



김 용 원

경북대학교 의과대학 신경과학교실

Neurocritical Care in the View of Interventional Neurologist

Yong-Won Kim, MD

Department of Neurology, Kyungpook National University Hospital, Daegu, Republic of Korea School of Medicine, Kyungpook National University, Daegu, Korea

Reperfusion of the occluded artery by intravenous thrombolytics and/or endovascular therapy and stroke unit care is considered the effective approach for improving the outcome of stroke. However, successful reperfusion can only be achieved in a limited cases of acute ischemic stroke. Moreover, there are much more patients who were not eligible for reperfusion therapy. Regardless of reperfusion therapy, neurocritical care in acute ischemic stroke can reduce risk of severe complications and lead improvement of patient outcomes.

Key Words: Stroke, Endovascular therapy, Neurocritical care

Introduction

Advancements of reperfusion therapies including intravenous rtPA and endovascular treatment lead to achieve improvement of reperfusion rates and clinical outcome than the past.¹⁻⁶ Considering the current guidelines for reperfusion therapy,^{7,8} eligible candidates for reperfusion therapy are increased. These advances have been paralleled by an increase in the number of ischemic stroke patients requiring neurocritical care management such as post-reperfusion therapy, and hemorrhagic complications.

However, patients who were not eligible for reperfusion therapy still exist. In these patients, post-stroke care is also important to decrease complications and improve the clinical outcome. Neurocritical care can play an important role

in neurological monitoring for early recognition and management of complications. Therefore, we review neurocritical care in the sight of interventional neurologist.

Therapeutic Hypothermia

Therapeutic hypothermia is reported to exert a neuroprotective effect and facilitate ICP control in various conditions such as hypoxia, stroke, and traumatic brain injury. The underlying neuroprotective mechanism is believed to involve a reduction in cerebral metabolism, oxygen consumption, glucose metabolism, neuroinflammation, free radical production, and cell death.⁹ Various neuroprotective measures have been proposed in cerebral ischemia.¹⁰ In addition, reduction of cerebral blood volume, vasogenic edema, blood-brain barrier disruption have been identified to be effective in ICP control.⁹ In experimental studies of stroke, initiation of therapeutic hypothermia early after from stroke onset revealed decrease in infarction volume and improvement in the functional outcome. Small pilot studies have been undertaken in stroke patients, which

Yong-Won Kim

Department of Neurology, Kyungpook National University Hospital, Kyungpook National University School of Medicine, 130 Dongduk-ro, Jung-gu, Daegu 42119, Korea

Tel: +82-53-420-5758 Fax: +82-53-422-4265

E-mail: yw.kim23@gmail.com

suggest the potential benefits of therapeutic hypothermia in terms of neurological outcome and mortality.

The optimal time for the initiation of hypothermia induction has not yet been clearly established. However, recent human studies on the induction of hypothermia immediately after reperfusion therapy have also shown an improvement in the clinical outcome.¹¹ Most clinical studies on hypothermia for acute ischemic stroke have mainly focused on mild hypothermia (core temperature of 32-35°C) and cooling duration of 5 h to 72 h.¹¹⁻¹⁵ The cooling duration differs with the purpose of hypothermia induction. Studies based on edema control pertain to the maintenance of hypothermia for a longer duration (34-72 h).^{12,16} The rewarming process should also be executed with caution due to the risk of rebound cerebral edema, increased ICP, and rebound hyperthermia. Gradual rewarming generally at the rate of 0.1-0.25°C/h, is important to prevent such complications.

The commonly used cooling methods are surface cooling and endovascular cooling. No differences have been reported in the outcomes achieved with both cooling methods.¹⁷ Surface cooling systems consist of cooling blankets or surface pads and offer the advantages of easy application, rapid initiation, and relatively low rate of complications. In the endovascular cooling method, a cooling catheter is introduced through the femoral or subclavian vein. Cooling is achieved by circulating cooled saline within the cooling catheter. This system offers the advantage of accurate temperature control during maintenance and rewarming period.

The most frequent complication is shivering, which can interfere with the cooling process and increase the rate of systemic metabolism.¹⁸ The cardiopulmonary complications associated with hypothermia include bradycardia, arrhythmia, hypotension, pneumonia, and pulmonary edema. Electrolyte imbalance such as hypokalemia, hypomagnesemia, hypophosphatemia, and hypocalcemia have also been reported as accompanying complications. Once therapeutic hypothermia is induced, it is difficult to recognize complications by clinical symptoms. Therefore, con-

tinuous monitoring via serial chest radiography and laboratory tests are important for the detection of complications.

ICP monitoring

The main complications of acute ischemic stroke are ischemic brain edema and intracranial hemorrhage (ICH). Symptomatic ICH occurs in 3.6% to 7.7% of the patients receiving intravenous or intra-arterial thrombolytic therapy and anticoagulation therapy.^{7,19-23} Massive cerebral infarction is associated with cytotoxic or vasogenic brain edema and ICH, which can in turn increase ICP. Increased ICP can reduce cerebral perfusion, cause tissue hypoxia, and finally lead to brain herniation.²⁴

Increased ICP can be recognized by serial changes in neurological symptoms, including pupillary dilatation, loss of brainstem reflex, and change in breathing patterns. However, the appearance of these symptoms may indicate that it may be too late for the effective management of ICP. Therefore, AHA/ASA guidelines recommend early monitoring of patients with high risk of brain edema.⁷ Establishment of a ventricular drain with an external pressure gauge is the current gold standard for ICP monitoring.²⁵ This drainage system provides information about ICP as well as intracranial compliance and can be used to control ICP through cerebrospinal fluid drainage. This measurement represents the global ICP. In cases of acute ischemic stroke, focal measurement of ICP may accurately reflect the pressure changes caused by compartmentalization.²⁶ Intraparenchymal monitoring devices are effective for focal ICP measurement and easier to apply than intraventricular devices.

