Feeding and Memory Disorder (Alzheimer Disease)

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Abstract: Progressive mental deterioration in old age has been recognized and described throughout history. However, it was not until 1906 that a German physician, Dr. Alois Alzheimer, specifically identified a collection of brain cell abnormalities as a disease. One of Dr. Alzheimer's patients died after years of severe memory problems, confusion and difficulty understanding questions. Upon her death, while performing a brain autopsy, the doctor noted dense deposits surrounding the nerve cells (neuritic plaques). Inside the nerve cells he observed twisted bands of fibers (neurofibrillary tangles). Today, this degenerative brain disorder bears his name, and when found during an autopsy, these plaques and tangles mean a definite diagnosis of Alzheimer's disease (AD). Since its discovery more than 100 years ago, there have been many scientific breakthroughs in AD research. In the 1960s, scientists discovered a link between cognitive decline and the number of plaques and tangles in the brain. The medical community then formally recognized Alzheimer's as a disease and not a normal part of aging. In the 1970s, scientists made great strides in understanding the human body as a whole, and AD emerged as a significant area of research interest. This increased attention led in the 1990s to important discoveries and a better understanding of complex nerve cells in the brains of AD patients. More research was done on AD susceptibility genes, and several drugs were approved to treat the cognitive symptoms of the disease. There is 450 thousand person to contract the Alzheimer disease in Iran. The lifetime risk of Alzheimer disease is estimated to be 1:5-2. More than 15% of individuals older than 65 years have AD, and the prevalence increases to at least 40% in individuals older than 80 years. This study showed that although Alzheimer's cure does have be improved conditions living the reduce stress and increase social activity and especially the control diet, the incidence avoided. The foods in which substances such as omega 3, omega 6, Vitamin A and Vitamin B12, there is more recommended and those 60 years above of age must have there is reduced animals fatty acids severely in the diet of red meat.

Key words: Alzheimer disease, Feeding, Malnutrition, Dementia.

INTRODUCTION

Alzheimer's disease is a seriously disabling neurodegenerative disease of the brain. Alzheimer's disease progressively damages and destroys such cognitive processes as memory, orientation, and speech. Alzheimer's disease is not curable and is the most common cause of dementia, a progressive and permanent loss of cognitive and mental performance.

Alzheimer's disease begins subtly but eventually progresses into severe disability and an inability to function safely and effectively in daily life and to meet basic needs. There is no cure for Alzheimer's disease, and it is one of the ten leading causes of death in the world.

The cause of Alzheimer's disease is not yet well understood. However, Alzheimer's disease is characterized by the formation of large numbers of abnormal features in the brain called plaques and tangles. Plaques are dense deposits of protein that build up over time between brain cells. Tangles are twisted fibers of protein that develop inside brain cells. It is believed that plaques and tangles can block communication between brain cells and play a role in brain cell degeneration and brain cell death.

Everyone develops some plaques and tangles as they age, but people with Alzheimer's disease have far greater numbers of them. Plaques and tangles first develop in areas of the brain responsible for memory and learning, but then spread to other areas that control language and thought. This leads to symptoms that include...
forgetfulness and other problems with memory that become progressively worse. Disorientation, poor judgement, speech difficulties, personality changes, and difficulty completing familiar tasks also occur. Symptoms of Alzheimer's disease eventually progress to become severely disabling.

For more details on symptoms and complications, refer to symptoms of Alzheimer's disease.

Risk factors for developing Alzheimer's disease include being over age 65 and having a family history of Alzheimer's disease. Other risk factors include a history of severe head injury, alcoholism, and having diabetes, high blood pressure, high cholesterol, and other types of cardiovascular disease.

There is no specific diagnostic test that can detect Alzheimer's disease. Making a diagnosis includes performing a variety of tests and assessments that evaluate the brain and can rule out other causes of Alzheimer's disease symptoms, such as vascular dementia or depression. Diagnosis and treatment may require the collaboration of a variety of providers, including a primary care physician, neurologist, psychiatrist, and/or psychologist.

The diagnostic process begins with taking a thorough personal and family history, including symptoms, and completing a physical examination. This includes a neurological exam. A neurological exam evaluates the nerves and nervous system and such functions as reflexes, sensation, movement, balance, coordination, vision, and hearing.

Commonly used tests include a mini-mental state examination (MMSE), which evaluates mental function by assessing the answers provided to a series of questions. Imaging tests that are used to help make a diagnosis include CT and MRI, which provide information about the structure of the brain. A PET scan and functional MRI are imaging tests that can show how well different areas of the brain are functioning.

It is possible that a diagnosis of Alzheimer's disease can be missed or delayed because symptoms develop gradually and are similar to symptoms of other diseases and conditions. For more information about diseases and conditions that can mimic Alzheimer's disease, refer to misdiagnosis of Alzheimer's disease.

Alzheimer's disease is not curable, and at this time there are no treatments that can slow the advancement of the disease. However, there are some medications that may help to reduce some symptoms and maximize independence and the quality of life. There are also clinical trials taking place to research a variety of potential treatments. For more information on treatment, refer to treatment of Alzheimer's disease. more

Alzheimer's disease (AD) affects the mental abilities including memory, language, and cognition. Progressively it leads to dementia and death. AD usually arises in late middle age or the elderly but there is a rare familial subtype that occurs earlier. Because AD is so well-known, other causes of dementia or memory loss may be overlooked. Other possible diagnoses include normal aging (if very mild symptoms), emotional problems, fatigue, depression, and certain medical conditions such as thyroid disease, brain tumors, multi-infarct disease, or Huntington's disease. In its early stages, a correct diagnosis of AD can also be overlooked itself and misdiagnosed as other conditions such as depression, dementia, simple forgetfulness, or senility.

