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Pharmacotherapy of Alzheimer's Disease

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Abstract

Alzheimer disease is one of most devastating brain disorders mostly occurring in elderly humans. It is neurodegenerative disease involved obtusely deface memory, thinking, behavior and social skills. These changes affect a person's ability to function. Symptoms of Alzheimer's are stage to stage Neuropsychiatric symptoms like apathy, social withdrawal, disinhibition, agitation, psychosis, and wandering are also common in the mid to late stages. The most common symptoms are dementia. the prevalence rate of Alzheimer in worldwide is estimated to be as high as 24 million. Amyloid and beta protein are responsible for this disease. Alzheimer is not diagnosed with single test. Different tests are performed to diagnose this disease like computerized tests, genetic testing, mental ability test, brain imaging, CT scan, PET, MRI. Cholinergic inhibitors are used to treat Alzheimer.

Keywords: Neurodegenerative disease; Neuroinflammation; Brain imaging; Non-pharmacological and pharmacological approaches; Genetic variation

Abbreviations: AD8: About Dementia; FAQ: Questionnaire on Functional Activities; NPI-Q: Questionnaire for the Neuropsychiatric Inventory; FDA: Food and Drug Administration; ANAM: Automated Measures for Neuropsychological Assessment; ADAD: Autosomal Dominant Alzheimer's Disease; CT: Computed Tomography; MRI: Magnetic Resonance Imaging; DMT: Disease Modifying Therapies

Alzheimer

Alzheimer is a neurodegenerative disease in which obtusely deface memory, cerebration, deliberations, intersectional skills. These variations affect a person's skill at work, and it will over time mostly it occurs in old age. Alzheimer's disease causes the brain to diminish and brain cells die. Alzheimer's disease is the most common cause of dementia.

Stages of Alzheimer's

Following are the stages of dementia.

a) Non dementia stage.

b) Memory loss.

c) Less cognitive disability.

d) Moderating cognitive decline lenient dementia.

e) Moderating severe cognitive decline moderating dementia.

f) Severe cognitive decline moderating severe dementia.

g) Very severe cognitive decline severe dementia.

Types of Alzheimer

There are two types of Alzheimer based on onset of action.

i) Early onset alzheimer

ii) Late onset alzheimer

Early onset alzheimer: Symptoms visible between a person 30's and mid-60s. It occurs due to the gene changes from parent to baby.

Late onset alzheimer: The symptoms appear after the 60 years older. It may or may not be transferred in families (Figure 1).

Etiology

Normally Alzheimer result in the accumulation of two type of protein

i) Amyloid protein

ii) Tau protein

Accumulation of amyloid protein form plaque. When plaques appear result in toxic effect in neurons and damage the

transmission between another Protein called tau protein normally attach to the microtubules and stabilize neuronal microtubules but due to some chemical changes occurred in tau protein it does not attach with microtubule and form tangles. These tangles block the synaptic communication between neurons.

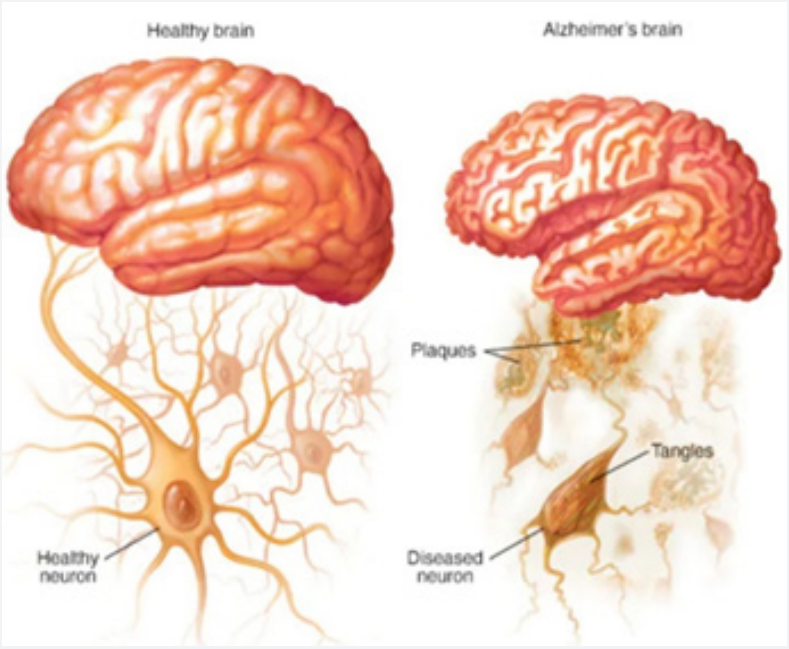

Figure 1: Types of alzheimer.

