

Parkinson's Disease Dementia: Etiology, Mechanisms, Diagnosis, Management and Future Directions

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Abstract

Parkinson's disease dementia (PDD) is a common, disabling, and prognostically important neurocognitive syndrome arising in the context of established Parkinson's disease (PD). It represents one of the major late-stage manifestations of synucleinopathy and reflects the convergence of cortical Lewy body pathology, cholinergic degeneration, dopaminergic network dysfunction, Alzheimer-type co-pathology, neuroinflammation, vascular injury, and age-related vulnerability. Clinically, PDD is characterized by progressive impairment in attention, executive function, visuospatial processing, memory retrieval, and behavioural regulation, typically accompanied by neuropsychiatric symptoms such as visual hallucinations, apathy, depression, anxiety, delusions, REM sleep behaviour disorder, and fluctuating cognition. The diagnostic distinction between PDD and dementia with Lewy bodies remains anchored in the one-year rule, although biological and clinicopathological evidence increasingly supports their conceptualization as overlapping Lewy body dementias. Diagnosis remains primarily clinical, supported by neuropsychological testing, structural and functional imaging, exclusion of reversible contributors, and emerging biomarkers including α -synuclein seed amplification assays, amyloid and tau biomarkers, and neurodegeneration markers. Rivastigmine remains the best-supported symptomatic pharmacologic therapy, while management requires systematic rationalization of

dopaminergic and anticholinergic medication, treatment of neuropsychiatric complications, sleep optimization, rehabilitation, caregiver support, and advanced-care planning. Disease-modifying therapies remain investigational, but future directions include biological staging, precision phenotyping, synuclein-targeted immunotherapy, lysosomal enhancement, neuroinflammation modulation, digital biomarkers, and integrated trials across the Lewy body disease spectrum.

Keywords: Parkinson's disease dementia; Lewy body dementia; α -synuclein; cognitive impairment; rivastigmine; dementia with Lewy bodies; neurodegeneration; synucleinopathy