference in demographic and clinical characteristics between groups. Patients group showed increased time and step for the task compared to control group. Short step length, increased cadence and slow walking speed were also found in patients group. During straight walking phase, there was significant difference in foot clearance height between freezer group and control group. Moreover, in freezer group, more affected foot clearance height was significantly decreased than less affected foot. During turning phase, normal control group showed similar cadence between the inside and outside footsteps, and compensatively increased step speed and length of outside footstep. But, patients group showed similar step speed and length between the two footsteps, and increased cadence of inside footstep. Decreased outside foot clearance height was prominent in freezer group than normal control group. ROM of knee extension and flexion was decreased in only freezer group compared to control group. **Conclusion:** Patients with PD showed slow walking speed, increased cadence and short step length at gait. PD patients with FOG showed decreased foot clearance height and knee ROM during straight walking and turning, but PD patients without FOG did not. Further study is needed to understand the gait characteristics of FOG in PD patients.

P-1-176

An application of smartphone tapper for assessment of bradykinesia in Parkinson's disease: A pilot study

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Background & Objectives: Smartphone applications have been increasingly used not only for the diagnosis of a disease but also for monitoring of patients' status. Motor section of Unified Parkinson's Disease Rating Scale (UPDRS) is considered gold standard in evaluation of motor symptoms in Parkinson's disease (PD). Mechanical tapper (MeTp) has been used to assess bradykinesia in PD. We developed an application of smartphone tapper (SmTp) and sought to determine whether SmTp is applicable for clinical purpose. To this end, we studied which parameter of tapping performance using SmTp discriminates most between PD and controls. We also investigated whether parameters of SmTp correlate with motor UPDRS scores and number of tapping obtained from MeTp. **Method:** Fifteen controls and fifteen age- and sex- matched PD patients (male:female=7:8, age range from 52 to 80) were recruited. Among patients with PD, four patients were drug-naïve-, and eleven patients were on dopaminergic medication. Hoehn and Yahr stage of all patients were between 2 and 3, and the mean motor UPDRS score was 22.3 +/- 7.2. Smartphone tapper application consists of two rectangles of 30 by 45 millimeter, separated by 15 millimeters. Subjects were asked to tap each side of rectangles alternatively at the fastest speed for ten seconds. The same trial of tapping task was repeated three times for each hand. The mean and maximum number of tapping scores were calculated for each hand. Regardless of dominance of hands, we compared parameters from better side or worse side between PD and controls. Tapping number of mechanical tapper was obtained according to CAPSIT protocol where patients repeatedly tap one tapper at the fastest speed for 10 seconds and two tappers alternatively for 20 seconds. Both one tapper and two tapper tasks were repeated three times. **Results:** In SmTp test, the mean number of tapping, performed by the better hands, was 43.5 +/- 7.2 in PD group, and 49.4 +/- 9.5 in control group ($P < 0.060$, t-test). However, mean tapping scores performed by the worse hands were significantly different between two groups (37.5 +/- 6.0 in PD group, and 44.0 +/- 7.6 in control group; $P < 0.014$, t-test). Maximum number of tapping scores were significantly different between PD and controls, no matter what hand was used ($p < 0.042$ using the better hands, $P < 0.013$ using the worse hands). In ROC curve analysis, maximum tapping score in the worse hand discriminates PD patients from control groups ($p = 0.02$, AUC 0.75). At the cut off of 44 taps, sensitivity was 60.00 (95% CI, 32.29%-83.66%), and specificity was 80.00% (95% CI, 51.91% to 95.67%). The maximum tapping number of the worse side using SmTp highly inversely correlated with motor UPDRS scores ($r^2 = 0.45$, $p = 0.0060$). However, correlation analysis between SmTp and MeTp showed trend of positive correlation in both one- tapper task ($p = 0.092$), and two- tappers task ($p < 0.055$). No trend was found for better side in one- and two-point tapping **Conclusion:** Maximum tapping score of SmTp appears to be the best parameter to discriminate PD from controls. Moreover, this parameter highly correlated with motor UPDRS which is a gold standard in the assessment of motor function in PD. A further study with larger sample size is warranted for evaluation of usefulness of SmTp in clinical practice

P-1-177

Central cholinergic dysfunction could be associated with oropharyngeal dysphagia in early Parkinson's Disease

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Background & Objectives: Dysphagia is an important issue for prognosis in Parkinson's disease (PD). Although cortical dysfunction may influence dysphagia in PD patients, the exact relationship between cortical and swallowing function is unclear. We investigated the association of cortical function assessed by specific neurophysiological biomarker and swallowing function measured by videofluoroscopic studies (VFSS). **Method:** We enrolled 29 early PD patients. Using Swallowing Disturbance Questionnaire (SDQ), we divided

enrolled patients into PD with dysphagia and PD without dysphagia group. Videofluoroscopic dysphagia scale (VDS) were applied to explore the nature of dysphagia. To assess central cholinergic dysfunction, short latency afferent inhibition (SAI) was evaluated. We analyzed the relationship between the central cholinergic dysfunction and oropharyngeal dysphagia and the characteristics of dysphagia. **Results:** BMI and MMSE score in PD without dysphagia was higher than PD with dysphagia. Modified H&Y stage and SDQ score was significantly higher in PD with dysphagia than PD without dysphagia. The SAI value was significantly different between the two groups. The comparison of each component in VFSS between PD with dysphagia and PD without dysphagia showed statistical significance in the most oral phase component and a single pharyngeal phase component. Total score of VDS was higher in PD with dysphagia than PD without dysphagia. **Conclusion:** Our findings suggest that cholinergic dysfunction is associated with dysphagia in early PD and abnormal SAI value is a good biomarker for predicting the risk of dysphagia in PD patients.

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Association of body mass index and the depletion of nigrostriatal dopamine in Parkinson's disease

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Background & Objectives: Objective: To investigate the association of body mass index and nigral dopaminergic density in patients with Parkinson's disease (PD) using quantitative analysis of dopamine transporter activity. **Method:** Methods: This study enrolled 398 drug-naïve patients with de novo PD underwent [18F] N-(3- Fluoropropyl)-2 β -carbon ethoxy-3 β -(4-iodophenyl) nortropine positron emission tomography scan and body mass index measurement at initial evaluation. The relationship between body mass index and dopamine transporter activity in striatal subregions was assessed using techniques of linear regression analyses. Additionally, logistic regression analysis was conducted to estimate independent predictors of the highest and lowest quartiles of dopamine transporter activity. **Results:** Results: After adjusting for age and gender, correlation analyses showed significant positive relationships between body mass index and dopamine transporter activity in the total striatum and all investigated striatal subregions including the anterior putamen, posterior putamen, and caudate nucleus. A multivariate analysis adjusted for age, gender, disease duration, smoking status, coffee and tea consumption and residence area revealed that body mass index remained independently and significantly associated with dopamine transporter activity in all striatal subregions. Moreover, a multiple logistic regression analysis revealed that body mass index was a significant independent predictor of the lowest quartile of dopamine transporter activity in the anterior putamen (odds ratio, 0.892; $p = 0.016$), caudate nucleus (odds ratio, 0.901; $p = 0.031$), and total striatum (odds ratio, 0.863; $p = 0.003$). **Conclusion:** Conclusions: The present findings suggest that a low body weight in patients with PD may have a detrimental effect on nigral dopaminergic neurons, which could lead to more severe dopaminergic neuronal degeneration in the substantia nigra.

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Validation of the Seoul-instrumental activity daily living in the detection of dementia in Parkinson disease

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Background & Objectives: A prevalence of PDD is about 31.3% and PD patients have a 4- to 6-fold increased risk of developing dementia compared to age-matched general population. In diagnosis of PDD, dementia symptoms with insidious onset and slow progression ought to reveal in at least two of the four core cognitive domains (e.g. attention, visuospatial function, executive function, and memory) and it has to be severe enough to impair daily life, which must be independent from motor or autonomic dysfunctions. However, there is no exact recommendation how to evaluate activities of daily living (ADL) in PDD patients. Many articles have assessed clinical rating of ADL dysfunction using by various ADL scales each other in order to evaluate functional impairment of PDD. In the various ADL scales, the Pill Questionnaire (PQ) is widely accepted as a ADL scale in the world that evaluates whether patients can take their anti-parkinsonian drugs independently, or not. The Schwab and England Activities of Daily Living Scale is another staging system for a standard assessment of disability in PD. The iADL is more complex and demanding in terms of cognitive control than pADL, and therefore more vulnerable to cognitive decline early in the course of Alzheimer's disease (AD). One report suggests that iADL total score is particularly correlated with PD duration and the Hoehn and Yahr (H-Y) score in the PDD. This results show that motor deficits remain the major contributor to iADL impairment in PDD. On the other hand, another study shows that cognition plays a unique role in iADLs in PDD and is consistent with what is found in AD. Seoul iADL (S-iADL) scale is well known to the tool for instrumental activities of Korean elders. However, cutoff point of S-iADL was also determined from patients, who were constituted from most of AD and other dementia patients. Although S-iADL has never been validated in PDD, this scale has been used for diagnosis of PDD in Korea. The aim of this study is to identify cutoff score of S-iADL for differentiation of PDD from PD-NC and PD-MCI. **Method:** Newly diagnosed PD patients was divided into two subgroups as follows: PD patients with normal cognition (PD-NC) and PD-MCI (n=114) versus PDD (n=13). Clinical diagnosis of PDD was defined by suggestions of the MDS-Task Force team. We obtained age at registration, age at onset, education, gender, disease duration, S-iADL, Mini-Mental State Examination (MMSE), the Montreal Cognitive Assessment (MoCA), Clinical Dementia Rating (CDR), CDR-sum of box (CDR-SOB), initial motor Unified Parkinson's Disease Rating Scale (mUPDRS) and Hoehn-Yahr (HY) stage. **Results:** mUPDRS ($p=0.036$), HY stage ($p=0.025$), MMSE z-score ($p=0.002$), MoCA ($p=0.002$) and CDR ($p=0.018$) shows significant differences between PDNC plus PD-MCI and PDD. Cutoff point of S-iADL was over 1.0. For diagnosing PDD, S-iADL showed that 80% of sensitivity, 82.93 % of specificity, 36.4% of positive predictive value (PPV), and 97.1% of negative predictive value (NPV). PDD was not correlated with S-iADL, but MMSE z-score, CDR and CDR-SOB are associated with PDD. **Conclusion:** S-iADL shows an limitation to diagnose PDD from PDNC and PD-MCI. Interesting point in this study, PDD is more correlated with CDR and CDR-SOB than S-iADL. In addition, MMSE z-score and MoCA are also well associated with PDD. Therefore, it needs to have another good ADL scale, not S-iADL, in the future.

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Two cases of Parkinson's disease accompanied by retinitis pigmentosa

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Background & Significance: Parkinson's Disease (PD) is a neurodegenerative disorder causing a pathologic dopaminergic deficiency in the basal ganglia of the brain. The retina of all mammals contains dopaminergic neurons that modulate the receptive field of ganglion cells to provide spatial contrast sensitivity and color vision. In PD patients' retina, dopamine extent is less than control adults. Retinitis pigmentosa (RP) is an inherited, degenerative eye dis-

ease that causes severe vision impairment due to the progressive degeneration of the rod photoreceptor cells in the retina. Here, we present two cases of PD in the patients who have retinitis pigmentosa. **Case:** Case 1. A 63-year-old female patient was admitted. She was diagnosed retinitis pigmentosa 5 years ago and suffered from blindness from 3 years ago. From 6 months ago, she presented tremor in the right great toe, and the symptom progressed to right leg tremor. She admitted our medical center to be treated for the tremor. Brain MRI showed no abnormality, CIT-PET showed decreased uptake of left posterior putamen. That suggests idiopathic Parkinson's disease. Case 2. A 49-year-old female patient was admitted. She suffered from visual disturbance and was diagnosed as retinitis pigmentosa. Five years ago, both arm and leg tremor developed, and she was diagnosed with Parkinson's disease but did not take medication by herself. From 6 months ago, visual disturbance and tremor got worsened, and she admitted our medical center. Brain MRI showed no abnormality, CIT-PET showed decreased uptake of left posterior putamen, which suggests idiopathic Parkinson's disease. **Conclusions or Comments:** Our cases showed idiopathic Parkinson's disease accompanied by retinitis pigmentosa. There is a relationship between Parkinson's disease and retina. However, there were few reports about the relationship between Parkinson's disease and retinitis pigmentosa. We reported two cases of Parkinson's disease accompanied by retinitis pigmentosa.

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A rare complication of rasagiline, acute cholecystitis, a case report

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Background & Significance: Rasagiline mesylate is irreversible selective monoamine oxidase type B inhibitor, as monotherapy or as adjunct to levodopa in patients with end of dose fluctuations Rasagiline PO QD, at the therapeutic dosage range of 0.5 to 1 g/day, is known to be effective and well tolerated. Furthermore once-daily rasagiline showed 24-hour clinical effect even before first dose of levodopa which was not seen with multi-dose entacapone. Despite these benefits of rasagiline, we should be aware of severe side effects that was not well known, such as acute cholecystitis even in patients without hepatic impairment. **Case:** A 50-year old male without any other underlying disease has presented with Lt. pill rolling tremor, rigidity, bradykinesia preceded by orthostatic hypotension and constipation, since 2002. He was treated with perkin 100/25mg (levodopa, carbidopa) 2 tablets twice a day, procyclidine 2.5mg twice a day and propranolol 5mg twice a day, for 7 years by continuously increasing dosage of levodopa. On 2015.03.24., a 1mg of rasagiline was added on his prescription as an adjuvant of levodopa, due to motor fluctuation. He was expected to follow up at out patient clinic after one month. He was social drinker without hepatic impairment, and took no other medication except anti-parkinson medication. On 2015.04.20., 26 days after taking newly added rasagiline, he visited our emergency room due to abnormal behavior, confusion, elevated liver enzyme. On laboratory findings, mild leukocytosis (WBC 12630, Neutrophil dominant), elevated CRP(10.9), elevated AST/ALT (158/108), normal bilirubin, ALP and r-GTP were shown. To evaluate whether elevated hepatic enzyme is due to obstructive lesion, we checked enhanced abdomen CT. Abdomen CT showed mildly dilated common bile duct without evidence of obstructive cause, concluded by acute cholecystitis with GB sludge. With the evidence of no definite obstructive lesion on abdomen CT, he had neither fever nor abdominal tenderness, we used triple antibiotics and hepatotonics without ERCP. Furthermore, we conducted EEG to exclude nonconvulsive seizure, concerning with acute confusion. With consideration of drug side effect of rasagiline, we discontinued rasagiline for 2 weeks, mild leukocytosis and elevated liver enzyme were improved as normal WBC and AST/ALT with regression of confusion and abnormal behavior. **Conclusions or Comments:**

The most common side effects as adjunct to levodopa therapy include dyskinesia, weight loss, hallucinations, postural hypotension, vomiting, anorexia, abdominal pain, constipation, dry mouth, and etc. Rasagiline is contraindicated in patients who have severe hepatic impairment. However, this case report has reminded us that rasagiline can cause acute cholecystitis without any obstructive lesion in patients without hepatic impairment. Short term studies regarding adverse effects of rasagiline have shown no major side effects, but reminding that rasagiline is a relatively new drug, every adverse effect should be reported and a cautious approach in clinical application is needed.

P-1-182

Clebopride-induced parkinsonism

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Background & Significance: Clebopride, a benzamide derivatives, acts as a dopamine receptor blocking agent (DRBA). It is widely used for dyspepsia and has been reported to cause diverse movement disorders including drug induced parkinsonism, tardive dyskinesia, acute dystonia, and tremor. No report has been presented about parkinsonism due to clebopride in Korea. **Case:** We describe a 73-year-old woman who developed parkinsonism after taking clebopride for about 6 months. Brain MRI and F-18 FP-CIT positron emission tomography (PET) imaging was normal. She completely recovered within 8 months after cessation of clebopride. **Conclusions or Comments:** Clinicians should be aware of this side effect of clebopride, which can be reversible.

P-1-183

Delayed parkinsonism after treatment of dural arteriovenous fistula

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Background & Significance: Dural arteriovenous fistula (DAVF) is one of the reversible cause of parkinsonism, however, delayed parkinsonism after treatment of DAVF is never reported. **Case:** A 73-year-old woman presented with progressive parkinsonism over several years. Three years previously, she was treated for DAVF by coil embolization. Follow up brain MRI showed that sequelar lesion of left lenticular nucleus, with atrophy and T2-hyperintensity. 18F-FP-CIT PET was normal. **Conclusions or Comments:** While the underlying pathophysiology of delayed parkinsonism after DAVF is not clear, it is possible that irreversible ischemic insults on basal ganglia secondary to previous severe venous hypertension of deep cerebral vein due to DAVF.

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Normal dopamine transporter imaging in a spinocerebellar ataxia type 17 with parkinsonism

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Background & Significance: Autosomal dominant spinocerebellar ataxia type 17 (SCA17) is a neurologic disorder caused by abnormal CAG/CAA repeat expansions within the TATA-box binding protein (TBP) gene. According to recent reports, 42 repeats in TBP gene is the smallest expansion which can manifest features of SCA17, including ataxia, dysarthria, and even parkinsonism, and it is already known that striatal dopamine uptake is reduced in dopamine transporter imaging such as 123I-iodophenylnortropane single photon emission computed tomography (123I-FP-CIT SPECT) in SCA17. To date, all SCA17 patients with parkinsonism revealed a decreased metabolism in this

presynaptic dopaminergic imaging. However, we experienced a case of SCA17 manifested as parkinsonism, but with normal presynaptic dopamine metabolism. **Case:** A 79-year-old male visited with tremor and slowness of moving, which occurred insidiously since 3 years ago. Before the onset of symptom, he also had constipation, though he could not remember when it had started. He was taking medication only for hypertension during several years. On neurologic examination, he did not have evidence of cognitive deficit, his eye movement, motor power and sensory function were also intact. However, he showed decreased facial expression, rigidity of neck, and resting tremor on right arm and leg. He walked with reduced arm swing and his step was short. He also had mild postural instability on the pull test. Levodopa was started to relieve gait disturbance and rigidity, but he started to experience dyskinesia and wearing-off in several months. Genetic tests for spinocerebellar ataxia (SCA) type 1, 2, 3, 6, 7 were all negative. However, increased 42 CAG/CAA repeats of TATA box-binding protein (TBP) gene was found. There was diffuse and severe brain atrophy, including brainstem and cerebellum in his brain magnetic resonance image (MRI). However, in the 123I-FP-CIT SPECT scan, there was no evidence of nigrostriatal dopaminergic degeneration. **Conclusions or Comments:** Presynaptic dopaminergic system seemed to be normal in our patient, which was quite different from previous reports with reduced presynaptic dopaminergic metabolism in SCA17. 123I-FP-CIT SPECT is an excellent method for nigrostriatal dysfunction in parkinsonism, so there is little chance of false negativity of presynaptic dopaminergic abnormality. Rather, considering limited response to dopaminergic medication, we could assume that postsynaptic dopaminergic system was selectively involved in our patient, although we could not get other images for postsynaptic dopamine receptor. In conclusion, SCA17 seems to be able to selectively involve the postsynaptic dopaminergic system, and normal DAT imaging cannot exclude SCA17 completely.

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Imaging of nigrosome 1 at 3T MRI for distinguishing drug-induced Parkinsonism from Idiopathic Parkinson's Disease

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Background & Objectives: To explore the feasibility of the nigrosome 1 at 3T MRI to differentiate idiopathic Parkinson's disease (IPD) from drug-induced parkinsonism (DIP) **Method:** The institutional review board approved this study, and participants gave informed consent. We enrolled patients with DIP (n=20) and IPD (n=29) who underwent N-3-fluoropropyl-2-β-carbomethoxy-3-β-(4-iodophenyl)nortropane (18F-FP-CIT) PET, and 20 age-matched healthy subjects. All participants underwent 0.5×0.5×1.0-mm oblique axial 3D multi-echo-data image combination imaging to visualize the nigrosome 1 at 3T. Two reviewers independently assessed the nigrosome 1 without clinical information. DIP was diagnosed when no abnormality is seen on 18F-FP-CIT PET. Diagnostic performance of nigrosome 1 imaging was tested between the patients with IPD and DIP, and between the patients with IPD and healthy subjects. Inter-rater agreement was assessed by Cohen's kappa. **Results:** Inter-rater agreement for nigrosome 1 in all participants was almost perfect (k = 0.825). Between the patients with IPD and DIP, 32 and 17 out of 49 patients were rated as abnormal and normal on nigrosome 1 imaging, respectively. Three patients with DIP were misclassified to the abnormal group (sensitivity, 100%; specificity, 85%; accuracy, 93.9%). Between the patients with IPD and healthy subjects, abnormal and normal nigrosome 1 were rated in 32 and 17 out of 49 participants, respectively. There were three false positive healthy subjects (sensitivity, 100%; specificity, 85%; accuracy, 93.9%). **Conclusion:**

Nigrosome 1 imaging at 3T can differentiate DIP from IPD with high accuracy, and may help to select patients who need dopamine transporter imaging in those with suspected DIP.

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Does the pattern of striatal dopamine depletion contribute to apathy in Parkinson's disease?

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Background & Objectives: Apathy has been recognized as an important factor for quality of life in Parkinson's disease (PD), but the mechanism underlying apathy in PD has not yet been elucidated well. To investigate whether the pattern of striatal dopamine depletion contributes to apathy in PD, we hypothesized that PD patients with greater apathy might have a different pattern of striatal dopamine depletion, particularly the areas other than the posterior putamen, compared to those with less apathy. **Method:** We conducted a survey of the degree of apathy (using the self-rated Korean version of the Apathy Evaluation Scale, AES-S) in 134 PD patients who had been initially diagnosed at our hospital by dopamine transporter (DAT) scanning, using a [18F] N-(3-Fluoropropyl)-2 β -carbon ethoxy-3 β -(4-iodophenyl) nortropine (FP-CIT) PET scan (from March 2009 to June 2013). **Results:** Patients with a high AES-S score (above the median) had a greater Beck Depression Inventory (BDI) score (14.0 ± 6.8 vs 11.1 ± 7.4 , $p = 0.023$), compared to those with a low AES-S score. However, DAT activities in 12 striatal subregions and 10 inter-subregional ratios (ISRs) were comparable between the two groups. There were no correlations between subregional DAT activities and the total AES-S score, except for the right ventral striatum /posterior putamen ISR, which was negatively correlated to the total AES-S score ($r = -0.175$, $p = 0.044$). **Conclusion:** This study demonstrates that the pattern of striatal dopamine depletion does not contribute to the degree of apathy in early PD, although severity of depression is different between the patients with greater apathy and those with less apathy. Apathy in PD may be more likely associated with extra-striatal lesions accompanied in PD than striatal dopaminergic deficits.

P-1-187

The impact of prolonged temporal discrimination threshold on kinematic parameters of finger tapping and dexterous finger movements of Parkinson's disease

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Background & Objectives: Patients with Parkinson's disease (PD) have various higher order sensory dysfunctions. However, the effects of abnormal higher order sensory dysfunction on specific kinematic parameters of PD have not been reported. We investigated the impact of temporal discriminative sensory dysfunction on various kinematic parameters of repetitive finger tapping and dexterous finger movements. **Method:** We included 33 patients with PD. Their mean (\pm SD) age was 64 ± 9.7 years, disease duration was 28.1 ± 30 months, and UPDRS score was 21.8 ± 8.9 . We measured the somesthetic temporal discrimination threshold (sTDT) in each side index fingers. Using gyroscopes, we measured the amplitude, speed, and frequency of finger tapping of each hand.

We also calculated the degree of progressive decrement and variability of amplitude, speed and frequency. Coin rotation task was performed to assess finger dexterity. We counted how many times patients could rotate the coin for 10 sec using each hand. **Results:** Prolongation of sTDT values did not correlate with the mean values and the degrees of progressive decrement in amplitude, speed, and frequency. However, there was a significant positive correlation between sTDT values and the degree of variability in amplitude, speed and frequency of finger tapping (amplitude variability, spearman's rho = 0.393, $p = 0.001$; speed variability, spearman's rho = 0.243, $p = 0.49$; frequency variability, spearman's rho = 0.279, $p = 0.023$). These significance remained after the regression analysis adjusted for age and gender (Benjamini-Hochberg corrected p-value for multiple testing; amplitude variability $p = 0.006$; speed variability $p = 0.036$; frequency variability $p = 0.036$). Also, there was a significant negative correlation between prolongation of sTDT and coin rotation values (Spearman's rho = -0.260, $p = 0.035$). **Conclusion:** In PD, higher order sensory dysfunction and consequent abnormal sensorimotor integration seem to have an impact on the irregularity of amplitude, speed, frequency of finger tapping and impaired dexterous finger movements.

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Gender difference in depletion of presynaptic nigrostriatal dopamine in de novo Parkinson's disease

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Background & Objectives: Background: Varied evidences have been reported of gender difference in dopamine transporter (DAT) activity in PD. However, an aging is another crucial factor to determine the deterioration of striatal DAT activity, and thus identifying age mixed gender effect is important to determine the true gender effect in PD. Objective: To explore an age-related gender difference in presynaptic nigrostriatal dopaminergic density in patients with de novo Parkinson's disease (PD) using dopaminergic positron emission tomography (PET) scans. **Method:** Methods: This study included 307 de novo PD patients (152 men and 155 women) who underwent a 18F-FP-CIT PET scan at initial diagnosis. Gender difference of age-related DAT activity alterations in striatal subregion was assessed using linear regression analysis. **Results:** Results: Women showed higher DAT binding in all striatal subregions compared to men. In linear regression analysis, age-related DAT decline was greater in women at anterior putamen (men vs women) (Estimated slope [ES], -0.003 vs -0.022; $p = 0.013$), anterior (ES, -0.018 vs -0.043; $p = 0.004$) and posterior (ES, -0.011 vs -0.03; $p = 0.003$) caudate nucleus, but not in elsewhere. **Conclusion:** Conclusions: We found that age-related DAT decline was more severe in women at antero-dorsal part of the striatum but not in posterior part in PD. Considering early stage of PD pathogenesis in participants, these features suggest that neuroprotective effect in women may not play a sufficient role against PD pathogenesis per se.

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HMPAO SPECT study of cerebral perfusion in Parkinson's disease with depression and Major depression disorder

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Background & Objectives: Depression, the most common psychiatric complication in Parkinson's disease (PD), affects 40-50% of PD patients. Diagnosis of depression in PD is complicated by overlapping symptoms of the two disorders. We performed SPECT in Major depression (MD) disorder and PD

patients with and without depression. The aim of this work was to investigate regional cerebral blood flow (rCBF) in patients with PD with depression and without depression, and compare it to healthy controls, and patients with MD. **Method:** 103 patients were studied, 38 PD with depression (PDMD), 46 PD patients without depression, 19 MD patients, and 32 age-matched healthy control subjects. SPECT images were analyzed using Statistic Parametric Mapping 2 (SPM2; Wellcome Department of Cognitive Neurology, Institute of Neurology, London, UK) implemented in MATLAB version 6.5 (MathWorks Inc., Sherborn, MA, USA). **Results:** Brain perfusion SPECT analysis revealed that PD and PDMD groups showed significant hypoperfusion in the bilateral frontoparietal cortex compared to control group. Also, in the MD group, hypoperfusion was significantly observed in the paracingulate gyrus compared to control group. More interestingly, PDMD group showed more significant hypoperfusion in the subcallosal cortex compared to PD group. Hypoperfusion was also observed in PDMD in the intracalcarine cortex, superior temporal gyrus and central opercular cortex compared to MD group. **Conclusion:** In the present study, we suggested that dysfunction of frontal cortex, especially in paracingulate gyrus, might be involved in the pathogenesis of depression in PD. Importantly, we found that hypoperfusion of the frontal cortex in PDMD group was less than in the MD group.

P-1-190

Analysis of White Matter integrity in Parkinson's disease

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Background & Objectives: Neurodegenerative change of dopaminergic neuron in nigrostriatal pathway is well known in Parkinson's disease (PD). However, alternation of white matter integrity of crossing brain in PD is inconclusive. We performed tract-based analysis of white matter integrity in PD. **Method:** We recruited 20 normal controls and sex, age matched 35 PD. Clinical assessments of patients were included mini-mental status examination, Unified Parkinson's Disease Rating Scale (UPDRS). We measured the fractional anisotropy, mean, radial and axial diffusivities with the TRActs Constrained by Underlying Anatomy tools available as part of FreeSurfer. **Results:** Age, and sex did not different between two groups. Compared with controls, there were significant increased mean diffusivities in corpus callosum, corticospinal tract, and inferior longitudinal fasciculus, uncinated fasciculus of PD ($p < 0.05$). **Conclusion:** These results suggest that deterioration of white matter integrity beyond basal ganglia system might result from pathologic process of PD, and play an important role of functional impairment of various clinical manifestation of PD.

P-1-191

Incomplete recovery and minimal dopamine transporter decrease in drug-induced parkinsonism

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Background & Objectives: Drug-induced parkinsonism (DIP) is generally considered to a reversible condition, however, recent many studies showed the patient with DIP who recovered incompletely from the parkinsonian symptoms. To determine whether the presynaptic dopaminergic status differs

according to the recovery from parkinsonism, we compared the dopamine transporter density of patients with DIP using 18F-FP-CIT PET scan data. **Method:** We reviewed the medical records of DIP patients who were taken 18F-FP-CIT PET at the diagnosis, and classified according to the degree of recovery. Also, we selected patients with essential tremor (ET) who were taken the PET scan as controls. **Results:** A total of 19 DIP patients with partial recovery (PR), 42 with complete recovery (CR), and 32 with ET were recruited. There is no difference in age and sex among three groups, and the UPDRS motor score was comparable between PR and CR groups. PR patients showed decreased dopamine transporter (DAT) binding in caudate ($p = 0.027$ vs. CR, $p = 0.030$ vs. ET) and posterior putamen ($p = 0.030$ vs. CR, $p = 0.015$ vs. ET) when compared with CR and ET patients. DAT binding of CR patients were similar to that of ET patients. **Conclusion:** This result demonstrates that incomplete recovery from DIP is associated with minimal decrease of DAT density during parkinsonian symptoms. Also, this suggests that incomplete recovery from DIP may be a result of presynaptic neuronal damage by offending drugs.

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Creutzfeldt-Jacob disease with gait disturbance as an initial manifestation mimicking progressive supranuclear palsy

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Background & Significance: Sporadic Creutzfeldt-Jacob disease (sCJD) is fatal prion disease characterized by rapid progressive dementia often associated with myoclonus and ataxia. However, varied manifestations with atypical presentation make it difficult to make proper diagnosis of sCJD. Here we describe sCJD case with gait disturbance as an initial manifestation mimicking progressive supranuclear palsy (PSP). **Case:** A previous healthy 53-year old man visited with complaint of unsteady gait with falls within one year. He also complained deterioration of cognitive function for time, person and place during the past 3 months. On neurological examination, he had symmetric bradykinesia, rigidity and prominent abnormal stooped posture and postural instability without tremor (H&Y stage 3.0, UPDRS part III score: 43), suggestive of atypical parkinsonism. He also showed slow vertical and horizontal saccades with near complete limitation of both up and down-gaze. We performed additional laboratory test because possibility of other secondary cause for parkinsonism should be excluded considering his onset age was relatively young and he showed too rapid progression, which revealed diffuse 7-8Hz background slow with FIRDA on EEG and elevated 14-3-3 protein in CSF. His condition was rapidly worsened with dysphagia in 3 months. In this stage, after 15 months after onset, He could not sit up independently and became bed-ridden state (H&Y stage 5.0). **Conclusions or Comments:** PSP has no definite biomarker so possibility of diagnostic error always exists. In our case, patient's clinical manifestation was in accordance with PSP and vertical gaze palsy, negative finding in MRI and no myoclonus were atypical for CJD. Nevertheless, his onset age and rapid progression gave us clue that there might be secondary cause of parkinsonism. What we want to say is that we should do the differential diagnosis for PSP-look alike, especially sCJD when there is atypical manifestation for PSP despite it is not classical symptoms for CJD.

P-1-193

A case of cerebellar dysfunctions in MSA-C and PSP-C

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Background & Significance: Multiple system atrophy (MSA) and progressive supranuclear palsy (PSP) are an adult-onset progressive neurodegenerative disorder that are known to display diverse clinical features and disease progression. Multiple system atrophy (MSA) is a sporadic, progressive, adult-onset disease characterized by parkinsonism, cerebellar syndrome and autonomic dysfunction. Progressive supranuclear palsy (PSP) is an adult-onset neurodegenerative disorder characterized by early postural instability, which leads to falls, and a vertical supranuclear-gaze palsy. Several studies reported cases of clinicopathological coexistence of PSP and MSA. Early clinical manifestations of MSA and PSP can make difficulty in diagnosis. We report a case of patient with cerebellar dysfunction combined with autonomic dysfunction and supranuclear vertical gaze palsy. **Case:** A 75-year-old man firstly visited to our clinic with symptoms of dysarthria, orthostatic dizziness and gait disturbance for 4 year in January 2014. His past medical and family histories were unremarkable. In review of systems, he had orthostatic dizziness, urinary frequency and impotence. He showed dysarthria, dysphagia, symmetric bradykinesia, action tremor, bilateral dysmetria, dysdiadochokinesia, decomposition, postural instability, stooped posture, wide based gait and impaired tandem gait. In 2 positional blood pressure test, systolic BP decreased 20mmHg after standing. Brain MRI was unremarkable except diffuse brain atrophy. Brain FDG PET-CT yielded mildly decreased glucose metabolism of left frontal cortex, left deep gray matter and right cerebellar hemisphere. Brain FP-CIT PET-CT revealed asymmetrically decreased DAT binding of bilateral striatum, especially posterodorsal portion of putamen. He diagnosed parkinsonism-plus syndrome, especially MSA with predominant cerebellar features (MSA-C). After two months later, he visited outpatient clinic. On neurological examinations, he presented echolalia, applause sign, bilateral downward gaze palsy suggesting PSP. **Conclusions or Comments:** In a recent study that compared early clinical features of PSP-C and MSA-C, older onset, early falls, and supranuclear vertical gaze palsy without dysautonomia may predict the diagnosis of PSP-C in patients with late-onset sporadic cerebellar ataxia than MSA-C. Here we report a patient with a complex phenotype characterized by MSA-C and PSP-C. Further pathological and clinical evaluations in the patient are needed for accurate diagnosis.

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Freezing of gait in extrapontine myelinolysis

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Background & Significance: Extrapontine myelinolysis (EPM) is rarely complicated with akinetic-rigid parkinsonism. Herein we report a case with EPM presenting with freezing of gait (FOG). **Case:** A 65 year-old female visited our clinic with a 10-day history of gait disturbance. She was a habitual drinker. She was hospitalized in a local hospital due to altered mental status, where she was diagnosed as hyponatremia (103 mmol/L). Gait disturbance was noticed after recovery. Neurologic examination at admission to our hospital showed cognitive impairment, myoclonus in both upper limbs, dystonic posturing of the right arm, generalized bradykinesia, postural instability and FOG. Brain MRI which was taken 3 days after onset of gait disturbance showed high signal intensities (HSIs) in both caudate nuclei (CN) and putamen and normal pons on T2 weighted images. Brain single photon emission tomography (SPECT) showed decreased perfusion in both basal ganglia (BG). L-dopa and methylphenidate was effective for her balance and gait problems. Follow-up MRI showed normalized CN and putamen, and HSIs on the pons. Follow-up SPECT showed improved perfusion in the both BG. **Conclusions or Comments:** FOG was reversible along with the disappearance of HSIs in the striatum and partial improvement in perfusion to BG. As HSIs in the pons was identified after the resolution of FOG, BG dysfunction was critical in the de-

velopment of FOG.

P-1-195

Coexistence of ocular neuromyotonia and hemifacial spasm

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Background & Significance: Ocular neuromyotonia is a rare oculomotor disorder, and only anecdotal cases have been reported so far. Various conditions including cranial irradiation and mechanical irritation by tumors or vascular structures have been suggested to cause ocular neuromyotonia, and overlapping pathology between ocular neuromyotonia and hemifacial spasm is suggested in that both are peripherally induced movement disorders. The co-occurrence of ocular neuromyotonia and hemifacial spasm has not been reported yet. **Case:** A 47-year-old woman presented with involuntary jerky movement on the right face and intermittent diplopia that had begun before 2 years. Neurologic examination revealed involuntary contraction of the right eyelid and perioral muscles combined with irregular tonic lateral deviation of the right eye, which was evoked rather spontaneously, not by gaze. There was no limitation related to gaze. Magnetic resonance imaging showed that the right anterior inferior cerebellar artery intersects with both the right facial nerve and the right abducens nerve. We concluded that the patient has right hemifacial spasm coexisting with ocular neuromyotonia on the lateral rectus muscle, caused by simultaneous mechanical irritation of the right facial and abducens nerves. **Conclusions or Comments:** Co-occurrence of hemifacial spasm and ocular neuromyotonia in a single patient is first to be reported. This case could be a good evidence for an overlapping pathophysiology between ocular neuromyotonia and hemifacial spasm.

P-1-196

Primary central nervous system lymphoma: clinical experience of a single institution

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Background & Objectives: Due to its rarity, primary central nervous system lymphoma (PCNSL) remains as a diagnostic challenge to practitioners including neurologists. By definition, PCNSL is confined to central nervous system (CNS); and its presentation can mimic symptoms of other inflammatory or demyelinating diseases. **Method:** Medical records from May 2011 to May 2015 were reviewed. Biopsy-proven PCNSL cases presenting with neurological deficits as their chief complaint were included. Subjects with systemic involvement identified by either abdomen/chest computed tomography (CT) or positron emission tomography-computed tomography (PET-CT) at presentation were excluded. Clinical characteristics, paraclinical test findings, laboratory and neuroimaging measures were assessed. **Results:** 7 patients fulfilled the inclusion criteria. Time to nadir tend to be subacute (<8 weeks) or chronic in the majority of patients. Multifocal involvement of periventricular white matter, basal ganglia was common, but diffuse confluent lesion was noted in one subject. Laboratory results were generally not helpful. Cerebrospinal fluid cytology results were unremarkable except one patient. IgH rearrangement analysis was positive in one subject. Hypermetabolic signal on PET-CT was observed in 3 patients. Most patients had diffuse large B cell lymphoma; however lymphomatoid granulomatosis with EBV(+) was diagnosed in one. **Conclusion:** PCNSL may be underrecognized since the diagnosis is often difficult even with recent neuroimaging techniques. Clinical attention should be made especially in case of atypical disease course.

P-1-197**Analysis of normal appearing white matter in multiple sclerosis using myelin water imaging**In Hye JEONG¹, Joon Yul CHOI², Su-Hyun KIM¹, Jae-Won HYUN¹, AeRan JOUNG¹, Jongho LEE², Ho Jin KIM¹¹Department of Neurology, Research Institute and Hospital of National Cancer Center, ²Seoul National University, Department of Electrical and Computer Engineering

Background & Objectives: Several advanced MRI studies have shown the microstructural damage in the normal appearing white matter (NAWM) of the brain in patients with multiple sclerosis (MS). Among them, conventional myelin water imaging (MWI) has shown the high specificity to detect demyelination. However, conventional MWI has limitation on image quality. Recently, a new method, direct visualization of short transverse relaxation time component (ViSta) MWI, that selectively acquires short T2 signal by suppressing the long T1 signal was developed. The aim of this study was to assess the myelin integrity of NAWM and the pattern of regional myelin loss in patients with MS using both conventional and ViSta MWI. We also investigated the correlation of MWF value with disability and disease duration.

Method: For the comparison of the whole brain myelin water fraction (MWF) in NAWM between healthy controls (HC) and MS patients, 14 HC (34.7 ± 7.2, years) and 28 MS patients (34.5 ± 7.2, years), were scanned with a 3T MRI scanner (Siemens). The conventional and ViSta MWIs were acquired by following parameters: 28 slices with voxel size of 1.5 x 1.5 x 4.0 mm³ and scan time of 14 minutes 5 seconds for conventional MWI and 32 slices with voxel size of 1.5 x 1.5 x 4.0 mm³ and scan time of 6 minutes 53 seconds for 3D ViSta MWI. Regions of interest (ROI)s were determined in the centrum semiovale (CS), periventricular white matter (PVWM), genu and splenium of the corpus callosum (CC) and optic radiation (OR). A generalized liner model was used to compare the MWF values between two groups using age and gender as covariates. Partial correlation analyses between MWF and clinical measures were performed and corrected for age and gender.

Results: We analyzed 156 ROIs from 28 MS patients and 112 ROIs from 14 HCs. MWFs of MS patients in NAWM of ROIs including CS, PVWM, genu of the CC and OR were significantly reduced compared to those of HCs in both conventional and ViSta MWI. MWF of splenium of the CC was not significantly different between two groups, though a similar trend was observed. A significant correlation between disease duration and MWF of OR was revealed in both conventional ($r = -0.4$, $P = 0.04$) and ViSta ($r = -0.63$, $P = 0.001$) MWI. In addition, EDSS score was negatively correlated with ViSta MWF of OR ($r = -0.76$, $P < 0.001$). Subgroup analysis of patients with early MS (disease duration < 2year) revealed early decrease of MWFs in NAWM (conventional MWF of CS and OR and ViSta MWF of CS, PVWM, genu of the CC and OR) in comparison with HCs.

Conclusion: This new in vivo myelin measurement, ViSta confirms the myelin damage of NAWM in MS and it appears sensitive enough to detect white matter changes early in the disease process.

P-1-198**Treatment outcomes with rituximab in 100 patients with neuromyelitis optica: influence of FCGR3A polymorphism on the therapeutic response to rituximab**Su-Hyun KIM¹, In Hye JEONG¹, Jae-Won HYUN¹, AeRan JOUNG¹, Hyo-Jin JO¹, Sang-Hyun HWANG², Sooin YUN³, Jungnam JOO³, Ho Jin KIM¹¹Department of Neurology, Research Institute and Hospital of National Cancer Center, Korea,²Department of Laboratory, Research Institute and Hospital of National Cancer Center, Korea,³Biometric Research Branch, Research Institute and Hospital of National Cancer Center, Korea

Background & Objectives: Despite increased use of rituximab therapy in neuromyelitis optica spectrum disorder (NMOSD), the overall efficacy and safety

of long-term rituximab treatment in a large group of patients remains uncertain. Furthermore, identification of predictor of rituximab response is an important issue for assessing the individual risk-benefit of therapy and making treatment decision. We assessed the long-term clinical efficacy and safety of rituximab treatment in patients with neuromyelitis optica spectrum disorder (NMOSD) and the influence of fragment c gamma receptor 3A (FCGR3A) polymorphisms on rituximab response.

Method: Clinical data for NMOSD patients treated with rituximab for at least 6 months were evaluated retrospectively. After induction therapy, a single infusion of rituximab (375 mg/m²) as maintenance therapy was administered whenever a reemergence of CD27+ memory B cells among peripheral blood mononuclear cells occurred. Using an allele specific polymerase chain reaction-based method, the gene polymorphisms FCGR3A V158F were assessed.

Results: As of January 2015, 100 patients had received repeated rituximab treatment over a median 67 months. Of these, 41 had >5 years' follow-up, and 24 patients had >7 years'. The relapse rate was reduced significantly, by 96%, and disability improved or stabilized in 96% of patients. Rates of adverse events generally remained stable. The FCGR3A-F allele was associated with a risk of relapse in the rituximab treatment model (additive model, $p = 0.047$; recessive model, $p = 0.038$; MAX, $p = 0.031$) and insufficient memory B-cell depletion (additive model, $p = 0.034$; recessive model, $p = 0.032$; MAX, $p = 0.032$).

Conclusion: Repeated rituximab treatment for NMOSD was observed in an increasing number of patients and increasing duration of exposure; it maintained good efficacy and a safety profile, consistent with previous reports. The finding of a relationship between FCGR3A genetic polymorphism and rituximab response suggests the importance of individualized rituximab treatment strategies in NMOSD.

P-1-199**Clinical characteristics of disabling attack at onset in patients with neuromyelitis optica spectrum disorder**

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Background & Objectives: Neuromyelitis optica (NMO) is an autoimmune relapsing inflammatory disease involving the central nervous system. Individual attacks of NMO are generally severe enough to cause disability. We aimed to elucidate clinical characteristics of disabling attacks, and factors predicting residual disability at the very early stage of NMO.

Method: We investigated clinical characteristics including disability and treatment profile at onset and at first relapse in NMO or NMO spectrum disorder (NMOSD) patients with anti-aquaporin-4-antibody. The each onset attacks were defined as Disabling Attack at Onset (DAO) when the best EDSS score within a year includes inability to walk without assistance (EDSS 6 or above) or functionally blindness (20/200 vision or worse) in at least one affected eye.

Results: A total of 57 patients were enrolled (53 females; age of onset, 41.9 ± 14.8 years). Among them, 43 patients (75.4%) experienced relapse and the median interval between onset and the first relapse was 12.0 months (interquartile range, 4.6 to 22.4). Ten patients (17.5%) had DAO; 4 patients became unable to walk without assistance following myelitis, and 6 patients had severe visual impairment following optic neuritis despite of treatment with steroids and plasma exchange. Attack severity was the only clinical factor predicting DAO. In addition, the use of immunosuppressants delayed the interval to the first relapse ($P = 0.003$).

Conclusion: We demonstrate that some of NMOSD patients can have severe residual disability following onset attack. Since there are no clinically modifiable factors predicting disabling attacks, the use of immunosuppressant should be weighed heavily in patients with NMOSD.

P-1-200**Clinical and radiologic characteristics of Alexander disease: A single center experience**

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Background & Objectives: Infantile-onset Alexander disease (IOAD) present symptoms secondary to extensive cerebral white matter (WM) abnormalities, whereas adult-onset (AOAD) may bulbospinal symptoms following the atrophy of medulla oblongata. The purpose of this study was to verify the clinical and radiologic characteristics in Alexander disease (AxD). **Method:** Seven unrelated Korean patients with AxD were classified into three types by age at onset following as IOAD, juvenile-onset (JOAD), and AOAD. **Results:** Two patients were classified as IOAD with R79H or R88C mutations, two as JOAD (A267P and I363V), and three as AOAD (E312Ter, R70Q, D128N). Five AxD are still alive including 2 IOAD, but one AOAD (R70Q) deceased secondary to dysphagia and pneumonia within 1 year after symptoms onset. Psychiatric symptoms were observed in 2 with a past history of epileptic seizure, bulbospinal symptoms in 2 AOAD, 1 JOAD, and 1 IOAD. An AOAD with E312Ter did not complain of both cerebral- and bulbospinal- symptoms and signs. On MRI of the brain, the extensive cerebral WM abnormalities were observed in 5 including one AOAD (E312Ter). Atrophies of medulla oblongata and upper cervical spinal cord as well as pial FLAIR-hyperintensity was observed in 2 AOAD, 2 JOAD, and 1 IOAD, but not in two (R79H and E312Ter). Focal signal abnormalities on diffusion-weighted MR image were observed in 5. **Conclusion:** Clinico-radiological features and prognosis in AxD may be variable. IOAD or JOAD can show the characteristic bulbospinal symptoms or MR images of AOAD with time. Some AxD patient may have only a- or pauci-symptomatic cerebral WM abnormalities.

P-1-201**A case of Chronic Lymphocytic Inflammation with Pontine Perivascular Enhancement Responsive to Steroids (CLIPPERS)**

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Background & Significance: Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) was first described in 2010 by Pittock, et al. CLIPPERS is a defined inflammatory central nervous system (CNS) disorder, mainly involving the brainstem and in particular the pons. The condition features a combination of clinical symptoms essentially referable to brainstem pathology and a characteristic magnetic resonance imaging (MRI) appearance with punctate and curvilinear gadolinium enhancement 'peppering' the pons. Another feature is clinical and radiological responsiveness to glucocorticosteroid (GCS)-based immunosuppression. Neuropathologic finding of biopsy material includes white matter perivascular, predominantly T lymphocytic, infiltrate without granulomas, infection, lymphoma or vasculitis. **Case:** A 41-year-old man developed dizziness, headache, left tilting tendency 24 hours after taking unidentified medicine for erectile dysfunction. Five days later he experienced tingling sensation and hypesthesia on his left side of face, arm and leg and left sided limb ataxia. Cerebrospinal fluid study showed 12 cells/ μ L (lymphocytes- dominant, 97%)

and mildly elevated protein level (43.0 mg/dl). His brain MRI showed diffuse swelling and perivascular enhancement in pons, and punctate enhancement in splenium of corpus callosum and subcortical white matter. PET CT showed no demonstrable abnormal FDG uptake. After seven days from the onset of symptoms, intravenous methylprednisolone 1g was given for five days, and his symptoms were improved on the 2nd day. After gradual reduction of steroid treatment during 42 days he recovered completely with remaining mild headache. **Conclusions or Comments:** This is a rare case of chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids. The cause is not certain, but possibly drug induced. Characteristic brain MRI features were helpful for the treatment decision.

P-1-202**Cerebral Salt Wasting Syndrome in a Patient with Neuromyelitis Optica Spectrum Disorder**

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Background & Significance: Neuromyelitis optica spectrum disorder (NMOSD) presents with symptoms such as intractable vomiting and hiccups. However, to our knowledge, hyponatremia associated with cerebral salt wasting syndrome (CSW) has never been described in a patient with NMOSD. In this report, we describe a case of a young male patient with NMOSD and systemic lupus erythematosus (SLE) who presented with CSW. **Case:** A 22-year-old man presented with intractable vomiting and hiccups. Five weeks after onset of these symptoms, he developed numbness in his right arm. Neurological examination revealed no abnormalities at first but, five weeks later, he began to show mild weakness in the right arm. Brain magnetic resonance imaging (MRI) showed hyperintense lesions around the third ventricle and hypothalamus, and MRI of the cervical cord demonstrated high signal intensities between the third and fifth cervical segments. Meanwhile, laboratory tests disclosed evidence of hypo-osmolar hyponatremia. Serological evaluation of anti-aquaporin 4 (AQP4) antibody, the antibody to double-stranded DNA (dsDNA) and the anti-nuclear antibody (ANA), was performed and found to be positive. The patient was finally diagnosed with NMOSD and SLE complicated by CSW. He was treated with methylprednisolone and hypertonic saline after which he demonstrated remarkable improvement of clinical symptoms and hyponatremia. **Conclusions or Comments:** The diagnosis of NMOSD and SLE in a young male patient is challenging at the time of onset. In addition, cerebral salt wasting syndrome is one of the cause of hyponatremia in NMOSD patients.

P-1-203**A case of Sjögren syndrome involving central nervous system with phonic tic**

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Background & Significance: Sjögren syndrome (SS) is a chronic autoimmune disease mainly characterized by the inflammation of exocrine glands. However, a broad spectrum of systemic manifestations may characterize the disease. Central nervous system (CNS) involvement is common in SS patients. But there were no reports of phonic tic in SS with CNS involvement. **Case:** A 62-year-old female had suffered idiopathic thrombocytopenic purpura for 30 years. From 2 years ago, she complained whole body tingling sensation and

weakness of all extremities. For 1 year, she took 5mg of oral prednisolone for idiopathic thrombocytopenic purpura. Motor weakness worsened and phonic tic - sounds like barking of a dog - was developed. She was admitted to our medical center. Brain MRI showed multiple white matter hyperintensity in T2 weighted image. Cervical and thoracic spinal MRI showed longitudinally extensive transverse myelitis. Anti-RO, anti-La antibody were positive. Schirmer's test and lip biopsy were also compatible with diagnostic criteria of SS. We increased the amount of prednisolone and risperidone for phonic tic. Symptoms improved slightly ever since. **Conclusions or Comments:** We report a case of SS involving central nervous system with phonic tic.

P-1-204

Multiple Sclerosis diagnosed by pain and paresthesia shortly after Cervical trauma

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Background & Significance: There is a hypothesis that spinal cord or brain trauma damage BBB, and this damage provoke autoreactive cell to migrate to the Brain. As a result, it suggest that could be involved in Multiple Sclerosis, MS appearance or deterioration to the genetically susceptible person. However, until now, Trauma and MS involvement study are almost about the patient-control group study, and furthermore the results are showing conflicting, so there is no definite conclusion. **Case:** A 39-years-old woman came to the hospital with symptom pain and paresthesia, which occurred shortly after treatment in Oriental Medicine Clinic. The Patient 18 years ago happened to sudden a binocular visual acuity and got a treatment for optic nerve inflammation. She took neuroimaging, analysis of cerebrospinal fluid and evoked potentials and heard that it had low chance of MS at that time. At that time, after one month, every symptoms got better. So the patient was living with regular check in the hospital nearby, no other treatment and medication. Due to the 5-6 years chronic, heavy and dull pains from the backside of neck, both of shoulders and in the larynx, She visited Oriental Medicine Clinic nearby the first time and received a new operation kairopeuraktik. During the operation, there was an action which bended the neck to the right and left extremely. And then she felt the sharp pain. So the operation was stopped after 5-6 minutes. After she came home, the pain on the head proceeded approximately one week, so she took cold pack, stretching, anti-inflammatory and muscle relaxant. But the symptom didn't get better. It changed from 10 days after starting the symptom. An oppressive and dull pain were gone, but both hands started to numb. And similar sensory change was felt also around the neck. When she bended the neck, she felt paresthesia in the back. And the region of paresthesia extended from the both hand to the upper extremity distal more and more. After 5 days of paresthesia change, she came to our hospital and then took a sensory medical checkup. Cranial nerve, motor, sensory examination were normal, but both upper and lower extremities brisk deep tendon reflex and Lhermittes sign were suspicious. In the Brain and Spinal MRI, it was founded T2 hyperintense lesion in mellula. (Fig.) Other extraordinary remarks which can suggest at different times and in different areas were not found In analysis of cerebrospinal fluid no specific finding, anti aquaporin4 antibody was negative, and every aurotimmune antibody checkup including rheumatic factor, etc. were all negative. We made a diagnosis as MS, because it was found that objective remarks such as paresthesia after optic nerve inflammation in the past and objective remarks The patient got IV high dose Steroid, and after that every symptoms were recovery. Even in the follow up neuroimaging, we found it improvement, so she was discharged from the hospital. (Fig.) **Conclusions or Comments:** Kairopeuraktik which aims correction of abnormal joint array with hand and decrease of pain is an operation,

but is known also that it may cause the mechanical damage or side-effects. In the early step of this disease, there is a change that Certain MS patients after having Trauma, it may get worse or cause new symptom, so it could affect to the MS occurrence, recurrence, and deterioration. Specially, trauma in the cephalosome, collum arouse the change in BBB so it would be involved with occurrence. Through many kinds of video study, animal experiment, and laboratory studies, Both sharp trauma and relatively light trauma also cause this BBB change, and furthermore The cervical spine trauma such as Whiplash injury could induce the brain fine structure change or Brain deterioration. In conclusion, regarding to this patient case or results up to now, The patients with predisposition of MS need to be cautious for the injury of Brain or Spine and extreme mechanical handling or operation.

P-1-205

Non-traumatic spinal cord infarction after surfing - a case of surfer's myelopathy

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Background & Significance: Surfing is a rare cause of spinal cord infarction without trauma, especially in novice surfers. We report a case of acute onset myelopathy soon after surfing, so-called 'Surfer's myelopathy'. The underlying mechanism is expected to be acute spinal cord infarction. This case is the first case of Surfer's myelopathy in South Korea. **Case:** A previously healthy 22 year old male was surfing for the first time, lying prone on a surfboard with the lumbar spine hyperextended. Thirty minutes later, the surfer suddenly felt cracking back pain followed by complete paraplegia, complete sensory loss of the lower extremities and urinary retention. Neurological examination revealed complete paraplegia and absent anal tone. Spine MRI revealed pencil-like T2 hyper-intense signal abnormality on the spinal cord extending from T8 level to the Conus medullaris. T1 weighted image was normal. The spinal diffusion weighted image showed high signal intensity with decreased ADC value in T11-T12 level, which was suggestive of spinal cord infarction. CSF findings were RBC 72/ μ L, WBC 18/ μ L and protein 73.7mg/dL. CT angiography of the thoracic aorta was normal. Therapeutic strategies, including intravenous methylprednisolone and daily aspirin showed no clinical improvement. **Conclusions or Comments:** This case is a typical case of Surfer's myelopathy, which is consistent with previously reported cases worldwide. Most of the patients are novice surfers who surf for the first time with prolonged hyperextension of the lumbar spine, soon followed by paraplegia. The etiology is poorly understood, but the most probable one is a vascular phenomenon caused by dynamic compression or vasospasm of the spinal arteries during prolonged hyperextension of the spine. The long-term prognosis depends on the severity of the initial symptom. The more severe the initial motor weakness is, the more likely to remain paraplegic afterwards. The expected prognosis of this case is very poor because he initially presented with complete paraplegia. However, since there are only a few cases reported worldwide, long-term prognosis is so far unknown. There is no known effective treatment reported yet. Clinical managements such as blood pressure management, lumbar drainage or intravenous corticosteroid pulse therapy lack sufficient evidence to support the effectiveness.

P-1-206

A case of acute disseminated encephalomyelitis following pneumococcus vaccination in adult

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Background & Significance: Acute disseminated encephalomyelitis (ADEM) is a monophasic inflammatory demyelinating disease of the central nervous system that typically follows a febrile infection or vaccination. Various viral or bacterial pathogens and a number of vaccinations have been associated with ADEM. Although some case reports of pneumococcal meningitis associated ADEM were reported, ADEM following pneumococcus vaccination was rarely reported. We report a patient who developed ADEM after pneumococcus vaccination. **Case:** A 62-year-old woman present with sudden onset weakness in both lower limbs and right upper limb along with decreased sense over trunk and both lower limb for one day. She had a history of rheumatoid arthritis for 10 years and Hashimoto's thyroiditis. She had taken methotrexate 2.5mg once per week. One month prior to her visit, she received pneumococcus vaccination for traveling abroad. The next day after vaccination she developed fever and swelling of injection site for two days but subsided spontaneously. Two weeks after vaccination she visited our hospital's endocrinology department complaining of fever and anorexia. During admission to endocrinology department, laboratory tests including complete blood counts, electrolyte, liver function test, thyroid function test, C-reactive protein, adrenocorticotropic hormone stimulation (ACTH) test were all normal. Her symptoms improved spontaneously and was discharged. Five days after discharge, she visited neurology department complaining of weakness and decreased sense over trunk and both lower limb. She was mentally alert and vital signs were stable. Neurologic examination revealed a reduced muscle power in both lower limbs and right upper limb assessed as MRC grade 4, sensory level below T4 dermatome. Deep tendon reflexes were hyperactive in all limb. Hoffmann signs and Babinski signs were positive bilaterally. Urinary and bowel function was normal. Spinal MRI showed intramedullary T2-high signal intensities from C1 to C6, and from C7 to T8 level with contrast enhancement. Cerebrospinal fluid analysis showed 50 WBC/ μ l (lymphocyte 92%), protein 90mg/dl, glucose 56mg/dl (serum : 100mg/dl). With the impression of acute transverse myelitis, she was treated with high dose of steroid (methylprednisolone 1g per day) for 5 days and tapered. Her symptoms gradually improved and follow up MRI after 5 days revealed improvement of previous lesion. Brain MRI performed after 5 days after treatment showed multifocal nodular T2-high signal intensities at left parietal lobe and frontal lobe and right pons consistent with ADEM. **Conclusions or Comments:** To our knowledge, although pneumococcal meningitis associated ADEM were reported, pneumococcus vaccination has not previously associated with ADEM. In this case is the first describing a possible association of ADEM with pneumococcus vaccination.

P-1-207

Neuromyelitis optica spectrum disorder associated with syphilis: case study

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Background & Significance: Neuromyelitis optica spectrum disorder (NMOSD) is characterized by optic neuritis, longitudinally extensive transverse myelitis (LETM), or area postrema syndrome. Chronic infection such as HIV or syphilis is one of the red flags of NMOSD. We report a case presenting with LETM and area postrema lesion following treatment of latent syphilis. **Case:** A 72-year-old man presented with diplopia and dizziness in November 2012. Brain MRI showed high signal intensity lesion involving the left dorsolateral medulla on T2 weighted image. He had a history of latent syphilis, and serologic test for syphilis was positive. He was treated with penicillin G intra-

venously, and his symptoms and MRI lesions were improved. Follow up brain MRI in May 2013 showed recent lesions on the left paramedian pons and the central medulla without clinical symptoms. He was treated with penicillin G intravenously again. In December 2014, he presented with altered mentality and dyspnea. Brain and spine MRI showed multiple abnormalities; cervical LETM extending to dorsolateral medulla, thoracic and lumbar myelitis. CSF study revealed pleocytosis (WBC 160) with elevated total protein (74 mg/dL). CSF test for syphilis was negative although serologic test for syphilis was positive. IgG index and oligoclonal band were negative and serum anti-aquaporin-4 antibody was negative. He was treated with methylprednisolone pulse therapy in tentative diagnosis of NMOSD. He was improved nearly completely and mild gait instability was left 6 months later. **Conclusions or Comments:** This case is compatible to NMOSD without AQP4-IgG in terms of clinical symptoms and MRI findings and his symptoms were improved completely after methylprednisolone pulse therapy. Syphilis could lead to chronic CNS infection mimicking NMOSD however syphilis might be one of causes of post-infectious NMOSD like this study. Clinician should have regard to these two aspects when diagnose NMOSD in a patient with syphilis.

P-1-208

Etiology of spontaneous downbeat nystagmus

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Background & Objectives: Downbeat nystagmus is one of the most common types of central nystagmus. However, no study has attempted analyses of clinical features in a large number of Koreans with downbeat nystagmus. **Method:** We analyzed the demographic and clinical features of spontaneous downbeat nystagmus in 218 patients who had an evaluation at the Dizziness Clinic of Seoul National University Bundang Hospital from 2003 to 2014. **Results:** Patients included 117 (53.7%) men with an age ranging from 15 to 92 (mean \pm SD=58.7 \pm 17.9). Underlying causes were found in 162 patients (74.3%, secondary group) while no etiology was identified in the remaining patients (idiopathic group). There were no differences in the age and sex between the idiopathic and the secondary groups. The underlying etiology included hereditary disorders (n=48, 22.0%), vascular diseases (n=46, 21.1%), and inflammatory diseases (n=28, 12.8%). Strokes (n=34, 15.6%) and episodic ataxia (n=32, 14.7%) were the most frequent underlying disorders. The most common site of lesions identified on MRIs was the cerebellum (69.1%) followed by brainstem (28.6%). Among the 74 patients with a confirmed lesion on MRIs, 25 (33.8%) patients showed an isolated cerebellar lesion, while 35 patients (47.6%) had lesions involving multiple areas. **Conclusion:** This study provides clinical features of downbeat nystagmus in Korean patients, which may be distinct from those observed in previous studies involving different ethnic background.

P-1-209

Diurnal variation of upbeat nystagmus: Is this the gravity effect?

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Background & Significance: There have been growing evidences that the gravity may influence vertical eye position and movements. The intensity of downbeat nystagmus decreases during the daytime, while upbeat nystagmus improves when the head is in the horizontal position. We report a patient with diurnal variation of upbeat nystagmus, suggesting a marked role of gravity in

vertical eye position. **Case:** A 37-years-old man presented with acute vertigo, oscillopsia and headache. He showed mixed clockwise torsional-upbeat nystagmus, which was mainly upbeat in the left eye and mostly torsional in the right eye. Horizontal head impulse test was normal in both directions. Brain MRI and MR angiography showed a small tiny infarction in the caudal medulla with a dissecting aneurysm in the right vertebral artery. Perfusion weighted images revealed perfusion defects in the right PICA territory including right medulla. Fundus photography showed counter-clockwise (from the patient's perspective) contraversive torsion of the left eye only (14.9° in the left eye, 7.7° in the right eye, normal range: 0-12.6°). He also showed contraversive leftward tilt of the subjective visual vertical (-10.0° in the left eye, -6.2° in the right eye, normal range: -3.0° to 3.0°, negative value indicates leftward tilt). Bithermal caloric tests were normal. After admission, he noticed that his oscillopsia improved after waking up in the morning, but became aggravated during the daytime. Video-oculography documented that the mean slow phase velocity (SPV) of upbeat nystagmus at 5 PM (18°/sec) was more pronounced than after waking up at 9 AM (3.2°/sec). The SPV of upbeat nystagmus was not changed immediately after lying down from an upright position. However, the intensity of upbeat nystagmus was more attenuated after a prolonged supine position than after taking daily activities. The diurnal variation of upbeat nystagmus has lasted two weeks or more. **Conclusions or Comments:** Diurnal variation of upbeat nystagmus in our patient suggests that vertical nystagmus can be affected by head position relative to gravity. The crossing ventral tegmental tract (CVTT) has been proposed as the efferent tract of an antigravitational pathway originating from otolith. In the horizontal position of the head, upbeat nystagmus may improve since the antigravitational activity is no longer required.

P-1-210

Assessment of vestibular recovery using video head impulse test after vestibular neuritis

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Background & Objectives: Recovery from symptoms of vestibular neuritis may take days to weeks. Factors affecting the speed of recovery is not well known. To evaluate the vestibular function with video head impulse test (vHIT) for long-term prognosis and to look for the association between poor recovery and initial vestibular function test results. **Method:** 31 (16 male, 15 female) vestibular neuritis inpatients with less than 48 hours after symptom onset from August 2014 to June 2015 are recruited. Recovery pattern of vestibular function was able to be determined in 22 patients with vHIT data. Rotatory chair test (RCT), vHIT, cervical and ocular vestibular myogenic potential (o- and c-VEMP), fundus photography, caloric test, subjective visual vertical (SVV), pure tone audiometry are done within 5 days from onset during admission period. Daily bedside examination includes observation of spontaneous nystagmus, head-shaking nystagmus, vertigo analogue scale, 3-point verbal rating scale of activities of daily living (ADL) impairment. Follow-up bedside examination and video head impulse test are performed at 1 week, 1, 3, and 6 months after discharge. **Results:** 3 patterns of serial vHIT gain are identified from 22 patients. Initial normal gain from onset (group 1, n=7), recovery from the low gain (group 2, n=9), poor recovery of gain (group 3, n=6). All 4 group 3 patients had residual symptom at 30 days after onset. In the 15 patients with normal gain at 30-day follow-up (group 1+2), 3 patients (20%) still complained residual symptom affecting ADL. Duration of spontaneous nystagmus (grade 2+grade 3) with fixation was 4 days in group 2 and 3, only 2 days in group 1. Among laboratory tests, SVV angle (mean 0.9, 5.3, 13.9 degree in group 1,2,3 respectively, $p<0.001$), ocular torsion (ipsilesional 7.6, 10,

21.9 degree, $p=0.002$, contralesional 3.5, -0.4, -9.2 degree, $p=0.012$), RCT vestibulo-ocular reflex gain (p value 0.01, 0.003, 0.009 in 0.02Hz, 0.08Hz, 0.64Hz respectively), and initial ipsilesional lateral canal vHIT gain (1.13, 0.52, 0.3 in each group, $p<0.001$). Ipsilesional o-VEMP wave was absent in 17% (1/5) in group 1, 66% (6/9) in group 2, 100% (6/6) in group 3. But caloric paresis was not associated with recovery. **Conclusion:** Initial bedside examination of spontaneous nystagmus and laboratory vestibular function tests including vHIT are useful in the assessment of prognosis after vestibular neuritis. The severity of vestibular impairment is associated with poor recovery at long-term follow-up.

P-1-211

A case of inferior vestibular neuritis shown at video head impulse test

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Background & Significance: Acute vestibular neuritis (VN) is a commonly encountered vertigo disorder, mostly involving the superior vestibular nerve. The vestibular neuritis with an involvement of only the inferior vestibular nerve is a rare condition which involves posterior semicircular canal exclusively. The video head impulse test is a useful method for evaluation of each semicircular canal. We present the result of video head impulse test of inferior vestibular neuritis. **Case:** A 46-year-old female visited the clinic with continuous vertigo. At vestibular function test, she showed spontaneous clockwise and downbeat nystagmus without positional nystagmus. Caloric test and subjective visual vertical test were normal, but fundus photograph showed ocular torsion to the left. Both cervical and ocular vestibular myogenic potential were normal. In video head impulse test, bilateral lateral and anterior semicircular canal gain were normal but in the left posterior circular canal, the gain was decreased to 0.43 and catch-up saccades were observed. **Conclusions or Comments:** As the inferior vestibular neuritis is rare and does not show typical features of vestibular neuritis, it can be misdiagnosed as central vertigo. Recently, the video head impulse has been introduced but so far there is little clinical experience. We experienced a case of inferior vestibular neuritis proven with the result of video head impulse test. The video head impulse test is the reliable and convenient method for evaluation of each semicircular canal quantitatively.

P-1-212

Persistent otolith dysfunction even after successful repositioning in benign paroxysmal positional vertigo

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Background & Objectives: The purpose of this study was to characterize utricular or saccular dysfunction in patients with BPPV using cervical and ocular VEMPs in response to air-conducted sound (ACS) and changes of VEMP findings after successful repositioning maneuvers. **Method:** ocular and cervical vestibular evoked myogenic potentials (VEMPs) were studied in 112 patients with BPPV and 50 normal controls in a referral-based University Hospital. Ocular (oVEMPs) and cervical VEMPs (cVEMPs) were induced using air-conducted sound (1000 Hz tone burst, 100 dB normal hearing level) at the time of initial diagnosis and 2 months after successful repositioning in patients with BPPV, and the results were compared with those of the controls. **Results:** Abnormalities of cVEMPs and oVEMPs in patients with BPPV were prevalent

and significantly higher compare to the healthy control group ($p < 0.01$ in each VEMP by chi-square test). In the patient group, difference between the proportions of abnormal responses of cVEMP and oVEMP was not significant in both affected ($p = 0.37$, chi-squared test) and non-affected ($p = 1.00$) ears. The abnormalities were more likely reduced or absent responses rather than delayed ones; reduced or absent responses are 17.6% in cVEMPs ($p = 0.04$, chi-square) and 21.6% in oVEMPs ($p < 0.01$). The non-affected ear in the BPPV group also showed significantly higher abnormalities of cVEMP and oVEMP when compared to the control group. The follow-up VEMPs after repositioning maneuvers were not significantly different compared to the initial values from both stimulated affected and non-affected ears. **Conclusion:** Although unilateral clinical presentation of BPPV, bilateral otolithic dysfunction revealing persistent reduced or absent response of cervical and ocular VEMPs suggests that BPPV may be a disorder caused by a significant bilateral damage of the otolith of both utricle and saccule.

P-1-213

Reduced resting-state functional connectivity in the vestibular cortical network in normal aging

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Background & Objectives: To evaluate age-related alterations in the functional connectivity of intrinsic brain activity in the vestibular cortex and correlation of vestibular functional connectivity changes with the vestibulo-ocular and vestibulo-otolith functions. **Method:** The spontaneous fluctuations within brain systems using resting-state functional magnetic resonance imaging were examined in healthy subjects ($n = 31$). We used seed voxel approach and chose the posterior insular and parietal operculum as seeds. Vestibulo-ocular reflex (VOR) gain using video head-impulse test and ocular and cervical vestibular-evoked myogenic potentials (VEMPs) were examined in all participants. We also performed a voxel-based morphometry as a control to detect age-related brain atrophy that might be unrelated to vestibular processing. **Results:** We found that in the resting state, the pattern of connectivity with both the posterior insular and the parietal operculum were changed in old age group. Connectivity with the vestibular cortex in the old subjects ($n = 16$) compared to young subjects ($n = 15$) was decreased with the lateral superior occipital and lingual gyrus, superior frontal gyrus, postcentral gyrus, the superior temporal gyrus, the inferior frontal gyrus, and with the cerebellum. These results remain significant after correction for resting state network-specific gray matter volume. In contrast, there was no significant correlation between the functional connectivity in the vestibular cortex and the VOR gain and VEMPs performance. **Conclusion:** Age-dependent functional connectivity decrease in the vestibular cortex with the somatosensory, language and cerebellar network independent of the vestibular functions indicate a disruption of vestibular integration of multimodal sensory inputs and cause deteriorated vestibular and balance function in elderly.

P-1-214

Positional nystagmus in lateral semicircular canal fistula

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Background & Significance: Typical symptoms of labyrinthine fistula are posi-

tional vertigo, severe disequilibrium, and sensorineural hearing loss, collectively making it difficult to differentiate from BPPV. The positional nystagmus is showed both conditions, but the patterns and mechanism of nystagmus is different. **Case:** A 77-years-old man was developed sudden onset of episodic vertigo and unsteadiness. He had not been listening in his left ear from left middle ear surgery 40 years ago. 4 months ago, he was diagnosed adhesive otitis media with cholesteatoma and performed left middle ear surgery again (the name was radical mastoidectomy with tympanoplasty). After second ear surgery, He was develop episodic vertigo with left beating nystagmus when compressing tragus of his left ear and the nystagmus was disappeared when stopping compression. He also showed right beating nystagmus in supine position and left beating nystagmus in sitting position. But, He didn't showed nystagmus in other positions. The temporal bone CT revealed left lateral semicircular canal fistula and the nystagmus was disappeared after closing the fistula. **Conclusions or Comments:** The mechanism of positional nystagmus might be related to the change of intracranial pressure.

P-1-215

Perverted head-shaking nystagmus; a few examples of peripheral vestibular disorder

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Background & Objectives: Perverted head-shaking nystagmus (pHSN) refers to the nystagmus that develops in the plane other than that being stimulated by head shaking, i.e., downbeat or upbeat HSN after horizontal head shaking. It has been considered to be a central pattern of HSN. Little data is available regarding how frequently pHSN occurs and its significance in subjects with central or peripheral vestibular disorders. In this study, we would like to determine clinical significance of the head-shaking nystagmus test (HSN) and perverted HSN in patients with unilateral peripheral and central vestibular disorders. **Method:** We reviewed the medical records of 889 consecutive subjects who were referred to a dizziness clinic of Chonbuk national university hospital from March 2010 to May 2015. Nystagmus was registered in darkness for 60 sec before head shaking, during head shaking, and for 100 sec after head-shaking. HSN was considered to develop when the post-head-shaking nystagmus last at least 5 beats with latency from the end of head-shaking of no more than 5 sec, and a velocity of at least 2.5 deg/sec. **Results:** Perverted HSN was observed in 8.2 % (73/889) of all patients including many kinds of central vestibular disturbances such as stroke, vestibular migraine, cerebellar ataxia, and vertebro-basilar insufficiency (VBI). However, pHSN was also observed in peripheral vestibular disorders including BPPV especially involving the vertical canals, Meniere disease and even in the vestibular neuritis. **Conclusion:** Our results show that the perverted HSN in dizzy populations was frequently appeared and that pHSN occurs not only in cases of central vestibular disturbances but also in cases of peripheral disorders. Perverted HSN may not be specific in distinguishing peripheral hypofunction from more central vestibular imbalances such as difference in the vestibular velocity storage mechanism.

P-1-216

Changes in resting-state functional magnetic resonance imaging in vestibular neuritis

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Background & Objectives: The aim of this study was to elucidate (i) whether there are changes of cerebral resting-state networks with respect to functional interregional connectivity (resting-state activity) in acute and chronic stage in patients of vestibular nucleus (VN), and (ii) whether these are related to neurophysiological, perceptual and functional parameters of vestibular-induced disability. And the aim was to understand the mechanism related to the activity of the vestibular cortex. **Method:** We studied 25 right-handed patients (mean age of 54.5 ± 11.9) with acute unilateral VN at two time points: in the acute stage and 3 months later. The age- and gender-matched 20 healthy subjects (mean age of 48.8 ± 11.7 years) served as normal controls. Patients were examined within the first 7 days (acute) of the disease using a battery of clinical and neuro-otological tests, subjective and objective disability scores (including dizziness handicap inventory, scale for the assessment and rating of ataxia), electrophysiological investigations (including videooculography, audiometry, calorimetry, rotator chair test, ocular and cervical vestibular evoked myogenic potential, subjective visual vertical), and functional magnetic resonance imaging (fMRI). And patients were examined within after 3 months (chronic) of the disease using a battery of clinical and neuro-otological tests, subjective and objective disability scores and fMRI. The normal control subjects examined the clinical test and fMRI. Using independent component analysis (ICA), we compared resting-state networks between the patients with VN and normal control subjects, and we compared the resting-state networks between the acute and chronic stage in the patients with VN. **Results:** A neural network (component 30) comprising the parietal lobe, medial aspect of the superior parietal lobule, posterior cingulate cortex, middle frontal gyrus, middle temporal gyrus, parahippocampal gyrus, anterior cingulate cortex, insular cortex, caudate nucleus, thalamus and midbrain was modulated between acute VN patients and normal controls and in patients over time. Within this network, acute VN patients showed decreased resting-state activity (ICA) in the contralesional intraparietal sulcus (IPS), supramarginal gyrus (SMG), posterolateral thalamus, posterior and retroinsular cortex, parietal operculum, parieto-insular vestibular cortex (PIVC), superior temporal gyrus (STG, BA 22), inferior parietal lobe (IPL), anterior cingulate cortex, hippocampus, which increased after 3 months. Resting state activity in IPS tended to increase over 3 months in VN patients. VN leads to a change in resting-state activity of the contralateral IPS adjacent to the SMG, which reverses during vestibular compensation over 3 months. **Conclusion:** In our data, the altered resting-state activity in a network of multisensory cortical areas important for vestibular processing, spatial orientation and spatial working memory. More specifically, there was a trend that the extent of the partial reversal of decreased resting-state activity of the contralateral IPS in VN patients was related to the amount of vestibular induced disability in daily life after 3 months. This may indicate powerful restitution-related or compensatory cortical changes in resting-state activity.

P-1-217

Analysis of vestibulo-ocular reflex in patients with cerebellar ataxia using video head impulse tests

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Background & Objectives: The cerebellum is not an essential structure for generating vestibulo-ocular reflex (VOR), but it is critical for modulating VOR. Various cerebellar ataxic disorders including spino-cerebellar ataxia (SCA) and the cerebellar type of multiple system atrophy (MSA-C) may have common symptoms such as ataxia and dysarthria. However, regarding VOR gain, various results have been reported even though VOR gain was usually decreased.

Video head impulse test (vHIT) is a useful test for differentiating acute stroke causing vertigo from acute peripheral vestibulopathy, and it allows objective and quantitative measurements of VOR. In this study, we analyzed VOR gains and compensatory saccades in various cerebellar ataxias using vHIT. Also, we want to know if there is any clinical factor that affect VOR gains in cerebellar ataxia. **Method:** From January 2014 to June 2015, we identified 30 patients with chronic, progressive cerebellar ataxia who had the VOR data measured by vHIT device. The cerebellar ataxia group (n=30) were classified into 4 clinical diagnosis; 1) genetically confirmed SCA, 2) MSA-C, 3) cerebellitis, 4) idiopathic cerebellar atrophy. We defined 42 patients as normal control subjects who showed normal findings on neurologic examination, brain MRI, and vHIT. We compared VOR gains of each canals between cerebellar ataxia and control. Furthermore, according to the result of VOR gain of the vertical canals, the ratio of VOR gain of anterior canal (AC) and that of posterior canal (PC) (AC/PC ratio) was calculated and compared with normal control. Cerebellar ataxia group was classified into 3 subgroups (i.e., increased AC/PC ratio, normal AC/PC ratio, decreased AC/PC ratio). And then, we analyzed the clinical characteristics (i.e., clinical diagnosis, demographic factors, disease duration and severity) between each subgroups. Finally, we summarize the result of VOR gains and patterns of compensatory saccades according to SCA subtypes. Statistical analysis was using the SPSS, statistical significance was set at $P < 0.05$. **Results:** In 30 patients with cerebellar ataxia, men were 13, and average age was 57 ± 14 (range: 21-76). The number of each clinical diagnosis was as follows; SCA were 6, MSA-C 13, idiopathic cerebellar atrophy 10, and cerebellitis 1. In the direct comparison of VOR gains between cerebellar ataxia and normal control, VOR gain of the horizontal canal was not different ($p=0.17$), but VOR gains of AC and PC were significantly decreased in cerebellar ataxia group ($p=0.007, 0.046$, respectively). To investigate more about the decreased VOR pattern in the vertical canals, AC/PC ratio of VOR gain was calculated in both cerebellar ataxia. In the comparisons among three subgroups, clinical diagnosis and disease duration were significantly different ($p=0.04, 0.008$, respectively). In detail, the increased AC/PC ratio group (n=7) had much longer disease durations and more common SCA6 diagnosis, while the others (17 normal and 6 decreased AC/PC ratio) had shorter disease durations and more common diagnosis of MSA-C and/or idiopathic cerebellar atrophy. SCA 6 (n=4) had markedly decreased VOR gains and significant compensatory saccades, while SCA 2 and 7 had almost normal VOR gain and no definite compensatory saccades. In addition, AC/PC ratio was mostly increased in SCA 6. **Conclusion:** We found that cerebellar ataxia patients had significantly lower VOR gain in vertical semicircular canal planes than normal control. Furthermore, some cerebellar ataxia patients had relatively increased VOR gains in the anterior canal, and it is correlated with the longer disease duration and the diagnosis of SCA6. The vestibulo-cerebellar dysfunctions including downbeat nystagmus may be compatible with the finding of increased AC/PC ratio of VOR gain due to a disinhibition of the AC pathway. In the future, analysis of VOR gain through vHIT may helpful for the differential diagnosis of various cerebellar ataxias.

P-1-218

Modulation of nystagmus by vision, proprioception, and efference copy signals: a systematic study

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Background & Objectives: The vestibular nucleus neurons use non-vestibular sensorimotor signals to modulate their activities in accordance with the behavioral goals. For adaptation, they also change their responsiveness to non-ves-

tibular signals in unilateral vestibular loss. These non-vestibular signals can generate nystagmus, known as arthrokinetic nystagmus in normal subjects. In lesions at or near to the vestibular nuclei, non-vestibular signals may induce nystagmus, such as mastication- or swallowing-induced nystagmus. However, influence of non-vestibular signals on these types of nystagmus has not been investigated in a systematic way, and the underlying mechanisms of modulation remain obscure. **Method:** In the present study, using several non-vestibular sensorimotor inputs, we evaluated modulation of central nystagmus observed in a patient with rhombencephalitis mostly involving the left dorso-lateral medulla. **Results:** The nystagmus was induced or significantly modulated by i) visual inputs, and ii) combined proprioceptive and efference copy signals (during voluntary motion) unrelated to spatial orientation. In contrast, isolated proprioceptive signal, mental set, or non-proprioreceptive somatosensory input had a negligible effect on the nystagmus. **Conclusion:** Based on these modulations, we suggest that i) the visually-mediated nystagmus is due to a lesion-induced pursuit asymmetry and ii) the nystagmus induced during voluntary motion is due to erroneous contribution of combined proprioceptive and efference copy signals in integrating the non-vestibular signals in human.

P-1-219

X-linked adrenoleukodystrophy presenting with positional downbeat nystagmus

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Background & Significance: Downbeat nystagmus is a sign of CNS dysfunction and observed in disease involving the vestibulocerebellum. There are various causes of downbeat nystagmus including brainstem infarction, multiple sclerosis, cerebellar ectopia, and cerebellar degeneration. X-linked adrenoleukodystrophy is a progressive neurodegenerative disease that characteristically affects the white matter of CNS, mainly presenting cognitive deterioration, hemiplegia or quadriplegia, cerebellar ataxia and often seizures. Even though clinical course appears to vary depending on the phenotype, initial dizzy presentation with downbeat nystagmus is very rare to our knowledge. Here, we report a case of X-linked adrenoleukodystrophy presenting with downbeat nystagmus. **Case:** A 20-year-old man was admitted due to positional dizziness and imbalance. The symptom began two months before the admission and did not show any improvement with antivertigo medications prescribed by other clinics. He had no personal or familial history of any underlying diseases. Neurologic examination revealed bilateral dysmetria and truncal ataxia. Babinski reflex and ankle clonus were positive on both sides. The video-oculography showed normal smooth pursuit, subtle spontaneous downbeat nystagmus (1 deg/sec) and aggravated downbeat nystagmus when changing position from sitting to head bending (10 deg/sec) or straight head hanging (24 deg/sec). The caloric test and vestibular evoked myogenic potentials were normal. Brain magnetic resonance imaging revealed high-signal intensity lesions on T2-weighted image involving the bilateral cerebellar white matter, internal capsules, splenium of corpus callosum, and the pyramidal tract of the brain stem. On spine MRI, no abnormal signal intensity was found. The fasting plasma levels of very long chain fatty acid were as follows: C22:0, 25.49 μmol/L (normal, 0-96.3), C24:0, 51.49 μmol/L (normal, 0-91.4), and C26:0, 2.78 μmol/L (normal, 0-1.3). The C24:0/C22:0 and C26:0/C22:0 ratios were elevated to 2.02 (normal, 0-1.39) and 0.109 (normal, 0-0.023), respectively. Genetic study was performed using a peripheral blood sample. The ABCD1 gene in the patient revealed a missense mutation (c.871G>A), which is a pre-

viously reported mutation in X-linked adrenoleukodystrophy. The patient was diagnosed with X-linked adrenoleukodystrophy and started treatment with Lorenzo's oil. **Conclusions or Comments:** The mechanism of downbeat nystagmus is thought to be caused by vestibulo-cerebellar dysfunction responsible for vertical semicircular canal or unbalance of vertical smooth pursuit system. With cerebellar white matter lesions involving the flocculo-nodular lobe, a release of inhibition upon anterior semicircular pathways may be a possible cause of downbeat nystagmus in this patient. When downbeat nystagmus occurs in young age accompanied by upper motor neuron signs and ataxia, X-linked adrenoleukodystrophy should be considered from an etiologic perspective.

P-1-220

Variants of windmill nystagmus

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Background & Significance: Windmill nystagmus is characterized by a clock-like rotation of the beating direction of a jerk nystagmus suggesting separate horizontal and vertical oscillators, usually 90° out of phase. **Case:** We report oculographic characteristics in three patients with variants of windmill nystagmus in whom the common denominator was profound visual loss. Two patients showed a clock-like pattern while in the third the nystagmus was largely diagonal (in phase or 180° out of phase) but also periodically changed direction by 180°. Different phase relationships between independent horizontal and vertical oscillators could account for these different patterns. **Conclusions or Comments:** We hypothesize that windmill nystagmus is a unique manifestation of "eye movements of the blind". It emerges when the central structures, including the cerebellum, that normally keep eye movements calibrated and fixation steady, can no longer perform their task because they are deprived of the retinal image motion that signals a need for adaptive recalibration.

P-1-221

Perverted Head-shaking nystagmus in central lesions: characteristics and mechanism

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Background & Objectives: Perverted head-shaking induced downbeat nystagmus (pHSDN), has been known as a possible sign indicating central pathology. There have been a few explanations for the pathophysiologic mechanism of pHSDN. However, the exact mechanism of this sign is still unknown. In present study, we try to disclose the mechanism of pHSDN by analyzing it from patients with obvious central lesion. **Method:** We recruited 16 consecutive patients with pHSDN from central lesions. We hypothesized that pHSDN is originated from anterior semicircular canals. To test our hypothesis, we measured or calculated the duration, temporal pattern, rotational axis, and time constant of pHSDN. In addition, 5 patients were also received vertical head-shaking test. Then, we compared time constant of downbeat nystagmus from horizontal and vertical head-shaking test. **Results:** The pHSDN was evident after ceasing the head-shaking, and reached its peak velocity within the first 2 to 3 beat of nystagmus. The intensities of following nystagmus decreased

over time with exponential fashion. The mean time constant of pHSDN was 5.8 ± 1.45 seconds and the rotational vector for pHSDN was located between both anterior canals. The downbeat nystagmus from horizontal and vertical head-shaking test had a similar time constant. **Conclusion:** Our study showed that the pHSDN was originated from central lesion induced anterior canal predominance. Based on this finding, we speculate that pHSDN can be observed in peripheral vestibular dysfunction.

P-1-222

Vitamin D status in patients with myasthenia gravis: a pilot study

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Background & Objectives: Myasthenia gravis (MG) is a chronic autoimmune neuromuscular disease generally mediated by ant-acetylcholine receptor autoantibodies. Vitamin D has important roles both in the autoimmune response and in skeletal muscles. We investigated the levels of 1, 25-dihydroxy vitamin D [1, 25(OH)D] and 25-hydroxy vitamin D [25(OH)D] in patients with MG and healthy subjects to determine whether vitamin D deficiency is present in MG. **Method:** Plasma levels of 1, 25(OH)D and 25(OH)D were analyzed in 18 patients with MG (11 females; mean age, 54 years) and in 18 healthy age- and sex-matched blood donors without vitamin D3 medication. **Results:** MG patients without pre-existing vitamin D3 supplementation had lower plasma 25(OH)D levels (mean, 17.3 ± 7.3 ng/mL) than healthy controls (26.9 ± 6.1 ng/mL) ($p < 0.05$). 1, 25(OH)D levels showed slightly high in MG patients (46.4 ± 21.9 ng/mL) than healthy controls (40.1 ± 7.0 ng/mL), but had no significant difference between two groups. **Conclusion:** Plasma 25(OH)D levels significantly lower in patients with MG compared with healthy controls. We recommend monitoring of vitamin D status in patients with MG to avoid direct negative effects on the muscles or autoimmune response.

P-1-223

Clinical and pathologic features of point mutations in Dystrophinopathy

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Background & Objectives: Most common type of muscular dystrophy is Dystrophinopathy, which is included with Duchenne muscular dystrophy (DMD) and Becker muscular dystrophy, and caused by DMD gene defect. Vast majority of mutations are deletions or duplications, and these mutations can be tested by commercially available gene tests in these days. Remaining 15-20% of mutations are point mutations in coding sequence or splicing sites. Relatively these point mutation, can provoke dystrophinopathy patients, have not been studied well yet. The aim of this study is to investigate clinical features of point mutations in DMD gene. **Method:** This study is a single-centered retrospective observational study of the patients with dystrophinopathy who underwent targeted sequencing to identify the genetic cause of myopathy. Targeted sequencing was performed to test 69 myopathy-causative genes including DMD gene. Medical records were reviewed to investigate clinical and pathologic features of the disease. Those variables were compared according to mutation types (nonsense mutation, splicing site mutation, frameshift mutation). **Results:** For December 2003 to July 2014, among clinically suspected dystrophinopathy patients without deletion or duplication in routine DMD gene test, we found various point mutations in 21 patients (mean age: 16.5 ± 12.3 years, 21 males). Of them, 10 (48%) nonsense mutations, 7 (33%)

splicing site mutations and 4 (19%) frameshift mutations were observed. Immune histochemistry pattern study showed partially decreased dystrophin in 4 cases. Clinical and pathologic features of the patients including onset age, age at diagnosis, blood creatinine kinase level, occurrence of cardiac abnormalities, or Dystrophin immunohistochemistry patterns of muscle specimen were statistically not different among mutation types. **Conclusion:** Since it is important to do early diagnosis and care dystrophinopathy patients with routine check-up and proper management for their prognosis, if dystrophinopathy is clinically suspected and there is no exon deletion or duplication, it is necessary to progress further genetic study to detect point mutation of DMD gene.

P-1-224

Ultrasound helps discriminating true carpal tunnel syndrome in diabetic polyneuropathy

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Background & Objectives: It is well known that carpal tunnel syndrome (CTS) and diabetic polyneuropathy (DPN) frequently occur concomitantly. Because symptoms of DPN may mimic those of CTS and nerve conduction studies of DPN may produce similar abnormalities in median nerve conduction as CTS, confirming a clinical diagnosis of CTS in patients with DPN is difficult. Median nerve ultrasound (US) shows increased cross-sectional area (CSA) in CTS and increasingly used for evaluating CTS. However, US measurement to assess of the median nerve CSA in DPN has rarely been reported. The aim of this study was to evaluate the diagnostic value of US in CTS with DPN by measuring median nerve CSA. **Method:** One hundred forty six patients with NCS confirmed CTS in at least one wrist were evaluated. Median nerves were assessed into five groups: diabetes with CTS but without DPN (n=27), DPN only (n=23), DPN with CTS (n=143), controls without CTS (n=30), and controls with CTS (n=69). The median nerve CSA was measured at 3 levels (inlet, outlet, and forearm), and the wrist-to-forearm ratio (WFR) and inlet-to-outlet ratio (IOR) were calculated. We also assessed clinical characteristics including symptom with historical-objective scale (Hi-Ob scale) and body mass index. **Results:** The median nerve CSAs at inlet and outlet were larger in DPN with CTS (13.94 ± 5.38 mm², 13.38 ± 4.52 mm², respectively) compared with DPN only (9.83 ± 2.55 mm²; $p < 0.001$, 11.35 ± 3.33 mm²; $p = 0.014$, respectively). The area under the curve (AUC) for CSA measurements at all three anatomical levels demonstrated that the CSA at inlet (AUC; 0.744, $P < 0.001$) had the most optimal threshold value for identification of CTS in DPN (12.5 mm²) with a sensitivity of 50.7% and a specificity of 87.0%. WFR and IOR were also significantly higher in DPN with CTS than DPN only. There was significant difference in median CSA at inlet between controls with and without CTS (12.51 ± 3.36 mm² vs 10.07 ± 1.31 mm², $p < 0.001$). However, the CSAs at inlet revealed no significant differences among the groups with CTS, including control with CTS, DPN with CTS, and diabetes with CTS but without DPN. There were no differences in median CSA between DPN only and control without CTS. The CSA at inlet was positively correlated with median terminal latency, and negatively correlated with amplitude and nerve conduction velocity (NCV) of median compound motor action potential (CMAP), and amplitude and NCV of the median sensory nerve action potential (SNAP). Furthermore, in NCS confirmed DPN with CTS, 26.6% presented no symptoms and 27.3% revealed no swelling in median nerve US, implicating that some portion of electrophysiologically diagnosed CTS with DPN may not have true CTS and may be just as the finding of distal median nerve involvement in the process of DPN. **Conclusion:** The CSAs of the median nerve at inlet are useful to detect CTS in diabetes and some NCS confirmed CTS with DPN presented neither swelling nor symptoms. These implicates that US may

provide supportive evidence for the diagnosis of true CTS in diabetic patients.

P-1-225

High serum B-cell activating factor levels in myasthenia gravis

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Background & Objectives: Myasthenia gravis (MG) is a B-cell-mediated disease in which the target autoantigen is the acetylcholine receptor at the post-synaptic membrane of the neuromuscular junction. B-cell-activating factor (BAFF) is an important factor in B cell development and activation. Because of its effects on B cells, the possibility that BAFF is playing a role in autoimmunity in patients with MG is important to investigate. **Method:** To test whether serum BAFF levels are increased in MG patients, we tested serum samples from 20 MG patients (11 females; mean age, 54) and in age- and sex-matched 20 healthy controls. We compared the serum BAFF values between ocular and generalized MG. In addition, we analyzed BAFF levels according to anti-acetylcholine receptor antibodies status. **Results:** Mean serum BAFF levels were 744.36±273.44 pg/mL in the healthy group and 1202.12±419.03 pg/mL in MG patients, which were significantly higher in the MG patients. Among 6 ocular and 14 generalized MG patients, serum BAFF levels were 837.6±352.72 pg/mL and 975.75±291.45 pg/mL, respectively, and did not differ significantly. Serum BAFF levels showed no significant differences between MG patient with both anti-acetylcholine receptor binding and blocking antibodies and with either antibodies (828.3±331.74 pg/mL vs 980.4±300.45 pg/mL). **Conclusion:** BAFF values did not correlate with disease severity and antibody status. Although serum BAFF levels do not correlate with disease severity, our data suggest that BAFF is likely to play a role in the pathogenesis of MG by promoting the survival and maturation of autoreactive B cells.

P-1-226

The overlap of Arnold-Chiari malformation type 1 in a patient with Charcot-Marie-Tooth disease type 2D

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Background & Significance: Charcot-Marie-Tooth disease type 2D (CMT2D), which is caused by mutation in GARS gene, is known to be more severe in the upper extremities. **Case:** A 21-years old Korean man complained of gait disturbance and frequent falls. Physical examination showed bilaterally-symmetric, distal dominant weakness on the both sides of the lower limbs (motor power of ankle joint - plantar flexion (MRC 3), dorsiflexion (MRC 4-)). Additionally, mild, asymptomatic weakness (MRC 4+) of the both hands were also observed. Nerve conduction study revealed reduced compound muscle action potentials of peroneal nerve, tibial nerve, and median nerves of both sides. Needle electromyography revealed active and chronic neurogenic changes on the atrophied muscles affected. MR images of the lower limbs revealed the significant fatty degeneration, more severe at the posterior group muscles. MR images of the brain and cervical spinal cord revealed the inferior herniation of the cerebellar tonsil through the foramen magnum, and focal syringomyelia at the level of C6-7, which is consistent with Arnold Chiari Malformation type 1 (ACM1). The patient's mother showed distal muscle weakness on physical examination, and the results of electrophysiologic studies was very similar to the patient. For the genetic evaluation of dominantly-inherited distal weakness, whole exome sequence analyses were performed in 3 (affected patient and his mother, and unaffected mother's sister). As a result, a

known heterozygous mutation of c.794C>T (p.Ser265Phe) was found in the affected, but not in the unaffected. **Conclusions or Comments:** Here, we report the selective involvement of lower limb and forearm in a case with the rare co-existence of CMT2D and ACM1.

P-1-227

Facial spasm associated with neuromyelitis optica spectrum disorder

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Background & Significance: The term neuromyelitis optica spectrum disorders (NMOSD) include definite NMO and limited forms of the central demyelinating disease in AQP4-IgG-seropositive patients. There have been some reports of movement disorder, such as paroxysmal dystonia associated with MNOSD or multiple sclerosis. Here we report a case of the NMOSD patient presenting a facial spasm that has not been previously reported. **Case:** A 31-year-old woman visited the hospital due to bilateral legs weakness and voiding difficulty on the day admission. 3 days before admission, burning sense had occurred in her whole body except the head. Neurologic examination of the patient showed proximal dominant weakness (MRC grade IV) with hypesthesia in bilateral legs. Brain and spinal cord MRI revealed diffuse T2 high signal intensity and swelling involving medulla and C1-T11 spinal cord. She was managed with intravenous methylprednisolone pulse therapy. However, bilateral legs weakness aggravated progressively (MRC grade I) and right arm weakness developed. Plasmapheresis was performed with five cycle every other day. To confirm the diagnosis of NMO syndrome, NMO-IgG was assessed and the result was positive. Weakness and sensory symptom had been improving progressively, however paroxysmal tonic spasm was developed in bilateral leg on admission day 10. On admission day 24, she complained of left lower facial involuntary movement, that was compatible with lower facial spasm. For the facial spasm she was given oxcarbazepine in escalating doses and then facial spasm was mildly improved. **Conclusions or Comments:** Hemifacial spasm in multiple sclerosis patients have been described in some reports. It has been shown to be caused by involvement of the ipsilateral facial nucleus. In our MNOSD patient, the lesion was revealed in the left medulla on brain MRI. Facial corticobulbar fibers seems to descend caudally to at least the middle medullary levels before most of them cross to the opposite facial nucleus, and for this reason, left lower facial spasm appeared in our case

P-1-228

A case of ocular myasthenia gravis initially presenting with sudden onset complete third nerve palsy

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Background & Significance: Ocular myasthenia gravis(MG) is an autoimmune disorder of the neuromuscular junction characterized by fluctuating ptosis and/or diplopia. It is the most common initial presentation of MG and has been known to mimic third, fourth, and sixth nerve palsies. Unlike the true third nerve palsies, MG rarely affects pupillary function. We report a patient who presented with complete third nerve palsy ultimately diagnosed with ocular myasthenia gravis. **Case:** A 54-year-old male patient presented with sudden onset diplopia and complete right ptosis. He had no past history of medical illness and no recent head trauma. Neurological examination showed complete limitation of elevation, depression and adduction on his right eye movement. Right pupil was asymmetrically dilated(0.5mm/0.3mm) but direct and con-

sensual light reflex of both eyes was normal. Other cranial nerve function tests were normal. The result of laboratory tests including thyroid function test were normal. Brain MRI and MRA yielded no significant findings. CSF analyses revealed normal cell count and pressure. With the impression of ischemic third nerve palsy, he was treated with anti-platelet. After 1 month, he reported that his symptoms worsened at the end of the day and seemed better in the morning. Jolly test performed in a hand muscle and orbicularis oculi was normal. Acetylcholine receptor antibody was not detected. Neostigmine test reveals some improvement of his symptoms. With the impression of ocular myasthenia gravis, he was treated with 30mg pyridostigmine every 12 hours, which dramatically improved his symptoms. **Conclusions or Comments:** In this case, sudden onset complete third nerve palsy without fluctuating weakness was an initial manifestation of ocular myasthenia gravis. Ocular MG is often underestimated if ophthalmoplegia comes on acutely over less than 1 week without fatigable weakness. This case suggest that ocular MG should be considered as differential diagnosis for any patients presenting with ophthalmoplegia without fluctuating weakness even if the results of laboratory, electrophysiological tests are unremarkable.

P-1-229

Hoffmann's Syndrome: a rare form of hypothyroid myopathy

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Background & Significance: Hoffmann's syndrome (HS) is a rare metabolic myopathy associated with hypothyroidism, which has characteristic findings of myopathy such as proximal weakness and pseudohypertrophy of muscles. We report a patient with myopathy, whose initial symptoms were fatigue and progressive muscle weakness with cramps, which was dramatically improved after thyroid hormone administered. **Case:** A 34-year-old man was admitted to our institution with complaints of progressive muscle weakness, cramps, and myalgia in his legs. He also had dry skin, loss of hair, puffy face, peri-orbital swelling, and calf muscle hypertrophy. On his initial neurological examination, the proximal and distal muscle strength of upper and lower extremities was checked of Medical Research Council (MRC) Grade 5. There was no abnormal finding in all sensory modalities. His deep tendon reflex (DTR) was normoactive, and there were no pathologic reflexes. Laboratorial investigation showed increased serum creatinine phosphokinase (5,493 IU/mL), primary hypothyroidism with raised titers of anti-thyroglobulin and anti-microsomal antibodies. Electrodiagnostic findings were compatible to diagnose the myopathy. On his electromyography, a couple of characteristic findings: (1) there was increased insertional activity along with positive sharp wave, and (2) polyphasic motor unit potential (MUP) with short duration. There was no specific finding on his nerve conduction study (NCS). Thyroid ultrasonography showed severely atrophied thyroid glands without any residual normal parenchyme, which suggesting atrophic thyroiditis. Vastus medialis histologic examination revealed non-specific myopathic findings without inflammation. He was diagnosed hypothyroid myopathy, probably result from Hashimoto's thyroiditis. Oral levothyroxine treatment was started and the dose was elevated gradually according to the response. After a week of thyroid hormone therapy, he showed improvement of his symptoms and decreased muscle enzyme levels. **Conclusions or Comments:** HS should be considered as one of the differential diagnoses in patients with myopathy who have hypothyroidism. The most characteristic finding is a dramatic response to thyroid hormone supply.

P-1-230

A case of lumbosacral plexopathy and avascular necrosis in carbon

monoxide(CO) poisoning

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Background & Significance: Carbon monoxide(CO) intoxication causes a variety of neuropsychological problems. But peripheral neuropathy and brachial or lumbosacral plexopathy are uncommon complication and rarely reported. Possible mechanisms cause peripheral neuropathy and plexopathy are nerve compression secondary to rhabdomyolysis or adjacent swelling tissue, direct CO toxicity and hypoxic injury due to methemoglobinemia. Avascular necrosis(AVN) is a aseptic necrosis of femur head due to anoxia and well known causes of AVN are chemotherapy, alcoholism, excessive steroid use, vasculitis, arterial embolism, radiation, bisphosphonates use and sickle cell anemia etc. **Case:** A 32-year-old man noted complaint of decreased right thigh muscle volume, decreased sensation of right leg and right hip joint pain during walking. Seven months ago, he was found unconsciousness in his room with charcoal firing then he was treated hyperbarbic therapy due to CO intoxication. In neurologic examination, Mild right thigh atrophy, slightly decreased motor power in right hip flexion, knee extension, knee flexion and decreased medial side calf area hypoesthesia were observed. In nerve conduction study and electromyography, there was an electrophysiologic evidence of right femoral and saphenous neuropathy. In right thigh muscle MRI, there was atrophy in vastus medialis, vastus intermedius, adductor magnus, hamstring muscles and Sartorius and gracilis muscle showed fatty degeneration. Also, there was bilateral avascular necrosis(right side more severe, Right: ARCO stage 3, left: ARCO stage 2) in Hip MRI. Other laboratory and CSF study were not remarkable. As we consider the results of neurologic examination, electrophysiologic test and MRI finding, final diagnosis of our patient was right lumbosacral plexopathy and combined bilateral avascular necrosis. **Conclusions or Comments:** CO intoxication induced peripheral neuropathy or plexopathy are very rare and to our knowledge, combined avascular necrosis with lumbosacral plexopathy after CO poisoning has not been reported ever. There are many hypothesis of pathophysiology that nervous system injury induced by CO poisoning. Considering the our case, CO induced tissue hypoxemia may play a significant role in PNS injury after CO intoxication.

P-1-231

A case of late-onset mitochondrial myopathy

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Background & Significance: Mitochondrial myopathy refer to a large heterogeneous group of disorders resulting from primary dysfunction of the mitochondrial respiratory chain and causing muscle disease. Mitochondrial myopathy presents with muscle weakness, muscle wasting, and exercise intolerance, which usually develop in the childhood or young age. **Case:** Here we report unusual case of late-onset mitochondrial myopathy. An 83-year-old man presented with a 2 year history of quadriparesis. Initially, he experienced mild weakness in the both legs that became progressively worse for 2 years. Neurological examination revealed proximal muscle weakness and wasting. Gower's sign was positive. Ocular muscle weakness, sensory symptoms or exercise intolerance was absent. Electromyography showed myopathic changes. A muscle biopsy confirmed the diagnosis of mitochondrial myopathy. **Conclusions or Comments:** Notably, this case illustrates that mitochondrial myopathy would be considered in the differential diagnosis of myopathy in the elderly

P-1-232**Subacute sensory-dominant thoracic polyradiculopathy associated with anti-GM1 IgM antibody**

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Background & Significance: Nonstructural polyradiculopathies result from various causes, including vasculitis, infectious, neoplastic, metabolic, and immune mediated inflammatory such as acute inflammatory demyelinating polyneuropathy or chronic inflammatory demyelinating polyneuropathy. However, there has not been a report about the association of polyradiculopathy with anti-GM1 IgM antibody, which has been known to be related to multifocal motor neuropathy. **Case:** A 59 year-old male with hypertension presented with weakness on both hands and numbness with tingling sensation on both hands and trunk aggravating for one month. Neurologic examination showed weakness in both distal upper extremities of grade IV and decreased tactile and vibration senses in both hands and from T4 to L1 dermatomes. Deep tendon reflexes were normal in both arms and legs. He had no urinary difficulty. Spine MRI showed no abnormality, except for mild disc bulging at cervical and lumbar spines. Nerve conduction studies (NCS) were normal on both upper and lower extremities except prolonged F latencies on right median, both peroneal, and posterior tibial nerves. Electromyogram studies were also normal in bilateral arm and thoracic paraspinal muscles. Sensory evoked potentials (SEPs) on median and ulnar nerves showed delay of signal conduction between the brachial plexus and the lower medulla, bilaterally. SEPs on posterior tibial and peroneal nerves were normal. Routine laboratory investigations including complete blood count, erythrocyte sedimentation rate, C-reactive protein, blood sugar, hemoglobin A1c, electrolytes, creatinine, Vitamin B12, total serum protein, creatine kinase, liver enzymes, albumin, thyroid-stimulating hormone, free T4, human immunodeficiency virus antigen/antibody, and rapid plasma reagin. Serological tests were also negative for rheumatoid factor, anti-nuclear antibody (Ab), anti-dsDNA, anti-Sjögren's syndrome A/B, anti-neutrophil cytoplasmic Ab, anti-phospholipid Ab, anti-cardiolipin Ab, anti-Hu, anti-Yo, anti-Ri. Antiganglioside Abs including anti-GD1b IgG/IgM and anti-GM1 Ab IgG were negative, however, anti-GM1 Ab IgM was positive. Cerebrospinal fluid examination showed a mild pleocytosis (10/mm³) and slightly increased protein level (79.4 mg/dL). Cytology for malignancy was negative. Treatment with intravenous methylprednisolone (1 g/day) was performed for eight days, followed by oral prednisolone, which resulted in a marked improvement of his symptoms. Follow-up NCS performed six weeks after the onset of symptoms was normal, however, the electromyography revealed active denervation potentials in lower thoracic paraspinal muscles. **Conclusions or Comments:** We report a case of subacute sensory-dominant polyradiculopathy probably related with immune-mediated inflammation, considering positive anti-GM1 IgM antibody and responsiveness to intravenous steroid therapy.

P-1-233**Guillain-Barré syndrome is not always presented with ascending and symmetric quadriparesis: A finger drop pattern of acute motor axonal neuropathy as a prominent variant form**Byeol-A YOON¹, Dong-Ho HA², Hwan-Tae PARK³, Susumu KUSUNOKI⁴, Jong-Kuk KIM¹¹Department of Neurology, Dong-A University College of Medicine, ²Department of Radiology, Dong-A University College of Medicine, ³Department of Physiology, Dong-A University College of Medicine, ⁴Department of Neurology, Kinki University School of Medicine

Background & Objectives: Traditionally, Guillain-Barré syndrome (GBS) is regarded as ascending and symmetric paralysis. It's typical presentation had been important diagnostic criteria for several decades. But, there was reported several type of variant GBS especially in asian countries including Korea. In such cases, methods to prove them as a GBS are mandatory because electrophysiological studies had limited values and urgent treatment is important for prognosis. Anti-ganglioside antibody study can serve us the significant clues in those situations. In recent years, we found interesting cases of GBS showing prominent finger drop sign in acute motor axonal neuropathy (AMAN). We suggest them as another important variant with immunological and image evidences. **Method:** From January 2012 to June 2015, eight AMAN patients with prominent finger drop sign were found from the GBS database from Dong-A University Neuroimmunology Team (DAUNIT). visited to Dong-A university hospital complaining progressive limbs weakness, especially characteristic pattern of predominant finger extensor weakness. Repeated electrophysiological studies and limb magnetic resonance image studies were performed. Anti-ganglioside antibody study including complex antibody were evaluated from acute stage serum. **Results:** Three patients were men and five were women. Seven patients out of 8 had preceding history of watery diarrhea. All patients had characteristic finger drop pattern of weakness especially in distal upper limbs with relatively sparing finger flexor and lower extremities. Powers of muscles innervated by posterior interosseous nerve were prominently impaired compared to muscles innervated by anterior interosseous nerve (1.9±1.1 vs 3.5±0.9 in medical research council grade). And also, all patients showed asymmetrical motor weakness. Cerebrospinal fluid analysis revealed albuminocytological dissociation. Anti-ganglioside antibody studies for IgG or IgM against ganglioside GM1, GM2, GD1a, GD1b, GD3, GT1a, GT1b and GQ1b were performed. They showed typical pattern of AMAN immunology. Electrophysiological study showed denervation in posterior group muscles compared with anterior group. Forearm MRI also showed prominent denervation signals exclusively in muscles innervated by posterior interosseous nerve. **Conclusion:** Our cases with finger drop AMAN showed the characteristic patterns. Those involve exclusively upper limbs especially posterior interosseous group. Forearm MRI and anti-ganglioside antibody study suggest us that antigenic distribution is not evenly distributed in some cases of AMAN type GBS. There is worth to think of the new diagnostic criteria in those patients.

P-1-234**Is decrement response at limb muscles on repetitive nerve stimulation the risk factor for generalization of ocular myasthenia gravis?**

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Background & Objectives: Myasthenia gravis (MG) is a neuromuscular junction disorder characterized by fatigable muscle weakness. Especially, extraocular muscle weakness which causes ptosis, diplopia, or both is the most common symptom of myasthenia gravis. About half of all myasthenia gravis patients initially present with symptoms restricted to the extraocular musculature (Ocular MG, OMG). Also, it has been already reported that approximately up to 50% of ocular MG patients subsequently progress to generalized form (Generalized MG, GMG). Currently, old age, high titer of acetylcholine receptor binding antibody (AChR-Ab) and presence of thymoma are known as the prognostic factors of generalization on ocular MG. Until now, however, the results of repetitive nerve stimulation test (RNST) on limb muscles could not predict the generalization of ocular MG. In this study, we test the hypothesis that decremental response at limb muscles on low frequency RNST is the risk factor of generalization of OMG to GMG. **Method:** We retrospectively reviewed the medical records of myasthenia gravis patients from Yonsei

Severance MG registry during the period of July 2009 to December 2013. The inclusion criteria were 1) ocular manifestation at the time of first RNST and 2) 1 year or more of the follow up period. The patients who had MG symptoms and sign other than ocular manifestation were excluded. The diagnosis of MG was based on clinical features, the AChR-Ab level, the RNST, and stigmine test. Total 51 patients were identified. Ten (19.6%) of the total 51 patients were progressed to GMG. The baseline characteristics were compared between the remained OMG (ROMG) and GMG groups. Predictive value of abnormal RNST at limb muscles for generalization was assessed by multivariable logistic regression analysis. **Results:** Generalization occurred in 10 (19.6%) of the 51 study subjects. The other 41 patients remained as OMG. The ROMG and GMG group did not differ by the age ($p=0.074$), presence of thymoma ($p=0.527$), and initial QMG score ($p=0.737$). Among the total 51 patients, 49% ($n=25$) had an abnormal RNST. Of the 10 GMG patients, 9 (90%) were abnormal in comparison to 16 (of 41; 39%) for ROMG patients ($p=0.005$). Limited to limb muscles (Abductor digiti quinti, Trapezius and Flexor carpi ulnaris) on RNST, 8 (80%) of GMG group had decremental response, but only 2 (of 41, 4.9%) had decremental response for ROMG group ($p<0.001$). Decremental response on RNST at any muscles or at limb muscles were more frequently observed ($p=0.005$, $p<0.001$) in the GMG group than remained OMG group. According to multivariate analysis, decrement response at limb muscles on RNST (95% confidence interval, 0.001-0.111, $p=0.006$) was associated with generalization of ocular MG. **Conclusion:** These results suggest decremental response of RNST at limb muscles was associated with generalization of ocular MG.

P-1-235

Steroid sparing effect of Tacrolimus in Myasthenia Gravis

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Background & Objectives: Tacrolimus is a macrolide T cell immunomodulator that is used in myasthenia gravis (MG) patients to affect muscle contraction, glucocorticoid receptors, and an increase in T cell apoptosis. The traditional MG treatments are cholinesterase inhibitor, prednisolone, and thymectomy. Among these, prednisolone or prednisone is a major treatment option for myasthenia gravis, and has rapid effect. However, complications of steroid are disturbing problem and sometimes it is impossible to increase the dose of steroid or it is needed to add on other immunosuppressants. Tacrolimus is considered as effective in clinical observations but there are no sufficient evidences so far. In this study, we aimed to figure out the safety and effectiveness of tacrolimus. **Method:** Medical records of MG patients who were treated with tacrolimus were reviewed retrospectively. We recorded the patient's age of tacrolimus initiation, gender, MG classification, the duration of tacrolimus treatment, the side effects of tacrolimus, the reason for start and discontinuation of tacrolimus, and the dose of corticosteroids. For assessment of steroid sparing effect of tacrolimus, we selected the patients who were treated with corticosteroids for 24 weeks or more before tacrolimus treatment and were treated with tacrolimus for 48 weeks or more. The dose of corticosteroids of each patient was investigated at 24 and 12 week before tacrolimus start day, tacrolimus start day, and 12, 24, 36, and 48 week after tacrolimus start day. **Results:** Total 98 MG patients (Female 77, Male 21) treated with tacrolimus were identified. Among them, 87 patients were classified as generalized MG and 11 patients were classified as ocular MG. The mean age of tacrolimus initiation was 41.8 years (standard deviation (SD) 13.8). Among total 98 patients, tacrolimus was added on corticosteroid in 38 patients. In the other 60 patients, tacrolimus was switched with other immunosuppressants (e.g. azathioprine, cyclosporine, and mycophenolate mofetil). The 60 patients had been treated with corticosteroids and other immunosuppressants and then switched to cor-

ticosteroid and tacrolimus. Among the 60 patients, 25 patients were switched to tacrolimus due to the side effects of the other immunosuppressants and the other 35 patients were switched to tacrolimus due to the lack of efficacy of the other immunosuppressants. Twenty patients discontinued tacrolimus. Among the 20 patients, lack of efficacy is the reason in four patients and the reason of discontinuation was uncertain in one patients. The other 15 patients stopped the tacrolimus because of the side effects, i.e. diarrhea ($n=8$), hair loss ($n=3$), abdominal pain ($n=1$), renal dysfunction ($n=1$), paresthesia ($n=1$), and oral ulcer ($n=1$). The mean duration of tacrolimus treatment was 76.8 weeks (SD 80.0). Among total 98 patients, the 46 patients were treated with prednisolone for 24 weeks or more before tacrolimus treatment and were treated with prednisolone and tacrolimus for 48 weeks or more. The mean dose of prednisolone showed increasing tendency just before tacrolimus, and steadily decreasing tendency after tacrolimus initiation. The mean dose of prednisolone was 15.7 mg (SD 9.1), 16.6 mg (SD 10.3), 21.0 mg (SD 13.3), 17.3 mg (SD 9.2), 14.9 mg (SD 6.9), 13.5 mg (SD 5.6), 12.6 mg (SD 5.6) at 24 and 12 week before tacrolimus start day, tacrolimus start day, and 12, 24, 36, and 48 week after tacrolimus start day, respectively. Prednisolone dose at 48 weeks using tacrolimus was significantly lower than at 24 weeks before tacrolimus (12.6 ± 5.6 vs. 15.7 ± 9.1 ; $p=0.03$). Prednisolone dose at 48 weeks using tacrolimus was significantly lower than at 12 weeks before tacrolimus (12.6 ± 5.6 vs. 16.6 ± 10.3 ; $p=0.02$). **Conclusion:** This study showed good long-term safety and tolerability of tacrolimus in patients with myasthenia. In addition, steroid sparing effect of tacrolimus also demonstrated. Tacrolimus may be considered as second-line treatment in patients with myasthenia gravis

P-1-236

Diabetes mellitus exacerbates the clinical and electrophysiological features of Guillain-Barré syndrome

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Background & Objectives: It is known that underlying diabetes mellitus (DM) can affect the clinical and electrophysiological pattern of coexisting peripheral neuropathies of other etiologies. The aim of this study were to identify the effect of underlying DM on the clinical and electrophysiological features of Guillain-Barré syndrome (GBS), and on the prognosis of GBS with regard to functional outcome. **Method:** This study prospectively included 27 GBS patients with DM (GBS-DM+) and 58 GBS patients without DM (GBS-DM-) from 2 university-based hospitals. The clinical and electrophysiological findings were compared between the two groups. The functional outcomes was quantified by measuring the Hughes grade, whose values were compared between the groups at 3 months after symptom onset. **Results:** All three sudden deaths that occurred during the acute stage of GBS were GBS-DM+ patients. GBS-DM+ patients had a tendency toward more frequent sensory involvement, and specific electrophysiological patterns and calculated indexes disclosed a distal accentuation of conduction abnormalities in these patients. In addition, multivariate analysis identified history of mechanical ventilation [odds ratio (OR)=10.057, 95% confidence interval (95% CI)=2.057-49.164, $P=0.04$] and DM (OR=9.049, 95% CI=2.152-38.044, $P=0.003$) as independent factors for poor functional outcome at 3 months. **Conclusion:** The findings of this study suggest that DM exacerbates the clinical and electrophysiological features of GBS and influences long-term disability. Both chronic inflammation and nerve ischemia in DM may intervene in the disease course of GBS, which is a prototype of acute inflammatory neuropathy.

P-1-237**Accelerated neuropathy of renal failure**

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Case: ESRD is rapidly growing health problem. In ESRD patients, many alterations have been described about hematologic, cardiovascular, and even central, peripheral nervous system. Effect of uremia in peripheral nervous system is well known as 'Uremic neuropathy', which clinical course is usually progress over several years and axonal feature in electrophysiological testing. A rare form of uremic neuropathy was described since 1960'. progressed more rapidly (over several weeks to months), and showed demyelinating feature on electrophysiological testing as well as axonal feature. This accelerated form of uremic neuropathy has been rarely reported in Korea. In this case, we described two ESRD patients who developed a progressive and symmetrical lower extremity weakness over several months, estimated to be caused by accelerated neuropathy of ESRD. Treatment guidelines have not been established, whereas renal transplant is generally been accepted as an only effective treatment. Further studies are needed to elucidate the pathogenesis of this disease.

P-1-238**Meralgia paresthetica caused by obturator hernia**

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Background & Significance: Meralgia paresthetica is a painful mononeuropathy of the lateral femoral cutaneous nerve, which is susceptible to compression beneath the inguinal ligament. It has known various etiologies including retroperitoneal mass, diabetes, post-operative complication after hip replacement or spine surgery. Obturator hernia represents low incidental rate among bowel herniation. In general, obturator hernia is associated with obturator neuropathy, because it leads to protrusion of sac through obturator foramen and canal along the obturator nerve. Meralgia paresthetica associated with obturator hernia have not been reported so far. Here we report a 1 case of meralgia paresthetica caused by pre-operative obturator hernia. **Case:** A 78 year-old female presented with right anterolateral thigh paresthesia with right inguinal pain. There is no history of diabetes, malignancy, operation and her occupation was homemaker. However, she was diagnosed incidentally right obturator hernia 10 years ago. She had not surgery before admission because she had no symptom associated with obturator hernia and strangulation had not been occurred. In neurological examination, her lower extremities motor power was normal and other neurological deficits was not found excluding right anterolateral femoral paresthesia. Because her clinical symptom was suspicious for right meralgia paresthetica, nerve conduction study was performed. Nerve conduction study revealed slow sensory nerve conduction velocity of right lateral femoral cutaneous nerve. She was diagnosed meralgia paresthetica, but we considered intermittent inguinal pain was associated with other medical conditions. So abdomen CT was performed and it showed right herniated bowel loop between obturator externus and pectineus muscles. Herniated bowel loop might be considered to compress lateral femoral cutaneous nerve. She was referred to the GS department and underwent herniorrhaphy. 3 months later after she had surgery, right femoral paresthesia and inguinal pain were disappeared. **Conclusions or Comments:** Meralgia paresthetica associated with hernia is usually post-operative complication of hernia and obturator hernia is well known for cause of obturator neuropathy. This report is the first case of meralgia paresthetica caused by obturator hernia itself. It was proved by relieved symptoms after the decompressive surgery.

Therefore if clinically suspicious for meralgia paresthetica, obturator hernia should be considered and then nerve conduction study including abdomen CT are important to decide treatment plan. Lateral femoral cutaneous nerve can be lead to significant disability when the diagnosis is missed or delayed. Therefore earlier surgical intervention would be a great help to prevent permanent neurological deterioration in compressive lateral femoral cutaneous neuropathy.

P-1-239**Ataxic form of Guillain-Barre syndrome associated with anti GD1b antibody-a case report of subacute stage**

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Background & Significance: Guillain-Barre syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy and the targets of immune reaction are gangliosides located in the plasma membrane of nerve tissue. Clinical features of GBS may vary depending on the different antiganglioside antibodies. Anti-GD1b antibody is reported to be associated with ataxic form of GBS, which shows cerebellar ataxia without profound weakness or ophthalmoplegia, or acute sensory ataxic neuropathy (ASAN). Nerve conduction finding in anti-GD1b antibody related GBS is mostly axonal type. We report a patient who was diagnosed to ataxic form of GBS associated with GD1b antibody showing atypical electrophysiological finding in subacute stage of disease. **Case:** A 15-year-old girl visited our clinic for gait difficulty and frequent falling during past 2 months. Initially, she had severe myalgia without remarkable infection signs including respiratory and gastrointestinal symptoms. Several days later, she noted bilateral leg weakness and stumbling gait. She also suffered from easy fatigability during that period. She had no past medical history and there was no family history of similar neurological disease. Neurological examinations showed no abnormal sign of cranial nerves including extraocular movement. Limb weakness was insignificant (MRC grade 5) and there was no sensory impairment including positional and vibratory sensation. Pathologic reflex was absent. However, finger to nose and heel to shin tests disclosed mild ataxia and tandem gait was slightly unstable. Deep tendon reflexes of elbow, wrist, knee and ankle were decreased (1+) bilaterally. Brain and cervical spine magnetic resonance imaging showed no abnormality. Laboratory findings including cerebrospinal fluid (CSF) examination were normal, except for slightly increased protein level (82mg/dl) in CSF. Nerve conduction studies demonstrated decrease of conduction velocities, conduction block on bilateral upper and lower motor nerves and absence of F-waves. She had borderline value in the anti-GD1b IgG antibody (34.9%) but negative to the others including anti-GD1b IgM antibody. Diagnosis of ataxic GBS had been made and she markedly recovered with supportive care. Follow up evaluation revealed positive anti-GD 1b IgG (67.3%) and anti-GM1 IgG (54.5%) antibodies. **Conclusions or Comments:** Because this patient had presented with a rare form of GBS in subacute stage (two months after the disease onset), initial neurological examination was not remarkable. Based on the symmetric hyporeflexia, minimal ataxia and detailed clinical history, electrophysiological studies were performed and it disclosed the demyelinating sensorimotor polyneuropathy. In this patient, antiganglioside antibody test was helpful to identify the clinical subtype of GBS, however, electrophysiological finding was not common in this ataxic form of GBS.

