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Alzheimer's Disease: An Overview Study

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ABSTRACT

Alzheimer's disease (AD) is a complex neurological ailment characterised by progressive loss of thinking, memory, and language that eventually results in the inability to perform social and functional tasks necessary for daily living. After being diagnosed with AD-related dementia, the average survival time is 4 to 8 years; however, other circumstances, such as co-occurring medical problems, can affect life expectancy. appear gradually and progressively, increasing with time from mild amnesia to severe mental impairment. To enable an earlier and more precise diagnosis, there is therefore rising interest in the development of biomarkers that may be used to identify these alterations in the brains of people who are at-risk. Successful therapies depend on an early diagnosis, although this can be challenging given the current reliance on clinical observation and cognitive testing. Brain pathology discovered after death can validate the diagnosis and show that AD is the root cause. Treatment to reduce or stop the disease's progression would be the gold standard; however, there is very little information available on the early stages of AD's cause. Though age is by far the biggest risk factor, age is not a necessary component of ageing; rather, the disease is more likely to develop in older people. The chance of acquiring the disease is impacted by both hereditary and environmental variables. Many quantitative surrogate measures of disease progression, such as imaging and biochemical biomarkers, have been proposed as effective endpoints for potential therapy. However, little is known about the dynamics of these many surrogate markers throughout time, especially with regard to the onset of disease. The definition of "Alzheimer's disease," its global spread, its causes, and the most important biomarkers that may offer assistance with this issue are all examined in this study.

Keywords: Alzheimer's Definition; Prevalence; Alzheimer's Disease Causes; Biomarkers of Alzheimer's Disease

Abbreviations: AD: Alzheimer's Disease; AB: Amyloid-Beta; GM: Grey Matter; APP: Amyloid Precursor Protein; PET: Positron Emission Tomography

Definition of Alzheimer's Disease

Three main sets of symptoms make up the chronic, progressive neurodegenerative condition known as Alzheimer's disease. Memory loss, language impairment, and executive dysfunction (loss of higher level planning and intellectual coordinating skills) are all included in the first category (cognitive dysfunction). The second category consists of behavioural and psychiatric symptoms, such as agitation, hallucinations, delusions, and depression, which are collectively referred to as non-cognitive symptoms (Burns, et al. [1]). The third category consists of people who have trouble conducting activities of daily life (considered "basic" for getting dressed and eating by themselves and "instrumental" for more complicated tasks like driving and shopping). Alzheimer's disease symptoms range from minor memory loss to extremely severe dementia (figure). Clinically, pathologically, and epidemiologically, the coexistence of vascular disease with Alzheimer's disease is being recognised more frequently (Snowdon, et al. [2]). The most frequent form of dementia is Alzheimer's disease (AD), which is clinically characterized by a progression from episodic memory issues to a gradual overall deterioration in cognitive function. (Citron [3]). One of the most prevalent factors contributing to dementia that affects nerve cells across the brain is Alzheimer's disease.

This neurodegenerative illness is pathologically caused by intracellular neurofibrillary tangles and extracellular amyloidal protein, and it leads in the development of plaques that impede communication between the nerve cells (Kaloni and Negi [4]). Dementia, which is characterized by a decline in thinking and independence in daily tasks, is mostly brought on by Alzheimer's disease (AD), a sickness that results in the degradation of brain cells. The cholinergic and amyloid hypotheses were put up as two key causes of AD, and AD is thought to be a complex illness. Additionally, the condition is influenced by a number of risk factors, including advancing age, hereditary variables, head injuries, vascular diseases, infections, and environmental factors. Breijyeh and Karaman [5]. Alzheimer's disease (AD) is a neurological condition marked by a progressive deterioration in cognitive function and memory. The disease's aetiology is poorly understood, and a number of mechanisms, including oxidative stress, neuroinflammation, cholinergic neuronal apoptosis, and the buildup of aberrant proteins in the brain, contribute to its onset (Hanafy, et al. [6]).

One of the risk factors for Alzheimer's disease that is developing at a rapid rate in middle age is coronary heart disease, greater homocysteine levels, which might cause withdrawal due to a change in lifestyle. In order to recollect memories, cigarettes help to avoid Alzheimer's disease. Although there is no known cure for Alzheimer's, drugs like Donepezil and the ability to raise acetylcholine levels and lower amyloid beta deposition have both been shown to be quite helpful in treating Alzheimer's (Esmaeili, et al. [7]). The most common cause of dementia in the world, AD is a neurodegenerative condition associated with ageing. The progressive accumulation of extracellular amyloid-(A) plaques, intracellular neurofibrillary tau tangles, neuroinflammation, cerebral small vessel disease, and neurodegeneration are just a few of the neuropathologic changes linked to AD. Many of these changes are known to start years before the appearance of clinical symptoms (Georgakas, et al. [8]).

