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Clinical Features of Parkinson's Disease in Early Stages. Diagnostic Errors of Different Forms

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Abstract: The article describes the clinical picture of Parkinson's disease (PD) with indication of disease stages according to classifications by different authors. Criteria for clinical diagnosis of PD are provided. Difficulties in making correct diagnosis are indicated. Clinical examples demonstrate cases of erroneous diagnosis, confirming the diversity of clinical symptoms of PD onset and indicating the necessity for thorough patient examination and history taking.

Key words: Parkinson's disease onset, sleep disorders, depression, imaging studies.

Introduction.

Parkinson's disease (PD) is a steadily progressive brain disease primarily associated with degeneration of dopaminergic neurons in the substantia nigra and manifested by hypokinesia, rigidity, rest tremor and postural instability, as well as a wide spectrum of non-motor disorders (mental, autonomic, sensory, etc.) [1, 2].

Already in early PD stages, patients' social adaptation significantly decreases, and their quality of life deteriorates [3]. According to some epidemiological studies, early PD stages account for 49.8% of disease cases (16.5% – stage I, 33.3% – stage II according to Hoehn-Yahr) [4].

In PD course, 5 stages are usually distinguished [5]. An interesting gradation of PD stages was developed by M.V. Stern [6] et al. in 2004-2010. They proposed the term "PD risk syndrome" (PARS), which can be subdivided into several consecutive stages:

• **Prephysiological stage** – presence of only certain genetic predisposition to PD development without any clinical signs (markers) of current neurodegenerative process;

• **Preclinical stage** – appearance of first positron emission tomography/single-photon emission computed tomography (PET/SPECT) signs of nigrostriatal deficit, still proceeding without clinical signs of neurodegenerative process;

• **Premotor stage** – appearance of various non-motor PD symptoms: autonomic dysfunction (orthostatic hypotension, urinary urgency and incontinence, impotence, constipation, hyper- or anhidrosis); sensory symptoms (olfactory

disturbance, color perception), pain; sleep disorders (REM sleep behavior disorder, restless legs syndrome, insomnia, daytime sleepiness and daytime sleep attacks, sleep fragmentation); neuropsychiatric symptoms (anhedonia, apathy, depression, psychoses, dementia, anxiety, panic attacks);

• **Prediagnostic stage** – appearance of certain "soft" neurological symptoms insufficient for PD diagnosis. Here should also be indicated subtle subclinical signs of motor control disturbance detected in such patients using specialized neurophysiological methods (visual-motor coordination tests, etc.);

• **Clinical stage** – stage of developed clinical symptoms.

Hyposmia is detected in 95-100% of PD patients; it is associated with degenerative changes in olfactory structures of the brain base and appears on average 10-13 years before motor symptom manifestation [7].

Various sleep disorders can occur long before PD motor phase manifestation [8]. They are mainly associated with reticular formation nuclei damage in the brainstem. Pathological behavior in rapid eye movement (REM) sleep occurs in 1/3 of PD cases; in 20-48% of these patients it appears 1-13 years before motor symptoms [9].

Depression is a very typical non-motor PD symptom, occurring in 27.6% of patients with initial disease stage [10]. Many observations confirm a clear association between affective disorders and PD development risk. Persons with depression are subject to 2.2-3.2 times greater risk of PD development compared to persons without affective disorders. Depression and anxiety can manifest many years (<20) before PD onset, but their frequency particularly increases 3-6 years before clinical diagnosis [7]. Depression and other affective disorders in PD are associated with involvement of several noradrenalin- and serotoninergic brain structures (locus coeruleus, raphe nuclei) and dopamine deficiency in the mesolimbic system.

Constipation, urinary disorders and erectile dysfunction can precede PD development (2-24 years, on average 18 years) [3]. Other early clinical PD markers include musculoskeletal and other pain syndromes; color vision deterioration (decreased color discrimination and contrast sensitivity) [4]; abnormalities in color visual evoked potentials recording; retinal thinning and degeneration; subtle motor disorders; impaired rapid saccadic eye movements, as well as coordinated eye and hand movements.

4-6 years before clinical PD stage onset, patients significantly more frequently visit doctors of various specialties compared to control group individuals [1].

Correct PD diagnosis made already in early stages allows adequate correction of motor and non-motor disease symptoms, improving patient quality of life and social adaptation. Thanks to adequate therapy selection, it is possible to postpone development of undesirable side effects to later periods.