P-1-240**A case of paraneoplastic myopathy related to very rapidly progressive malignant lymphoma**

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Background & Significance: The association between inflammatory myopathy, especially dermatomyositis and cancer has been reported through some case reports and population-based studies. The pathogenesis is not fully understood, but it may occur through cross-reaction of autoantibody against tumor cells with normal muscle tissues. Though there's some reports of polymyositis with malignant tumors, cases related to malignant B cell lymphoma were rare. We present here a case of paraneoplastic polymyositis associated with very rapidly progressive B-cell lymphoma. **Case:** A 66-year-old man was referred to our department by internal medicine doctor with elevated muscle enzyme (CK:2125IU/L) at August 2014. He had a history of advanced gastric cancer (stage IIIA, tubular adenocarcinoma) diagnosed at Feb 2013, and underwent total gastrectomy followed by adjuvant chemotherapy. For cancer, he maintained NED state from May 2013. At first visit to our clinic, he showed very mild myalgia at both proximal thigh since about 2 weeks ago, but had no weakness and no neurological abnormalities. On NCS/EMG test, there was no definite electrophysiologic evidence for myopathy. Blood tests including autoantibodies, RF, tumor markers, TFTs and serologic markers were all normal. We considered muscle MRI and muscle biopsy for more confirmative diagnosis, but patient refused. So we decided to follow up with close observation. At April 2015, he revisited to us with progressive proximal weakness at 4 extremities started from one month ago. On examinations, he showed symmetric, proximal-dominant motor weakness (MRC Gr 4-), but otherwise were normal. There's no skin lesions such as heliotrope rash or Gottron's papules etc. Blood tests showed more elevated CK (18466IU/L), LDH (1504IU/L), AST/ALT (652/544IU/L), and FANA(+). EMG showed myopathic changes and denervation potentials in all muscle examined. His muscle MRI showed diffuse muscle edema, more severe at both upper arms and thighs, suggestive of inflammatory myositis. However muscle biopsy showed just mild myofibers size variation, endomyseal fibrosis and some degenerating/regenerating myofibers. There's no inflammatory cell infiltration. Though the absence of inflammatory cells at biopsy, we considered polymyositis as the most possible diagnosis based on clinical and laboratory findings. So we started a high-dose (1g/d) IV methylPd therapy for 5 days. We didn't consider paraneoplastic myopathy at that time regarding to his past cancer history since the abdomen/pelvis CT checked at just 1 month ago was normal. During pulse therapy and following oral prednisolone maintenance (60mg/d), his weakness was improving slowly, and CK level decreased to 2931IU/L. He was discharged, and we planned to maintain steroids for at least several months. After 1 month, he was rushed to ER room with severe abdominal pain, and abdomen/pelvis CT at ER showed diffuse peritoneal thickening with some mass formation, suggestive of peritoneal seeding. Whole body PET showed extensive hypermetabolic peritoneal mass with pleural seeding and mediastinal multiple LNs metastasis. Tissue biopsy was done and the pathologic diagnosis was B cell lymphoma with features intermediate between DLBCL and Burkitt lymphoma (Ann arbor stage IV). He was started on the R-CHOP regimen. After first chemotherapy, CK level was more decreased to 1567IU/L, but his motor power did not improve, maybe due to poor medical conditions. **Conclusions or Comments:** In this case, malignant lymphoma was diagnosed just 2 months after from symptom onset, with preceding period of only elevated muscle enzyme for about 8 months. Because the abdomen/pelvis CT imaging at near symptom onset was normal, malignant tumor might be progressed very rapidly for 2 months. Or maybe there's latent tumor cells at uncovered areas. Clinicians should keep in mind the possibilities of latent cancer or rapid development of malignant tumor (lymphoma) at patients with polymyositis, albeit the absence of inflammatory cells at biopsy.

P-1-241**Acute motor axonal neuropathy in association with Graves' disease**

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Background & Significance: Graves' disease (GD) is an inflammatory autoimmune thyroid disease. Thyroid-stimulating hormone (THS) receptor is the major autoantigen in GD. GD is caused by autoimmune reaction to the receptor for THS receptor antibodies. GD has been known to manifest with neurological complications, rare of the peripheral nervous system. Guillian-Barré syndrome is autoimmune peripheral neuropathy and occurs when immunologic tolerance for peripheral nerve components is lost. Acute motor axonal neuropathy (AMAN) is an axonal variant of GBS that presents with acute ascending quadriparesis. This is a primary axonal polyneuropathy frequently associated with Campylobacter jejuni enteritis and antidodies against ganglioside. The association between AMAN and GD has not been previously described. We report a case with clinical and electrophysiological evidence of AMAN in association with GD. **Case:** A 46-year-old man presented to the emergency room with the acute onset of ascending paralysis that had progressed over 3 days. Three days ago, he awoke with weakness in his ankles and toes resulting in slight difficulty with ambulation and then his weakness progressed to proximal lower extremities. Two days ago, his weakness also developed hands. He had no significant past medical history. Ten days prior to the onset of symptoms, he had experienced a severe upper respiratory infection (URI). On neurological examination, motor examination showed Medical Research Council (MRC) 4+ strength in upper extremities muscles, symmetrical MRC 4 strength in lower proximal muscles, and symmetrical MRC 4+ strength in lower distal muscles. His tendon reflexes were reduced in upper and lower extremities. He had normal cranial nerve examination and no sensory loss to all modalities. On admission, he had symptoms of thyrotoxicosis in the form of palpitation, anxiety along with proptosis for last 4 months. Physical examination revealed resting tachycardia. Laboratory tests showed a low concentration of thyroid stimulating hormone (0.06 mU/L, normal value 0.17-4.05 mU/L) and raised values of T3 (433.69 ng/dL; 78-182 ng/dL) and free T4 (50.6 pmol/L; 11.5-23.0 pmol/L). TSH-receptor antibodies (5.95 U/L; 0-1.5 U/L) was raised. Graves' disease with ophthalmopathy was diagnosed, and the patient received carbimazole (20 mg daily), propranolol (80 mg daily), and prednisolone (starting dose of 100 mg daily). However, his weakness deteriorated. On hospital day 3, motor examination showed MRC 4 strength in his upper extremities muscles, symmetrical MRC 2 strength in his lower proximal muscles, and symmetrical MRC 3+ strength in his lower distal muscles. On hospital day 4, he underwent nerve conduction studies, which showed reduced compound muscle action potentials in right median, bilateral ulnar and tibial motor nerves and normal sensory nerve action potentials in the upper and lower extremities. The cerebrospinal fluid analysis showed protein at 32 mg/dl, glucose at 53 mg/dl (serum glucose 108 mg/dl), and no white blood cells. Clinical and electrophysiological findings of our patient were compatible with AMAN. Further analysis showed normal bacterial and viral cultures and serology. Serum viral serologies, including HIV, Hepatitis A and B, Epstein-Barr virus, Lyme titers, and stool cultures for Campylobacter jejuni and other enteric pathogens were negative. VDRL tests were negative in both CSF and serum. Anti-GM1 and anti-GD1b antibodies were negative. Due to the fulminant course of his paralytic illness, a comprehensive vasculitis work-up was initiated, however, there was no significant findings. Brain and cervical spine MRI were normal. On hospital 7 days, he received a 5-day course of intravenous immunoglobulin (IVIg; 2 g/kg) over 5 days. He showed a rapid and dramatic clinical response. **Conclusions or Comments:** we report an unusual variant of AMAN in association with deterioration of GD. Studies are

still needed to ascertain the pathophysiologic relationships between GD and autoimmune polyneuropathies.

P-1-242

Respiratory failure as an initial symptom of amyotrophic lateral sclerosis

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Background & Significance: Although respiratory complications are common causes of morbidity and mortality in patients with advanced amyotrophic lateral sclerosis (ALS), it is very rare that ALS presents with respiratory failure as the initial manifestation. However, we have identified three cases of ALS presenting with respiratory impairment. **Case:** All three patients visited hospital due to dyspnea and eventually required mechanical ventilation. Electrophysiological study revealed wide spread denervation and confirmed the diagnosis of ALS. Two patients complained some degree of muscle weakness before respiratory failure but one patient didn't recognize muscle weakness or atrophy. **Conclusions or Comments:** These cases emphasize that ALS would be considered in the differential diagnosis of unexplained respiratory failure.

P-1-243

A case of Guillain-Barre Syndrome and myositis associated with preceding hepatitis E virus infection

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Background & Significance: Hepatitis E virus (HEV) infection is an emerging cause of acute hepatitis around the world. Neurological manifestations have been previously reported to be associated with acute HEV infection. In Korea, several cases have been reported about HEV hepatitis, but there is no report about HEV infection with neurological manifestations. We experienced HEV infection induced Guillain Barre syndrome (GBS) and myositis. This case is the first report about acute HEV infection with neurological manifestation in Korea. **Case:** A 66-year-old man previously healthy except for hypertension was admitted to the local hospital with acute onset both leg weakness and myalgia. A laboratory test performed at local hospital showed elevated muscle enzyme (creatinine kinase (CK) > 4000 UI/L). Doctors at the hospital considered rhabdomyolysis and treat with conservative management including massive hydration. 8 days after, the patient complained of dyspnea and he was admitted to department of Internal Medicine of our hospital. On hospital day 7, altered mental status and CO₂ retention was observed in arterial blood gas analysis. He transferred intensive care unit and his mental status was improving during ventilator care, but ventilator weaning was fail without lung problem. So, the patient was consulted to the neurologist. Neurological examination showed proximal weakness in both arm and leg and hypoactive deep tendon reflex of both biceps, knee and ankle. Nerve conduction studies showed axonal type sensorimotor polyneuropathy with abolition or prolongation of F-wave responses. Needle electromyography showed diffuse abnormal spontaneous activity in all four limbs with myopathic MUAPs. Laboratory test on admission showed an aspartate aminotransferase (AST) level of 234 U/l (normal range 14-36 U/l), alanine aminotransferase (ALT) of 536 U/l (normal range 9-52 U/l), CK of 5204 UI/L and myoglobin 3974 UI/L. Serum IgM and IgG against HEV were positive. Thigh MR showed high signal intensity and diffuse enhancement on bilateral medial thigh muscle. A biopsy specimen of the Right adductor magnus muscle showed marked myofiber necrosis with

lympho plasma cell infiltration in endomysium. Based on these findings the diagnosis of GBS and myositis related to acute HEV hepatitis was made. But anti ganglioside antibodies (GM1, GD1b, GQ1b) were negative. Intravenous immunoglobulins were given for 5 days. Ventilator weaning was done after 5 days, and he could walk without assist after 2 weeks and discharged. **Conclusions or Comments:** Several cases have been reported with HEV hepatitis associated with neurologic disorders, including GBS, brachial neuritis, and polyradiculopathy. GBS and brachial neuritis are most frequently reported neurologic manifestations in HEV infection. Although several GBS cases caused by HEV hepatitis are being reported, only one case of myositis has been reported so far, and it was manifested along with GBS. In this case, GBS combined myositis was induced by HEV hepatitis. GBS and myositis seem to be related to an auto-immune response. GBS associated with HEV hepatitis mostly showed a better prognosis, and the patient in this case also improved rapidly after IVIG treatment and myositis was self limiting course. We have to consider that HEV infection can be a cause for neurologic disorders, and a simple HEV serologic test is recommended for patients with neurologic manifestations accompanied by abnormal liver function.

P-1-244

Discrepancy between the electrodiagnostic study and ultrasonography in clinically suspected carpal tunnel syndrome

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Background & Objectives: Ultrasonography has emerged as a complementary tool to nerve conduction study (NCS) in diagnosing carpal tunnel syndrome (CTS). However, some patients showed discrepancy between the result of electrodiagnostic studies and ultrasonographic finding. The aim of this study was to investigate the characteristics of patients who met diagnostic criteria for CTS in ultrasound but showed normal result in NCS. **Method:** Ultrasound and NCS were performed on patients with clinically suspected CTS. NCS-proved CTS was confirmed by the criteria of the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM). Blinded measurements of cross section area (CSA) were made from transverse sonographic images of the median nerve at the wrist and mid-forearm. Patients were divided in two groups; group 1 included NCS-proved CTS patients, group 2 included patients who met diagnostic criteria for CTS in ultrasound but showed normal results in NCS. Subjects who had normal results in both NCS and ultrasonography were excluded. The demographic and laboratory data of all patients were assessed. **Results:** In total, 84 patients with NCS-proved CTS (group 1) and 15 patients with normal NCS and increased CSA in ultrasonography (group 2) were studied. Patients in group 2 were younger (56.7 ± 11.8 vs. 49.4 ± 13.1 , $p=0.036$) and had shorter duration of symptom (31.9 ± 64.9 vs. 12.1 ± 17 , $p=0.022$) than patients in group 1. Proportion of unilateral CTS was significantly higher in patients in group 2 (26.2 % vs. 61.5 %, $p=0.01$). Seventy three percentage of patients in group 2 had left CTS despite 93% of them were right-handed people. This discrepancy between Handedness and side of CTS was significantly higher in group 2 patients (32.1 % vs. 71.4 %, $p=0.005$). **Conclusion:** Clinically suspected CTS with normal NCS and abnormal ultrasound finding tend to have younger age and shorter duration of symptoms. Also, most of them showed unilateral CTS on opposite side of mainly-used hand. Further prospective studies about discrepancy between the NCS and ultrasound are required.

P-1-245

Clinical and pathological characteristics of genetically confirmed autosomal dominant Emery-Dreifuss muscular dystrophy patients

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Background & Objectives: Clinical triads of Emery-Dreifuss muscular dystrophy (EDMD) are joint contracture, slowly progressive muscle weakness and cardiomyopathy. Due to early presentation of non-specific muscle weakness, later involvement of other clinical features, and various heterogenetic subtypes of EDMD, clinical diagnosis and differentiation with other type of muscular dystrophy is often challenging in early-onset EDMD. In this study, 4 patients, who were previously diagnosed other type of muscular dystrophy in childhood, were confirmed to have reported mutations in LMNA gene. We evaluate clinical and pathological characteristics retrospectively to elucidate the clinical course of early-onset EDMD. **Method:** We performed targeted sequencing of 69 genes known to be responsible to myopathy, including LMNA genes. DNA was extracted from either blood sample or muscle sample. Targeted sequencing were done using illumine Hiseq2000 platform. Clinical manifestations, neurologic examination findings, electromyography, and cardiac evaluation result of those genetically confirmed EDMD patients were reviewed retrospectively using our hospital electrical medical record. Muscle samples from the patients were pathologically confirmed, and immunohistochemical staining were performed including Emerin and Lamin, if possible. **Results:** We identified 4 recurrent LMNA mutations, c.1357C>T;p.R453W, c.1366A>G;p.N456D, c.746G>A;p.R249Q and c.1583C>G;p.T528R from 4 sporadic EDMD patients. Clinical symptoms occur at early childhood between 2 to 5 years and most of chief complain was waddling and slow gait. Neurologic examination revealed proximal motor weakness and, notably, calf pseudohypertrophy was marked in 3/4 patients. Serum CK level was mildly elevated and muscle biopsy shows dystrophic myopathic changes. From the available initial electrocardiogram data, 3 patients showed normal sinus rhythm and two of them were converted to having conduction block in adult age. There was no family history in these patients suggesting sporadic genetic mutation. **Conclusion:** In early-onset EDMD patient, first clinical symptoms and neurological examination findings are not pathognomonic, so they are likely to be diagnosed more prevalent other muscular dystrophic diseases. By targeted sequencing of known 64 myopathy causative genes, 4 patients show mutations in LMNA exclusively. Retrospective follow up data reviews show different onset time of both contracture and cardiac disease. Our data suggest that clinical follow up and early genetic screening are important to those non-specific muscular dystrophy patients for predicting the disease course and preventing respiratory and cardiac complications.

P-1-246

Expiration to inspiration ratio as a prognostic factor in systemic AL amyloidosis

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Background & Objectives: Autonomic failure is frequently observed in patients with AL amyloidosis. The aim of this study was to elucidate whether autonomic dysfunction can predict poor prognosis in patients with AL amyloidosis. **Method:** We retrospectively analyzed 67 consecutive patients with AL amyloidosis who had performed autonomic function test in a prospectively collected

database. Autonomic parameters including heart rate variability, beat to beat heart rate response to deep-breathing and expiration : inspiration ratio (E:I ratio), beat-to-beat blood pressure and heart rate response to Valsalva maneuver and head-up tilt were examined in all enrolled patients. Clinical, laboratory and echocardiographic characteristics were also examined. Univariate and multivariate analyses were performed to evaluate the prognostic value of autonomic parameters on survival. **Results:** Among parameters of autonomic function test, decreased E:I ratio was a significantly associated with survival ($p = 0.017$). E:I ratio was decreased in 35 patients (52.2%). The overall survival rates at 1 year for patients with decreased E:I ratio were 42.9% versus 68.8% for patients with normal E:I ratio ($p = 0.033$). In multivariate analysis comparing E:I ratio and well-known prognostic parameters (difference between involved and uninvolved light chain, β 2-microglobulin, NT-proBNP, ECOG), decreased E:I ratio was significantly and independently associated with survival with a hazard ratio of 4.5 ($p = 0.018$; 95% confidence interval, 1.3-16.0). **Conclusion:** This study demonstrated that the decreased E:I ratio was significantly associated with increased mortality in patients with AL amyloidosis.

P-1-247

Mutations in the muscle adenylosuccinate synthetase-like 1 gene cause an autosomal recessive distal myopathy

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Background & Objectives: Distal myopathy is a clinically and genetically heterogeneous group of predominant distal muscle degenerative diseases. Distal myopathy is classified into subgroups based on clinical aspects and genetic causes. To date, more than 14 causative genes have been reported in different forms of distal myopathies. Recent genomic analysis has steadily updated novel genetic causes; however, considerable myopathy patients still wait for the genetic cause to be uncovered. In this study, we have done to identify the underlying cause of autosomal recessive adolescent-onset distal myopathy. **Method:** Four patients from two unrelated Korean families were evaluated. To isolate the genetic cause, exome sequencing was performed. In vitro and in vivo assays using myoblast cells and zebrafish models were performed to examine the ADSSL1 mutation causing myopathy pathogenesis. **Results:** Patients had an adolescent-onset distal myopathy phenotype which included distal dominant weakness, facial muscle weakness, rimmed vacuole, and mild elevation of serum creatine kinase. Exome sequencing identified completely cosegregating compound heterozygous mutations (p.D304N and p.I350fs) in ADSSL1 which encodes a muscle-specific adenylosuccinate synthase in both families. None of the controls had both mutations, and the mutation sites were located in well conserved regions. Both the D304N and I350fs mutations in ADSSL1 led to decreased enzymatic activity. The knock-down of the adssl1 gene significantly inhibited the proliferation of mouse myoblast cells, and the addition of human wild-type ADSSL1 reversed the reduced viability. In an adssl1 knock-downed zebrafish model, muscle fibers were severely disrupted, which was evaluated by myosin expression and birefringence. In these conditions, supplementing wild-type ADSSL1 protein reversed the muscle defect. **Conclusion:** We suggest that mutations in ADSSL1 are the novel genetic cause

of the autosomal recessive adolescent-onset distal myopathy, and this study broadens the genetic and clinical spectrum of distal myopathy. It appears that the loss-of-function mutations may develop distal myopathy by the defect of de novo purine nucleotide biosynthesis due to a deficiency in enzyme activity. This is the first report that ADSSL1 is involved with a human genetic disease, and will be useful in performing exact molecular diagnostics of distal myopathy.

P-1-248

A case of antiphospholipid syndrome with transverse myelitis mimicking Guillain-Barré Syndrome

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Background & Significance: Antiphospholipid syndrome (APS) is an autoimmune disease, which is associated with the antiphospholipid antibodies (aPL), including anticardiolipin, lupus anticoagulant and anti-beta2-glycoprotein I (β 2GPI). Rarely, APS manifests as myelopathy or peripheral neuropathy related to vasculitis of small and medium-sized vessels. We present a case of APS with transverse myelitis (TM) accompanied by inflammatory multiple mononeuropathies. **Case:** A 10-year-old girl presented with quadripareisis. Three days ago, she complained of fever, nausea and cough. With motor weakness, she also complained of voiding and defecation difficulties. There was no family history of autoimmune diseases. Neurologic examinations revealed motor weakness of both lower extremities (Medical Research Council (MRC) grade I/I) and both upper extremities (MRC grade II/II). She did not show sensory loss, dizziness nor diplopia, but upbeating nystagmus was observed. The deep tendon reflexes were hyporeflexive in both upper and lower limbs, and Babinski sign was not evoked. CBC, blood chemistry and creatine kinase, ESR and CRP were normal. Brain CT did not show any abnormality, and there were no infection or inflammatory evidence in CSF study (WBC 0/mm³, RBC 0/mm³, glucose 66mg/dL (serum 134 mg/dL), protein 31mg/dL). On electrophysiological test of the first day, F-waves were unobtainable in bilateral median, ulnar, radial and peroneal nerves, but motor and sensory NCS were normal on tested nerves of upper and lower limbs. IVIG treatment for five days began under suspicion of Guillain-Barré syndrome. After 1 week, NCS were compatible with motor axonal multiple neuropathies. CMAPs (compound motor action potential) of bilateral median and right ulnar motor nerves showed lower amplitudes than lower limit of normal. NCS of lower extremities were normal. But, results of F-waves were same as previous study, also H-reflexes were not evoked. The patient complained of dyspnea, so oxygen supply via nasal prong provided. Lab tests for anti-MAG antibody and anti-ganglioside antibodies including anti-GM1, GD1b and GQ1b were negative. For two months, motor weakness of upper extremities improved gradually, so MRC grade of left arm was V, but recovery of lower extremity was poor. And voiding difficulties got no better. Serial NCS showed gradual normalization of F-waves and increment of CMAP amplitudes. We planned further evaluation of upper motor system, because on her neurologic exam, deep tendon reflexes on all extremities became more active, and even hyperactive on left lower extremity. On sensory and motor evoked potential studies, sensory conduction pathway defect between cauda equina and cervical cord, and central motor conduction pathway defect were suspected. In spine MRI, T2-weighted imaging showed high signal intensity lesions at anterior and left lateral spinal cord from C5 to T2 level, suggestive of transverse myelitis. On evaluation of myelitis, anti-phospholipid IgG was positive (46.4 GPL/mL), and anti-cardiolipin IgM and IgG were weak positive. Lupus anticoagulant and anti- β 2GPI were negative. Anti-NMO (neuromyelitis optica) antibody was negative. Other tests for autoimmune diseases including ANA, anti-dsDNA, anti-MPO and anti PR3 antibody were also within normal range. She is treated with low-dose prednisolone and aspirin for 6 months. Her motor weakness

has been slightly improved, but she is still on state of assisted standing. **Conclusions or Comments:** We experienced a rare case of APS which showed concurrent transverse myelitis and peripheral neuropathies. Vasculitis related to aPL could cause myelitis, Guillain-Barré syndrome and also multiple mononeuropathies. It is rare, but for APS patients, closed observation, detailed neurological examination and proper laboratory study are needed for prompt diagnosis and management of complicated transverse myelitis.

P-1-249

NTRK1 mutations in Korean patients with congenital Insensitivity to pain with anhidrosis

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Background & Significance: Congenital Insensitivity to pain with anhidrosis (CIPA) is a extremely rare recessively-inherited neurological disease, which is known to be caused by the mutation in NTRK1 gene. **Case:** Eight Korean patients, who clinically diagnosed as having CIPA, were enrolled for this study. Mutational analyses revealed 2 known mutations (c.2020G>T [p.674Y], IVS7-33T>A) and 4 novel mutations (c.704C>G [p.Ser235Stop], c.1786G>A [D596N], IVS14+3A>T, c2350_2363del [p.Leu784fsX79]) in NTRK1 gene. Clinical manifestations, which were similar to the previous reports, showed a history of multiple repeated fracture, complete or partial anhidrosis with heat intolerance, repeated fever, self-mutilating behavior, and developmental delay. Mental retardation were observed in 6 (75%). Nerve conduction study and somatosensory evoked potentials were unremarkable. Quantitative sudomotor axon reflex test showed absent or significantly reduced sweating in the foot and forearm. **Conclusions or Comments:** Here, we reported 4 novel mutations in NTRK1 gene with the clinical features of the patients with CIPA.

P-1-250

Respiratory failure due to restrictive chest wall deformity in muscular dystrophy

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Background & Significance: Patients with muscular dystrophy in an advanced stage often develop respiratory failure, most likely related to respiratory muscle or diaphragmatic weakness. Facioscapulohumeral muscular dystrophy (FSHD) is a slowly progressive dystrophy involving primarily the facial and shoulder musculature. Respiratory muscles are usually known to be spared. One of the common characteristics is pectus excavatum, which is an inward growth of the sternum. Herein, we report a case of FSHD with respiratory failure resulting from pectus excavatum. **Case:** A 57-year-old female with muscular dystrophy was presented with recently aggravated dyspnea for 1 week with cough and sputum. She had progressive muscle weakness which started from the face since early 30's. Proximal limb muscles, earlier in the arms and later in the legs, were involved and worsened to the point that she could not walk. However, she still was able to use her hands even for sewing. In the past, she was diagnosed with muscular dystrophy without genetic typing. Her two brothers and her daughter were diagnosed with muscular dystrophy as well. Her 29-year-old daughter underwent genetic testing at the age of 27 and was informed that she has FSHD although she does not have any clinical symptoms. On admission, neurologic examination revealed severe muscle weakness and atrophy of all limbs and facial diplegia but with relatively intact

finger and hand movements. She has significant pectus excavatum. ABGA showed hypercapnea. The chest CT scan showed an almost completely compressed left bronchus due to the position of the sternum, atelectasis of the left lung, and a severely enlarged pulmonary artery. Bronchoscopic findings revealed a very small left bronchus full of sputum. She had to be mechanically ventilated in order to prevent the collapse of the bronchus and maintain ventilation and expectoration. **Conclusions or Comments:** Our case shows that when the pectus excavatum induces significant compression of the bronchus, the result is a decreased ventilation with consequent pulmonary artery hypertension and respiratory failure, although patients with FSHD usually do not develop respiratory insufficiency. Restrictive chest wall deformity requires attention by neurologists, especially in patients with muscular dystrophy as well as respiratory muscle weakness.

P-1-251

A case of acute inflammatory demyelinating polyradiculoneuropathy in chronic diabetic patient

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Background & Significance: Peripheral neuropathy is a common complication of diabetes. But, other causes have to be considered if peripheral motor dysfunction is involved in diabetes. For diabetic patients, especially who has diabetic polyneuropathy (DPN), motor symptoms would be ignored, so proper evaluation and treatment could be delayed. We experienced a case of acute inflammatory demyelinating polyradiculoneuropathy (AIDP) with DPN and report the clinical features and laboratory findings **Case:** A 72-year-old woman presented with numbness and tingling sensation in upper and lower limbs with hand clumsiness. Her medical history was diabetes of 25 years and hypertension of 15 years. Symptoms in upper extremities have started four months ago, and endocrinologist diagnosed with diabetic polyneuropathy. Despite symptomatic treatment of diabetic neuropathy, she still complained of worsening pain and novel symptoms under both ankles. The nerve conduction study revealed severe sensorimotor mixed polyneuropathy. In cerebrospinal fluid analysis, protein was slightly increased (59.0 mg/dL) without pleocytosis. And serum Anti-GM1Ab IgG (89.25%) and anti-GD1bAb IgG (>100%) were positive. Ultrasonography showed diffuse swelling of peripheral nerves of upper limbs. We diagnosed her as AIDP on DPN. She was treated with intravenous immunoglobulin and her neurologic deficits were gradually improved. **Conclusions or Comments:** Motor function involvement in DPN is uncommon. But modest motor symptoms are likely to be mistaken for DPN. Ignored symptoms could be early manifestations of other diseases such as AIDP, periodic paralysis, myelopathy and myasthenia gravis. Closed observation and early diagnosis are needed for diabetic patients with motor dysfunction to improve prognosis.

P-1-252

Hypokalemic paralysis caused by renal tubular acidosis type I: a case report

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Background & Significance: Severe hypokalemia can cause paralysis (so-called hypokalemic paralysis), presented as non-periodic and periodic paralysis which is caused by acute shift of potassium into cell or by excessive excretion of potassium. Among the etiologies of hypokalemic periodic paralysis, the most common are thyrotoxic periodic paralysis and sporadic periodic paralysis in Asia and familial periodic paralysis in Western countries. This phenomenon is

a potentially reversible medical emergency with unique diagnostic and therapeutic challenges. We report a case of hypokalemic non-periodic paralysis caused by renal tubular acidosis type I. **Case:** A 71-year old male presented with acute onset of quadriparesis evolving over a period of 3 hours and reaching upto the extent that he could not sit or stand alone. This was the first episode and similar event was not preceded. He denied fever, diarrhea, trauma, overeating or strenuous exercise. He was taking medications including calcium channel blocking agent, proton-pump inhibitor and methylon 8mg per day for hypertension, pneumoconiosis and psoriasis. His vital signs were normal. In neurological examination, muscle power of upper limb was 2/3 and lower limb was 3/4; deep tendon reflexes were present but diminished. Cranial nerve and sensory function test were normal, and pathologic reflex was not observed. The initial laboratory examination showed abnormalities that were serum sodium of 138 mmol/L, potassium of 2.2 mmol/L, chloride of 111 mmol/L, creatinine of 1.42 mg/dL, BUN of 25mg/dL and urine potassium of 39.2 mmol/L. Serum aldosterone, renin, free T4 and TSH were in normal range. ABGA was performed: pH 7.17, pO₂ 103 mmHg, pCO₂ 29 mmHg, HCO₃ 11 meq/L. This ABGA report showed a combination of metabolic acidosis with hypokalemia. Anion gap was 16, and urine anion gap was 30.2, indicating defects of renal tubule that is responsible for acid-base balance. These findings were compatible with renal tubular acidosis(RTA) type I. In the nerve conduction study(NCS), compound motor action potential(CMAP) was decreased moderately in all extremities, but sensory nerve action potential(SNAP) was normal. There was no decremental response nor incremental response in the exercise test. Spine MRI showed no abnormalities. Intravenous KCl supplementation was initiated with alkali replacement. We performed further laboratory test to find secondary etiology of RTA type I including antinuclear antibody(ANA), anti-double strand DNA antibody, anti-neutrophil cytoplasmic antibody(ANCA), which were negative. Schirmer test, salivary scan and abdomen ultrasonography were normal. In the following NCS next day, CMAP was normalized completely with serum potassium of 3.2 mmol/L. Motor weakness was fully recovered to normal. **Conclusions or Comments:** We report a case with hypokalemic paralysis because of RTA type I. Hypokalemic paralysis can be cured completely with potassium replacement. Delayed diagnosis and treatment could worsen the prognosis of patients. Therefore, we recommend clinicians must consider hypokalemic paralysis in the condition of acute onset of quadriplegia.

P-1-253

Polymyositis in a patient with Charcot-Marie-Tooth disease type 1A; diagnostic delay in unusual combination

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Background & Significance: Polymyositis (PM) is one of inflammatory myopathies that typically presents with symmetric weakness of proximal limbs and trunk. Charcot-Marie-Tooth disease type 1A (CMT1A) is the most common inherited polyneuropathy caused by the duplication of peripheral myelin protein 22 (PMP22). It manifests as progressive distal muscle weakness, atrophy and absence of deep tendon reflexes (DTR). The clinical diagnosis of PM may be complicated in those with a coexisting neuropathy that remains unnoticed for a long time. In this report, we present a rare case of PM that developed in a patient with CMT1A. **Case:** A 25-year-old female came to our clinic complaining of bilateral proximal muscle weakness. She had a history of recovery from the same symptoms with the treatment of steroid and azathioprine for about 3 years. On neurologic examination during first visit, power in bilateral hip and hand muscles was grade (Gr) 4; power in distal leg and proximal arm muscles

was Gr 4+. Deep tendon reflexes were absent. Serum creatinine kinase level was elevated up to 1460 IU/L. Initially, she was diagnosed with chronic inflammatory demyelinating polyneuropathy (CIDP) by nerve conduction study, combined with polymyositis (PM) by needle EMG and muscle biopsy. Weakness of proximal arms and legs was dependent on the treatment of prednisolone and azathioprine, while distal weakness was not responsive to treatment modalities. After 6 years of follow-up, her brother who had bilateral finger weakness was diagnosed as CMT1A and subsequently, she was also diagnosed after testing positive for PMP22 gene duplication. **Conclusions or Comments:** We described a patient with unusual combination of polymyositis and CMT1A. If polymyositis develops in a patient with underlying inherited neurologic disease, the diagnosis may be delayed due to the complicated clinical features and unrelated pathogenesis.

P-1-254

A case of carpal tunnel syndrome with bifid median nerve and concurrent persistent median artery confirmed by ultrasonography

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Background & Significance: Frequency of bifid median nerves (BMN) in carpal tunnel syndrome (CTS) is varies from 0.8% to 21%. And a persistent median artery (PMA) is found in approximately 10% of the population. Each of the abnormalities could influence diagnosis, treatment and the consequent prognosis of CTS. We experienced a case of CTS with BMN and concurrent PMA, and report the clinical features and sonographic findings. **Case:** A 75-year-old man presented with pain of bilateral hands for several months. He was on medication of hypertension. The symptoms are same degree on both sides. Motor power including handgrip was normal, but tingling sense evoked by hand movements were in bilateral third and fourth fingers. Tinel's signs were positive and Phalen's maneuver produced paresthesia in both hands. Deep tendon reflexes were normal. And examination did not reveal any limitation of motion. The nerve conduction study showed low sensory nerve action potential (SNAP) amplitudes over finger-wrist and palm-wrist segments on right median nerve. And, finger-wrist segment in left median nerve showed low SNAP amplitude and slow sensory nerve conduction velocity (NCV). By neurosurgery, we found relative swelling of median nerve at wrist crease. Right median nerve did not show swelling at the wrist or carpal tunnel inlet, but PMA and concurrent BMN were discovered. He was treated with steroid injection under ultrasonography-guided with caution of BMN and PMA. Procedure was done without any complication. His symptoms got better gradually. **Conclusions or Comments:** PMA may result in some complications including CTS, anterior interosseous nerve syndrome and pronator syndrome. Association between BMN and occurrence of CTS is controversial, but it seems to be certain that BMN may lead to false negative influences in electrophysiological tests. By using ultrasonography, we could avoid misdiagnosis and malpractice of the invasive procedure. This case shows effectiveness and safety of ultrasonography in diagnosis of CTS patients with anatomic variations.

P-1-255

Ulnar nerve conduction study using the first dorsal interosseous muscle recording in healthy Korean subjects

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Background & Objectives: Ulnar neuropathy at the wrist (UNW) can be diffi-

cult to localize by nerve conduction studies. The standard approach has been the demonstration of prolonged distal motor latency to first dorsal interosseous (FDI), in conjunction with a normal dorsal ulnar sensory response and denervation on needle EMG of ulnar innervated hand muscles with sparing of ulnar innervated muscles proximal to the wrist. However, none of these electrophysiologic abnormalities in isolation clearly differentiate patients with UNW from the more numerous patients with ulnar neuropathy at the elbow (UNE). Recently, conduction block (CB), along with conduction slowing, of FDI motor fibers across the wrist using a simplified version of the short segment incremental study (SSIS) were reported in patients with UNW. The purpose of this study was to describe normative values for ulnar nerve conduction study using the FDI recording before a simplified version of the short segment incremental study (SSIS) was applied in patients with UNW. **Method:** Ulnar nerve motor conduction study with FDI and abductor digiti minimi muscle (ADM) recording was performed in 132 hands of 66 healthy subjects. Ulnar NCS was performed with 2 different recording electrode montages (ADM-base of 5th finger; FDI-thumb) and differences in latency and amplitude were compared. The ulnar nerve was stimulated at the wrist (5 cm proximal to the active recording electrode for ADM). **Results:** The maximal values for terminal motor latency to the ADM and FDI muscle were 2.5 msec and 4.3 msec, respectively. The maximal side-to-side terminal motor latency difference to FDI was 0.4 msec. In addition, the maximal side-to-side terminal motor latency difference to ADM was also 0.4 ms. The maximal ipsilateral latency difference between ADM and FDI was 1.9 msec. There was a tendency for the terminal motor latency of FDI to increase with advancing age. **Conclusion:** We obtained the normative values for ulnar nerve conduction study using the FDI recording, which is useful in applying a simplified version of the SSIS in patients with UNW.

P-1-256

Mutation in heat shock 27 kDa protein causes axonal neuropathy in mouse model

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Background & Objectives: Mutations in heat shock 27 kDa protein 1 (HSP27 or HSPB1) cause distal hereditary motor neuropathy (dHMN) or Charcot-Marie-Tooth disease type 2F (CMT2F) according to unknown factors. Mutant HSP27 proteins affect axonal transport by reducing acetylated tubulin. In this study, we generated new mouse model for HSP27 mutation. **Method:** We generated a transgenic mouse model overexpressing HSP27-S135F mutant protein driven by Cytomegalovirus (CMV) immediate early promoter. Then the phenotype of the model was evaluated by behavior, electrophysiology, MRI, and immunohistochemistry. **Results:** The mouse phenotype was similar to dHMN patients in that they exhibit motor neuropathy. To determine the phenotypic aberration of transgenic mice, behavior test, magnetic resonance imaging (MRI), electrophysiological study, and pathology were performed. Rotarod test showed that founder mice exhibited lowered motor performance. MRI also revealed marked fatty infiltration in the anterior and posterior compartments at calf level. Electrophysiologically, compound muscle action potential (CMAP) but not motor nerve conduction velocity (MNCV) was reduced in the transgenic mice. Toluidine staining with semi-thin section of sciatic nerve showed the ratio of large myelinated axon fiber was reduced, which might cause reduced locomotion in the transgenic mice. Electron microscopy also revealed abundant aberrant myelination. Immunohistochemically, neuronal dysfunctions included elevated level of phosphorylated neurofilament and reduced level of acetylated tubulin in the

sural nerve of transgenic mice. There was no additional phenotype besides motor neuronal defects. **Conclusion:** Overexpression of HSP27-S135F protein causes peripheral neuropathy. The mouse model can be applied to future development of therapeutic strategies for dHMN or CMT2F.

P-1-257

Neuroprotective approach of uric acid in ALS; clinical and therapeutic role

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Background & Objectives: Uric acid (UA) is an important natural antioxidant that may reduce oxidative stress. The association between serum UA levels and ALS, and the cellular and molecular mechanisms underlying its neuroprotective actions remain poorly understood. The objective of this study was to investigate serum UA levels in Korean ALS patients and to relate them to progression of the disease, and demonstrate neuroprotective effect of UA against cell death in vitro ALS model. **Method:** ALS patients and healthy controls individually well-matched for sex, age and body mass index underwent blood tests for serum uric acid levels, which were then correlated with clinical factors and survival rate. In vitro analysis, cells were treated with several concentrations of UA after excitotoxicity, then we evaluate the protective effect of UA in NSC34 cells against toxicant-induced cell damage. **Results:** 136 ALS patients and 136 matched controls were included in the study. The mean UA level of the ALS patients was lower (4.50 ± 1.17 mg/dl) than that of the controls (5.51 ± 1.22 mg/dl) ($p < 0.001$). Levels of serum UA in patients were inversely correlated with progression rate of disease. With Kaplan-Meier analysis, high tertile levels of serum UA correlated with better survival rate than low tertile levels (logrank test: $p = 0.035$). Also, to evaluate the protective effect of UA in NSC34 cells against toxicant-induced cell damage, cells were treated with several concentrations of UA after excitotoxicity. Cell viability was significantly decreased at sodium arsenite exposure and increased cells treated with UA after cell damage. Also UA treatment was increased the expression of PI3K, p-AKT, and p-GSK3 β which are proteins related to neuronal cell survival and decreased levels of cytosolic cytochrome c, activated caspase 3, and AIF, which are associated with neuronal cell death, in oxidative stress condition. **Conclusion:** ALS patients had lower serum UA levels than healthy individuals. UA levels are negatively correlated with rates of disease progression, suggesting that UA levels may contribute to the progression of ALS. Therefore UA levels could be considered as a biomarker of disease progression in ALS patients. In vitro, these results indicated that UA mediates its neuroprotective effects by reducing oxidative stress, enhancing survival signals, and inhibiting death signals. These findings may offer the possibility of treating the patients of ALS, and UA could be an option of multimechanistic approaches.

P-1-258

X-linked dominant Charcot-Marie-Tooth disease type 6 (CMTX6) with mutation in the pyruvate dehydrogenase kinase isoenzyme 3

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Background & Objectives: Charcot-Marie-Tooth disease (CMT), also called as Hereditary motor and sensory neuropathy (HMSN), is one of the most common inherited neuromuscular disorders, with a population prevalence of 1 in 2,500. It encompasses a clinically and genetically heterogeneous group of disorders characterized by distal muscle weakness, wasting, and sensory loss. X-linked dominant Charcot-Marie-Tooth disease type 6 (CMTX6), caused by R158H mutations in the pyruvate dehydrogenase kinase isoenzyme 3 (PDK3), was first reported in Australia. Here, we report two Korean CMTX6 patients to harbor R158H mutation in PDK3 with axonal sensorimotor neuropathy. As far as our knowledge, this is a second case of CMTX6 family with PDK3 mutation. **Method:** We analyzed 845 Korean families including 1,386 CMT patients with to assess causative genetic mutations. Two patients from one family were CMTX6 and three members of kindred underwent a clinical history taking and neurological examination. Nerve conduction studies (NCS) of median, ulnar, peroneal, tibial, and sural nerves are performed on all patients with a surface electrode. **Results:** Two male patients from one family had mutation in PDK3 gene, missense mutation, missense mutation c.G473A (p.R158H). Gait disturbance had developed since 10-year-old age and progressed. In the neurological examination at age of 33, the patient presented weakness and atrophy of bilateral upper and lower extremities and sensory impairment in all modalities. A son of his oldest sister had developmental delay and bilateral limb weakness, and other members of family were normal. Nerve conduction studies showed distal dominant sensorimotor axonal neuropathy. **Conclusion:** In this study, we report second family with CMTX6 related to PDK3 mutation. Although the most common causative mutation is GJB1 mutation in X-linked CMT, we should consider PDK3 mutation in analyzing the cause of X-linked CMT.

P-1-259

A case of Isaac's syndrome with unknown origin

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Background & Significance: Isaacs' syndrome is characterized by continuous muscle fiber activity by a peripheral nerve hyperexcitability. The pathomechanism of this syndrome remains unknown, but a channel abnormality has been speculated as the underlying etiology of this syndrome. Association with myasthenia gravis, thymoma, malignancy, amyloidosis or paraproteinemia has been reported. **Case:** A 54-year-old man was admitted to our hospital due to persistent, generalized muscle contraction and painful muscle stiffness for 2 months. Widespread fasciculation, myokymia were noted in distal and proximal extremities, trunk and facial muscles. His myokymia was worsened by exercise and observed even during sleep. Laboratory examination showed a mild elevation in his serum creatine kinase level (358 U/l, normal range 58-348 U/l) and normal thyroid hormone level. Acetylcholine receptor binding antibody, paraneoplastic antibody, rheumatoid factor, anti-nuclear and anti-SSA/SSB antibodies, serum and urine protein electrophoresis were negative. Sensory and motor nerve conduction studies (NCS) were within the normal range, except for after-discharges on motor NCS (Figure 1.) Needle electromyography demonstrated continuous myokymic discharges in his limb muscles (Video 1). Chest CT and bone scan were non-remarkable. The antibody against voltage-gated potassium channel was not performed. Administration of an anti-convulsant (phenytoin 300 mg/day, carbamazepine 500 mg/day and clonazepam 1 mg/day) resolved his myokymia. However, his leg pain was not improved, and high-dose methylprednisolone therapy (60 g/day for 2 days) was tried. But high-dose steroid therapy markedly aggravated myokymia. **Conclusions or Comments:** This case is our first case of Isaacs' syndrome with unknown origin.

P-1-260**A case of autosomal dominant Emery-Dreifuss muscular dystrophy associated with a heterozygous LMNA mutation**Youn-Eun PARK¹, Jae-Yong SHIN¹, Jin-Hong SHIN¹, Boram KANG², Dae-Seong KIM¹¹Department of Neurology, Pusan National University School of Medicine, ²Research Institute for Convergence of Biomedical Research and Technology, Pusan National University Yangsan Hospital

Background & Significance: Emery-Dreifuss muscular dystrophy (EDMD) is caused by dominant or recessive mutations in the gene encoding nuclear protein, lamin A/C (LMNA). Limb-girdle muscular dystrophy type 1B (LGMD1B) is an allelic disorder of the same gene. EDMD is clinically characterized by multiple joint contractures, as well as progressive muscle weakness. Elbow, ankle and knee joints can be usually involved and rigid spine is also a frequent feature of the patients. We report a patient presented with progressive muscle weakness and significant limitation of neck flexion and rigid spine, who was finally diagnosed as AD-EDMD by whole exome sequencing. **Case:** A 31-year old man presented with progressive muscle weakness. He normally achieved motor developmental milestones during infancy. Since age 4, he lost independent ambulation. He was operated for correction of Achilles tendon contractures at age 10. Since then, upper limbs also became weak, and neck flexion was limited. He reported his mother was slender and had walking and climbing difficulty, and his elder sister had muscle weakness and atrophy and her clinical status was worse than his. Motor power was graded as 1 in neck flexion, 2~3 in proximal upper limbs, 4~4+ in distal upper limbs, 2~3 in proximal lower limbs and 2~3 in distal lower limbs. Facial muscles were spared. He displayed ankle contractures and rigid spine. Nerve conduction study was unremarkable, but needle electromyography revealed myopathic changes in all tested muscles. Muscle biopsy done in left biceps muscle revealed mild myopathic changes with type 1 fiber predominance, but no definite dystrophic changes. For genetic diagnosis, whole exome sequencing was performed, and a known heterozygous mutation of c.1580C>T (R527P) was detected in LMNA. The mutation was confirmed by Sanger sequencing. **Conclusions or Comments:** Joint contractures by sustained immobilization can be developed in various neuromuscular disorders. Congenital myopathies especially can cause multiple joint contractures from birth, which is called as arthrogyrosis multiplex congenita. Otherwise, EDMD is famous for the development of joint contractures during the disease course. Our patient had remarkable rigid spine and neck flexion limitation, as well as progressive muscle weakness. Although muscle pathology failed to suggest a specific muscle disease, clinical features shown in the patients may be the important clue for the specific diagnosis. Here, we searched out a LMNA mutation by whole exome sequencing, but primary Sanger sequencing targeting the gene of LMNA can be recommended in the clinical setting as shown in our patient.

P-1-261**Presenilin 1(PSEN1) mutation in patient with primary lateral sclerosis**Jinseok PARK¹, Ki-Wook OH¹, Young-Eun KIM², Chang-Seok KI², Seung Hyun KIM¹¹Department of Neurology, College of Medicine, Hanyang University, Seoul, Korea, ²Departments of Laboratory Medicine and Genetics, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

Background & Significance: Presenilin 1(PSEN1) mutations were well known as a common cause of early-onset familial Alzheimer's disease. Concerning genotype-phenotype correlation in PSEN1 mutations, myoclonus, seizure, extrapyramidal sign, and spastic paraparesis were common clinical feature. But

patient with motor neuron disease had not been reported. Herein, we describe patient with primary lateral sclerosis accompanied with PSEN1 mutation. **Case:** A 28-year-old male presented with four year history of aggravating forgetfulness and progressive gait disturbance followed by dysarthria. The family history that obtained from his parents showed sudden death tendency on paternal line. But his parents and younger brother showed no cognitive nor motor problem. Neurologic examination showed cognitive decline combined with weakness of lower limbs and dysarthria. He revealed spasticities on both upper and lower limbs with hyper-reflexia and Babinski reflex, ankle clonus, and Hoffman sign. Sensory examination was normal. Neuropsychological evaluation showed loss of verbal and visual memory for immediate and delayed recall, confrontation naming, and visuospatial function with relatively preserved attention and recognition. Electrophysiologic study including needle electromyography(EMG) was normal. In genetic analysis, PSEN1 mutation(c.497T>C) was detected. **Conclusions or Comments:** To our best knowledge, this is first case about PLS patient coexist with PSEN1 mutation.

P-1-262**Spinal xanthomatosis, a rare phenotype of Cerebrotendinous xanthomatosis : a case report**Jin-Sung PARK¹, Gun-Hee LEE¹, Jin-Hong SHIN²¹Department of Neurology, Kyungpook National University, School of Medicine, ²Department of Neurology, Pusan National University, yangsan hospital

Background & Significance: cerebrotendinous xanthomatosis (CTX, OMIM:213700) is a rare autosomal-recessive lipid storage disorder caused by the deficiency of the mitochondrial enzyme sterol 27-hydroxylase (CYP27) with resulting cholestanol and cholesterol accumulations in various central nervous as well as peripheral nervous systems. CTX can be diagnosed by high plasma cholestanol level with the confirmation of molecular genetic testing of CYP27A1 gene. CTx can manifest various neurological symptoms and we report a case of young male who presented with spastic paraplegia and he was finally diagnosed as spinous xanthomatosis, a more rare variant of CTX. **Case:** A 30 years old male visited our hospital with progressive leg weakness, and he had xanthomas in both achilles tendon for 10 years. He was previously an active athlete during high school. When about he was 17, he complained of ankle pain and at that time he was diagnosed as achilles tendinitis. In the early twenties, he was unable to walk by himself and he was diagnosed as spastic paraplegia. His weakness progressed and he was wheel-chair bound when he visited our hospital. His neurologic exam showed a lower leg weakness of MRC grade 2 with spasticity and he showed positive babinski signs and ankle clonus. The laboratory tests including thyroid function test were within normal range. His lipid profile including total cholesterol, low-density lipoprotein, high-density lipoprotein, triglyceride were also normal. However, the cholestanol level was increased by 30.93ug/ml(normal range: 0.86~3.71ug/ml). His spinal magnetic resonance imaging (MRI) was unremarkable but his brain MRI showed a symmetric high signal intensity in the T2 weighted images. The neuropsychology test was performed but he had no significant mental deterioration. His ophthalmologic exam showed peripheral retinal degeneration and there was no evidence of cataract. The electrophysiological study was normal and cardiovascular system testing including echocardiography, transcranial doppler, carotid ultrasound, carotid contrast angiogram were also normal. However Bone densitometry showed a T-score or -1.3 that was compatible with osteopenia. Under the tentative diagnosis of CTX, we proceeded with the genetic study and we found a compound heterozygous mutation of c.1214G>A and c.1263+1G>A in CYP27A1 gene. He was finally diagnosed as spinal xanthomatosis, a rarer variant of CTX. He is currently on chenodeoxycholic acid treatment and we are looking forward for a neurological improvement. **Conclusions or Comments:** Classic clinical manifes-

tations of CTX are childhood onset diarrhea, premature cataract and tendon xanthomas in the adolescence. The common neurological manifestations are early onset cataract, pyramidal signs, ataxia, dystonia, cognition impairment, seizure and parkinsonism in the descending order. MRI abnormality can be observed in two-thirds of the patients that show cerebral and cerebellar atrophy, hyperintense signs on dentate nucleus and globus pallidus with extension to adjacent white matter on T2 weighted images. However, our case presented with isolated progressive spastic paraplegia with achilles xanthomas which is atypical compared to classical CTX. A rare spinal phenotype has been described before and although it is genetically indistinguishable from classic CTX, this rare phenotype is sometimes known as spinal xanthomatosis. SPX does not present with cerebellar signs, seizures or peripheral neuropathy and usually show a benign course compared to the classical CTX. Although CTX can present with various phenotypes, early diagnosis is crucial as CTX is a treatable disease and neurological progression can be slowed or even prevented after appropriate treatment.

P-1-263

Effective treatment with tacrolimus in corticosteroid-resistant polymyositis

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Background & Significance: The idiopathic inflammatory myopathies, including polymyositis (PM), dermatomyositis (DM) and sporadic inclusion-body myositis (IBM), are characterized by moderate to severe weakness and inflammation in the skeletal muscle. It is clinically important since they are treatable diseases. Corticosteroids are considered as the first-line treatment of choice in PM/DM. If there were no response to steroid, or if there had adverse effects of steroid, second-line treatment should be considered. Here, we report two cases of successful treatment with tacrolimus in corticosteroid-resistant polymyositis. **Case:** Case 1. A 46-year-old woman was admitted with progressive weakness developed 2 months ago. She had diabetes mellitus without medication. She denied of any sensory symptoms. Physical examination showed decreased muscle strength in both upper and lower extremities, predominantly affecting the proximal muscle groups. Deep tendon reflexes (DTR) were decreased at the knee and ankle jerk. On laboratory test, creatine kinase (CK) was elevated up to 8470 IU/L. Nerve conduction studies (NCS) were unremarkable except bilateral carpal tunnel syndromes, and electromyography (EMG) revealed myopathy. Based on the clinical and electrophysiological test, we could assume that she was diagnosed with inflammatory myopathy. To confirm the diagnosis, muscle biopsy was performed and demonstrated inflammatory myopathy, especially CD8 positive T cell dominant infiltration in endomysium. Finally, we diagnosed as PM. Initially, we treated with corticosteroid 60mg per day. However, there was no improvement of weakness 4 weeks later. We thought that she had corticosteroid-resistant PM. So, intravenous immunoglobulin (IVIG) therapy (0.4g/kg/day for 5 days) was administered. Although CK level were decreased after IVIG administration, her symptoms were not improved. Therefore, we decided to add tacrolimus (3mg once daily), and taper the dosage of corticosteroid. After 1 month, her weakness was improved as much as she could stand up by herself with minimal assistance. After 4 months, the muscle strength was nearly normal in all muscles. Case 2. A 56-year-old man was admitted with progressive weakness developed 1 month ago. His weakness was started from both lower extremities, following by both upper extremities, and became progressively worse. Before he admitted to hospital, he could not walk without assistant. He had hypertension and hyperlipidemia with medication. Physical examination showed

decreased muscle strength in both upper and lower extremities, and hyporeactive DTR at the biceps, knee and ankle joint. On laboratory test, CK was elevated up to 16730 IU/L. NCS were unremarkable. EMG confirmed an active myopathy, and muscle biopsy demonstrated inflammatory myopathy, especially CD8, CD3, and CD 68 positive T cell dominant. In conclusive, we diagnosed as PM. We started treatment with corticosteroid 60mg once daily. However, 4 weeks after, his symptoms were no improvement. We thought that he had poor response to corticosteroid. So, we administered IVIG (0.4g/kg/day for 5 days) for reducing an inflammation. In addition, oral tacrolimus (3mg once daily) was administered and the dosage of corticosteroid was tapered. His condition was better after add-on therapy with tacrolimus. After 5 months, his muscle strength was nearly normal in all muscles, and he could go up the stairs without help. **Conclusions or Comments:** We experienced that corticosteroid-resistant PM was successfully treated with tacrolimus. Severe adverse effects of tacrolimus were not observed in our cases. Add-on therapy with tacrolimus might be the acceleration of recovery, and facilitate rapid reduction of the corticosteroid. When steroid or other immunosuppressive agents are not response, tacrolimus could be considered as treatment option in PM.

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Treatment-induced neuropathy of diabetes at newly diagnosed diabetes mellitus

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Background & Significance: Treatment-induced neuropathy in diabetes (TIND) is a painful, autonomic neuropathy that develops in rapid improvements in glycemic control in individuals with a long history of hyperglycemia. The neuropathy is predominantly small fiber, affecting autonomic and somatosensory fibers, with little or no involvement of large myelinated nerve fibers on examination or nerve conduction studies. Here, we report the patient with TIND after intensive glycemic control at newly diagnosed diabetes. **Case:** A 30-year-old female was admitted for management of tension headache in neurology department. In admission laboratory, she was diagnosed with type 2 diabetes. She was operated for distal pancreatectomy caused by pancreatic pseudocyst 2 years ago. Neurological examination did not show any evidence of focal neurological deficit and peripheral neuropathy. HbA1c was 12.8% and initial serum glucose was 340 mg/dl. And glucosuria and proteinuria were also detected. The other laboratory tests including thyroid function test did not show any remarkable finding. A nerve conduction study (NCS) was no evidence of neuropathy. The patient started with Insulin treatment (glargine and lispro) immediately and the dose of insulin was escalated for 6 days (glargine, 14 IU to 40 IU; lispro, 4 IU 3 times a day to 10 IU 3times a day). Six days later from insulin applying, she complained right dominant, generalized, severe burning pain, tingling sensation, orthostatic intolerance, and recurrent presyncopal episodes. These symptoms were accompanied with sweating and tiredness and aggravated at early morning. She had hypoglycemic symptoms in the morning and evening at 10 days after insulin applying. Follow-up NCS did not show significant interval change. After reducing the dose of insulin (insulin aspart, 24 IU before breakfast, 14 IU before dinner) for 2 weeks, she relieved burning pain and orthostatic dizziness was also improved. **Conclusions or Comments:** This case describes the patient with TIND which is relatively rapidly appeared than previous reports due to aggressive glycemic control. TIND is an underestimated iatrogenic disorder associated rapid glycaemic change in patients with uncontrolled DM. Although the underlying mechanism is not yet known, there is a clear relationship between a rapid rate

of glycemic control and the development of microvascular complications. All physicians including neurologists keep in mind that rapid glycemic change in patients with uncontrolled DM, especially newly diagnosed, could lead to TIND.

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A case of generalized Myasthenia Gravis with spasmodic dysphonia

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Background & Significance: Myasthenia gravis (MG) is an autoimmune disease mediated by various antibodies leading to a subsequent loss of post synaptic receptors. In about 75% of MG patients, the initial symptoms are diplopia, ptosis and sometimes present with bulbar symptoms such as dysarthria, nasal dysphonia and dysphagia. Spasmodic dysphonia (SD) is a type of focal dystonia caused by intermittent laryngeal spasms affecting voluntary speech production. Here we report a case of a generalized Myasthenia Gravis patient who presented with spasmodic dysphonia. **Case:** A 32-year-old woman complained of dysphonia and non-fluctuating limb weakness for 3 months. However her dysphonia worsened during the afternoon, with fatigability. The initial neurological examination showed dysphonia, but she had no ptosis or diplopia. The neurologic examination showed no abnormalities in the cranial nerves but she showed a symmetric weakness of MRC grade 4+ in her limbs and MRC grade of 4 in her neck flexors and extensors. The laboratory data revealed no significant findings including acetylcholine receptor antibody (<0.01nmol/L) and anti-MuSK (Muscle Specific Kinase) antibody. Her chest computed tomography (CT) was also unremarkable. However, the repetitive nerve stimulation test at low rate stimulation showed a significant decremental response in the orbicularis oculi and trapezius muscles. We additionally performed a voice analysis and laryngoscopy, and the result was compatible with spasmodic dysphonia. Interestingly, we repeated the voice analysis after the pyridostigmine test and there was significant improvement in the numerical and clinical data in the follow up voice analysis. The patient was finally diagnosed as double seronegative Myasthenia gravis with spasmodic dysphonia and she received steroid treatment along with pyridostigmine. After one month of the treatment, she is stable and has no spasmodic dysphonia or weakness. **Conclusions or Comments:** The hallmark of Myasthenia gravis (MG) is fatigability of the peripheral skeletal muscles. Rarely MG is described to have dysphonia and it is described to have hypernasality, glottal incompetence, vocal fatigue, intermittent aphonia, and stridor, which are characteristic features of a flaccid type of dysphonia. Spasmodic dysphonia (SD) is a focal dystonia affecting the vocal cords causing intermittent muscle spasm. The diagnosis of spasmodic dysphonia can be made by three-tiered approach. First, it is screened by questionnaires that are suggestive of a possible spasmodic dysphonia. Then voice analysis is performed to identify probable spasmodic dysphonia. Finally, laryngoscopy is detrimental for a definite diagnosis. The etiology of SD is unknown but it is hypothesized to be related to an abnormal inhibition in the sensory feedback causing intermittent laryngeal spasm via an unknown inhibitory neurotransmitter that need to be elucidated. This further consolidates the findings of our patient who was double-seronegative but she was a good responder to pyridostigmine. A retrospective study showing the characteristic features of laryngeal MG, also showed only 3% positivity of anti-acetylcholine receptor antibody (AChR Ab) but all the patients showed a consistently good response to edrophonium test, that correlate to the findings of our patient. According to recent reports, double seronegative MG patients seem to show a mild disease severity compared to AChR Ab positive patients. Therefore we must include Myasthenia gravis in the differential diagnosis even in patients who present with spasmodic dysphonia.

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Exploration of modifier genes in Korean patients with facioscapulohumeral muscular dystrophy

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Background & Objectives: Facioscapulohumeral muscular dystrophy type 1 (FSHD) is one of the most common forms of autosomal dominant muscular dystrophy, caused by a contraction of the D4Z4 macrosatellite repeat array on the subtelomeric region of chromosome 4q. The SMCHD1 and FAT1 genes were recently identified as modifiers of disease severity. To identify the roles of modifier genes in Korean patients with FSHD, we screened 71 FSHD patients for potentially pathogenic variants in SMCHD1 and FAT1. **Method:** We retrospectively selected DNA samples of 71 FSHD patients from 57 unrelated families. They were tested by targeted next-generation sequencing for SMCHD1 and FAT1 genes. **Results:** Thirty-seven (52%) men and thirty-four (48%) were enrolled. Median D4Z4 copy number was 4 units and 69 (97%) patients carried 1-6 units. The mean age at diagnosis and symptom onset was 31.8±15.1 and 13.1±8.0 years old, respectively. We identified c.3586A>G SMCHD1 variant (based on cDNA sequence NM_015295.2) was identified in FN59 patient. Even though FN59 patient, an 18-year-old girl, had preserved D4Z4 repeats (8 units), she was severely affected. She demonstrated delayed motor milestone and muscle weakness after birth. She was wheelchair-bound at 13 years old. c.3586A>G SMCHD1 variant was considered as benign variants using Sorting Intolerant from Tolerant (score=1.0) and Polymorphism Phenotyping version 2 (score=0.001). However, the potential alteration of splicing of c.3586A>G variant was investigated using three different in silico splice site prediction programs. In the analysis, the variant was predicted to activate an exonic cryptic acceptor site (Human Splicing Finder (range: 0-100), score=87.3, MaxEntScan (range: 0-12), score=8.19, and Splice Site Prediction by Neural Network (range: 0-100), score=0.94). This result suggests the possibility of disease-modifying effects of c.3586A>G SMCHD1 mutation in FN59 patient. **Conclusion:** We identified one c.3586A>G SMCHD1 mutation as modifier of disease severity in severely affected FSHD patient with preserved D4Z4 repeats.

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A novel mutation in DNAJB6, p.(Phe91Leu), in childhood-onset LGMD1D with a severe phenotype

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Background & Objectives: To identify and characterize genetic mutation in a Korean family with limb-girdle muscular dystrophy 1 (LGMD1). **Method:** We analyzed in the affected family members clinical features, DNAJB6 by Sanger sequencing, muscle structures by magnetic resonance imaging (MRI), and functional consequences of the identified mutation using a zebrafish model. **Results:** The clinical phenotypes along with identification of a novel c.271T>C (p.(Phe91Leu)) mutation in DNAJB6 led to the diagnosis of LGMD1D in the affected family members. This mutation presents unique clinical and radiological features compared with other DNAJB6 mutants. All affected members examined showed reduced pulmonary function, and had nasal voice and dys-

phagia except the two members who were thirteen and twelve years of age at the time of examination. Muscle phenotypes developed between 8 to 11 years of age and were more severe as compared to previously reported LGMD1D patients with mutant DNAJB6. Patients' MRI scans exhibited early involvement of the lateral head of gastrocnemius, in contrast to its late involvement in reported LGMD1D cases. Functional study using zebrafish embryos demonstrated that p.Phe91Leu elicits more severe muscle defects than the reported p.Phe93Leu and p.Pro96Arg mutations. **Conclusion:** We conclude that a novel p.(Phe91Leu) mutation in DNAJB6 is associated with severe childhood-onset LGMD1D

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Late-onset myasthenia gravis in Korea: comparison with early-onset and very late-onset myasthenia gravis

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Background & Objectives: To identify the clinical characteristics of patients with myasthenia gravis (MG) according to age at onset in Korea. **Method:** We retrospectively recruited 227 non-thymomatous MG patients with adult onset who had been followed up for at least 1 year. The patients were classified as "early-onset MG" (EOMG) with onset at <50 years old (N=135), "late-onset MG" (LOMG) with onset at 50-64 years old (N=53), and "very late-onset MG" (VLOMG) with onset at ≥65 years old (N=39). Clinical features and serological findings were compared between these groups. **Results:** LOMG patients showed more frequent ocular MG (55%) and less frequent thymic hyperplasia (9%) than EOMG patients (ocular MG, 31%; thymic hyperplasia, 38%) (p=0.009; p<0.001). Particularly, when comparing female patients, more ocular MG (69%) and less frequent secondary generalization (10%) were found in the LOMG group, compared to the EOMG (27% and 47%; p<0.001 and 0.012) and the VLOMG (23% and 59%; p=0.003 and 0.006) groups. There were no significant differences between VLOMG and EOMG patients, regardless of sex, except less frequent thymic hyperplasia (p<0.001) in VLOMG patients. No factors were associated with secondary generalization in LOMG and VLOMG patients, whereas AChR-Ab positivity (HR, 5.48; 95% CI, 1.73-17.37; p=0.004) was independently associated with the secondary generalization in the EOMG group. **Conclusion:** Our study suggests that the female LOMG group was characterized by more frequent ocular MG and less frequent secondary generalization, distinguished from the female EOMG and VLOMG groups, although the VLOMG group shared many similar features with the EOMG group. Further large epidemiologic studies in Korea are needed to determine the characteristics of late-onset MG patients, especially female, according to the age at onset.

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Experience of myasthenia gravis in single tertiary center for 5 years: epidemiological and clinical characteristics of elderly MG

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Background & Objectives: Myasthenia gravis (MG) is the most common neuromuscular junction disorder associated with autoimmune mechanism. It has been known to be prevalent in young adult and female, however, the increase is

more found in the elderly patients at age over 50 years now than before. We aimed to identify the epidemiological and clinical characteristics of elderly MG. **Method:** We reviewed medical records of who admitted to the hospital for management of myasthenia gravis between Jan 2009 and Dec 2013. Total 46 patients were included in this study. We analyzed the clinical characteristics according to the age of onset: type of onset (ocular or generalized), sex, thymic pathology, interval time to generalize, initial acetylcholine-receptor (AChR) autoantibody titer, associated autoimmune disease, clinical course and other clinical features. **Results:** Of the 46 patients, 28 (61%) were young-MG (age of onset ≤ 50 years) and 18 (39%) were elderly-MG (age of onset > 50 years). The mean age of onset was 37.7±10.5 years in young-MG and 63.7±12.2 years in elderly-MG. The proportion of female (50.0% vs. 83.3%, p = 0.030), ocular-type onset (60.7% vs. 88.9%, p = 0.049) and its mean concentration of AChR autoantibody (2.6 vs. 6.3 nmol/L, p = 0.016), normal thymus (32.1% vs. 61.1%, p = 0.072) was significantly larger in elderly-MG. The interval time to generalize from ocular-type onset (p = 0.915), the number of immunosuppressant agent (p = 0.157), and associated thyroid disease (p = 1.00) was not significantly different. **Conclusion:** Unlike other previous study for describing elderly-MG, our results showed that non-thymomatous MG was common among older patients with female predominance. The mean concentration of autoantibodies to acetylcholine receptor of ocular-type onset was higher in elderly-MG than young-MG. It needs to be studied with larger populations to determine whether the non-thymic immune-mediated mechanism is present in elderly-MG.

P-1-270

Vitamin B12 (cobalamin) deficiency with extreme hyperhomocysteinemia presenting as subacute combined degeneration, pancytopenia and splenic MRI lesion

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Background & Significance: Vitamin B12 (Cobalamin) is a water-soluble vitamin which is essential for the integrity of nervous system and the formation of blood. It is also required as a cofactor in the metabolism of homocysteine. Its deficiency is usually due to malabsorption, malnutrition, and genetic disorders, and known to cause megaloblastic anemia, subacute combined degeneration and hyperhomocysteinemia. The mild decrease in vitamin B12 level is commonly seen in clinical practice in Korea but the severe form is relatively rare these days. Here we report a case with severe vitamin B12 deficiency with multiple involvement of nervous and hematopoietic systems. **Case:** A 60-year-old man presented with progressive gait disturbance for 9 months. He also reported a weight loss of 15 kg. He had no remarkable past history of disease, but he has been eating very small amount of food but alcohol. Regarding the family history, his elder brother had been diagnosed as vitamin B12 deficiency several years ago. The patient first noticed his gait problem when he was trying to climb mountain. Since then, the gait disturbance and generalized weakness gradually worsened. At admission, neurological examination revealed decreased proprioception on bilateral lower extremities, positive Romberg test, and impaired tandem gait. Electrophysiologic tests including somatosensory evoked potential showed central conduction block suggesting cervical cord lesion. Laboratory tests discovered non-detectable vitamin B12 level (lower than minimal detecting threshold, 30 pg/ml), megaloblastic anemia (hemoglobin 4.4 g/dl, MCV 119.3 fL), thrombocytopenia (platelet count 70,000 /ul), leukopenia (1,300 /ul), mild hyperbilirubinemia (1.8 mg/dl), and extreme hyperhomocysteinemia (104 uMol/L). Magnetic resonance imaging (MRI) of cervico-thoracic spine revealed T2 high signal intensity on dorsal cervical cord. On brain MRI, there were T2 high signal intensity and diffusion restriction in splenium of corpus callosum and left corona radiata. Intramuscular

cobamide injection was performed once a day for a week, and his gait difficulty was rapidly recovered. The serum cobalamin level at that time was above the detecting threshold (> 2000.0 pg/ml), and repeated Brain MRI revealed decreased signal intensity on diffusion-weighted imaging. **Conclusions or Comments:** Vitamin B12 deficiency should be considered in differential diagnosis of anemia with various neurological symptoms but high signal intensity in splenium of corpus callosum is uncommon. In this case, we suggested that both splenial lesion and ischemic stroke were caused by extreme hyperhomocysteinemia due to very low vitamin B12 level.

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Optic neuritis in patient with overlapping Bickerstaff's Brainstem Encephalitis and Guillain-Barre' Syndrome

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Background & Significance: Bickerstaff's brainstem encephalitis (BBE) is characterized by acute ophthalmoplegia, ataxia, impaired consciousness and hyperreflexia. BBE is an autoimmune disease affecting both the central and peripheral nervous system associated with anti-GQ1b antibody. In the CNS, GQ1b gangliosides are less abundant, but they are present in relatively high concentration in the human optic nerve. There are some reports about optic neuropathy in MFS patients, but BBE with optic neuritis is rare. **Case:** A 34-year-old man was admitted with confusion. Since eight days before presentation, he complained abdominal pain. After four days, he experienced voiding and defecation difficulty. And then, confusional mentality and abdominal distension was observed via his parents. At presentation, he had a fever and mentality was confused. Lateral and dawn gaze limitation of the left eye was seen. There was no weakness and deep tendon reflex (DTR) was normal. CSF study showed 54 cells and a protein level of 92.12 mg/dL with normal glucose level. Brain MRI revealed diffuse leptomeningeal enhancement, but brainstem signal change was not observed. He was diagnosed as BBE based on clinical symptoms and CSF findings, and started receiving high dose steroid pulse therapy. Next day, respiratory difficulty, weakness (below MRC grade II in all extremity) and right facial palsy was newly developed. DTR was decreased generally. Nerve conduction study showed motor dominant sensorimotor polyneuropathy that does not meet demyelinating criteria. We diagnosed as BBE with GBS. We started intravenous immunoglobulin (IVIg, 2g/kg, for 5 days) therapy. Thirteen days after admission, his mental was recovered fully, however, he complained dark vision on the left eye. We reviewed his initial MRI, and found that optic nerves were enhanced at that time. We checked follow-up MRI. Leptomeningeal enhancement was improved but new multiple high signal intensity was revealed in medulla, pons, midbrain and splenium. Visual evoked potential (VEP) showed poor wave formations on the left side. We started high dose steroid pulse therapy once more. However, improvement of his visual acuity was not enough. We decided another steroid pulse therapy. His weakness and visual acuity was improved gradually. At discharge, he could walk with mild ataxia and his left visual acuity was also improved to 0.1 from blind. After three month, his gait was more improved and he could walk alone without ataxia. His left visual acuity was improved to 1.0 and left side VEP waves were observed well compared to previous study. **Conclusions or Comments:** We report a patient of optic neuritis with BBE and GBS. We can diagnose as BBE with clinical, laboratory, MRI findings and positive anti-GQ1b antibody. We concluded optic neuritis is one of CNS findings of BBE. Because of visual symptom is covered with altered mentality, careful observation of optic nerve function and MRI is important.

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A case of recurrent Miller-Fisher syndrome

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Background & Significance: Miller-Fisher syndrome (MFS) is a variant of Guillain-Barre syndrome, which is characterized by external ophthalmoplegia, areflexia and gait ataxia. Besides the typical triad symptoms, additional signs and symptoms can accompany the clinical condition, such as, bulbar palsy, sensory disturbances, or limb weakness. MFS is usually monophasic and recurrence is quite rare. Here, we aim to report a case of anti-GQ1b antibody positive, recurrent MFS in an elderly patient with ten-year interval period between attacks. **Case:** A 72-year old man presented dysarthria, ataxia, and tingling sensation in both forearms and thighs two days ago. He had a cold for one week prior to this symptom noted. He has begun to take a medication for hypertension and diabetes mellitus since last month. Neurological examination revealed marked oculomotor disturbances with the limitations of ocular movements in all direction. Dysarthria and swallowing difficulty were also observed. The limb power was normal, but all reflexes were absent and plantar responses were flexor. He needed assistance when walking due to gait ataxia. A few days later after the initial examination, he had facial diplegia with bilateral ptosis. In nerve conduction study sensory nerves were all abnormal: sensory action potential was not recordable or conduction velocity was slower than lower limit of normal. In contrast, motor nerves were within the normal ranges. The repetitive stimulation test showed neither a decrement nor an increment. Blink reflex test suggested demyelinating polyneuropathy involving bilateral blink reflex pathway. A thorough laboratory examination was performed and included the following tests: vitamin B12, folic acid, liver and renal function, glycosylated hemoglobin A1c, complete blood counts, thyroid-stimulating hormone (TSH) and thyroxine, anti-acetylcholine receptor antibody, analysis of CSF; tests for varicella zoster, HIV, syphilis, and hepatitis; tests for the immunologic parameters antinuclear antibody (ANA), rheumatoid factor and antibodies in Sjogren disease. All of these tests were within normal limits or negative, but CSF protein (51.7 mg/dL) was elevated without pleocytosis. IgG antibodies against GQ1b were tested, and markedly positive using ELISA method. MR imaging of the brain was normal. Interestingly, he had a history of same symptoms and signs ten years ago, which was completely in remission within several weeks. At that time, incomplete ophthalmoplegia, gait ataxia and both hands numbness were noted in his medical records. As we diagnosed recurrent MFS, we initiated treatment with intravenous immunoglobulin for 5 days. At follow-up one month later, the patient showed normal ocular movements and gait. **Conclusions or Comments:** We reported a case of recurrent MFS in an old age, with 10-year interval time between recurrences. Recurrent MFS is a rare clinical condition. One paper reviewed 28 patients suffering a total of 70 episodes of MFS, and a number of recurrences ranged from single to seven in each patient. The mean interval between attacks was 9.45 years. The authors found that clinical features of all described episodes were quite uniform: the classical features of ophthalmoplegia, ataxia and areflexia were present. In addition, recurrent episodes of MFS were quite similar to the first episode. The overall outcome was favorable in all patients. It is unclear what promotes recurrent MFS, either genetic background or immune-mediated mechanism. Diagnosis is dependent on clinical aspects, but strongly supported by positive autoantibodies to GQ1b. Although MFS is a self-limited disease, immunomodulating therapy with IVIGs is favored.

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Churg-Strauss syndrome can also mimic chronic inflammatory demyelinating polyneuropathy

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Background & Significance: Churg-Strauss syndrome is a vasculitis of small to medium-sized blood vessels. It usually accompanied with airway inflammation such as asthma, allergic rhinitis or recurrent sinusitis. It characterized by hyper-eosinophilia, which causes tissue damage, commonly in the lung, followed by skin. It may develop severe or life threatening condition. Here, we present a man who showed chronic symmetric weakness and no typical symptoms of vasculitis, later diagnosed Churg-Strauss syndrome by nerve biopsy. **Case:** The 62-year-old man with no specific medical history was admitted for progressive four limb weakness and gait difficulty since 3 years. He denied any history of asthma, allergic rhinitis and other systemic vasculitis suspecting symptoms. On neurologic examination, he showed symmetric weakness on four limbs (Medical Research Council (MRC) grade IV in both proximal muscles and MRC grade III in both distal muscles) with hyporeflexia. Babinski signs were negative. He showed distal paresthesia in four limbs. Blood tests revealed slight eosinophilia (white blood cell count, 4,100/ μ l with an 7.8% eosinophil count) and cerebrospinal fluid were normal (WBC 0/ μ l, glucose 61mg/dL (serum glucose 113mg/dL), protein 24.5mg/dL.). Laboratory tests for vasculitis including rheumatoid factor, anti-nuclear antibodies, anti-neutrophil cytoplasmic antibody, anti-Ro/SS-A, anti-La/SS-B and immunofixation electrophoresis/protein electrophoresis for urine and serum were all negative. Anti-GM1 IgG/IgM antibodies were absent. He underwent electromyography and nerve conduction study and showed sensorimotor polyneuropathy that is not consistent with demyelinating criteria of chronic inflammatory demyelinating polyneuropathy (CIDP). Brain MRI and cervical spine MRI were within normal. Even though he was initially suspected as having a CIDP, we processed sural nerve biopsy because his laboratory findings were not typical for CIDP. Sural nerve biopsy revealed vasculitic peripheral neuropathy with chronic granulomatosis with polyangiitis. Finally, he was diagnosed as Churg-Strauss syndrome with peripheral nerve involvement and started high dose steroid treatment. His chest CT revealed ill-defined nodule and ground glass opacities in bilateral upper lung area which indicates a pulmonary inflammation. **Conclusions or Comments:** Even in the patient who has symmetric chronic weakness and no prominent vasculitic symptoms, nerve biopsy is still critical if supportive laboratory findings not typically fit with CIDP or other suspected disease. Early diagnosis and treatment prevent organ damage and mortality in this rare form of systemic vasculitis, Churg-Strauss syndrome.

P-1-274

Delayed facial diplegia in a case with serologically confirmed acute motor axonal neuropathy form of Guillain-Barré syndrome

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Background & Significance: Facial diplegia is an isolated subtype or one of major accompanying feature of Guillain-Barré syndrome (GBS). It also found in some cases of Miller Fisher syndrome (MFS) as a form of delayed involvement during recovery stage. But, delayed bilateral facial nerve involvement was not reported in acute motor axonal neuropathy (AMAN) subtype of GBS yet. **Case:** A 49-year-old man visited emergency center due to progressive quadriplegia for 4 days. He had history of 5 days watery diarrhea since 9 days prior to symptom onset. In neurological examinations, his cranial nerves function was intact on admission. Limb motor power was 1 to 4 in medical research council grade with proximal/distal discrepancy and bilateral asymmetry. There was no objective sensory loss. Deep tendon reflexes were absent in all limbs.

Nerve conduction study on 3rd day of symptom onset revealed the early pattern of motor conduction block neuropathy. Anti-ganglioside antibody study using acute stage serum revealed positivity to IgG anti-GM1 antibody with high titer. Intravenous immunoglobulin treatment was effective and his limb weakness was began to recover from 8 admission day. During the improvement of limb weaknesses, unexpected bilateral facial nerve palsy was detected at 19 day. Additional administration of oral steroid for 2 weeks was effective for delayed facial diplegia. **Conclusions or Comments:** This is a very rare serologically confirmed AMAN case accompanied with delayed facial diplegia during the recovery phase. The mechanisms of delayed involvement of facial nerves in GBS or MFS are not clarified yet. Further series of cases will be needed in the future.

P-1-275

2 cases of delayed cranial nerve palsy in Miller-Fisher syndrome

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Background & Significance: Miller Fisher syndrome is considered a variant form of Guillain-Barré syndrome. Ophthalmoplegia, ataxia, areflexia, pupillary abnormalities, blepharoptosis and facial palsy are frequent in MFS. Patients with MFS usually show monophasic event and have good recovery. We report 2 cases of MFS who developed cranial palsy including cranial III, VII and X while the first symptoms were recovering. **Case:** Case 1: A 23-year old man admitted with bilateral facial palsy. He complained of diplopia with ocular pain, ataxia and dizziness after massive diarrhea a week prior to admission. 7 days after symptom onset, while the initial symptoms had fully recovered, bilateral facial palsy and mild dysarthria had newly developed. House-Brackmann facial nerve grade IV and left uvula deviation was observed. Deep tendon reflexes were absent. CSF study showed White blood cell (WBC) count 10cells/mm², protein 80mg/dL and glucose 67/101mg/dL. On serologic test, Anti GQ1bIgG antibody was positive. Blink response revealed absent potentials. Distal sensory nerve conduction test revealed slow velocities. He had no previous medical history. Brain MRI was normal. The patient was diagnosed with Miller-fisher syndrome. He was treated with oral prednisolone 50mg for 2 weeks. After 3 weeks, facial palsy improved gradually, and he was discharged from our hospital. Case 2: A 42-year old woman presented to our hospital with ataxia and diplopia after a week of diarrhea. On examination, she had total ophthalmoplegia, left ptosis, ataxia and areflexia without limb weakness. Brain MRI was normal, CSF study showed WBC count 2cells/mm², protein 41mg/dL and glucose 99/129mg/dL. She was diagnosed as MFS and was treated with IVIg followed by a tapering course of oral prednisolone (30~10mg). On serologic test, Anti GQ1b IgG Ab was positive. Although left ptosis and panophthalmoplegia had improved, left facial palsy had newly developed 2weeks after IVIg. Blink reflex showed left peripheral facial lesion. She was treated with prednisolone 40mg. 2weeks later, delayed facial palsy had improved, left ptosis had newly developed once more. Left ptosis recovered a week later and all neurologic deficits had fully recovered after 2months. **Conclusions or Comments:** Recently, a few case of delayed facial palsy in MFS had been reported. However, patients with delayed cranial nerve palsies, other than facial nerve, are rare. Our case suggests that not only delayed facial palsy but also other cranial nerve palsies may develop at first-symptom recovery stage in MFS. Delayed cranial nerve palsies show a favorable prognosis.

P-1-276

Brachial plexopathy after robotic thyroidectomy using Da Vinci robotic system

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Background & Significance: Since the adoption of the Da Vinci robotic system for thyroid surgery, robotic thyroidectomy (RT) has become a popular option for patients who want to avoid neck scar. Injury to the brachial plexus is a rare complication associated only with RT because of the unique patient positioning and trans-axillary approach. This case report describes an uncommon case of brachial plexus injury that developed after a RT using Da Vinci robotic system. **Case:** A 26-year old female presented with a left thyroid nodule on neck ultrasonography. Fine needle aspiration demonstrated papillary carcinoma. Following lengthy discussions regarding the advantages and disadvantages of conventional open versus robotic assisted trans-axillary thyroidectomy, she consented to the latter, based on factors relate to comesis. In operation room, the patient was positioned with the neck slightly extended. Her left arm was rotated to full abduction at 180 degrees and supported on an armrest wrapped. Incisions were made and dissection proceeded in a traditional manner. Adequate visualization of the thyroid was obtained by the Da Vinci Robot identifying and saving the recurrent laryngeal nerve. Immediately after her surgery, she presented with pain in left arm, subsequently, developed numbness and muscle weakness. Neurological examination revealed left upper arm paralysis and hypoesthesia involving the deltoid (shoulder abduction), biceps (elbow flexion) muscles. The flexion and extension of the wrist and fingers were essentially normal. The deep tendon reflexes of the left biceps and brachioradialis tendons were decreased but otherwise were normal. The active range of motion of the neck and passive ROM of the left shoulder joint were normal. There was no other neurological deficit, such as cranial nerve dysfunction, dysarthria, of incontinence. According to the physical examinations, the brachial plexus injury should be taken into consideration. To confirm the diagnosis, nerve conduction studies (NCS) and electromyography (EMG) were performed 4 weeks after the onset of symptoms. The results of the NCS revealed decreased compound motor action potential (CMAP) amplitude on Lt axillary and musculocutaneous nerves. The NCS of median and ulnar nerves were normal. EMG of the left biceps, deltoid, and brachioradialis showed increased insertion activity and positive sharp waves, along with reduced interference pattern. Furthermore, the EMG results showed no sign of abnormal denervation in the cervical paraspinalis, first dorsal interosseous, and abductor pollicis brevis muscles. Ultrasonography of the brachial plexus in the interscalene and supraclavicular area revealed no structural lesion causing plexopathy. She was discharged from hospital on day 3 post-injury. The patient was assessed at 1 and 3 month postoperatively. The pain previously described had totally disappeared at 1 week after surgery. At 1 month follow-up, the motor weakness was almost improved. **Conclusions or Comments:** This is a case report of a patient, who developed brachial plexopathy after RT using Da Vinci robotic system. Several review articles reported RT-associated traction injury of brachial plexus because of the patients positioning with arm fully abducted. But, this kind of upward traction usually causes injury of lower part of brachial plexus. Our patient showed main injury of axillary and musculocutaneous nerves which are located posterolateral area of the axilla. Not only traction effect but also direct injury by device or compression by tissue edema should be considered as new mechanism of brachial plexuopathy in trans-axillary RT.

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Changes of cortical excitability in obstructive sleep apnea syndrome

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Background & Objectives: Abnormalities in cortical excitability have been proposed to underlie the pathophysiology of various neurocognitive manifestations of obstructive sleep apnea syndrome (OSAS). Transcranial magnetic

stimulation (TMS) provides a noninvasive method for study and modulation of cortical excitability in the human brain. We aimed to investigate cortical excitability in patients with OSAS using TMS. **Method:** We recruited 62 patients affected by OSAS (apnea-hypopnea index [AHI] > 5) and 49 non-OSAS subjects from Seoul St. Mary's Hospital. All the subjects had undergone overnight polysomnography (PSG), and measures of motor cortical excitability (resting motor threshold [RMT], motor-evoked potential [MEP] amplitude and latency, cortical silent period [CSP] as well as intracortical inhibition [ICI] and intracortical facilitation [ICF]) were taken on the day after polysomnography (PSG). We used Pearson correlation coefficient for regression analysis between TMS values and PSG parameters. **Results:** In OSAS subjects, AHI, RDI and arousal index (AI) were significantly correlated with MEP latency ($r=0.484/P < 0.001$, $r=0.45/P < 0.001$, $r=0.518/P < 0.001$) and ICF ($r=0.395/p=0.001$, $r=0.347/P=0.006$, $r=0.418/P = 0.01$). A significant negative correlation was found between RDI and MEP amplitude ($r=-0.271/P=0.033$). Also, sleep efficiency in OSAS patients was significantly correlated with MEP amplitude ($r=0.672$, $P < 0.001$). But, in non-OSAS subjects, no correlation was found between TMS values and PSG parameters. **Conclusion:** We found alterations of motor cortical excitability in OSAS patients were correlated with severity of respiratory parameters and arousal index during sleep. We believe that frequent arousal and sleep deprivation may change the excitability of motor cortex and modifying excitatory and inhibitory cortical circuits. These findings require clarification with further exploration.

P-1-278

Relationship between obstructive sleep apnea syndrome and cerebral microbleeds

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Background & Objectives: Obstructive sleep apnea (OSA) was identified as a risk factor for cerebrovascular disease (CVD) independent of other risk factors, such as hypertension. Silent brain lesion, including cerebral microbleed (CMB), silent brain infarction, and leukoencephalopathy, is considered to be a predictor of stroke. We aimed to investigate the prevalence of cerebral microbleeds in patients with OSA and the correlation between OSA severity and prevalence of microbleeds. **Method:** We enrolled the subjects who underwent both brain MRI, including gradient echo imaging, and polysomnography (PSG) within one year. CMB was defined as the rounded foci of <10 mm in size that appear hypointense and distinct from vascular flow voids, leptomeningeal hemosiderosis, or non-hemorrhagic subcortical mineralization. Overnight PSG was applied to diagnose the presence of OSA and to estimate the severity of OSA. Univariate and multiple logistic regression models were used to identify the predictive factors, including OSA, for the presence of CMB. **Results:** Of 157 cases who underwent both polysomnography (PSG) and MRI, 60 patients were finally included in this study by our criteria. Mean age was 62.4 ± 13.2 years and 37(61.7%) patients were male. Out of 60 patients, 40 (66.7%) patients were diagnosed to obstructive sleep apnea with the apnea-hypopnea index (AHI) of higher than 5/hr. In univariate regression model, the predictive factor of CMB was hypertension ($p=0.041$) and AHI ($p=0.075$). Multiple regression analysis with the adjustment of age, hypertension, smoking revealed that the patients with $AHI \geq 15/hr$ had higher predictive value for the presence of CMB, compared to the patients with $AHI < 15/hr$. ($P=0.014$; odds ratio, 6.661; 95% confidence interval 1.474-30.107). **Conclusion:** We reported that CMB was significantly correlated with hypertension and sleep apnea. In our preliminary results, moderate to severe OSA could be the predictive factor for the risk of CMB.

P-1-279**Long-term Adherence of positive airway pressure therapy in Patients with Obstructive Sleep Apnea Syndrome; What are the main determining factors?**Jung Hwa LEE¹, So-Hee KIM², Seung Bong HONG², Eun Yeon JOO²¹Department of Neurology, Samsung Changwon Hospital, Sungkyunkwan University School of Medicine, ²Sleep Center, Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

Background & Objectives: Positive airway pressure (PAP) is the gold standard treatment for obstructive sleep apnea syndrome (OSA). Yet, adherence to PAP is still challenging in many patients. We investigate the main determining factors influencing the long-term adherence to PAP treatment in patients with OSA. **Method:** We gathered PAP data of 1690 patients who were recommended to use PAP from Jan. 2008 to Feb. 2015. 1226 patients (72.5%) agreed while 464 patients (27.5%) declined to use PAP after instruction about PAP. We investigated the overall adherence of PAP treatment and compared the clinical characteristics including age, sex, Body mass index (BMI), Apnea hypopnea index (AHI), other comorbidities such as insomnia, restless leg syndrome (RLS), periodic leg movement disorder (PLMD), hypertension (HTN), benign prostatic hyperplasia (BPH), cardiac disease and so on between the patients with PAP use and those who discontinued PAP. Additionally, we observed the adherence rate between CPAP (n=175) and AutoPAP (n=585) users. **Results:** Among 1226 patients, 56.1% (n=688) demonstrated long-term adherence (defined as PAP use at least 1 year, < 2 y, n=140; 2-4 y, n=213; 4-6 y, n=206; > 6 y, n=129) and 38.0% (n=466) discontinued the treatment, where 76.8% stopped within 1 month and 21.7% stopped within 2-6 months. Therefore 98.5% stopped within 6 months. In the group of good adherence among the long adherence (defined as PAP > 70% of the days, 41.9% of patients used PAP > 90% of days and 60% slept with PAP > 5 h of total sleep time. The long term adherence group showed significantly higher AHI (38.8/h vs. 27/h, p < 0.001), larger BMI (26.8 vs. 25.5 kg/m²), higher ESS (9.9 vs. 8.8, p < 0.001), higher HTN incidence (51.8% vs 39.0% p < 0.001) but lower insomnia incidence (17.8% vs 29.3% p < 0.001) before treatment than discontinued PAP group. About a third of patients who discontinued PAP complained about discomfort from mask. In contrast, there was no difference in adherence rate and time of use (both 70% of total sleep time) between CPAP and APAP users. In the PAP group, there was an obvious negative correlation between AHI during PAP use and average usage time (P < 0.001 correlation co-efficiency = -0.179). **Conclusion:** Overall 56.1% of patients have continued PAP use for more than 1 year and about 41.9% of patients have used PAP more than 70% of days. Severe OSA, high blood pressure, lower AHI during PAP and daytime sleepiness are the important factors to adhere to the PAP more strongly. Contrastingly, insomnia is an important negative influencing factor.

P-1-280**Acute response of heart rate variability to continuous positive airway pressure treatment in obstructive sleep apnea**

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Background & Objectives: Obstructive sleep apnea (OSA) is known to be associated with autonomic dysregulation and has been recognized as one of major risk factors for cardiovascular diseases. Heart rate variability (HRV), one of the quantitative markers of autonomic function, has been well known to be altered in cardiac diseases such as congestive heart failure and myocardial infarction. A number of studies showed significant changes in HRV in OSA patients relative to controls, suggesting that increased nocturnal sympathetic

activity may play an important role in the pathogenesis of cardiovascular diseases. Continuous positive airway pressure (CPAP) is the most effective treatment in OSA. However, an effect of CPAP treatment on HRV in OSA has not been well understood. We therefore examined an acute response of HRV to CPAP in order to identify beneficial effect of CPAP treatment on autonomic dysregulation in OSA. **Method:** We retrospectively studied patients with moderate to severe OSA (apnea-hypopnea index, AHI \geq 15) who underwent baseline overnight polysomnography (PSG) and second PSG with CPAP titration. Patients with complete correction of apnea/hypopnea (AHI < 5) during CPAP titration were included in the analysis. Patients with proven cardiac arrhythmia or dysautonomia were excluded. Two 5-min epochs of REM sleep and non-REM sleep (N2 stage) periods were extracted from baseline PSG and second PSG with CPAP titration data, with avoidance of ECG artifacts and recording discontinuation. The following 6 parameters were used to assess HRV: (1) SDNN, standard deviation of all RR intervals; (2) NN50, number of adjacent RR intervals differing by more than 50 ms in the entire analysis interval; (3) VLF, very low-frequency band; (4) LF, low-frequency band; (5) HF, high-frequency band; and (6) LF/HF ratio. Within-subject comparison (baseline PSG vs. PSG with CPAP) was performed using paired t-test to examine changes in HRV parameters. Pearson's correlation was performed between HRV parameters from baseline PSG and AHI, sleep efficiency, and oxygen desaturation index to identify relationship between HRV and apnea-related parameters in OSA patients. **Results:** A total of 169 patients (135 males, mean age = 53 \pm 10 years) were finally selected for analysis. Mean AHI from the baseline PSG was 44.5 \pm 21.6 and mean body mass index was 27.6 \pm 3.9. During REM periods, SDNN (t = 10.8, P < 0.000001), NN50 (t = 5.6, P < 0.000001), VLF (t = 3.1, P = 0.002), and LF (t = 2.8, P = 0.005) were reduced in PSG with CPAP titration as compared to baseline PSG. During non-REM period, SDNN (t = 9.8, P < 0.000001), NN50 (t = 10.3, P < 0.000001), VLF (t = 6.3, P < 0.000001), LF (t = 4.6, P < 0.000001), and HF (t = 3.3, P = 0.001) were also reduced in PSG with CPAP titration as compared to baseline PSG. AHI of REM periods was correlated with LF (r = 0.243, P = 0.002) and LF/HF ratio (r = 0.192, P = 0.013) during REM periods. Oxygen desaturation index was correlated with LF (r = 0.184, P = 0.017) during REM periods. No significant relationships were found between HRV parameters and apnea-related parameters in non-REM period. **Conclusion:** We found that significant reductions of SDNN and NN50, representative indicators of cardiac autonomic irregularity, in response to CPAP in both REM and non-REM periods. We also observed that CPAP resulted in significant reductions of LF and VLF, markers of sympathetic activity, in both REM and non-REM periods. Autonomic dysregulation, especially increased sympathetic activity, has been considered as a possible mechanism of increased risk of cardiovascular diseases in patients with OSA. Our findings of acute changes in HRV parameters in response to CPAP suggest that long-term CPAP treatment may reduce the occurrence of cardiovascular diseases by way of reducing excessive sympathetic activity. Further studies are needed to clarify effects of long-term CPAP on HRV, and eventually on prevention of cardiovascular comorbidity in OSA patients.

P-1-281**Significance of snoring for predicting obstructive sleep apnea severity**

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Background & Objectives: The aim of this study was to investigate the predictive value of snoring frequency in the diagnosis and severity of obstructive sleep apnea. **Method:** This observational study was performed retrospectively at a single tertiary hospital. Patients who underwent overnight polysomnography at our hospital with one or more of the following characteristics were eligible

for inclusion in the study: 1) sleepiness, non-restorative sleep, fatigue, or insomnia symptoms; 2) arousal due to cessation of breathing or the occurrence of gasping or choking when waking up; and 3) habitual snoring, breathing interruptions, or both, noted by a bed partner or other observer. We analyzed the differences in clinical and polysomnographic variables between patients with and without obstructive sleep apnea and investigated the associations of those variables with obstructive sleep apnea severity. **Results:** One hundred ninety-three patients met the inclusion criteria. Of those 193 patients, 145 patients were diagnosed with obstructive sleep apnea. The multiple logistic regression analysis showed that snoring frequency was an independent significant predictor of obstructive sleep apnea. However, there was no significant difference in snoring frequency according to obstructive sleep apnea severity, and snoring frequency was not correlated with apnea-hypopnea index. **Conclusion:** We confirmed that snoring frequency is an important predictor of obstructive sleep apnea, and we showed for the first time that snoring frequency is not correlated with obstructive sleep apnea severity. These findings suggest that clinicians should be cautious when using snoring frequency to determine obstructive sleep apnea severity and a patient's response to treatment.

P-1-282

The quadratic relationship between apnea severity and depressive symptoms in patients with obstructive sleep apnea

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Background & Objectives: Obstructive sleep apnea (OSA) is a common sleep disorder, characterized by repetitive obstruction of the upper airway during sleep. The prevalence of depression in OSA has been reported to be high. However, the relationship between severities of OSA and depressive symptoms in patients with OSA is still unclear. **Method:** A relationship between apnea severity and depressive symptoms was determined by univariate and multivariate analyses. The dependent variable was the scores of BDI and the independent variables were AHI, RDI, ODI, and MinSaO₂. The independent variables were analyzed for nonlinear effects using univariate and multivariate quadratic regression model for BDI. We also examined whether the effects of apnea severity on depression differ between men and women with OSA. **Results:** All indicators of apnea severity (AHI, RDI, ODI, and MinSaO₂) used in this study had no linear relationship with BDI scores. In contrast, univariate quadratic regression model showed that BDI had the nonlinear relationships with RDI ($p = 0.008$), ODI ($p = 0.009$), and MinSaO₂ ($p = 0.003$), but not with AHI ($p = 0.058$). In the analyses using multivariate quadratic regression model, ODI ($p = 0.03$) and MinSaO₂ ($p = 0.02$) were identified as factors that had significant nonlinear relationships with BDI. And these relationships were significant only in men (ODI ($p = 0.024$) and MinSaO₂ ($p = 0.011$)), not in women. **Conclusion:** In this study, ODI and MinSaO₂ showed non-linear relationship with BDI in multivariable quadratic regression. And this association is significant only in men, not in women

P-1-283

Comorbid Insomnia in Korean patients with obstructive sleep apnea

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Background & Objectives: Chronic insomnia and obstructive sleep apnea

(OSA) are two of the most common sleep disorders. Increasing evidence from studies suggests that insomnia and OSA frequently coexist. The aim of this study was to investigate the prevalence of patients diagnosed with obstructive sleep apnea (OSA) with insomnia and to compare the clinical characteristics, subjective symptoms related sleep, health-related quality of life (QoL) and compliance of using CPAP between patients who suffer from OSA only and OSA with insomnia. **Method:** We have retrospectively screened the patients who visited two sleep centers. A total of 476 adult patients diagnosed with OSA by polysomnography (PSG), were divided into two groups based according to insomnia severity index (ISI) score: OSA with insomnia ($ISI \geq 15$) and OSA without insomnia ($ISI < 15$). Demographic factors, PSG results, and compliance of using continuous positive airway pressure (CPAP) were compared between two groups. And for all of the subjects, subjective symptoms were evaluated using various questionnaires, including the Korean versions of the Medical Outcome Study Short Form-36 (SF-36), the Pittsburgh Sleep Quality Index (PSQI-K), the Epworth Sleepiness Scale (KESS), the Insomnia Severity Index (ISI), and the Beck Depression Inventory (KBDI)-2. **Results:** Of the 476 patients diagnosed with OSA, 142 (29.8%) patients had insomnia symptom. OSA with insomnia group were more likely to be women ($p < 0.001$) and had more prevalence of heart disease ($p < 0.001$) compared with OSA only group. Subjects in the OSA with insomnia group had lower quality of life (SF-36), lower quality of sleep (PSQI-K), higher KESS score and higher KBDI score. The PSG showed that subjects in the OSA with insomnia group had lower total sleep time and sleep efficiency than those in OSA only group ($p < 0.05$). There was no significant difference in the number of current CPAP user (8.4% vs. 10.2%) and compliance of CPAP between two groups. **Conclusion:** The prevalence of insomnia symptoms in patients with OSA was about 30%. There were significant differences in PSG-based sleep architecture, subjective symptoms including excessive daytime sleepiness and depression and quality of life between OSA with insomnia and OSA without insomnia. There was no significant difference in compliance of CPAP between two groups. However, further investigation about CPAP compliance is needed because of the small sample size of CPAP user.

P-1-284

Electrophysiological characterization of subjects with obstructive sleep apnea syndrome presenting as insomnia

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Background & Objectives: To characterize sleep and sleep quality of subjects with obstructive sleep apnea syndrome (OSA) with insomnia-related symptoms compared to OSA without insomnia in terms of polysomnography (PSG) and cardiopulmonary coupling (CPC) analysis. **Method:** Subjects with OSA (apnea-hypopnea index, $AHI \geq 5/h$, $n = 200$) were enrolled and divided into OSA subjects with insomnia (OSA-I) and subjects with OSA only (OSA-O). Subjects of OSA-I complained of difficulty falling asleep and/or difficulty staying asleep at an initial interview in sleep clinic. CPC parameters were obtained using CPC analyzer in RemLogic (Embla, USA). Sleep spectrogram by CPC analyses is decomposed into the frequency bands: high-frequency coupling (HFC) and low-frequency coupling (LFC), and very low frequency coupling (VLFC), which indicates stable, unstable sleep, wake and rapid eye movement sleep respectively. We compared demographics including mood status and daytime sleepiness, PSG as well as CPC parameters between groups and performed correlation analyses for each group. **Results:** Female proportion was significantly higher in OSA-I (61.1%) than OSA-O (0.8%). Subjects with OSA-I were older (56.4 y) and slimmer (body mass index, BMI 24.4 kg/m²) than OSA-O (44.6 y, 26.0). Subjects with OSA-O were much

sleepier (Epworth sleepiness scale 10.0 vs. 6.8 in OSA-I), however, mood status was not different (Beck depression inventory, 9.6 in OSA-I vs. 8.4 in OSA-O). OSA-I showed significantly longer sleep latency and lower sleep efficiency than OSA-O. Despite of higher arousal index and apnea-hypopnea index of OSA-O, wakefulness after sleep onset was greater in OSA-I. LFC duration among CPC parameters was greater in OSA-O (45.0%) than OSA-I (40.4%), however, it was not significant after adjustment of AHI. In correlation analyses, LFC was positively associated with AHI and arousal index and negatively correlated with lowest SaO₂ after adjusting of age, gender, and BMI in both OSA-O and OSA-I groups. HFC showed the opposite results to LFC with the same PSG parameters in both groups. **Conclusion:** Subjects with OSA-I demonstrated poorer sleep quality regarding PSG parameters in spite of lower respiratory related distress than subjects with OSA-O. CPC parameters are limited to differentiate sleep of OSA subjects with or without insomnia. It needs to explore factors causing poor sleep quality rather than respiratory disturbances in OSA subjects complaining of insomnia-related symptoms.

P-1-285

The relationship between sleep and biophysiological measures in subjects with psychophysiological Insomnia

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Background & Objectives: Since it has not been fully studied whether objective sleep study parameters are related to biophysiological measures in subjects with insomnia. **Method:** we enrolled consecutive 392 subjects with psychological insomnia (PI) who participated in the Wedical checkup program. They completed the overnight polysomnography (PSG) to evaluate sleep disorders causing insomnia. Of them, subjects who had sleep disordered breathing (apnea-hypopnea index, AHI $\geq 15/h$, n=43), REM behavior disorder (n=1), and periodic limb movement disorder (PLMS index $\geq 119.3/h$, n=1) were excluded. 347 participants (age 52.0 \pm 13.09 y, female=231, 66.6%) were included in the study for analyses. **Results:** Overall, sleep quality revealed by PSG was not good; 5.7 h of total sleep time (TST), 76.8% of sleep efficiency (SE), and 17.4/h of arousal index (AI). After adjustment of age and gender, serum hemoglobin and hematocrit level was significantly correlated with mean arterial pressure (MAP), REM latency, N1, N3, and REM sleep (%). Serum WBC level was significantly related to AHI and N1 sleep (%). Body mass index (BMI) of PI subjects correlated with serum HDL-cholesterol, triglyceride (TG), and liver enzyme. The greater scores of movement related arousals and PLMS index were significantly associated with the higher levels of serum TG and liver enzymes. However, TST, SE, or AI did not show any correlation with biophysiological data. Compared to subjects with longer sleep time (TST > 6h/d, n=149, 42.9%), subjects with shorter sleep time (≤ 6 h/d, n=198, 57.1%) were younger (age 49.6 y vs. 53.7y) and showed worse sleep quality. Biophysiological data were not different between two groups. BMI was significantly correlated with short sleep (p=.031). Subjects with low SE (<85%, n=235, 67.6%) showed higher age (54.6 y vs 46.5 y) and unfavorable biophysiological data than subjects with normal SE ($\geq 85%$, n=112, 32.4%), however the data were within normal ranges. Serum glucose level and BMI showed significant correlation with lower SE. **Conclusion:** PI subjects have objectively poor sleep, however, their biophysiological measures are within normal range in general. Several PSG parameters have significant relationships with biophysiological data. It is notable that worse sleep quality was associated with lower BMI in subjects with PI.

P-1-286

White matter hyperintensity in RLS; a comparison of subtypes of RLS

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Background & Objectives: Restless legs syndrome (RLS) shows a tendency to have vascular risk factors and there is a different pathogenesis between early and late onset RLS. However, the results of previous studies that investigated the association between RLS and cardio- or cerebro-vascular disease were mixed. In this study, RLS was classified into early and late onset to investigate the association between the type of onset and white matter hyperintensity (WMH). **Method:** Thirty-nine subjects with primary RLS, were divided into early (n=21) and late (n=18) onset, and 44 healthy controls completed a magnetic resonance imaging scan. To avoid the impact of symptom duration, RLS subjects were selected in light of the symptom duration. Two researchers independently graded WMHs and compared the results to determine the grade. We compared the WMHs in each type of RLS patient to those of age- and gender-matched controls. We also used a series of questionnaires including all Korean versions of the International RLS Severity scale as well as the Insomnia Severity Index and the Pittsburg Sleep Quality Index. **Results:** Inter-rater reliabilities were 0.862 (p=0.000) for periventricular hyperintensity and 0.906 (p=0.000) for deep white matter hyperintensity (DWMH). The mean ages of early and late onset RLS patients were different (47.4 \pm 9.3 vs. 59.8 \pm 5.6, p=0.000). Late onset RLS patients showed a significantly higher grade of DWMH than early onset RLS patients (p=0.043) and age- and gender-matched controls (p=0.031), even after we controlled the variables for cardiovascular risk factors. **Conclusion:** We found late onset RLS to be related to the presence as well as the severity of DWMH, although we cannot determine the causality.

P-1-287

Transient global amnesia impairs small-world topology

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Background & Objectives: Optimal brain network requires a balance between local specialization and global integration, which is known as small-world network. A key step in the pathophysiological cascade of transient global amnesia (TGA) is acute perturbation of hippocampus that is one of the major hubs in the brain network. In this study, we evaluated whether functional brain networks during the acute stage of TGA are characterized by a loss of small-worldness using graph theory. **Method:** A consecutive series of 21 patients with TGA who visited Seoul National University Bundang Hospital and underwent resting-state EEG twice, once in the acute stage (<24 hours after symptom onset) and once in the resolved stage (>2 months after symptom onset), were retrospectively identified. The synchronization likelihood was calculated for all pairwise combinations of EEG channels in the delta, theta, alpha, beta 1, beta 2 and gamma frequency bands. The resulting connectivity matrices were converted to graphs by applying a threshold. Clustering coefficient (CC) and characteristic path length (CPL) were computed as a function of degree K and were then normalized by comparing with random control networks. Small-worldness index (SMI) were also computed using CC and CPL. A paired Student's t test or Wilcoxon rank sum test was used to evaluate the difference in the CC, CPL and SMI between the acute and resolved stage of TGA. **Results:** In the acute stage of TGA, the CC decreased (P = 0.029), the

CPL increased ($P = 0.031$), and the SMI decreased ($P = 0.031$) in the beta 1 frequency band ($K = 13.5$). There were no differences between the TGA stages in the other frequency bands. **Conclusion:** Our study demonstrated that the brain networks during the acute stage of TGA shift away from small-world characteristics in the beta 1 frequency band. Disruption of functional connectivity might play a crucial role in the pathophysiology of TGA.

P-1-288

Brain areas involved in performing TTCT (Torrance Tests of Creative Thinking)

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Background & Objectives: TTCT (Torrance Tests of Creative Thinking) is an exemplar test to evaluate divergent thinking styles. (Torrance, 1998). The aim of this study is to elucidate the neural correlates of performing TTCT. Additionally, we tried to find the discriminative brain areas between high and low score groups and the highly-correlated to the subcategories TTCT scores. **Method:** Fifty seven teachers' college students at Dongguk University, Seoul, were evaluated using the TTCT Figural form A. From that sample fifteen right-handed (screened by Edinburgh handedness scale) participants (6 males, 9 females) were recruited using their creativity index (CI) gained with the TTCT figural form A for a high (group A) and a low score group (group B). While group A consisted of 8 students with CI= 106-123, group B included 7 students with CI= 67-97. The mean CI of each group was 127.6 and 81.6. The mean age of the group A was 18.8 ± 0.7 (range: 18-20) and the mean age of the group B was 20.0 ± 2.2 (range: 18-24). The fMRI task consisted of two blocked conditions: creative drawing imagery (CDI) and eye tracking (EC). In the CDI blocks as a task condition, participants were asked to draw figures in his mind while they were lying in MRI scanner based on the lines presented which were extracted from the TTCT figural form B that the participants had not conducted. In the ET blocks as a control condition, they let their eyeballs track the lines presented repetitively. Each block's duration was 30 minutes and 6 CDI and 6 EC blocks were arranged with alternative manner. The imaging was done by the 3T Magnetom Skyra MRI scanner (Siemens, Germany). fMRI data analysis was carried out using SPM8. (Wellcome Department of Cognitive Neurology, London, UK). In first-level statistical analysis (individual analysis), epoch-related BOLD response was modelled by box-car function with the convolution of canonical HRF. Task related brain activation was identified with the contrast of task > control epoch. To identify the creativity related brain activation, the second-level analysis (group analysis) was carried out on the task related brain activity with the comparison between group A and B and multiple linear regression approach. Individual creativity score was categorized into innovative (originality, fluency) and adaptive (resistance, abstractness, elaboration) categories. Sums of each category were used as covariates of interest, and age and gender were also used as covariates to adjust the potential confounding effects. **Results:** Group comparison (between group A and B) revealed increased brain activities of group A (red colour, Fig 1) in left dorsal prefrontal (BA6), medial frontal (BA6), lingual (BA 18), posterior cingulate and precuneus (BA 5/7/24/31). And activations of right prefrontal (BA6) and anterior cingulate (BA32) gyrus were also increased. On the other hand, in group B (green colour, Fig 1), activations of right fusiform (BA 37) and inferior frontal gyrus (45/47) were increased. Multiple linear regression revealed higher correlation in left insula (BA13), inferior frontal (BA45), middle temporal (BA39), posterior cingulate (BA23/30), cuneus and lingual gyrus (BA18/30) with innovative subcategories of TTCT scores (green color, Fig 2). With adaptive subcategories, many bilateral cortical areas were correlated (red color, Fig 2). **Conclusion:** With this fMRI study, we found the brain areas

which subserves doing TTCT figural form and correlated activation sub-categorical scores (innovative Vs adaptive).

P-1-289

Annotating system for PhosphoVariant

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Background & Objectives: Protein phosphorylation is one of the most important biochemical processes in the living organisms. By phosphorylation, proteins transduce signals to other proteins and the signal transduction pathways are involved in development and learning at the organism level, and the cell cycle, differentiation and apoptosis at the cellular level. Various diseases are caused by mutations in phosphorylation sites. For an example, in Alzheimer's disease, the hyperphosphorylated tau protein is one of pathological proteins and various kinases are known to involve in pathogenesis. In the whole genome/exome sequencing studies, tremendous mutations are found out. In this study, we made a computer program to annotate such mutations that changes phosphorylation sites and corresponding kinases. **Method:** In the previous study, we made program called PredPhospho that predicts phosphorylation sites and their kinases. Our PredPhospho and PhosphoVariant annotation system were implemented using the PERL (<https://www.perl.org>) programming language and MySQL (<https://www.mysql.com/>). **Results:** We thought that phosphovariants change phosphorylation sites or their interacting kinases. We classified phosphovariants as type I, II, and III according to mutation locations and changed kinases. If users input protein ID and locations of mutations, the program predict phosphovariants and their kinases. **Conclusion:** Because phosphorylation is important in protein functions, phosphovariants may change protein functions. Users can use our program to screen important polymorphisms and identify the mechanisms of genetic diseases and traits.

P-1-290

Ubiquitination-dependent and - independent tau clearance via SQSTM1/p62

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Background & Objectives: For the clearance of tau proteins both the ubiquitin-proteasome system and autophagy are involved. Polyubiquitination of tau proteins would be an early and essential step for these processes. There is ubiquitin binding domains (UBA) within SQSTM1/p62 through which SQSTM1/p62 binds to tau proteins thereby transferring these into clearance machinery. To understand the molecular mechanisms in relation to the polyubiquitination we performed this study. **Method:** Various mutant constructs of SQSTM1/p62 were established. Further, the point mutation of ubiquitin constructs to impair K-63 polyubiquitination was established. Under the overexpression of wild-type and mutant ubiquitin constructs the efficacy of SQSTM1/p62 proteins on tau clearance were evaluated. **Results:** SQSTM1/p62 was demonstrated to enhance the clearance of tau proteins in neuronal cells, both total and phosphorylated form. The deletion of UBA within SQSTM1/p62 markedly diminished its efficacy on tau clearance, but not completely. Under the condition of ubiquitin overexpression, the overall increased ubiquitination was linked to highly enhanced clearance of tau proteins. The remaining clearance of tau proteins even after deletion of Δ UBA constructs were identified, which was thought to be mediated through ubiquitination-independent pathway. In addition, when K-63 polyubiquitination was inhibited via K to R point mutations the pattern of wild-type of SQSTM1/p62-dependent tau clearance was altered compared to the condition of wild-type ubiquitin

overexpression. **Conclusion:** We identified SQSTM1/p62-mediated tau clearance is dependent on polyubiquitination. However, ubiquitin-independent tau clearance by SQSTM1/p62 was also noted, which is considered cooperatively mediates tau clearance with ubiquitin-dependent pathway.

P-1-291

A mini-gene for measuring alternative splicing of tau exon 10

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Background & Objectives: Tau plays an important role in maintenance of neuronal cytoskeleton and axonal transport as a neuronal microtubule-associated protein. Alternative splicing of exon 10 of tau gene determines the expression of either three-repeat (3R) or four-repeat (4R) tau isoforms which differ in the number microtubule binding domains. The imbalance between 3R and 4R tau isoforms leads to several neurodegenerative diseases called tauopathy. However, the mechanism underlying the alternative splicing of exon 10 has not been determined. **Method:** As the first step to investigate the mechanism of exon 10 splicing, we developed a tau mini-gene construct encompassing exons 9-11 and part of introns 9-10, which are required for the correct splicing of exon 10. Firefly luciferase reporter was tagged to the c-terminal of this construct, and stop codon was inserted at 73 base-pair of the exon 10. **Results:** To check the efficacy of the luciferase reporter system in measuring the exon 10 alternative splicing, we co-transfected this tau mini-gene with the constructs expressing Tra2 β or SRp55 proteins in CHO cells. Tra2 β or SRp55 proteins are chosen due to their well-known role to regulate tau exon 10 alternative splicing in the opposite way. When Tra2 β construct is co-transfected with tau construct, the expression of luciferase was faint. In contrast, SRp55 co-transfected cells revealed the increased luciferase production. These findings demonstrate that the luciferase acts as a reporter of alternative splicing of exon 10. **Conclusion:** We have made an efficient and specific tau mini-gene that can be used to identify alternative splicing of exon 10 of tau gene. It can be used to explore the mechanism of tau exon 10 splicing and to test the efficacy of candidate molecules in regulation of tau splicing.

P-1-292

The differential caspase activity in cortex and striatum by 3-nitropropionic acid chronic infusion

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Background & Objectives: The systemic administration of 3-nitropropionic acid (3-NP) facilitates the development of selected striatal lesions, and there some reports have provided the clues or the different points about the pathology. In this study, we investigated the relationship between reduced brain derived neurotrophic factor (BDNF) level in lesioned brain region and active caspase, and also the involvement of Apoptosis signal-regulating kinase 1 (ASK1) in caspase activation. **Method:** We analyzed the apoptotic cell death, BDNF distribution, caspase-3 activity, caspase-6 activity, and ASK1 expression level also active ASK1 in the cortex and striatum each. **Results:** Above factors presented differential and distinct level and distribution in each subregion. We also demonstrate that by downregulating of ASK1 in the cortex, caspase-6 activity was reduced; BDNF protein levels were prevented to decrease in the cortex; and severely restricted BDNF was replenished in the striatum. **Conclusion:** With these findings, the present study suggests that the increased ASK1 of the damaged cortex is related with caspase-6 activation, and involved in BDNF restriction of striatum from cortex. Furthermore, under same damage by systemic infusion of 3-NP, the differential expression level of ASK1 between cor-

tex and striatum might be elucidated a kinase factor to modulate the caspase activation and striatal degeneration.

P-1-293

DGAT2 mutation relevant with an autosomal dominant early-onset axonal Charcot-Marie-Tooth Disease

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Background & Objectives: Charcot-Marie-Tooth disease (CMT) is a clinically and genetically heterogeneous group of inherited peripheral neuropathies. Further genetic and phenotypic classification of CMT2 has grown to more than 20 subtypes. However, considerable CMT2 patients still wait for the genetic cause to be uncovered. Recently, zebrafish have been widely used to model human disorders including CMT, and manipulated to mimic the pathological phenotype of CMT. In this study, we investigated to identify the underlying cause of a Korean autosomal dominant axonal CMT family. **Method:** Two patients from an autosomal dominant family were evaluated. To isolate the genetic cause, exome sequencing was performed. In vivo assays using zebrafish models were performed to examine the DGAT2 mutation causing CMT neuropathy pathogenesis. **Results:** Patients had an early-onset axonal CMT phenotype characterized by sensory ataxia, tremor and slow disease progression. Large myelinated fibers were markedly decreased in the sural nerve, and lower limb MRI showed length-dependent axonal degeneration. Pedigree analysis and exome sequencing analysis identified a heterozygous missense mutation (p.Y223H) in the diacylglycerol O-acyltransferase 2 (DGAT2) gene. DGAT2 encodes an endoplasmic reticulum-mitochondrial-associated membrane protein, acyl-CoA:diacylglycerol acyltransferase (EC 2.3.1.20), which catalyzes the final step of triglyceride (TG) biosynthesis pathway. Patient showed consistently decreased serum TG levels, and over-expression of the mutant DGAT2 significantly inhibited the proliferation of mouse motor neuron cells. Moreover, the mutant form of human DGAT2 inhibited the axonal branching in the peripheral nervous system of zebrafish. Therefore, we suggest that mutation in the DGAT2 is the novel underlying cause of an autosomal dominant axonal CMT2 neuropathy. **Conclusion:** We suggest that mutation in DGAT2 gene is the novel genetic cause of the autosomal dominant axonal CMT neuropathy, and this study broadens the genetic and clinical spectrum of axonal CMT. We believe this finding will help in better understanding the pathophysiology and function of the DGAT2 gene in relation to axonal neuropathies and also contribute to the exact molecular diagnostics of an axonal CMT.

P-1-294

Patient fibroblasts-derived induced neurons demonstrates neuropathology of Krabbe Disease

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Background & Objectives: Krabbe disease (KD) is an autosomal recessive neurodegenerative disorder caused by the defects of β -galactosylceramidase (GALC) activity, a lysosomal enzyme that is responsible for cleavage of substrates including galactosylceramide (GalCer) and psychosine. Defects in GALC activ-

ity causes demyelination, which is thought to trigger neural abnormalities in KD. However, recent reports have indicated myelin-free neuronal and axonal defects in the Twitcher mutant mouse models of KD. **Method:** In the present study of the patient who had KD at 12 years of age with low GALC activity had one novel nonsense mutation K563X combined with a known missense mutation L634S in compound heterozygous patterns. To investigate the neuropathophysiology of KD, we generated induced neurons (iNeurons) derived from the patient fibroblast. **Results:** The amount and morphology of GALC, GalCer and psychosine, the lysosomal proteins, and mitochondria were determined in cultured iNeurons of the patient and control. The length of neurites was shorter in the KD patient than in the control iNeurons. The lysosomal-associated membrane proteins 1 and 2 (LAMP1 and LAMP2) were expressed at higher levels than in controls, and the LAMP1-positive vesicles were enlarged in the patient cells. Moreover, undigested GalCer and psychosine were observed in the KD iNeurons. Incubation of control iNeurons with psychosine resulted in neurite defects, mitochondrial fragmentation, and lysosomal alterations. **Conclusion:** KD patient iNeuron model provides a disease-relevant cell model and gives insights that the accumulation of GALC substrates contributes to perturbed organelles and neuropathology in KD. KD patient iNeurons are important research tools for the visualization of neuronal defects in adult-onset KD pathogenicity.

P-1-295

Novel mutations in MKS3/TMEM67 genes in COACH syndrome

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Background & Significance: COACH syndrome is a rare autosomal recessive developmental disorder, a subtype of Joubert syndrome and related disorders (JSRD) with multiorgan involvements, characterized by cerebellar vermis hypoplasia, oligophrenia, ataxia, coloboma, and hepatic fibrosis. Mutation in TMEM67/MKS3 was recently reported to cause COACH syndrome. **Case:** A 21-year-old Korean man with chronic hepatopathy was referred to our hospital with a provisional diagnosis of cerebellar ataxia. Clinical evaluation revealed that he had cerebellar ataxia, isolated elevations in serum γ -glutamyl transpeptidase activity, oligophrenia, the molar tooth sign (MTS) in the brain MR images and congenital hepatic fibrosis (CHF). However, chorioretinal colobomas and nephronophthisis were not noted. In addition, his parents and sister were clinically unaffected. Direct sequence analysis of TMEM67 in the patient showed two novel compound heterozygous mutations: 1) missense mutation (c.395G>C and p.Gly132Ala) in exon 3, and 2) deletion in exon 26 (c.2758delT and p.Tyr920ThrfsX40). **Conclusions or Comments:** Finally, we found that the two mutations facilitate turnover of TMEM67 protein using Western blotting. The two mutations in TMEM67 and their effect on TMEM67 protein stability will promote diagnosis of COACH syndrome and understanding of molecular function of TMEM67.

P-1-296

Ketamine increases excitability of hippocampal neurons through KCNQ Currents

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Background & Objectives: Recent studies showed that ketamine, an ionotropic

glutamatergic NMDAR (N-methyl-D-aspartate receptor) antagonist, produces a fast-acting antidepressant response in patients with major depressive disorder. However, little is known about the effects of ketamine in the regulation of ion channels in the brain. KCNQ channels regulate neuronal excitability and KCNQ channel inhibitor XE991 reverts cognitive impairment. **Method:** We tested the action of ketamine on KCNQ2/3 channels in HEK293 cells and hippocampal neurons using patch clamp technique. **Results:** Ketamine inhibits KCNQ2/3 currents heterologously expressed in HEK293 cells. Current inhibition by ketamine was voltage independent but concentration-dependent. The IC₅₀ for current inhibition was $50.7 \pm 13.4 \mu\text{M}$. The voltage-dependent activation of the channel was not modified. The powerful effects of ketamine on cloned KCNQ channels imply that ketamine action on KCNQ channels didn't involve NMDAR. The effects of MK801 and DL-2-amino-5-phosphonopentanoic acid (AP-5), NMDAR blockers that are structurally similar to and distinct from ketamine, respectively, were also examined. MK801 had similar inhibitory effects on KCNQ2/3 channels, but AP-5 showed no effects on KCNQ2/3 activity, suggesting the direct effects of ketamine and MK801 on KCNQ2/3 channels. In hippocampal neurons, which endogenously express KCNQ2/3 channels, application of ketamine produced an increase in neuronal excitability and input resistance. Subsequent application of XE991 had little effect on ketamine-treated cells, indicating that ketamine mimicked and occluded blockade of KCNQ2/3 channels with XE991. **Conclusion:** Taken together, these data suggest that ketamine is a KCNQ2/3 channel modulator and the modulation of the neuronal excitability by ketamine may contribute to the fast-acting antidepressant action of ketamine.

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A case of baclofen-induced encephalopathy presenting reversible structural brain lesions

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Background & Significance: Baclofen, the derivative of the neurotransmitter gamma-aminobutyric acid (GABA), is the centrally acting GABA agonist. Baclofen is commonly used drug to relieve spasticity. Few cases of baclofen-induced encephalopathy have been reported in patients with end stage renal disease. Including baclofen, drug-induced encephalopathy is caused by impaired cerebral metabolism that is not attributed to structural brain lesions in general. However, no previous study has reported reversible encephalopathy with structural brain lesions in a patient receiving baclofen therapy. **Case:** A 58-year-old wheelchair-bound woman with history of traumatic cervical spinal cord injury was admitted to our hospital due to altered mentality. She had a medical history of muscle relaxant for spasticity. The medication was changed to baclofen from dantrolene recently. At the time of admission, she had a body temperature of 36.7°C . A neurological examination revealed a drowsy mental status and quadriplegia. Neck stiffness and signs of meningeal irritation were not observed. Routine laboratory tests showed a leukocyte count of $21.3 \times 10^3/\text{mm}^3$ with 80.3% neutrophils, and a C-reactive protein level of 0.8 mg/dL. Magnetic resonance imaging revealed bilateral symmetric T2 hyperintensity lesions in whole gray matter and subcortical white matters of both cerebral hemisphere. The cerebrospinal fluid (CSF) examination was normal. Gram staining, acid-fast bacillus staining, KOH mount, India ink preparation, and tuberculosis polymerase chain reaction of CSF were negative. Cultures for bacteria, fungi, and mycobacteria, as well as viral cultures, from the patient's blood, urine, and CSF were also negative. Autoimmune antibody tests were entirely negative. On day 2 after the baclofen treatment termination, the patient's neurological examination showed marked improvement. After 1 week,

she had recovered completely, and follow up MRI showed completely resolved diffuse T2 hyperintense lesions. With baclofen discontinuation, her symptoms did not recur. Because extensive laboratory analyses were entirely normal, and brain image and neurologic manifestations improved after discontinuation of baclofen, we therefore suspected baclofen as an acquired cause of reversible encephalopathy. **Conclusions or Comments:** To our knowledge, this is the first reported case of reversible encephalopathy most likely due to baclofen treatment and with no evidence of any other causative disorder or medication. The fact that immediate baclofen discontinuation led to a prompt recovery regarding both brain images and neurologic manifestations supports a causal relationship. Baclofen is the common drug for the management of spasticity from spinal cord lesions and multiple sclerosis. However, neurological adverse effects of baclofen could be induced. Baclofen has been revealed to cause neuronal hyperpolarization and decrease the release of neurotransmitters such as glutamate, catecholamine, and substance P. Our observation is particularly important, as this patient with apparent baclofen-induced encephalopathy made a complete recovery with immediate baclofen discontinuation. Therefore, in acutely confused patient on baclofen usage, drug-encephalopathy should be considered as one of possible causes.

