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PARKINSONISM: CLASSIFICATION, CLINICAL FEATURES, AND TREATMENT

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Annotation: Parkinsonism is a clinical syndrome of polyetiological nature, characterized by the cardinal features of tremor, muscle rigidity, and akinesia (bradykinesia). The development of this syndrome is associated with organic or functional lesions of certain localized brain structures.
The main pathogenetic mechanism involves a deficiency of the neurotransmitter dopamine, resulting from the degeneration of dopamine-producing neurons in the nigrostriatal system.

Keywords: Parkinsonism; Parkinson's disease; Dopaminergic neurons; Tremor; Bradykinesia; Deep brain stimulation (DBS); Levodopa therapy

Introduction. Dopamine plays a crucial role in coordinating motor activity within subcortical structures of the brain.
These biochemical changes lead to relative hyperactivity of the cholinergic system and a decrease in noradrenergic and serotonergic activity. Consequently, the balance of motor control is disrupted, forming the characteristic clinical picture of parkinsonism.

Classification of Parkinsonism

In modern neurology, parkinsonism is conventionally divided into three main groups:
1. Primary (Idiopathic) Parkinsonism — includes Parkinson's disease and hereditary autosomal-recessive juvenile parkinsonism. This type accounts for approximately 70–80% of all parkinsonism cases.
2. Secondary (Symptomatic) Parkinsonism — develops as a result of cerebrovascular disorders, drug-induced effects, head trauma, toxic exposure, encephalitis, or tumor processes. This form constitutes 10–15% of all cases.
3. Parkinsonism with Multisystem Involvement ("Parkinsonism Plus" Syndromes) — characterized not only by parkinsonian features but also by additional lesions of cerebellar, pyramidal, or autonomic systems.
These include progressive supranuclear ophthalmoplegia (Steele–Richardson–Olszewski syndrome), multiple system atrophy, Shy–Drager syndrome, olivopontocerebellar and striatonigral degeneration, corticobasal degeneration, Guam syndrome, and parkinsonism in Alzheimer's disease.

Clinical Features

The clinical manifestation of parkinsonism mainly consists of four cardinal symptoms:
akinesia (bradykinesia), muscle rigidity, tremor, and postural instability.
The order and severity of these signs depend on the type of disorder and its stage of progression.

Akinesia (Bradykinesia) is the most important and persistent symptom of parkinsonism. It manifests as difficulty initiating movement, slowness of motor activity, and reduced facial expression. Patients often struggle to begin walking or change direction; speech becomes soft and monotonous, and facial expression turns mask-like (hypomimia), with infrequent blinking.
Muscle Rigidity results from increased muscle tone and is felt as stiffness or resistance to passive movement — the so-called "cogwheel" or "lead-pipe" phenomenon. Rigidity usually begins in the upper limbs and later spreads to the neck and trunk.

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Tremor commonly occurs at rest and presents as rhythmic oscillatory movements, typically of the fingers — resembling the motion of "pill-rolling" or "counting coins." Emotional stress or fatigue may intensify tremor, while voluntary movement tends to suppress it.
Postural Instability is caused by impaired balance and postural reflexes, leading to frequent falls or retropulsion. In later stages, this symptom alters the patient's gait: the patient walks with small, shuffling steps, leaning forward as if "chasing" their own center of gravity.
Additional symptoms of parkinsonism may include:

- Hypersalivation (sialorrhea),
- Dysarthria (speech impairment),
- Dysphagia (difficulty swallowing),
- Orthostatic hypotension,
- Sleep and affective disorders,
- Cognitive decline and depression.

These changes are linked to the progressive degeneration of the dopaminergic system and the worsening of biochemical imbalance.

Pathogenesis

The pathogenesis of parkinsonism is based on a degenerative process affecting dopaminergic neurons in the substantia nigra pars compacta, which project to the striatum (caudate nucleus and putamen). As a result of this degeneration, the synthesis, release, and reuptake of dopamine are severely impaired.
Microscopically, the neurons of the substantia nigra show loss of pigmentation, and the remaining cells often contain Lewy bodies — eosinophilic cytoplasmic inclusions composed mainly of α-synuclein and ubiquitin.
These inclusions are considered a morphological hallmark of Parkinson's disease and reflect a disorder of intracellular protein metabolism.
The reduction of dopaminergic activity disrupts the balance between excitatory (cholinergic) and inhibitory (dopaminergic) influences within the basal ganglia. This leads to overactivation of the indirect motor pathway, resulting in motor inhibition and the classic clinical signs of parkinsonism.
Additional biochemical disturbances may involve serotonergic, noradrenergic, and glutamatergic systems, contributing to affective and cognitive disorders commonly observed in later stages of the disease.

Diagnosis of Parkinsonism

The diagnosis of parkinsonism is primarily clinical, based on careful evaluation of motor and non-motor symptoms and neurological examination findings.
I. Anamnesis and Clinical Criteria
Diagnosis is suggested by the presence of at least two of the four cardinal symptoms:
1. Tremor at rest,
2. Bradykinesia (akinesia),
3. Muscle rigidity,
4. Postural instability.

Among these, bradykinesia is mandatory for diagnosis.
The asymmetrical onset of symptoms, good response to dopaminergic therapy, and absence of secondary causes support idiopathic Parkinson's disease.

