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Parkinson's Disease. Diagnostic Criteria. Differential Diagnosis

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Abstract: Parkinson's disease (PD) is one of the most common neurodegenerative diseases. Disease diagnosis is based on generally accepted international clinical criteria. Currently, there are no research methods that confirm the diagnosis of PD. Degenerative diseases in early stages may mimic PD, however, some research methods can help recognize various forms of atypical parkinsonism. As the disease progresses, non-motor symptoms become the main sources of disability. Long-term observation and careful evaluation are the most important methods for making a correct diagnosis. The article presents the classification of parkinsonism and modern criteria for PD diagnosis. The sequence of diagnosis according to international standards is shown. Attention is paid to diseases accompanied by parkinsonism syndrome. Modern concepts of diagnostic research methods are presented.

Key words: Parkinson's disease, parkinsonism syndrome, parkinsonism classification, diagnostic criteria, diagnostic research methods.

Introduction.

The effectiveness of PD treatment depends on the timing of its detection, diagnosis, and correct therapy selection. Disease diagnosis remains clinical, based on characteristic motor manifestations. Clinical diagnosis can only be probable or possible. For a definitive diagnosis, pathomorphological examination is necessary. According to international reviews, even in specialized clinics dealing with movement disorders and having access to various modern research methods, the error rate in PD diagnosis ranges from 10% to 20%. Therefore, the PD diagnosis should be regularly reviewed, as atypical clinical signs characteristic of other neurodegenerative diseases may appear over time.

Parkinson's Disease Diagnosis. PD diagnosis is conducted in three stages. The first stage involves making a syndromic diagnosis, differentiating parkinsonism from externally similar conditions, such as essential tremor, affective disorders (apathy, depression), paratonia, hysteria, gait apraxia in cerebrovascular lesions (Alzheimer's disease, Binswanger's disease), normal pressure hydrocephalus (Hakim-Adams syndrome), hypoparathyroidism (basal ganglia calcification - Fahr syndrome). The second stage involves establishing a nosological diagnosis of parkinsonism

and differential diagnosis of PD with other diseases that manifest as parkinsonism syndrome (atypical and secondary parkinsonism). The third stage involves searching for symptoms confirming PD.

For establishing PD diagnosis, internationally recognized diagnostic criteria developed by the Brain Bank of the Parkinson's Disease Society of Great Britain are used [1], possessing high specificity (98% of cases are subsequently confirmed as PD), with sensitivity of these criteria approaching 90%, meaning that in 10% of PD cases, the diagnosis is not made when present.

Clinical diagnostic criteria for PD are:

1. Presence of bradykinesia in combination with at least one of the following symptoms:
   a) muscle rigidity;
   b) rest tremor;
   c) postural instability not related to primary visual, vestibular, cerebellar, or proprioceptive disorders.
2. Asymmetric onset
3. High effectiveness of dopaminergic agents (levodopa preparations).

Clinical criteria confirming PD diagnosis are:

1. Disease onset with unilateral manifestations.
2. Presence of rest tremor.
3. Constant asymmetry with more pronounced symptoms on the body side where the disease began.
4. Good response (70-100%) to levodopa.
5. Progressive disease course.
6. Presence of severe levodopa-induced dyskinesia.
7. Maintenance of levodopa effectiveness for 5 years or more.
8. Disease course for 10 years or more.

Criteria for excluding PD diagnosis are:

1. Acute onset and spontaneous symptom regression.
2. Rapid progression or prolonged remission.
3. Early development of postural disorders (1st-4th year of disease) and severe bulbar, autonomic, and cognitive disorders (2nd-3rd year of disease).
4. Cerebellar and pyramidal signs.
5. Anamnestic indications of repeated strokes with stepwise progression of parkinsonism symptoms, repeated traumatic brain injuries, or confirmed encephalitis.
6. Oculogyric crises.
7. Neuroleptic treatment before disease debut.
8. Strictly unilateral manifestations for more than 3 years.
9. Supranuclear gaze palsy.
10. Early appearance of severe dementia.
11. Lack of effect from high doses of levodopa.
12. Intoxication with substances causing parkinsonism (neuroleptics, manganese, etc.).

Clinical Manifestations of PD

According to modern concepts, the preclinical period of PD can last from five to twenty years. PD precursors include unexplained deterioration of smell, vivid dreams, depression, and constipation. The above symptoms may occur many years before motor manifestations of the disease. These non-motor manifestations of PD may help diagnose the disease at its premotor stage and conduct therapeutic measures in the future.

Clinical-neuroimaging comparisons show that the first symptoms of the disease appear with the death of approximately 50-70% of substantia nigra neurons, while dopamine content in the striatum decreases by more than 80%. The severity of the clinical picture is directly proportional to the number of dopaminergic neurons in the substantia nigra. The fewer neurons, the more severe the clinical manifestations of the disease, primarily the severity of bradykinesia.

