

# Alzheimer's disease

The elderly are especially at risk for malnutrition. Disease, depression, polypharmacy, poverty, inadequate care, malabsorption and social isolation are contributing factors. The 1993 Nutrition Screening Initiative found that 1/4 of all elderly patients and 1/2 of all hospitalized elderly may suffer from malnutrition. The study results prompted a spokesperson for the American Dietetic Association to state: "Our health care system is a little malnourished itself." In long-term care settings, where it is estimated that from 10 percent to 85 percent of older adults may be malnourished, the nutrition provider "must carefully consider many complex physical, medical and psychosocial factors to deliver individualized nutritional care."

A European initiative (the "Health Promotion Program") has been undertaken to prevent weight loss in patients with Alzheimer's disease. Weight loss in Alzheimer's patients was first observed by Alois Alzheimer in 1901. A number of studies have consistently observed weight loss in patients with dementia and, in particular, Alzheimer's disease. This weight loss increases the risk of infections, skin ulcers and consequently decreases quality of life in Alzheimer's patients. The cause is certainly related in part to reduced functional capacity in Alzheimer's patients, but other hypotheses hold that neuro-endocrine or metabolic factors are implicated. One study has even correlated the burden the disease places on the caregiver with the degree of weight loss in the Alzheimer's patient. This implies that supportive measures aimed at relieving caregivers' stress may indirectly impact patient's nutritional status.

The progression of neurological disease also may be slowed by application of antioxidant support. In the treatment of Parkinson's disease, Kedar N. Prasad of the University of Colorado Health Sciences Center proposes an antioxidant-laden

cocktail of natural beta-carotene (30mg/day), d-alpha tocopheryl succinate (600 IU/day), vitamin C (4g/day), coenzyme Q10 (200mg/day), NADH (10mg/day), N-acetyl-cysteine (500 mg/day), zinc (30 mg/day) and selenium (200 mcg/day).

Inflammation may contribute to the progression of neurodegenerative diseases. Attenuation of inflammation can be achieved via the aforementioned antioxidant support; it also can be accomplished via nutritional modulation of prostaglandin metabolism. Administration of essential fatty acids such as EPA and GLA and supplementation with nutrient co-factors such as zinc and vitamin C that favor endogenous production of anti-inflammatory eicosanoids can positively impact many disorders.

Defects in methylation results from aberrant homocysteine metabolism. Homocysteine may play a role in the etiology of cardiovascular disease, stroke, venous thrombosis, arthritis, Alzheimer's disease and vascular dementia. The key nutrients for reversal of homocysteinemia are vitamin B6, vitamin B12 and folic acid. Screening for vitamin B12 deficiency in the elderly can yield a high number of abnormal results even in the absence of anemia. Vitamin B12 deficiency may result from malabsorption or lack of hydrochloric acid common in the elderly. Measurements of serum methylmalonic acid and homocysteine are more sensitive methods for confirmation of B12 deficiency than radioisotope assays or serum B12 levels. In one study of 86 B12 deficient patients who responded to cobalamin therapy, neuropsychiatric abnormalities were noted in 28 percent, many times in the absence of anemia.

Once relegated to the status of a rare genetic disorder, the concept of mitochondrial dysfunction is taking on new importance in a more nuanced approach to understanding of degenerative processes. Ensuing nutritional deficiencies attendant to aging or debilitation might further compromise efficiency of ATP production. The results could be failure of energy production in the cells of the brain. Nutrients that

support mitochondrial function include coenzyme Q10, vitamin E, riboflavin, niacin, l-carnitine and magnesium. Recently, a new "stabilized" form of NADH for oral administration ("Enada") has been proposed for treatment of dementia, Parkinson's disease, chronic fatigue syndrome and other debilitating ailments.

Even hidden food intolerances may contribute to the development of Alzheimer's disease, for example, atypical presentation of coeliac disease in the absence of classic intestinal manifestations. Recent research has demonstrated a high prevalence of antigliadin antibodies (IgG, IgA or both) in patients with neurological dysfunction of obscure etiology (57 percent versus 5 percent in neurological controls and 12 percent in normal controls). There was a nearly tenfold increase in neurological dysfunction or dementia in gluten-sensitive individuals compared to those not gluten-sensitive. Detection was made more elusive by the fact that only 35 percent of patients who developed neurological problems had histological evidence of celiac disease. Two-thirds had no clinical signs of celiac disease.

