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A Brief Note on Parkinson's Disease and Its Pathology

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DESCRIPTION

Parkinson's Disease (PD), Parkinson's Dementia (PDD), and Dementia with Lewy Bodies (DLB) are part of a group of neurodegenerative disorders known as synucleinopathies because of the aggregation of synuclein into intracellular Lewy Bodies (LBs). The presence of LBs in the brainstem is linked to dopaminergic neuron loss and motor impairment in people with Parkinson's disease. However, up to 80% of Patients Acquire Dementia (PDD) throughout the course of their condition and this development is linked to the existence of LBs in cortical regions. Progression to dementia is similarly linked with higher tau co-pathology in a manner similar to Alzheimer's Disease (AD), but with a larger temporal neocortical distribution. These findings imply that -synuclein and tau pathologies are linked in Parkinson's disease and may directly impact one other or be driven by a similar mechanism to accelerate neurodegeneration progression. Identifying characteristics that may impact illness progression might give a viable target for changing disease trajectory.

While most Parkinson's disease is idiopathic, genetic abnormalities that increase the risk of the condition have provided insights into disease pathophysiology. Leucine-Rich Repeat Kinase (LRRK2) is one of the most often altered genes in Parkinson's disease. The most common LRRK2 mutation, p.G2019S, confers a 25 to 42.5 percent risk of Parkinson's disease with a comparable age of start and illness duration as idiopathic PD indicating that it may phenocopy idiopathic PD (iPD). Indeed, as with iPD, the most prevalent hallmark of LRRK2 PD is the loss of substantia nigra neurons. While LBs are another characteristic of certain LRRK2 PD, about 21-54 percent of reported LRRK2 mutation carriers have no visible LBs indicating that another disease component is to responsible for the observed clinical illness.

The discovery of Progressive Supranuclear Palsy (PSP)-like tau inclusions in numerous cases adds further evidence to tau pathology as the neuropathological substrate of the parkinsonism found in LRRK2 mutant carriers. PSP-like tau pathology was modest in these instances, while AD-like tau was also a major neuropathological characteristic. LRRK2 mutations are exceedingly infrequent in pathologically established primary tauopathies PSP or Corticobasal Degeneration (CBD) indicating that LRRK2 mutations are predominantly linked with PD. But genetic investigations do not reveal whether LRRK2 mutations can cause tau pathology in the setting of LRRK2 Parkinson's disease. It's also unclear if the tau seen in LRRK2 mutation carriers is PSP tau or AD tau, and whether this disease is significant enough to be designated as the neuropathological substrate of dopaminergic neuron loss. In most cases, AD tau staging in LRRK2 PD follows a similar pattern to iPD and iPDD and is accompanied by widespread contemporaneous Aβ pathology. Furthermore, tau is not an independent disease factor in LRRK2 PD, but it is linked to the degree of synuclein pathology and dementia progression. Studies show that LRRK2 PD is comparable to iPD in terms of tau accumulation. Future research must determine if LRRK2 directly impacts the development of tau pathology and whether LRRK2 inhibitors alter tau pathology.

CONCLUSION

The occurrence of a hereditary disease that clinically mimics idiopathic disease can give valuable mechanistic insights regarding disease causation. The mutant protein may be implicated in the illness's molecular process and might be a therapeutic target for both familial and idiopathic sickness. Patients with and without LRRK2 mutations appear to have similar illness onset and symptoms in the case of Parkinson's disease. However, it is uncertain if LRRK2 PD and iPD have the same pathogenic substrate.