History of Alzheimer's Disease:

Progressive mental deterioration in old age has been recognized and described throughout history. However, it was not until 1906 that a German physician, Dr. Alois Alzheimer, specifically identified a collection of brain cell abnormalities as a disease. One of Dr. Alzheimer's patients died after years of severe memory problems, confusion and difficulty understanding questions. Upon her death, while performing a brain autopsy, the doctor noted dense deposits surrounding the nerve cells (neuritic plaques). Inside the nerve cells he observed twisted bands of fibers (neurofibrillary tangles). Today, this degenerative brain disorder bears his name, and when found during an autopsy, these plaques and tangles mean a definite diagnosis of Alzheimer's disease (AD).

Since its discovery more than 100 years ago, there have been many scientific breakthroughs in AD research. In the 1960s, scientists discovered a link between cognitive decline and the number of plaques and tangles in the brain. The medical community then formally recognized Alzheimer's as a disease and not a normal part of aging. In the 1970s, scientists made great strides in understanding the human body as a whole, and AD emerged as a significant area of research interest. This increased attention led in the 1990s to important discoveries and a better understanding of complex nerve cells in the brains of AD patients. More research was done on AD susceptibility genes, and several drugs were approved to treat the cognitive symptoms of the disease.

Over the last decade, scientists have substantially progressed in understanding potential environmental, genetic and other risk factors for AD, the processes leading to formation of plaques and tangles in the brain, and the brain regions that are affected. Specific genes related to both the early-onset and late-onset forms of AD have been identified, but genetic risk factors alone do not fully explain its causes, so researchers are actively exploring environment and lifestyle to learn what role they might play in the development of this
More effective treatment options have been approved by the Food and Drug Administration (FDA). However, AD is still incurable. The drugs currently in use treat only the symptoms, not the cause of the disorder, and they only slow the progression of cognitive decline.

Patients with Alzheimer disease most commonly present with insidiously progressive memory loss, to which other spheres of cognitive impairment are added over several years. After memory loss occurs, patients may also have language disorders (eg, anomia) and impairment in their visuospatial skills and executive functions.

The National Institutes of Health-Alzheimer's Disease and Related Disorders Association (NIH-ADRDA), the Diagnostic and Statistical Manual of Mental Disorders, Fourth Revision, Text Revision (DSM-IV-TR), and the Consortium to Establish a Registry in Alzheimer's Disease (CERAD) have formulated several clinical guidelines for the diagnosis of AD. The NIH-ADRDA criteria for the diagnosis of AD require the finding of a slowly progressive memory loss of insidious onset in a fully conscious patient. AD cannot be diagnosed in patients with clouded consciousness or delirium. Toxic metabolic conditions and brain neoplasms must also be excluded as potential causes of the patient's dementia.

The main focus of these diagnostic guidelines consists of verifying the initial finding of mild, slowly progressive memory loss, that additional spheres of cognition are also compromised, and that other possible causes for dementia (eg, cerebrovascular disease, cobalamin deficiency, syphilis, thyroid disease) are ruled out with a combination of clinical examination and ancillary radiologic and laboratory tests. These guidelines are widely believed to be 90-95% accurate (as histopathologically verified) when followed carefully, and they are important not only for routine management but also for selecting and enrolling patients in therapeutic trials.

Substantially less common, but biopsy or autopsy-proven, presentations include right parietal lobe syndrome, progressive aphasia, spastic paraparesis, and impaired visuospatial skills, which is subsumed under the visual variant of Alzheimer disease.

Depression and AD and other dementias have an association that is likely to be complex and depression may be misdiagnosed in the realm of dementia. Recent Framingham data have helped bolster the epidemiological association. The study showed a 50% increase in AD and dementia in those who were depressed at baseline. During a 17-year follow-up period, a total of 21.6% of participants who were depressed at baseline developed dementia compared with 16.6% of those who were not depressed (HR 1.72, 95% CI, 1.04-2.84, p = 0.035 and AD HR 1.76, 95% CI, 1.03-3.01, p =0.039). In another related study, recurrent depression was noted to be particularly pernicious, with one depression episode conferring an 87-92% increase in dementia risk, while having 2 or more episodes nearly doubled the risk (Giubilei et al., 2001).

Pathophysiology:

The anatomic pathology of Alzheimer disease includes neurofibrillary tangles (NFTs); senile plaques (SPs) at the microscopic level; and cerebrocortical atrophy, which predominantly involves the association regions and particularly the medial aspect of the temporal lobe. NFTs and SPs were described by Alois Alzheimer in his original report on the disorder in 1907; they are now universally accepted as a hallmark of the disease. Although NFTs and SPs are characteristic of Alzheimer disease, they are not pathognomonic. NFTs are found in several other neurodegenerative disorders, including progressive supranuclear palsy and dementia pugilistica. SPs may occur in normal aging. Therefore, the mere presence of these lesions is not sufficient to diagnose Alzheimer disease. These lesions must be present in sufficient numbers and in a characteristic topographic distribution to fulfill the current histopathologic criteria for Alzheimer disease.

In addition to NFTs and SPs, many other lesions of Alzheimer disease have been recognized since Alzheimer's original papers were published. These include (Morris) the granulovacuolar degeneration of Shimkowicz; (Morris, 1998) the neuropil threads of Braak et al (Morris, 2003) and (Sano, et al.,1997) neuronal loss and synaptic degeneration, which are thought to ultimately mediate the cognitive and behavioral manifestations of the disorder.

