Treatment Of ALZHEIMER'S Disease In Ayurveda

There is ray of hope by ayurvedic treatment that either the disease can be prevented or/and few presentations can be revert back by improving cerebral perfusion.

by **Dr. Sunil Kr. Gupta,** MD (Paed. Med.), PhD

Alzheimer's disease (AD) is a senile dementia and is the most common form of dementia. This incurable, degenerative, and terminal disease was first described by German psychiatrist and neuropathologistAlois Alzheimer in 1906. Mostly it is prevalent after 60 years of age. Alzheimer's can also occur at early ages. In 2006 about 26.6 million cases were reported worldwide.

Approximately 10% of all persons over the age of 70 years have significant memory loss, and in more then half the cause is AD. It is estimated that the minimal annual total cost of caring for a single AD patient in an advanced stage of disease is >$50,000 (Harrison's Principals of internal medicine, 18th edition, 2011).

Path physiology

The aetiopathogenesis of Alzheimer's disease are not well understood. Pathologically it is associated with deposition of plaques and tangles in the brain. So far as the treatment is concerned it offers minimal symptomatic relief. Even progression of disease cannot be prevented.

Till date many clinical studies have been conducted for a possible treatment of the disease, but no study have reported promising positive results. Even the preventive strategies suggested so far are of no help. Hence this disease is putting a pressure on social, psychological, physical, and economic elements of the caregiver's life. It is reported that AD is one of the most costly diseases to society in developed countries.

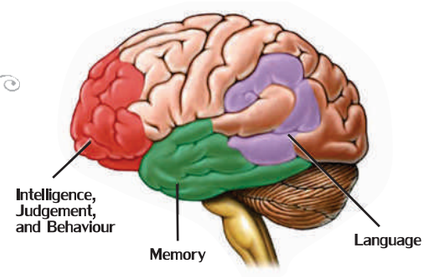
Biochemistry of AD

In Alzheimer's disease there is accumulation of abnormally folded A-beta and tau proteins in the brain. Beta-amyloid (A-beta or Aá) is a transmembrane protein that penetrates through the neuron's membrane. APP (amyloid precursor protein) is critical to neuron growth, survival and post-injury repair. In AD, this APP is divided into smaller fragments by enzymes through proteolysis. One of these fragments gives rise to fibrils of beta-amyloid, which form clumps that deposit outside neurons in dense formations known as senile plaques. In Alzheimer's disease, changes in tau protein lead to the disintegration of microtubules in brain cells. In AD, tau protein gets hyperphosphorylated and begins to pair with other threads, creating neurofibrillary tangles and disintegrating the neuron's transport system.

Cerebral Perfusion in AD

Apart from other Pathophysiological changes in neural tissue in cases of AD that there is definite increasing deficit in cerebral perfusion in cases of AD. Studies reported that SPECT studies on patients with AD showed Regional cerebral perfusion deficits. AD patients showed significant reductions in cortical/cerebellar activity ratio. Cortical perfusion is globally depressed with the largest reductions in frontal and posterior temporo-parietal cortices. Scientists reported that even mildly demented patients, had parietal and temporal perfusion deficits, often unilateral. Moderate to severely demented patients had bilateral temporal and parietal perfusion deficits. Further studies reported that in AD a consistent finding in all subgroups was a significant deficit in temporoparietal blood flow of both hemispheres. Distinct group differences were seen in frontal, central and occipital areas with different combinations of involvement. Apart from this there many more studies reporting cerebral perfusion defects in AD.

Presentation of Problem in AD



The deficits in AZ may be the defects in form of:

- Language
- Memory
- Perception
- Emotional behavior or personality
- Cognitive skills (such as calculation, abstract thinking, or judgment)

The disease usually started with Pre-dementia stag. In this stage it present with forgetfulness. Mild cognitive impairment (MCI) are noted between normal forgetfulness due to aging, and the development of AD. It presents with difficulty in remembering recently learned facts and inability to acquire new information. It may take as long as 8 years between presentation of mild cognitive difficulties and fulfilling the clinical criteria for diagnosis of AD.

Other presentations may be in form of Subtle problems e.g. executing functions of attentiveness, planning, flexibility, and abstract thinking or impairments in sementic memory (memory of meanings, and concept relationships) in early stages of AD. Apathy can also be observed at this stage. This presentation of apathy remains the most persistent neurophychiatric symptom throughout the course of the disease.