P-1-298

Spinal tuberculosis

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Background & Objectives: Spinal tuberculosis (ST) is a destructive form of tuberculosis. It accounts for approximately half of all cases of musculoskeletal tuberculosis. ST is more common in children and young adults than the others. Since typical tuberculous spondylitis spreads to involve the adjacent disk spaces while ST does not. Moreover, the radiographic appearance of ST often mimics skeletal neoplasm. Despite its common occurrence and the high frequency of long-term morbidity, there are no straightforward guidelines for the diagnosis and treatment of spinal tuberculosis. Early diagnosis and prompt treatment are necessary to prevent permanent neurological disability and to minimize spinal deformity. **Method:** A 27-year-old woman with no significant medical history visited our hospital due to progressive gait disturbance and numbness of both legs over 3 weeks. Her complaints were accompanied with upper back pain and a weight loss (5kg). At the time of admission, vital sign was stable and physical examination was normal. Neurologic examination revealed bilateral lower extremities anesthesia and paresthesia with vague sensory level at T12-L1, bilateral spastic gait with hyperreflexia in lower limb, positive Babinski sign, and positive Romberg sign. But other neurologic exam was normal. Laboratory studies were not remarkable except for slightly elevated erythrocyte sedimentation rate (ESR) of 31mm/hr. Thoracic CT and MRI revealed destruction of the vertebral body of T4- T5, anterior epidural space, paravertebral space, and bilateral posterior element, encroaching dural sac and both neural foramen. There were no remarkable spinal cord signal intensity change. At first, primary or metastatic malignancy was highly suspected. Lumbar puncture was also performed to exclude other conditions such as pyogenic infection, revealing normal CSF findings. The patient underwent laminectomy of T4- T5; the soft tissue mass that encased the dura was successfully debulked. The histopathology of the removed soft tissue revealed necrotizing granulomatous inflammation with a few acid-fast bacilli, suggestive of Mycobacterial infection. No malignancy was evident. The patient regained part of normal neurologic function (improved gait and relieved lower extremities numbness) after surgical treatment and she was started treatment with antimycobacterial agents. **Conclusion:** Although the prevalence of tuberculosis had been rapidly decreased in past by the country widely expanded na-

tional control program, improvement of nutrition and development of medical services, Tuberculosis is still a serious communicable disease in Korea. Typical granulomatous inflammation in tuberculosis resembles malignant deposits in the spine, which are more frequent. Our case is an excellent example of wrong interpretation of TB as primary or metastatic neoplastic disease. An MRI scan showed compression fracture of the T4-5 vertebral body with infiltration of paraspinal tissue at the vertebral column with indentation of osseous masses into the spinal canal. This case was similar to those of bone and spinal neoplastic metastasis. The rapid diagnosis of ST demands a high index of suspicion and expertise regarding the appropriate diagnostic procedures. Due to the devastating consequences of a missed diagnosis, Mycobacterium tuberculosis should be considered early.

P-1-299

A case of ventriculitis associated with extended spectrum beta-lactamase producing *Klebsiella pneumonia* after acupuncture

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Background & Significance: Ventriculitis is an uncommon infection of central nervous system caused by infection of the ventricular drainage system. Although it can occur as a primary infection, almost cases of ventriculitis develop as the complication of meningitis, cerebral abscess, intra-ventricular hemorrhage or iatrogenesis. In addition, ventriculitis following invasive brain surgery or procedure has been reported. Although, Staphylococci is the leading pathogen of ventriculitis, other strains has been reported as possible causes of ventriculitis. Acupuncture is widely performed in the East Asia, particularly in Korea, to relieve musculoskeletal pain. Here, we report a case of ventriculitis caused by extended spectrum beta-lactamase (ESBL) producing *Klebsiella pneumonia* after acupuncture. **Case:** A 72-year-old woman was admitted to our hospital with a reduced level of consciousness and irritability. She has been suffered from chronic low back pain and underwent acupuncture on the day before admission. After acupuncture, she complained of headache and vomiting and had become drowsy. Her past medical history was unremarkable except for diabetes mellitus. At admission, body temperature was 37.2°C. On neurological examination, she was stupor, and had sluggish light reflex, pupil dilatation and ptosis at left eye and marked neck stiffness. Laboratory study revealed increased C-reactive protein (38.39mg/dl) and normal leukocyte count (7880/mm³). Cerebrospinal fluid analysis revealed frank pus, elevated leukocyte count 30750/ml (89% were neutrophils), low glucose concentration, and elevated protein level (441mg/dl). On fastidious anaerobic broth media, small white hemolytic colonies that were susceptible to chloramphenicol, Piperacillin/Tazobactam, Imipenem and meropenem grew after 48 hours after culture. Biochemical profiling demonstrated that the colonies was *K. pneumonia*. Brain MRI showed mild dilatation of the both lateral ventricle, surrounded by increased signal, subtle diffusion high signal fluid-debris levels in both occipital horns and enhancement of the ependyma. These features were compatible with ventriculitis. The patient received empirical antibiotic therapy within 10 hours of presentation. Following the result of biochemical profiling, antibiotics were changed to meropenem. Surgical management including intraventricular drains for intrathecal administration of antibiotics was given. After the end of treatment, she became to be alert, albeit tetraplegia was remained. **Conclusions or Comments:** To our best knowledge, this is the first report of ventriculitis associated with *Klebsiella pneumonia* after acupuncture. In this case, primary infection that leads to ventriculitis was not found. In addition, the patients did not receive any neurosurgical intervention. It is therefore reasonably assumed that insufficient aseptic technique during acupuncture may inoculate skin normal flora into cerebrospinal fluid at lumbar level. Given that acupuncture is extensively used in Korea, more attention should be given to

the thorough sterilization before acupuncture.

P-1-300

Infection related cerebral venous thrombosis after phlebotomy and dermopuncture on cervical muscle

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Background & Significance: The incidence of cerebral venous thrombosis has dropped dramatically in recent years. In the past, before the introduction of antibiotics, infection was the main cause of cerebral venous thrombosis (CVT). Recently, the occurrence of septic CVT is rare, which leads to an increased chance of misdiagnosis and treatment delay. Early suspicion and recognition is very important for improving prognosis of the disease. For this, we always consider infection as a source of severe headache. CNS infection such as septic CVT can result from many causes. We reported a case of dural venous sinus thrombosis with bacterial meningitis after phlebotomy and dermopuncture on cervical muscle. **Case:** A 59-year-old male visited our outpatient-department(OPD) of neurology due to headache. He had history of long-term cervical muscular pain, so had visited to a herbal clinic and taken phlebotomy and dermopuncture on cervical muscle on repeat a few days before. And soon after, he presented with diffuse bursting and squeezing headache. At first, the severity of headache was mild. So at OPD, he was prescribed analgesic medicines such as NSAIDs and went back after being confirmed that his brain CT findings were negative. But his headache were worsening and accompanied with high fever, nausea, vomiting, and severe neck pain. So he visited emergency room 4 days after last OPD visit. His vital sign showed Body temperature 38.3°C and otherwise nonspecific. He complained of severe headache with severe motion sickness and nuchal rigidity. Emergency brain CT was done. CT revealed no definite acute brain lesion. But, in comparison with previous CT in OPD, mild ventricular dilatation was noted. In that findings, we could doubt silent SAH(CT-negative SAH) and meningoencephalitis. So CSF study was done to distinguish the possible fetal disease. The pressure of cerebrospinal fluid was 250mmHg and the color was yellowish and turbid like pus. WBC counts was 4,000/ μ m with neutrophil dominant pattern and CSF/Serum glucose ratio was 3/150(2%), which strongly suggests bacterial meningitis. Routine blood laboratory test showed WBC counts >27,000/ μ m and CRP >270mg/L, which suggests septicemia with systemic infection. Immediately, broad-spectrum antibiotics including vancomycin and 3rd generation-cephalosporin were administered intravenously and brain MRI was done in series. Brain MRI showed multifocal restricted diffusion lesions at CP angle cistern in posterior fossa and cavernous sinus, which suggest pus(empyema) in subarachnoid space. And enhance T1 imaging revealed diffuse leptomeningeal enhancement and no enhancement of left transverse sinus which suggest thrombosis of left transverse sinus. The patient was hospitalized in Intensive care unit for close monitoring of vital and neurologic signs, and treated with continuous antibiotics intravenous injection. After long term treatment, his symptoms such as headache, fever and neck pain were improved and laboratory findings were also normalized. **Conclusions or Comments:** CNS infection related CVT is fetal disorder which needs immediate aggressive treatment. Because this disease is rare, high suspicion is compulsory for immediate detection and treatment. CNS infection related CVT can originate from many cause; upper respiratory infection, previous infection like pneumonia, neurosurgical procedure, otitis media, sinusitis, previous history of skull fracture and invasion from dura mater. And like this patient, dermopuncture or phlebotomy also can result in CNS infection. So we should always consider CNS infection in patients who has any history of invasive procedure with possible source of infection related to central nerve system.

P-1-301

A case of Lemierre's Syndrome with chronic otitis media

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Background & Significance: Before the introduction of antibiotics, Lemierre's syndrome is relatively common. This disease was caused by an acute oropharyngeal infection. It is developed to secondary septic thrombophlebitis of the internal jugular vein complicated with frequent metastatic infections. It can be followed to septicemia rapidly and death within 1-2 weeks. After the widespread use of antibiotics began, the incidence of Lemierre's syndrome has decreased dramatically. **Case:** A 56 year-old-woman presented with severe headache and fever. She was diagnosed with chronic otitis media and had pus culture in her left ear 7 days ago. She was prescribed the oral ciprofloxacin 250mg three times a day and drop ear ciprofloxacin two times a day. Her headache was getting worse, nausea and vomiting has been occurred. Her body temperature was 38.5°C. Laboratory finding revealed C-reactive protein of 24.517 mg/dL. On neurologic examination, she had no neurologic deficit except neck stiffness. In CSF study, the opening pressure was 12cm H₂O. The color was turbid. The leukocyte count was 3,380/ μ l with 96% polymorphonuclear neutrophils, 4% monocytes. The protein was 149 mg/dL. The glucose was 72 mg/dL. The Blood sugar test was 152 mg/dL. She was diagnosed with bacterial meningitis and admitted to the neurologic department. We started immediately to intravenous vancomycin 500mg four times a day, ceftriaxone 2g two times a day, ampicillin 2g six times a day. On day 4 of hospitalization, in the left ear pus culture, the pseudomonas aeruginosa was grown. She complained of swelling of her Left side neck and tenderness. Neck CT showed the thrombophlebitis in the Left IJV. We diagnosed with 'Lemierre's syndrome'. We changed antibiotics to IV tazobactam/piperacillin 4.5g four times a day for targeting P. aeruginosa. We followed up the neck CT after 10 days, the amount of the thrombophlebitis was larger rather than first, unexpectedly. We started with IV heparin for thrombolysis. On day 31 of hospitalization, we confirmed by transfemoral cerebral angiography the blood flows that are connected to right IJV from left ICA through the transverse sinus and to left EJV through the left pterygoid plexus. The blood flow is well, we did not thrombectomy. On day 33 of hospitalization, she was discharged receiving the prescription of oral levofloxacin 750mg a day, oral warfarin 5mg a day. After 2 months, we confirmed that the thrombosis in the left IJV was reduced greatly. She has no neurological deficit. **Conclusions or Comments:** In 1936, Lemierre reported that Fusobacterium necrophorum is isolated from blood culture of the 20 patients who has occurred an acute oropharyngeal infection with septic thrombophlebitis of the IJV. Almost the primary infection is tonsillitis. Otitis media is rare cause of Lemierre's syndrome. Lemierre's syndrome could be suspected if there are symptoms of sore throat and submandibular swelling. Lemierre's syndrome can be diagnosed when the thrombophlebitis of IJV. The symptoms of Lemierre's syndrome usually are sore throats at first. The primary infection is spread to the surrounding tissues, and provoked to formation of IJV thrombophlebitis. It can be cause sepsis with metastatic infection. The most common metastatic target is the lungs, followed by the joints, muscle, liver, spleen, meninges. The main treatment of Lemierre's syndrome is the early administration of antibiotic, the drug of choice is metronidazole or clindamycin. The duration of antibiotics is not proven to date. The role of anticoagulant to the patient with Lemierre's syndrome is controversial. This case is suspected to be caused by P. aeruginosa. If a patient with otitis media, fever, neck swelling, tenderness were observed, Lemierre's syndrome should be discriminated by blood culture, neck image study, such as neck CT, MRI. The important of management is the early administration of antibiotics.

P-1-302

Mumps-virus-associated mild encephalopathy with a reversible splenic lesion in an adult patient

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Background & Significance: Mumps is a common childhood infection caused by the mumps virus. The hallmark of infection is swelling of the parotid gland. Aseptic meningitis and encephalitis are common complications of mumps, together with orchitis and oophoritis which can arise in adult men and women respectively. Other complications include deafness and pancreatitis. Previously, Tada et al identified clinically mild encephalitis/encephalopathy with a reversible splenic lesion (MERS) as a new type of acute encephalopathy, characterized by transient splenic lesions with high-signal intensity on diffusion-weighted magnetic resonance imaging, a mild clinical course and a good outcome. MERS has been associated with various infectious diseases. Influenza virus A and B are the most common pathogens, following by mumps virus, adenovirus, rotavirus, streptococci, and Escherichia coli. Although there are some reports of MERS associated with mumps virus infection, these cases mostly developed in childhood. We report a case of MERS arose in adult patient. **Case:** A 59-year-old female presented with febrile sense for a day. She has had upper respiratory tract infectious symptoms since about 2 weeks prior to admission. Five days before admission, she experienced headache. Three days before admission, the headache was aggravated, presented with VAS score 7 and she also had blurred vision, drowsiness, dizziness, nausea and vomiting. She had fever of 38.2 °C. On physical examination, there were no sign of parotitis, thyroiditis, pancreatitis or oophoritis. Neurological examination showed no abnormality. Initial laboratory values revealed thrombocytopenia (25,000/μL), low serum level of sodium (130 mEq/L), increased serum level of erythrocyte sedimentation rate (26 mm/hr), C-reactive protein (14.88 mg/dL), aspartate aminotransferase (193 IU/L), alanine aminotransferase (117 IU/L), alkaline phosphatase (101 IU/L), gamma guanosine triphosphate (96 U/L), and lactic acid dehydrogenase (935 IU/L). Cerebrospinal fluid study was normal. Serum mumps-specific IgM antibody by enzyme-linked immunosorbent assay was positive but CSF mumps-specific IgM antibody was negative. Diffusion-weighted and fluid-attenuated inversion recovery MR images showed a focal, ovoid high signal intensity lesion in the splenium of the corpus callosum. This lesion revealed low signal intensity on apparent diffusion coefficient. A follow-up MRI on the 4th day of admission revealed complete resolution of the splenic lesion. She was given conservative therapy and her headache, blurred vision, dizziness, nausea and vomiting were relieved. After 8 days, she was discharged without any neurological sequelae. **Conclusions or Comments:** Splenic lesion caused by mumps infection may occurred in adult. In this case, MRI findings might be able to mimic acute ischemic stroke. Follow up imaging study is crucial.

P-1-303

Fulminant bacterial meningitis with intracisternal and intraocular abscess

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Background & Significance: Acute bacterial meningitis is an acute purulent infection within the subarachnoid space. Brain abscess is a focal collection within the brain which can arise as a complication of a variety of infections. Bacterial meningitis with brain abscess can cause significant morbidity and mortality in adults. We report a case of intracisternal and intraocular abscess

associated with purulent bacterial meningitis. **Case:** A 50-year-old male was admitted to the emergency department because of altered consciousness. He had presented at a local clinic 2 weeks earlier with otitis media; at that time he was given cefixime to treat an ear infection. The blood pressure was 150/90 mmHg, respiration rate 20 and temperature 34°C. Physical examination revealed a sign of meningeal irritation and chemosis of both eyelids. Neurological examinations showed stuporous mentality, fixed eyeball and right hemiparesis (MRC grade III). Results of laboratory investigations revealed a white blood cell count of 31,450/mm³ with 96.5% polymorphonuclear cells. The C reactive protein was elevated up to 15.65mg/dl and creatine phosphokinase 1002 U/L, serum myoglobin 2542.4 ng/ml. The sodium was decreased down to 124 mEq/L. The cerebrospinal fluid (CSF) was straw with 2484 leukocytes/mm³ (90% polymorphs and 10% lymphocytes). The CSF sugar was 65mg/dl and proteins were 243.7mg/dl. Direct examination of CSF showed negative for Gram stain or Cryptococcus. The PCR analysis of CSF for Mycobacterium tuberculosis and herpes simplex virus were negative. The peripheral blood culture grew Staphylococcal epidermidis. The brain diffusion MRI showed abnormal high signal intensity on right thalamus, left internal capsule, basal cistern and both intraocular spaces. The T1 or T2-weighted MRI and FLAIR images showed abnormal signal intensity of materials on basal cistern, carotid sinus and intraocular space. Contrast-enhanced brain MRI showed abnormal enhancement of such structures. Brain MRA revealed no stenosis of carotid artery. He was initially treated with intravenous ceftriaxone and vancomycin. But, the high fever (40.2°C) and hypotension persisted and he expired on day 5 after admission due to cardiac arrest. **Conclusions or Comments:** The mortality rate of fulminant bacterial meningitis with multiple abscess is extremely high, depending on the variability in clinical presentation which may delay the early diagnosis and treatment of the disease. Multiple abscesses with bacterial meningitis and inadequate response to medical therapy require early repeated aspiration or surgical drainage.

P-1-304

A case of isolated spinal neurocysticercosis

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Background & Significance: Neurocysticercosis is caused by the larval stage of the tapeworm Taenia solium. Spinal cysticercosis is rare and isolated spinal neurocysticercosis is extremely rare. We reported a case with isolated spinal neurocysticercosis. **Case:** A 71-year-old female patient presented with pain at left shoulder and posterior neck. In her neurologic exam, pain and temperature sensation was decreased below T10 level. Also vibration sense was decreased from anterior superior iliac spine to ankle on right leg. Spine MRI revealed a cystic mass in spinal cord at T4-5 level. Spinal cystic mass lesion was single intramedullary and intradural-extramedullary cystic lesion which is well-defined round shape at T4 and T5 vertebral levels. It was hyperintense at T2WI and hypointense at T1WI. There was no abnormality at brain parenchyma. Isolated spinal neurocysticercosis was suspected. Treatment with oral praziquantel 3.6g daily was administered for 4 weeks. After antiparasitic treatment, sensory changes of pain, temperature were improved, but decreased vibration sensation was remained. **Conclusions or Comments:** Most effective treatment of spinal cysticercosis is still unknown. We have experienced and reported a case of isolated spinal cysticercosis treated with praziquantel.

P-1-305

Septic cavernous sinus thrombosis as a complications of an dental infection

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Background & Significance: Septic cavernous sinus thrombosis(CST) is a rare and fatal disease. This condition is usually secondary to infections of the paranasal sinuses or the skin around the eyes and nose. Rarely, the source appears to be odontogenic. We report a patient with septic CST along with dehiscence of right medial wall of orbit and herniation of adjacent orbital fat and medial rectus muscle after dental infection. **Case:** A 47-year-old male with hypertension and diabetes mellitus visited emergency department for 2 weeks of headache, right periorbital pain. Three weeks prior to our visit, he had periodontal abscess which had been treated by local dental clinic. All investigation at that time, including brain CT angiography, fundoscopy were normal. Ten days later, he visited emergency department again complaining of persistent severe headache, diplopia and blurred vision of right eye. Vital signs were stable without fever. On examination, there was limited movement of the right eye in all directions of gaze and right ptosis. Right pupil was dilated and sluggish to light reflex. There was mildly decreased sensation over right periorbital area. Other cranial nerve function tests were normal. The laboratory tests were normal except mild leukocytosis and elevated CRP (7.19mg/dl). Brain MRI showed enlargement of right cavernous sinus with diffusely infiltrating enhancing lesions involving cavernous sinus and dehiscence of right medial wall of orbit with herniation of adjacent orbital fat and medial rectus muscle. MR angiography demonstrated narrowing of the intracavernous segment of the internal carotid artery. With the impression of granulomatous inflammation of cavernous sinus, high doses of steroid was started. However, headache get worse and we performed CSF analysis. CSF was clear and opening pressure was 26cmH₂O. CSF analysis showed WBC 960cells/ μ l with domination of polynuclear leukocytes (89%), protein 88mg/dl and glucose 121mg/dl (the synchronous serum value was 268mg/dl). MR venography showed absence of flow within cavernous sinus. We treated with vancomycin, ceftriaxone, metronidazole and high dose methylprednisolone. At 27th hospital day, he discharged in improved state and keep following on outpatient clinic. **Conclusions or Comments:** Since septic CST is rare condition with nonspecific initial presentation, it is often missed, underestimated, or diagnosed late. In our case, correct diagnosis was not made until third imaging study and CSF analysis were performed. This case emphasizes that CST should be considered as a differential diagnosis for any patients presenting with continuous headache who have a history of recent paranasal sinusitis or odontogenic infection.

P-1-306

Multiple cranial neuropathy possibly due to invasive aspergillosis

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Background & Significance: Multiple cranial neuropathy, which are the uncommon neurologic disorder, may demand to clinicians for the broad evaluation including some life-threatening disorders. We report an immunocompromized patient with multiple cranial neuropathy possibly due to invasive aspergillosis. **Case:** A 49-year-old man presented with ptosis in the right eye and severe headache right orbitofrontal area for three days. On the initial neurologic examination, he showed complete ptosis with total limitation of adduction and supraduction in the right eye and hypesthesia on the right face. On the second hospital day, his right eye could not perceive anything even the light and revealed complete ophthalmoplegia. Brain MRI revealed mildly contour-bulging and enhanced right cavernous sinus and abnormally enhanced right optic nerve. Laboratory study showed high glucose (473mg/dl) and increased Hb A1c (14.7%). Serum markers for vasculitis, syphilis, and tumors

were all negative. Cerebrospinal fluid study showed pleocytosis; WBC 152/ μ l (Neutrophil 79%), protein 37.6 mg/dl. Bacterial, tuberculosis, and fungal stain and culture using CSF were negative. Viral marker, which were included herpes simplex, varicella zoster, cytomegalovirus, Epstein-Barr virus, were negative. Only serum aspergillosis antigen were positive. High dose IV prednisolone (1g/day), ceftriaxone, acyclovir, and amphotericin B were started. However, right facial palsy were developed on the third hospital day, and moreover, severe vertigo and vomiting with hearing loss were arised on the next day. After that day, his symptoms did not progress during two months. Follow-up brain MRI revealed that inflammatory enhance lesion spreaded out the right cerebellopontine angle, temporal bone near the geniculate ganglion, Meckel's cave, and cavernous sinus. He has treated with amphotericin B, but, did not make a change the better. **Conclusions or Comments:** Aggressive aspergillosis involving the central nervous system can be fatal especially to the immunocompromized patients. A proper antifungal agent with or without surgical therapy should be considered immediately on the suspicious case.

P-1-307

Rhino-orbito-cerebral mucormycosis presented with painful ophthalmoplegia

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Background & Significance: Orbital apex syndrome (OAS) is an uncommon clinical presentation consisting of complete ophthalmoplegia with vision loss, involving cranial nerves II, III, IV, V1 and VI. Some of these conditions, particularly among the infectious causes, can be life-threatening with delayed diagnosis and treatment. Rhino-orbito-cerebral mucormycosis (ROCM) is an invasive and often fatal fungal infection. We present a case of a successfully treated diabetic patient with OAS resulting from rhino-orbital mucormycosis who presented to the department of neurology for treatment of headache and eyeball pain. **Case:** A 59-year-old male presented to the department of neurology for severe headache, dizziness, and diplopia. Current medical history revealed poorly controlled type 2 DM. He was noted to have significantly decreased ocular motility and loss of vision in his left eye. Ophthalmologic examination revealed no light perception and complete ophthalmoplegia with ptosis in the left eye. Orbit MRI showed inflammatory change involved left optic nerve and diffuse T2 high signal intensity with enhancement in left presptal area, retromaxillary fossa, masticator space and temporalis muscle suggesting diffuse inflammatory or infectious condition. Endoscopic surgical resection was performed and a pathology sample was taken. Histopathology of tissue stains showed positivity for mucormycosis. The patient subsequently underwent aggressive endoscopic removal of the involved tissue. The patient was treated with liposomal amphotericin B 400 mg, IV, postsurgery. **Conclusions or Comments:** OAS is a common presentation of ROCM, but unfortunately blindness is permanent and the risk of mortality is increased at this stage of progression. Mucormycosis is a highly aggressive infection known to have a high mortality rate when not treated. Survival rates depend on the interval period from appearance of symptoms to onset of treatment or surgery, extent and location of the infection, type of treatment, and amount of immunosuppression. Patients with ROCM need extensive surgical and medical treatment to maximize outcomes. Success requires multidisciplinary management.

P-1-308

Unusual manifestation of tuberculous cerebral vasculitis on vessel wall MRI

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Background & Significance: Background: Ischemic strokes in 'tubercular zone' associated with vasculitis is a well-recognized complication of tuberculous meningitis (TBM). Brain magnetic resonance imaging (MRI) contributed to the diagnosis of tuberculous cerebral vasculitis (TCV) in most cases. We experienced a case of unusual manifestation of cerebral infarction caused by TCV on vessel wall MRI. **Case:** A 77 year old female patient, who has been on antituberculosis agents since 8 months ago under the impression of TBM, was presented by sudden left hemiparesis due to acute posterior internal capsule infarction. Brain MRI and contrast enhanced magnetic resonance angiography (CE MRA) showed 1.2 cm sized enhancing nodule at left sylvian cistern with compression of left middle cerebral artery (MCA) M2 segment whereas normal right MCA flow. Forty days later new right basal ganglia and left cerebral white matter infarctions were occurred. Vessel wall brain MRI CE revealed no change of enhancing mass-like lesion around left MCA 2-3 junction and new segmental wall thickening and diffuse enhancement in bilateral MCAs, posterior cerebral arteries and basilar artery, which were suggestive of cerebral vasculitis. She was started on dexamethasone in combination with four-drug treatment for TBM. **Conclusions or Comments:** Although MRI-detected abnormalities suggestive of vasculitis induce usually infarction in TBM, unusual manifestation showing initial focal concentric vessel wall thickening and enhancement followed by multisegmental cerebral vasculitis findings on vessel wall MRI in our case can cause diagnostic uncertainty. We should be well acquainted with the diverse manifestations of TCV on vessel wall MRI because urgent treatment of TCV is based on corticosteroids.

P-1-309

Prognostic factors of acute encephalitis

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Background & Objectives: Acute encephalitis is well known as a syndrome consisted of fever, mental change, focal neurologic deficit with or without convulsions. Although other infections of central nervous systems have favorable outcome, encephalitis sometimes causes high morbidity and mortality by directly involving brain parenchyma. We analyzed clinical characteristics and prognosis of patients with encephalitis to find out possible predictors of outcomes. **Method:** We retrospectively analyzed patients who were admitted and diagnosed as acute encephalitis in Chungnam National University hospital from January 2005 to June 2015. Using ANOVA, independent T-test, Pearson's chi-square test, we analyzed clinical factors (initial presenting symptoms of altered mentality or abnormal behavior or seizures, initial EEG, brain MRI abnormalities, blood chemistry, CSF findings, and etiology) which may affect outcomes (hospital days, modified Rankin scale) of acute encephalitis. **Results:** 112 patients (65 male, 47 female) were enrolled and mean age was 46 ± 18.73 years. Fourteen patients (20%) were diagnosed as viral encephalitis, 3 patients (4%) as autoimmune encephalitis, 4 patients (6%) as bacterial or fungal encephalitis. In remaining 49 patients (70%), etiology was not identified. Presence of encephalitis related brain MRI signal change was well correlated with poor outcome and longer hospital stays ($p=0.013$). Also, serum creatinine ($p<0.05$), C-reactive protein (CRP) ($p<0.01$), increased number of cerebrospinal fluid WBC ($p<0.05$) and protein ($p<0.01$) had statistically significant correlation with poor prognosis. EEG abnormality and primary presenting symptoms were not related to patient's outcome in our study. **Conclusion:** This study suggested that the brain imaging abnormality, serum creatinine, CRP, CSF WBC, and CSF protein levels have strong correlation with prognosis of acute encephalitis. Further prospective studies are needed to figure out the clinical significance of these variables.

P-1-310

A case of mild encephalopathy with a reversible splenial lesion after mumps infection

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Background & Significance: Mild encephalopathy with a reversible splenial lesion (MERS) is related to viral infection, bacterial infection, electrolyte imbalance, drug use and other conditions. Mumps virus is one of the common pathogens implicating MERS. We report a successfully treated case of mumps infection with central nervous system involvement and many other complications. **Case:** A 30-year-old man came to emergency room with sudden onset headache, parotid gland swelling which started 3 days ago. Headache was throbbing nature involving whole cranial area, and initial body temperature was elevated to 37.8°C . The initial neurologic examination was normal, except positive meningeal irritation sign. Lumbar puncture revealed elevated opening pressure with 180mmCSF and lymphocyte-dominant pleocytosis with $380/\text{mm}^3$ (lymphocyte 100%). He was admitted and insulated with an impression of mumps meningitis. Brain MR imaging with gadolinium enhancement showed high signal intensity lesion involving the splenium of the corpus callosum on T2 weighted image. Serum IgM antibody against mumps virus was positive. On hospital day 7, he complained of scrotal pain due to orchitis. On day 13, his headache and scrotal pain were relieved and discharged with no neurologic deficit. **Conclusions or Comments:** This case describes a mumps meningitis which involved the splenium of corpus callosum in an immunocompetent adult. Mumps meningitis should be suspected when a patient had a meningitis with other systemic symptoms associated with mumps infection, and corpus callosum involvement from brain MR imaging.

P-1-311

A case of Meningitis-retention syndrome : a rare complication of aseptic meningitis

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Background & Significance: Acute urinary retention is a urological emergency and common in elderly men with prostate hyperplasia. Among patients with acute urinary retention, children, young adults, and women are extremely rare. Therefore we should consider a neurologic etiology in these cases. The meningitis-retention syndrome (MRS), a combination of acute urinary retention and aseptic meningitis has not been well recognized. **Case:** A previously healthy 42-year-old man visited our emergency room (ER) with complaints of mild fever, mild headache 2 weeks ago and voiding difficulty from 10 days ago. He had visited a local clinic with these symptoms and been diagnosed as acute pyelonephritis. Even though he treated for 1 week with anti-bacterial and anti-pyretic agent, voiding difficulty was getting worse. When he came to our ER, he could not urinate on himself. Transurethral catheterization revealed 1,050mL of residual urine, and an indwelling Foley catheter was inserted into the bladder. On admission, vital signs including the blood pressure and heart rate were stable, body temperature was normal (36.6°C). Neurologic examination revealed slightly brisk deep tendon reflexes on both legs. Nuchal rigidity was not marked. Limb motor and sensory including the perineal area were normal. There was no skin lesion. Cranial nerve dysfunction and pathologic reflexes including ankle clonus, babinski's reflex were not seen. Routine laboratory tests showed no specific finding. Erythrocyte sedimentation rate was

mildly elevated (18mm/hr) and C-reactive protein was normal. Cerebrospinal fluid (CSF) examination showed mononuclear cell dominant pleocytosis (WBC 43/uL), elevated protein level (86.9 mg/dl) and normal glucose level (50 mg/dl). Viral antibody test (IgM and IgG), culture and polymerase chain reaction (PCR) of Herpes simplex virus (HSV) and Varicella zoster virus (VZV) were negative. Bacterial and AFB stain, culture and PCR were negative, too. Brain magnetic resonance image (MRI) showed no abnormal enhancing lesion. On lumbosacral spine MRI, linear enhancing lesion along the leptomeninges was observed. Nerve conduction studies and somatosensory evoked potentials on median and posterior tibial nerves were normal. We treated him conservatively with anti-pyretic agent and NSAIDs. He also took alfuzosin (alpha-1 adrenergic receptor antagonist) and bethanechol (parasympathetic muscarinic receptor agonist). On the 8th days in hospital, we removed his indwelling Foley catheter. He felt comfortable in urination, and post-voiding residual urine volume was 15mL. On the 13th days, he discharged from the hospital. His symptom was fully recovered 3 weeks later after the discharge.

Conclusions or Comments: Pathophysiology of MRS is not fully understood. However myelin basic protein (MBP) level was increased with CSF examination in recent MRS study, which is suggestive of central nervous system demyelination. From these results, it seems possible that MRS is a mild variant of acute demyelinating encephalomyelitis (ADEM), which selectively affects the lower urinary tract innervations. MRS is a rare clinical condition. But neurologist and urologist should pay attention to young adults who shows acute urinary retention with headache and fever.

P-1-312

Neurosyphilis presenting with committing arson

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Background & Significance: Neurosyphilis is an infectious disease involving the central nervous system caused by spirochete *Treponema pallidum*. Herein, we report a patient presented with acute cognitive impairment and behavioral changes, which was setting fire to the others car as the initial presentation of neurosyphilis. **Case:** A previously healthy 57-year-old man was transferred to our hospital from the Gongju Institute of Forensic Psychiatry with complain of acute memory impairment and abnormal behavior from one month ago. During the last month, he had made several attempts of setting fire to his house, but, did not seem to care all about these situation at that time. At last, he had set fire to the others car and was arrested for an arson. He had no other previous crime record. During his time in prison, he had shown memory impairment, incoherently speech, and muttering to himself. Vital signs were normal and there was no recent infection history. Any recent or remote evidence of intoxication, head trauma, or medication overuse was denied by himself and his family. Brain T2-weighted MRI revealed high signal on the bilateral medial temporal cortex. Serum venereal disease research laboratory (VDRL), *treponema pallidum* hemagglutination assay (TPHA), and the fluorescent *treponema pallidum* antibody absorption (FTA-ABS) tests were all positive. Human immunodeficiency virus (HIV) antibody test was normal. Cerebrospinal fluid (CSF) study showed leukocytosis (139 / μ l) and the rapid plasma reagin (RPR) was reactive with a titre of 1:128. He was treated for 14 days with intravenous potassium penicillin G potassium (40,000,000 units/day) every four hours. At the time of discharge, memory dysfunction and muttering were improved, but incoherency was still remained. **Conclusions or Comments:** It is difficult to diagnose neurosyphilis without suspicion and serological tests for syphilis because of its non-specific clinical and radiological manifestations. Clinicians should consider serologic testing for the disease if patients present with acute neurocognitive change. If the serologic testing for syphilis is positive, cerebrospinal fluid should be analyzed for reactive VDRL or RPR tests.

P-1-313

Serial MRI findings of slowly progressing probable Creutzfeldt-Jakob disease: case report

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Background & Significance: Creutzfeldt-Jakob disease (CJD) is one of prion diseases and is rapidly progressing, fatal, potentially transmissible dementia. Clinical diagnosis is combination of rapidly progressive dementia, myoclonus and multifocal neurological dysfunction associated with an electroencephalogram showing generalized periodic sharp wave complexes and/or a positive 14-3-3 protein test in CSF. Recently, magnetic resonance image (MRI) finding has been being important diagnostic criteria. In T2 weighted-image or fluid-attenuated inversion recovery (FLAIR), hyperintensities of basal ganglia, thalamus and cerebral cortex are observed. Hyperintensities in striatum and cortex in diffuse weighted image (DWI) are also observed. CJD is rapidly progressive. Some articles report patients with CJD will be dying within month to year of onset of illness. Finding out brain anatomic changes of these patients is difficult because of rapid progressive course. We observed follow-up MRI finding in a patient with very slowly progressing CJD patient. **Case:** A 46-year-old man presented to our hospital with lethargy, delusion of persecution, and auditory hallucinations. He had been depression and compulsive behavior for one year. In that time he washed his hands repeatedly and couldn't find his way. The clinical diagnosis of schizophrenia was initially considered and started neuropsychiatric medication. His symptom had been rapidly progressed. He had akinetic mutism and couldn't have a meal by himself in six months. At admission, he had alert mental status but neurological examination revealed impaired prosody of speech, mild tremulous movement and lead pipe rigidity in the 4-extremities. Routine blood examination and other infection tests are normal. In CSF study, there were no abnormal findings including 14-3-3 protein. In view of the unusual manifestations, MRI of the brain was done. MRI showed diffuse cortical hyperintensities indicating typical cortical "ribbon" on DWI and FLAIR, and EEG revealed periodic triphasic waveforms with background slowing. Collectively, sporadic Creutzfeldt-Jakob disease was considered. Follow-up CSF study had revealed positive of 14-3-3 protein. One-year later, patient had akinetic mutism, dysphasia and lead pipe rigidity in the 4- extremities. He loses the ability to care for himself and has a seizure attack. Follow-up MRI showed devastating brain atrophy. Diffuse cortical diffusion restriction in cerebrum has been decreased but still noted. Significant enlargement of ventricle with interstitial edema is detected. Two-year later, patient has no further progression. He still has akinetic mutism, dysphasia and lead pipe rigidity and tracheostomy and PEG insertion are done. Follow-up MRI showed interval progress severe diffuse atrophy of both cerebrum and cerebellum with passive ventriculomegaly. Three-year later, patient's symptom not changed. Follow-up MRI showed no significant changes except more progressive severe diffuse atrophy of both cerebrum and cerebellum. In serial following MRI, atrophy of optic nerve is detected and in fundus examination, optic disc atrophy is also detected. Western Blot Finding of 14-3-3 Protein showed weakly positive of patient CSF. And examination of Pathogenic prion protein (PrP^{Sc}) in Human CSF Using RT-QUIC Assay showed elevation from normal value. **Conclusions or Comments:** CJD is rapidly progressing disease. Because of this rapid progressive course, finding out long-term MRI following up is difficult. Common MRI findings in CJD are hyperintensities of basal ganglia, thalamus and cerebral cortex in T2 weighted-image or FLAIR, additionally striatum and cortex in DWI. Differences of our slowly progressing patient are there are no striatum and thalamus lesion. He has diffuse cerebral dysfunction and seizure attacks but stable vital sign. These differences may contribute to slowly progress and stable vital sign. Further follow-up MRI may be needed.

P-1-314

A case of disseminated Group B streptococcal infection with Nicolau syndrome

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Background & Significance: Group B streptococcal (GBS) infection is known as an important cause of invasive infections in neonates. Despite the decline in neonatal GBS infections, the rate of invasive GBS disease in nonpregnant adults is increasing, especially in elderly patients with underlying chronic disease. Urinary tract infection, pneumonia, and soft tissue infection are common presentations of GBS disease in adults. Nicolau syndrome (NS) is a rare cutaneous adverse reaction at the site of intramuscular (IM) injection, characterized by severe pain, erythematous patch resulting in a necrotic ulcer. We report a previously healthy woman with disseminated GBS infection and NS after IM injections. **Case:** A 60-year-old woman presented with sudden onset of altered mental status, headache and fever. She had been diagnosed with spinal stenosis and received several times of IM injections for pain relief around back and buttock a few days ago. On physical examination, auscultation of heart and lung was unremarkable and violaceous skin lesions were observed on buttock (Fig 1). Stuporous mentality and neck stiffness were noted. Other neurological examinations were unremarkable. Initial CSF analysis showed marked leukocytosis (WBC count 921/mm³) with increased total protein (245.7 mg/dL) and significantly decreased glucose (16mg/dL) level. Her diffusion-weighted brain MRI revealed diffusion restrictions at right anteroinferior cerebellum, superior vermis to superior cerebellum, left tail of hippocampus, right inferior frontal gyral cortex, right superior parietal lobular cortex and bilateral high frontoparietal cortices (Fig 2-A). Additionally, diffuse leptomeningeal enhancement along both cerebral cortical sulci was observed (Fig 2-B). These findings were compatible with meningitis and multifocal acute multi-embolic infarction. Spine MRI was insignificant. Intravenous dexamethasone and triple broad-spectrum antibiotics (ceftriaxone, vancomycin and ampicillin) were started. Three days later, streptococcus agalactiae was detected in CSF, blood and urine cultures. Her mental status was improved, but she showed intermittent confusion, mild gait ataxia and left arm weakness. And her fever was not controlled and she complained of back pain repeatedly. After about 15 days, we performed contrast enhanced abdomen-pelvic CT, lumbar spine MRI and follow-up brain MRI to find other causes of fever. The CT revealed severe pyelonephritis bilaterally and multiple abscesses of the right psoas and left gluteus maximus muscles. The spine MRI showed infectious spondylitis of lumbar 3rd-4th vertebral bodies and increased leptomeningeal enhancement. We changed the antibiotics to imipenem and ampicillin and introduced a vacuum-drainage catheter into the gluteal abscess. After then, her vital sign and general condition has been improved. After 6 weeks, follow-up brain and spine MRI showed improving state of meningitis and infectious spondylitis. **Conclusions or Comments:** We surmised that this disseminated GBS disease was attributed to the soft tissue infection at the site of IM injections. Although NS is a rare adverse reaction, it is a preventable complication. This case suggests that clinicians should be aware of this complication and take the precautions.

P-1-315

Cryptococcal meningoencephalitis associated with systemic lupus erythematosus

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Background & Significance: Cryptococcal meningoencephalitis is a rare complication of systemic lupus erythematosus (SLE). Cryptococcal meningitis in patients with systemic lupus erythematosus (SLE) can be difficult to diagnose, especially in patients receiving immunosuppressive therapy. The clinical presentation can be non-specific with a subacute onset of symptoms, which may lead to missed diagnosis or delayed treatment. We describe a patient who have been diagnosed with cryptococcal meningitis and SLE. **Case:** A 40-year-old female was admitted because of one month history of confused mentality and general weakness. She had no history of diabetes mellitus, hypertension and any other medical problem. However, she had been confirmed with SLE before 3 months ago. She had managed with immunosuppressive agents (iv steroid, iv cyclophosphamide) and has lupus nephritis as a complication of SLE. After discharging from the hospital, she had complained general weakness and poor oral intake, but she could continue her general activity of life. She had taken prednisolone orally and her general condition had aggravated. Brain magnetic resonance imaging showed multifocal acute lesions of high signal intensities. Her vital signs was normal except for body temperature. In neurologic examination at admission, she was showed confused mental status and multidirectional nystagmus. Her laboratory test including elevation of CD4/CD8 reactivity and C3, C4 activity was unremarkable. Her cerebrospinal fluid test were presented with abnormal findings (pressure 45cmH₂O, WBC 240, RBC 2, protein 106, glucose 3). Furthermore, the antigen of Cryptococcus was positive and we could find india ink preparation sign. Although we prescribed amphotericin B, her mentality and abnormal neurology had been aggravated. She had respiratory failure and we performed intubation and ventilator care but sepsis occurred to be expired. **Conclusions or Comments:** Cryptococcal meningitis should be considered in SLE patients presenting with non-specific systemic symptoms of headaches, fevers, nausea, and vomiting, particularly in those on cyclophosphamide and steroid therapy. Physicians should heighten their suspicion for cryptococcal infection in order to establish an early and aggressive treatment.

P-1-316

A case of the fulminant cerebellitis in adult

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Background & Significance: Acute cerebellitis (AC) is a rare inflammatory syndrome characterized by rapid onset of cerebellar dysfunction. It has been reported to have a variable course, from a benign to fulminant. But, those that represent a life-threatening condition are rare. Also, the majority of the cases with severe complications have been reported in the pediatric population, with extremely few adult cases. We describe the case of a 33-year man who presented with a life-threatening complication. **Case:** A 33-year-old man was admitted to the ENT department of our hospital with complaints of postural vertigo from a 1 day ago. There was no past medical or family history except a history of a flu-like prodrome from a 2-weeks ago. His neurologic examination was normal. Also, there was no remarkable findings at the initial brain magnetic resonance imaging (MRI) examination. He was diagnosed with benign paroxysmal positioning vertigo (BPPV). But, after four days of hospitalization, he began to report headache with the dizzy symptom. Follow up brain MRI revealed a mild swelling of the cerebellum with increased signal intensity in T2-weighted, suggesting of cerebellitis. He treated with acyclovir and methylprednisolone in intensive care unit (ICU). But after three days, his clinical condition deteriorated dramatically. His mental state was changed to a semi-coma and he began to show irregular respiratory pattern. Emergent brain CT revealed a complicated obstructive hydrocephalus, so ventricular catheter was promptly placed at the right lateral ventricle. But, there was no clinical improvement. He was persisted of coma state for the 11 days, relying on a

ventilator. He passed away on day 23 of hospitalization, despite all our efforts. **Conclusions or Comments:** Acute cerebellitis is a rare syndrome characterized by a sudden onset of symptoms of the cerebellar damage. Though some clinicians believe that acute cerebellitis is a self-limiting condition, but there were also the cases showing a poor outcome with rapid progression, such as our patient. Generally, MRI was the examination of choice due to a higher diagnostic accuracy. But points to be careful, normal MRI findings are also possible. Therefore, we should don't exclude the possibility of the cerebellitis, if you clinically doubt it. And a repeated radiological examination is required to establish diagnosis and to apply adequate treatment at a more earlier stage. This also highlights the importance of timely surgical interventions, as well as early diagnosis.

P-1-317

Eosinophilic meningitis associated with toxocariasis

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Background & Significance: Eosinophilic meningitis due to *Toxocara canis* comes from neural larva migrans and is a rare disease related with toxocariasis. **Case:** A 39-year-old man was admitted with febrile sensation, headache, and myalgia for 10 days. He had peripheral blood eosinophilia with eosinophilic meningitis. Abdominal and chest CT revealed multiple nodular infiltrations in liver and lung. Serological test was positive for *Toxocara canis*. His symptoms were improved after oral administration of steroids and albendazole. **Conclusions or Comments:** We report a case of eosinophilic meningitis associated with toxocariasis.

P-1-318

Bilateral optic neuropathy after influenza vaccination in Korean middle aged female

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Background & Significance: Adult onset optic neuritis is usually a monocular condition. The presentation of bilateral optic neuritis suggests a systemic cause. We present a case of bilateral optic neuritis after the influenza vaccination. **Case:** A 59 year old female presented to our department with a progressive visual loss of left eye. She had headache and pain on ocular movement. Past medical history was negative and no history of any preceding illness was reported. 10 day prior to her admission she had received inactivated influenza vaccine. Upon admission visual acuity was decreased in bilateral eye. Optic disks were swollen in both eye with congestion of retinal vein as well as blurring of disk margins. Brain MRI revealed no abnormality. After IV steroid therapy, her visual acuity was improved slowly. After 1month, her visual acuity was fully recovered. **Conclusions or Comments:** Influenza vaccination-associated optic neuropathy is a very rare condition and was not reported in South Korea. We believed that in any case of unusual bilateral optic neuritis, attention should be directed to post-vaccination neuropathy.

P-1-319

Syndrome of inappropriate antidiuretic hormone associated with herpes simplex encephalitis

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Background & Significance: The syndrome of inappropriate antidiuretic hor-

none (SIADH) is a common consequence of neurologic infection. The mechanism was known that the spread of inflammation to the hypothalamus and pituitary gland upregulates ADH secretion directly. However, we report a case of SIADH of herpes simplex encephalitis (HSE) patient without obvious inflammatory signs in the pituitary gland and hypothalamus. **Case:** A 55-year-old man was admitted with a complaint of fever, headache, nausea and allotriopia for five days. He had no any past medical history. Neurologic examination was unremarkable except anomia. Brain magnetic resonance imaging showed high-signal intensity on the T2-weighted images in the left medial temporal and insular lobes. There is no signal enhancement following gadolinium injection. The cerebrospinal fluid (CSF) study showed pleocytosis of 51 /uL leukocytes with 3% neutrophilic cells, 89% lymphocytes and 8% monocytes. The CSF protein concentration was 23.3 mg/dl and the glucose level was 143mg/dL. The herpes simplex virus (HSV) type 1 was detected by polymerase chain reaction, the patient was diagnosed as HSE. On admission, blood analysis revealed hyponatremia (124 mmol/L), decrease of plasma osmolality (262 mOsm/kg), spared secretion of urine sodium (200 mmol/L) leading to the diagnosis of SIADH. The patient had no other conditions that might have contributed to SIADH, we concluded that HSE caused SIADH. The patient treated with anti-viral agent and fluid restriction. Serum sodium level was normalized after three days and the patient improved gradually. **Conclusions or Comments:** We report a case of SIADH associated with HSE without obvious inflammatory signs in the pituitary gland and hypothalamus. SIADH might be caused by the breakdown of the limbic-hypothalamic-pituitary system rather than direct inflammation of the hypothalamus and the pituitary gland.

P-1-320

Rhabdomyolysis in anti-NMDA receptor encephalitis

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Background & Objectives: Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis is the most common type of autoimmune synaptic encephalitis and often treatable. It may cause rhabdomyolysis which is major cause of morbidity and mortality. We analyzed clinical presentation and outcome of anti-NMDAR encephalitis patients with rhabdomyolysis. **Method:** We prospectively analyzed clinical presentation of anti-NMDAR encephalitis from Jan 2013 to Mar 2015. Precipitating factor and treatment outcome of patients with rhabdomyolysis were reviewed. **Results:** Of the 19 anti-NMDAR encephalitis patients, nine were identified with elevated CK enzyme level and clinical evidence of rhabdomyolysis. Six patients had been receiving immunotherapy when CK level was elevated. Three had been administered dopamine blockers a few days before the CK level elevation and abnormal movement such as dyskinesia and dystonia got worsened at that time point. Dopamine receptor blockers were discontinued, three received urinary alkalization, one treated with continuous renal replacement therapy, two were administered with dopamine agonist, and three received benzodiazepine. Seizure was aggravated in one patient before rhabdomyolysis, but not a status epilepticus. One patient died because of multiple organ failure. **Conclusion:** Rhabdomyolysis is a common presentation in anti-NMDAR encephalitis and early detection and prevention is important. Status epilepticus is not a precipitating factor, but dopamine receptor blockers may lead rhabdomyolysis.

P-1-321

Invasive pituitary macroadenoma

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Background & Objectives: Pituitary adenomas are the most common tumors of the sellar region. They constitute approximately 10% of all intracranial tumors. They tend to have slow but severe impact on vision due to compression of the optic nerves, optic chiasm and cavernous sinus. Grossly, depending on the size they are classified as microadenomas if their diameter is <10 mm or as macro adenomas if it is >10 mm. The few reports published to date have not reported series of sufficient size to establish the characteristic clinical, pathologic, and imaging attributes of clival invasion by pituitary adenoma. Pituitary adenomas are almost always benign with no malignant potential. **Method:** A 47-year-old female presented with headache of gradual onset slowly progressive increasing in severity since 2 weeks with recent onset of diplopia and left eye ptosis of sudden onset. On advice, patient was evaluated using brain magnetic resonance imaging (MRI). MRI Brain with Contrast Non homogenous heterogeneous mass lesion measuring : 21x11x13 mm with relatively high signal intensity in T1(Gd enhancement) lesion at posterior aspect of main mass. Clivus involvement is evident by cortical breakdown, thinning. Mass lesion seen to encase both intracavernous internal carotid arteries left cavernous sinus more than right. **Results: Conclusion:** Truly aggressive pituitary tumors are uncommon, with the incidence of not more than 2%. Such tumors prove their atypical behavior by invading adjacent tissues, by proliferating rapidly. Nonsecretory pituitary tumors are called null-cell tumors measuring a few millimeters are common and found in up to 25% of autopsies. These may grow slowly, destroying normal pituitary function (hypopituitarism), or they may compress nearby structures and cause neurologic problems. The invasive nature of pituitary macroadenoma is well documented, but while invasion of the cavernous sinuses and carotid arteries and along the dura is common, invasion of the clivus is relatively rare.

P-1-322

Survey of clinical practice of north Korean defectors in neurology department: a university hospital experience

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Background & Objectives: The number of North Korean refugees who are entering South Korea has been rapidly increasing since the late 1990s, and the total in 2014 was 28,000. Although their health remains one of the most critical factors affecting the settlement of North Korean refugees, there was no report on the current clinical practice among these people in neurological field. **Method:** We analyzed the data on medical chart reviews of North Korean refugees in Chungnam National University Hospital for 2003 to 2015. **Results:** Of total 324 patients as North Korean refugees, 47 (14.5%) patients visited to the department of neurology. The patient included 38 women (age range=28-78 years, mean age±SD=49.0±12.5 years) and 9 men (age range=27-78 years, mean age±SD=50.4±17.7 years). Headache (7 tension-type headaches, 10 migraines, 3 medication overuse headaches, and 1 subarachnoid hemorrhage) and dizziness (20 vestibular migraines, 2 vestibulopathies and 1 benign paroxysmal positional vertigo) were most common symptoms. Of these, 40 (19/47) % patients also complained mental illness such as anxiety and depression. Most symptoms were resolved by medication and supportive care. Other diagnoses included 1 cerebral infarction, 2 seizure disorders, 1 cervical myelopathy, and 1 hemifacial spasm. **Conclusion:** Considering the high proportion of middle age in North Korean refugees, the headache and dizziness combined by mental illness were prominent presenting symptoms in a department of neurology. Further epidemiological study is needed for validation. Systemic

medical support based on evidence is necessary for their successful adaptation of the vulnerable minority group to South Korean Society.

P-1-323

NUDT15 R139C causes Azathioprine-induced early leukopenia in Korean patients with neurological diseases

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Background & Objectives: Azathioprine (AZT), a prescription drug commonly used to treat various neurological diseases, could be complicated by life-threatening leukopenia. This leukopenia has been associated with genetic variation in thiopurine S-methyltransferase (TPMT). And, in a recent study, NUDT15 R139C was strongly associated with AZT-induced leukopenia in Korean and Caucasian populations with inflammatory bowel disease. We performed an association study to investigate and replicate the association of both TPMT and NUDT15 variants with AZT-induced leukopenia in Korean patients with various neurological diseases. **Method:** A total of 73 patients with neurological diseases for whom AZT treatment was indicated were enrolled from multiple centers. DNA sequencing for exons of TPMT and NUDT15 was performed by the Sanger method. Clinical data for these patients were analyzed from medical records with a specific focus on leukopenia (early vs. late; early leukopenia was defined as cases in which leukopenia developed within 8 weeks of AZT therapy). **Results:** Of the 73 patients, 18 (24.4%) showed leukopenia (7 patients with early leukopenia, 11 patients with late leukopenia). Known intronic and exonic TPMT polymorphisms do not correlate with AZT-induced leukopenia in this study. Five patients developed early leukopenia with severe grade (white blood cells <1,000 mm⁻³), all of whom were risk homozygous (T/T) for NUDT15 R139C. Patients with the TT genotype were exquisitely sensitive to AZT, as compared to those with the T/C or C/C genotype, among whom, 80% and 75%, respectively, tolerated a maintenance dose (50-100 mg). The interval from onset of AZT therapy to the development of leukopenia was shorter, and the grade of observed leukopenia was more severe in patients with the T/T genotype compared to those with the C/T genotype (p=0.001, p=0.000, respectively). Patients with the C/T genotype showed a significantly high risk of early leukopenia compared to those with the C/C genotype. Overall, in our study, the association of R139C with early leukopenia was replicated in our Korean patients with various neurological diseases (OR 48.9, p=7.61 \diamond 10-5). However, we could not confirm an association with late leukopenia. The mean maintenance dose (mg) of AZT and discontinuation rate (%) were not significantly different between patients with the C/T genotype and C/C genotype (68.75±45.81 vs. 70.42±40.26; 25% vs. 20%). **Conclusion:** We verified the previously reported association of the R139C variant of NUDT15 with early leukopenia in Korean patients with various neurological diseases. Moreover, treatment with AZT should be avoided for patients with the T/T genotype. Finally, late leukopenia could not be predicted by R139C genotype.

P-1-324

lipoatrophy after local steroid injection, two cases

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Background & Objectives: Local corticosteroid injections are frequently included as treatment option in clinical guidelines in the field of musculoskeletal disorders. Adverse effects of systemic steroid medication are well known to the public, whereas the side effects of local steroid injections, were not known to even clinicians and there is a potential threat to misdiagnosis. We report two cases of localized lipoatrophy and depigmentation following local steroid injection. **Results:** A 43-year-old woman was referred to our clinic with a 4-month history of depressed skin and numbness in back of the hand. She underwent a local triamcinolone injection around the right first metacarpal joint to alleviate the pain of radial collateral ligament injury 4 months ago. Examination revealed atrophy of the skin, hypopigmentation, and hypoesthesia in the right first dorsal interosseous area (Figure 1) without weakness. An 18-year-old female visited our clinic with a 3-month history of depression in her left medial foot. Four months ago, she had been treated for a presumed plantar fasciitis with local steroid injection. Examination revealed a prominent tissue atrophy and depigmentation between navicular prominence and instep (Figure 2). Electrodiagnostic studies of the two cases showed no abnormalities. **Conclusion:** Although the incidence of soft tissue atrophy after local steroid injection is reported less than 1%, it will increase in proportion to the frequency of the procedure. Many practitioners classify these adverse effects as 'trivial' and leave out of consideration. However, even clinicians, who do not perform the injection, should be aware of the occurrence of this cosmetically disturbing side effect.

P-1-325**Unilateral isolated hypoglossal nerve palsy caused by arachnoid cyst**

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Background & Significance: Hypoglossal nerve palsy is uncommon cranial nerve palsy. Hypoglossal nerve palsy caused by compression of the cisternal segment of the hypoglossal nerve is an even rarer entity. Damage to the hypoglossal nerve produces characteristic clinical manifestations. Dysfunction of the hypoglossal nerve may be a consequence of supranuclear, nuclear, infranuclear disease. The nuclear and infranuclear hypoglossal nerve can be divided into five segments. The segmental approach to the anatomy and pathologic conditions of the hypoglossal nerve is used to clarify the differential diagnosis. We present a rare case of an arachnoid cyst causing isolated unilateral hypoglossal nerve palsy. **Case:** A Previously well 57-year-old man with no significant past or family history was referred to the neurology clinic with a 4-week history of dysarthria and dysphagia. On admission, general physical examination revealed no significant abnormal finding. The initial neurological examination showed dysarthria, right tongue deviation with associated unilateral tongue atrophy. There were no other cranial nerve palsies. The rest of his neurological examination being normal. Laboratory findings were normal except for elevated levels of triglyceride (173.3 mg/dl). Electromyography demonstrated denervational potential of the right side of the tongue. Brain magnetic resonance angiography images (MRA) revealed no remarkable findings. A gadolinium-enhanced cranial MRI scan revealed cystic lesion in cisternal segment of right hypoglossal nerve. We prescribed oral prednisolone 15mg daily, but his symptom was not improved. We recommended surgical procedure, but he refused it. **Conclusions or Comments:** The prevalence and natural history of arachnoid cysts in adults are not well defined. Prior studies of adults with arachnoid cysts have estimated cyst prevalence as between 0.3% and 1.7%. Men had a greater prevalence of arachnoid cysts than women. The most

common locations for arachnoid cysts were the middle fossa, retrocerebellar, and cerebral convexity. Tongue weakness may be due to a supranuclear, nuclear, or infranuclear lesion. Supranuclear lesions cause weakness but no atrophy, and the weakness is rarely severe. Supranuclear tongue weakness may occur with a destructive lesion of the cerebral cortex or the corticobulbar tract in the internal capsule, cerebral peduncle, or pons. Deviation of the tongue will occur away from the side of the lesion. In addition to weakness, nuclear and infranuclear lesions cause atrophy, fasciculation of the involved side. The tongue protrudes toward the weak side, which is also the side of the lesion. Hypoglossal nerve palsy usually occurs in association with involvement of lower cranial nerves (CN IX, X and XI). Isolated hypoglossal nerve paralysis is rare but has been reported to be caused by hypoglossal nerve schwannomas, dural arteriovenous fistulas, enlarged emissary veins of the hypoglossal canal, aneurysms of the stump of a persistent hypoglossal artery, occipital condyle fractures, traumatic arachnoid cysts, metastatic lesions to the skull base, and internal carotid and vertebral artery dissections. It has also followed neck surgery and has occurred with no apparent cause. Isolated unilateral hypoglossal nerve palsy caused by arachnoid cyst is very rare but detectable through appropriate imaging tools. In conclusion, arachnoid cyst in cisternal segment of hypoglossal nerve should be considered as a candidate lesion presenting tongue weakness.

P-1-326**Developmental abnormalities of the craniocervical junction resulting in Collet-Sicard syndrome**

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Background & Significance: Collet-Sicard Syndrome describes paralysis of cranial nerves IX, X, XI, and XII. Collet-Sicard syndrome is the most frequently reported neurological complication associated with Jefferson (C1) fractures. As the lateral mass of the atlas is displaced laterally away from the spinal cord toward the styloid process and the stylohyoid ligament, it impinges on cranial nerves IX, X, XI, and XII. In our report, we discuss a patient whose only initial presentation of developmental abnormalities of the craniocervical junction was most akin to a Collet-Sicard syndrome. **Case:** A 70-year-old man with a history of hypertension and left facial palsy, presented to our hospital with 2 week of difficulty articulating words and 10 days of dysphagia to solids. He had a left lower motor facial nerve palsy, a decreased gag reflex on the left, the soft palate pulling to the right, and weakness of left trapezius and sternocleidomastoid. His tongue deviated to the left. He had no Horner's syndrome. Direct laryngoscopy revealed left vocal cord paralysis. The overall clinical picture was consistent with dysfunction of the cranial nerves VII (a previous Bell's palsy), and IX to XII (a Collet-Sicard syndrome). Brain diffusion-weighted imaging was normal and brain magnetic resonance angiography showed absent right vertebral artery flow and the elongated basilar artery. Computed tomography and magnetic resonance imaging of cervical spine showed a congenital occiput-C1-C3 fusion, hypoplastic dens, and scoliosis. The left transverse processes of the atlas and axis were hypertrophic. On computed tomography, the distance between the styloid process of the skull and atlas transverse process was 3 mm left and 13 mm right. **Conclusions or Comments:** Cranial nerves IX, X, and XI exit the skull through the jugular foramen; cranial nerve XII exits through the hypoglossal foramen. After exiting the skull, the cranial nerves pass through the space between the styloid process and the transverse process of the atlas. There is normally 8-10 mm of space between them. In our patient, the distance from the styloid process to the transverse process of the atlas was only 3 mm on the left. We propose that as the left transverse process of the atlas was hypertrophic and displaced laterally toward the styloid process, it impinged on cranial nerves IX-XII. In sus-

pected Collet-Sicard syndrome, developmental abnormalities of the cranio-cervical junction should be considered in the differential diagnosis.

P-1-327

Leukoencephalomyelopathy in chlorfenapyr intoxication

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Background & Significance: Chlorfenapyr [4-bromo-2-(4-chlorophenyl)-1-(ethoxymethyl)-5-(trifluoromethyl)-1H-pyrrole-3-carbonitrile], widely used pesticide, interferes with mitochondrial oxidative phosphorylation, resulting in disruption of adenosine triphosphate (ATP) production and eventual cell death. Fatal effect of chlorfenapyr on human brain has been reported; however, chlorfenapyr intoxication presented with involvement of spinal cord has not been reported. We present a case of chlorfenapyr intoxication with involvement of encompassing brain and spinal cord. **Case:** A 52-year-old man was admitted through the emergency department because of spatiotemporal disorientation and progressive mental deterioration. He ingested 100 ml of chlorfenapyr for suicide attempt about 1 hour before arrival to the hospital. His vital signs on arrival were as follows: blood pressure 70/40 mmHg, heart rate; 98 beats/minute, respiratory rate; 22 breaths/minute and body temperature; 36°C. Laboratory tests including electrocardiography, arterial blood gas, serum calcium, and electrolyte were unremarkable. On admission, he was unresponsive to external stimuli, and showed neither focal nor lateralizing neurological deficit. Neither seizure nor tetany was observed. Electroencephalography was unremarkable, and follow up laboratory tests on 3 days after admission were unremarkable. Brain MRI revealed extensive restricted diffusion selectively on the white matter without any changes in the grey matter. Fifteen days after the admission, he showed progressive weakness of both lower extremities, prompting spine MRI study. Spine MRI revealed selective white matter abnormalities in whole levels of spinal cord. We started intravenous steroid immediately after the admission. About 10 days after the treatment, his mental state has gradually improved, and to normal over the next 20 days. However, his lower extremities weakness has not been improved, and thus he has continued to take rehabilitation treatment thereafter. **Conclusions or Comments:** The present case illustrates the first report of selective white matter damage in the spinal cord as well as brain caused by chlorfenapyr ingestion. Axons in the white matter have an extremely high-energy demand, which is responsible for maintenance of the ionic gradient and keeping the structural integrity necessary to support neurotransmission. Given that most of the motor disabilities in the mitochondrial diseases can be attributed to axonal loss or degeneration, we speculate that MRI findings of extensive white matter abnormalities are caused by vulnerability to energy deprivation, as in other mitochondrial leukoencephalopathy.

P-1-328

γ-aminobutyric acid B receptor (GABABR) antibody encephalitis misdiagnosed as post traumatic psychotic disorder after traffic accident

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Background & Significance: GABABR encephalitis is a relatively rare disease entity that occurs in lesser than 5% of autoimmune encephalitis which usually involves the typical symptoms and MRI finding of limbic encephalitis. Because the patient can represent prominent acute psychiatric symptoms, it is very difficult to distinguish autoimmune encephalitis from psychiatric disorder until

patient progress to advanced stage of the illness and frank serological or MRI evidence is apparent. We present a patient of GABABR antibody encephalitis who was initially diagnosed as post traumatic psychotic disorder. **Case:** A 58 years old man visited a local hospital with first unprovoked seizure and discharged after negative MRI and EEG results. Five days later, he had a traffic accident and he visited emergency room because of persistent amnesia and confusion after the accident. He showed agitation, insomnia, affective lability, memory impairment but there were no detectable abnormalities in the 2nd MRI and 2nd CSF analysis. The initial impression in emergency room was post-traumatic syndrome considering afferent relations between symptom onset and probable head trauma therefore the patient was transferred to a local hospital to conservative treatment. Three weeks after traffic accident, he was admitted to our hospital due to progressive neuropsychiatric deterioration including dysarthria and gait disturbance. The repeated 3rd routine CSF study including virology and bacteriology and MRI still revealed no abnormal findings. Other tumor markers and toxicology screen were negative. First and second EEG was showed no epileptiform discharge and even slowing. Because unusual course of post traumatic psychosis, we checked the paraneoplastic and autoimmune antibodies. Ten days after admission, he developed 3 times of GTC seizures. Followed up EEG showed subclinical seizures arising from right hemisphere and the 4th brain MRI demonstrated high signal intensity at T2 and FLAIR imaging in the right uncus and hippocampus consistent with limbic encephalitis. His serum was found to be positive for anti-GABABR antibodies and he was diagnosed as anti-GABABR encephalitis without hidden systemic malignancy. Because IV methyl prednisolone (1g/day for 5 days) showed only partial response, IV immunoglobulin (0.4g/kg/day for 5days) and aggressive antiepileptic management was used. Over the next 1month, he showed gradual improvement and had complete resolution from all of neuropsychiatric symptoms after 3 month later, corresponding to remarkable MRI resolution. **Conclusions or Comments:** GABAB antibody mediated limbic encephalitis is a spectrum of autoimmune encephalitis with prominent seizures and memory dysfunction. Even if initial and repeated MRI and EEG is negative, psychiatrists and neurologists should be concerned about the possibility of autoimmune encephalitis in any patient who develops new onset neuropsychiatric symptoms and whom etiology has not been confirmed.

P-1-329

Can we diagnose recurrent attack of acute intermittent porphyria (AIP) only by measuring delta-aminolevulinic acid (ALA) and porphobilinogen (PBG)?

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Background & Significance: The porphyria is a group of disease affecting the haem synthesis pathway. Acute intermittent porphyria(AIP) is caused by deficiency of hydroxyl- methylbilane and delta-aminolevulinic acid(ALA) and porphobilinogen (PBG) are elevated in urine. In n patient with abdominal pain, peripheral polyneuropathy, psychosis and epileptic seizure, elevation of delta-ALA and PBG in 24 hour urine collection is helpful in diagnosing AIP. However, delta-ALA and PBG remains significantly high for many years after acute attack of AIP. So demonstration of elevated level of delta-ALA and PBS does not mean the recurrent attack of AIP. To diagnose a recurrence, clinical signs as abdominal pain or exacerbation of peripheral polyneuropathy can be used for detection. We experienced a patient with AIP, who complained aggravation of her abdominal pain which was improved significantly after normosang[®] treatment. **Case:** On november 2014, 37 year old woman developed abdominal pain and quadriparesis which prevented walking alone. At that time, she got only symptomatic treatment with morphine and other pain kill-

ers at other university hospital. She visited our clinic at December 17th 2014. On neurological examination, she showed quadriplegia with distal predominance, paresthesia below bilateral inguinal area and generalized areflexia. She complained pain at her abdomen and lower extremities. The concentration of delta-ALA and PBG in 24 hour urine collection was elevated up to 21.3mg/day (normal : 0-7.5mg/day) and 77.6mg/day(normal 0-2.5mg/day) respectively. For that time, we also did symptomatic treatment for her abdominal and extremity pain. Later part of June 2015, she was admitted to our neurology department due to recent aggravation of pain of her whole abdomen and lower extremities. We elevated the dose of general analgesic but they did not work. The concentration of delta- ALA and PBG in urine (29.4mg and 51.4mg respectively) did not show any significant interval change comparing to previous exam. Because of lack of efficiency of many pain medication, we treated her with Normosang®(3mg/kg for 4 days) Then, her pain was dramatically improved even though previous pain medication was maintained. **Conclusions or Comments:** As delta-ALA and PBG can be elevated for many years after acute attack of AIP, we have to consider clinical situation such as aggravation of abdominal pain or weakness to decide whether recurrence of AIP occurs or not rather than depending on elevated delta ALA and PBG levels. Further study regarding why urine delta ALA and PBG levels are elevated long after acute porphyria event could help us understand the mechanisms of acute recurrence.

P-1-330

A case of Brainstem astrocytoma presenting with persistent hiccups

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Background & Significance: Hiccup (singultus) is a poorly understood phenomenon. An involuntarily powerful spasm of the diaphragm, followed by a sudden inspiration with a closure of the glottis. Usually hiccups terminate spontaneously within hours and these brief episodes in adults are usually benign and self-limiting. However, Persistent or intractable episodes are more likely to result from serious pathologic processes. Here we present a case of persistent hiccups that was caused by brainstem tumor. **Case:** A 18 year-old-man visited neurology department for evaluation of headache and intractable hiccups lasting more than 6 months. The patient had contractions at the rate of one every few seconds. He take metoclopramide 100 mg and undergone tongue retraction treatment for hiccup but there were no effect. He had no medical history except viral meningitis for 5 years ago. His all laboratory tests including metabolic and hematological tests showed normal results. General examination was normal. In neurological examination, cranial nerve involvement was not detected. Swallowing and speech were normal. However, we found impaired tandem gait and positive Romberg test. At first, we suspected possibility of medulla oblongata involvement such as space occupying lesion, brainstem encephalitis, or demyelinating disease. And then we performed Brain MRI. The MRI study showed 2.3cm hemorrhagic non-enhancing mass of foramen magnum with vascular pedicle in inferior aspect. The MRI showed low signal intensity in T1 and T2. And severe edematous change of cervicomedullary junction of spinal cord and medulla. And then, we consulted for Neurosurgical department for management of this case. They recommended the surgical dissection. The patient undergone surgical treatment and his clinical condition was resolved. Biopsy of the tumor showed low grade astrocytic tumor, suggestive of pilocytic astrocytoma. Now, the patient is in good clinical condition without a relapse and complication. **Conclusions or Comments:** Hiccups often are associated with underlying organic disease and often induce social and emotional distress. It is typically not associated with any particular disease. In contrast, intractable hiccup is an uncommon, chronic, and incapacitating disturbance defined as hiccup bouts lasting more than

48 hours or recurring despite various treatments. Patients with chronic hiccups may face a variety of conditions, such as dehydration, insomnia, depression, gastroenteric disorders, such as gastroesophageal reflux, and even death. It may occur as a component of the lateral medullary syndrome, masses in the posterior fossa or medulla, and occasionally with generalized elevation of intracranial pressure, brainstem encephalitis, or with metabolic encephalopathies such as uremia. Medulla oblongata has a central role in this reflex and lesions in this site could cause hiccup, usually persistent and intractable. A neural center for hiccupping is now considered to exist in the reticular formation of the medulla oblongata. In this case, the possibility of underlying lesions in the medulla oblongata should be evaluated by MRI in patients with intractable hiccups, even if the patients do not reveal other central nervous system manifestations.

P-1-331

Gliomatosis cerebri mimicking Herpes Simplex Encephalitis

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Background & Significance: Gliomatosis cerebri (GC) is rare primary brain tumor characterized by diffuse infiltration of neoplastic glial cells that typically involve multiple brain areas. Magnetic resonance imaging (MRI) is the gold standard method for the diagnosis of GC. However, it can be difficult to distinguish GC from herpes simplex encephalitis (HSE), if they are presented as the temporal lobe lesion with acute encephalitic symptoms. We report a case of GC, which mimicked the clinicoradiological course of HSE. **Case:** A 54-year-old man, presented with mild headache, drowsy mental status and loss of consciousness, was admitted in other hospital. Initial brain MRI showed high-signal intensity on the T2-weighted sequence in the left frontal, temporal and insular lobes, suggestive of HSE. The patient was diagnosed with HSE and received acyclovir treatment. The patient recovered gradually. At four months later, the patient visited our hospital due to aggravated symptoms, including dysarthria, anomia and right hemiparesis. Brain MRI showed that the lesion had increased in size with more edema around lesion in the left frontal, temporal and insular lobes. Irregular enhancement was observed, and hemorrhagic transformation is combined. Magnetic resonance spectroscopy showed increased choline/creatine and Cho/N-acetylaspartate ratios, suggestive of glioma. However, the cerebrospinal fluid (CSF) study showed pleocytosis of 114 /uL leukocytes with 44% neutrophilic cells, 46% lymphocytes and 10% monocytes. The CSF protein concentration was increased (117 mg/dl), and the glucose level was 47mg/dL. CSF gram stain, bacterial and viral cultures and HSV PCR were negative. Brain biopsy was performed for differential diagnosis between GC and HSE, and the pathological diagnosis was malignant glioma with marked atypia. **Conclusions or Comments:** It is important to differentiate GC from HSE in patients who have temporal lobe lesions, even in patient with CSF pleocytosis. Brain biopsy should be considered in these cases.

P-1-332

Primary diffuse large B-cell lymphoma of the choroid plexus

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Department of Neurology, Yonsei University College of Medicine

Background & Significance: Primary central nervous system lymphoma (PCNSL) is defined as extranodal non-Hodgkin lymphoma located in the craniospinal axis without the evidence of a systemic primary tumor. The most frequent locations are frontal lobes, basal ganglia, and corpus callosum at presentation. PCNSL lesions may be divided into parenchymal, subependymal

and leptomeningeal, also with the possibility of a ventricular involvement (2-8.6%) nearly always secondary to an extension from a subependymal location. Primary ventricular lymphomas are extremely rare and a choroid plexus origin is even rarer. Here we describe a case of a primary lymphoma (diffuse large B-cell type) of the choroid plexus. **Case:** A 79 year-old female patient presented with headache, short-term memory loss, and drowsy mental status over one month. She did not report any previous cognitive impairment and weight loss recently. Neurologic exam showed drowsy mental status and severely impaired short term memory, but other cranial nerve function, motor, and sensory exam were intact. In CSF study, CSF opening pressure was 120mmCSF and revealed elevated WBC count (252/ μ L) and protein (2100mg/dL). CSF Glucose was 41mg/dL (serum glucose 101mg/dL). Her Brain MRI showed enhancing choroid plexus mass in bilateral lateral ventricle and edema in bilateral medial temporal lobes, mimicking ventriculitis. Repeated CSF cytology did not show any malignancy, and bacterial, tuberculosis, fungal culture showed negative results. We started to treat her with high dose dexamethasone, 20mg per day. To cover cryptococcus and tuberculosis which were the most frequent pathogen of choroid plexitis, anti-tuberculous agents and amphotericin B were given. But, her clinical symptoms were gradually worsening, and brain imaging revealed the size of mass increased, and the mass infiltrated into right thalamus and basal ganglia. Invasive brain biopsy was done, and it finally confirms diffuse large B cell lymphoma, primary on central lesion. **Conclusions or Comments:** Primary choroid plexus lymphoma is a very rare tumor that should be included in the differential diagnosis of intraventricular neoplasms.

P-1-333

Parkinson's disease in a patient with Klinefelter's syndrome

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background & Significance: Klinefelter syndrome(KS) is the most common sex chromosomal disorder. The prevalence is 1/600 male live births and it is a frequent cause of hypogonadism and infertility. Patients with KS may present with several neurological symptoms including tremor, slow motor development and cognitive dysfunction. However, parkinsonism has been rarely reported in KS. We report a case of Parkinson's disease(PD) in a patient with KS. **Case:** A 60-year-old man presented with 1-year history of bradykinesia. He was previously diagnosed with Klinefelter syndrome, karyotype 47 XXY and had no family history of parkinsonism. On neurological exam, he had a masked face. His speed and amplitude of finger and foot tapping were reduced on both sides and the reduction was prominent on left side. Cogwheel rigidity was found on both sides and resting tremor was absent. Results of muscle power and sensory exams were normal. No abnormalities were observed on cerebellar function tests. His posture was mildly stooped. He walked with a widened base, and reduced stride and cadence. On pull tests, he displayed no postural instability. Unified Parkinson's Disease Rating Scale part III total motor score was 25 and Hoehn and Yahr stage was 2. Brain MRI showed several periventricular non-specific high signal intensities on a T2-weighted image. [18F]-FP-CIT PET studies revealed decreased striatal uptake in both hemispheres, more prominent in the putamen. Mini-mental state examination was 21/30 and global deterioration scale was 3. Neuropsychological tests revealed that verbal and visual memory, confrontational naming, generative naming, visuospatial function and inhibitory control were impaired. Bradykinesia and gait were improved with pramipexole and rasagiline. **Conclusions or Comments:** This is the first CIT-PET proven case of sporadic PD in a patient with KS. The development of PD in KS might be coincidental. However, there's a possible explanation for the development of PD in terms of melatonin and its effect on neuronal cells. Alteration of melatonin secretion is uncertain in KS. It is con-

sidered that low levels of testosterone influences the homeostasis of melatonin and gonadotropins. Significant evidence exists regarding melatonin's neuro-protective role in animal models of PD and KS. Overproduction of reactive oxygen and nitrogen causes oxidative cell damage and oxidative stress and free radicals are involved in dopaminergic neurodegeneration in PD. Melatonin was found to have antioxidant functions by scavenging free radicals and increasing antioxidant enzyme gene expression, cellular antiapoptotic effects and anti-inflammatory action by preventing microglial activation. Melatonin also normalizes mitochondrial metabolism in dopaminergic neurons resulting in a decrease in oxidative stress and maintenance of cell function and survival. Thus, melatonin prevents dopaminergic neuronal cell death by blocking different apoptotic pathways and reduces disease burden in animal models of PD. The relationship between melatonin and parkinsonism has not been thoroughly investigated in humans. Down-regulation of melatonin receptors in the substantia nigra was found in an autopsy study of patients with PD. Since melatonin is considered a potent endogenous antioxidant, decreased expression of melatonin receptors might be involved in PD pathogenesis. In a previous case, a 27-year-old KS patient was diagnosed with early-onset familial PD. However, genetic analysis tests for PD and other diseases were normal. The authors suggested his early symptoms might be related to reduced melatonin concentration. As our patient was older, neurodegenerative changes might be more involved in the disease progression. However, we hypothesized that the reduced melatonin secretion might also cause oxidative stress and apoptosis in the nigral dopaminergic neuronal cells, resulting in parkinsonism. In summary, we report a rare case of development of parkinsonism in an older patient with KS. Further studies investigating the link between melatonin and pathophysiology of PD are needed.



2015년 대한신경과학회 제34차 추계학술대회

- Poster Presentation II -

【Poster Presentation 좌장명】

Stroke VII	P-2-1~P-2-12	이경복(순천향의대)
Stroke VIII	P-2-13~P-2-24	서우근(고려의대)
Stroke IX	P-2-25t~P-2-36	유성욱(고려의대)
Stroke X	P-2-37~P-2-48	한시령(가톨릭의대)
Stroke XI	P-2-49~P-2-60	최재철(제주의대)
Stroke XII	P-2-61~P-2-72	정근화(서울의대)
Stroke XIII	P-2-73~P-2-84	박만석(전남의대)
Dementia V	P-2-85~P-2-96	양영순(보훈공단 중앙보훈병원)
Dementia VI	P-2-97~P-2-108	심용수(가톨릭의대)
Dementia VII	P-2-109~P-2-120	서상원(성균관의대)
Epilepsy III	P-2-121~P-2-131	이서영(강원의대)
Epilepsy IV	P-2-132~P-2-141	이은미(울산의대)
Headache III	P-2-142~P-2-152	정필욱(성균관의대)
Movement IV	P-2-153~P-2-163	송인욱(가톨릭의대)
Movement V	P-2-164~P-2-175	안태범(경희의대)
Movement VI	P-2-176~P-2-186	김상진(인제의대)
Movement VII	P-2-187~P-2-197	마효일(한림의대)
MS/NMO II	P-2-198~P-2-210	김우준(가톨릭의대)
NO II	P-2-211~P-2-222	최정윤(고려의대)
NO III	P-2-223~P-2-234	이학승(원광의대)
Muscle and Nerve VI	P-2-235~P-2-246	나상준(건양의대)
Muscle and Nerve VII	P-2-247~P-2-257	남태승(전남의대)
Muscle and Nerve VIII	P-2-258~P-2-268	박기종(경상의대)
Muscle and Nerve IX	P-2-269~P-2-279	강사윤(제주의대)
Sleep II	P-2-280~P-2-334	김광기(동국의대)
Neuroscience II	P-2-290~P-2-299	고성호(한양의대)
Neurophysiology	P-2-300~P-2-309	한선정(원광의대)
Infection III	P-2-310~P-2-319	김영수(성균관의대)
Systemic involvement	P-2-320~P-2-333	조중양(인제의대)

Poster Presentation II

• 시간: 13:00~14:10

• 장소: 컨벤션센터 1st Floor

P-2-1

Long-term follow up results after carotid and vertebral artery stent placement (CVAS)

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Background & Objectives: Carotid endarterectomy (CEA) is the preferred surgical procedure over carotid and vertebral artery stent placement (CVAS) for the older population due to the commonly believing less side effects. Carotid endarterectomy (CEA) is performed under general anesthesia in operating room. Due to the risk factors of general anesthesia, it can potentially be harmful to the older population which include in increasing patient recovery time, leaving a surgical scar, and hospitalization. Carotid and vertebral artery stent placement (CVAS) is also critical surgical method if anything was to happen during the procedure and the patient may die. We monitored registered patients at Yonsei Stroke Network who received carotid and vertebral artery stent placement (CVAS) for at least four to nine years. We monitored mortality rate of each patient within a month post carotid and vertebral artery stent placement (CVAS) and carotid and vertebral artery stent (CVAS) placement related symptoms such as re-stroke and restenosis. Post carotid artery stent placement (CAS) maintain with dual anti-platelet therapy to follow current percutaneous transluminal coronary angioplasty (PTCA) guideline, but since there is no specific carotid vertebral stent (CVS) guideline for post carotid artery stent placement (CAS) care is available, we treated patient with single anti-platelet therapy and statin. **Method:** In the prospective analysis of 51 patients who had more than 70% carotid stenosis required carotid and vertebral artery stent placement (CASP) between 2006 and 2012, we classified patients with age, gender, past medical history, occupation, and region. To find the most up to date patients' current health status, we conducted continuous medical observations to find three to nine years of mortality rate due to cardiovascular accident (CVA), restenosis, and occurrence of symptoms as re-stroke. Activities of daily living (ADL) measured by Katz Index of Independence in Activities of Daily Living assessment tool. The number of anti-platelet resistances was checked by VerifyNow in Ex Vivo and platelet leukocytes activation and aggregation were checked by Fluorescence-Activated Cell Sorter Scan (FACS). **Results:** A total of randomized 51 patients who underwent carotid and vertebral artery stent placement (CVAS) consisted of 43 male (84.31%) and 8 female (15.69%). Randomized patient population's mean age was 65.82 and SD was 8.08 and we calculated patient age as when they admitted to Severance Hospital at Yonsei University. Total of 18 patients (35.29%) had previous cardiovascular accident (CVA) and 36 patients (70.59%) had hypertension (HTN) and 12 patients (23.53%) had diabetes mellitus (DM). Total of 28 patients (54.90%) had history of smoking and 10 patients (19.61%) had history of drinking. **Conclusion:** After the carotid artery and vertebral stent placement (CVAS), no restenosis was observed in our patient population and none of our patients had undergone replacement of carotid and vertebral artery stent placement (CVAS). There was no significantly increased platelet leukocyte interaction observed in our patient group. Carotid and vertebral stent placement may safe and effective treatment of symptomatic stenosis patients at any age group.

P-2-2

Association between serum alkaline phosphatase level and cerebral small vessel disease

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Background & Objectives: Alkaline phosphatase (ALP) promotes vascular calcification by inhibiting organic pyrophosphate. Serum ALP level is a marker of vascular calcification, which plays a role in the development of atherosclerosis. Several epidemiologic studies reported that serum ALP level is associated with an increase in cardiovascular events, and predicts poor functional outcome and mortality in ischemic stroke. We evaluated whether serum alkaline phosphatase (ALP) is associated with cerebral small vessel disease (cSVD) and cerebral arterial stenosis (CAS). **Method:** The present study had a hospital-based, cross-sectional design. Assessment of vascular risk factors and brain magnetic resonance image (MRI) were analyzed in 1,011 neurologically healthy subjects. On brain MRI, the presence of silent lacunar infarction (SLI, lacunar lesion ≤ 15 mm in diameter) and overt cerebral white matter hyperintensities (cWMH, lesion with Fazekas scale score ≥ 2) were evaluated as indices of cSVD. On brain MR angiography, the presence of extracranial arterial stenosis (ECAS, stenosis $\geq 50\%$ in extracranial arteries) and intracranial arterial stenosis (ICAS, stenosis $\geq 50\%$ in intracranial arteries) were evaluated as indices of CAS. Serum ALP levels were measured by the p-nitrophenyl phosphate and diethanolamine method. **Results:** The prevalence of SLI and overt cWMH was 11.9% and 29.1%, respectively. The prevalence of CAS, ICAS, and ECAS was 19.1%, 10.0%, and 11.7%, respectively. Univariate analysis showed that the 3rdALP tertile group was associated with the female gender, higher WBC and platelet counts, higher triglyceride levels, the presence of SLI, and overt cWMH compared with the 1stALP tertile group. The presence of CAS, ICAS, and ECAS was not different across serum ALP tertiles. The multivariate analysis indicated that the presence of SLI (odds ratio [OR]: 2.09; 95% confidence interval [CI]: 1.27-3.42; $p = 0.004$) and overt cWMH (OR: 1.48; 95% CI: 1.03-2.13, $p = 0.036$) were prevalent in the 3rd ALP tertile group compared with the 1st ALP tertile group after adjusting cardiovascular risk factors. The mean serum ALP level was significantly higher in the presence of SLI and overt cWMH. In the multivariate model with adjustments for confounding factors, the statistical significance of serum ALP level remained when the presence of SLI (OR: 1.05 per 10 IU/L increase in ALP; 95% CI: 1.02-1.08; $p = 0.003$) and overt cWMH (OR: 1.03 per 10 IU/L increase in ALP; 95% CI: 1.00-1.06; $p = 0.025$) were added to the model. In the spline curve based on a generalized additive model, we observed a significant positive association between ALP level and the probability of SLI and overt cWMH. **Conclusion:** Our study found a positive association between serum ALP and indices of cSVD. No association was found between serum ALP and indices of CAS. Further studies are needed to clarify the pathomechanism of ALP in the development of cSVD.

P-2-3

Different risk factors between intracranial and extracranial arterial stenosis in ischemic stroke

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Background & Objectives: Intracranial or extracranial arterial atherosclerosis is an important cause of ischemic stroke. Because intra- and extracranial arteries have different structures and hemodynamics, there are many reports which suggest that risk factors between intra- and extracranial stenosis are dissimilar. However, the results are inconclusive. So we investigated the different association between cervicocerebral arterial stenosis and traditional vascular markers, radiologic markers including cerebral microbleed (CMB), white matter hyperintensities (WMH) and silent brain infarct (SBI), and blood markers by location of the stenosis. **Method:** Total 2,535 patients, with acute ischemic stroke admitted to our stroke center within 7 days after symptom onset, were enrolled from January 2002 to September 2010. We excluded patients with cardioembolic stroke (n = 607), no available MR or CT angiography (n = 90), no significant arterial stenosis (<50%) (n = 767), and no available brain MRI (n = 10). Arterial stenosis degree was evaluated using MR or CT angiography by trained neurologists. Patients were divided into four groups, intracranial stenosis (ICAS) group, extracranial stenosis (ECAS) group, combined intra- and extracranial stenosis (COMS) group, and isolated PCAS (posterior circulation arterial stenosis) group. We defined ICAS as $\geq 50\%$ stenosis or occlusion of the large intracranial vessels (proximal portions of the middle cerebral artery, anterior cerebral artery, posterior cerebral artery; intracranial portion of the internal carotid artery; or basilar artery). ECAS was defined as $\geq 50\%$ stenosis or occlusion of extracranial vessels (extracranial portions of the internal carotid artery, vertebral artery, and common carotid artery). PCAS was defined as $\geq 50\%$ stenosis or occlusion of posterior cerebral artery or vertebral artery or basilar artery. **Results:** Patients' baseline characteristics are presented in Table 1. Heart disease, higher BMI, and CMB were significantly associated with ICAS compared with to ECAS. Old age, female sex, and smoking were prevalent in ECAS group rather than ICAS group. In subgroup analysis, except COMS and isolated PCAS group, the multivariate logistic regression analysis showed CMB had significant association with ICAS [adjusted OR, 2.162; 95% CI, 1.282–3.646]. **Conclusion:** In conclusion, our study demonstrated that intra- and extracranial stenosis have different risk factors and radiologic markers, especially CMB. This finding suggests that ICAS may be related to not atherosclerotic plaque in ECAS, but endothelial dysfunction which is major cause of CMB in the aspect of pathogenesis.

P-2-4

Does sleep apnea cause ischemic stroke during sleep?

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Background & Objectives: Current literature indicates that the sleep breathing disorder (SBD) increases a risk of cardiovascular disease including ischemic stroke. It has been suggested that mechanisms related to ischemic stroke include hypoxia, hemodynamic changes, hypercoagulability, paradoxical embolization, carotid atherosclerosis and secondary effects of hypertension or cardiac disease. In this study, we examined whether preexisting SBD affects the onset of an acute stroke. **Method:** We prospectively investigated patients consecutively who were admitted with acute ischemic stroke from October 2013 to March 2015. We collected data on symptoms suggesting SBDs during the month preceding the stroke using the Korean version of the Berlin questionnaire taken from the caregiver or the patient. Stroke data including time of onset, demographics, risk factors, and subtypes were also collected. Based on the time of onset, strokes were classified as wake-up stroke and non-wake-up stroke. Logistic regression analysis was used to determine the factors related with strokes upon awakening. **Results:** We identified 277 ischemic strokes which presented to the ER, 63 (22.7%) of which were strokes upon awakening.

A prior history of observed or self-recognized sleep apnea was the only risk factor of strokes upon awakening based on a univariate analysis (odds ratio 2.144; 95% confidence interval 1.069-4.303). Multivariate analysis showed that a prior history of apnea was associated with wake-up stroke (odds ratio 2.346; 95% confidence interval 1.110-4.958), whereas atrial fibrillation was negatively associated with stroke wake-up stroke (odds ratio 0.326; 95% confidence interval 0.117- 0.904). **Conclusion:** SBD was associated with occurrence of ischemic stroke during sleep. This suggests that SBD may contribute to ischemic stroke not only as a predisposing risk factor but also at the onset of the stroke. Direct injury to the brain such as hypoxia, hemodynamic changes, hypercoagulability, paradoxical embolization, and plaque disruption associated with vibration may be a plausible mechanism for SBD-associated acute ischemic stroke.

P-2-5

Two cases of endovascular treatment in stenosis of cavernous- petrous segment of ICA

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Background & Significance: Procedural safety and high rates of in-stent recurrent stenosis lesion remain a concern in the endovascular treatment of curvature lesion of ICA. In this cases, we performed with PTA(percutaneous transluminal angioplasty) for stenosis of petrous-cavernous ICA. **Case:** Case 1) A 80-year-old man presented to the emergency room with a suddenly developed altered mentality and right hemiparesis for three hours. He had hypertension, diabetes mellitus and atrial fibrillation. Brain MR imaging showed a left MCA infarction and ICA occlusion. Recombinant tissue plasminogen activator(rt-PA 0.6mg/kg) was administered intravenously. Cerebral angiography revealed an occlusion of petrous-cavernous junction of left ICA. The lesion was recanalized by solitaire. But residual stenosis was persisted at petrous-cavernous junction portion. This lesion was treated with PTA and intra-arterial infusion of tirofiban. After 30 minutes, follow up angiography demonstrated a widely patent petrous-cavernous junction portion of the ICA. On 2 days, mentality and right hemiparesis was much improved. Case 2) A 71-year-old man visited with dysarthria and left hemiparesis for 3 days. He had a hypertension and hyperlipidemia. Brain MR imaging showed a right corona radiata infarction and focal stenosis of posterior genu portion of right cavernous ICA. We treated with clopidogrel and atrovastatin. After 2 months, follow up MR angiography showed that a stenosis of cavernous segment deteriorated. The PTA was performed without stent. Follow up CT angiography performed at 1 year, PTA placement showed successful dilatation with no new neurologic deficit. **Conclusions or Comments:** In cases, PTA is effective for stenosis of curvature portion of petrous-cavernous ICA. We suggested that PTA may be effective treatment option in stenosis of curvature portion of ICA.

P-2-6

A case of recurrent limb-shaking transient ischemic attack improved after contralateral carotid artery stenting

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Background & Significance: Limb shaking transient ischemic attacks (TIAs) is a rare condition presenting with a hyperkinetic involuntary movement, which caused by flow insufficiency at the vascular territory and exhausted vascular reactivity. Therefore the aim of treatment usually focuses on restoring the cere-

bral blood flow. We report a case of recurrent limb shaking TIA caused by ipsilateral carotid occlusion and improved after carotid artery stenting for contralateral internal carotid artery (ICA). **Case:** A 78-year-old man presented emergency room (ER) with recurrent episodes of involuntary movement of left side limbs lasting for less than 10minutes each in duration. Involuntary movement was characterized vigorous, irregular, wide-amplitude movement involving left side proximal arm and leg which seemed like hemiballismus. In ER, symptom did not appear anymore. Brain MR was done and there were no acute ischemic lesion in diffusion weighted image. MR angiography showed occlusion of right proximal ICA and severe stenosis in left proximal ICA. Electroencephalography (EEG) was normal. The serum glucose level and other laboratory results were also normal. A few days later, he revisited ER with recurrent episodes of involuntary movement, which were similar with previous episodes. Brain SPECT showed significant flow decrease in right frontal and parietal lobe. On transfemoral cerebral angiography, Rt proximal ICA was completely occluded and the flow of right anterior circulation was mainly supplied by contralateral anterior circulation via A-com. Balloon angioplasty and stenting was undergone for severe stenosis of left ICA. The patient discharged without any neurologic deficit. No further event of involuntary movement occurred for 3- month follow up period. **Conclusions or Comments:** For this patient, ipsilateral carotid occlusion and contralateral carotid severe stenosis both contributed to development of limb-shaking TIAs. Aggressive intervention for the contralateral carotid stenosis resulted in restoring cerebral blood flow to the vulnerable region seems to prevent further occurrence of involuntary movement.

P-2-7

Cortical subarachnoid hemorrhage and acute ischemic stroke associated with intracranial atherosclerosis

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Background & Significance: Cortical subarachnoid hemorrhage (cSAH) is an uncommon form of the subarachnoid hemorrhage (SAH) localized along the convexity of brain. The etiology of cSAH have been reported including pial arteriovenous malformation, dural arteriovenous fistula, arterial dissection, cortical venous thrombosis, vacuities, reversible cerebral vasoconstriction syndrome, posterior reversible leukoencephalopathy syndrome, endocarditis, cerebral amyloid angiopathy, coagulation disorder, abscess, cavernoma, brain tumor, and high-graded extracranial stenosis. We describe a rare case of cSAH with acute ischemic stroke because of intracranial stenosis and discuss the potential mechanism. **Case:** The 45-year-old male suffered sudden onset thunderclap headache without neurologic deficit. The initial blood pressure was 200/100 mmHg. He had a history of hypertension, but didn't take medication regularly. He was a 30PY current smoker. There was no another past medical history, or familial history. Noncontrast computed tomography scan was performed to rule out secondary headache, and showed a left frontal cSAH and multiple low intensity lesions in left fronto-temporo-parietal area. Magnetic resonance image also revealed a left frontal cSAH and multiple acute ischemic lesions in left fronto-temporo-parietal lobes. Magnetic resonance angiogram demonstrated severe stenosis of left middle cerebral artery To exclude vascular malformation, he underwent digital subtraction angiography(DSA). DSA revealed moderate to severe (about 69%) stenosis at superior division of left MCA without any vascular malformation. Routine lab tests including coagulation function were normal. Additional lab test including protein S, protein C, antithrombin III, and anti-cardiolipin antibody were also normal. He was treated with aspirin and his symptoms resolved in 3days. **Conclusions or Comments:** We report a case with the cortical SAH concomitant with acute

stroke. Severe atherosclerotic carotid disease is associated with cSAH, and rarely caused cSAH concurrent to acute ischemic infarction. In our case, cSAH developed concomitant with acute ischemia due to intracranial artery stenosis. One retrospective study demonstrated that cSAH was observed in 0.14% (8/4953 cases) of acute ischemic stroke patients. Among eight patients, extracranial atherosclerotic disease was detected in four patients, and only one patient had intracranial artery occlusion. While cSAH have been reported with many different etiologies, in cases of cSAH concurrent with ischemic stroke, arterial occlusive disease was the most frequent etiology. Several explanations have been proposed for the association between carotid stenosis and cSAH. Hemodynamic stress may damage dilated fragile pial collateral vasculature and rupture of pial vessels produce cSAH. In our case, possible mechanism of ischemic infarction is arterial embolization from MCA stenosis. Patterns of acute infarction may provide a clue to the mechanism of cSAH. Small emboli occlude blood vessels close to the cortex, which subsequently may recanalize spontaneously, and reperfusion injury lead to cSAH.

P-2-8

A case report: the effect of valproate on dystonic movement of women who suffer from moyamoya disease

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Background & Significance: Moyamoya disease (MMD) is an uncommon cerebrovascular disorder that is characterized by progressive occlusion of the supraclinoid internal carotid artery (ICA) and its main branches within the circle of Willis. A common symptom of moyamoya disease is usually ischemic episode in children and intracranial hemorrhage in adults. We report the rare case that manifest a dystonic movement in adult women suffering from moyamoya disease. In this case, we also report the effect of valproate on the dystonic movement. **Case:** The 57-year-old female patient presented with dystonic movement of the left arm. Seven years ago, she had stoked at bilateral F-P lobe and then she had been diagnosed with moyamoya disease. She has been improved and discharged. Although some cognitive dysfunction occurring after that. But there was no ischemic symptom or other discomfort of living her life. Six months ago, left hemifacial spasm and left arm dystonic movement was began, and there was no other neurological symptoms. We had performed MRI and MRA of brain, diamox SPECT, and EEG. There was no epileptic discharge on EEG record. And the result of MRA, we found that the moyamoya disease was progressed. And the result of SPECT, Mildly increased perfusion is observed in right frontal, temporal, parietal region with increased vascular reserve. We started valproate and her dystonic movement was improved significantly until 2 months later. **Conclusions or Comments:** We report a special case of dystonic movement on moyamoya disease of adult female, and that was responds to valproate.

P-2-9

Brainstem infarction caused by dissecting aneurysm of a basilar artery in a child

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Background & Significance: Giant aneurysms are often observed in the pediatric population, however dissecting giant aneurysm of the posterior circulation is rarely reported in child. We report a giant dissecting basilar artery

(BA) aneurysm and thrombosis causing brainstem infarction in a child. **Case:** A 5 year-old boy was consulted to neurologic department with decreased response and agitation since 3 days ago, and left side weakness since last night. He did not have any perinatal problem and developmental delay. On neurologic examination, he was alert and fully cooperative. Verbal fluency was decreased and ataxic hemiparesis was observed on his upper and lower extremities of the left side. Diffusion weighted MR showed high signal intensity on bilateral anteromedial pons suggesting acute ischemic stroke. A large thrombus occupying BA was also observed in Gradient echo image. MRA revealed occlusion of basilar artery from the level of vertebrobasilar junction, and both posterior cerebral arteries were supplied by anterior circulation via P-com. Digital subtraction angiography also showed complete occlusion of BA with development of collateral circulation toward brainstem and cerebellum. Follow-up MRI after 15 days showed encephalomalatic change in bilateral pons. Even though the BA had not been recanalized, the lesion was not extended since initial presentation. His symptoms had been stabilized and left side weakness had been completely improved on neurologic examination after 1 month from the symptom onset. **Conclusions or Comments:** It seems that spontaneous BA dissection and a large thrombosed aneurysm occurred simultaneously, which resulted in occlusion of perforating branches of BA and pontine infarction. Although the follow up information has not been provided beyond 1 month from the onset, we report a rare case of completely thrombosed dissecting aneurysm of BA in a child.

P-2-10

A case of bilateral intracranial ICA stenoses causing ischemic stroke in the patient with chronic myeloid leukemia treated with nilotinib(Tasigna[®])

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Background & Significance: Progressive atherosclerosis has been recognized as an adverse effects of nilotinib(Tasigna[®]) for the treatment of chronic myeloid leukemia(CML). Several reports described the association of nilotinib with vascular disease such as peripheral arterial occlusive disease(PAOD), coronary artery disease, and extracranial internal carotid artery(ICA) stenosis. There was only one report for the intracranial atherosclerosis which was in a chain of progressive peripheral atherosclerosis. However, there has been no case of ischemic stroke from intracranial arterial involvement without peripheral artery disease(PAD). We report a patient who developed cerebral ischemic stroke, caused by atherosclerotic stenoses in bilateral intracranial ICAs. **Case:** A 35-year-old man presented with a sudden onset of right hemiparesis and dysarthria. He has taken nilotinib for 3 years for the treatment of CML. MRI showed scattered diffusion-restricted cortical and subcortical lesions in the left frontal and tempo-parietal areas. In CT angiography, bilateral luminal narrowings from both sides of distal ICAs to proximal MCAs. Vessel wall image with MR technic to distinguish atherosclerosis from moyamoya syndrome showed eccentric lateral wall thickening with plaque enhancement in the left cavernous ICA. Multifocal stenoses with enhancement also detected in bilateral MCAs. Laboratory studies related to autoimmune disease or coagulopathy were not remarkable. Both trans-thoracic and trans-esophageal echocardiography could not detect any potential source of embolism. There was no other systemic atherosclerosis in coronary CTA and dynamic kidney CT scan. **Conclusions or Comments:** The patient had no vascular disease risk factors prior to treatment with nilotinib and developed isolated intracranial atherosclerosis leading to stroke without evidence of PAD. Clinicians should be aware of the potential risk of cerebrovascular disease with nilotinib even in the absence of systemic arterial diseases.

P-2-11

Huge giant cervical carotid artery aneurysm with ipsilateral cerebral infarction: ultrasonographic findings of two cases

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Background & Significance: Extracranial carotid artery aneurysms are rare clinical entity. These aneurysms have provided diagnostic and therapeutic challenges for decades. Here, we present two cases of huge extracranial carotid artery aneurysm with interesting ultrasonographic findings that could have an association with thromboembolic risk. **Case:** Case 1. A 60-year-old, woman was brought to the emergency room with global aphasia and right side hemiparesis (NIHSS score 18) which developed abruptly 2 hours before her arrival. She had a huge mass on her left neck with the thrill on it. Diffusion-weighted MRI revealed left ICA territorial infarction. Urgent ultrasonographic examination showed a huge carotid aneurysm (7.2cm of diameter) with dynamic spontaneous echo contrast (SEC) and hyperechoic fragment in the sac. Intravenous t-PA thrombolysis failed to recanalize the cerebral vessel. The patient showed little neurologic improvement after the administration of the IV t-PA and the day after the admission, she expired of massive brain edema. Case 2 A 62-year-old woman who had been suffered from ischemic stroke 1 month ago, presented recurrent ischemic stroke On her both sides of neck, large, soft, and pulsatile mass was palpated which had been diagnosed as extracranial ICA aneurysms (Rt - 7cm of diameter, Lt - 10cm) Diffusion-weighted MRI revealed acute cerebral infarction on her left posterior corona radiata and left posterior watershed area. Carotid ultrasonography also showed dynamic SEC and hyperechoic fragment in the sac. We administered LMWH during the admission, and clopidogrel was applied after the discharge. After another 1 month, multiple territorial infarction recurred again with global aphasia and quadriparesis. Conservative management was done. **Conclusions or Comments:** There are several modalities (Ultrasonography, angiography, CT angiogram etc.) to diagnose the extracranial carotid aneurysm. It is well known that the spontaneous echo contrast (SEC) in left atrium is related to increased thromboembolic risk. The cases shows a huge common carotid aneurysm with SEC and ipsilateral ICA occlusion. Although the direct association between SEC in the extracranial carotid aneurysm and thromboembolic risk was not identified, SEC or hyperechoic substances in the giant cervical carotid artery aneurysm may be helpful to estimate the thromboembolic risk of the patients with giant cervical carotid artery aneurysm.

P-2-12

Multiple intracranial aneurysms in a patient with systemic sclerosis

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Background & Significance: The presence of multiple intracranial aneurysm may be a sign of significant risk factors for aneurysmal rupture. Systemic sclerosis (SSc) is an autoimmune connective tissue disease and associated with variant vascular disease. It is rare, but recent study reported a higher prevalence of cerebral aneurysms in SSc patients than in the general population. We report a patient with SSc associated with multiple intracranial aneurysms. **Case:** A 48-year-old woman visited our hospital for evaluation of headache. The patient had developed Raynaud's phenomenon and sclerodactyly 2 years ago. She diagnosed as SSc based on her clinical features and the interstitial lung disease. She had no history of hypertension, diabetes mellitus. Magnetic angiography incidentally revealed cerebral aneurysm. Digital subtraction angiography (DSA) identified two irregularly shaped 7mm right internal carotid

artery aneurysm in the paracaloid area and 5mm aneurysm in the left ACA pericallosal area. Coil embolization was performed on two separate occasions. The post embolization course was unevenful and post DSA showed that the aneurysmal coil embolization were successfully done. **Conclusions or Comments:** Systemic sclerosis is an autoimmune multiorgan connective tissue disease and micro- and macrovessels are a direct target of the disease. The mechanisms of vascular damage in systemic sclerosis are poorly understood, but recent studies suggest that endothelial cell injury by specific endothelial autoantibodies, inflammatory cytokines or oxygen radicals, underlies the development of vascular damage. Endothelial cell injury has been found to play a primary role in the pathogenesis of ordinary cerebral aneurysms. Several recent reports have suggested that the presence of anti-centromere antibodies correlates with severe vascular damage, and that anti-centromere antibodies may be directly toxic to endothelial cells. In conclusion, the prognosis for patients with collagen disease after rupture of cerebral aneurysm seems to be poor because the multiplicity and atypical location. Thus early treatment of SSc and other collagen diseases may prevent vasculitis from progressing to affect the intracranial vessels and thus reduce the occurrence of aneurysm, and early detection and treatment are important to improve the prognosis.

P-2-13

The influence of minimal daily temperature upon the incidence of stroke in Seoul

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Background & Objectives: Although atmospheric temperature has been associated with stroke incidence, the evidence is conflicting. The relationship between daily temperature and incidence of stroke is largely unknown in Korea. This study attempted to evaluate whether maximal or minimal daily temperatures were associated with incidence of stroke and its subtypes among Seoul citizen. **Method:** From Jan 1, 2005 to Dec 31, 2014, we obtained maximal and minimal daily temperature from Korean Metrological Administration. A consecutive patients with acute stroke were registered, who visited emergency room or outpatients clinic in Soonchunhyang University Seoul Hospital. The addresses of cases were restricted within 3-kilometers radius of our hospital. The clear onsets of stroke were prospectively recorded, and the median time between last normal time and first abnormal time was regarded as stroke onset if unclear. The sums of incidence for the next 2 days and 3 days were counted in each day, and were classified by stroke subtypes. The categories of daily temperature were divided by 10 degrees Celcius from the mean temperature. One-way ANOVA test was applied to compare the 2-days and 3-days incidence rate among the temperature groups. **Results:** A total 2,309 acute stroke were identified during the period: 1,643 ischemic stroke and 666 hemorrhagic stroke. The mean was 17.1 ± 10.8 for maximal daily temperature and 9.0 ± 10.7 for minimal daily temperature. The mean of 2-days and 3-days incidence rate of stroke were 1.26 and 1.90 in each. In maximal daily temperature, those were 1.24 and 1.89 for over 27.0 degree (n=844 days), 1.24 and 1.83 for 17.1~27.0 degree (n=1,138 days), 1.30 and 1.96 for 7.1~17.0 degree (n=829 days), 1.26 and 1.89 for -3.1~7.0 degree (n=731 days), and 1.43 and 2.16 for less than -3.0 degree (n=111 days). In minimal daily temperature, 1.27 and 1.89 for over 19.0 degree (n=860 days), 1.22 and 1.84 for 9.1~19.0 degree (n=1,044 days), 1.26 and 1.91 for -1.1~9.0 degree (n=927), 1.28 and 1.91 for -11.1~1.0 degree (n=748 days), and 1.65 and 2.42 for less than -11.0 degree (n=74 days). Less than -11.0 degree of minimal daily temperature has significantly higher 2-days and 3-days incidence rates of acute stroke in ANOVA (P=.043 and P=.017 in each) **Conclusion:** We report a measurable effect of atmospheric temperature upon stroke incidence. The lowest minimal daily temperature increased stroke incidence in Seoul, Korea.

P-2-14

Clinical characteristics of ischemic stroke in the 80 year-old or older compared with younger patients

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Background & Objectives: The incidence of ischemic stroke increases with age due to improvements in health care and living conditions. With increasing proportion of old age, the proportion of old age is rapid expansion in acute ischemic stroke patients. Young and old age groups in acute ischemic stroke patients have different risk factors and clinical features. But, many patients of old age are excluded from active treatment like thrombolysis due to hemorrhagic transformation, poorer clinical outcome etc. So we studied clinical features, risk factor, outcome in ≥ 80 years old patients compared with <80 years. **Method:** We enrolled 1445 patients, who diagnosed acute ischemic stroke in Chosun university hospital, from January 2010 to January 2013. Patients were divided into two groups: ≥ 80 years versus <80 years. We compared with two groups about risk factors, stroke subtypes, thrombolysis, complications in hospital, initial National Institutes of Health Stroke Scale(NIHSS) score, prognosis. **Results:** Of the total 172 patients, 54(31.4%) patients were 80 years or older. Symptomatic hemorrhage and asymptomatic hemorrhage was not different between both groups [over 79 years patients: 4/54 (7.4%) vs. under 80 years patients: 10/118 (8.5%), P>0.302], [16.7% vs. 17.7%, P>0.701]. There were no difference in mortality and favorable prognosis at 3 months later between both groups [over 79 years patients: 5/54 (9.3%) vs. under 80 years patients: 5/118 (4.2%), P=0.290], [over 79 years patients: 13/21 (24.1%) vs. under 80 years patients: 56/86 (47.5%), P=0.803]. There was difference in early neurological improvement rate(improvement > NIHSS 3) and degree of improvement (NIHSS at discharge - NIHSS at admission) were significant [over 79 years patients: 24/54 (44.4%) vs. under 80 years patients: 77/118 (65.8%), P=0.012], [2.69 vs 5.55, P=0.017]. **Conclusion:** Intracranial hemorrhage(symptomatic and asymptomatic) and favorable prognosis are not different significantly between two groups. Elderly patients is not a absolute contraindication for intravenous thrombolysis. For intravenous thrombolysis in elderly ischemic stroke patients, we need more study to exclude the poor prognostic factors and to consider of gain of patients.

P-2-15

Effect of lesion location on the development of dementia after acute ischemic stroke

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Background & Objectives: A considerable proportion of individuals who have had a stroke develop post-stroke dementia (PSD), and several factors have been suggested as risk factors. However, the role of stroke lesion location is not clearly established. We hypothesized that the disruption of specific neural networks by stroke could contribute to PSD. **Method:** A matched case-control design was applied to a predetermined cohort with acute ischemic stroke. Cases were defined as newly developed dementia diagnosed more than 3 months after stroke. Each case was matched to 2 controls for age, education, and initial stroke severity. Involvement of major hub locations of the cholinergic pathway, functional neural networks, central executive network, and salient network were used. **Results:** We enrolled patients with PSD and matched controls in stroke registry cohort. Male sex, diabetes mellitus, smoking, history of stroke, and several acute and chronic neuroimaging variables were possibly different between the 2 groups (p < 0.2). Acute ischemic lesions affecting the default

mode and central executive networks were consistently associated with PSD in various regression models using several sets of adjusting variables. Lesion location analysis showed that patients with PSD were more likely to have acute lesions in the left centrum semiovale, hippocampal complex, and posterior parietal cortex. **Conclusion:** Our results suggest that the disruption of specific location in subcortical white matter could explain newly developed dementia after acute ischemic stroke.

P-2-16

Association between polymorphisms in microRNA machinery genes (DICER1, DROSHA, RAN, and XPO5) and ischemic stroke

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Background & Objectives: Stroke is the third leading cause of death in the world. Ischemic stroke is a complex, multifactorial disease influenced by multiple genetic and environmental factors. MicroRNAs (miRNAs) play a role in atherosclerosis-related diseases, such as cerebrovascular and cardiovascular disease. In addition, miRNA machinery genes, such as DICER1, DROSHA, RAN and XPO5, play an important role in vascular disease. The objective of this study was to investigate the association between ischemic stroke and six known polymorphisms in miRNA processing genes [DICER1 rs13078 3' untranslated region (UTR) A>T, DICER1 rs3742330 3'UTR A>G, DROSHA rs10719 3'UTR T>C, DROSHA rs6877842 3'UTR G>C, RAN rs14035 3'UTR C>T, and XPO5 rs11077 3'UTR A>C] with ischemic stroke in Koreans. A total of 1000 participants (589 ischemic stroke patients and 411 controls) were enrolled in the study. **Method:** We analyzed the associations between these polymorphisms and both disease status and clinical factors in 585 ischemic stroke patients and 403 controls. Genotyping was performed with the polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) method. **Results:** We identified an association between the DICER1 rs3742330 and DROSHA rs10719 polymorphisms and ischemic stroke. The frequency of the rs3742330 and rs10719 polymorphisms was higher in stroke patients than in controls, suggesting these polymorphisms may be associated with increased risk of ischemic stroke. **Conclusion:** Our study provides the evidences for DICER1 and DROSHA genes as potential biomarkers for use in ischemic stroke prevalence.

P-2-17

Bilateral paramedian thalamic infarction: a case report of artery of percheron occlusion

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Background & Significance: The artery of Percheron (AOP) is a rare anatomic variant that arises from one of the P1 segments and supplies thalamus and midbrain. The incidence of all ischemic strokes is about 0.1% to 2% and of all thalamic strokes is 4% to 18%. The most common clinical symptoms and signs are visual disturbance (55%), confusion and decreased alertness (49%), coma (30%), memory impairment (28%), and behavior problems (23%). **Case:** We report a case of a patient with acute bilateral thalamic infarcts and a truncated AOP demonstrated on transfemoral cerebral angiography (TFCA). A 29-year-old man came to the emergency department with impaired mental status and somnolence. He was founded unresponsive with urination and defecation. He had a known history of right cochlear implantation 12 years ago due to congenital hearing loss. He was drowsy but had no asymmetry in

cranial nerve and motor system on neurologic examination. Electrocardiogram showed a normal sinus rhythm. Brain CT revealed symmetric hypodense lesions in the medial parts of thalami. TFCA showed that AOP was originated from the P1 segment of left posterior cerebral artery. There were no other steno-occlusive vessels. Because of cochlear implantation, brain MRI could not be performed. We founded new hyperlipidemia (LDL-cholesterol 165mg/dL) and hyperhomocysteinemia (24.6 μ mol/L) but there was no evidence of hypercoagulopathy in blood test. No other embolic sources were found on transthoracic echocardiography, transesophageal echocardiography, transcranial doppler for patent foramen ovale, and holter monitor for 24 hours. **Conclusions or Comments:** The presence of AOP must be suspected when bilateral symmetric paramedian thalamic infarcts. We couldn't find any embolic sources of stroke in this patient, however, cardioembolism should always be considered in relatively young patients.

P-2-18

Malignant middle cerebral artery infarction with thyrotoxic atrial fibrillation

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Background & Significance: Atrial fibrillation (AF) is a common type of supraventricular tachyarrhythmia and characterized by uncoordinated atrial activation. Patients with AF have a five times higher risk of stroke, and it is estimated that up to 25 percent of all strokes. AF, commonly considered as a cardiac embolic source, can itself be induced by stroke. Experimental and clinical studies implicates that several cortical structures, especially the insula, are associated with cardiac rhythm control. However, in patients with newly detect AF after stroke, assessment for underlying causes are necessary. We describe a case of malignant middle cerebral artery (MCA) infarction subsequently developed AF with rapid ventricular response (RVR) due to thyrotoxicosis. **Case:** A 62-year-old-women with history of hypertension was admitted because of left hemiplegia. On the neurologic examination, mental status was drowsy. Motor examination revealed left hemiplegia (MRC grade 2). Severe neglect symptoms including anosognosia and asomatognosia were present. Her blood pressure was 160/100 mmHg. She is noted to be in atrial fibrillation with a rapid ventricular response of 135 beats per minute. Magnetic resonance image showed right MCA total infarction with M1 occlusion. We tried to intra-arterial thrombolysis, but recanalization was not achieved. She was intubated and transferred to the intensive care unit. Heart rate was greater than 130 beats per minute under sedation, and we used esmolol for control of heart rate. Transthoracic echocardiogram was normal. Thyroid function studies showed a suppressed thyroid stimulating hormone (TSH) level of 0.01 with elevated free thyroxine (T4) of 4.20 ng/dL (normal, 0.7-1.48 ng/dL). She met the diagnostic criteria for thyroid storm. **Conclusions or Comments:** Newly discovered AF, especially AF with RVR, require assessment for an underlying causes, such as heart failure, pulmonary problems, or hyperthyroidism. Thyrotoxicosis may be complicated with AF and cerebral thromboembolism is frequent in such patients. One retrospective study revealed that AF was occurred 14.9% of thyrotoxicosis patients. The incidence of stroke in thyrotoxic AF is less clear, but stroke risk was related to the presence of associated clinical stroke risk factors. This case developed MCA infarction associated with newly detected AF. Thyroid function test will be helpful in patient with newly detected AF and without history of thyroid disease

P-2-19

A case of Takayasu's arteritis with assessment of response to treatment using high-resolution dark blood MRI

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Background & Significance: Assessment of disease activity and response to treatment in Takayasu's arteritis is important for predicting prognosis. But it is challenging because of its non-specific clinical symptoms and lack of specific laboratory and imaging markers. Recently, high-resolution dark blood MRI is used for evaluating the vascular wall abnormalities. We recently have assessed the disease activity and response to treatment using high-resolution dark blood MRI (DBMRI) in a patient with active Takayasu's arteritis. We describe here our case. **Case:** A 28 year-old woman complained of chronic fatigue, mild fever, and intermittent paresthesia of left arm either at rest or on exertion. On admission, her temperature was 36.5°C and blood pressure was 120/70 mm Hg in the right arm and 90/60 mm Hg in the left arm. Bruit was audible in the bilateral carotid and supraclavicular regions. Neurological examination did not show any focal neurological sign. Laboratory studies revealed an increased erythrocyte sedimentation rate (ESR, 52 mm/h) and increased high-specific C-reactive protein (CRP, 1.24 mg/L). B-mode ultrasonography of the carotid arteries showed long, smooth, and homogeneous concentric thickening of the bilateral common carotid arteries. Contrast-enhanced MR angiography showed diffuse stenosis of the left subclavian and bilateral common carotid arteries. 18F-fluorodeoxyglucose-positron emission tomography/computed tomography demonstrated intense linear uptake in the bilateral common carotid arteries, ascending aorta, aortic arch, and right brachiocephalic trunk. DBMRI showed concentric thickening and double ring enhancement pattern of the bilateral common carotid arteries. She was diagnosed with active Takayasu's arteritis by National Institutes of Health criteria. She was medicated with 60 mg/day of prednisolone and tapered to 10 mg/day over 2 months. ESR and CRP were normalized in response to steroid therapy. Follow-up DBMRI showed the improvement of enhancement of the bilateral carotid arteries itself and connective tissue enhancement adjacent to the bilateral common carotid arteries. **Conclusions or Comments:** In our case, the change on DBMRI was correlated with change of ESR in response to treatment. Our case shows that DBMRI may be useful surrogate for assessment of disease activity and response to treatment in active Takayasu's arteritis.

P-2-20

Transient ischemic attack in hereditary hemorrhagic telangiectasia (HHT) patient

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Background & Significance: Paradoxical embolism is the process that thrombotic material causes end-organ damage by crossing from the right-sided (pulmonary) circulation to the left-sided (systemic) circulation through a congenital or acquired shunt. The most commonly recognized shunt mechanism is intracardiac shunting through a patent foramen ovale, however, pulmonary arteriovenous malformations (AVMs), due to hereditary hemorrhagic telangiectasia (HHT) can also cause paradoxical embolism resulting stroke or transient ischemic attack (TIA). HHT is a rare genetic disease characterized of multiple arteriovenous malformations in skin, mucosal membranes and visceral organs: cerebral, spinal, hepatic, pancreatic and pulmonary AVM. TIA or ischemic stroke in HHT is frequent in clinical practice and often associated to pulmonary arteriovenous malformations. **Case:** We present the case of a 31-year-old man with a previous history of recurrent epistaxis and hemoptysis admitted to our department for a transient episode of right side weakness. Since 2005, he had multiple episodes of hemoptysis and his computed tomography (CT) scan for chest revealed pulmonary AVMs which is needed

embolization. The last episode of apparent bleeding was three months before the current presentation. Physical examination showed a blood pressure of 129/93 mm Hg, pulse of 87 beats per minute and no fever. Though he had a right arm weakness for 5 minutes, it was completely resolved and neurologic examination was normal. Magnetic resonance imaging (MRI) of the brain showed no acute infarction. There was no steno-occlusive lesion on his brain angiography. However, his lung perfusion CT scan revealed residual multiple pulmonary AVMs in LUL since embolization, which was lastly done three months ago. Transcranial Doppler detected 170 embolic tracks during normal respiration, and 250 embolic tracks during Valsalva manual. His mother and two aunts had also history of pulmonary AVMs. Therefore genetic test was done on him, which revealed HHT type 1 (ENG gene mutation). We considered lobectomy as management since there was recurrent pulmonary AVMs even he had several times of embolization, there was increasing risk of massive bleeding or embolic stroke. We started Plavix monotherapy before surgery, planning changing to low-molecular heparin before few days before surgery. We consulted to Cardiac surgery part, and he had an operation 3 weeks after the presenting symptom. Since the first symptom appeared, there was no recurrent neurologic symptom. **Conclusions or Comments:** The presumed mechanistic diagnosis in this case was underlying HHT resulting in paradoxical embolism into the cerebral circulation across the shunt provided by the pulmonary AVM. HHT is a rare disease, however often is associated with epistaxis, gastrointestinal bleeding, congestive heart failure, and hemoptysis. Patients with this syndrome are at risk of developing brain arteriovenous malformation or fistula, stroke, or transient ischemic attack due to paradoxical embolism. Pulmonary AVMs should be carefully screened in the patient with stroke or TIA symptoms and genetic consultation for HHT is recommended if there is a recurrent episodes of bleeding or evidence of family history. Percutaneous embolization is a treatment of choice of pulmonary AVM, but surgery is considerable in specific cases as our patient.

P-2-21

A case of forced conjugate eye deviation in small pontine infarction

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Background & Significance: Forced conjugate eye deviation is an indicator of large hemispheric infarction. However, we recently experienced a case showing forced conjugated eye deviation in a patient with a small pontine infarction. We describe here our case. **Case:** A 58-year old man with hypertension and diabetes mellitus presented with sudden onset vertigo and ocular movement limitation. Neurological examination showed forced conjugate eye deviation to the right side, which was not overcome by vestibulo-ocular reflex. There were no other neurological signs including cranial nerve signs, sensory disturbance, and motor weakness. Initial diffusion weighted image (DWI) showed no diffusion restriction lesion. The next day, forced conjugate eye deviation was slightly released. However, saccade to the left side was still slow and incomplete. Right beating nystagmus became prominent in right gaze. On the third day, follow-up DWI revealed a tiny high signal on tegmentum in left lower pons. **Conclusions or Comments:** Our case shows that small pontine lesion could be considered as the possible cause of forced conjugate eye deviation in patient without symptoms and signs of hemispheric lesion.