Some authorities believed that NFTs, when present in low densities and essentially confined to the hippocampus, were part of normal aging. However, the histologic stages for Alzheimer disease that Braak et al formulated include an early stage in which a low density of NFTs is present in the entorhinal and perirhinal (ie, transentorhinal) cortices. Therefore, even small numbers of NFTs in these areas of the medial temporal lobe may be abnormal.

In contrast, there is consensus that the presence of even low numbers of NFTs in the cerebral neocortex with concomitant SPs is characteristic of Alzheimer disease. Granulovacuolar degeneration occurs almost exclusively in the hippocampus. Neuropil threads, which are an array of dystrophic neurites diffusely distributed in the cortical neuropil, more or less independently of plaques and tangles. This lesion suggests neuropil
alterations beyond those merely due to NFTs and SPs and indicates an even more widespread insult to the cortical circuitry than that visualized by studying only plaques and tangles. NFTs are initially and most densely distributed in the medial aspect and in the pole of the temporal lobe; they affect the entorhinal cortex and the hippocampus most severely. As Alzheimer disease progresses, NFTs accumulate in many other cortical regions, beginning in high-order association regions and less frequently in the primary motor and sensory regions. SPs also accumulate primarily in association cortices and in the hippocampus. Plaques and tangles have relatively discrete and stereotypical patterns of laminar distribution in the cerebral cortex, which indicate predominant involvement of corticocortical connections.

**Frequency:**

**In Iran:**
There is 450 thousand person to contract the Alzheimer disease in Iran. The lifetime risk of Alzheimer disease is estimated to be 1:5-2. More than 15% of individuals older than 65 years have AD, and the prevalence increases to at least 40% in individuals older than 80 years.

**International:**
Prevalence of Alzheimer disease similar to those in the United States have been reported in industrialized nations. Countries experiencing rapid increases in the elderly segments of their population have rates approaching those in the United States.

**Mortality/Morbidity:**
In the United States, Alzheimer disease is frequently considered a leading cause of death. In 2006, Alzheimer disease was the seventh leading cause of death; however, Alzheimer disease as an underlying cause of death is frequently underreported (Sapolsky, 1986). The primary cause of death is intercurrent illness, such as pneumonia, in a patient who has become severely demented from Alzheimer disease. Patients lose the ability to walk and swallow. Difficulty swallowing may lead to aspiration pneumonia.

**Race:**
Alzheimer disease and other dementias are more common in African-Americans compared to whites. According to the Alzheimer's Association, in the population aged 71 and older, African-Americans are almost 2 times as likely to have Alzheimer disease and other dementias than whites (21.3% of African-Americans vs 11.2% of whites). The number of Hispanic patients studied in this age group was too small to determine the prevalence of dementia in this population. In individuals age 65 and older, 7.8% of whites, 18.8% of African-Americans, and 20.8% of Hispanics have Alzheimer disease or other dementias, and the prevalence of Alzheimer disease and other dementias is higher in older versus younger age groups (Morris et al.,).

**Sex:**
Alzheimer disease affects both men and women; however, Plassman et al found the risk of developing Alzheimer disease to be significantly higher in women than in men, primarily due to the higher life expectancy in women compared with men (Bremner, 1999).

**Age:**
The prevalence of Alzheimer disease increases with age. Alzheimer disease is most prevalent in individuals older than 60 years. Some forms of familial early-onset Alzheimer disease can appear as early as the third decade, but this represents a subgroup of the less than 10% of all familial cases of Alzheimer disease. More than 90% of cases of Alzheimer disease are sporadic and occur in individuals older than 60 years. Of interest, results of some studies of nonagenarians and centenarians suggest that the risk may decrease in individuals older than 90 years. If so, age is not an unqualified risk factor for the disease, but further study of this matter is needed. Savva et al found that the association between the pathological features of Alzheimer disease and dementia (eg, neuritic plaques, diffuse plaques, tangles) is stronger in younger old persons (ie, age 75 years) than in older old persons (ie, 95 years). These results were achieved by assessing 456 brains donated to the population-based Medical Research Council Cognitive Function and Ageing Study from persons 69-103 years of age at death.

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These results demonstrate that the relationship between cerebral atrophy and dementia persist into the oldest ages, but the strength of association between pathological features of Alzheimer disease and clinical dementia diminished. It is important to take age into account when assessing the likely effect of interventions against dementia on the population.

**Malnutrition:**

Demented patients who stop eating become malnourished rapidly. Development of abnormal markers of nutritional status are often used to justify feeding-tube placement in the belief that tube feeding would help prevent the consequences of malnutrition, which include pressure sores, infection, and death.

A sample of 40 chronically tube-fed patients with poor functional and cognitive status demonstrated that weight loss, severe depletion of lean and fat body mass, and micronutrient deficiencies persisted even if generous amounts of standard enteral formulas were provided. Other studies have demonstrated that weight loss increased in amount and frequency as the duration of the tube feeding lengthened (Giubilei et al., 2001; Seshadri et al., 2002). Other nutritional markers such as hemoglobin, hematocrit, albumin, and cholesterol levels also did not show any significant improvement after a feeding tube was placed (Giubilei et al., 2001; Seshadri et al., 2002).