Initial PD motor symptoms usually begin on one side of the body and appear on the other side only after 2-5 years of disease. Asymmetric onset of motor symptoms is one of the most reliable diagnostic factors for PD.

The main clinical manifestations of the disease are motor symptoms: hypokinesia, rest tremor, muscle rigidity, and postural disorders:

1. **The main clinical manifestation of PD, as noted above, is bradykinesia,** representing slowness of movement due to difficulty in their planning, initiation, and execution. Hypokinesia development up to

complete akinesia is characteristic. This reduces spontaneous locomotor activity, absence of synergistic limb movements when walking, difficulty initiating movement ("freezing" phenomenon), and impaired accuracy in movement execution. Gait becomes "shuffling" - step length shortens, feet do not lift off the floor when walking. Patients are characterized by propulsion phenomenon (moving by inertia, it's difficult for the patient to stop) and retropulsion (imperatively backing up). Hypokinesia combined with hypertonia leads to amimia, rare blinking, speech changes (quiet monotonous, fading), and handwriting - letter size decreases (micrographia).

For early detection of hypokinesia, the following tests can be used [2]:

- **Fournier test:** the patient is asked to perform a series of movements as quickly as possible: stand up, sit down, turn, bend, etc. Even in early stages of hypokinesia, movement slowness can be noticed when performing the test;
- **Thumb and index finger tapping test:** the patient performs table tapping with thumb and index finger alternately with both hands at maximum possible tempo and maximum amplitude. The test is especially informative in hemiparkinsonism formation - one hand lags behind in tempo and amplitude;
- **Hand clenching and unclenching test:** the patient is asked to clench and unclench the hand as quickly as possible (the hand on the side of forming hypokinesia lags behind).

2. **Tremor in parkinsonism** is observed in 75% of patients and is characterized by low frequency oscillations (4-6Hz). Trembling usually begins on one side in the hand and is more pronounced at rest ("rest" tremor). Tremor is represented by multidirectional movements of the thumb and other fingers, creating a peculiar picture of "pill rolling" or "coin counting." With disease progression, tremor captures proximal parts of upper and lower extremities and may spread to chin and lips. During active movements, parkinsonian tremor decreases. Head trembling is not characteristic of parkinsonism and occurs predominantly in essential tremor.
3. **Skeletal muscle rigidity ("plastic hypertonia")** is characterized by uniform tone increase during passive movements. The "cogwheel" phenomenon may also be detected. Due to predominance of muscle tone in flexor muscles, the patient's posture changes - stooping increases ("supplicant" posture).
4. **Postural instability,** characterized by impaired ability to maintain balance in a particular pose or when changing pose, develops with disease progression. It is caused by hypokinesia, muscle rigidity, postural tone and reflex dysfunction. The patient's body is inclined toward movement direction and may precede leg movement, accompanied by patient falls.

PD manifests clinically not only with movement disorders but also has a whole spectrum of non-motor manifestations that occur in all patients regardless of disease onset age and disease stage. Non-motor symptoms include: intellectual disorders, emotional sphere disorders, and autonomic disorders manifesting from the gastrointestinal tract (constipation, dyspepsia), cardiovascular system (hypotension, bradycardia), autonomic-trophic disorders (oily and peeling skin).

Most non-motor manifestations appear and increase with disease progression, parallel to movement disorder aggravation. However, some non-motor manifestations, such as olfactory disturbances, constipation, sleep disorders, pain syndromes, occur before classic PD motor symptoms develop. In this regard, a preclinical, premotor stage of PD is currently discussed. In late PD stages, non-motor manifestations begin to dominate as factors affecting patient quality of life, becoming at certain moments more important and disabling than motor fluctuations caused by levodopa therapy, constituting insurmountable difficulties for patients themselves and their caregivers.

PD Classification by Clinical Disease Form

1. **Mixed form (hypokinetic-rigid-tremulous)** - detected in 60-70% of PD cases.
2. **Akinetic-rigid form** detected in 15-20%.
3. **Tremulous form** detected in 5-10% of cases.

As the disease progresses, its clinical form may change.

Classification by Progression Rate

Three variants of PD progression rate are distinguished:

1. **rapid progression rate,** where disease stage change (first→second or second→third) occurs within 2 years or less;
2. **moderate progression rate,** where stage change occurs in more than 2 years but not more than 5 years;
3. **slow progression rate** with stage change in more than 5 years.

The modified Hoehn and Yahr scale evaluates PD severity on a five-point system. It is the most frequently used PD

stage classification. It was first published in 1967 in Neurology journal by Margaret Hoehn and Melvin Yahr [3]. Initially, it described 5 stages of PD progression. Subsequently, the scale was modified, supplemented with stages 0, 1.5, and 2.5.

