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Research Article

MODERN METHODS FOR PREDICTING COGNITIVE DISORDERS IN PARKINSON'S DISEASE

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ABSTRACT

In Parkinson's disease (PD), among the clinical and symptomatic manifestations of the disease in recent years, attention has been increasingly paid to the so-called non-motor symptoms, that is, cognitive disorders. In PD, cognitive disorders can manifest themselves in the form of a decrease in executive, visual, and visuospatial activity and memory impairment from mild to severe dementia. Cognitive impairments of a subjective and objective nature are often found at the level of early and mild manifestations. In such situations, it is important to develop a strategy for predicting the progression of cognitive disorders and developing methods for their prevention. Long-term and multicenter studies conducted over the past 30-40 years have shown that after the diagnosis of PD, dementia develops after ten courses of the underlying disease. This, in turn, aggravates the already complex course of PD with a pronounced decrease in the quality of life of patients.

KEYWORDS

Parkinson's disease, cognitive disorders, dementia.

INTRODUCTION

In the literature, there is no consensus on the chronology of the development of cognitive disorders; that is, there is no information about the specific timing, pathology profile, and rate of cognitive ability loss in Parkinson's disease (PD). In this regard, we believe that the relevance of research in this area of unsolved problems of neurology is determined by the usefulness of both scientists and doctors of practical health care.

To date, all diagnostic and prognostic signs of the development of dementia in PD can be divided into two large groups: clinical signs and biological markers. At the same time, such signs as the patient's age and

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the presence of non-tremor symptoms of PD are considered more or less carefully studied.

The International Parkinson's Society and the Society of Movement Disorders have identified a consensus on the division of cognitive disorder syndrome into mild impairment and dementia [1, 2]. Dementia in PD was defined as a severe impairment of the patient's daily life in the form of a decrease in social and functional activity, requiring the intervention of a second person to provide care. Moreover, such changes in the patient occur regardless of motor or vegetative disorders.

METHODS

In order to solve the problem of identifying the features of etiological and pathogenetic factors in the development of cognitive disorders in PD, we analyzed the literature sources from the PubMed and CrossRef catalogues. The keywords "Parkinson's disease", "cognitive disorders", "aetiology of cognitive disorders", and "pathogenetic factors of cognitive disorders" were included. In total, information was obtained from 1241 scientific sources, of which 56 were of a review nature. Clinical examples were not considered.

RESULTS AND DISCUSSION

According to P. Svenningsson et al. [3], among the examined patients without PD dementia, up to 30% of cases of cognitive impairment have a mild form, which can be clinically determined only in 10-20% of patients. In general, studies have shown that dementia can develop in a short time after a diagnosis of mild cognitive impairment. However, along with this, there is information that the timing of the development of dementia does not have an approved chronology. Moreover, there is evidence of regression of cognitive disorders. For example, a scientific article by K.F. Pedersen et al. [4] presents the results of observation of patients with cognitive disorders in the early stage of PD, who, after 12 months of treatment, achieved regression of cognitive impairment in 20% of cases. However, as the author of the article himself points out, all of them initially had a mild form of cognitive disorders in PD.

Various authors ambiguously assessed the importance of assessing the duration of PD in predicting the development of dementia. Although most authors are inclined to a borderline period of high probability of cognitive decline in PD as 10 years, more recent literature data provides information regarding the earlier period of possible development of not only cognitive disorders but also dementia.

Thus, C.H. Williams-Gray et al. [5, 6] conducted a long-term study dividing the results into two stages. In both cases, they found an increase in the incidence of dementia in PD at baseline for mild cognitive impairment. In particular, in the first term of the study (five), cognitive properties decreased to the level of dementia in 20% of patients with PD, and after the second term (ten) - in 46%. At the same time, in the studies of G. Santangelo et al. [7], after a four-year follow-up period, loss of cognitive properties in PD was noted in only 5% of cases. In comparison, in the observations of K. Pigott et al. [8], This figure reached 50% over six years.

Investigating the clinical manifestations of cognitive disorders in PD, C.H. Williams-Gray et al. [9] proved that cortical posterior cognitive deficits in memory and speech disorders manifest cognitive disorders in PD. A.A. Kehagia et al. [10] share a similar opinion, believing such a conclusion leads to the hypothesis of a "double syndrome" of cognitive abilities in PD, given the absence of frontal dysfunction.