Epidemiology

In 2018, Alzheimer's Disease International estimated the number of people living with dementia worldwide to be approximately 50 million people, expected to triple by 2050, with two-thirds living in low- and middle-income countries It is expected [7] The latest data shows that the prevalence of dementia in Europe is predicted to double by 2050 [5] Although increasing evidence suggests that the incidence of dementia is decreasing in high-income countries [8], the evidence for a decline in prevalence is less convincing [6].

Incidence and prevalence of alzheimer's disease projected to the year 2000 to 2050

The incidence and prevalence of AD rise with increasing age and are higher in women in part because of their increased longevity. The incidence of AD ranges from 1% at ages 65 to 70 to approximately 4% over age 85. In the United States, the number of new cases per year is expected to triple from ap- proximately 420,000 in 2000 to more than 1.3 million in 2050 [8]. Estimates of prevalence of AD range from the lowest figure of 3% of the population at 65 years to the highest reported estimate of 47% of people over age 85. The prevalence of AD in the United States in 2000 was estimated to be 4.5 million. By 2050, this number will increase by almost threefold, to 13.2 million. In the United States, AD currently is the eighth leading cause of death, with approximately 63,000 deaths per year and a death rate of 21.8 deaths per 100,000 population. The death rate of AD is increasing by approximately 6% per year. The median survival from initial diagnosis recently was estimated to be 4.2 years for men and 5.7 years for women [12].

Pathophysiology

Basic scientists refer to the preclinical stage of Alzheimer's disease as the cellular stage. Changes in neurons, microglia, and astroglia drive the insidious progression of the disease before any cognitive impairment is observed [9]. Neuroinflammation, vascular changes, aging, and glymphatic system dysfunction [10] act upstream or in parallel to amyloid-β accumulation in the context of this cellular disease Amyloid-β induces the spread of tau pathology in an unknown manner. This is associated with the appearance of necrotic markers in neurons exhibiting granulovacuolar degeneration [11]. Two pathological features of Alzheimer's disease are:

i) Extracellular beta-amyloid deposits (in neurite plaques)

ii) Intracellular neurofibrillary bands (paired helical filaments)

Beta-amyloid deposition and neurofibrillary tangles cause loss of synapses and neurons, causing extensive atrophy of affected brain areas, usually beginning in the mesial temporal

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lobe. The mechanism by which beta-amyloid peptide and neurofibrillary tangles cause such damage is not fully understood. There are several theories. The amyloid hypothesis postulates that progressive accumulation of beta-amyloid in the brain initiates a complex cascade of events that culminates in neuronal death, loss of neuronal synapses, and progressive neurotransmitter deficiency; all these effects contribute to the clinical symptoms of dementia. A persistent immune response and inflammation has been observed in the brains of Alzheimer's patients. Some experts have suggested that inflammation is the third major pathological feature of Alzheimer's disease [13]. It has been shown that disturbances in glucose metabolism play a potentially important role in the development of Alzheimer's disease and #039 [14]. Prion mechanisms have been identified in Alzheimer's disease. In prion diseases, a normal cell surface brain protein called prion protein is misfolded into a pathogenic form called a prion. The prion then causes other prion proteins to misfold in the same way, resulting in a significant increase in abnormal proteins that cause brain damage. In Alzheimer's disease, beta-amyloid in brain amyloid deposits and tau in neurofibrillary tangles are thought to have prion-like self-propagation properties.

Diagnosis of Alzheimer's Disease

A person cannot be diagnosed with Alzheimer's disease with a single test. To accurately diagnose patients, doctors use a variety of diagnostic methods in addition to the patient's medical history and other data. These tools include neurological exams, cognitive and functional assessments, brain imaging (MRI, CT, PET), cerebrospinal fluid testing, and blood tests.

Physical exam and diagnostic test

i) A medical professional will probably enquire about nutrition, food, and alcohol consumption.

ii) Examine every drug. (Bring a list of all the medications you now use, including over-the-counter medications and supplements, as well as their containers.)

iii) Verify your temperature, pulse, and blood pressure.

iv) Pay attention to the lungs and heart.

v) Carry out additional procedures to evaluate general health.

vi) Gather samples of blood or urine for analysis in a lab.

