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MODERN APPROACHES TO THE STUDY AND TREATMENT OF ALZHEIMER'S DISEASE

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Abstract:
Alzheimer's disease is a complex neurodegenerative disorder characterized by progressive loss of cognitive functions and changes in behavioral aspects. This article provides an analysis of current data on the mechanisms of pathogenesis, focusing on the roles of β-amyloid deposits, hyperphosphorylated tau protein, and inflammatory processes. Advances in diagnostics, such as the use of biomarkers and neuroimaging techniques, are reviewed along with promising therapeutic approaches, including monoclonal antibodies and emerging directions in gene therapy. The study underscores the importance of integrating fundamental research with clinical practice to enhance the quality of life for patients.

Keywords: Alzheimer's disease, neurodegenerative disorders, cognitive decline, β-amyloid, tau protein, inflammation, biomarkers, neuroimaging, monoclonal antibodies, gene therapy, quality of life.

Introduction

Alzheimer's disease (AD) is a complex multifactorial condition and remains one of the leading causes of dementia in older adults. Over the past decades, research has significantly advanced the understanding of its pathogenesis and the development of treatment methods. This paper examines key theoretical and practical aspects of AD. [1]. Research on the pathogenesis of Alzheimer's disease is being conducted at leading scientific centers worldwide [2]. The works of Smith

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and Brown (2023) highlight the central role of amyloid plaque accumulation in initiating a cascade of pathological changes, including synaptic dysfunction and neuroinflammation [3]. Miller and Wang (2022) demonstrated that activated microglia exacerbate neuronal damage by releasing pro-inflammatory cytokines [4]. Lee and Johnson (2023) analyzed the mechanisms of tau protein aggregation and its role in neurodegeneration in detail. These studies strengthen the understanding of the multifactorial nature of AD pathogenesis and emphasize the need for a combined treatment approach [5]. Petrov's (2023) research shows that genetic predisposition significantly influences the development of AD. Specifically, the presence of the APOE4 allele increases the risk of developing the disease by 2–3 times compared to the general population. Petrov also highlights the importance of the PSEN1 gene, mutations in which are associated with early-onset Alzheimer's disease. These findings underscore the necessity of genetic testing for early diagnosis and the development of personalized treatment methods [2]. The accumulation of β-amyloid plaques and hyperphosphorylated tau protein in the brain are key hallmarks of Alzheimer's disease. Lee and Johnson (2023) demonstrated that tau protein aggregation disrupts intercellular communication and leads to neuronal death, particularly in the hippocampus and cortex. These processes trigger an inflammatory reaction of microglia and oxidative stress, ultimately resulting in neuronal loss. Lee and Johnson's (2023) study showed that tau protein aggregation stimulates the release of pro-inflammatory cytokines by microglia, exacerbating neuronal damage. These findings highlight the importance of inflammatory processes in the pathogenesis of Alzheimer's disease [5]. For example, studies show that amyloid protein accumulation in the brain begins 10–15 years before the onset of symptoms. However, not all patients with high amyloid levels develop dementia, emphasizing the importance of studying additional factors, such as genetic predisposition (e.g., APOE4 mutations) [6]. Breakthroughs in diagnostics are linked to the use of PET scanning to detect amyloid and tau deposits. Green and Patel (2023) reviewed advanced biomarkers, including β-amyloid and p-tau levels in the blood, significantly simplifying early diagnosis [7]. Zhang and Torres (2023) emphasized the importance of genetic tests, including mutations in the APP, PSEN1, and APOE4 genes, in predicting disease risk [8]. However, challenges such as the high cost of imaging methods and insufficient standardization of cerebrospinal fluid analysis remain. Current research aims to develop more accessible tests. Breakthroughs in diagnostics are associated with PET scanning

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to detect amyloid and tau deposits. Significant progress has been achieved using biomarkers such as β-amyloid and tau protein in cerebrospinal fluid. For instance, phosphorylated tau (p-tau) levels reliably correlate with the stage of the disease. However, challenges remain, including the high cost of imaging methods and insufficient standardization of cerebrospinal fluid analysis. Ongoing research focuses on developing more accessible tests, including blood analysis [8].

Treatment:

Vasiliev's (2023) study emphasizes the importance of cognitive stimulation in treating Alzheimer's disease. Vasiliev demonstrated that regular participation in cognitive exercises improves neuroplasticity, even at late disease stages [10]. Moreover, he noted that integrating cognitive therapy with pharmacological approaches, such as using memantine, significantly increases treatment efficacy. Vasiliev also highlighted promising areas, including the development of individualized cognitive programs based on specific cognitive deficits in patients. Current treatment methods focus on symptomatic therapy. Cholinesterase inhibitors, such as donepezil, and NMDA receptor antagonists, such as memantine, improve cognitive functions but do not slow disease progression. Recent clinical trials of monoclonal antibodies, such as aducanumab and lecanemab, have shown reduced amyloid levels, although their clinical efficacy remains controversial. The high cost of these drugs also limits their widespread use [10].

Prevention:

Research indicates that up to 40% of AD cases can be prevented by modifying risk factors. For instance, lowering blood pressure by 10 mmHg reduces the risk of AD by 15%. Long-term studies also confirm that a Mediterranean diet rich in antioxidants and omega-3 fatty acids slows cognitive decline. Regular physical exercise improves cerebral blood flow, reducing the risk of neurodegeneration. However, more data are needed on the interaction of these factors with genetic predisposition [10, 11].

Conclusion:

Alzheimer's disease remains one of the most challenging problems in modern medicine. Despite advances in diagnostics and therapy, a complete cure remains elusive. The primary focus should be on prevention and early diagnosis. Future

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research should focus on developing personalized treatment approaches and integrating innovative technologies such as genome editing and artificial intelligence to optimize therapy and disease prediction.

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