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ALZHEIMER'S DISEASE: CURRENT STATE OF KNOWLEDGE, DIAGNOSIS, TREATMENT, AND THE ROLE OF LIFESTYLE IN PREVENTION AND THERAPY

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ABSTRACT

Background: Alzheimer's disease (AD) is a progressive neurodegenerative disorder that leads to gradual memory loss, impaired thinking, and cognitive decline. It is characterized by the accumulation of amyloid plaques and neurofibrillary tangles in the brain, which damage nerve cells. As the disease progresses, patients lose the ability to function independently, resulting in complete dependence on care.

Aim: This review aims to summarize current knowledge about AD and evidence on dietary and lifestyle factors associated with AD

Material and methods: A narrative review of recent literature was conducted using peer-reviewed articles published in PubMed and Google Scholar.

Results: Based on a literature review, a proper balanced diet, rich in antioxidative and anti-inflammatory nutrients, may have a beneficial effect on the prevention of AD. Additionally, the diet should avoid highly processed foods, rich in saturated and trans fatty acids, and poor quality food, which may increase exposure to the consumption of pollution and toxins, such as heavy metals.

Conclusions: Research, in addition to classical pharmacological treatment and modern imaging techniques, highlights the significant role of lifestyle changes—especially diet and regular physical activity—in the prevention and treatment of AD. Numerous studies indicate that maintaining a healthy, active lifestyle and a proper diet can significantly reduce the risk of developing cognitive impairments and dementia, including AD.

KEYWORDS

Alzheimer's Disease, Dementia, Genes, Lifestyle, Physical Activity, Risk Factors, Prevention

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Description of the current state of knowledge

Alzheimer's disease (AD) is the most common form of dementia, accounting for 60–80% of all cases. It is currently estimated that around 50 million people worldwide suffer from this debilitating neurodegenerative disorder. The disease also indirectly affects the lives of tens of millions of caregivers and loved ones who witness the gradual decline in cognitive function over the years. [2]

AD was first described in 1906 by Alois Alzheimer, based on the case of Auguste Deter—a 51-year-old woman suffering from cognitive impairment, disorientation, delusions, and other behavioral changes. Alzheimer first encountered her in 1901, and she died 4.5 years later. The diagnostic criteria for AD were updated in 1984 and subsequently refined in 2011 and 2018 to incorporate the use of biomarkers and enable the identification of preclinical stages of the disease. Although the pathological features characteristic of AD were qualitatively described by Alzheimer, it was not until the mid-1980s that the molecular nature of the disease's two hallmark features was discovered: beta-amyloid peptide, found in amyloid plaques, and hyperphosphorylated tau protein, which forms neurofibrillary tangles. [5]

AD is going to more and more common. It is currently estimated that 5.7 million Americans suffer from dementia caused by AD. Projections indicate that by the middle of the 21st century, this number will rise to 13.8 million. In 2015, AD was the cause of 110,561 deaths according to official death certificates, making it the sixth leading cause of death in the United States and the fifth among individuals aged 65 and older. Between 2000 and 2015, the number of deaths due to stroke, heart disease, and prostate cancer declined, while deaths related to AD increased by as much as 123%. [7]

AD is a condition with a complex and heterogeneous pathobiology. The key neuropathological criteria for its diagnosis include extracellular deposits of amyloid- β forming neuritic plaques and intracellular accumulation of hyperphosphorylated tau in the form of neurofibrillary tangles. [1]

The pathogenesis of AD has not yet been definitively established. In the vast majority of cases, it is sporadic and multifactorial, with both environmental and genetic factors contributing to its development. The main risk factors include age, level of education, quality of interpersonal relationships, and polymorphism of the APOE gene, which encodes apolipoprotein E — the only genetic risk factor for AD confirmed to date. [3]

The $\epsilon 4$ allele of the APOE gene, compared to the most common $\epsilon 3$ allele, remains the strongest known genetic risk factor for the development of sporadic AD. In contrast, the relatively rare APOE $\epsilon 2$ allele is the most potent genetic protective factor against the disease, clearly highlighting the crucial role of the APOE gene in its pathogenesis. [4]

The multifactorial form should be clearly distinguished from the rare familial cases caused by mutations in single genes, which account for approximately 1.5–2% of all cases. [3]

Diagnosis

The mainstay of AD diagnosis remains the clinical assessment, which primarily includes an interview with the patient and an informant, as well as cognitive evaluation and a focused physical examination. However, with the growing recognition that pathological changes begin many years before the onset of symptoms, and with the emergence of biomarkers for β -amyloid and tau pathology, along with MRI-based brain atrophy imaging, diagnostic criteria have undergone significant changes. These advancements now allow for earlier detection of the disease and greater diagnostic specificity. The latest criteria developed by the National Institute on Aging (NIA) and the International Working Group (IWG-2) now incorporate one or more preclinical stages of AD, in which the presence of biomarkers indicates underlying AD pathology despite the absence of clinically observable symptoms. [6]