Neurological exam: The doctor will attentively monitor the patient throughout a neurological exam to look for issues that could indicate brain illnesses other than Alzheimer's. The physician will search for indications of brain tumours, Parkinson's disease, stroke, accumulation of fluid in the brain, and other disorders that could cause problems with thinking or memory. The doctor is going to test Reflexes. muscle tone, strength, and coordination. Eye motion. Speaking.

Mental ability test: Typically, a professional would use tests called cognitive assessments to evaluate your mental capacities, such as memory or reasoning. Most cognitive evaluations consist of multiple-choice, paper-based tests and questions with scoring. These examinations evaluate several mental skills, such as:

i) Memory, both short- and long-term.

ii) Focus and attention span language and communication abilities temporal and spatial awareness (orientation).

iii) Vision-related skills (visuospatial skills).

iv) It's critical to keep in mind that an individual's educational background may have an impact on their exam results.

v) A person with poor reading or writing skills, for instance, can score lower even if they do not necessarily have Alzheimer's disease.

vi) In a similar vein, a person with more schooling may score better. but dementia persists.

vii) As a result, these tests can aid medical professionals in diagnosing dementia; however, they should never be used in place of a proper diagnosis.

Cognitive, functional and behavioral tests: Test in cognition, function, and behaviour include, for example:

i) Find Out About Dementia 8 (AD8).

ii) Questionnaire on Functional Activities (FAQ).

iii) Small-Cog.

iv) MMSE, or Mini-Mental State Exam.

v) The MoCA, or Montreal Cognitive Assessment.

vi) Questionnaire for the Neuropsychiatric Inventory (NPI-Q).

Computerized cognitive tests and devices: Longer traditional tests may not be feasible in clinical settings or randomised clinical trials. Digital computerised assessments, on the other hand, are intended to quantify an individual's performance on a range of cognitive or functional activities. Several digital cognitive assessment products have been approved for commercialization by the US Food and Drug Administration (FDA):

a) Automated Measures for Neuropsychological Assessment (ANAM).

b) Automated Battery for the Cambridge Neuropsychological Test (CANTAB Mobile®)

c) Think ICA.

d) Think gram.

e) Cognition

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Genetics test

Genes associated with risk: The biggest risk gene for Alzheimer's, APOE-e4, can be tested for blood, however this test is mostly used in clinical trials to identify participants who are more likely to develop the disease. Being a carrier of this gene mutation does not guarantee the development of Alzheimer's disease, nor does it determine an individual's current state of Alzheimer's. The decision to pursue APOE-e4 genetic testing is debatable and should only be made after consulting a doctor or genetic counsellor. Deterministic genes: Autosomal dominant Alzheimer's disease (ADAD), commonly known as "familial Alzheimer's," is a rare form of Alzheimer's disease that affects 1% or fewer of cases. Testing for these genes is also possible. A person with ADAD may start having symptoms as early as their 30s and is more likely to run strongly in families. Although many members of these families do not want to know their genetic makeup, some get tested to find out if they are predisposed to the illness. A few ADAD families have participated in clinical trials to further our understanding of Alzheimer's.

Brain imaging

Computed tomography (CT) or magnetic resonance imaging (MRI) with structural imaging is frequently used in the usual medical workup for Alzheimer's disease. The main purpose of these tests is to rule out other illnesses that can generate symptoms comparable to Alzheimer's but need a different course of care [15].

Causes of Alzheimer's Disease

Age-related alterations in the brain, in addition to genetic, environmental, and lifestyle variables, are most likely the reasons. Depending on the individual, each of these characteristics may have varying effects on the likelihood of getting Alzheimer's disease [16].

Age-related causing

Age-related alterations in the brain have the potential to destroy neurones and other brain cell types, which could exacerbate the effects of Alzheimer's. Atrophy (shrinking) of specific brain regions, inflammation, blood vessel damage, the generation of unstable chemicals known as free radicals, and mitochondrial malfunction are some of these age-related alterations.

