



# A Brief Overview on Alzheimer’s disease

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Alzheimer’s disease (AD) is a neurodegenerative disorder characterized by progressive memory loss and cognitive decline. It is the most common cause of dementia, accounting for 60-70% of cases. The disease is caused by a combination of genetic and environmental factors, leading to the accumulation of amyloid plaques and neurofibrillary tangles in the brain. These pathological changes disrupt neuronal function and lead to the death of brain cells. The clinical course of AD is characterized by a gradual decline in memory and cognitive abilities, eventually leading to severe dementia. The disease is currently incurable, but early diagnosis and management can help to slow down the progression of the disease and improve the quality of life of affected individuals. Research is ongoing to identify potential therapeutic targets and develop effective treatments for AD.

The pathogenesis of AD is complex and involves multiple pathways. The amyloid hypothesis is the most widely accepted theory, suggesting that the accumulation of amyloid-beta (Aβ) plaques in the brain is the primary driver of neurodegeneration. Aβ is a peptide derived from the amyloid precursor protein (APP), which is cleaved by beta-secretase and gamma-secretase. The resulting Aβ monomers aggregate to form oligomers and eventually large plaques. These plaques are thought to be toxic to neurons, leading to synaptic dysfunction and neuronal death. Other theories include the cholinergic hypothesis, which suggests that a deficiency of acetylcholine (ACh) in the brain leads to cognitive impairment. This hypothesis is supported by the fact that ACh levels are significantly reduced in AD brains, and cholinergic agonists have been shown to improve cognitive function in AD patients. The tau hypothesis suggests that the hyperphosphorylation of tau protein leads to the formation of neurofibrillary tangles, which are also toxic to neurons. The interplay between these different pathways is still unclear, and further research is needed to fully understand the underlying mechanisms of AD.

Genetic factors play a significant role in the development of AD. The APOE ε4 allele is the most common genetic risk factor for late-onset AD, with carriers having a 2-3 fold higher risk of developing the disease. The ε4 allele is thought to promote the aggregation of Aβ and impair its clearance from the brain. In contrast, the ε2 and ε3 alleles are considered protective, with the ε2 allele being particularly so. The ε3 allele is the most common, accounting for about 65% of the population. The ε4 allele is present in about 15% of the population, but its frequency increases significantly in AD patients. The ε4 allele is also associated with a higher age at onset and a more rapid decline in cognitive function. In addition to APOE, several other genes have been identified as being associated with AD, including PSEN1, PSEN2, and APP. These genes are primarily associated with early-onset, familial AD, but they also influence the risk of late-onset AD. The APP gene encodes the APP protein, which is the precursor of Aβ. Mutations in APP lead to increased production of Aβ and a higher risk of developing AD. PSEN1 and PSEN2 encode presenilin-1 and presenilin-2, which are γ-secretase subunits. Mutations in these genes lead to increased production of Aβ and a higher risk of developing AD.

Environmental factors also play a role in the development of AD. Education, lifestyle, and diet are all thought to influence the risk of developing the disease. Higher levels of education are associated with a lower risk of AD, possibly due to cognitive reserve. Physical activity and a healthy diet, such as the Mediterranean diet, are also associated with a lower risk of AD. The Mediterranean diet is rich in antioxidants, which may help to reduce oxidative stress and inflammation, both of which are thought to contribute to the development of AD. Smoking is also associated with a lower risk of AD, although the mechanism is unclear. The use of statins, which are cholesterol-lowering drugs, has also been associated with a lower risk of AD. However, the use of statins should be weighed against the risk of side effects, such as muscle pain and liver damage. The use of anti-inflammatory drugs, such as NSAIDs, has also been associated with a lower risk of AD. However, the use of NSAIDs should be weighed against the risk of side effects, such as stomach ulcers and kidney damage. The use of hormone therapy in postmenopausal women has been associated with a higher risk of AD. However, the use of hormone therapy should be weighed against the risk of side effects, such as blood clots and breast cancer.

Diagnosis of AD is based on a combination of clinical history, physical examination, and cognitive testing. The Mini-Mental State Examination (MMSE) is a widely used cognitive test that assesses memory, orientation, and other cognitive functions. A score of 24 or higher is considered normal, while a score of 23 or lower is considered abnormal. The MMSE is a simple and easy-to-use test that can be administered by a healthcare professional. Other cognitive tests, such as the Montreal Cognitive Assessment (MoCA) and the Clock Drawing Test, are also used to diagnose AD. The MoCA is a more comprehensive cognitive test that assesses a wider range of cognitive functions, including memory, attention, and executive function. The Clock Drawing Test is a simple test that involves drawing a clock face and labeling the hours. It is used to assess visuospatial abilities and executive function. In addition to cognitive testing, physical examination and laboratory tests are also used to diagnose AD. Physical examination may reveal signs of systemic disease, such as hypertension and diabetes, which can contribute to cognitive decline. Laboratory tests, such as blood and urine tests, may be used to rule out other causes of cognitive decline, such as vitamin deficiencies and thyroid disease. The diagnosis of AD is a complex process that requires a thorough evaluation of the patient’s clinical history and cognitive function. Early diagnosis is important for the management of AD, as it allows for the implementation of interventions that can help to slow down the progression of the disease and improve the quality of life of affected individuals.

The management of AD is primarily focused on symptom management and the use of medications to improve cognitive function. Cholinesterase inhibitors, such as donepezil, rivastigmine, and galantamine, are the first-line medications for AD. These drugs work by increasing the levels of ACh in the brain, which can help to improve memory and cognitive function. Memantine, an NMDA receptor antagonist, is also used to improve cognitive function in AD patients. It works by blocking the NMDA receptor, which is thought to be overactive in AD. The combination of a cholinesterase inhibitor and memantine is often used to improve cognitive function in AD patients. In addition to medications, non-pharmacological interventions, such as cognitive stimulation therapy and exercise, are also used to improve cognitive function in AD patients. Cognitive stimulation therapy involves a series of structured activities that challenge the patient’s memory and cognitive abilities. Exercise, particularly aerobic exercise, has been shown to improve cognitive function in AD patients. The use of these interventions can help to slow down the progression of the disease and improve the quality of life of affected individuals. The management of AD is a complex process that requires a multidisciplinary approach involving healthcare professionals, family members, and the patient. The goal of management is to improve the patient’s quality of life and to help them to live as independently as possible for as long as possible.

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