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A REVIEW OF DIAGNOSTIC AND TREATMENT OF ALZHEIMER'S DISEASE

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ABSTRACT

***Alzheimer's disease** (AD) is a neurodegenerative disease that usually starts slowly and progressively worsens. It is the cause of 60–70% of cases of dementia. The most common early symptom is difficulty in remembering recent events. As the disease advances, symptoms can include problems with language, disorientation (including easily getting lost), mood swings, loss of motivation, self-neglect, and behavioral issues. As a person's condition declines, they often withdraw from family and society. Gradually, bodily functions are lost, ultimately leading to death. Although the speed of progression can vary, the typical life expectancy following diagnosis is three to nine years. The disease is named after German psychiatrist and pathologist Alois Alzheimer, who first described it in 1906. Alzheimer's financial burden on society is large, with an estimated global annual cost of US$1 trillion. Alzheimer's disease is currently ranked as the seventh leading cause of death in the United States. No treatments stop or reverse its progression, though some may temporarily improve symptoms More than 6 million Americans, many of them age 65 and older, are estimated to have Alzheimer's disease. That's more individuals living with Alzheimer's disease than the population of a large American city. Many more people experience Alzheimer's in their lives as family members and friends of those with the disease.*

The symptoms of Alzheimer's disease — changes in thinking, remembering, reasoning, and behavior — are known as dementia. That's why Alzheimer's is sometimes referred to as "dementia." Other diseases and conditions can also cause dementia, with Alzheimer's being the most common cause of dementia in older adults.

Alzheimer's disease is not a normal part of aging. It's the result of complex changes in the brain that start years before symptoms appear and lead to the loss of brain cells and their connections. The course of Alzheimer's is generally described in three stages, with a progressive pattern of cognitive and functional impairment. The three stages are described as early or mild, middle or moderate, and late or severe. The disease is known to target the hippocampus which is associated with memory, and this is responsible for the first symptoms of memory impairment. As the disease progresses so does the degree of memory impairment.

KEYWORDS: *Alzeimer's, Diagnosis, Treatment, Genetics, Neurological*

INTRODUCTION

Alzheimer's disease is a brain disorder that slowly destroys memory and thinking skills and, eventually, the ability to carry out the simplest tasks. In most people with the disease — those with the late-onset type symptoms first appear in their mid-60s. Early-onset Alzheimer's occurs between a person's 30s and mid-60s and is very rare. Alzheimer's disease is the most common cause of dementia among older adults. [1]

In 1906, Dr. Alzheimer noticed changes in the brain tissue of a woman who had died of an unusual mental illness. Her symptoms included memory loss, language problems, and unpredictable behavior. After she died, he examined her brain and found many abnormal clumps (now called amyloid plaques) and tangled bundles of fibers (now called neurofibrillary, or tau, tangles).

These plaques and tangles in the brain are still considered some of the main features of Alzheimer's disease. Another feature is the loss of connections between nerve cells (neurons) in the brain. Neurons transmit messages between different parts of the brain, and from the brain to muscles and organs in the body. Many other complex brain changes are thought to play a role in Alzheimer's, too. This damage initially takes place in parts of the brain involved in memory, including the entorhinal cortex and hippocampus. It later affects areas in the cerebral cortex, such as those responsible for language, reasoning, and social behavior. Eventually, many other areas of the brain are damaged.

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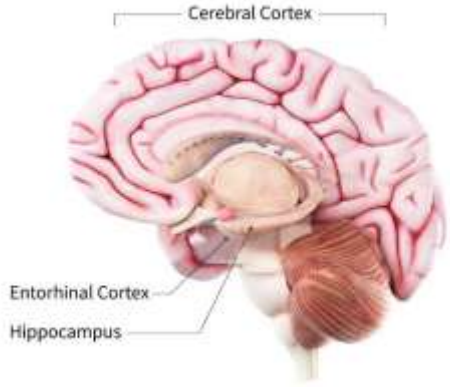

How many Americans have Alzheimer's disease?