P-2-22

Cerebral infarction with massive intra-aortic mass in young female

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Background & Significance: Primary aortic sarcoma is a rare and aggressive tumor. Because of their low prevalence and presentations mimicking non-malignant vascular disease, diagnoses are often made postmortem or after disseminated metastases have already occurred. Since the first description in 1873, several cases of cerebral infarction related to aortic sarcoma were reported. We describe a first case of cerebral infarction due to aortic sarcoma in Korea. **Case:** A 20-year old woman presented with an acute onset global aphasia and right side weakness. She had no vascular risk factor and a family history of stroke. No history or risk of hypercoagulability. MRI showed a left middle cerebral artery territory (MCA) infarction. MRA showed left MCA proximal M1 occlusion. As the patient was young and did not have any risk factor for stroke, extensive work-up to find the embolic source was performed including cardiac work up, coagulation labs and CT of the aortic arch. No embolic source was confirmed from the trans-thoracic echocardiography. D-dimer and other coagulation factors were within normal range. CT neck angiography showed a large thromboembolus in aortic arch, left common carotid artery (CCA) and right brachiocephalic trunk. The patient started anti-coagulation with warfarin. Afterwards through rehabilitation, the patient's symptoms improved to an almost normal. 2 months later, the patient's symptom was aggravated. When she stand up she complained of transient left side weakness and syncope. Though the prothrombin time was in the therapeutic range, and the D-dimer was normal, the CT showed progression of the aortic arch atheroma extending through the left subclavian artery, left distal internal carotid artery (ICA), right distal ICA and left proximal vertebral artery. CT showed about 2.2 cm sized round shape mass lesion in the right supra-clavicular area. Patient's symptoms were not improved and thrombolysis was aggravated despite of proper anti-coagulation, so we consulted to department of thoracic surgery for bypass surgery. The surgery was progressed in the direction of removing the thrombolus inside the aortic arch as much as possible. The first surgery connected the ascending aorta with the left subclavian artery, and the second surgery consisted of a bypass surgery that connects the aorta with the right distal CCA while linking the ascending aorta with the descending aorta to prevent a sudden death. Biopsy result was very low grade aortic intimal sarcoma but clinical condition was very aggressive. **Conclusions or Comments:** Primary aortic sarcoma is a rare tumor, with approximately 200 cases reported in literature. In this case, tumor involved the branch vessels. Clinically, patients mostly present with symptoms of tumor embolus, including cerebral infarction or peripheral artery thromboembolism. Generally, prognosis is poor and median survival from diagnosed is 11 months. Treatment choices are surgery, chemotherapy and radiotherapy. Resective surgery improves the prognosis and is associated with an improvement in median survival. Other thromboembolism, occasionally d-dimer is increased. But this case and other thromboembolic cases due to aortic sarcoma showed normal ranges of coagulation lab including d-dimer. The mechanisms leading to activation the clotting system and multiple embolization are unknown. osteopontin, an extracellular matrix protein was found to be upregulated in macrophages and malignant cells from primary sarcomas of the pulmonary artery, thus supporting a possible role in both progression and tumor spreading through the activation of both cellular adhesion and chemotaxis. Our patient was diagnosed with left MCA infarction due to emboli from aortic intimal sarcoma, and it was first case in Korea. Aortic sarcoma is extremely rare cause of stroke, but in cases when a massive thrombolus forms in large vessels without any particular cause and coagulation labs are normal, the possibility of such primary aortic tumor should be taken into account.

P-2-23

Acute intracerebral hemorrhage with a spot sign during computed tomographic angiography

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Background & Significance: The spot sign is an indicator of active hemorrhage and has been associated with an increased risk of hematoma expansion and mortality in patients with ICH. The spot sign refers to one or more foci of contrast enhancement within an acute primary parenchymal hematoma. It is also associated with larger hemorrhage, more severe clinical presentation, and decompression of the hemorrhage into the intraventricular space. Hematoma expansion is an independent predictor of increased mortality and poor outcome in ICH. Hematoma expansion is common, and usually occurs in the early phase of ICH. However, an ICH occurring during CTA is very rare. **Case:** A 73-year-old woman presented with dysarthria followed by right hemiparesis. Unenhanced computed tomography (CT) revealed a hypodense lesion in the left middle cerebral artery (MCA) territory. Computed tomographic angiography (CTA) with contrast was performed immediately, after which she suddenly lost consciousness and her initial symptoms began to progress. Enhanced CT revealed an acute intracerebral hemorrhage (ICH) in the left MCA territory, the same area as identified previously. A small focus of enhancement was seen peripherally on enhanced CT, consistent with the spot sign (maximum dimension = 5.82mm, maximum attenuation = 221 Hounsfield Unit). Coronal CTA demonstrated the same focus of the spot within the hematoma. According to the criteria of Jorner E. et al., the calculated spot sign score was three points (mortality = 61%, poor outcome = 54%). Follow-up unenhanced CT performed 1 h later revealed hematoma enlargement with intraventricular and subarachnoid hemorrhage. **Conclusions or Comments:** This patient had an acute cerebral infarction when the left MCA was blocked before arrival at our hospital. We hypothesize that the reason for ICH development was the recanalization of the blocked vessel, which was caused by the pressure of the contrast medium infusion while performing CT. In addition, the pressure of the contrast medium created the spot sign, and hematoma expansion occurred rapidly over the course of 1 h, resulting in a poor outcome. As in this case, a spot sign caused by high blood pressure accompanying ICH is highly likely to result in a poor outcome. Therefore, immediate surgical treatment is needed.

P-2-24

Endovascular therapy for acute stroke in patients with current malignancy

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Background & Significance: Cancer causes a hypercoagulable state, increasing the risk of thromboembolism including acute ischemic stroke. Intravenous thrombolysis appears to be safe in patients with cancer; however, it is often contraindicated in this population because of coagulopathy or recent surgery. Endovascular therapy may be a more suitable recanalization strategy for some patients with cancer and stroke. But the safety of endovascular therapy for acute stroke in patients with cancer is not well established. The aim of our study is to evaluate outcomes of endovascular therapy in patients with current malignancy. **Case:** We reviewed our acute ischemic stroke (AIS) database for clinical outcomes and complications in patients with current malignancy (CM) who received endovascular therapy. Patients with CM who received endovascular therapy were identified from the 2011 and 2014 in CNUH. Baseline clinical and demographic were taken from our prospective stroke registry and charts were reviewed for history of current malignancy. Patients with current malignancy were defined as patients undergoing treatment for malignancy

during acute ischemic stroke. Of 10 AIS patients treated with endovascular, 3 patients received intravenous tPA plus endovascular therapy, 7 patients received endovascular therapy only. Total occlusion (TIMI 0) was found in 10 patients (100%). 5 patients are ICA occlusion (5/10, 50%) and 5 patients are MCA occlusion (5/10, 50 %). After endovascular therapy, total recanalization was achieved in 100% (TIMI 3). In six of the ten patients treated with endovascular therapy, significant improvement was seen with no symptomatic hemorrhage (6/10, 60%) and four patients were not changed NIHSS compared to admission (4/10, 40%). Postprocedural hemorrhage occurred in 2 patients (20%), of which one patient was symptomatic hemorrhage and one patient was petechial hemorrhage. Postprocedural restenosis or reocclusion occurred in 2 patients (20%), of which one patient was MCA reocclusion and one patient was MCA severe stenosis. **Conclusions or Comments:** Endovascular treatment has become a prominent tool for the management of AIS, resulting from proximal large artery occlusions. The good outcomes in our 6 patients suggest that select patients with cancer and AIS may benefit from endovascular therapy especially if reperfusion can be achieved. However, the efficacy of endovascular therapy for acute stroke in patients with current malignancy has not been proven in clinical trials, and this report only includes 10 patients; therefore, further studies are needed to determine the utility of endovascular therapy in patients with current malignancy.

P-2-25

Transient global amnesia-like tiny hippocampal lesion in patients without transient global amnesia

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Background & Objectives: Tiny hippocampal lesions on diffusion weighted MR (DWI) has been reported as a typical radiological finding in patients with transient global amnesia (TGA). However, TGA-like tiny hippocampal lesions were occasionally observed in patients without clinical symptoms of TGA. We report eight cases of TGA-like tiny hippocampal lesions which were incidentally found in patients without TGA. **Method:** Clinical characteristics including age, sex, vascular risk factors, cognitive function including mini-mental status examination (MMSE), and clinical symptoms and radiological findings including hippocampal lesions in DWI, hippocampal cavity, and underlying white matter hyperintensities based on Fazekas scale were evaluated. **Results:** Median age was 61 years (53-73); five were women (62.5%); and four had conventional vascular risk factors including hypertension in 3, diabetes in 1, smoking in 3, and previous stroke in 1. 7 of 8 patients performed MMSE. Median MMSE score was 27 (24-29). Registration was complete in all patients. Recall was 3/3 in 3, 2/3 in 3, and 1/3 in 1 (median 1/3). None of the patients presented typical TGA-like symptoms such as loss of recent memory such as anterograde and partial retrograde amnesia resolving in 24 h. Reason for DWI investigation was dizziness in 4, finally diagnosed with vestibular neuronitis in 2, benign paroxysmal positional vertigo in 1, and anxiety in 1. One patient for dementia workup and 2 patient for stroke workup (1 with recurrent right facial and arm paresthesia and 1 with right facial palsy and dysarthria). The other patient suffered from feelings of unreality for about 30 minutes, after swimming. All patients had a single lesion; right in 4 and left in 4. Lesion location in the hippocampal was body in 6; head-body junction in 1; and tail in 1. Hippocampal cavities were detected in 7 patients (7/8, 87.5%); unilateral in 5 and bilateral in 2. Hippocampal cavities were on the same side of tiny hippocampal DWI lesions in 6 patients (6/7, 85.7%). The width of the cavities in the axial section was 2.0 (1.5 - 2.2) mm. White matter hyperintensities measured by Fazekas scale were 1-2 (median 1) in the periventricular white matter and 0-2 (median 1) in the deep white matter. **Conclusion:** We present eight cases of TGA-like tiny hippocampal lesions which were incidentally found in patients

without typical clinical symptoms of TGA. Pre-existing structural vulnerability to amnesia including hippocampal cavity, limbic structure, or white matter integrity may have worked as a determining factor for the development of clinical symptoms of TGA or not. Further study to compare clinical and radiological factors using with age-sex matched patients with typical TGA are warranted.

P-2-27

The ischemic lesion volume of diffusion weighted images is much more important to determined the prognosis after using thrombolysis in acute MCA occlusion than that of perfusion weighted images on MRI

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Background & Objectives: There is a mounting evidence in support of the notion that vessel recanalization has the most powerful effect on clinical outcomes in acute ischemic stroke (AIS). However, It has been focusing the importance of remaining saving brain tissue after ischemic events to get more favorable outcome as much as a recanalization after thrombolysis. Many studies demonstrated the usefulness the volume of perfusion-weighted images on MRI to estimate the presence of ischemic penumbra after acute ischemic stroke. Until now, its reliability is yet doubtful in clinical fields. In this study, we studied the relationship between lesion volumes of diffusion weighted and perfusion weighted images (DWI and PWI) and outcomes after thrombolysis based on MRI in patients with acute MCA occlusion. **Method:** In this study, we enrolled 200 patients with AIS arriving at Dong-A university stroke center within 6hr after onset of their ischemic events. Among this population, we selected 62 patients using IV thrombolysis under initial MRI screening. We measured volume of initial DWI on MRI. The volume of baseline PWI (Tmax>6) was calculated by voxel-by-analysis. Poor outcome is defined as 90-day modified Rankin score >2. **Results:** Successful recanalization ((thrombolysis in cerebral infarction grade 2b or 3) was achieved in 67.7% and favorable outcomes in 56.5% of cases. The median volume of DWI was 64 mL in those patients with poor outcome and much larger than those with showing favorable outcomes (8.7 mL) in 90 day after using IV thrombolysis. Those patients with poor outcomes also had larger PWI (Tmax>6) than those with favorable outcomes after using IV thrombolysis. In multivariate analysis, the non- recanalization (OR, 34.7; 95%CI, 2.7-450.5; p<0.01) and larger size of baseline DWI volume (more than 14 mL, OR, 7.1; 95% CI, 1.3-38.9; p=0.02) on MRI had an independent significance for an occurrence of poor outcomes after using thrombolysis. However, the volume of PWI showed no any significance for its occurrence. At 60 mL DWI cutoff, the prediction sensitivity and specificity for poor outcome are 97% and 56%, respectively (area under curve 0.75, 95% CI 0.38-0.72). **Conclusion:** In this study, the baseline volume of DWI on MRI is a significant important prognostic factor for predicting outcomes after IV thrombolysis. Therefore, it would be very important to estimate the ischemic lesion volumes and to select proper candidates for IV thrombolysis after AIS.

P-2-28

Atypical bilateral medial medullary infarction due to unilateral vertebral arterial dissection

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Background & Significance: Vertebral dissection presents with various clinical

presentations from asymptomatic to major strokes. The authors experienced one patient with bilateral medial medullary infarction caused by unilateral vertebral arterial dissection who developed quadriplegia despite of early treatment with intravenous thrombolysis. **Case:** A 60-year-old woman came to emergency room with sudden onset of dizziness and nausea/vomiting. She was on long-term treatment for essential hypertension and had no recent head trauma and neck manipulations. Neurologic examination showed bilateral gaze evoked nystagmus, bilateral arm ataxia, truncal ataxia and bilateral positive Babinski signs. Initial brain computerized tomography was unremarkable. Blood test, biochemistry, chest x-ray, and EKG were normal. Intravenous Tissue plasminogen activator (TPA) was infused within 1 h after arrival. During preparation for infusion of TPA, she became stuporous with depressed respiration and quadriplegia developed quickly. Immediately following endotracheal intubation and mechanical ventilation, her consciousness regained. However, quadriplegia persisted in spite of IV thrombolysis. Brain MR imaging at second weeks of admission revealed acute bilateral medial medullary infarction and right cerebellar infarction with hemorrhagic transformation. T1 weighted sagittal MR images showed the intramural hematoma of the right vertebral artery, suggesting of arterial dissection. **Conclusions or Comments:** We presumed that anterior spinal artery and its dominant perforator for median medulla from vertebral artery was occluded by intramural hematoma, which resulted in bilateral medial medulla infarction. We reported an atypical patient who had unilateral vertebral dissection evolving into bilateral medial medullary infarction that did not respond to intravenous thrombolytic therapy.

P-2-29

Small vessel TIA or stroke detected with perfusion-weighted MRI

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Background & Significance: The clinical diagnosis of transient ischemic attack (TIA) or minor stroke is highly subjective especially if the brain imaging does not reveal any evidence of ischemic lesion. We report 2 cases of small vessel TIA or stroke detected with the initial perfusion-weighted MRI. **Case:** Case 1. A 60-year-old hypertensive lady presented with left side weakness. She experienced slurred speech and weakness of her left arm and leg, which lasted for 5 minutes. On neurological examination, she did not show any deficit. The initial multimodal MRI, taken 2 hours after the symptom, revealed no abnormality on DWI but showed perfusion defect in her right basal ganglia. The intracranial and extracranial MR angiography did not show any vascular stenotic lesion. The patient has not experienced further symptom. Follow-up DWI was carried out 2 days later and it revealed the diffusion restriction corresponding to the initial perfusion defect. Case 2. A 66-year-old hypertensive female presented with the clinical findings of cheiro-oral syndrome. Neurological examination revealed sensory deficit in her left fingers and left hemi-mouth. Multimodal MRI taken 18 hours after the onset revealed no abnormality on DWI but showed perfusion defect in her right lateral thalamus. MR angiography did not show any significant vascular lesion. Follow-up DWI taken 2 days later showed acute infarction on the right thalamus, which corresponded to the initial perfusion defect. **Conclusions or Comments:** Whereas perfusion-weighted imaging has been known to be helpful for detecting ischemic lesion caused by large vessel disease, our cases show its usefulness for small vessel TIA or stroke as well.

P-2-30

Two cases with only abnormal perfusion MRI findings in the transient ischemic attack

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Background & Significance: Transient ischemic attacks (TIAs) are brief episodes of neurological dysfunction resulting from focal cerebral ischemia not associated with permanent cerebral infarction. TIAs are defined as any focal cerebral ischemic event with symptoms lasting < 24 hours. Recently, however, 30% to 50% of classically defined TIAs show brain injury on diffusion-weighted imaging (DWI). Perfusion weighted imaging (PWI) using gadolinium based dynamic-susceptibility contrast provides information on ischemia. The mismatch between the core of the infarction revealed on DWI and the region of critical hypoperfusion estimated by PWI has been proposed as a surrogate of the ischemic penumbra. Several studies have demonstrated that PWI lesions are present in about 30-40% of TIA patients, and in many of these cases there are no DWI lesions. Herein, we describe 2 cases with TIA, which was completely resolved within 24 hours and just showed abnormal PWI without no acute lesions in the DWI. **Case:** Case I: A woman, who was 31 years old, visited our emergency room because she could not understand some written languages for 20 minutes. When she was waiting for bus with watching her cell phone, she suddenly did not understand the words and sentences on her cell phone. Her symptom was prolonged for 20 minutes and the symptom had resolved. She performed magnetic resonance imaging (MRI) at 12 hours after symptom onset. Although there was no acute cerebral infarction in the DWI, T2-weighted and FLAIR images, PWI only showed a lesion with increased MTT in the left parietal lobe. Case II: A 47 year-old woman visited our emergency room because of sudden weakness and tingling sensation on right upper limb. When she arrived at emergency room, her symptoms were being improved and completely resolved within several hours. In the neurologic examination, she showed slightly mild weakness of right hand flexion and finger abduction. Sensory was normal. There were no vascular risk factors. Her perfusion MRI showed increased time delay in the left parietal area. Other MR images were all normal findings. **Conclusions or Comments:** These cases suggest the importance of perfusion MRI to diagnose TIA. Two cases showed short duration of symptoms and completely resolution of their symptoms. Therefore, many clinicians ought to keep in mind watching the perfusion MRI carefully.

P-2-31

Predictive ability of focal perfusion abnormality on perfusion weighted MRI in diffusion negative transient ischemic attack

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Background & Objectives: Multi-modal MRI which has recently become strong diagnostic tools for the assessment of transient ischemic attacks (TIA) includes diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI). DWI has been shown to be useful for diagnosis and prognostication of TIA. If initial DWI is normal in TIA patients, PWI may be helpful in predicting the true vascular event. Therefore we investigated that presence of initial PWI abnormality implies the true vascular event by showing the new DWI lesion on follow-up MRI in corresponding area of initial PWI abnormality. **Method:** In addition to the previous prospective cohort study of our researchers which has investigated 162 of TIA patients who visited Asan Medical Center (AMC) within 72 h of symptom onset between July 2009 and December 2011, we retrospectively investigated 281 of TIA patients who visited AMC within 72 h of symptom onset from the AMC Stroke Database, collected from just after completion of the prospective cohort study to May 2015. Among them, patients who had initial DWI lesions were excluded. Finally we

enrolled 87 of TIA patients who conducted initial multimodal MRI which included DWI, PWI, fluid-attenuated inversion-recovery (FLAIR) imaging, MR angiography (MRA) and follow-up DWI at three-days. PWI abnormalities were visually divided by 3 patterns which are normal perfusion, focal perfusion abnormality and territorial perfusion abnormality. Focal perfusion abnormality was confined to a specific region of increased signal intensity in the TTP(time to peak) map and territorial perfusion abnormality was defined as large areas involving 1 or more vascular territories of increased signal intensity in the TTP map. **Results:** Of the 443 patients who presented with TIA, 119 patients were conducted follow-up DWI at three-days. 87 of whom had no DWI lesions on initial multi-modal MRI. The result shows initial PWI abnormalities were significantly associated with follow-up DWI abnormalities ($p < 0.0001$). 13 out of 16 patients (81.2%) with focal PWI abnormality were significantly associated with the follow-up DWI abnormality : OR = 15.8 (95% CI = 3.7-66.7). On the other hand, 14 out of 24 patients (58%) with territorial PWI abnormality were associated with the follow-up DWI abnormality. Territorial PWI abnormality had relatively little connection with follow-up DWI abnormality compared with focal PWI abnormality : OR = 3.6 (95% CI = 1.21-10.5). **Conclusion:** We found that presence of initial focal PWI abnormality on multi-modal MRI is a strong predictor of development of new DWI ischemic lesions and implies the true vascular event in TIA patients who had no initial DWI lesions. These results are in agreement with those of two separate cohort studies which are the prospective and retrospective cohort study.

P-2-32

Increased plasma homocysteine levels not MTHFR variant are associated with cerebral microbleeds

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Background & Objectives: Cerebral small vessel diseases such as leukoaraiosis and silent brain infarction are known to be associated with increased levels of plasma homocysteine. However, there are no clear association between homocysteine level and cerebral microbleeds. We aimed to seek the relationship between cerebral microbleeds and total homocysteine level or methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism. **Method:** Eight hundred sixty-one patients with subjective memory disturbance who visited dementia clinic were consecutively included in this study. We collected demographic, clinical and laboratory data including total homocysteine level and MTHFR C677T polymorphism. All patients performed brain MRI including T2*-weighted gradient-echo MRI. We used logistic regression analysis to examine an independent association between homocysteine and cerebral microbleeds. **Results:** One hundred seventy-three patients had cerebral microbleeds. The TT genotype of the MTHFR C677T polymorphism was associated with hyperhomocysteinemia ($p < 0.001$), but not with the presence of cerebral microbleeds. After adjusting confounders (hypertension, diabetes, stroke, age, sex, serum creatinine, serum uric acid), age (OR, 1.038; 95% CI, 1.017-1.058, $p < 0.001$), hypertension (OR, 2.122, 95% CI, 1.435-3.137, $p < 0.001$) and homocysteine (OR, 1.017, 95% CI, 1.000-1.035, $p = 0.049$) were independently associate with the presence of cerebral microbleeds. **Conclusion:** Hyperhomocysteinemia was associated with cerebral microbleeds in cognitive declined patients. Although TT genotype of the MTHFR C677T polymorphism had great association with hyperhomocysteinemia, it was not associated with cerebral microbleeds.

P-2-33

Susceptibility vessel sign with bright vessel appearance that differentiates clot composition and its association with stroke etiology

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Background & Objectives: Red thrombi, composed of fibrin and trapped erythrocytes, have magnetic susceptibility effect. Susceptibility weighted imaging (SWI) was more sensitive than T2*-weighted imaging to visualize susceptibility vessel sign (SVS). Bright vessel appearance (BVA) on arterial spin labeling (ASL) imaging can visualize arterial occlusions, especially more sensitive in distal, fine branches. We hypothesized SWI-SVS with BVA better delineates thrombus composition, especially within distal and small branches, and is related to stroke etiologies suspected to generate red thrombi. **Method:** From a total of 564 stroke cases who admitted to Seoul National University Hospital in 2014, the authors collected eligible cases with the following inclusion criteria; (1) Lesion-documented ischemic stroke (N=425); (2) Susceptibility-weighted imaging (SWI) and arterial spin labeling (ASL) MRI performed (N=407); (3) Symptomatic occlusion of intracranial arteries evaluated by the presence of bright vessel appearance (BVA) in ASL image (N=141). All images were analyzed for the presence or absence of susceptibility vessel sign in SWI (SWI-SVS), BVA in ASL, arterial occlusion on time-of-flight MR angiography (TOF-MRA). The location of SWI-SVS and BVA were classified into (1) proximal, large arteries; distal ICA, M1, M2, A1, P1, basilar artery, V4 and (2) distal, small arteries; M3, M4, P2, A2, lenticulostriate arteries, the three cerebellar arteries. The relationships between SWI-SVS in the presence of BVA and stroke etiologies are explored. **Results:** Of the 141 analyzable cases, male was 58.2% (n=82), mean age was 65.7 ± 14.3 , and median NIHSS score was 4 [2-11] point. Thirty-four percent (48 of 141) of BVA was located within distal, small arteries. SWI-SVS was observed in 99 patients (70.2%) and 30.3% (30 of 99) was located within distal, small arteries. SWI-SVS was more commonly associated with the patients with other determined etiology (21 of 23, 91.3%) and cardioembolism (38 of 44, 86.4%) than other etiologies (40 of 74, 54.1%). Among those with other determined etiology with SWI-SVS (n=21), the most common was cancer-related hypercoagulability (62%, n=13). Multivariate analysis showed that SWI-SVS was an independent predictor of other determined etiology (adjusted OR, 7.80; 95% CI, 1.59-38.28) and cardioembolism (adjusted OR, 5.07; 95% CI, 1.27-20.18). **Conclusion:** SWI-SVS, in the presence of BVA, may predict ischemic stroke with other determined etiology and cardioembolism, mainly suggestive of red thrombi. Especially, occlusions of distal, small branches can be more visualized with BVA and composition of the thrombus can be identified by the presence or absence of SWI-SVS.

P-2-34

Venous infarction related to dural arteriovenous fistula directly drained into cortical veins

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Background & Significance: A dural arteriovenous fistula (DAVF) has diverse symptoms according to the location and venous drainage. We report a case of venous infarction related to DAVT presenting aphasia with slow responsiveness. **Case:** A 73-year woman had a 3 weeks history of aphasia and slow responsiveness. She had a past history of hypertension, diabetes, and cerebral palsy. Neurological examination showed transcortical sensory aphasia, right hemiparesis (MRC grade 4/5), and areflexia. Brain MRI demonstrated hemorrhagic infarction at the left tempo-occipital cortex and dilated temporo-occipital horn. MR angiography with enhancement revealed left proximal and distal

ICA multifocal severe stenosis. We initially considered cerebral infarction due to artery to artery embolism and focal epilepsy. We conducted conventional angiography for identification of carotid stenosis. Conventional angiography showed left temporal DAVF directly drained with cortical vein without engorgement of dural sinus. Finally we diagnosed venous infarction with intracranial DAVF and tried embolization. Transarterial embolization of the branches of left middle meningeal artery and occipital artery with Onyx obliterated the DAVFs completely. **Conclusions or Comments:** We should consider DAVF in patient with venous infarction although there is no dilatation of the dural sinus and signal voids of feeding arteries and draining vein in MR scan.

P-2-35

Lateralized MRI findings of cerebral hypoxia in patients with carotid artery stenosis

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Background & Significance: Hypoxic-ischemic brain damage shows different brain MRI findings at basal ganglia, thalami, cerebral cortex (in particular the sensorimotor and visual cortices), cerebellum or hippocampi. Brain damage of CO intoxication follows the pathophysiology of hypoxic-ischemic encephalopathy and also shows different brain MRI findings at basal ganglia (especially GP), WM or cerebral cortex. These generally damage bilateral hemisphere, and lateralized or unilateral brain MRI findings are rare. These findings can be shown in patients having underlying carotid artery stenosis. **Case:** Case 1 : 91/M, mental deterioration, asphyxia event at breakfast, semi-coma Case 2 : 75/M, mental deterioration, Breathing gases from burning coal briquettes, stupor Case 3 : 50/F, mental deterioration, hanging, stupor Case 4 : 61/M, heart attack, acute MI and cardiac arrest, semi-coma MRI/MRA findings of Case 1 (severe Lt CCA stenosis) show left lateralized lesions and those of Case 2 (severe Lt ICA stenosis) indicate left unilateral lesions. But those of Case 3 and 4 with unremarkable MRA show bilateral and symmetrical lesions. **Conclusions or Comments:** Profound hypoxic insults can affect the entire cerebral cortex or just the perirolandic cortex, the cerebellum and the deep grey matter structures. Less severe insults may affect only the watershed regions. Watershed infarctions caused by a diffuse anoxic-ischemic insult appear to be more common in neonates and children. It is hypothesized that watershed infarction occur in cases of severe hypoperfusion due to carotid occlusion without anoxia. In CO intoxication patients, GP lesions are often considered as pathognomonic signs and WM lesions are the most common findings either in the acute phase or in those with delayed neuropsychiatric sequelae. Consequently, these diseases generally damage bilateral hemisphere. If patients who already had carotid artery stenosis have hypoxic or CO intoxicative events, lateralized or unilateral brain lesions can be shown vulnerable to hypoperfusion. These brain MRI findings can have clinicians confuse to early diagnosis, and this study can help to differentiate from other possibly lateralized brain diseases such as post-seizure, encephalitis, CJD and so on.

P-2-36

Extensive cerebral microhemorrhage after extracorporeal mechanical oxygenation

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Background & Significance: Extracorporeal mechanical oxygenation (ECMO) is used to provide cardiac or pulmonary support in case refractory to conventional therapies. We describe a patient who demonstrated neurologic deficit and extensive cerebral microbleeds after ECMO. **Case:** 58 year-old woman pre-

sented with acute respiratory distress syndrome secondary to community acquired pneumonia by streptococcus pneumoniae. Ventilator and venovenous ECMO were applied for 8 days. Thereafter despite of improved medical problem, the patient failed to awaken fully and revealed cognitive dysfunction including global aphasia and mild symmetric motor weakness with bilateral Babinski sign. Diffusion weighted image of brain magnetic resonance imaging (MRI) was not distinctive except a few lesions with diffusion restriction, but extensive petechial hemorrhages across the all brain area was shown, especially in gray-white matter junction on susceptibility-weighted imaging. Neurological injury occurs frequently in ECMO-treated patients with otherwise reversible cardiopulmonary injury. (1, 2) The cause of extensive cerebral microhemorrhage in this case was complicating because sepsis or heparin induced thrombocytopenia was noted during ECMO. Her neurologic deficit was rapidly improving and recovered normal function. **Conclusions or Comments:** Neurologist and physician should be aware of this rare complication after ECMO support and comparing to extensive imaging finding, neurologic deficit showed favorable outcome.

P-2-37

Transient ischemic attack in patient with an unruptured aneurysm

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Background & Objectives: Cerebral aneurysm, located on the superior cerebellar artery (SCA), is uncommon, and transient neurologic sign, such as hemiparesis, is a rare presentation. We report a case of an aneurysm located on the SCA caused transient hemiparesis and then ruptured. **Method:** A 43-year-old man with a history of hypertension presented with sudden onset left side weakness. The symptom lasted for two hours and completely resolved when he visited the emergency center. Neurological examination revealed neither focal neurological signs nor meningeal irritation signs. **Results:** Brain magnetic resonance imaging (MRI) showed no evidence of acute ischemic lesion, and magnetic resonance angiography (MRA) revealed unruptured saccular aneurysm at basilar tip area. Trans-femoral cerebral angiography (TFCA) showed a giant aneurysm filled with thrombus in the left SCA. We planned endovascular treatment, but he wanted to delay. Three days after admission, he collapsed and had a generalized tonic-clonic seizure with comatose mentality. Follow up computed tomography (CT) showed subarachnoid hemorrhage in the peripontine, basal cisterns, posterior fossa as well as Sylvian fissure. Coiling of aneurysms and insertion of external ventricular drain (EVD) were performed immediately. However he remained comatose state and died ten days later. **Conclusion:** Thrombotic emboli from the aneurysm might cause transient cerebral ischemia presented with hemiparesis. Thrombosed aneurysm is often large and aneurysm thrombosis may increase the risk of rupture. Therefore, we have to consider transient neurologic symptom in unruptured aneurysm as a red flag and early treatments include surgical clipping and endovascular coiling may be helpful to prevent severe complications.

P-2-38

Factors associated with Do-Not-Resuscitate order in acute ischemic stroke patient

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Background & Objectives: Do not resuscitate (DNR) order is patient or his family preferences for limitation in cardiopulmonary resuscitation, but not attempted to limit other medical care. However, according to past reports, early DNR order impacted on poor outcome or mortality in acute stroke patients.

In Canada and USA, about 30% of patients with acute stroke received the DNR order during their hospitalization period. DNR order was associated with age, cognitive function, health policy, preadmission status, disease severity, patient or family beliefs and values and etc. Although, DNR order might be affected by social or community difference, there was a few reports for DNR order in Korea and most of them were for cancer or Intensive care unit patients. Acute cerebral infarction (AIS) is important cause of death in Korea, there was no report about DNR order of them during hospitalization. **Method:** The consecutive series of patients who were diagnosed AIS between January 2011 and December 2014 in Ulsan university hospital were selected. The medical data and expire day were collected retrospectively from the prospective stroke registry or a review of electronic data in hospital. All DNR consent were confirmed in electronic copies in medical records. Early DNR was defined as the consent form was underwent within one day after hospitalization. If guardian withdrew a consent, patient were considered to no DNR patients. **Results:** A total of 1700 patients were included. Among them, sixty patients (3.5%) took the DNR consent. All case were received the consent from guardians. The DNR consents were underwent by partner (11.7%), their children (85.0%) and etc (3.3%). Mean day from admission to DNR consent was 2.97 days. The patients with DNR order were associated with old age, female, hypertension, previous disability, and initial stroke severity. There were 40 mortality cases in study period, thirty-six patients (90.0%) had the DNR order. Among the patients with DNR, patients received the early DNR were 39 cases (65%). The patients with the Early DNR were associated with old age and initial severity. Specially, early DNR was high proportion in patients over age 80 (41% vs. 9.5%). Previous disability was not associated with early DNR. **Conclusion:** Our study shows that DNR order was associated with old age. For explaining that association, more extensive research about social and economic factors related DNR order is necessary in acute cerebral disease.

P-2-39

Acute ischemic stroke in a patient with a native Aortic valve thrombosis

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Background & Significance: Spontaneous native aortic valve thrombosis is very rare event that usually associated with local valve injury after cardiac catheterization or surgery, or occurs as a complication of bacterial endocarditis. we report a case that patient with acute embolic stroke caused by native aortic valve thrombosis without other anatomical or functional cause of embolism, no evidence of any hypercoagulable state or infection process. **Case:** A 39-year-old male was admitted to Emergency room due to decreased mentality with right-side hemiplegia developed 20 minutes ago. He had no history of illness and no previous history of thrombosis or valve dystrophy. Brain MRI showed acute cerebral infarction in the Lt MCA territory and brain CT angiography revealed occlusion of left MCA at M1 segment. Patients treated with IV t-PA and IA thrombolysis within 3 hours of symptom onset. Coronary CT showed irregular, low density, non enhancing filling defect on aortic valve. Transesophageal echocardiogram revealed a echogenic round mass, which a small (1.55 × 0.87 cm) mass was attached on NCC of aortic valve and aortic sinus of Valsalva. it was most likely thrombus than vegetation. To prevent additory risk of embolization to the cerebral or coronary circulation, surgery was performed and thrombus on the aortic valve was removed 8 days after the stroke. A pathologic report was not achieved due to loss of specimen during operation. Seven months after surgery the patient is doing well. **Conclusions or Comments:** Spontaneous native aortic valve thrombosis is rare but this case suggests that it could be a possible sources of embolism and acute ischemic

stroke.

P-2-40

A case of a negative DWI MRI within 12 hours of acute ischemic stroke symptoms

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Background & Significance: Diffusion weighted image(DWI) in MRI is the most sensitive tool for detection of ischemic stroke, even in 1hours from first onset. But its sensitivity can not approach to 100%, so the term 'Diffusion-negative stroke' was made. Diffusion-negative stroke has been known to be more frequent in posterior circulation ischemia, mainly in brain stem than in anterior circulation. We present a case of diffusion-negative stroke in anterior circulation, which was confirmed as ischemic stroke in F/U DWI-MRI. **Case:** A 62 years old women arrived to the emergency department with dysarthria with hemiparesis of right upper and lower extremities. Her neurologic Symptoms emerged about 1 hour and 20minutes before the arrival. And the Symptoms were spontaneously resolved just before arrival. Her vital signs showed: Blood pressure 140/90 mmHg, Blood sugar 110mg/dl, Body temperature 36.8°C. She was on long term medication due to known COPD, but no other regular medication. the Electrocardiography showed normal sinus rhythm. Brain CT was normal findings. So aspirin 300mg was administered to her immediately and MRI was done in series. DWI MRI didn't show any significant abnormality. T1, T2, SWI and enhanced T1 images, either. Of course, MRA was also normal findings. But only perfusion MRI (TTP) showed subtle minimal perfusion deficit in left basal ganglia(globus pallidus) and corona radiata. She was hospitalized for closed observation for neurologic symptom and treated with oral clopidogrel in accordance with TIA. 3hours after admission, she complained of right sided hemiparesis and slurred speech again. MRC grades were 3 in right upper and lower limbs. Her symptoms continued to the next day morning, so we tried follow up DWI-MRI, which was done at 12 hours after the first onset of acute stroke symptoms. But DWI was negative. And soon her symptoms was resolved again. In the next day(HD#3), she showed dysarthria and mild right sided hemiparesis(MRC grade 4). We repeated diffusion & perfusion MRI once more. Finally, DWI showed very subtle high signal intensity in left basal ganglia and corona radiata, which correlated with the perfusion deficit area in previous perfusion MRI. Of course, f/u perfusion MRI showed no significant difference from the previous study, which had showed perfusion deficit in left basal ganglia(globus pallidus) and corona radiata. her symptoms were continued. she was treated with antiplatelet agent, anti-hypertensive drugs and statins, in accordance with ischemic stroke (TOAST classification: small vessel occlusion). After acute management and evaluation of stroke, she was discharged at the hospital day#6 with mild sequelae of right sided hemiparesis, but could gait alone without any help. **Conclusions or Comments:** Diffusion MRI imaging has been noted with the most sensitive and reliable tool for diagnosis of acute ischemic stroke. In acute cell death, the failure of sodium-potassium ATPase pumps induces the shift of water from extracellular space to intracellular compartment. The intracellular shift of water makes high signal intensity in DWI. So we can detect acute ischemic lesion in DWI. But this patient did not showed any abnormal finding in DWI. We propose that the DWI can be the most sensitive tool but, even in negative result in DWI, we can not rule out ischemic stroke perfectly. So close monitoring for the patient's neurologic symptoms are necessary and we should be always concerned about DWI negative stroke.

P-2-41

Cavernous malformation in pineal gland

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Background & Significance: Cavernous malformation is one of the most common vascular malformation of the CNS. It occurs throughout cerebral hemisphere with diverse clinical symptoms including seizure, focal neurologic deficit and potentially lethal hemorrhagic stroke. Although pineal gland is one of the rarest sites for cavernous malformation, its presence can lead to detrimental hemorrhage and hydrocephalus due to anatomic position of pineal gland, which is near third ventricle and tectum of midbrain. We report a case of pineal gland cavernous malformation. **Case:** We experience a 53-year-old female patient who visited our clinic for headache and presyncopal attacks. No neurologic deficit was present on her visit. Brain MRI was performed and revealed 9mm pineal gland mass lesion appearing heterogeneous on T1 image, and high signal with hypo-signal rim, suggesting various bleeding including subacute hemorrhage. (Figure 1.) She referred to neurosurgery department and did not visit our clinic afterward. **Conclusions or Comments:** Hemorrhage in pineal gland with relevant symptoms casts on diagnostic possibilities including germ cell tumor, pineal cyst, and vascular malformation. Vascular malformation such as cavernous malformation in pineal gland is extremely rare, but bleeding can lead to detrimental result such as hydrocephalus and re-bleeding rate is high, prompting surgical removal. Therefore, when brain MRI suggests unequivocal cavernous malformation with relevant symptom, clinicians must consider surgical resection of cavernous malformation. In previous reported literature, there have been 16 cases of pineal cavernous malformation. Patients with pineal cavernous present with severe headache, upward gaze palsy, and diplopia. 1, 2, 3 Total resection of the tumor is generally recommended, unless high surgical risk obliterates surgical exploration. 2, 4 In Korean literature, our patient represent the third reported case. 2

P-2-42

Are the anomalous vertebral arteries more hypoplastic? CT and MR angiographic analysis

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Background & Objectives: Vertebral artery hypoplasia (VAH) is an important risk factor for posterior circulation ischemia. However, no standard criteria exist on the definition of VAH. VAH is the product of the anomalous development of the vertebral artery. This study was undertaken to assess whether vertebral artery anomalies contributes to the diameters of the vertebral artery. **Method:** We retrospectively included subjects who underwent neck CT and MR angiography within a month, simultaneously. We defined V1 anomaly as the abnormal origin of the VA in MR angiography and V2 anomaly as the VA not passing through the C6 transverse foramen in axial CT scan. Linear mixed model with the side of the VA as within subject variable was used to evaluate possible determinants for the size of VAs. **Results:** A total of 238 subjects included in the final study population; proportion of female (41.6%) and age (65.9 ± 12.1 years). Twenty-four subjects (10.1%) had more than one anomaly of its origin or the transverse foramen. Among 476 vertebral arteries, 2.3 percent (11/476) of vertebral arteries had anomalous origin of the aorta, directly. Additionally, 27 out of 476 (5.7%) vertebral arteries had an abnormal entrance to the transverse foramen not through the C6 transverse foramen. In linear mixed model analysis of the vertebral arterial diameter, male (0.2mm larger than female, $p=0.015$), right vertebral artery (0.4mm smaller than the left one, $p<0.001$), V1 anomaly (0.9mm smaller, $p<0.001$) and V2 anomaly (0.8mm smaller, $p<0.001$) was a significant predictor for the vertebral arterial diameter. **Conclusion:** Vertebral arteries with the aortic origin or abnormal entrance to

the C6 transverse foramen is smaller than the others. These developmental characteristics of the vertebral artery might be a clue for the VAH.

P-2-43

Acute ischemic stroke caused by IgA nephropathy without nephrotic syndrome

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Background & Significance: IgA nephropathy is one of the most common glomerulonephritis, which can be lead to nephrotic syndrome. Its another complications are progressive chronic kidney disease, vascular disease such as coronary heart disease and cerebrovascular disease, although there is the exact mechanism is highly contentious. IgA nephropathy induced acute ischemic stroke have been reported only the case provoked by nephrotic syndrome. We report a patient with ischemic stroke caused by IgA nephropathy without nephrotic syndrome. **Case:** A 21-year-old male was found out aphasia about 2 hours and a half ago. He was taking prednisolone 30mg daily after diagnosed as IgA nephropathy. In neurological examination, sensory aphasia was found and MRC grade of right side was IV. Initial his laboratory findings were revealed as follows; BUN/Cr 18.8/2.84, eGFR 30.3ml/min/1.73m², albumin 3.21g/dl, triglyceride 158.5mg/dl, LDL-cholesterol 154mg/dl and proteinuria was found(urine protein 3+ and urine microalbumin 575mg/l). Kidney sonography presented with increased cortical echogenicity and cortical thinning of both kidney and then he was diagnosed chronic kidney disease. Because sequential laboratory examination to exclude other causes of chronic kidney disease had no specific findings, we considered that chronic kidney disease was induced by IgA nephropathy. Diffusion weighted magnetic resonance image showed cortex involving left MCA territory infarction and magnetic resonance angiography showed distal MCA occlusion. Transthoracic echocardiography showed ejection fraction 28%, left ventricular global hypokinesia. However, coronary angiography and magnetic resonance image of heart didn't show coronary heart disease and myocarditis. So, heart failure was considered to be induced by chronic kidney disease. Warfarin was started and neurological deficits including aphasia was slightly improved during hospitalization. **Conclusions or Comments:** IgA nephropathy induced acute ischemic stroke have been reported so far only the case induced by nephrotic syndrome which cause thrombogenic condition. In this case, acute ischemic stroke was due to heart failure caused by IgA nephropathy without nephrotic syndrome. Therefore in early stage of IgA nephropathy, active initial management such as normovolemic state, plasma lipid abnormality, proteinuria, blood pressure control and infection control is necessary to prevent chronic kidney disease which can be lead to vascular events.

P-2-44

Clinical outcomes associated GI bleeding following ischemic stroke

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Background & Objectives: Gastrointestinal (GI) bleeding is a potentially serious complication after stroke. Some reports show that GI bleeding is associated with poor clinical outcomes after ischemic stroke. During or after GI bleeding period, that many clinician stop or change stroke preventive medications such as antiplatelet (AT) and anticoagulant (AC) therapy, which might increase stroke recurrence. The aim of this study was to evaluate the incidence of GI bleeding and its clinical outcomes regarding to adjustment of stroke preventive medications after GI bleeding in patients with ischemic stroke.

Method: We collected 3060 patients admitted with ischemic stroke from January 2010 to December 2014. GI bleeding was defined as any episode of fresh blood or coffee ground material in nasogastric aspirate, hematemesis, melena or bloody stool on admission or during hospitalization. Among them, 72 patients underwent endoscopy and confirmed GI bleeding. We examined the incidence of GI bleeding and reviewed the change of stroke preventive medication such as AT and AC before and after GI bleeding. Also, the correlation of medication change and clinical outcome during 6 months follow-up period analyzed in 54 patients excluding 18 patients of follow-up loss. **Results:** Total seventy-two patients (2.35%) experienced GI bleeding after stroke. The source of GI bleeding was the upper GI tract in 69% of the cases; causes included peptic ulceration (62.5%) and malignancies (6.5%), and other or unidentified causes accounted for 31%. Total 54 patients were followed up for 6 months. In these patients, 41 patients suffered GI bleeding during hospitalization for treatment of acute ischemic stroke(GIBH). Other 13 patients suffered GI bleeding during out-patient clinic follow-up period(GIBO). Among 41 patients of GIBH, 30 patients started first stroke preventive medications and 11 patients have been medicated because of previous cardiac disease or ischemic stroke. 30 patients discharged with following medications; mono AT (9/30), dual AT (11/30), mono AT plus AC (5/30), AC (2/30) and no stroke preventive medication (3/30). In other 11 patients, medications were adjusted from dual -> mono AT (3/11), dual AT plus AC -> mono AT plus AC (3/11), mono -> mono AT (aspirin -> clopidogrel or cilostazol) (3/11) and discontinued (2/11). In 13 patients of GIBO, medication were adjusted from dual -> mono AT (1/13), dual AT plus AC -> mono AT plus AC (1/13), mono -> mono AT (aspirin -> clopidogrel or cilostazol) (2/13) and discontinued (3/13) etc. During 6 months follow-up period, recurrent GI bleeding was not occurred. The stroke recurrence in GIBH occurred in 9.96%(4/41) and in GIBO did not occur. All-cause death in GIBH was 4.88%(2/41) and in GIBO 15.4%(2/13). **Conclusion:** Our study showed 2.35% incidence of GI bleeding in patients with ischemic stroke, mostly due to peptic ulcer, which compatible with previous other studies. When GI bleeding during medication for ischemic stroke was occurring, it seems that using fewer number of drug and less GI toxic drug is available without increasing stroke recurrences. However, after large randomized controlled study, systematic treatment guideline for selection of stroke preventive medication after GI bleeding in ischemic stroke is necessary.

P-2-45

Effects of 5-lipoxygenase inhibitors on intracranial hemorrhage

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Background & Objectives: 5-Lipoxygenase is one of the major enzymes in leukotrienes biosynthesis and is widely recognized as an important role in acute inflammation and asthma. The 5-lipoxygenase inhibitor has been reported to have neuroprotective and antiapoptotic effects in ischemia models. Inflammation is observed in ICH and contributes to brain injury in several ways and its treatment reduces edema formation and tissue injury and improves outcome in ICH models. In this study, the authors examined whether a 5-lipoxygenase inhibitor reduces cerebral edema and hemorrhage after intracerebral hemorrhage (ICH). **Method:** Male Sprague-Dawley rats weighing 200 to 220 g were used in these experiments. Rats were subjected to ICH with or without administration of zileuton, a 5-lipoxygenase inhibitor. Experimental ICH was induced by the stereotaxic intrastriatal administration of bacterial collagenase. We evaluated hemorrhage volume, brain water content, and whole blood leukotriene B4 level. Seventy-two hours after the induction of ICH the rats were anesthetized and sacrificed for measuring water content

(n=6/group)). Water content was expressed as a percentage of wet weight. Hemorrhage volume was quantified at 72 h via a spectrophotometric assay. Measurements from perfused brains subjected to ICH were compared with the standard curve to obtain data in terms of hemorrhage volume (n=6/group). Leukotriene B4 level was measured using an enzyme-linked immunosorbent assay kit. **Results:** Immunoassay showed lower levels of leukotriene B4 level in the group treated with zileuton compared to the group without zileuton administration. A trend to have lesser brain water content was seen in the zileuton-treated group compared to the group without zileuton administration, although a trend to develop larger hemorrhage volume was observed. **Conclusion:** Zileuton may reduce cerebral edema following intracerebral hemorrhage. Though beneficial effects of 5-lipoxygenase inhibitors were implied in various models of neurological diseases such as cerebral ischemia or epilepsy, the benefits in hyperacute stages of intracerebral hemorrhage is questionable due to larger hemorrhage volumes seen in the group treated with zileuton. Considering 5-lipoxygenase inhibitors may alter platelet function and lead to a larger hemorrhage volume, different administration schedules must be investigated to minimize the harm. Long-term functional outcome may also be studied to elucidate the effects.

P-2-46

Rt. Pontine tegmental infarction with contralateral ataxia

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Background & Significance: The symptoms of pontine tegmental infarction are paralysis of ipsilesional conjugate gaze, abducens nerve palsy, facial palsy, trigeminal sensory involvement, contralateral hemiplegia or pure sensory syndrome. Ataxia are presented when cerebellar structure or related tract is damaged. Here, we report unusual case of pontine tegmental infarction showing contralateral ataxia and tremor. **Case:** 68 year-old man visited ER due to abrupt onset of non-whirling dizziness and left side falling tendency. On the neurological examination, nystagmus was not shown and eye position was neutral with no gaze limitation. He had mild to moderate ataxia on finger-to-nose and heel to shin testing on the left side. On attempted tandem gait he fell to the left side. He also showed tremulous movement of left upper extremity. Diffusion-weighted magnetic resonance imaging(MRI) showed high signal intensity in right pontine tegmentum and ADC map revealed restricted diffusion in the same area. Magnetic resonance angiography (MRA) showed no definite abnormality. **Conclusions or Comments:** Pontine tegmental structure is associated with multiple nuclei and fiber tracts. As affecting those structures, motor or sensory symptoms may present. In our case, main symptoms were ataxia and tremor of contralateral side. There are several reports that pontine lacunar infarction can cause contralateral ataxia. However, concomitant presence of contralateral ataxia and tremor is very rarely reported in patient with pontine tegmental infarction.

P-2-47

Brain abscess masquerading as brain infarction

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Background & Significance: Occasionally, patients can present with the sudden onset of focal neurological symptoms, and it can be difficult to differentiate cerebral abscess from acute ischemic stroke on clinical grounds. To illustrate this potential pitfall, we present the case of a patient with a brain abscess who was initially misdiagnosed as having an acute cerebral infarction based on his clinical and imaging presentation. **Case:** We describe a patient

who presented with sudden onset right hemiparesis and fever. MRI was consistent with an acute stroke, showing multiple lesions with restricted diffusion in the left middle cerebral artery territory. These lesions were not enhancing and were not associated with vasogenic edema. A diagnosis of acute stroke was made based on clinical and radiographic data. Follow-up MRI obtained after eleven days showed interval development of ring enhancement and vasogenic edema surrounding the previously noted core of restricted diffusion. Based on these findings the diagnosis was revised to cerebral abscesses and the patient was treated successfully with antibiotics. In retrospect, the largest DWI lesion on baseline MRI demonstrated two characteristics that are atypical for stroke: it had an ovoid shape and a subtle T2 hypointense core. **Conclusions or Comments:** This case demonstrates that the acute clinical and radiographic presentation of cerebral abscess and ischemic stroke can be strikingly similar. Follow-up imaging can be instrumental in arriving at an accurate diagnosis.

P-2-48

A case of acute subdural hemorrhage after carotid artery stenting

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Background & Significance: As the endovascular techniques improve, the incidence of ischemic stroke following carotid artery stenting(CAS) have markedly reduced. As a result, Intracranial hemorrhage (ICH) and hyperperfusion syndrome(HPS) became major causes of morbidity and mortality following CAS. ICH and HPS have been reported as serious complications in <1% of CAS procedures. To our knowledge, there is no prior report of subdural hemorrhage(SDH) subsequent to CAS. Here in, we report a case of acute SDH as an immediate complication just after CAS **Case:** A 42-year-old woman presented with a transient episode of amaurosis fugax and right sided weakness lasting for 15 minutes a few days ago. There was no past medical or family history. The MRI showed the left internal borderzone infarction and the MRA showed a severe stenotic lesion in the short segment of the proximal portion of the left internal carotid artery. Laboratory tests including complete blood cell count, routine chemistry and coagulation studies were all within normal range. Dual antiplatelet therapy with aspirin plus clopidogrel was initiated. On the second day of admission, CAS was performed. During the procedure, before guide wire passage, the patient was anticoagulated with an 5000 IU (80IU/kg) heparin bolus. Selective angiography confirmed 90% narrowing in diameter in the long segment of the left internal carotid artery. Wallstent was implanted successfully, and the final angiogram confirmed excellent result and brisk blood flow. After CAS the patient was admitted to stroke unit and Electrocardiogram, blood pressure monitoring and frequent neurological examination were done, The blood pressure (BP) varied between 140/86 mm Hg and 125/73 mm Hg, and mental status remained alert without any neurological deficit. However, about 2 hours after CAS, severe headache developed, and mental status rapidly evolved to stupor, The BP remained at 155/61 mm Hg. Sluggish reacting pupils were found. Brain CT revealed acute SDH on the left side with slight shift of the midline structures. At that time, there was no history or physical evidence of preceding head trauma. And Follow-up laboratory tests were all normal except active partial thromboplastin time(aPTT) 96.6 seconds(29.1-45.1), supposed to be the result from administration of intravenous heparin during CAS. Three and a half hours after the CAS, her level of consciousness deteriorated to coma and bilateral pupils dilated without reactivity. Decompressive craniectomy and hematoma removal were immediately undertaken. At operation, the left frontotemporoparietal craniectomy revealed an extensive subdural hematoma. Removal of large amount of fresh clot disclosed bleeding point at a left cortical middle cerebral artery on the superior temporal gyrus. There was no apparent damage to the underlying cortex but

swollen. Neither aneurysm nor arteriovenous malformation was found around the bleeding site. The postoperative recovery was unsatisfactory and the patient was remained comatose. **Conclusions or Comments:** We present a patient who experienced an acute SDH within 2 hours after CAS. In this case, there is no evidence of head or neck trauma, vascular abnormalities. And the arterial origin of bleeding had been verified at operation. The pathological mechanism of SDH in this case remains unclear. We postulate that SDH may be caused by sudden rise in intracranial pressure due to the hyperperfusion after CAS which displaced brain tissue within the cranium and disrupted a corticodural bridging artery and heparin bolus infusion during CAS also attributed partly. To our knowledge, this is the first case report of SDH subsequent to CAS.

P-2-49

Prognostic impact of the collateral status in the vertebrobasilar occlusive stroke

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Background & Objectives: Although therapeutic time window for revascularization in the acute vertebrobasilar occlusive stroke is known to be longer than anterior circulation stroke, there is no conclusive date regarding therapeutic time window. So, we analyzed prognostic impact of collateral status using the baseline angiography in patients achieved successful endovascular recanalization for vertebrobasilar occlusive stroke. **Method:** We analyzed 33 consecutive patients from our prospective collected registry. Inclusion criteria were follows: (1) basilar arterial occlusion, unilateral vertebral artery occlusion with extension to the proximal basilar artery or contralateral hypoplastic vertebral artery, (2) Time from symptom onset to groin puncture was within 8 hours, (3) endovascular treatment using current generation devices (stent-retrievers or penumbra catheter). The American Society of Interventional and Therapeutic Neuroradiology/Society of Interventional Radiology collateral grading system was used for evaluating collateral status. **Results:** Baseline NIHSS score was not statistically different according to the collateral status (18.94 vs 19.18, $p=0.073$). Symptom onset to reperfusion time (252 min vs 312 min, $p=0.073$) and endovascular procedural time (49 min vs 74 min, $p=0.069$) tended to be longer in the poor collateral group. Favorable outcome was frequent in the good collateral group (50% vs 5.9%, $p=0.004$) On linear regression analysis, good collateral status (odds ratio 13.674 [1.272-147.002], $p=0.031$) was independently associated with Favorable outcome **Conclusion:** Good collateral status is an important prognostic factor for clinical outcome regardless of time to reperfusion. Noninvasive imaging technique for identify collateral status is needed, it will be aid for screening a candidacy for reperfusion therapy.

P-2-50

Cerebral small vessel disease score determines short and long-term prognosis in acute ischemic stroke patients

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Background & Objectives: The recently developed "total small vessel disease (SVD) score" might appropriately reflect total burden or severity of cerebral SVD better than considering multiple SVD pathologies as separately. We investigated that whether total SVD score could predict short and long-term outcome and type of mortality after index stroke in acute ischemic stroke

patients. **Method:** In total, 1096 consecutive ischemic stroke patients, who underwent brain magnetic resonance imaging, were enrolled. We investigated presence of cerebral microbleeds (CMBs), white matter hyperintensities (WMHs), perivascular spaces (PVs) and asymptomatic lacunar infarctions (ALIs). Functional outcomes at three months were determined based on the modified Rankin scale. We determined the date and causes of death from Korean National Statistical Office, which were identified based on death certificates. **Results:** CMBs were found in 26.8% of subjects (294/1096), HWCs in 16.4% (180/1096), HPVs in 19.3% (211/1096) and ALIs in 38.0% (416/1096). After adjusting age, sex and variables with $p < 0.1$ in univariate analysis (smoking (inversed relation), previous stroke, thrombolysis, stroke subtype and NIHSS score), the "total SVD score" was independently associated with poor functional outcome (odds ratio (OR): 1.22, $p = 0.006$). In multivariate Cox regression analyses, total SVD score was a predictor for all-cause (hazard ratio (HR): 1.20, $p = 0.001$), ischemic stroke (HR: 1.22, $p = 0.015$) and hemorrhagic stroke mortalities (HR: 2.08, $p = 0.002$) but not cardiac related death (HR: 1.23, $p = 0.382$). **Conclusion:** Total SVD score is a powerful predictor and potential image biomarker in acute ischemic stroke patients for short-term functional outcome and long-term prognosis.

P-2-51

Paradoxical role of vascular stiffness in lacunar stroke in progression

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Background & Objectives: Although the progressing lacunar infarction consists of 12 to 36% of all lacunar infarctions, the clinical predictors and therapeutic methods for progression are not clear. We investigated the role of vascular stiffness in lacunar infarction with motor progression. **Method:** Consecutive 2427 patients over 4 years, who were diagnosed with stroke at the Gachon University Gil Hospital, were reviewed retrospectively. We analyzed lacunar infarction patients in aspect of clinical, laboratory, and pulse wave velocity. Motor progression is defined as one or more increase in the motor score in NIHSS. Good outcome was designated as a modified Rankin scale 0-2 at discharge. **Results:** Among all 662 patients who had lacunar infarct, 66 patients experienced motor progression (9.97%). The induced- hypertension therapy group ($n=25$) received phenylephrine and conventional group ($n=41$) received anticoagulation such as heparin, volume expansion, or both. Although there were not significantly different from baseline clinical and laboratory findings (ie. age, sex, stroke risk factors, initial BP, and NIHSS), motor progression group showed significantly more frequent BP drop at progression ($p < 0.0001$) and higher pulse wave velocity ($p = 0.001$). Induced- hypertension group (vs. conventional group) had a lower NIHSS and good outcome at discharge. In multiple regression analysis, pulse wave velocity (odds ratio, OR 1.005, 95% CI 1.001-1.009, $p = 0.021$) was independent predictor for good outcome in the induced-hypertension group. Side effects of phenylephrine treatment was dysuria ($n=1$). **Conclusion:** The present study suggests that vascular stiffness can be not only a predictor for motor progression but also a predictor for motor improvement after induced- hypertension therapy using phenylephrine in lacunar stroke.

P-2-52

Higher pulsatility index of the middle cerebral artery is associated with lacunar stroke progression

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Background & Objectives: Stroke progression in patients with lacunar infarction is not an uncommon phenomenon. The pulsatility index measured by transcranial doppler sonography (TCD) reflect downstream arterial resistance and its association with small vessel pathology have been proposed. Herein, we present clinical and sonographical factors associated with stroke progression within 7 days after symptom onset in lacunar infarction. **Method:** From total 4,478 acute ischemic stroke patients who had admitted to the tertiary academic stroke center between January 2002 and January 2015, we selected lacunar infarction cases which occurred in the middle cerebral artery (MCA) territory. MRI findings of each case were thoroughly reviewed. Patients with cardiac or proximal embolic sources were excluded. Stroke progression was defined as increment of NIH stroke scale (NIHSS) by two or more points or motor scale of NIHSS by one or more points within 7 days after stroke onset. TCD data of the ipsilateral middle cerebral artery at the depth of 64 to 68mm were collected. Baseline demographic information and clinical data were also gathered during hospitalization. **Results:** A total of 89 lacunar infarction cases were analyzed. Among them, 26 (29.2%) patients experienced stroke progression. The patients who experienced stroke progression had more history of diabetes (38.5% vs. 17.5%, $P = 0.034$), and higher HbA1c ($6.23 \pm 0.80\%$ vs. $5.97 \pm 0.74\%$, $P = 0.033$) than those who did not. Initial NIHSS (3 (2-4.75) vs. 2 (1-3)) and functional outcome at discharge (modified Rankin scale, 1 (1-2.75) vs. 1 (1-1)) was poorer in progression group. Pulsatility index was higher in the progression group among lacunar infarction patients (0.99 ± 0.19 vs. 0.90 ± 0.14 , $P = 0.048$). By the multivariate analysis, higher pulsatility index was associated with progression in lacunar infarction patients (OR 1002.45, 95% CI 11.95-84101.39, $P = 0.002$) after adjusting age, sex, history of diabetes and initial NIHSS. **Conclusion:** This study demonstrates clinical and sonographical factors associated with stroke progression in lacunar infarction patient. Especially, higher pulsatility index was associated with stroke progression. This might indicate underlying pathophysiology of lacunar stroke progression.

P-2-53

Prevalence and characteristics of unruptured cerebral aneurysms in ischemic stroke patients

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Background & Objectives: The prevalence of unruptured cerebral aneurysms (UCAs) in ischemic stroke patients has not been investigated. The purpose of this study was to measure the prevalence and characteristics of UCAs in acute ischemic stroke (AIS) and compare our findings with those of the general population. In addition, we investigated the factors associated with cerebral aneurysms in AIS patients. **Method:** We retrospectively reviewed brain magnetic resonance angiography (MRA) of 955 patients with AIS and 2,118 controls who had received a brain MRA as part of a health check-up. We investigated the prevalence, size, location and risk factors of the subjects in the context of UCAs. **Results:** UCAs were found in 74 patients with AIS (7.7%) and in 79 individuals who received a health check-up (3.7%). The prevalence of UCAs was significantly higher in the AIS group compared with the health check-up group (OR 2.17, 95% CI 1.56-3.01, $p < 0.001$). The mean aneurysm diameter was larger in AIS group than the health check-up group (3.95 mm vs 3.10 mm, $p = 0.01$). UCAs were primarily located in the internal carotid artery, and there were no significant differences in the pattern of UCA distribution between the two groups. According to multivariate analysis, only hypertension ($p = 0.028$) was correlated with an increased prevalence of UCA in stroke

patients. **Conclusion:** This study identified a higher prevalence and larger size of UCAs in AIS patients compared with the general population. Hypertension was associated with the coexistence of cerebral aneurysms and ischemic stroke.

P-2-54

Influence of the interval between serum levels of admission and fasting glucose on the functional outcome in hyperglycemic patients with acute ischemic stroke

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Background & Objectives: The previous studies have demonstrated that elevated admission or fasting glucose increases the risk of worse outcome in ischemic stroke. In this study, we investigated the impact of the interval between serum levels of admission and fasting glucose on the functional outcome in hyperglycemic patients with acute ischemic stroke. **Method:** From January 2011 to December 2014, we retrospectively recruited ischemic stroke patients admitted within 24 hours who had admission hyperglycemia. Hyperglycemia was defined as serum glucose > 7.8 mmol/L. Blood samples for the fasting glucose level were taken in the next morning following admission. We performed multivariate analysis to determine whether the interval between admission and fasting glucose levels is an independent factor to determine the functional outcome, which was assessed by measuring the percent decrease between the levels of admission and fasting glucose. Unfavorable outcome was defined as MRS 4-6 at 3 months. **Results:** Of the 274 patients recruited, 177 (65%) were male and the mean age was 67.5±11.4 years. Diabetes mellitus was found in 168 (61.3%) patients. The mean serum levels of admission and fasting glucose were 11.3±4.1 mmol/L and 7.4±1.9 mmol/L, respectively, with a mean percent decrease of 32.0±15.7%. On multiple regression analysis, higher fasting glucose (OR 1.249, 95% CI 1.028-1.518, P=0.025) and lower percent decrease of glucose level between admission and fasting state (OR 0.972, 95% CI 0.946-0.998, P=0.036) were significant and independent predictors of unfavorable outcome. Functional outcomes according to the percent decrease of glucose level reached statistical differences in tertile analysis, which demonstrated the patients with lower percent decrease had higher prevalence of unfavorable outcome (P<0.001). **Conclusion:** This study showed that the lower interval between the serum levels between admission and fasting glucose were independently associated with unfavorable outcome in hyperglycemic patients with acute ischemic stroke. We suggest that not only a single glucose measurement but also the pattern of change should be addressed in the prediction of stroke outcome.

P-2-55

A case of contrast leakage mimicking intraventricular hemorrhage in patient with intravenous thrombolysis

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Background & Significance: Contrast leakage on non-enhanced brain CT is a well-known phenomenon after intravenous thrombolysis (IVT) or intra-arterial thrombolysis. However, it is sometimes difficult to differentiate from intracranial hemorrhage. Recently, we experienced a case of contrast leakage mimicking intraventricular hemorrhage in patient with IV tPA. We describe here our case. **Case:** A 66-year-old male with hypertension presented with a sudden-onset left hemiparesis. Neurologic examination revealed dysarthria, left facial palsy, left hemiplegia, and left hemineglect (NIHSS score of 7). His blood pressure was 130/80 mmHg. Initial non-enhanced brain CT re-

vealed no hemorrhage. CT angiography revealed M1 occlusion of right middle cerebral artery. IVT was started within 100 minutes after onset and administered according to standard regimen (alteplase of 0.9mg/kg). His NIHSS score was immediately improved from 7 to 4 after IV tPA. Non-enhanced CT after IV tPA was immediately performed, which showed fluid collection with hyperintensity in both lateral ventricle. However, the fluid collection with hyperintensity did not look as dark signal on susceptibility weighted imaging (SWI). The fluid collection with hyperintensity was rapidly decreased on the second follow-up CT, and almost completely cleared on the third follow-up CT. In the meantime, his neurological status remained stable. **Conclusions or Comments:** Our case shows that contrast leakage into lateral ventricle could be misinterpreted as intraventricular hemorrhage after IVT and SWI may be helpful to differentiate the contrast leakage from intracranial hemorrhage after IVT.

P-2-56

Successful repeated mechanical thrombectomy in patients with recurrent basilar artery occlusion

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Background & Significance: The top of basilar artery syndrome is associated with extremely poor outcome and high mortality. Although intra-arterial mechanical thrombectomy is reported to have high recanalization rate and favorable outcome, the safety and feasibility of repeated mechanical thrombectomy in patients with recurrent basilar artery occlusion is unknown. We report a patient who underwent repeated mechanical thrombectomy for recurrent top of basilar syndrome in acute period of ischemic stroke. **Case:** A 64-year-old woman arrived emergency room with sudden vertigo and diplopia. She had no remarkable past medical history. Three days before, she had transient vertigo and left arm weakness for 1 hour. Her initial NIHSS score was 2. Brain MRI showed acute infarction on the bilateral cerebellum, the thalamus, and the occipital lobe. MRA revealed occlusion on tip of the basilar artery with collateral from bilateral the posterior communicating arteries. The left proximal vertebral artery and the subclavian arteries were also occluded. She suddenly got drowsy 3.5 hour after admission. Follow-up neurological examination revealed the NIHSS score was worsened to 7. Mechanical thrombectomy targeted to the basilar tip occlusion was performed through the right vertebral artery. After recanalization, she recovered to the NIHSS score 0. On evaluation, she did not have any potential cardiac sources of embolism. Laboratory findings revealed that positive of the anti-cardiolipin antibody and anti beta2glycoprotein antibody. She received dual antiplatelet with aspirin and clopidogrel. On the hospitalization day 6, she suddenly got stupor, and the NIHSS score was 22. CTA revealed same site occlusion on the basilar tip. Second mechanical thrombectomy was performed and complete recanalization was achieved. She returned to previous neurological state except dizzy feeling. She discharged without neurological deficit. **Conclusions or Comments:** We report a successful case of repeated mechanical thrombectomy for recurrent the basilar artery occlusion. In regard of intracranial hemorrhage, repeated mechanical thrombectomy might be attractive treatment option in acute period of ischemic stroke.

P-2-57

A case of sudden hearing loss in PICA infarction

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Background & Significance: It is well known that acute audiovestibular loss commonly occurs in acute ischemic stroke around the AICA (anterior inferior cerebellar artery). But acute hearing loss associated with non-AICA territory cerebellar infarction has been reported rarely. The patient was initially diagnosed as left PICA infarction, but complained of ipsilateral hearing loss. **Case:** A 77-year-old woman visited ER for vertigo. She had been treating hypertension and diabetes mellitus for 7 years. Neurologic examination revealed right gaze-evoked nystagmus and head impulse test turned out negative. Brain MRI showed acute infarction of left PICA territory and MR angiography showed no significant stenotic lesion of both intracranial and extracranial arteries. The Video-Nystagmogram(VNG) and caloric test suggested central origin nystagmus, which showed right beating nystagmus in all positions and 20% of canal paresis in left ear. We started 100mg of aspirin for the treatment of stroke. On the second day of admission, she started complaining hearing impairment of left ear. Pure tone audiogram demonstrated a sensorineural deafness. After a week, vertigo improved, but deafness did not disappear despite steroid treatment. **Conclusions or Comments:** We report a case of sudden unilateral hearing loss associated with PICA infarction.

P-2-58

Middle cerebral artery infarction caused by large-sized artery vasculitis in systemic lupus erythematosus

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Background & Significance: Systemic lupus erythematosus (SLE) is a autoimmune disease with a wide variety of clinical and serological manifestations that may affect multiple organs. Vasculitis prevalence in SLE is reported to be between 11 % and 36 %, however, involvement of cerebral large vessel artery is rare. We report a patient who had been diagnosed as SLE, who developed left middle cerebral artery infarction and severe stenosis at left middle cerebral artery caused by vasculitis. **Case:** A 32-year old female patient with systemic erythematosus lupus visited rheumatology outpatient clinic due to her speaking difficulty and subjective right side weakness. She was diagnosed as SLE in two years prior to this event by malar rash, thrombocytopenia, arthritis, and ANA positive by ACR criteria. However her compliance to medical treatment was not good and lost follow up. She presented with headache at her left temporal area with throbbing character and mild nausea during one week. She admitted at neurology department and performed brain magnetic resonance imaging (MRI) including MR angiography(MRA). Brain MRI/MRA revealed acute infarction on the left middle cerebral artery territory with severe narrowing of left middle cerebral artery. Initial NIHSS was 3. She also underwent digital subtract angiography, multifocal mild stenosis and severe left middle cerebral artery stenosis were compatible vasculitic lesion. Therefore we diagnosed SLE vasculitis, and she underwent 3-day steroid pulse therapy for 1g every day. Transthoracic echocardiography and transesophageal echocardiography revealed mild echogenic turbulence in left atrium with no other cardio-embolic sources. Other laboratory tests revealed 1:80 positive at ANA titration and anti-SS-A antibody. During admission, she had no progression of stroke symptoms and she discharged with prednisolone 60mg daily and hydroxychloroquin 200mg twice a day. At discharge, her NIHSS was 1. **Conclusions or Comments:** Among clinical manifestations of SLE, neuropsychologic SLE is not common, but crucial for an adequate early and aggressive treatment. As seen in our patient, cerebral large vessel vasculitis in SLE should be considered as stroke etiology in patients with young age stroke.

P-2-59

A case of superficial siderosis presenting with progressive hearing loss

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Background & Significance: Superficial siderosis (SS) of the central nervous system (CNS) is a rare syndrome resulting from hemosiderin deposition caused by repeated slow hemorrhage into the subarachnoid space in the subpial layers of the brain and spinal cord. SS of CNS is characterized by sensorineural deafness, cerebellar ataxia and signs of pyramidal tract dysfunction. Magnetic resonance imaging (MRI) in SS of CNS shows the characteristic marginal T2 hypointensity around the brainstem, cerebellum, and spinal cord. We report the case of a 45-year-old male patient who was diagnosed with SS using MRI. The patient presented with progressive hearing loss without any definite neurological complications. **Case:** A 45-year-old man presented with a several months history of hearing disturbance. His hearing disturbance worsened progressively over two months. His medical history was unremarkable without recent trauma history. Initial neurological examination was normal except both sensorineural hearing loss. Initially, the patient was admitted to the department of otorhinolaryngology. However, his gradient echo T2*-weighted brain MRI revealed multiple hypointense lesions around both temporal area and brainstem on image (Figure1). We diagnosed him with SS, and checked his whole spine MRI, a lumbar puncture and transformoral cerebral angiography (TFCA) to evaluate recurrent intracerebral hemorrhage causes. Laboratory tests were normal, consisting routine blood and urine analyses, blood coagulation, liver and kidney functions and blood ion concentrations. In addition, his antinuclear antibody, erythrocyte sedimentation rate, thyroid function and tumor markers were all normal. A lumbar puncture revealed clear and colorless cerebrospinal fluid with a pressure of 135mmH2O. Whole spine MRI scans were normal. TFCA showed a small wide neck aneurysm (2.5mm size) of left posterior cerebral artery (P1) segment (Figure2). 7 days after admission, he discharged with no improvement of his hearing loss. **Conclusions or Comments:** SS is a rare disease caused by chronic repeated subarachnoid hemorrhage. However, our case showed no definite intracerebral hemorrhage causes including trauma history. The characteristic clinical features of SS of CNS are bilateral sensorineural deafness, progressive cerebellar ataxia and pyramidal tract sign. Our case presented with only progressive hearing loss. The present case suggests that the etiology of idiopathic progressive sensorineural hearing loss could be considered SS of CNS.

P-2-60

Delayed reversible cerebral vasoconstriction syndrome following traumatic brain injury: a case report

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Background & Significance: Reversible cerebral vasoconstriction syndrome (RCVS) is an entity characterized by severe headache and transient multifocal narrowing of cerebral arteries which usually resolves spontaneously within 3 months. RCVS can be precipitated by several factors including traumatic brain injury (TBI) that leads to cerebral vasospasm in 19-68% of patients. Vasospasm is a delayed, secondary consequence that can profoundly impact neurological recovery and functional outcome after TBI. Although vasospasm may result from traumatic subarachnoid hemorrhage (SAH), other mechanisms such as blast-induced neurotrauma are increasingly recognized as causative factors. We report a patient presenting with sudden-onset neurological

deterioration that occurred on day 4 after traumatic brain injury, which is associated with delayed RCVS without SAH. **Case:** In March, 2014, a 36-year-old woman presented to our hospital with transient loss of consciousness lasting a few minutes followed by severe headache. Her husband reported spontaneously resolving generalized tonic-clonic convulsions. Since then, she had developed sudden onset, severe occipital headache accompanied by nausea. Four days earlier, she had a history of head trauma following sliding down from the floor. She complained of only mild headache directly after trauma and resolved after a few minutes. She had no medical history and denied substance abuse. On initial examination, her vital sign was within normal range. Neurologic examination showed mild weakness on the right extremities. Magnetic resonance imaging (MRI) of the brain, performed after the administered gadolinium, revealed multifocal high-intensity lesions in the left hemisphere on T2 weighted image. (Fig.A) Magnetic resonance angiography (MRA) showed vasoconstriction of the left inferior branch of middle cerebral artery (Fig.B). Routine blood tests including inflammatory markers, thrombophilia and connective tissue screens were normal. A cerebrospinal fluid (CSF) examination was normal except for mild elevation of protein (151.9mg/dL). On the third day, follow-up MRI was done, which revealed a completely improvement of the segmental vasoconstriction (Fig.C). We concluded that the patient's RCVS resulted in the cerebral infarctions due to the severe vasospasm of regional cerebral artery, most likely because of the head trauma. The arterial vasoconstriction in this case was reversible, but the infarction remained as an irreversible complication. **Conclusions or Comments:** We report the patient with delayed RCVS following traumatic brain injury. Clinical and radiological findings fulfilled the diagnostic criteria of RCVS. Although the pathophysiology remains unknown, the prevailing hypothesis is of a transient disturbance in the control of cerebral vascular tone leading to segmental and multifocal arterial constriction and dilatation. Our patient had a certain time window between the initial injury and delayed-onset neurological symptoms provoked by cerebral vasospasm. This finding is well known phenomenon in patients with TBI combined with SAH, which is believed to be produced by spasmogenic substances generated during the lysis of subarachnoid blood clots which cause endothelial damage and smooth muscle contraction. The vascular endothelium produces nitric oxide, which tonically dilates the cerebral vasculature; endothelial damage may interfere with nitric oxide production, leading to vasoconstriction and an impaired response to vasodilators. In addition, increased release of the potent vasoconstrictor endothelin may play major role in the induction of cerebral vasospasm after SAH. Of great interest is that our patient showed delayed vasoconstriction without SAH following TBI. The mechanism of this phenomenon is unclear but it is considered as a cascade of molecular injury mechanisms that are initiated at the time of initial trauma and continues for hours or days.

P-2-61

Modified dysphagia screening test in stroke patients also reduce the incidence of in-hospital pneumonia

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Background & Objectives: Dysphagia is a common complication in stroke patients. Acute onset dysphagia after stroke is related with an increased risk of aspiration pneumonia. The Gugging Swallowing Screen (GUSS) test is a simple and stepwise bedside screening test. Original GUSS consists of 4 steps, including indirect, semisolid, liquid, solid swallowing test. But solid swallowing test using dry bread is not proper to Korean life style. So we used modified GUSS, including indirect, semisolid, liquid swallowing test, and evaluated the effect of modified GUSS to prevent aspiration pneumonia. **Method:** From January 2007 to December 2009, medical records of 885 patients who admit-

ted for stroke was reviewed. 439 patient was admitted before modified GUSS test and 362 patients was admitted after modified GUSS test. We found patients developed fever ($\geq 38^\circ\text{C}$ or using antipyretic drugs) and classified etiology of fever. Aspiration pneumonia was defined as pneumonia with predisposing conditions, such as reduced consciousness or dysphagic symptoms, or aspiration events. We compared fever and pneumonia rate between before and after modified GUSS test. Chi-square test and Student T test was used. **Results:** Between non modified GUSS (group 1) and modified GUSS group (group 2), general demographics had no statistical significance. The mean age was 63.95 ± 13.08 and 64.25 ± 13.42 ($p=0.609$). Male was 635 (58.9%) and 530 (59.9%) ($p=0.608$) Initial NIHSS score was 5.40 ± 5.80 and 5.17 ± 5.64 ($p=0.375$). Fever was developed at 11.7% in group 1 and 13.2% in group 2 ($p=0.487$). Among the etiology of fever, rate of aspiration pneumonia was changed with statistically significant. (40 patients (8%) in group 1 and 18 patients in group 2, $p=0.021$) Other etiology had no statistical significance. **Conclusion:** Modified GUSS is more simple test than original GUSS. But modified GUSS was also useful test to predict the dysphagia patient and prevent aspiration pneumonia.

P-2-62

Stabilizing course of blood pressure at acute stage of ischemic stroke and 3-month functional outcome

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Background & Objectives: Stroke provoked the unstable and BP response, which is gradually stabilized in following few days. Even though BP at this stage is regarded as important factor for stroke outcome, however, there is limited information how BP is changed at acute stage and its effect on stroke outcome. **Method:** Using prospective stroke registry, we consecutively identified a series of patients who arrived the Seoul National University Bundang Hospital due to ischemic stroke within 24 hours of symptom onset between January, 2010 and November, 2014. Change of BP is estimated by second steps, which are within 24-h standard deviation of systolic BP (SBPSD) (first step) and their day-by-day course during first 3 days (second step). For each day, SBPSD was dichotomized into high (SBPSD > 15 mmHg) and low BPV. Primary outcome was 3-month poor functional outcome (modified Rankin Scale, 3 to 6). Using classification and regression tree method, proportions of poor outcome were classified according to BPV status for first 3 days. **Results:** A total of 2545 patients were enrolled (mean age, 67.1 ± 13.5 year-old; median baseline NIHSS score, 3 (interquartile range, 1-9)). While mean values of SBP were similarly remained 134.5 ± 16.7 at 1st day, 132.9 ± 16.7 at 2nd day and 134.7 ± 16.7 mmHg at 3rd day, SBPSD were changed from 14.4 ± 5.0 at 1st day to 12.5 ± 4.5 at 2nd day and 12.2 ± 4.6 mmHg at 3rd day, respectively. The association between change of BP and primary outcome were demonstrated (Figure). In total subjects, poor outcome was 32%. At first day, poor outcomes of patients with high BPV and low BPV were 37% and 28%, respectively ($P<0.001$). In second day, proportions of poor outcome were divided into 51% of high BPV and 29% of low BPV in patients with high BPV at first day ($P<0.001$) and those divided into 40% of high BPV and 26% of low BPV in those with low BPV at first day ($P<0.001$). With additional classification according to the BPV at third day, proportions of poor outcome were distributed from 25% to 57%. In dividing steps, BPV at 2nd day were most important factor (normalized importance = 43.7%), which followed by 3rd and 1st day. **Conclusion:** In ischemic stroke, high BPV at acute stage is rapidly stabilized at acute stage. When patients sustain or return to the high BPV state at 2nd and 3rd day, they would be associated with poor functional outcome.

P-2-63

Initial glucose fluctuation increased poststroke cardiovascular events in diabetic patients

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Background & Objectives: Recent studies have shown that blood glucose fluctuation in the patients with type 2 diabetes mellitus (DM) is related to recurrent cardiovascular events after acute myocardial infarction (MI). This study attempted to evaluate whether initial glucose fluctuation is associated with cardiovascular events in type 2 DM patients with acute ischemic stroke. **Method:** We recruited 639 consecutive patients with type 2 DM and acute non-cardioembolic infarction within 72 hours in Soonchunhyang university hospital from March 2005 to December 2014. The blood glucose levels were checked 4 times per day in initial 3 days. J-index, coefficients of variation (CV) and standard deviation (SD) were calculated for glycemic variability. We divided the patients into 4 groups according to quartile of glycemic variability: normal, mild, moderate and severe. Composite outcome (nonfatal stroke, nonfatal MI, cardiovascular death), death of all causes and modified Rankin Score (mRS) at 3 months were prospectively captured. Multivariable logistic regression analyses were performed adjusting covariates which can influence on recurrent cardiovascular events (age, sex, NIHSS, previous stroke, atrial fibrillation, significant vessel stenosis, WBC, HbA1C, initial fasting blood sugar and taking anti-thrombotics agent). **Results:** We recruited 639 consecutive patients with type 2 DM and acute non-cardioembolic infarction within 72 hours in Soonchunhyang university hospital from March 2005 to December 2014. The blood glucose levels were checked 4 times per day in initial 3 days. J-index, coefficients of variation (CV) and standard deviation (SD) were calculated for glycemic variability. We divided the patients into 4 groups according to quartile of glycemic variability: normal, mild, moderate and severe. Composite outcome (nonfatal stroke, nonfatal MI, cardiovascular death), death of all causes and modified Rankin Score (mRS) at 3 months were prospectively captured. Multivariable logistic regression analyses were performed adjusting covariates which can influence on recurrent cardiovascular events (age, sex, NIHSS, previous stroke, atrial fibrillation, significant vessel stenosis, WBC, HbA1C, initial fasting blood sugar and taking anti-thrombotics agent). **Conclusion:** In diabetic patients, fluctuating initial blood glucose was significantly associated with cardiovascular events after acute ischemic stroke. Further multicenter studies are needed to confirm the relationship between glycemic variation and poststroke cardiovascular events.

P-2-64

Impact of nocturnal desaturation in the stroke unit on early neurological deterioration following ischemic stroke

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Background & Objectives: Early neurological deterioration (END) is classified as substantial neurological deterioration that occurs following acute ischemic stroke. The mechanisms of END in patients with acute stroke remain unclear. Although hypoxia is associated with stroke risk and poor outcome following acute stroke, the impact of nocturnal desaturation after admittance to the stroke unit (SU) on the risk of END is not completely understood. We aimed to investigate the relationship between nocturnal oxygen desaturation (NOD) in the SU and END in patients with acute stroke. **Method:** A total of 225 patients with acute ischemic stroke or transient ischemic attack (TIA) who were

admitted to the SU within 7 days after stroke onset were evaluated retrospectively. NOD was evaluated based on the oxygen desaturation index (ODI) using oxygen saturation minute data during 9 hours of the first night (10:00 PM to 7:00 AM) following SU admission. The patients were divided into two groups based on their ODI as follows: normal ODI, where patients showed < 5 events per hour, and NOD ODI, where patients showed ≥ 5 events per hour. We compared clinical characteristics, NOD, laboratory findings and radiological findings, and the patients were divided into two groups according to the presence of END. **Results:** The proportion of patients with nocturnal desaturation was significantly greater in the group of patients who also showed END (40.0% vs. 9.8%, $P < 0.001$). Moreover, more patients who developed END showed poor outcomes ($mRS \geq 3$) at 3 months compared to those without END (65.0% vs. 28.8%, $P = 0.001$). For the stroke subtypes, the percentages of LAA and SVO were significantly higher among patients in the END group, while CE was more common among patients without END ($P = 0.030$). However, the stroke lesion locations did not significantly differ between the two groups ($P = 0.822$). After controlling for relevant confounding factors, we found that NOD within the SU was associated with a risk of END (OR, 5.81; 95% CI, 1.57-21.51). **Conclusion:** This study demonstrates that nocturnal desaturation in the SU is associated with a risk of END. Therefore, intensive monitoring of nocturnal desaturation in the SU might be an important factor in preventing the risk of END and poor outcomes following acute stroke.

P-2-65

Prognosis of cerebral venous thrombosis and provoking risk factors

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Background & Objectives: Little is known about the relationships between the presence/type of provoking risk factors and the prognosis and optimal duration of anticoagulation in patients with cerebral vein and dural sinus thrombosis (CVT). **Method:** Prospectively recorded data of a tertiary medical center were retrospectively reviewed. Patients with CVT were categorized into three groups by risk factors: unprovoked, those with possibly resolved provoking factors (PR), and those with persistent provoking factors (PP). The baseline characteristics, treatment, and prognosis of these three groups were analyzed. **Results:** From 2000 to 2015, 61 patients were registered for CVT, 19 (31.1%) with unprovoked, 11 (18.0%) with PR, and 31 (50.9%) with PP. Median follow-up and duration of anticoagulation were 35 and 8 months, respectively. The PR group consisted of patients with causative drugs or arteriovenous fistula successfully treated. Despite the similarities in baseline characteristics of the three groups, deaths ($n=3$; $P = 0.256$) and recurrences ($n=7$; $P = 0.020$) were observed only in the PP group. The median interval to death and recurrence were 9 and 13 months, respectively. Death was associated with underlying disease activity, not with CVT progression. Recurrences were associated with lack of initial administration of anticoagulation ($P = 0.028$); of the seven patients with recurrence, five (71.4%) did not receive anticoagulation at the second event. **Conclusion:** Although the prognosis of CVT is generally benign, recurrence and death were observed in the patients with persistent risk factors, suggesting their need for long-term treatment with anticoagulants.

P-2-66

Neutrophil-to-lymphocyte ratio (NLR) predicts short-term functional outcome in acute ischemic stroke

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Background & Objectives: As indicators of the systemic inflammatory response, the neutrophil-to-lymphocyte ratio (NLR) have been proposed to predict the clinical outcome in some cardiovascular disease. We assessed the significance of NLR as a predictor of the outcome in patients with acute ischemic stroke. **Method:** The study included 356 patients (62.2% men, mean age 65.8 ± 13.0 years) within 3 days after the onset of acute ischemic stroke. We measured complete blood count and white blood cell differential count in all patients. All subjects were divided into four groups according to quartiles of the NLR in the initial blood test. Outcomes were measured as 3-month modified Rankin Scale (mRS) score. A good functional outcome was defined as a mRS of 0-2 points, whereas a poor outcome was defined as a mRS of >2 points. Multivariate logistic regression was used to assess association among the clinical, inflammatory and serological parameters including NLR and mRS scores. **Results:** The frequency of atrial fibrillation, heart failure, hypertension, and diabetes, the NIHSS score at admission, and the level of hs-CRP, D-dimer and the NLR were each significantly higher in the poor outcome group ($p < 0.05$). The cut-off values of NLR and NIHSS score at admission for prediction of the poor outcome were 2.135 (sensitivity 0.864, specificity 0.533) and 3.5 (sensitivity 0.862, specificity 0.787), respectively. In age-adjusted analysis, the NLR were significantly correlated with 3-month mRS score (partial $r = 0.329$, $p < 0.001$) and NIHSS score at discharge (partial $r = 0.301$, $p < 0.001$). Multivariate logistic regression analysis demonstrated that age of ≥ 65 (OR, 10.2; 95% CI, 3.31-31.21, $p < 0.001$), presence of diabetes mellitus (OR, 3.3; 95% CI, 1.36-8.12, $p = 0.008$), NIHSS score of ≥ 4 (OR, 26.4 95% CI, 9.81-71.15, $p < 0.001$), NLR of ≥ 2.135 (OR, 9.2; 95% CI, 3.18-26.4, $p < 0.001$) were independently associated with poor functional outcome. **Conclusion:** The NLR is a useful marker for short-term outcome in acute ischemic stroke. NLR may have a role in risk stratification for predicting poor outcome.

P-2-67

Association of aortic valve disease and stroke initial neurological severity

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Background & Objectives: Association between aortic valve disease and ischemic stroke has been reported by several studies. As its pathophysiology suggested that, valve thickening by calcification, stenosis, or congenital structural abnormality such as bicuspid aortic valve causes formation of microthrombus and red clot. And cardioembolic stroke represents generally severe neurological deficits than other conventional stroke mechanisms such as small vessel occlusion or large artery disease. It can be explained that red clot caused by turbulence in left atrium has more notorious effect than white clot. We investigated the association of initial stroke neurological severity and aortic valve disease by analyzing transthoracic echocardiographic parameters. **Method:** Total 1707 ischemic stroke patients from Seoul National University Hospital were enrolled and their transthoracic echocardiograms, medical documents and routine lab were reviewed. Echocardiographic parameters such as ejection fraction, left atrium size, aorta diameter, mitral E/A, mitral inflow DT, regional wall motion abnormality, valve status are evaluated. Neurological severity of stroke was categorized in two groups (mild: National Institutes of Health Stroke Scale (NIHSS) 0-7, moderate to severe: NIHSS 8-14, NIHSS: 15 and more). **Results:** Among 1707 patients 389 (21.4%) patients had aortic valve diseases. Patients with aortic valve diseases had higher median of initial stroke neurological severity (NIHSS 4 vs 5, p -value < 0.01). Patients with aortic valve disease had moderate to severe initial stroke neurological severity than patients without aortic valve. (moderate :OR1.51; 95% CI 1.07 to 2.11, p -value: 0.02). This association was still significant after adjusting. **Conclusion:** In our study,

patients with aortic valve diseases had significantly more moderate or severe stroke at admission than patients without aortic valve diseases.

P-2-68

Comparison of gastrointestinal bleeding risk among different statin exposures with warfarin: An electronic health record-based retrospective cohort study

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Background & Objectives: Patients who require both warfarin and statin are frequently seen in vascular clinics. When a physician prescribes both medications, bleeding risk should be deeply considered. Gastrointestinal (GI) bleeding is one of the most frequently occurring complications when a patient is administered warfarin, and this complication hinders continuous administration of warfarin, thus increasing the frequency of other thrombotic events. This study aimed to compare GI bleeding risk among different statin exposures with warfarin. **Method:** We observed patients who were exposed to one of four statins (pravastatin, simvastatin, atorvastatin, or rosuvastatin) and warfarin concomitantly for up to 2 years (730 days). Observation was ended when GI bleeding occurred or observation was censored. To evaluate the risk of co-administration of different statins and warfarin on GI bleeding, within-class comparison was used. We performed 1:1 matching using a propensity score for comparison of each single statin and others. Propensity scores were calculated on the basis of age, sex, Charlson comorbidity index, underlying liver cirrhosis, coagulation disorder, and the prescription of antithrombotics, nonsteroidal anti-inflammatory drugs, steroid, or fibrate for more than 30 days during the observation period. Kaplan-Meier analysis with log-rank test and Cox proportional hazard regression were conducted for comparison. **Results:** We identified 1,686 patients who were administered statin concomitantly with warfarin. The patients were classified by each statin: 287 patients were exposed to pravastatin, 317 to simvastatin, 716 to atorvastatin, and 366 to rosuvastatin. Log-rank test on GI bleeding-free survival rate showed that GI bleeding risk was significantly low in the pravastatin group ($p < 0.05$) and high in the rosuvastatin group ($p = 0.009$). Baseline mean age was 61.2 ± 13.7 , 61.0 ± 12.1 , 63.3 ± 13.2 , and 62.3 ± 13.9 years in the pravastatin, simvastatin, atorvastatin and rosuvastatin groups, respectively ($p = 0.031$). Mean levels of total cholesterol during the observation period were 167 ± 44 , 155 ± 36 , 145 ± 35 , and 139 ± 37 mg/dl, respectively ($p < 0.001$). The proportion of time in the therapeutic range of warfarin international normalized ratio was 0.45 ± 0.34 , 0.42 ± 0.35 , 0.43 ± 0.34 , and 0.47 ± 0.34 , respectively ($p = 0.217$). On Kaplan-Meier curve analyses, GI bleeding was less frequent in the pravastatin group than in others ($p < 0.05$). It was more frequent in the rosuvastatin group than in others ($p = 0.009$). However, its frequency was similar in simvastatin versus others ($p = 0.973$) and in atorvastatin versus others ($p = 0.946$). In the Cox proportional hazard regression analysis, age, sex, Charlson comorbidity index, nonsteroidal anti-inflammatory drug exposure, steroid exposure, antiplatelet exposure, and the proportion of time in the therapeutic range of warfarin international normalized ratio were adjusted. Hazard ratio (HR) of GI bleeding on statin exposure in the rosuvastatin group was significantly high (HR, 5.0 [95% confidence interval, 1.1-23.3], $p = 0.038$) while the ratio in the pravastatin group tended to low (0.3 [0.1-1.0], $p = 0.051$). **Conclusion:** Rosuvastatin administration has a relatively high risk of GI bleeding among patients who are concomitantly administered warfarin, whereas pravastatin administration appears to carry a low risk.

P-2-69

Imaging features and prognostic factors for the imaging outcomes in

cervicocerebral artery dissection

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Background & Objectives: Cervicocerebral artery dissection is a common cause of ischemic stroke in young adults and accounts for 10-25% of strokes. Prognostic factors for the improvement of the imaging findings are not well established, although the previous randomized trials have reported the clinical outcomes of cervicocerebral artery dissection. We aim to identify the imaging outcomes of a cervicocerebral artery dissection and prognostic factors which influence on the reverse of the imaging outcome. **Method:** We included 162 consecutive patients who were diagnosed with a cervicocerebral artery dissection on the baseline vascular images from January 2004 through December 2014. Demographic characteristics, putative risk factors, lesion sites, and modes of therapeutic interventions were collected. The imaging findings on the baseline within 7 days after symptoms onset and follow-up vascular images those were performed at 1 month, 3 months, 6 months, or 1 year were obtained. We compared the radiological features of the first and follow-up images and the degrees of recovery (complete or partial recovery) between baseline and follow-up vascular images. **Results:** A total of 70 patients who underwent baseline and 6 months or a year vascular imaging was compared for these analysis. Mean age was 48 year-old (SD 11) and the number of female was 21 (30%). The median interval between symptoms onset and the first vascular imaging was in a day (IQR 0, 1). Median baseline NIHSS score was 1 (IQR 0, 3). The proportion of patients with hypertension, diabetes mellitus and dyslipidemia were 43.3%, 8.6% and 11.4%, respectively. The site of a dissection was more common in the posterior circulation (71.4%, 95% CI 60% to 81%) compared to the anterior circulation and presence of infarction on baseline MRI was 84.3% (95% CI 73% to 92%). Regarding the lesion sites, a dissection was more common in the intracranial (82.9%, 95% CI 72% to 90%) than the extracranial arteries (17.1%, 95% CI 10% to 28%). The baseline vascular images identified an aneurysm in 40% (95% CI 29% to 52%), stenosis or occlusion in 77.1% (95% CI 65% to 86%) and occlusion in 21.4% (95% CI 13% to 31%). Follow-up images showed that a complete reverse of the dissected vessels in 77.8% (95% CI 40% to 96%) at 3 month and 83.3% (95% CI 51% to 91%) at 6 month. Patients who had an occlusion on the baseline images had complete reverse in 20% (95% CI 5% to 49%) and partial or complete reverse in 37.5% (95% CI 16% to 64%) on the follow-up vascular images. Follow-up images in patients with an occlusion on the baseline images identified continued occlusion in 22% (95% CI 36% to 99%) at 6 months and 50% (95% CI 16% to 64%) at the first year. There were no significant difference in efficacy for the partial or complete reverse of vascular lesion on the follow-up images in patients who were treated with antiplatelet (OR 0.68; 95% CI 0.15-3.10, p=0.72) and anticoagulation treatment (OR 1.38; 95% CI 0.48 to 3.97, p=0.60). In the multivariate analysis, the odds ratio for complete or partial reverse on the follow-up vascular images from vertebral artery was 0.16 (95% CI 0.48-0.52, p=0.002) and from dyslipidemia was 0.09 (95% CI 0.01 to 0.94, p=0.045) after adjustment for the age, gender, and eGFR. **Conclusion:** In patients who were diagnosed with cervicocerebral artery dissection on the baseline vascular images, the rate of complete or partial reverse was over 80% on the follow-up images at the 6 month or 1 year. Dyslipidemia and vertebral artery dissection are poor prognostic factors for the recovery of the vascular lesion after cervicocerebral artery dissection.

P-2-70**Pseudoathetosis associated with loss of proprioception after acute ischemic stroke**

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Background & Significance: A profound loss of proprioception leads to the pseudoathetosis resulting from the failure of proper integration of cortical sensory function. However, pseudoathetosis has rarely reported in patients with acute aschemic stroke. We describe a patient who exhibited abrupt loss of proprioception and combined pseudoathetosis following acute cerebral infarct that involved in the postcentral gyrus. **Case:** A 72-year-old female was brought to the emergency department 1 hours after she suddenly had the tingling sense and involuntary movements of the left arm. She had atrial fibrillation with dual anti-platelet medications. Neurological examination shows the loss of position sense and limb-kinetic movement in the distal part of the left arm. The most remarkable sign was a dystonic posturing and pseudoathetosis of the left arm and leg, which was only reveal when the patient outstretched her extremities with the eyes closed. Diffusion-wieghted MRI showed acute infarcted lesion involving right parietal cortex. There was total occlusion of the right proximal internal carotid artery and inferior branch of the right middle cerebral artery in MR angiography. Nerve conduction studies were normal. **Conclusions or Comments:** The proprioceptive sensory loss without overt damage to the motor system can lead to pseudoathetosis, which rarely occurs following acute ischemic stoke on the parietal cortex lesion. Postulated that the loss of proprioception causes alterations in the cortical sensory inputs to the striatum and, finally, variable mixtures of involuntary movements. However, why only a small proportion of patients with proprioceptive sensory loss develop involuntary movements is unknown.

P-2-71**Detection of aorta and common carotid artery dissection in acute stage of stroke**

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Background & Significance: Common carotid artery (CCA) occlusion is a rare cause of cerebrovascular events. In acute stroke combined with CCA occlusion, dissection of aorta and CCA could be one of cause. In general, contrast-enhanced computed tomography angiography (CTA) is a diagnosis tool to detect dissection of aorta and CCA. **Case:** During last four years, four patients who showed CCA occlusion with hemispheric stroke symptom were visited to emergency room and underwent neck CTA or neck MRA by acute stoke imaging protocol in our institution. Among them, one patient was evaluated with neck CTA and diagnosed with CCA occlusion caused by aortic dissection. In another three patients, contrast enhanced neck-MRA was used for evaluation and showed occlusion from CCA to intracranial carotid artery. In two of three patients, neck-MRA source image demonstrated a longitudinal flap from ascending aorta to CCA. Those two patients were diagnosed with aortic dissection combined with CCA occlusion. Another patient was diagnosed with thrombotic occlusion of CCA, and treated with endovascular thrombectomy. **Conclusions or Comments:** CTA, especially with arterial contrast enhancement is the investigation of choice. However, neck-MRA source image could be also useful to detect dissection of aorta and CCA in acute stage of stroke

P-2-72**A case of cryptococcal meningitis presenting as acute ischemic stroke**

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Background & Significance: Cryptococcal meningitis is usually subacute or

chronic in nature and it is a serious fungal infection of the central nervous system (CNS). The most common symptoms are headache and altered mental status, including personality changes, confusion, lethargy, obtundation, and coma. We report a rare case of cryptococcal meningitis presenting as acute ischemic stroke. **Case:** A 67-year-old woman visited our hospital presented with headache, dysarthria, left hemiparesis, mild confusion without fever for 4 days. She had a past medical history of hypertension, dyslipidemia. Initial neurologic examination at emergency room showed confused mentality, mild dysarthria, left central type facial palsy and left limb weakness medical research council grade 4/5. Meningeal irritation signs were negative. Brain magnetic resonance imaging (MRI) obtained immediately and showed no definite diffusion restriction lesions but showed hyperintense acute reperfusion marker (HARM) in fluid-attenuated inversion-recovery (FLAIR) image. Brain magnetic resonance angiography showed unremarkable study. Although there was no definite diffusion restriction lesions, we initially diagnosed her as acute ischemic stroke on the basis of focal neurologic symptoms with acute onset and of HARM sign in FLAIR imaging. Under diagnosis of acute ischemic stroke with high risk of hemorrhagic transformation, we managed her by controlling blood pressure tightly within normal range. Ten hours later after admission, patient's left arm shows involuntary tonic-clonic movement without loss of consciousness. Electroencephalography showed background asymmetry as 3~6Hz in right hemisphere and 8Hz in left hemisphere without epileptiform discharge. Even if her symptoms did not show common symptoms of CNS infection like fever or meningeal irritation signs, we performed cerebrospinal fluid (CSF) study because her symptoms also showed non-typical symptoms of acute ischemic stroke such as constant headache, confusion and involuntary movement of affected limb. CSF examination showed opening pressure of 26.5cmH₂O with slight yellowish color, white blood cell count of 205/ul (neutrophil 10%, lymphocytes 90%), glucose level 40mg/dl (serum glucose level 90mg/dl) and elevated protein level (183mg/dl). Cryptococcal antigen was found positive in CSF with titer of 1:1 and also in serum with titer of 1:2. Cryptococcus neoformans were grown in subsequent CSF culture and india ink were positive in CSF as well. We performed enhanced T1-weighted image additionally and leptomeningeal enhancement on right cerebral cortex were shown. Under the diagnosis of cryptococcal meningitis, we started amphotericin B and fluconazole. Follow up CSF analysis was performed every week and subsequent CSF profile showed gradual improvement. Patient's neurologic symptoms were recovered gradually and follow up brain MRI performed at 7 weeks after started antifungal agent showed much reduced leptomeningeal enhancement at enhanced T1-weighted image. We treated with antifungal agent for total duration of seven months and her symptoms completely improved with no relapse until one year of our serial assessment via out-patient department. **Conclusions or Comments:** Cryptococcal meningitis usually manifests as nonspecific neurologic symptoms such as headache or altered mental status in subacute, or chronic nature. We report a patient with cryptococcal meningitis initially presented as acute ischemic stroke. If left untreated, cryptococcal meningitis may lead to serious symptoms such as hearing loss, dementia, coma, occasionally even to death. So, we suggest that physicians should consider cryptococcal meningitis as differential diagnosis with patients presenting focal neurologic deficit.

P-2-73

Effectiveness of intravenous magnesium on the progressive lacunar infarction

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Background & Objectives: About a quarter of lacunar infarctions unexpectedly get worsened during hospitalization. Currently there is little strategy to effectively prevent the progression of this type of lacunar infarctions. Intravenous magnesium is one of the good potential candidates as a neuroprotective agent via its peculiar neuronal and vascular mechanisms. The purpose of this study is to elucidate whether intravenous magnesium can halt the further progression of the lacunar infarction or not. **Method:** We prospectively enrolled patients with lacunar infarction within 7 days of symptom onset. The study period is from March 2011 to June 2015. All patients got worsened their neurologic deficits from initial lacunar infarction during hospitalization. Patients with recurrence of ischemic stroke to another vascular territory were excluded from the study. Depending on the use of intravenous magnesium, enrolled patients were divided into the group with the intravenous magnesium (IVMg) and without the intravenous magnesium (non-IVMg). Demographic characteristics, National Institute of Health Stroke Scale (NIHSS) scores at admission and discharge, vascular territory, modified Rankin Score at discharge were compared between the two groups. We also compared the percentage of patients showing dramatic recovery from the worsening of initial stroke between the groups. **Results:** Of the fifty patients enrolled during the study period, sixteen patients (32%) were treated with the intravenous magnesium. Lenticulostriate artery was the most common territory of progressive lacunar infarction (26/50, 52%). There were no significant differences in the demographic characteristics, NIHSS scores at admission and discharge. Dramatic recovery from worsening defined as three points or more improved from the nadir tended to occur more frequently in the IVMG group (43.8% vs 17.6%, $P=0.082$). In the multivariate analysis this tendency did not change after adjusting the initial NIHSS score and age (Odds ratio 3.99, 95% CI [0.989 - 16.097], $P=0.052$). **Conclusion:** Using the intravenous magnesium infusion can be an alternative option for preventing from the further aggravation of lacunar infarction. More large-scale prospective study is warranted to make clear the role of intravenous magnesium as a possible therapeutic candidate in the progressive lacunar infarction.

P-2-74

Lipoic acid use and functional outcome after tissue plasminogen activator treatment in patients with acute ischemic stroke and diabetic polyneuropathy

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Background & Objectives: Alpha-lipoic acid (aLA) is a strong antioxidant commonly used for treatment of diabetic polyneuropathy (DPNP). Previously, we demonstrated the neurorestorative effects of aLA after cerebral ischemia in rats. However, its effects on stroke patients remains unknown. This study investigated whether patients treated with aLA for acute ischemic stroke (AIS) after reperfusion therapy have a better functional outcome than aLA-naïve patients. **Method:** In a prospective observational cohort study of 172 patients with DPNP and AIS treated with tissue plasminogen activator (tPA), we investigated the relationship between aLA use and functional outcome at 3 months and 1 year, the occurrence of hemorrhagic transformation (HT), early neurological deterioration (END), and early clinical improvement (ECI). The functional outcomes of patients were categorized as favorable (modified Rankin Scale [mRS] score 0-2) or unfavorable (mRS score 3-6). We defined END as an increase of ≥ 1 point in motor power or an increase of ≥ 2 points in the total National Institute of Health Stroke Scale (NIHSS) score and ECI as a decrease of ≥ 4 points in NIHSS score within 7 days. Ischemic stroke subtypes were defined according to the TOAST classification. **Results:** Of the 172 AIS with DPNP patients included, 47 (27.3%) used aLA for DPNP. In the entire cohort, those treated with aLA had a significantly higher rate of a favor-

able outcome at 3 months (55.3 vs. 33.6%, $p < 0.01$) and 1 year (57.4 vs. 34.4%, $p < 0.01$) compared with their counterparts. The risks of END and HT were significantly lower and the percentages of ECI were significantly higher in those treated with aLA. In the multivariable analysis, aLA use was associated with favorable outcome at 3 months (OR = 2.13, 95% CI = 1.01-4.51, $p = 0.048$) and 1 year (OR = 2.26, 95% CI = 1.06-4.84, $p = 0.036$). Age and HT were significantly associated with unfavorable outcome at 3 months and 1 year. **Conclusion:** aLA use for AIS with DPNP patients treated with tPA is associated with favorable outcome. These results indicate that aLA could be a useful intervention for the treatment of AIS after reperfusion therapy.

P-2-75

Plasma biomarker IGF level is associated with functional outcome in patients with acute ischemic stroke

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Background & Objectives: Insulin-like growth factor is involved in the maintenance of endothelial function and may contribute to the neuroprotective effect. In previous studies, insulin-like growth factor is increased after hypoxic brain injury, and animal model with administration of insulin-like growth factor have shown reduced infarct volume. We aim to evaluate the prognostic value of insulin-like growth factor in acute ischemic stroke patients. **Method:** In this retrospective observational study, a consecutive series of patients hospitalized for ischemic stroke within 7 days of onset were enrolled. The National Institutes of Health Stroke Scale (NIHSS) score was assessed on admission before reporting of serum insulin-like growth factor levels. For the assessment of functional outcome at 90 days Modified Rankin Scale (mRS) was used. Serum IGF-1 levels were determined by chemiluminescence immunoassay on admission. The influence of IGF-1 levels on functional outcome and death was assessed by multivariate logistic regression analysis. **Results:** 213 Patients compatible with eligibility criteria were enrolled. The mean age was 67.7 ± 12.3 years. (64.6% males, median baseline NIHSS 3) Patients with an unfavorable outcomes and death had significantly decreased serum IGF-1 levels on admission ($P < 0.0001$ for both). Serum insulin-like growth factor levels < 105 ng/mL was as an value for unfavorable functional outcome (OR 2.07, 95% CI: 1.35-4.48; $P < 0.0001$), after adjusting for other significant confounders. However, there was no relation of hormone levels to either the clinical subtype of stroke or the early neurologic deterioration. **Conclusion:** This study shows that a considerable correlation between decreased insulin-like growth factor level and unfavorable functional outcome at 3 months. Low level of insulin like growth factor may play a role in the progression of acute ischemic stroke. Limitations of this study are lack of long term follow up functional outcome after 3 months, measurement of insulin-like growth factor were performed once at admission, and retrospective study design.

P-2-76

Exome sequencing nominates the ACOX3 gene as a cause of recurrent extracranial internal carotid artery vasospasm

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Background & Objectives: Recurrent extracranial internal carotid artery vasospasms (EICAV) are a rarely recognized but important cause of ischemic

stroke mainly in young individuals. Although there was evidence that altered autonomic innervations at the affected intermediate ICA segment with up-regulated sensitivity to sympathetic vasomotor stimuli contributed to vasospasm, the exact pathophysiology of EICAV remains largely unknown to date. **Method:** We adopted whole exome sequencing to search for causative mutations in Korean dizygotic twin brothers who, both of them suffered from recurrent EICAV and their unaffected parents. **Results:** Under the recessive compound mutation model in which each asymptomatic parent was required to have a heterozygous mutation at the different loci in the same gene and each symptomatic case was required to have both of the parental recessive mutant alleles, 2 candidate genes (ACOX3 and MUC6) fulfilled the requirements. The variants from the MUC6 gene were proved to be common polymorphisms in Korean by Sanger sequencing while 2 different heterozygous alleles in the ACOX3 gene were absent in 400 in-house DNAs from healthy Koreans. One of the 2 ACOX3 variants derived from paternal DNA locates at position 8411961 on Chromosome 4 where c.665G>A mutation substitute a glycine for a glutamic acid at position 222 (G222E) of the ACOX3 gene. The second variant derived from maternal DNA locates at position 8417636 Chromosome 4 where c.235T>G mutation substitute a phenylalanine for a valine at position 79 (F79V) of the ACOX3 gene. Both loci were found in phylogenetically highly conserved domains in the ACOX3 gene. An asymptomatic sister of the twin brothers was proved by Sanger sequencing to carry c.665G>A mutation only but not c.235T>G mutation. **Conclusion:** We report herein the candidate compound missense mutations for EICAV in the ACOX3 gene on chromosome 4.

P-2-77

The correlation of SNPs of rs17501010, rs893051 and rs9290927 with small vessel disease

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Background & Objectives: The blood-brain barrier (BBB) plays a major role in development of leukoaraiosis (LA). The junctional complex of BBB consists tight junction (TJ) and adherens junction (AJ). Claudin-1 is the integral component of TJ. The aim of this study was to evaluate whether genetic variations in claudin-1 gene are associated with the development of LA. **Method:** The blood-brain barrier (BBB) plays a major role in development of leukoaraiosis (LA). The junctional complex of BBB consists tight junction (TJ) and adherens junction (AJ). Claudin-1 is the integral component of TJ. The aim of this study was to evaluate whether genetic variations in claudin-1 gene are associated with the development of LA. **Results:** Among the three SNPs of claudin-1, a significant genetic difference was found only between control and LA (both LA-PVWM and LA-DWM) with SNP rs9290927. However, their haplotypes G-G-T and G-C-A were significantly different between LA-PVWM and control, which increase the development of LA-PVWM with odd ration 1.45 and 0.57, respectively. **Conclusion:** This study demonstrated first evidence of genetic polymorphism of TJ component claudin-1 and their haplotypes associated with LA.

P-2-78

Association study of between Plasminogen activator inhibitor-1 (PAI-1) polymorphisms and ischemic stroke risk in a Korean population

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Background & Objectives: Stroke is the third most common cause of death in many developed countries, and 80% of strokes are ischemic in origin. Plasminogen activator inhibitor-1 known as endothelial plasminogen activator inhibitor or serpin E1 is a protein that in humans is encoded by the SERPINE1 gene. The PAI-1 gene is located on seventh chromosome (7q21.3-22). We designed a genetic epidemiological study of seven PAI-1 polymorphisms to investigate the association between PAI-1 and ischemic stroke in Korean population. **Method:** We conducted a case&control study of 999 individuals who were screened for the seven polymorphisms (PAI-1 -844G>A [rs2227631], -6754G>5G [rs1799889], 43G>A [rs6092], 9785A>G [rs2227694], 10692T>C [rs11178], 11053T>G [rs7242], 12068G>A [rs1050955]) by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP). A total of 574 patients (mean age \pm SD: 62.63 \pm 10.89 years, men: 41.7 %) with ischemic stroke were enrolled. Ischemic stroke was diagnosed by a rapid developing neurological symptoms and concurrent acute infarction documented by brain magnetic resonance imaging (MRI). A total of 425 subjects without any history of neurological disorders were enrolled as controls (mean age \pm SD: 62.82 \pm 10.61 years, men: 42.1%). **Results:** The PAI-1 -675 4G>5G, 10692T>C and 12068G>A polymorphisms were significantly result with ischemic stroke prevalence. In ischemic stroke subgroup analyses, LAD was significantly associated with the PAI-1 12068G>A polymorphism. SVD was significantly result with PAI-1 10692T>C polymorphism. The PAI-1 -675 4G>5G polymorphism showed significantly associated with the CE. **Conclusion:** We identified genetic associations of PAI-1 -844G>A, -675 4G>5G, 43G>A, 9785 A>G, 10692T>C, 11053T>G and 12068G>A polymorphisms with ischemic stroke prevalence and prognosis in Korean population. Our findings suggest that polymorphisms of PAI-1 may contribute to ischemic stroke and are potential biomarkers to diagnosis for ischemic stroke risk.

P-2-79**The early recognition of cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is important: two cases review**

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Background & Significance: Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is the heritable disease of small cerebral arteries. Although patients with CADASIL have an earlier age at onset of stroke events, the clinical findings and neuroimaging resemble those of small artery disease. CADASIL is relatively rare small vessel disease than sporadic one but it is common to have recurrent ischemic event and progressive neurologic deficits. No characteristic findings of CADASIL are pathognomonic. There are also variable with the onset of clinical and neuroimaging features in each patient. The symptom seems to be more common with aging but it is difficult to suspect CADASIL and to make an early diagnosis. A genetic test is the diagnostic gold standard but because it is costly and time-consuming, we need the screening tool to select patient to be subjected to the genetic testing and to make a diagnosis in good time for management. **Case:** This paper describes the screening test using CADASIL scale on two patients presenting acute lacunar stroke, who are finally diagnosed patients with CADASIL. **Conclusions or Comments:** It is important for clinician to select patients with high suspicion of CADASIL before genetic testing. The selection using screening tool as a CADASIL scale may allow iden-

tifying patients with CADASIL even in hospitals with less expertise in this disease. An early diagnosis can provide for them to do more strict regulation of risk factors and can open up the genetic counseling for their family.

P-2-80**CADASIL initially presented with isolated internuclear ophthalmoplegia**

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Background & Significance: Internuclear ophthalmoplegia (INO) is sign due to dorsal brainstem lesion. The most common etiology of INO has been shown to be stroke that is caused by branch atheroma, small vessel occlusion or artery-to-artery embolism. Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is a hereditary disease of the cerebral small blood vessels, and it is caused by mutations in the Notch 3 gene. The main clinical manifestations are recurrent ischemic stroke, migraine, and progressive cognitive impairment. Here we present a case of CADASIL initially presenting as an isolated INO that has not been previously reported. **Case:** A 63-year-old man suddenly developed horizontal diplopia 1 day ago before admission. He was former smoker with 10-pack-year and has suffered from hypertension, but has not taken anti-hypertensives. He denied diabetes, hyperlipidemia, and previous headache or stroke history. Neurologic examination of the patient showed mild exotropia and abduction limitation in the left eye. He had also abducting nystagmus of the right eye and deficiency of convergence of the left eye. There were no ptosis, dysarthria and cognitive dysfunction. Diffusion-weighted MRI showed an acute infarction in the left upper paramedian pontine tegmentum adjacent to the fourth ventricle and in left midbrain. T2-weighted and fluid attenuated inversion recovery (FLAIR) MRI revealed multiple hyperintensity lesions on bilateral periventricular white matter. The MR angiogram showed right intracarotid artery occlusion and multiple intracranial arterial stenosis. He was found to have a heterozygote R544C mutation at exon 11 of Notch 3 gene. **Conclusions or Comments:** Our case suggest that INO should be manifestation of CADASIL, which be considered as a diagnosis especially in case of extensive white matter change, even though the patient has risk factors for ischemic stroke.

P-2-81**Primary angitis of central nervous system in a patient with CADASIL**

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Background & Significance: Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is an inherited small vessel disease caused by mutations of the NOTCH3 gene. Immunological derangement is not expected in CADASIL. Herein we report a case of CADASIL and primary angitis of central nervous system (PACNS) presenting with territorial cerebral infarction. **Case:** A 37-year-old male baker visited us due to visual field defect and cognitive impairment. Previous medical and familial history was unremarkable. Neurologic examination revealed aphasia, left homonymous hemianopsia and right lower quadrantanopsia, without motor and sensory abnormalities. Brain magnetic resonance imaging showed acute cerebral infarction in left parieto-occipital lobe, a gadolinium enhancing lesion in the head of left caudate nucleus, severe diffuse white matter changes, cerebromalacia in right parieto-occipital lobe, left cerebellar hemisphere, bilateral basal ganglia and thalami. Cerebral angiography showed beaded appearance of right posterior cerebral artery. c-ANCA was positive. Genetic testing disclosed a mutation (Exon18, c.368G>A) on NOTCH3. Prednisolone was prescribed

with gradual but incomplete recovery of visual impairment. **Conclusions or Comments:** Although the combination of CADASIL and PACNS is hardly expected, our case confirmed the coexistence of two rare conditions. It remains to be studied whether the gene product of NOTCH3 has a role on immunologic function.

P-2-82

A case report of cerebral infarction in a patient with a large atrial septal defect and pulmonary hypertension

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Background & Significance: Cyanotic congenital heart disease (CCHD) is defined by the presence of congenital cardiac defect that causes either bidirectional or right-to-left shunting with systemic hypoxemia. Even though CCHD is a rare cause of cardioembolic stroke, those patients with CCHD have a relatively high prevalence of a thrombotic event, especially cerebral infarction or pulmonary thrombosis. We present the case of a stroke patient with no prior medical history who was found to have a large atrial septal defect (ASD) with pulmonary hypertension. **Case:** A 46-year old male was admitted for sudden onset of gait difficulty accompanied by headache that started the day before he presented to the emergency room. The patient was alert and fully oriented. His extraocular movements were intact and spontaneous nystagmus was not seen. On neurologic examination, he had mild dysmetria of the left arm and leg. He did not show motor or sensory abnormality. The initial non-contrast CT scan of the brain revealed a 5cm-wide hypodensity in the inferior portion of left cerebellum and an old infarct in left occipital lobe. Diffusion-weighted imaging confirmed the lesion in the left cerebellum as an acute infarct in the territory of the left posterior inferior cerebellar artery. MR angiography showed occlusion of the distal portion of left vertebral artery. Even though the patient claimed to have had no significant medical history, the initial chest x-ray alone was sufficient to suspect the presence of a long-standing left-to-right shunt with pulmonary hypertension; the pulmonary trunk was severely dilated along with bilateral pulmonary arteries and cardiomegaly was predominantly right-sided. The chest CT confirmed the x-ray findings. Even though he denied ever having symptoms of chest pain or exercise intolerance, oxygen saturation was 93% on arterial blood gas analysis at room air. Upon transthoracic echocardiography (TTE), the patient was found to have a large ASD, 2.67cm in size and right-sided cardiomegaly. On right-sided cardiac catheterization, the mean pulmonary artery pressure (mPAP) was 49 mmHg (pulmonary hypertension is diagnosed when mPAP \geq 25 mmHg at rest). The Qp/Qs ratio, the ratio of total pulmonary blood flow to systemic blood flow, was calculated to be 1.89; the ratio was greater than 1, implying the intracardiac shunt, most likely bidirectional given the large size of ASD, was predominantly left to right in direction. Because the cardiac shunt was considered the cause of embolic stroke, he was given low-molecular-weight heparin. He has also been taking bosentan, an endothelin receptor antagonist specifically aimed at pulmonary hypertension. He has been transferred to cardiology to evaluate whether surgical repair is needed for the cardiac defect. **Conclusions or Comments:** Cyanotic congenital heart disease is rare cause of ischemic stroke, but the patients with CCHD are at increased risk of thrombotic events despite their relatively young age and even in the absence of other cardiovascular risk factors. The prevalence of thromboembolic complication ranges from 5% to as high as 30 to 40% when silent infarct is included. Despite having a large ASD with pulmonary hypertension, the patient had not been aware of his congenital cardiac anomaly. When a patient presents with acute neurological complaints with abnormal features on chest x-ray, the possibility of stroke with congenital heart disease should be considered.

P-2-83

Catastrophic venous thrombosis refractory to conventional treatment

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Background & Significance: Venous thrombosis has been conventionally treated with anti-coagulation or thrombolytic agents. We presented a catastrophic case of venous thrombosis which were refractory to intense anti-thrombotic treatments. **Case:** A previously healthy 72-year-old male was transferred to our institution due to severe headache after the introduction of intravenous steroid (dexamethasone) as the preparation step for the bronchoscopic biopsy targeting bronchial mass in the right upper lung in the other hospital. The mental state was drowsy and quadriplegia (more severe in the both lower extremities compared to the upper extremities) was found on the neurologic examination. The entire obstruction of superior sagittal sinus and left transverse sinus, and small cortical hemorrhage in the left parietal lobe were found in the brain CT and MR venography. The CSF study and routine blood laboratories were normal. The autoimmune laboratories were normal. D-dimer were markedly elevated and the lupus anticoagulant was positive. Initial five day trial of Intravenous heparin was ineffective and the symptoms were aggravated. With the infusion of the intravenous urokinase, neurologic symptoms were gradually improved, however deep vein thrombosis occurred in right popliteal to infrapopliteal vein and left common femoral vein to infrapopliteal veins. Mechanical thrombectomy for deep vein thrombosis was tried twice with the interval of one week and the combination of intravenous heparin and oral dabigatran were continued after the discontinuation of urokinase infusion (10 days). Despite of above intense medical and mechanical treatments, the deep vein thrombosis was not controlled and the patient's neurologic status was again deteriorated gradually. In the follow-up digital subtraction angiography, the contrast dye with usual injection pressure could not fill the venous sinus due to increased intracranial pressure. The patient was passed away due to sepsis and multi-organ failure 40 days after the admission. **Conclusions or Comments:** We illustrated a patient with catastrophic venous thrombosis storm supposed to be developed by the bronchial neoplasm and concomitant usage of steroid. The intense anti-thrombotic treatment was not effective.

P-2-84

Stroke-like manifestation of High-dose MTX toxicity

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Background & Significance: Acute lymphoblastic leukemia (ALL) is a relatively rare hematologic malignancy occurring in adults. Methotrexate (MTX) is often employed as a therapeutic agent. Although multiple drugs are used in addition to MTX, the acute neurotoxicity reported in patients with ALL who are undergoing chemotherapy is usually attributed to MTX. MTX-induced CNS neurotoxicity and acute cerebral infarction are hard to differentiate in terms of acute onset neurologic deficit. Herein, we report a 32-year-old woman who had this rare complication. **Case:** A 32-year old woman visited our emergency room complaining of aphasia 7 hours ago. On examination, she featured global aphasia without motor weakness. Other cranial nerve, sensory exams were normal. There were no pathologic reflexes. Careful history taking from her caregiver gave us crucial information that she had seemed dull about 7 days ago, after receiving intrathecal and high dose IV MTX. She already experienced high dose MTX before. But, at past, she didn't suffer from neuro-

logic complication of MTX. Because of the acute onset, we first assessed her diagnosis as acute stroke. But, the brain MRI showed diffusion restriction on left cerebral hemisphere without vessel occlusion and perfusion defect. The diffusion restricted lesion showed no FLAIR signal change. It more resembled toxic leuko-encephalopathy rather than acute infarction. She was received hypervolemic therapy for probability of stroke. Leucovorin was given to reduce MTX toxicity. Her symptoms were fully recovered after the leucovorin infusion. Because of the probability of acute infarction, we maintained hydration and did echocardiography for ruling out cardiac embolism. The echocardiography was normal. The follow-up brain MRI was done at 1 month later. The FLAIR image showed high signal intensity corresponding to the previous diffusion restricted area. Previous diffusion high signal was gone. On neurologic examination, she didn't present any deficit. **Conclusions or Comments:** For neurologists who are treating stroke, acute onset neurological problems of patients who are receiving MTX treatment are hard to differentiate adverse effect of MTX from stroke. Since the time of intervention for stroke is crucial for patient's prognosis, neurologists have to decide whether their symptoms are due to MTX toxicity or stroke. Careful history taking about the dose and the time of MTX use is critical. Abnormal DWI image, but normal vessel and perfusion image are key indicators for distinguishing toxicity from stroke.

P-2-85

Posterior ventricular enlargement sign differentiating dementia with lewy bodies from Alzheimer's disease

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Background & Objectives: Enlargement of lateral ventricle has been noted in dementia including Alzheimer's disease (AD) as well as dementia with Lewy bodies (DLB). The pattern of ventricular shape change specific for DLB was investigated. **Method:** A total of 76 patients with DLB, 76 patients with AD, and 45 subjects with normal cognition (NC) underwent structural brain MRI and detailed neuropsychological tests. Ventricular shape was compared across the three groups using visual inspection and automated shape analysis. The degree of posterior ventricle enlargement (PVE) was measured based on the ratio of the distance between temporal and occipital horns of the lateral ventricle to the distance between temporal horn of the lateral ventricle and occipital pole of the brain. **Results:** After controlling for age, gender, and education, DLB group (PVE, $68.5 \pm 8.3\%$) had more severe PVE than AD group ($62.8 \pm 9.0\%$, $P = 0.002$) and NC group ($61.9 \pm 9.9\%$, $P = 0.004$). Ventricular shape analysis showed that DLB group had outward ventricular deformity mainly in the anterior and posterior horns of the lateral ventricle, compared to AD group. Higher PVE was associated with poorer neuropsychological performance in the Luria loop, phonemic item of controlled oral word association test, and Stroop color reading tests. **Conclusion:** PVE on visual inspection could be a useful imaging marker suggesting DLB in dementia patients.

