Dementia



Life Style and Alzheimer's Disease

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Introduction:

Erik Ericson (1993) has identified eight stages of psychosocial development behaviors that may be associated with healthy and unhealthy expressions of the self's development and ego boundary growth. During the Late Adulthood stage and reflecting on life, the elderly person may experience satisfaction, or a sense of failure. This is identified as achieving either integrity or despair.

Expressions of achieving integrity, are where the person is self approving, proud, and content with self and life. He/she remains active in thinking about the future, and is comfortable giving and sharing with others. Also being an example to others and accepting the aging process and death gracefully, as part of life cycle.

Expressions of achieving despair and distrust is personified with a feeling of low self-esteem, deep resentment, and uselessness. He/she is angry at self, others, and society, resulting in being closed off to others. The person does not like being old, is irritable, feels cheated, and complains frequently.

Brown (2014) views chronological age as a predictor of health and functional abilities, as a proxy measure. Determining how well a person accomplishes tasks of daily living is a better indicator of health and functional status. During this life stage, gradual mental and physical decline occurs, and the person is tasked with adjusting to these changes, that will eventually result in the person's death. Many elders are also adjusting to retirement and a decrease in income. This often results in roles changing in

terms of social and civil obligations. Loss, such as the deaths of a spouse and friends occur, and maintaining social relationships may be a challenge.

As the culmination of old age is death, the elderly have to develop a vision and world view about death to prepare themselves for the impending future. They have to find some positive thoughts and aspects about death and accept the fact. Religious views and philosophies on death can contribute to this vision.

Alzheimer's disease:

Old people often have limited regenerative abilities and are more susceptible to disease, syndromes, and sickness than younger adults. There is a decline in lean body mass, weight gain, changing sensual awareness, and nutritional risk factors (Brown 2014). Memory impairment and mild cognitive impairment (MCI) often appear in Late Adulthood. Alzheimer's disease ranks as the fifth leading cause of death for adults aged 65 and older in the United States.

Definition of the need or condition:

Alzheimer's disease (AD) is an irreversible, progressive disorder in which neurons (brain cells) deteriorate, resulting in the loss of cognitive (thought) functions, primarily memory, judgment, reasoning, movement coordination, and pattern recognition. In advanced stages of the disease, all memory and mental functioning may be lost. At present, it is a terminal illness. Abnormal changes in the brain worsen over time, eventually interfering with many aspects of brain function. Memory loss is one of the earliest symptoms, along with a gradual decline of other intellectual and thinking abilities, called cognitive functions, and changes in personality or behavior.

Diagnosis and management of the disease:

There is no one test to diagnose Alzheimer's disease (AD). Typically, doctors start the diagnostic process by ruling out other diseases and conditions that may also cause memory loss. Genetic factors are known to play a role in some cases of Alzheimer's disease (AD). A gene, called the amyloid beta precursor protein (APP) gene, has been linked to the occurrence of AD in Down's syndrome patients who survive beyond 40 years. The causes of Alzheimer's disease (AD) are poorly understood, but its effect on brain tissue has been demonstrated clearly. AD damages and kills brain cells. Plaques and tangles in brain tissue are considered hallmarks of Alzheimer's disease. Studies of plaques and tangles from the brains of people who have died of AD suggest several possible roles these structures might play in the disease.

In individuals with AD, changes in the brain may begin 10-20 years before any visible signs or symptoms appear. Some regions of the brain may begin to shrink, resulting in memory loss and the first visible sign of AD. Over time, AD progresses through three main stages including mild (early), moderate, and severe.

The specific goals of treatment are to preserve cognitive and functional ability, minimize behavioral disturbances, slow disease progression, and provide patients with a high level quality of life. Although current drugs cannot alter the progressive loss of cells, they may help minimize or stabilize symptoms. These medications may also delay the need for nursing home care. Cholinesterase inhibitors (donepezil, rivastigmine, and galantamine) are effective for mild to moderate Alzheimer's disease, and memantine for moderate to severe Alzheimer's disease. Until further evidence is available, other drugs including statins, anti-inflammatory drugs, vitamin E and Ginkgo biloba, cannot be

recommended either for the treatment or prevention of Alzheimer's disease. An increasing number of psychosocial therapies are now available for people with dementia, including behavioral therapy, reality orientation, art therapy, music therapy, complementary therapy, aromatherapy and bright-light therapy, as well as cognitive behavioral therapies (Natural Medicines, 2015).