The persistent malnutrition in these chronically tube-fed patients in the face of adequate amounts of formula suggest that "the long-term effects of chronic disease, immobility, and neurologic defects may undermine attempts at long-term nutritional support." Negative outcomes may be unavoidable in these patients despite tube feeding.

**Pressure Sores:**

The data linking malnutrition to the development or worsening of pressure sores are limited. Two retrospective cohort studies (Sachdev et al., 2002; Duthie et al., 2002) did demonstrate that during six months of follow-up, poor oral intake was associated with non-healing pre-existing pressure sores and the formation of new pressure sores. Malnutrition is often cited as a risk factor for developing pressure sores, and feeding tubes are often placed to improve nutritional status and theoretically improve skin integrity. However, one retrospective study observed that the incidence of decubitus ulcers was not statistically different between those patients with (21 percent) and without (13 percent) feeding tubes.

A MEDLINE search from 1985 to 1994 was performed to review the relationship between malnutrition and pressure sores and to gauge the effectiveness of tube feeding in improving the outcomes of pressure sores. The conclusion suggests that the data linking malnutrition and the development of pressures sores were incomplete and contradictory and that "the routine use of tube feeding to prevent or treat pressure sores is not clearly supported by data (Bottiglieri et al., 2001)." In a follow-up review, there were still no data to support the use of feeding tubes to improve pressure sores.

**Aspiration Pneumonia:**

Interrupting the cycle of eating, aspiration, and subsequent pneumonia is one of the most commonly cited reasons for using a feeding tube. However, there are no data that show feeding tubes reduce the risk of aspiration pneumonia in patients with dementia (Durga et al., 2005). In fact, some data have shown that the risk of aspiration is increased. One study examining the risk of aspiration pneumonia in 104 severely demented nursing home patients found that patients with feeding tubes experienced significantly more episodes of aspiration pneumonia (58 percent) than the patients without feeding tubes (17 percent; \( P < 0.01 \)).

In assessing whether one site of feeding tube placement was superior to others, investigators compared the incidence of aspiration between patients with jejunostomy tubes and those with gastrostomy tubes. A meta-analysis of 45 studies between 1978 and 1989 with a total of 2,976 gastric tubes and 386 jejunal tubes found that aspiration rates were highly variable across different patient populations and studies. The authors concluded that there were no data to demonstrate decreased risk of aspiration at one feeding tube site compared with another (Eussen et al., 2005). The continued risk of aspiration despite feeding tube placement may result from continued reflux of gastric contents and aspiration of oropharyngeal secretions (Rifat et al., 1990).

**Quality of Life:**

Caretakers and physicians often project sensations of hunger and thirst onto severely demented patients with poor oral intake. "We can't just let him starve to death," is a common refrain heard from family members. Clearly, it is impossible to ask patients suffering from severe dementia if they are truly uncomfortable in such a state. Data about thirst and hunger can be extrapolated from patients dying with other terminal illnesses.
One study (Suay Llopis, 2002) surveyed (Mix) patients dying of cancer and stroke. The patients had anorexia or profound dysphagia, and they retained sufficient awareness to express sensations of hunger and thirst at least 75 percent of the time from initial admission until their death. All those who experienced hunger received small amounts of food for alleviation. Patients who complained of thirst and dry mouth were given mouth swabs, sips of water, ice chips, and lubrication of the lips. It is important to mention that the amount given to attempt to alleviate these symptoms was much less than the amount needed to replenish losses. Interestingly, an overwhelming majority of these patients (84 percent) reported that their thirst and hunger were successfully alleviated by these minimal interventions.

In another prospective study, (Campbell, 2002) there were no significant differences in the mean patient comfort scores between the predehydration and dehydration phases. [Evidence level B: clinical cohort study] A perfect comfort score was realized in 85 percent of cases. The results of these studies suggest that patients with a terminal illness can experience comfort despite minimal intake of food and fluids.

**Functional Status and Survival:**

Artificial nutrition and hydration are often provided to improve functional status and survival. Currently, there are limited data about the impact of feeding tubes on improving functional status in patients with advanced dementia. One retrospective observation11 of nursing home residents who received feeding tubes found no improvement in bowel and bladder function, mental status, speech, activities of daily living, or ambulation during the 18 months after PEG tube placement. Patients in one community-based study21 did experience some improvement in upper and lower body function over a period of four months.

Patients and their surrogate decision makers often expect the prolongation of life to be the outcome of feeding-tube placement (Kirschbaum, 1996). A few cohort studies (Ritchie, 2003; Wenstrup) comparing nursing home residents with and without feeding tubes have not shown a survival advantage in patients with feeding tubes (Ritchie, 2003; Wenstrup). In a recent MEDLINE review, (Hock et al.,) only 38 percent of the patients were alive one year after placement of a feeding tube. For acutely ill patients with severe dementia, there was no survival advantage among patients who received a feeding tube during their index hospitalization, compared with those without a feeding tube (Leong et al., 2001). With or without a feeding tube, these patients have a 50 percent six-month median mortality rate. Perhaps the inability to eat marks the point at which the patient has entered the final stages of the illness that may be intractable even in the face of aggressive intervention. This would account for the lack of improvement in functional status and survival rate.

Dementia is a loss of brain function that occurs with certain diseases.

It affects memory, thinking, language, judgment, and behavior.