Prevalence of Alzheimer's Disease

Given the rise in life expectancy around the world, this poses a significant social and economic problem that affects people with AD, as well as their family members and carers. Short-term memory loss is a common beginning symptom of the condition, and as it advances, other symptoms may include language difficulties, disorientation, mood swings, and behavioural disorders (such as agitation, irregular sleep patterns, and psychosis). The condition eventually advances to loss of physiological function and, eventually, death.

Like any other disease, dementia is very prevalent in different parts of the world. This is partly because different studies use different diagnostic criteria, have varied mean population ages, and have diverse socioeconomic loads and educational backgrounds, which result in a lack of scientific homogeneity. By 2025, it's anticipated that the majority of people in the world who are 60 or older will reside in emerging nations (Rizzi, et al. [9]). About 50,000,000 people worldwide are affected by Alzheimer's disease (AD), which is one of the commonest and most significant causes of dementia in the elderly population (Hodson [10]). Dementia typically lasts 8 to 10 years, and its average patient is 80 years old. However, scientists believe that by 2050, the prevalence of the disease will have tripled (Liu and Li [11]). Clinical indicators appear 20 years after the onset of physiological symptoms, and an increase in extracellular amyloid-beta (AB) is the diagnostic criterion for the condition (Masters, et al. [12]).

In the next 20 years, there may be a 35% increase in the number of new instances of AD, according to a report (Alzheimer's Disease Facts and Figures [13]). According to this estimate, there are roughly 7.7 million new cases of dementia annually worldwide, or one instance every four seconds. The prevalence is 4.4 million in 2015, and by 2050 it is expected to have doubled (Sadleir and Vassar [14]). The prevalence of AD is rising in Asia, Europe, and North America, in that order. (Ghara and Roy [15]). An early-onset disease is one that affects people under the age of 60 in 1-6% of recorded cases. As the condition worsens, the fatality rate rises. Tomike, et al. [16]. AD has been labelled a global health crisis that could affect up to 139 million people globally by 2050 due to the disease's rapidly increasing prevalence and associated economic cost (World Health Organization [17]). As various clinical trials have demonstrated minimal therapeutic benefit when experimental treatments are employed after the accumulation of several insults, biomarkers will likely play a significant role in assuring the success of future treatments by assisting in early diagnosis (Salloway, et al. [18]). Memory loss results from Alzheimer's disease (AD), a neurodegenerative condition that worsens over time and causes major disruptions to normal brain structure and function (Korolev [19]).

It is a neurodegenerative condition that progresses fatally and for which there is no viable treatment or cure. Treatment to reduce or halt disease development would be the gold standard therapy, however there is very little understanding of the underlying causes of AD in its early stages (Lawrencea, et al. [20]). With advancing age, the risk of acquiring AD rises, especially after age 65. A syndrome called dementia is used to describe how intellectual function gradually deteriorates. The most prevalent kind of dementia is AD (Long and Holtzman [21]). According to the 2019 World Alzheimer Report, there are already more than 50 million individuals living with dementia worldwide, and as the population ages, this figure is projected to rise to 152 million by 2050 Alzheimer's Disease International. World Alzheimer Report [22]. Reviewing the incidence data for AD reveals that it is the sixth most common cause of mortality in the US. In 2019, 5.8 million Americans of all ages (approximately 1.7%) were predicted to have Alzheimer's dementia (Alzheimer's disease facts and figures [13]). Nearly 459,000 Australians (or 1.8% of the population) already live with dementia, and it is predicted that this figure will rise to 590,000 by 2025 and 1,076,000 by 2050. In Australia, dementia is the second most common cause of death. (Dementia Australia Home Page [23]).

