USANA Technical Bulletin

Alzheimer's

General Description

- Alzheimer's disease is a progressive neurodegenerative disorder which primarily affects brain structures involved in memory processes and motor skills. Alzheimer's disease is the most common form of dementia, which generally refers to a progressive decline in mental function, memory and acquired intellectual skills. [1]
- Pathologically, the brain is reduced in size (atrophy), especially in the frontal occcipital and temporal regions. Histologically, it is characterized by thickening, conglutination, and distortion of the intracellular neurofibrils (neurofibrillary tangles) and by plaques composed of granular or filamentous masses, found predominately in the nerve cells of the cerebral cortex, amygdala, and hippocampus. [1]
- Major clinical criteria for the clinical diagnosis of probable Alzheimer's include: 1) deficits in two or more areas of cognition, 2) progressive worsening of memory, 3) absence of other medical or psychological disorders that could account for memory impairment and 4) clear consciousness despite memory impairment.

Causes

- The exact cause of Alzheimer's is unknown. The etiology is complex and may involve several genes and possible environmental factors.[2]
- Chronic exposure to aluminum has been suggested as a possible causative agent in Alzheimer's. However, clinical evidence for this link is inconclusive.[3]
- Oxidative stress may play a role in the pathogenesis of neuron degeneration and death in Alzheimer's [4,5] An increase in free radical production has been demonstrated in Alzheimer's disease brain tissue.
- In particular, iron has been shown to be a significant component of senile plaques in Alzheimer's disease[6] and may contribute to the disease process by initiating lipid peroxidation, leading to membrane damage and ultimately cell death.[7]

At Risk

- Alzheimer's disease is obviously related to age, - that is, the older you get the more likely you are to develop Alzheimer's disease or dementia. For instance, the prevalence of dementia is roughly 3% for individuals aged 65 to 74, whereas it is 18.7% for individuals between the ages of 75 and 84 and nearly 50% for those over age 85. In

addition, there is evidence to suggest that genetic factors may play a part in some forms of AD.

- Clinical manifestations of mental deterioration, memory loss, confusion, and disorientation may begin in late mid-life (>45 years old). Death usually results in about 5 to 10 years after diagnosis.

Prevention and Management

- Nutritional support is important in the treatment of Alzheimer's.[2]
- In patients with moderately severe impairment from Alzheimer's disease, treatment with alpha-tocopherol (vitamin E) or selegiline slows the progression of disease.[8]
- There is an association between Alzheimer's and low serum cobalamin (vitamin B12) levels.[9]
- Deficiency of choline, an important component of membrane phospholipids and the neurotransmitter acetylcholine, may play a role in the etiology of Alzheimer's[10]

Abstracts

Sano M et al. A controlled trial of selegiline, alpha-tocopherol, or both as treatment for Alzheimer's disease. N Engl J Med 1997 Apr 24;336(17):1216-22. BACKGROUND: There is evidence that medications or vitamins that increase the levels of brain catecholamines and protect against oxidative damage may reduce the neuronal damage and slow the progression of Alzheimer's disease. METHODS: We conducted a double-blind, placebo-controlled, randomized, multicenter trial in patients with Alzheimer's disease of moderate severity. A total of 341 patients received the selective monoamine oxidase inhibitor selegiline (10 mg a day), alpha-tocopherol (vitamin E, 2000 IU a day), both selegiline and alpha-tocopherol, or placebo for two years. The primary outcome was the time to the occurrence of any of the following: death, institutionalization, loss of the ability to perform basic activities of daily living, or severe dementia (defined as a Clinical Dementia Rating of 3). RESULTS: Despite random assignment, the baseline score on the Mini-Mental State Examination was higher in the placebo group than in the other three groups, and this variable was highly predictive of the primary outcome ($P<0.001$). In the unadjusted analyses, there was no statistically significant difference in the outcomes among the four groups. In analyses that included the base-line score on the Mini-Mental State Examination as a covariate, there were significant delays in the time to the primary outcome for the patients treated with selegiline (median time, 655 days; $P=0.012$), alpha-tocopherol (670 days, $P=0.001$) or combination therapy (585 days, $P=0.049$), as compared with the placebo group (440 days) CONCLUSIONS: In patients with moderately severe impairment from Alzheimer's disease, treatment with selegiline or alpha-tocopherol slows the progression of disease.

References

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