Dementia (meaning "deprived of mind") is a cognitive disorder. It serious may be static, the result of a unique global brain injury or progressive, resulting in long-term decline in cognitive function due to damage or disease in the body beyond what might be expected from normal aging. Although dementia is far more common in the geriatric population, it may occur in any stage of adulthood. This age cutoff is defining, as similar sets of symptoms due to organic brain syndrome or dysfunction, are given different names in populations younger than adult. Up to the end of the nineteenth century, dementia was a much broader clinical concept.
Dementia is a non-specific illness syndrome (set of signs and symptoms) in which affected areas of cognition may be memory, attention, language, and problem solving. It is normally required to be present for at least 6 months to be diagnosed; cognitive dysfunction that has been seen only over shorter times, in particular less than weeks, must be termed delirium. In all types of general cognitive dysfunction, higher mental functions are affected first in the process. Especially in the later stages of the condition, affected persons may be disoriented in time (not knowing what day of the week, day of the month, or even what year it is), in place (not knowing where they are), and in person (not knowing who they are or others around them). Dementia, though often treatable to some degree, is usually due to causes that are progressive and incurable.

Symptoms of dementia can be classified as either reversible or irreversible, depending upon the etiology of the disease. Less than 10 percent of cases of dementia are due to causes that may presently be reversed with treatment. Causes include many different specific disease processes, in the same way that symptoms of organ dysfunction such as shortness of breath, jaundice, or pain are attributable to many etiologies. Without careful assessment of history, the short-term syndrome of delirium (often lasting days to weeks) can easily be confused with dementia, because they have all symptoms in common, save duration, and the fact that delirium is often associated with over-activity of the sympathetic nervous system. Some mental illnesses, including depression and psychosis, may also produce symptoms that must be differentiated from both delirium and dementia. Chronic use of substances such as alcohol as well as chronic sleep deprivation can also predispose the patient to cognitive changes suggestive of dementia.

Dementia is a word for a group of symptoms caused by disorders that affect the brain. It is not a specific disease. People with dementia may not be able to think well enough to do normal activities, such as getting dressed or eating. They may lose their ability to solve problems or control their emotions. Their personalities may change. They may become agitated or see things that are not there.

Memory loss is a common symptom of dementia. However, memory loss by itself does not mean you have dementia. People with dementia have serious problems with two or more brain functions, such as memory and language.

Many different diseases can cause dementia, including Alzheimer's disease and stroke. Drugs are available to treat some of these diseases. While these drugs cannot cure dementia or repair brain damage, they may improve symptoms or slow down the disease.

Dementia Causes:

Most types of dementia are nonreversible (degenerative). Nonreversible means the changes in the brain that are causing the dementia cannot be stopped or turned back. Alzheimer's disease is the most common type of dementia.

Lewy body disease is a leading cause of dementia in elderly adults. People with this condition have abnormal protein structures in certain areas of the brain.

Dementia also can be due to many small strokes. This is called vascular dementia.

The following medical conditions also can lead to dementia:

- Parkinson's disease
- Multiple sclerosis
- Huntington's disease
- Pick's disease
- Progressive supranuclear palsy
- Infections that can affect the brain, such as HIV/AIDS and Lyme disease.

Some causes of dementia may be stopped or reversed if they are found soon enough, including:

- Brain tumors
- Changes in blood sugar, sodium, and calcium levels (see: Dementia due to metabolic causes)
- Low vitamin B12 levels
- Normal pressure hydrocephalus
- Use of certain medications, including cimetidine and some cholesterol-lowering medications
- Chronic alcohol abuse.

Dementia usually occurs in older age. It is rare in people under age 60. The risk for dementia increases as a person gets older.
Dementia Prevention and Dementia Support:
The best way to prevent vascular dementia is to lower the risk of stroke. Studies have shown that the rate of prevalence for vascular dementia is (Newcomer, 1999) times higher following a stroke than the rate of prevalence in control groups. There are ways you can reduce your risk of high blood pressure, stroke, and vascular dementia.

Make a Commitment to a Healthier Lifestyle:
Not smoking, exercising regularly, and eating a healthy diet (limiting your intake of alcohol, salt and saturated fat) is a great way to reduce your risk of heart disease and many other diseases. Stress is a major contributor to high blood pressure and heart disease, so it's helpful to learn to manage your stress through relaxation techniques or meditation. Try to get your blood pressure checked at least once a year, as well as your body fat levels measured periodically, especially if you are over 65 or have a history of heart disease.

Physicians about Medications:
Medications can control high blood pressure and heart disease. Blood thinners, for example, are commonly used to correct an irregular heart beat. In more advanced cases of arteriosclerosis, or hardening of the arteries, surgery may be necessary to restore the blood flow to the brain.

Education, lifestyle changes, and adequate medical advice are the best safeguards. Know the warning signs and start making the necessary life adjustments. The most important thing is to begin! Seek support and encouragement from friends, family, support groups (see References and resources), and health care experts. And remember that even if you have already been diagnosed with vascular dementia, it is not too late to do anything about it.

Dementia Signs:
The phrase "signs of Dementia" should, strictly speaking, refer only to those signs and symptoms of Dementia that are not readily apparent to the patient. The word "symptoms of Dementia" is the more general meaning; see symptoms of Dementia.

The signs and symptom information on this page attempts to provide a list of some possible signs and symptoms of Dementia. This medical information about signs and symptoms for Dementia has been gathered from various sources, may not be fully accurate, and may not be the full list of Dementia signs or Dementia symptoms. Furthermore, signs and symptoms of Dementia may vary on an individual basis for each patient. Only your doctor can provide adequate diagnosis of any signs or symptoms and whether they are indeed Dementia symptoms.

