Journal of Dementia

Liu, J Dement 2023, 7:3

Review Article Open Access

A Short Note on Management of Alzheimer's Dementia

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Abstract

A common form of dementia, especially among the elderly, is Alzheimer's disease. Neurodegeneration and progressive cognitive decline are its hallmarks. The exact cause of Alzheimer's disease is still unknown, despite numerous studies. Several theories have been proposed, including tau protein hyper-phosphorylation and A amyloid deposition in the brain. This survey article investigates the possible pathogenesis of Alzheimer's illness, zeroing in on the impacts of disturbances in the degrees of vitamin B12, folate, and homocysteine, as well as the effect of oral microorganisms causing periodontitis and insulin opposition, and their relationship to Alzheimer's. High homocysteine levels and low folate and vitamin B12 levels have been linked to an increased risk of Alzheimer's disease, according to research. The article also looks into the connection between oral bacteria and Alzheimer's disease, specifically dental infections and periodontitis, which contribute to the inflammatory processes in Alzheimer's patients' nervous systems.

Keywords: Alzheimer's disease; Neurodegeneration; Homocysteine; Neuropathologist; Hyper-phosphorylation

Introduction

Alzheimer's disease may represent a form of type 2 diabetes mellitus known as type 3 diabetes that is associated with the brain because there may be a disruption in insulin signaling that further disrupts glucose metabolism in the brain. The predictive characteristics of Alzheimer's disease can be identified using neuroimaging techniques like MRI, PET, and tau PET, with amyloid PET being the most useful for excluding the disease. The article concludes by emphasizing the significance of comprehending genetic and neuroimaging factors in Alzheimer's disease diagnosis and treatment [1].

A broad term used to describe a person's cognitive decline is dementia. Alzheimer's disease is thought to be the leading cause of dementia in older people. It is a neurodegenerative disease that gets worse with age and makes life difficult. It has been more than 100 years since German psychiatrist and neuropathologist Alois Alzheimer first described Alzheimer's disease in 1906. Clinically, it is characterized by progressive cognitive impairment, and pathologically, it is established by the presence of neurofibrillary tangles and senile plaques caused by amyloid accumulation [2].

Method

The ideal biomarkers for diagnosis and effective treatment for managing the disease are still unavailable, despite the fact that it has been regarded as the most prevalent form of dementia. As a result, it is essential to develop diagnostic procedures that are simple, cost-effective, and easy to use for the early detection of disease and the introduction of appropriate treatments that can reduce its severity. Numerous studies have been conducted to determine the pathogenesis of Alzheimer's disease, and a number of theories, such as tau protein hyper-phosphorylation and A amyloid deposition in the brain, have been proposed [3]. However, the precise etiology that lies behind the mechanisms of pathogenesis remains a mystery, and extensive research is still being conducted on these topics.

A common neurological condition known as Alzheimer's disease (AD) is characterized by a gradual decline in memory, language, emotion, and cognition. It mostly influences older individuals. The FDA has approved pharmaceutical medications and anticholinesterases as a form of AD therapy because of the effects of AD. However, it became increasingly clear that these medications did not address the underlying factors that contribute to AD pathogenesis; rather, they focused on the symptoms to improve the cognitive outcomes of the patients. Subsequently [4], a chase after prevalent infection changing choices is sent off. Planning new helpful specialists requires a careful comprehension of the neuroprotective cycles and shifted capabilities completed by specific qualities, and antibodies. The history of Alzheimer's disease, the significance of the blood-brain barrier in determining the scope of treatment options, and the advantages and disadvantages of the current therapeutic treatment options for stem cell therapy, immunotherapy, regenerative therapy, as well as improved Alzheimer's disease care and diagnosis are all discussed in detail in this comprehensive review article. Additionally, we have included a discussion of the possibility that aducanumab and lecanemab could serve as cutting-edge treatments for patients with refractory Alzheimer's disease. The FDA recently granted lecanemab treatment approval for Alzheimer's disease [5].

Result

Late examinations on the etiology of Alzheimer's sickness have raised a few variables impacting its pathogenesis. Derangements in vitamin B12, folate, and homocysteine levels, oral bacteria that cause periodontitis and insulin resistance, and their effects on the brain as a whole, all play a significant role in the pathogenesis of Alzheimer's disease, as we discuss in this review article [6].

