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CORRECTION OF VEGETATIVE DISORDERS IN PARKINSON'S DISEASE AND PARKINSONISM.

Musayeva Yulduz Alpisovna
Shamsiyeva Umida Abduvaxitovna
Melikulova Nilufar Shuxratboyevna
Erniyozov Asliddin Amiriddin O'g'li

ANNOTATION
According to data over the past decades, non-motor symptoms of Parkinson's disease (PD) have attracted special attention of scientists, among which autonomic disorders occupy an important place. According to various sources, the frequency of autonomic disorders in PD reaches 70% - 100%. The structure of autonomic disorders in PD is mainly composed of: cardiovascular disorders (20-63%), gastrointestinal disorders (20-97%), 92%), trophic skin disorders (20-34%), thermoregulation disorders (10-50%).

Keyswords: Parkinson's disease; Parkinsonism; Vegetative disorders ; Correction.

Parkinson's disease (PD) is a chronic progressive neurodegenerative disease of the central nervous system, clinically manifested by hypokinesia, rigidity, rest tremor, postural instability, and a wide range of non-motor symptoms [1].]. Vegetative symptoms (constipation) may be one of the first symptoms of PD or appear in the early stages of the disease (sympathetic cardiac denervation) [11]. The reason for the development of autonomic disorders in PD is the involvement of both the central and peripheral autonomic nervous systems in the degenerative process [12]. Autonomic disorders are one of the most common causes of a decrease in the quality of life and disability in patients with PD in the late stages of the disease [13]. About 25% of patients claim that autonomic disorders have a more adverse effect on their condition than motor fluctuations [3].

To assess autonomic disorders in PD, various instrumental diagnostic methods are used, as well as scales and questionnaires: the Scale for Outcomes in Parkinson's disease for Autonomic Symptoms (SCOPA-AUT) [14], the scale for assessing non-motor symptoms of PD (Non-Motor Symptoms Scale) [15], Quantitation of non-motor symptoms in Parkinson's disease [16], a comprehensive scale for assessing autonomic disorders in PD patients [17].

Cardiovascular disorders and STN DBS

Cardiovascular disorders in PD are represented by orthostatic hypotension, postprandial hypotension, arterial hypertension in the supine position, and impaired heart rate variability [25]. Cardiovascular symptoms are present at all stages of the disease, some of them occurring before the onset of motor symptoms [25]. Patients present primarily non-specific complaints such as transient blurred vision, nausea, lightheadedness, and dizziness. The most common cardiovascular symptom in patients with PD is orthostatic hypotension, which occurs in more than half of patients. Clinically, orthostatic

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hypotension can be identified as a 20 mm Hg drop in systolic blood pressure. or diastolic blood pressure by 10 mm Hg. during an orthostatic test. Orthostatic hypotension significantly affects the quality of life of patients, worsening the daily activity of patients. The etiology of orthostatic hypotension is multifactorial. Orthostatic hypotension in PD is considered a symptom of the primary disease itself, which is then exacerbated by additional external factors. The tendency to hypotension is characteristic in the treatment of patients with levodopa and dopamine receptor agonists. It should be noted that the hypotonic effect of these drugs is especially evident in the initial phase of therapy, and after longer treatment, blood pressure values are restored to previous levels.

Gastrointestinal disorders and STN DBS

Widespread autonomic symptoms in PD are disorders of the function of the gastrointestinal tract. Gastrointestinal disturbances in patients with PD include salivation (20–89%) [9], gastroesophageal symptoms (20–89%) [9], dysphagia (22–97%), constipation (29–79%), and anorectal dysfunction (30–66%). Deceleration of gastric and intestinal motility in PD associated with autonomic denervation significantly affects the quality of life of patients. This violates. absorption of levodopa preparations and motor fluctuations develop. One study reported that STN DBS improves gastric motility, while dopaminergic drugs do not. Cases of improvement in gastric motility function have also been described, while dopaminergic preparations do not have this effect. There have also been reports of improvements in swallowing and constipation associated with STN DBS. Constipation in PD often precedes the development of motor symptoms, they are mainly associated with the deposition of α-synuclein in the enteric nervous system, and the involvement of central autonomic structures also plays a role in its development.

Thermoregulation disorders and STN DBS

Thermoregulation disorders in PD are detected already in the early stages of the disease and are represented by a disorder in sweating and maintaining body temperature [9]. Most often, patients are disturbed by episodic and severe bouts of sweating at night. It is interesting to note that in two-thirds of cases, episodes of sweating occur simultaneously with severe akinesia, or "OFF-period".

Genitourinary disorders and STN DBS

Genitourinary disorders occupy an important place among the autonomic symptoms of PD; they are clinically manifested by an overactive bladder (47–87%) [25], obstructive urination disorders (2–3%), decreased libido, dissatisfaction with sexual life, erectile dysfunction and ejaculation in men (41–79%). The severity of the main symptoms of urination disorders in PD depends on the clinical form, severity, duration of the disease, and the rate of progression. The mechanisms by which urination disorders occur are not yet fully understood. It is assumed that these disorders arise due to the degeneration of the dopaminergic system. Because the basal ganglia and dopamine have an inhibitory effect on micturition, damage to these structures results in overactivity in the bladder detrusor muscle [11] and underregulation of the external urethral sphincter. There is also an opinion that urinary disorders arise

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due to a violation of prefrontal inhibition. It is important to note that all forms of urination disorders occur in PD, although detrusor overactivity is most often observed. Currently, there is an opinion that levodopa at the beginning of PD therapy has a negative effect on bladder function, but then with long-term therapy it has a positive effect. Bladder dysfunction, in particular urinary incontinence, is a poor prognostic sign in PD, because this increases the risk of falls in PD patients, leading to fractures. STN DBS may affect urinary symptoms through D1-GABAergic receptors in the subthalamic nucleus [21]. Several studies have found a positive effect of STN DBS on the symptoms of urinary disorders in the form of a decrease in the effect of STN DBS on the symptoms of urinary disorders in the form of a decrease in the severity of nocturia. Despite this, 3 years after the operation, in 20% of cases, the symptoms of urinary disorders continued to progress. Research papers have shown a positive effect of STN DBS on detrusor hyperreflexia.

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