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Euro Dementia 2019: Parkinson's disease dementia and dementia with Lewy bodies: the same disease or two entities? - Helena Sarac, University Hospital Centre Zagreb, Croatia

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Abstract:
Dementia with Lewy bodies (DLB) and Parkinson's disease dementia (PDD) share many clinical, neurochemical and pathological features with each other as well as with Alzheimer's disease.the clinical features of DLB and PDD include cognitive impairment, parkinsonism and visual hallucinations. Their diagnosis is based on an distinction concerning the time of motor and cognitive symptoms, as early cognitive impairment in DLB and later onset following that of motor symptoms in PDD. Pathological hallmarks, cortical and subcortical α-synuclein/Lewy body plus β-amyloid and tau-pathologies are similar. Despite clinical overlap, clinical differences at onset indicate different entities and they have been considered as subtypes of an α-synuclein-associated disease spectrum (Lewy body diseases) and classified into DSM-5 as two separate entities of major neurocognitive disorders with Lewy bodies. Invivo PET and post-mortem findings revealed cortical atrophy, elevated cortical and limbic Lewy pathologies (with APOEε4), and a higher prevalence of Alzheimer pathology in DLB than PDD. While these hallmarks may account for earlier onset and greater severity of cognitive impairments in DLB, PET studies revealed no differences in cholinergic and dopaminergic deficits. Based on recent publications, including the fourth consensus report of the DLB Consortium, we prefer to view DLB and PDD as two entities or subtypes of an unified nosological continuum-α-synuclein-associated disease spectrum.

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Discussion: The clinical constellations of DLB and PDD embrace psychological feature impairment, brain disease, visual hallucinations, and unsteady attention. Intravitam PET and postmortem studies have disclosed a additional pronounced animal tissue atrophy, elevated animal tissue and structure Lewy body pathologies, higher Aβ and letter masses in cortex and striate body in DLB compared to PDD, and earlier psychological feature defects in DLB. Conversely, multitracer PET studies have shown no variations in animal tissue and striatal cholinergic and dopaminergic deficits. Clinical management of each DLB and PDD includes enzyme inhibitors and alternative medicine and non-drug methods, however with solely gentle symptomatic effects. Currently, no disease-modifying therapies are offered.

The clinical options of DLB and PDD square measure similar and embrace insanity, psychological feature fluctuations, and (visual) hallucinations within the setting of clinical or latent Parkinson's. The psychological feature domains of each disorders overlap, with progressive govt dysfunctions, visual-spatial abnormalities, and memory disorders. supported international accord, DLB is diagnosed once psychological feature impairment precedes parkinsonian motor signs or begins at intervals one year from its onset, whereas in PDD, psychological feature impairment develops within the setting of well-established Parkinson’s illness (PD). DLB patients will develop Parkinson's of skyrocketing severity over the years, though twenty fifth of them ne'er develop parkinsonian symptoms. Despite totally different temporal sequences of motor and psychological feature deficits and several other quantitative clinical variations, each disorders show mostly convergent , albeit regionally and quantitatively divergent neuropathological lesions, related to related to and letter masses in DLB. The overlap of clinical and morphological options has junction rectifier to the talk of whether or not DLB and PDD square measure constant illness, totally different phenotypical expressions of constant αSyn/Lewy body illness (LBD) spectrum, or distinc disease sharing genetic risk options with atomic number 46 and Alzheimer’s illness, despite recent studies indicating a regional overlap of pathologies, the current paper can critically review the main current findings in DLB and PDD, their potential nosologic interrelations, and therefore the on the market biological markers and therapies. Of note, this review doesn't embrace delicate psychological feature impairment in LBD.

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Conclusion: DLB and PDD ar necessary dementedness syndromes that overlap in several clinical options, genetics, neuropathology, and management. they're presently thought-about as subtypes of AN α-synuclein-associated unwellness spectrum (Lewy body diseases), from incidental Lewy body unwellness and non-demented brain disease to PDD, DLB, and DLB with Alzheimer's disease at the foremost severe finish. psychological feature impairment in these disorders is elicited not solely by psychological feature neurodegeneration however by multiple regional pathological scores. each DLB and PDD show heterogeneous pathology and neurochemistry, suggesting that they share necessary common underlying molecular pathological process with Alzheimer's disease and alternative proteinopathies. whereas we tend to opt to read DLB and PDD as extremes on a time, there remains a pressing have to be compelled to additional clearly differentiate these syndromes and to know the synucleinopathy processes resulting in either one.

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