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II. Differential Diagnosis

It is important to differentiate idiopathic Parkinson's disease from secondary parkinsonism, which may arise due to:

- Cerebrovascular pathology (multi-infarct state),
- Neuroleptic or other drug-induced parkinsonism,
- Post-encephalitic parkinsonism,
- Traumatic brain injury,
- Carbon monoxide or manganese intoxication,
- Normal pressure hydrocephalus,
- Wilson's disease,
- Progressive supranuclear palsy, or other parkinsonism-plus syndromes.

III. Instrumental Methods

- To confirm the diagnosis and exclude other causes, the following instrumental methods may be used:
- MRI or CT of the brain – to assess structural changes, vascular lesions, or atrophy.
- DAT-SPECT (dopamine transporter imaging) – to evaluate the functional integrity of the nigrostriatal pathway.
- EEG, EMG, and autonomic function tests – to identify associated abnormalities.
- Laboratory tests (such as ceruloplasmin, serum copper, or toxicological screening) are indicated when secondary parkinsonism is suspected.

Treatment of Parkinsonism

The treatment of parkinsonism is aimed at restoring dopaminergic balance, reducing motor symptoms, and improving the patient's quality of life.

Therapy is usually individualized, depending on the etiology, disease stage, and tolerance to pharmacological agents.

1. Dopaminergic Therapy

The mainstay of treatment remains Levodopa, a dopamine precursor that crosses the blood-brain barrier and is converted into dopamine in the striatum.

To prevent peripheral metabolism of levodopa and increase its central bioavailability, it is combined with decarboxylase inhibitors (such as carbidopa or benserazide) — e.g. Sinemet, Madopar.

Advantages: marked improvement of bradykinesia, rigidity, and tremor.

Disadvantages: long-term use often leads to motor fluctuations ("on-off" phenomenon) and dyskinesias (involuntary movements).

2. Dopamine Agonists

Drugs such as bromocriptine, pramipexole, ropinirole, and rotigotine directly stimulate dopamine receptors and can be used in early stages or as adjuncts to levodopa therapy.

They reduce motor fluctuations and allow lower doses of levodopa, but may cause hallucinations, hypotension, and impulse control disorders.

3. MAO-B and COMT Inhibitors

MAO-B inhibitors (selegiline, rasagiline, safinamide) slow down dopamine breakdown in the brain, thus prolonging levodopa's effect.

COMT inhibitors (entacapone, tolcapone) inhibit peripheral metabolism of levodopa, extending its duration of action.

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Combination therapy with these agents provides smoother control of symptoms and reduces "off" periods.

4. Anticholinergic Agents

Medications such as trihexyphenidyl and biperiden are particularly useful in younger patients with predominant tremor, but are less tolerated in the elderly due to side effects like dry mouth, blurred vision, and confusion.

5. Amantadine

Amantadine acts as a glutamate (NMDA) receptor antagonist, increasing dopamine release and reducing levodopa-induced dyskinesias. It also has mild anticholinergic and antiviral properties.

Non-Pharmacological Treatment

- Physiotherapy and kinesitherapy are important to maintain mobility, balance, and posture.
- Speech therapy improves articulation and communication skills.
- Occupational therapy helps patients adapt to daily life activities.
- Psychological support and social rehabilitation are essential to improve mental well-being.

Surgical Treatment

In advanced stages where medication becomes insufficient, neurosurgical interventions may be considered:

1. Deep Brain Stimulation (DBS)

This is the most effective and widely used surgical method.

Electrodes are implanted into the subthalamic nucleus or globus pallidus internus, providing high-frequency electrical stimulation that modulates abnormal neuronal activity.

DBS significantly reduces tremor, rigidity, and motor fluctuations, improving quality of life.

2. Lesioning Procedures

Techniques such as pallidotomy or thalamotomy are rarely used today but can still be effective in selected cases.

Prognosis

Parkinsonism is a chronic progressive disorder, but with appropriate and timely treatment, patients can maintain functional independence for many years.

The rate of progression depends on the etiology:

Idiopathic Parkinson's disease progresses slowly,

Secondary parkinsonism and multisystem degenerations progress more rapidly and respond poorly to therapy.

Early diagnosis, regular follow-up, and multidisciplinary management significantly improve long-term outcomes and quality of life.

References:

1. Nasriddinov A.N., Khakimov B.A. Neurology. Tashkent: 2021.
2. Rakhimov I.R., Kholmatov F.Sh. Fundamentals of Clinical Neurology. Tashkent; 2020.
3. Mamazhonov B. Basics of Diagnosis and Treatment of Nervous System Diseases. Tashkent; 2019.
4. Fedorova N.V., Illarioshkin S.N. Parkinson's Disease and Parkinsonism. Moscow: GEOTAR-Media; 2020.
5. Nasonov A.V. (Ed.) Neurology: National Guidelines. Moscow: GEOTAR-Media; 2021.

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INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 5,October ,2025

6. Gusev E.I., Konovalov A.N., Skvortsova V.I. Neurology. Moscow; 2019.
7. Ministry of Health of the Republic of Uzbekistan. Medical Protocol: Parkinson's Disease and Parkinsonism Syndromes. Tashkent; 2022.
8. Parkinson's Foundation. Available from: https://www.parkinson.org
9. National Institute of Neurological Disorders and Stroke (NINDS). Available from: https://www.ninds.nih.gov
10. World Health Organization (WHO). Neurological Disorders. Available from: https://www.who.int