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Perspective

Atypical Parkinsonism: Classification and Clinical Features

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ABOUT THE STUDY

Parkinsonism is defined as a hypokinetic syndrome characterized by restless tremors, muscle stiffness, bradykinesia or akinesia and postural instability. Although there are many secondary or acquired causes of Parkinsonism, the most common primary or neurodegenerative cause of Parkinsonism is Parkinson's Disease (PD). A small but significant number of patients suffer from Parkinson's syndrome, which is characterized by early dementia, frequent falls, ocular dysmotility, prominent dysautonomia or ataxia. These syndromes are commonly referred to as multicellular degeneration and are referred to as atypical Parkinsonian syndromes. They commonly include Progressive Supranuclear Palsy (PSP), Multiple System Atrophy (MSA), Corticobasal Degeneration (CBD), and Dementia with Lewy Bodies (DLB), as well as other rarer causes. The main difficulty with these conditions is that they can easily be confused with Parkinson's disease and there are almost no reliable diagnostic tests to tell them apart.

The most effective Parkinson's drug is levodopa, which when passes into the brain is converted to Dopamine (DA). Levodopa is combined with Carbidopa (Drug) to create the combination drug Sinemet. The Carbidopa protects from premature conversion to DA outside the brain, hence prevents nausea. These typical Parkinsonism conditions respond very poorly, Levodopa progresses more rapidly than Parkinson's disease, and the diagnosis is often clear because they have additional symptoms or signs that are not usually associated with Parkinson's disease. However, the distinction of typical conditions is also very challenging. However, it was first medically described as a neurological syndrome in 1817 when James Parkinson published a detailed medical essay on the shaking palsy. The most complete pathologic analysis of Parkinson's disease and the clear description of the brain lesions were performed in 1953 by Greenfield and Bosanquet when low dopamine presence were identified in the brains of PD patients (Parkinson, 2002).

Progressive Supranuclear Palsy (PSP) is a late-onset degenerative disease involving the gradual deterioration and death of specific volumes of the brain. The condition leads to symptoms including loss of balance, slowing of movement, difficulty moving the eyes, and cognitive impairment. PSP may be mistaken for other neurodegenerative diseases such as Parkinson's, frontotemporal dementia and Alzheimer's. The cause of the condition is uncertain, but involves accumulation of tau protein within the brain. This is 'tauopathy'-a disorder associated with abnormal aggregates. Tau proteins are the most frequent microtubule-associated proteins in the brain and are characterized as intrinsically disordered proteins. They are abundant in the neurons of the Central Nervous System (CNS) and have roles primarily in maintaining the stability of microtubules in axons. Medications such as levodopa and amantadine may be useful in some cases. PSP affects about six people per 100,000. The first symptoms typically occur at 60-70 years of age. Males are slightly more likely to be affected than females. No association has been found between PSP and any particular race, location, or occupa. Current clinical research criteria for diagnosing potential progressive supranuclear palsy emphasize a drop in symptoms within the first year of appearance, but in most cases come much later than this.

Progressive supranuclear palsy has no diagnostic tests and remains a clinical diagnosis. There is no cure for the disease. Auxiliary features in the research include MRI results such as superior cerebellar peduncle degeneration, midbrain degeneration, high signal in the midbrain, diminished or signal increase in the red nucleus, globus pallidus and lack of cardiovascular autonomic dysfunction. Adjuvant therapy, especially with regard to prevention of swallowing and falls, prolongs survival and improves quality of life. The trail of Levodopa and amantadine is valuable.

Multiple Systemic Atrophy (MSA) is a degenerative disorder characterized by Parkinsonism, and/or cerebellar signaling and autonomic failure. The name Multiple System Atrophy was coined in 1969, combining Shy-Dragger Syndrome, olivopantocerebellar atrophy, and sporadic striatonigral atrophy with a few causes. Multiple System Atrophy was then subdivided into Multiple System Atrophy-P (Parkinsonism predominant) and Multiple System Atrophy-C (cerebellar signs predominant).

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Prevalence of the disease per is about 4 per 100,000. Normal age onset is 55-60. It has never been reported to have started more than 30 years ago. All cases are sporadic; Family Multiple System Atrophy has never been documented. Women and men are equally affected. Two-thirds of patients have MSA-P; One-third has MSA-C. Both types of survival times are similar; 6-9 years on average from symptom onset. Functional impairment usually precedes MSA-P. The symptomatic feature of multiple system atrophy is the glial cytoplasmic inclusions containing the α-syncycline, mainly found in the basal ganglia, cerebellar structures, and motor cortex.

Parkinsonism with symptoms of autonomic failure, such as a postural drop in blood pressure of more than 20 mmHg systolic/10 mmHg diastolic, urine incontinence or incomplete bladder emptying, and erectile dysfunction, are all hallmarks of MSA-P. Multiple System Atrophy has a variety of distinct clinical characteristics that help differentiate it from other parkinsonian disorders. These symptoms may appear years before Parkinsonism or cerebellar indications appear. Early falls and postural instability, dystonia affecting the orofacial muscles occurring spontaneously or after starting levodopa treatment, dystonia affecting the trunk and neck leading to anterocollis and trunk flexion, and emotional incontinence: inappropriate crying/laughing are some of the 'red flags'. Dementia, hallucinations, a positive family history, and ocular paresis are some of the clinical symptoms that tend to rule out MSA.

Cortico-basal degeneration was first defined by Rebeiz and colleagues, cortico-basal degeneration in 1968 as a syndrome characterised by progressive slowness and stiffness in the limbs, dystonia, numbness or "deadness" of the afflicted limbs, and gait instability. Cortico-nigral degeneration with neuronal achromasia, cortical degeneration with enlarged chromatolytic neurons, and cortico-basal ganglionic degeneration have all been used to describe the syndrome in the past. Clinical criteria for the diagnosis of cortico-basal degeneration have mostly been developed from the research of clinical aspects of individuals referred to movement disorder clinics.

However, symptomatic cases of cortico-basal degeneration have been reported almost entirely with the appearance of a 'non-movement' disorder, usually with prominent dementia. As a result, our understanding of the clinical aspects of cortico-basal degeneration is evolving, even though the diagnostic criteria for cortico-basal degeneration remain focused on movement disorder symptoms and signs for the time being.