Dementia Complications:
Dementia causes serious memory, personality, and behavioral problems that the person can not recognize. Someone with dementia may at first remember how to operate an automobile and how to travel to familiar places. However, at some point as the disease progresses, their driving abilities do become

What Causes Dementia and Alzheimer's?
AD is a complex disease without a single cause, but with many contributors. Many researchers are now converging on the idea that it is a degenerative inflammatory disease that damages a particular part of the brain called the Medial Temporal Lobe. This may be largely due to the long-term consequence of faulty nutrition plus certain negative lifestyle factors, much like cardiovascular disease, and that any long-term solution must involve fundamental changes to a person's diet. The contributory factors may include:

- Lack of antioxidant nutrients
- Lack of omega 3 Fatty Acids
- Excessive stress and the stress hormone cortisol
- Raised homocysteine and a lack of B vitamins
- Excess aluminium and/or mercury
- Acetylcholine and precursor deficiency
- A genetic predisposition
- A lack of physical and mental exercise - use it or lose it.

You can find out which of these factors is likely to affect you be completing the FREE Mental Health Check.
Diet & Nutrition—what Works:
UP Your Antioxidants:
Inflammatory reactions invariably mean increased production of oxidants, and hence an increased need for antioxidants such as vitamin A, beta-carotene, and vitamins C and E, all of which have been shown to be low in those with AD. Other antioxidants, including cysteine, glutathione, lipoic acid, anthocyanidins, and co-enzyme Q10, possibly other quinones, and melatonin may also prove important. In simple terms this means eating a lot more fresh fruit and vegetables - at least six portions a day - and oily fish and seeds.

Vitamin E appears to have a protective effect. A study published in the Journal of the American Medical Association found that the risk of developing AD was 67 per cent lower in those with a high dietary intake of vitamin E, versus those with a low intake.

A US study gave 633 disease-free 65-year-olds large amounts of either vitamin E or vitamin C. A small number in each group would have been expected to show the signs of AD five years later. None did.

Vitamin E appears to not only play a key role in early prevention in this way, but also in slowing down the progression of the disease. In a study reported in the New England Journal of Medicine in 1997, AD patients received either 2,000ius of vitamin E, the drug Selegiline, or a placebo.

Where's the Evidence?
Enter 'dementia' and 'antioxidants' into the search field for a summary of studies on antioxidants and Alzheimers/dementia.

Side Effects?
None known, however some studies indicate that high intakes from vitamin E supplements (above 300mg) may increase cardiovascular risk. We recommend supplementing vitamin E with Co-enzyme Q10, and ideally in an all-round antioxidant supplement.

Contraindications with Medication?
Vitamin E, in large amounts above 300mg may thin the blood. This is a benefit however if you are already on blood thinning medication you should discuss this with your doctor first. Statin drugs interfere with the body's ability to use vitamin E. If you are on a statin drug and wish to take vitamin E also supplement 90mg or more of Coenzyme Q10.

Omega 3 Fatty Acids:
Omega-3 fats are most prevalent in carnivorous, cold water fish such as salmon, tuna, herring and mackerel. According to recent a study by Dr. Martha Morris and colleagues at Chicago's Rush Institute for Healthy Aging, eating fish once a week reduces your risk of developing AD by 60 percent.

The researchers followed 815 people, aged 65 to 94 years, for seven years and found that dietary intake of fish was strongly linked to AD risk. They found that the strongest link was the amount of DHA, a form of omega 3-fat found in fish. The higher a person's DHA, the lower their risk of developing AD. The lowest amount of DHA per day that offered some protection was 100 mg. While intake of EPA (another omega-3 fat) did not reach significance, the highest intake of EPA consumed was 30 mg a day.

But why exactly might fish have this protective effect? One theory is that it helps to ease brain inflammation, which, in turn damages brain cells. Omega-3 fatty acids are also a vital component of brain cell membranes and help control calcium flow in and out of cells. This is important because too much calcium inside brain cells is known to contribute to the production of the toxic beta-amyloid protein, which is found in excessive levels in most people who develop Alzheimer's.

Where's the Evidence?
Enter 'omegas' and 'dementia' into the search field for a summary of studies on omega 3 and Alzheimers/dementia.

Contraindications with Medication?
Omega 3 oils, in large amounts, can thin the blood. This is a benefit however if you are already on blood thinning medication you should discuss this with your doctor first.
Stress, Cortisol and Memory Loss:

Under prolonged stress, the body produces the adrenal hormone cortisol. The research of Professor Robert Sapolsky at Stanford University has shown that although cortisol is a powerful anti-inflammatory hormone, raised cortisol can damage the brain. In studies with rats he found that two weeks of induced stress causing raised cortisol levels causes dendrites, those connections between brain cells, to shrivel up. He believes that brain cell loss in ageing and AD may be, in part, due to high levels of cortisol and recommends that corticosteroid drugs should not be used in AD patients for other medical problems like asthma or arthritis.

Using a brain imaging technique, Douglas Bremner of Yale University has shown that the part of the brain responsible for learning and memory is smaller in patients with post-traumatic stress disorder, and that this correlates with poorer memory. Researchers at the La Sapienza University have shown that cortisol levels are significantly higher in AD patients than in controls, and correlate with the severity of the disease. Linda Carlson and colleagues at McGill University in Montreal have confirmed that in AD patients, the higher the cortisol, the worse their memory. They also found that the higher the levels of DHEA the better their memory.

Adrenal exhaustion can also lead to a lack of cortisol, which increases inflammation. It's a question of balance. All this research implies that the ability to create a lifestyle that minimises prolonged stress is also important for reducing AD risk.

