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What's My Age Again? Dementia and the Impact on an Aging Population

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Introduction

- Dementia describes a complex syndrome arising from abnormal brain function and impacting cognitive abilities, decision making, and behavior (World Health Organization [WHO], 2020).

- Some causes of dementia may be reversible, if induced by pharmacological agents, infections, or as secondary disease. However, in many cases, dementia is progressive (WHO, 2020).

- Dementia is an overarching term used to refer to many discrete causes of cognitive decline(Alzheimer's Association, 2020).
  - Alzheimer's Disease
  - Lewy Body Dementia
  - Vascular Dementia
  - Frontotemporal Dementia
  - Parkinson's and Huntington's Dementia
  - Mixed Dementia

- Dementia influences the physical, psychological, emotional, social, and economic well-being of patients and families (WHO, 2020). Financial costs significantly impact the quality of life of the entire family unit and complicate to decision making.

- Dementia is currently the "fifth leading cause of death for Americans over the age of 65," affecting 5 million adults in the United States and accruing $236 billion in care costs (Falk et al. ,2018).

- Worldwide incidence of dementia is expected to triple by 2050 (WHO, 2020), bringing the number of affected American seniors to 14 million, and increasing the American cost of dementia care to $1 trillion (Falk et al., 2018).

- Increasing incidence and cost adds urgency to the development of diagnostic and treatment modalities.

- Dementia is complex and practitioners must be educated about all aspects of dementia care to provide effective holistic treatment.

Figure 1. Dementia as an overarching term. Retrieved from https://www.alz.org/alzheimers-dementia/what-is-dementia

Signs and Symptoms

- Cognitive domains affected (Falk et al., 2018):
  - Language
  - Complex Attention
  - Executive Function
  - Learning and Memory
  - Perceptual-Motor
  - Social Cognition

- Early stage: Gradual cognitive decline. Changes include easily dismissed memory problems (WHO, 2020).
  - Forgetfulness, such as missed appointments or misplaced keys
  - Disorientation or getting lost in familiar places
  - Losing track of time (WHO, 2020)
  - Intermittent difficulty remembering the correct word or name (Alzheimer's Association, 2020)
  - Difficulty performing some tasks of independent living, such as paying bills

- Middle stage: Symptoms become more evident and well defined as dementia (WHO, 2020).
  - Behavioral changes, such as wandering, insomnia, and repeated questioning
  - Delusional and compulsive behavior
  - Becoming lost at home (WHO, 2020)
  - Difficulty with communication and remembering names of loved ones
  - Bowel and bladder incontinence
  - Anomic aphasia – inability to name easily recognizable objects and their purpose
  - Difficulty with planning or completion of daily tasks; need for assistance (WHO, 2020)

- Late stage: Near total dependence and lack of activity. Severe memory deficits and progression to being bed-bound. Lack of awareness or acknowledgement of surroundings (WHO, 2020).
  - Behavioral escalation including inappropriate vocalization and aggression (WHO, 2020)
  - Difficulty with mobility, progressing to inability to balance well enough to stand
  - Difficulty swallowing – increasing risk for aspiration pneumonia
  - Lack of ability to perform hygiene related tasks (Alzheimer's Association, 2020)

Neuro-psychological and behavioral symptoms (Kazui et al., 2016):

- Agitation
- Depression
- Hallucinations
- Anxiety
- Euphoria
- Apathy
- Disinhibition
- Irritability
- Aberrant motor behavior
- Sleep disturbances
- Eating Abnormalities

Pathophysiology
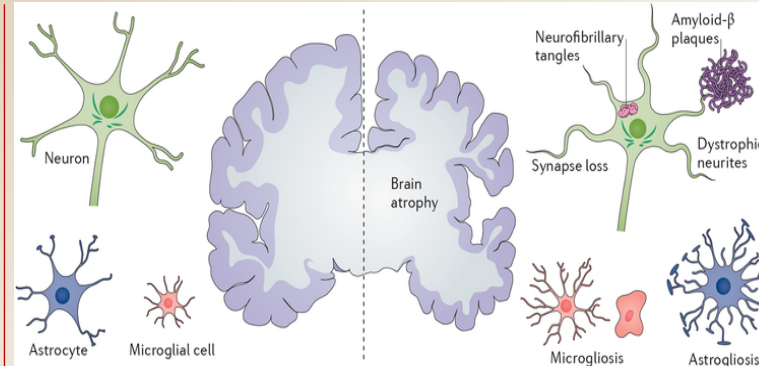
Figure 2. Graphic depiction of gross anatomy and histology of a normal brain and neural cells in comparison to an Alzheimer's affected brain and neural cells. Retrieved from https://europepmc.org/article/PMC/6463489#free-full-text