Discussion of the role nutrition plays in managing the need or condition:

According to Web MD, "there's no special diet for people with Alzheimer's disease, but good nutrition can ease some symptoms and help them feel good." The following is recommended:

- Eat a variety of foods, especially fruits and vegetables, whole grains, lean protein, and low-fat dairy.
- Keep a healthy weight. Proper portion sizes and exercise are a key part of this.
- Limit foods with high saturated fat and cholesterol, like fatty meats, processed meats, and fried foods.
- Cut down on sugar.
- Avoid eating too much salt.
- Avoid processed grains to include alcohol.
- Drink plenty of water. (WebMD, 2015)

Deficiencies of vitamin B 12 and folate are related to high concentrations of homocysteine, an amino acid associated with the promotion of poor vascular health and cognitive decline. In order to prevent the buildup of homocysteine in the blood and neural tissue, vitamin B 12 and folate are needed to convert it to the amino acid

methionine. Excess homocysteine in brain tissue is thought to contribute to the development of Alzheimer's disease either through vascular mechanisms or as a neurotoxin. High-dose vitamin B supplementation has decreased homocysteine levels but has not slowed progression of cognitive decline in people with AD (Aisen, 2008). The Mediterranean diet has shown promise in delay of cardiovascular disease and prolonging longevity. Researchers in a prospective cohort study in Bordeaux, France, developed an adherence score and assessed the dietary patterns of 1410 adults aged 65 and older (Feart, 2009). After adjustment for cardiovascular risk factors, higher adherence to the Mediterranean diet was associated with slowed cognitive decline in one of the four cognitive function tests (the Mini- Mental State Exam). In contrast, a prospective cohort study of 1880 older adults in New York showed that the Mediterranean-type diet and physical activity were independently related to lower AD risk (Scarmeas, 2009).

Other promising research into the mechanisms of cognition in aging, deals with the theory of caloric restriction and inflammation (Witte, 2009). A small intervention study in humans (29 healthy women, mean age 60.5 years) showed that a reduced- calorie diet (30 percent below normal in-take, not to drop below 1200 kcal) improved insulin sensitivity, inflammatory response (tumor necrosis factor-alpha), and memory performance on the Rey Auditory Verbal Learning Task test. Both obesity and long-term underweight were associated with lower cognitive scores in the larger Whitehall Cohort Study (Sabia, 2009).

There is growing evidence for possible dietary interventions in the development of AD and cognitive decline with age, such as antioxidant nutrients, fish, dietary fats, and

B-vitamins. Numerous animal and laboratory studies have shown that antioxidant nutrients can protect the brain from oxidative and inflammatory damage, but there are limited data available from epidemiological studies. There is more substantial epidemiological evidence from a number of recent studies that demonstrate a protective role of omega-3 fatty acids, such as docosahexaenoic acid, in AD and cognitive decline (Morris, 2009).

Isaacson and Ochner (2015) reviewed many dozens of studies and identified dietary interventions that included the Mediterranean diet, omega-3 fatty acids, antioxidants, B vitamins, and low-carbohydrate diets. On the basis of this review, a combination of B vitamins (folic acid, B6, and B12) probably improves cognitive impairment in MCI (mild cognitive impairment), whereas a Mediterranean diet may improve cognitive function in AD and probably decreases the risk for AD in both MCI patients and non-demented persons. Omega-3 fatty acids are likely to decrease cognitive impairment in MCI, and flavonoids (strawberries and blueberries) may delay symptoms.

Nutrition must not be considered alone but must be joined with the following interventions in response to the treatment of Alzheimer's disease, representing a holistic approach.

- Healthy diet
- Regular exercise
- Quality sleep
- Mental stimulation
- Stress management
- An active social life.

http://www.helpguide.org/articles/alzheimers-dementia/alzheimers-and-dementia-prevention.htm

References:

- Aisen, P. S., Schneider, L. S., Sano, M. et al. (2008). High- dose B vitamin supplementation and cognitive decline in Alzheimer disease: A randomized controlled trial. JAMA; 300(15): 1774–1783.
- Brown, J. (2014). *Nutrition through the life cycle*. Stamford, CT: Cengage Learning Feart, C., Samieri, C., Rondeau, V. et al. (2009). Adherence to a Mediterranean diet, cognitive decline, and risk of dementia. JAMA; 302(6): 638–648.
- Erickson, E. (1993). *Childhood and society*. New York, NY: W. W. Norton & Company. Glazer, H. et al. (2014). Evidence on diet modification for Alzheimer's disease and mild cognitive impairment. Program and abstracts of the 66th Annual Meeting of the American Academy of Neurology. Abstract P5.224.
- Help Guide (2015). Retrieved from http://www.helpguide.org/articles/alzheimers-dementia/alzheimers-and-dementia-prevention.htm
- Isaacson, R. & Ochner, C. (2015). The Alzheimer's prevention and treatment diet. Miami. FL: AD Education Consultants Inc.
- Morris, M. (2009). The role of nutrition in Alzheimer's disease: Epidemiological evidence. *Eur J Neurol. Suppl (1) 1-7.*
- Natural Medicines (2015). *Alzheimer's disease*. Natural Medicines Bottom Line Monograph.Somerville, MA: Natural Medicines.
- Sabia, S., Kivimaki, M., Shipley, M., et al. (2009). Body mass index over the adult life course and cognition in late midlife: The Whitehall II Cohort study. Am J Clin Nutr; 89: 601–7.
- Scarmeas, N., Luchsinger, J. A., Schupf, N. et al. (2009). Physical activity, diet, and risk of Alzheimer's Disease. JAMA; 302(6): 627–637.
- WebMD (2015). Retrieved from http://www.webmd.com/alzheimers/guide/alzheimersnutrition
- Witte, A. V., Fobker, M., Gellner, R. et al. (2009). Caloric restriction improves memory in elderly humans. Neuroscience, National Academy of Sciences of the USA. PNAS; 106(4) 1255– 1260.