Streszczenie w języku angielskim

Introduction:

Parkinsonian syndrome occurs primarily in Parkinson's disease (PD). It is less commonly observed in the group of atypical parkinsonisms (APS), which includes progressive supranuclear palsy (PSP), corticobasal syndrome (CBS), and multiple system atrophy (MSA). The phenotypic overlap between these conditions complicates accurate diagnosis. The definitive diagnosis can only be established by postmortem neuropathological examination.

In vivo, only a probable diagnosis of APS can be made, which carries a relatively high risk of misclassification. The aim of this publication series is to present the diagnostic challenges of APS and additional tools that may be potentially useful in differentiating these diseases, as well as to analyze factors potentially related to their pathophysiology.

Methodology:

To conduct the review, a detailed literature search was performed using medical sources available in PubMed. Selected studies were evaluated and analyzed in the context of potential asymmetry in clinical and imaging studies observed in atypical parkinsonism. In a publication comparing two tauopathies - PSP and CBS, participants were divided into three groups: those diagnosed with PSP, CBS, and a control group with Parkinson's disease (PD). Perfusion of selected brain structures was assessed using single-photon emission computed tomography (SPECT), followed by the collection of peripheral blood samples from each participant. Based on the obtained parameters, peripheral inflammatory factors were calculated, which may be associated with neurodegeneration. In the second study investigating the two most common PSP subtypes—progressive supranuclear palsy-Richardson syndrome (PSP-RS) and progressive supranuclear palsy-parkinsonism predominant (PSP-P)—an analysis was conducted using inflammatory markers and neuropsychological assessments. Cognitive function was evaluated in each participant using the Montreal Cognitive Assessment (MoCA) and the Frontal Assessment Battery (FAB). Similarly to the previous study, serum samples were analyzed to determine peripheral inflammatory factor levels. Additionally, the analysis was expanded to include interleukin concentrations measured in both serum and cerebrospinal fluid (CSF).

Results:

In the study assessing the relationship between brain perfusion in selected regions and peripheral inflammatory markers, a linear negative correlation was observed between neutrophil-to-high-density lipoprotein ratio (NHR) and bilateral perfusion of the insulae

and thalami in patients diagnosed with CBS. In the study analyzing cognitive function impairments in relation to peripheral inflammatory markers, a negative correlation was found between MoCA scores and platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR) levels. Additionally, NLR also showed a negative correlation with FAB test results. Furthermore, a positive association was identified between interleukin-1β (IL-1β) levels in serum and cognitive function as measured by MoCA. No correlation was found between neuropsychological parameters and other inflammatory peripheral parameters evaluated in serum or the interleukins analyzed in CSF.

Conclusion:

The series highlights the relevance of assessing peripheral inflammatory markers as part of routine examinations in patients with suspected APS. Despite relatively low sensitivity and specificity, the increase in these markers might have a negative impact on cognitive function in these disorders. However, the data suggest that interleukin 1β may be potentially associated with a protective effect on cognitive function. Nevertheless, due to the lack of correlation between MoCA scores and this parameter in cerebrospinal fluid, further analysis based on a larger patient cohort is recommended. Based on the review analysis, significant differences in the presence of symmetry in both clinical and imaging assessments were identified across specific types of APS.

Summary:

The presented publications highlight the potential usefulness of assessing inflammatory markers alongside neuropsychological evaluation and symmetry in clinical manifestation and imaging as an additional diagnostic approach. This series emphasizes the need for further studies on larger patient cohorts, including broader panels of inflammatory parameters in order to enhance understanding of disease mechanisms and identify potential therapeutic targets in APS in the future.

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Date / Data:
2025-02-10 23:01

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