Genetic causing

Most of the time, there is more than one hereditary component to Alzheimer's. Rather, it is probably controlled by several genes together with environmental and lifestyle variables. Genetic variants are alterations in the genes that can either raise or lower a person's risk of contracting the illness. As of right moment, researchers have identified around 70 genetic areas linked to Alzheimer's disease. Only three of the genetic variations linked to Alzheimer's thus far have been shown to be the disease's cause. An individual who inherits a mutation in one of these genes (APP, PSEN1, or PSEN2) is likely to develop Alzheimer's disease before the age of 65, and in some cases, much sooner, even though this is an uncommon occurrence. Individuals who have Down syndrome are also more likely to experience Alzheimer's disease early in life. The APP gene, which generates the amyloid precursor protein, is located on an extra copy of chromosome 21, which causes Down syndrome. An excess of this protein causes beta-amyloid plaques to accumulate in the brain. Alzheimer's disease is predicted to strike at least 50% of those with Down syndrome, with symptoms starting to show in their 50s and 60s.There is a further genetic variation in the APOE gene that influences the risk of Alzheimer's disease. This gene has many variants. More specifically, for some groups, having APOE ε4 raises the risk of Alzheimer's and causes the disease to manifest earlier in life. A possible defence against Alzheimer's disease could be APOE ε2 [17].

Health, Environmental and Lifestyle Factors

Studies indicate that a multitude of variables other than heredity could contribute to the onset and progression of Alzheimer's disease. For instance, there is a lot of interest in the connection between metabolic diseases like diabetes and obesity and vascular disorders like high blood pressure, heart disease, and stroke, as well as cognitive loss. We will learn more about if and how lowering risk factors for these illnesses may also lower the chance of Alzheimer's disease through ongoing study. Maintaining one's health as one ages can be facilitated by a balanced diet, exercise, social interaction, and mentally challenging activities. These elements may also lessen the chance of Alzheimer's and cognitive decline. Clinical studies are being used by researchers to examine some of these theories [18].

Risk Factors of Alzheimer's Disease

i) **Age:** Increasing age is the greatest known risk factor for Alzheimer's disease [19]. Alzheimer's isn't a part of typical aging. But as you grow older, the chances of developing it increase [19]. Alzheimer's disease doubles every five years after you reach the age of 65 [20]. Younger people can also get it. Around 1 in 3 people with younger onset Dementia have Alzheimer [21].

ii) **Family history:** People who have parents or siblings with Alzheimer are more likely to get it themselves [22]. But in few families Alzheimer disease is caused by inheritance of single genes and risk of the condition being passed are much higher [20].

iii) **Genetics:** Four genes are currently known to be involved in developing Alzheimer disease. Presenilin on chromosome 14, Presenilin 2 on chromosome 1, the amyloid, B-protein precursor on chromosome 21, and Apolipoprotein E gene on chromosome 19 [23]. APOE e4 is a form of the gene that increases risk of Alzheimer disease [19]. There are two types of genes 'familial' genes and risk genes [21]. Risk genes are more common than familial genes [21].

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iv) **Down Syndrome:** Many people with Down syndrome develop Alzheimer disease [20]. Because of genetics changes Down syndrome can cause [21]. Amyloid plaques build in the brain which leads to Alzheimer disease in most people [20]. The chromosome 21 gene have an extra copy in Down syndrome disease [22,23].

v) **Head Trauma:** People who have traumatic brain injury (TBI) above 50 years old have increased risk of dementia and Alzheimer disease [19]. Head injuries are the most common cause by cars, motorcycles, bicycle accidents, military exposures, firearms and sports [24].

vi) **Mild cognitive impairment:** People who have mild cognitive impairment have decline thinking skills and memory [19]. People who have MCI increase significant risk of dementia. When MCI affects mainly memory the condition is worse due to Alzheimer disease [19].

vii) **Excessive alcohol consumption:** Drinking large amounts of alcohol will cause brain changes [19]. According to several studies, alcohol misuse of more than 21 units weekly increased the risk of dementia [24]. There is a causal relationship between harmful use of alcohol, other mental and behavioral disorders, and non-communicable disease as well as injuries [24].

viii) **Smoking:** Smoking increases the risk of dementia and Alzheimer disease [24]. Smoke increases oxidative stress, generation of free radical [25]. It promotes inflammatory action in immune system leading to activation of phagocytes and oxidative disease [26]. Smoking may lead to cerebrovascular disease which increases the risk of Alzheimer disease.

ix) **Hypertension:** Hypertension in midlife increases the risk of Alzheimer disease [24]. Hypertension can cause changes in vascular walls which can lead to cerebral hypoxia, ischemia hypoxia and hypoperfusion increase development of AD [25]. Cerebral ischemia is capable of leading to the accumulation of APP and AB [26]. Hypertenstion may lead to dysfunction in blood brain barrier [25].

x) **Type 2 diabetes:** Type 2 diabetes is a clear risk of development of future Alzheimer [24]. Several mechanisms for this, including insulin resistance impaired insulin receptor, insulin deficiency, toxicity of hyperglycemia [25].

xi) **Depression:** Depression is very common in the people who have Alzheimer dementia [27]. 40 percent of people with Alzheimer disease suffer significant depression [28]. Depression may be the reaction of early cognitive disease [28].