P-2-86

Dissociation between β -amyloid burden and neurodegenerative changes in Alzheimer's disease: report of two cases

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Background & Significance: Recently, amyloid positron emission tomography (PET) is widely used to detect Alzheimer's disease (AD) pathology in pre-clinical or clinical stage of the disease. However, several studies raised questions that distinct distributions of amyloid did not correspond with clinical presentations or specific neurodegenerative/regional distributions in early onset AD, AD presenting as aphasia, or posterior cortical atrophy (PCA). Here, we report two cases with amyloid pathology who presented with aphasia and visuospatial dysfunctions and discuss the dissociation between amyloid burden and clinical features, glucose metabolism or cortical atrophy. **Case:** Case I A right-handed 72-year-old man visited neurology outpatient clinic in December, 2009 with impairment in naming and memory, which started from 2007. Series of studies were done on February, 2010. Neuropsychological test showed significant memory, language, and frontal dysfunctions. His K-MMSE score was 11/30, CDR 1, and GDS 5. Language evaluation revealed severely impaired naming and repetition which was compatible with moderate conduction aphasia (AQ 49.4, LQ 55.1). Brain MRIs showed atrophy in bilateral temporal lobes, worse on the left, including the frontoparietal area. FDG-PET showed severely reduced glucose metabolism in the left frontoparietotemporal areas. Based on the clinical and neuroimaging findings, his clinical diagnosis was Logopenic variant primary progressive aphasia or Alzheimer's dementia. His aphasia and cognitive dysfunctions continued to decline. Amyloid PET image taken in July, 2015 demonstrated asymmetric amyloid load on the right temporoparietal area. Case II A right-handed 61-year-old female patient presented neurology outpatient clinic in June, 2010 with impairment in visuospatial function, which started from 2009. Series of studies were done. Neurological and neuropsychological test showed optic ataxia, oculomotor apraxia, significant visuospatial and visual memory dysfunctions, and simultanagnosia. Her K-MMSE score was 27/30, CDR 0.5 and GDS 5. Brain MRIs showed bilateral parietotemporal atrophy, worse on the right. FDG-PET images showed severe glucose hypometabolism in the bilateral parietotemporal (worse on the right) and right occipital areas. Based on the clinical and neuroimaging findings, her clinical diagnosis was visual variant of early onset Alzheimer's dementia or PCA. Her clinical symptoms continued to worsen. Follow-up FDG-PET and amyloid PET images taken in July, 2015 demonstrated glucose hypometabolism in bilateral parietotemporal and frontal areas, more severe in the right hemisphere and amyloid load in bilateral parietotemporal and ventrolateral prefrontal areas, more severe in the left hemisphere. **Conclusions or Comments:** There may be two possibilities to explain the dissociation between distribution of amyloid and clinical symptomatology or neurodegenerative patterns in AD, regardless of clinical phenotypes. First, given that a recent case report showing patterns of neurodegeneration, such as cortical atrophy or glucose hypometabolism, mirror tau distribution, not amyloid distribution, tau pathology may be more closely linked to clinical presentations and spatial distributions than amyloid pathology in AD. That means amyloid pathology may have an early and subclinical impact on cognition and at moderate and later stages of disease, tau pathology may play an important role in ongoing symptomatology. Second, previous studies demonstrating discordance between amyloid burden and clinical presentation in focal cortical atrophy syndrome showed a typical AD-like pattern of amyloid deposition distributed throughout the association cortex. However, in our cases, the amyloid distribution was totally opposite to neurodegenerative changes. Thus, possible methodological or technical problems about tracer uptake in atrophic regions may be a tentative cause of the dissociation.

P-2-87

Transient global amnesia: a study of 97 cases

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Background & Objectives: Transient global amnesia (TGA) is characterized by a sudden onset anterograde and retrograde amnesia lasting less than 24 hours benign outcome. In this study, we investigated retrospectively 97 patients, and analyzed combined disease. **Method:** We retrospectively identified total 107 patients (mean age \pm SD, 59.62 \pm 11.01 years; M/F = 26/71) clinically diagnosed as TGA who had been admitted to Ewha Womans University Mokdong Hospital from Jan 2004 to July 2013. Patient's demographic and clinical profiles were obtained through medical record review. Of 107 patients, 97 subjects underwent brain MRI including coronal oblique DW imaging within 72 hours of symptom onset. Electroencephalography and mini mental status examination-K were also recorded for data analysis. **Results:** Mean age of patients with TGA was 59.62 \pm 11.01 years (range, 41-74 years); Female was more prevalent than males. (78.1 vs. 26.8%) Predisposing factors such as physical (n=20, 20.62%) and psychological stress (n=17, 17.53%) were identified. Other symptoms such as headache (n=12, 12.37%), dizziness (n=15, 15.46%) or nausea (n=2, 2.06%) were associated in some patients. Diffusion weighted MR images showed high signal abnormalities in 53.61% of patients; mostly unilateral hippocampal lesion (Left > Right; 22 > 12; 42.31% > 23.08%) but sometimes bilateral distributions (n=18, 34.6%). The body of hippocampus was shown to be involved in TGA than head or tail of hippocampus (n=28, 66.7%). EEG abnormalities were found in 9 (13.1%) out of 69 patients and epileptiform discharges were seen in 5 (8.33%) out of these 9 patients. **Conclusion:** In this study, we investigated the epidemiologic and clinical characteristics of TGA. This study confirms the clinical characteristics and neuro-imagings of TGA patients.

P-2-88

Does serum uric acid act as a modulator of CSF AD biomarker-related cognitive decline?

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Background & Objectives: Controversies are present regarding the effect of serum uric acid on cognitive decline. We evaluated the effect of serum uric acid on the relationship between cerebrospinal fluid (CSF) biomarker of Alzheimer's disease (AD) and longitudinal cognitive decline using the database of Alzheimer's Disease Neuroimaging Initiative. **Method:** Serum uric acid was measured at baseline in 281 healthy subjects, 624 patients with mild cognitive impairment, and 206 patients with AD. They were categorized into lower, middle, and higher tertile groups according to serum uric acid level. Baseline CSF AD biomarkers including beta amyloid (A β 1-42) and tau were measured. We evaluated the longitudinal effect of serum uric acid level being independent of baseline AD CSF biomarkers, and tested whether the effects of baseline CSF AD biomarkers on longitudinal cognitive decline differ according to serum uric acid tertile groups, using linear mixed effect models for the mini-mental state examination and AD Assessment Scale-Cognitive Subscale scores. **Results:** After controlling for the longitudinal effects of CSF A β 1-42 and tau, the lower tertile group showed faster cognitive decline, while the higher tertile group showed slower cognitive decline than the middle tertile group. When interactions between the CSF biomarkers and uric acid were tested, the detrimental effects of CSF A β 1-42 and tau were alleviated in the higher tertile group but accentuated in the lower tertile group. **Conclusion:** Serum uric acid level had protective effect on longitudinal cognitive decline independently and interactively with CSF biomarkers.

P-2-89

The neuropsychological profiles of logopenic variant primary

progressive aphasia

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Background & Objectives: PiB-PET imaging is a biomarker of Alzheimer's disease (AD). Logopenic variant primary progressive aphasia (lvPPA) is considered as an atypical clinical presentation of AD. In this study, we aimed to analyze the results of the neuropsychological profiles in PiB-PET positive lvPPA patients. **Method:** From Jan. 2004 to Dec. 2012, we recruited 16 PiB-PET positive lvPPA patients with MMSE scores greater than 15 according to the classification of primary progressive aphasia (PPA) proposed by an international group of PPA investigators. All patients underwent an extensive standard neuropsychological battery including memory, visuospatial function, and executive function; as well as a neuropsychiatric inventory within 3 days of patients' initial hospital visit. **Results:** The mean age of the lvPPA patients at their first evaluation was 64.3 \pm 8.3 years (range: 51-81 years), a disease duration of 4.4 \pm 2.0 years (range: 2-8 years), and a mean age of 60 when symptom onset was first reported. Mean MMSE scores were 23.6/30. Neuropsychological test results revealed that most of the lvPPA patients had verbal (11/16), and non-verbal memory loss (12/16), dyscalculia (8/16), and executive dysfunction, including digit span forward (16/16), animal fluency tests (16/16), and modified TMT (14/16). According to clinical history, there was no visuospatial dysfunction; however, 2 patients performed below the normal range on the modified ROCF copy test, and VOSP scores were abnormal in 5 cases. **Conclusion:** The first manifestation of PPA is a language problem that persists for two years or more. Other cognitive and functional deficits usually follow. PiB-PET positive lvPPA patients with diffuse neuropsychological deficits involved dominant hemispheric function rather than non-dominant hemispheric function. A longitudinal prospective study is needed to define the neuropsychological deficits along the disease progress in lvPPA patients.

P-2-90

Peri-operative rivastigmine patch reduces the delirium occurrence in the elderly at risk of dementia

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Background & Objectives: To date, there is inconsistent and conflicting data regarding the efficacy of acetylcholinesterase inhibitors in preventing post-operative delirium. The elderly who has cognitive dysfunction is expected to show delirium more frequently. To verify the preventive effect of the cholinesterase inhibitor, rivastigmine patch, on the post-operative delirium, we limited to the patients with decreased cognition. **Method:** We had enrolled consecutively 75 patients who were going to operation after femur neck fracture, whose K-MMSEs were 10 to 26 and global dementia scales were 3 to 5. Finally, 62 patients were enrolled. Thirty-one patients were randomly applied the 5 unit patches from 3 days before and 7 days after the operation. **Results:** The patch non-applied group showed higher delirium frequency (14/31 vs 5/31, p=0.013) and severity (Delirium Rating Scale 6.2 \pm 8.5 vs 2.2 \pm 5.7, p=0.001). However, once delirium developed, the patch did not decrease the severity of delirium. **Conclusion:** Peri-operative rivastigmine patch application reduced the delirium occurrence in the elderly with lower cognition.

P-2-92

Investigation of neural substrate of transient global amnesia using

positron emission tomographySangHak Yi¹, Young Ho PARK¹, Jae-Won JANG², SangYun KIM¹¹Department of Neurology, Clinical Neuroscience Center, Seoul National University Bundang Hospital, Seongnam, Korea, ²Department of Neurology, Kangwon National University Hospital, Chuncheon, Korea

Background & Objectives: Transient global amnesia (TGA) is characterized by the abrupt onset of episodic memory impairment. Although injury to hippocampal CA1 neurons is supposed to play a crucial role in the pathophysiological cascades, further involvement of neocortex still remains elusive. In this study, we investigated which brain regions show metabolic changes during the acute stage of TGA using 18 fluoro-2-deoxyglucose positron emission tomography (FDG-PET). **Method:** A consecutive series of 14 patients with TGA who visited Seoul National University Bundang Hospital and underwent FDG-PET within 3 days after symptom onset were retrospectively identified. We used statistical parametric mapping to compare cerebral glucose metabolism of TGA patients with 25 age-matched normal controls. **Results:** The patients with TGA demonstrated reduced glucose metabolism in the left middle temporal pole, right cuneus and both calcarines with a statistical significance of $P < 0.005$ (uncorrected). **Conclusion:** The brain regions with decreased glucose metabolism are main components of posterior medial system which is known to be implicated in the process of episodic memory. This study provides evidence for disruption of posterior medial system during the acute stage of TGA.

P-2-93**The association between cognitive status and abnormal findings on MR spectroscopy in the patients with hypoxic brain damage**

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Background & Objectives: Hypoxic brain damage is one of main causes leading to permanent brain damage and severe cognitive impairment. However, it remain unclear whether brain image could predict the long-term cognitive status. We investigated the association between long-term cognitive outcomes and abnormal findings on MR spectroscopy in acute period. **Method:** We selected the patients with acute hypoxic brain damage, who were admitted from 2005 to 2014. They took the MR scans between 7 and fourteen days after hypoxic event. Left frontal, occipital, temporal and basal ganglia were chosen for the evaluation of MR spectroscopy. The patients with the history of stroke or aged less than 20 years, were excluded. All patients also took the minimal status examination (MMSE) and Glasgow Coma Scales on 7 days and one month after hypoxic insult. The patients with good cognitive outcomes were defined as one having 18 or more points on MMSE at one month of onset. **Results:** A total of nine patients were included in this study (51.7 ± 11.0 years). The median point of MMSE was 18. Five patients had good cognitive outcomes. Compared to the subject with poor outcomes, those with good outcomes had the higher NAA/Creatine peak ratio on frontal and temporal area, higher GCS and MMSE on seven days of hypoxic insult and less frequency of ventilator care. Hypothermia was tried to only two patients and they seemed higher NAA/Creatine peak ratio in the frontal cortex than the others. **Conclusion:** MR spectroscopy has several limitations. Most of all, the MR spectroscopy was only available in the patients with stable vital sign. However, MR spectroscopy could be helpful for the prediction of good cognitive status in chronic period in the patients with acute hypoxic insult.

P-2-94**Differences in hippocampal surface and white matter structure in early and late MCI: ADNI Study**Peter LEE¹, Hojin RYOO², Jinah PARK², Yong JEONG¹¹Department of Bio and Brain Engineering, KAIST, ²Department of Computer Science, KAIST

Background & Objectives: Alzheimer's disease (AD) is a progressive neurodegenerative disease and the most common type of dementia yet actual progression is unclear. Main symptom of AD is cognitive decline mainly with memory failure. However, recent studies show that the clinical symptom follows later than the onset of the disease which delayed the diagnosis and its early intervention. To overcome this problem, Alzheimer's Disease Neuroimaging Initiative (ADNI) proposed Early (EMCI) and Late mild cognitive impairment (LMCI) stage with respect to Wechsler Memory Scale Logical Memory II scores. In this study, we investigate the difference of hippocampal surface and white matter structure in EMCI and LMCI to see the early events in AD progression. **Method:** We compared MRI T1 volumes of hippocampal subfields from 20 controls, 17 EMCI patients, and 20 LMCI patients. Hippocampal shape modeling based on a progressive template surface deformation was used to compare group atrophy. This method induces a large-to-small scale deformation of a template surface to build the pairwise correspondence by minimizing geometric distortion while robustly restoring the individuals' shape characteristics. We also made a template surface of the control to use as a ROI for diffusion tensor image (DTI) voxel-based morphometry analysis. For DTI analysis, additional 20 AD patients' data were included to show disease progression characteristics. FSL TBSS pipeline was used to register FA, MD, RD, and AD map images to study-specific template in MNI space. Cluster-wise group comparison on DTI metrics within hippocampus was performed using FSL randomise with Threshold-free Cluster Enhancement option along 10,000 permutations. For further analysis on the relationship between structural changes with clinical score, linear regression was performed to find significant clusters. Modified Alzheimer Disease Assessment Score and MMSE were used for general clinical score. All images and clinical data were gathered from ADNI database (adni.loni.usc.edu). **Results:** Hippocampal surface analysis showed significant atrophies on bilateral CA1 regions and right ventral subiculum of EMCI compared to control, whereas bilateral CA1, CA2-4, and subiculum in LMCI. LMCI showed more significant atrophy regions on right ventral subiculum part of hippocampus than EMCI ($P < 0.05$ uncorrected). Normative hippocampal template was created on MNI space 1mm isotropic 5,674 and 5,477 voxels. DTI VBM results showed increased axial diffusivity on the right CA2-4 region in EMCI compared to control. LMCI compared to control showed increased axial diffusivity on bilateral CA2-4 and subiculum regions. AD compared to control showed atrophy on most of the hippocampal regions. Lastly, axial diffusivity showed significant correlation with the scores of M-ADAS Cog and MMSE ($P < 0.05$ FWE corrected). **Conclusion:** We analyzed the hippocampal structural changes with EMCI and LMCI with respect to structural MRI and DTI. Right hippocampal change was observed in EMCI and LMCI showed additional extended change to left hippocampus in terms of both surface structure and white matter integrity. It suggest that in the AD progression process, right hippocampal change starts first and propagate to the other side as disease progress. We also demonstrate that the white matter integrity (axial diffusivity) within the hippocampus correlates well with the cognitive performance indicating possible use of it as an early feature of AD progression.

P-2-95**Unusual idiopathic normal pressure hydrocephalus patient with marked asymmetric and upper body parkinsonism**

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Background & Significance: Asymmetry of parkinsonian symptoms is strong

evidence toward the diagnosis of Parkinson's disease (PD). Lower body parkinsonism is characteristic in idiopathic normal pressure hydrocephalus (INPH). **Case:** We report an unusual INPH patient with marked asymmetric and upper body parkinsonism. An 83-year-old man presented with gait impairment and asymmetric clumsiness of movement. According to the Unified Parkinson's Disease Rating Scale, the motor subscore was 12 in the left limb and 8 in the right. The score was 14 for both the upper body and lower body. After the cerebrospinal fluid tap test, he showed marked improvement in the upper body score. A loss of asymmetry of parkinsonian signs, with the greater improvement in the left limb, was presented. Fluorinated N-3-fluoropropyl-2- β -carboxymethoxy-3 β -(4-iodophenyl)-nortropane (F-18 FP-CIT) positron emission tomography imaging was normal. **Conclusions or Comments:** In the differential diagnosis of elderly patients presenting with parkinsonism compatible with PD, we might need to consider a diagnosis of INPH.

P-2-96

Heterogeneous etiology of SNAP in MCI Stage: longitudinal changes of cortical thinning

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Background & Objectives: We aimed to investigate the longitudinal outcome of amnesic mild cognitive impairment (aMCI) patients with significant Pittsburgh Compound B deposition [PiB(+) aMCI] and those without [PiB(-) aMCI]. **Method:** The amount of brain fibrillar β -amyloidosis was measured in 48 patients with aMCI using PiB positron emission tomography (PET). They were categorized as PiB(+) aMCI (N = 32) and PiB(-) aMCI (N = 16) using a cut-off value of 1.5 in global PiB uptake ratio. Sixteen PiB(-) aMCI patients were further categorized according to the clinical and imaging properties. Clinical follow-up (mean duration of 2.7 ± 1.4 years after PiB-PET scans), and follow-up with brain MRI (N = 38) and PiB-PET (N = 30) were performed. **Results:** Twenty-two PiB(+) aMCI patients (68.8%) and four PiB(-) aMCI patients (25.0%) progressed to dementia, and PiB(+) aMCI had a higher risk of progression to dementia than PiB(-) aMCI (hazard ratio = 3.23, 95% CI = 1.07-9.78). PiB(+) aMCI showed faster rate of cortical thinning in the bilateral precuneus and right medial and lateral temporal cortices compared to PiB(-) aMCI. Among six PiB(-) aMCI patients who had regional PiB uptake ratio > 1.5 in the posterior cingulate cortex (PCC), three (50.0%) progressed to dementia, and two of them had global PiB uptake ratio > 1.5 at the follow-up PiB-PET. **Conclusion:** Our findings suggest that PiB-negativity predicts better prognosis in aMCI patients, but regional PiB-positivity in the PCC predicts poor prognosis in the PiB(-) aMCI group.

P-2-97

Slower progression of subcortical vascular dementia patients who met the Seoul criteria compared to those who did not

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Background & Objectives: Differentiating subcortical vascular dementia (SVaD) from AD is important from therapeutic perspectives. Recent studies revealed that many patients with clinically diagnosed SVaD have coexistent AD pathology. The Seoul criteria (age ≤ 75 , lacunar numbers ≥ 5 and medial temporal lobe atrophy ≤ 3 with severe WMH) has been proposed to help discriminate pure SVaD from mixed SVaD using clinical and MRI variables without resorting to amyloid PET. In this study, we hypothesized that patients who meet the Seoul criteria would have slower deterioration than those do not. **Method:** Out of 282 SVaD patients who met DSM-IV criteria for vascular dementia and had severe white matter hyperintensities (cap or band ≥ 10 mm and deep white matter lesion ≥ 25 mm) from January 2007 to December 2013, we retrospectively recruited 113 patients who, after baseline evaluation, underwent at least once follow-up neuropsychological tests. Participants were divided into 36 patients who met the Seoul criteria (S+) and 77 patients who did not (S-). S+ patients did not differ from S- patients in terms of gender proportion, educational level, APOE4 carrier proportion, disease duration and vascular risk factors except body mass index. The time interval from the initial and the final neuropsychological evaluation ranged from 1 year to 3 years with 2.4 years on average. **Results:** S+ patients showed slower deterioration in global cognitive scales such as dementia version of Seoul Neuropsychological Screening Battery, Clinical Dementia Ratio (CDR) and CDR-sum of Boxes compared to S- patients. S+ patients had better memory function at baseline and showed slower decline in stroop-color reading test during the follow-up period than S- patients. **Conclusion:** S+ patients who were presumed to have pure SVaD showed slower deterioration in global cognitive tests than S- patients who were presumed to have combined vascular and Alzheimer pathologies. These findings are noteworthy for understanding and predicting the prognosis of SVaD and making decisions for caring of the patients in the clinics.

P-2-98

Donepezil induced Rhabdomyolysis

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Background & Significance: Donepezil is a cognitive enhancing medication for treating mild to moderate dementia and inhibits the cholinesterase, a neurotransmitter. Common side effects of donepezil include diarrhea, nausea, vomiting, loss of appetite, muscle cramps and trouble in sleeping. We present a rare case of acute rhabdomyolysis probably induced by donepezil. **Case:** A 86-year-old male presented with decreased level of consciousness for 2 days. He had a medical history of Alzheimer's disease for two years and he had taken donepezil 5mg and choline alfoscerate 400mg daily for 2 months. He did not have any other diseases and he had not taken any other medications. There is no medical history of crush injury, convulsion, alcohol intake, strenuous exercise or infection. His temperature was 36.0°C, blood pressure was 140/90mmHg, pulse rate was 86/minute and respiratory rate was 14/minute. Initial laboratory values revealed creatine kinase: 1830 IU/L, Myoglobin: 3903.7 ng/ml, aspartate aminotransferase: 72 U/L, creatine kinase-myocardial band: 20.40 U/L, blood urea nitrogen: 36.5 mg/dl, creatinine: 1.82 mg/dl, erythrocyte sedimentation rate: 64 mm/hr and C-reactive protein: 6.09 mg/dL. Cerebrospinal fluid analysis was normal. Neurological examination revealed stupor consciousness level, he would only react to pain and decreased muscle strength. Brain MRI showed no acute lesions. He was admitted to neurologic intensive care unit and intravenous hydration and conservative therapy were given. Laboratory findings improved gradually. Seven days after admission, his consciousness level improved to alert. **Conclusions or Comments:** Rhabdomyolysis may be associated with taking donepezil. Clinicians pay attention to this side effect.

P-2-99**Early- versus late-onset subcortical vascular cognitive impairment**

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Background & Objectives: The aim of this study was to evaluate the differences between early-onset subcortical vascular cognitive impairment (EO-SVCI) and late-onset SVCI (LO-SVCI) with regard to pathologic burden, structural changes, and cognitive function. **Method:** We prospectively recruited 142 patients from a single referral center. Patients were divided into EO-SVCI (n=30, age of onset <65 years) and LO-SVCI (n=112, age of onset ≥ 65 years) groups. All patients underwent neuropsychological tests, 3T brain MRI, and [11C] Pittsburgh compound B (PiB)-PET. We compared pathologic burden such as small vessel disease and amyloid burden; structural changes such as structural network, cortical thickness, and hippocampal volume; and cognitive function between EO-SVCI and LO-SVCI. **Results:** EO-SVCI patients had more lacunes, while LO-SVCI patients had higher PiB standardized uptake value ratios. EO-SVCI patients exhibited more severe structural network disruptions in the frontal area, while LO-SVCI patients exhibited more severe cortical and hippocampal atrophy. Although disease severity did not differ between the two groups, frontal-executive dysfunction was more severe in EO-SVCI patients. **Conclusion:** EO-SVCI patients showed more vascular related factors, while LO-SVCI patients exhibited more Alzheimer's disease-related characteristics. The greater number of lacunes in EO-SVCI might account for the more severe frontal network disruption and frontal-executive dysfunction, while the greater amyloid burden in LO-SVCI might account for the more severe cortical and hippocampal atrophy. Our findings suggest that the age of onset is a crucial factor that determines distinct features in SVCI patients, such as pathologic burden, structural changes, and cognitive function.

P-2-100**Association of sleep qualities and cortical thickness in Subjective Memory Impairment patients**

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Background & Objectives: Individuals with subjective memory impairment (SMI) but no objective deficits are at increased risk of developing cognitive decline and dementia. SMI individuals often present depression and sleep problems, both of which are associated with neurodegeneration. While there are numerous studies on the relationship between depression and neurodegeneration such as cortical atrophy, little attention has been paid to the relationship between sleep and cortical atrophy in patients with SMI. Thus, the aim of this study was to evaluate the relationships between sleep quality, cortical thickness, and cognition in SMI individuals with or without depression. **Method:** We prospectively recruited 200 SMI individuals from September 2011 to December 2013 in a single referral center. All the participants underwent detailed neuropsychological tests and 3 Tesla MRI. Subjective sleep quality and depressive symptoms were assessed using the Pittsburgh Sleep Quality

Index (PSQI) and geriatric depression scale, respectively. We performed cortical thickness analyses using a surface-based morphometry method. **Results:** SMI with depression group (n=123) showed higher PSQI-K score than SMI without depression group (n=77) (p=0.001). In SMI with depression group, poor habitual sleep efficiency was associated with cortical thinning in the bilateral anterior temporal, right precuneus, and bilateral dorsolateral prefrontal cortices. In addition, cortical thinning in these areas was associated with verbal memory and language dysfunction. However, in SMI without depression group, there was no association between sleep quality and cortical thickness. **Conclusion:** Our findings showed that in depressed SMI individuals, those who have poor habitual sleep efficiency have cortical thinning in the areas that are vulnerable to Alzheimer's disease, which accounts for decreased memory and language function. We suggest that individuals with depressed SMI and poor sleep efficiency are at a higher risk of developing cognitive decline.

P-2-101**Diffuse type of frontal lobe atrophy shows a poorer prognosis than focal type in the behavioral variant frontotemporal dementia: A CREDOS-FTD Study**

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Background & Objectives: We hypothesized that there are different clinical characteristics between diffuse and focal type of frontal lobe atrophy on axial MRI scans in patients with behavioral variant frontotemporal dementia (bvFTD). **Method:** A total of 74 patients with bvFTD were retrospectively recruited from 8 dementia clinics across Korea. MRI visual rating scale was performed to classify patients with bvFTD into diffuse (n = 33) and focal type (n = 41) of frontal lobe atrophy. We compared baseline characteristics, progression in motor and cognitive symptoms, and survival time between diffuse and focal type. Survival analyses were performed for 62 patients. **Results:** We found that MRI visual rating subtypes were associated with distinct clinical features across the subtypes. The score of the Unified Parkinson's Disease Rating Scale (UPDRS) Part III at baseline in diffuse type was higher compared with that in focal type (13.5±14.8 vs 5.8±9.9). The motor symptoms were more likely to occur earlier in diffuse type than in focal type. Also, diffuse type tended to have more depressive moods and higher caregiver-stress in agitation and nighttime behavior. Moreover, it was an intriguing finding that the median survival time in diffuse type (6.8 years) was shorter than in focal type (9.5 years). The Cox regression revealed that the high score of UPDRS Part III at baseline contributed to increased risk of mortality. However, bvFTD patients with diffuse type had significantly better neuropsychological performance than those with focal type at both baseline and follow-up. **Conclusion:** There were distinct clinical characteristics between diffuse and focal type of frontal lobe atrophy in patients with bvFTD. We speculate that bvFTD patients with diffuse type show a poorer prognosis than those with focal type. Shorter survival in diffuse type may be associated with motor symptoms.

P-2-102

Paraneoplastic limbic encephalitis presenting with persistent anterograde amnesia

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Background & Significance: Paraneoplastic limbic encephalitis (PLE) is characterized by acute or subacute amnesia, personality changes, other psychiatric syndromes, and seizures. It is often associated with characteristic MRI changes of increased T2 signal in the mesial temporal structures, inflammatory cerebrospinal fluid (CSF) parameters, and positive paraneoplastic proteins. The commonly associated neoplasms are lung cancer, testicular tumor, thymoma, and ovarian cancer. The present report describes a case of a 69-year-old man presenting with only acute persistent amnesia, who was diagnosed later as PLE combined with small cell lung cancer (SCLC). **Case:** A 69-year-old male presented with 3 days short-term memory loss. There was no neurologic deficit other than persistent anterograde amnesia. The patient was a current smoker and had type 2 diabetes diagnosed 2 years previously. MRI of the brain showed hyperintensity on FLAIR and T2-weighted image in right inferior frontal region, bilateral amygdala and hippocampi, suggesting PLE. CSF study revealed no evidence of malignancy or infection. As we suspected the possibility of hidden malignancy, we checked chest CT, tumor markers and paraneoplastic autoantibodies. Chest CT showed multiple subpleural nodules suggestive of lung cancer. We consequently performed PET/CT and CT-guided percutaneous needle biopsy and finally diagnosed the patient with PLE associated with SCLC. Paraneoplastic autoantibody testing was positive for anti-Hu antibodies. The patient was treated with combination chemotherapy and his symptoms gradually improved, but completely did not recovered. **Conclusions or Comments:** The diagnosis of malignancy in patients with PLE sometimes comes from evaluation of a neurologic complaint. The neurologic presentation varies depending on the site of the lesion. In cases in which risk factors for malignancy are present, any patient with an unexplained neurologic manifestation should be investigated for paraneoplastic neurologic syndrome. When a neurologic abnormality leads to suspicion of paraneoplastic neurologic syndrome, screening for hidden malignancy including paraneoplastic autoantibodies should be conducted.

P-2-103

Comparisons of hippocampal subfields volumes between mild cognitive impairment and Alzheimer's disease

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Background & Objectives: To assess whether there are differences of the hippocampal subfields volume between Alzheimer's disease (AD), mild cognitive impairment (MCI) and controls, and to investigate which subfields volumes of hippocampus are correlated with severity of cognitive dysfunction. **Method:** We included consecutive 102 participants, who were 61 AD, 12 MCI and 29 controls. All of the participants underwent detailed neuropsychological evaluation, 3D T1-weighted MRI at 3-Tesla. Hippocampus was segmented into seven subfields using FreeSurfer: CA1, CA2-3, CA4-dentate gyrus (DG), subiculum, presubiculum, fimbria, and hippocampal fissure. We compared hippocampal subfields volumes in AD, MCI and controls, and also analyzed the volume changes respect to MMSE score in AD. Correlation analyses between cognitive function and subfields volumes were performed. **Results:** Analysis of variances, corrected for age, gender and education, showed that the volumes of bilateral CA1, CA2-3, CA4-DG, presubiculum and subiculum, and left fimbria were significantly different between AD, MCI and controls. These differ-

ences were found in the comparison between mild stage of AD (MMSE 19-24), moderate stage of AD (MMSE 10-18), MCI and controls. Volume loss in all the hippocampal substructures besides bilateral fimbria and hippocampal fissures, and left CA1 was associated with the cognitive function measured by K-MMSE with correction for age, gender and education ($p < 0.001$). **Conclusion:** Our results suggest that the volume reductions in the hippocampal presubiculum, subiculum and CA2-3, CA4-DG might be ones of the important changes in the spectrum of AD like disease.

P-2-104

The relationship between neuropsychiatric symptoms and hippocampal subfields volumes in Alzheimer's disease

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Background & Objectives: To assess whether there are differences of the hippocampal subfields volume between Alzheimer's disease (AD) and controls, and to investigate which subfields volumes of hippocampus are correlated with neuropsychiatric symptoms (NPS) in Alzheimer's disease. **Method:** We included consecutive 110 patients with AD and 30 controls. All of the participants underwent detailed neuropsychological evaluation, 3D T1-weighted MRI at 3-Tesla. Hippocampus was segmented into seven subfields using FreeSurfer 5.3: CA1, CA2-3, CA4-dentate gyrus (DG), subiculum, presubiculum, fimbria, and hippocampal fissure. We compared hippocampal subfields volumes in AD and controls, and also analyzed the volume changes respect to Neuropsychiatric Inventory (NPI) sub-score (NPI_behavioral and NPI_psychological) in AD. Correlation analyses between NPI total scores, NPI-sub-scores and subfields volumes were performed. **Results:** Comparisons of the hippocampal subfields volumes between AD and controls exhibited that AD had smaller volumes of bilateral CA2-3, CA4-DG, presubiculum, subiculum, and right fimbria and CA1 than controls. The correlation analyses of each NPI scores with subregional volumes of the hippocampus revealed that the same regions which had smaller volumes in AD than in controls correlated positively with NPI total score and NPI_psychological score. NPI_behavioral score was correlated with the volumes of the right subiculum and right CA4-DG, after correction for age, gender, K-MMSE and intracranial volume. **Conclusion:** Our results suggest that the hippocampus, which was known to be vulnerable to stress, had the volume loss in the specific areas of right CA4-DG and subiculum with relation to the NPI_behavioral scores.

P-2-105

Relative cognitive impairment in neuropsychological tests as a predictor for future cognitive decline

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Background & Objectives: Clinical diagnosis of Alzheimer's disease (AD) often relies on comparison of performance on neuropsychological tests with group norms. However the sensitivity of this approach is relatively poor in the early phase of the disease and subjects with subtle decline in performance were often considered to be cognitively normal. However, recent studies showed that subtle cognitive decline could be associated with preclinical AD. The aim of this study is to describe the demographic and clinical characteristics of subjects who showed relative cognitive impairment (RCI) in cognitively normal elderly subjects and determine which cognitive domain can predict future cognitive decline. **Method:** A total of 1,586 cognitively normal elderly subjects were enrolled from a nationwide multicenter study of dementia, the Clinical Research for Dementia of South Korea (CREDOS) study. A total of 279 sub-

jects (17.6%) had at least 1 follow-up evaluation of neuropsychological tests and enrolled in this study. One representative neuropsychological test for each cognitive domain (Seoul Verbal Learning Test (SVLT) for memory, Korean version of Boston Naming Test (K-BNT) for language, Rey Figure Copy Test (RCFT) for visuospatial function and phonemic Controlled Oral Word Association Test (COWAT) for frontal function) was selected to define RCI which was designated as a lower percentile score than the mean of other test percentile scores by 1 or more standard deviation. Kaplan-Meier survival analysis and Cox regression analysis were used to determine independent predictors for conversion to mild cognitive impairment (MCI) or AD. **Results:** Fifty seven subjects were classified as having RCI-memory and 28 RCI-language, 41 RCI-visuospatial function and 57 RCI-frontal function group were classified. There was no significant difference in age, gender and Mini-Mental State Examination (MMSE) score among RCI groups. RCI-language group had significantly higher years of education than other 3 groups. RCI-language group was the only significant predictor for conversion to MCI or dementia after adjustment for age, gender, years of education and Mini-Mental State Examination ($p=0.01$, Odd ratio=1.89, 95% CI 1.16-3.12). The mean conversion time of RCI-language group was 704.3 days compared to 897.9 days in non-RCI group. **Conclusion:** These findings suggest that relative decrease in language function in cognitively normal elderly subjects with higher education level could be associated with underlying AD neuropathology and predict future cognitive decline.

P-2-106

Independent effects of physical exercise and education on age-related cortical thinning in cognitively normal individuals

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Background & Objectives: Physical exercise and education are generally accepted as modifiable risk factors for age-related cognitive decline. However, the evidence is limited and indirect. In this study, we aimed to investigate the effects of physical exercise on the cortical thickness in a large sample of cognitively normal individuals. We also determined whether physical exercise and education affect cortical thickness, independently or interactively. **Method:** A total of 1,842 participants were included in this analysis. Physical exercise was assessed using the questionnaire which consisted of three exercise parameters such as intensity, frequency, and duration. Cortical thickness was measured using surface based method. Multiple linear regression analyses were performed after controlling for possible confounders. **Results:** Increased duration of exercise (≥ 1 hr/day), but neither intensity nor frequency, was associated with increased mean thickness in the global (p -value = 0.020) and in the frontal regions (p -value = 0.009). Especially, the association of exercise with cortical thinning had regional specificity for the bilateral dorsolateral prefrontal, precuneus, and inferior parietal regions. The effects of exercise on cortical thickness were more prominent in highly educated individuals than poorly educated individuals. Tests for trends across combined exercise and education effects showed that groups with exercise (≥ 1 hr/day) and education (≥ 12 yrs) had more thickness in the bilateral dorsolateral prefrontal, supplementary motor, anterior and posterior cingulate, precuneus, and insular regions, than groups without exercise (< 1 hr/day) and education (< 12 yrs). However, there were no interactive effects of exercise and education on cortical thickness (p -value = 0.116-0.352). **Conclusion:** Our findings suggest that physical exercise and education show protective effects on age-related cortical thinning, independently, but not interactively. Furthermore, considering the paucity of modifiable risk factors for age-related cortical thinning, our results have im-

portant public health implications.

P-2-107

An autopsy confirmed case of nonfluent/agrammatic variant primary progressive aphasia combined with generalized chorea with corticobasal degeneration pathology

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Background & Significance: Nonfluent/Agrammatic variant of primary progressive aphasia (naPPA) is a clinical syndrome of slowly progressive language disorder characterized by effortful and halting speech with inconsistent speech sound errors/distortion and agrammatism. Other clinical features include impaired comprehension of syntactically complex sentences, spared object knowledge and single word comprehension. Many patients with nonfluent variant will eventually to generalized motor syndrome involving myoclonus, dystonia, and rigidity. Clinico-pathological studies have demonstrated that naPPA can be associated with various kinds of neurodegenerative pathology, such as progressive supranuclear palsy, corticobasal degeneration (CBD), transactive response DNA binding protein (TDP)-43 proteinopathy, and Alzheimer's disease. Here, we report an autopsy confirmed case of naPPA combined with generalized chorea which was a rare clinical phenotype and finally turned out to be CBD pathology. **Case:** A 78-year-old female patient presented with 4-year-history of progressive dysarthria and difficulty with expressive language. Past medical history was significant for diabetes mellitus and surgical resection of cerebellar meningioma 1 year after symptoms onset. Neurological examination revealed severe dysarthria, generalized bradykinesia, and mild clumsiness and rigidity on bilateral upper extremities. In language evaluation, the patient showed severely impaired spontaneous speech with relatively spared comprehension. Speech hesitancy or labored speech was prominent. Varying degrees of phonological errors and agrammatism were also present in speech production and repetition. In neuropsychological evaluation, she showed impaired attention, naming, and executive function. Her K-MMSE score was 25/30, CDR 0.5 and GDS 4. Laboratory tests including complete blood count, routine chemistry, electrolyte, thyroid function test and vitamin levels were normal. Brain MRI revealed diffuse cortical atrophy and FDG-PET showed glucose hypometabolism in left inferior frontal and opercular areas. Based on clinical and neuroimaging findings, her clinical diagnosis was nonfluent/agrammatic variant primary progressive aphasia. At the age of 79, 5 years after the symptoms onset, she developed generalized choreic movements involving neck, trunk and four extremities while walking or sitting. Genetic testing for Huntington disease was negative. She continued to decline with worsened gait and reached a vegetative state. She died at the age of 83, 9 years after the disease onset. The brain weight was 1012g. Grossly, moderate atrophy was detected in dorsolateral prefrontal cortex, inferior frontal gyrus, basal ganglia and hippocampus. Microscopically, numerous tau immunoreactive neuronal cytoplasmic inclusions, neuronal threads, and astrocytic plaques in cerebral cortices, especially anterior cingulate, and superior,

middle and inferior frontal gyri. Profound frontal subcortical white matter tau pathology was also identified, which was compatible with a diagnosis of CBD. No beta-amyloid, alpha-synuclein and TDP 43-immunoreactive pathology was observed. **Conclusions or Comments:** The original literature of CBD reported patients with choreiform movements and dystonia in limbs. However, chorea or ballism was rarely observed in patients with CBD. Thus, this autopsy proven case of naPPA combined with generalized chorea with CBD pathology would extend clinical boundaries of phenotypes of CBD pathology.

P-2-108

Clinical impacts of lobar microbleeds in patients with clinically probable cerebral amyloid angiopathy: a pilot study

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Background & Objectives: Cerebral amyloid angiopathy (CAA) is the cerebrovascular disease caused by amyloid accumulation. Strictly lobar microbleed (MB) is one of key features of patients with clinically probable CAA. Also, cortical superficial siderosis (cSS) was recently included in the clinical diagnostic criteria of CAA. In this study, we aimed to determine whether lobar MBs or cSS would better predict cognitive impairments in patients with clinically probable CAA. **Method:** Participants consisted of 38 patients with probable CAA who fulfilled the modified Boston criteria. All patients underwent brain magnetic resonance imaging (MRI) and neuropsychological test. The number of lobar MBs and cSS were manually counted. Participants were divided into three and two groups according to number of lobar MBs and cSS, respectively. Linear regression analysis was performed after controlling for possible confounders. **Results:** The frequency of patients with no lobar MBs, single lobar MB and multiple lobar MB was five, three, and twenty nine, respectively. The frequency of patients without and with cSS was twenty and eighteen, respectively. Multiple lobar MBs were an independent predictor of poorer cognitive performance in executive function. However, there were no differences in cognitive functions between with and without cSS. **Conclusion:** Our preliminary results suggested that that multiple lobar MBs affect executive dysfunction in clinically probable CAA patients.

P-2-109

Functional connectivity of the posterior insular in Wernicke's encephalopathy with impaired vestibular ocular reflexes

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Background & Objectives: Wernicke's encephalopathy (WE) is a syndrome characterized by ataxia, ophthalmoplegia, nystagmus, confusion, and impairment of short-term memory. With its classical representations, a loss of the horizontal vestibulo-ocular reflex (VOR) which is probably the major cause of acute ataxia in Wernicke's encephalopathy is another important feature. Previous functional MRI (fMRI) studies revealed that visual stimulation in patients with bilateral vestibulopathy generated enhanced activations within the visual and ocular motor systems which suggested an upregulation of visual sensitivity during visual motion stimulations. More recently, it has been recognized that slow (<0.1 Hz), spontaneous fluctuations in the fMRI blood oxygen level-dependent (BOLD) signal have been shown to exhibit phase coherence within functionally related areas of the brain. Goal of this study was to establish whether the acute impairment of bilateral vestibular input in patients with

WE causes a resting state functional connectivity changes in three patients with radiologically normal brain imaged of the visual and vestibular cortex. **Method:** Three patients of WE and twenty healthy right-handed subjects participated in this study. All participants (patients and healthy controls) had no previous history of vertigo and they received detailed neurological and otological examination. They did not reveal hearing impairment on pure tone audiogram, or spontaneous and positional nystagmus on video Frenzel goggle examination, abnormal catch-up saccade on head-impulse test. They also were undertaken psychiatric interviews for screenings to detect psychiatric disorders such as depression and anxiety disorders. **Results:** Main finding we found is that reduced functional connectivity of the posterior insula and parietal operculum compared to healthy control subjects. These areas are supposed to vestibular cortex regions integrating multisensory signals into a percept of spatial orientation and self-motion. This reduced connectivity may be related to the impaired input of vestibular signals revealing abnormal head impulse test and caloric hyporeflexia, and ataxia in patients with WE. **Conclusion:** Using whole brain resting-state connectivity analysis in WE patients with bilateral hVOR deficits, we show that enduring bilateral deficient or missing vestibular input leads to changes in resting-state connectivity of the brain. They may account for the patients' persistent/transient deficits in visuo-spatial attention, spatial orientation and unsteadiness.

P-2-110

Anatomical subtypes of subjective memory impairment individuals

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Background & Objectives: Although it is well known that subjective memory impairment (SMI) is a heterogeneous group, its subtypes are not well defined. We investigated whether individuals with SMI can be categorized into anatomical subtypes using 3-dimensional MRI. **Method:** We consecutively recruited 613 SMI individuals from the Memory Disorders Clinic at Samsung Medical Center from July 2007 to December 2012. All individuals completed neuropsychological tests and 3-tesla T1 weighted MRI. The images were processed to measure cortical thickness and hierarchical agglomerative cluster analysis was performed using Ward's clustering linkage. We compared cortical thinning pattern of SMI subtypes with each other using a general linear model with random field theory corrected $p < 0.05$. In addition, scores on neuropsychological tests were compared between the subtypes. **Results:** At the 3-cluster level, individuals were divided into diffuse atrophy subtype (n=212, 34.6%), bilateral medial temporal atrophy subtype (n=79, 12.9%) and minimal atrophy subtype (n=321, 52.4%). Individuals in the medial temporal atrophy subtype were older and had more vascular risk factors such as diabetes and hyperlipidemia compared to other two subtypes. Individuals in the minimal atrophy subtype showed significantly high frequency of female and high geriatric depression scale scores compared to other two subtypes. Compared with individuals in other subtypes, those in the medial temporal atrophy subtype scored the lowest on memory and frontal-executive function tests. However, after controlling for age, gender, and education, the significant differences in neuropsychological test score disappeared. **Conclusion:** Our findings suggest that SMI can be categorized into several anatomical subtypes with distinct demographics features.

P-2-111**A case of rapid progressive cognitive impairment caused by human immunodeficiency virus**

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Background & Significance: Since the introduction of HAART (Highly active antiretroviral therapy), the focus of HIV (human immunodeficiency virus) treatment became to manage chronic complications. Cognitive impairment is one of the long-lasting complication related to HIV, but it generally occurs in advanced stage. We report a case of rapid progressive cognitive impairment who had no specific past medical history but was finally diagnosed to HIV-associated neurocognitive disorders. **Case:** A 48 year-old man who had no previous medical history was referred to our memory and dementia clinic for evaluation of his progressive memory impairment and spatial disorientation. His memory deterioration had exacerbated during a month. He could not easily remember even the names of his friends and close relatives. On neurologic examination, his mentality was alert and orientation was relatively intact. He had no motor weakness, sensory deficits, or any other neurological deficits. The patient scored 29/30 on the Korean version of Mini-Mental state examination (K-MMSE), but detailed neuropsychological test showed significant multiple cognitive impairment in all cognitive domains. FLAIR & T2-weighted brain MR imaging revealed ill-defined small high signal intensity lesions on left medial temporal gyrus. Analysis of his cerebrospinal fluid revealed normal levels of sugars and proteins and mild pleocytosis (WBC count was 8 cells/mm³). Other causes of infections, such as, bacteria, tuberculosis, Aspergillus, cryptococcus, toxoplasma and cytomegalo virus were not observed in CSF. However, the serum and CSF anti-HIV were strongly positive, and CD4 T-cell count was decreased in 206 cells/mm³. After diagnosis of mild cognitive impairment due to HIV-associated neurocognitive disorder, we started HAART. In 3 months follow-up examination after starting HAART, significant cognitive deterioration was not observed compared to previous assessment when he admitted to hospital. **Conclusions or Comments:** We might consider as a differential diagnosis for HIV-associated neurocognitive disorder if young adult shows progressive cognitive impairments without any relevant causes of cognitive disorders.

P-2-112**The distribution and clinical impact of Apolipoprotein E4 among subjective memory impairment and early mild cognitive impairment**Hanna CHO¹, Young-Eun KIM², Duk L. NA³, Chang-Seok KI², Sang Won SEO³

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Background & Objectives: Recent advances of Alzheimer's disease (AD) biomarkers have prompted more focus on the subjective memory impairments (SMI) and early-stage amnesic mild cognitive impairment (E-aMCI). These preclinical or prodromal stages of AD is important to evaluate underlying mechanism for the future development of AD, however, little is known about the distribution of the APOE e4 genotype, which is the most important genetic variant associated with AD, and its clinical impact in these groups. Therefore, the aim of this study was to investigate the distribution of APOE e4 genotypes and to compare the neuropsychological results between APOE e4 carrier and non-carrier across the full clinical AD spectrum including AD, late-aMCI (L-aMCI), E-aMCI, SMI, and control groups. **Method:** We prospectively recruited 713 patients with AD, 735 patients with aMCI, and 575 pa-

tients with SMI, all of whom had been clinically diagnosed at Samsung Medical Center. After obtaining of informed consent, all of the patients underwent a battery of neuropsychological tests, conventional brain MRI scans, and APOE4 genotyping. A large dataset from 8,260 individuals who took routine health examination from 1995 to 2002 at the Health Promotion Center of Samsung Medical Center was used as controls. **Results:** The frequency of the APOE e4 allele showed an ordered fashion in the AD (30.8%), L-aMCI (24.0%), E-aMCI (15.1%), SMI (11.7%), and control (9.1%) groups. In the statistical comparisons of APOE e3/e3 vs. e3/e4 genotype between diagnostic groups, relative to control group, all patients' group had higher frequency of APOE e3/e4. Relative to SMI or E-aMCI group, AD and L-aMCI groups had higher frequency of APOE e3/e4 genotype. Relative to L-aMCI group, AD group had higher frequency of APOE e3/e4 genotype. However there was no significant difference between the E-aMCI and SMI groups. In the neuropsychological test results between APOE e4 carrier and non-carrier, SMI and E-aMCI group showed no significant difference. On the contrary, L-aMCI group showed memory impairment in APOE e4 carrier, and AD group showed cognitive impairment in the attention, language, visuospatial, memory, and frontal functions in APOE e4 carrier. **Conclusion:** Our findings suggested that SMI and E-aMCI had higher frequency of APOE4 genotype, but the clinical impact of APOE4 were not prominent in two groups, unlike L-aMCI and AD.

P-2-113**Tract-specific correlates of neuropsychological deficits in patients with subcortical vascular cognitive impairment**Na-Yeon JUNG¹, Cheol E HAN², Hee Jin KIM³, Sang Wook YOO², Hee-Jong KIM², Eun-Joo KIM⁴, Duk L. NA³, Joon-Kyung SEONG², Sang Won SEO³

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Background & Objectives: We investigated the tract-specific correlates of neuropsychological deficits in patients with subcortical vascular cognitive impairment (SVCI) using Tract-Specific Statistical Analysis (TSSA) which is our new method of diffusion tensor imaging (DTI) analysis. **Method:** A total of 114 SVCI and age, sex, and education matched 55 normal cognition (NC) subjects who underwent DTI and neuropsychological tests were included. Using TSSA, we classified fiber tracts into major fiber tracts such as anterior thalamic radiation (ATR), cingulum (CG), corticospinal tract (CST), inferior fronto-occipital fasciculus (IFO), inferior-longitudinal fasciculus (ILF), superior-longitudinal fasciculus (SLF), and uncinata fasciculus (UNC). We selected a representative streamline from each fiber tract. The group comparison between NC and SVCI and correlational analysis between fractional anisotropy (FA) values and scores of neuropsychological tests were performed. **Results:** Relative to NC subjects, patients with SVCI showed significantly decreased FA values in the bilateral ATR, CG, SLF, UNC, and CST and left ILF. FA values in the middle portion of CG were associated with scores in language, visuospatial, memory and frontal functions. FA values in the anterior portion of the ATR were associated with scores in attention, memory and frontal executive functions, while FA values in its middle portion were associated with score in language function. FA values in the posterior portion of SLF were associated with visuospatial dysfunction while FA values in its middle portion were associated with memory impairments. **Conclusion:** Our findings suggested that disconnection of specific white matter tracts, especially in neighboring regions to corresponding gray matter, contributed to specific cognitive impairments in patients with SVCI. Furthermore, our findings provided us better under-

standings of patho-mechanism of cognitive impairments in patients with SVCI.

P-2-114

Novel PSEN1 (G209A) mutation in a case of early onset Alzheimer dementia

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Background & Significance: Three main genes are described as causative genes for early onset Alzheimer dementia (EOAD): APP, PSEN1 and PSEN2. **Case:** We describe a 54-year-old woman with Alzheimer dementia had a novel PSEN1 mutation. One of two daughters also has same mutation, G209A in the TM-IV of PS1 protein. Her mother had unspecified dementia began at the age of 40s. **Conclusions or Comments:** PolyPhen2 and SIFT prediction suggested that G209A might be a damaging variant with high scores. 3D modeling revealed that G209A exchange could result significant changes in the PS1 protein. We report a case of EOAD patient with her daughter of novel PSEN1 (G209A) mutation.

P-2-115

Screening of cognitive dysfunction in chronic hemodialysis patients - comparison of the mini mental state exam to the Montreal cognitive assessment

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Background & Objectives: The prevalence of cognitive impairment in end stage renal disease patients undergoing hemodialysis (HD) therapy is much higher than general population. We compared two commonly used screening tests; the Korean version of Montreal Cognitive Assessment (K-MoCA) and Mini-mental State Examination (K-MMSE) to detect cognitive dysfunction in ESRD patients. The cognitive profile of HD patients was also explored using comprehensive neuropsychological test battery. **Method:** 20 HD patients and 24 healthy controls, with matching age, education, and gender ratio, were recruited and assessed with the validated K-MoCA, the K-MMSE and a comprehensive neuropsychology (NP) test battery. The NP test battery included to measure 5 cognitive domains; attention, visuospatial function, language, memory and executive function/working memory. Korean version of Geriatric depression scale was also applied. All test scores were transformed to standardized z-scores with appropriate age and education level prior to group comparison for more accurate cognition measures. Receiver operating characteristic (ROC) with area under the curve (AUC) was plotted to compare the appropriateness of the K-MMSE and K-MoCA to differentiate the MCI and NC groups. Composite scores for 5 cognitive domains were calculated by averaging each sub-test to investigate cognitive impairment profile of HD patients. **Results:** No significant group difference was found in the K-MMSE score regardless of using total score or standardized z-score. However, HD patients achieved lower scores in the K-MoCA and the significant group differences were found in both the total score and the standardized z-score. The K-MMSE ROC AUC (95% CL) in the total score was 0.57 (0.39-0.75) and the ROC AUC (95% CL) when converted to z-score was 0.60 (0.42-0.78). The K-MoCA ROC AUC in the total score was 0.72 (0.57- 0.87) and the ROC AUC (95% CL) in

z-score was 0.77 (0.63-0.91). The correlation analysis revealed strong positive correlations between the z-score of K-MoCA and the composite scores of visuospatial function, executive function/working memory, and the language domains whereas the memory and the attention showed a moderate positive correlation. HD patients performed significantly worse than the controls in all cognitive domains as well as all of the neuropsychological sub-tests except for the trail making test part B. **Conclusion:** The K-MoCA might be more sensitive to detect cognitive impairment in HD patients while the K-MMSE failed to detect it. The K-MoCA demonstrated to have moderate to strong positive correlations with existing comprehensive neuropsychological tests. The K-MoCA is a more appropriate screening test for assessment of cognitive function in HD patients. HD patients seemed to have significant cognitive impairments in 5 cognitive domains; attention, language, visuospatial function, memory and executive function even without subjective cognitive impairment.

P-2-116

Glucose metabolism in early onset versus late onset behavioral variant frontotemporal dementia

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Background & Objectives: The aim of this study was to compare the overall glucose metabolism between early onset and late onset behavioral variant frontotemporal dementia (bvFTD) to investigate whether their metabolic deterioration was different even at the same dementia severity. **Method:** Thirty one patients with early onset bvFTD (mean age: 61.6±4.9), 12 with late onset bvFTD (mean age: 75.3±5.3) and 65 healthy volunteers (mean age: 64.9±7.3) were recruited from the dementia clinics of 3 tertiary referral hospitals. Glucose hypometabolic patterns were evaluated by comparing patients with early onset bvFTD and late onset bvFTD with 65 healthy controls using voxel-based statistical parametric mapping. **Results:** There were no significant differences in Korean version of Mini-Mental State Examination, clinical dementia rating (CDR), and sum of boxes scores of FTD-CDR between early onset and late onset bvFTD patients. However, overall glucose hypometabolism of early onset bvFTD patients was much greater in magnitude and extent involving bifrontal and anterior temporal areas than that of late onset bvFTD patients. **Conclusion:** The results of greater hypometabolism in early onset than late onset patients with bvFTD even in the same severity of dementia were consistent with those of our previous research comparing glucose metabolism between patients with early onset versus late onset Alzheimer's disease, reflecting greater functional reserve in younger than in older subjects.

P-2-117

The correlation between CSF biomarkers, regional brain atrophy, cognitive performance in early onset neurodegenerative dementia

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Background & Objectives: Although neurodegenerative process starts decades before the onset of the illness, the diagnosis of ND dementia requires clinical symptoms and relevant regional cerebral atrophic changes. The early onset neurodegenerative dementia (EOND) is clinically different from late onset

neurodegenerative dementia by genetic predisposition, more rapid progression and without definite relevant cerebral atrophy. Amyloid beta ($A\beta$) and hyperphosphorylated tau (p-tau) are neuropathological hallmarks of Alzheimer's disease (AD) but total tau (T-tau) pathology is also evident in other neurodegenerative dementia such as Frontotemporal dementia (FTD), Corticobasal ganglionic dementia (CBD) or progressive supranuclear palsy (PSP). In recent years, these biochemical markers has enabled earlier diagnosis of these ND diseases, but the functional model to form the association between $A\beta$, tau pathology and regional brain atrophy has not been well defined. To determine the relationship between these neuropathological changes, we aimed to find out whether each known CSF biomarkers contribute differently to the grades of focal cerebral atrophy and also cognitive dysfunction in EOND dementia. **Method:** We performed quantitative brain MRI analysis using Visual Rating Atrophy Scale and CSF biomarkers evaluations in 25 patients of de novo early onset dementia from 2013 to 2014. The cerebral atrophy were rated separated for medial temporal lobe (MTL), Lateral temporal lobe (LTL), anterior temporal lobe (ATL) orbitofrontal lobe (OFL), parietal lobe (PAR), ventricular enlargement (VE) and whole brain (WB) using Visual Rating Scale by the method according to the literature. Three CSF biomarkers, $A\beta$ -42, T-tau and p-tau epitopes, has been evaluation using ELISA test from one of the author's center (Park, SUH). Cognitive performance was tested by the Seoul Neuropsychological Screening Battery in all subjects. **Results:** The 24 EOND patients had mean onset ages of 58.46 ± 10.11 ; 18 of EOAD, 4 of FTD and 2 of CBS. Of those EOND patients, two patients had PSEN1 and PSEN2 mutations. In whole group, there was no correlation between levels of CSF biomarkers and severity of each or whole cerebral atrophy ratings in whole EOND, as well as the sub-analysis in clinical probable EOAD including genetic EOAD. Only in clinical probable EOAD group without genetic EOAD, the levels of T-tau showed a positive correlation with severity of LT, PAR and WB atrophy ($R=0.675$ $p=0.023$, $R=0.642$ $p=0.033$, $R=0.5489$ $p=0.08$). **Conclusion:** Overall, the literature indicates that $A\beta$ -42, T-tau, p-tau, T-tau/ $A\beta$ -42 and p-tau/ $A\beta$ -42 predict development of dementia. The level of T-tau suggested possible pathologic role in the focal cerebral atrophy in EOAD, although which warrants further analysis for the conclusion.

P-2-118

A episode like transient global amnesia associated with intraventricular tumor adjacent to the fornix

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Background & Significance: Transient global amnesia (TGA) is a syndrome of temporary and reversible disruption of short-term memory accompanied with repetitive questioning. Symptoms of TGA resolve within 24 hours. Although the etiology is incompletely understood, TGA may be related to vulnerability of hippocampal CA1 neurons to metabolic stress. In the vast majority of cases, TGA is not associated with structural abnormalities on standard brain imaging techniques. We report a case with a TGA-like episode associated with intraventricular hemorrhagic metastatic tumor which disrupted the fornix **Case:** A 62-year-old right-handed woman presented to the cognitive neurology clinic with a chief complaint of headache and amnesia. She was in generally good health. According to her son, she suddenly began to be confused and disoriented around at noon. She asked repetitively questions. The patient stated that she had a severe headache 4 hours before the amnesic episode. There was no history of headache. Her repetitive asking recovered on the day of admission, but, her son reported that the patient developed memory problem after the episode. On the past medical history, she had hypertension,

diabetes mellitus, angina pectoris and depressive disorder. There was no history of recent head injury or epilepsy. She was suffering from right side facial pain and edema and dentist was planning to remove the mass of maxillary oral cavity thought to be cause of buccal abscess. She worked as a baby sitter and regularly enjoyed taking photographs. There was no family history of cognitive disorders. The physical examination revealed right side facial erythematous swelling. Neurologic examination and laboratory workup was unremarkable. On the neuropsychological test, the patient scored below one standard deviation (16 %tile) on verbal immediate recall, verbal and visual recognition of Seoul Verbal Learning Test and Rey copy test. Brain image revealed hemorrhagic metastatic tumor in right lateral ventricle with minimal intraventricular hemorrhage and multiple bone metastasis. Intraventricular tumor was adjacent to the fornix. Biopsy of masses of skull and maxillary oral cavity was done and pathologic diagnosis was confirmed as undifferentiated pleomorphic sarcoma. Although she received chemotherapy and radiotherapy, she died of cancer progression after 5 months. **Conclusions or Comments:** Our patient presented with similar symptoms of TGA and had evidence of intraventricular hemorrhagic metastatic tumor adjacent to the fornix. The fornix, one of structures in Papez circuit. There was a report of transient global amnesia associated with a unilateral infarction of the fornix. TGA is a benign syndrome. However, other constitutional symptom like severe headache can be a red flag for other etiologies like our case.

P-2-119

Neuropsychiatric characteristics of PiB-negative subcortical vascular dementia versus behavioral variant frontotemporal dementia

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Background & Objectives: Neuropsychiatric symptoms of subcortical vascular dementia (SVaD) are mainly associated with damage to frontal-subcortical circuits and may be similar to symptoms of behavioral variant frontotemporal dementia (bvFTD). The aim of this study was to determine whether the neuropsychiatric manifestations of the Pittsburgh compound B (PiB)-negative SVaD and bvFTD groups differ. **Method:** We compared the Caregiver-Administered Neuropsychiatry Inventory (CGA-NPI) between 48 patients with PiB(-) SVaD and 31 patients with bvFTD. A stepwise logistic regression was applied to determine the best model to predict SVaD. **Results:** The SVaD group showed a higher frequency of depression, whereas the bvFTD group had a higher frequency of elation, aberrant motor behavior and appetite/eating disorders. Regarding NPI subscores, the bvFTD group had greater severity of elation, apathy, disinhibition, aberrant motor behavior and appetite/eating disorders, whereas SVaD did not have significantly higher subscores in any domains. The most predictive models that compare the SVaD and bvFTD groups included as follows: (1) presence of depression and appetite/eating disorders, (2) NPI subscores of depression, irritability and aberrant motor behavior. **Conclusion:** Apart from apathy, SVaD differed from bvFTD in that negative symptoms were more common in SVaD than bvFTD, whereas positive symptoms were predominant in bvFTD compared to SVaD.

P-2-120

Identification of PSEN1 variants in Korean patients with early-onset Alzheimer's Disease

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Background & Objectives: Alzheimer's disease (AD) is the most common type of dementia in the elderly with 50-70% of dementia cases. There are three known causative genes for AD: the amyloid precursor protein gene (APP) on chromosome 21q21.3, the presenilin-1 gene (PSEN1) on chromosome 14q24.3, and the presenilin-2 gene (PSEN2) on chromosome 1q42.13. In Korean population, only four EOAD cases with PSEN1 mutations and one case with APP mutation have been reported thus far. However, there is no data on the frequency of PSEN1 variants in a consecutive series of AD cases. In this study, we investigated the frequency and spectrum of PSEN1 variants in a consecutive series of AD patients for determining the genetic background of AD patients in Korea. **Method:** Among 1,101 patients with AD who visited the Memory Disorder Clinic at Samsung Medical Center from January 2007 to December 2013, 32 patients with clinically suspicious familial AD were included in this study. They had early onset of disease (< 50 years) or strong family history of autosomal dominant pattern. After obtaining informed consent, peripheral blood specimens were collected and genetic analysis performed. **Results:** A total of 8 (25%) of the 32 patients had PSEN1 variants: two pathogenic variants and six variant of unknown significances (VUSs). Two pathogenic variants (p.E120K and p.S170F), were detected in exon 5 (E120K) and exon 6 (S170F). Six VUSs were found in exon 5 (T119I, Y159C), exon 8 (L282P A285S), and exon 11 (T389H, T389S). Their phenotypes were consistent with those of previously reported cases: early onset, frequent parietal symptoms and abnormal movements, and rapid progression. **Conclusion:** We identified two known mutations and six VUS of PSEN1 gene in 8 EOAD patients. Although mutations in these genes are a rare cause of AD, the identification of these genes and mutations will contribute to a better understanding of the genetic background in Korean EOAD patients.

P-2-121

Decreased blood pressure after rapid intravenous fosphenytoin infusion

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Background & Objectives: Fosphenytoin (FOS) is a phosphate ester pro-drug of phenytoin and offers several benefits over intravenous (IV) phenytoin, including marked decreased in pain and irritation at the infusion site, fewer reductions in infusion rate and lower cardiovascular complications. We investigated clinical characteristics of hypotension following IV FOS infusion with loading dose. **Method:** We reviewed medical records of consecutive patients received with IV FOS between July 2013 and June 2015. We analyzed various clinical features; demographic data, comorbidity, drug history, seizure etiologies, seizure types, incidence of hypotension/cardiac arrhythmia and dosing data (total dose, concentration and infusion rate of intravenous FOS). We identified the difference between patients with hypotension after IV FOS and those without hypotension. **Results:** Among a total of 28 patients, 11 (39%) had hypotension associated with IV FOS and 2 of these individuals also had atrioventricular block during IV FOS infusion. The others (17, 61%) had no cardiovascular complications. Analysis of demographic and clinical data found statistically significant associations between hypotension and both old age (above 60 years, P=0.034) and the presence of systemic infection (P=0.04). Among dosing data, high total dose (>1400mg PE) increased the risk of hypotension after IV FOS infusion (P=0.04). There were fatal complications in four

patients; one patient died of a cardiac arrhythmia directly related to an infusion of IV FOS and the other patients received inotropic agents due to severe hypotension. **Conclusion:** Hypotension associated IV FOS infusion frequently occurred, especially in elderly patients. The presence of systemic infection and total dose of FOS influenced the development of hypotension. FOS can produce more hypotension than expected and should be infused under careful cardiovascular monitoring.

P-2-122

Seizure disorder presenting as stroke-like symptoms

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Background & Objectives: In cases admitted with only postictal neurological deficit in seizure disorder, which other persons did not witness seizure or seizure-like movements, it is difficult to differentiate from strokes, especially in patients with cerebrovascular risk factors. Seizure is reversible disorder that can be treated with appropriate therapies and should be diagnosed at an early stage. We investigated the characteristics of patients presenting with stroke-like symptoms. **Method:** Of patients admitted with seizure or epilepsy on our hospital between 2005 and 2015, 7 patients had a history of stroke-like symptoms. We investigated the clinical features and brain imaging and electroencephalogram (EEG) findings using electronic medical records. **Results:** Of the seven patients, three had lobar intracranial hemorrhage, two had cortical infarctions, and two had lacunar infarctions on brain MRI. With the exception of one patient who presented with amnesia, all patients complained of hemiparesis, and three out of the seven patients presented with confusion or drowsy mental status. Several symptoms, such as aphasia, subtle facial twitching, gaze preference, and hemianopsia were also observed with hemiparesis in each patient. The EEG findings revealed that three out of the seven patients had NCPSE with subclinical seizures. In two patients with NCPSE, brain single-photon emission computed tomography (SPECT) showed hyperperfusion in the whole hemisphere, including a previous focal stroke lesion, and one of these patients showed high signal intensity around the previous stroke lesion on diffusion-weighted imaging. Other four patients had focal slowing or interictal sharp waves on EEG with hypoperfusion in SPECT that were suspicious findings of postictal neurological deficit. All patients had recovered to their last normal status after appropriated anticonvulsant treatment, and the median duration of symptoms was 4 days (range, 30 min-9 days). **Conclusion:** Seizure could be presented with stroke-like symptoms or signs such as unexplained mental status and focal neurological deficit. Early EEG and brain imaging should be performed to reach the accurate diagnosis.

P-2-123

Prolonged loss of consciousness in patients with reflex syncope: diagnostic challenge between epilepsy and syncope

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Background & Objectives: Syncope is a transient loss of consciousness (LOC) due to global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneously complete recovery. Consciousness may be lost in several disorders, but the mechanism is different from transient global cerebral hypoperfusion. Among these disorders epilepsy is a most common condition from which clinicians should differentiate syncope. **Method:** The consecutive patients with LOC of uncertain origin were initially evaluated with 24 hours video-EEG monitoring under the suspicion of 1st unprovoked seizures. If

there were no definite interictal epileptiform discharges during 24 hours video-EEG monitoring, the patients were consulted to the department of cardiology for the possibility of syncope including cardiac evaluation. The diagnosis of reflex syncope was made of tilt table test and the positive result was considered when the induction of either reflex hypotension/bradycardia or delayed orthostatic hypotension (decrease in systolic blood pressure ≥ 20 mmHg and in diastolic blood pressure ≥ 10 mmHg) associated with syncope or pre-syncope. Inclusions for this study were as follows; the patients with 1) prolonged LOC for at least 15 minutes, 2) evidence suggesting reflex syncope: positive with tilt table test, 3) normal MRI, 4) no evidence of cardiac syncope, 5) no evidence of epileptiform discharges during 24 hours video-EEG monitoring. The clinical variables included age, sex, duration of LOC, frequency of the previous syncope, the history from a witness using a standardized questionnaire. Also, we evaluated the distribution of the eccentric cortical thickness in the brain expressed as percentile comparing to age, sex matched normal control using freesurfer 5.1. **Results:** Of 26 patients evaluated with 24 hours video-EEG monitoring and cardiac evaluation for LOC of uncertain origin, 9 patients had epilepsy, 9 patients had syncope and 8 patients were able to be diagnosed neither epilepsy nor syncope. Of 9 patients with syncope, 7 patients met the inclusion criteria. The duration of LOC was variable from 15mins to 30mins. The age of onset was 45 ± 24 years old, and 4 patients were male. All of the patients had a history of at least once attack of typical reflex syncope before attacking the prolonged LOC attack. The distribution of eccentric cortical thickness was wider as comparing to normal control; the eccentric cortical thickness was distributed in the right superior temporal, superior frontal, superior parietal, insular, paracentral cortex and left superior frontal, paracentral. **Conclusion:** A prolonged LOC can be possible in the case of syncope, and detailed history taking of previous typical reflex syncope is critical for differentiating it from epilepsy.

P-2-124

Alcohol-related seizures presenting with nonconvulsive status epilepticus and a thalamic lesion: an atypical presentation of subacute encephalopathy with Seizures in Chronic Alcoholism (SESA) Syndrome

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Background & Significance: Subacute encephalopathy with seizures in chronic alcoholism (SESA) syndrome is a rare clinical manifestation in patients with chronic alcohol abuse. Typical clinical features are characterized by seizures, alteration of mental functions, focal neurological deficit, and focal electroencephalography (EEG) abnormality. In most reports, cortical, but not subcortical, lesions are detected. **Case:** A 52-year-old man presented with prolonged confusion and aphasia after a generalized seizure. Continuous rhythmic focal ictal discharges were observed on EEG and a focal thalamic lesion was detected by MRI. Symptoms, epileptiform discharge, and the thalamic lesion disappeared after treatment with an antiepileptic drug. **Conclusions or Comments:** This is the first report of atypical subacute encephalopathy with seizures in chronic alcoholism (SESA) syndrome characterized by reversible EEG-demonstrated partial NCSE and subcortical MRI lesion.

P-2-125

Different EEG pattern between waking and sleeping status during hypoglycemia in type 1 DM patient

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Background & Significance: EEG changes of hypoglycemia are known as mixed frequency theta to delta activity with increased amplitude than the initial background rhythm. However, there are few reports about hypoglycemia induced EEG changes between waking and sleeping status. **Case:** We experienced a patient who showed different pattern of EEG changes after hypoglycemia according to waking and sleeping status. A 45-year-old man who had been diagnosed type 1 DM for 15 years presented EEG pattern of increased amplitude of theta slowing after hypoglycemia. Such EEG finding was disappeared as the patient was going to fall asleep which reappeared when he woke up. **Conclusions or Comments:** This case suggests a possibility of different EEG pattern between waking and sleeping status during hypoglycemia.

P-2-126

Reversible MRI abnormalities in a patient with complex partial status epilepticus

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Background & Significance: As computed tomography (CT) started to be used in patients with seizures, there were some reports that some periaxial abnormalities in CT disappeared spontaneously in follow-up studies. With the advance in imaging modalities, magnetic resonance imaging (MRI) is essential for evaluation of patients presenting with seizures. In MRI, these seizure-induced abnormalities were also described in several case reports and small case series. These periaxial abnormalities were reversible with no specific treatment except antiepileptic drugs, suggesting that these were not structural but functional, possibly resulting from the cerebral edema induced by seizure activity. Recognizing these reversible changes in MRI is clinically important to avoid unnecessary treatment and test. We reported reversible MR diffusion-weighted imaging in a patient with complex partial status epilepticus. **Case:** A 71-year-old man was brought to the emergency room for prolonged tonic posture with myoclonic movement of left arm and altered mental status. 4 months ago, he had clipping surgery of A-com aneurysm, which was incidental findings without symptoms. 2 months after surgery, bilateral chronic subdural hemorrhage was observed in follow-up CT scan and burr-hole drainage was done. He was discharged after operation without acute complications but remnant subdural hematoma existed along bilateral cerebral convexity. 5 days before admission, he presented 1 episode of complex partial seizure with clonic movements of left leg. After the event, drowsy mental status and tonic posture with intermittent myoclonic movement of left arm were prolonged. Weakness of left arm and leg was observed at the time of admission. MRI showed high signal intensity in right frontal, parietal lobe cortex, insular cortex, anteromedial temporal lobe including amygdala and hippocampal head, and ventromedial thalamus in DWI and FLAIR image. Apparent diffusion coefficient (ADC) showed high signal and partially low signal in the corresponding area. Very frequent periodic lateralized epileptiform discharges (PLEDs) on right frontocentral area (Fp2, F4) were observed on EEG. In CSF analysis, a WBC count of $0/\mu\text{L}$, a glucose level of 38.7mg/dL , a protein level of 69mg/dL was confirmed and viral PCRs including herpes simplex virus-1, II, herpes zoster virus, Epstein-Barr virus and cytomegalovirus were negative. There was no evidence of bacterial, tuberculosis or fungal infection in CSF analysis. He was treated with IV acyclovir and antiepileptic drugs. DWI abnormalities were normalized in follow-up MRI after 4 days after first MRI, but persisted abnormalities were observed in FLAIR images. His mental status and weakness of left side was recovered after seizure was controlled, but mild cognitive impairment was persisted at the time of discharge. This case was concluded as com-

plex partial status epilepticus induced by chronic subdural hematoma. **Conclusions or Comments:** MRI is now essential to evaluate a patient with seizures. Seizure-induced MRI changes are variable among patients. They are usually high signals in DWI, T2 and FLAIR images. They might be focal around the seizure focus, or diffuse changes which are remote from the seizure focus. And they are fully reversible in the follow-up MRI after few days, persistent for months, or are permanent and induce atrophic change. Our patient revealed reversible changes in DWI but persisted T2/FLAIR high signal intensity. It may be associated with duration of seizure. It was previously reported that there was a trend between the duration of seizure and the possibility that MR changes may persist. It is important to understand the MRI changes induced by seizure activity so that we can avoid unnecessary treatment and diagnostic studies that might delay proper management for the patient.

P-2-127

Lesional focal seizures triggered by the hyperglycemia

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Background & Significance: Focal seizures could be triggered by the hyperglycemia. However the reasons for the development of focal seizures in the hyperglycemic patients are still unclear. Herein, we investigated the seizure types in the hyperglycemic patients and describe the factors affecting the semiology. **Case:** We retrospectively identified patients with hyperglycemia and focal seizures who were admitted or consulted to the department of neurology at our institution between January, 2013 and January, 2015, and investigated semiology, brain images, and electroencephalography (EEG) findings. The patients with previous seizure attack and unwitnessed seizure events were excluded. Total four patients were selected. Mean age was 59.3 years. Three were male. All patients had underlying structural brain lesions. One had hemosiderin rim in the right temporo-occipital region. Other three patients had encephalomalacia in the left middle cerebral artery territory, bi-frontal, and right frontal regions, respectively. The semiology and EEG features were corresponded with the underlying brain lesions in these patients. Blood glucose levels were 337.3 mg/dl. **Conclusions or Comments:** Focal seizures are frequent in the hyperglycemic patients. The pre-existing abnormal cortical structures might be more vulnerable to the hyperglycemic stress than without. Further large studies are required to understand the characteristics of seizure in the hyperglycemia.

P-2-128

Anti-GAD antibody associated encephalitis

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Background & Significance: Anti-glutamic acid decarboxylase (GAD) antibody plays a crucial role in neurologic conditions and diabetes. GAD is the biosynthesizing enzyme of neurotransmitter γ -aminobutyric acid (GABA). In neurological disease, antibodies against GAD associated with stiff person syndrome, cerebellar ataxia, dementia and epilepsy. These are considered to be associated with dysfunction of the GABAergic system. Treatment usually focused on modification of the immune response and enhancement of GABAergic activity. We report a patient with anti-GAD antibody encephalitis and diabetes who developed relapsing-remitting pattern seizure and stroke. **Case:** A 44-year-old man with type 2 diabetes admitted ER with facial twitching. Brain MRI showed focal T2 hyperintense lesion in right precentral gyrus. Brain SPECT showed increased regional cerebral perfusion on right fronto-parietal cortex. After 6 months later, generalized tonic clonic seizure was occurred and newly developed focal T2 high signal in right occipital and

disappeared previous lesion. Analysis of the cerebrospinal fluid (CSF) showed no pleocytosis. Thyroid function test and chest CT showed no significant findings. Serum lactate level was found to be increased to 5.4 mmol/L and MELAS gene test was negative. HbA1c was 7.2% with serum anti-GAD antibody level was 1.22 U/mL (normal range: 0-1.0). Anti-GAD antibody in CSF showed also positive. Other autoimmune study was normal. He implemented intravenous immunoglobulin and immunosuppressant. However, 6 months later, he developed sensory aphasia with newly developed diffusion restriction in Lt. temporal lobe. Multifocal areas of decreased glucose metabolism at right frontal lobe, right parietal lobe, and Lt. cerebellum and mildly at right occipital lobe with relatively increased FDG uptake at Lt. temporal lobe. A five-day course of high-dose (0.4g/kg/day) intravenous steroid pulse, followed by 60mg oral prednisone was implemented and immunosuppressant. **Conclusions or Comments:** We report a patient who manifests neurological symptoms as epilepsy with progressive as well as a relapsing-remitting course. Anti-GAD antibodies associated encephalopathy is rare, and shows various neurological manifestations. Thus, we should be careful who pharmaco-resistant epilepsy with diabetes patients.

P-2-129

Sleep deprivation electroencephalographic findings in the patient with sporadic hemiplegic migraine

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Background & Significance: Hemiplegic migraine is an uncommon type of migraine accompanied by various degrees of motor weakness during the attack, which is classified into a sporadic and a familial subtype. The EEG findings of hemiplegic migraine during severe headache are often diffuse slowing contralateral to the weakened limb, but are normal in many cases between attacks. In this report, we would like to describe a case with hemiplegic migraine who revealed abnormal EEG findings after sleep deprivation, which was not observed in the routine EEG study without sleep restriction. **Case:** We present a 52-year old woman who suffered migrainous headache accompanying right arm weakness and mild aphasic symptoms for 2 years. She had a history of diabetes mellitus and hypertension for several years, but her laboratory and imaging studies revealed normal. During the medical treatment for migraine, she underwent five times of EEG. The first four of routine EEG exams (without sleep deprivation) were non-specific. However, on the seventh day of hospitalization, the fifth conventional EEG after 24 hours deprivation of sleep showed intermittent slowing on the left parieto-occipital area, and this pattern of EEG was detected during photic stimulation. Additionally, larger (than normal range) amplitude of POSTs (Positive occipital sharp transients) on the left side was observed compared to the other side during natural sleep. **Conclusions or Comments:** There have been several reports on the relationship between EEG and hemiplegic migraine. Nevertheless, EEG findings after sleep deprivation has not been reported in the hemiplegic migraine patients. Because insufficient sleep time can trigger the recurrence, the difference of EEG waves before and after sleep deprivation would be another clue for diagnosis of the illness.

P-2-130

Selective serotonin reuptake inhibitor induced posterior reversible encephalopathy in a normotensive patient

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Background & Significance: Posterior reversible leucoencephalopathy syndrome (PRES) is a clinico-radiological syndrome. The most common clinical features of PRES are headache, nausea and vomiting, altered mental status, seizures, cortical blindness and other visual abnormalities, and transient motor deficits. The main finding in neuroimaging is posterior white matter edema, with symmetrical involvement of the parietal and occipital lobes. Nevertheless PRES can be associated with several conditions, including acute or chronic renal failure, blood transfusion, organ transplant, infection, autoimmune disorder, immunosuppression. **Case:** We report a case of PRES in normotensive patient with SSRI medication. 22-year-old woman without underlying disease developed acute onset of generalized tonic-clonic seizure. This condition spontaneously was resolved within 2 minutes. Her altered consciousness gradually improved and then she was alert state when she arrived at our hospital. In Hematological tests, hyponatremia and hypoosmolarity were showed. Cerebrospinal fluid (CSF) analysis was normal. Magnetic resonance imaging (MRI) showed regions of T2 signal in bilateral parieto-occipital lobes. Diffusion weighted neuroimaging (DWI) was carried out which revealed finding suggestive of PRES. The patient was treated with supportive care which followed improvement in vital sign. **Conclusions or Comments:** Although rare, SSRI should be considered as a potential cause of PRES in patient with diuretics. With this case report we have tried to create awareness and vigilance about prescribing SSRI and diuretics.

P-2-131

A case of sick sinus syndrome as seizure-like manifestation

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Background & Significance: Although pathophysiologically distinct, syncope and seizures share clinical characteristics which may make diagnosis difficult. Several studies have reported that 30-42% of patients initially thought to have epileptic seizures were later found to have convulsive syncope due to cardiovascular cause. **Case:** A 89-year-old man who was diagnosed COPD and CAOD, came to neurology out-patient clinic with a history of several attacks of convulsive movement in the preceding 3 months. The episodes usually happened after a feeling of spreading from the stomach to the brain and. After convulsive movement, he was confused and disoriented for 2-3 seconds. While EKG and Echocardiography showed normal, video EEG combined with Holter monitoring revealed that his attacks were synchronous with periods of sick sinus syndrom. Permanent cardiac pacemaker was implanted and his symptoms disappeared. **Conclusions or Comments:** Epileptic disorders and cardiogenic syncope may both manifest with seizure like motor manifestation. Combined EEG/EKG monitoring is important diagnostic tool in the diagnosis of seizures and syncope.

P-2-132

Can pursuit eye movements reflect the efficacy of antiepileptic drugs?

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Background & Objectives: We evaluated whether eye movements could reflect the efficacy of antiepileptic drugs in patients with epilepsy. **Method:** Thirty patients with epilepsy of unknown cause as well as age- and sex-matched normal controls were enrolled in this study. We divided the patients into drug-con-

trolled epilepsy (n=22) and drug-resistant epilepsy (n=8) groups according to their seizure controls. We analyzed the differences in the parameters of the eye movements in these two groups compared with normal controls using video-based electro-oculography. In addition, we investigated the differences in the cerebellar volumes of these two groups using whole-brain T1-weighted MRI images. **Results:** The latency and accuracy of saccade in patients with epilepsy were significantly different from normal controls, but they were not different between patients with drug-controlled epilepsy and drug-resistant epilepsy. However, the gain of pursuit was significantly decreased in patients with drug-resistant epilepsy compared with normal controls (p=0.0010), whereas it was not different between patients with drug-controlled epilepsy and normal controls (p=0.9646). In addition, the patients with drug-resistant epilepsy had lower cerebellar volumes than normal controls (p=0.0052), whereas the cerebellar volumes in patients with drug-controlled epilepsy were not different from normal controls (p=0.5050). **Conclusion:** We demonstrated that pursuit eye movements could reflect the efficacy of antiepileptic drugs in patients with epilepsy, a finding that may be related to cerebellar dysfunction.

P-2-133

Difference in heart rate change between temporal and frontal lobe seizures

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Background & Objectives: Heart rate(HR) change is easily seen in seizures. Tachycardia is frequently seen in temporal lobe epilepsy (TLE) than extra temporal lobe epilepsy (XTLE). We report the difference HR pattern between temporal lobe epilepsy(TLE) and frontal lobe epilepsy(FLE). **Method:** The ECG data collected during EEG monitoring was used. To compare the HR pattern between FLE and TLE, we investigated the baseline HR, maximum HR, seizure onset to peak, HR change, and time to return to baseline. **Results:** Total 12 patients were enrolled, 6 patients were men and 6 were women, aged 15 to 66 years. 4 patients were FLE and another 8 were TLE. Total 147 seizures were analyzed, 70 were TLE and 77 were FLE. 67 seizures were right oriented, 18 were left, 61 were bilateral and 1 was undetermined. Figure 1 shows the typical examples of TLE and FLE. The change of HR is larger and longer in TLE than FLE. Table 1 shows the difference between FLE and TLE. The mean duration of seizures in TLE were 98.39 seconds, in FLE were 34.11 seconds, and the difference was 64.28 seconds (p < 0.0001). Baseline HR was similar within two groups, as 74.71 bpm. The peak HR was 136.92 seconds in TLE, 122.93 seconds in FLE, and the difference was 13.99 seconds (p < 0.0001). The HR change was 62.06 bpm in TLE, 48.37 bpm in FLE, and the difference was 13.69 seconds (p < 0.0001). The mean duration of seizure onset to peak was 49.13 seconds in TLE, 18.39 seconds in FLE, and the difference was 30.74 (p < 0.0001). Figure 2 shows the difference of return to baseline between TLE and FLE. The numbers of seizures that return to baseline during ictal period were 1 in TLE (1.3 %), 3 in FLE (4.3 %). Before 60 seconds were 15 in TLE (19.5 %), 40 in FLE (57.1 %). Between 60 seconds and 120 seconds were 5 in TLE (6.5 %), 11 in FLE (15.7 %). Over 120 seconds were 56 in TLE (72.7 %), 16 in FLE (22.9 %). In TLE, many seizures were returned to baseline HR when longer than 120 seconds but in FLE, were before 60 seconds. The p value was < 0.0001. **Conclusion:** The HR change of TLE was bigger and longer than that of FLE. The HR change can be the points that differentiate from TLE to FLE.

P-2-134

Epileptic nystagmus in a patient with occipital lobe epilepsy: a case report

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Background & Significance: Epileptic nystagmus is defined as a quick, repetitive jerky movement of the eyeball associated with seizure activity. It usually represents as episodic vertigo, visual hallucination, and cortical blindness. Because an epileptic nystagmus is a rare phenomenon, it may be difficult to diagnose with epileptic nystagmus when a patient complains recurrent episodic vertigo. We have experienced a case of epileptic nystagmus that was diagnosed with occipital lobe epilepsy. **Case:** A 47 years-old woman was admitted with a recurrent rotatory vertigo. Symptom usually lasts about a minute and occurs about 20 times a day. During interictal phase, there was no spontaneous nystagmus and she was normal on neurological examination including head shaking test, head impulse test, Dix-hallpike test and head turning test. However, during the attacks, she showed leftward deviation of both eyes followed by horizontal left-beating nystagmus without any convulsive movement and abnormal posture. The slow component of nystagmus did not cross the midline. She was able to stay alert, and she felt rotatory vertigo with nausea, seeing colored dots on her left visual field. Brain MRI showed lissencephaly of right cerebral hemisphere. And a hyperintensity in the right parieto-occipital cortex and left pulvinar on T2-weighted image, with contrast enhancement, which was considered a seizure-related change (figure 1). To confirm epileptic nystagmus, the patient had continuous video electroencephalography (EEG) monitoring. During interictal phase, EEG showed continuous theta slowing and low amplitude on right cerebral hemisphere. Ictal EEG showed rhythmic fast activity initially in the right occipital area (O2) with rapidly spreading to both occipital area (figure 2). It was followed by periodic lateralized epileptiform discharges (PLEDs) in right occipital area. The patient was diagnosed with right occipital lobe epilepsy accompanied with epileptic nystagmus. We started fosphenytoin and levetiracetam. With initial treatment, frequency and duration of seizure attack was decreased. After two weeks of treatment, she did not suffer from vertigo as well as epileptic nystagmus. **Conclusions or Comments:** Epileptic nystagmus is a rare phenomenon characterized by rapid, repetitive eye movement caused by epileptic activity. Based on the clinical features, in the case of recurrent unexplained vertigo or with other symptoms such as loss of consciousness, visual loss or visual hallucinations, the epileptic nystagmus should be included in the differential diagnosis. If so, continuous EEG monitoring may be helpful to reveal this phenomenon, and patient's symptoms of vertigo and nystagmus can be totally subsided with anti-epileptic medication.

P-2-135

Somatosensory evoked potential (SEP) induced myoclonic status epilepticus in hypoxic ischemic encephalopathy

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Background & Significance: Somatosensory evoked potential (SEP) study is used for evaluation of prognosis after cardiopulmonary resuscitation (CPR). Bilateral absent of cortical response on SEP study recorded 3 days after CPR predict poor outcome. We report unusual case of re-emerging seizure after SEP study which performed for prediction of prognosis after hypoxic event. **Case:** A 77 year-old man who diagnosed as amyotrophic lateral sclerosis (ALS) had hypoxic event for 6-7 minutes. After resuscitation, and generalized my-

oclonus occurred and persisted for two days. Three days after disappearance of myoclonus, median nerve SEP study was performed and myoclonus re-emerged right after SEP study. Electroencephalogram (EEG) after vecuro-nium injection to remove generalized muscle artifacts revealed frequent bi-hemispheric synchronous spike and waves with maximum amplitude in central area (C3, 4), which disappeared temporarily after lorazepam injection. Levetiracetam was partly helpful and myoclonus disappeared 10 days later. **Conclusions or Comments:** SEP study can provoke myoclonic status epilepticus. In this patient, underlying degeneration of cortical neuron in peri-central areas might play a role in hypersensitivity. We should be cautious about applying SEP study to those who experienced myoclonus after hypoxic event.

P-2-136

Posterior reversible encephalopathy syndrome(PRES) involving deep brain structures as the presentation of eclampsia

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Background & Significance: Posterior reversible encephalopathy syndrome (PRES) has been described in a number of medical conditions, such as severe hypertension, eclampsia etc.. PRES commonly shows posterior regions of the brain on brain MRI, and lesions of deep brain structure such as brain stem are rare but reported. Generally, the symptoms and brain MRI finding resolve by treatment of causing diseases. **Case:** In the 31st+4 week of gestation, a 39-year-old woman presented at the emergency department with altered mentality and seizure. The patient had no history of other diseases except mild hypertension with no medication, her previous pregnancy and delivery were normal, and she took no regular medication. At emergency department, she showed stuporous mentality and generalized tonic-clonic seizure during 30 seconds. Initial blood pressure was 220/122 mmHg. Serum platelet was 72,000/mm³, BUN/Cr were 26.6/1.5 mg/dl, AST/ALT were 731/419 IU/L. And there was proteinuria 4+ by dipstick test. Brain MRI showed hyperintense lesions in the bilateral thalamus, midbrain, pons, cerebellum, parietal and occipital cortex and subcortex with posterior dominant on FLAIR and DWI.(Figure 1) Emergency Cesarean section was operated immediately. And blood pressure control, magnesium and levetiracetam intravenous injection were performed. At 2 days after Cesarean section, the patient's mentality was recovered to alert state. And the performed EEG was unremarkable. The patient showed improved symptom by blood pressure control and conservative care, and she was discharged at hospital day #8. After the discharge, she had no neurologic signs and other symptoms. Follow up brain MRI at 19th day after discharge showed no lesion.(Figure 2) **Conclusions or Comments:** In our case, the symptoms and brain lesions completely improved by delivery and blood pressure control. PRES involving deep brain structures as the presentation of eclampsia is very rare, but its prognosis is not bad compared with the typical PRES. As like this case, although deep brain structures are involved, the prognosis are good if immediate delivery and proper blood pressure control are performed. Further study to difference PRES involving deep brain structures from typical PRES should be required.

P-2-137

Human herpesvirus-6 encephalitis presenting non-convulsive status epilepticus

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Background & Significance: Viral etiologies constitute majority of the cases with encephalitis including herpes simplex virus. A major challenge in treating patients with symptoms of encephalitis is the prompt identification of the specific disease-inducing agents. Like most human herpes viruses, human herpesvirus-6 (HHV-6) is ubiquitous and capable of latent infection of its host. Primary infection with the virus is associated with roseola infantum (exanthem subitum) and, most commonly, an undifferentiated febrile illness within the first 2 years of life. Viral reactivation has been linked to a variety of diseases, including encephalitis. Herein we report a case with HHV-6 encephalitis presenting non-convulsive status epilepticus mimicking herpes simplex encephalitis. **Case:** A 75-year-old woman with acute renal failure and sepsis had suffered from delirium during the initial hospitalization period. She was fully restored to awareness and mood after several days. Continuously she had been treated with antibiotics (vancomycin, piperacillin/tazobactam and trimethoprim/sulfamethoxazol) because of persistent MRSA bacteremia and fever. At the hospital day 14, the patient developed confusion and then no verbal response with alert mental status in one day. She showed chewing and right hand fumbling movements intermittently, considering automotor seizure. Brain MRI including diffusion image showed no definite parenchymal lesion but cerebrospinal fluid (CSF) analysis showed pleocytosis and elevation of protein (WBC 99/mm², RBC 0/mm², protein 120.6 mg/dl, glucose 64 mg/dl, serum glucose 265 mg/dl). The EEG showed ictal discharges and PLED from left temporoparietal area. Non-convulsive status epilepticus in encephalitis was considered. Seizures were treated with phenytoin, valproate and lorazepam intravenously. Empirical acyclovir was started and the antibiotics were changed (vancomycin and bactrim were stopped and meropenem was added). CSF and blood bacterial culture were negative. Polymerase chain reactions (PCR) for HSV -1 and -2, varicella zoster virus, cytomegalovirus, Epstein Barr virus, enterovirus and M. tuberculosis were negative in CSF. CSF HHV-6 DNA PCR was positive. The patient was treated in the intensive care unit with ventilator support, anticonvulsants, intravenous ganciclovir (10mg/Kg per day). Follow-up brain MRI showed gyral swelling with hyper-intensities in right medial temporal area, left entire temporal lobe, left posteromedial thalamus and both lower frontal area on T2WI and FLAIR. Repeat CSF HHV-6 PCR at 6+ days was also positive. During acyclovir therapy her seizure activity was not controlled well. After the change to ganciclovir, it had taken a favorable turn. The clinical status and her EEG were improved. Intravenous ganciclovir was discontinued after a total treatment of 2 weeks because of thrombocytopenia and acute massive gastric bleeding. Fortunately she had made a slow recovery with neurologic and general medical conditions. Follow-up CSF analysis revealed absence of WBC and mildly increased protein level and CSF HHV-6 PCR was negative a month after the onset of encephalitis. **Conclusions or Comments:** HHV-6 encephalitis was considerably similar to herpes simplex viral encephalitis in terms of clinical, brain MRI and EEG findings. Awareness of HHV-6 infection may help with early diagnosis, prompt treatment and improved outcome.

P-2-138

Creutzfeldt-Jakob disease presenting as frontal lobe epilepsy with migraine

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Background & Significance: Creutzfeldt-Jakob disease (CJD) is the prototype of a family of rare and fatal human degenerative conditions characterized by progressive brain dysfunction. People with CJD are unable to care for themselves within 6 months or less after symptoms begin. The first symptoms of

most CJD are rapidly progressive dementia, personality changes, hallucination, ataxia, myoclonic seizure. Early diagnosis of CJD is important because that able to explain patient's poor prognosis to prepare for resting life expectancy and prompt conservative treatment alleviate patient's discomfort. **Case:** A 62 year old men with a long history of alcoholism was admitted to the hospital due to migraine with aura. He complained intermittent cognitive dysfunction and tingling sensation of right forearm. He treated for hypertension 10 years and had one episode of loss of consciousness. There was no abnormalities in physical and neurological examination. Routine laboratory examinations were normal. Brain magnetic resonance imaging showed normal. In electroencephalogram for exclusion of seizure intermittent spike and slow wave complex on right frontal area was shown. As a result, suspected diagnosis of migraine and right frontal lobe epilepsy was made and conservative treatment including antiepileptic drug was followed. After the 30th day after discharge patient presented myoclonus and cognitive function impairment and became apathic and akinetic. Later 14-3-3 protein was detected in the CSF study and characteristic generalized periodic sharp wave pattern was observed in follow up electroencephalogram. We finally diagnosed the patient as Creutzfeldt-Jakob disease. **Conclusions or Comments:** We report a rare case of Creutzfeldt-Jakob disease presenting as frontal lobe epilepsy with migraine.

P-2-139

Convulsive movement as an initial manifestation of sporadic Creutzfeldt-Jakob disease

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Background & Significance: Creutzfeldt-Jakob disease (CJD), or subacute spongiform encephalopathy, is one of the prion diseases and may be sporadic, familial or iatrogenic. The common presentations include rapidly progressive cognitive decline, behavioral changes, cerebellar dysfunction and visual disturbances. It affects the brain diffusely, although predominantly unilateral presentation has been described. Myoclonic jerks are typically seen at more advanced stages of the disease. Focal motor or generalized seizures have been reported to occur in only 10-15% of patients. Seizures as a presenting symptom in patients with CJD were reported only occasionally and rare. We describe 3 patients who were diagnosed as having CJD following hospitalization for convulsive movements. **Case: Case 1:** A 73-year-old woman developed tonic-clonic seizure of right upper and lower extremities that is 1st attack in her life. She had progressive memory impairment, myoclonic jerks, visual disturbance, visual hallucination and dizziness lately. In MRI, mildly increased signal intensity was shown in both parieto-occipital area on diffusion images. EEG finding was periodic lateralizing epileptiform discharges (PLEDs) over the Lt. hemisphere. In CSF study, 14-3-3 protein was detected. So she was diagnosed sporadic CJD after expired 1 month later from admission. **Case 2:** A 70-year-old man was admitted because of simple partial motor (left arm) and secondarily generalized tonic clonic seizures. Four 4 days earlier, his family noticed that he had fever, headache and myoclonic jerks. Due to without regaining full consciousness and EEG monitoring showed occipital periodic sharp wave complexes (PSWCs), he was treated with a loading dosage of fosphenytoin 30 mg per kg followed by maintaining fosphenytoin and levetiracetam. Brain MRI showed only revealed hydrocephalus & non-specific white matter high signal intensity in T2WI and diffusion images. 14-3-3 proteins was detected in CSF study. He died 1 week after admission. **Case 3:** A 61-year old woman with generalized tonic-clonic seizure and Todd paralysis (right side) was admitted. She had been diagnosed schizophrenia 30 years ago and was bed ridden patient. Brain MRI showed high signal intensity in Lt. thalamus and Lt. P-T-O high signal on flair in F/U study (HD#2). The CSF was colorless but WBC counts were 10 per cu-

bic millimeter (mm3) and protein 14-3-3 was positive. Only generalized intermittent slow wave was seen in EEG. After using aciclovir, valproic acid and clonazepam, visible convulsive movement was not seen but, full consciousness was not regained either. **Conclusions or Comments:** Myoclonic jerks are one of the most common symptoms in patients with CJD. While focal and generalized seizures may occur in the course of CJD, they are not frequent events as initial symptom, and have been reported to occur in only 10-15% of patients. Our patients was hospitalized following the development of partial or generalized seizure. They all had no history of previous seizures. We propose that CJD should be added to the list as one of the rare but possible causes of generalized or partial seizure with recent progressive cognitive decline or altered consciousness.

P-2-140

Neurosyphilis presenting with non-convulsive status epilepticus

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Background & Significance: Neurosyphilis is an infectious disease of the central nervous system caused by spirochete *Treponema pallidum*. Classic clinical presentations of disease are including symptomatic meningitis, meningovascular syphilis, general paresis, and tabes dorsalis but atypical manifestation of neurosyphilis have always existed. As atypical manifestations, several case reports described progressive cognitive dysfunction and seizures just like herpes encephalitis. Seizure is usually accompanied with neurosyphilis (14~60%) but most of cases are associated with Jarisch-Herxheimer Reaction and initial presentation of status epilepticus is extremely rare. Here we present a patient of neurosyphilis whose initial symptom was NCSE. **Case:** A previously healthy 29-year-old man presented with sudden onset global aphasia and altered mentality. MRI revealed DWI high signal intensity in the cortex of left temporoparietal lobe with normal MRA and mild T2 high signal intensity at the same area. 24 hours video EEG monitoring recorded while the patient was unresponsive and without visible spontaneous movements revealed four seizures with onset over the left occipito-temporal area (O1>T5) and postictally PLEDs were noted over the left O-T areas. Serum Venereal Disease Research Laboratory Test (VDRL) and *Treponemal* hemagglutination (TPHA) were reactive and CSF VDRL was reactive with 1:32 titer. At ophthalmologic exam, Argyll Robertson pupils were described. Intravenous penicillin (4 million U every 4 hours) was started for treatment of symptomatic neurosyphilis and 300mg bid of carbamazepine and 500mg bid of levetiracetam for controlling seizure. After treatment, his mental state became alert and his language dysfunction have been improved. Follow up EEG still showed intermittent spikes on left temporoparietal area (O1>T5) but no more ictal pattern was documented **Conclusions or Comments:** The incidence of Syphilis had been decreased after effective use of penicillin. But recently in last decade, incidence has risen as a result of HIV epidemic and became more common in immunosuppressant patient. And neurosyphilis is a well treatable disease with penicillin but if not treated, there will remain some severe neurologic deficits like dementia, recurrent seizures, paresis, blindness, etc. So early diagnosis and quick administration of antibiotics is really important in the prognosis of neurosyphilis patient. Although a lot of cases of seizure in neurosyphilis are related to Jarisch-Herxheimer Reaction which is systemic immunologic reaction to endotoxin-like products released by when microorganisms killed to antibiotics, seizure and status epilepticus can be the initial presentation of the disease. Therefore, When evaluating sudden first onset NCSE patient, neurosyphilis must be considered as one of the differential diagnosis list.

P-2-141

Aphasic status epilepticus associated with uremia

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Background & Significance: Aphasic status epilepticus (ASE) is a rare disorder characterized by recurrent aphasia without impairment of other cognitive functions. **Case:** A 76-year-old woman with chronic kidney disease developed ASE after neglecting peritoneal dialysis. MRI failed to demonstrate a corresponding lesion. EEG demonstrated ictal discharges in the left frontotemporal leads. ASE was cured after intravenous valproic acid administration. **Conclusions or Comments:** This is the first case report of ASE in a patient with acute aggravation of uremia.

P-2-142

Field testing the criteria for primary stabbing headache according to the third beta edition of the international classification of headache disorders

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Background & Objectives: The diagnostic criteria of primary stabbing headache (PSH) have been recently redefined in the third beta edition of the International Classification of Headache Disorders (ICHD-3 Beta) such that the distribution is not limited to the V1 region but included all the head and neck regions. This study is to investigate the validity of ICHD-3 beta criteria for PSH. We also examined the clinical differences between PSH and secondary headache with stabbing nature and the differences between the first and recurrent stabbing headache among PSH. **Method:** Clinical characteristics of 289 patients were prospectively collected from subjects referred to our hospital for stabbing headache during the previous 5 years. With neurological examination and constructional headache questionnaire, we investigated the onset, month, location, pain characteristics, mean visual analogue scale (VAS), time to recovery and presence of referred pain, preceding infection, stressful condition and allodynia. A database of 236 patients with PSH and 9 patients with secondary headache with stabbing nature were collected. We used IBM SPSS 21 for the statistical analysis. **Results:** Out of 245 patients who followed up two weeks after initial visit, 7 patients developed zoster infection at the respective dermatome, and 2 patients suffered from Bell's palsy. The remaining 236 patients fulfilled the diagnostic criteria for PSH according to the ICHD-3 beta, while only 22 patients satisfied that of the ICHD-2. There were no significant clinical differences between patients with PSH according to ICHD-2 and 3 beta without out V1 dermatome except that frequency of stressful condition prior to the event was more than 3 times higher in V1 exclusive group. Though subjects with secondary headache with stabbing nature lacked in numbers, there were several clinical differences between PSH and this group. Age of onset in male, Mean VAS, frequency of stressful condition prior to event, and frequency of involvement of V2 dermatome were significantly low in PSH group. **Conclusion:** All the patients with PSH according to ICHD-2 fulfilled the criteria of ICHD-3 Beta obviously. ICHD-3 Beta criteria for PSH was far more sensitive than ICHD-2; only 9% of patients with PSH according to ICHD-3 Beta fulfilled ICHD-2. Clinically there were no notable statistically significant differences between the dermatome distributions or recurrence.

P-2-143**Headache as an aura of focal seizures; video-EEG monitoring study**Jae Hyun JIN¹, Dong Wook KIM¹, Sang Kun LEE²¹Department of Neurology, Konkuk university hospital, ²Department of Neurology, Seoul National University hospital

Background & Objectives: Headache, and especially migraine, is often linked to epilepsy. Headache can be associated with epilepsy as preictal, ictal, or postictal phenomenon, but there are only limited reports of headache as an epileptic aura followed by other epileptic manifestation with EEG changes. **Method:** Auras described by the patients were classified into 54 categories and analyzed retrospectively. We obtained the detailed features of headache in patients who described headache as an aura. Video-recorded clinical seizures, EEG findings and neuroimaging data were used to determine the ictal onset areas of the patients who had headache as an aura. **Results:** Among the 831 patients included, 55 had generalized seizures and 775 had partial seizures. While 457 patients did not experience an aura, 374 patients reported at least one aura. Five patients described headache as the most frequent aura, and headache was the second aura in one patient. All patients had partial seizures (two frontal lobe seizures, two temporal lobe seizures, and two parietal lobe seizures). The characteristics of headache were migraine-like in two patients, tension-type headache in another two patients, and hemicrania epileptic in the other two patients. **Conclusion:** Our study showed that headache is an uncommon feature of aura in adult epilepsy patients, and it can present as diverse features including tension-type headache, migraine-like headache, and hemicranias epileptica. Additional studies would be needed to localizing value of various types of headache in adult epilepsy patients.

P-2-144**Cluster headache associated with intracranial carotid artery dissection**

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Background & Significance: Cluster headache (CH) has the characteristics of unilateral pain with ipsilateral cranial autonomic features lasting 15 to 180 minutes. It would be easier if the diagnosis is based on the criteria of ICHD. Although most CH is primary, we should not overlook the fact that it can have secondary causes such as tumors, infections and vascular abnormalities. We present a patient with CH associated with internal carotid artery dissection (ICAD). **Case:** A previously healthy 57-year-old man had suffered the sudden onset squeezing headache around left front-temporal and periorbital area. Initially he took NSAIDs and OTC to abolish pain, but there was no improvement over 30 days. Pain attacks lasted 30~180 minutes and occurred 2 or 3 times a day. Examination during headache revealed left ptosis and miosis, accompanied by ipsilateral conjunctival injection and lacrimation. These features satisfied criteria "A to D" for CH of the ICHD, so we prescribed sumatriptan 100mg, verapamil 80mg, topiramate 100mg and prednisolone 60mg a day for 7 days. But these medication was not effective. We performed brain MRI to rule out other cause of headache and brain MR angiography revealed dissection at left internal carotid artery (C1) with intimal flap. He admitted for TFCA and anti-coagulation. After treatment, headache had been improved markedly. Follow-up CT angiography revealed interval improvement 2 months later. **Conclusions or Comments:** This case suggests that ICAD should be considered for differential diagnosis of secondary cluster headache, even though the typical characteristic neuralgic pain is presented. The underlying pathophysiology of CH is incompletely understood. Autonomic dysfunction in carotid artery by dissection may play a role in the pathogenesis.

P-2-145**Management of diffuse idiopathic skeletal hyperostosis presenting with neck pain**

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Background & Significance: Diffuse Idiopathic Skeletal Hyperostosis (DISH) is characterized by calcification and ossification of soft tissues, mainly ligaments and entheses. This is termed senile ankylosing hyperostosis. **Case:** Eight years ago, 72-year-old man had been admitted to our out patient hospital due to right posterior nuchal discomfort with cramping nature. Neurologic examinations were normal. He had no limitation in motion except pain so that he was prescribed some medicine. However, the symptoms were getting worse. Later, Cervical spine X-ray revealed continuous marginal osteophytosis in C2 to C7 which was enough to diagnose the Diffuse Idiopathic Skeletal Hyperostosis (DISH). We had tried to manage the symptoms with various medications such as analgesics, muscle relaxants, NSAIDs, and physiotherapy for five years, but symptoms had been persistent. Since he had took lesser occipital nerve block, headache was partially relieved. But However, 3 months after the procedure, symptoms were aggravated, and eventually he complained about motional limitation in extension and flexion at 30 degrees. Now he is taking cetamadol 325/37.5mg, joins, lycrica 75mg, and sensival 5mg per twice a day. Headache somewhat has been improved after taking these medications. **Conclusions or Comments:** DISH can attribute to complication such as dysphagia, spinal fractures, spinal stenosis, difficult intubation, difficult gastroscopy, aspiration pneumonia, myelopathy, et cetera. Despite improvement in our understanding about DISH, specific therapeutic interventions are not yet available.

P-2-146**Reversible cerebral vasoconstriction syndrome with vasogenic edema after blood transfusion**

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Background & Significance: Reversible cerebral vasoconstriction syndrome (RCVS) is characterized by thunderclap headache with reversible vasoconstriction in cerebral angiography. It is more common in women than in men. RCVS has many potential etiology - hypertension, eclampsia, autoimmune disease and several medications, such as immunosuppressants, anti-depressant, oral contraceptive. However, the cause and effect relationship between blood transfusion and RCVS is not well known. In here, we report a RCVS case which is triggered by blood transfusion. **Case:** A 46-year-old female visited emergency center because of severe headache and loss of consciousness. She presented with thunder-clap headache which was abruptly onset, spontaneous resolution, and repetition several times for a week. Moreover, she showed tonic posturing in her right arm with loss of consciousness. These events also repeated 3 times at the visited day. In past medical history, she was diagnosed with endometriosis, and otherwise healthy. She took medroxyprogesterone for 9 days 1 month ago. And before 10 days, she received blood transfusion (packed red blood cells, 4 packs) because of menorrhagia with severe anemia. At that time, she took oral contraceptive agent, which was composed of ethinyl estradiol and drospirenon complex. Her initial vital signs, neurologic examination, and laboratory test including cerebral fluid analysis were normal. There were hyper-intense lesions on fluid-attenuated inversion recovery image (FLAIR) and T2-weighted image in left occipital lobe and basal ganglia. Also, there were diffuse leptomeningeal enhancement in T1 weighted gadolinium enhanced image (T1Gd), diffuse intracranial vessel stenosis in MR angiography (MRA). Based on clinical manifestations and image findings, she was di-

agnosed with RCVS and took calcium channel blocker. Her symptoms showed improvement after taking medication. Follow up Brain MRI was done after 2 weeks. It showed that hyper-intense lesions on FLAIR and leptomeningeal enhancement on T1Gd were disappeared. However, multifocal stenosis of intracranial vessels on MRA was no significant change. Some improvement in right middle cerebral artery (MCA) and newly appeared stenosis in left MCA were observed on MRA. Trans femoral cerebral angiography was also done, and it showed that improvement but residual multi-focal intracranial vessels stenosis. **Conclusions or Comments:** Although medroxyprogesterone, which effects on the vacular endothelium, is well known the cause of RCVS, the oral pills, ethinyl estradiol and drospirenon, in this case dose not seem to cause the headache. In anemic patients, there were a few reports about the effect of blood transfusion in vascular tone. Blood transfusion in chronic anemic patients could alteration of vascular tone, and finally results in overwhelming cerebral vascular constriction. In conclusion, blood transfusion is one of the possible etiology in RCVS.

P-2-147

Acromegaly caused by pituitary adenoma in patient with normal hormone level

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Background & Significance: Pituitary adenomas are common tumors in sellar, it represent approximately 17% of all intracranial tumors. Pituitary adenomas are generally classified according to the size of mass and their biological functioning. Pituitary adenomas which exceed 10 mm as considered macroadenomas and the smaller one than 10mm considered as microadenomas. About secreting pituitary adenomas, the functional classification is based on the endocrine activity of the tumors (e.g., prolactin, growth hormone, adrenocorticotrophic hormone, gonadotropic hormone or thyroid stimulating hormone). We report a case of acromegaly caused by pituitary adenoma which have normal hormone level. **Case:** 37-year old man was admitted with severe headache after heavy drinking since 2 weeks ago. He had a history of chronic headache and recurrent spontaneous pneumothorax. His usual headache characteristics was dull nature, mild intensity without nausea or vomiting and usually response to acetaminophen. The day before the admission, headache was suddenly aggravated. There was no preceding trauma or any infection sign. Of note, his facial features were coarse and hands and feet were enlarged and eye brows were pronounced on physical examination. We checked CSF study, brain MR imaging and routine laboratory test. CSF analysis revealed acellular and normal glucose, protein level and normal opening pressure. After he took naproxen, headache was disappeared, but MRI with enhanced showed mass on sellar area. Sellar MR imaging showed hemorrhagic pituitary adenoma. The results of pituitary hormone level was normal range. **Conclusions or Comments:** The clinical presentation of pituitary adenomas depending on their endocrine dysfunction. In this case, the clinical symptoms of pituitary adenoma might be happened without significant endocrine dysfunction.

P-2-148

Intracranial hypertension after massive blood transfusion

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Background & Significance: Idiopathic intracranial hypertension is an uncommon syndrome presenting with chronic headache, papilledema, diplopia,

tinnitus and other neurological symptoms. However, the pathophysiology of the syndrome is unknown, although many hypotheses have been proposed, including hormonal change, cerebrospinal fluid (CSF) dynamics, and cerebral venous blood pressure change. We experienced a patient with idiopathic intracranial hypertension after rapid massive blood transfusion, who had chronic anemia due to multiple uterine myomas. **Case:** A 38-year-old woman presented to the emergency department with uncontrolled vaginal bleeding for 2days. She had history of uterine myoma operation in 2007, otherwise there was no relevant medical or family history. The body mass index was 20.78. Initial vital signs were stable. She had mild dizziness and anemic conjunctiva, however her mental status was alert and the neurologic exam showed no focal deficits. Anemia with uterine myoma was considered as a possible cause. The hemoglobin level was 2.4g/dl, mean cell volume (MCV) 51.5fL, mean cell hemoglobin (MCH) 14.3pg, mean cell hemoglobin concentration (MCHC) 27.7g/dL, Fe <10ug/dL, total iron binding capacity (TIBC) 468ug/dL, ferritin 2.4ng/mL and laboratory findings were compatible with iron deficiency anemia. She had 7 pints of red blood cell transfusion for 2days and follow up hemoglobin level was 10.8g/dl. Three days after last transfusion, she started to suffer severe pulsatile headache from occipital area radiating to frontal area with visual analog scale score of 10. She also had nausea and excessive daytime somnolence. The CSF study through lumbar puncture was done, the pressure was 25.0 cmH₂O and the other profiles were within normal range. The magnetic resonance imaging with angiography and venography showed suspicious leptomeningeal enhancement without any vessel abnormalities. She had multiple retinal hemorrhages on both eyes but no papilledema on the ophthalmic exam. In this case, the papilledema was absent and the CSF pressure was not high enough to diagnose idiopathic intracranial hypertension, however, suddenly developed severe headache and multiple retinal hemorrhages on both eyes suggested idiopathic intracranial hypertension. She was treated with 20% intravenous dextrose mannitol and oral acetazolamide. The headache was improved gradually for next 7 days and no other complications occurred. The multiple uterine myomas were confirmed on sonography and myomectomy was done 1month later. Her hemoglobin level was stable and the headache has resolved completely. **Conclusions or Comments:** The chronic iron deficiency anemia is well known as risk factor of idiopathic intracranial hypertension with young, obese women. In this case, the severe chronic anemia was present before headache and the onset of headache was the time after massive rapid blood transfusion was done. We made a hypothesis that the sudden blood transfusion in chronic anemia patient might damage cerebral vascular endothelium, releasing free radicals, resulting in vasogenic cerebral edema. Finally, the massive rapid blood transfusion might be the cause of idiopathic intracranial hypertension. Therefore, it is important to investigate carefully when the chronic anemia patient have sudden headache after blood transfusion.

P-2-149

A case of overdose chemotherapy-associated intracranial hypotension mimicking dural metastasis in breast cancer

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Background & Significance: Intracranial hypotension occurs spontaneously or secondary to iatrogenic manipulation including a lumbar puncture or spinal surgery. It is characterized by an orthostatic headache to upright posture and its relief within moments after assuming the recumbent position. The diagnostic radiologic finding is prominent dural enhancement in gadolinium-enhanced brain MRI. In a patient with metastatic cancer, this finding may be misinterpreted as a dural metastasis. Therefore, the accurate differential diagnosis

is especially important in patient with malignancy. Although intracranial hypotension is a disease entity of diverse origin, there have been few reports associated with chemotherapy. We report a case of intracranial hypotension followed by overdose chemotherapy. **Case:** In 2013, a 47-year-old Russian woman with right breast cancer received radiotherapy and tamoxifen treatment following a partial mastectomy with lymph node dissection in our hospital. In February 2014, hepatic metastatic tumors were detected. During 2 months, she received 4 cycles of monoxol 75 mg/m² and herceptin and then went back to Russia. In her country, she received intravenous taxotere 175 mg/m² for about 3 months. She revisited our hospital due to disabling headache, nausea, vomiting and blurred vision. Her headache and nausea markedly worsened by upright posture and was relieved by the recumbent position. She denied trauma history. Gadolinium-enhanced brain MRI showed diffuse thin dural enhancement with even thickness and distended transverse sinus (Fig 1). For differential diagnosis, a lumbar puncture was attempted several times, but cerebrospinal fluid (CSF) pressure was too low to be measured. With a clinical impression of intracranial hypotension, she was instructed to absolute bed rest and undergone medical treatment with a plentiful supply of fluid and oral caffeine-containing medicine. Her symptoms slowly subsided over the next few days. **Conclusions or Comments:** Taxotere is classified as an antimicrotubule agent, suppressing microtubule dynamic assembly within the cells. This patient suffered from disabling postural headache followed by overdose intravenous taxotere therapy. This case suggests that overdose taxotere might affect CSF production.

P-2-150

Secondary stabbing headache caused by skull metastasis: a case report

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Background & Significance: Primary stabbing headache is characterized by transient and localized stabs of pain in the head. Secondary causes have been infrequently reported. **Case:** A 77-year-old woman with stage IIIb cervical cancer visited our headache clinic complaining of stabbing headache restricted to left parietal area. The patient did not present any neurological deficit or signs of increased intracranial pressure or meningeal irritation. Initial diagnosis was compatible with primary stabbing headache. The headache worsened gradually despite medical treatment. Brain MRI showed multiple enhancing bony lesions including left temporoparietal bone, suggesting skull metastases from cervical cancer. **Conclusions or Comments:** Localized stabbing headache can be a manifestation of skull metastasis. Clinical suspicion and neuroimaging with contrast enhancement can be helpful for the early diagnosis of secondary causes, especially in patients with alleged cancer.

P-2-151

Headache caused by chronic carbon monoxide exposure

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Background & Significance: Carbon monoxide (CO) intoxication is the most common type of gas poisoning. Headache is one of the common intoxication symptoms. However, diagnosis of headache caused by chronic carbon monoxide exposure is difficult because the clinical features are nonspecific and ambiguous. We report a case of a chronic carbon monoxide induced headache. **Case:** A 40-year-old woman visited hospital because of headache that began one month ago. She had no headache like that before. Her family history and past history were not specific. She was a hairdresser. Her headache started at bilateral occipital area, neck, and shoulders, which developed throbbing pain

around both eyes. The headache was moderate in intensity, and accompanied by nausea and vomiting. She denied the presence of auras, photophobia and phonophobia. It was not aggravated by physical activity. The headache got worse at the workplace, and improved when she came home. Neurological examination did not show any focal neurological deficit. The vital sign including blood pressure, pulse rate, and body temperature was normal. There were no specific findings in blood test, urinalysis, brain magnetic resonance imaging, and magnetic resonance angiography. Based on the medical history and test results, she was diagnosed with a probable chronic tension type headache. She took painkillers and muscle relaxants, but they did not work. After 10 days, the patient was transferred to our emergency medical center for loss of consciousness with her colleague working together in the hairshop. At the time of admission, their consciousness became normal. She had no symptoms except mild headache. Her vital sign, physical examination and neurological examination was normal. No significant abnormalities were noted on her complete blood count (CBC), blood chemistry analyses, arterial blood gas analysis, except for the high levels of carboxyhemoglobin in patient (23%) and her colleague (27%). She was diagnosed with CO poisoning, and was immediately given 100% oxygen using a nasal prong device. The next day, all symptoms were improved and level of carboxyhemoglobin returned to normal range. After discharge, patients were checked gas facilities of her shop, and found gas leak at the water heater system. She had no symptoms after fixing the water heating system. **Conclusions or Comments:** Our case suggested that it is important to ask the questions about living environment, heating systems, occupation, and similar symptoms among coworkers and family members in history-taking of a sub-acute or chronic headache patient to make a differential diagnosis of headache caused by chronic carbon monoxide exposure.

P-2-152

Cerebral paragonimiasis presenting as chronic headache

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Background & Significance: *Paragonimus westermani* (*P. westermani*) is a common human parasite in the Far East, especially prevalent in Korea, China, Japan, and Taiwan. Most patients with *P. westermani* infection are asymptomatic. However, some patients can have pulmonary and extrapulmonary manifestation. Cerebral paragonimiasis is rare (less than 1% of symptomatic patients), but can cause several serious complications; seizure, motor or sensory disturbance, mental retardation and blindness. Paragonimiasis is a fluke infection transmitted via consumption of raw or undercooked crab or crayfish. Sometimes, *P. westermani* migrate from pleural cavity to brain through soft tissues around vena jugularis interna and lateral sinus of the brain. By this migrating route, one cerebral hemisphere, especially the occipital lobe, was affected frequently in cerebral paragonimiasis. **Case:** A 77-year-old woman of age presented with complaints of daily headache for three years. Her medical history was unremarkable without trauma history. The patient was alert and oriented. Initial neurological examination showed right homonymous hemianopsia. Computerized tomography (CT) scans and magnetic resonance images of the brain revealed multiple densely calcified regions with widespread inflammatory changes in the surrounding tissue in the left temporal and occipital areas (Figure 1). Chest CT scans showed linear opacities and bronchiectasis in right upper lobe, right lower lobe, and focal tiny centrilobular nodules in left upper lobe (Figure 2). In enzyme-linked immunosorbent assays for cerebrospinal fluid, IgG antibody for *P. westermani* was detected. The patient was born and has been lived along the riverside area near the Sumjin river in Hadong-gun, Gyeongsangnam-do. Especially the

Sumjin river basins are well known endemic areas of *P. westermani*. She used to eat freshwater crabs, were caught in the stream nearby the village. And she sometimes drank ditch water around a paddy field. We diagnosed her with cerebral paragonimiasis and initiated praziquantel 1000mg for three days. She refused the biopsy for confirm the diagnosis. After 2 months later, her visual disturbances remained, but chronic headache was disappeared. **Conclusions or Comments:** We report a chronic headache patient with cerebral paragonimiasis, who was diagnosed by history, neurologic examination, radiological and serological evaluations. Cerebral paragonimiasis could be considered as a cause of chronic daily headache, especially endemic area.

P-2-153

The prevalence of Parkinson's disease in south Korea: a 11-year nationwide, population-based study

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Background & Objectives: The prevalence and incidence of Parkinson's disease (PD) are important for supporting the better comprehension of disease aspects and helping public health planning. Our aim is to evaluate the prevalence of PD in South Korea between 2002 and 2013. **Method:** This retrospective, nationwide, longitudinal study used National Health Insurance Service National Sample cohort (NHIS-NSC) 2002-2013, which was released by the KNHIS in 2014, comprising 1,025,340 (~2.2%) random subjects who were selected from 46,605,433 Korean residents in 2002. This database was based on having KCD (Korean Classification of Diseases) code G20, which were assigned by neurologists, and being prescribed PD medication. **Results:** The prevalence of PD was 0.03% in 2002 and 0.10% in 2013, and there was a 3.33 fold increased during over the 11 years. In 2013, the percentage of seventh and eighth decade patients was 68.5% and that of the female patients were 60% of the total PD patients. According to the age groups, 40-49, 50-59, 60-69 and ≥ 70 years prevalence was respectively 0.02%, 0.05%, 0.23% and 0.79%. The prevalence ratio of 2013 to 2002 by the age group was higher in 40-49 and ≥ 70 years. That of 30th decade was 2.0 and that of 60th and 70th decade was 2.72. **Conclusion:** The prevalence of PD in 2013 using NHIS-NSC database was 0.10%, so the prevalence of PD increased 3.33 fold over the 11 years and it tends to be more prevalent in female. The increasing rate of PD prevalence was higher in the 30th decade and over than 60th decade group.

P-2-154

Comparison of dysphagia between patients with Parkinson's disease and progressive supranuclear palsy

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Background & Objectives: Dysphagia is a common symptom in patients with parkinsonian syndrome. These patients usually have difficulties of bolus management in the oral phase as well as frequent aspirations in the pharyngeal phase of swallow. The aim of this study was to compare the characteristics of dysphagia between patients with idiopathic Parkinson's disease (IPD) and progressive supranuclear palsy (PSP). **Method:** The data of thirty patients with IPD (17 males and 13 females with a mean age of 72.4 ± 7.8 years) and seventeen patient with PSP (8 males and 9 females with mean age of 68.6 ± 8.1 years) referred to a speech-language pathologist due to dysphagia were analyzed retrospectively. Disease duration, MMSE, rigidity, and bradykinesia scores of each patient were obtained from their medical records. Also, for dysphagia, the oral transit time (OTT) of bolus (thin liquid) was analyzed from the video-fluoroscopic swallowing study (VFSS) and an 8-poin scale was used for as-

sessing the severity of penetration/aspiration. The analysis of the general linear model and the chi-squared test were performed to compare the two groups. The correlations between dysphasia measurements and motor symptoms were analyzed. **Results:** There were no differences in age, gender, and MMSE scores between IPD and PSP groups. The scores of rigidity and bradykinesia were also not significantly different between two groups. However, the disease duration of patients with PSP (3.9 ± 2.9 years) was significantly shorter ($p < .01$) than that of IPD patients (7.9 ± 5.5 years). For dysphagia measurement, there was no difference in OTT between the two groups but penetration/aspiration was more severe ($p < .05$) in patient with IPD. The results of correlation analysis showed a significant correlation between rigidity and bradykinesia score ($r = .447$, $p < .01$). However, there was no correlation between OTT and severity of penetration/aspiration. **Conclusion:** These results indicate that patients with PSP show early progress of motor dysfunction such as rigidity and bradykinesia compare to those with IPD. However, the pharyngeal stage of dysphagia is relatively intact in PSP group although OTT was delayed as similar to IPD group.