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We believe that such contradictory data testify, on the one hand, to the lack of unification of all factors for the development of cognitive disorders in PD (the choice of the variant of the course of the disease, chronology from the appearance of the first signs, dependence on the onset of PD, the criteria used inherent in cognitive disorders or for dementia, the frequency of research, etc.), and the assessment of only the clinical manifestations of the disease, on the other hand.

An attempt to unify the clinical manifestation of cognitive disorders was made by a group of scientists at the University of Canterbury in 2016 [11], their studies divided patients with PD into four groups: with normal cognitive functions, subjectively reduced cognitive functions, with mild cognitive impairment and dementia. Such a division of patients was due to the continuity of the PD process. The clinical tests affected the criteria of the same cognitive domain, which turned out to be a more reliable prognostic criterion for the progression of dementia in PD.

Studying the risk factors for the development of cognitive disorders in PD, D.H. Ffytche et al. [12] concluded that visual hallucinations can also be harbingers of dementia. At the same time, it is necessary to clearly distinguish between the appearance of visual hallucinations and dementia, which in many studies may reflect both the progression of cognitive impairment and the onset of hallucinations against the background of the above-mentioned dysfunctions.

As a confirmation of this judgment, we can cite the data of a long-term study by a group of gerontological psychiatrists from Norway, who determined the prognostic risk of developing cognitive dysfunctions 8 years after the onset of visual hallucinations [13]. Neurologists at McGill University (Montreal, Canada) [14] conducted a similar study and set a prognostic period of 4 years.

Thus, as can be seen from the information presented, clinical studies conducted to identify prognostic criteria for developing cognitive disorders in PD have shown multidirectional results and do not have a single interpretation system. It should also be noted that predicting the development of cognitive disorders in PD by assessing clinical signs requires a long time to conduct research and monitor patients, which creates difficulties in fully implementing the entire assessment program. In this regard, in recent years, more and more scientists have been paying attention to assessing the significance of predicting cognitive disorders in PD using biomarkers, which by their nature are more committed to predictive assessment.

Given that pathomorphological disorders of the brain play a leading role in the multifactorial pathogenesis of PD, cerebrospinal fluid is one of the leading areas in the study of biomarkers. For this purpose, the following biological markers of amyloid-β pathology (Aβ42), markers of neurodegeneration and phosphorylation (t-tau and p-tau), α-synuclein as a direct marker of the development of PD were studied in the cerebrospinal fluid.

Given that the prognostic value of cerebrospinal fluid in predicting cognitive impairment in PD remains unknown, D.C. Backstrom et al. [15] conducted a study to assess the diagnostic and prognostic value of several biomarkers of this biological environment in patients with an early form of PD. A regional, population-based, prospective cohort study was conducted on 128 patients without cognitive impairment with PD. In PD, high levels of light chain neurofilament protein, low levels of β-amyloid, and high levels of heart fatty acid-binding protein have

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been linked to the development of cognitive impairment.

Swedish scientists from Lund University led by S. Hall [16] examined the cerebrospinal fluid of 42 patients with PD for the level of changes in α-synuclein, β-amyloid-42, tau, phosphorylated tau, and neurofilament light. The researchers found evidence of an association between higher levels of α-synuclein at baseline and worsening motor symptoms and cognitive speed over 2 years in PD. Elevated levels of α-synuclein may be a marker of more intense synaptic degeneration in PD. The results indicate that cortical amyloid pathology (low levels of cerebrospinal fluid Aβ-42) is associated with memory decline.

Similar results, with slight discrepancies, were obtained by other researchers in different years of the study. So, in 2014, G. Alves et al. [17] published the results of a study of 104 patients with PD who were followed for 5 years, and the results obtained indicated that low levels of β-amyloid predict early dementia. After another 2 years, M.Jr. Terrelonge et al. [18] conducted a study in 341 patients and proved that low levels of amyloid β predict cognitive impairment at follow-up. Similar results were obtained by other researchers, confirming the reliability of the method of studying cerebrospinal fluid to determine biomarkers for the development of cognitive disorders in PD.

