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## **APPROACH TO THE TREATMENT OF COGNITIVE IMPAIRMENT IN PATIENTS WITH PARKINSON'S DISEASE**

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Parkinson's disease (PD) is one of the most frequent neurological pathologies of the elderly. According to epidemiologic data, its prevalence among people over 65 years of age reaches 2%. The classic signs of Parkinson's disease are motor disorders in the form of slow movements (hypokinesia), muscle rigidity and resting tremor [1,2, 5].

Cognitive impairment often comes to the forefront in the clinical picture of Parkinson's disease, among non-motor manifestations, and becomes the main cause of maladaptation of patients [2,4,6-8,11,14]. According to current research, dementia occurs in 20-40% of patients with Parkinson's disease [1,6-8,11,14]. The risk of severe cognitive impairment increases with significant disease duration.

Cognitive impairment is a serious social problem, affecting the quality of life of patients and their relatives, as well as being one of the causes of disability in patients with Parkinson's disease. In most cases of PD, cognitive impairment is mild to moderate, but in elderly patients it can reach dementia severity, having a negative impact on quality of life. Cognitive impairment in PD includes: decreased concentration, difficulty in planning activities during the day, difficulty in maintaining a long conversation, solving complex problems, slow thought formation, difficulty remembering events and their details [4-8,11,14]. In the initial stages of PD, cognitive disorders are more common, associated with slowing of mental processes, decreased attention, and limited ability to memorize. The patients' thinking and intellect remain relatively intact, but the ability to solve complex tasks may decrease. These disorders are characterized by decreased attention and speed of psychomotor reactions and are defined as mild cognitive impairment (MiCI)[3,4, 6]. As PD progresses, cognitive deficit may increase and turn into moderate cognitive impairment (MoCI) and dementia, which is facilitated by older age of patients, male gender, gross motor deficit and impaired regulatory, visual-spatial functions according to neuropsychological testing. The presence of concomitant pathology (diabetes mellitus, arterial hypertension, hypercholesterolemia, etc.) aggravates the course of PD and gradually leads to the development of dementia [1,2,5-8].

In extended neuropsychological testing, cognitive impairment in this disease is

detected in 90-95% of cases [1,8,12,13]. At late stages of the disease, 24-31% of PD patients have cognitive impairment severity reaching the level of dementia, whereas in the initial age group of the general population the proportion of persons with dementia is almost 10 times lower (3-4%). According to some researchers [1,2,7], dementia develops in 17% of patients with PD onset before 70 years of age, whereas in patients developing the disease after 70 years of age, dementia develops in 83% of cases [6-8].

Recently, the issue of the influence of glutamate on cognitive functions in PD has been discussed. The imbalance in the dopamine-glutamate system leads to the predominant excitatory effect of the latter on brain neurons. Glutamate activates NMDA-receptors regulating the influx of calcium ions inside the cell. The massive entry and increased calcium concentration in nigrostriary dopaminergic neurons results in the triggering of oxidative stress. High sensitivity and glutamatergic hyperstimulation of NMDA receptors, as well as cytotoxic concentration of calcium ions, triggering a cascade of excitotoxicity, accelerate the degenerative process of neurons and lead to disruption of signaling pathways involved in memory formation [1,2,9,10].

Amantadine has the ability to block NMDA(N-methyl-D-aspartate) type glutamate receptors, thereby slowing down the degenerative process of neurons and is of great interest for the treatment of PD. Amantadine is believed to have a positive effect on hypokinesia and rigidity, to a lesser extent on tremor. In the early stages of PD, amantadine can be used as monotherapy. A favorable response to therapy is noted in approximately 80% of patients, with overall improvement in 20-40%. Positive in the early administration of amantadine is its neuroprotective effect, which probably slows the progression of the disease. It reduces the severity and shortens the duration of the off-period (off-periods) and results in reduction of peak dose dyskinesias in a greater proportion of patients. Marked clinical improvement in advanced stages of PD. Amantadine 300 mg administration reduces the severity of dyskinesias in 60-70% of patients [9,10]. These changes are directly related to the stimulation of dopamine synthesis, reduction of dopamine reuptake, increase of dopamine receptor sensitivity to it, mild anticholinergic action of amantadine sulfate [1,2,9].