Estimates vary, but experts suggest that more than 6 million Americans age 65 and older may have Alzheimer's. Many more under age 65 also have the disease. Unless Alzheimer's can be effectively treated or prevented, the number of people with it will increase significantly if current population trends continue. This is because increasing age is the most important known risk factor for Alzheimer's disease.

What does Alzheimer's disease look like?

Memory problems are typically one of the first signs of Alzheimer's, though initial symptoms may vary from person to person. A decline in other aspects of thinking, such as finding the right words, vision/spatial issues, and impaired reasoning or judgment, may also signal the very early stages of Alzheimer's disease. Mild cognitive impairment (MCI) is a condition that can be an early sign of Alzheimer's, but not everyone with MCI will develop the disease.

People with Alzheimer's have trouble doing everyday things like driving a car, cooking a meal, or paying bills. They may ask the same questions over and over, get lost easily, lose things or put them in odd places, and find even simple things confusing. As the disease progresses, some people become worried, angry, or violent. [2]

Middle-stage and later symptoms include disorientation, confusion, behavioral changes and problems with speech or language [5,8,9]. These symptoms also have a neurobiological basis and can be monitored based on the assessment of biological substances reflecting pathological changes in human fluids decades before disease onset [3,12]. It ispostulated that, in addition to obtaining the patient's medical history, several tests should be performed to assess decline of cognitive function related to AD, including neuropsychological tests, neuroimaging tests and assessment of biochemical markers [3,5]. CSF biomarkers are widely discussed in working groups and included in international guidelines for clinical practice [4,5,7,10,13,14]. Clinicians may encounter a number of challenges in diagnosing AD [12] due to mixed pathologies related to cerebrovascular disease or Lewy body dementia (LBD). Furthermore, the diagnostic process may be complicated because of the use of different diagnostic techniques or presence of other, pre-analytical factors [8,11,12,15–16].

DIAGNOSIS

An important part of diagnosing Alzheimer's disease includes being able to explain your symptoms. Input from a close family member or friend about your symptoms and their impact on your daily life helps. Tests of memory and thinking skills also help diagnose Alzheimer's disease.

Blood and imaging tests can rule out other potential causes of the symptoms. Or they may help your health care provider better identify the disease causing dementia symptoms.

In the past, Alzheimer's disease was diagnosed for certain only after death when looking at the brain with a microscope revealed plaques and tangles. Health care providers and researchers are now able to diagnose Alzheimer's disease during life with more certainty. Biomarkers can detect the presence of plaques and tangles. Biomarker tests include specific types of PET scans and tests that measure amyloid and tau proteins in the fluid part of blood and cerebral spinal fluid.

Tests

Diagnosing Alzheimer's disease would likely include the following tests:

Physical and Neurological Exam

A health care provider will perform a physical exam. A neurological exam may include testing:

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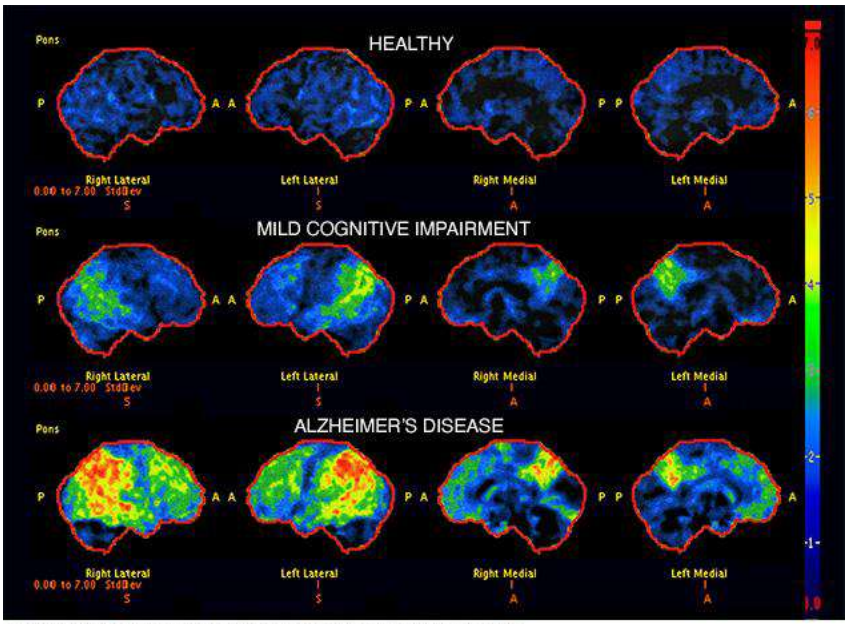
- Reflexes.
- Muscle tone and strength.
- Ability to get up from a chair and walk across the room.
- Sense of sight and hearing.
- Coordination.
- Balance.