More over 30 million people had Alzheimer's disease in 2015, and by 2050, that number may rise to more than 114 million. A patient's capacity to think clearly may be adversely affected by dementia brought on by AD, which can induce tissue loss in every area of the brain and cause serious harm to the nervous system (Wang, et al. [24]). Unlike age-related memory decline, Alzheimer's disease (AD) is a kind of dementia. AD is prevalent in seniors 65 years of age and older. It is a progressive, neurological, and irreversible brain condition) Kerppers, et al. [25]. Early signs include forgetting previous conversations or occurrences, apathy, and depression. These symptoms may progress to impaired speech, disorientation, confusion, poor judgement, behavioural changes, and eventually trouble speaking, swallowing, and walking. Numerous variables, including genetic mutations (less than 5%), improper protein folding, damage to membrane neurons, mitochondrial malfunction, oxidative stress, the creation of toxic molecules, and neuroinflammatory processes are linked to neurodegeneration. The absence of effective biomarkers for early diagnosis, prevention, and therapy approaches will be the biggest difficulty in treating Alzheimer's. (Zvěřová [26]).

Alzheimer's Disease Causes

The pathology of AD is characterised by the complex interaction of a number of biochemical changes, such as modifications in the metabolism of amyloid precursor proteins, phosphorylation of tau protein, oxidative stress, impaired energetics, mitochondrial dysfunction, inflammation, membrane lipid dysregulation, and disruption of neurotransmitter pathways (Kaddurah Daouk, et al. [27]). The majority of these clinical characteristics are directly related to metabolic abnormalities, and it is now understood that metabolic dysfunction plays a significant role in AD (Cai, et al. [28]). For instance, decreased cerebral glucose absorption, an unchanging aspect of AD, develops decades before the start of cognitive loss (Chen and Zhong [29]). Heart failure, atrial fibrillation, and heart and structural abnormalities limit blood flow to the brain and harm its nerve cells (Perry et al. [30]). Cerebrovascular accidents (strokes), which raise the risk of Alzheimer's disease through cerebrovascular injury and cerebral ischemia, are one of the most significant risk factors (Hickman, et al. [31]). The blood-brain barrier becomes dysfunctional as a result of strokes, which also cause ischemia and an increase in beta amyloid and hyperphosphorylation of tau proteins in the brain. The most significant risk factor for developing Alzheimer's disease is a stroke, and heart conditions account for 20% of all strokes (De Bruijn and Ikram [32]). The risk of Alzheimer's disease is increased by hypercholesterolemia and hyper homocysteine. Hypercholesterolemia raises cholesterol, and its ability to penetrate the blood-brain barrier contributes to an increase in beta amyloid deposition. It raises the prevalence of Alzheimer's by having an impact on the brain. Cardiovascular disease is also influenced by high cholesterol levels. Middle-aged high cholesterol levels are a significant risk factor for Alzheimer's disease with a late onset. Vascular endothelium, cerebrovascular disease, and stroke are all caused by high homocysteine levels (Tumminia, et al. [33]).

AD develops from a variety of factors (Korolev [19]), a number of theories are available to explain the neurodegenerative process as well (Obied, et al. [34]). The primary characteristics of AD, coupled with synaptic and neuronal loss, include the buildup of aberrant proteins, amyloid-(A) deposits (senile plaques), and hyperphosphorylated tau protein (neurofibrillary tangles) inside the neurons. (Long and Holtzman [21]). Following the cleavage of the amyloid precursor protein (APP), cells typically release soluble A-. Senile plaques and aberrant APP cleavage caused by - and -secretases in AD result in the deposition of A into dense beta sheets (Désiré, et al. [35]). In neurons, the protein tau maintains the microtubules (Chen, et al. [36]). Increased phosphorylation in the cerebrospinal fluid may help distinguish AD from other forms of dementia. The abnormal post-translational modification of the tau protein, particularly by phosphorylation, induces conformational changes and the aggregation of tau, resulting in the formation of neurofibrillary tangles. Increased phosphorylation in the CSF fluid may aid in distinguishing AD from other forms of dementia. The level of tau phosphorylation is a marker of the aberrant activity of kinases and phosphatases during the evolution of the disease. A-oligomer levels that are abnormally low or high, as well as hyperphosphorylated tau proteins, can impede synaptic plasticity, lead to pre- and post-synaptic dysfunction, and worsen AD symptoms. (Chen, et al. [36]).

Grey matter (GM) is where the tissue loss begins, and it then spreads to the white matter (WM), corpus callosum (CC), and hippocampus (HC) (Chitradevi and Prabha [37]), gradually. Thus, by examining the differences in the specific structural components of the brain, the early stages of AD can be identified and diagnosed. Mild cognitive impairment (MCI) sufferers are most at risk of developing into the final stage of irreversible brain disorders. (Li, et al. [38]).

