Alzheimer's
Disease

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Introduction to Alzheimer's

Alzheimer's disease is a progressive degenerative disease of the brain. It is first described by the German neuropathologist Alois Alzheimer (1864-1915) in 1905. This disease worsens with advancing age, although there is no evidence that it is cause by the aging process.

The average life expectancy of a person with the disease is between five and ten years, but some patients today can live up to 15 years due to improvements in care and medical treatments. The cause of Alzheimer's has not been discovered yet and it cannot be possible to confirm a person has Alzheimer's until their autopsy following death.

How does Alzheimer's develop

What causes Alzheimer's? Well no one know exactly the development
of this debilitating disease. But recent advances has produced several clues as to how it is born. Initially when we study the brain of a Alzheimer's victim, we focus on two specific areas. One is the cortex of the frontal and cerebral lobes1. The second is the hippocampus (meaning seahorses in Greek which it resembles2) which is located below the cerebral cortex and responsible for short-term memory. If we study samples of these two section, we would find three irregularities which are not found in normal brain matter. These three are called neurofibrillary tangles, neuritic plagues and granulovacuolar degeneration3.

A nerve cell has numerous axons and dendrites coming out of it. A neurofibrillary tangle is when the neuron changes. A number of dendrites are missing and the nucleus is filled with protein filaments resembling steel wool4.
Although all elderly people has a few of these helix shaped bundles in their brain for they are normal indicators of aging, Alzheimer's patients has more than usual. Their presence usually in the frontal and temporal lobes is a indication of AD.

Senile neuritic plagues are small round objects. They are masses of amyloid protein material composed of residue left over from healthy nerve endings that were broken off and decayed. Their presence near the cell further indicates something gone wrong. Neuritic plaques is the best evidence for diagnostics to make the determination of AD.

A third sign of neuron deterioration is granulovacuolar degeneration. This is when fluid-filled vacuoles are seen crowding inside the nerve cell, specifically in the triangular shaped cells of the hippocampus. This condition can only be observed in carefully sliced, stain and analyzed brain tissue.

The cell having lost all it's dendrites and nucleus soon disintegrates entirely, vanishing into the body's waste disposal system. With the depletion of enough nerve material the brain actually shrinks, sometimes by as much as ten percent5. The more cells the AD sufferer loses, the more mental functions he loses. Soon the person will have limited motor skills. People who were once witty and quick on their feet were reduced to the

mental status of small children.

Diagnosis of Alzheimer's

How would you now if a person you knew has Alzheimer's? There are certain telltale signs that point to it. There was one patient6 that was convinced she was suffering from AD. As proof of her condition, she bought the a meeting several recent newspaper clippings, which she began to quote from memory. Obviously this person did not have the disease, she wouldn't have memorized complex and lengthy information. But forgetting on a regular basis doesn't indicate Alzheimer's either.

Stages of AD

In the initial stage, there is no clear evidence of memory trouble and deterioration in brain functions. The individual performs well on exams that test mental abilities (psychometric tests7) similar to those given to measure IQ.

In the second stage, the patient shows very mild memory problems with difficulty in remembering names of friends. The changes at this point is still very small. Occasionally, the patient might make a surprising statement such as inquiring about the health of a friend who everyone knows, died years ago. Only extensive psychometric testing can determine if the person's mental ability changed. A close family member like a husband or wife might suspect something is wrong.

By the third stage, there is definite evidence of memory loss, which might interfere with job performance, The person might have difficulty competing a job that use to be routine. The person may avoid social situations because he or she realizes there's a problem

In stage four there is clinical evidence of memory impairment when the mental status is tested by doctors. The disease has now become obvious to the family. A sign of this stage is when the patient keeps asking the same question which has already been answered, this make daily companionship difficult because his friends and family are frustrated.

By stage 5, the patient show problems with both recent and past memories, they even forget events that are important like Christmas, birthdays, friendships and interests. Judgment is failing, the individual is no longer able to select clothes for a particular weather of season and cannot match items by color. Eventually, the victim of AD may leave the water running, the stove on, or the front door open. At this point wandering becomes a major problem.

In stage six, understanding of languages diminishes and simple commands aren't understood. Victims may go back to their first language if they have one. Eventually languages disappear entirely.

In stage seven or the terminal stage, the victim becomes bedridden and totally dependent for all functions. He cannot speak coherently and can't eat unassisted. Death usually occurs at this stage form aspiration pneumonia8, pneumonia caused by breathing in food or other objects because the victim doesn't remember how to swallow food safely, or from urinary infections.

Recent Research on Alzheimer's

Some progress has been made in understanding the nature of the Alzheimer's disease. Scientists has recently found medicine that can slow down the progress of AD. The average survival period from the time of diagnosis to death in 1985 is 10 years. Today the rate has increase a third to 15 years9.

A recent media release stated the discovery of a mutant gene called "triplet repeat" disease genes10. These genes produce proteins that may

block from properly functioning key enzymes that are important to the production of energy in the brain. This gene was found in several diseases like AD, Huntingtons, and Haw River Syndrome as well as three other rare neurological disorders.

Another press release from the Alzheimer's Association is one concerning the new study of an important advance toward early detection of AD11. "Through investigations such as these, in addition to those involving apolipoprotien E (APOE), positron emission tomography (PET), and other approaches, we will improve our ability for accurate detection of individual at risks for the disease"12 said Zaven Khachaturian Ph.D., director of the Alzheimer's Association's Ronald and Nancy Reagan Research Institute.

Among the drugs being tested to treat AD are
-Cholinergic agents: choline, lecithin, and the agonist (RS 86, arecholine, and bethanechol)
-Peptides: vasopressin, ACTH 4-10, naloxone
-Nootropic agents: pramiracetem, CI 911, Praxilene, Oxiratem
-other general drugs: chelating agents, Nimodipine, Vinpocetine13
most of these are still in experimental stages. Some has proved to work slightly but is generally unsafe, others has tested safe but not beneficial, but none has been both. People who offer "cures" to Alzheimer's are either frauds or ignorant14.

When people realizes that AD is a serious disease, perhaps as much as HIV, then maybe they will pay attention. The reason why there hasn't been a cure is because scientists tries to attract grants by working on a problem that people think is serious and controversial. If there was as much attention that was paid to AIDS as there was in AD, then maybe there will be an answer.