



Understanding the Ayurvedic Approach to Alzheimer's Disease

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"...from delusion comes loss of memory; from loss of memory; the ruin of discrimination; and on the ruin of discrimination, the individual perishes".

Bhagavad Gita; Samkhya Yoga; Chapter 2, Sl 62

The concept of memory loss and death due to it was first described in the Bhagavad Gita, however no specific term or name was ascribed to this concept. We now recognize this concept as Dementia and Dementia due to ALZHEIMER'S DISEASE is one of several age associated neurodegenerative diseases characterized by loss of memory.

Introduction:

Alzheimer's disease (AD) is an irreversible, progressive neurodegenerative disease that is characterized by severe memory loss, unusual behavior, personality changes and a decline in thinking abilities. These losses relate to the death of brain cells and the breakdown of the connections between them. Its exact cause is still unknown, but environmental as well as genetic factors are thought to contribute to the disease progression. The disease is the most common form of dementing illness among middle aged and older adults affecting more than 4 million Americans, a number estimated to increase to 7.7 million by 2030. Symptoms typically appear after age 60, with some early-onset forms of the disease linked to a specific genetic defect. People with AD eventually require comprehensive care. Thus, AD presents a considerable problem in patient management, as well. No cure exists for Alzheimer's, and the drugs currently available to treat the disease address only its symptoms, and with limited effectiveness. It is believed that therapeutic intervention that could postpone the onset or progression of AD would dramatically reduce the number of cases over the next 50 years.

Etiology and Pathology:

The disease is characterized by two key abnormalities in the brain: amyloid plaques and neurofibrillary tangles. Amyloid plaques are abnormal clumps of a protein known as beta amyloid. They are found in the tissue between nerve cells in the brain, along with degenerating bits of neurons and other cells. The plaques accumulate to neurotoxic levels compressing the nerve fibers that lie in their path, leading to the production of the brain lesions characteristic of AD. Neurofibrillary tangles, largely comprising a protein called tau, are bundles of twisted filaments found within neurons. In healthy individuals, the tau protein plays a role in the cell's structural support and in the transport of various substances within the cell. In AD, the structure of tau is transformed abnormally and it accumulates as tangles. When this occurs, the normal function of the protein is disrupted resulting in the collapse of the neurons' transport system that eventually impairs communication between nerve cells, and causes them to die. The progression of AD depends on the accumulations of plaques and tangles and the subsequent cerebral

degradation that follows. This destruction of cerebral tissue triggers the behavioral changes associated with Alzheimer's dementia.

Three competing hypotheses exist to explain the cause of the disease. The oldest hypothesis is the "cholinergic hypothesis". It states that Alzheimer's begins as a deficiency in the production of acetylcholine, a vital neurotransmitter. All of the first-generation anti-Alzheimer's medications were based on this hypothesis and work to preserve acetylcholine by interfering with acetylcholinesterases (enzymes that break down acetylcholine) and restoration of the "cholinergic nuclei". Results from these medicines have not been promising and have led to the conclusion that acetylcholine deficiencies may not be causal but are a result of widespread brain tissue damage.

The other two hypotheses are of generally equal acceptance. "Tau-ists" believe that the tau protein abnormalities come first and lead to a full disease cascade. "bA-ptists" believe that beta amyloid deposits are the causative factor in the disease. A third protein, alpha synuclein, which has already been shown to be important in Parkinson's disease, has recently been proposed as the etiological candidate, giving rise to the "syn-ners". Several researchers now believe that AD may be a "triple-protein pathology", wherein interactions among all three lesions give rise to AD. There is compelling evidence that genetic predispositions underlie the development of AD. However, the most obviously genetic cases are also the rarest. Most cases identified are 'sporadic' with no clear family history. It is probable that environmental factors have to interact with a genetic susceptibility to cause development of disease.

Prevention and Cure:

Efforts to find a cure for AD have so far been disappointing. Many studies have indicated that non-steroidal anti-inflammatory drugs (NSAIDs) like ibuprofen and aspirin delay the onset, and lower the ultimate risk, of AD. Prolonged intake of these drugs results in a greater risk of developing fatal stomach ulceration and gastrointestinal bleeding. Several studies have also reported that the combination of vitamins E, C and folic acid might reduce the risk of AD. Other research findings conclude that quitting smoking, weight reduction, and avoidance of diabetes all reduce Alzheimer's risk. Some evidence suggests that Alzheimer's risk may also be reduced by inclusion of fish in the weekly diet. The spice turmeric reduces Alzheimer's incidence in a mouse model and actually dissolves human senile plaques in the test tube. Turmeric is a powerful antioxidant and a powerful anti-inflammatory and presence of turmeric or curry spice in the diet may provide preventive value. Studies are also ongoing to investigate the role of cholesterol-lowering drugs like lovastatin, simvastatin etc. as a means of preventing or delaying Alzheimer's. There seems to be a connection between the cholesterol level inside the brain cells and the deposition of toxic amyloid plaques which make the brain cells die. In addition to lowering cholesterol, the statins may have a beneficial role in reducing inflammation. Some work is being done to investigate the role of raised levels of homocysteine, and possible nutritional prevention or treatment through taking of foods high in B vitamins and antioxidants to control the levels of homocysteine.