P-2-155

Cognitive and motor aspects of Parkinson's disease associated with dysphagia

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Background & Objectives: Dysphagia is a common symptom and an important prognostic factor in Parkinson's disease (PD). Although cognitive and motor dysfunctions may contribute to dysphagia in patients with PD, any specific association between such problems and swallowing functions is unclear. Here, we examined the potential relationship between cognitive/ motor components and swallowing functions in PD. We evaluated the contributions of cognition and motor function to the components of swallowing via video fluoroscopic swallowing (VFS) experiments. **Method:** We prospectively enrolled 56 patients without dementia having PD. PD severity was assessed by the Unified Parkinson's Disease Rating Scale (UPDRS). All participants received neuropsychological tests covering general mental status, visuospatial function, attention, language, learning and memory, and frontal executive function. The well-validated "modified barium swallow impairment profile" scoring system was applied during VFS studies to quantify swallowing impairments. Finally, correlations between neuropsychological or motor functions and impairment in swallowing components were calculated. **Results:** The most significant correlations were found between the frontal/executive or learning/memory domains and the oral phase of swallowing, though a minor component of the pharyngeal phase correlated with frontal function as well. Bradykinesia and the UPDRS total score were associated with both the pharyngeal and oral phases. **Conclusion:** Our findings suggest that cognitive dysfunctions are associated with the oral phase of swallowing in patients with early stage PD while the severity of motor symptoms may be associated with overall swallowing function.

P-2-156

Diabetes and risk of Parkinson's disease: an updated meta-analysis of observational studies

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Background & Objectives: Increasing body of evidence indicated link between diabetes mellitus and Parkinson's disease (PD). Several epidemiologic studies addressed the issue that diabetes is a potential risk factor for PD. Recently, a series of large-scale prospective cohort studies reported the association between PD and diabetes. However, the results of epidemiologic studies are conflicting. The association between diabetes and PD was ranging from a risk factor, no association, or even to a protective factor. Even the meta-analyses of observational studies showed inconsistent results. A meta-analysis of cohort studies reported pooled estimates that diabetes is a risk factor for PD (pooled RR of four prospective studies = 1.37, 95% CI 1.21-1.55; $p < 0.0001$). (Cereda et al, 2011) In contrast, a meta-analysis of case-control studies showed that diabetic patients have lower risk for later development of PD (pooled OR for 14 case-control studies = 0.75; 95% CI 0.58-0.98). (Lu et al.2014) The aim of this updated meta-analysis, which incorporated additional epidemiologic studies, is to synthesize evidence from observational studies that evaluated the association between diabetes and the risk of PD. **Method:** Studies were retrieved from PubMed, Cochrane Library, Embase, secondary references, clinical trials registries and a thesis database. Original contributions written in English published up to 30 June 2015 were included. Prospective cohort and case-control studies providing risk estimates relating to pre-existing diabetes and PD were considered eligible. **Results:** We could find one additional more recent cohort study through literature search. In all, five prospective cohort studies and 14 case-control studies included in this meta-analysis. In prospective studies, the preceding diabetes showed no association with PD in random effect model (relative risk = 1.239; 95% CI 0.887-1.731; $p = 0.209$) with marked heterogeneity (I square statistic = 84.992, $p < 0.001$). The direction of risk unchanged even after removal of newly added cohort study from the analysis in random effect model (RR = 1.343; 95% CI 0.926-1.950, $p = 0.12$). Meta-analysis of case-control studies showed that diabetics have lower incidence of later PD (odds ratio = 0.755; 95% CI 0.583-0.978; $p = 0.033$), also with a marked heterogeneity (I square statistic = 75.201, $p < 0.001$). **Conclusion:** Our updated meta-analysis showed that diabetes has no association with PD. Although several large-scale prospective studies showed a tendency that prior diagnosis of diabetes may be a risk factor for later development of PD, our meta-analysis failed to reach statistical significance. Data from case-control studies suggest that diabetes may have a role as a protective factor for PD. This inconsistency may be in part due to the difference in study design and methods. In spite of recent advances, there is neither unified diagnostic criteria nor screening biological tests for PD, so the diagnosis of PD largely depends on clinical decision of individual physician. The exclusion/inclusion criteria and the method of ascertainment for diabetes vary study to study. Diabetes itself is a major risk factor for vascular disorders. It may be challenging to differentiate vascular parkinsonism from idiopathic PD even for experienced movement disorder specialists. It may be impossible to exclude vascular responsibility completely in epidemiologic studies. In addition, possible role of surveillance bias and resulting false positive association also cannot be excluded completely. Diabetic subjects under medical service may have more chance for diagnosis of PD than general population. Drugs used for treatment of diabetes and PD may play a role as an additional confounder. Further large-scale prospective studies considering a wide variety of confounders are required to elucidate the relationship between diabetes and PD.

P-2-158

Stridor occurred in a patient with SCA17

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Background & Significance: Autosomal dominant spinocerebellar ataxia type 17 (SCA17) is a rare neurologic disorder caused by abnormal CAG/CAA repeat expansions within the TATA-box binding protein (TBP) gene. Various phenotypes have been reported to manifest in SCA17. Even rare, progressive ataxia, autonomic dysfunction were also reported in a patient with mutated SCA17 allele, who was diagnosed as a multiple systemic atrophy. However, there has been no reported case reporting respiratory symptoms in SCA17, while respiratory symptom is not rare in advanced stage of MSA. **Case:** A 48 year-old male visited our hospital with a progressive gait imbalance, which occurred insidiously. Before the onset of gait imbalance, he had suffered from urination difficulty, constipation, and postural dizziness for 3 years. He had a history of hyperthyroidism which was treated 18 years ago, since then he had not experienced recurrence. Among his family members, no one had similar symptoms like him. On neurologic examination, he had a cogwheel pursuit, hypermetric saccade in both direction, but limitation in gaze was not found. His muscle tone and motor power was normal, and tremor was also absent. He did not have a sensory dysfunction. However, he showed a bilateral, symmetric limb ataxia. And he walked with a wide base, bilateral swaying pattern. Romberg's sign was absent. He showed a marked drop in blood pressure without responsive tachycardia. On brain MRI, there was no significant evidence of cerebral cortical atrophy, but mild brainstem and cerebellar atrophy was found. Levodopa was started to relieve gait disturbance, with no significant improvement. Based on his clinical feature and brain image, he was initially diagnosed as multiple system atrophy. However, increased 44 CAG/CAA repeats of TATA box-binding protein (TBP) gene was found in this patient, which suggested positivity for SCA type 17. Genetic tests for spinocerebellar ataxia (SCA) type 1, 2, 3, 6, 7 were all negative. During the 2-year-follow-up period, he started to have a severe snoring when asleep. To assess his severe snoring at night, he underwent polysomnography, and we found severe snoring, sleep apnea, and stridor. During that episode, he also showed transient oxygen desaturation. To protect him from the respiratory failure during sleep, continuous positive airway pressure (CPAP) was applied, and he was discharged to nearby health care facility. **Conclusions or Comments:** Although rare, it has been already reported that increased repeats in TBP gene can be detected in clinical MSA. Features to note is that the patient we report developed severe obstructive apnea and stridor during sleep in the late stage of the disease. This is the first case of SCA17 to be reported with stridor, and clinical consideration should be given about the possibility of respiratory involvement of SCA17, especially when the patient manifest as MSA.

P-2-159

Parkinsonism in spinocerebellar ataxia type 7

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Background & Significance: Spinocerebellar ataxia type 7 (SCA7) is characterized by progressive ataxia and macular degeneration. Parkinsonism has not been reported in SCA7 previously. **Case:** The patient first visited our clinic with a two-year history of a gait disturbance. The neurological examination revealed dysarthria and cerebellar ataxia. The ophthalmological evaluation showed macular dystrophy. Pathological expansion of CAG on the SCA7 gene was found. The patient had not been followed up for 5 years until he revisited our clinic for aggravated movement disorders and behavioral problems. The patient was alert and asthenic on neurological examination. Follow-up neurologic examination showed cognitive impairment, slow saccade, asymmetric bradykinesia, postural instability, limb and gait ataxia. Positron emission tomography (PET) using 18F-fluorinated N-3-fluoropropyl L-2-beta-carboxymethoxy-3-beta-(4-iodophenyl) nortropane (18F-FPCIT) revealed decreased uptake of 18F-FPCIT in the bilateral putamen and caudate nuclei. **Conclusions or**

Comments: Parkinsonism has not been reported in a patient with SCA7 previously. Further studies are needed to extend clinical spectrum of SCA7 suggested by the novel features observed in our patient.

P-2-160

Escitalopram induced extrapyramidal symptoms: A case report

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Background & Significance: In 1979, Meltzer et al. published one of the earliest report of extrapyramidal side effect to serotonin reuptake inhibitors. It was a case of fluoxetine-induced parkinsonism rigidity. Among the SSRIs, fluoxetine has been reported in highest numbers as the casual agent, but recently other SSRI induced parkinsonism is reported quite high. We would like to present a case of escitalopram-induced dystonia to increase awareness for this clinical problem. **Case:** A 60 year old male, who has a history of left MCA infarction in 2005 and 2014, an anxiety disorder, and insomnia was taking alprazolam 0.25mg and escitalopram 10mg for about 20days. He was complaining of sudden onset and quite rapid progressing dysphagia, and walking disturbance. Neurologic exam showed right side dominant tremor, cogwheel rigidity in rt. hand, bradykinesia, stooped position, festination in walking, disturbance of speech and masked facial expression. Because we thought escitalopram was quite safe SSRI to induced parkinsonism, we added perkin(carbidopa 25-levodopa 100) three times a day thinking, it might be genuine parkinson disease or other cause of parkinsonism. And three day later he was suffering with more symptoms as oromandibular dyskinesia and dystonia. There was minimal response to levodopa than we expected. So escitalopram and alprazolam was discontinued and change to clonazepam 0.5mg for insomnia and anxiety, thing of parkinsonism induced by escitalopram. Four days later he was improving in movement. he was able to walk more without festination, and tremor and bradykinesia was markedly improved. OMD and slurred speech was still noticed so we increase dose in levodopa. **Conclusions or Comments:** Although not used in this particular case, diagnostic options such as single photon emission tomography (SPECT) in the work-up of patients with clinically uncertain parkinson syndromes should also be considered. As a sensitive neuroimaging method, SPECT is used for the assessment of nigrostriatal dopaminergic system integrity and degeneration. Results indicate that SPECT can distinguish other neurodegenerative diseases, such as dopa-responsive dystonia, and clarify clinical dilemmas. In conclusion, although evidence pointing to Parkinsonian syndrome induced by escitalopram is rare and relatively recent, this particular SSRI should be included in the list of causal agents in vulnerable patients.

P-2-161

Facial diplegia masqueraded as an aggravating parkinsonism

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Background & Significance: Parkinson's disease (PD) is a degenerative disorder of the central nervous system. As the clinical manifestations of PD are complicated, the clinicians sometimes confuse the cause of new-onset symptoms. We described a patient with Parkinson's disease who had gradual facial diplegia. **Case:** Case 1. A 74-year-old man presented with gradual worsening of dysarthria. He was diagnosed with PD 3 years ago and had stable parkinsonism with levodopa treatment. He noted that, 15 days before admission, he found that he was having difficulty speaking, swallowing, and chewing. He took regular levodopa medication and had no additional drug history. His neurological examination did not show any clinical evidence of ag-

gravating parkinsonian symptoms except dysarthria, dysphagia. However bilateral facial paralysis was remarkable. Nerve conduction study (NCS) and electromyography (EMG) were underwent and revealed sensory motor polyneuropathy in his facial nerves and muscles. A diagnosis of facial Guillain-Barre syndrome variant was made on the basis of the patient's clinical findings and electrophysiologic results. His facial diplegia continued to improve gradually with a return of 80% function of his facial muscle **Conclusions or Comments:** Bilateral facial paralysis is a rare condition and therefore represents a diagnostic challenge. Among patients with prominent facial diplegia, a facial GBS variant should be considered based on the neurological examination and clinical history.

P-2-162

Apraxia of lid opening caused by ropinirole withdrawal

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Background & Significance: Apraxia of lid opening (ALO) is defined as a non-paralytic motor abnormality characterized by difficulty in initiating the act of eyelid elevation. However, the pathogenesis of ALO is not well understood. We report on one patient who suffered from ALO after dopamine agonist withdrawal. **Case:** An 80-year-old woman with a history of diabetes, and Parkinson's disease was referred for evaluation of decreased mentality. She was diagnosed with hypoglycemia and regained alertness following glycemic control. On neurologic examination, she had no parkinsonian feature, although she was on 4mg of ropinirole per day for Parkinson's disease. Thus, we withdrew ropinirole. Two days later, we noticed that she had difficulty in lifting her eyelids despite frontal muscle contraction. There was no evidence of ptosis or abnormal contraction of orbicularis oculi muscles, and parkinsonian features were not found. Diffusion-weighted magnetic resonance imaging was negative. Based on previous literatures reporting levodopa responsive ALO, we put her on levodopa/benserazide 100/25 mg three times a day, which dramatically improved inability to open her eyelids on that day. **Conclusions or Comments:** The reversible eyelid opening difficulty in our patients was consistent with ALO. Our case suggests the importance of considering changes in dopaminergic medications when dealing with ALO and possible role of dopaminergic pathways in the pathogenesis of ALO.

P-2-163

Mirror movements in a patient with pontine infarction

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Background & Significance: Mirror movements are involuntary synchronous movements of a limb associated with the intended movements of the contralateral homologous limb. Mirror movement has been classified as congenital or acquired. Although activation of ipsilateral uncrossed corticospinal pathway or disinhibition of interhemispheric inhibitory mechanism via transcallosal pathway have been suggested, the exact mechanism of mirror movement has to be clarified. Mirror movement secondary to stroke may be a good example for guessing its mechanism. We describe a patient with acute pontine infarction who presented mirror movements on the unaffected hand. **Case:** A 73-year old man admitted to neurology department of our hospital due to dysarthria and weakness of the right leg. One year earlier, he experienced an ischemic stroke in the left pons, for which he had been treated with antiplatelet agent and a statin. He did not any neurological deficit after the first stroke including motor weakness and dysarthria. On initial neurological examination, he showed mild dysarthria and clumsiness on the right arm. The motor power

of his right leg was lessened to grade GIV. In addition, he showed synchronous movement of the left hand corresponding to the intended movement of right hand. Sensory function was normal. Brain MRI with diffusion-weighted image showed acute infarction on the left median pons, near to the previous infarction. Synchronous involuntary movement of the left hand had been spontaneously improved over one year. **Conclusions or Comments:** The synchronous movement of the left hand during intended movement of the right hand in this patient can be defined as mirror movements. The temporal relationship between the onset of the mirror movements and acute stroke event with brain MRI findings suggest the mirror movement might be caused by acute pontine infarction. The mirror movement in this patient can be explained by disrupted balance between the bi-hemispheric reorganization of motor networks, which had occurred during recovery from the previous hemiparetic stroke.

P-2-164

Subthalamic electrode Insertion with deep brain stimulation for impulse control disorder

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Background & Objectives: Subthalamic deep brain stimulation (STN-DBS) has revolutionized the management of advanced PD with drug-induced complications. It is particularly effective for motor fluctuations by improving off Parkinsonism through continuous neuronal stimulation. Impulse control disorder (ICD) is a representative non-motor complication in PD, which is also induced by dopaminergic drugs. We investigated if STN DBS also improves ICD as it does to motor fluctuations in patients with PD. **Method:** From March 2013 to July 2015, 98 patients underwent DBS surgery targeting the subthalamic nucleus (STN), globus pallidus internus (GPi) or posterior subthalamic area (PSA). Among them, seven patients with ICD received bilateral STN DBS. We evaluated these 7 patients using the Unified Parkinson Disease Rating Scale part III (UPDRS III), the Questionnaire for ICD in PD (QUIP) and Addenbrooke's cognitive examination (ACE-R). Neuroimaging studies with MR, [18F]FDG-PET, and [18F]FP-CIT PET were performed. The outcome of STN-DBS was assessed by the patient's global impression (PGI) and clinician's global impression (CGI). **Results:** The mean age of patients with ICD was 48 ± 3.86 years (mean \pm SD), and the mean age at onset of PD was 37.4 ± 2.63 years. The mean interval between the onset of Parkinsonism and the onset of ICD was 6.14 years. The mean of the UPDRS III score before DBS was 56.4 ± 13.15 at med-off, and 22.14 ± 14.8 at med-on. [18F] FDG PET showed glucose hypometabolism in the ventral striatum in patient with ICD. The mean of levodopa-equivalent daily dose (LEDD) was 1,421mg/day before DBS, and 364 mg/day after STN-DBS ($p < 0.01$). The QUIP score was 4.85 ± 1.68 before STN-DBS, and 1.43 ± 1.73 after STN-DBS ($p < 0.01$). PGI score after STN DBS was 2.57 ± 0.79 . **Conclusion:** Our data showed that ICD is more common in young patients with PD, and that STN-DBS reduced LEDD markedly to a quarter of pre-DBS LEDD, and provided evidence that STN-DBS improved ICD. While the pathogenic mechanism of ICD is largely unknown, and seems different from that of motor fluctuations, our observations that STN DBS improves both motor fluctuations and ICD suggest that both motor and non-motor complications may share, in part, the common pathogenic mechanism.

P-2-165

Effect of unilateral subthalamic deep brain stimulation in highly

asymmetric Parkinson disease: 7-year follow-up

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Background & Objectives: Usually bilateral STN DBS is performed in advanced PD to control motor complications such as fluctuation and dyskinesias. However some authors suggested that unilateral STN DBS can be a reasonable treatment option for highly asymmetric PD because unilateral STN DBS may ameliorate the ipsilateral and axial motor symptoms and bilateral surgery inherently involves more surgical complications. We previously reported 2-year follow-up result showing that unilateral surgery was not satisfactory in majority of the patients. The present study is a 7-year follow up of unilateral STN DBS on highly asymmetric patients with PD. **Method:** Eight patients with highly asymmetric PD who underwent unilateral STN DBS were followed up for mean 7 years after the first surgery. The assessment included motor Unified Parkinson Disease Rating Scale (UPDRS), Hoehn-Yahr (HY) stage, levodopa equivalent daily dose (LEDD), Mini- Mental State Examination (MMSE), and Beck Depression Index (BDI) were assessed preoperatively, and postoperative evaluation was carried out at 3, 6, 12 months and yearly after surgery. Serial change of the ipsilateral, axial, and contralateral motor UPDRS was analyzed. **Results:** During the total follow-up period (mean 91.5 months, range 36-105), seven of 8 patients wanted the second-side surgery as the effect of unilateral surgery combined with best medical treatment has gradually become unsatisfactory. Four of 7 patients underwent the second surgery at 58.5 ± 11.6 months after the first surgery. Three patients could not have the second-side surgery due to irrelevant reason to PD. The total UPDRS III score, the mean axial UPDRS score, and LEDD returned to the baseline 3 years after the first unilateral surgery. The improvement in contralateral UPDRS subscore continued through 3 years. The ipsilateral UPDRS subscore worsened gradually and doubled at 3 years. The asymmetry of motor symptoms measured by UPDRS was reversed at 6 months. **Conclusion:** Overall, the benefit from unilateral STN DBS attenuated over time, and 3 years appears to be the maximum. At least for the first few years, the unilateral procedure clearly demonstrated a clinical benefit. Therefore selected patients with prominently asymmetric parkinsonism, mild disease progression, and elderly patients can be proper candidates for unilateral STN DBS.

P-2-166

Risk factors for surgical site infections after deep brain stimulation of patients with Parkinson's disease in a 13-years period

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Background & Objectives: Deep brain stimulation (DBS) has been an established therapeutic option for patients with advanced Parkinson's disease (PD). However, infection is one of the most serious complications of DBS surgery for PD patients. We aimed to investigate the incidence, clinical characteristics, and risk factors of the infection in DBS surgery for PD patients. **Method:** We analyzed the infection rate in a consecutive series of 170 PD patients who underwent DBS surgery between April 2001 and October 2014, using a systemic review of electronic medical records. Since 76 PD patients underwent simultaneous bilateral DBS surgery and 94 PD patients received the staged unilateral DBS surgery, we analyzed 245 DBS surgery procedures. The subthalamic (STN) DBS surgery was performed in 154 PD patients, globus pallidus pars interna (GPi) DBS in 10 PD patients, unilateral STN and unilateral GPi DBS in 5

PD patients and the thalamic DBS in 1 PD patients were performed. **Results:** The total incidence of infection in our series was 7.1% (12 patients) of 170 PD patients and 4.9% (12 procedures) of 245 DBS surgery procedures. Patients were followed for a mean of 99.2 ± 182.9 days (ranging from 3 to 650) for infections. Gram-positive bacterium, such as *Staphylococcus epidermidis* and *Staphylococcus aureus*, were the most common pathogens (75%). Out of the twelve infections, four involved the frontal site, four involved the anterior chest, one involved the lateral chest, and one involved the periauricular site. Hardware replacement was performed in 25% (3 patients) of 12 infected PD patients. Mean duration of hospital stay for infection management was 18.2 ± 12.2 days, and mean cost for treatment was \$3,830 USD. Within 6 months of following the procedure, 10 Infected and 235 non-infected patients were compared. There were no significant differences in the following factors: age, sex, job, smoking, history of surgery, steroid usage, underlying disease (such as diabetes mellitus, hypertension, obesity and malnutrition), characteristics for DBS implantation and surgery procedure (such as ASA score, prophylactic antibiotics usage within one hour of procedure, and first dressing days). However, there were significant differences in the duration of operation time ($p=.006$), length of prophylactic intravenous antibiotic ($p=.002$) and total prophylactic antibiotic therapy ($p=.014$). The intensive care unit (ICU) management after DBS surgery ($p=.044$), procedure type ($p=.001$) and DBS model ($p=.022$). Multivariate analysis showed that the ICU management after DBS surgery and the short duration of prophylactic intravenous antibiotic treatment were correlated with the occurrence of infection. A shorter duration of prophylactic intravenous antibiotic treatment was correlated with a higher risk of surgical site infection compared to those with longer duration of prophylactic intravenous antibiotic treatment ($OR=0.606$, $p=.002$). In addition, the infection rate in ICU management after DBS surgery was shown to be significantly different from non-ICU management after DBS surgery ($OR=5.007$, $p=.043$). **Conclusion:** Our study suggests that infection still remains an unresolved issue after DBS surgery for PD patients, and ICU management after DBS surgery and the short treatment period of prophylactic intravenous antibiotics were associated with the increased risk of infection. However, it is unclear why higher infection rates are correlated with shorter prophylactic intravenous antibiotics treatment periods and ICU management after surgery. In the future, multicenter studies with a larger sample size should be conducted.

P-2-167

Bilateral deep brain stimulation of the subthalamic nucleus under sedation with propofol and fentanyl

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Background & Objectives: Awakening during deep brain stimulation (DBS) surgery is very stressful to patients. The aim of the current study was to evaluate the effect on MER signals and their applicability to subthalamic nucleus (STN) DBS surgery for patients with Parkinson's disease (PD) under sedation with propofol and fentanyl. **Method:** Sixteen consecutive patients with PD underwent STN-DBS surgery with propofol and fentanyl. Their MER signals were achieved during the surgery. To identify the microelectrodes positions, the preoperative MRI and postoperative CT were used. Clinical profiles were also collected at the baseline and at 6 month after surgery. **Results:** The firing rates of MER signals did not show any differences between both sides. All that signals were slightly attenuated and contained only bursting patterns, compared with our previous report. All electrodes were mostly located in the middle one third part of the STN on both sides of the brain in the fused images. Six months later, the patients were improved significantly in the medication-off state and they met with less dyskinesia and less off-duration. **Conclusion:** Our

study revealed that the sedation with propofol and fentanyl was applicable to STN-DBS surgery. There were no significant problems in precise positioning of bilateral electrodes. The surgery also improved significantly clinical outcomes in 6-month follow-up.

P-2-168

Electrode reposition cases in subthalamic nucleus deep-brain stimulation for Parkinson's disease

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Background & Objectives: Review clinical characteristics of lead reposition cases in subthalamic nucleus DBS. **Method:** Among 100 patients treated by deep brain stimulation from March 2013 to July 2015 in the Asan Medical Center by one neurosurgeon and one neurologist (J.K. Lee and C.S. Lee), lead reposition cases were collected. Four patients were referred and four patients were reoperated. Patient's global impression (PGI) were scored postoperatively. The distances to ideal targets (dist-I) and active electrodes were measured in all pre and postoperative leads. **Results:** Leads were repositioned in seven patients of 11 electrodes. Intervals between the initial lead insertions and revisions ranged from 1 to 157 months (median: 38 months). Unilateral revisions were done in three patients, and bilateral revisions were done in four patients. All revised patients showed various symptoms and side effects including tremor, dyskinesia, freezing of gait that were uncontrolled medically. DBS stimulation had suboptimal effects in these patients. Mostly, patients with dist-I at least 2 mm at one electrode were revised. One 47 years old female patient with minimal electrode malposition of 1.2 mm was also revised because of severe tremor. Five patients were considerably improved with PGI score 2 or 3. In addition, dist-I significantly reduced in these patients (3.2 ± 2.1 to 1.3 ± 0.9 , $p = 0.005$, Wilcoxon signed rank test calculated by the exact method). In two advanced Parkinson's disease patients with preoperative UPDRS score greater than 50, the dist-I were about 1.5 - 2.7 mm, dist-I were improved moderately after operations (1.3 to 1.5, 1.2 to 1.0, 2.7 to 1.6, 2.0 to 0.9). These patients were minimally improved with PGI score by one. No patient became worse after revision. **Conclusion:** Our result shows that lead reposition can be sometimes required in suboptimal outcome DBS to improve the outcome. Especially the patients with dist-I greater than 2 mm accuracies are effectively improved with the current surgical techniques. Our results support the findings in the previous studies that revision is clearly indicated in patients with dist-I > 2mm. A finite element modeling study suggested that electrode reposition by only 1 mm may cause significant changes in charge distribution. Our study actually showed that one relatively young patient with dist-I = 1.2 mm also improved considerably. On the other hand, improvements seemed lesser in advanced Parkinson's disease patients with about 2mm dist-I and about 1mm improvements of dist-I postoperatively, as judged by PGI.

P-2-169

Mortality after deep brain stimulation surgery for patients with advanced Parkinson's disease

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Background & Objectives: Despite the widespread use of deep brain stim-

ulation (DBS) for patients with Parkinson's disease (PD), long-term outcomes are still required to be investigated. We aimed to analyze the mortality of advanced PD patients who received DBS surgery. **Method:** We assessed the survival rate of consecutive 160 advanced PD patients who underwent DBS surgery between April 2002 and May 2014. Kaplan-Meier survival curves were constructed using death as the endpoint. Cox proportional hazards regression models were used to assess the association of clinical risk factors with survival. **Results:** Twenty-seven (16.9%) PD patients (13 men and 14 women) had died by July 15, 2015 with the mean follow-up period of 4.9 ± 3.1 years. The survival rate was 97% at three years after DBS surgery and 85% at five years after DBS surgery. Pneumonia (N = 7) was the most common specific cause of death. In a step-wise Cox regression analysis, male gender (hazard ratio (HR) = 3.0; 95% confidence interval (CI) = 1.35 - 6.65; P = 0.007), hallucinations (HR = 3.95; 95% CI = 1.60 - 9.78; P = 0.003), and the placement of nursing home (HR = 2.81; 95% CI = 1.30 - 6.09; P = 0.009) predicted poor survival. **Conclusion:** The poor survival of advanced PD patients who underwent DBS surgery was predicted by male gender, hallucinations, and the placement of nursing home. Further studies with long-term follow-up will confirm these factors influencing mortality of these PD patients.

P-2-170

Incongruent hemiatrophy and hemiparkinsonism in a patient with schizencephaly

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Background & Significance: Hemiparkinsonism-hemiatrophy (HPHA) syndrome is characterized by atrophy of hemibody with parkinsonism, which can be accompanied by the atrophy of contralateral hemisphere. Schizencephaly is a rare congenital developmental disorder. Herein we report a case with schizencephaly presenting with hemiatrophy and contralateral hemiparkinsonism. **Case:** A 66-years-old male visited us due to left hand tremor. He had a history of limping gait and a small size of the right hand since childhood. Physical examination showed hemiatrophy of the right hemibody. Neurologic examination showed resting tremor and mild bradykinesia in the left hand. Brain MRI showed closed-lip schizencephaly in left parietal lobe. 18F-fluorinated N-3-fluoropropyl-2-beta-carboxymethoxy-3-beta-(4-iodophenyl) nortropane positron emission tomography showed decreased dopamine transporter bindings in the bilateral putamen. Levodopa was effective for his parkinsonism. **Conclusions or Comments:** Hemiatrophy has never been associated with contralateral schizencephaly. Parkinsonism is not reported in the patients with schizencephaly, either. In HPHA syndrome, the hemiatrophy is congruently present in the same hemibody affected by parkinsonism. In this case, parkinsonism in the right hemibody could be masked by precedent hemiatrophy, resulting incongruent combination of hemiatrophy and hemiparkinsonism.

P-2-171

Selective fascicle injection of botulinum toxin at the flexor digitorum superficialis and flexor digitorum profundus in patient with focal dystonia affecting fingers

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Background & Significance: Focal dystonia is often observed in adult onset primary dystonia that affect a specific area of the body. It often aggravated by purposeful actions and may be specific to a particular task. Botulinum toxin has known as the treatment of choice for the majority of focal dystonias. However, weakness of the target muscle is the most common side effect and it

was seen in 53-100% of those patients who have been treated with botulinum toxin for focal dystonia. Especially in patients with focal hand dystonia, weakness can be an influential factor that cause a functional deficit **Case:** A 37-year-old man with right hand dominant presented to our clinic with progressive stiffness of fingers for a year whenever he try to write. The patient used to develop excessive flexion of the thumb, index and middle fingers of right hand. He could not continue writing without taking his pen again. On examination he had good range of motion and normal grip strength. The patient had tried oral medications with no effect. On repetitive examination we counted flexors of the index finger as responsible muscles of his writing difficulty. We decided to inject botulinum toxin at the selective fascicle of flexor digitorum superficialis digit 2(FDS2) and flexor digitorum profundus digit2(FDP2) under electromyography guidance. FDP2 separates from the muscle belly earlier and located on the deepest portion of the muscle belly from the surface (at a proximal one third of forearm, OIS of electromyographic approach). We injected 40 unit of abobotulinum at the highest MUAP point when the patient flexed an index finger. To inject FDS2, landmarking line(LL) was established between the medial epicondyle and the pisiform. This was taken to be 100% of lenth. Estimated OIS of FDS2 is 72%(%LL) and 14mm at right angles from the LL. We injected 70 unit of abobotulinum A under electromyography guidance. 2 weeks later, although the patient had mild weakness of flexors of index and middle fingers, he notified much improvement of his writing. And he also did not complaint of any other functional impairment with his hands. **Conclusions or Comments:** Selective fascicle injection of botulinum toxin in patients with focal hand dystonia is more effective and safe treatment because it can minimize unwanted weakness after botulinum toxin injection.

P-2-172

Unilateral limb asterixis related to hypoperfusion of middle cerebral artery territory

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Background & Significance: Unilateral limb asterixis related to cortical infarct or drugs is an unusual clinical feature. We found this association in one patient. Magnetic resonance imaging (MRI), MR anginography (MRA), somatosensory evoked potentials (SEPs), electroencephalogram (EEG) and electromyographic recording were performed. **Case:** 72-years old man developed an acute left ataxia with left limb asterixis. This consisted of frequent arrhythmic loss of extensor muscle tone on instruction to maintain the wrist and fingers extended. Voluntary electromyographic activity in the left extensor digitorum communis muscle showed abrupt periods of interruption ranging from 50 to 100 milliseconds in duration. SEPs were normal. No definitive acute brain lesions were detected in MRI. But MRA represented right middle cerebral artery M1 occlusion. EEGs showed continuous slow on right hemisphere. One day later, acute left ataxia with left limb asterixis was disappeared. Rechecked EEGs were normal. **Conclusions or Comments:** Our case suggest that the hypoperfusion of cerebral hemisphere without structural lesions might cause unilateral asterixis.

P-2-173

Dystypia without aphasia in a patient with Parkinson's disease after deep brain stimulation

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Background & Significance: Previously, two case reports showed that the acute infarction in the left frontal lobe explained the clinical manifestation of dystyopia without aphasia. We add another case report of dystyopia without aphasia, which are considered to be caused by the edematous change around the electrode insertion site in the left frontal lobe after the operation of deep brain stimulation. **Case:** We report a 50-year-old right-handed Korean woman who showed a sudden typing impairment using a cell phone without aphasia after she received a surgical procedure for deep brain stimulation. She had no specific underlying disease but has suffered her right leg tremor as her chief complaint since 2008. She first visited our neurology department in 2008 and was diagnosed as Parkinson's disease. She has been regularly followed up in our outpatient clinic with anti-PD medications until recently. Her daily activity was improved with a proper dosage of levodopa, but interrupted with wearing-off and on-dyskinesia phenomena. On June 25th, 2015, she was admitted into our neurology department for the preoperative workup for deep brain stimulation. After the preoperative workup was finished, she was transferred to the department of neurosurgery and received a stereotactic operation for bilateral subthalamic nucleus deep brain stimulation. After electrode insertion, she was retransferred to our neurology department to adjust anti-PD medications. We daily maintained 400mg of Perkin divided by three instead of our previous cocktail regimen including levodopa, dopaminergic agonist and COMT inhibitor before the operation of deep brain stimulation. She showed neuropsychiatric changes which contained apathy and depressive mood. She also lost the whole score specifically from the part of attention and calculation in K-MMSE. We considered the possibility of dopaminergic agonist withdrawal syndrome and supplied her with 4mg of Requip. She was almost completely recovered from apathy and depressive mood to the previous neuropsychiatric status. In the preoperative workup, she obtained the score of 30/30 in K-MMSE. After the operation of deep brain stimulation, she obtained the score of 25/30 in K-MMSE, showing the complete loss of score in the part of attention and calculation. Supplemented with dopaminergic agonists, she gained the score of 29/30 in K-MMSE, especially 4/5 in the part of attention and calculation. After the recovery from dopaminergic agonist withdrawal syndrome, she was retransferred to the department of neurosurgery and received an operation of bilateral transaxillary subpectoral implantation of IPGs. After the successful operation, she was retransferred to our neurology department for the adjustment of electrical pulse in deep brain stimulation and anti-PD medications. Her anti-PD medication were adjusted to 300mg of Perkin, 4mg of Requip and 1.5mg of Mirapex. While we talked with her husband about her medical status after the operation of deep brain stimulation, we found out that she often sent incomprehensible messages to her husband through her cell phone after she received the operation of deep brain stimulation. We investigated her language functions including speech, comprehension, reading and writing. We concluded that she did not show any aphasic signs, but displayed clinical manifestation of dystyopia. For the anatomical localization of the brain lesion that is clinically presented with dystyopia without aphasia, we performed the magnetic resonance imaging of her brain. The T2-weighted MRI image showed that the electrode insertion site in her left frontal lobe was accompanied by the edematous change. **Conclusions or Comments:** In conclusion, the brain lesion in the left frontal cortex and subcortical white matter caused by acute cerebral infarction or electrode insertion for deep brain stimulation might play an important role in the clinical manifestation of dystyopia without aphasia.

P-2-174

Parkinsonism in EPM and CPM without striatal dopamine binding dysfunction: a case report

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Background & Significance: Extrapontine and central pontine myelinolysis are related to rapid correction of hyponatremia or metabolic disequilibrium syndrome. Clinical symptoms include conscious disturbance, pseudobulbar palsy and behavior problems. Some previous studies reported the parkinsonian feature could be seen in EPM and CPM. We report a single case of EPM with Parkinson feature. We performed [18F] FP-CIT PET to investigate the pathophysiology of EPM with Parkinsonism. **Case:** A 66 year old, man with a history of chronic alcoholics visited emergency room presented with tremor, gait disturbance and bradykinesia. He shows bilateral resting tremor, masked face, rigidity, bradykinesia and postural instability. His symptoms were compatible to Parkinson disease, modified Hoehn and Yahr stage was 5. His family told these parkinsonian features started abruptly, at one month ago. One month ago, he was admitted to nephrology for general weakness and hyponatremia. When he admitted to nephrology, his laboratory studies showed hyponatremia (96mEq/L), hypokalemia (2.9mEq/L) and hypoosmolarity (204mOsm/Kg) without any other abnormality. His brain MRI was normal. After infusion of isotonic saline, the sodium concentration increased to 107mEq/L over 3days. At 4th hospital day, tolvaptan and isotonic saline administered, serum sodium concentration increased to 122mEq/L (1mEq/L/hr). At this day, he experienced generalized tremor, mutic feature and confused mental state. His family did not want further treatment, he transferred to other hospital. His tremor, confusion and bradykinesia were continued after discharge. At 10days after discharge, he performed brain MR imaging at other hospital. T2 and FLAIR images shows abnormal high signals in central pons, bilateral caudate and putamen. These lesions were high signal intensity at diffusion and ADC image that suggests vasogenic edema. These MR finding were compatible to CPM and EPM. We performed [18F] FP-CIT PET, there was no striatal dopamine dysfunction. We tried dopaminergic medication (levodopa/carbidopa: 100/25mg), his symptom were markedly improved. After 6 month later, the patient is neurologically normal state with small dose dopaminergic medication (levodopa 300mg/day) **Conclusions or Comments:** This case demonstrates Parkinsonism in EPM and CPM without striatal dopamine binding dysfunction. Previous studies reported that dopamine deficiency may be main cause of extrapyramidal symptom in EPM or CPM patients. Other study reported EPM with parkinsonism patient shows striatal dopamine binding dysfunction in [123I]IPT SPECT image. They suggest the damage of nigrostriatal dopaminergic neuron may be main pathogenesis of EPS. But our case suggests pathogenic mechanism may be not confined to nigrostriatal dopaminergic pathway.

P-2-175

Glutamic acid decarboxylase antibody associated paraneoplastic cerebellar syndrome in thymoma

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Background & Significance: Glutamic acid decarboxylase (GAD) antibody was described in a few patients with idiopathic cerebellar ataxia. Although GAD antibody is frequently encountered neuronal autoantibody in thymoma, GAD antibody associated paraneoplastic cerebellar degeneration in thymoma was very rarely reported. We reported a case with GAD antibody and thymoma associated paraneoplastic syndrome presenting positional vertigo and severe truncal ataxia, which was completely resolved by thymectomy **Case:** A 63-year-old woman without underlying disease presented aggravated positional vertigo over 2 weeks. Over time, she could not walk without assistance due to severe truncal ataxia and vertigo aggravated with motion. Repeated

brain magnetic resonance imaging (MRI) which had been checked with 2-week interval did not show abnormal finding. Radius fracture of left wrist and small subdural hemorrhage in left parietal lobe occurred due to frequent fall down. Video-oculography showed positional nystagmus initially and multi-directional nystagmus on follow-up. After 1-month duration of symptom, computed tomography (CT) scan of chest and abdomen was evaluated with suspicion of paraneoplastic syndrome. On her chest CT, there was about 3*4cm size lobulating mass at left side anterior mediastinum (invasive extracapsular type B2). GAD antibody was elevated (215.62U/mL). With steroid pulse therapy, her dizziness was rather improved. After thymectomy, her vertigo and truncal ataxia completely resolved **Conclusions or Comments:** Paraneoplastic cerebellar degeneration is rare in thymoma, and its combination with elevation of GAD antibody was even rarely reported. In this case, her cerebellar syndrome associated with GAD antibody and thymoma was improved by steroid therapy and completely resolved by cancer treatment. In addition, her initial neurologic manifestation mimicked with peripheral vertigo and later on, her symptom progressed to truncal ataxia without limb ataxia and multidirectional nystagmus was observed. Atypical positional vertigo or ataxia associated with GAD antibody should be considered as a symptom of paraneoplastic syndrome by thymoma.

P-2-176

The quality of life in patients with hemifacial spasm

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Background & Objectives: Hemifacial spasm (HFS) is a movement disorder manifested by unilateral spasms of the muscles innervated by the facial nerve. Botulinum toxin provides an effective medical treatment. Since HFS is a chronic disease, it frequently interferes with social life, causing social isolation and depression and having a significant impact on the quality of life. The aim of the study was to assess the quality of life in patients with HFS in respect of influence of the severity of depression symptoms. **Method:** Fifty-two patients included from the Outpatient Clinic, Department of Neurology, Jeju National University Hospital, who fulfilled the inclusion criteria and had no exclusion criteria (suffering from concomitant movement disorders, other severe chronic diseases or cognitive impairment). Demographic and clinical data were collected. Severity of HFS was assessed by the five-point clinical scale. Quality of life was assessed with the 36-Item Short-Form Health Survey (SF-36) questionnaire and severity of depressive symptoms was evaluated with the Beck Depression Inventory. Accompanying HFS non-motor and motor-related symptoms were asked by a questionnaire without quantifying their severity. **Results:** (Among fifty-two patients included in this study, twelve patients have taken only medication without Botulinum toxin therapy.) The mean global score of SF-36 was 100.9 ± 18.8 , and BDI was 15.9 ± 6.6 . Over 75% of patients reported HFS non-motor and motor-related symptoms. Decreased score of SF-36 was affected by increased severity of HFS and the number of accompanying non-motor and motor-related symptoms. **Conclusion:** Our data suggest that the severity of HFS and the accompanying non-motor symptoms affect the quality of life in HFS patients.

P-2-177

Clinical implication of initially affected side in typist's cramp

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Background & Objectives: The aim of this study is to characterize typist's cramp and to know whether initially affected hand may be a prognostic factor for progression. **Method:** We collected patients with typist's cramp from our hospitals and conducted PubMed search to identify previous published cases. **Results:** 11 typist's cramp (mean age: 33.0 ± 13.7 years, 6 women) were analyzed. All of them were right-handed. We classified the patients into three subgroups based on previous definitions (simple, dystonic, and progressive cramp). 7 of 11 typist's cramp (63.6%) was simple cramp. Among them only one patient was right-handed, whereas the other 4 patients (36.4%) with dystonic or progressive cramp were all left-handed ($p=0.015$). **Conclusion:** Our study suggests that initially affected side may be a determinant for the disease progression, which may be related to more frequent use.

P-2-178

Respiratory dysfunction in patients with Parkinson's disease and multiple system atrophy

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Background & Objectives: Respiratory function abnormalities are critical symptoms in patients with both Parkinson's disease (PD) and multiple system atrophy (MSA), however there have been few studies of respiratory dysfunction in patients with PD and MSA. In the present study we investigated respiratory function in early to mid-stage of PD and MSA patients to identify the characteristics of respiratory dysfunction. Specifically, we examined association between respiratory dysfunctions of patients and their clinical factors. **Method:** 24 patients with PD (12 females, mean age = 68.04 ± 9.28 years, Hoehn & Yahr stage (HYS) 1.0-3.0) and 5 patients with probable MSA (4 females, mean age = 61.20 ± 6.14 years, HYS 2.0-3.0) with no history of respiratory disease were recruited from the movement disorders clinic of Korea University Guro Hospital and performed respiratory function tests using a spirometer. All patients were non-smoker. The respiratory parameters (spirometry, lung volumes and diffusing capacity) were collected and expressed as percentages of predicted values. Demographic data was obtained through interviews with the patients and motor severity was assessed with UPDRS motor subscale score, HYS and tremor/PIGD score. Autonomic dysfunction was assessed with SCOPA-Aut scale, head-up tilt test, valsava ratio, heart rate variability and sympathetic skin test. The respiratory parameters were compared between the groups. Also, the respiratory parameters were correlated with clinical factors. **Results:** There were no differences in age and gender distribution between PD patients and MSA patients ($P > 0.05$). Four patients with PD and one patient with MSA were classified as restricted. Two patients with PD were classified as obstructed. 18 of 24 PD patients (75%) and 4 of 5 MSA patients (80%) had lung function within normal limits. PD patients had lower FVC, FEV1, FEV1/FVC and MVV values than those expected for normal controls. MSA patients had lower FVC, FEV1, FEV1/FVC, PEF, MVV, TLC and DLco values than those expected for normal controls. Predicted maximal voluntary ventilation (MVV) and predicted diffusing capacity of the lung for carbon monoxide (DLco) were significantly reduced in MSA patients compared to PD patients ($P < 0.05$). In PD patients, respiratory parameters were not correlated with disease duration, UPDRS score or HYS. Between-group comparisons in PD patients (abnormal vs. normal sympathetic function and abnormal vs. normal parasympathetic function) revealed no differences in respiratory parameter. Some parameters were correlated with SCOPA-Aut scores

(total score - RV/TLC, Gastrointestinal subscore -FEV1, Urinary subscore - PEF, RV/TLC). **Conclusion:** The results of the study might be interpreted that abnormalities of respiratory parameters preceded the onset of clinical respiratory symptoms even in early to mid-stage of PD and MSA patients. Abnormality of obstructive parameters was dominant in PD patients and abnormality of restrictive parameters was more distinctive in MSA patients. Respiratory muscle weakness is common in both groups, but insufficient to cause clinical symptoms. Our findings of reduced MVV and DLco in MSA patients might reflect more severe extrapulmonary restriction such as reduced axial muscle power in MSA patients than PD patients. These results showed that MVV and DLco have the potential to become clues for differential diagnosis of MSA from PD. Association between SCOPA-Aut scores and respiratory parameters in PD patients might suggest that respiratory dysfunction in PD patients is related to autonomic dysfunction, especially obstructive dysfunction. Further studies are needed to elucidate precise Respiratory function abnormalities and relationship between respiratory dysfunction and dysautonomia in PD and MSA patients.

P-2-179

Restless legs syndrome in Parkinson's disease patients: a comparative study on prevalence, clinical features, motor and non-motor symptoms

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Background & Objectives: Restless legs syndrome (RLS) is a common neurological disorder that can coexist with Parkinson's disease (PD). However, the precise pathophysiology of co-existence of two disease remains unknown, it is assumed that dopaminergic mechanisms play a central role. Previous studies have shown an association between restless legs syndrome and Parkinson's disease. We investigate the prevalence of restless legs syndrome in Parkinson's disease patients and to identify associated clinical features, motor and non-motor symptoms. **Method:** Exclusion criterion were patients with Mini Mental State Examination score of less than 21/30 and atypical parkinsonism. The International Restless Legs Syndrome Study Group criterion was used to identify patients with restless legs syndrome. Further assessments were performed on blood test, clinical features, PD severity scales, PD sleep scale, anxiety, depression, QoL, and autonomic dysfunction (SCOPA-AUT) in PD patients with and without RLS. **Results:** A total of 24 patients were recruited. The prevalence rate of restless legs syndrome in our cohort was 20.8% (n=5). There was significantly associated with a longer disease duration of PD **Conclusion:** Our study demonstrated a higher prevalence of RLS in patients with PD compared to general population. PD patients with RLS suffer from more anxiety, depression, poorer sleep quality, and worse autonomic dysfunction, especially urinary problem. This is a preliminary and baseline study, more patients recruiting and further researches are needed.

P-2-180

Association between urine protein/creatinine ratio and cognitive dysfunction in Parkinson's disease

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Background & Objectives: Impaired renal function and proteinuria have been associated with cognitive impairment and dementia. Chronic kidney disease is considered to be an independent risk factor for Parkinson's disease. However,

few studies have mentioned an association between proteinuria and cognition in Parkinson's disease. We investigated the relationship between proteinuria and cognitive dysfunction in patients with Parkinson's disease. **Method:** Among 172 patients with Parkinson's disease, 53 had normal cognition, 76 had mild cognitive impairment, and 43 had dementia based on comprehensive neuropsychological tests. The urine protein/creatinine ratio was calculated using the spot urine test. **Results:** The urine protein/creatinine ratio was significantly higher in patients with dementia than in those with mild cognitive impairment or cognitively normal patients. Each abnormal neuropsychological test result was associated with increased urine protein/creatinine ratio, except for those associated with the language and calculation domains. After controlling for age, diabetes mellitus, hypertension, symptom duration, and parkinsonian motor severity, the urine protein/creatinine ratio was significantly associated with decreased cognition. **Conclusion:** The urine protein/creatinine ratio was associated with dementia in Parkinson's disease. These finding suggests that increased protein excretion is associated with cognitive dysfunction in patients with Parkinson's disease.

P-2-181

Improvement of standing stability after weight-shifting training in spinocerebellar staxia type 6: a case report

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Background & Significance: Progressive postural imbalance is a devastating symptom in people with degenerative cerebellar ataxias. Currently, there are no pharmacologic treatments available to reverse or even substantially reduce immobility associated with postural imbalance. With a review of literature, we describe a patient with spinocerebellar ataxia type 6 (SCA type 6) who showed a remarkable improvement in the performance of standing stability after short-term training of weight-shifting. **Case:** A 39-year-old male who had been diagnosed with SCA type 6 at age 31 complained of recent worsening of balancing and gait with usual symptomatic management. Smart Equi test (NeuroCom® Inc., Clackamas, OR, USA), a widely accepted clinical instrument that has been used to measure balance performance, was employed for balance training in order to improve his standing stability. Total eight sessions of balance training were performed with a schedule of 2 times a week for one month. Balance performance of the patient was assessed using clinical scales and computerized dynamic posturography (CDP) before the first and after the final session. Follow-up CDP showed remarkable improvement in all measures of limit of stability (i.e., reaction time, movement velocity, endpoint excursion, maximum excursion and directional control). In addition, sensory organization test showed a prominent change in its pattern of abnormality suggesting improvement of ability to suppress inaccurate visual information (over-reliance on vision) for maintaining upright posture. **Conclusions or Comments:** This case suggests that weight-shifting training using CDP may be an effective way to improve standing stability in selected individuals with degenerative cerebellar ataxia.

P-2-182

Corticobasal syndrome-like sporadic Creutzfeldt-Jakob disease

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Background & Significance: As a rapidly progressive neurodegenerative disorder, CJD are characterized by progressive dementia, ataxia and myoclonus. The typical subtype of CJD is sCJD, which accounts for over 85% of CJD. Creutzfeldt-Jakob disease (CJD) presented with CBS is very rare. We report a

patient that presented with the corticobasal syndrome (CBS), including limb apraxia, dystonia, asymmetrically **Case:** 2015 February, A 60-year-old, right-handed, man without history of other disease, complained of an paresthesia his right hand and pressing type mild headache over the course of the last one month. He seems right hand clumsiness and paresthesia on right hand. Over the 2weeks, the symptoms like as right upper arm's dystonia, hyp-esthesia are progressed. On admission, he was found to have right hand alien phenomena with ideomotor apraxia, myoclonus and mild rigidity affecting his right arm. Over two weeks, loss of balance, postural instability, so he can't stand, so he visited our hospital by wheelchair. Furthermore visual hallucination began to occur. His initial vital signs were blood pressure of 122/68 mmHg, pulse of 74/min, and body temperature of 36.7c. His electrocardiography was within normal limit. In neurologic examination, her mental status was alert, and relately his languafe function, metal fuction was normal. However, he showed neither attention and calculation nor recall memory and visuospatial function in bedside K-MMSE, without meningeal irritation sign. First brain MRI, EEG resulted normal, over the next two weeks, there was a worsening in gait, progressively involving right arm paresthesia, dystonia, a spontaneous myoclonus. The second brain MRI revealed an extensive and diffuse DWI hyperintense signal in the caudate and lenticular nuclei, cerebral cortices on the bilateral hemisphere. The second EEG also showed diffuse slowing with periodic lateralized sharp wave in the left centroparietal area

Conclusions or Comments: We presented a patient with only motor, sensory symptoms without the cognitive dysfunction Corticobasal syndrome (CBS) can occur in neurodegenerative disease including CJD CJD should be suspected in patients presenting with CBS when clinical progression is rapid (<12 months)

P-2-183

Inherited cerebellar ataxia type 17 (SCA17) associated with 41 trinucleotide repeats of TATA-box binding protein gene(TBP) in a family

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Background & Significance: Autosomal dominant spinocerebellar ataxia type 17 (SCA17) is a neurologic disorder with diverse manifestations, such as limb ataxia, dysarthria, myoclonus, dementia, parkinsonism and psychiatric symptoms, and it is caused by increased CAG/CAA repeats within the TATA- box binding protein (TBP) gene. The expansion more than 42 repeats in TBP gene was thought to be pathologic, but single cases with 41 CAG/CAA repeats manifesting clinical SCA17 have been reported, meaning that the border of pathologic margin of TBP repeats could be lowered. Here, we report a family with members manifested the feature of SCA17 with 41 repeats. **Case:** A 57-year-old female (patient 1) visited with progressive gait imbalance, which occurred 8 years ago. Among her family members, her younger sister and her daughter (patient 2) had similar symptoms. On neurologic examination, she had slight cognitive impairment, with mini-mental status examination (MMSE) score of 28/30 and Montreal cognitive assessment (MoCA) score of 19/30. She had hypomimia, dysarthria, and rigidity of all extremities. She also showed bilateral, symmetric limb ataxia on heel-to-shin test and rapid alternating movement test. Her brain MRI revealed diffuse brain atrophy which is especially severe in cerebellum. Her daughter (patient 2) also visited us for tremor. She had mental retardation from childhood, and had been taking anti-psychotics (olanzapine 15mg/day) under the diagnosis of schizophrenia since adolescence because of obsession and persecutory delusion. Although there was no recent change in medication, she started to have tremor from several months ago. She showed cognitive impairment (MMSE 11/30, MoCA 5/30),

hypomimia, dysarthria, and bradykinesia. She also showed mild limb ataxia on rapid alternating movement and postural instability on the pull test. As her mother, her brain MRI revealed diffuse brain atrophy. Because of the similar pattern of neurologic symptoms in the family members, we performed gene tests under suspicion of genetic disorders, especially hereditary ataxia. Tests for SCA type 1, 2, 3, 6, and 7 were all negative, but expansion of 41 CAG/CAA repeats of TATA box-binding protein (TBP) gene was found in both patients. **Conclusions or Comments:** It is already known that abnormal expansion of CAA/CAG trinucleotide within TBP gene plays a key role in SCA17. However, the margin of pathologic expansion is still not clear. A few obstacles, like variable manifestation of the disease, and reduced penetrance in smaller expansion less than 48 repeats make the problem difficult in setting the boundary. To date, 41 repeats are suggested as the minimum size for clinical onset of SCA17, but these reports relied on a single case, so it is difficult to certainly tell this number of TBP expansion was crucial for the onset of the disease. From that point, our case can be a good evidence for setting 41 repeats categorized to be pathologic. The patients we describe here, the mother and the daughter, showed various neurologic manifestations in common, which implies that this complex of symptoms is inherited. Furthermore, the onset of psychiatric symptom of the daughter in her adolescence can be explained by the influence of unstable trinucleotide repeats in TBP gene. Diffuse atrophy of cerebrum and cerebellum shown in MRI also advocates this speculation. Genetic study also revealed only slightly increased 41 trinucleotide repeats of TBP gene, otherwise normal. This case report can support previous reports of single case with 41 TBP repeats, and also be a strong evidence for lowering the margin of pathologic expansion of SCA17 gene locus. However, further study is needed to investigate what decides the clinical onset of SCA17 in the small expansion of repeats.

P-2-184

Aspergillus abscess presenting as chronic progressive cerebellar ataxia

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Background & Significance: Cerebellar ataxia has a wide variety of causes. Chronic cerebellar degeneration may be slowly progressive and untreatable. In acute disease, on the other hand, acute cerebellar swelling due to infarction, edema, or hemorrhage can have rapid and catastrophic effects and is a treatable disease. So, Special emphasis is placed on causes of cerebellar ataxia, both acquired and genetic, that are reversible when timely therapy is initiated. However, not always such officialisfit. We experienced progressive cerebellar ataxia could be focal lateralized mass. **Case:** A 72-year-old woman presented with a 2-year history of slowly progressive gait disturbance. She had dysarthria and bilateral limb and gait ataxia. She had history of mastoidectomy due to right otomastoiditis. Brain MRI showed an intracranial abscess at the right cerebellopontine angle along with recurring otomastoiditis (Panel A). The abscess was drained and aspergillus flavus grew from the cultures. She eventually underwent a subtotal petrosectomy with voriconazole and improved neurologically over two weeks. Late-onset, chronic slowly progressive cerebellar ataxia is usually observed in neurodegenerative diseases. However, treatable causes should be ruled out even in a chronic course of ataxia. **Conclusions or Comments:** This report highlights that underlying etiology of a patient with chronic bilateral progressive cerebellar ataxia could be focal lateralized mass rather than untreatable neurodegenerative diseases. Our patient had aspergillus abscess at her right cerebellopontine angle, which was originated from right mastoiditis, but her clinical symptoms were chronic bilateral cerebellar ataxia in which neurodegenerative diseases such as multiple system atrophy were initially suspected.

P-2-185

A case of acute focal myelitis presented with painful tonic spasm involving both legs

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Background & Significance: Painful tonic spasms, a form of paroxysmal dyskinesias, are rare movement disorder characterized by sudden-onset dystonic posturing. It has been reported mostly in patients with multiple sclerosis or neuromyelitis optica (NMO). With a review of literature, we report a rare case of focal myelitis presented with painful tonic spasms and its characteristic surface electromyographic (EMG) finding. **Case:** A 28 year-old unmarried man presented with painful tonic contraction of muscles in both lower extremities. There were no history of infectious diseases, vaccination, and overseas travel within last 4 weeks. Vital signs including body temperature were stable. Neurologic examination showed mild paraparesis, hypoesthesia in left leg, bilateral heightened deep tendon reflexes, and extensor toe sign in the left lower extremity. Both lower extremities were rigidly extended with sustained paroxysmal tonic spasms which lasted briefly (<1min). The movement occurred spontaneously or by the slightest stimulations (light touch or noise). Surface EMG in vastus lateralis and peroneus longus muscles revealed characteristic interference pattern compatible with tonic spasm. CSF studies showed subtle pleocytosis (5-7 cells/mm³) with normal range of glucose, protein, immunoglobulin G(Ig G) index, without oligoclonal band. Laboratory studies, including rheumatoid factor, antinuclear antibody titers, double stranded DNA, and NMO-IgG were normal or negative. Spine MRI revealed subtle high signal intensity at T4-T5 level in T2 weighted image. 4 weeks later, the patient discharged with nearly full recovery of painful tonic spasms after standard antiviral and symptomatic treatments with high dose corticosteroid. **Conclusions or Comments:** This case suggest that painful tonic spasms can be a presenting symptom of acute focal myelitis, and characteristic surface EMG finding have diagnostic implication for similar paroxysmal dyskinesias in a certain clinical setting.

P-2-186

The first case report of adult onset Niemann-Pick disease type C in Korea

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Background & Significance: Niemann-Pick type C is a rare, autosomal recessive disease with visceral, psychiatric and neurological symptoms. We report Niemann-Pick type C in two siblings. **Case:** First case was 25-year-old male. He started to develop delusions and limb clumsiness at the age of eighteen years. Hallucination, cognitive impairment and gait disturbance followed within a few years. On examination, He showed severe ataxic gait, limb dystonia with ataxia, dysarthria, vertical gaze limitation, hyperreflexia, and severe cognitive deficits. These progressive symptoms were refractory to symptomatic medical treatment. The abdominal CT scan revealed hepatosplenomegaly which was not detected on routine physical examination. There was no definite abnormal finding in the brain MR image, but widespread hypoperfusion was found in the brain perfusion SPECT. Second case was 23-year-old female, younger sister of the first case. Her symptom was started with right hand clumsiness at the age of nineteen years. And then psychosis, cognitive impairment and gait disturbance developed during the next few years. On examination, she had similar symptoms with her brother, but was milder than him. She also had hepatosplenomegaly in the abdominal CT scan, and mild diffuse brain atrophy was found in the brain MR image. NPC1 gene sequencing re-

vealed compound heterozygote for p.R518W and p.A927V mutations in both cases, already known as a genetic cause of Niemann-Pick type C. And filipin staining tests in cultured fibroblasts to evaluate amount of free cholesterol were positive in both cases. **Conclusions or Comments:** This is the first case report of adult form of Niemann-Pick type C in Korea. When a patient with generalized dystonia shows either or both vertical supranuclear ophthalmoplegia and prominent psychotic features which are unusually accompanied symptoms with dystonia, hepatosplenomegaly should be searched for the diagnosis of Niemann-Pick type C even though it is not noticeable on physical examination.

P-2-187

Inhibitory effect of apocynin on proteasome inhibition-induced apoptosis in differentiated PC12 cells

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Background & Objectives: The dysfunction of the proteasome system has been implicated in neuronal degeneration. Apocynin, a natural organic compound structurally related to vanillin, has been shown to have anti-inflammatory and antioxidant effects. However, the effect of apocynin on the neuronal cell death induced by proteasome inhibition has not been studied. **Method:** Using differentiated PC12 cells, in the respect of cell death process the preventive effect of apocynin on the proteasome inhibition-induced apoptosis in neuronal cells was examined. **Results:** The proteasome inhibitors MG132 and MG115 caused a decrease in cytosolic levels of Bid and Bcl-2 proteins, an increase in the levels of Bax, loss of the mitochondrial transmembrane potential, cytochrome c release, activation of caspases (-8, -9 and -3) and an increase in the tumor suppressor p53 levels. Treatment with apocynin attenuated the proteasome inhibitor-induced changes in the levels of apoptosis-related proteins, formation of reactive oxygen species, GSH depletion and cell death. **Conclusion:** Apocynin may attenuate the proteasome inhibitor-induced apoptosis in PC12 cells by suppressing the activation of the mitochondrial pathway and the caspase-8- and Bid-dependent pathways. The preventive effect of apocynin appears to be attributed to its inhibitory effect on the formation of reactive oxygen species and depletion of GSH.

P-2-188

Biochemical protective effect of 1,25-dihydroxyvitamin D3 through autophagy induction in the MPTP mice model of Parkinson's disease

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Background & Objectives: Dysregulation of the autophagy pathway has been suggested as an important mechanism in the pathogenesis of Parkinson's disease (PD). Therefore, modulation of autophagy may be a novel strategy for the treatment of PD. Recently, an active form of vitamin D3 has been reported to have neuroprotective properties. In our previous study, the neuroprotective effect of calcitriol has been confirmed in SH-SY5Y cells. In this article, we explored if calcitriol exerted neuroprotection in a sub-chronic 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-treated mouse model of Parkinson's disease. **Method:** An in vivo model of Parkinson's disease, MPTP treated mouse model was adapted. After treating with MPTP only, or co-treated with calcitriol, the substantia nigra pars compacta (SNpc) was dissected and related protein level was detected by western blot **Results:** Our results suggested that MPTP injected mice treated with calcitriol had been attenuated deficiency of

tyrosine hydroxylase (TH) expression and increased LC3-II conversion compared with those which did not. **Conclusion:** In conclusion, we have confirmed that the calcitriol has the anti-PD and neuroprotective effects in MPTP-induced in vivo PD model in mice. And the possible mechanism may be associated with the autophagy induction.

P-2-189

The neuroprotective effect of erythropoietin on rotenone-induced neurotoxicity in SH-SY5Y cells through the induction of autophagy

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Background & Objectives: Currently, the autophagy pathway is thought to be important for the pathogenesis of Parkinson's disease (PD), and the modulation of autophagy may be a novel strategy for the treatment of this disease. Erythropoietin (EPO) has been reported to have neuroprotective effects through anti-oxidative, anti-apoptotic, and anti-inflammatory mechanisms, and it has also been shown to modulate autophagy signaling in an oxygen toxicity model. Therefore, we investigated the effects of EPO on autophagy markers and evaluated its neuroprotective effect on rotenone-induced neurotoxicity. **Method:** We adapted the rotenone-induced neurotoxicity model in SH-SY5Y cells as an in vitro model of PD. We measured cell viability using MTT and Annexin V/propidium iodide assays and measured intracellular levels of reactive oxygen species. Immunofluorescence analysis was performed to detect the expression of LC3 and α -synuclein. Intracellular signaling proteins associated with autophagy were examined by immunoblot analysis. **Results:** EPO mono-treatment increased the levels of mTOR-independent/upstream autophagy markers, including Beclin-1, AMPK, and ULK-1. Rotenone treatment of SH-SY5Y cells reduced their viability, increased reactive oxygen species levels, and induced apoptosis and α -synuclein expression, and simultaneous exposure to EPO significantly reduced these effects. Rotenone enhanced mTOR expression and suppressed Beclin-1 expression, indicating suppression of the autophagy system. However, combined treatment with EPO restored Beclin-1 expression and decreased mTOR expression. **Conclusion:** EPO protects against rotenone-induced neurotoxicity in SH-SY5Y cells by enhancing autophagy-related signaling pathways. The neuroprotective effects of EPO against rotenone-induced dopaminergic neurotoxicity provide an experimental basis that may significantly impact the development of future PD treatment strategies.

P-2-190

Novel compound heterozygous mutations of PLA2G6 in a Korean pedigree of young-onset Parkinson's disease: a study of whole genome sequencing

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Background & Objectives: The PLA2G6 (Park 14) mutation causes neurodegenerative disorders with protean clinical phenotypes; particularly, it is reported to be associated with young-onset Parkinsonism. The purpose of this study is to understand the Park 14 gene mutation among familial Parkinsonism and to apply whole genome sequencing (WGS) for molecular diagnosis of familial Parkinsonism. **Method:** We performed whole genome sequencing (WGS) with genomic DNAs from two affected brothers and two unaffected

parents on Illumina HiSeqXTM Ten. All subjects in the study underwent 3Tesla MRI and PET with [18F]FP-CIT (a ligand for dopamine transporter). **Results:** [18F]FP-CIT PET showed a marked loss of ligand binding in the striatum in both affected brothers, but normal in unaffected parents. The regional pattern of reduced ligand binding was indistinguishable from sporadic PD. Brain MRI did not show significant abnormal findings. WGS studies identified novel compound heterozygous mutations c.359 G>A (p.W120*) in exon 3 and c.1742G>A (p.R581Q) in exon 13 of PLA2G6 in two brothers. The father carried the c.359 G>A variant whereas their mother carried c.1742 G>A. The mutations were not found in healthy controls. **Conclusion:** Two affected brothers with a clinical phenotype of typical PD revealed two novel compound heterozygous mutations in PLA2G6, each of which was inherited from the parents who have heterozygous mutation without loss of nigrostriatal dopamine neurons. This is the first report of familial PD with PLA2G6 (PARK 14) mutation in Korea.

P-2-191

Impaired vascular endothelial function in patients with idiopathic restless leg syndrome: a new aspect of vascular pathophysiology

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Background & Objectives: Restless legs syndrome (RLS) is a common sleep disorder in which patients feel unpleasant leg sensations and the urge to move their legs during rest, particularly at night. Leg movement improves these symptoms. Although several studies have demonstrated an association between cardiovascular disease and RLS, the mechanisms underlying this relationship remain unclear. Recent studies have shown the change of peripheral microvasculature including altered blood flow and capillary tortuosity, and peripheral hypoxia. Vascular endothelial dysfunction can be assessed non-invasively with ultrasound measurements of brachial artery flow-mediated dilatation (FMD). Therefore, this study investigated FMD in RLS patients to determine the involvement of microvascular alterations in this disorder. **Method:** The study enrolled 25 drug-naïve RLS patients and 25 sex- and age-matched controls and compared the FMD values of the two groups. RLS was diagnosed according to the criteria of the International Restless Legs Syndrome Study Group. **Results:** FMD was significantly lower in the RLS patients ($6.6 \pm 1.2\%$) compared to the controls ($8.4 \pm 1.8\%$; $p < 0.05$) and the RLS patients showed a negatively but weak correlation between RLS severity and FMD ($r = -0.419$, $p = 0.04$). Multivariate linear regression analysis revealed that RLS ($B = -1.87$, 95% confidence interval [CI] -2.72 to -1.02 ; $p < 0.001$) and age ($B = -0.06$; 95% CI -0.12 to -0.02 ; $p < 0.001$) were significantly and inversely correlated with FMD. **Conclusion:** This study demonstrated that RLS patients have poorer vascular endothelial dysfunction than normal healthy subjects. This may explain the increased risk of cardiovascular disease in the RLS patient group.

P-2-192

Ataxia with oculomotor apraxia type 1 without oculomotor apraxia: A case report

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Background & Significance: Ataxia with oculomotor apraxia type I (AOA1) is a recessively inherited ataxic disorder which is clinically characterized by childhood onset of progressive cerebellar ataxia, oculomotor apraxia and peripheral axonal sensorimotor neuropathy. Dystonia, chorea and cognitive impairment

are commonly associated symptoms and hypoalbuminemia and hypercholesterolemia are often observed. AOA1 is caused by mutations of the APTX gene. Although AOA is a common form of autosomal recessive ataxia in Japan, AOA1 has never been reported in Korea. Here, we present a 32-year-old man with progressive ataxia caused by compound heterozygous mutations of APTX gene, who did not show oculomotor apraxia but had bilateral gaze-evoked nystagmus. **Case:** We observed a 32-year-old Korean man who presented with slowly progressive gait disturbance and unsteadiness that developed at the age of 14. His ataxic gait has progressively deteriorated and he was confined to wheelchair two years ago. On physical examination, he had pes cavus, but no scoliosis or other musculoskeletal deformity. On neurological examination, he had mild cerebellar dysarthria without dysphagia. Limb motor powers were normal. Light touch, pinprick, temperature and pain sensations were normal, but vibration and positional senses were decreased on the lower limbs. Deep tendon reflexes were hyporeflexic on all the limbs. Babinski signs were absent bilaterally. He had bilateral upper and lower limb dysmetria and truncal ataxia. He was not able to stand still without assistance. Ranges of extraocular movement were full in all direction. He did not show oculomotor apraxia, but showed gaze evoked nystagmus in all directions. His mini-mental status exam score was 30. Laboratory tests were unremarkable except for hypercholesterolemia, hypoalbuminemia, and mildly elevated alpha-fetoprotein (AFP) level. Nerve conduction study and electromyography revealed sensorimotor axonal polyneuropathy. Brain magnetic resonance imaging study showed pure cerebellar atrophy without involvement of brainstem or cerebral cortex. Sanger sequencing after PCR amplification of APTX revealed compound heterozygous mutations of deletion of two nucleotides (c.359_360delAC, p.Asp120Lysfs2) in exon 3 and a missense mutation (c.617C>T, p.Pro206Leu, rs121908131) in exon 5. The mutation of c.359_360delAC is novel and c.617C>T is a known pathogenic mutation. His father was a carrier of single heterozygous mutation (c.617C>T), but we were not able to perform genetic test on his mother. **Conclusions or Comments:** To the best of our knowledge, this is the first case of AOA1 with confirmed APTX mutation in Korea. Hereditary ataxia with oculomotor apraxia is genetically heterogeneous. Currently four genes (APTX, SETX, PIK3R5, and PNKP) have been found to cause AOA syndromes (AOA1, AOA2, AOA3, and AOA4, respectively). Oculomotor apraxia can be observed in other hereditary ataxic disorders such as ataxia-telangiectasia, ataxia-telangiectasia-like disorder 1, autosomal recessive spinocerebellar ataxia 5 and 18, autosomal recessive spastic ataxia, Joubert syndrome, COACH syndrome, pyruvate dehydrogenase E2 deficiency and orofacioidigital syndrome VI. In AOA1, the most common presenting symptom is gait disturbance and abnormal eye movement or head thrust was recognized as an initial symptom in less than 10%. Although gaze-evoked nystagmus was reported to be present over 70% in AOA1, it has limited diagnostic value as GEN in all directions occurs in many neurodegenerative disorders with impaired function of the flocculus/ paraflocculus. Oculomotor apraxia was reported not to be present in 34.5% of AOA1 and may progress to external ophthalmoplegia. Given that our patient had 18-year-history of gait unsteadiness and cerebellar atrophy was documented 15 years ago, absence of oculomotor apraxia in this patient is unusual. In summary, our case indicates that AOA1 should be included in differential diagnosis of early onset ataxia in Korea, even in the absence of oculomotor apraxia.

P-2-193

Correlation between presynaptic dysfunction on F-18 FP-CIT PET and trinucleotide CAG repeat in Huntington's disease

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Background & Significance: Huntington's disease (HD) is a neurodegenerative

genetic disorder that affects muscle coordination and leads to mental decline and behavioral symptoms. HD is produced by the expression of mutant forms of the protein HTT (huntingtin) containing a pathologically expanded polyglutamine repeat. Moreover, a loss of presynaptic terminals or a reduced expression of dopamine transporter in the nigrostriatal dopaminergic system is observed in Huntington's disease. We present a correlation between the expression of dopamine transporter in the nigrostriatal dopaminergic system on F-18 FP-CIT PET and the number of trinucleotide CAG repeat in HTT gene of 2 clinically suspicious HD patients. **Case:** Two clinically suspicious HD patients admitted to the hospital. The first patient, 38-year-old women showed marked chorea, depressive mood and gait disturbance for a month. Another patient, 42-year-old women with a long history of alcoholism presented both hand tremor with dyskinesia and memory impairment for several months. In both patients, brain magnetic resonance imaging and routine laboratory examinations were normal. Finally, we suspected the patients as a Huntington's disease, F-18 FP-CIT PET, and HD gene test was followed. In the first patient who presented marked clinical symptoms, F-18 FP-CIT PET showed reduced striatal uptake, dominant in left posterior putamen. Moreover, in HTT gene test one allele had 47 abnormal CAG expansion which means full penetrance HD causing allele. The other patient with mild clinical symptoms, F-18 FP-CIT PET, showed no significant abnormalities, but one allele had 29 intermediate CAG repeats in HTT gene test. **Conclusions or Comments:** We report 2 cases of Huntington's disease showed abnormal CAG repeats in HTT gene. Of 2 patients, a patient with marked clinical symptom showed more prominent CAG repeats and reduced striatal uptake in F-18 FP-CIT PET. We presume that the number of trinucleotide CAG repeats in HTT gene is associated with the degree of presynaptic dysfunction in Huntington's disease.

P-2-194

CSF1R mutations presenting with atypical Parkinsonism

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Background & Significance: Mutations in the colony stimulating factor 1 receptor gene (CSF1R) have recently been shown to cause adult-onset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP). Clinically, most patients present with neuropsychiatric symptoms, cognitive decline, and motor impairment particularly affecting gait. We describe here the parkinsonian features in two genetically confirmed cases of ALSP. **Case:** The genetic analysis of the CSF1R gene was done in one autopsy-confirmed ALSP case and one clinically suspected case based on the clinical and neuroimaging characteristics. Both patients commonly exhibited clinical features such as personality changes (apathy, irritability) and cognitive impairment (memory decline and executive dysfunction), which were similar to those reported in the literatures. They did not have a clear family history of similar symptoms. Case 1. A 55-year-old male developed Parkinsonian features two years after the initial symptoms of cognitive impairment. He showed hypomimia, hypotonic dysarthria, bradykinesia and impaired dexterity without muscle weakness or overt spasticity, and gait disturbance characterized by broad-based, reduced stride, shuffling, decreased arm swing hesitancy on turning, and postural instability. Brain magnetic resonance image (MRI) showed widespread white matter changes and corpus callosum atrophy. Autopsy revealed generalized demyelination with frequent axonal spheroids and pigmented macrophages. A heterozygous missense mutation of c.2381T>C (p.I794T) was found in exon 18 of CSF1R gene. Case 2. A 56-year-old female initially presented with asymmetrical, left-sided, action

tremor, bradykinesia and rigidity without clear evidence of pyramidal signs. Her gait showed mildly reduced stride and dragging her left foot. Brain MRI revealed frontal dominant white matter changes more affecting right hemisphere. The basal ganglia was only minimally affected like case 1. A heterozygous mutation within splice donor site of intron 18 (c.2442+5G>A) in CSF1R gene was detected. **Conclusions or Comments:** Atypical Parkinsonism characterized by bradykinesia and gait impairment was dominant clinical feature in ALSP with CSF1R mutations. It can be an initial or asymmetric clinical presentation.

P-2-195

Dentatorubropallidolusian atrophy (DRPLA) with recurrent seizure and esotropia

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Background & Significance: Dentatorubro-pallidolusian atrophy (DRPLA) is a rare neurodegenerative disorder with various clinical phenotypes and has a cytosine-adenine-guanine (CAG) trinucleotide repeat in a gene on chromosome 12. It has been known that trinucleotide repeat disorders show strong inverse correlations between the CAG repeat number and the age of onset and genetic anticipation. DRPLA can show various symptoms like cerebellar ataxia, choreoathetoid movement, dementia, myoclonic movement and progressive myoclonus epilepsy. But DRPLA patients with esotropia and without eye paralysis are not common. We report a case of DRPLA patient who confirmed by DNA analysis and showed esotropia and recurrent seizure. **Case:** The 36-year-old male admitted because of worsening gait disturbance and recurrent seizure. Since childhood, he had frequent convulsive movement and mental retardation so he could not keep his social life and had a problem in finding job. 21 years ago, he started anti-epileptic drug by suspicion of juvenile myoclonic epilepsy (JME). But his symptoms gradually worsened and at any moment he noticed diplopia. But he could not explain diplopia description exactly due to mental retardation. His cousin (uncle's daughter) also had mental retardation when she was young and suffered with deafness. She had stayed only home in her whole life and died in 3rd decades. His another uncle has also mental retardation. In neurologic examination, we observed esotropia of right eye. Slow saccade, hypometric saccade were seen. Also in smooth pursuit test, he showed saccadic pursuit and divergence insufficiency. Motor and sensory exams were normal. In cerebellar function test, bilateral dysmetrias were seen in rapid hand alternating test and finger to nose test. We could not perform romberg test and tandem gait because of severe myoclonic movement. On brain MRI, we found diffuse cerebral, brain stem, cerebellum atrophy compare with normal group. Furthermore we checked gene test such as SCA 1, 2, 3, 6, 7, 17, Friedreich's ataxia and DRPLA by PCR amplification. We diagnosed DRPLA because the gene test showed demonstration of expanded CAG repeat 61 times. **Conclusions or Comments:** In this case, there was no structural lesion in brain MRI which can cause diplopia. In addition, the progression of ataxia with recurrent seizure means some kind of congenital disorders. Normally, eye movement disorders are mostly accompanied by SCA 1, 2, 3, 7. However, without eye paralysis, cerebellar dysfunction can cause abnormality in horizontal position. Cognitive disorders, behavioral disorders can be observed in SCA 1,2,3, 13,17,19,21, DRPLA and seizures in SCA 10, 17, and DRPLA. There are a lot of symptoms in DRPLA patients so we have difficulty in initial diagnosis. Esotropia in spinocerebellar ataxia patients is due to degenerative change of pontine and cerebellum. We can use symptom of esotropia in diagnostic clue in DRPLA patients.

P-2-196

Whole-exome sequencing identifies the first Asian patient with ovarioleukodystrophy related to AARS2 mutation

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Background & Significance: To report the novel mutations in the gene AARS2 (NC_000006.11) responsible for a ovario-leukodystrophy (adult-onset vanishing white matter disease with premature ovarian failure; OMIM #615889). **Case:** A 36-year-old woman presented with a 1-year history of progressive gait disturbance and cognitive decline. Her medical history showed premature ovarian failure. Brain magnetic resonance imaging (MRI) uncovered a diffuse leukoencephalopathy with profound central hypomyelination. Whole-exome sequencing (WES) revealed two pathologic AARS2 mutations: nonsense c.C963A (p.Y321X) and missense c.T452C (p.M151T). Both identified mutations with a frequency <0.01% in public databases predicted to be deleterious in silico, and have never been previously reported. **Conclusions or Comments:** This is the second report of ovario-leukodystrophy related to AARS mutation, and the first, to our knowledge, Asian case report of the vanishing white matter disease with premature ovarian failure due to mutations in AARS2 gene which encodes mitochondrial alanyl-tRNA synthetase.

P-2-197

Korean patients with spinocerebellar ataxia type 6 presenting with unusual manifestations

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Background & Significance: Spinocerebellar ataxia type 6 (SCA6) is a dominantly inherited form of spino-cerebellar ataxia, which usually manifests as a syndrome of progressive pure cerebellar ataxia with a late onset, and caused by a small expansion of CAG repeats in the CACNA1A gene. We describe two unrelated Korean SCA6 patients presenting with dystonia and apraxia of eyelid opening, respectively, aiming to extend the clinical heterogeneity of SCA6. **Case:** Case1 A 51-year-old man presented with a 3 year-history of progressive gait disturbance. He had complained stiffening of the right leg. Neurological examination showed gait ataxia, dysarthria, downbeat nystagmus, gaze evoked nystagmus (GEN), hypermetric horizontal saccade and saccadic pursuit. He also had right foot dystonia elicited by walking. Genetic testing was positive for SCA6. Case2 A 58-year-old woman visited our clinic with a 1 year-history of progressive gait difficulty, clumsy hand, slurring of speech, and difficulty in eye opening. Neurological examination showed apraxia of eyelid opening, horizontal GEN, dysmetric saccades and mild dysarthria. There were mild bilateral intention tremor, bradykinesia and rigidity. Gait was normal based, but arm swing and walking speed were decreased. Genetic testing was positive for SCA6. She was treated with levodopa, but the symptoms were not alleviated. **Conclusions or Comments:** SCA6 is clinically characterized by a severe form of late onset slowly progressive pure cerebellar ataxia with nystagmus. These two cases expanded the clinical phenotypes of SCA6 and emphasized the existence of dystonia, eyelid apraxia, and parkinsonism in SCA6 patients.

P-2-198

Comparison of myelin water fraction values in white matter lesions between multiple sclerosis and neuromyelitis optica spectrum disorder

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Background & Objectives: Both multiple sclerosis (MS) and neuromyelitis optica spectrum disorder (NMOSD) are inflammatory autoimmune diseases of the central nervous system, which commonly involve the white matter of the brain. We hypothesized that loss of myelin integrity of white matter lesions in MS and NMOSD would differ as much as pathophysiology of the two diseases is different. We investigated whether myelin damage of white matter lesions differs quantitatively between MS and NMOSD using myelin water imaging (MWI). **Method:** Twenty-seven MS and 20 NMOSD patients without acute relapse within 3 months prior to scans were enrolled in this study. MWIs were acquired using a 3T MRI scanner (Siemens) with the following parameters: 28 slices, voxel size: 1.5 x 1.5 x 4.0 mm³ and scan time: 14 minutes 5 seconds. As the baseline MWF value varies according to the location in the brain, we selected 6 regions including anterior corona radiata (ACR), posterior corona radiata (PCR), superior corona radiata (SCR), genu and splenium of corpus callosum (CC), and optic radiation (OR) as regions of interest (ROI). Lesions were classified as either isointense or hypointense (black holes) on T1WIs. T1 hypointense lesions were further classified mild hypointense (similar with gray matter) and strongly hypointense (similar with CSF signal) lesion. Generalized linear models adjusted for age and gender was used to compare the lesion MWF values between two groups. Multiple linear regression statistics were used to assess the association between lesion MWF and T1 signal intensities. **Results:** One hundred sixty ROIs from 27 MS patients and 79 ROIs from 20 NMOSD patients were analyzed. There was no significant difference in the current age. However, female to male ratio was higher in NMOSD (17:3 vs. 15:12) than those in MS. NMOSD patients had longer disease duration (134±65.1 vs 75.3±57.7, months) and also higher EDSS score than MS patients (4.0, range 0-8.0 vs 2.0, range 0-6.5). The mean MWF value of ACR (3.4±2.1 vs 5.4±1.9, p-value 0.001) and PCR (4.6±3.0 vs 6.6±3.6, p-value 0.008) lesions was significantly lower in MS compared to NMOSD. Multiple linear regression analysis indicated that lesion MWF of PCR and OR in both MS and NMOSD was significantly associated with T1 signal intensities, even after adjusted for age and gender. **Conclusion:** To the best of our knowledge, this is the first study comparing the lesion MWF value between MS and NMOSD. Although tissue damage of the optic nerves and spinal cord is known to be severe in NMOSD, PVWM lesions appear to exhibit more severe myelin loss in MS than in NMOSD. We also demonstrated that degree of demyelination is associated with the degree of T1 hypointensities by MWI.

P-2-199

MRI characteristics of short segment myelitis in NMO-IgG-positive neuromyelitis optica spectrum disorders

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Background & Objectives: Longitudinally extensive transverse myelitis (LETM) is the most characteristic MRI findings of spinal attacks in neuromyelitis optica spectrum disorders (NMOSD). However, recent studies suggested short segment myelitis, less than 3 vertebral segments, is not infrequent in NMOSD. The purpose of this study was to determine the frequency of short segment myelitis in NMO-IgG-seropositive NMOSD patients and evaluate the MRI features of short segment myelitis lesions. **Method:** We analyzed 116 spinal MRIs obtained within 30 days from symptom onset in 42 NMOSD patients. Longitudinal and axial extents of spinal cord lesions were investigated.

Results: LETM were observed in 89 spinal attacks, whereas short segment myelitis was observed in 27 attacks. Multifocal involvements were observed in 16 attacks. In LETM, median lesion length was 5 segments (range, 3-19). The lesions most frequently affected the upper- thoracic cord (n=49), followed by middle-thoracic (n=44), upper-cervical cord (n=37). The medulla oblongata was affected in 15 spinal attacks. The entire spinal cord was affected in 3 attacks. On the axial plane, most lesions occupied the holocord (n=38), and central cord or H-type gray matter (n=42). In short segment myelitis, median lesion length was 1 segment (range, 1-2). The upper cervical cord (n=8) was the most commonly involved, followed by middle-thoracic (n=7), upper-cervical (n=5) and lower-thoracic cord (n=5). Axially, lesions were centrally located in 14 attacks and hemicord involvement was in 9 attacks. Eight patients have short segment myelitis in the initial manifestations of spinal attacks, and 3 patients have only short segment myelitis in their disease course. **Conclusion:** Short segment myelitis was not uncommon in NMO-IgG-seropositive NMOSD, and it can be the initial manifestations of spinal attacks. Although it is very rare, some patients may show only short segment myelitis in the disease course. Therefore, NMO-IgG testing should be performed even in short myelitis lesions.

P-2-200

Anti-Ro/La antibody may be associated with time to next relapse in neuromyelitis optica spectrum disorder

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Background & Objectives: Presence of antinuclear antibody, including anti-Ro/La antibody, in patients with multiple sclerosis is associated with shorter disease duration and lower disability. The clinical role of these antibodies is still uncertain in neuromyelitis optica spectrum disorder (NMOSD). In this study, we aim to investigate the association between the presence of anti-Ro/La antibody and the time to next relapse (inter-attack duration) in NMOSD. **Method:** We reviewed consecutive 59 anti-aquaporin-4 antibody (AQP4 ab)-positive NMOSD patients between March, 2005 and June, 2014. After excluding 9 patients with interferon beta treatment and 10 patients with less than 6 months follow-up, we enrolled 40 patients. AQP4 ab statuses were determined using cell-based indirect immunofluorescence assays. All patients underwent anti-Ro/La antibody (ab) testing and were classified into two groups according to the presence of anti-Ro or anti-La antibody. The time to next relapse was compared between two groups with Cox multiple regression analyses after controlling for age, sex, disease duration, higher disability (≥ Expanded Disability Status Scale [EDSS] 6) and immunosuppressant treatment. **Results:** Fourteen (35%) patients were positive for either anti-Ro or -La ab and among them, 15 (37.5%) patients had more than one relapse until last follow-up. In anti-Ro/La ab negative group, four (4/26, 15%) patients had relapses. The median time to next relapse was statistically longer in anti-Ro/La ab-positive group compared with the anti-Ro/La ab-negative group (47 months vs. 26 months; p = 0.026). **Conclusion:** In our study, anti-Ro/La ab-positivity was associated with more benign course in patients with AQP4 ab-positive NMOSD. Further, large-scale studies will be needed to confirm this association.

P-2-201

Antibody to myelin oligodendrocyte glycoprotein in adults with inflammatory demyelinating disease of the CNS

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Background & Objectives: Several recent studies have shown the presence myelin-oligodendrocyte glycoprotein antibody (MOG-Ab) in the serum of adult patients with the NMOSD phenotype. However, the clinical relevance of MOG-Abs among adult patients with IDD is not yet clear, and there are some differences between the assays used. Here, using an improved cell based assay method a large number of sera from adults with IDD, and controls, were tested for antibodies to MOG and AQP4. To evaluate the clinical relevance of myelin-oligodendrocyte glycoprotein antibody (MOG-Ab) in a large cohort of consecutive adult patients with inflammatory demyelinating disease (IDD) of the CNS. **Method:** Live cell based assays for antibodies to MOG-Ab (IgG1 antibodies only) and aquaporin-4 antibody (AQP4-Ab) were performed in a cohort of 288 adult patients with IDD and in 72 controls. **Results:** Eighteen patients with IDD (6.3%) had MOG-Ab (MOG group) and fifty patients (17.4%) had AQP4-Ab (AQP4 group); none had both antibodies. The MOG group tended to manifest as a symptoms of isolated optic neuritis (15/18, 83%). All relapses in MOG group involved only the optic nerve within 1 year of disease onset. At onset, MRI in the MOG group uniquely demonstrated extensive peri-neural enhancement (6/18; 33%). They had fewer brain abnormalities and less periventricular lesions than the MS group, and fewer spinal involvement than the AQP4 groups. There was no female bias in the MOG group and none of them met the criteria for definite NMO. In MOG group, 3/13 monophasic patients suffered a poor visual outcome (<0.2) or paraplegia from the initial attack, while 1/5 relapsing patient suffered poor visual outcome due to repeated optic neuritis. **Conclusion:** MOG-Ab may be a disease specific biomarker in adult patients with IDD who have a disease distinct from NMO or MS. Some patients with MOG-Ab may be left with severe disability, most often after the initial attack suggesting the need for early active immune modulating or suppressing treatment.

P-2-202

Visual evoked potentials and optic coherence tomography in neuromyelitis optica spectrum disorders: which is more sensitive?

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Background & Objectives: Background: Visual evoked potentials (VEP) reflects dysfunction of visual pathway, while optic coherence tomography (OCT) is useful to detect morphological changes of retinal nerve fibers in optic neuritis (ON). The diagnostic sensitivity of each test is not known during the course of neuromyelitis optica spectrum disorders (NMOSD). Objectives: We investigated the sensitivity of OCT and VEP to detect ON in patients with NMOSD at early and later phase of attack. **Method:** Methods: Both VEP and OCT were performed in 17 patients with NMOSD. Twenty-four eyes (11 eyes with ON and 13 eyes without ON) in the acute phase (within 1 month after attack) and 23 eyes (12 eyes with chronic ON and 11 eyes without ON) in the chronic phase (at least 6 months after attacks) were examined. Demographic and clinical data including disease duration, Expanded Disability Status Scale and visual acuity were collected. **Results:** Results: In the acute phase VEPs showed abnormality in 91 % of eyes, and OCT was abnormal in only 9% of eyes. The difference in sensitivity between VEP and OCT in acute phase was not statistically significant, but it showed a tendency to be unbalanced in favor of VEPs (McNemar $p=0.065$). In the chronic phase, no sensitivity difference was found between OCT and VEPs (McNemar $p=0.125$). Average thickness of retinal nerve fiber layer (RNFL) in chronic phase was significantly thinner in

affected eyes than in unaffected eyes (48.8 ± 8.2 vs. 92.9 ± 15.3 , $p < 0.001$). For overall examined eyes, global RNFL (G-RNFL) correlated only with disease duration ($\rho = -0.426$, $p = 0.043$). **Conclusion:** Conclusions: We demonstrated that VEP was better tool to detect earlier changes of ON in NMOSD patients compared with OCT, while the sensitivity of both tests were similar in chronic ON. G-RNFL correlated well with the duration of ON, and might be a useful surrogate marker of disease burden in NMOSD. Supplementary use of VEP and OCT may enhance the diagnostic accuracy of ON during the course of NMOSD.

P-2-203

A case of neuromyelitis optica spectrum disorder presenting as area postrema syndrome with posterior reversible encephalopathy syndrome

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Background & Significance: With the discovery of anti-aquaporin-4 antibody (AQP4 Ab), several clinical and radiological features beyond optic nerve and spinal cord were described in neuromyelitis optica (NMO). Area postrema syndrome was known to be a characteristic feature of NMO, and the association between posterior reversible encephalopathy syndrome (PRES) and NMO was reported with the pathogenic role of AQP4 Ab. We report a case of NMOSD initially presented with concurrent area postrema syndrome and posterior reversible encephalopathy syndrome. **Case:** A 15-year-old girl presented with recurrent nausea, vomiting and hiccup. Symptoms persisted even with the gastrointestinal medications for about a week and then generalized tonic clonic seizure occurred. On admission to our hospital, she showed obtunded mental status, disorientation, confused speech and visual hallucination. Her vital signs were stable, but systolic BP was elevated to 145~160mmHg and diastolic BP to 70~100mmHg. She also showed dysarthria, dysphagia and four limb weakness of MRC grade IV. Analysis of cerebrospinal fluid revealed 6 WBC/mm³, marked elevated level of protein 141.8mg/dL and oligoclonal band was not observed. Electroencephalogram showed diffuse cerebral dysfunction without focal epileptiform discharge. Brain MRI showed multifocal T2 hyperintense lesions in the lower medulla to the cervicomedullary junction, peri-4th ventricular area, periaqueductal gray matter, hypothalamus and symmetric cerebral regions mainly in the parieto-occipital lobes. Bilateral symmetric cerebral lesions, especially in the parieto-occipital lobes, were compatible with PRES. After treatment with high dose steroid pulse therapy and anti-hypertensive medication, there was clinical improvement and follow-up brain MRI showed markedly decreased extent of above mentioned lesions. After 2 months later from initial presentation, she had right optic neuritis and despite the second high dose steroid pulse therapy, a week later she developed left optic neuritis. In addition, there was progressive muscle weakness in all four limbs to MRC grade II~III in upper limbs and MRC grade I in lower limbs. She also complained hypesthesia below T10 sensory level and urinary retention, so spine MRI was done. Spine MRI showed diffuse hyperintense lesion in the whole spinal cord on T2 weighted image. Tests revealed positive AQP4 Ab and she was finally diagnosed with NMO. **Conclusions or Comments:** Our case showed characteristic brain involvements at the initial clinical presentation of NMO, which were encephalopathy caused by PRES and area postrema syndrome. Brain abnormalities at onset in NMO were studied previously, which showed that initial manifestation of brain abnormalities could be classified in to two clinically distinct group, either having encephalopathy or brain-stem symptoms like intractable hiccup and vomiting. But in our case, she showed both concurrently, and her symptoms might be difficult to be considered as the initial presentation of NMO. Area postrema syndrome showed

characteristic clinical features and compatible lesions in area postrema; like the other circumventricular organs, area postrema has a leaky blood-brain barrier and express abundant aquaporin-4 water channels thus could be the preferential target for NMO. PRES includes clinical symptoms of altered mental status, confusion and visual hallucination plus radiologic findings of symmetric cerebral lesions. A substantial proportion of patients with PRES have underlying autoimmune conditions and small number of patients with NMOSD manifest as PRES. Water flux impairment due to aquaporin-4 autoimmunity may predispose to PRES in patients with NMOSD who experience blood pressure fluctuations or who are treated with therapies that can cause rapid fluid shifts. In this case, autonomic dysfunction associated with brainstem involvement may cause acute hypertension and autoimmune condition could make her vulnerable to this change in the blood pressure, presenting as PRES.

P-2-204

Diffuse large B cell lymphoma with open ring enhancement mimicking tumefactive multiple sclerosis

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Background & Significance: Tumefactive multiple sclerosis is a demyelinating disease that mimics intracranial neoplasm, infection, infarction or others on magnetic resonance imaging(MRI). The MRI is characterized by a solitary lesion, larger than 2cm with mass effect, edema and open-ring enhancement. Among these features, open-ring enhancement is considered of great value in the differential diagnosis of tumefactive multiple sclerosis. However the diagnosis of tumefactive multiple sclerosis without biopsy is no more than speculation in some cases. We report a biopsy-proven case of diffuse large B cell lymphoma with open-ring enhancement on MRI. **Case:** A 71 year old man was admitted for dizziness with mild headache and gait disturbance since last 4 weeks. He had no previous history of visual impairment, sensory change or weakness. On neurologic examination, there were all normal except truncal ataxia. Brain MRI on admission revealed about 3.4x2.3cm sized solitary ovoid heterogenous signal mass in right cerebellum with open-ring enhancement pattern that compressing brainstem and 4th ventricle. On perfusion MRI, localized decrement in cerebral blood flow and cerebral blood volume in right cerebellar white matter was noted. Cerebrospinal fluid study showed normal pressure and mild pleocytosis (WBC 7, RBC 40). Protein and IgG index were normal and oligoclonal band was negative. He had preliminary diagnosed as tumefactive multiple sclerosis and treated with intravenous pulse methylprednisolone 1,000mg for 5 days. After 3 weeks of methylprednisolone treatment, MRI revealed near complete regression. One month later, the patient revisited our hospital because of severe headache, dizziness and gait disturbance. Emergent brain MRI showed marked enlargement of the lesions. Therefore an open biopsy was performed for definite diagnosis. Histological investigation was BCL-2, BCL-6, MUM-1, CD3/20 + and pathology review was consistent with diffuse large B cell lymphoma(DLBCL). The patient was started on a regimen of cyclophosphamide, doxorubicin, vincristine and prednisolone (CHOP) plus rituximab. **Conclusions or Comments:** Open- ring enhancement may not be so specific for the diagnose of the multiple sclerosis. Early biopsy still important for suspected tumefactive multiple sclerosis patients not to delay for definite diagnosis and treatment.

P-2-205

Complete tongue paralysis as a rare presentation of seropositive

neuromyelitis optica spectrum disorder affecting dorsal medulla oblongata

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Background & Significance: Neuromyelitis optica spectrum disorder (NMOSD) is characterized by optic neuritis and myelitis. Some of the patients with NMOSD have typical brain lesions including dorsal medulla, dorsal brainstem, hypothalamus and thalamus and so on. Despite lesion involving dorsal medulla is typical for NMOSD, complete tongue paralysis has not been reported. Herein, we report a patient who showed complete tongue paralysis due to a lesion affecting dorsal medulla oblongata. **Case:** A 55-year-old woman presented to the emergency room with anorexia, dysphagia, and tongue swelling from 1 month ago. On neurological examination, her tongue could neither to be protruded nor moved from side to side. Dysarthria and gait ataxia were observed. Magnetic resonance imaging (MRI) showed T2-high and diffusion-high signal intensities on the tegmentum of medulla, dentate nucleus of both side cerebellum, and periventricular white matter of the left occipital lobe. CSF IgG index was within normal range and oligoclonal band was negative. Serologic tests for SS-A/anti-Ro and SS-B/anti-La, and anti-nuclear antibodies were positive. Tongue paralysis sustained despite steroid pulse therapy. On oropharyngeal MRI, the transverse diameter of tongue base was enlarged and there was no infiltrative or inflammatory disease on the tongue. Serum anti-aquaporin 4 (AQP-4)-IgG antibody was positive. Immune suppressive therapy with azathioprine started on a diagnosis of NMOSD with AQP4-IgG. She reported gradual improvement of tongue mobility and neurological symptoms with rehabilitation therapy. There was no recurrence for 2-year-follow up period. **Conclusions or Comments:** This is the a rare case of NMOSD presenting complete tongue paralysis and swelling due to intrinsic tongue muscle paralysis, which was caused by bilateral involvement of hypoglossal nuclei located in lower dorsal medulla oblongata.

P-2-206

Magnetic resonance imaging of trigeminal neuritis after influenza vaccination

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Background & Significance: Patients with symptoms related to the trigeminal nerve may present with a broad spectrum of clinical findings, including facial pain, either typical trigeminal neuralgia or atypical pain, numbness, paresthesias, and weakness or trismus of the masticator muscles. These patients may have lesions anywhere from the brain stem nuclei to the distal extracranial branches. The cause of lesion include various entities such as inflammation, demyelinating, vasculitis, infection, granuloma and tumor. As we experienced a case of trigeminal neuritis with right facial (V3) area numbness after influenza vaccination, we report this case with Magnetic resonance imaging and electrophysiologic studies. **Case:** The 45-year-old woman admitted with a 10-day history of sensory change in right lower lip, chin and right lateral side of tongue. She described numbness and paresthesia without skin lesion. She had a history of influenza vaccination and right upper tooth extraction 1 month ago. Her underlying disease was hypothyroidism and she took medicine regularly. Initial vital signs are blood pressure 120/90 mmHg, pulse 76 beats / min, breathing 18 times / min, temperature 36.3°C. The results of laboratory tests, including a complete blood count, basic chemistry, antinuclear antibody, anti-SS(A,B) antibody, rheumatoid factor, cytoplasmic antineutrophil cytoplasmic antibody, and perinuclear antineutrophil cytoplasmic antibody, were

all normal. In neurologic examination, mental status was alert and motor was all grade V. Sensory decrease was checked in right V3 distribution of the trigeminal nerve. Including facial motor, other cranial nerve examinations were normal. Deep tendon reflexes were normoreflexia and there was no pathologic reflex. A differential diagnosis of trigeminal neuropathy or trigeminal trophic syndrome was considered. In electrophysiologic study, there is no electrophysiological evidence of conduction defect on blink reflex test, facial nerve conduction study and jaw jerk response. (Figure 1, 2) In brain MRI, about 1.0-cm high signal intensity lesion at right middle cerebellar peduncle and entry nerve root of right trigeminal nerve was seen on FLAIR and enhanced FLAIR. (Figure 3) Considering MRI findings and the history, diagnosis of trigeminal neuritis was concluded, and hence, the patient was prescribed steroid pulse therapy for 3 days and tapered for 2 weeks. The patient reported with minimal improvement after steroid treatment. In follow MRI, near complete resolution of right trigeminal nerve entry zone lesion was seen. (Figure 4)

Conclusions or Comments: The trigeminal (gasserian) ganglion is situated near the apex of the petrous bone in the middle cranial fossa and project to the principal sensory nucleus of the trigeminal nerve and to the spinal nucleus of the trigeminal nerve. The principle sensory nucleus is located in lateral pons, posterolateral to the motor nucleus of the trigeminal nerve. Lateral pontine tegmental lesion may present as an isolated trigeminal sensory neuropathy, with numbness and paresthesia of face due to the involvement of the principle sensory nucleus of the trigeminal nerve. In this case, Magnetic resonance imaging showed signal changes of lateral pontine tegmental lesion and pre-ganglionic trigeminal nerve roots. Considering patient's symptom, this finding of this MRI is radiologically significant in that it clearly shows the principle sensory nucleus.

P-2-207

Spinal dural arteriovenous fistula as a potential mimic of transverse myelitis

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Background & Significance: Spinal dural arteriovenous fistulae comprise the majority of spinal vascular malformations. The most common clinical presentation is that of progressive myeloradiculopathy, probably related to venous hypertension. **Case:** The patient is a 52-year-old female, who first noted tingling sensation in her Rt.leg. 3 month later, she developed progressive weakness in both leg motor weakness. MRI of the thoracic spinal cord demonstrated increased signal of central portion of spinal cord at T5-T12 on T2 weighted image. Initially, the patient was diagnosed with transverse myelitis. However, unlike most case of transverse myelitis, several features suggestive of venous hypertensive myelopathy **Conclusions or Comments:** The history, neurological findings and radiological changes on MRI scan should alert clinicians to the possibility of spinal dural arteriovenous fistula. Early diagnosis and treatment may significantly improve outcome and prevent permanent disability

P-2-208

A case of acute combined central and peripheral demyelination

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Background & Significance: Combined central and peripheral demyelination is a rare disease entity. Demyelination can affect the central and peripheral

nervous system simultaneously. **Case:** We describe a 31-year-old woman who presented with gait disturbance, dysarthria and vertigo. She had complained vertigo from 1 month ago. Gait disturbance and dysarthria started after 2 weeks from vertigo symptom. In the past history, she had optic neuritis about 9 years ago. She had abnormalities of demyelinating disease in the electrophysiologic study and magnetic resonance imaging of the brain and cervical spine. She responded to treatment with intravenous immunoglobulin and corticosteroids. **Conclusions or Comments:** Our case illustrates that acute combined central and peripheral demyelination may be considered in demyelinating disease with various symptoms

P-2-209

Reversible corpus callosal lesions associated with a use of Adalimumab in patient with ulcerative colitis

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Background & Significance: Reversible corpus callosal lesion is a unique phenomenon aroused curiosity to many researchers although its exact pathophysiology is still not exactly understood. Various etiologies have been suggested for reversible lesion in corpus callosum, including toxins, encephalitis, influenza A and several metabolic disorders. We herein report a patient showing reversible corpus callosal lesions who has been treating for Ulcerative colitis (UC) with adalimumab (ADA). **Case:** A 43-year old man, who was diagnosed UC 15 year ago, admitted via emergency department for acute inflammatory lesion in his left inguinal area accompanied with acute dysarthria. On May 14th, 2015, he started ADA therapy for UC on a doctor's recommendation. However, he stopped the ADA after last injection on May 27th due to adverse effects as tingling sensation and insomnia. On July 11th, 16 days after last ADA administration and 28 days after first administration, he had pain, swelling and redness on his left inguinal area and dysarthria with mild confusion. The Brain diffusion weighted image showed focal, well defined diffusion restriction lesion at corpus callosal body and splenium. Thorough neurologic exam revealed no other symptoms related to corpus callosum lesion including interhemispheric disconnection syndrome. Symptoms in left inguinal area and dysarthric speech were dramatically improved 5 days after admission with treatment of empirical antibiotics and other supportive managements without any immune modulating therapies. Follow up brain MRI after 8 days of symptom onset revealed significantly improved lesion **Conclusions or Comments:** ADA is a human monoclonal immunoglobulin G1 antibody to TNF- α which may cause various kinds of acute and delayed adverse effects. However, it is the first report of the possible relationship of reversible corpus callosal lesion with ADA. We suggest that the reversible corpus callosum lesion is related with ADA therapy due to following reasons. A hypothesis of pathomechanism for reversible corpus callosum lesion is exocytotoxic edema due to toxic effects by various causes. There are some reports of demyelinating brain lesions and posterior reversible encephalopathy syndrome (PRES) which was associated with the use of TNF- α antibodies such as infliximab or etanercept. These findings might share similar pathomechanisms with our case. Delayed type hypersensitivity may occur up to 2 to 3 weeks after exposure, however, the longest interval from exposure to symptom onset is not clear yet. In the previous studies, ADA antibodies could be detected 4 weeks after even single dose infusion of nadalimumab. This finding can be a clue to our hypothesis of late ADA hypersensitivity reaction. Our patient's inguinal cellulitis may be another clue because cutaneous inflammation and infection is one of the most common adverse effects of TNF- α antibodies. Our report highlights that the ADA therapy may cause delayed adverse effects and the reversible corpus callosal lesion can be a one of the manifestations.

P-2-210

Listeria monocytogenes central nervous system infection mimicking Neuromyelitis optica spectrum disorder

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Background & Significance: The *Listeria monocytogenes* has a particular tropism for the central nervous system and can produce infection in the meninges and brain substance. We report a case of listerial rhombencephalitis mimicking NMOSD features. **Case:** A 54 year-old man was brought to our emergency room with a sudden onset of quadriplegia 24hrs prior to the arrival. He was working in the livestock and agriculture industry. He had been diagnosed as Henoch-Schonlein nephritis 3 month ago, and taking 60mg of oral prednisolone with a medication for hypertension and diabetes daily. The neurologic examination revealed alert consciousness, but weakness in bilateral upper and lower extremities of MRC grade 2~3. Pain and temperature sense was decreased below T2 dermatome and also vibration sense was decreased below T10 level with hyperreflexia and bowel & bladder dysfunction. The blood test disclosed normal except showing mild leukocytosis and anemia. The CSF exam showed a normal range of ICP, protein, glucose level with leukocytosis. T2 weighted spinal MRI showed edematous change with a high signal intensity from medulla to C5 level, also showed a high signal intensity in gadolinium enhanced MRI from medulla to C2 level. No gross abnormal lesion was seen in the brain MRI. These findings suggested the diagnosis of NMOSD (neuromyelitis optica spectrum disorder) and high dose intravenous methylprednisolone was initiated. On the 3rd day of admission, he had a high fever and a follow-up csf examination showed increased ICP of 350mmH₂O, and increased leukocytosis, protein, decreased level of glucose compared to the initial exam. Considering the bacterial meningitis at the age over 50, vancomycin, ceftriaxone and ampicillin was added. On the 9th day, listeria monocytogenes infection was identified from both CSF and serum culture. Through the negative result of serum aquaporin-4 antibody, a diagnosis of listerial rhombencephalitis was confirmed. Unfortunately, he did not respond to the treatment and died on the 24th day of admission. **Conclusions or Comments:** This case suggests that listerial rhombencephalitis should be considered as one of the causes of CNS infection especially in the old age and the immunocompromised patients if there are abnormal lesions in the brainstem or spinal cord although the typical clinical features suspicious of encephalitis is not presented.

P-2-211

Unilateral ptosis with partial third nerve paresis in midbrain infarction

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Background & Significance: Warwick demonstrated that the oculomotor nucleus in monkeys is arranged as discrete subnuclei with the central caudal nucleus providing a common output to both levator palpebrae muscles. Lesions involving the central caudal nucleus, which supplies both levator muscles, are well known to cause bilateral ptosis. Herein, we present a patient with unilateral ptosis and partial oculomotor palsy resulting from a midbrain infarct that may involve the oculomotor nucleus. **Case:** A 40-year-old woman visited the neurology department with a chief complaint of binocular diplopia with ptosis on the left eye starting two days prior. The patient reported that the initial symptoms occurred abruptly with mild headache and nausea, and the ocular symptom persisted for several hours after the onset. She had a history of central retinal artery occlusion on the left side two years prior, treated with ante-

rior chamber paracentesis. Her systolic blood pressure ranged between 120 and 140, and no febrile sense, chilling and fever were noted. Her electrolytes, renal function, and complete blood count were within normal ranges. The patient exhibited moderate-degree left eyelid ptosis with medial gaze limitation without contralateral gaze palsy, eyelid, and pupillary involvement (Fig. 1A). No other neurologic signs were abnormal. Magnetic resonance imaging (MRI) of the patient's brain was performed at her admission. Focal diffusion restriction in left dorsomedial portion of midbrain on superior colliculus level was seen in the diffusion restriction image. No vascular abnormality or other parenchymal lesion was observed (Fig. 1B). The diagnosis was an acute midbrain infarction. Antiplatelet agent infusion was initiated immediately. Two days later, ptosis and medial gaze limitation were partially resolved. **Conclusions or Comments:** Although our patient had evidence of a nuclear lesion on MRI, her only symptoms were unilateral ptosis and palsy of the ipsilateral medial rectus muscle. The oculomotor nucleus complex is located near the midline in the midbrain at the level of the superior colliculus. A central caudal nucleus provides bilateral innervation to the levator palpebrae muscles while its four paired subnuclei of the oculomotor complex innervate different ipsilateral extra-ocular muscles, except the most medial portion, which innervates the contralateral superior rectus muscle. Because the common central nucleus contributes bilaterally, a nucleus lesion, either unilateral or bilateral, can result in paresis of both levator palpebral muscles and bilateral ptosis. This patient presented with unilateral ptosis and medial gaze limitation of her left eye, and MRI showed diffusion restriction of the ventromedial portion of midbrain where the third nerve nuclear complex lies. A possible explanation is that the lesion was located at the junction where fascicles exit the oculomotor nucleus complex rather than the central caudal nucleus. Because the extremely proximal parts of fascicles are involved, the MRI lesion displays as if the nucleus is involved. The fascicles are arranged in topographic manner and the fascicles for medial rectus and levator palpebrae are located adjacently on medial portion of organization, therefore, the patient presented only two deficits simultaneously (Fig. 2). In conclusion, a nuclear lesion of midbrain can cause unilateral ptosis. A lesion of junction or very proximal part of fascicles from oculomotor nucleus can explain such phenomena. Physicians should consider a nuclear lesion of the midbrain or an adjacent structure of the nucleus when a patient presents with unilateral ptosis.

P-2-212

A case of multiple orbital myositis accompanied by herpes zoster ophthalmicus

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Background & Significance: Herpes zoster ophthalmicus (HZO) may cause a variety of ocular symptoms including: dermatitis, conjunctivitis, uveitis, scleritis, keratitis, neuralgia and cranial nerve palsies. In patients with HZO, limitation of ocular movement is generally interpreted as diseases of III, IV or VI cranial nerves. However, external ocular muscle palsies due to orbital myositis has been rarely diagnosed. We report a patient with multiple orbital myositis on both eyes accompanied by unilateral HZO. **Case:** A 71-year-old woman complained of diplopia over last 4 days. She had been diagnosed with left sided herpes zoster ophthalmicus two weeks prior to the visit and had taken oral Acyclovir. However, her physical examination demonstrated still left sided periocular edema and grouped vesicles on erythematous skin lesions with distribution of ophthalmic division of the trigeminal nerve. On neurologic examination, she showed left sided abduction deficit. Her brain MRI showed swelling and enhancement in the bellies of bilateral external ocular muscles. She was diagnosed with multiple orbital myositis on both eyes accompanied by left sided HZO. She was treated with methylprednisolone in-

jection (1g/day for 5 days) and intravenous Acyclovir (1g/day for 10days). During treatment, abduction deficit disappeared. **Conclusions or Comments:** This case highlighted that acute orbital myositis should be considered in cases with ocular limitation accompanied by HZO. It showed rapid response after treatment with intravenous steroid and acyclovir.

P-2-213

Unilateral hemorrhage restricted to the middle cerebellar peduncle

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Background & Objectives: The middle cerebellar peduncle (MCP) is a large nerve fiber bundle formed by the fibers projecting to the cerebellum. Despite the MCP serves a main passage for the visual and ocular motor inputs to the cerebellum, abnormal ocular motor findings have not been clarified in hemorrhages restricted to the MCP. **Method:** Five patients with acute hemorrhage from a cavernous hemangioma restricted to unilateral MCP had a recording of eye movements using three-dimensional video-oculography within seven days from the symptom onset. The diagnosis was based on the axially oriented high-resolution MRI scans. **Results:** Vertigo was the main presenting symptom in all patients. Other findings included mild dysarthria (n=4), ipsilesional sensory change in the face (n=3), mild ataxia of the ipsilesional upper limb (n=3), veering to the lesion side on Romberg testing with the feet apposed (n=3), and transient ipsilesional hearing difficulty or ear fullness without evidence of hearing loss on audiometry (n=2). None had facial or limb weakness. All patients showed spontaneous torsional nystagmus, contraversive in three and ipsiversive in two. One patient showed hemiseesaw nystagmus. Horizontal or vertical gaze did not affect the direction of torsional nystagmus. Skew deviation or ocular torsion was also noted in all patients, ipsiversive in three and contraversive in two. The OTR was in the opposite direction of spontaneous torsional nystagmus. Horizontal smooth pursuit was impaired in all patients, only ipsilesionally in two and in both directions but more to the lesion side in the remaining three. Two of the three patients with VOG recording of vertical pursuit showed accentuated torsional eye movements during downward smooth pursuit. All five patients showed gaze-evoked nystagmus, and, of interest, the vertical components were disconjugate between the eyes in three of them. One patient showed centripetal and downbeat nystagmus during sustained contralateral gaze, followed by transient reversal of the nystagmus directions on resuming the straight-ahead gaze. Another two patients showed rebound nystagmus. Recording of head impulse tests documented decreased gains of the horizontal and posterior canals on both sides, but without canal paresis on bithermal caloric tests. However, bedside head impulse tests were normal in the remaining four patients. **Conclusion:** Patients with unilateral MCP hemorrhage show a distinct pattern of eye movement abnormalities that include spontaneous torsional nystagmus along with an OTR, horizontal GEN with disconjugate vertical components, impaired horizontal pursuit more to the lesion side, and accentuated torsional movements during vertical pursuit. The inappropriate torsional nystagmus during vertical pursuit may be due to failure of symmetric cancellation of torsional signals from the vertical canals on both sides.

P-2-214

Isolated anterior cerebellar vermian infarction

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Background & Significance: The anterior cerebellar vermis (the lingula, central

lobule, and culmen) is mainly involved in coordination of the half-automatic movements of walking and postural adjustment of the lower limbs in the gravity. In humans, however, the clinical features have not been described in isolated anterior cerebellar vermian infarction. **Case:** A 59-year-old man with hypertension, diabetes mellitus, and coronary artery disease suddenly developed non-rotatory dizziness and unsteadiness when walking up the stairs. He also reported heaviness in bilateral lower extremities and unsteadiness during stance and gait. He denied nausea, vomiting, headache, diplopia, dysarthria, or other neurological symptoms. One day from symptom onset, the patient showed no head tilt, skew deviation on alternating cover test, or spontaneous nystagmus with and without visual fixation using Frenzel goggles. He had no gaze-evoked nystagmus. Smooth pursuit tracking sinusoidal target motion at a velocity of 10°/s was impaired to the left. Horizontal and vertical saccades were normal. Head turning to either side while lying down produced small apogeotropic nystagmus (slow phase velocity=3.5°/s), which lasted for about 10 seconds without positional vertigo. Bedside head impulse tests were normal. He showed increased postural sway from side to side on Romberg testing with the feet apposed. The neurological examination was otherwise normal including proprioception, past pointing, and muscle tone. Bithermal caloric tests were normal. The video head impulse test documented normal gains for horizontal and vertical canals in the both sides. He showed normal gains and phases of the vestibulo-ocular reflex during sinusoidal harmonic acceleration and normal time constants of the pre- and postrotatory nystagmus during step velocity rotations. Tilt suppression of the postrotatory nystagmus was normal. Ocular torsions assessed by fundus photographs showed abnormal extorsion of the right eye (15.1°, normal range 0 to 12.6°) but normal of the left eye (9.1°). The patient showed no tilt of the subjective visual vertical. Cervical and ocular vestibular evoked myogenic potential tests were normal. Diffusion-weighted MRI scans showed a focal infarction restricted to the right lingula and central lobule. Cerebral angiography disclosed no steno-occlusive lesion. **Conclusions or Comments:** Unilateral anterior vermian infarction may present acute spontaneous dizziness with imbalance. The apogeotropic positional nystagmus and impaired smooth pursuit indicate a participation of this area in the control of ocular motion.

P-2-215

Recurrent nonpupil-sparing isolated complete third nerve palsy without compressive neuropathy

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Background & Significance: A third cranial nerve palsy can result from lesions between the oculomotor nucleus in the midbrain and the extraocular muscles within the orbit. This is classified to neurologically isolated or non-isolated according to the presence of other neurologic deficits. It is further characterized isolated lesions as having normal internal function (pupil-sparing) or abnormal internal function (nonpupil-sparing). A complete oculomotor palsy includes external (motility of the globe) and internal dysfunction. The pupillo-motor fibers lie in the outer portion of the nerve, directly beneath the epineurium, and are vulnerable to compression by trauma, tumors, or aneurysm. **Case:** A 45-year-old male presented with a sudden onset of a drooping left eyelid and binocular diplopia for 12 days. He experienced same symptom 17 years ago and improved spontaneously. On examination, the left pupil was fixed and dilated, and there as complete left ptosis. The patients could not elevate, depress, or adduct the left eye. The left pupil measured 7mm in diameter and unreactive to light. MRI and MR angiography of the brain revealed high signal intensity distributed along the left third nerve. Intracerebral aneurysm, tumor and hemorrhage were not observed. Lyme disease, herpes zoster in-

fection, multiple sclerosis and sarcoidosis were excluded. After administration of high-dose steroid, neurological deficits improved spontaneously. **Conclusions or Comments:** We report a rare case of recurrent nonpupil-sparing isolated complete third nerve palsy without compressive neuropathy.

P-2-216

Wall-eyed bilateral internuclear ophthalmoplegia in unilateral midbrain infarction

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Background & Significance: Internuclear ophthalmoplegia (INO) is caused by lesion in the medial longitudinal fasciculus (MLF) and clinically this syndrome is characterized by adduction weakness on the side of lesion and monocular nystagmus of the abducting eye. Convergence is intact in the most of INO because the vergence pathway is spared. Lesion affects the MLF within the upper midbrain, vergence pathways and the oculomotor apparatus can be disrupted. Wall-eyed bilateral INO (WEBINO) syndrome is associated with bilateral MLF lesion of midbrain, it has been mostly reported in patients with multiple sclerosis and brainstem stroke. **Case:** A 62-years-old male with diabetes came to our clinic with complaints of 5 days of binocular diplopia. Neurologic examination reveals exotropia of both eyes and bilateral internuclear ophthalmoplegia. Convergence was absent. Brain MRI demonstrated a small acute infarction in the right midbrain on fluid attenuation inversion recovery and diffusion weighted images. **Conclusions or Comments:** We report a rare case of WEBINO in unilateral midbrain infarction.

P-2-217

A case of ophthalmoplegia and blepharoptosis mimicking incomplete 3rd nerve palsy following injection of filler into the nasal bridge

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Background & Significance: To report a case of sudden external ophthalmoplegia mimicking incomplete 3rd nerve palsy following injection of filler into the nasal bridge **Case:** A 26-year-old woman was referred for sudden double vision and blepharoptosis. The patient was injected with facial filler into the nasal bridge 2 hours ago. The patient complained for headache and dizziness. In examination, visual acuity was normal, but incomplete ptosis and external ophthalmoplegia combined with medial gaze and downward gaze palsy were showed on the left eye. The patient had developed skin necrosis and a surrounding reddish reticular pattern on her face around the nasal bridge, glabella and forehead. Other neurological examination was normal. Brain MRI showed no acute ischemic lesions of parenchyma. Orbit MR showed enhanced lesions at medial rectus and inferior rectus muscle in left eye. **Conclusions or Comments:** Following previous reports, retrograde embolization to the ophthalmic artery from fillers injected to the glabellar or the nasolabial area was caused blindness and total ophthalmoplegia, and rarely cerebral infarction. We thought that selected ophthalmoplegia without visual loss after filler injection was mimicked incomplete 3rd nerve palsy and caused by retrograde embolization into the muscular branches of ophthalmic artery from facial anastomoses.

P-2-218

Clinical characteristics and prognosis of patients presenting with binocular diplopia

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Background & Objectives: Binocular diplopia is the double vision related to a misalignment of the eyes, which is usually the result of impaired function of the extraocular muscles. Many causes of binocular diplopia have been documented including CNS lesion, ocular motor nerve palsy, neuromuscular junction disease and muscle disease. In clinical practice, ocular motor nerve palsies are commonly encountered and often diagnosed with "microvascular cranial nerve palsy (MCNP)". Vascular risk factors such as old age, hypertension, diabetes mellitus (DM), hypercholesterolemia and smoking are possibly associated with MCNP. Until recently, there have been few studies describing characteristics and prognosis of MCNP in Korean patients. The aim of this study is to identify the etiologies of binocular diplopia and evaluate the clinical characteristics, underlying vascular risk factors and long-term prognosis of MCNP by retrospective chart review. **Method:** Medical records between January 2005 and December 2010 were reviewed and we included 189 patients who presented with double vision and diagnosed with binocular diplopia by neurology specialists. Among them, 56 patients who did not perform any brain imaging study or did not visit for follow-up were excluded. The collected data included patient demographics, clinical characteristics including neurological deficits, underlying etiology, past medical history, investigation methods and its results, and clinical progression of 133 patients. In view of etiology, we classified patients as having MCNP when there was no any identifiable cause of binocular diplopia after investigation. The frequency of newly diagnosis of DM was investigated among 93 patients who had no DM at the onset of diplopia and visited for follow up over 6 years. **Results:** A total of 133 patients (79 men (59%) and 55 women (41%)) was included. Mean age of onset was 53.0 ± 13.9 years and mean duration of follow-up was 25.1 ± 34.7 months (1 to 125 months). MCNP (96 patients, 72.2%) was the most common cause of binocular diplopia. There were nine patients with acute cerebral infarction (3 in pons, 2 in midbrain, 2 in cerebellum, 2 in others), nine with intracranial tumor (7 with primary brain tumor, 2 with metastatic cancer), four with intracranial hemorrhage, three with multiple sclerosis, two with meningitis and each one of intracranial aneurysm (ACA) and cavernous AVF. Among 96 patients with MCNP, 53 patients showed isolated ocular motor nerve palsy as oculomotor nerve palsy in 20 patients (37.7%), trochlear nerve palsy in 15 (28.3%) and abducence nerve palsy in 18 patients (34.0%). 75 patients (78.4%) had known vascular risk factors as hypertension in 32 patients, diabetes mellitus in 34 patients, and current smoking in 21 patients. 21 (21.6%) patients had no vascular risk factor at all. Steroid was administered in 42 patients and antiplatelet or anticoagulant were given in 32 patients. In 21 cases, conservative medicine including vitamin B or herb extracts like ginkgo was used. Among 111 patients who had the medical records of the progress, 64 patients (68%) showed good prognosis and mean duration for recovery was 22.5 ± 40.1 days (range, 0-222). Among initially non-diabetic patients, 2 patients were newly diagnosed as diabetes mellitus after each of 71 and 82 months since diplopia. One patient had with diplopia as first presentation and was diagnosed as diabetes at the same time. **Conclusion:** MCNP is the most common cause of binocular diplopia and the frequency is higher compared to the previous report from ophthalmology clinic. It might be due to that patients with isolated ocular symptom or obvious etiology such as trauma mostly visit ophthalmology clinic and be excluded in this study. Established vascular risk factors are also associated with MCNP and the prognosis of these patients was relatively good regardless of the various treatment.