- **Stage 0** — no signs of disease.
- **Stage 1** — symptoms manifest in one extremity.
- **Stage 1.5** — symptoms manifest in one extremity and trunk.
- **Stage 2** — bilateral manifestations without postural instability.
- **Stage 2.5** — bilateral manifestations with postural instability. Patient can overcome movement inertia caused by push.
- **Stage 3** — bilateral manifestations. Postural instability. Patient capable of self-care.
- **Stage 4** — immobility, need for outside help. Patient can walk and/or stand without support.
- **Stage 5** — patient confined to chair or bed. Severe disability.

Clinical Scales for PD Severity Assessment

Standard international scales are used to assess disease severity:

1. **UPDRS, MDS-UPDRS.** The Unified Parkinson's Disease Rating Scale (UPDRS) is used to assess the degree and dynamics of long-term PD course [4]. This scale can analyze the following indicators: thinking, behavior, mood, daily activities, movement, and treatment complications. These indicators are assessed during doctor-patient communication.
2. **Schwab and England Activities of Daily Living Scale** [5]. Assessment of impairment degree of one of the most important life activity categories - self-care, is determined according to the international Schwab and England scale. This scale reflects the degree of daily activity of a sick person, their dependence on others when performing self-care.

Differential Diagnosis

Differential diagnosis of PD and other diseases causing parkinsonism is possible in most cases without additional research methods - based on clinical data totality. Differential diagnosis is conducted between all diseases accompanied by parkinsonism syndrome: PD, secondary parkinsonism, parkinsonism within degenerative diseases ("parkinsonism plus" or atypical parkinsonism).

Juvenile parkinsonism (early-onset parkinsonism) represents a special hereditary form of primary parkinsonism with early onset, studied in detail only in the last 15-20 years [6]. The gene of this parkinsonism form was isolated in 1998, encoding a new protein "parkin" consisting of 465 amino acids. The disease is widespread and somewhat more common in women. Inheritance type in most cases is autosomal recessive, so many juvenile parkinsonism cases are sporadic. Symptom debut most often occurs at age 20-40 years, less often at earlier age.

The clinical picture of juvenile parkinsonism consists of classic parkinsonism symptoms but has several features compared to classic PD. The main ones are: absence of dementia and other mental disorders and frequent combination of parkinsonism with pyramidal symptoms. Disease course is slowly progressive, prognosis is relatively favorable.

Multiple System Atrophy (MSA)

MSA accounts for up to 12.5% of parkinsonism cases [7]. This is the most common variant of atypical parkinsonism, where the latter combines with various cerebellar, autonomic, pyramidal, and other manifestations.

MSA course variants by dominant clinical manifestation:

1. **Striatonigral Degeneration (SND).** In SND, akinetic-rigid parkinsonism phenomena predominate.
2. **Sporadic Olivopontocerebellar Atrophy (OPCA).** In OPCA, cerebellar symptoms predominate (especially ataxia and dysarthria).

Shy-Drager Syndrome. In Shy-Drager syndrome, autonomic nervous system dysfunction is observed, manifested by orthostatic hypotension, pelvic organ function disorders, impotence. Present in both disease manifestation variants.

Parkinsonism syndrome is initially indistinguishable from that in PD, but disease development shows more symmetric manifestations. Already in the first-second year of disease, cerebellar symptoms begin to be determined (gait ataxia and intention during pointing tests, nystagmus), pathological plantar signs appear: Babinski symptom develops, and obvious early autonomic insufficiency develops.

The latter manifests as urinary incontinence and imperative urges, severe constipation, hypo- or anhidrosis, impotence. A striking manifestation of autonomic insufficiency is orthostatic hypotension. Its first signs will be patient complaints of dizziness, instability, desire to sit or lie down after prolonged vertical position.

Characteristic of MSA are signs of pseudobulbar disorders: early dysarthria, dysphagia, focal dystonias (including

early severe trunk flexion forward when standing and walking - antecollis) and focal myoclonus (sudden, involuntary, repetitive flexor contractions of muscle groups, like jerking, involving body parts, more often arms or upper trunk), forced laughter or crying, respiratory disorders (inspiratory stridor), cyanosis and swelling of hands and feet.

Most patients, especially initially, respond adequately to levodopa therapy, although effect duration is short - about two years. MRI examination reveals cerebellum atrophy, brainstem atrophy, hyperintense T2 signal from putamen as striation and specific phenomenon as white cross in pons area ("hot cross bun" sign).

Conclusion

Conducting quality diagnostics is very important, as timely disease detection allows controlling its course and development using various groups of dopaminergic drugs. Based on principles and methods of modern diagnostics at preclinical and initial disease stages and differential diagnosis with parkinson-like syndromes of other nosological forms allows us, receiving necessary therapy, to maintain previous active life activity for patients for many years.

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