Symptoms of MCI include:

- Forgetfulness of recent events or conversations
- Difficulty performing more than one task at a time
- Difficulty solving problems
- Taking longer to perform more difficult activities

The early symptoms of AD can include:

Language problems, Misplacing items, Getting lost on familiar routes, Personality changes and loss of social skills, patient losses interest in things previously enjoyed, Difficulty in performing tasks that take some thought.

As the AD becomes worse:

Symptoms are more obvious and interfere with your ability to take care of yourself. Such as forgetting details about current events, Change in sleep patterns, often waking up at night, Difficulty reading or writing, Poor judgment and loss of ability to recognize danger, Using the wrong word, mispronouncing words, speaking in confusing sentences, Withdrawing from social contact, Having hallucinations, delusions, depression, agitation and Difficulty in doing basic tasks.

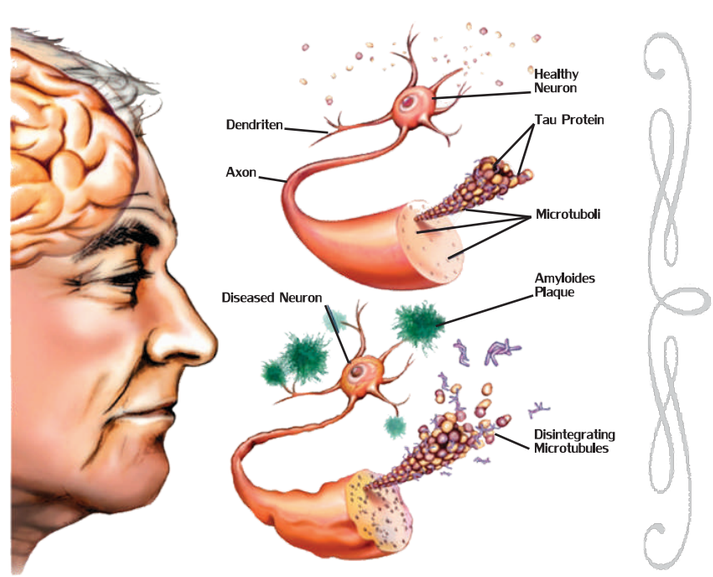
People with severe AD can no longer:

Understand language, Recognize family members, Perform basic activities of daily living, such as eating, dressing, and bathing

Other symptoms that may occur with AD:

- Incontinence
- Swallowing problems

The mean life expectancy after diagnosis is approximately seven years. Fewer than three percent of individuals live more than fourteen years after diagnosis.



Infographic Source : Dementia Today

Treatment

There is no cure for AD in Allopathy. The goals of treatment are:

- Slow the progression of the disease (although this is difficult to do)
- Manage symptoms, such as behavior problems, confusion, and sleep problems
- Change your home environment so you can better perform daily activities
- Support family members and other caregivers

Medicines are used to help slow down the rate at which symptoms become worse. The benefit from these drugs is usually small. You and your family may not notice much of a change.

Medicines for AD include:

- Donepezil (Aricept), rivastigmine (Exelon), and galantamine (Razadyne, formerly called Reminyl). Side effects include stomach upset, diarrhea, vomiting, muscle cramps, and fatigue.
- Memantine (Namenda). Possible side effects include agitation or anxiety.
- Other medicines may be needed to control aggressive, agitated, or dangerous behaviors. Examples include haloperidol, risperidone, and quetiapine. These are usually given in very low doses due to the risk of side effects including an increased risk of death.

It may be necessary to stop any medications that make confusion worse. Such medicines may include painkillers, cimetidine, central nervous system depressants, antihistamines, sleeping pills, and others. Never change or stop taking any medicines without first talking to your doctor.

Some people believe certain vitamins and herbs may help prevent or slowdown AD.

- There is no strong evidence that Folate (vitamin B6), vitamin B12, and vitamin E prevent AD or slows the disease once it occurs.
- High-quality studies have not shown that ginkgo biloba lowers the chance of developing dementia. DO NOT use ginkgo if you take blood-thinning medications like warfarin (Coumadin) or a class of antidepressants called monoamine oxidase inhibitors (MAOIs).

AYURVEDIC TREATMENT

Since AD cannot be cured or prevented, there is ray of hope by ayurvedic treatment that either the disease can be prevented or/and few presentations can be revert back by improving cerebral perfusion.

There are two aspects of treatment by Ayurveda

PART 1

Improving the cerebral perfusion so that brain function of dying neuronal cells can be improved

PART 2

Aiming to improve the immune system by ayurvedic system.Since in AD immune response initially aiming at maintaining the integrity of the body may fail and consequently lead to tissue destruction and neuronal loss.

