the effect of aerobic exercise in migraine patients. The study was conducted in Pakistan [4], comprising 28 migraine outpatients aged between 20-50 years. Patients of experimental group had received supervised exercises including aerobic exercise followed by progressive muscle relaxation along with prophylactic medicine while the control group only received prophylactic treatment. Treatment was carried out for 6 weeks three times a week. Overall results depict that experimental group had better outcome post-intervention arriving to a conclusion that prophylactic medicine, aerobic exercises and progressive muscle relaxation used together have a depressing effect on migraine [4].

Moreover, in a community-based study of 480 medical students, revealed significantly lower migraine associated disability in who practiced regularly exercise compared to those who did no exercise. Physical exercise included both aerobic and strength training [5]. In a later randomized, controlled, clinical trial in Denmark evaluating the effect of aerobic exercise involving cross-training, biking and brisk-walking on 26 persons with migraine and co-existing tension-type headache and neck pain, it was revealed that exercise caused a reduced incidence of migraine and improved ability to engage in physical activity. Moreover, migraine frequency, pain intensity and duration were also reduced [5].

Conclusions

Based on the evidences it justifies that exercise regimens can be a valuable tool in the therapy of migraines because of pronounced efficacy, minimized side effects, innumerable health benefits and affordability. Thus, it can be concluded that physical exercises can be prescribed as a non medicative method of treating migraine. Additionally, headache specialists and general practitioners encouraged to incorporate physical exercises as a part of their patients' treatment strategy.

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УДК 616.892.32-08-037

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COMPARATIVE ANALYSIS OF THE ROLE OF NOVEL BIOMARKERS IN PARKINSON'S DEMENTIA AND LEWY BODY DEMENTIA

Introduction

Dementia is a broad term used to describe a range of symptoms associated with cognitive decline and memory loss that interfere with daily functioning. It is a progressive condition that affects thinking, behavior, and the ability to perform everyday tasks [1]. Parkinson's dementia, a subtype of dementia, occurs in individuals with Parkinson's disease, a neurodegenerative disorder characterized by motor symptoms like tremors and stiffness. Parkinson's dementia is

marked by cognitive impairment that develops as the disease progresses [2]. On the other hand, Lewy body dementia is a distinct type of dementia characterized by the presence of abnormal protein deposits called Lewy bodies in the brain. Individuals with Lewy body dementia experience cognitive decline, visual hallucinations, and fluctuations in alertness and attention, in addition to motor symptoms similar to Parkinson's disease [3]. Biomarkers play a crucial role in identifying specific molecular changes associated with these neurodegenerative disorders [1]. This study seeks to compare and contrast the relationship between biomarkers and Parkinson's dementia and Lewy body dementia by exploring the differences and similarities in the biomarker profiles such as Alpha-Synuclein, Tau and Amyloid Beta, Biomarkers of neuroinflammation and Neurodegeneration markers and assess the utility of these biomarkers in clinical practice.

Goal

To compare and evaluate novel biomarkers in Parkinson's dementia and Lewy body dementia through a systematic review.

Material and methods of research

The analysis and generalization of scientific literature on this topic from PubMed, National library of medicine and other scientific articles were done. The search terms were "Parkinson's dementia", "Lewy body dementia", "biomarkers", "neurodegenerative disorders", "alpha-synuclein", "tau protein", "genetic markers".

Results of the research and their discussion

In recent research, several novel biomarkers have been identified in Parkinson's dementia and Lewy body dementia. These biomarkers include alpha-synuclein species, Tau and Amyloid Beta, Biomarkers of neuro inflammation (cytokines, microglial activation markers) and neurodegenerative markers. These biomarkers play a crucial role in the development and progression of Parkinson's dementia and Lewy body dementia by providing valuable insights into the underlying pathophysiological mechanisms, aiding in early diagnosis, monitoring disease progression, and potentially guiding targeted treatment strategies.

Alpha-synuclein is a key protein implicated in both Parkinson's dementia and Lewy body dementia, sharing similarities in terms of its pathological role in these neurodegenerative disorders [1]. In both conditions, abnormal aggregation of alpha-synuclein forms insoluble clumps known as Lewy bodies, which are characteristic pathological features observed in the brains of affected individuals. These aggregates contribute to neuronal dysfunction and cell death, leading to cognitive decline and motor symptoms associated with Parkinson's dementia and Lewy body dementia [2]. However, there are also notable differences in the distribution and presentation of alpha-synuclein pathology between Parkinson's dementia and Lewy body dementia. In Parkinson's dementia, alpha-synuclein pathology primarily affects the substantia nigra region of the brain, leading to motor symptoms such as tremors, rigidity, and bradykinesia [1]. On the other hand, in Lewy body dementia, alpha-synuclein aggregates are more widespread throughout the brain, including regions involved in cognitive function, resulting in a combination of motor and cognitive impairments [2]. Furthermore, the timing of alpha-synuclein pathology in relation to the onset of symptoms differs between Parkinson's dementia and Lewy body dementia. In Parkinson's disease, motor symptoms typically manifest first, followed by cognitive decline later in the disease course, whereas in Lewy body dementia, cognitive impairment may occur early on, alongside or even preceding motor symptoms [1, 2].