P-2-219

Vertical diplopia with unilateral ptosis as an initial manifestation of pleomorphic adenoma of the in a lacrimal gland

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Background & Significance: Pleomorphic adenoma is the most common benign epithelial neoplasm of the lacrimal gland. It commonly presents as an insidiously growing and painless mass, and like other orbital masses, may also cause exophthalmos. To our knowledge, it is very rare for lacrimal gland pleomorphic adenoma to cause ptosis and diplopia as initial symptoms without proptosis. Here we describe a case of lacrimal gland pleomorphic adenoma with unusual clinical manifestations. **Case:** A 72-year-old man visited our hospital complaining gradually aggravating binocular diplopia for six months. The symptoms were persistent, and did not improved even in the morning or after resting. He did not have any underlying diseases, and underwent cataract surgery in both eyes. He presented with ptosis and hypotropia in the right eye, but there were no significant exophthalmos, subconjunctival hemorrhage, and periorbital ecchymosis. Neurologic examination revealed upgaze limitation of the right eye and diplopia was aggravated on upgaze. The patient's pupils were round, equal in size, and reactive to light. Repetitive nerve stimulation test was performed, and there was no progressive declining response of compound muscle action potentials. The laboratory tests including plasma levels of acetylcholine receptor antibody and thyroid hormones were normal. Brain MRI revealed a well-defined enhancing mass with internal cystic components in the right lacrimal gland. The patient was finally diagnosed with pleomorphic adenoma in the right lacrimal gland, and surgical treatment was considered. **Conclusions or Comments:** We report a case of lacrimal gland pleomorphic adenoma presenting with slowly progressive ptosis and diplopia. In our patient, compression the superior rectus muscle and the levator palpebrae muscle might be a considerable cause of vertical diplopia and ptosis. Long-term painless exophthalmos is common symptom of lacrimal gland pleomorphic adenoma. However, even if proptosis is not present, lacrimal gland pleomorphic adenoma should be considered in patients with slowly progressive ptosis and diplopia.

P-2-220

A case of midbrain infarction causing isolated fourth nerve palsy

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Background & Significance: Trochlear nerve innervates the superior oblique muscle, which adducts and intorts the eye. Trochlear nerve palsy affects eye to be hypertropic and extorted, resulting in vertical diplopia. Traumatic, congenital, and microvascular disorders are the most common etiologies of trochlear palsy. Trochlear palsy mostly accompanies other neurological deficits such as Horner's syndrome, sensory change, internuclear ophthalmoplegia, nystagmus and ataxia. However, isolated trochlear palsy due to midbrain infarction is uncommon. Few cases of isolated trochlear nerve palsy with midbrain infarction has been reported. We report a patient with isolated trochlear nerve palsy due to midbrain infarction. **Case:** A 78-year-old woman complained of sudden vertical diplopia which suddenly developed 1 day ago. She was taking medication for hypertension and type 2 diabetes for 7 years. Examination revealed left eso-deviation and left hypertropia at left eye in the neutral position. Vertical diplopia aggravated in downward and right gaze and on left head tilting. She has no history of head trauma. Laboratory evaluations were all normal. Brain diffusion weighted image showed a small infarction restricted to right trochlear nucleus. And magnetic resonance angiography showed vascular irregularity at both vertebral arteries. She was started an-

ti-platelet therapy. And appropriate fluid therapy was applied. And then, she take absolute bed rest. After 5 days later, she has no diplopia and her symptom has been completely resolved. And she discharged with medication. **Conclusions or Comments:** The isolated trochlear nerve palsy can be developed in various conditions. Most common causes of isolated trochlear nerve palsy are trauma and vascular diseases. Also, birth trauma, tumors, aneurysms, connective tissue diseases and infections can be presenting as isolated trochlear nerve palsy. In case of midbrain infarction, there are many anatomic structure in midbrain such as spinothalamic tracts, medial longitudinal fasciculus, descending sympathetic tract and superior cerebellum. So, midbrain infarction usually presents as gaze paresis, pure motor symptom, limb ataxia, hypesthesia, and Horner's syndrome. However, midbrain stroke presenting as isolated trochlear palsy is rare. In this case, we suggest that isolated trochlear palsy with no other neurological deficits can be focal midbrain infarction.

P-2-221

Idiopathic hypertrophic cranial pachymeningitis presenting as Tolosa-Hunt syndrome

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Background & Significance: Idiopathic hypertrophic cranial pachymeningitis (IHCP) is a rare condition, which is a fibrosing inflammatory process that involves the dura mater. Tolosa-Hunt syndrome often represent a focal manifestation of IHCP. So neuroimaging such as brain MRI is needed in patients suspected for Tolosa-Hunt syndrome for detection of IHCP. We reported a case of IHCP presenting as Tolosa-Hunt syndrome. **Case:** A 29 years old male visited our emergency room (ER) with headache in left parietal area. The first onset of headache was 2 weeks before visiting ER. Its nature was compressive and accompanied with left eyeball pain, but no visual symptoms. Neurological examination revealed nonspecific findings. His vital signs showed; blood pressure 110/70 mmHg, body temperature 36.5°C, and Blood glucose level 94mg/dL. There was no history of diabetes, hypertension or other specific medication. Routine evaluation for systemic illness, Brain CT, MRI and CSF study was done for evaluation of secondary headache and nonspecific findings were noted, so he was discharged with some analgesic drugs and started to visit our outpatient department (OPD) regularly. On regular observation in OPD, his headache did not show meaningful improvement in severity and frequency. But any neurological signs suggesting for other specific disease was not visible, so conservative treatment such as analgesic drugs was continued in according to primary headache. 1 month after the first visit to our hospital, sudden ptosis and gaze palsy in left eye were noted, so he complained diplopia, periorbital pain, left facial and hemispheric pain. Neurologic examination revealed left third and fourth nerve palsy. In clinical diagnosis of Tolosa hunt syndrome, he was hospitalized and retried laboratory study, brain imaging, blink reflex test and CSF study. Lab findings including HIV test, VDRL, Tuberculin test, auto-immune markers, thyroid function test and tumor markers were normal. CSF study showed mild increased WBC counts (30/ μ g, lymphocytes-dominant) and otherwise nonspecific finding, which suggests mild inflammatory condition. Brain MRI revealed mildly bulging small soft tissue mass lesion in left cavernous sinus and superior orbital fissure. In addition, diffuse left tentorial thickening and enhancement were also noted. Blink reflex test revealed left trigeminal nerve lesion, suggesting left trigeminal neuralgia. A confirmative diagnosis of IHCP was made, he was started on steroid therapy. 3weeks after initiation of steroid therapy, his ptosis and extra-ocular movement was mildly improved, but headache was intermittently repeated. Now we are on observation for his neurologic symptoms regularly in our OPD with gradually tapering oral prednisolone. **Conclusions or Comments:** IHCP is diffuse inflammatory disease which causes thickening of the dura mater and often pres-

ent as Tolosa-Hunt syndrome. We suggest that in a patient with clinical diagnosis of Tolosa-Hunt syndrome, neuroimaging study is absolutely needed and the relationship between Tolosa-Hunt syndrome and IHCP should be discussed and studied within the framework of etiology.

P-2-222

A case of spontaneously resolved isolated oculomotor nerve palsy due to cavernous sinus fistula

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Background & Significance: A carotid-cavernous sinus fistula (CCF) is an abnormal communication between the cavernous sinus and the carotid arterial system. CCF usually presents with conjunctival injection, proptosis, loss of visual acuity and ophthalmoplegia. There have been some CCF case reports presenting with isolated oculomotor, abducens and trochlear nerve palsy. We report a case of spontaneously resolved isolated oculomotor nerve palsy due to CCF. **Case:** A 61-year-old woman admitted with complaint of headache and ptosis. Before one day, she had vascular type right temporal headache, retro-orbital and periorbital pain, and ptosis. The patient had no past medical/surgical history and recent trauma/injury history. Physical & neurologic examination revealed ptosis, limitation of medial, upward and downward gaze in right eyeball without unisocoria, which indicate oculomotor nerve palsy. Brain MRI showed no remarkable change. MR angiography showed dilated bilateral superior ophthalmic veins and cavernous sinus, more severe in right side. We diagnosed CCF, and decided emergent cerebral angiography and embolization. However, her symptoms and signs were spontaneously resolved within a few hours after admission. We underwent only angiography which found right CCF with multiple feeders from bilateral ICA & ECA cavernous dural branches. She discharged without other procedure due to disappearance of symptoms and signs without recurrence. **Conclusions or Comments:** Spontaneously resolved isolated oculomotor nerve palsy with CCF were not yet reported. When ocular symptoms and signs are transient, the diagnosis of CCF may be overlooked easily. Therefore, although ocular symptoms and signs are transient, these could be caused by CCF and then neuroimaging study was recommended immediately to rule out CCF.

P-2-223

Recurrent acute isolated third cranial nerve palsy in the anti-GQ1b antibody syndrome

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Background & Significance: Anti-GQ1b IgG antibody is associated are strongly associated with Miller-Fisher Syndrome, acute ophthalmoplegia in Guillain-Barre´ syndrome, Bickerstaff's brain stem encephalitis, and with isolated ophthalmoplegia. We report an exceptional case of recurrent isolated third cranial nerve palsy with pupillary involvement without areflexia or ataxia. We report a case of recurrent isolated third cranial nerve palsy with pupillary involvement without areflexia or ataxia which presented a seropositivity of antibody of anti-GQ1b IgG. **Case:** Previously healthy 23-year-old man presented left ptosis and diplopia. His left eye extraocular movement was paralyzed at adduction, supraduction & infraduction, but spared at abduction, intorsion during down-ward gaze, suggestive of left third cranial nerve palsy. In 5 years ago & 2 years ago, he had suffered twice monocular ptosis and diplopia, suggestive of isolated third nerve palsy. He showed normoactive and symmetric tendon reflexes. Nerve conduction studies were within normal range

and CSF finding was non-specific except mildly increase protein (protein 50.8mg/dL). He showed normal thyroid function test, and normal repetitive nerve stimulation test. The brain MR imaging did not show abnormality. The anti GQ1b IgG antibody test was positive at CSF & blood. Left ptosis and diplopia were partial recovery after steroid therapy. **Conclusions or Comments:** Only few patients were been in which different anti-GQ1b IgG antibody syndromes have recurred at different times in a single patient, had presented 1)ophthalmoplegia, ataxia, and areflexia, 2)internal ophthalmoplegia and 3)complete ophthalmoplegia, ataxia, marked drowsiness, respiratory paralysis . The present finding of recurrent unilateral ptosis and restriction of extraocular movement with anti-GQ1b IgG antibody helps confirm the existence of the syndrome. Detection of anti-GQ1b antibody is important in the differential diagnosis of ophthalmoplegia, especially at the recurrent isolated cranial nerve palsy.

P-2-224

Optic chiasm involvement in ethambutol-induced optic neuropathy

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Background & Significance: A few cases of bitemporal hemianopsia suggesting optic chiasm involvement associated with ethambutol toxicity have been previously reported. However, only one case has demonstrated optic chiasmal lesion in neuroimage. We report a patient with ethambutol-induced optic neuropathy who revealed optic chiasm involvement using high-resolution MRI. **Case:** A 52-year-old woman was admitted to our hospital for progressive blurred vision of both eyes that began 1 month ago. She had taken rifampin, clarithromycin, and ethambutol for last 8 months after diagnosis of pulmonary nontuberculous mycobacteria infection. She didn't have certain symptoms suggesting optic neuritis such as pain with ocular movement or dyschromatopsia. On admission, visual acuity was 20/2000 in the right eye and counting finger at 10 cm in the left eye. Automated perimetry showed bitemporal visual field defects. The MRI showed high signal intensity lesion along the optic chiasm and tracts in FLAIR sequence, but no other structural lesion or enhancement of optic nerve pathway. Immunologic profile including NMO-IgG antibody and cerebrospinal fluid analysis were normal. She was diagnosed ethambutol-induced optic neuropathy. On follow-up examination at 2 months after discontinuing ethambutol, visual acuity was improved as 20/200 in the right eye and 20/200 in the left eye. **Conclusions or Comments:** Our case suggests that ethambutol-induced optic neuropathy can involve optic chiasm like other severe demyelinating diseases. Thorough history taking related to toxic effect should be accompanied to evaluate patients with optic chiasm involvement.

P-2-225

Immunoglobulin G 4-related disease presenting headache and diplopia

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Background & Significance: Immunoglobulin G (IgG) 4-related diseases are systemic syndromes characterized by elevated serum levels of IgG4 and IgG4-positive lymphoplasmacytic infiltrative lesions in the pancreas, liver, biliary tract, lung, breast, kidney, retroperitoneum, lymph node, thyroid gland or salivary gland. Orbital tissues especially the lacrimal gland and orbitare also affected by IgG4-related conditions. **Case:** A 72-year-old male with diabetes and ischemic heart disease complained of headache and diplopia for 2 weeks.

He had dull nature headache on the left frontal and left periorbital pain with mild nausea. His diplopia was worsened when he saw the left side and disappeared when he covered one eye. He did not experience any symptoms of dry eyes or dry mouth. He had no prominent proptosis but showed eyelid swelling on both sides. Mild lateral gaze limitations on both eyes were found. Thyroid function and antinuclear antibody test scores were within normal limits. The level of serum IgG was elevated to 5,000 mg/dL. Magnetic resonance imaging (MRI) demonstrated bilateral lacrimal gland enlargement but there was no thickening of extraocular muscles. Light microscopic examination of lacrimal gland tissue stained with hematoxylin and eosin showed dense lymphocyte hyperplasia and lymphoid follicles. Immunostaining for IgG4 also revealed numerous IgG4-positive plasma cells. After starting systemic steroid therapy, the intermittent diplopia and headache disappeared and both lacrimal glands started to decrease slowly. **Conclusions or Comments:** IgG4-related disease should be considered as one of the cause of diplopia, especially in patients with bilateral lacrimal gland enlargement.

P-2-226

A case of dural arteriovenous fistula misconceived as carotid cavernous fistula

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Background & Significance: Arterial-cavernous sinus fistulas may result from a direct communication between the intracavernous carotid artery and the venous channels of the cavernous sinus (direct carotid-cavernous sinus fistula) or between the smaller meningeal branches from either the internal or the external carotid arteries and the sinus (dural-cavernous sinus fistula). The direct fistula is often the result of trauma or rupture of an intracavernous aneurysm, and generally presents with sudden, marked signs and symptoms of orbital vascular engorgement. We report a case of a dural cavernous sinus fistula fed by right dural arteries, which presented with mainly left-sided signs and symptoms, which is less common with dural-cavernous sinus fistulas. **Case:** A 73-year-old man developed sudden onset of diplopia last for two days. He complained ophthalmic pain during eye movement 3 month ago. Recently, he had no trauma or infection history. He took a hypertension medication. He did not show headache, nausea, dizziness. On neurological examination, there was no gross limitation of extraocular muscle movement. Red glass test revealed horizontal diplopia in all direction. Orbital bruit was auscultated on left eye. Pupil light reflexes were normal. He had no relative afferent pupillary defect, his visual fields were full to confrontation. Manual motor and sensory examination were normal. Brain MRI showed left superior ophthalmic vein enlargement, we suspected of that was carotid cavernous fistula on left. But, carotid angiography performed on the left side was normal. Carotid angiography performed on the right side showed a dural-cavernous sinus fistula, with shunting from right meningeal artery directly to left cavernous sinus. Onyx embolization was done successfully. Follow-up brain MRI showed multiple dot-like diffusion restrictions in bilateral cortex, suggestive of embolic infarction. But, there was no additional neurological deficit. And compared to previous MRI, left superior ophthalmic venous lumen diameter was slightly decreased. On one month of the follow-up, diplopia was improved. **Conclusions or Comments:** Clinical symptoms of a cavernous sinus dural arteriovenous fistula result from an influx of arterial blood into the cavernous sinus itself and subsequent reflux into veins around the sinus. In our case, contrary to the clinical symptoms and brain MRI impression, the dural AV fistula was located in the opposite site. This is an unusual case of a dural-cavernous sinus fistula with manifestations which were mainly contralateral to the arterial supply.

P-2-227

Isolated horner syndrome in infective cervical myelitis

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Background & Significance: Horner syndrome occurs as a result of disruption to the sympathetic pathway. Depending on the cause of Horner syndrome, early recognition and intervention can be lifesaving. **Case:** A 54-years-old man presented with ptosis in right eye for 2 days. He had sustained headache with fever 4 days before. Examination showed ptosis and inverted ptosis in his right eyelid and 1 mm of anisocoria with the right pupil being smaller than the left. Other neurologic examination was unremarkable, and pharmacologic test using 0.5% apraclonidine was showed reversed ptosis. Neuroimaging for localizing the lesion of Honer syndrome, brain and cervical MRI disclosed a myelitis extending to the C5 to C7 level without brain abnormalities. Cerebrospinal fluid analysis also disclosed infection presumed that the viral origin. After treatment with antiviral agent and methylprednisolone, his symptoms were slowly improved. **Conclusions or Comments:** An isolated Horner syndrome may be the presenting manifestation of infective cervical myelitis.

P-2-228

Neural networks related to Opsoclonus-myoclonus syndrome revealed by PET study

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Background & Objectives: Opsoclonus is a disorder of involuntary eye movements with irregular, multidirectional saccades, without intersaccadic intervals. Opsoclonus arise because of the synaptic organization of burst neurons in the brainstem, in which positive feedback loops and postinhibitory rebound properties of burst neurons predispose to saccadic oscillations. Disinhibition of the fastigial nucleus in the cerebellum causes opsoclonus. The objective of this study is that which specific region is related to the deactivation in opsoclonus using 18F FDG-PET. And that which region is related to the post-treatment change of activation compared with a group of normal scans. **Method:** A 22-year-old woman presented with rapidly progressive vertigo, oscillopsia, and severe gait imbalance beginning 3 days before. On initial clinical examination, the patient showed intermittent bursts of large conjugate omnidirectional saccadic eye oscillations. CSF showed a mild lymphocytosis of 26cells/ μ L with normal opening pressure. Abdomen CT scanning revealed a huge heterogeneous adnexal mass of 9-cm which was confirmed histologically as a mature teratoma. She received consequently IV immunoglobulin and methylprednisolone, and then tumor removal was followed. Clinical improvement was noted after operation. Functional imaging data were gathered including FDG-PET within a week after the symptom onset and 2 months after her ocular movement improved. **Results:** In pretreatment PET study, the cerebral distribution of FDG uptake in the patient showed a diffuse decrease in the bilateral parieto-temporo-occipital cortex. These diffuse decrease FDG uptake area was distributed in Middle Temporal Gyrus (Brodmann area(BA) 37), Inferior Occipital Gyrus (BA19), Lingual Gyrus(BA 17), Middle Occipital Gyrus (BA 19), Inferior Temporal Gyrus (BA 20), Middle Temporal Gyrus(BA 39), Middle Occipital Gyrus (BA 18). In contrast, there were showed focal increase in cerebellar deep nuclei, including fastigial nucleus. In post treatment PET study, the cerebral distribution of FDG uptake in patient showed a bilateral occipital lobe in Lingual gyrus(BA17), middle occipital gyrus(BA 18, BA 19). And showed focal decrease in cerebellar deep nuclei, including fastigial

nucleus, showing decreasing uptake compared to previous study. **Conclusion:** Deactivation of specifically the peripheral visual field projection supports the view that opsoclonus inhibits visual motion processing along the Magnocellular pathway. Alterations in the properties of the membranes of burst neurons underlie saccadic oscillations. The concept of a membrane-based etiology of saccadic oscillations stems from the characteristics of the neural connections in the brainstem saccade generator. In a longitudinal ECD-SPECT study, an increased blood flow in the cerebellar vermis disappeared in analogy to clinical symptoms. Moreover, in a study, described in a short-term follow-up 7 days after treatment with clonazepam, a decrease of the glucose metabolism in the inferior cerebellar vermis. Our PET studies have revealed the striate and the extrastriate visual cortex deactivation in involuntary ocular oscillations, reciprocal hyperactivation in cerebellar deep nuclei including fastigial nucleus, and reversal of deactivation of visual cortex and hyperactivation of cerebellar deep nuclei.

P-2-229

A case of normal pressure glaucoma presenting as monocular nasal visual field defect

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Background & Significance: Previous studies reported visual field defect with structural lesion traumatic cataract, optic disc drusen, aneurysm, meningioma, pituitary tumor. however, it had not considered visual field defect without structural lesion, especially monocular nasal field defect. **Case:** We report a 75-year-old man who presented monocular nasal visual field defect without structural lesions on the afferent visual pathways. His monocular field defect progressed rapidly into complete visual loss. However, he showed relatively normal fundi with slightly increased cup to disc ratio with normal intraocular pressure and was diagnosed as the normal pressure glaucoma. **Conclusions or Comments:** Progressive monocular nasal field defect without structural lesion should be considered as the ocular problem such as glaucomatous visual field defect. Differential diagnosis was discussed.

P-2-230

Topographic lesion analysis of cerebellar tumor related positional nystagmus

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Background & Objectives: Positional nystagmus and vertigo syndromes are usually originated by peripheral vestibular disorder, mostly benign paroxysmal positional vertigo (BPPV), however, central positional nystagmus (CPN) is encountered. and it may be difficult to differentiate from BPPV. The differential diagnosis from BPPV may be difficult, but it is important since CPNs have critical underlying diseases such as infarction and tumor. We attempted to analyze the clinical characteristics and define the central structures responsible for CPN associated with brain tumors. **Method:** Four patients with positional nystagmus were enrolled. All subjects were initially misdiagnosed as BPPV involving the horizontal canal with apogeotropic nystagmus and then finally diagnosed with a brain tumor. Spontaneous and positional nystagmus were analyzed using a video Frenzel goggle and/or video nystagmography system. Brain MRI was obtained in all patients. We investigated the lesion site of the brain

tumor. Neuro-ophthalmological and radiological findings were analyzed. **Results:** The enrolled 4 patients (3 men; age range = 19-77 years) showed apogeotropic positional nystagmus in the supine roll tests. Except the positional nystagmus, neurologic examination showed no definite abnormalities. Since all subjects were initially diagnosed as BPPV, canalith repositioning maneuvers (Gufoni's maneuver), head-shaking maneuver and Brandt-Daroff exercise were applied. However, positional nystagmus persisted in all subjects. Brain MRI showed brain tumors involving midline cerebellar structures around the 4th ventricle and the nodulus. The pathological diagnosis was hemangioblastoma in 2 patients and metastatic tumor in the other patients. **Conclusion:** Apogeotropic pattern of CPN was observed in patients of positional nystagmus associated with cerebellar tumor. It can mimic BPPV involving the horizontal canal with apogeotropic nystagmus, so clinicians should be aware of the possibility of CPN especially in cases of the failure of the canalith repositioning maneuver for the treatment of BPPV and the presence of associated central neurologic symptoms and signs.

P-2-231

A case of opsoclonus myoclonus ataxia syndrome: transition of coulomotor findings from ocular flutter to opsoclonus

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Background & Significance: Adult-onset opsoclonus myoclonus ataxia syndrome (OMA) is a rare disorder associated with cancer, infection, demyelinating disease or unknown causes. The opsoclonus is known as a main oculomotor abnormality but the ocular flutter could be present in the recovering phase or an isolated oculomotor finding. Here we present a patient with OMA with changing oculomotor findings from ocular flutter to opsoclonus. **Case:** A 41-year old man visited out-patient clinic complaining of walking difficulty. He had suffered from severe upper respiratory tract infection three weeks ago and then the gait disturbance developed two weeks later. Headache, dizziness, nausea, vomiting and mild fever were accompanied by. On initial examination, he showed mild neck stiffness and trunkal ataxia. He was admitted under the impression of central nervous system infection. He revealed ocular flutter and four limbs myoclonus after two days. And then the eye movement disorder changed into opsoclonus two days later. The serial oculomotor findings are recorded by video camera and video oculography. Full laboratory battery for paraplastic syndrome, autoimmune encephalitis, and demyelinating disease were performed. High-dose steroid, intravenous immunoglobulin and plasma exchange resulted in minimal improvement of his clinical manifestations. **Conclusions or Comments:** We observed ocular flutter changing into opsoclonus in the same patient with OMA. These serial observations are necessary in a patient because there is a continuum between ocular flutter and opsoclonus.

P-2-232

Delayed loss of visual acuity after closed head trauma: a case with indirect traumatic optic neuropathy

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Background & Significance: Indirect traumatic optic neuropathy is rarely occurred after closed head trauma. Indirect traumatic optic neuropathy is caused by the transmission of forces to the optic nerve without injury of normal tissue structures. Here, we report a patient presented with delayed loss of visual acuity

ty after head trauma, who was diagnosed clinically, as indirect traumatic optic neuropathy and reviewed the literature. **Case:** A 38-year-old male developed visual disturbance of both eye, more severe in left eye. He was healthy without any past medical histories, except auto-bicycle traffic accident 2 months ago. At the time of the accident, he suffered from multiple fractures of left leg and nose. However, he had no traumatic lesion of orbit structure on facial computed tomography. His visual acuity of left eye gradually dropped to 0.02 and he complained of blurred vision of right eye. No neurological deficits were noticed except for the visual disturbance. Electrophysiologic tests showed prechiasmatic conduction defect in bilateral VEP pathways. Any responsible lesions in both visual pathways were not noticed on orbit magnetic resonance image (MRI). No brain parenchymal lesions were noticed on brain MRI. Immunologic tests including NMO autoantibody showed negative results. He was taken high dose oral steroid(methylprednisolone 48mg per day) during three weeks. However, he had no benefit at all from the steroid therapy. Furthermore, his visual acuity of right eye gradually also dropped to 0.02 over two month. Inflammatory optic neuropathy was doubtful because of unusual clinical course, no response of steroid therapy, and no laboratory evidences of autoantibodies. We diagnosed him clinically as having indirect traumatic optic neuropathy. **Conclusions or Comments:** There have been few studies on indirect traumatic optic neuropathy in Korea. Indirect traumatic optic neuropathy need to be considered as a cause of visual loss after closed head trauma.

P-2-233

Optic tract syndrome due to posterior communicating artery aneurysm

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Background & Significance: Lesions involving the optic tract may cause visual field defects, relative afferent pupillary defects, and optic atrophy. However, optic tract syndrome has not been described in a posterior communicating artery aneurysm. **Case:** A 66-year-old man was referred for evaluation of visual field defects that was incidentally found during routine evaluation a month before. Examination confirmed right upper quadrantanopia and additional found a relative afferent pupillary defect in the right eye. Extraocular movements, ocular alignment, and other cranial nerve function were normal. Corrected visual acuity was 0.4 OD and 0.9 OS. Color was normal OU on Hardy-Rand-Rittler and Ishihara tests. Humphrey perimetry documented right homonymous superior quadrantanopsia. Optical coherence tomography revealed atrophy of the retinal nerve fiber layer inferior quadrant in either eye. Magnetic resonance imaging and angiography revealed a 2 cm sized aneurysm originating from the left posterior communicating artery and compressing the left optic tract. He was treated successfully with coil embolization of the aneurysm. **Conclusions or Comments:** Posterior communicating artery aneurysm is a rare cause of homonymous hemianopsia due to compression of the optic tract.

P-2-234

Periodic pupillary change in Cheyne-Stokes respiration

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Background & Significance: Pupillary oscillations can be present in normal subjects, but associated with various neurological diseases such as diabetic

neuropathy, tabes dorsalis and epileptic seizure. We report a patient with Cheyne-Stokes respiration showing periodic pupillary change. **Case:** A 50-years-old woman with poorly controlled hypertension and diabetes mellitus presented with visual blurring in both eyes and decreased mentality. On admission, her blood pressure was 240/120 mmHg. She was semicomatose, and both pupils were miotic with reaction to light. Horizontal vestibulo-ocular reflex was impaired bilaterally. T2-weighted brain MRIs showed bilateral symmetric hyperintensities at periventricular deep white matter, basal ganglia, thalamus and cerebellum. Fundus photography revealed Grade 4 hypertensive retinopathy. These findings suggested posterior reversible encephalopathy syndrome due to malignant hypertension. Three days later, Cheyne-Stokes respiration first appeared with periodic pupillary change. At the end of hyperpnea, both pupils were constricted with transient right-beating nystagmus and roving eye movements. The miosis continued during the phase of apnea. After that, both pupillary dilatation occurred at the end of apnea, which were maintained during the phase of hyperpnea. This periodic pupillary change occurred repeatedly with a cycle of one minute 30 seconds. After 2 weeks, her consciousness improved and Cheyne-Stokes respiration with periodic pupillary change disappeared. **Conclusions or Comments:** In our patient, baseline miosis and periodic pupillary change during Cheyne-Stokes respiration suggest that arterial hypoxia during the end phase of apnea may lead to pupillary dilatation through the activation of peripheral adrenergic innervation.

P-2-235

Miller-Fisher syndrome antecedent acute hepatitis A in a patient with chronic hepatitis B

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Background & Objectives: Miller-Fisher syndrome (MFS) is a disease which is characterized by clinical triad of ophthalmoplegia, ataxia, and areflexia. It is considered as a variant form of Guillain-Barré syndrome (GBS). Many cases of GBS follow an infection, usually a viral infection, especially in the upper respiratory organs. And there are some reports linking GBS and acute hepatitis, and vaccination related GBS. Korea is one of the most endemic areas of hepatitis B virus (HBV) infection and has a high prevalence of chronic HBV infection. More over, prevalence of acute hepatitis A virus (HAV) infection in young adult is now increasing, it became an important social issue. We report a rare case of MFS which is developed after acute hepatitis and exacerbation of chronic hepatitis B. **Method:** A 37-year-old man presented to our hospital with diplopia and gait disturbance. The patient had a vertical transmission of chronic HBV infection. Six weeks ago, he admitted to our hospital for fever, myalgia, and jaundice, diagnosed with acute HAV infection and exacerbation of chronic HBV infection, and discharged with improvement of symptoms. One week before the second admission, the initial symptoms were perioral numbness, glove and stocking pattern tingling sense, right side hypoesthesia, and mild gait disturbance. The symptoms were progressed, and at the time of admission the patient suffered from biocular diplopia, dysphagia, aggravated tingling sense and hypoesthesia, and he cannot walk without assistance. We found ophthalmoplegia, facial diplegia, upper and lower extremity dysmetria, areflexia, and there were no muscle weaknesses in lower extremities but ataxia was severe enough that the patient hardly walks. **Results:** He was diagnosed with MFS, six weeks after acute HAV infection. Cerebrospinal fluid study was normal with anti-GQ1B antibody negative. He underwent nerve conduction study and electromyography and the results were consistent with sensorimotor polyneuropathy. The patient was treated with intravenous immunoglobulin and by hospital day 6, the gait disturbance started to improved. **Conclusion:** GBS is commonly triggered by antecedent viral infections, which is started one to four weeks before the symptoms. But the exact mechanisms of a link be-

tween infection and demyelination remain to be established. GBS is classified as acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor sensory axonal neuropathy, and variant. We described MFS occurred after HAV and HBV infection. There are some case histories reports on the development of GBS following acute hepatitis and the symptoms were motor weakness, ambulatory disturbance, leg numbness, and facial palsy. But only few cases about typical MFS, which is variant of GBS, with ataxia, ophthalmoplegia, and areflexia after hepatitis are reported. While most of cases about GBS with hepatitis are developed at acute phase of hepatitis, our patient was at remission state of hepatitis. More over, the patient has suffered from acute exacerbation of chronic HBV infection. It is hard to think HBV infection as a direct risk factor of HAV infection, but it might had a synergistic effect to infection and course of HAV.

P-2-236

Prognostic value of brain MRI, blink reflex in Bell's palsy

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Background & Objectives: Bell's palsy (BP) is an idiopathic peripheral facial palsy with weakness of facial muscles with acute onset and no readily identifiable cause, due to unilateral dysfunction of the seventh cranial nerve. There are still controversies regarding the enhancement of the facial nerve on MRI and its correlation with disease severity, electrophysiological tests, and prognosis. This study is conducted to investigate the correlation between the facial nerve enhancement on MRI, abnormality of electrophysiologic studies and prognosis of BP. **Method:** This prospective study includes 520 patients of BP from July 2005 to June 2015. History taking, neurologic examination, electrophysiologic test and brain MRI were performed at the time of diagnosis. The severity of facial palsy was assessed by House-Blackmann (H-B) facial nerve grading scale. Follow-up physical examination and electrophysiologic test were conducted about 8 weeks after the initial assessment. **Results:** Brain MRI showed that 292 of them had facial nerve enhancement, while the rest patients did not. Patients with facial nerve enhancement had more severe facial palsy than patients without enhancement at the initial assessment (p-value: 0.001). After 8 weeks, H-B grade between the two group were statistically significant (p-value: 0.023). Higher H-B grade was seen in the group with enhancement. Patients with abnormality on blink reflex had more severe facial palsy than with normal at time of diagnosis (p-value: 0.001). At the 8 weeks after diagnosis, group of abnormal in blink reflex had higher H-B grade (p-value: 0.008). However, facial nerve conduction study did not reflect the prognosis of Bell's palsy (p-value: 0.514). **Conclusion:** Facial nerve enhancement on brain MRI have more severe facial weakness and poor prognosis. And blink reflex also reflect the severity and prognosis of BP. Brain MRI and blink reflex is an effective tool in predicting severity of facial muscle weakness and prognosis of BP.

P-2-237

Early prediction of poor outcome in Guillain-Barré syndrome

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Background & Objectives: Guillain-Barré syndrome (GBS) is an acute post-infectious immune-mediated peripheral neuropathy with a highly variable clinical course and outcome. Although intravenous immunoglobulin or plasma exchange is beneficial, some patients recover incompletely. Identifying early patients with poor outcome might provide opportunity for effective or additional treatment in early stage when nerve degeneration is potentially

reversible. We aimed to determine early clinical factors associated with poor functional outcome in GBS. **Method:** Forty-seven patients with GBS were enrolled, who were hospitalized and follow up at one university hospital during May 2005-June 2015. Data collected retrospectively were used to identify risk factors of being unable to walk at 6 months. Various epidemiological, clinical and electrophysiological parameters were assessed. Potential predictors of poor outcome (unable to walk unaided) at 6 months were analyzed. **Results:** Mean days from onset to admission were 6 days. Twenty-five Patients (53%) were unable to walk at admission and 11 patients (23%) were unable to walk at 6 months. Older age (> 60 years) (p=0.002), low MRC sum score (p=0.001), or high GBS disability score (p=0.001), and respiratory failure (p=0.001), severe weakness unable to walk (p=0.005), or voiding difficulty (p=0.042) at hospital admission were associated with being unable to walk at 6 months. Preceding infections including diarrhea, subtype of acute motor axonal neuropathy (AMAN), and existence of GM1 antibody were not associated with poor prognosis in our study. **Conclusion:** Our study confirms that the poor outcome is associated with older age and severity indicated by GBS disability score or MRC sum score, but was not consistent with the previous studies suggesting preceding infection, AMAN subtype, and GM1 antibody as poor prognostic factors. Initial voiding difficulty was proved as poor prognostic factor in our study, which implicates that severe involvement of lumbosacral root is associated with difficulty in both urination and walking. This study suggests that severe weakness unable to walk or voiding difficulty at admission, and older age could predict poor outcome at 6 months in GBS.

P-2-238

High risk group screening of later onset Pompe disease in unspecified myopathy patients

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Background & Objectives: Pompe disease is a rare autosomal recessive disorder caused by the deficiency of a lysosomal enzyme, acid alpha- glucosidase (GAA). Pompe disease is broadly divided into two onset forms, such as infantile onset Pompe disease (IOPD) and later onset Pompe disease (LOPD). Also, early diagnosis and start with enzyme replacement therapy (ERT) have remarkable effects on the prognosis of Pompe disease. But, in South Korea, only around 20 patients with Pompe disease are on treatment as of now. This means that most of Pompe disease patients in Korea are underdiagnosed and cannot have opportunity for treatment. So we performed risk group screening of later onset Pompe disease in unspecified myopathy patients using GAA activity measurement and genotype analysis of GAA gene. **Method:** From June 1, 2013 to August 31, 2014, we enrolled 100 myopathic patients, who are suspected as Pompe disease with clinical manifestation. For diagnosis of Pompe disease, GAA activity were evaluated with direct blood smear (DBS) and mixed leukocytes. We studied GAA activity by reaction with only substrate, substrate and acarbose, and their ratio (GAA activity with only substrate by with substrate and acarbose). For patients with low GAA activity, we performed Sanger sequencing of GAA gene. DNA was extracted from either blood sample or muscle sample. And we compared GAA activity of the patients with between Pompe disease, pseudodeficiency mutations, by Mann-Whitney U test. **Results:** Among 100 patients, there are 58 males and their mean age is 38.9 years old. 65 patients presented symptoms over the age of 15 years. Their mean CK level is 2014.8 and about 70% patients show symmetric arm and leg, proximal dominant weakness. We decide low absolute value of reaction substrate and acarbose or low ratio with DBS and mixed leuko-

cyte sample. 18 patients decided as low GAA activity. Among them, 3 patients is true Pompe disease with genetic confirmation (2 patients: c.1822C>T [p.R608*] + c.2238G>C [p.W746C], 1 patient: c.546G>G [leaky splicing] + c.1316T>A [p.M439K]). But 15 patients have at least one G576S or E689K mutation, known as pseudodeficiency allele. All values of GAA activity except GAA activity of mixed leukocyte with only substrate show significant difference between Pathogenic and Pseudodeficiency mutations. **Conclusion:** Through risk group screening study, we found 3 Pompe disease patients among 100 unspecified myopathy patients. It is higher detection rate than previously reported prevalence, up to 1 per 40,000 people. And 18 patients showed low GAA activity with DBS and mixed leukocyte and 15 patients have pseudodeficiency mutations. Therefore, we have to consider possibility of Pompe disease in diagnosis of myopathy and it is needed to confirm genotype of GAA gene, even though patients have low GAA activity with DBS and mixed leukocyte.

P-2-239

Pontine venous malformation mimicking ocular myasthenia gravis

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Background & Significance: Myasthenia gravis (MG) is a neuromuscular junction transmission disorder, characterized by fluctuating neurological symptoms, such as diplopia, ptosis or limb muscle weakness. MG can be diagnosed with the typical history, neurological examination, and alleviation of symptoms after administration of acetylcholine esterase inhibitor (AChEI). In addition, repetitive nerve stimulation test (RNST) and serum antibody to acetylcholine receptor (AChR) are utilized for the objective tools for the MG diagnosis. Nonetheless of above, ocular MG may show normal RNST and negative AChR antibody test, especially in early or minimal stage of the disease. Therefore, clinicians should carefully exclude other diseases or conditions mimicking ocular MG. There have been several reports of brain stem lesions that clinically resemble ocular MG. Here, we present a case of pontine venous malformation mimicking ocular MG. **Case:** A 29-year-old male with no known medical history presented with fluctuating horizontal diplopia for several years. He complained that diplopia aggravated with exhaustion or after strenuous exercise and diplopia was absent or minimal in the morning. At the time of initial interview, he complained of horizontal diplopia that was aggravated with gaze to the right side. His extraocular muscle movement showed no restrictions on all the six directions, bilaterally. However, red glass test revealed a typical pattern of isolated right 6th cranial nerve palsy. Other components of neurological examination showed no abnormalities. Interestingly, his diplopia was aggravated with Valsalva maneuver. Both RNST and AChR antibody test were normal. To differentiate other structural lesions around the right 6th cranial nerve or nucleus, we performed brain MRI with the thin sections through brainstem. Brain MRI revealed venous malformations on bilateral pons and medulla. The draining vein on the right posterior pons was considerably dilated and showed high signal intensity on gadolinium enhanced image. He was empirically treated with 60mg of oral pyridostigmine, 3 times per day. After one week of medication, his diplopia was remarkably improved to even remission of symptom. **Conclusions or Comments:** Diagnosis of ocular MG has often been a challenging problem. Although fluctuation of neurological symptom is the hallmark of MG, other diseases rarely show a similar pattern of fluctuation, like our case. Some researchers have already reported various brainstem lesions mimicking MG: vertebral artery malformation, Chiari type I malformation, pontine glioma and other brain stem tumors. Our case raised two interesting but mysterious issues. First issue is how pontine venous malformation could develop the fluctuation of diplopia in this case. Second issue is unknown mechanism regarding the favorable re-

sponse to AChEI drugs. Although rather speculative explanations, we suggest followings for these issues: Diplopia in this case can be associated with mechanical compression or electrical interference over neural conduction by congestions of venous malformation in pons. Thus, Valsalva maneuver evokes peripheral venous stasis, and subsequent structural compression or physiological block of right 6th nucleus. The effectiveness of AChEI is unknown but may be related with the resolution of decreased safety factor. Because of proximal 6th nerve derangement, our patient probably had decreased safety factor. Because of proximal 6th nerve derangement, our patient probably had decreased safety factor in his extraocular muscle. AChEI can increase the amount of acetylcholine at junctional cleft and increase the success rate of neuromuscular junction transmission. To our knowledge, this is the first case of pontine venous malformation clinically mimicking ocular MG. Venous malformation of brainstem should be added as one of differential diagnoses for ocular MG. Brain imaging is crucial if this mimic is suspected for the differential diagnosis.

P-2-240

Multifocal motor neuropathy with conduction block presented with calf muscle rippling

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Background & Significance: Multifocal motor neuropathy is an extremely rare disease that shows progressive and asymmetric limb motor weakness with electrophysiologic evidence of conduction block at a corresponding nerve. Motor weakness is a major clinical characteristic of multifocal motor neuropathy. However, the disease has usually chronic course, therefore motor weakness may not be obvious at initial symptom presentation. Herein we described a case with continuous calf muscle rippling without motor weakness, and electrophysiologically proven motor neuropathy with conduction block. **Case:** A 69-years old man presented with continuous rippling of bilateral calf muscles started 2 month ago. He had medical history of liver cirrhosis related to hepatitis C and hypertension. Except for calf muscle rippling, he did not complain of limb weakness or sensory symptoms. In neurologic exam, nearly continuous rippling on bilateral calf muscle was clearly visible. There was no weakness or sensory change in four extremities. Deep tendon reflexes of knee and ankle were bilaterally hypoactive, and no pathologic reflexes were found. In electrophysiologic studies, right posterior tibial nerve motor nerve conduction study (NCS) showed conduction block (negative peak CMAP area reduction more than 50%, CMAP duration increase less than 30%) with marked slowing of conduction velocity over ankle- popliteal fossa segment. In left posterior tibial motor NCS, pseudo-conduction block with conduction velocity slowing was noted in ankle-popliteal fossa segment. Despite of normal distal CMAP amplitude, F-wave was not evoked in left posterior tibial nerve. Terminal latencies of both median motor nerves delayed slightly, and right peroneal motor nerve showed slowed conduction velocity in ankle-fibular head segment. All sensory NCSs were normal. Needle electromyography in calf muscles revealed positive sharp wave with neuropathic motor unit potential. Serum anti- GM1 IgM antibody was positive with high titer. **Conclusions or Comments:** Although motor weakness was not present, continuous muscle rippling represents motor involvement. In addition, conduction block in electrophysiologic studies and positive anti-GM1 IgM antibody are key criteria for diagnosis of multifocal motor neuropathy with conduction block. This case suggest that motor weakness at initial presentation could be subtle and atypical in multifocal motor neuropathy. Muscle rippling movement must be considered as manifestation of motor involvement in neu-

romuscular disorders.

P-2-241

Neurofibromatosis presenting as chronic inflammatory demyelinating disease

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Background & Significance: Neurofibromatosis type 2 is a rare autosomal dominant disorder characterized by the occurrence of schwannomas, peripheral nerve and cutaneous tumors. Patients seldom present peripheral nerve lesions unrelated to tumor masses, mostly axonal type. We hereby report a case of CIDP patient whose nerve biopsy confirmed neurofibromatosis. **Case:** A 32-years-old female patient with history of ovarian cyst suffered from abdominal cramp and over 3 months, developed severe headache, both leg tingling sensation, limb tremor, knee pain and limb weakness. Neurologic exam revealed right facial hypoesthesia, right lower extremity hypoesthesia and MRC grade IV~IV+ weakness in all limbs. Deep tendon reflexes were normoactive. Brain MRI demonstrated multiple, bilateral cranial nerve enlargement with enhancement including cranial nerve 3, 5, 7 and 8. Diffuse thickening of bilateral spinal nerve roots and ganglions were shown in cervical spine MRI. Nerve conduction study found demyelinating type polyneuropathy. Facial motor NCVs were normal, but blink reflex revealed bilateral trigeminal nerve defect. On laboratory studies autoimmune studies including anti-GM1, GD1b antibodies were negative. Protein was elevated to 59.4mg/dL in CSF, without pleocytosis. She was treated with oral steroids and later mycophenolate was added. Treatment response was poor so further evaluations were done to identify other etiologies mimicking CIDP. PET CT whole body scan did not reveal any malignancy. Nerve biopsy of left supraorbital nerve identified neurofibroma. The patient did not have any skin lesions, hearing impairment nor family history that could imply neurofibromatosis. Conventional gene studies with direct sequencing did not reveal mutations of NF1 and NF2 genes. But since neurofibroma was found on biopsy and brain MRI supports bilateral involvement, she was diagnosed as neurofibromatosis type 2. **Conclusions or Comments:** This case implies that in atypical or treatment unresponsive patients, we should always take account that other etiologies could mimic CIDP. Even though it is not well recognized, neurofibromatosis could present as CIDP. Neuropathy in neurofibromatosis type 2 is known to be usually axonal type, but slowing in the demyelinating range could be shown. Its peripheral nerve involvements are rare in incidence but it could be the sole presenting feature.

P-2-242

A case of anti-GQ1b antibody syndrome presenting with acute unilateral ophthalmoplegia without ataxia and areflexia

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Background & Significance: Anti-GQ1b antibody syndrome is one of the causes which induce pupil-sparing third nerve palsy. Although Miller-Fisher syndrome which shows ophthalmoplegia, ataxia and areflexia is the representative, various subtypes are associated with anti-GQ1b antibody. We experienced a case of anti-GQ1b antibody syndrome presenting isolated unilateral third nerve palsy with subtle facial weakness and report the clinical features and laboratory findings. **Case:** A 70-year-old male presented with right ptosis, diplopia and periocular pain for a week. He was on medication due to allergic rhinitis for 40 years. Neurological examinations showed right oculomotor nerve palsy but both pupils were isocoric with prompt light reflexes. Visual

acuity and visual field test were normal. Mild right facial palsy was suspected, although patient did not complain. Other cranial nerves were normal. There was no limb weakness or sensory disturbance. Cerebellar function test was normal and the deep tendon reflexes were normoactive. Brain MRI showed no abnormalities in brainstem and cerebral hemispheres, but also inflammation in orbit or hypertrophy of extraocular muscles was not revealed. Electrophysiological test showed right facial neuropathy but unremarkable findings on RNS. Hematological evaluations including complete blood count, erythrocyte sedimentation rate was within normal limits and immunologic serum study such as anti-Ach receptor antibody, anti-MAG antibody was negative. Thyroid function test showed elevated TSH level, but thyroid hormones were normal, which indicated asymptomatic hypothyroidism. There was no evidence of diabetes mellitus on HbA1c and 75g oral glucose tolerance test. In CSF study, it shows mild increased protein but normal IgG index (WBC 1/mm³, RBC 0/mm³, protein 50 mg/dL, glucose 67 mg/dL, IgG index 0.39). Enzyme linked immunosorbent assay from acute stage in serum revealed positive anti-GQ1b antibody IgM. He was treated with IVIG (0.4mg/Kg) for 5 days. His symptoms showed some improvement and he is under clinical follow-up. **Conclusions or Comments:** Unilateral pupil-sparing ophthalmoplegia has multiple potential etiologies (e.g. Tolosa-Hunt syndrome, myasthenia gravis, multiple sclerosis, diabetes, thyroid ophthalmopathy, vasculitis, sarcoidosis and structural lesions in brain). In this case, we could not find any structural lesions in brain MRI. And endocrine dysfunction consistent with symptoms was not revealed. Also there was devoid of evidence for neuromuscular junction disease. Anti-GQ1b antibody titers are related to various clinical manifestations including Miller-Fisher syndrome, Guillain-Barre syndrome (GBS) with ophthalmoplegia, Bickerstaff brainstem encephalitis and pharyngeal-cervical-brachial type GBS. This case informed that the patient with unilateral isolated third nerve palsy without ataxia or areflexia has showed elevated anti-GQ1b antibody. It may be necessary to test anti-GQ1b antibody in patients with unilateral isolated painful ophthalmoplegia for precise diagnosis.

P-2-243

Guillain-Barré Syndrome with persistent and prominent asymmetric weakness

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Background & Significance: Guillain-Barré syndrome (GBS) is a heterogeneous disease in terms of clinical and electrophysiological findings. Typically, it is characterized as progressive weakness in both arms and legs with areflexia or hyporeflexia. Other supporting features include relative symmetry, mild sensory signs, cranial nerve involvement, cessation of progression within 2-4 weeks of onset, autonomic dysfunction, absence of fever at onset and typical cerebral spinal fluid(CSF) and nerve conduction study(NCS) features. However, other diagnosis may be considered in patients with asymmetrical weakness, persistent bladder and bowel dysfunction and distinct sensory levels. Here we present a patient with prominent and persistent asymmetrical weakness who was diagnosed as acute motor axonal neuropathy according to NCS findings, clinical courses and serologic test including anti-ganglioside autoantibodies. **Case:** An 18-year-old male with no known underlying diseases presented with right lower limb weakness with acute onset. He had history of fever and watery diarrhea 2 weeks prior to the visit. On neurological exam, he was alert and oriented. His cranial nerve function showed mild dysphagia without facial palsy or ophthalmoplegia. On motor exam, his motor grades were all 0 on his right leg, from hip to toe. He also had distal motor weakness on the left leg but was much less severe compared to the right leg. The knee and ankle jerks were absent bilaterally. Cerebellar function test was not remarkable. He did not com-

plain of any sensory symptoms. On CSF laboratory results on his first day, his white blood cell count was 10 but protein and glucose was within normal range. Porphobilinogen and delta aminolevulinic acid did not show increment in 24 hour urine collection. Anti-GM1 IgM and IgG autoantibodies and Anti-GD1b IgM and IgG autoantibodies were positive. (Anti-GM1 IgG was borderline positive.) Nerve conduction study on the 3rd day of onset revealed purely motor axonal neuropathy in which right side was much more severe. His weakness worsened fast, reaching nadir within the first few days. He was treated with intravenous immunoglobulin 2mg per kg for 5 days and his symptoms gradually improved. On 6 month follow up, the patient's symptoms were greatly improved in his left leg, but his right leg was minimally improved. His follow up nerve conduction studies showed minimal improvement, with features for purely motor axonal neuropathy with prominent asymmetry. **Conclusions or Comments:** Monophasic symmetric ascending paralysis is the most prominent feature of typical Guillain-Barré syndrome. However, atypical findings such as persistent and prominent asymmetry can only be seen rarely. Asymmetric patterns are known to be related to multifocal nerve involvement of autoimmune attack especially when cranial nerves are involved. Even though he had persistent prominent asymmetry in clinical symptoms and signs, we diagnosed acute motor axonal neuropathy in this patient according to electrophysiological, clinical and abnormal anti-ganglioside autoantibodies findings.

P-2-244

A case of Pompe disease having a diagnostic difficulty based on genetic study

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Background & Significance: Pompe disease (Glycogen-storage disease type II, OMIM #232300), an autosomal recessive disorder caused by a deficiency of acid α -glucosidase (GAA), results in the accumulation of glycogen in the lysosome, followed by muscle damage. We presented a case of Pompe disease having a diagnostic difficulty based on genetic study because of the change of pathogenicity of the single nucleotide mutation and rare autosomal recessive inheritance patterns. **Case:** A 45-year-old female admitted to slowly progressive muscular weakness and respiratory difficulty. She felt bilateral lower limb weakness and axial muscle weakness from 5-6 years old. At the age of 37 years, she started ventilator care in intensive care unit due to respiratory failure. At the age of 40 years, she admitted to our hospital. She had a son, who died by aspiration pneumonia at 14 months old, diagnosed as Pompe disease with muscle hypotonia, severe cardiomegaly and muscle biopsy finding. On physical examination, she presented generalized muscle atrophy, Gower's sign, and axial and proximal dominant limb weakness. Routine blood laboratory tests showed mild liver enzyme and creatinine kinase (CK) elevation and muscle biopsy showed numerous clear vacuoles in the muscle fiber, showing weak stainability to PAS stain. But GAA enzyme activity and genotype assay showed negative results. So we only had done conservative management, including respiratory care. 5 years later, we re-evaluated GAA enzyme activity and genotype, which showed that GAA enzyme activity of leukocyte with acarbose was abnormal (1.0 nmol/hr/mgprotein, reference range: ≥ 10), and genotyping analysis showed compound heterozygote pathogenic mutations - c.1822C>T (p.R608X) and c.2238G>C (p.W746C) with heterozygous c.1726G>A (p.G576S). Now, she has been treated with Myozyme, and she felt mild improvement when she tried to stand with assistance. **Conclusions or Comments:** Through this case report, we suggest that the limitations of genetic diagnosis exist and more careful genetic diagnosis is needed in neuromuscular disease because, like our case, mother and son with one genetic disease were possible in genetic disease with autosomal recessive inheritance.

P-2-245

Chronic relapsing axonal neuropathy

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Background & Significance: Chronic axonal neuropathy is common and usually slowly progressive. It is mostly caused by metabolic, toxic or hereditary disorders. But chronic relapsing axonal neuropathy is rare. Some case reports showed that intravenous immunoglobulin or other immunosuppression drugs may help in patients with chronic relapsing axonal neuropathy as in patients with chronic inflammatory demyelinating polyneuropathy. We report a woman with chronic relapsing axonal neuropathy who showed relapses with long term interval. **Case:** A 53-year-old woman was admitted due to 5 days of newly developed weakness of right lower extremity and exacerbation of pre-existing weakness of left lower limb weakness. She was able to stand still and walk independently. She had no diplopia, ptosis, facial palsy or other cranial nerve symptoms. About 20 years ago, she suffered from left lower extremity weakness of unknown etiology. After the episode, she had remaining weakness in dorsiflexion of her toes on the left side. On neurological examination, she was alert with intact orientation. She could stand still but complained of steppage gait, worse on the left side. Using MRC score, right ankle dorsiflexion was grade IV, right ankle inversion and eversion grade III. On the left side, ankle dorsiflexion was grade I and ankle inversion was III, eversion I. She also complained of hypesthesia on her left whole arm, left trunk and bilateral lower extremities below knees, especially on the lateral side. Position senses were normal, while vibration senses below knees were absent bilaterally. Ankle reflexes were absent bilaterally. Pathologic reflexes were absent. Laboratory tests including complete blood count, electrolytes, liver enzymes, creatinine, serum and urine protein electrophoresis, ASO, IgA, Anti-SS-A and B, Anti-dsDNA, p-ANCA, c-ANCA, FANA, Anti gangliosides antibodies, CRP, Rheumatoid factor, thyroid function test, porphobilinogen, delta-aminolevulinic acid and vitamin B12 were all within normal range. CSF examination on the day of admission was normal. (RBC 0, WBC 0, protein 26.5mg/dL, glucose 72mg/dL). Nerve conduction studies and electromyography revealed decreased compound action potential(CMAP) and sensory nerve action potential(SNAP) amplitudes on bilateral lower limb, worse on the left side. Motor nerve conduction velocity and sensory nerve conduction velocity was only mildly decreased with no definite evidence of demyelination. F waves on right median and bilateral tibial nerves were unobtainable and H-reflexes were absent bilaterally. We were not able to perform nerve biopsy due to patient's refusal. With the diagnosis of possible chronic relapsing axonal neuropathy, we treated the patient with Intravenous immunoglobulin(IVIG) for 5 days, with total dose of 2g/kg. We are waiting whether this IVIG trial will alleviate her symptoms or not. **Conclusions or Comments:** There is no known definite treatment regimen in chronic progressive(not relapsing) idiopathic axonal neuropathy. However we may consider immunosuppressants in patients with chronic relapsing idiopathic axonal neuropathy(CRAN) as we usually do the kind of treatment in treating chronic inflammatory demyelinating polyneuropathy. (CIDP)

P-2-246

Isolated compressive temporal branch palsy of facial nerve after drug intoxication

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Background & Significance: Temporal branch of facial nerve runs superficially along a temporal area. The branch can be damaged by trauma, cutaneous surgery or biopsy around temporal area, resulting in isolated forehead palsy. However, isolated temporal branch palsy of facial nerve by compression or entrapment has been rarely reported. Herein we reported isolated temporal branch palsy of facial nerve which occurred after drug intoxication and secondary rhabdomyolysis. **Case:** A 25-years old man visited emergency room for altered mentality. He had a history of schizoaffective disorder and had taken quetiapine, escitalopram and lorazepam. For suicidal attempt, he took large dosage of quetiapine, escitalopram and lorazepam at least 6 hours before the visit. At the time that the patient was rescued, he lay on right side and placed his head on a floor. At initial evaluation, mentality was stupor and multiple skin ulcerative lesions at prolonged compression sites were found. Serum muscle enzymes were elevated, creatine kinase as 6173 IU/dL and myoglobin as >3000 ng/mL, indicating rhabdomyolysis. Brain CT revealed soft tissue swelling on right zygomatic and temporal area. He was treated with supportive care including massive hydration. After the recovery of mentality and soft tissue swelling on right head, wrinkling impairment of right forehead was detected. Neurologic exam revealed forehead paralysis without involvement of any other facial muscles. The other cranial nerve exams were normal. Blink reflex and facial nerve conduction study recorded at lower orbicularis oculi were normal. However, facial nerve compound motor action potential recorded at frontalis was not evoked on right side, whereas left side showed clearly visible potential. Based on clinical history, symptoms, neurologic exams and electrophysiologic studies, isolated temporal branch palsy of facial nerve was diagnosed. **Conclusions or Comments:** The isolated temporal branch palsy of facial nerve may be resulted from compression over zygomatic arch by prolonged immobilization, or entrapment between swollen muscles and soft tissues. Rhabdomyolysis and drug intoxication occasionally be accompanied with peripheral nerve injuries. Thorough history taking and physical exams were needed for accurate detection and proper management of the nerve injuries.

P-2-247

Acid alpha-glucosidase pseudodeficiency allele in normal Korean populations

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Background & Objectives: Pompe disease is a rare autosomal recessive disorder caused by the deficiency of a lysosomal enzyme, acid alpha-glucosidase (GAA). Up to 500 nucleotide variants have been identified in this gene, among which 350 are reported to be pathogenic either in infantile or late-onset Pompe diseases. Two non-pathogenic variants, c.1726 G>A (p.G576S) and c.2065 G>A (p.E689K) are of special interest, as they cause moderately reduced GAA activity without overt manifestation of Pompe disease. The presence of those mutations cast diagnostic challenge in newborn screening as well as in adult patients without typical clinical manifestation. We investigated the frequency of GAA pseudodeficiency allele in normal Korean populations to address the importance of those pseudodeficiency mutations. **Method:** One hundred and fifty control subjects without muscle weakness were enrolled. The DNA was extracted from peripheral blood leukocytes. The genotype analysis was performed by PCR-restriction fragment length polymorphism (RFLP), utilizing the enzymes HaeIII, BceAI, and TaqI. Sanger sequencing was performed in homo mutant of c.1726 G>A to confirm the genotype. The characteristic of compound heterozygote was determined through synteny test. **Results:** The c.1726 G>A mutation was found in forty-nine individuals (homo mutant 7.3%, hetero mutant 25.3%), giving the mutant allele frequency of 0.2. Fifty-six controls are harboring c.2065 G>A mutation (homo mutant 4.7%, hetero mutant 32.6%). The homozygous of c.1726 G>A and c.2065 G>A was found in 2 persons. The allele frequencies of this study are comparable to those

of Japanese population. **Conclusion:** We confirmed the pseudodeficiency allele is significantly prevalent in Korean population. These pseudodeficiency alleles have to be taken into account interpreting the enzyme activity and genotype of GAA, to prevent false positive diagnosis. Linkage of some potentially pathogenic variants with the pseudodeficiency allele is another topic worth investigation.

P-2-248

Genetic characteristics of Korean patients with amyotrophic lateral sclerosis using multi-gene panel testing

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Background & Objectives: Amyotrophic lateral sclerosis (ALS) is a rapidly progressive neurodegenerative disease involving motor neurons and mutations in a number of genes have been identified in patients with ALS. Since a growing number of genes have been identified in the genetic etiology of ALS, simultaneous screening of mutations in multiple genes is likely to be more efficient than gene-by-gene testing. In this study, we tested a multi-gene panel by using next-generation sequencing to define genetic characteristics of Korean patients with ALS. **Method:** We collected DNA samples from patients who were diagnosed as having ALS from 2010 to 2012. One hundred and fifty-two Korean patients with a clinical diagnosis of definite, probable, or probable with laboratory support ALS according to the revised El Escorial criteria were enrolled. All patients were of Korean descent. Multi-gene panel testing including 18 ALS-related genes (SOD1, SETX, FUS, ANG, TARDBP, TAF15, VCP, UBQLN2, SQSTM1, SIGMAR1, ALS2, FIG4, VAPB, OPTN, DAO, MAPT, SPG11, and GRN) was developed and applied to simultaneously identify mutations in 4 familial (fALS, 2.6%) and 148 sporadic (sALS, 97.4%) patients with ALS. **Results:** We identified four known mutations in SOD1, ALS2, MAPT, and SQSTM1 and 28 variants of unknown significance (VUSs) in nine genes. We found a known causative mutation in one of the four patients with fALS (25.0%, 1/4) and three of the 148 patients with sALS (2%, 3/148). Thirty-one patients (20.4%) had potentially pathogenic variants. Interestingly, there were 2 (1.3%) patients with more than two variants in different genes. **Conclusion:** These results suggest that multi-gene panel testing is a useful approach for mutation screening in ALS-related genes. Moreover, the relatively low frequency of mutations in known ALS genes implies marked genetic heterogeneity in Korean patients with ALS.

P-2-249

Genetic profiles in patient with nemaline myopathy

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Background & Objectives: Nemaline myopathy (NM) is a clinically heterogeneous congenital myopathy characterized by the presence of nemaline rods in the skeletal muscle fibers. We investigated the clinical characteristics and pathological features in Korean patients with NM. **Method:** The whole exome

sequencing (WES) and Sanger sequencing were performed to find out the pathogenic mutations. Fifteen patients were diagnosed through muscle pathologic findings. **Results:** Seven patients of classic congenital type showed the motor developmental delay and hypotonia at birth. Six patients showed mild childhood type with the gait difficulty. Two patients showed intermediate congenital type and one of them needed mechanical ventilation. Regarding the distribution of weakness, seven patients presented distal dominant weakness across the clinical type. Dysmorphic features including pes cavus, equinovarus, and elongated face were identified in eight patients. Hypertrophic cardiomyopathy was identified in one patient. Typical nemaline rods were recognized on light microscopy with muscle biopsy from 14 patients, while scattered distribution was found in 9 patients. However, we could identify the nemaline filamentous aggregation in electro microscope (EM) from 1 patient. One of them showed a mitochondrial abnormality in EM. Type 1 fiber predominance was in 14 patients, while there was type 2 fiber predominance and atrophy in 1 patient. Variation in fiber size was prominent in 9 patients. Genetic analysis revealed missense, nonsense, and frameshift mutations in NEB gene in 11 patients. A missense heterozygous mutation in TPM3 gene (c.32T>A, p.Met11Lys) and ACTA1 gene (c.715G>A, p.Glu239Lys) were also identified. However, we could not detect pathogenic mutation in 2 patients. This is most likely due to uneven coverage of WES especially between exon 82 and 105 of NEB gene, which contains highly repetitive sequences. **Conclusion:** In this study, the most severe motor weakness is correlated with increased variation of fiber size and scattered distribution of nemaline rods. However, the types of mutations did not prove significance among the clinical features. In genetic analysis in NEB, mapping error of WES should be considered in repetition sequences. Functional study will be required to prove pathogenicity related with types of mutations in the patients with NM.

P-2-250

Proteomic analysis of the alteration of nuclear cytoplasmic distribution of intracellular proteins in motoneuron cell lines expressing mutant SOD1 G93A

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Background & Objectives: The appropriate nuclear cytoplasmic localization of proteins is essential for eukaryotic cellular function and biological processes such as RNA processing, translation, protein-protein interaction and post-translational modification. Aberrant localization of proteins has been implicated in many human diseases, like cancer and neurodegenerative conditions. Evidence suggests that the cytoplasmic mislocalization of nuclear proteins including transactive response DNA-binding protein 43kDa (TDP-43) and fused in sarcoma (FUS) in amyotrophic lateral sclerosis and frontotemporal lobar degeneration is associated with neurotoxicity. In this study, we investigated the proteome-wide alteration of nuclear cytoplasmic distribution in the motor neuron cell lines expressing mutant human SOD1 (G93A). **Method:** The motor neuron-like cell line NSC34 transfected with wild or mutant human SOD1 (G93A) underwent for subcellular fractionation. The proteome in the nuclear and cytoplasm fractions were analyzed using liquid chromatography-tandem mass spectrometry. Abundance difference of proteins for genotype, subcellular fraction and their interaction were analyzed. Bioinformatics analysis using TargetMine and DAVID were used to understand the function of the identified proteins. Finally, immunoblots of the significant proteins that changed the subcellular distribution in mutant cells were

performed for validation. **Results:** In total, 11,216 peptides and 1,925 proteins were identified, with 23% of the peptides and 32% of the proteins being found in both nuclear and cytoplasmic fractions. Using the intersection data set common to both fractions, we found that a considerable number of proteins (79%) were differentially distributed in the nuclear versus cytoplasmic compartments in the wild type. A total of 37 proteins showed a significant alteration in the nuclear cytoplasmic distribution in the mutant cells ($p < 0.05$). The subcellular distribution in the mutant cells was shifted from the cytoplasm to the nucleus for the proteins of RNA transport and processing (Dhx9, Fmr1, Srsf3, Srsf6, Tra2b), whereas the opposite was the case for the pathways of protein folding (Cct5, Cct7, Cct8), the aminoacyl-tRNA biosynthesis (Farsb, Nars, Txnrd1), the synaptic vesicle cycle (Cltc, Nsf), the Wnt signaling (Cltc, Plcb3, Plec, Psm3, Ruvb1) and the Hippo signaling (Camk2d, Plcb3, Ruvb1). **Conclusion:** This study allows a comprehensive understanding of the nuclear cytoplasmic distribution of intracellular proteins in motor neuron cell lines, and their alteration by the mutant SOD1 expression. Further studies are warranted, in order to elucidate the pathomechanistic implications of the aberrant localizations of the candidate proteins.

P-2-251

A case of arterial type Thoracic outlet syndrome (TOS) presenting with an axillary pain

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Background & Significance: Thoracic outlet syndrome (TOS) is a complex syndrome that involves compression of the neurovascular structures as they pass from the base of the neck to the arm. Arterial type TOS is the most rare form. Here, we report a case of arterial TOS who present with an axillary pain. **Case:** An 18-year-old high school female student visited our hospital with recurrent right axillary pain during a year. Her symptoms used to be aggravated after carrying a heavy back pack and with an arm elevation posture. Physical examination showed a positive Adson maneuver on the right side. A palpable supraclavicular rib, pallor, necrosis and edema were not found. Neurological examination showed no motor/sensory deficits. Nerve conduction and electromyographic studies revealed no abnormalities. Laboratory findings were also unremarkable. A chest radiograph showed no cervical rib and abnormal first rib on both side. Arterial duplex scan with TOS maneuver (continuous scanning with neutral and abduction position of right arm) did not showed a significant reduction of blood flow in radial and brachial arteries. Computed tomographic (CT) angiography imaging with postural maneuver was performed. Moderate to severe compression of bilateral subclavian arteries by anterior scalene muscle was found and it was remarkable with an arm elevation posture. MRI revealed no abnormal findings of cervical spine and brachial plexus. Finally, we diagnosed her as an arterial type TOS. **Conclusions or Comments:** Arterial TOS is the rarest type, occurring less than 5% of all TOS, and it can cause ischemic pain mimicking various neurogenic diseases. A diagnosis of TOS is challenging because there is no single gold standard method. In our case, axillary pain was not a typical symptom of TOS and duplex scan failed to demonstrate a reduction of arterial flow, CT angiography with postural maneuver was helpful to find a dynamic vascular compression and to determine location of the structure producing compression.

P-2-252

Serial ultrasonographic findings of herpes zoster neuralgia with motor weakness

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Background & Significance: Herpes zoster (HZ), caused by reactivation of the latent varicella zoster virus (VZV) in the dorsal root is most common after the sixth decade of life. It usually presents as a unilateral dermatomal vesicular rash, associated with severe pain. VZV usually persists asymptomatic in the sensory ganglia of anyone who has suffered from chickenpox. It reactivates in about 25% of people to travel along the sensory nerve fibers causing vesicular lesions in the dermatome supplied by the nerve. One of the most common and debilitating sequelae of HZ is post herpetic neuralgia (PHN), defined as pain persisting more than 3 months after the rash has healed. PHN is one of the most common causes of severe neuropathic pain. The diagnosis of both HZ and PHN is usually made clinically on the basis of the characteristic rash and patient's symptoms. Recently, the ultrasound (US) has shown diagnostic information or additional data for peripheral nerve diseases. We report the results of a serial ultrasonographic study which showed sequential change of cross-sectional areas (CSA) of right median, ulnar and radial nerves in a patient with C5-8 ganglionopathy due to VZV reactivation, followed by PHN. **Case:** A 65-year-old man presented with right hand weakness and neuropathic pain (burning discomfort and hyperalgesia) starting after a HZ rash on the right forearm surface (C6-7 dermatome). He was treated with antiviral drugs and steroid in the acute phase of HZ. Clinical evaluation showed right finger flexion, hand grip, wrist flexion/extension weakness and allodynia at finger, hand and wrist. Nerve conduction studies showed, on the right side, reduction of median and ulnar nerve sensory action potential (SAP) amplitudes; on the left SAPs were normal. Sensory conduction velocities (SCV) were decreased on the right side. Median and ulnar motor nerve studies showed low compound muscle action potentials (CMAP) and slow conduction velocities. Hence clinical and neurophysiological findings were consistent with the suspicion of right C5-8 radiculoplexopathy due to reactivation of VZV. US study of both median and ulnar nerves with cervical roots was performed and the CSA of right median and ulnar nerves with cervical roots were significantly larger than those of the left. Two months after treatment, CSA of the nerves have decreased before improvement of NCS. Five months after treatment, CSA of the nerves have decreased more and partial improvement of NCS was observed. After nine months, the CSA of the nerves have nearly normalized compared to healthy side and more improvement of NCS was revealed. These findings were also founded at 13 month's follow-up study. This case report suggests that US may show nerve swelling in acute phase of herpes zoster neuralgia and sequential change of CSA may reflect the natural course of disease, along with clinical and electrophysiological improvement. **Conclusions or Comments:** Modern anatomic study by Watson, who compared autopsy tissue from patients with and without PHN, found degeneration of the spinal cord dorsal horn in patients with PHN. The virus or the associated inflammation may spread from the sensory ganglion to the spinal cord, as shown by various cases of myelitis and to the roots or peripheral nerves. US in Renna's prior case report showed reduction of nerve CSA. Probably the best explanation in previous case might be distal axonal degeneration because of ganglionopathy. To our knowledge, no US data on nerve imaging in PHN of acute stage are available in the literature. This case report suggests that HZ might cause ganglionic/nerve degeneration with initial demyelination resulting in "nerve hypertrophy". In conclusion, this case report suggests that US may show changes of CSA in PHN at acute phase and repeated study may be helpful to predict an improvement, earlier than electrophysiological study. Hence further studies are needed to reveal its diagnostic value.

P-2-253

Myasthenia gravis accompanied by adrenal gland tumor

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Background & Significance: It is unknown that hormone secreting adrenal gland can cause myasthenia gravis **Case:** 55-year old man visited our clinic due to episodic diplopia which aggravated at evening. He had long standing history of uncontrolled hypertension and taken antihypertensive medication including thiazide. 1 year before his visit, he had transient quadripareisis with hypokalemia (Potassium level 1.9 mmol/L). Anti-acetylcholine antibody test was negative. Repetitive nerve stimulation showed generalized decremental response at low and high frequency stimulation. Left adrenal gland mass was detected at chest CT scan incidentally. Hormonal test was compatible with primary hyperaldosteronism. Left adrenalectomy was done. Repetitive nerve stimulation test that was taken after surgery did not show decremental response. **Conclusions or Comments:** Active hormone secreting adrenal tumor may be cause of secondary myasthenia gravis. More extensive chest CT scan for detecting adrenal gland mass is warranted in myasthenia with uncontrolled hypertension.

P-2-254

Spontaneous temporal meningocele, a rare cause of facial synkinesis

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Background & Significance: Facial synkinesis is common after facial nerve injury of varying causes, such as Bell's palsy, surgical or non-surgical trauma, infection, etc. Here we report the clinical case of a young male who developed an ocular to oral and oculostapedial synkinesis without previous history of facial palsy. **Case:** A 19-year-old asian man complained of contraction of the right corner of his mouth during blinking from Jan 2015. He had no history of medical and neurological diseases and was not taking any medications. The patient had not a previous history of facial palsy and no signs of facial hemiatrophy were detected. The patient felt awkward and nervous about the involuntary movement of his face. He also complained of eye-watering while eating (crocodile tears or gustolacrimal reflex) and concomitant buzzing noise in his right ear during blinking. However, He did not complain of facial pain, sensory change, hearing loss, hyperacusis or facial muscle weakness. On physical examination, the smiling was completely symmetric, and all the facial muscles of the right side had a normal strength, including the orbicularis oris. Voluntary eyelid closure resulted in involuntary oral movement laterally. This action also served to deepen the nasolabial fold, making facial asymmetry. Examination of the nervous system did not reveal any other abnormality. Electrophysiological evaluation revealed normal blink reflex on both side and normal amplitude on facial motor study of orbicularis oculi, orbicularis oris and nasalis with normal latency. Lateral spread responses were detected when a mentalis muscle response was recorded upon stimulation of the zygomatic branch and when an orbicularis oculi response was obtained upon stimulation of the mandibular branch. Needle electromyography showed seldom denervation potentials and neurogenic motor unit potentials on right orbicularis oris muscles. A magnetic resonance imaging scan showed a temporal meningocele with T2 high signal intensity consistent with cerebrospinal fluid and widened labyrinthine portion of facial nerve canal. **Conclusions or Comments:** Synkinesis, an abnormal synchronous activity of muscles that normally do not contract together, with voluntary and reflex movements, is an important clinical marker of mis-reinnervation. We experienced a case of temporal meningocele that developed through abnormal lateral extension of subarachnoid space following the labyrinthine segment of facial nerve, producing injury of the facial nerve. Although its rarity, temporal meningocele should be considered on the differential list of the causes of facial synkinesis, especially when accompanied by an atypical presentation.

P-2-255**Alternating recurrent painful ophthalmoplegia as an idiopathic hypertrophic tentorial pachymeningitis**

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Background & Significance: Idiopathic hypertrophic pachymeningitis is a rare chronic fibrosing inflammatory disease characterized by diffuse thickening of cranial dura mater. Its usual clinical manifestations are headache, cranial neuropathy, ataxia, seizure and so on. We experienced a case manifested with recurrent painful ophthalmoplegia occurring on different side as a rare presentation of idiopathic pachymeningitis. **Case:** A 72-year-old woman visited due to double vision with left ocular pain. Neurologic examination revealed ptosis, hypertropia, lateral and inferior rectus muscle weaknesses on left eye. Brain MRI and MRA showed no cavernous enhancement or aneurysmal sac. Two months of oral prednisolone improved her symptom completely. After 7 months from the first attack, she felt double vision with right periocular pain. In neurologic examination, right superior oblique muscle weakness was detected. Follow up brain MRI showed thickened enhancement at right cerebellar tentorium. There was no significant abnormality in cerebrospinal fluid examination with normal pressure. VDRL, anti-HIV, Tb PCR, mycoplasma, aspergillus antigen and cryptococcus were negative. All rheumatological profile was negative. Repeated oral steroid was also effective to her right ocular pain and superior oblique palsy. **Conclusions or Comments:** Alternating recurrent painful ophthalmoplegia is caused by various kinds of neurological condition including diabetic cranial neuropathy, sellar mass, aneurysm or Tolosa-Hunt syndrome etc. Idiopathic pachymeningitis also will be one of the candidate for differential diagnosis and follow up imaging study show important clues in such cases.

P-2-256**Stiff-person Syndrome in a patient with myasthenia gravis after thymectomy : a case report**

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Background & Significance: Stiff person syndrome (SPS) is a very rare autoimmune or paraneoplastic disorder, characterized by continuous systemic muscle stiffness and painful paroxysmal muscle spasm. We report a case of SPS developed in myasthenia gravis (MG) after removal of thymoma. **Case:** A 48-year-old woman visited emergency room for generalized weakness and dyspnea. She was diagnosed with myasthenia gravis. Thymectomy was performed for a small thymoma found in the chest CT, and intravenous immunoglobulin (IVIg) treatment was initiated for myasthenic crisis. The histology of thymus was WHO grade B1 (Masaoka stage I), non-invasive cortical type thymoma. Six months after surgery, she was admitted due to dyspnea, continuous muscle stiffness and painful spasm aggravated by emotional stress. She also presented proximal muscle weakness, hyperhidrosis and urinary urgency. Anti-AChR antibodies was elevated to 10.184 nmol/L (reference range : <0.5 nmol/L). Anti-GAD antibodies was 300 U/ml (reference range : <1.0 U/ml). Additional diagnosis of SPS was confirmed. IVIG treatment partially relieved the stiffness and spasm. **Conclusions or Comments:** This is the first case report of serologically proven SPS in a patient with MG. The pathogenesis of SPS in this case was not certain. Thymoma might be the cause of SPS, but the possibility of autoimmune SPS triggered by thymectomy itself could not be excluded.

P-2-257**Facial palsy after dental procedure**Bom CHOI¹, Chae Young LEE¹, Bohee KIM¹, Im Suk KOH²¹Department of Neurology, Sacred heart hospital, Hallym university, ²Department of Neurology, National medical center

Background & Significance: Facial paralysis after dental anesthesia is rarely reported. It did not recover immediately, and was thought to be other cause rather than simple block effect of local anesthesia. **Case:** 53-year old woman visited our clinic due to right facial palsy. She had visited dental clinic and been injected local anesthesia. At injection, she felt severe pain at injection site. Procedure lasted about forty minutes. Immediate after procedure, her friend noticed distortion around her mouth. Six hours later, she could not close her right eye. She did not complain hyperacusis or change in taste. MRI which was done 24 hours after injection showed facial nerve enhancement in labyrinth portion in affected side. **Conclusions or Comments:** The mechanism of facial palsy after dental procedure is not only due to local anesthetic effect but combined with unique anatomical structure of facial nerve.

P-2-258**Clinical characteristics of MuSK-MG in Korea: comparison with double seronegative MG**Kee Hong PARK¹, Jung-Joon SUNG¹, Jin-Sung PARK², Ohyun KWON³, Jee-Eun KIM⁴, Byung-Jo KIM⁵, Kwang-Kuk KIM⁶, Dae-Seong KIM⁷, Jeong-Geun LIM⁸, Suk-Won AHN⁹, Jiyoung OH¹⁰, Jae Young AN¹¹, Tai-Seung NAM¹², Byung-Nam YOON¹³, Yoon-Ho HONG¹⁴

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Background & Objectives: Myasthenia gravis (MG) is an autoimmune disease caused by auto-antibodies against components of muscle membrane at the neuromuscular junction. Anti-acetylcholine receptor (AChR) antibody positive patients accounts for about 85% of generalized MG patients, while the seropositivity rate of MuSK antibody varies widely depending on the region and ethnicity. MuSK-MG is known to have different clinical manifestation and treatment response from AChR- MG. Here, we investigated the seropositivity rate of MuSK antibody and clinical characteristics of MuSK MG patients in Korea. **Method:** Serum samples of 57 patients (53 MG, 2 LEMS and 2 motor neuron disease) from 13 hospitals were collected and anti- MuSK antibody was assayed by commercial ELISA kit. Clinical characteristics of AChR-negative generalized MG patients were analyzed, comparing MuSK-positive and negative groups. **Results:** Among the MG patients, 38 patients were negative to AChR antibodies (6 ocular, and 32 generalized MG), and 15 were positive. Frequency of anti-MuSK antibody positivity was 28% (n=9) in AChR- seronegative generalized MG group, and none was positive to both antibodies. Noted was female predominance (89% vs. 44%), rare incidence of thymic abnormalities (0% vs. 30%), more frequent bulbar-dominant (78% vs. 44%, MGFA B classification) and severe (44% vs. 13%, MGFA class III or worse)

symptoms at presentation, less frequent use of acetylcholinesterase inhibitors (11% vs. 70%), and more frequent crisis (56% vs 13%) in MuSK-positive compared to double-seronegative patients. There was no difference regarding onset age, follow-up duration, current symptom severity measured using MG composite score, and the rate of remission between groups. **Conclusion:** MuSK-MG accounts for 28% of AChR-seronegative MG patients in Korea. Demographic and clinical features of MuSK- MG in Korea are remarkably similar to those previously reported in Western countries. Future studies are warranted for serological diagnosis and further characterizing of double seronegative generalized MG.

P-2-259

Charcot-Marie-Tooth (CMT) with Acute Demyelinating Disease

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Background & Objectives: The hereditary peripheral neuropathies are classified based upon clinical characteristics, mode of inheritance, electrophysiologic features, metabolic defect, and specific genetic markers. The primary hereditary neuropathies predominantly affect peripheral nerves and produce symptoms of peripheral nerve dysfunction. Hereditary motor sensory neuropathy (HMSN), also known as Charcot-Marie-Tooth (CMT) disease, is a spectrum of disorders caused by a specific mutation in one of several myelin genes that result in defects in myelin structure, maintenance, and formation. This disorders can affect both central and peripheral nervous systems rarely. **Method:** A 23 year old man was admitted with gait disturbance which episode developing for several days. He had quadriparesis with sluggish speech but his cognitive functions are intact. On his medical history, he was diagnosed hereditary motor and sensory neuropathy six years ago. Family history was significant in mother, maternal brother, maternal grandfather. Examinations revealed bilateral hammer toes, mild distal muscle wasting in feet and hands. Subjective sensations were normal but position and vibration sensation was mild impaired in both limbs. Reflexes were all absent in both extremities. **Results:** On magnetic resonance imaging of his brain, Diffusion restriction in both periventricular white matter, diffusion high signal intensity with increased T2 signal change in splenium of corpus callosum, bilateral periventricular and centrum semiovale. Demyelinating disease was doubtful and he was supplied oral steroids 60mg /day for 14 days. His dosages were tapering down day by day. He improved completely over one month. **Conclusion:** Charcot-Marie-Tooth Disease is an inherited, progressive disease of the nerves with weakness and numbness more pronounced in the legs than the arms. Rarely X-linked CMT can present neurologic deficits with signal changes in MRI like acute demyelinating CNS disease. we describe a patients who had CMT with acute demyelinating changes in white matters on brain MRI.

P-2-260

Diffusion-weighted MRI: useful diagnostic tool for early detection of spinal cord infarction

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Background & Objectives: Acute spinal cord infarction (ASCI) is rare, but can lead to severe neurological condition. According to recent studies, however, prompt treatments of ASCI such as hemodynamic augmentation, CSF decompression and hypothermia may reduce clinical deterioration and also lead to dramatic improvements. Thus, early detection of ASCI is important and, by

extension, is laying the foundation which can try the recanalization therapy as cerebral infarction. Although, diffusion weighted image (DWI) is using as a method of choice for early detecting patients with acute cerebral infarction, early use of DWI in the patients with spinal cord injury were relatively rare. Therefore, we assessed about clinical finding of ASCI patients, time course of diffusion abnormality and usefulness of DWI for early detection of ASCI. **Method:** We included thirty-eight patients who were diagnosed as ASCI at a tertiary hospital from October 2003 to June 2015. Through the review of medical records, we investigated the baseline characteristics, clinical time course and image findings of the patients. DWI was performed at 1.5 Tesla(T) and 3.0T within 4 hours to 8 days after the onset of clinical symptoms. **Results:** Mean age was 62 years and 60.5% (N=23) of patients were male. Presumed etiologies were: 1) iatrogenic cause: bronchial artery embolization or transarterial chemoembolization (N=8, 21%), 2) atherosclerosis: features of vascular disease on computed tomography (CT) aortic angiography or cerebral angiography (6, 16%), 3) arterial embolism or thrombosis (2, 5%). The cause of ASCI remained unclear in sixteen patients (42%). However, half of these had vascular risk factors such as hypertension, diabetes, cigarette smoking, hypercholesterolemia, history of cerebrovascular events, or history of coronary artery disease. On DWI and T2-weighted spinal cord images, ischemic lesion was most commonly observed in thoro-conus area (N=17, 44.7%) and also observed in thoracic area (N=9, 23.7%), in cervicothoracic area (N=7, 18.4%), in cervical area (N=5, 13.2%). Most common Lesion pattern of MRI (Magnetic Resonance Imaging) was anterior portion which was observed in twenty-nine patients (76.3%). Other lesion pattern of MRI were posterior portion in two patients (5.3%), central portion in one (2.6%), whole portion in four (10.5%) and unilateral portion in two (5.3%). Among the patients with Positive findings on DWI (N=28, 73.7%), two cases were hyper-acute stage which diffusion abnormalities were found at 4 and 5 hours after the onset of symptoms in spite of no signal change in T2 weighted image. twenty-six cases were acute stage which diffusion and T2 weighted images showed hyper-intense at 5 hours to 7 days after the onset of symptoms. one case was subacute stage which diffusion restriction with normalized ADC value was found at 120 hours after the onset of symptoms. Four cases were chronic stage which diffusion abnormalities were not found at 6 to 8 days after the onset of symptoms because of pseudonormalization. In the rest six patients, it was hard to distinguish the lesion because of artifact, image quality or small lesion size. As the time course of ASCI patients in hospital, on average, it took 9 hours to admit, 28 hours to take first MRI, 66 hours to take first DWI and 35 days to discharge. It took patients who had MRI scan without DWI at first, more time to diagnose ASCI and they need second or third MRI scan evaluation. **Conclusion:** Our study has shown that spinal cord infarcts: (1) had a clearly definite cause in more than 50% of patients; (2) were more frequently located in the anterior spinal cord; (3) were more often involved the thoacic area. Diffusion abnormalities can be found after a few hours in patients with ASCI and they may persist for about 1 week. Early performing of DWI is more economical and helpful in early detection of ASCI.

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Dynamic electromyography and gait analysis in patients with Charcot-Marie-Tooth neuropathy

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Background & Objectives: Charcot-Marie-Tooth disease (CMT) is a genetically heterogeneous group of diseases on the peripheral nervous system characterized by progressive and symmetrical distal weakness. One of the main

symptoms of CMT is a gait disturbance due to muscle weakness and atrophy. The purpose of this study is to evaluate the ambulatory function in young CMT1A patients. Furthermore, the usability of the dynamic EMG and gait analysis was assessed to correct gait disturbance of CMT1A patients. **Method:** A total of 20 individuals (10 CMT1A patients and 10 normal controls) in the age of 20s were enrolled in this study. Written informed consent was obtained from all the subjects according to the protocol approved by the Institutional Review Board of Samsung Medical Center. Gait motion with self-selected speeds consistent with the subjects' usual gaits was captured. Two gait cycles in each subject were used to analyze the gait and the averaged kinematic data of each group in the frontal, sagittal, and transverse planes. Simultaneously, the dynamic EMG was recorded from 6 muscles which are the gluteus medialis (GMED), gluteus maximus (GMAX), biceps femoris (BF), rectus femoris (RF), tibialis anterior (TA), and gastrocnemius medialis (GCM). The RMS EMG during the gait was calculated from the dynamic EMG and normalized with the maximum value of RMS EMG during the maximum voluntary contraction to determine the muscle activation. **Results:** In this study, we confined the subjects to CMT1A patients in their 20s so we could obtain homogeneous and comparable data to healthy controls with respect to kinematic pattern. To evaluate the gait deficit in CMT1A patients, the kinematic patterns were compared to normal controls in the frontal, sagittal, and transverse planes. Since muscle weakness in CMT1A begins in the distal limbs, gait disturbances are initially perceived in the ankle angle. The CMT1A group had less dorsiflexed ankle during the swing phase, which was considered to be a footdrop due to ankle dorsiflexor weakness. The maximum ankle angle for swing phase was significantly lower in the CMT1A group (2.8 ± 3.4 degrees) compared to the control group (6.5 ± 3.5 degrees) ($p = 0.008$). The infirmity of dorsiflexor also results in a dorsiflexion failure at initial contact with the ground: The ankle angle of the CMT1A patients at initial contact was -1.6 ± 3.6 degrees and that of the normal controls was 2.5 ± 4.1 degrees ($p = 0.062$). The muscle activation of the CMT1A group was higher than that of the controls with analogous patterns throughout a gait cycle. The activation of TA was significantly increased compared to other muscles and the increase was sustained for a full gait cycle. The increased and prolonged muscle activation can be explained by the motor neuron axonal sprouting due to the neuropathy, as well as the demand for a larger activation to compensate for the muscle weakness. Since the MRC scale is the most frequently used scale to estimate muscle force, we analyzed whether the distinctive parameters found in this study correlated with the MRC scale. We found a linear correlation between the maximum ankle angle during mid-swing and the MRC scale of the dorsiflexor. And the MRC scale also showed linear correlation with muscle activation during the swing phase of TA. **Conclusion:** We evaluate the gait disturbance in young CMT1A patients with kinematic data and muscle activations. Gait analysis provided various parameters that represent CMT1A patients' mobility. Moreover muscle activation can be used to evaluate the individual muscle functions. Therefore, the combined analysis of gait and muscle activation could provide crucial information to assess the patient's ambulatory function. In near future, application of both can suggest the way to correct the gait disturbances in CMT patients.

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Anti-GD1b antibody-associated acute motor conduction block neuropathy with reversible conduction failure

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Background & Significance: Guillain-Barré syndrome (GBS) has demyelinating and axonal subtypes with different immunopathologic mechanisms. Segmental demyelination and conduction block is a pathological and electro-

physiological feature of acute inflammatory demyelinating polyradiculoneuropathy (AIDP). Acute motor axonal neuropathy (AMAN) is characterized by an antibody-mediated primary axonal degeneration. However, conduction block has been described in some patients with anti-ganglioside antibodies, which was called as reversible conduction failure (RCF) since it rapidly recovers without further development of increased temporal dispersion or secondary axonal degeneration. We report a patient showing RCF in sequential electrophysiological studies, who was diagnosed as anti-GD1b antibody related GBS. **Case:** A 65-year-old man developed numbness and paresthesia on bilateral finger tips followed by progressive limb weakness. He had preceding upper respiratory infection 7 days before. He had no significant past medical history except for hyperthyroidism and depressive disorder. Neurological examination showed that motor weakness was predominant in distal limbs, and graded by MRC scale as follows: shoulder elevation 4-, elbow flexion 4-, wrist extension 3, finger fanning 3, leg elevation 3, knee flexion 4-, ankle plantarflexion 3, great toe flexion 4. Ataxia was detected on finger-to-nose and heel-to-shin examinations. Deep tendon reflex was all disappeared and pathologic reflex was not detected. Nerve conduction study revealed distal conduction block in left median nerve, and prolonged terminal latency and conduction block in left ulnar nerve. In peroneal nerves, F-wave latency was prolonged and motor conduction velocities were slowed. The study also revealed reduced sensory nerve action potentials (SNAP) in left median nerve and left ulnar nerve and no SNAP in left superficial peroneal nerve. In laboratory findings, serum IgG anti-GD1b antibody was positive. Cerebrospinal fluid analysis showed mildly elevated protein level (52.7 mg/dL) without pleocytosis (2 cells/mm³). After 5-day course of intravenous immunoglobulin rapid improvement in motor power was observed; shoulder elevation 4, elbow flexion 4, wrist extension 4-, finger fanning 3, leg elevation 4, knee flexion 4, ankle plantarflexion 4, great toe flexion 5. Through sequential nerve conduction studies conduction blocks were shown to be rapidly disappeared, which is compatible with RCF. **Conclusions or Comments:** This study represents anti-GD1b antibody-associated acute motor conduction block neuropathy (AMCBN) based on the presence of RCF. AMCBN is characterized by the presence of RCF on nerve conduction studies, and has been related to detection of anti-GM1 or GD1a antibodies. Since patients with AMCBN usually showed excellent prognosis, the disease has been considered as a mild form of AMAN. The RCF has known to be a physiologic conduction block caused by involvement of axolemma at Ranvier nodes. It is distinct that our patient clearly showed ataxia and abnormalities in sensory nerve conduction, which is correlated with high titer of anti-GD1b antibody. Serial electrophysiological studies are mandatory for identification of GBS subtypes and to elucidate the pathophysiological mechanisms of muscle weakness among demyelination, axonal degeneration and physiologic conduction block. We underline that serial NCSs might be required for proper characterization of clinical and electrophysiological features.

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GNE myopathy showing diffuse lumbosacral root thickening

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Background & Significance: GNE myopathy is an autosomal recessive muscle disease caused by mutations in GNE, a gene encoding for UDP-N-acetylglucosamine 2-epimerase and N-acetylmannosamine kinase, which are key enzymes in sialic acid biosynthetic pathway. Typically, it presents with slowly progressive distal weakness in early adulthood, however it scarcely affects the quadriceps muscles. Characteristic pathological finding includes 'rimmed' vacuole. Although such information is quite helpful for the correct identi-

fication, the rarity of the disease may delay the diagnosis. **Case:** A 19-year-old woman was referred our department because of slowly progressive distal leg weakness gait for 2 years. She had difficulty in fast walking for 1.5 years, reported frequent falls for 1 year and atrophy of calf muscles for about 6 months, without any sensory deficits. She had neither preceding infection nor family history. Neurological examination showed distal weakness of both lower extremities with foot drops and slightly decreased deep tendon reflexes without sensory abnormality. Slow motor conduction velocities on the bilateral peroneal and tibial nerves, with compatible range of demyelinating neuropathy (28 m/sec), were noted in nerve conduction study. Conduction block or temporal dispersion was not noted. Following lumbar spine MRI revealed hypertrophy of diffuse lumbosacral roots without enhancement. Referring physician's opinion was chronic inflammatory demyelinating polyradiculoneuropathy or Charcot-Marie-Tooth disease despite of lack of sensory abnormality. However, myopathic changes were shown in the electromyography (EMG) performed in the tibialis anterior, peroneus longus, and extensor digitorum communis muscles with fibrotic change in the gascrocnemius muscle. Serum CK level was minimally elevated of 233 U/L. CSF findings were normal, and anti-GM1 and anti-MAG antibodies were not detected. Muscle biopsy obtained from the right peroneus longus muscle was conclusive of distal myopathy, showing marked fiber size variation with some degenerating, regenerating and atrophic fibers, mild endomysial fibrosis, and vacuolar changes. Subsequent genetic analysis confirmed GNE myopathy with compound heterozygous mutation (c.662T>C, c.1807G>C). Peripheral myelin protein 22 gene duplication was not detected. We tried intravenous immunoglobulin treatment to supply Neu5Ac. Though individual muscle strength was not dramatically improved, gait became markedly stable. She has been doing well with oral supplement of sialic acid (3,000 mg/day). **Conclusions or Comments:** The reason why motor nerve conduction slowing and lumbosacral root thickening were presented in GNE myopathy is obscure. But these findings could lead an initial diagnosis as a motor neuropathy rather than myopathy. Clinical suspicion and needle EMG were crucial in this case. Timely and accurate diagnosis of GNE myopathy has become more important since recent ongoing clinical trials have shown the improvement after metabolic supplementation and gene therapy.

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Multifocal motor neuropathy in patient with rheumatoid arthritis receiving infliximab treatment

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Background & Significance: Tumor necrosis factor (TNF) alpha inhibitors are used as immunosuppressant in some medical conditions, especially in rheumatologic disease. They have been associated with diverse adverse effects, rarely including peripheral neuropathy. Multifocal motor neuropathy (MMN) is a chronic and progressive motor neuropathy characterized by motor conduction blocks in nerve conduction studies with normal to nearly normal sensory nerve findings. We report herein a patient who developed a multifocal motor neuropathy after using a TNF - alpha inhibitor. **Case:** 58-year-old woman came to outpatient complaining about the weakness of extremities. Suffered from rheumatoid arthritis (RA) about ten years, she had dosed MTX for 5-years ago. At first, the MTX was sufficient for her disease. But, 2-years ago, her RA progressed rapidly. So rheumatologist added infliximab to control it. 1-year after the beginning of infliximab, she felt weakness in left leg, spreading to all the other extremities. Relatively involving distal part of the extremities, it progressed very slowly. On neurologic examination, she showed right and distal dominant four-extremity weakness, resisting to gravity. On a sensory exam, subtle decrease in pinprick sensation below knee level of both

legs was present. Nerve conduction study (NCS) showed conduction block in multiple nerves, especially in bilateral median nerves, and nearly normal SNAPs. Both findings were suggestive of multifocal motor neuropathy. The ganglioside antibody, especially Anti-GM1 antibody was negative. The Brain MRI was normal. There were no significant findings on other laboratory exams. Treatment plans were discontinuation of infliximab and use of intravenous gammaglobulin (IVIg). She discharged without improvement in the short term. Follow-up NCV after 2 months showed no interval change. But her symptoms improved progressively. **Conclusions or Comments:** The reports overseas are suggestive of the relationship between infliximab and multifocal-motor-neuropathy-like manifestation. To our knowledge, there are no reports about above description in South Korea. It may be the first time to report the multifocal-motor-neuropathy-like findings in patients who treating with infliximab. Infliximab is an emerging drug for treating many inflammatory diseases. Neurologists have to keep their mind that when they meet patients with progressive limb weakness using infliximab, it can be a possible cause for their weakness.

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Frontotemporal dementia with motor neuron disease in a patient with antiphospholipid syndrome :cases report

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Background & Significance: Frontotemporal dementia (FTD) is a syndrome of progressive changes in behavior and language due to loss of function of neurons in the frontal and temporal lobes. In about 10-15% of patients with FTD, the disease also involves the nerve cells controlling voluntary movement, called motor neurons. When this happens, the syndrome is called FTD with motor neuron disease (MND). FTD with MND is common but FTD with MND in antiphospholipid syndrome (APS) is very curious. A 71-year-old male person who had been diagnosed in FTD with MND, was positive in lupus anticoagulant test. To the best of our knowledge, this is a very rare case of FTD with MND in APS. **Case:** A 71-year-old man presented with 2 years history of right upper extremity weakness, 1 year history of left upper extremity weakness and 6 month history of both lower limbs weakness was admitted to out neurology department in October 2014. When the patient referred to our hospital, he was wheelchair ambulation and could stand for a few seconds. He had underlying diseases; epilepsy, angina, pulmonary thromboembolism (PTE). There was no family history of similar disease or exposure to any toxins or drugs. Physical examination was unrevealing and mentation was normal. Weakness of upper limbs with Medical Research Council (MRC) grade 4 and weakness of lower limbs with MRC grade 3~4, proximally and distally. He had slowly progressive motor weakness. Upper motor neuron sign and lower motor neuron sign were seen together. To rule out MND, Electromyography (EMG) was done and EMG were seen denervation on all region. Based on EL Escorial criteria, MND was made because upper motor neuron sign was seen in cervical region (Hoffman sign) and lower motor neuron sign were seen in bulbar (Dysphagia, Tongue atrophy and fasciculation) and lumbar region (Lower extremity atrophy and fasciculation). He also had memory impairment and apathy so evaluation his cognition, SNSB and Brain PET CT was done and showed frontal lobe and temporal lobe dysfunction. We concluded that he had FTD with MND. To rule out secondary causes of MND, further laboratory tests were done. Lupus anticoagulant was positive, anti-cardiolipin Ab and Anti-B2 glycoprotein 1 were negative. He was diagnosed APS because he had clinically episode of PTE and laboratory lupus anticoagulant. He was treated anticoagulant because he has APS and PTE. He was also took conservative treatment on FTD with MND but there was no improvement of symptoms and signs. **Conclusions or Comments:** We think that this is the first

case with FTD with MND in APS in Korea. Up to know, Aetiology and pathogenesis of MND are still uncertain. Among numerous hypotheses, autoimmune mechanisms are taken into account. Antiphospholipid antibodies may also be important in different neurological symptoms such as stroke, seizures, dementia, chorea, transverse myelopathy, migraine, ocular ischemia and cerebral phlebitis. Other associations, like Guillain-Barre syndrome, and MND are more controversial. This association is not simple but possibly a causal correlation; antiphospholipid antibodies may urge their effect immunologically or by thrombosis of small arterioles and venules, microinfarcts of motor neurons and resultant development of MND manifestations. These hypotheses lead us to elucidate the autoimmune pathogenesis and explanation for immunosuppressive therapy in MND. Further evaluation and evidence in the pathophysiology of the disease must be needed.

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Anterior interosseous nerve palsy in neuralgic amyotrophy due to fascicular involvement of the median nerve proper

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Background & Significance: Anterior interosseous neuropathy (AION), a pure motor palsy of the flexor pollicis longus (FPL), flexor digitorum profundus (FDP) to the 2nd and 3rd fingers, and pronator quadratus (PQ) has long been known as one of frequent presentations of neuralgic amyotrophy (NA). Being one of terminal branches of the median nerve, a causative lesion for isolated involvement of the anterior interosseous nerve is usually considered to reside in itself, not in the proximal median nerve proper. Against this long-held assumption, we have recently identified two cases of AION in the clinical setting of NA whose lesions were in the median nerve proper in the upper arm. **Case:** Both cases, 35 year-old and 25-year-old men, had quite similar clinical presentations. man was visited to the our department for weakness of right hand grip and upper arm pain. The neurological examination showed weakness of isolated flexion of the interphalangeal joint of the thumb and distal interphalangeal joint of the index(MRC grade I). Pronation and supination were normal. Electrophysiological studies confirmed the motor deficit in the distribution of the AIN, with axonal loss of the flexor pollicis longus, flexor digitorum profundus, pronator quadratus. However, there are not decreased in sensory potential amplitudes. Radiologic study was performed one week later. On MRI, anterior interosseous neuropathy is evident when on T2-weighted image increased signal intensity in the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus muscles and nerves are visible. So we concluded that location of lesion is proximally to anterior interosseous nerve, that is median nerve trunk. **Conclusions or Comments:** In cases of AION, radiological evaluations such as MRI or US are strongly recommended because AION can result from fascicular involvement of the median nerve proper as shown these cases. In the same context, the other proximal lesion than the median nerve might manifest as one of clinical syndromes of its distal branch presentations, especially in the clinical setting of NA. In selected cases, direct lesion visualization covering parent nerves is recommended in addition to clinical and electrophysiological evaluation unless there is other clear evidence of localization.

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Colchicine induced myoneuropathy manifested by polyradiculoneuropathy

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Background & Significance: Colchicine is the one of the most available drugs for treatment of gout and rheumatic disease. Neuromyopathy induced by colchicine is rare and usually presented with subacute onset of proximal weakness, generalized symptoms and elevated serum creatine kinase (CK). Electrophysiological study can support the diagnosis by showing length dependent pattern of sensorimotor polyneuropathy and 'irritable myopathy' in electromyography (EMG). We report a patient with colchicine myoneuropathy mimicking recurrent polyradiculoneuropathy. **Case:** A 57-year-old female admitted because of acute progressive quadriparesis following common cold. She has been taking colchicine for 4 years for treating Behcet disease. She had bilateral severe dorsiflexor (MRC grade 1/5) and plantarflexor (MRC grade 2/5) weakness with generalized hyporeflexia. Electrophysiological study had supported the diagnosis of acute axonal polyradiculoneuropathy. Clinical symptoms and electrophysiological findings had been recovered after intravenous immunoglobulin therapy. Eight months after the event, she had complained relapsed weakness of both lower limbs. The symptom had been started on bilateral proximal region; hip flexors (MRC grade 2/5) and knee extensors (MRC grade 2/5) with severe myalgia. Prominent paralytic ileus with severe constipation and abdominal pain was accompanied with limb weakness. Serum CK was elevated up to 1349 U/mL (normal range 52-336 U/mL). Nerve conduction study was compatible with axonal polyradiculoneuropathy but myotonic discharges with mixed neurogenic and myopathic motor unit action potentials were notes in EMG. Muscle biopsy obtained in the deltoid muscle revealed atrophic myofibers with numerous vacuoles. We concluded colchicine myoneuropathy on the basis of clinical and laboratory findings. Her neurological deficits improved with normalization of serum CK level after discontinuation of colchicine. **Conclusions or Comments:** We had some difficulties in making a prompt diagnosis of colchicine myoneuropathy, because of previous history of acute axonal polyradiculoneuropathy. We first thought recurrent polyradiculoneuropathy and considered additional immunotherapy. Severe ileus, relatively uncommon in chronic inflammatory demyelinating polyradiculoneuropathy, elevated serum CK, and myotonic discharges in EMG gave us a clue. Though colchicine myoneuropathy is rare, it should be included as a differential diagnosis in any patient with polyradiculoneuropathy who has treated with colchicine, because discontinuation of colchicine is ultimate for treatment.

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Surfer's myelopathy- An unusual case of nontraumatic myelopathy

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Background & Significance: Surfer's myelopathy (SM) is a rare acute spinal cord ischemia caused by kinking of blood vessels supplying spine by hyperextension of the back. The SM was named because this condition is most often seen in surfing beginners. Here we describe a case with SM that was needed to differentiate from neuromyelitis optica spectrum disorder with long extensive transverse myelitis and spinal cord infection. **Case:** A previously healthy 34 year-old man was transferred for complete paraplegia and fever from a hospitals in Hawaii, where he took a surfing lesson for the first time in his life. About 1 and half hours after his first surfing lesson, he felt back pain radiating down to his lower limbs without any traumatic events. The back pain was progressed to complete paraplegia and total sensory loss on both legs in 30 minutes. Next day, he was transferred to our hospital by airplane. On admission day 1, his body temperature was 38.0 °C. Neurologic examination revealed complete paraplegia as MRC grade 0, decreased deep tendon reflexes on both knee and ankle, T10-level sensory deficit, and absence of rectal tone. His bladder was

distended. Initial MRI taken 2 days after symptom onset showed T2 hyperintensity from T5 to the conus medullaris. CSF analysis revealed white blood cell count of 280/ μ L, protein of 76.5 mg/dL, IgG index of 0.683, and an absence of oligoclonal bands. Brain MRI was normal. Immunologic profile including NMO-IgG antibody was all negative. Due to fever and CSF pleocytosis, infectious myelitis was initially suspected. He was treated with intravenously administered broad-spectrum antibiotics, acyclovir, and high-dose steroids. Despite of treatments including high dose steroid pulse therapy of 2 cycles for 2 weeks, no neurologic improvement was observed. Follow-up MRI at 2 weeks showed more increased T2 weighted signal intensity on previous lesion and new abnormal enhancement from T10 to T12 level. Spine diffusion weighted image showed restricted diffusion at the affected level, suggesting spinal cord infarction. His clinical status has not been significantly changed despite of rehabilitation for 2 months until now. **Conclusions or Comments:** Although there was some evidence for infection, sudden onset of symptoms after surfing, restricted diffusion on DWI, and lack of any improvement in neurological symptoms led us to a diagnosis of SM rather than infectious myelitis. Increased awareness of this injury may be able to make physicians to recognize the SM earlier.

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Phenotypic and genetic profiles in 121 Korean patients of X-linked dominant Charcot-Marie-Tooth disease type 1

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Background & Objectives: Charcot-Marie-Tooth disease (CMT) is clinically and genetically heterogeneous hereditary neuropathies characterized by progressive distal muscle weakness, sensory loss, and areflexia. CMT is mainly divided into three groups including demyelinating (CMT1), axonal (CMT2), and intermediate type (CMTX) neuropathy. Mutation in Gap junction protein, beta 1 (GJB1) is the genetic cause of Charcot-Marie-Tooth disease type X1 (CMTX1), the second most frequent CMT. In this study, we analyzed clinical characteristics and genotype-phenotype correlation in a cohort of Korean CMTX1 with 1092 patients from 604 families. **Method:** We enrolled and reviewed clinical features of a Korean CMT cohort with 2,044 individuals (1,092 affected and 952 unaffected) from 604 unrelated families. **Results:** A total of 121 patients (64 male and 57 female) from 58 unrelated families have a mutation in GJB1, and the genetic frequency is 9.6%, which is higher than our previous report. In addition, the frequency is 14.8% when calculated within genetically identified cases. Among them, 22 females were asymptomatic. Clinical features from 99 symptomatic patients include foot deformity, ataxia and tremor with a frequency of 86.9%, 42.4%, and 35.4%, respectively. Because of the locus of GJB1 (Xq13.1), the inheritance occurs as X-linked dominant with gender dependent phenotypic severity. Besides 38.6% of asymptomatic female, symptomatic female also exhibited milder phenotype compared to male patients. There were significant differences in age at onset, FDS, CMTNS, foot deformity, and ataxia between genders. Electrophysiologically, MNCV and CMAP in motor nerve were significantly reduced in male patients. Genetically, a total of 39 mutations were found including 35 missense mutations and 4 stop/frameshift mutations. Among them, 31 mutations were previously reported and 8 were novel mutations. Ten mutations were found from more than two independent families. V95M and R164Q were found from six families. Structurally, Cx32 is divided into 9 domains: 3 intracellular (IC), 4 transmembrane (TM) and 2 extracellular (EC) domains. Although mutations were found in all domains, extracellular domain 2 (EC2) has the

highest frequency with 12 mutations (38.7%) from 23 families (39.7%), while transmembrane domain 4 (TM4) has only one mutation. To determine genotype-phenotype correlation, we evaluated phenotypic severity of male patients according to GJB1 domain. A total of 56 male patients who had full medical records were divided into 3 or 9 groups according to the mutation sites. Patients with mutation in EC domain has the most severe phenotype and mutation in IC domain exhibited mildest phenotype. There were significant difference in MNCV of median and ulnar nerve among the domains. When analyzed in 31 female patients, domain dependent phenotypic severity was similar to male patients, however, significant difference was found only in SNAP of median sensory nerve. **Conclusion:** CMTX1 is the second most common CMT in Korea, which implies that genetic examination of GJB1 should be included in the diagnosis of CMT. There are strong gender-dependent and partial genotype-dependent phenotypic severity in Korean CMTX1 patients. In conclusion, this is the first large-scale report on Korean CMTX1 patients by characterizing genotype and phenotype features, therefore, this report will provide crucial information on diagnosis and treatment of CMTX1.

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Clinical efficacy of pulsed radiofrequency neuromodulation for intractable meralgia paresthetica

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Background & Objectives: Meralgia paresthetica (MP) is a neurologic disorder of the lateral femoral cutaneous nerve characterized by a localized area of paresthesia and numbness on the anterolateral aspect of the thigh. In most patients with MP, successfully managed with conservative treatment. However, small group of MP patients who are refractory to medical treatment, more aggressive treatment with lower risk should be considered. The objective of this study was to evaluate clinical outcomes of pulsed radiofrequency (PRF) neuromodulation to the lateral femoral cutaneous nerve (LFCN) in MP patients who refractory to conservative treatment. **Method:** We retrospectively reviewed clinical data of 11 patients with medically intractable MP who underwent PRF neuromodulation of the LFCN. These MP patients underwent diagnostic LFCN block using 2.0% lidocaine. Temporary pain relief > 50% was considered a positive response to the diagnostic nerve block. After the positive results of the diagnostic nerve blocks, these patients underwent PRF neuromodulation at 42°C for 2 min. The pain of the patients was evaluated by 10cm Visual Analog Scale (VAS). From all MP patients who received PRF, pain measured VAS scores, and complication during at least six or more months were collected for statistical evaluation. **Results:** Although the mean initial VAS scores of the patients were 6.4 ± 0.97 cm, it was decreased to 0.91 ± 0.70 cm, 0.82 ± 0.75 cm, and 0.63 ± 0.90 cm at 1, 3, 6 month follow-ups, respectively ($P < 0.001$). Sixty-four percent of patients achieved complete pain relief (free of pain) in the last follow-up, whereas twenty-seven percent of patients achieved successful pain relief (VAS \geq 50% reduction in pain). Furthermore, we did not observe any complications after procedure. **Conclusion:** PRF neuromodulation of the LFCN provide immediate and long lasting pain relief effect without complications. Therefore, PRF of LFCN can be used as an alternative treatment in patients with MP who are refractory to conservative medical treatment.

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A case of Guillain-Barre syndrome with pain dominant initial symptoms

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Background & Significance: Guillain-Barre syndrome (GBS), the most frequent cause of acute flaccid paralysis worldwide. GBS is usually a predominantly motor disorder with areflexia and subjective more than objective sensory symptoms. Pain is a common but underappreciated symptom in GBS. We report a case of Guillain-Barre syndrome with pain dominant symptoms. **Case:** 62-year-old female patient visited the outpatient generalized pain and complained of diarrhea and for a week. The patient visited the hospital before the day accompanied by a generalized weakness. The patient was difficult to walk generalized muscle weakness. Previous her medical history is none. Patients are accompanied by diarrhea and generalized pain for a week and were treated at another hospital with gastroenteritis. At the time the patient has received treatment and outpatient hospital three days. The symptoms of diarrhea patients was improved a lot. For patient who did not know the specific cause of enteritis. But the patient did not improve generalized pain. The patient complained of generalized muscle weakness and pain the day before the hospital visit. The patient complained of severe pain in the lower back and a lot of pelvis were arms and legs had also complained of pain. Patients with limb muscle strength was MRC grade 4, hyporeflexia findings showed. The patient underwent magnetic resonance imaging of the spine and brain examination revealed no abnormalities. Patient showed normal CSF finding. Nerve conduction study and electromyography in patients with the findings of acute diffuse peripheral neuropathy associated with motor dominant partially axonal involved was shown findings. We diagnosed with Guillain-Barre syndrome with pain dominant initial symptoms. Patients were treated intravenous immunoglobulin. The patient has been hospitalized for a week after the treatment of symptoms of pain and muscle weakness worse. Patients with limb muscle strength was MRC grade 3. 2 weeks later, the patient is not worsening of symptoms were concurrent physical therapy. 3 weeks later, the patient was pain in the lower back and pelvis is reduced. A month after admission, the patient was discharged to outpatient and ambulatory possible to walk through the brace. Currently the patient is under physical therapy and symptomatic treatment, while outpatient hospital visits. **Conclusions or Comments:** Guillain-Barre syndrome is a rare disorder. Pain may precede the onset of weakness by 2 weeks in 1/3 of patients, and about 2/3 of patients have modest discomfort early in the illness. The discomfort in GBS has been described as aching, usually confined to muscles of the back, hips, or upper legs. We have experienced the above symptoms, we report on the case.

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Phenotypic characteristics of Charcot-Marie-Tooth type 1E (CMT1E) patients from cohort study

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Background & Objectives: Charcot-Marie-Tooth disease (CMT) is clinically and genetically heterogeneous disorders. Point mutations of PMP22 are known to cause CMT type 1E. In this study, we investigated the phenotypic characteristics in 23 patients from 15 families carrying PMP22 point mutations. **Method:** Patients were evaluated by taking a detailed history and neurologic examination including motor and sensory impairments, deep tendon reflexes, and muscle atrophy. Muscle strength of limb muscles was assessed manually using the standard medical research council scale. The motor

and sensory nerve conduction studies (NCS) were performed on the median, ulnar, peroneal, tibial, and sural nerves of four extremities using a standard method. Their mutations were identified by the exome sequencing. **Results:** A total of 23 patients were recruited from 15 Korean families. The mean age was 32 years and the gender ratio was 42 percent female and 58 percent male. Their onset age varied from 6 months to 40 years. We identified 9 different mutations including one novel mutation after filtering 200 Korean control exomes. 15 patients showed markedly severe clinical manifestations; early onset age, prominent delayed motor development, generalized areflexia, and high functional disability score. On the other hand, 8 patients showed an adult-onset and from mild to severe neurologic deficit. Four patients of them suffered from spasm and neuropathic pain and other two patients had hearing impairment. **Conclusion:** Our 23 patients with PMP22 point mutations presented genotypic and phenotypic diversity. This study may be the basis in establishing the clinical, electrodiagnostic, and molecular characteristics of patients with PMP22 point mutations.

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Ocular myasthenia gravis in patients with sarcoidosis

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Background & Significance: Neurologic complications occur in 5 to 10 percent of patients with sarcoidosis. Neurosarcoidosis is a diagnostic consideration in patients with known sarcoidosis who develop neurologic deficits. Common syndrome of neurosarcoidosis include a cranial neuropathy, neuroendocrine dysfunction, encephalopathy, myelopathy, peripheral neuropathy and myopathy. Diagnostic evaluation requires distinguishing neurosarcoidosis from a variety of other inflammatory, neoplastic, and infectious condition. **Case:** A 52-years-old female came with history of drooping eyelid. She had been diagnosed with pulmonary sarcoidosis in 1990 and was being treated medically with oral prednisolone (7.5mg/day). The diagnosis of ocular myasthenia gravis was confirmed by positive immunology. Patient's acetylcholine receptor antibody was 0.81 nmol/L. After administration of pyridostigmine, ptosis was improved slightly. **Conclusions or Comments:** Ocular myasthenia gravis in patient with sarcoidosis is rare case. MG should be considered cause of cranial nerve dysfunction in patient with sarcoidosis.

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Exome sequencing reveals compound heterozygous DYSF mutations in a myopathy family with decreased acid-alpha glucosidase activity

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Background & Significance: We describe herein the application of whole exome sequencing (WES) for the molecular genetic diagnosis of a large Korean family with recessively inherited myopathy. **Case:** The proband (a 57-year-old woman, II-2) and her sister (a 54-year-old woman, II-3) presented to our neurologic clinic with proximal muscle weakness. They recalled very active and sporty life since adolescence. At the late teens, they first noticed difficulty to climb stairs and calf atrophy. They felt proximal arm weakness at early 40s, and were wheelchair-bound at early 50s. Neurological examination revealed muscle weakness and atrophies of proximal muscles, predominantly at lower limbs than upper limb muscles. Sensitivity to pinprick, touch, position and vibration were normal. Knee and ankle jerks were decreased. No pyramidal or cerebellar signs were detected. Electromyography revealed chronic

myogenic process. Elevated serum CK levels were revealed (789 IU/l) in the proband. In addition, they had decreased acid-alpha glucosidase activity (Total acid-alpha glucosidase, 19.6 nmol/hr/mg protein in II-2 and 17.0 nmol/hr/mg protein in II-3; Total acid-alpha glucosidase, 5.5 nmol/hr/mg protein in II-2 and 8.4 nmol/hr/mg protein in II-3). However, the acarbose/total ratio were the lower limit of normal (27.9% in II-2 and 49.4% in II-3). WES and subsequent capillary sequencing identified a compound heterozygous mutations (c.5713C>T and c.937+1G>A) in DYSF, which was previously reported to be an underlying cause of Bethlem myopathy. In addition, compound heterozygous pseudomutations (c.1726G>A and c.2065G>A) in GAA were found. **Conclusions or Comments:** We identified compound heterozygous DYSF mutations in a recessive myopathy family with decreased acid-alpha glucosidase activity. This suggested the importance of massively parallel sequencing for the diagnosis of inherited myopathy.

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Clinical Heterogeneities in NEFL Mutations from 24 Korean Patients with Charcot-Marie-Tooth disease: CMT type 1F, CMT type 2E, and intermediate type CMT

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Background & Objectives: Charcot-Marie-Tooth disease (CMT) is clinically and genetically heterogeneous inherited neuropathies characterized by progressive distal muscle weakness, sensory loss, and areflexia. Mutations in the neurofilament light chain polypeptide (NEFL) gene are known to cause Charcot-Marie-Tooth type 1F (CMT1F) and Charcot-Marie-Tooth type 2E (CMT2E). Recently, one Spanish family with NEFL mutation was reported to have intermediate CMT. However, further ICMT cases with NEFL mutation had not been reported, and clinical heterogeneities of NEFL mutations were not well established. We had found three families with ICMT caused by NEFL mutations. In this study, we investigated the clinical and genetic differences among CMT1F, CMT2E, and ICMT. **Method:** We enrolled 24 patients from 12 families with NEFL mutations identified with whole exome sequencing (WES). Nerve conduction studies (NCS) were measured with surface electrode. A magnetic resonance imaging (MRI) of lower-limb and pelvic musculature was done. **Results:** 5 families (E396K, P22R, P22T, and P8L mutations) had CMT1F phenotype and 4 families (E396K, L333P, Y443N, and K467N mutations) showed CMT2E phenotype. Also, three families (E396K and I384) were associated with ICMT. The mean onset age of CMT1F, CMT2E, and ICMT were 15.8±11.4, 13±7.4, and 8.0±6.4, respectively. The gender ratio was 42 percent female and 54.2 percent male. The mean motor nerve conduction velocities (MNCV) of median nerves among each type presented significant differences: the median nerve MNCV of CMT1F was 20.0±10.6, MNCV of CMT2E was 49.5±9.1, and MNCV of ICMT was 38.4±4.2. The sensory nerves were more severely affected than motor nerves, but sensory nerves of CMT1F were most impaired. The annual changes of MNCV, reflecting disease progression, were minimal in all types. Muscle weakness was most severe in CMT1F and deep tendon reflexes (DTR) were decreased or absent in all cases. Pes cavus was most commonly observed skeletal deformity and the frequencies of pes cavus in each groups were not significantly different. The joint contracture and developmental delay were mainly observed in CMT1F and the hearing loss was found in ICMT. MRI revealed length-dependent axonal degeneration. E396K mutation was a hotspot mutation in NEFL gene in that 10 patients from 5 families (41.7%) were associated with E396K mutation. 58.3 percent of mutations lied in rod domain and 25.0 percent of mutations

was in head domain. 16.7 percent of mutations was detected in tail domain. Among them, the mutations, including E396K and I384F, causing ICMT lies in Rod domain. **Conclusion:** As far as our knowledge, this is the second report about intermediate type CMT with NEFL mutations. We suggested that E396K mutation is the mutational hotspot of NEFL gene relevant to CMT1F, CMT2E, and ICMT. This study broadens the genetic and clinical spectrum of clinical features in NEFL mutations and will be useful in performing exact molecular diagnostics of CMT patients.

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A case of recurrent Tolosa-Hunt Syndrome

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Background & Significance: A recurrent isolated oculomotor nerve palsy is an extremely rare clinical condition that to date has been described in ophthalmoplegic migraine, possible nerve compressive conditions (ethmoidal mucocele, neurofibromatosis type 2, recurrent hemorrhage in pituitary adenoma, pseudotumor cerebri, carotid basilar anastomosis, a Reye-like syndrome), essential mixed cryoglobulinemia, diabetes mellitus, and postvaccinal. In Tolosa-Hunt syndrome, recurrent oculomotor nerve palsies also may occur. We report on the case of a 53-year-old man presenting with repeated unilateral internal and external oculomotor nerve palsies caused by unknown origins. We think this patient recurrent Tolosa-Hunt Syndrome. **Case:** 53-year-old male patient complained of headache accompanied by pain in the whole body weakness and eye two days ago. A day ago, the patient complained of diplopia. The patient complained about right ptosis, ocular pain, headache and diplopia. The patient had been taking hypertensive medication as there was no other disease. Brain magnetic resonance imaging in patient with no specific findings. Cerebrospinal fluid (CSF) of patients also had normal findings. Other tests were normal. Viruses marker test and bacterial culture tests were normal. We thought the patient to Tolosa Hunt Syndrome and underwent steroid therapy. Three days later, the patient was symptomatic improvement. The patient was discharged six days later. The patient showed no symptoms at 2 months. 60 days after discharge, the patient complained of diplopia was accompanied by ptosis, ocular pain, headache. Patient is readmitted to our neurology department. Patients with brain magnetic resonance imaging, CSF examination, conducted several tests with repeated nerve stimulation test. Patient examination was normal. We think the cause of recurrent tolosa-hunt syndrome and treat to the patient with steroid, conservative medication. A week later, the patient was discharged symptom is improved. 50 days later, the patient complained of diplopia and was admitted through outpatient departure. The patient had normal findings on examination. The patient treated in the same way as before, and was discharged after 10 days. The patient went through four months after discharge. Currently the patient has no abnormal findings. **Conclusions or Comments:** The idiopathic granulomatous painful condition has been termed Tolosa-Hunt syndrome. A recurrent isolated oculomotor nerve palsy is an extremely rare clinical condition. We report a case of recurrent Tolosa-Hunt syndrome patient who has recurrent isolated oculomotor nerve palsy.

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Overlap case of pharyngeal-cervical-brachial variant of Gullain-Barre Syndrome and Miller-Fisher Syndrome

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Background & Significance: Guillain-Barre Syndrome (GBS) is acute inflammatory neuropathies with multiple interactions involving immunologic systems. In GBS, there are many variations of typical presentation. Those types of GBS have different clinical features and findings of electro-physiologic and serologic tests. Among several subtypes of GBS, pharyngeal-cervical-brachial variant (PCB) is known for a rare type of GBS and is characterized by ptosis, facial, pharyngeal, neck flexor muscle weakness that spreads to the arms and spares leg strength, sensation, and reflexes. We report a patient with an overlap of PCB and Miller-Fisher syndrome (MFS) showing atypical manifestation. **Case:** A 42-year-old female was admitted with diplopia for 4 days. She has had sore throat, cough and sputum for a week. She has no past medical history. In neurologic examination at admission, there were down & up gaze limitation with multidirectional nystagmus on bilateral eyes, facial diplegia with bilateral ptosis, dysphonia, and dysphagia in cranial nerve functions. In motor examination, she showed prominent weakness of neck muscles (flexion/ extension GII/GII) and bilateral proximal upper (shoulder flexion GII/GII, abduction GII/GII) without weakness of distal limbs and symmetric hyporeflexia(1+) in biceps and triceps jerks. On the next day of admission, her weakness of upper proximal limbs has progressed (shoulder flexion GI/GI, abduction GI/GI, elbow flexion GIV+/GIV+) and developed weakness of proximal lower extremities (hip flexion GII/GII, extension GII/GII, abduction GIV/GIV, adduction GIV/GIV) with hypoactive(1+) knee jerks. The CSF study performed on admission day -demonstrated normal finding (opening pressure 12cmH₂O, WBC 0/mm³, RBC 2/mm³, protein 10, glucose 85, blood sugar 108) The nerve conduction study (NCS) on admission day showed reduced compound muscle action potentials (CMAPs) in bilateral median and ulnar nerves and F waves were absent in the same nerves suggesting acute axonal motor neuropathy (AMAN) and other findings showed normal. We diagnosed as PCB/MFS and started immunoglobulin (IVIg) therapy. After 5 days of IVIg therapy, her ophthalmoplegia and weakness stayed stable. The results of follow-up NCS on 7 days showed more reduced CMAPs in bilateral median and ulnar nerves and no F waves in bilateral peroneal nerves additionally. There were negative results in anti-GQ1b and anti-GM1 antibodies. As time goes by, her ophthalmoplegia and weakness has steadily improved. In follow-up NCS on about 40 days later, decreased CMAPs on bilateral median and ulnar nerves showed similar while F waves on median, ulnar, and peroneal nerves returned to normal. Her diplopia disappeared after a month and her weakness fully recovered after about 3 months. **Conclusions or Comments:** In this case, initial presenting symptom was diplopia by ophthalmoplegia, which suggests MFS. However, the findings of neurologic examination on admission day demonstrated prominent face, neck, and proximal weakness, which suggest PCB. We diagnosed as an overlap of PCB/MFS based on her clinical presentations. However, weakness of proximal lower limbs not presented in typical PCB/MFS, no history of diarrhea, and negative results of immunologic markers could make diagnosis confused and difficult. Nevertheless, we should consider a combined form of GBS in case presenting atypical presentations of GBS. Furthermore, when pharyngeal- cervical-proximal limb weakness is combined with ophthalmoplegia or ataxia, we should try to suspect PCB/MFS first of all.

