

Alzheimer's Disease and Its a Diagnosis, Symptoms, Pathophysiology, and Treatment

Devram B. Sodha¹, Devang H. Joshi², Dhruvik B. Joshi³, Rajesh M. Vala⁴, Dharmesh R. Makvana⁵

^{1,2,3,4,5}Student at Gyanmanjari Pharmacy College, Bhavnagar-364001 Gujarat

Abstract

Alzheimer's disease (AD) is the most prevalent form of dementia, predominantly affecting individuals over 65 years old, though a rare form can manifest in younger people. Globally, AD accounts for approximately 75% of the more than 35 million dementia cases, with projections suggesting the number could rise to 115 million by 2050. The precise etiology of Alzheimer's remains largely unknown. Historically, dementia has been recognized since ancient times, with significant milestones in understanding and terminology occurring from the 17th century onwards. Alzheimer's disease is identified by symptoms such as confusion, personality changes, language difficulties, mood swings, and short-term memory disturbance. Pathophysiologically, AD is characterized by amyloid plaques and neurofibrillary tangles (NFTs) which cause brain damage. Diagnosis involves clinical evaluation, neuroimaging, neuropathological findings, and cerebrospinal fluid analysis. Risk factors include genetic predisposition, vascular issues, and psychosocial elements. Current treatments focus on acetylcholinesterase inhibitors to manage symptoms, particularly in the mild to moderate stages of the disease.

Keywords: Alzheimer's disease(AD), diagnosis, symptoms, risk factors, and treatment, dementia.

Introduction

Alzheimer's disease is the most common type of dementia. It is a eventually leads to death. The disease is usually found in people over 65, a rare from can appear in younger people. At globally Alzheimer's disease affect about 75% of the more than 35 million people with dimentia. The people with a d is expected to double every 20 years.so by 2050, around 115 million people omight have the disease. The exact cause of Alzheimer's are mostly unknown.

Historical background

History of dimentia dates back to ancient times. Around 2000 BC, she Egyptian noted the memory decline with age growth. In the second century A.D, arethus ditiguished between reversible delirium and irreversible dementia. During middle ages, dementia was often viewed as decline punishment. Thomas willis coined neurology and academically describe vascular dementia in 17th century . The term dementia was accepted in late 18th century by Philippe pinel. In 19th century otto binswanger and alois alzheimal studied dementia causes, leading to the term Alzheimer's disease.



Symptoms

- Confusion
- Personality changes
- Language difficulties
- Unexplained mood swings
- Disturbance in short term memory

Pathophysiology

Alzheimer's disease is characterized by two main factors; one is amyloid plaques and NFTS. These are causes brain damage and leads to symptoms of AD.

1)amyloid plaques: these are dumps of proteins call beta-amyloid outside brain cells. They mainly consist of two type of beta-amyloid proteins,AB40 and AB42, with AB42 being more common in plaques. Amyloid plaques usually form in the outer layer of the brain and affected deeper brain structures later.

2)NFTs: these are twisted fibres inside brain cell made of a protein called tau, which become abnormally modified. Tau langles Start in areas related to memory and then spread to other part of the brain.

Amyloid precursor protein (app)

Developed by beta secretase and alpha secretase

Mutation of amyloid precursor are protein on chromosome number 21

Increase the production of APP

Produces of amyloid protein

Accumulation of beta amyloid protein

Directly neurotoxin

\downarrow

Alzheimer's disease

Diagnosis

Key diagnostic steps:

- 1. clinical evolution: slow, progressive dementia is primary sign.
- 2. **Neuroimaging**: brain scans (CT,MRI) may show cortical atrophy, and PET scan may show reduce brain metabolism.
- 3. **Neuropathology findings**: at autopsy, the present of beta amyloid plaques , and amyloid angiopathy confirm AD.
- 4. Cerebro spinal fluid (CSF):lower level of AB amyloid 42 and higher level of tau protein are indicators of AD.



