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Neuropsychiatric Disorders in Parkinson's Disease

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ABSTRACT: This article discusses neuropsychiatric disorders in Parkinson's disease. We have demonstrated high efficiency in relation to fatigue in PD ADR Mirapex. So, if before therapy a pronounced degree of fatigue according to the corresponding scale for assessing fatigue was noted in 64% of patients, then after treatment - only in 26%. It is important that no significant correlation was found between the dynamics of indicators of fatigue and motor functions, depression, sleep disorders during therapy, which indirectly indicates independent genesis of this symptom and specificity the Mirapex effect.

KEYWORDS: neuropsychic disorder, sensory disorder, autonomic disorder, depression, dementia, patients, fatigue, significant correlation, dynamics of indicators, independent genesis.

INTRODUCTION

The traditional concept of Parkinson's disease (PD) as a disease predominantly of the motor sphere, which is based on an isolated lesion of the dopaminergic system, has now been drastically corrected. PD is considered as a multisystem neurodegenerative disease with damage to various mediator systems and a wide range of both motor and non-motor (neuropsychic, sensory, autonomic) disorders.

Neuropsychic (emotional, cognitive, psychotic, behavioral) disorders, as well as pathogenetically close to these symptoms, have a significant negative impact on the quality of life of patients and their loved sleep disturbances and fatigue. Depression, dementia, psychotic symptoms are predictors of a poor quality of life in patients, regardless of the severity of the motor symptoms of the disease. However, these disorders are often not diagnosed, which deprives patients of adequate therapy. The diagnosis of a number of neuropsychiatric disorders

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is complicated by their clinical similarity (overlap) with some motor and non-motor manifestations of PD, as well as frequent comorbidity.

THE MAIN FINDINGS AND RESULTS

Neuropsychiatric disorders accompany all stages of PD; their frequency generally increases with the severity and duration of the disease. Some of the neuropsychiatric disorders appear or worsen with the development of fluctuations in symptoms. In this case, some disorders are manifested with limited motor activity of patients, others - with optimal motor health or when the phases of "on" – "off" are changed.

In some cases, neuropsychiatric disorders precede the manifestation of movement disorders by 5-10 years or more, manifesting themselves at the "pre-motor" stage of the disease. Depression and rapid eye movement (REM) sleep behavior disorders - symptoms (along with hyposmia and constipation), the connection of the appearance of which with the subsequent development of PD is the most convincing. Depression occurs in 20% of patients with PD even before the diagnosis is made. Thus, the presence of depression in middle-aged and elderly people is considered as a factor that increases the risk of PD by 2–3 times. This syndrome is characterized by motor activity in the REM phase due to the absence of atony, which is physiological for this stage of sleep. Behavioral disorders are manifested by vocalization, limb movements during sleep, associated with dreams. Neuroimaging studies of patients with an isolated syndrome of sleep behavior disorder with REM showed a small but significant decrease in the density of striatal neurons, which confirms the pathogenetic relationship of the syndrome with PD. Possible "pre-motor" neuropsychic symptoms, the relationship of which with the further development of PD is being clarified, include anxiety, daytime sleepiness, fatigue, apathy. The emergence of neuropsychic symptoms in the "pre-motor" phase is explained by the popular concept of H. Braak et al., According to which one of the first targets of the degenerative process in PD is the nuclei of the lower part of the brain stem, which manifests itself, in particular, by depression, anxiety, sleep and wakefulness disorders.

The pathophysiology of most neuropsychiatric symptoms of PD is multifactorial. The pathomorphological basis for the appearance of a wide range of symptoms is the spread of the degenerative process (Lewy bodies) as the disease progresses to many structures of the brain with a violation of their functional interaction. A significant role in the pathophysiology of a number of neuropsychic symptoms (depression, anxiety, insomnia, cognitive disorders) is

Published: October 15, 2021 | Pages: 176-180

played by degeneration of the brain stem nuclei with the development of dysfunction of neurotransmitter systems (noradrenergic, serotonergic, acetylcholinergic, glutamatergic) and impaired functioning of the brainstem and cortical brains. There is more and more evidence of the involvement of the dopaminergic system in the formation of most neuropsychiatric disorders. It is assumed that a number of neuropsychiatric disorders (depression, apathy, fatigue, behavioral disturbances) are caused by dysfunction of the brain areas responsible for motivationally determined behavior. These cerebral "reward centers" include dopaminergic projections that connect the ventral tectum with the mesolimbic and mesocortical regions of the brain. A key structure that ensures the functional interaction of these parts of the brain and the modulation of targeted behavior is considered the nucleus accumbens. Dopaminergic dysfunction in the hypothalamus in PD patients, revealed in a recent neuroimaging study, is probably a significant component of the pathogenesis of sleep disorders and daytime sleepiness. Requires further clarification the possibility of linking hypothalamic dysfunction with manifestations of depression and fatigue. Premorbid psychological personality traits, motor and other non-motor symptoms of PD, undesirable effects of antiparkinsonian drugs can serve as additional factors contributing to the manifestation of certain neuropsychiatric disorders.

Parasomnias in PD are manifested by disturbances in sleep behavior with REM, vivid dreams (nightmares), and nighttime hallucinations. Sleep behavioral disorders with REM occur in about 15-30% of patients with PD; they serve as a risk factor for the progression of cognitive disorders, joining psychotic disorders. With a dysadaptive nature of motor activity during sleep, correction of pharmacotherapy may be required. All antidepressants have a negative effect on the course of the syndrome, therefore, if possible, drugs of this class should be discontinued. Clonazepam is considered the most effective treatment for the syndrome, and the positive effects of gabapentin and melatonin have also been reported.

Hypersomnia in PD is a heterogeneous syndrome with multifactorial pathogenesis. The main manifestations of hypersomnia are excessive daytime sleepiness and sudden short periods of sleep during the day (sleep attacks). Excessive daytime sleepiness (IDS) occurs in 15-50%, and sudden falling asleep occurs in 4-8% of patients with PD. The incidence of IDS in PD exceeds the incidence of this disorder in the age population by about 2 times, which indirectly indicates a connection between the syndrome and PD. In 40% of patients with daytime sleepiness, a

Published: October 15, 2021 | Pages: 176-180

phenotype similar to narcolepsy is noted with a reduction in the time to fall asleep (less than 5 minutes) and the onset of sleep from the REM stage.

It is assumed that against the background of the progression of the disease and the intake of dopaminergic drugs, dopamine regulation disorders occur in the ventral striatum and structures of the mesolimbic system with the formation of their hypersensitivity. Probably, violations of the control over the intake of drugs and ITP are realized against the background of a decrease in the activity of the prefrontal frontal cortex. To correct SDS, first of all, a revision of antiparkinsonian therapy is required - a decrease in the dose of levodopa in compliance with the schedule of taking the drug. With INP, cancellation or reduction of the dose of ADR that caused the disorder is effective. Transfer to another ADR in a lower equivalent dose is possible. In a number of cases, the additional appointment of atypical antipsychotics is effective. It is advisable to conduct cognitive-behavioral psychotherapy.

CONCLUSION

Thus, neuropsychiatric disorders occur in the majority of PD patients; they are significant, and in some cases the leading factors that reduce the quality of life. The similarity of individual clinical manifestations of a number of neuropsychic disorders and their frequent comorbidity require specialists from a subtle differentiated approach in the diagnosis of these disorders. Timely identification of these symptoms provides an opportunity for adequate therapeutic correction of a wide range of manifestations of PD.

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