The clinical stages of AD can be divided into the following phases: **Preclinical phase** – a period that may last for many years. During this stage, mild memory impairment and early pathological changes in the cerebral cortex and hippocampus are observed; however, they do not yet affect the patient's daily functioning, and no clear clinical symptoms of the disease are present. **Mild stage of AD** – this is when the first noticeable symptoms begin to appear, such as difficulties in everyday activities, problems with memory and concentration, disorientation in time and place, mood swings, and the development of depression. **Moderate stage of AD** – the disease progresses and affects additional areas of the cerebral cortex. This stage is marked by worsening memory loss, difficulty recognizing close relatives, loss of impulse control, and problems with speaking, reading, and writing. **Severe stage of AD** – in this phase, the disease affects almost the entire cerebral cortex, with significant accumulation of neuritic plaques and neurofibrillary tangles. There is a profound decline in both cognitive and physical functions – the patient no longer recognizes loved ones, is often bedridden, and experiences difficulties with swallowing and urination. At this stage, the disease ultimately leads to death due to complications. [10]

The diagnosis of AD also relies largely on positron emission tomography (PET) using tracer molecules and the analysis of proteins in cerebrospinal fluid (CSF). One of the key biomarkers used for confirming and predicting AD is phosphorylated tau protein 181 (P-tau181). It accumulates in the brain, is released into the CSF, and then crosses the blood–brain barrier into the bloodstream, where it can be detected and serve as an indicator of the disease. In recent years, diagnostic accuracy has significantly improved with the use of specialized PET scans, which demonstrate 100% specificity and 96% sensitivity in both typical AD cases and its milder forms. An alternative approach is CSF analysis for levels of P-tau, A β 42, and total tau protein, which achieves a diagnostic accuracy of 85–90%. However, this method is associated with longer waiting times for results, primarily due to the invasive nature of the procedure (lumbar puncture) and the limited availability of specialized laboratories. [8]

Both PET imaging and CSF analysis show comparable diagnostic performance; thus, the choice of method should depend on patient and physician preference, cost, and availability of resources. Studies indicate that pathological changes in the brain may begin up to twenty years before the appearance of clinical symptoms. For this reason, relying solely on symptom-based diagnosis may be insufficient, as the disease is already in an advanced stage by that time. Therefore, for more effective diagnosis and treatment of AD, the use of biomarkers enabling early detection is strongly recommended. [9]

Treatment

A physically active lifestyle is linked to brain health. In large observational studies with follow-up periods extending decades, physically active people seem less likely to develop cognitive decline, all-cause dementia, vascular dementia and AD when compared with inactive people. Especially, other potential mechanisms underlying the association are most likely indirect, such as the positive effects of physical exercise on other modifiable cardiovascular risk factors, including hypertension, insulin resistance and high cholesterol levels as well as other biological mechanisms, including but not limited to enhancing the immune system function, anti-inflammatory properties, and increasing neurotrophic factors. [11]

The Finnish FINGER study was the first large-scale, long-term randomized clinical trial to demonstrate that a comprehensive lifestyle intervention can effectively reduce the risk of cognitive decline in individuals at risk of dementia. This intervention included a healthy, balanced diet, regular physical activity, cognitive training, social engagement, and management of vascular and metabolic risk factors. The study showed a beneficial impact on cognitive functions, even among individuals with a genetic predisposition to AD. In a substudy of the French MAPT project, which used PET imaging to detect amyloid deposits, it was found that a lifestyle intervention—either alone or in combination with omega-3 supplementation—was associated with improved cognitive performance in individuals with a positive amyloid β status. This suggests that even if lifestyle modifications do not directly affect the pathological changes characteristic of AD, they can still offer benefits to individuals already affected by the condition. [12]

It is suggested that the best solution in the case of people who have AD, also in the case of a protein supply, is a diet guided by the principles of rational nutrition in the elderly. According to current nutritional standards, the percentage of protein in the diet in the elderly should be increased and it should be around 20% of the daily energy requirement. In AD, the amount of fat in the human diet is not the only important aspect. Its quality is equally important, particularly from which fatty acids it is made. It is suggested that unsaturated fatty acids have a protective effect against AD, while the consumption of SFA has a higher risk of developing the disease according to the WHO, daily energy taken from fat should not exceed 30%. In addition, it is recommended that saturated fatty acids—SFAs should not exceed 10% of the total energy supply, and the daily supply of omega-3 and omega-6 unsaturated fatty acids should be about 250 mg. The diet should also be rich in B vitamins, as well as vitamins E, C, and D, and polyphenols. [13]

