COMPLEXITY OF ALZHEIMER'S DISEASE: INTRODUCTION AND ITS PATHOPHYSIOLOGY

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Resume: Alzheimer's disease (AD), which is typified by gradual cognitive loss and neurodegeneration, continues to be a major global health concern. In the pathophysiology of AD, tau neurofibrillary tangles and amyloid-beta (Aβ) plaques gradually build up in the brain as a result of the complex interaction of hereditary, environmental, and lifestyle variables. Furthermore, lifestyle therapies have demonstrated promise in lowering the risk of dementia and cognitive decline. These include dietary changes, cognitive stimulation, and physical exercise. To enable early intervention and improve patient care, an accurate and prompt diagnosis of AD is crucial. Recent developments in biomarker research, such as neuroimaging methods and cerebrospinal fluid (CSF) markers, allow for more accurate illness progression tracking and diagnostic classification. highlights the complex nature of AD and new developments in the field.

Keywords: Alzheimer's disease, Amyloid-βplaques, diagnostic techniques, neurofibrillary tau tangles**,** therapeutic modalities.

Introduction

A neurological disorder that deteriorates over time, Alzheimer's disease (AD) is characterized by cognitive decline, memory loss, and functional impairment [1]. In elderly people, it is the most common cause of dementia, accounting for 60–70% of cases worldwide. Since Dr. Alois Alzheimer initially brought up Alzheimer's disease (AD) in 1906, it has grown to be a significant global health issue that impacts millions of individuals and their families [2]. Approximately 35.6 million cases of dementia were reported worldwide in 2010; according to a 2013 World Health Organization (WHO) assessment, this number is expected to quadruple by 2050 [3]. As people age, the prevalence of dementia increases to 25% to 50%, and the incidence affects between 5% to 8% of people over 65. The aggregation of specific proteins in the brain is responsible for the development of structures called "plaques" and "tangles." The defining markers of AD are these microscopic characteristics, which include neurotic plaques that contain amyloid beta peptide (Aβ42) and neurofibrillary tangles (NFTs) made of hyperphosphorylated tau [7].

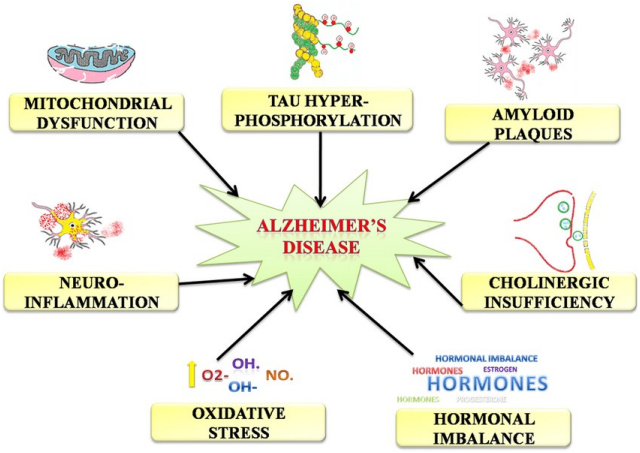
These proteins help break down the connections between nerve cells, which eventually results in the death of neurons and the loss of brain tissue [8]. The highest estimated prevalence rate of neurodegenerative diseases worldwide in 2019 was 62% for Alzheimer's disease, followed by 17% for Parkinson's disease, 10% for mixed dementia, 4% for Lewy body dementia, 2% for frontotemporal dementia, 2% for vascular dementia, and 3% for other neurodegenerative diseases.

Although the precise mechanisms behind AD are still unclear, a number of important processes have been linked to the disease's etiology [10]. Neuronal dysfunction, synapse degradation, and ultimately neurodegeneration are the results of these processes, which include a complex interaction of genetic, molecular, and cellular abnormalities [11].

Genetic predispositions like mutations in the APP gene or genes controlling Aβ metabolism (e.g., presenilin mutations) are among the many causes that lead to Aβ accumulation in the brain [16]. Furthermore, because Aβ clearance mechanisms become less effective with age, allowing Aβ to accumulate, becoming older is a major risk factor. Furthermore, Aβ accumulation is made worse by the brain's inability to remove Aβ, which is mediated via the lymphatic system and other clearance mechanisms [17]. Aβ accumulation can be increased by impairing these processes through disruptions in the blood-brain barrier or malfunctions in microglial cells, the immune cells in the brain that are in charge of clearing Aβ.

Aβ can also cause tau protein hyperphosphorylation, which is another characteristic of AD pathogenesis and results in the development of neurofibrillary tangles inside neurons. In AD, tau pathology and Aβ interact to promote neurodegeneration and cognitive decline in a synergistic manner (Table 1.1) [28].

Aspects of the pathogenesis of Alzheimer's disease, including hereditary variables, diagnostic biomarkers, treatment approaches, and future directions.



Materials and method of research

Scientists are investigating new approaches to treat AD, such as using green nanoparticles as drug delivery vehicles. These are some cutting-edge methods that are being research

A possible method of delivering medicinal substances to the brain is through green nanoparticles, which are made from natural sources such as plant extracts or biopolymers. In order to address AD pathology, these nanoparticles can contain medications like neuroprotective or anti-amyloid medicines and deliver them across the blood-brain barrier (BBB). The efficacy and safety of AD therapy may be improved by green nanoparticles through increased drug stability, extended circulation time, and precise targeting.

Results

Some substances that come from plants, such resveratrol from grapes and curcumin from turmeric, have neuroprotective properties that may lessen AD-related neuronal damage and cognitive loss. These chemicals' bioavailability and brain penetration can be improved by encapsulating them in green nanoparticles, which increases their potential as AD treatments. Green nanoparticles can also be designed to release medications in response to particular stimuli, which would maximize drug delivery to the areas of the brain that are impacted.

Their biocompatibility, biodegradability, and environmental friendliness make green nanoparticles superior to their synthetic counterparts. These qualities lower the possibility of negative consequences and environmental damage, making them desirable for drug administration. A greener approach to AD treatment is provided by green nanoparticles, which use natural ingredients and sustainable production techniques.

By engineering green nanoparticles to transport several therapeutic drugs at once, combination therapy techniques for AD are made possible. The ability of these nanoparticles to encapsulate medications with complimentary modes of action, such as antioxidants and anti-amyloid compounds, allows them to work in concert to target several pathogenic pathways linked to the progression of AD. This strategy could improve therapy effectiveness and address AD's complex character.

In conclusion

The main risk factor that contributes to the beginning of AD is age. The accumulation of Aβ plaques and neurofibrillary tangles, which result from the aggregation of hyperphosphorylated beta-protein, is the main cause of AD development. Cognitive dysfunction and impairment are directly linked to these pathological characteristics. As our understanding of AD develops, it becomes more and more evident that a thorough approach is essential to understanding its intricacies and developing effective treatment strategies. Researchers can find new

avenues for prevention and intervention by looking at how lifestyle, environmental, and genetic factors combine to cause AD onset. This chapter also highlights the urgent need for interdisciplinary collaboration and creative research approaches to address the challenges posed by AD.

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