Biomarkers of Alzheimer's Disease

In the context of treatment, the use of biomarkers is crucial for ensuring that AD patients have access to existing symptomatic medicines as well as for early disease prognosis in the event that AD disease-modifying drugs become accessible (Abbasi [39]). The biomarker used to identify the disease should be capable of differentiating between dementia's many causes and identifying it at an early stage. Additionally, using induced pluripotent stem cells has shown to be a successful method of treating this illness (Kaloni and Negi [4]). The area of AD biomarker research is gradually transitioning from group-level association studies to subject-level diagnosis and prognosis in practical settings. Recent research trying to evaluate the risk for cognitive decline using biomarkers in patients with moderate cognitive impairment (MCI) is one example of this. (Van Maurik et al. [40-42]). However, at this stage, there has already been a sizable permanent neuronal loss, which may lessen the possibility that disease-modifying medicines may be able to delay the onset of dementia (Sperling, et al. [43]). However, at this stage, there has already been a sizable permanent neuronal loss, which may lessen the possibility

that disease-modifying medicines may be able to delay the onset of dementia (Cullen, et al. [41]).

A biomarker is a biological marker that can detect molecular alterations both physiologically and pathologically (Molinuevo, et al. [44]). An ideal biomarker should be able to distinguish between identical situations without placing an excessive amount of technical burden on the user. It should also be reproducible, highly accurate, non-invasive, affordable, and quick and easy to assess (García, et al. [45]).In this regard, the identification of novel, less invasive blood-based biomarkers for AD may be useful for presymptomatic diagnosis, disease progression monitoring, drug discovery and development, patient stratification, and targeted therapy Blennow and Zetterberg [46].A molecular definition of AD has been made possible by AD biomarkers, virtually eliminating the need for a clinical definition. In our nation, a variety of AD biomarkers are accessible. Each biomarker represents a distinct disease process and stage. The use of biomarker analysis may be justified in some clinical situations, such as an early beginning or an unusual presentation of the disease, despite the fact that prices limit their utilisation. Today, there is no doubt about the value of biomarkers in clinical studies on AD. Additionally, even in the absence of disease-modifying medications, the advent of biomarkers into medical practise has significantly changed therapeutic treatments. (Méndez, et al. [47]).

Blood-based biomarkers for Alzheimer's disease were thought to be impossible for a long time, but new findings suggest that they might indeed be possible. New high-sensitivity tests have produced convincing data that are not only consistent across many cohorts but also independent of the precise analytical technique applied. Amyloid and phosphorylated tau protein concentrations in the blood correlate with the equivalent levels in the CSF and with amyloid-PET or tau-PET scans. Other blood-based biomarkers of neurodegeneration, including glial fibrillary acidic protein and neurofilament light chain, also seem to offer information on disease progression and the ability to track the effects of treatment (Teunissen, et al. [48]). A-42, t-Tau, and p-Tau are the three traditional biomarkers used today to diagnose AD. Cerebrospinal fluid biomarkers' diagnostic utility is constrained by the invasive nature of lumbar puncture-based collection and associated risk of adverse outcomes. Because they need little to no invasive procedures and are therefore simple to collect and analyse, plasma/serum measurements are the gold standard in clinics. Neuroimaging methods like positron emission tomography can be used to visualise the two key proteins A and Tau that are thought to be involved in the disease process (Janeiro, et al. [49]).

List the most promising therapeutically useful and novel AD biomarkers. These consist of cerebrospinal fluid (CSF), blood-based assays, positron emission tomography (PET), [50,51] functional magnetic resonance imaging (fMRI), structural neuroimaging, and fMRI. The potential clinical applications of these biomarkers are then discussed, including the identification and management of neuropsychiatric symptoms of dementia, aiding in planning for end-of-life care, aiding in the differential diagnosis of dementia and mild cognitive impairment (MCI), and screening at-risk populations for disease. (Georgakas, et al. [8]) [52].

Conclusion

- The risk factor for Alzheimer's disease to date is age; The older you are, the more likely you are to develop the disease, but it is not an inevitable part of aging along with genetic and environmental factors.

- Attention must be given to developing biomarkers that can be used to detect these changes in the brains of individuals at risk to facilitate early and accurate diagnosis of successful treatment.

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