Similar results were also obtained regarding the role of total α-synuclein and its modified forms, including phosphorylated, nitrate, and other oligomeric forms, in cerebrospinal fluid. All this indicates the promise of the studies carried out as markers of the progression of cognitive disorders in PD, but further research is needed to identify the pathogenetic causes and conditions for developing these changes.

Advances in radiological and chemical imaging technologies made it possible, at the turn of the 21st century, to make a breakthrough in understanding changes in structural, functional, and molecular changes in the brain in PD. Studies such as magnetic resonance imaging (MRI) and positron emission tomography (PET) scans of the brain have significantly advanced the understanding of the complex mechanisms that underlie the development of cognitive disorders in PD.

In 2016, the COPPADIS research group, led by D. Santos-García [19], presented the developed protocol for the concept of PD as a global disease with motor and non-motor symptoms, issues of the level of quality of life of patients and their care, biomarkers and their prognostic role in the progression of the disease. Eight hundred patients with PD from various scientific and medical centres in Spain were examined; one of the leading methods was an MRI of the brain. It turned out that the MRI imaging method occupies one of the leading places in the ability to predict the development of cognitive disorders in Parkinson's disease.

Meanwhile, a few years earlier, British scientists from the Imperial Medical College under the leadership of M. Politis et al. [20] PET of the brain concluded that this method of research using translocator protein radioligands makes it possible to monitor the progression and severity of neuroinflammation in neurodegenerative diseases, including Parkinson's disease, in vivo.

According to K. Wu et al. [21], A common functional polymorphism (Val(158) Met) in the catechol-O-methyltransferase (COMT) gene is associated with changes in executive functions when performing tasks that have a frontostriatal basis. According to the authors, this is due to changes in dopamine levels in the cerebral cortex since catechol-O-methyltransferase is

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the primary way to inactivate dopamine in the frontal regions. To prove this assumption, the researchers investigated in vivo changes in presynaptic dopamine accumulation in patients with PD depending on the polymorphism of the Val(158)Met catechol-O-methyltransferase using positron emission tomography (18)F-DOPA. The results showed that Met homozygotes have higher presynaptic levels of dopamine in the frontal regions than Val homozygotes, which may help explain how this genotypic variation may affect frontostriatal cognitive deficits in Parkinson's disease.

In 2015, independent studies conducted by D. Yildiz et al. [22], E. Mak et al. [23], as well as J.B. Pereira et al. [24] came to a consensus that patients with PD and mild cognitive impairment have a pattern of loss of cortical volume in the posterior, parietal, and frontal cortex, as well as atrophy in the hippocampus, which correlates with memory deficits. It is these signs that can directly act as predictors of cognitive disorders in Parkinson's disease.

According to A. Hanganu et al. [25], longitudinal assessment of cerebral cortex thickness and subcortical volumes in patients with PD and mild cognitive impairment may be manifested by progression of cortical thinning in the temporal, occipital, parietal, and frontal cortex and further loss of hippocampal volume, which is associated with cognitive decline.

According to the results of the study conducted by B.Borroni et al. [26], a cortical thinning scheme has been proposed to differentiate dementia in Parkinson's disease, which is characterized by predominant thinning of the frontal cortex, from Lewy body dementia, which is characterized by predominant thinning of the parietal and occipital cortex.

Neurodegenerative disorders are characterized by damage to the white matter tracts (structural cohesion), increasing mean diffusion, and decreased fractional anisotropy. In patients with PD without a formal diagnosis of Parkinson's disease, mild cognitive impairment or dementia leads to an increase in mean diffusion in the hippocampus, as well as in the frontal and parietal tracts of white matter. All of this is associated with impaired performance in terms of verbal and visuospatial memory, semantic fluency, and other executive functions. In addition, a decrease in fractional anisotropy of the hippocampus correlates with indicators of global cognitive decline.

Parkinson's patients with mild cognitive impairment or dementia have a progressive loss of functional connectivity at rest over 3 years in many brain regions, especially in the posterior parts of the brain, which has been correlated with cognitive decline.

Other studies have demonstrated that cognitive decline in dementia caused by PD is associated with impaired functional connectivity between the corticostriatum and frontal cortex.

The literature analysis showed a large amount of scientific literature on studies related to neurovisual methods for predicting cognitive disorders in Parkinson's disease. Among them, the most studied, along with those described above, are methods for assessing perfusion imaging of the brain, dopaminergic molecular imaging, PET imaging of glucose metabolism, complex versions of PET studies with assessment of metabolic processes in the brain, cholinergic molecular imaging of PET, PET imaging of amyloid-β, PET imaging of tau-cortical aggregates.