Alzheimer's disease (AD) is one of the most prevalent debilitating, irreversible, age-related neurodegenerative conditions that ultimately leads to death. Amyloid beta (A) plaque deposition in the parenchyma external to the neurons and neurofibrillary tangles are two pathological characterizations of AD.1, 2, 3, and 4 In the elderly adult population, the prominent impaired memory and cognitive loss is the pathological characterization [7].

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Received: 1-May-2023, Manuscript No: dementia-23-97167; **Editor assigned:** 4-May-2023; Pre QC No: dementia-23-97167 (PQ), **Reviewed:** 17-May-2023; QC No: dementia-23-97167; **Revised:** 24-May-2023; Manuscript No: dementia-23-97167 (R), **Published:** 30-May-2023, DOI: 10.4172/dementia.1000155

Citation: Liu X (2023) A Short Note on Management of Alzheimer's Dementia. J Dement 7: 155.

Moreover, there are favorable to fiery phagocytic microglial cells, receptive astrocytosis, dystrophic neurites, tau-positive neuronal strings, and different designs. AD is brought on by these structural and inflammatory abnormalities, which result in the loss of neurons in brain regions that are vulnerable. There is evidence to suggest that structural changes in the brain [8], such as an increased reduction in the volume of the hippocampus and a decreased metabolism of glucose, may take place prior to amyloid deposition and tau pathology in AD. Clinical attributes incorporate loss of memory, engine issues, and social dependence at last begin to show up after it [9].

Discussion

The course of ongoing neuro-aggravation in the mind has been straightforwardly associated with mental deterioration, loss of memory, and dementia, as per various writing discoveries, and it adds to the pathogenesis of Promotion. Studies in vitro, in vivo, and using data from transgenic mice have all supported these observations, which show that there is an increased amount of cytokines, proteases, and chemokines in addition to oxidative stress around senile A-plaques.5, 6, 7 Amyloid Precursor Protein (APP), a transmembrane protein that is widely expressed in brain neurons [10], has long been thought to be the catalyst for A synthesis.15, 16, 17 The two main types that are the most prominent hallmarks of Alzheimer's disease are (i) neurofibrillary tangles (NFTs), which are mostly made of hyperphosphorylated microtubule-associated tau proteins, and (ii) significant atrophy of brain amyloid plaques, which are The distinction, if any, between Alzheimer's dementia and Alzheimer's disease has been the subject of a great deal of debate and misunderstanding [11]. The terms were frequently used interchangeably until fairly recently. They both referred to dementia that was found to be associated with tau-containing neurofibrillary tangles and beta-amyloid plaques, the neuropathological hallmarks that Dr. Alois Alzheimer first described in 1906, according to autopsy results. In 1989, when I first started practicing neurology, we called suspected Alzheimer's disease senile dementia of the Alzheimer's type (SDAT) [12]. We based our diagnosis on our experience with patients who were similar to ourselves at the time because there was no practical way to make a definitive diagnosis during life. The availability of useful amyloid and tau biomarkers has significantly improved diagnosis accuracy throughout life [13]. These include PET scans of the brain, tests on spinal fluid, and, most recently, a number of very sensitive and specific blood tests that should soon be available in the commercial market.

Conclusion

Let's return to the question of what makes Alzheimer's disease distinct from Alzheimer's dementia and why this distinction is so significant. Alzheimer's disease progresses over time [14]. Dementia reaches one extreme: cognitive impairment that makes it hard to do things every day, gets worse over time, and eventually kills you. The Alzheimer's dementia phase typically lasts eight to ten years. Most of the time, the first signs of cognitive problems don't affect daily activities. Work might in any case be conceivable. Mild cognitive impairment (MCI) is the term for this stage. MCI, like dementia, can be caused by other disorders, but when amyloid or tau biomarkers are present, almost always Alzheimer's disease is the cause [15]. We once only cared about MCI and dementia. However, it is essential to comprehend that the amyloid plaques and neurofibrillary tangles of Alzheimer's disease begin to manifest in the brain up to twenty years prior to the onset of cognitive impairment.

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