Treatment of Alzheimer's Disease

i) Symptomatic and disease modifying therapies are the two important pharmacotherapeutic approaches to Alzheimer's Disease. Symptomatic treatment shows a significant effect not only on cognition but also in the symptoms including psychosis, agitation and sleep disturbance. On the other hand, the disease modifying therapies (DMT) mainly focused on the interventions that are based on the amyloid cascade hypothesis and Tau biology [29].

ii) Disease modifying treatments: While symptomatic treatments have proven helpful, it is the finding of a cure that is most vital. Since the amyloid hypo- thesis indicates that Aß generation and deposition from overexpressed APP cleavage make up the fundamental basis of Alzheimer's disease, interest centers on anti-amyloid therapies. These therapies result in decreased production of Aß, increased clearance of Aß and the prevention of Aß aggregation into amyloid plaques. Immunotherapy has also been an area of interest as it targets the clearing of Aß peptides, which can either directly or indirectly impact cognitive decline. Focusing on decreasing Aß generation, several methods can be employed to achieve this, mainly by targeting the amyloidogenic and nonamyloidogenic pathways. ß and secretases both compete for APP, with ß- and ϒ-secretase processing ultimately resulting in amyloid deposition and ϒ-secretase generating soluble APPSC. 2Inhibiting ß- and ϒ-secretases while simultaneously potentiating ϒ-secretase action would thus reduce Aß generation and deposition overall [30].

iii) Approved Drug treatments

iv) Cholinesterase inhibitors: The first-generation cholinesterase inhibitor was Tacrine, but its use was limited by hepatotoxic side effects. Donepezil, rivastigmine and galantamine then followed, with the former probably the most widely used agent. Efficacy appears similar between these different agents so choice should be based on cost, individual patient tolerance and physician experience. Donepezil is prescribed at an initial dose of 5 mg in the evening, increased to 10 mg after one month if appropriate. Response is gauged by a rating of better memory, function or behaviour by the patient or carer: there is no point in trying to measure change with brief mental status schedules such as the mini mental state examination, as these are not designed to detect clinically relevant change. If there is no response after three months of treatment it is reasonable to consider stopping the medicine at that stage although opinions around this can differ. Common side effects are gastrointestinal, fatigue and muscle cramps, and all patients should have an electrocardiogram prior to commencing cholinesterase inhibitor because of the risk of sick sinus syndrome and other conduction abnormalities. Care should be taken if considering commencing a cholinesterase inhibitor in a person with a history of peptic or duodenal ulcer disease. Small numbers of patients may exhibit an acute worsening of cognition or agitation on starting; in which case, the medicine should be stopped immediately. Average effects on cognition and function are generally modest and response rates are variable, with around one third of patients showing no benefit and a smaller proportion (around one-fifth) showing larger benefit. It is expected that about

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one-third of patients may not tolerate cholinesterase inhibitor because of side effects [31].

v) Memantine: Memantine uncompetitively blocks the NMDA receptor and may be neuroprotective by preventing neuron loss as well as improving symptoms by helping to restore function of damaged neurons [32].

Conclusion

The past 5 years have seen significant progress in understanding the pathophysiology and genetic basis of Alzheimer's disease The amyloid-β cascade hypothesis has been revised by a more thorough understanding of the cells in the preclinical stages of Alzheimer's disease Genetic research has evolved from identifying three causative genes and one risk gene to identifying a variety of genes that may be included in a polygenic risk score for Alzheimer's disease Advances in biomarker diagnostics have completely reconsidered the external and pre-clinical labeling of Alzheimer's disease, especially now that blood biomarkers are available and seem within reach, patients can now be enrolled in studies much earlier in the disease Molecular imaging further refines the diagnostic classification and pathological basis of diseases, allowing visualization of co-pathology and local protein aggregation These developments have been followed by insights in the areas of risk reduction, primary and secondary prevention, non-pharmacological and pharmacological approaches, which can ultimately be tested in parallel at much earlier points than previously possible It will be done If the industry maintains this pace, very early identification and multidisciplinary treatment of patients could become a reality.

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