Lab Tests
Blood tests may help rule out other potential causes of memory loss and confusion, such as a thyroid disorder or vitamin levels that are too low. Blood tests also can measure levels of beta-amyloid protein and tau protein, but these tests aren't widely available and coverage may be limited.

Mental status and Neuropsychological Testing
Your provider may give you a brief mental status test to assess memory and other thinking skills. Longer forms of this type of test may provide more details about mental function that can be compared with people of a similar age and education level. These tests can help establish a diagnosis and serve as a starting point to track symptoms in the future.
Brain imaging



Brain scan images for diagnosis of Alzheimer's disease Open pop-up dialog box
Images of the brain are typically used to pinpoint visible changes related to conditions other than Alzheimer's disease that may cause similar symptoms, such as strokes, trauma or tumors. New imaging techniques may help detect specific brain changes caused by Alzheimer's, but they're used mainly in major medical centers or in clinical trials.
Imaging of brain structures include:
Magnetic resonance imaging (MRI). MRI uses radio waves and a strong magnetic field to produce detailed images of the brain. While they may show shrinkage of some brain regions associated with Alzheimer's disease, MRI scans also rule out other conditions. An MRI is generally preferred to a CT scan to evaluate dementia.

Computerized tomography (CT). A CT scan, a specialized X-ray technology, produces cross-sectional images of your brain. It's usually used to rule out tumors, strokes and head injuries.

Positron emission tomography (PET) can capture images of the disease process. During a PET scan, a low-level radioactive tracer is injected into the blood to reveal a particular feature in the brain. PET imaging may include:
Fluorodeoxyglucose (FDG) PET imaging scans show areas of the brain in which nutrients are poorly metabolized. Finding patterns in the areas of low metabolism can help distinguish between Alzheimer's disease and other types of dementia.

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Amyloid PET imaging can measure the burden of amyloid deposits in the brain. This test is mainly used in research but may be used if a person has unusual or very early onset of dementia symptoms.

Tau PET imaging, which measures the tangles in the brain, is generally used in the research setting. [17]

ALZHEIMER'S DISEASE TREATMENT

The early treatment will improve the quality of life of the patient. The pharmacological treatment in early stages of the disease is useful to improve the cognitive disorders, to slow down the advance of the deficit and to diminish the psychiatric symptoms such as agitation, depression and psychosis. - Non Pharmacological Treatment Non-pharmacological treatments consist of the care of the patient through the implementation of psychotherapeutic and stimulation interviews. The adaptation of the context, the work with the family, and persons in charge are very important. Psycho-education is important as it can produce some behavioral changes, and it can diminish the need of a symptomatic treatment in the same way that an organized routine can be established, the avoiding of isolation, the cognitive stimulus when it is possible and the affective contention.

