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PARKINSON'S DISEASE: DIAGNOSIS AND TREATMENT

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Abstract: Parkinson's disease (PD) is a chronic progressive disorder characterized by the degeneration of nigrostriatal neurons and dysfunction of the central nervous system and basal ganglia. The disease was first described in 1817 by J. Parkinson as “shaking palsy.” Its prevalence worldwide is approximately 1%, with the average onset age being 60–65 years. In 5–10% of cases, the disease begins before the age of 40. Men are affected 1.5 times more often than women. The etiology of PD remains unclear, but age-related, genetic, and environmental factors are thought to contribute to its development. PD is primarily sporadic; however, the risk of developing the disease doubles if close relatives are affected. Genetic factors play only a minor role, accounting for approximately 10% of cases. Genetic predisposition may increase susceptibility to damaging factors affecting the nigrostriatal system and age-related processes. Environmental influences implicated in PD pathogenesis include infections, toxic exposures, metals, pesticides, well water consumption, and rural living conditions.

According to modern concepts of pathogenesis, PD arises from the degeneration of nigrostriatal neurons due to intracellular metabolic disturbances: oxidative stress, glutamate excitotoxicity, excessive calcium ion influx, increased intracellular protease activity, impaired mitochondrial respiration, neuronal deficiency, and metabolic dysregulation. These factors activate apoptosis, although the precise mechanisms of neurodegeneration remain uncertain. Pathomorphological examinations in PD reveal degeneration of nigrostriatal neurons, substantia nigra neurons, and intraneuronal inclusions resulting from Lewy body proteins.

The main neurotransmitter disturbances in PD include deficient dopamine synthesis and excessive excitatory amino acids, such as glutamate and acetylcholine, as well as altered noradrenaline and serotonin synthesis. Clinically, PD manifests due to decreased dopamine levels in the basal ganglia. Its symptoms develop gradually and asymmetrically, often starting in one limb. Core clinical features of PD include hypokinesia, resting tremor, rigidity, and postural instability. Hypokinesia presents as reduced movement activity, impaired initiation, bradykinesia, and decreased movement amplitude (decrement). Severe hypokinesia makes it difficult for patients to rise from a chair, turn in bed, and walk, often accompanied by bent elbows and flexed posture (“pleading posture”), shuffling gait, and freezing episodes.

Characteristic speech disturbances include dysphonia, bradylalia, monotony, and dysarthria. Resting tremor typically begins in the distal parts of the hands during tasks such as counting coins or rolling pills, later involving the legs. Rigidity in PD is predominantly plastic. In advanced stages, patients experience postural instability, frequent falls, and reliance on assistive devices.

Clinically, PD can present in akinetic-rigid, tremor-rigid, or mixed forms. The Hoehn and Yahr scale defines five stages of disease severity:

1. Stage 1 – unilateral parkinsonism (hemiparkinsonism);
2. Stage 2 – bilateral parkinsonism without postural impairment;

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3. Stage 3 – moderate postural instability;
4. Stage 4 – marked limitation of movement but still able to walk independently;
5. Stage 5 – patient confined to bed.

Late-stage PD often includes motor complications (motor fluctuations, drug-induced dyskinesias, gait disturbances, freezing episodes) and non-motor impairments (vegetative, cognitive, and neuropsychiatric). Motor fluctuations result from loss of nigrostriatal buffering function and non-physiological stimulation of dopamine receptors, as well as altered pharmacokinetics and pharmacodynamics of levodopa. Dyskinesias induced by levodopa appear in approximately 50% of patients after 5 years of therapy and manifest as choreoathetosis, dystonia of limbs, or oromandibular dystonia. In late stages, akinetic crises can occur, presenting with severe episodes of immobility, dysphagia, vegetative disturbances, hyperthermia, oliguria, and altered consciousness.

Clinical diagnosis of "definite" PD requires:

1. Absence of absolute exclusion criteria;
2. Presence of two or more supportive criteria;
3. Absence of "red flags."

Clinical diagnosis of "probable" PD applies when:

1. Absolute exclusion criteria are absent;
2. One or two red flags are compensated by supportive criteria.

Supportive criteria include: dramatic response to dopaminergic therapy, presence of levodopa-induced dyskinesias, resting tremor, hyposmia, or cardiac sympathetic denervation. Absolute exclusion criteria include prominent cerebellar signs, vertical gaze palsy, frontal dementia, primary progressive aphasia within 5 years, parkinsonism limited to lower limbs for over 3 years, treatment with dopamine receptor blockers, non-responsiveness to high doses of levodopa, and primary cortical sensory or motor deficits. Red flags include rapidly progressing disability, absence of motor progression over 5+ years, early bulbar symptoms, respiratory failure, severe autonomic failure within 5 years, periodic falls within 3 years, dystonic contractures, absence of characteristic non-motor features, presence of pyramidal signs, and bilateral symmetric parkinsonism.

Differentiation between primary and secondary parkinsonism is necessary, including vascular, toxic, drug-induced, post-traumatic, tumor-related, or normal-pressure hydrocephalus, as well as parkinsonism-plus syndromes in multisystem neurodegeneration.

Treatment of PD focuses on pharmacotherapy, medical-social rehabilitation, physiotherapy, and neurosurgical interventions.

Pharmacotherapy aims to halt or slow neurodegenerative processes in the nigrostriatal system (neuroprotective therapy) and restore biochemical balance (symptomatic therapy). Neuroprotective agents include MAO-B inhibitors, dopamine receptor agonists, and dopamine transport inhibitors. Symptomatic therapy aims to normalize neurotransmitter imbalance and requires continuous drug administration. Key principles involve: increasing dopamine synthesis, stimulating DA receptors, promoting presynaptic dopamine release, inhibiting dopamine reuptake, and inhibiting dopamine catabolism. Drugs used include dopamine precursors, receptor agonists, MAO-B inhibitors, COMT inhibitors, and amantadine. Choice of initial therapy depends on patient age, symptom severity, clinical form, individual drug response, comorbidities, cognitive status, and pharmacoeconomic considerations. Early-stage therapy favors monotherapy, escalating to combination therapy as efficacy diminishes.

Medical-social rehabilitation addresses high disability levels and socio-economic challenges, involving a multidisciplinary team (neurologist, physiotherapist, psychotherapist,

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social worker, nurse) and individualized programs, including therapeutic exercises, occupational therapy, and physiotherapy.

Neurosurgical treatment is indicated when pharmacotherapy efficacy declines, or motor fluctuations and dyskinesias appear. Techniques include stereotactic destructive procedures (ventrolateral thalamotomy, pallidotomy, subthalamotomy) and deep brain stimulation, targeting pathological pallido-thalamic and thalamo-cortical circuits. Neurosurgical intervention reduces movement disorders, allows lower pharmacological doses, and mitigates side effects.

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