There were 32 PD patients with cognitive impairment under our observation, among whom there were 21 men and 11 women aged from 68 to 76 years. The duration of the disease was on average 5-8 years. The diagnosis of PD was established in accordance with the international clinical diagnostic criteria of the Brain Bank of the Parkinson's Disease Society of Great Britain [Hughes A.J. et al., 1992].

The severity of PD was determined using the Unified Parkinson's Disease Rating Scale (UPDRS) Part I to assess non-motor manifestations of the disease in daily life [1,2] - part I to assess non-motor manifestations of the disease in daily life [1,2]. Disease stage was assessed using the Hoehn and Yahr Hoehn and Yahr scale (Hoehn, Yahr, 1967) as modified by Lindvall (1989), Tetrad, Langston (1989) from 2.5 to 3.5 stages. [1,2]. The akinetic-rigid form was most common in 23 people, and the mixed form in 9 people.

MoCI was analyzed using the criteria proposed by J. Touchon and R. Petersen in 1997 and modified in 2004. The neuropsychological study included the use of the Mini-Mental State Examination (MMSE) scale [12], the results of which had the following values: MiCI corresponds to 28-27 points; MoCI -26-24 points;

initial stage of dementia-23-20 points and more than 20 points - dementia. The Frontal Assessment Battery (FAB) test for the assessment of frontal dysfunction was used, according to the results of which cognitive impairment was interpreted as follows: MiCI corresponds to 16-18 points; MoCI -12-15 points; dementia-11 points and below [13]. We also used the Clock Drawing Test (CDT), Schulte's test, which allowed us to identify work capacity (workability, exhaustion), the presence or absence of attention deficit disorder [13].

All patients (26 patients) diagnosed with MiCI (14 patients) and MoCI (12 patients), in addition to the main pathogenetic treatment with levodopa and dopamine receptor agonists, were treated with amantadine sulfate (PK-Merz, Germany) in tablet form at a dose of 300 mg daily.

The duration of follow-up was 3 months; clinical and neuropsychological examinations were performed at the first visit to diagnose cognitive impairment and 3 months after the start of amantadine sulfate treatment.

The severity of motor disorders and the degree of severity of the disease were more pronounced in patients with MCI, compared to those with LCI. Interestingly, the akinetic-rigid form of PD was more frequent in patients with MoCI (12 people - 46.2%).

After a course of treatment with amantadine sulfate (PK-Merz) at a dose of 300 mg in 3 months, 21 patients (80.8%) showed improvement in motor activity and reduction in the severity of symptoms of parkinsonism and UPDRS scale score.

Positive dynamics of neuropsychological status in 3 months from the beginning of treatment with amantadine sulfate was observed in PD patients with MoCI, and in 10 people (38.5%) against the background of treatment MoCI decreased to MiCI, which, apparently, was also associated with a positive effect on motor activity of patients.

The dynamics of neuropsychological status in patients with MoCI in PD treated with amantadine sulfate showed that according to the MMSE scale indicators, the indices of constructive praxis, counting, memory and speech improved (increased from  $24\pm 5$  points to  $27\pm 5$  points). There was a significant increase in the total score on the battery of tests to assess frontal dysfunction (from  $13.7\pm 2.1$  to  $17\pm 2.3$  points), as well as increased performance and mental stability to perform tasks on the Schulte tables.

Thus, the use of amantadine sulfate (PK-Merz) in complex treatment at different stages of PD had a positive effect on motor functions in patients, and also reduced the severity of levodopa-induced dyskinesias. Neuroprotective capabilities of amantadine sulfate associated with suppression of glutamate excitotoxicity led to improvement of cognitive function indices in patients with MiCI and MoCI. This makes it possible to improve the quality of life, reduce the risk and rate of dementia development in patients with Parkinson's disease and use this drug both in monotherapy at early stages and in complex pathogenetic treatment at late stages of Parkinson's disease.

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