A) Cholinesterase Inhibitors

This pharmacological group is the most studied for the treatment of AD. The cholinesterase inhibitors improve the cholinergic transmission inhibiting the enzyme in a reversible, pseudo reversible or irreversible way. They have a moderate effect in patients at early stage of the disease, and its long-term use delays the advance of the cognitive deterioration [19-24]. The favorable answer predictor would be the following: - slight to moderate stage of the disease - late onset - absence of Apo E, allele 4 [25]. The cholinesterase inhibitor medications are: taurine, rivastigmine, donepezil and galantamine. They must be used with caution in patients with bradycardia, heart block, peptic ulcer or asthma. Due to the fact that a cholinergic deficit is related to the appearance of the psychiatric symptoms of AD, there are studies that support the hypothesis that administrating cholinesterase inhibitors symptoms such as apathy, psychosis, disinhibition, agitation could be improved regulating the cholinergic transmission in the frontal, temporal, and orbitofrontal areas. The nucleus basalis has projections to the cerebral cortex, the limbic and paralimbic system would be like a station between the emotional and the cognile inhibitor of the cholinesterase. Eight studies controlled with placebo have shown effectiveness since 1981. Some of them show significant improvement [26-30]. Tacrine is metabolized in liver (in cytochrome P450 of the system it has a half-life of 2-4 hours. The most important side effects of tacrine are nausea, vomits, stomachache, anorexia, bradicardia, mialgias, ataxia and the increase of hepatic enzymes especially the transaminase glutamic-oxalacetic in 40 % of the cases already studied. It cannot be used in patients with hepatic deficiency. The increase of the hepatic enzymes is produced during the first 12 weeks of treatment and it is more frequent in women. If we stop the treatment the enzymes go back to normal levels within 4 or 6 weeks [31]. There must be weekly controls of the enzymes in the first 6 weeks, then monthly during, 2 month and then every 3 months.

Galantamine is the newest reversible inhibitor of the cholinesterase that has an allosteric modulatory action on the nicotine receptors. The presynaptic activation of the latter increases the release of acetylcholine, glutamate, monoamines and GABA. So, it has a dual action: it has proved effectiveness over the cognitive symptoms and the psychiatric symptoms, of AD [32-33]. It has a half-life of 7 hours. It is metabolized by cytochrome P450 and glucuronidation, and also excreted in urine without changes. Two double-blind studies (1,500 patients with Alzheimer's slight to moderate level of the disease during 6 months) have proved effectiveness on the cognitive symptoms and the global functioning with doses of 16 to 32 mg daily [34,35]. The negative side effects are: nausea, vomits, anorexia and shaking. The drug is well tolerated when the dose is increased gradually. Other inhibitors of the cholinesterase that are under research are eptastigmine, huperzine and velnacri.

B) Neuroprotectors/Neuronal Metabolism

Estrogen It has been observed that women are more likely to suffer from AD and to have greater deficits. Women who have taken estrogen after menopause had reduced AD risk, or a later onset of the disease than women without estrogen replacement [36-42]. Estrogen is thought to have a protective effect against AD through the following mechanisms: Cholinergic neurons activation Antioxidant effect Diminution of plasmatic levels of APOE Increase in the use of glucose by neurons Promotion of neuronal survival In-vitro estrogen breaks the APP in more soluble fragments than the Beta A4 [43]. The use of estrogen is now controversial. On one side, in different studies the effectiveness of the administration of estrogen in monotherapy or associated to the cholinesterase inhibitors has been observed. [44-46]. On the other side, recent studies have demonstrated no effectiveness with a higher number of deep vein thrombosis than in the control group [47, 48]. It is important to consider the risks/benefits of estrogen therapy. Basically, the risks are breast cancer, endometria's cancer and deep vein thrombosis, and the benefits are over the memory, and over cardiovascular diseases.

C) Anti-inflammatory Drugs

The incidence of AD is significantly minor in patients treated with anti-inflammatory drugs in the same way as the cognitive deficit [49-53]. There are some clinical studies carried out in the last years with ibuprofen, indomethacin, aspirin and prednisone that have proved beneficial effects [54-56]. Other drugs that are still being studied are the colchicines, the hydroxichloroquine and the

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methotrexate. With the prednisone and colchicines a decrease of the cognitive decline has been observed. A new study with the hydroxichloroquine didn't show a delay in the progression of the disease [57]

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