Ayurvedic Treatment for Alzheimer's disease:

There is nothing specifically written in the classical Ayurvedic texts regarding AD. However just by looking at the symptoms one can predict that as it is an age associated

neurodegenerative condition (Vata time of life), AD is primarily a Vata disorder. The condition affects memory-function of Udana Vayu. There is a failure to absorb healthy impressions from the 5 senses a condition associated with imbalance in Prana, Samana (afferent impulses) and Vyana Vayu (efferent impulses) thus affecting the thought process. Ayurvedic rasayana (rejuvenative) and vajikarana (to increase ojas and immunity) therapies are recommended for long term care of the elderly with AD. Herbs include Ashwagandha, Kapikachhu, Brahmi, calamus (vacha), Bala, and turmeric. A classical ayurvedic formula for AD like condition is Saraswati Churna. PanchaKarma is recommended depending on the status of the patient. Palliative therapy (Shamana chikitsa) is generally recommended for weaker patients. Nasya preparations are highly recommended, Pranayama and Meditation sessions are also extremely useful.

1) Therapy with B vitamins that lower homocysteine levels: Includes B6, folic acid, and B12 (methylcobalamin) It is thought that high homocysteine levels in the brain causes neuronal damage leading to progression of Alzheimer's disease.

Fenugreek, Licorice root, and sea vegetables such as dulse, kelp, kombu, and nori.

2a) The use of antioxidants, such as vitamin E and Guggul. Vitamin E (tocopherols and tocotrienols) is an efficient free radical scavenger and thus prevents oxidative damage to the fatty tissues that make up the brain.

Carrots, spinach, turnips, broccoli, mustard and other green leafy vegetables, almonds, kiwi, papaya are rich sources of Vit E.

2b) Guggul contains ferulic acids, phenols and other nonphenolic aromatic acids which are potent scavengers of superoxide radicals. Guggul is useful for its cholesterol and lipid lowering effects, anti-inflammatory property and as an antioxidant

3) Providing Acetyl-L-carnitine: Acetyl-L-carnitine protects against amyloid-beta neurotoxicity and improves the cognitive function in subjects with Alzheimer's disease. Acetyl-L-carnitine is believed to block death of nerve cells owing to its antioxidant properties.

Amaranth, fenugreek, ginger, kelp, spirulina

4) Use of curcumin: Curcumin is getting a lot of attention lately. It has potent anti-inflammatory and anti-oxidant activities and can suppress oxidative damage, inflammation, cognitive deficits and amyloid accumulation.

5) Use of Docosahexaenoic acid (DHA): Patients who consumed Docosahexaenoic acid (DHA), a omega-3-fatty acid found in fish oils, had about 40% less Alzheimer's disease and other dementia, compared with people who consumed less DHA. DHA slows down toxic amyloid production and reduces brain plaques seen in AD.

In addition to fish oils herbal sources of DHA include flax seed, spirulina, sunflower and the blue-green algae AFA.

6) Mood improvement through nutrients and herbs: Studies show that all the herbs listed below increase memory, prevent degeneration of vision, hearing, taste and smell. Studies also show that these herbs increase the flow of glucose and ATP (the main energy unit

produced by our metabolism) into all cells, and also stabilize cell membranes including those forming the blood brain barrier. They have been shown to slow the onset of dementia, stimulate the biosynthesis of natural vasodilating agents, destroy free radicals and inhibits lipid peroxidation

Herbs: *Ashwagandha, Brahmi, Gingko, Rosemary oil.*

7) Providing herbs that are anti-inflammatory agents. Many scientists now believe that inflammation may be an important component of the Alzheimer's disease process. The amyloid and protein plaques found in Alzheimer's patient's brains, which are hallmarks of the disease, may be indicative of an inflammatory response. Researchers believe that Non-steroidal anti-inflammatory drugs (NSAIDs) may influence inflammation by interfering with the actions of some proteins and thus lessening their harmful effects. NSAID's have also been shown to produce a lot of side-effects

Ashwagandha, Brahmi, Ginger, Turmeric, Tulsi

Lifestyle changes:

- 1) Pranayama (alternate nostril/anulom/vilom)
- 2) Meditation (so hum; mindfulness)
- 3) Yoga postures that will provide mental stability
- 4) Mindful activities (crossword puzzles, knitting, stitching, gardening etc)
- 5) Building and stabilizing PTO (prana, tejas, ojas)
- 6) Food and dietary changes