In Parkinson's dementia and Lewy body dementia, the roles of tau and amyloid beta proteins differ from those seen in Alzheimer's disease. While Alzheimer's disease is characterized by the accumulation of amyloid plaques and tau tangles in the brain, the involvement of these proteins in Parkinson's dementia and Lewy body dementia is less prominent and differs between

the two conditions [2].In Parkinson's dementia, tau pathology is typically less pronounced compared to Alzheimer's disease. However, some individuals with Parkinson's disease may develop tau pathology in the form of neurofibrillary tangles, especially in later stages of the disease. These tau tangles are associated with cognitive impairment and dementia in Parkinson's disease [1]. On the other hand, Lewy body dementia is characterized by the presence of alpha-synuclein aggregates in the form of Lewy bodies, as previously discussed. While amyloid beta plaques are not a primary feature of Lewy body dementia, some individuals with this condition may show co-existing Alzheimer's pathology, including amyloid plaques and tau tangles. This overlap in pathology can complicate the clinical presentation and diagnosis of Lewy body dementia [3].

Neuroinflammation, characterized by the activation of microglia and the release of inflammatory cytokines, plays a significant role in the pathogenesis of both Parkinson's dementia and Lewy body dementia [1]. While there are similarities in the underlying mechanisms of neuroinflammation in these conditions, there are also differences in the specific biomarkers and patterns of inflammation observed. In both Parkinson's dementia and Lewy body dementia, activated microglia are found in the brain regions affected by alpha-synuclein pathology, such as the substantia nigra and cortical areas. These activated microglia release pro-inflammatory cytokines, such as interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), contributing to neuroinflammation and neuronal damage [2]. However, differences in the extent and distribution of neuroinflammation markers may exist between Parkinson's dementia and Lewy body dementia. For example, some studies suggest that microglial activation and neuroinflammation may be more widespread and severe in Lewy body dementia compared to Parkinson's dementia, reflecting the broader distribution of alpha-synuclein pathology in the former condition. Additionally, differences in the levels of specific cytokines and chemokines may be observed between the two disorders, potentially reflecting distinct inflammatory profiles associated with each condition [1, 2].

Neurodegenerative markers, such as neurofilament light chain (NFL) and markers of neuronal damage are important indicators of neuronal injury and degeneration in various neurodegenerative disorders, including Parkinson's dementia and Lewy body dementia. These markers can provide valuable insights into the extent of Neurodegeneration and disease progression in affected individuals [2]. In both Parkinson's dementia and Lewy body dementia, elevated levels of neurofilament light chain have been reported in cerebrospinal fluid (CSF) and blood samples. NFL is a structural protein found in neurons and is released into the CSF and bloodstream following neuronal damage or degeneration. Increased NFL levels are indicative of axonal injury and neuronal loss, reflecting the ongoing neurodegenerative processes in these conditions [2]. Markers of neuronal damage, such as total tau protein and phosphorylated tau (p-tau), may also be elevated in the CSF of individuals with Parkinson's dementia and Lewy body dementia. Tau is a microtubule-associated protein found in neurons, and its abnormal accumulation is associated with neuronal injury and degeneration. Elevated levels of total tau and p-tau in the CSF indicate tau pathology and ongoing Neurodegeneration in affected individuals [3]. While similarities exist in the elevation of neurodegenerative markers such as NFL and tau proteins in both Parkinson's dementia and Lewy body dementia, differences may also be observed in the specific patterns and levels of these markers between the two conditions. For example, some studies suggest that individuals with Lewy body dementia may exhibit higher levels of neurodegenerative markers compared to those with Parkinson's dementia, potentially reflecting the more widespread neuronal damage and pathology seen in Lewy body dementia [1, 3].

Biomarkers like alpha-synuclein, tau, amyloid beta, neuroinflammation markers, and neurodegenerative markers are crucial for diagnosing and treating Parkinson's dementia and Lewy body dementia. They provide objective measures of underlying pathology, aiding in early de-

tection, differentiation from other disorders, and monitoring disease progression. Elevated levels of alpha-synuclein and tau indicate neuronal dysfunction, while neuroinflammation markers reflect disease processes. These biomarkers help clinicians make informed decisions and customize treatments. They also offer insights into disease mechanisms and treatment responses. By utilizing these biomarkers, healthcare professionals can enhance diagnostic accuracy, personalize treatments, and improve outcomes for individuals with these conditions.

Conclusions

The identification and characterization of novel biomarkers in Parkinson's dementia and Lewy body dementia have significantly advanced the understanding of the underlying pathophysiological mechanisms of these neurodegenerative disorders. Biomarkers such as alpha-synuclein species, tau, amyloid beta, neuroinflammation markers, and neurodegenerative markers play crucial roles in the development, progression, diagnosis, and treatment of Parkinson's dementia and Lewy body dementia. These biomarkers provide valuable insights into the specific pathological processes driving neuronal dysfunction, cognitive decline, and motor symptoms in these conditions. By utilizing these information provided by these biomarkers, healthcare providers can enhance diagnostic accuracy, personalize treatment strategies, and ultimately improve outcomes for individuals affected by Parkinson's dementia and Lewy body dementia. But the study found persistent gaps in knowledge regarding biomarkers in Parkinson's dementia and Lewy body dementia, conflicting findings across various articles, and it is necessary to do further future researches in these areas.

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УДК 579.61:616.34]:616.8

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THE ROLE OF GUT MICROBIOME IN NEUROLOGICAL DISEASES

Introduction

The gut microbiome significantly influences the diseases of human body by altering energy balance and lipid synthesis, leading to enhanced energy storage and systemic inflammation. Emerging evidence increasingly supports the gut microbiome's profound impact on neurological health, suggesting that the balance of gut bacteria can influence brain function and contribute to the pathogenesis of neurological diseases. This connection, often referred to as the gut-brain axis, has been implicated in conditions ranging from neurodevelopmental disorders to neurodegenerative diseases, highlighting the potential of microbiome-targeted therapies [1, 2].

Goal

The primary objective of this review is to explore and elucidate the mechanisms through which the gut microbiome exerts its influence on neurological diseases, thereby contributing to a deeper understanding of the gut-brain axis and its potential therapeutic targets.