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AD and LBD: biomarkers perspective

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Dementia with Lewy bodies (DLB) is the second most common neurodegenerative dementia, associated with cognitive decline with fluctuation, visual hallucination, rapid eye movement sleep behavior disorder (RBD), and parkinsonism. The pathological hallmark of DLB is Lewy bodies, which are intracellular inclusions of protein that are predominantly composed of α -synuclein. As one of the α -synucleinopathies, aggregation and spreading of α -synuclein are thought to have a central role in progressive neuronal cell death in the cortex and subcortex in patients with DLB. In addition, co-occurrence of Alzheimer's disease (AD)-related pathologies, which have synergistic interactions with α -synuclein, confer a distinct clinical phenotype in DLB.

The significance of parkinsonism in AD has been over-

looked because these motor signs are often considered as part of the normal aging process or frequently manifested in the late stage of AD. Despite the large degree of variation in prevalence, parkinsonian motor symptoms are observed in the early stage of AD in up to 13% of patients. Ample evidence has suggested that the presence of parkinsonism in AD is associated with more rapid cognitive and functional decline. Moreover, given that differentiation between AD with parkinsonism and DLB is a major diagnostic challenge, it is important to understand the neuroanatomical basis of clinical manifestation in patients with DLB.

Here, I will briefly touch clinical, biofluid, and neuroimaging markers of DLB that would be helpful in the understanding of α -synuclein-related dementia.

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