• Email: editor@ijfmr.com

Risk factor

- 1. Genetic hypothesis
- 2. Vascular hypothesis :
- Smoking
- Obesity •
- High cholesterol •
- Hypertension •
- Diabetes and •
- Stroke associated with higher AD risk. •
- 3. Phychosocial hypothesis :
- Low education increases AD risk. •
- Strong social network and engagement lower AD risk, while isolation increases it.
- Physical activities etc. •

Treatment

Medication:

1. Acetylcholinesterase inhibitors:

- Includes doneperil, galantamine, and rivastigmine. •
- Disease Ach in the brain to improve symptoms. •
- Effective in mild to sever AD, particularly in mild to moderate stages. •

Conclusion

Alzheimer's disease remains a significant global health challenge with increasing prevalence expected in the coming decades. Despite its unclear etiology, advancements in diagnostic techniques and understanding of its pathophysiology, such as the roles of amyloid plaques and NFTs, have improved the ability to identify and study AD. Risk factors encompass genetic, vascular, and psychosocial aspects, highlighting the importance of a comprehensive approach to prevention and management. While treatments like acetylcholinesterase inhibitors provide symptomatic relief, ongoing research is crucial for developing more effective interventions to combat this debilitating disease

Reference:

- 4. Lobo A, Launer LJ, Fratiglioni L, Andersen K, Di Carlo A, Breteler MM, Copeland JR, Dartigues JF, Jagger C, Martinez-Lage J, Soininen H, Hofman A. Prevalence of demen tia and major subtypes in Europe: A collaborative study of population-based cohorts. Neurology 2000;54:S4-9.
- 5. Plassman BL, Langa KM, Fischer GG, Hee ringa SG, DR Weir, Ofstedal MB, Burke JR, Hurd MD, Potter GG, Rodgers WL, Steffens DC, Willis RJ, Wallace RB. Prevalence of dementia in the United States: the aging, demographics, and memory study. Neuroepidemiology 2007;29:125-32.
- 6. Office of National Statistics. Deaths Registered in England and Wales. 2016;1–15.
- 7. Wu Y-T, Fratiglioni L, Matthews FE, et al. Dementia in western Europe: epidemiological Evidence and implications for policy making. Lancet Neurol. 2016;15:116–24.
- 8. Matthews FE, Stephan BCM, Robinson L, et al. A two decade dementia incidence Comparison from the Cognitive Function and Ageing Studies I and II. Nat. Commun. 2016;7:11398.



E-ISSN: 2582-2160 • Website: www.ijfmr.com • Email: editor@ijfmr.com

- C. Caspersen, N. Wang, J. Yao, A. Sosunov, X. Chen, J.W. Lustbader, H.W. Xu, D. Stern, G. McKhann, S.D. Yan, Mitochondrial abeta: a potential focal point for neuronal Metabolic dysfunction in Alzheimer's disease, FASEB J. 2005; (19): 2040–2041.
- 10. X. Chen, S.D. Yan, Mitochondrial abeta: a potential cause of metabolic dysfunction in Alzheimer's disease, IUBMB Life 2006; (58): 686–694.
- 11. Alzheimer A. UbereineeigenartigeErkrankung der Hirnridne [About a Peculiar Disease of The Cerebral Cortex]. Allg Z Psychiatr 1907; 64: 146-48.
- Alzheimer A. About a peculiar disease of the cerebral cortex. Alzheimer Dis AssocDisord 1987; 1: 3-8.
- 13. Koukolík F., Jirák R. Alzheimerova nemoc a další demence, Galén, 1998.
- 14. Fratiglioni L, von Strauss E, Qiu CX. Epidemiology of the dementias Of old age. In Dening T, Jacoby R, Oppenheimer C, Thomas A, eds. The Oxford Textbook of Old Age Psychiatry. 4th ed. New York, NY: Oxford University Press 2008;391-406.
- 15. Blennow K, de Leon MJ, Zetterberg H. Alzheimer's disease. Lancet 2006;368:387-403.
- 16. Green RC, Cupples LA, Go R, Benke KS, Edeki T, Griffith PA, Williams M, Hipps Y, Gr aff-Radford N, Bachman D, Farrer LA.Risk of demen-Tia among white and African American relatives of patients with Alzheimer disease. JAMA 2002;287:329 -36.
- 17. Revesz T, McLaughlin JL, Rossor MN, Lantos PL. Pathology of familial Alzheimer's disease With Lewy bodies. J. Neural Transm. Suppl. 1997;51:121–35.
- 18. James BD, Wilson RS, Boyle PA, Trojanowski JQ, Bennett DA, Schneider JA. TDP-43 stage, Mixed pathologies, and clinical Alzheimer's-type dementia. Brain. 2016;139:2983–93.