The following is an actual case study of a patient with Alzheimer's disease treated at our center. E.K. began developing memory lapses at the age of 73. She became increasingly forgetful, crying every morning, believing her dead parents were alive, getting lost while going out alone, hiding her possessions, accusing her family of wanting to steal her things. Over a course of three years she developed profound memory loss and began to forget who her immediate family was. A neurologist rendered a diagnosis of Alzheimer's dementia. Blood tests performed by her neurologist revealed a normal SMAC and CBC. A serum B12 was 280, considered in the normal range. She was started on tacrine, with minimal response.

When first seen at our center at the age of 76, E.K. could state her name but did not know where she was or the date. Her

physical exam was otherwise unremarkable, but she was uncooperative and suspicious. Homocysteine was found to be elevated at 16.8 (optimal <10) and methylmalonic acid (a sensitive measure of B12 status) was found to be elevated at 455 (normal 73-271 nm/L). Antigliadin IgA (but not IgG) was positive at 1:40. Serum DHEA sulfate was noted to be low at 17. (Age-appropriate norm >25.)

The patient was given a series of B12 shots 3 times weekly for two weeks, then started on monthly injections. She was started on a gluten-free diet. She was placed on a combined regimen of nutritional supplements, which were faithfully administered to her by her son who lived with her, as follows:

Multivitamin

Vit E 800 IU

Folate 5mg

B6 25mg

Vit C 4 gms

Inositol 12 gms

DHEA 25 mg

Thiamine 400mg

Alpha-lipoic acid 2,000 mg

Acetyl-L-carnitine 1,000mg

DHA (Docosahexaenoic acid) 1,000 mg

Ginkgo biloba extract 320 mg

Coenzyme Q10 200 mg

NADH 10mg

N-acetylcysteine 200mg

Phosphatidylserine 300 mg

The patient underwent gradual improvement in memory, mood and functional status. An early observation by her family was that she became relatively calmer and day/night reversal of her sleep pattern abated. She no longer could not be left alone, which had in the past prompted panic and calling out of windows that she needed help. Spells of anger, paranoia and obstinacy became less frequent. The patient's ability to name objects returned, and her ability to dress, bathe and eat with minimal assistance returned. After two years, her improvements were so significant that her neurologist noted the following on an insurance report:

"Patient seems to have recovered significant memory over the last 2 years from natural process/or the employment of vitamin supplements in collaboration with family members. Improvement has been seen especially in areas of ADL (Activities of Daily Living), i.e. independence in self dressing, eating and light cooking. There is absolutely no issues [sic] about continence. There is no evidence of alteration of sleep-wake cycle, mood changes, agitation, wandering or other affective or personality disorders. She has reached a stable plateau in her neurological state with no evidence of progressive deterioration. I would presently classify her as having minimal dementia in the the order of Age Related Memory Loss."

**Discussion:** This is an example of the potential of multifaceted nutritional intervention in the face of a seemingly intractable progressive neurological disease. To what extent each component of the complex regimen undertaken by E.K. affected her outcome is unclear. But the essence of targeted nutritional therapy is the synergy between diverse agents simultaneously addressing multiple regenerative pathways.

Of interest in this case is the apparent efficacy of B12 shots, despite a normal B12 blood test. Elevated homocysteine, despite E.K.'s unremarkable lipid profile, may have set the stage for a component of vascular dementia. The trio of B12, folate and B6 help to reverse elevated homocysteine and may improve cognitive functioning.

High-dose inositol supplementation, in addition to its value in relieving symptoms of depression and anxiety, may ameliorate dementia. Thiamine also may play a role in optimizing cerebral function in Alzheimer's Disease. The effects of an "antioxidant cocktail" and mitochondrial enhancers such as CoQ10, NADH and carnitine in neurodegenerative disease have been alluded to here previously, and DHA and phosphatidylserine may help brain function. (Low red blood cell levels of omega 3 fatty acids and especially DHA have been correlated with depression). The adrenal androgen DHEA, replaced after found to be low in E.K., is responsible for improvements in memory, well-being and sleep quality in dementia. Finally, ginkgo biloba has an increasingly established role in ameliorating Alzheimer's symptoms.

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