Where's the Evidence?

Enter 'stress' and 'dementia' into the search field for a summary of studies on stress and Alzheimers/dementia.

UP Your B Vitamins to Lower Homocysteine:

The single, hottest nutritional discovery is that your risk of developing Alzheimer's disease (AD) is strongly linked to your level of the toxic amino acid homocysteine, which can be measured from a pinprick of blood on a home test kit. The lower your level throughout life the smaller your chances of developing serious memory decline. Homocysteine is a neurotoxin, capable of directly damaging the medial temporal lobe, which is the area of the brain that rapidly degenerates in AD. Homocysteine is easily lowered with inexpensive B vitamins.

A study in the New England Journal of Medicine, published in 2002 charted the health of 1,092 elderly people without dementia, and measured their homocysteine levels. Eight years later, 111 were diagnosed with dementia, 83 of whom were given the diagnosis of AD. Those with high blood homocysteine levels (above 14 units) had nearly double the risk of Alzheimer's. There's also evidence that even before a decline in mental function starts to show up in so-called "healthy" elderly individuals, high homocysteine predicts physical degeneration in certain parts of the brain. In Scotland, researchers have found that reduced mental performance in old age is strongly associated with high homocysteine and low levels of vitamins B12 and folic acid. Following up participants in the Scottish Mental Surveys of 1932 and 1947, which surveyed childhood intelligence, they found that the most mentally agile had the highest levels of B vitamins and lowest levels of homocysteine; high homocysteine was linked with a 7 to 8 percent decline in mental performance. A similar Californian study, asked 579 men and women aged 60 and over to keep track of their diet and the supplements they took. After nine years, 57 of them developed Alzheimer's. Those with the highest folate intake reduced their risk of developing Alzheimer's by 55 percent.

A research group led by Dr. Teodoro Bottiglieri at the Baylor University Metabolic Disease Center in Dallas, Texas, suggests that low levels of folic acid (which leads to raised homocysteine) may cause brain damage that triggers dementia and Alzheimer's. Their research has found that one-third of those with both dementia and homocysteine levels above 14 units were deficient in folic acid.

So there is a lot of research that points to a link between high homocysteine, low B vitamin intake and a raised risk of brain degeneration. The link between brain deterioration - memory loss, cognitive deficits, depression, and personality breakdown - and B vitamin deficiency is well established. But why? What is the link between B vitamins and damaged brain cells. The reason for this link may be that the body needs B vitamins to convert the toxic and brain damaging homocysteine into two very useful chemicals called glutathione - an antioxidant - and the amino acid called SAMe. SAMe is vital for 'methylation', which is a key chemical process happening millions of times every second, which keeps the brain's chemistry in balance.

So the theory makes sense but does supplementing with vitamins prevent, or actually reverse memory loss? In truth, it's early days but large amounts to seem to be effective. There are trials going on right now giving
B vitamins to people with age-related cognitive decline and AD; a Dutch one involving 818 people aged between 50 and 75 was completed last year.

They either got a vitamin containing 800mcg of folic acid a day - almost three times the RDA and the equivalent of 2.5 pounds of strawberries - or a dummy pill. After three years supplement users had scores on memory tests comparable to people 5.5 years younger. On tests of cognitive speed, the folic acid helped users perform as well as people 1.9 years younger.

Mega-doses are also being used in a trial currently being run by Professor David Smith of the Optima Project at the University of Oxford. He's giving people with age-related memory decline 1,000mcg of folic acid, 20mg of B6 and 500mcg of B12, which is 250 times the RDA; a far cry from the amount you could get by 'eating a well balanced diet'.

Such high amounts are being used for the simple reason that they work. 'The lowest dose of oral cyanocobalamin (B12) required to normalize mild vitamin B12 deficiency older people is more than 200 times the recommended dietary allowance,' concludes a paper by scientist at the University of Wageningen in Holland, one of the top B12 research centres in the world.

Although more needs to be done to find out both how early supplementation has to begin in order to halt or even reverse memory loss, and what is the most effect combination of diet and supplements, it certainly makes sense to ensure an optimal intake of B6, B12 and folic acid along with an amino acid N-Acetyl Cysteine (NAC) which is used to make the valuable brain antioxidant glutathione.

Nutritional supplements aimed at lowering homocysteine not only produce reduction in symptoms, but also potentially stop the disease progression. Although this has not yet been proven in double-blind controlled studies, case studies do show this type of improvement.

Where's the Evidence?
Enter 'vitamins' or 'homocysteine' and 'dementia' into the search field for a summary of studies on homocysteine, B vitamins and Alzheimer's/dementia.

Side Effects?
None known. However, supplementing folic acid on its own can mask the symptoms of B12 deficiency. Hence we recommend only supplementing extra folic acid if you also supplement extra vitamin B12.

Contraindications with medication? Certain drugs such as methyltraxate are anti-folate. If you are on an anti-folate drug you should discuss with your doctor before taking large amounts of folic acid.

Toxic Minerals-aluminium and Mercury:
Another brain toxin sometimes found in the plaques of dementia and Alzheimer's sufferers is aluminium. While some studies have shown this increased accumulation of aluminium, what isn't clear is whether this is a cause or a consequence of the disease. The likelihood is that it's a bit of both and may be a significant contributor to memory problems. Numerous epidemiological surveys have linked aluminium intake in water to increased risk of AD. Other sources (food, medicines, toiletries and cosmetics) are less well investigated. In a study in the 1980s of 647 Canadian gold miners who had routinely inhaled aluminium since the 1940s (this used to be a common practice, thought to prevent silica poisoning), all tested in the 'impaired' range for cognitive function, suggesting a clear link between aluminium and memory loss. A number of recent review papers have kept aluminium firmly on the map of potential contributors to dementia and AD. While the mechanism for action of aluminium in brain degeneration is far from clear, aluminium does exert a pro-oxidant effect in combination with copper, but not an inflammatory effect.