Treatment of Part 1

Few cases of AD indicated definitive clinical improvement in cases of AD when treated with herbal nutritional supplement (using Cerebro Flo containgZingiberofficinale, Asparagus racemosus,Terminaliachebula etc.). This herbal nutritional supplement documented the improvement in the cerebral perfusion when given to the patients with higher mental function disorder.

The reversal in clinical presentation is expected with the herbal nutritional supplement and the quantum of clinical improvement depends upon the level of improvement in cerebral perfusion and anatomical damage in the neural tissue.

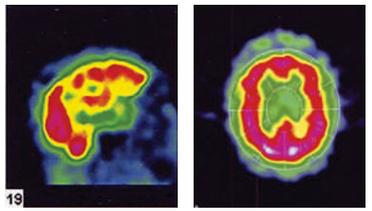
The changes can be expected in 3-6 month of starting the supplement. Early cases may show clinical improvement as early as 3 weeks of starting the supplement.

Treatment of Part 2

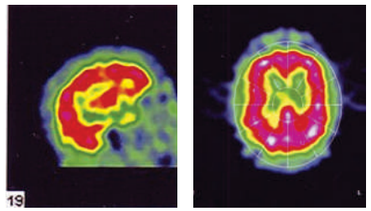
Use of purified Vishtinduk (StrichnusNux-vomica) is useful in improving the immune system and consequently inhibits tissue destruction and neuronal loss.

Case studies showing improvement in Cerebral Perfusion

Here one case report is given documenting improvement in cerebral perfusion after supplementing said herbal nutritional supplement.



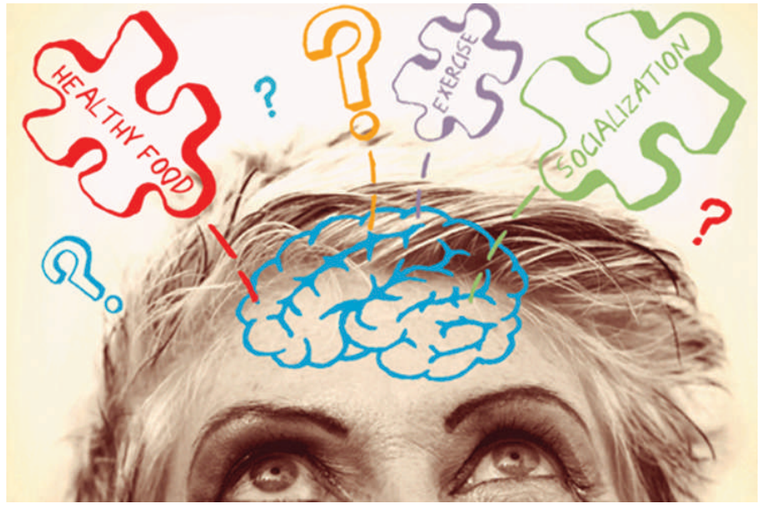
Pretreatment



Post Treatment (6 Months)

BHU Efforts

Times of India has reported that in a major achievement in the field of medicine, Banaras Hindu University (BHU) is making all efforts to introduce medicines for Alzheimer's and Parkinson's diseases soon.



Prof GP Dubey of the Institute of Medical Sciences(IMS), BHU has claimed to have received global patent for the plant based products prepared from three medicinal plants. These plants have been found useful in prevention and management of not only Azheimer's and Parkinson's disease but also in various age related neurogenerative changes. These plants are BacopaMonnieri (Brahmi), Hippophaerhamnoides (Amlavetus) and Dioscoreabulbifera (Varahikand).

A private drug manufacturing company has shown its interest in manufacturing Ayurvedic drugs from these plants. The BHU has entered into an agreement with the company for globalising the BHU patented drugs into global market. The decision about manufacturing drug was taken during a meeting held in the chairmanship of vice-chancellor, BHU, Lalji Singh on Saturday.

Giving details about the drugs and the patented products on Sunday, Prof Dubey along with director, IMS-BHU, DrRanaGopal Singh informed that Ayurvedic products from medicinal plants are believed to be the first global patent of the country in Ayurveda.

According to them, this is going to be the first Ayurvedic drug of the country which will fulfill all the international regulatory norms. The royalty earned from these drugs will be donated to BHU, Genome Foundation, Aadesh Institute, Bhatinda and SRM University, by Prof Dubey. He has been working on these drugs for past 30 years. The drugs developed by the company will come in the form of capsules and their cost will be nominal so that even poor people can buy them.