In recent years, there has been growing interest in the use of the ketogenic diet (KD) in the treatment of various conditions, including neurodegenerative diseases. Its effects are associated with a reduction in inflammatory responses and a decrease in oxidative stress. Key features of ketogenic therapy include lowered blood glucose levels and elevated concentrations of ketone bodies. In AD, significant metabolic disturbances are observed, such as impaired glucose uptake, weakened mitochondrial brain energy metabolism, altered neurotransmitter release, and an intensified inflammatory response. Although current evidence is limited, studies in animal models and clinical trials suggest that KD may have beneficial effects on mitochondrial function and cellular metabolism. Moreover, improvements in cognitive performance have been reported in older adults with AD. The effectiveness of the diet depends on the level and duration of ketosis, with the most promising results observed in the early, pre-symptomatic stages of the disease. However, implementing this form of therapy requires a practical diagnostic approach. [14]

Recent systematic reviews indicate that adherence to the Mediterranean diet—characterized by meals rich in fresh fruits and vegetables, whole grains, olive oil, legumes, and seafood, while limiting dairy and poultry and avoiding red meat, sweets, and processed foods—is associated with a reduced risk of cognitive decline and AD. [17]

The neuroprotective and probiotic potential of *Lactiplantibacillus plantarum* AM2 in cognitive impairment has also been studied. In one study, a probiotic strain producing acetylcholine was isolated and characterized, and its potential anti-Alzheimer's properties were evaluated in a D-galactose-induced cognitive impairment rat model. In cognitive function tests, rats treated with *Lactiplantibacillus plantarum* AM2 showed a reduced latency time in the Morris Water Maze test, suggesting improved cognitive abilities. Biochemical analyses demonstrated improved blood glucose levels, decreased oxidative stress markers, and increased levels of glutathione and total antioxidant capacity. Histopathological analyses revealed mitigation of hippocampal damage and restoration of normal cellular architecture. These results indicate that *Lactiplantibacillus plantarum* AM2 is a promising probiotic candidate with neuroprotective effects and the ability to produce acetylcholine. Further research is needed to assess its therapeutic applications. [15]

One study evaluated the effects of a six-month aerobic exercise program on patients with AD and their caregivers. The results showed that regular physical activity significantly reduced neuropsychiatric symptoms

in patients and lowered caregiver burden. Participants who engaged in aerobic exercise demonstrated improvements in areas such as irritability, anxiety, apathy/indifference, and appetite disturbances compared to the control group, which may be attributed to both the alleviation of symptoms in the intervention group and the worsening of symptoms in the control group. [16]

Despite medical advances, we do not have a causal treatment for AD. Symptomatic treatment is used. In addition to the non-pharmacological approaches described above, there are few groups of medications approved for the treatment of this condition. The classic group of drugs we can use are cholinesterase inhibitors. This group of drugs includes donepezil, rivastigmine, and galantamine. The goal of this treatment is to improve cognitive function. The response is assessed based on improved memory, function, or behavior reported by the patient or caregiver. [18]

Another group of drugs that can be used are anti-glutaminergic agents, which work by regulating glutamate levels through noncompetitive inhibition of NMDA receptors. Excessive levels of this neurotransmitter can lead to harmful changes in the brain, including neuronal death. The goal of this therapy is to slow the progression of the disease, stabilize or temporarily improve cognitive functions, and manage behavioral disturbances. Although these drugs are not curative, they help maintain the patient's independence and improve the quality of life for both individuals with AD and their caregivers. [19]

Unfortunately, due to the complex nature of AD, its treatment remains a significant challenge. Currently approved pharmacological therapies are limited to cholinesterase inhibitors, memantine, or a combination of these drugs. Despite promising results in early-phase studies, many new medications have failed to demonstrate efficacy in phase III clinical trials, falling short of the predefined endpoints. [20]

Antidepressants are frequently used in the treatment of AD. Sertraline and mirtazapine are commonly prescribed antidepressants for depression associated with dementia; however, their effectiveness remains uncertain. A randomized, multicenter, double-blind, placebo-controlled trial evaluating the effects of various antidepressants in patients with dementia showed no significant benefits compared to the control group. No statistically significant differences were found in scores on the Cornell Scale for Depression in Dementia between 107 participants treated with sertraline or mirtazapine and 111 individuals in the control group, both at 13 and 39 weeks. [21]

Conclusions

AD is characterized by a complex and multifactorial pathology. Diagnosis is based on clinical assessment supported by imaging studies and biomarker analysis, which allows detection of changes long before symptoms appear. Prevention and treatment of AD include lifestyle changes such as regular physical activity, a healthy diet (e.g., Mediterranean diet), and cognitive training, as well as pharmacological treatment. Regular physical exercise not only supports the cardiovascular system but also improves brain metabolism, reduces inflammation, strengthens the immune system, and promotes increased levels of neurotrophic factors that support neuron regeneration and function. Regarding diet, a nutritional model consistent with the principles of rational nutrition for seniors is recommended, with particular emphasis on increased protein intake and the quality of fats. It is advised to limit saturated fats while increasing the proportion of unsaturated fatty acids, especially omega-3 and omega-6. The diet should also be rich in B vitamins, vitamins E, C, D, and polyphenolic compounds, which exhibit antioxidant and anti-inflammatory properties.

Disclosures**Author's contribution**

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