Beta-amyloid is a metaloprotein that contains zinc, copper, and iron. One hypothetical cause of its build-up and neurotoxic effect is that it mops up surplus metals and that these metals make beta-amyloid produce more hydrogen peroxide, a toxic free radical linked to its neurotoxic effect. Copper encourages this effect, while zinc appears to render beta-amyloid less harmful. This hypothesis, proposed by Dr Ashley Bush from Harvard Medical School, and colleagues from the University of Melbourne, Australia, has only been put to the test in a small randomized trial, giving AD patients clioquinol, a drug that prevents copper and zinc binding to beta-amyloid, thereby potentially promoting its dissolution and diminishing its toxic properties. This resulted in a reduction in deterioration of AD patients versus those receiving placebos. This line of investigation is likely to encourage further research into the possible toxic effects of copper and protective effects of zinc.
Mercury is another potential cause for concern. Autopsies of brains from AD patients, compared to control patients of the same age, have shown raised levels of mercury. Researchers from the University of Basel, Switzerland, have also found high blood mercury levels, more than double those of the control groups, in AD patients, with early-onset AD patients having the highest mercury levels of all.

Trace amounts of mercury can cause the type of damage to nerves that is characteristic of AD, according to recent research at the University of Calgary Faculty of Medicine, strongly suggesting that the small amounts we are exposed to, for example from amalgam fillings, may be contributing to memory loss. Although the research on the link of mercury to AD is in its infancy, it is certainly logical to reduce exposure to this highly toxic metal. It is possible that a small amount of cases of dementia may be due to mercury toxicity, although it is unlikely to be a major contributor to the epidemic of Alzheimer's disease.

Acetylcholine Enhancers and Memory:

Whatever the contributory causes to the brain damage seen in AD, once the brain damage occurs, there is memory loss. Understanding how that occurs opens up avenues for treatment.

A memory is not held in one, but in several brain cells joined together in a network. The memory itself is thought to be put into storage by the neurotransmitter acetylcholine, and stored by altering the structure of a molecule called RNA within brain cells. The limbic system, which is the 'doughnut' on top of the brain stem, then has to decide if the memory is worth keeping. The amygdala, part of the limbic system, decides about more emotional memories, while the hippocampus decides about others. In AD the memory with others. For example, you know the face but can't remember the name.

Most currently prescribed medication for dementia and AD block the breakdown or re-uptake of acetylcholine. An alternative approach would be to supplement the nutrients the brain uses to make acetylcholine in the first place. The primary precursor nutrient is phosphatidyl choline, the conversion of which is dependent on pantothenic acid (vitamin B5). However, phosphatidyl choline is synthesized from phosphatidyl dimethylaminoethanol (DMAE), itself synthesized from phosphatidyl ethanolamine (PE) and phosphatidyl serine (PS), reactions dependent on good methylation. That's where B vitamins, zinc and and magnesium are needed. While each of these nutrients has demonstrated mild memory promoting effects the combined therapeutic effect of these 'acetylcholine friendly' nutrients has yet to be adequately explored in the prevention or treatment of AD patients.

Also of interest in the amino acid pyroglutamate, from which the drug Piracetam (and various other nootropic drugs) is derived. Early animal research indicated a potential to increase acetylcholine reception. A recent meta-analysis of studies demonstrates a difference between those individuals treated with piracetam and those given placebo.

Herbs-ginkgo Biloba and Vinpocetine:

The herbs Ginkgo Biloba and the herbal extract Vinpocetine, have also demonstrated potential memory enhancing effects in the elderly. While a systematic review of all research up to 2002 concluded 'promising evidence of improvement in cognition and function with Ginkgo' three recent randomized trials on Gingko have failed to confirm earlier positive results for those with cognitive impairment, however one showed mild improvement for those who were not diagnosed with dementia. Ginkgo may therefore have a role to play in prevention. Research on Vinpocetine, an extract of the Periwinkle plant, is also promising, but in its infancy.

Study in Iran:

In studying by scholars,100 persons between 65 and 80 years That were studied from 100 patients,80 of them,the Alzheimer's disease patients were not despite old age and20 of them suffering from Alzheimer's had in terms of living conditions,that had almost status a good stress, social activity.of the even 20 percent subjects of genetic status had similar The studies showed that 80 percent of those Alzheimer's disease patients were not people who were in the diet daily foods such as olive oil,walnuts,fish,chicken meat,vegetables such as Broccoly and fruits .That are there especially green leafy vegetables.In general the was formed structure of these people food compounds omega3,omega6,vitamin E,and vitaminB12.the 20 percent of people who had family structure,40percent were diagnosed with Alzheimers patients in the diet ther was a lot of animal fatty acid and red meat.
Result:

This study showed that although Alzheimer's cure does/the can be improved conditions living the reduce stress and increase social activity and especially the control diet, the incidence avoided. The foods in rich substances such as omega 3, omega 6, Vitamin A and Vitamin B12, there is more recommended and those 60 years above of age must have there is reduced animals fatty acids severely in the diet of red meat.

REFERENCES


