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Editorial

Clinical Pathogenicis of Alzheimer and Parkinson

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Abstract

The common neurodegenerative diseases are mostly idiopathic disturbances of unclear pathogenesis. Most common diseases are possibly expected to be induced from a complex interaction among multiple predisposing genes and other factors. In this concern, genetic mapping and gene-isolation created by the Human Genome Project helped in investigation of genes involved in the inherited forms of Alzheimer's disease (AD) and Parkinson's Disease (PD) [1]. The aggregates of Aβ42 and α-synuclein which are neurotoxic in both diseases, may explain the pathogenesis not only of the inherited forms of these diseases but also of the idiopathic variety. Such hypothesis on the cause and pathogenesis help to identify new treatment targets for these debilitating disorders. PD begins between ages 50 and 65, before onset of AD, with a few cases starting before the age of 40. With a prevalence of around 3 cases per 1,000 people, PD is less common than AD but still an important cause of neurological disorder among people [2]. As in AD, symptoms of PD result from destruction and loss of brain cells that produce dopamine, which is necessary in nerve cell communication, and in a movement-related part of the brain called the substantia nigra. The cells in this part are thought to contain abnormal accumulations of aprotein, called “Lewy bodies” [3]. Alzheimer's Disease The most common neurodegenerative disease, Alzheimer's disease forms about two thirds of total dementia cases. The disease is progressive and results in the irreversible loss of neurons, particularly in the cortex and hippocampus. The clinical hallmarks are progressive impairment in memory, judgment, decision making, orientation to physical surroundings, and language. The pathological symptoms are neuronal loss, extracellular senile plaques containing the peptide β-amyloid, the product of cleavage of a much larger protein, the β-amyloid precursor protein, and neurofibrillary tangles, consisting of a hyperphosphorylated form of the micro tubular protein tau [4]. Excluding persons with clinically questionable dementia, Alzheimer's disease has a prevalence of approximately 1 percent among those 65 to 69 years of age and increases with age to 40% to 50% among persons 95 years of age and over. Parkinson's Disease Parkinson's disease is the second most common neurodegenerative disorder, after Alzheimer's disease. It is characterized clinically by Parkinsonism (resting tremor, bradykinesia, rigidity, and postural instability) and pathologically by the loss of neurons in the substantial nigra and the presence of protein deposits in the cytoplasm of neurons (Lewy bodies) [5]. Parkinson's disease has a prevalence of approximately 0.5% to 1% among persons 65 to 69 years of age, rising to 1% to 3% among persons 80 years of age and older. Cognitive Decline Cognitive decline is a general symptom in both AD and PD diseases, though its incidence is lower in PD. As many as half of the people with PD develop cognitive disorders which may occur as a mild loss of memory to complete dementia. The dementia of PD is called “sub cortical” because of the location of affected brain areas, and these dementias have somewhat different clinical symptoms than a “cortical” dementia like AD.

In PD dementia, slowed physical activity can be accompanied by slowed thinking and by disorders in mental abilities that are more responsive to reminders than those of AD because the difficulty is with memory retrieval rather than, as in AD, with storage of new learning [6].

Behavioral Symptoms

Depression, Anxiety, Psychotic Symptoms, Sleep Disturbances, Lewy Body and Dementia.

Treatment and Outcomes

There are no treatments to slow or stop the brain cell damage caused by Parkinson's disease dementia. Current strategies focus on helping symptoms. Treatment considerations involving medications include the following issues: Cholinesterase inhibitors, Antipsychotic drugs, L-DOPA, Antidepressants, Clonazepam.

Conclusion

In conclusion, differentiating between various types of neurodegenerative diseases is necessary as it aids in deciding on best treatment suggested. Medications suitable for one of these conditions may not be suitable for a patient with another condition. Although PD-related cognitive changes could get worse over time, there is no sign that the patient will develop AD and in fact at this point there is no diagnosis of dementia.

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