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PARKINSONISM: AN ACADEMIC OVERVIEW OF ETIOLOGY, CLINICAL FEATURES, AND TREATMENT

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Introduction

Parkinsonism is a clinical syndrome characterized by bradykinesia, rigidity, resting tremor, and postural instability. While Parkinson’s disease (PD) is the most frequent cause, the term “Parkinsonism” encompasses a broader group of neurological disorders that share similar motor and non-motor symptoms but arise from diverse etiologies. Accurate differentiation between Parkinson’s disease and Parkinsonism is essential, as prognosis and therapeutic responses vary significantly across subtypes.

Etiology of Parkinsonism

1. Idiopathic Parkinson’s Disease (PD)

Idiopathic PD arises from progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to striatal dopamine deficiency. Although its pathogenesis remains incompletely understood, evidence suggests that genetic predisposition, mitochondrial dysfunction, oxidative stress, and environmental exposures contribute to neuronal loss.

2. Drug-Induced Parkinsonism (DIP)

Drug-induced Parkinsonism results from medications that block dopamine receptors or reduce dopaminergic transmission. Common causative agents include:

- Typical and atypical antipsychotics

- Antiemetics such as metoclopramide

- Certain calcium-channel blockers

DIP often presents symmetrically and may improve after discontinuation of the offending medication.

3. Vascular Parkinsonism (VP)

Vascular Parkinsonism is caused by ischemic lesions involving the basal ganglia or subcortical white matter. Patients frequently exhibit lower-body Parkinsonism, gait disturbance, and poor responsiveness to dopaminergic therapy.

4. Atypical Parkinsonian Syndromes

These include:

- Multiple system atrophy (MSA)

- Progressive supranuclear palsy (PSP)

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- Corticobasal degeneration (CBD)

They typically progress rapidly and respond poorly to dopaminergic treatments.

5. Toxin- and Infection-Related Parkinsonism

Exposure to manganese, carbon monoxide, or certain pesticides may induce Parkinsonism. Infectious etiologies, such as post-encephalitic Parkinsonism, are less common today.

Clinical Manifestations

Common symptoms include:

- Bradykinesia

- Muscular rigidity

- Resting tremor

- Postural instability

Non-motor symptoms include cognitive decline, mood disorders, sleep disturbances, and autonomic dysfunction.

Diagnostic Evaluation

Diagnosis involves clinical assessment, medical history review, and neurological examination. Imaging tools include:

- MRI and CT scans

- DaTscan for dopamine transporter imaging

Response to levodopa helps differentiate idiopathic PD from atypical forms.

Treatment Approaches

Pharmacological Therapy:

- Levodopa: most effective for motor symptoms

- Dopamine agonists: pramipexole, ropinirole

- MAO-B inhibitors: selegiline, rasagiline

- Anticholinergics and amantadine for specific symptoms

Surgical Treatment:

- Deep Brain Stimulation (DBS) for advanced or refractory cases

Rehabilitation and Supportive Care:

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- Physical, occupational, and speech therapy

- Structured exercise programs such as Tai Chi and aerobic training

Prognosis

Prognosis varies by cause. Idiopathic PD progresses gradually and responds to therapy, while drug-induced Parkinsonism may be reversible. Atypical syndromes progress more rapidly and respond poorly to treatment.

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