Thus, methods for predicting cognitive disorders in PD using neuroimaging deserve close attention. In the near future, research in this direction, using molecular

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and mitochondrial targets, can improve the understanding of the mechanisms underlying cognitive impairment in Parkinson's disease and accelerate the development of new methods for their prevention and treatment.

The search for reliable methods for predicting the development of dementia in PD has led scientists to assess the significance of changes in electroencephalography (EEG) and the degree of manifestation of symptoms of cognitive disorders in this disease.

For example, in the studies of S. Kamei et al. [27], It has been proven that in PD, there is a natural direct relationship between EEG slowing down and the severity of cognitive deficits. Moreover, along with the presence of a link between EEG changes and executive dysfunction, a group of Japanese scientists from Nihon University found an association between the level of scores on the Mini-Mental State Survey (MMSE) scale and the spectral ratio of fast and slow activity [28].

American neurologists from the Mayo Clinic found an increase in the delta power of the EEG in Parkinson's disease patients with deficits in attention, executive function, and memory [29], and R. Zimmermann et al. [30] revealed a connection between such cognitive impairment and a decrease in the median EEG rate.

It was also proven that there is a connection between changes in the background pattern of the EEG and a slowdown in the answers to a mini-exam on the mental state and tests for drawing a clock. Along with this, studies by L.C. Fonseca et al. [31] revealed an increase in low-frequency, i.e., delta and theta spectral power of the EEG, which was different in PD dementia and Alzheimer's disease dementia.

This indicates the need for close attention to such research methods since studies assessing long-term results may show a prognostic value of EEG in Parkinson's disease. However, such studies are still not reflected in the literature.

Attempts to unify the predictors of dementia in PD have led to the popularization of combined studies of various biomarkers. However, the complexity of such studies comes down to certain difficulties in interpreting the data obtained in patients with reduced cognitive abilities in Parkinson's disease since, in most cases, they are heterogeneous and sometimes may not even be associated with brain pathology. However, this research approach can provide additional information and improve the accuracy of predicting cognitive decline in Parkinson's disease.

Attempts to conduct such studies were made using Alzheimer's disease as an example. Certain interrelated changes in cerebrospinal fluid and blood markers were identified, corresponding to different variants of the neuroimaging picture of the brain.

In the studies of Y. Compta et al. [32], A link has been found between cerebrospinal fluid markers and decreased grey matter volume associated with several neuropsychological functions. The authors interpreted this by saying that markers of Alzheimer's disease pathology in cerebrospinal fluid are associated with measures of brain atrophy in Parkinson 's-associated dementia and in cognitive impairment and deserve further attention as putative biomarkers of cognitive impairment and dementia in Parkinson's disease.

The revealed low level of β-amyloid in the cerebrospinal fluid, according to Y. Compta et al. [33], was associated with cortical thinning, and the combination of biomarker pathologies was a stronger

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predictor of cognitive decline and dementia than the pathology of a single biomarker.

Also, low levels of α-synuclein in the cerebrospinal fluid have been associated with thinning of the frontal cortex in Parkinson's patients without dementia. At the same time, in patients with dementia in Parkinson's disease, cortical atrophy was associated with an increase in the total amount of cerebrospinal fluid α-synuclein and t-tau.

In the study, neuroimaging activity in brain motor networks correlated with total cerebrospinal fluid α-synuclein, indicating its ability to develop resting state motor networks in Parkinson's disease. In patients with PD without dementia, cerebrospinal fluid α-synuclein was associated with the dorsal attention network, suggesting that α-synuclein is also involved in cognitive function. No associations have been found between functional magnetic resonance imaging (fMRI) at rest and cerebrospinal fluid β-amyloid.

CONCLUSION

All of the above-combined studies support the hypothesis that cognitive-related volume loss in PD is associated with three key pathologies: α-synuclein, tau, and amyloid. This idea is consistent with morphological studies demonstrating that amyloid, tau, and α-synuclein pathologies contribute to neuronal loss and cognitive decline in the relatively early stages of the disease and can be used as predictors of dementia in Parkinson's.

Conflict of interest – none

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