CONCLUSIONS

Until now, there are substantial differences between reperfusion rate and favorable clinical outcome though advancement of reperfusion therapy. And, there are much more patients who were not received reperfusion therapy. Neurocritical care in acute ischemic stroke can reduce risk of severe complications and lead improvement of patient

outcomes, regardless of reperfusion therapy.

References

1. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995; 333:1581-1587.
2. Berkhemer OA, Fransen PSS, Beumer D, et al. A randomized trial of intraarterial treatment for acute ischemic stroke. *N Engl J Med* 2015;372:11-20.
3. Goyal M, Demchuk AM, Menon BK, et al. Randomized assessment of rapid endovascular treatment of ischemic stroke. *N Engl J Med* 2015;372:1019-1030.
4. Jovin TG, Chamorro A, Cobo E, et al. Thrombectomy within 8 hours after symptom onset in ischemic stroke. *N Engl J Med* 2015;372:2296-2306.
5. Saver JL, Goyal M, Bonafé A, et al. Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke. *N Engl J Med* 2015;372:2285-2295.
6. Campbell, B. C. V., Mitchell, P. J., Kleinig, T. J., Dewey, H. M., Churilov, L., Yassi, N., et al. Endovascular therapy for ischemic stroke with perfusion-imaging selection. *N Engl J Med* 2015;372:1009-1018.
7. Jauch EC, Saver JL, Adams HP, et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the american heart association/american stroke association. *Stroke* 2013; 44:870-947.
8. Powers, W. J., Derdeyn, C. P., Biller, J., Coffey, et al. 2015 American Heart Association/American Stroke Association Focused Update of the 2013 Guidelines for the Early Management of Patients With Acute Ischemic Stroke Regarding Endovascular Treatment. *Stroke* 2015;46:3020-3035.
9. Polderman KH. Mechanisms of action, physiological effects, and complications of hypothermia. *Critical Care Medicine* 2009;37:S186-202.
10. MD DT-CW, MD PJCG. Hypothermia for acute ischaemic stroke. *Lancet Neurol* 2013;12:275-284.
11. Hwang Y-H, Jeon J-S, Kim Y-W, et al. Impact of immediate post-reperfusion cooling on outcome in patients with acute stroke and substantial ischemic changes. *J NeuroInterv Surg* 2017;9:21-25.
12. Schwab S, Georgiadis D, Berrouschot J, et al. Feasibility and safety of moderate hypothermia after massive hemispheric infarction. *Stroke* 2001;32:2033-2035.
13. De Georgia MA, Krieger DW, Abou-Chebl A, et al. Cooling for Acute Ischemic Brain Damage (COOL AID): a feasibility trial of endovascular cooling. *Neurology* 2004;63:312-317.
14. Milhaud D, Thouvenot E, Heroum C, Escuret E. Prolonged moderate hypothermia in massive hemispheric infarction: clinical experience. *J Neurosurg Anesthesiol* 2005;17:49-53.
15. Hong JM, Lee JS, Song HJ, et al. Therapeutic Hypothermia After Recanalization in Patients With Acute Ischemic Stroke. *Stroke* 2013;45:134-140.
16. Els T, Oehm E, Voigt S, et al. Safety and therapeutical benefit of hemicraniectomy combined with mild hypothermia in comparison with hemicraniectomy alone in patients with malignant ischemic stroke. *Cerebrovasc Dis* 2006;21:79-85.
17. Gillies MA, Pratt R, Whiteley C, et al. Therapeutic hypothermia after cardiac arrest: a retrospective comparison of surface and endovascular cooling techniques. *Resuscitation* 2010;81:1117-1122.
18. Badjatia N, Strongilis E, Gordon E, et al. Metabolic impact of shivering during therapeutic temperature modulation: the Bedside Shivering Assessment Scale. *Stroke* 2008;39:3242-3247.
19. Berkhemer OA, Fransen PSS, Beumer D, et al. A randomized trial of intraarterial treatment for acute ischemic stroke. *N Engl J Med* 2015;372:11-20.
20. Goyal M, Demchuk AM, Menon BK, et al. Randomized assessment of rapid endovascular treatment of ischemic stroke. *N Engl J Med* 2015;372:1019-1030.
21. Jovin TG, Chamorro A, Cobo E, et al. Thrombectomy within 8 hours after symptom onset in ischemic stroke. *N Engl J Med* 2015;372:2296-2306.
22. Saver JL, Goyal M, Bonafé A, et al. Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke. *N Engl J Med* 2015;372:2285-2295.
23. Anderson CS, Robinson T, Lindley RI, et al. Low-Dose versus Standard-Dose Intravenous Alteplase in Acute Ischemic Stroke. *N Engl J Med* 2016;374:2313-2323.
24. Mokri B. The Monroe-Kellie hypothesis: applications in CSF volume depletion. *Neurology* 2001;56:1746-1748.
25. Steiner LA, Andrews PJD. Monitoring the injured brain: ICP and CBF. *Br J Anaesth* 2006;97:26-38.
26. Schwab S, Aschoff A, Spranger M, et al. The value of intracranial pressure monitoring in acute hemispheric stroke. *Neurology* 1996;47:393-398.