However, in initial PD stages, even experienced specialists sometimes find it difficult to diagnose this pathology. Probable PD diagnosis is established based on clinical data according to brain bank criteria.

A promising diagnostic method is transcranial ultrasound brain scanning, which reveals a zone of pathological hyperechogenic signal in substantia nigra projection in 80-90% of PD patients, apparently caused by increased iron content. This sign remains stable during subsequent disease course and, consequently, does not allow tracking its progression rate.

Due to relative accessibility, the method can be used for early PD diagnosis, possibly in the prodromal disease stage, however, obtained results should be supported by clinical data. Substantia nigra hyperechogenicity on the side contralateral to motor disorders is detected in more than 90% of patients in initial PD stages.

Functional neuroimaging methods are the only additional research methods that allow antemortem confirmation of PD presence, detection of changes long before clinical disease signs appear, and assessment of nigrostriatal neuron death rate. These include PET and SPECT.

PD is characterized by decreased fluorodopa (18F) uptake by putamen neurons on the side opposite to motor symptoms. Similar changes are noted on the other side, but to a lesser extent, reflecting neurodegenerative process asymmetry [4].

PET use allowed calculation of dopaminergic neuron loss rate per year. This amount, according to different authors, ranges from 2 to 9% annually, and correspondingly calculated PD preclinical stage duration is 6±3 years.

SPECT performance with tropane-based preparations ([123I]-beta-CIT, [123I]-FP-CIT or [11C]-CFT) allows determination of dopamine transporter quantity in synaptic cleft. Radioligands bind in nigrostriatal neuron terminals with membrane dopamine transporter ensuring dopamine reuptake [2]. PD is characterized by asymmetric uptake decrease in putamen.

However, these methods are not used in daily practice as they are quite expensive.

PD diagnosis is conducted in two stages. The first stage involves making a syndromic diagnosis of parkinsonism,

differentiating it from externally similar conditions, including essential and dystonic tremor, affective disorders (apathy, depression, abulia), hysteria, gait apraxia in cerebrovascular brain lesions or hydrocephalus, hypothyroidism. The second stage involves establishing nosological diagnosis of parkinsonism and differential diagnosis of PD with other diseases causing this syndrome [9].

In some cases, diagnostic difficulties arise in early stages, which may be related to later rigidity development, detection of focal brain changes using additional research methods, more often associated with vascular parkinsonism, and less often regarded as consequences of traumatic brain injury or neuroinfection.

Sometimes hypomimia and reaction slowness are diagnosed as depression manifestations; clumsiness and stiffness in hand – as joint damage or spinal osteochondrosis manifestation. At the same time, parkinsonism overdiagnosis cases are frequent, primarily in patients with essential tremor or gait disorders due to normal pressure hydrocephalus and discirculatory encephalopathy [2].

Let us consider some situations arising in patients with early PD stages using three clinical examples.

Clinical Diagnostic Criteria for PD by the Brain Bank of Parkinson's Disease Society of Great Britain [9]

Step 1

Parkinsonism Syndrome Diagnosis

Hypokinesia combined with ≥1 of the following symptoms:

a) muscle rigidity;

b) rest tremor 4-6 Hz;

c) postural instability not related to primary visual, vestibular, cerebellar disorders, or deep sensibility impairment

Step 2

PD Exclusion Criteria

- Repeated strokes in history with stepwise parkinsonism symptom progression
- Repeated traumatic brain injuries in history
- Encephalitis in history
- Oculogyric crises
- Neuroleptic treatment at symptom manifestation time
- Familial disease character (presence of >1 relative with similar disease)
- Presence of prolonged remission
- Strictly unilateral symptoms for >3 years
- Downward gaze palsy
- Early rapidly progressive autonomic insufficiency
- Cerebellar signs
- Early developing dementia with memory, speech and praxis disorders
- Babinski sign
- Presence of cerebellar atrophy or communicating hydrocephalus on computed tomography
- Absence of reaction to high levodopa doses
- Contact with toxic substances causing parkinsonism

Step 3

Criteria Confirming PD Diagnosis

- Unilateral onset
- Progressive course
- Symptom asymmetry preservation with predominance on initially involved side
- High levodopa preparation effectiveness (symptom reduction by 70-100%)
- Severe choreiform dyskinesias induced by levodopa
- Levodopa reaction preservation for ≥5 years
- Disease course for ≥10 years

Conclusions: Thus, the indicated clinical cases confirm the diversity of clinical symptoms of PD onset, therefore establishing correct diagnosis requires thorough patient examination and history taking.

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