P-2-278

Co-occurred case of Guillian-Barre syndrome and acute transverse myelitis

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Background & Significance: Acute transverse myelitis(ATM) is an inflammatory

disorder of the spinal cord associated with multiple sclerosis, neuromyelitis optica and autoimmune disorders. Alternatively, it can be occurred an isolated illness or association with preceding infections or vaccinations. Simultaneous development of inflammatory disorders of the central nervous system(CNS) and the peripheral nervous system(PNS) is generally considered to be uncommon, and there have been an extremely limited number of cases of concurrent ATM and inflammatory peripheral neuropathy. Here we report a case of ATM accompanied by Guillian-Barre syndrome(GBS). **Case:** A 72-year-old female patient was admitted with tingling sensation and weakness of both legs. She had 3 days history of touchable pain with gradual weakness and paresthesia of the legs. There were also hyperesthesia and allodynia below the T5 level. With progressed motor weakness to both lower extremities, she was unable to stand and walk. She had no prior history and symptoms of infection. Manual motor examination revealed hip flexion/extension and knee flexion/extension weakness of Medical Research Council (MRC) grade 3/3 and 2/2, respectively. Ankle dorsiflexion and plantar flexion were also reduced MRC grade 1/1. The deep tendon reflexes on knee, ankle were absent on right and decreased on left. There was positive Babinski sign bilaterally. Laboratory examinations including blood count, renal, and liver function analyses, C-reactive protein, erythrocyte sedimentation rate revealed no abnormality. CSF examination was normal in protein and glucose levels with no pleocytosis, and oligoclonal bands was negative. Spine MRI revealed hyperintense nodular lesions in the cervicothoracic spinal cord, extending from C5 to T2 vertebral level on T2 weighted series. Initial motor conduction studies on 3 day of illness revealed a decrease in motor conduction velocity in bilateral median, left common peroneal, and tibial nerves. The F-wave latency of bilateral tibial and peroneal nerves was prolonged. The right median nerve and bilateral sural nerves conduction velocities were slow. She was treated with intravenous immunoglobulin and pulse methylprednisolone for 5 days followed by prednisolone administration, which started at 60 mg/day and was gradually tapered off. The patient's leg strength improved over the following weeks. A month later, muscle strength was improved in bilateral lower extremities and she was able to walk independently after two month. **Conclusions or Comments:** Rarely, demyelination can affect the central and peripheral nervous system simultaneously described as acute severe combined demyelination. Previously, a few cases of ATM combined with GBS which usually associated with infection or vaccination. However, there were no prior viral infection, no history of diarrhea, and any other symptoms in this case.

P-2-279

A case of Polymyalgia rheumatica with extremities weakness dominant symptoms

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Background & Significance: Polymyalgia rheumatica (PMR) typically manifests as inflammatory pain in the shoulder and/or pelvic girdles in a patient over 50 years of age. This condition was long underrecognized and therefore under diagnosed. Patients who have polymyalgia rheumatica may also have temporal arteritis, an inflammation of blood vessels in the face which can cause blindness if not treated quickly. Who visited the extremities weakness in patient with polymyalgia rheumatica symptoms are rare. We report a case of polymyalgia rheumatica with extremities weakness symptoms. **Case:** Patient visited the outpatient departure present the right side weakness symptoms for 3 weeks. 3 weeks ago, the patient is whole body pain, had to get something to hold up. Previous his medical history is none. 2 weeks ago, it was patient with normal findings in X-ray test and visit a local clinic. The patient was observed

that right side weakness appeared during hospital admission, had complained of thigh pain. Patient with limb muscle strength was MRC grade 4+. The patient was hospitalized and underwent radiographic examination, including blood tests. The blood tests were normal except for elevated ESR and CRP. Evoked Potentials, nerve conduction tests, EMG was also normal. Brain magnetic resonance imaging scan was normal. In spinal magnetic resonance imaging findings of this enhancement made to the lumbosacral joint and soft tissue area sites. The pain was more severe in patient with a history on the shoulder and thigh, stiff feeling complained. Looking at the patient as a whole showed a systemic pain and muscle weakness, inflammation of the blood test phase reaction, and the radiographic examination showed an inflammatory response in the brain or spinal cord rather than the problem itself. We treated the patients with polymyalgia rheumatica and start. We were treated with a daily dose glucocorticoid 20mg. Patient symptoms were much improved after 3days dosing. 6 days later after admission, the patient was discharged, because the symptoms was improved. After discharge, the patient is currently under outpatient departure without focal neurologic findings. **Conclusions or Comments:** Polymyalgia rheumatica (PMR) is characterized by inflamematory pain and stiffness of the shoulder and/or pelvic girdles accompanied with laboratory evidence of severe inflammation in a patient older than 50 years of age. We reports a case of polymyalgia rheumatic patient admitted with symptoms of extremities weakness.

P-2-280

Late use of electronic media and its impact on insomnia, depression and suicidality among Korean adolescents

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Background & Objectives: The aim of this study was to investigate the impact of adolescent's last media use-time on their sleep and mood disturbances, including depression and suicidality. Furthermore, we also examined whether sleep disturbances mediated the relationship between the time of last media use and mood disturbances. **Method:** This cross-sectional, school-based online survey was administered by the Korea Centers for Disease Control and Prevention (KCDC) in 2011. A total of 26,395 participants were recruited from 150 middle and high schools (7th to 12th school grade) representative of nationwide adolescents from 15 administrative districts (metropolis/province) in Korea. Students were requested to fill out self-report questionnaires regarding their sleep habits, sleep disturbances, daytime sleepiness, depressive symptoms, and suicide feelings and attempts on the website. The question about media use was "When do you end the use of media (television including digital multimedia broadcasting (DMB), computer, or mobile phone) for the day on weekdays?". We used the Korean version of the Global Sleep Assessment Questionnaire (GSAQ) for sleep disturbances, Epworth Sleepiness Scale (ESS) for daytime sleepiness, Beck Depression Inventory (BDI) for depressive symptoms, and Beck 19-item Scale for Suicide Ideation (SSI-Beck) for suicidality. To establish mediation, path analysis was conducted to examine the relation between the time of last media use and depression and suicidality with a mediator of insomnia. **Results:** Subjects ending electronic media use around bedtime (between one hour before and after bedtime) were the most prevalent (48.4%, N=11,950) in our four categories regarding the temporal relationship between media use and actual bedtime. The models using path analysis were constructed to show the relationship between the time of the subject's media use and depression and suicidality when controlling for sleep duration, gender, and grade. Subjects who ended the media use later were more likely to have higher chance of depression and suicidality. This relationship was significant both with and without the mediation of insomnia. **Conclusion:** This large

study of Korean adolescents shows that almost half of the adolescents end their electronic media use around the time of going to bed. Later use of electronic media was found to be a risk factor for sleep and mood disturbance including depression and suicidality regardless of sleep duration with or without mediation by insomnia. These results of our study suggest that adolescents, in spite of the factors affecting their sleep and mood which may be difficult to change, such as quantitatively insufficient sleep, can benefit from the restricted use of their electrical media after bedtime. Moreover, education regarding electrical media use in the night time might be helpful to prevent youth suicide, a large burden to public health.

P-2-281

The anti-obesity effect of weekend catch-up sleep among adults in Korean population

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Background & Objectives: Obesity has become a global public health issue and the prevalence of obesity has being grown. Increasing reports have been focused on the association between short sleep duration or chronic sleep deprivation and increased weight. This study aimed to evaluate the association about sleep compensation by extending sleep duration in weekend and weight in general population in Korea. **Method:** Subjects were recruited randomly among the general population adults between 19-65 year old, excluded shift workers. 2083 subjects (929 women and 1154 men) were asked to fill out the questionnaires about their sleep duration separated by week days and weekends. The participants also completed several scales such as Epworth Sleepiness Scale, Pittsburgh Sleep Quality Index, Insomnia Severity Index, Goldberg anxiety scale, Patient Health Questionnaire-9 depression scale and Headache Impact Scale-6. Additionally demographic and behavioral information such as age, sex, alcohol, smoking, education, job, monthly income, frequency of physical exercise and BMI were obtained. Participants were divided into two groups with or without extended weekend sleep duration. Logistic regression was used to evaluate the association with weekend catch-up sleep and BMI. **Results:** Catch-up sleep (CUS) group was consisted of 45.1% among total participants. The subjects of CUS group were in younger age, more women, less educated, in higher socioeconomic status and having more headache and less physical activity in proportion. They slept longer (0.2 hour) per average day than non-CUS group, and had extended sleep on weekend about 1.7 hours than weekday. After covariate adjustment with age, sex, education, job status, socioeconomic status, alcohol, current smoking, physical activity and sleep duration, CUS group had lower BMI than non-CUS group regardless sleep duration and more sleep extension in weekend showed lower BMI. There was linear association in decreased BMI according to amount of compensated weekend CUS. **Conclusion:** The present study emphasizes on the association between sleep compensation in weekend and obesity. Presence of weekend CUS per se had significant anti-obesity effect and extended sleep has decreasing BMI effect dose-dependently. Sleep compensation during free weekend have benefit in preventing obesity for people with sleep deprivation, even for short sleepers.

P-2-282

Different cutoff value of the Korean Version of the REM sleep behavior disorder screening questionnaire between patients with

obstructive sleep apnea and healthy peopleJoon Hyun BAEK¹, Sang-Ahm LEE¹, Su-Hyun HAN¹, Han-Uk RYU²¹Department of Neurology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea, ²Department of Neurology, Chonbuk National University Medical School, Jeonju, Korea

Background & Objectives: To explore the utility of a Korean version of the rapid eye movement sleep behavior disorder screening questionnaire (RBDSQ-K) to discriminate patients with idiopathic REM sleep behavior disorder (iRBD) from patients with obstructive sleep apnea (OSA) and healthy subjects. **Method:** Participants with iRBD (n = 47) and OSA (n = 213) were diagnosed by polysomnography. In healthy subjects (n = 58), RBD was excluded by medical history without polysomnography. Receiver operating characteristic curve analysis was used to identify the optimal cutoff value of the RBDSQ-K for iRBD. **Results:** RBDSQ-K score was higher in iRBD subjects than in OSA subjects and healthy subjects (both $p < 0.001$). The optimal cutoff was 6.5 to distinguish iRBD subjects from OSA subjects and 4.5 to distinguish iRBD subjects from healthy subjects. The corresponding sensitivity and specificity was high for detecting iRBD from OSA and healthy subjects. The percentages of individuals with RBDSQ-K scores ≥ 5 and ≥ 7 were higher in OSA subjects with daytime sleepiness (36.1% and 13.8%, respectively) than in OSA subjects without daytime sleepiness (12.0% and 3.1%, respectively). Apnea-hypopnea index had no influence on RBDSQ-K score. Cronbach's alpha for the RBDSQ-K was 0.768, indicating a high degree of internal consistency. **Conclusion:** The RBDSQ-K had acceptable sensitivity and specificity for screening persons with probable RBD from healthy subjects and OSA subjects when the cutoff score was 4.5 and 6.5 points, respectively. However, attention must be paid to the possibility of false positives when using this scale, especially in OSA subjects with daytime sleepiness.

P-2-283**Preliminary study for REM sleep behavior disorder (RBD) in Parkinson's disease using RBD screening questionnaire**

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Background & Objectives: REM sleep behavior disorder (RBD) is associated with α -synucleinopathies such as Parkinson's disease (PD). We aimed to assess prevalence of RBD and differences in clinical characteristics between Parkinson's disease accompanied with and without RBD. **Method:** Forty two patients previously diagnosed with PD were evaluated for clinical history, motor and cognitive function using UPDRS and MMSE, autonomic symptoms, sleep characteristics using PSQI (Pittsburg sleep quality index), and the presence of RBD using RBDSQ-K (the Korean version of REM sleep behavior disorder screening questionnaire). The prevalence of RBD and demographic features of the patients were evaluated. The patients were classified into two groups such as PD with RBD and PD without RBD, as a result of RBDSQ-K. The motor and cognitive function as well as other clinical features of the two groups were compared. **Results:** A total of 42 PD patients were enrolled. 18 patients were classified as RBD+PD. There was no difference in prevalence between genders. Compared to the PD without RBD, the PD with RBD showed higher incidence of rigidity in UPDRS subscale. Regarding sleep problems, RBD without PD revealed higher sleep disturbance, low sleep efficiency, and low overall sleep quality in PSQI. There was no difference of cognitive dysfunction using K-MMSE between the two groups. **Conclusion:** PD with RBD was associated with worse sleep and motor symptoms. Therefore, RBD symptoms in PD might be a possibility of one of the poor prognostic markers

P-2-284**Quality of life in idiopathic REM sleep behavior disorder in Korea**

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Background & Objectives: Background: The rapid eye movement sleep disorder (RBD) is a parasomnia characterized by acting out during rapid eye movement (REM) sleep. There have been few quality of life (QoL) studies of patients with RBD, especially idiopathic RBD (iRBD). Moreover, it is debatable which affects their QoL, sleep itself or mood disorder. We studied the QoL of patients with iRBD and compared it to healthy controls and patients with hypertension (HTN), uncomplicated type 2 diabetes mellitus (DM) or restless legs syndrome (RLS). And we investigated the factors that may have influence on the QoL in patients with iRBD. **Method:** A total of 60 consecutive iRBD patients (24 female; mean age 61.43 \pm 8.99) were enrolled. The RBD diagnosis was based on a sleep history and overnight polysomnography (PSG) following the guidelines of the international classification of sleep disorders, 3rd. All patients underwent a baseline neurological examination to exclude secondary RBD with neurodegenerative disease. Cognitive function was evaluated using the Mini-Mental State Examination. All patients completed the questionnaires, including all Korean versions of Short Form 36-item Health Survey (SF-36) for QoL, the insomnia severity index (ISI), the Hospital Anxiety Scale (HAS), the Beck Depression Inventory-2 (BDI-2), and the Hamilton Depression Rating Scale (HDS). These results were compared with the scores from healthy controls (N=60), patients with HTN (60), DM (60) and RLS (60). **Results:** Total SF-36 score of iRBD patients was lower than healthy controls, but higher than that of HTN, DM and RLS patients. The total SF-36 score has a negative correlation with the PSQI ($r=-0.498$, $p=0.000$), ISI ($r=-0.645$, $p=0.000$), HAS ($r=-0.497$, $p=0.000$), BDI-2 ($r=-0.694$, $p<0.000$) and HDS ($r=-0.435$, $p=0.001$). Multiple regression identified that the SF-36 score has a negative correlation with the ISI ($\beta=-0.366$, $p=0.000$) and BDI-2 ($\beta=-0.482$, $p<0.000$). **Conclusion:** These findings show that iRBD has a considerable impact on the QoL of Koreans, although it is not as severe as those with other chronic medical diseases. The QoL impairment relates to the degrees of insomnia and depression with iRBD for Koreans.

P-2-285**Comparing the quality of life between patients with narcolepsy only and comorbid narcolepsy patients**Yong Won CHO¹, Mei Ling SONG², Hye-Jin MOON¹, Doh-Eui KIM³, Kwang Ik YANG³¹Neurology, Keimyung University, Dongsan Medical Center, Daegu, South Korea, ²Graduate school of Nursing, Keimyung University, Dongsan Medical Center, Daegu, South Korea, ³Neurology, Soonchunhyang University College of Medicine, Cheonan Hospital, Cheonan, South Korea

Background & Objectives: People with narcolepsy are likely to become drowsy or fall asleep, often at inappropriate times and places. And these symptoms will interfere with a normal education or career, further more it will affect the quality of life (QoL). We hypothesize that the quality of life in comorbid narcolepsy patients will be lower than the patients who have only narcolepsy. The purpose of our study is to investigate the prevalence of comorbid narcolepsy, and compare the health-related QoL between patients who suffer from narcolepsy only and narcolepsy with other comorbid sleep disorders. **Method:** We have retrospectively screened the patients who visited two sleep centers, and we screened 61 subjects with narcolepsy and enrolled them in our research. The narcolepsy was defined according to the ICSD-3 diagnostic criteria. We reviewed the patients' medical records as well as their sleep tests, and divided them into two

groups: narcolepsy only and narcolepsy with comorbid sleep disorders. Data analysis was performed using SPSS version 18.0, and the Mann-Whitney U test was used for comparing the differences between the two groups. **Results:** 24.6% (n=15) of the narcolepsy patients had another sleep disorders. And narcolepsy only patients were younger (24.07 ± 7.92 , vs 40.93 ± 18.25 , $p=0.001$), and had fewer comorbid medical diseases (17.4%, vs 46.7%, $p=0.022$) than those with comorbid narcolepsy. In the PSG data, narcolepsy only patients had less wake after sleep onset (39.73 ± 43.32 , vs 77.62 ± 65.88 , $p=0.011$), AHI (0.74 ± 1.13 , vs 9.01 ± 9.94 , $p<0.001$), PLMI (1.60 ± 5.02 , vs 7.79 ± 12.85 , $p=0.014$), total arousal index (9.85 ± 8.52 , vs 19.12 ± 12.15 , $p=0.002$), and had higher sleep efficiency (91.18 ± 8.60 , vs 84.48 ± 13.80 , $p=0.039$), compared with comorbid narcolepsy patients. However, there were no significant differences in QoL and psychiatric related factors between the two groups. In general, according to the SF36, the both groups of narcolepsy patients were lower in the mental health dimension. **Conclusion:** In our research comorbid narcolepsy patients were older than the narcolepsy only patients, had more medical diseases, and poorer sleep architecture. However there were no significant differences in QoL and psychiatric related factors between the two groups. From these results, we can make an inference that the QoL in narcolepsy is more affected by narcolepsy itself rather than age, comorbid medical disease, or comorbid sleep disorders. However, further investigation on a national level is needed to verify our results.

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Health-related quality of life in patients with narcolepsy type1 and type2

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Background & Objectives: Narcolepsy is one of the chronic sleep disorders, which is characterized by excessive daytime sleepiness, cataplexy, sleep paralysis, and hypnagogic hallucination. Narcolepsy has negative impact on patients and their families. Recently, new version (the third) of International classification of sleep disorders (ICSD-3) categorized narcolepsy into type I and type II. We evaluated the health-related quality of life (HRQoL) in narcolepsy patients, and compared the results of type I and type II narcolepsy patients. **Method:** In total, 38 patients were enrolled in this study (type I, n=17; type II, n=21). 27 patients were male and 11 patients were female. Mean age was 21.1 (aged between 14 and 40 years). All patients were diagnosed as narcolepsy with night polysomnography and multiple sleep latency test (MSLT). Type I narcolepsy was defined as having cataplexy or lower cerebrospinal fluid hypocretin (<110 pg/mL) level. We used the Short Form-36 health survey (SF-36), the Pittsburgh Sleep Quality Index (PSQI), the Epworth Sleepiness Scale (ESS), the Beck depression index (BDI), and questionnaires about their daily life. **Results:** During MSLT, mean sleep latency was shorter in type I narcolepsy although type I and type II had similar numbers of sleep onset REM period. ESS and BDI scores were higher in Type I. The results of PSQI demonstrated that type I patients had lower sleep efficiency and more daytime dysfunction. Global PSQI score was lower in type I as well. Type I patients had considerably lower scores on vitality and mental health dimensions of SF-36. The scores of other dimension and total SF-36 score were not different between type I and type II. **Conclusion:** Type I narcolepsy patients had more excessive daytime sleepiness and depression, and they suffered more from sleep problem. However, HRQoL of type I was more deteriorated only in vitality, mental health dimensions when compared with type II patients.

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Anti-Ma2-associated encephalitis presented with hypersomnia

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Background & Significance: Ma2 is an intracellular protein that can be isolated from neurons, particularly in the brainstem, hippocampus, amygdala, hypothalamus and thalamus. It is also found in testis and some neoplastic lesions but whose exact function remains unknown. Anti-Ma2 antibodies have been shown to be associated with neoplasm, mainly testicular tumors, lung cancer, and breast cancer. Neurologic symptoms usually precede detection of these conditions and main symptoms are related to limbic encephalopathy and diencephalopathy. However, there were few case studies on anti-Ma2 associated encephalitis that presented with excessive daytime sleepiness. **Case:** A 66-year-old woman presented with excessive daytime sleepiness over 2 months. The patient frequently felt faintness and light-headedness during walking and fell asleep. She had been on medication for diabetes mellitus. There was no preceding infection or trauma history. On neurological examination she was fully oriented and showed no focal neurological deficits. Brain MRI showed hyperintensity in bilateral midbrain, hypothalamus, thalamus and medial temporal lobe including amygdala and hippocampus on fluid attenuation inversion recovery (FLAIR) images. Cerebrospinal fluid (CSF) analysis showed mildly elevated protein level (78.7 mg/dL) without pleocytosis (5 cells/mm³). Laboratory findings for infection, thyroid function, and vitamin level were unremarkable. Serum autoimmune testing presented cytoplasmic FANA 1:40, anti-cardiolipin IgA(-) IgG(+) IgM(-), anti-phospholipid IgG(+) IgM(-). CSF autoimmune testing showed positive for anti-Ma2 antibody. Hypersomnia was gradually improved after five-day course of intravenous steroid pulse therapy and maintained low dose oral corticosteroid therapy. Chest CT showed atelectasis at right major fissure and both lower lobes with pleural effusion, but no significant lymph node (LN) enlargement and tumor. Abdomen CT showed no evidence of primary malignancy in abdominopelvic cavity but possible metastatic LN around common hepatic artery. Further work-up for an underlying cancer is ongoing. **Conclusions or Comments:** In our best knowledge, anti Ma2-associated encephalitis presented with hypersomnia is rare condition. We could not find a definite evidence of malignancy so far in this case study. However, neurological symptoms precede the diagnosis of cancer in 50% of patients with paraneoplastic syndromes. Clinicians should be concerned continuously and evaluate a risk of development of malignant conditions.

P-2-288

Sleep and related symptoms in shift workers and day workers in a single enterprise corporation

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Background & Objectives: YS Enterprise Corporation manufacture and supply various types of cast iron piston rings, stainless steel oil rings, cylinder liners, valve guides and camshafts applicable to automobile, marine, agricultural engines. The company operates 24 hours a day. The shiftwork schedule assembly time is 10am to 10pm. We compare any sleep and related symptoms in shift-workers and day-workers in single YS Enterprise Corporation. **Method:** A cross-sectional study was conducted among Questionnaires were distributed to 217 participants inof YS Enterprise Corporation located in

Chungnam, Korea. Total of 208 participants were enrolled after excluding 9 participants (1 missing data on work schedule status, 7 irregular shiftwork and daytime work schedule, 1 8 hour shift. Questionnaires were distributed. Questions were about the work schedule, duration of sustained employment, various division of work. Individual excessive daytime sleepiness were assessed by Epworth Sleepiness Scale (ESS), presence of insomnia symptom by Insomnia Severity Index (ISI), depression and anxiety index by Hospital Anxiety and Depression Scale (HADS), Individual chronotype by MEQ (Morningness and Eveningness Iq Questionnaire). **Results:** Total of 208 participants were enrolled (shift workers (SW), n=142 (68.26%), day workers (DW), n=66 (31.7%)). Mean age was not significantly different between groups, and both groups had significant male predominance. The division of work revealed more manufacture team in SW, and more maintenance, and inspection team in DW. Sleep symptom by questionnaire revealed severe daytime sleepiness in both group (ESS: SW, 13.70±5.98, vs. DW, 12.59±4.74, p=0.052), however insomnia symptoms were significantly worse in SW group (ISI: SW 16.05±5.98, vs. 12.41±6.29, p<0.001). Clinically significant insomnia symptoms were seen in 60.3% of SW, and 40% of DW (p=0.005). Depression and anxiety were not significantly different between two groups. Individual chronotype were 24 (11.5%) morningness (M), 159(76.4%) intermediate (I), and 25 (12.0%) eveningness (E). The morningness were related to older age, long sustained employment. The eveningness were related to sleep related symptoms of both excessive daytime sleepiness and insomnia (ESS, M 11.66±5.58, I (13.15±4.66), vs E (16.16±4.90), p=0.008, rho= 0.213), (ISI, M 13.04±7.15, I (14.68±6.14), vs. E (18.12±5.45), p=0.034, rho= 0.115). **Conclusion:** In this sample of Manufacture Company, shift workers were predominant in manufacture division. While severe daytime sleepiness were concerning in both SW, and DW, insomnia were more presented in SW. According to individual chronotype by MEQ, eveningness had more severe excessive daytime sleepiness along with insomnia. The eveningness chronotype individual did not correlate with better accommodation in shift worker.

P-2-289

Regional grey matter changes in shift workers: a voxel-based morphometry study

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Background & Objectives: Shift work, in particular night shift, disrupts the sleep-wake cycle and its synchrony with the physiologic circadian rhythms. As a consequence, shift workers frequently suffer from excessive sleepiness and insomnia. Previous MRI studies showed a negative relationship between the degree of daytime sleepiness and volume of prefrontal cortex in healthy subjects, and grey matter decreases in prefrontal cortex, postcentral gyrus, and lateral temporal cortex in chronic insomniacs relative to good sleepers, implicating that disruption of sleep-wake cycle can alter regional brain structures. To our knowledge, there is no currently available study investigating brain structural changes in shift workers. We used voxel-based morphometry (VBM) to identify structural changes of the grey matter in shift workers compared to day workers. We then correlated regional structural changes with clinical variables such as degree of daytime sleepiness and sleep quality. **Method:** Nineteen shift workers (all males, age = 21.6 ± 1.8) and 19 day workers (all males, age = 21.2 ± 2.0) participated in this study. All participants were healthy military servicemen and scanned on a 1.5 Tesla MR scanner. Conventional MR images and 3D volumetric T1-weighted images were acquired for identification of structural abnormalities and VBM analysis, respectively. Epworth Sleepiness Scale (ESS) and Pittsburgh Sleep Quality Index (PSQI) were obtained and compared between shift workers and day workers. Between-group comparisons of grey matter volumes were made using ANCOVA adjusting for age and intracranial

volume (familywise error-corrected $P < 0.05$ with small volume correction). Correlation analysis was performed between regional grey matter volume of significant cluster from VBM and scores of ESS and PSQI (Pearson's correlation, $P < 0.05$). **Results:** ESS score was higher in shift workers (10.3 ± 4.1) than in day workers (7.5 ± 3.4, $P = 0.029$). PSQI global score was also higher in shift workers (9.0 ± 2.2) than in day workers (6.0 ± 2.7, $P = 0.001$), as with subjective sleep quality domain score ($P = 0.003$) and daytime dysfunction domain score ($P = 0.008$). VBM showed significant gray matter volume reduction in dorsal upper midbrain in shift workers compared with day workers (MNI coordinate = 8/-35/-12, cluster size = 739 mm³, peak z score = 3.78). Correlation analysis showed an inverse relationship between regional grey matter volumes in dorsal upper midbrain and PSQI global score ($r = -0.370$, $P = 0.022$), subjective sleep quality domain scores ($r = -0.398$, $P = 0.013$), and sleep duration domain scores ($r = -0.331$, $P = 0.043$). **Conclusion:** We observed a novel finding of regional grey matter volume reduction in dorsal upper midbrain which corresponds to the pedunculopontine and laterodorsal tegmental (PPT/LDT) nuclei. The PPT/LDT, a major component of ascending reticular activating system, produces acetylcholine and plays a role in regulation of sleep-wake cycle. Given the known finding that light stimulus through retinohypothalamic pathway is a crucial zeitgeber for sleep state switching, we speculate that decreased exposure to light stimuli in shift workers may cause chronic decreases in activation of ascending reticular activating system, resulting in volume reduction of PPT/LDT nuclei. Our finding of relationship between smaller PPT/LDT volume and poorer sleep quality suggests that PPT/LDT grey matter decrease might be a consequence of chronic burden of shift work in association with disruption of circadian rhythm.

P-2-334

The sleep pattern of Korean woman professional basketball players

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Background & Objectives: Sleep has numerous important physiological and cognitive functions that may be particularly important to athletes. However, there are few published data about sleep pattern in elite sportman. We investigated sleep patterns of Korean woman professional basketball players by using sleep-related questionnaires. **Methods:** For this study, 23 Korean woman professional basketball players and 40 age and sex-matched control group were recruited. All subjects were asked to complete the self-administered sleep questionnaire consisting of habitual sleep patterns (sleep onset time, sleep latency, awakening time in the morning, day time napping time), exercise habits, Epworth Sleepiness Scale (ESS), Insomnia Severity Index (ISS), Pittsburgh Sleep Quality Index (PSQI), validation of the Perceived Stress Scale (PSS), and Beck Anxiety Inventory (BAI). **Method:** For this study, 23 Korean woman professional basketball players and 40 age and sex-matched control group were recruited. All subjects were asked to complete the self-administered sleep questionnaire consisting of habitual sleep patterns (sleep onset time, sleep latency, awakening time in the morning, day time napping time), exercise habits, Epworth Sleepiness Scale (ESS), Insomnia Severity Index (ISS), Pittsburgh Sleep Quality Index (PSQI), validation of the Perceived Stress Scale (PSS), and Beck Anxiety Inventory (BAI). **Results:** The sleep latency was significantly prolonged (45.43 ± 40.139 and 26.25 ± 22.92; $t = -2.103$, $p < 0.05$) and the daytime nap time was greater (59.35 ± 39.436 minute and 18.75 ± 33.907 minute; $t = -4.310$, $p < 0.001$) in the professional basketball players compared to the control group. The total PSQI scores (20.00 ± 8.852 and 15.25 ± 4.389) and ISI (11.73 ± 5.470 and 8.65 ± 3.255) were higher in basketball players, but estimated sleep ESS, PSS, and BAI scores were not different between the two groups. To compared with man professional basketball player, we investigated sleep questionnaire in 54 man professional basketball players. The daytime nap

time was greater (82.96 ± 37.347 and 59.35 ± 39.436 minute; $t = -2.498$, $p < 0.05$) in the man basketball players. The total PSQI scores (20.00 ± 8.852 and 12.57 ± 6.074), ISI (11.73 ± 5.470 and 6.91 ± 4.691) and BAI (17.00 ± 11.623 and 8.04 ± 6.269) were higher in female basketball players than male players, but estimated sleep ESS and PSS were not different between the two groups. **Conclusion:** This study suggests that Korean female basketball players have higher anxiety and insomnia score than control group and man basketball players.

P-2-290

Mesenchymal stem cells modulate the functional properties of microglia via TGF- β secretion

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Background & Objectives: The regulation of microglial cell phenotype is a potential therapeutic intervention in neurodegenerative disease. Previously, we reported that TGF- β levels in mesenchymal stromal cells (MSCs) could be used as potential biological markers to predict the effectiveness of autologous MSC therapy in patients with amyotrophic lateral sclerosis. However, the underlying mechanism of TGF- β in MSCs was not fully elucidated in determining the functional properties of microglia. In this study, we aimed to clarify the role of TGF- β that is involved in MSC effectiveness, especially focusing on microglia functional properties that play a pivotal role in **Method:** MSCs (5×10^5 /mL) were plated into 6-well plates. After 24 hours, the medium was replaced and the cells were incubated for another 48 hours, at the end of which time (MSCs number; $1-2 \times 10^6$ /mL) the medium was collected and centrifuged for obtaining MSC conditioned media (MSC-CM). The MSC-CM was treated for 24, 48, and 72 h to investigate effects of the releasing factors on microglia functional properties in LPS-stimulated microglia. The effect and mechanism were analyzed with FACS, immunofluorescence study, qPCR, and western blot. **Results:** We found that MSC-conditioned media (MSC-CM) inhibited pro-inflammatory cytokine expression, restored alternative activated microglia phenotype (M2-like phenotype) markers such as fractalkine receptor (CX3CR1) and mannose receptor (CD206), and enhanced phagocytosis in LPS-stimulated microglia. In addition, TGF- β in MSC-CM played a major role in these effects by inhibiting the NF- κ B pathway and restoring the TGF- β pathway in LPS-stimulated microglia. Recombinant TGF- β (rTGF- β) also induced similar effects to MSC-CM in LPS-stimulated microglia. **Conclusion:** We propose that MSCs can modulate the functional properties of microglia via TGF- β secretion, switching them from a classically activated phenotype to an inflammation-resolving phenotype. The latter role may be associated with the inhibition of neuroinflammatory processes in neurodegenerative disorders.

P-2-291

Neuroprotective effects of uric acid against sodium arsenite-induced motor neuronal cell death

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Background & Objectives: Uric acid (UA) is the main end product of purine metabolism in humans due to the loss of uricase activity during the evolution of hominoids. Serum UA is a natural antioxidant that can scavenge super-

oxide, thus helping to prevent the reaction of superoxide with nitric oxide leading to the formation of the strong oxidant, peroxynitrite. Based on the antioxidant effects of UA, many studies have suggested that high concentrations of UA may modulate the progression of several disease including Parkinson's disease (PD), Alzheimer's disease (AD), Amyotrophic lateral sclerosis (ALS). The association between serum UA levels and motor neuronal cell death remain poorly understood alone with the molecular mechanism underlying the neuroprotective effects of UA. The main purpose of this study was to evaluate whether the neuroprotective effects of UA against sodium arsenite-induced oxidative stress involved activation of PI3K. **Method:** To evaluate the protective effect of UA in NSC-34 cells against oxidative stress induced cell death, we measured cell viability and free radical production that cells were treated with different concentrations of UA for 24 h after exposure to sodium arsenite. To ascertain the neuroprotective mechanisms of UA against sodium arsenite-induced neuronal cell death, cells homogenates were treated under several different conditions, and the resulting effects on various intracellular signaling pathways were analyzed by immunoblotting. To establish the role of the PI3K/Akt pathway in neuroprotection by UA against sodium arsenite-induced neurotoxicity, we further assessed its effects in the presence of the selective PI3K inhibitor LY294002. **Results:** In sodium arsenite-induced oxidative stress conditions, treatment with UA resulted in a dose-dependent stepwise increase in neuronal cell viability and decreased production of free radicals. In addition, expression of PI3K, p-Akt, and p-GSK3 β was increased after UA treatment, while the expression of cytosolic cytochrome c, cleaved caspase-3, and AIF were decreased. These finding indicated that the neuroprotective effects of UA are inhibited in the presence of the PI3K inhibitor LY294002, suggesting that the protective effects of UA in the context of sodium arsenite-induced neurotoxicity are partially mediated through PI3K/Akt signaling pathway. Taken together, these results suggested that UA mediates neuroprotective effects by reducing ROS, enhancing survival signals through the PI3K/Akt pathway, and inhibiting apoptotic signals in motor neurons. **Conclusion:** Therefore, we proposed that PI3K/Akt signaling pathway may play a critical role in neuroprotection induced by UA on motor neuronal cells treated with sodium arsenite, suggesting that increased levels of UA may be a good target for neuroprotective therapy for the treatment of neurodegenerative disease.

P-2-292

Abi, an Abl tyrosine kinase-interacting protein, regulates synaptic development and neuronal survival via inhibition of BMP signaling

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Background & Objectives: The nonreceptor tyrosine kinase Abl plays important roles in several steps of neuronal morphogenesis through regulation of F-actin dynamics. Abl is known to bind and phosphorylate an adaptor protein Abi (Abl interactor) that regulates Arp2/3-dependent actin polymerization via Wiskott-Aldrich syndrome protein (WASP) and WASP family verprolin-homologous protein (WAVE). However, the functional roles and mechanisms of Abi in Abl-regulated neuronal morphogenesis remain unclear. Here we show that Abi functions together with Abl to regulate synaptic development and neuronal survival. **Method:** We used Drosophila larval neuromuscular junctions (NMJs) as a model synapse to study synaptic roles of Abi. When stained with an antibody against the axonal membrane marker HRP, the Drosophila NMJ at the crawling third instar stage displays stereotyped, segment-specific numbers of branches and boutons. Using this histochemical approach, we analyzed larval NMJ morphology of mutants affecting Abi-Abl signaling pathway. To test whether Abi loss is associated with neurodegeneration, we ex-

amined progressive vacuolization in the adult brain using hematoxylin and eosin (H&E) staining. Apoptotic cell death was assessed by TUNEL staining on cryosections of adult heads. For pharmacological manipulation of microtubule stability, animals were grown on the medium containing vinblastine sulfate. **Results:** Abi localizes pre- and postsynaptically at the NMJs. Loss of Abi causes synaptic overgrowth with an excess of satellite boutons and increase the depth of NMJ and the level of the abundance of glutamate receptors. Transgenic rescue and RNAi experiments demonstrate that presynaptic Abi functions to regulate synaptic growth, while postsynaptic Abi is required for normal NMJ depth and glutamate receptor abundance. Abi interacts with Abl or mutations in components of the WAVE complex, supporting a key role for Abi in Abl/WAVE-dependent regulation of synaptic growth. Importantly, loss of Abi or Abl significantly increases the synaptic level of phosphorylated Mad, the major effector for the bone morphogenetic protein (BMP) signaling pathway that is known to promote synaptic growth at developing NMJs and neuronal survival in adult brains. Consistent with a functional link between Abi/Abl and the BMP signaling pathway, depletion of neuronal Abi and Abl causes excessive brain neurodegeneration in the adult fly. Finally, we observe dose-sensitive genetic interactions between *abi* and *abl* with respect to brain neurodegeneration. **Conclusion:** Taken together, our results support a model in which Abl/Abi/WAVE restrains synaptic growth and neuronal cell death by negatively regulating BMP signaling. We are currently investigating the mechanism underlying Abl/Abi/WAVE-mediated inhibition of BMP signaling.

P-2-293

Neuroprotective effects of atorvastatin against oxygen-glucose deprivation-induced neural stem cell death through the activation of the PI3K pathway

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Background & Objectives: Oxidative stress is known to play a key role in the development of neuronal cell death, thus much research effort has focused on antioxidants as potential treatment agents for that. Current evidence suggests that atorvastatin reduce blood lipids and have antioxidant effects. In this study, we investigate the neuroprotective effects of atorvastatin against oxygen-glucose deprivation (OGD)-injured neural stem cells (NSCs) through the activation of the PI3K pathway. **Method:** We incubated NSCs in the anaerobic chamber with different exposure time. To evaluate the effect of atorvastatin on OGD-induced NSC death, we treated several concentrations of atorvastatin under OGD. We measured cell viability by MTT (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide) assay, trypan blue staining and lactate dehydrogenase assay. We performed BrdU staining and colony formation assays to evaluate cell proliferation. DAPI and TUNEL stainings were also done to examine the effect of atorvastatin on apoptosis. And, with western blotting, effect on intracellular signaling proteins was evaluated. **Results:** NSCs viability and proliferation were decreased by OGD, but combined treatment with atorvastatin increased them. The percentage of apoptotic cells under a OGD was markedly increased under a hypoxic condition, but was significantly decreased with the treatment of atorvastatin. The immunoreactivities of phosphorylated Akt, phosphorylated glycogen synthase kinase-3 β and B-cell lymphoma 2 increased in NSCs that were treated with atorvastatin, compared with the levels in cells under a hypoxic condition. However, the expression of Bax, cleaved caspase 9 and cleaved caspase 3 decreased in response to treatment with atorvastatin. **Conclusion:** This results indicate that atorvastatin has a neuroprotective effect against OGD-induced NSC death by the activation of PI3K

pathway. Further studies on atorvastatin may reveal a new therapeutic target to protect NSCs.

P-2-294

Effects of aspirin and clopidogrel on neural stem cells

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Background & Objectives: Cerebral infarction causes permanent neuronal loss, which is associated with severe morbidity and mortality. Most patients with cerebral infarction should take daily anti-platelet drugs, so the effects and mechanisms of those drugs on the regeneration of the brain need to be investigated. Aspirin and clopidogrel are widely used anti-platelet drugs for the primary and secondary prevention of ischemic stroke. We investigated the effects of aspirin and clopidogrel on neural stem cells (NSCs) in terms of viability, proliferation and intracellular protein level. **Method:** After treatment with several concentrations of aspirin and clopidogrel, we measured cell viability by MTT (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide) assay and trypan blue staining. To evaluate their effects on NSC proliferation, we performed BrdU labeling and colony formation assays. And, we evaluated several proteins using proteomics and antibody microarray. **Results:** Various viability tests showed that aspirin decreased NSC viability and proliferation in a concentration-dependent manner but not clopidogrel. After aspirin treatment, it was significantly decreased several survival signals and increased death signals in proteomics while clopidogrel did not affect those signals. **Conclusion:** These results suggest that aspirin might be helpful for NSCs and the recovery of the brain damaged by ischemic stroke. Further studies should be performed to confirm whether this effect is also shown in vivo condition.

P-2-295

Interaction between sublethal dose of amyloid beta and hypoxia in neural stem cells

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Background & Objectives: Alzheimer's disease (AD) is the most common form of dementia and leads to a progressive deterioration of cognitive function. Amyloid- β peptide (A β) is considered to be one of the main pathological factors of AD. However, the effect of A β itself on neural cells exposed to ischemic insult has not been reported yet. We investigated the effect of A β on neural stem cells (NSCs) exposed to ischemic insult in this study. **Method:** To evaluate the effect of A β on NSCs, we treated primary cultured embryonic NSCs and adult NSCs with several doses of A β for 48h, and then we added hypoxia condition. To evaluate the interactions between A β and hypoxia, we measured cell viability by MTT (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide) assay and trypan blue staining. To evaluate the effects of A β and hypoxia on proliferation of NSCs, we performed BrdU labeling and colony formation assays. And, western blotting for the evaluation of effect on intracellular signaling proteins was achieved. **Results:** The viability tests showed that A β and hypoxic insult decreased NSC viability in a concentration- and time-, respectively, dependent manners. But the cells pretreated with sublethal dose of A β up to 5 μ M showed significant resistance against hypoxic insult and restored the proliferation of NSCs. Pretreatment with sublethal dose of A β up to 5 μ M significantly increased the survival signals. **Conclusion:** These results suggest that NSCs overcome from sublethal dose of amyloid beta increase in

the resistance to ischemic insult by increasing survival signals in NSCs.

P-2-296

Apolipoprotein E deficiency worsens small vessel pathology and cognitive dysfunction in a mouse model of subcortical vascular dementia

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Background & Objectives: Apolipoprotein E (APOE) plays a central role in controlling blood-brain barrier (BBB) permeability and maintaining cerebrovascular integrity. Subcortical vascular dementia (SVaD) is a subtype of vascular dementia and is associated with white matter damage, lacunar infarction and BBB breakdown. However, the relationship between APOE deficiency and factors associated with SVaD remains elusive. Here, we describe small vessel pathology (microinfarcts and white matter disruption) and hippocampal neuronal loss after chronic cerebral hypoperfusion in APOE deficiency mice. **Method:** Ten weeks old APOE-deficient mice, or wild type C57BL/6J mice were subjected to chronic cerebral hypoperfusion with bilateral common carotid artery stenosis (BCAS) using microcoils or sham operation. Behavioral performance (anxiety, locomotion, spatial working memory, contextual and cued memory), and histopathological findings (white matter damage, microinfarcts, astrogliosis, hippocampal neuronal damage, and IgG extravasation) were investigated. **Results:** APOE deficiency in BCAS operated mice leads to BBB breakdown, microinfarcts, and hippocampal neuronal damage, and to accelerated white matter damage and behavioral deficits. In contrast, wild-type BCAS operated mice did not show BBB breakdown, microinfarcts, and hippocampal neuronal damage. **Conclusion:** When combined with chronic cerebral hypoperfusion, APOE deficiency enhances cognitive decline, neuronal damage, and small vessel pathology in mice. The current mouse model reflects the phenotypes of human VaD, including small vessel pathology with hippocampal neuronal damage and cognitive impairment thus can be a plausible model for studying SVaD pathophysiology and drug study for it.

P-2-297

Paracrine action of High Mobility Group Box 1 (HMGB1) via Toll-like receptor 2 prevents ischemia-induced oligodendrocyte death

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Background & Objectives: Demyelination with oligodendrocyte (OL) loss is a prominent histopathological feature of ischemic white matter injury. Protection of OLs from ischemic insults would be an important therapeutic strategy to overcome neurologic disabilities caused by ischemic white matter injury. Previously, we found that Toll-like receptor 2 (TLR2) expressed in OL lineage cells provides cell-autonomous protective effects. Based on our previous result, we hypothesized certain endogenous ligands can activate TLR2 in sterile pathological conditions. In the present study, we identified high mobility group box 1 (HMGB1) as an endogenous TLR2 ligand to promote protective effect in ischemia-induced OL death. **Method:** We used primary oligodendrocytes and primary microglia from shaking method by using mixed glia culture. LDH assay was performed 24 hours after oxygen-glucose deprivation. Immunodepletion was performed with HMGB1 specific antibody. HMGB1-siRNA was used in knock-down of HMGB1 in primary oligodendrocytes. All collected media from primary oligodendrocytes or microglia were centrifuged to remove cell debris. Endothelin-1 injection into the right internal capsule was used to make in vivo focal ischemic stroke model. Corner test and pole test

were used for evaluating of neurobehavioral impairment after endothelin-1 injection. **Results:** After oxygen-glucose deprivation (OGD), OLs released HMGB1 into bathing media as early as 1 hour after OGD start. Conditioned media collected from cultured OLs exposed to OGD exhibited protective activity against OGD-induced OL death in a TLR2-dependent manner. Immunodepletion of HMGB1 from the conditioned media or application of glycyrrhizin, a specific HMGB1 inhibitor, abolished the protective activity of the conditioned media. Knock-down of HMGB1 by using siRNA also aggravated OGD-induced OL death. Furthermore, exogenous recombinant HMGB1 application after OGD in OL culture also reduced OGD-induced OLs death. In addition, HMGB1 also affected to microglia expression of cytokines. Contrary to LPS applied microglia CM, HMGB1 applied microglia CM showed no harmful effect to OGD-induced OL death. Because HMGB1 application increased both pro-inflammatory and anti-inflammatory cytokines in microglia. We confirmed the in vivo protective effects of HMGB1 in an endothelin-1 induced focal white matter stroke model. Animals with glycyrrhizin co-injection showed worsening neurobehavioral parameters measured by corner test and pole test compared to those with vehicle co-injection. Glycyrrhizin co-injection animals showed also increased ET-1 induced demyelinating lesion with increased OL death. Furthermore, TLR2(-/-) mice showed similar impairment in neurobehavior parameters with glycyrrhizin co-injection WT mice. **Conclusion:** These results suggests paracrine action of HMGB1 through TLR2 protects oligodendrocytes from ischemic cell death with cell autonomous and non-autonomous manner

P-2-298

Amelioration of abnormal genomic alteration as a molecular therapeutic mechanism of intravenous administration of human mesenchymal stem cells in rodent stroke model: transcriptome analysis

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Background & Objectives: In cerebral ischemia, numerous genes are dysregulated in ischemic brain in response to hypoxia and inflammation. Intravenous administration of mesenchymal stem cells (IV-MSC) showed neuroprotective effect on cerebral ischemia in animal stroke model. However the molecular mechanism of therapeutic effect of IV-MSCs on cerebral ischemia is unclear. Using mRNA microarray technique, we performed transcriptome analysis to investigate the molecular mechanism of therapeutic effect of IV-MSCs on cerebral ischemia. **Method:** We compared the genomic profiling using mRNA microarray technique in rodent stroke model with or without IV-MSCs. Rats with transient middle cerebral artery occlusion (MCAo) were treated with 1×10^6 IV-MSCs 2 hours after MCAo. The mRNA microarray of brain tissue were conducted at 72 hours after MCAo in normal rats (normal group), MCAo rats (sham group), and MCAo rats treated with IV-MSCs (MSC group). Predicted pathway analysis of differentially expressed genes (DEGs) was performed across the tested groups. Functional tests and immunohistochemistry for inflammation were performed. **Results:** In cerebral ischemia, numerous genes were differentially expressed (857 DEGs between sham group vs. normal group), and most of them (88.7%) were upregulated. IV-MSCs attenuated the extent of gene expression that were dysregulated in cerebral ischemia (218 DEGs between MSC group vs. sham group). Predicted pathway analysis revealed that cerebral ischemia abnormally activated a total of 10 signaling pathways that are mainly related to inflammation and cell cycle. IV-MSCs partially attenuated the activation of signaling pathways in ischemic brain. IV-MSCs reduced the inflammatory cells (ED1+ and Iba1+ cells) at peri-infarct area,

reduced the overall infarct size, and induced functional improvement in MCAo rats. **Conclusion:** Using transcriptome analysis, we found that IV-MSCs ameliorated the extent of post-ischemic genomic alteration in ischemic brain. Amelioration of activated inflammation- and cell cycle-related genes in host brain is a molecular mechanism of therapeutic effect of IV-MSCs on cerebral ischemia.

P-2-299

Cilostazol reduces huntingtin accumulation in cultured astrocytes

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Background & Objectives: Huntington's disease (HD) is an autosomal-dominant neurodegenerative disorder caused by a CAG trinucleotide repeat expansion, which results in an abnormally long polyglutamine (polyQ) tract in the N terminus of the huntingtin protein. HD is accompanied by the formation of intracellular inclusions (also known as aggregates) derived from the mutant huntingtin, which process may play a key pathogenic role in inducing neuronal death. The clearance of mutant huntingtin fragments is largely dependent on macroautophagy, generally referred to as autophagy. In this study, we examined the effect of cilostazol, an inhibitor of phosphodiesterase 3, on huntingtin protein accumulation, as well as the possible associated mechanism. **Method:** We used cultured astrocytes transfected with GFP-tagged Huntingtin protein. Transfected cells were treated with cilostazol and observed under a fluorescence microscope. Huntingtin accumulation was also measured by Western blot. The effect of cilostazol on autophagy flux was assessed by Western blot for autophagic markers, light chain 3 (LC3) and p62. **Results:** Cultured astrocytes were transfected with GFP-mHttQ74. At 24 hour after transfection, cells were sham-washed or exposed to 10 μ M cilostazol. Fifteen hours after exposure, sham-washed control cells showed a number of green fluorescent aggregates in their cell bodies. On the other hand, astrocytes treated with cilostazol showed substantially reduced huntingtin aggregates. Western blot analysis confirmed the reduction of huntingtin accumulation. In autophagy flux study, when autophagic flux at the lysosomal stage was arrested with bafilomycin A1, the level of LC3 was increased. Additional treatment with cilostazol further increased the level of LC3, probably indicating that autophagosome synthesis was enhanced. When treated with cilostazol alone, on the other hand, levels of LC3 decreased below that in control, likely indicating that cilostazol increased autophagosome degradation by lysosomes under normal conditions. Further supporting that cilostazol increased autophagy flux, the level of p62 was decreased by cilostazol. **Conclusion:** Cilostazol reduces huntingtin accumulation in GFP-mHttQ74-transfected astrocytes by enhancing autophagy flux.

P-2-300

The Distribution of Cerebral Microbleeds Determines Their Association with Vascular Resistance in Noncardioembolic Stroke Patients

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Background & Objectives: Cerebral microbleeds (CMBs) are assumed to be manifestations of cerebral small vessel disease. Meanwhile, the pulsatility index (PI) measured by transcranial Doppler Doppler, has long been proposed to reflect vascular resistance. Therefore, we investigated whether the presence, burden and location of CMBs are associated with PI. **Method:** Between January 2007 and December 2012, we enrolled 702 consecutive patients diagnosed

with noncardioembolic acute ischemic stroke mechanism. The peak systolic velocity, end diastolic velocity and PI were investigated in bilateral middle cerebral arteries and mean values of PI acquired from bilateral/and or unilateral MCAs were entered for analysis. Binary and multinomial logistic regression analysis was used for determining factors related to the existence of CMBs according to their location. **Results:** The mean age was 62 \pm 11 years and 69.1% were male. PI was higher in patients with CMBs (1.02 \pm 0.25) than in those without (0.86 \pm 0.36) ($p=0.002$). Moreover, PI was higher in the nonlobar CMBs group than the strictly lobar CMBs group ($p=0.016$). In multivariate multinomial logistic regression, PI was independently associated with the nonlobar CMBs group (odds ratio: 2.99, 95% confidence interval: 1.12 - 7.97, $p=0.042$), but not with the strictly lobar CMBs group. **Conclusion:** PI, which representing cerebral vascular resistance, was independently associated with nonlobar CMBs, but not strictly lobar CMBs. These findings suggest a pathophysiological association between PI and CMBs in the nonlobar region.

P-2-301

Sustained attention is linked to the spectral content of sleep EEG activity

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Background & Objectives: During prolonged wakefulness, the homeostatic drive for sleep competes with effort to remain awake, resulting in impaired and/or unstable cognitive function. With increasing degrees of sleep loss, experimental participants exhibit marked decreases in vigilant attention. Despite the extensive empirical literature, there is a lack of knowledge about function of sleep architecture. The goal of current effort is to find out the component which frequency band is correlated with sustained attention. **Method:** The data from Korean Genome and Epidemiology Study (KoGES) conducted since 2009 (fifth evaluation) were utilized in this study. 1166 subject (614 men and 552 women) who completed portable PSG monitoring and psychomotor vigilance task (PVT) were elected for this analysis. An unattended home polysomnography was performed which is consisted of one channel electroencephalogram (C4-A1). Sleep architecture was assessed using visual sleep-stage scoring. The discrete fast Fourier transform was used to calculate the EEG power spectrum for the entire night within contiguous 30-s epochs of sleep for the following frequency bandwidths: delta (0.5-4 Hz); theta (4-8 Hz); alpha (8-12 Hz); sigma (12-14 Hz); beta1 (14-30 Hz); and beta2 (30-45 Hz). **Results:** Conventional sleep quality marker including apnea-hypopnea index, total sleep time, sleep efficiency, Pittsburgh sleep quality index and factor affecting sleep quality as Beck depression inventory were not correlated with PVT performance. However, REM sleep sigma band power increased as the median reaction time (RT) and lapse increased. ($p<0.001$) Total sleep time and REM sleep sigma band power is negatively correlated. ($p=0.002$) **Conclusion:** We expect that NREM delta band which is increased after sleep deprivation or NREM sigma band as sleep spindle frequency associated with PVT performance. However, increasing REM sigma band power is correlated with poor PVT performance, and shorter the total sleep time in PSG, larger the REM sigma band power. Therefore, We hypothesize that sleep deprivation lead increasing REM sigma band power, poor sustained attention.

P-2-302

Dysphagia in familial dysautonomia assessed by surface electromyography

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Background & Objectives: Dysphagia, which is a prominent and disabling feature of a variety of neurologic diseases, may be caused by lesions affecting to different degrees sensory input and motor output. And it is associated with complications in pneumonia, malnutrition, dehydration, and reduced quality of life. Familial dysautonomia (FD) is a rare hereditary sensory and autonomic neuropathy (type III). Neurogenic dysphagia is a common disabling feature and aspiration pneumonia remains a leading cause of death in FD patients. No previous study has investigated the clinical usefulness of surface EMG in the assessment of swallowing in FD. The purpose of this study was to demonstrate the clinical utility of surface EMG and to identify electrophysiological features of swallowing in FD patients. **Method:** We investigated 8 patients with genetically proven FD and 9 age-matched normal controls. The degree of dysphagia was graded by EAT-10 and Functional Oral Intake Scale (FOIS). Surface electromyography was recorded over the submental muscles. The laryngeal movements were recorded by piezoelectric sensor. The subjects were asked to swallow saliva and 20 ml of water in one gulp three times. The limit of dysphagia was obtained by offering different volumes of water. And they drank 100 ml of water as usual. Video was also monitored during the procedure. **Results:** The FD patients showed lower amplitudes of swallowing saliva and longer durations of swallowing 20 ml of water than normal control significantly. The dysphagia limit was significantly lower in FD patients. The clinical severity of dysphagia by EAT-10 had significant negative correlation with dysphagia limit in FD patients ($r=-0.91$, $p=0.002$). The amplitudes of swallowing saliva and durations of swallowing 20 ml of water correlated positively with EAT-10 scores. There were no significant differences of time and needed swallows to drink 100 ml water between two groups. **Conclusion:** Surface EMG provides useful physiological information for the evaluation of swallowing in FD patients. And dysphagia limit may be used as a screening and monitoring test for dysphagia in FD patients.

P-2-303

Different alarm criteria in muscle MEP between cervical and thoracic OPLL surgery

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Background & Objectives: The risk of neurological complications during surgery for thoracic ossification of posterior longitudinal ligament (OPLL) is known to high. Information on change of muscle motor evoked potential (mMEP) between cervical and thoracic OPLL surgery is lacking. Thus, we exploited mMEP in abductor hallucis (AH) muscle and post-operation motor deficit, and evaluated the predictive value of mMEP in individual leg muscles for OPLL surgery monitoring. **Method:** A retrospective study of 111 patients who underwent cervical or thoracic spine OPLL surgery between 2000 and 2014 with intraoperative mMEP monitoring in the bilateral AH muscles. mMEP in the AH muscle that reflect corticospinal tract divided right and left, respectively. So, data were obtained 216 limbs excluded no potential of mMEP in the AH muscle from the onset. Lowest mMEP at during surgery and surgery terminal mMEP compared to baseline, its relationship with postoperative motor deficit, and sensitivity and specificity were analyzed. **Results:** The post-op motor deficit/mMEP change were more frequent in the thoracic spine (8.3%/20.8%) than cervical spine (2.6%/7.3%). In area under the receiver operating characteristic curve (AUC-ROC) of cervical and thoracic OPLL were higher in lowest mMEP at during surgery (0.948) than in terminal mMEP (0.907). At cervical spine, AUC were over 0.90 in lowest mMEP during surgery and

terminal. At thoracic spine, the AUC were 0.864 in terminal while no significant in during surgery mMEP (0.716, $p=0.14$). The cut-off value were higher in thoracic spine (35%) than cervical spine (11%). In mMEP at terminal surgery, sensitivity was 100% in cervical and thoracic spine. However, specificity was higher in cervical spine than thoracic spine. **Conclusion:** This study showed thoracic OPLL surgery is more vulnerable to motor deficit than cervical spine. Therefore, in thoracic OPLL surgery, we suggest that alarm criteria setting would be 60% decrement.

P-2-304

Association of BDNF Val66Met with memory dysfunction and cortical thickness changes in Parkinson's disease: an imaging genetics study

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Background & Objectives: Cognitive dysfunction in Idiopathic Parkinson's disease (PD) which involves diverse cognitive domains is relatively common and progressive. Studies investigating genetic risk factors for cognitive dysfunction in PD are controversial except for GBA mutation. A common polymorphism of BDNF Val66Met that is associated with learning and memory, synaptic plasticity and neuronal survival has been studied for association with cognitive dysfunction in PD. However, results are controversial. Recently, cortical thickness measured by structural magnetic resonance imaging has been used as an endophenotype in genetic association studies. We tested whether genotypes of BDNF Val66Met are associated with dysfunction of a specific cognitive domain and then relationship of BDNF Val66Met-associated cognitive dysfunction with changes of cortical thickness was investigated. **Method:** From a single movement disorders center, 72 patients with idiopathic PD and 35 healthy adult volunteers were recruited. All participants underwent clinical evaluation based on a standardized protocol, three-dimensional volumetric MR Images and genotyping of BDNF Val66Met. In idiopathic PD group, detailed neuropsychological tests were performed to assess multiple cognitive domains; attention, memory, executive, visuospatial and language function. Cortical thickness was analyzed by FreeSurfer software. **Results:** In PD, Global cognitive function assessed by MMSE or criteria by MDS-Task for dementia in PD was not associated with genotypes of BDNF Val66Met. Multivariate analysis showed that total recall and delayed recall scores in Seoul Verbal Learning test (SVLT) were significantly lower in methionine carrier group (Val/Met + Met/Met) than in non-carrier group (Val/Val). In PD, voxel-based analysis of cortical thickness showed that cortical thickness is reduced more in bilateral caudal middle frontal and bilateral precentral regions in methionine carrier group ($p_{corrected}<0.05$). Lesser degree of statistical significance ($p_{uncorrected}<0.005$) for cortical thickness reduction in methionine carriers were in left superior frontal gyrus, right paracentral gyrus, left postcentral gyrus regions, right middle temporal gyrus, left inferior temporal gyrus, right entorhinal gyrus, right superior temporal gyrus, bilateral supramarginal gyrus, right inferior parietal, and right precuneus. Regression analyses with mean cortical thickness of left caudal middle frontal gyrus, right precentral gyrus, right in-

sula, left middle temporal gyrus, left inferior temporal gyrus or left entorhinal gyrus as a covariates showed loss of statistical significance in total recall and delayed recall scores of SVLT. However, in healthy controls, cortical thickness in any region was not associated with genotypes of BDNF Val66Met. **Conclusion:** In PD, BDNF Val66Met is associated with verbal memory dysfunction attributed to cortical thickness changes in frontal and temporal regions.

P-2-305

Cerebral-perfusion reserve after carotid-artery stenting: relationship with power spectrum of electroencephalography

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Background & Objectives: Carotid-artery stenosis may reduce cerebral perfusion, and affects cerebral neuronal activities. Electroencephalogram (EEG) power-spectrum analysis has been used as a tool to evaluate cerebral neuronal activities. We investigated whether the recovery of cerebral-perfusion reserve after carotid-artery stenting (CAS) affects the EEG power spectrum. **Method:** Nineteen patients with carotid-artery stenosis, who were candidates for CAS, were recruited. The subtraction imaging of SPECT and an EEG were taken twice, before and 1 month after CAS. The EEGs recorded twice before and after the injection of acetazolamide (ACZ) in each time point (pre-ACZ EEG and post-ACZ EEG). Twelve patients were excluded due to follow-up loss or difficulty to obtain proper EEG data. Thus 7 patients were included in this study finally. We did the power-spectrum analysis of the EEGs. Firstly, we obtained spectral ratio (SR) of each hemisphere with the data of each EEG. SR was defined as the divided value of power-spectrum sum of fast activities (alpha and beta) by power-spectrum sum of slow activities (theta and delta). And we compared the power-spectrum values between the hemisphere with CAS (CAS-H) and contralateral hemisphere (Contra-H), using "inter-hemispheric index of spectral ratio (IHISR)": $IHISR = (SR \text{ of CAS-H}) / [(SR \text{ of CAS-H}) + (SR \text{ of Contra-H})]$. We observed the changes of SR and IHISR, and tried to find the relevance of them to the recovery of cerebral-perfusion reserve. **Results:** In the subtraction images of SPECTs, cerebral-perfusion reserve was improved after CAS on the stent side in 6 of 7 patients. The other 1 patient without the improvement had bilateral carotid-artery stenosis. On the pre-ACZ EEGs among 3 patients with unilateral carotid-artery stenosis, CAS increased SR in 2 of 3 patients and IHISR in all 3 patients. On the post-ACZ EEGs of them, CAS increased SR in all 3 patients and IHISR in 2 of 3 patients. On the pre-ACZ EEGs among 4 patients with bilateral carotid-artery stenosis, CAS increased SR in 2 of 4 patients, and IHISR in none of them. On the post-ACZ EEGs among them, CAS increased SR and IHISR in 2 of 4 patients respectively. **Conclusion:** The IHISR of pre-ACZ EEGs and SR of post-ACZ EEGs may be electrophysiological tools to evaluate the changes of blood-flow reserve after CAS in patients with unilateral carotid-artery stenosis.

P-2-306

Evaluation of extensor digitorum brevis thickness in healthy subjects: a comparative analysis of nerve conduction studies and ultrasound scans

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Background & Objectives: 1) To evaluate the relationship between the thickness and compound muscle action potential (CMAP) of the extensor digitorum brevis (EDB) muscle 2) to obtain reference values for distal lower ex-

tremity muscle thickness as a possible measure of peripheral neuropathy; and 3) to evaluate various factors associated with unexplained EDB atrophy. **Method:** We measured the thickness of EDB, abductor hallucis brevis (AHB) and tibialis anterior (TA) muscles in 80 healthy volunteers with ultrasound and assessed EDB CMAP with fibular nerve stimulation. Two foot muscle-associated lifestyle factors were assessed and sociodemographic information was collected. **Results:** A significant correlation was observed between the amplitude of the fibular nerve CMAP and EDB thickness. The thickness of each of the three muscles was greater in men compared to women. EDB thickness decreased significantly with age although the thicknesses of the AHB and TA muscles were not correlated with age. **Conclusion:** EDB thickness was closely associated with fibular nerve CMAP but has less variation and differed among groups by age and sex and was not associated with lifestyle factors.

P-2-307

Difference in the responses to head-up tilting test depending on the age

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Background & Objectives: Causes of orthostatic intolerance were classified into three types, such as paroxysmal orthostatic tachycardia syndrome (POTS), reflex syncope, and neurogenic orthostatic hypotension, according to the behaviors of BP and HR after orthostatic stress. And positive responses to HUTT (head-up tilt table test) are classified by VVM (vasovagal mixed), DOH (delayed orthostatic hypotension), DOH+VV (delayed orthostatic hypotension plus vasovagal) type in the 2009 ESC syncope guideline. In VVM type, reflex syncope occurs in persons with normal baroreflexes and it often occurs suddenly triggered by physical pain or emotional stimulus. But, in DOH type, the prevalence of OH increases with age, so OH in the elderly is common. Because BP control becomes progressively more impaired with aging, due to a multitude of reasons, including impaired baroreflex sensitivity, volume status, and venomotor tone. Therefore, we analyzed difference in responses to head-up tilting test depending on the age. **Method:** We included 246 patients, which have done autonomic functions tests due to orthostatic intolerance from January, 2013 to December, 2014. First, we excluded 21 patients with a negative response to HUTT at both phases. Second, we divided 225 patients with a positive response to HUTT into BRG (baseline phase response group) and PRG (provocation phase response group). Third, we subclassified positive responses to HUTT in both phases into 4 groups, such as 1st cerebral and POTS group, 2nd VVM and VVI (vasovagal cardioinhibitory) group, 3rd DOH group, 4th DOH+VV group. Fourth, we statistically analyzed the age distribution in 4 groups on both BRG and PRG each. One-way ANOVA test was used for comparison for age with HUTT response type, and Scheffé' method was used for multiple comparison. The threshold for statistical significance was set at $p < 0.05$. **Results:** 225 patients with a positive response to HUTT were included for study, and mean age was 45.04 ± 20.71 (mean \pm SD) years. In BRG, we included 32 cases and mean age was 28.47 ± 19.30 . In PRG, we included 193 cases and mean age was 47.79 ± 19.67 . We subclassified the patients with positive responses to HUTT in both phases into 4 groups. Four groups of BRG included 20, 6, 4, 2 cases each and 4 groups of PRG included 37, 80, 58, 18 cases each. Comparison for age with HUTT response type used by one-way ANOVA test in each group of BRG, mean ages were 23.95 ± 16.14 , 34.67 ± 23.71 , 48.00 ± 20.80 , 16.00 ± 2.83 ($P = 0.076$), and in PRG, 39.25 ± 17.85 , 42.68 ± 20.25 , 57.47 ± 19.57 , 38.05 ± 15.16 ($P = 0.000$). Therefore, in each group of BRG, the distribution of age is neither different nor statistical significant. But, in each group of PRG, the distribution of age is different and statistical significance, shows vasovagal type syncope was more common in the younger group, orthostatic hypotension was more common in the older group.

Cerebral and POTS type were common in the more younger group, maybe this group has good compensatory function especially baroreflex. **Conclusion:** The age distribution in 4 response types of HUTT presented in PRG with statistical significance, and same age distribution presented in BRG without statistical significance. In PRG, vasovagal type was more common in the younger group, orthostatic hypotension was more common in the older group.

P-2-308

A case of Ross syndrome with segmental anhidrosis and anisocoria: application of finger wrinkle test

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Background & Significance: Ross syndrome is first described by Ross in 1958 characterized by segmental anhidrosis, tonic pupil, and generalized areflexia. Selective postganglionic autonomic denervation could be differential diagnostic point to other autonomic nerve system diseases. Here is the patient who suffer from focal anhidrosis in his left hand and sole, and occasionally found anisocoria and confirmed generalized areflexia who diagnosed Ross syndrome. Finger wrinkle test performed to prove damages in terminal branches of sympathetic nerves. **Case:** 43-year-old man visited our hospital complaining recent aggravation of decreased sweating in his left palm and sole started 5 years ago. Additional history revealed that he occasionally found his pupillary asymmetry 3 years ago, and urinary frequency initiated in his childhood. He has no orthostatic dizziness, tachycardia, heat intolerance or blurred vision in light and no family, medication, or heavy metal exposure history. On exam, his left palm was smooth and dry and relatively his right palm and sole were damp. His right pupil was bigger than left, and didn't response to light stimulation bilaterally. There was no light-near dissociation. 0.125 % pilocarpin test proved bilateral tonic pupil with denervation supersensitivity. Other neurologic exams including cranial nerve exams, motor and sensory function were not remarkable except generalized areflexia. Quantative sudomotor axon reflex test (QSART) and starch test revealed segmental anhidrosis in left palm and hypohidrosis in left sole. Finger wrinkle test was done for evaluation of distal autonomic nerve system impairment. 5 minutes after soaking of both hands in warm water, fingertip of left hand showed loss of wrinkles and red color change different from right hand which representing damage in terminal branch of sympathetic nerve in left hand. Other autonomic function test including adrenergic or cardiovagal function test and nerve conduction study were normal except loss of H-reflex bilaterally. Blood test excluded endocrinopathies, autoimmune and inflammatory diseases. Based on the clinical presentations and the test results, we diagnosed him as Ross syndrome. **Conclusions or Comments:** Ross syndrome is a selective autonomic nerve system dysfunction and should make ensure of clinical triad and exclude other diseases involving autonomic nerve system for diagnosis. Partial dysautonomia including Harlequin syndrome, Holmes-Adie syndrome, and Ross syndrome appears clinical spectrum involving segmental sweating abnormality, papillary changes and decreased deep tendon reflex. In Ross syndrome, selective degeneration of unmyelinated postganglionic autonomic fibers are thought to be cause of this clinical triad. Finger wrinkle formation is caused by sympathetic nerve system like sweating. About 5 minutes after soaking both hands under warm water, normal unmyelinated sympathetic nerve distributing fingertip constrict glomerulus connected to dermis and subcutaneous layer of skin and cause finger wrinkle. It is easy to evaluate injury in terminal branch of sympathetic nerve observing loss of wrinkle, especially, unilateral sympathetic nerve damage like this case. The patient who present atypical tonic pupil, segmental anhidrosis, loss of deep tendon reflex should consider the possibility of Ross syndrome and autonomic function test including finger wrinkle test have a important role replacing skin

biopsy for diagnosis.

P-2-309

Ventricular tachycardia imitating epileptic seizures

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Background & Significance: Syncope are the most common non-epileptic attacks mimicking epileptic seizures. Among them, cardiogenic syncope is potentially life threatening and has a high morbidity and mortality up to 50% within the first 3 years after the initial attack. **Case:** A 49 year old man was referred for the recurrent episodes of loss of consciousness with tonic posture and upward eyes deviation. The electrocardiogram (ECG) showed multimorphologic ventricular tachycardia during attacks, which normalized during intervals. He was treated with isoproterenol and symptoms subsided. **Conclusions or Comments:** Here, we report a case of ventricular tachycardia manifested as epileptic seizures.

P-2-310

Potential risk factors for developing herpes zoster-associated aseptic meningitis in patients with herpes zoster

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Background & Objectives: Herpes zoster (HZ) is a disease caused by re-activation of the latent varicella-zoster virus (VZV) infection. The incidence of HZ has been increasing in the last two decades, and numerous complications have been associated with herpes zoster. HZ-associated aseptic meningitis is considered a rare complication of HZ. However, the condition usually requires hospitalization and a longer treatment period. A retrospective study was performed to identify the potential factors associated with developing HZ-associated aseptic meningitis. **Method:** All outpatients and admitted patients of neurology and dermatology departments of a single tertiary center who were diagnosed with HZ in 2013 and 2014 were included. Total of 794 patients were initially included, and 554 patients were eligible for analysis. The demographics and potential risk factors were compared between the uncomplicated HZ group and HZ-associated aseptic meningitis group. Among the potential factors, the dermatological distribution of skin rash and gender showed statistically significant difference between the two groups. **Results:** Patients with craniocervical distribution of HZ accounted for 87.5% (N = 21) in the HZ-associated aseptic meningitis group, and 54.9% (N = 301) in the uncomplicated HZ group (p = 0.043). The aseptic meningitis group showed higher proportion of males (66.7%, N = 16) than the uncomplicated HZ group (42.8%, N = 237) (p = 0.033). Additional logistic regression analysis was performed, and the gender difference between the two groups remained significant (OR 2.591 in male, p = 0.033, 95% confidence interval 1.078 - 6.228). The dermatome distribution also showed an odds ratio of 5.470 in craniocervical involvement for complication of aseptic meningitis (p = 0.007, 95% confidence interval 1.602 - 18.680). **Conclusion:** In herpes zoster patients, craniocervical distribution of the skin rash and male gender are associated with a higher risk of HZ-associated aseptic meningitis. Closer observation is needed for signs and symptoms of meningitis in patients bearing the risk factors. Clinicians should take it into account when treating patients with HZ.

P-2-311**Unique color perception recovery following optic neuritis after acute Hepatitis A**Jeong Bin BONG¹, Hyun Goo KANG¹, Dae Soo SHIN², Hyung Suk HAN²¹Department of Neurology, Chosun University School of Medicine, ²Department of Neurology, St. Carollo Hospital, Suncheon, South Korea.

Background & Significance: The mechanism underlying optic neuritis in patients with acute hepatitis is unknown. Complement activation and auto-immune-mediated neurotoxicity have been proposed. A case of optic neuritis after hepatitis A with good visual recovery has been reported, but our patient showed improvement with respect to a specific color in retrobulbar optic neuritis. **Case:** A 37-year-old man presented with a short history of jaundice, lethargy, nausea, vomiting, and abdominal pain. Serum transaminase levels were markedly elevated and an infectious screen detected hepatitis A. One week later, the patient developed pain around the left eye, which was exacerbated by eye movement. After few days, he reported worsening vision in his left eye with relative afferent pupillary defect (RAPD), and color vision defect (0/15 Ishihara color plates). Three days later, he experienced complete blindness in his left eye, but his ocular motility and alignment were normal. There was no associated uveitis or optic disc swelling. Brain magnetic resonance imaging revealed high signal intensity and enlargement of the left optic nerve on T2-weighted images but did not show any white matter lesions on brain parenchyma. Visual evoked potentials showed no signal in the left eye. Cerebrospinal fluid analysis was normal, with negative results for the VDRL test and absence of fluorescent treponemal antibodies. The patient was treated with 1 g intravenous methylprednisolone daily for 5 days, followed by a 14-day oral taper. The day after the start of treatment, vision in the left eye began to improve. After 2 days, he could count the number of fingers shown, and began to recognize the color red. One week later, vision in the left eye was even more improved, but he could not recognize any color other than red. In the Ishihara color plate test, he could read only red colored letters. Two weeks after treatment, he could recognize all colors but vision in the left eye did not recover completely. A follow-up examination 3 weeks after onset showed marked improvement in vision. **Conclusions or Comments:** This type of recovery is unique and shows that improvement can occur in perception of a certain color when retrobulbar optic neuritis is resolved.

P-2-312**A case of ramsay Hunt Syndrome that initiated from the palate**

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Background & Significance: Ramsay-Hunt syndrome is caused by the varicella zoster virus, which results in zoster around the ears and face, accompanying pain in the ear, and peripheral facial palsy. Often 5th, 7th, or 8th cranial nerve palsy is present, while 4th, 6th, 9th, 10th, 11th, and 12th cranial nerve palsy is rare. Other symptoms such as hearing loss, ringing in the ear, and vertigo may be present as well. **Case:** A 67-year-old male patient complaining of mouth pain that persisted for 5 days was admitted to the hospital with symptoms of left otalgia and vertigo. Upon admittance, the initial symptoms were oral pain and multiple vesicles on the left side of his palate. At the time of his admission, left facial palsy was not observed. However, it was noted that he had pain in the left trigeminal nerve area and showed decreased facial temperature and tactile sense. The patient developed a left facial rash with associated pain 4 days after admission. Other symptoms included accompanied swelling and periorbital inflammation. There was no history of previous medication or trauma.

Although there were no unusual findings on brain MRI and CSF study, a serum antibody test was positive for anti-varicella zoster virus IgM antibodies. The patient was later diagnosed with Ramsay-Hunt syndrome. Upon admission to the hospital, treatment with oral steroids (prednisolone 60 mg/day) and acyclovir (4,000 mg/day) was initiated. His symptoms improved, and he was released after 2 weeks of treatment. This patient was diagnosed with Ramsay-Hunt syndrome involving the V1-V2 distribution of the trigeminal nerve. The oral mucosal lesions did not represent other dermatomal involvement and were extensions of the V2 branch of the trigeminal nerve via the pterygopalatine ganglion. These types of lesions are associated with involvement of both the greater and lesser palatine nerves, as well as the nasopalatine nerve, which are extensions of the V2 branch of the trigeminal nerve via the pterygopalatine ganglion. **Conclusions or Comments:** In this case, the distribution of lesions were very uncommon, but because lesions may begin from the palate, a diagnosis of Ramsay-Hunt syndrome should be considered in patients presenting with oral lesions and pain.

P-2-313**Acute myeloradiculitis associated with herpes simplex virus type2**

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Background & Significance: Herpes simplex virus 2 (HSV-2) infection is associated with significant diverse neurological manifestation and is frequently observed in immunosuppressed individuals. Myelitis or radiculitis are also observed in HSV-2 infection and skin lesions are often preceded. We present the case report of a HSV-2 myeloradiculitis in immunocompetent woman who showed no skin lesion, no pleocytosis in CSF and negative HSV-2 PCR at initial state. **Case:** A previously healthy 67-year old women presented to a hospital with flaccid paraplegia, paresthesias, urinary retention and constipation. Lower limb weakness and radiating lumbar pain were sudden onset during defecation, after than weakness was progressed but pain was disappeared. She was brought to a local emergency department, magnetic resonance imaging (MRI) revealed L4-5 mild disk herniation but no abnormal signal in spinal cord on first day, where she was diagnosed as spinal stenosis and underwent right L4-5 hemilaminectomy. After 4 day from symptom onset, she was transferred to our hospital due to worsening of weakness of legs. She has no skin lesion or fever. Neurological examination revealed paraplegia (MRC grade 0) and sensory loss of pain, temperature, touch, vibration, proprioception below the level of L1 dermatome. Deep tendon reflex was normal in upper extremities, but areflex in bilateral knee and ankle. Initial CSF analysis was notable for a WBC count of 1/mm³, and RBC count of 20/mm³, and a protein level of 62mg/dL. Result of PCR analyses was negative for HSV-1 PCR, HSV2 PCR, HSV-IgM. Oligoclonal band was positive but AQP-4 antibody was negative. Nerve conduction study was normal. A MRI showed high signal intensity in T2 weighted image from T10 to T12 level where were no significant enhancement in gadolinium enhanced T1 weighted image. She was diagnosed as idiopathic myelitis and treated with intravenous methylprednisolone followed by plasmapheresis but there was no recovery. On 15 day after symptom onset, follow-up MRI revealed patch enhancement on spinal cord below T10 level and nerve root including cauda equina. Follow-up CSF study showed pleocytosis (WBC 327/mm³, 89% lymphocyte dominant and protein 115mg/dL). Follow-up HSV-2 PCR was positive. She was diagnosed as HSV-2 myeloradiculitis and treated with IV acyclovir for 3weeks. Follow-up CSF study showed no pleocytosis and negative conversion for HSV-2 PCR. She was transferred to a rehabilitation hospital on day 65 with no definite clinical improvement. **Conclusions or Comments:** Although initial CSF pleocytosis was negative in our patient, repeated MRI, CSF study including PCR for HSV2 DNA in helped verify the diagnosis. Absent skin rash and negative initial CSF finding, making

diagnosis difficulty, led to delays antiviral treatment and poor neurological outcome. Our case supports the growing evidence that even in an immunocompetent patient, follow-up MRI study and CSF including HSV PCR test should be considered in unexplained myeloradiculitis patients presenting with flaccid paralysis.

P-2-314

Acute respiratory paralysis as an unusual presenting symptom of Japanese encephalitis

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Background & Significance: Japanese encephalitis (JE) is an endemic disease in Korea. Since 1971, the first year that the mass vaccination program against JE was implemented, the incidence rate of JE declined markedly. The usual clinical picture of JE is characterized by several non-specific febrile illnesses initially. The prodromal symptoms are usually followed by headache, vomiting, and a reduced level of consciousness, often heralded by a convulsion. Acute respiratory paralysis is a rare clinical manifestation in JE. To our knowledge, this is the first report of acute JE virus infection characterized by acute respiratory paralysis in an adult in Korea. **Case:** A 56-year-old female presented to our emergency room (ER) with fever, headache, myalgia, nausea and vomiting for 2 days. Her medical history was unremarkable without recent vaccinations. Initial neurologic examination showed neck stiffness and disorientation. The cerebrospinal fluid (CSF) profile was done in the ER disclosing a white cell count (WBC) of 356 per cumm (neutrophil 81%, lymphocyte 19%), protein 53mg/dl, and glucose 126mg/dl (serum glucose 212mg/dl). The initial brain magnetic resonance imaging (MRI) showed focal hyperintense lesion in splenial portion of the corpus callosums with prominent meningeal enhancement. We diagnosed her with viral encephalitis, and initiated intravenous acyclovir (0.1mg/kg for 14 days). However, on the day after admission (day 4 of illness), she developed dyspnea, cyanosis and acute respiratory failure requiring endotracheal tube intubation and mechanical ventilation. Her fever and headache persisted with worsening muscle power (muscle power grading of both proximal upper and lower limbs were 2+). Deep tendon reflexes were decreased but the Achilles reflex remained present. Sensory impairment and cranial nerve involvement were absent. On the 3th day of admission, her consciousness was worse with flaccid paralysis. The electroencephalography showed Frequent, polymorphic or semirhythmic theta to delta slow waves of medium amplitude. The nerve conduction study revealed axonal motor polyneuropathy. The diagnosis of JE was based on the presence of anti-JE virus IgM in the CSF by the ELISA method on day 14 of illness. About forty days after admission, she was still coma with flaccid paralysis, and discharged long-term hospital receiving ventilator care. **Conclusions or Comments:** Our case suggest that acute respiratory paralysis preceding flaccid paralysis presenting in an adult has many different diagnoses, however, Japanese virus encephalitis infection should still be considered as one of the diagnosis in endemic areas.

P-2-315

Acinetobacter baumannii meningoenephalitis related to Incidental durotomy

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Background & Significance: Postoperative bacterial meningitis is a rare com-

plication of spinal surgery and is considered to be a complication related to intraoperative incidental durotomy. A delay in diagnosis or treatment can lead to unfavorable outcome **Case:** 73yrs old in-patient woman was referred to neurology due to suddenly developed semi-comatous mentality. She was underwent lumbar fusion surgery 5days ago. Brain MRI and CSF examination revealed meningoencephalitis and ventricular empyema caused by Acinetobacter baumannii coexistent CSF leakage around OP site. In spite of intrathecal and intraventricular collistin, mental status didn't improved and she died of septicemia **Conclusions or Comments:** Due to the low incidence of postoperative meningitis, few studies have reported these complications. Patients' age and underlying disease can affect occurrence of meningoencephalitis. In our hospital we experienced acinetobacter baumannii meningoencephalitis after lumbar fusion and incidental durotomy

P-2-316

A case of influenza-associated acute necrotizing encephalopathy

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Background & Significance: Influenza virus infection may cause CNS dysfunction including post-influenza encephalitic Parkinsonism, encephalitis lethargica, or influenza-associated acute encephalopathy/encephalitis (IAE). These acute neurologic complications are more common in childhood less than 5 years old. Here, we report an adult case of IAE. **Case:** A 64-year-old woman with a history of hypertension presented to emergency room because of altered mental status. Three days before presentation, she visited a local clinic with headache, nausea, and vomiting. The patient was prescribed with oseltamivir (tamiflu®), which did not relieve her symptoms. One day prior to presentation, her mental status deteriorated and this patient was transferred to our hospital. Her husband and daughter denied recent travel history, recent intake of raw food, infection, or trauma. Vital signs are as follows; body temperature was 38.0°C, blood pressure, 122/82 mmHg; heart rate, 66 beats per minute; respiratory rate, 20 per minute. On neurological examination, her mental status was stuporous and she did not follow commands. Pupils were dilated bilaterally to 5mm/5mm. Oculocephalic reflex was absent, but corneal reflex was intact. She could localize painful stimuli and knee jerks were hyperreflexive with up-going toe signs bilaterally. On cerebrospinal fluid exam, color was yellow with opening pressure of 10 cmH₂O. Protein level was markedly elevated (1542.3 mg/dL) without any abnormalities in terms of cell count (RBC 0, WBC 0) and glucose level (108 mg/dL with 176 mg/dL in serum), Brain MRI/A/D at presentation was unremarkable. She was intubated for airway protection and was admitted to the ICU. She was initially treated with acyclovir on top of oseltamivir with an assumption of viral encephalitis. One day after admission, pupils were more dilated to 5.5mm/5mm, and corneal reflex became absent. Follow-up MRI on hospital day 3 showed ill-defined cystic lesions in the corpus callosum, left thalamus, both inferior cerebellar peduncles, and left pons on T2-weighted image, suggesting necrotic changes. Despite with empirical treatments, this patient had rhabdomyolysis, severe ileus and metabolic acidosis, and expired on hospital day 5. Later, nasopharyngeal swab identified influenza A antigen, and no other pathogen was identified in blood or CSF culture. **Conclusions or Comments:** We reported a rare case of adult-onset influenza-associated acute necrotizing encephalopathy. Pathogenesis of IAE has not clearly defined, and it is still unclear whether influenza virus directly invades the CNS or not. Rapid elevation of proinflammatory cytokines may lead to injury in the vascular endothelium, glial cells, neurons, and other organs, which may result in acute respiratory distress syndrome. Her CSF finding showed cytoalbuminologic dissociation, which made the diagnosis difficult at presentation. This case illustrates that IAE should be included in patients with altered mentality when seasonal influenza is at its peak.

P-2-317**The case of intramedullary spinal abscess**

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Background & Significance: The intramedullary spinal cord abscess is extremely rare spinal cord infection and are generally associated with congenital dermoid lesions or sinus tract, most commonly presenting in the lumbar or midthoracic cord, but may also occur in conjunction with congenital dermal sinuses or trauma. Most of cases were reported in children, adult cases are uncommon. we present a case of Intramedullary spinal cord abscess in multiple spinal level. **Case:** A previously healthy 58-year-old man visited was transferred with progressive on both legs weakness and voiding difficulty. He has DM, but not treated (HbA1C 7.2%). At first day, he felt both leg weakness with numbness and urinary retention were suddenly developed 4 day ago. he could not walk due to paraparesis. On admission, he had both leg muscle power with Medical Research Council (MRC) grade 4-/4 and the hypesthesia below T5 dermatome. The anal sphincter tone was decreased and and extensor plantar reflex were positive in both sides. There was no history of infection, trauma, fever, or surgery of the thoraco- lumbar spine. The CSF study showed leukocytosis (1200) and high protein level. Spinal MRI revealed multiple lobulated cyst with enhancement in spinal cord in level T2-3 and C5-9 level suggested intramedullary spinal cord abscess. Because his leg weakness were worsen progressively and both arm weakness and numbness were newly developed although he was treated extensive antibiotics medication, he was underwent decompressive laminectomy with aspiration and drainage of intramedullary cyst drainage. associated extensive antibiotic medication. **Conclusions or Comments:** Intramedullary spinal cord abscess is extremely rare, but it can make poor prognosis depending on medical treatment time window. So it is mandated prompt surgical drainage accompanied by extensive antibiotics administration in IMSC management is essential to decrease its complication and sequeale.

P-2-318**Atypical mitochondrial encephalopathy, lactic acidosis and stroke-like episodes with false positive 14-3-3 protein mimicking Creutzfeldt-Jakob disease**

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Background & Significance: Mitochondrial encephalopathy, lactic acidosis and stroke-like episodes (MELAS) is characterized by stroke-like episodes, seizures, dementia, and lactic acidosis due to mitochondrial DNA mutation. Positive 14-3-3 protein in cerebrospinal fluid (CSF) is crucial to diagnose with Creutzfeldt-Jakob disease (CJD). We report a patient with MELAS who developed stroke-like episode at relatively later age, had no elevation of lactic acid and positive 14-3-3 proteins in CSF. **Case:** He had disorientation and myoclonus on the left neck, arm and leg. Diffusion weighted image of brain showed right middle & posterior cerebral arterial territory lesion involving pulvinar and cortex suggesting ischemic stroke. Electroencephalography showed 1 Hz periodic lateralizing epileptiform discharges on the right parasagittal area. He was improved after conservative medical therapy. CSF 14-3-3 proteins were positive and genetic analysis confirmed mitochondrial DNA A3243G mutation. **Conclusions or Comments:** MELAS should be considered even if there were positive 14-3-3 proteins as well as no elevation of lactic acid in serum and CSF in patient with atypical vascular territorial stroke.

P-2-319**A case of neurosyphilis presenting as unilateral oculomotor nerve palsy**

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Background & Significance: Neurosyphilis can present with various clinical symptoms. Approximately 5% of patients with secondary syphilis develop associated central nervous system involvement such as headache, meningitis and cranial nerve palsies (mainly, in descending order of frequency, VII, VIII, VI, and II). Here, we report a case of neurosyphilis presenting as unilateral oculomotor nerve palsy. **Case:** A 30-year-old male presented with diplopia in both eyes at a neurology clinic 2 days before. On his familial history, he and his mother and sister had diagnosed ulcerative colitis before 3 years. He is currently no taking the medication related ulcerative colitis. On neurologic examination, he had right paralytic ptosis, and right pupils were fixed with 5mm and were not observed direct and indirect reflex and anisocoric. He had 35 prisms of right exotropia, 2 prisms of right hypertropia at near and distance in the state of primary deviation and 60-prism right exotropia and 4 - prism hypertropia in the state of secondary deviation. He had extraocular movement disorder in all gazes except abduction. Brain MRI showed that increased T2 signal intensity with contrast enhancement in the right oculomotor nerve, cisternal to intraorbital segment, that suggested oculomotor neuritis. On serologic examination, he was diagnosed with syphilis as a result of CSF protein 93, glucose CSF 59/Peripheral 106.0, CSF WBC 60, CSF VDRL (+) 1: 64, and blood TPHA (+) and no evidence of other co-infections (tuberculosis, herpes simplex, enterovirus PCR - negative, ADA 4.7U/L (0 - 10), angiotensin converting enzyme) or vasculitis and autoimmune disease. After administrating continuous intravenous penicillin, his symptom was gradually improved. Two week after, his oculomotor nerve palsy was improved. **Conclusions or Comments:** In patient with unilateral oculomotor nerve palsy, neurosyphilis should be considered one of differential diagnosis.

P-2-320**Trigeminal neuropathy associated with aggressive NK-cell Leukemia**

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Background & Significance: Among the subtypes of NK-cell neoplasm, aggressive NK cell leukemia (ANKL) is the most aggressive and extremely rare neoplasm. It shows a geographical preference to Asians¹. Patients usually present with systemic symptoms; such as general weakness, fatigue and recurrent bleeding. In addition, multiorgan failure, hemophagocytosis and disseminated intravascular coagulation are frequent, and the majority died within a few week. We describe an expired ANKL patient with partial trigeminal neuropathy as the first presentation, which was more sensitively detected with the facial blink test. **Case:** A 36-year-old Korean woman admitted with a chief complaint of severe, paroxysmal, electric-like headache. She also complained of the skin numbness on the left forehead. She had no peculiar past, family, and social history. She had no recent history of fever, night sweat, or weight loss. Neurological examination revealed the diminished sensation on the left forehead, cranial nerve V1 area. Under the clinical diagnosis of trigeminal neuropathy, the magnetic resonance imaging (MRI) study was taken, and the initial report was nonspecific. Blink reflex test showed a delay of ipsilateral R1 latency when left side was stimulated, and there is a problem on the afferent pathway. Then, we closely reviewed the patient's MRI and found an asymmetrical soft tissue enhancing mass like lesion in the left cavernous sinus, and it was ex-

tended to the orbital apex. This lesion was initially thought to be most consistent with inflammatory or metastatic lesion. After we checked her cerebrospinal fluid cytology was within normal range, we managed her symptom with a total five-days-course of steroid pulse therapy targeting to the inflammation. Her initial blood profile was within normal range, except for a mild thrombocytopenia, platelet counts of 124,000/uL, and her liver function test was mildly increased. On the fourth day of admission, we found out that her hemoglobin level was suddenly decreased from the initial result. While we underwent a further study on newly developed anemia, she was discharged with her initial symptoms improved after steroid pulse therapy. Her next out-patient's visit was a week later from the discharge. She complained of general weakness accompanying to recurrent nasal bleeding. Her blood laboratory results revealed pancytopenia with worsen liver function since the last week. She readmitted to hematologic department and received several transfusions, however her pancytopenia became worse. With her newly developed symptoms, her brain lesion might be thought as hematologic malignancy-related problem. Her whole body positron emission tomography showed intense hypermetabolism in multiple areas including left cavernous sinus. Considering hematogenous malignancy, we further performed the bone marrow biopsy, and the result consistent with ANKL. Regardless of treatment, her symptoms became worse and she expired on the 4th week from the initial symptom. **Conclusions or Comments:** We described a patient with ANKL that was initially presenting as trigeminal neuropathy. Our report emphasizes two concerns when treating patients with mild symptoms of trigeminal neuropathy in the clinical practice: first, Blink test might be useful, especially the lesion is localized to the ophthalmic division of the trigeminal nerve. Second, in our knowledge, our patient is the first case that the initial symptom of ANKL was presented as trigeminal neuropathy. Our patient's atypical presentation of leukemia illustrates that systemic malignancy should be considered in the differential diagnosis for various type of cranial neuropathies.

P-2-321

Anti-Ma2 antibody paraneoplastic encephalitis patient diagnosed with peripheral T cell type lymphoma

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Background & Significance: Anti-Ma2 antibody-associated encephalitis is a rare type of paraneoplastic neurological syndromes (PNS) present with various clinical symptoms. Most patients are presented with isolated or combined limbic, diencephalic or brainstem dysfunction. Less commonly, it can also result in cerebellar dysfunction, developing severe limb or truncal ataxia. Testicular germ cell tumors are the most commonly-associated cancers in anti-Ma2 encephalitis followed by lung cancer. It is also reported that breast, ovary, cervical, colon, kidney cancer and lymphoma is associated with anti-Ma2 antibodies. We report a case of anti-Ma2 encephalitis associated with peripheral T-cell lymphoma and showing lesions in bilateral parieto-occipital lobes and basal ganglia in MRI. **Case:** A 41-year-old man had memory dysfunction for 4 months and presented with seizure 10 days ago. He was previously healthy and had no history of exposure to toxins or drugs, except for being a HBV carrier. In neurologic examination, mental status was alert and oriented, there were no abnormalities in cranial nerves, motor, sensory, or cerebellar function examination. He had no psychotic symptoms including confusion, hallucination or delusion, and sleep disorders. In SNSB, MMSE was 28, but visuospatial dysfunction, memory dysfunction in delayed recall and recognition test, and frontal dysfunction were observed. Brain MRI revealed T2/FLAIR high signal intensity in the bilateral parieto-occipital white matter, right cau-

date nucleus and left putamen without gadolinium- enhancement in the corresponding lesions. Hypermetabolism of the bilateral caudate nucleus and putamen was observed in brain 18FDG-PET. CSF analysis showed a white blood cell count of 1/ μ L, a protein level of 34.4mg/dL, and a glucose level of 47mg/dL. Anti-Ma2 antibody was confirmed in both serum and CSF. Other paraneoplastic and autoimmune antibodies including anti-Hu, anti-Yo, anti-Ri, anti-LG11, anti-CV2/CRMP5, anti-amphiphysin, anti-NMDA, anti-AMPA1, anti-AMPA2, anti-CASPR2 and anti-GABA-B antibodies were absent. Enlarged lymph nodes in cervical, supraclavicular, axillary, mediastinal, retroperitoneal and inguinal area were observed in CT scan, and whole body 18FDG-PET revealed hypermetabolism in multiple lymph nodes and bones. Peripheral T-cell lymphoma was confirmed by biopsy of the cervical lymph node. We diagnosed him as anti-Ma2 paraneoplastic encephalitis associated with peripheral T-cell lymphoma. **Conclusions or Comments:** Anti-Ma2 encephalitis is frequently associated with testicular cancer and lung cancer. It usually involves the limbic system, diencephalon, or brainstem, and most had combined areas of involvement. We report herein a patient with subacute memory dysfunction and seizure confirmed as Ma2-antibody-positive encephalitis related to peripheral T-cell lymphoma. Lymphoma is a rare cause of anti-Ma2 encephalitis. And our patient showed involvement of bilateral parieto-occipital subcortex and basal ganglia, which is unusual in anti-Ma2 antibody-associated encephalitis. It is suggested that anti-Ma2-associated encephalitis can be presented with variety of clinical symptoms and abnormalities in brain MRI, and all of typical involvement of the limbic, diencephalic or brainstem may not be seen. So, paraneoplastic encephalitis should be considered and thorough search for occult neoplasms is recommended in patients with unusual presentation of encephalopathy.

P-2-322

Recurrent oculomotor neuritis related to autoimmune hypothyroidism

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Background & Significance: Recurrent oculomotor neuropathy is extremely rare. It has been described in ophthalmoplegic migraine, compressive conditions, essential mixed cryoglobulinemia, diabetes mellitus, and as a post-vaccinal phenomenon. Here we present a patient with recurrent oculomotor neuritis documented by magnetic resonance imaging (MRI) that may have been caused by autoimmune hypothyroidism. **Case:** A 16-year-old girl presented with left ptosis. She had no previous history of medical illness and no headache. On neurologic examination, complete ptosis was noticed in the left eye. The left pupil was dilated and the papillary light reflex was preserved. Ocular movement showed limitations in the medial gaze and upward gaze on the left side. In a neutral position, the left eyeball deviated to the lateral side and downward. The remainder of neurologic examination was normal. Brain MRI revealed dense enhancement and enlargement of the left oculomotor nerve. The right oculomotor nerve was also weakly enhanced, but it was not enlarged. No vascular abnormalities were seen. The patient's complete blood counts and routine chemistry were all normal. The blood tests used to screen for autoimmune diseases were all negative or within normal limits. The anti-nuclear antibody showed a dense fine speckled pattern at a dilution of 1:80. The cerebrospinal fluid analysis was normal. The thyroid stimulation hormone (TSH) level was elevated at 32.7 mIU/L; the normal range is 0.17-4.05 mIU/L. The level of T3 (101 ng/dl) and free T4 (0.94 ng/dl) were within normal limits. The thyroglobulin antibody level (278 IU/ml; normal range 0-30 IU/ml) and antithyroid microsomal antibody (500 IU/ml; normal range 0-100 IU/ml) were increased. The TSH receptor antibody level was normal (0.6

IU/ml; normal range, 0-1.0 IU/ml). The patient was treated with steroid pulse therapy and thyroid hormone replacement. On the third day of steroid pulse therapy, her symptoms began to resolve, and eye symptoms were resolved completely after the 5-day steroid pulse therapy. Five months later, the patient came back to the neurology department due to ptosis in the right eye. She had been on thyroid hormone replacement therapy since her previous visit. The neurologic examination revealed right ptosis. There was no profound extra-ocular movement limitation; however, the patient reported subjective vertical diplopia. Brain MRI revealed enhancement of the bilateral oculomotor nerve. There was less enhancement of the left oculomotor nerve than on the previous MRI. Results of a thyroid function test prior to admission were within normal limits, and the TSH level was 0.17 mIU/L. The levels of the thyroglobulin antibody (1156 IU/ml) and the antithyroid microsomal antibody (585 IU/ml) remained elevated and were even higher than determined at the patient's first visit. The TSH receptor antibody level was normal (0.6 IU/ml). After treatment with high-dose steroid therapy for five days, her symptoms were completely resolved. A follow-up MRI which was performed five months after symptom resolution, showed no enhancement in the left oculomotor nerve and remaining enhancement in the right oculomotor nerve. After discharge, the patient had no recurring eye symptoms during the 5-year follow-up period. **Conclusions or Comments:** This patient showed recurrent ophthalmopathy related to having a high titer of autoantibodies against the thyroid. Brain MRI revealed high signal intensity in the involved oculomotor nerves.

P-2-323

Gamma amino butyric acid B receptor(GABABR) Encephalitis that developed after viral encephalitis: a case report

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Background & Significance: Autoimmune encephalitis is caused by immune response against specific neuronal antigens. One of these antigens was found to be directed against the Gamma amino butyric acid B receptor (GABABR). GABABR antibody is associated to small cell lung cancer (SCLC). Some previous studies reported the viral encephalitis can activate the autoimmune encephalitis. These reports suggest there is robust link between viral encephalitis and NMDA encephalitis. In this study, we reports a GABABR encephalitis that developed after viral encephalitis **Case:** A 53-years-old man with diabetes, were admitted to emergency room for sudden onset of altered mental status. On admission, he showed alert mental state, not confused. There was no fever or other neurologic deficits. We performed routine laboratory test and brain MR imaging that were normal. EEG showed intermittent generalized slow wave, without ictal discharges. 1 day after, his mental state was confused with fever and started recurrent seizure. We performed CSF tapping and EEG monitoring. CSF analysis showed lymphocytic pleocytosis (WBC 60/mm³, lymphocyte 83%) and subtle high protein (58.8mg/dl), but glucose level was normal. Herpes virus type1,2 PCR enterovirus PCR, Tuberculosis PCR, Cryptococcus antigen and Japanese encephalitis antibody and classic paraneoplastic antibody were all negative. Thyroid function test, TPO antibody and thyroglobulin antibody were normal range. EEG showed recurrent ictal discharges. We diagnosed that viral encephalitis with status epilepticus. Acyclovir (30mg/kg/day) and antiepileptic treatment (valproic acid 30mg/kg/day and levetiracetam 3000mg/day) started. 2 weeks later, his mental state was recovered with mild confusion. Follow CSF study showed mild lymphocytic pleocytosis (WBC 17/mm³, lymphocyte 89%) and otherwise unremarkable. We planned to discontinue the antiviral medication and rehabilitation treatment. At 30th hospital day, he restarted recurrent seizure and confusion without fever. We performed chest and abdomen CT and autoimmune encephalitis antibody study. The Chest CT revealed 5.2Cm mass on the right upper

lung with liver metastasis. We could confirm the diagnosis as small cell lung cancer by pathologic study. Brain MRI showed high signal intensity lesion on right medial temporal area. The autoimmune antibody study, showed positive result for GABA-B receptor antibody. These results and clinical symptoms were consistent to GABABR encephalitis. Immunomodulation therapy started with IV immunoglobulin (400mg/kg/day) and IC steroid (1g/day). After immunomodulation treatment, seizure did not recur and clinical symptoms were recovered to near normal state. His family did not want to treat lung cancer that included chemotherapy or radiotherapy. **Conclusions or Comments:** We present a case with GABABR encephalitis secondary to viral encephalitis. Our result suggests that post infective immune mediated mechanism could be a trigger for autoimmune GABAB encephalitis

P-2-324

Accompanied myelitis in a patient with mild encephalitis with a reversible splenic lesion(MERS)

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Background & Significance: Mild encephalitis/encephalopathy with a reversible splenic lesion(MERS) typically shows lesions within the splenium of corpus callosum on Brain MRI. Central nerve system disturbances are mostly accompanied with MERS, but myelitis is a rare manifestation. Here, we present a case with accompanied myelitis in a patient with encephalitis with reversible splenic lesion **Case:** A previous healthy 35-year-old male was admitted due to acute onset of confusion and irritability which developed 2days after receiving intravenous levofloxacin 750mg for 2 days to treat urinary retention in local hospital. 1 week before admission, he initially had fever and headache, followed by urinary retention 5 days later. On arrival, he showed violent behavior and language and was out of control. He had no known history of previous neurologic disorder, including meningitis, encephalitis or epilepsy. Neurological examination were unremarkable except for his mental status though he was not fully cooperative. Physical examination was unremarkable except for mild fever of 37.7°C. The routine laboratory examination showed mild leukocytosis (14,201/ μ l) and slightly elevated procalcitonin (0.08ng/mL). CSF study showed polymorphonuclear pleocytosis (286/mm³), markedly elevated protein(286mg/dL) and glucose level was within normal range. There were no evidences of CNS infection, including bacteria, syphilis, cryptococcus, fungus, anti HSV, anti VZV, EBV, CMV and enterovirus. The level of myelin basic protein was normal value(3.1mcg) in CSF. The serology test of antibodies including FANA, p-ANCA, c-ANCA, RF, Anti dsDNA, anti-cardiolipin and anti-aquaporin- 4-Ab were also all negative. In Brain MRI T2, T2 Flair and diffusion-weighted images, focal high signal intensities were seen in posterior midline corpus callosum. There were correlated low signal intensities on ADC map. The abnormality of MRI was resolved 10days later. After diagnosis of MERS with urinary retention, we started intravenous 10mg/kg of acyclovir and 10mg/6hr of dexamethasone for 4 days. He recovered his mentality one day later and showed no personality change including abusive language. However, he continuously had urinary retention with ataxic gait. In Cervical MRI T2 and CE, cord swelling with diffuse subtle high signals were seen in C to upper T level and focal nodular enhancement in C2-3 and T1. We suspected parainfectious myelitis and additionally treated with 500mg of methylprednisolone intravenously for 4days. After 20 days, the patient was able to urinate himself and was discharged. **Conclusions or Comments:** Though etiology and pathophysiology of MERS is not fully understood, it has been suggested that this condition was induced by infection origin and usually manifested with CNS symptoms. There have been few studies on MERS with encephalitis

phalitis or encephalopathy with urinary retention associated with the spinal cord lesion. To the best of our knowledge, this is the first case of MERS with myelitis in Korea. In case of MERS with unexplained urinary symptom, it is recommend to evaluate possibility of coincidental spinal cord lesion.

P-2-325

Central nervous system involvement in the necrotizing vasculitis presenting bilateral facial palsy

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Background & Significance: Pachymeningitis is a rare inflammatory disease which could be accompanied as the secondary manifestation of systemic vasculitis. We present a 62-year-old male with vasculitis associated pachymeningitis presenting bilateral facial palsy and briefly discusses the clinical and radiologic findings. **Case:** A 62-year-old male admitted to our institution with progressive painful right facial swelling and headache. Five days after the admission, left facial palsy was noticed and consulted to the neurology department. The neurological examination showed bilateral peripheral type facial palsy (incomplete bilateral eyelids closure, worse in the left side) and uvular deviation to the left side. Facial sense and extra-ocular muscle functions were normal at that time. The endoscopic examination revealed inflammation of nasopharyngeal mucosa and mucopurulent exudate. Right ethmoidal sinusitis and thickened dura mater in the bilateral middle cranial fossa were found in the brain MRI. The CSF study and routine blood laboratory were normal except for the elevation of WBC (10,5000/ μ l, reference 4,000 - 10,000), CRP (11.79 mg/dl, reference < 0.3), BUN (40.1 mg/dl, reference 10-26), and creatinine (2.4 mg/dl, reference 0.7-1.4). Blood laboratories for syphilis, anti-nuclear antibody, and anti-neutrophil cytoplasmic antibody were negative. Culture studies for micro-organisms were negative. Necrotizing suppurative inflammation in small vessels and a few giant cells were found in the mucosal biopsy. The patient showed limited improvement with the empirical antibiotics and high dose steroid (prednisolone 1mg/Kg) treatment. **Conclusions or Comments:** Pachymeningitis is a potentially devastating illness leading to severe neurologic deficits even death. A careful neurologic examination and clinical suspicion is important to detect early signs of this disease.

P-2-326

MPNST presenting only unilateral leg pain in a patient with neurofibromatosis type I

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Background & Significance: Neurofibromatosis (NF) is an autosomal dominant disorder which affects the bone, soft tissue, skin and nervous system. Although most neurofibromas are benign, malignant peripheral nerve sheath tumors (MPNST) may occur and this is highly aggressive to have potential to recur as well as to metastasize. We present a case of young male who presented progressive unilateral leg pain, which was unresponsive to analgesics and caused disability of daily life, and finally diagnosed with MPNST on neurofibromatosis-1 **Case:** A 23-year-old male visited orthopedic department with right posterior thigh and calf pain on prolonged sitting or supine position. After walking for about 5 minutes, he could not continue walking due to severe right ankle pain, which was unresponsive to simple analgesics. He was previously healthy and didn't have trauma history at all. Initially, He felt only mild intermittent tingling sense of right leg since his middle school age, but it developed to severe pain gradually. He had never been experienced hearing impairment, vertigo or visual disturbance. On physical examination, multiple

café-au-lait macules and nodules were found on whole body. And he had also multiple freckles on both axillary areas. Neurofibromatosis type I was suspected, so he was referred to the department of neurology. On neurologic examination, we could not find any focal neurologic deficit. There were no family members similar with him. Brain MRI showed normal findings with no evidence of optic glioma or schwannoma and spine MRI also presented no intramedullary lesions. Based on the National Institute of Health (NIH) diagnostic criteria, the patient was diagnosed with neurofibromatosis type I. Electrodiagnostic study revealed right common peroneal neuropathy. We performed MRI for right leg and it showed large mass about 12cm at right distal femur, originated from sciatic nerve and at right proximal tibia level, a 7cm mass, probably originated from deep peroneal nerve. Both lesions were with heterogenous enhancement. And, no evidence of distant metastasis was found on PET-CT. He underwent mass excision. The pathology of distal femur mass was neurofibroma, and malignant peripheral nerve sheath tumor was proven at the proximal tibial lesion. After complete surgical resection, postoperative adjuvant chemotherapy is ongoing. **Conclusions or Comments:** Imaging study should be considered with possibility of neurofibroma or MPNST when persistent or atypical pain in a young adult especially who has specific skin lesion suggesting neurofibromatosis. And also when patient who diagnosed with neurofibromatosis presents uncontrolled pain, new neurological deficits, or alteration in the characteristics of known neurofibroma, we should evaluate with high possibility of MPNST.

P-2-327

Green tobacco sickness in elderly patient who had never been exposed to nicotine

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Background & Significance: Green tobacco sickness is an illness caused by dermal exposure to nicotine. Common symptoms of disease are dizziness, headache, nausea, vomiting, severe general weakness, fluctuation of blood pressure or heartbeat, abdominal cramping, chills, increased sweating, salivation and difficulty breathing. Sometimes it could be life threatening. Green tobacco sickness usually tends to be occurred when the leaf of tobacco is wet. Here we report an elderly person who had severe nausea, vomiting and nonspecific type dizziness after harvesting tobacco for first time in her life. **Case:** A 79-year-old female who had pulmonary tuberculosis and osteoarthritis and never been drinking or smoking arrived emergency room for evaluation of sudden onset dizziness. She had been taken medicine including steroid for her osteoarthritis. She felt severe nonspecific type dizziness, nausea, vomiting and sweating. Her dizziness aggravated when moving her head but dizziness never disappeared while not moving. She had dry mouth and decreased skin turgor. There were no specific findings in motor examinations, head roll test, Dix-hallpike test, finger to nose test, and heel to shin test. We couldn't perform other neurologic examination including Romberg test because she refused. Initial laboratory tests shown iron deficiency anemia and mild adrenal insufficiency. The type of dizziness and results of neurologic examination were not matched with vestibulopathy, central type dizziness or presyncopal type dizziness. Magnetic resonance imaging and angiography of the brain did not show relevant abnormal findings. Four days later with supportive care, she said that she harvested green tobacco for 6 hours the day of admission and tobacco harvest was her first time in life. She sweated a lot during the hot and humid weather and tobacco leaves got wet in rain the night before. Serum cotinine which is metabolite of nicotine was tested at 5 days of admission and serum cotinine level was 16 ng/ml (serum cotinine level in non-smoker even if secondhand tobacco smoke < 1ng/ml). Ten days later, she could be discharged.

Conclusions or Comments: This report describes a case of green tobacco sickness. The patient had dermal exposure to high level of nicotine because the level of serum cotinine level was significantly high despite the test of serum cotinine was performed 5 days later. We couldn't get her standing and check her gait ability because she had severe dizziness and headache. Cerebellar stroke could not be ruled out before taking brain imaging. Usually green tobacco sickness occurs to young adults, who had never been exposed to tobacco, after harvesting wet green tobacco leaf, green tobacco sickness could be happened to all people who had never been exposed to nicotine.

P-2-328

Severe hypothermia caused by organophosphate poisoning

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Background & Significance: Organophosphate (OP) self-poisoning is a significant cause of morbidity and mortality in the developing world, with the in-hospital case fatality usually reported to be between 10% and 20%. Because OP are acetylcholinesterase (AChE) inhibitors, the rapid accumulation of acetylcholine (ACh) in the synaptic junctions of central nervous system and peripheral tissues results in a cholinergic crisis that characterized by nicotinic neuromuscular (fasciculation, hypercontraction, and paralysis), muscarinic parasympathetic (hypersalivation, diarrhea, loss of bladder control, lacrimation, miosis, bradycardia, and hypotension) and central cholinergic (agitation, vomiting, seizure, and coma). Review of the literature regarding human responses to OP poisoning appears to suggest that an elevation in body temperature is a frequent outcome. We report a case of hypothermia caused by OP poisoning. **Case:** A 48-year-old man was referred to emergency room for the evaluation of mental change. He was diagnosed diabetes mellitus 13 years ago and operated for distal pancreatectomy caused by pancreatic pseudocyst and chronic pancreatitis 4 years ago. He was chronic alcoholics, but oral intake was not poor. In neurological examination, he had semicomatous mentality, bilateral miosis (2mm/2mm), sluggish light reflex, negative vestibulo-ocular reflex, negative corneal reflex, quadriplegia, absent deep tendon reflex, and absent neck stiffness. He was showed intermittent vertical and horizontal opsoclonus. On vital sign, systolic blood pressure 60 mmHg, sinus tachycardia (heart rate 120~130/minute), body temperature (BT) by tympanic thermometry 32.6°C was checked. Because metabolic and respiratory acidosis was checked in arterial blood gas analysis, he was intubated immediately. Initial blood sugar was 174mg/dl. He was showed hypersalivation, and there was no specific odor such as alcohol and solvent. The laboratory tests including serum thiamine and inflammatory marker, and brain CT, MRI did not show any remarkable finding and 12-channel EEG was showed continuous semirhythmic theta activities without epileptiform discharges. We started supportive care including warming, but his neurologic deficits were not improved even recovered BT of normal range. Three days later, his body temperature dropped to 34°C again. Because we considered the possibility of organophosphate intoxication, atropine (12mg/day) and pralidoxime (12g/day) was started and pseudoacetylcholinesterase (PChE) and RBC acetylcholinesterase activity (AChE) test were done. He was slowly improved after applying atropine and pralidoxime. On the 5th day of admission, OP intoxication was confirmed by low level of PChE (172 U/I, normal range 3400 -14200) and AChE activity (4963 U/I, normal range 11188-16698). **Conclusions or Comments:** This case describes the patient with severe hypothermia that unusual clinical course of OP poisoning. OP poisoning has been reported to interfere with the control of ACh-regulated homeostatic mechanisms such as temperature regulation. OP poisoning appears to suggest that an elevation in BT is frequently occurred.

However, the understanding of the pathophysiology of OP poisoning is incomplete in many respects. And cases are complicated by concurrent illnesses (in particular aspiration pneumonia) and interventions such as atropine. Neurologists keep in mind that hypothermia caused by organophosphate poisoning.

P-2-329

Pseudotumor cerebri in a patient undergoing sexual reassignment therapy

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Background & Significance: Pseudotumor cerebri means a syndrome of headache, papilledema, absent focal neurologic signs, or an intracranial mass on brain imaging. Pseudotumor cerebri has a no firmly established cause in most cases, and is generally referred as idiopathic intracranial hypertension. We report a case of pseudotumor cerebri from testosterone therapy in young female. **Case:** A 23-year-old female presented with a two-month history of blurred vision in both eyes. She was overweight and looked man like. For sexual reassignment, she was on intramuscular testosterone propionate 250mg every three-week. Her past medical history was otherwise unremarkable. Fundoscopic exam revealed bilateral optic disc swelling. Her visual acuity, color vision, visual field was normal on ophthalmological examination. Other neurological examinations were normal. She went on orbit and brain MRI with enhancement, which showed no evidence of optic neuritis and space occupying lesion. Lumbar puncture revealed an increased cerebral spinal fluid(CSF) pressure of 270mmCSF. CSF profile was normal. Serum free testosterone level was increased to 12.35 pg/mL. Estradiol level was normal. Intracranial hypertension due to increased serum testosterone was diagnosed. **Conclusions or Comments:** This case describes papilledema due to secondary intracranial hypertension caused by sexual reassignment therapy. Rarely, papilledema is only manifestation of intracranial hypertension. But, it can cause visual loss. Pseudotumor cerebri should be suspected when a patient on sexual reassignment therapy has visual disturbance and lumbar puncture should be performed.

P-2-330

Stroke-like manifestation in metastatic pancreatic cancer

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Background & Significance: Pancreatic cancer occurs in old ages and is not infrequently presented with distant metastasis. However, central nervous system (CNS) is rarely affected. **Case:** 79-years-old man was admitted due to sudden right arm dysesthesia and weakness. Neurologic examination showed dysarthria, right central type facial palsy and right hemiparesis (4/5). Brain MRI showed multifocal lesions in left cerebral and cerebellar hemisphere, which were high signal intensities on fluid inversion recovery images with diffusion restriction in diffusion weighted image. Leptomeningeal and parenchymal enhancement was found on gadolinium-enhanced T1 weighted image. Abdominal imaging showed multifocal metastatic mass and pancreatic cancer. No malignant cell was found in cerebrospinal fluid examination. **Conclusions or Comments:** Stroke-like manifestation can be an initial presentation of CNS tumor. High degree of suspicion is needed for an early and prompt diagnosis of CNS metastasis.

P-2-331

Relapsing polychondritis with encephalitis: a case report

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Background & Significance: Relapsing Polychondritis is a rare autoimmune disease characterized by affecting cartilage and other proteoglycan rich structures. The clinical manifestations of relapsing polychondritis include recurrent chondritis of the ears, nose, larynx and tracheobronchial tree. However, central nervous system involvement is rare in cases of relapsing Polychondritis. We have experienced a case of relapsing polychondritis with encephalitis, who was treated effectively with steroid **Case:** A 62 years-old man was admitted with swelling in both ears and fever for several days. The patient showed drowsy mentality, disorientation, fever and redness, heatness in both ears. He had no past history related to immunological Disease. Blood test revealed high level of white blood cell (WBC, 16530/u) and serum C-reactive protein (CRP, 6.82 mg/dL). Brain MRI showed prominent leptomeningeal enhancement without other parenchymal involvement, and Contrast-enhanced MRI revealed bilateral auricle enhancement. Cerebrospinal fluid (CSF) study reveals pleocytosis (135 leukocyte/uL, lympho 70%), protein 206, Glucose 69 (serum glucose 151). At first, the patient was suspected to have viral encephalitis and treated with acyclovir and steroid. After being treated with steroid and acyclovir for 14 days, the patient had symptomatic relief and discharged. Two days later, a flare up of both ear symptoms including swelling, heatness, redness was developed, which was considered as cellulitis of bilateral ear. Antibiotics was administrated but symptoms was not relieved. Two weeks later, The patient showed drowsy mentality, confusion and disorientation again. CSF study was done again, and reveals pleocytosis (630 leukocyte/uL, lympho 70%), protein 215.0 mg/dL, Glucose 76 mg/dL (serum glucose 200 mg/dL). The patient also complained visual disturbance of both eye and hearing difficulty on both ear. Fundoscopy reveals uveitis and optic neuritis of right eye. Audiometry showed sensorineural hearing loss of both ear. In combination auricular chondritis, uveitis and sensorineural hearing loss, relapsing polychondritis with meningoencephalitis was suspected. Intravenous methylprednisolone at a dose of 1g daily was administered for 5 days, and the patient's symptoms improved considerably. The patient discharged without any neurologic problem. **Conclusions or Comments:** Encephalitis is a rare, but serious complication of relapsing polychondritis. If a patient diagnosed with encephalitis has other symptoms such as auricular chondritis, nasal chondritis or ocular involvement, relapsing polychondritis should be included in the differential diagnosis. Early diagnosis and treatment with steroid may be important for a better outcome

P-2-332

Anti-NMDA receptor encephalitis presenting as an acute psychotic symptom in a man

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Background & Significance: The disorder, named "anti-NMDAR encephalitis," usually present with acute behavioral change, psychosis, and catatonia that evolve to include seizures, memory deficit, speech problems, and autonomic and breathing dysregulation. When severe, the disorder may become life-threatening and intensive care treatment is warranted. This case report

outlined below discusses the clinical presentation, workup, and treatment of a patient diagnosed with anti-NMDAR encephalitis. Key features of this disorder, such as a clinical presentation dominated by psychosis, are emphasized. This report thus highlights the increasing need for psychiatrists and other relevant medical disciplines to become aware of this underdiagnosed disorder and consider it in their differential diagnosis. **Case:** A 33-year-old man was referred to our emergency room following a 7-day history of agitation, and visual hallucinations in the form of "It seems a strange woman." Since the patient's agitation and hallucinations worsened, he underwent psychiatric consultation, in which a psychotic state had been diagnosed. One day later, due to further exacerbation in his mental state, the patient was referred to neurology. Neurological examination did not disclose any focal signs and routine laboratory workup was normal. Upon admission, the patient experienced a generalized tonic-clonic seizure. EEG revealed intermittent slow activity on right frontotemporal region, and valproic acid loading was initiated. CSF analysis revealed 17 lymphocytes, no red blood cells, and normal protein and glucose concentrations. Intravenous acyclovir was initiated until obtaining negative PCR results in the CSF. Brain MRI showed subtle increased signal change at right frontal, parietal and temporal cortex on T2WI. The laboratory examination of serum and CSF for relevant infectious and immune causes was unremarkable. Given the acute presentation of a severe psychotic state, with cognitive impairment, and seizures, a clinical diagnosis of probable autoimmune encephalitis was made, and anti-NMDAR encephalitis was suspected at the top of the differential diagnosis. Hence, CSF and serum samples were sent to the autoimmune encephalitis research group of SNUH, for serological testing of anti-NMDAR antibodies. As prompt initiation of immunotherapy has been recently shown to predict better outcome, the patient was started on steroid pulse therapy for a total of five days. Seven days later, CSF serologic results returned, demonstrating reactivity with the NMDAR, thus confirming the diagnosis of anti-NMDAR encephalitis. **Conclusions or Comments:** This report illustrates the case of a man admitted following an abrupt-onset episode of hallucinations and confusion. Eventually, the appearance of fluctuations in mental status combined with repeated generalized seizures led the medical staff to suspect the diagnosis of anti-NMDAR encephalitis. Notably, rapid institution of immunosuppressive treatment served as a first-line therapy. These interventions resulted in marked improvement over several days' time. Recent data suggest that over half of patients respond to first-line immunotherapy (steroids, IVIG, and plasmapheresis, alone or combination) within four weeks. The psychiatric symptom often predominate in the early phases. Such a presentation often leads patients to first seek psychiatric evaluation and treatment, causing a crucial delay in diagnosis and institution of immunotherapy. Moreover, a recent large cohort-based study revealed that 4% of patients presented with isolated psychiatric episodes. In this cohort MRI of the brain, EEG and CSF studies were abnormal in 33%, 90%, and 79% of patients, respectively. In summary, the present case illustrates the pertinent need for psychiatrists, neurologists, and emergency-room physicians to become aware of anti-NMDA-R encephalitis. The inclusion of this disorder in the differential diagnosis is critical, as prompt initiation of immunotherapy, if appropriate, could dramatically affect outcome.

P-2-333

Cerebral syncope versus neurally mediated syncope

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Background & Objectives: Syncope is sudden transient loss of consciousness and spontaneous complete recovery due to transient global cerebral hypoperfusion. Cerebral syncope is abnormal cerebral hypoperfusion without significant changes of blood pressure and heart rate. However, the study pop-

ulation of previous reports was too small. The aim of our study was to characterize cerebral syncope as a disease entity compared with neurally mediated syncope (NMS). **Method:** We reviewed patients with presyncope or syncope history who had undergone head up tilt test with transcranial doppler monitoring during 10 years in Asan Medical Center. A total of 1,137 patients with recurrent syncope or presyncope were recruited from January 2000 to March 2015. We defined cerebral syncope as decrement of mean cerebral blood velocity of MCA more than 30 percentage from baseline without significant change of blood pressure and heart rate during tilt test. Control group was consisted of neurally mediated syncope, which was defined as hypotension with bradycardia or no reflex tachycardia in tilt test. Patients with risk factors undergone other diagnostic tests such as EKG, echocardiography, 24hr holter monitoring, heart SPECT, EEG, and brain imaging. **Results:** We found 55 patients with cerebral syncope and 78 patients with NMS in this study. There were significant differences in mean age (45.5 ± 15.3 vs 59.6 ± 15.8) and past medical history of hypertension (23.6% vs 66.3%) and diabetes (9.1% vs 23.6%) between the cerebral syncope and NMS groups. More patients were detected for old stroke, leukoaraiosis, and cerebral vessel stenosis in NMS group. The mean ratio of change in terms of sBP, dBP, and MCV were 7.1%, 2.29%, and 35.33% in cerebral syncope group and 20.91%, 11.8%, and 21.93% in NMS group. The time trends of cerebral velocities between groups were not significantly different. **Conclusion:** In this study, we demonstrated that the cerebral syncope group was differed from the NMS group in baseline characteristics. In addition, we found cerebral blood flow velocity decreased more prominently in cerebral syncope group compared with the NMS group.

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