- Alzheimer's Disease – exhibition of diffuse neuronal degeneration, neuroinflammation, blood brain barrier dysfunction, and activation of glial cells (Raz et al., 2015)
  - Accumulation of extracellular senile plaques
  - Neurofibrillary tangles inside the neuron
  - Beta-amyloid peptide and tau protein are involved in neurodegeneration

- Dementia with Lewy Bodies – vascular changes cause hypoperfusion and protein agglutination, a profound inflammatory process occurs, and glial activation is present (Raz et al., 2015)
  - Abnormal accumulation of alpha-synuclein at synapses

- Frontotemporal Dementia – brain atrophy and neuronal degeneration is concentrated in the frontal and temporal lobes, glial activation is present, and aggregation of proteins is present (Raz et al., 2015)
  - Tau and ubiquitin protein aggregations are responsible for neural degeneration
  - Vascular dysfunction may play a role in disease development

- Vascular Dementia – causes include disease of large vessels, small vessels, and cardioembolic disease; vascular changes cause permeability of the blood brain barrier and neuronal hypoxia (Raz et al., 2015)
  - Hypertension is thought to be a risk factor
  - Lacunar infarct and white matter hyperintensities are present

- Mixed Dementia – Alzheimer's disease and vascular dementia are often seen as co-pathologies; unclear which pathology is initially developed (Raz et al., 2015)
  - Beta-amyloid plaques are present in the same areas as evidence of hypoperfusion
  - Studies of mixed dementia are limited
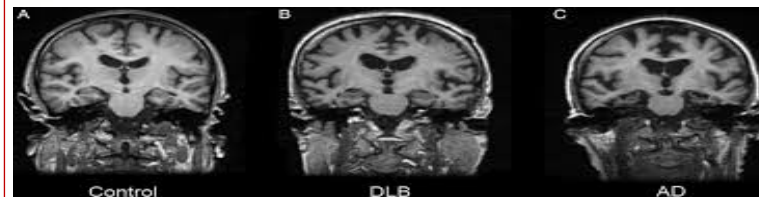
Figure 3. Magnetic resonance imaging depicting a normal control brain (A) in comparison with a brain with Lewy Body Dementia (B) and a brain with Alzheimer's Disease (C). Notice the relatively preserved temporal lobes in Lewy Body Dementia. Retrieved from https://jnnp.bmj.com/content/jnnp/86/1/50.full.pdf

Significance of Pathophysiology

All forms of dementia involve pathophysiological processes exhibited in other diseases, such as type II diabetes, hypertension, and obesity. By researching the similarities present in the inflammatory reactions and vascular degeneration of neural tissue in dementia patients to the inflammatory markers and vascular remodeling processes involved in other chronic illnesses, targets for pharmacologic reversal and management may be discovered.

In a research population observed by Clark et al. (2020), asymptomatic adults with increased beta-amyloid levels did not experience increased risk of cognitive decline compared with the control group. When adults with increased beta-amyloid levels were also obese, hypertensive, or obese and hypertensive (displaying vascular risk factors) the annual risk of cognitive decline doubled (Clark et al., 2020). While cognitive decline may still occur for study participants, data indicates a potential for individuals with increased beta-amyloid and hypertension or obesity to decline significantly more rapidly than individuals without vascular risk factors (Clark et al., 2020).

If a causal link is discovered between hypertension and obesity leading to blood brain barrier dysfunction, reduced clearance of beta-amyloid, neuronal atrophy, and ultimately cognitive decline, early mitigation or reversal of hypertension or obesity may slow cognitive decline (Clark et al., 2020).

Tussing-Humphreys et al. (2020) utilized dietary changes to manipulate cardiovascular risk factors previously linked to obesity. Obesity correlates strongly with cognitive decline, cognitive impairment, and dementia (Tussing-Humphreys et al., 2020). In the United States many adults over the age of 60 are obese and eat a poor diet (Tussing-Humphreys et al., 2020). Obesity, increased blood lipids, inflammatory biomarkers, and poor glucose metabolism can all be reversed through intentional dietary choices, avoiding high fat, high calorie foods. In turn, risk for cognitive decline will decrease.

Walker and Harrison (2015) researched the significance of symptoms found in both type 2 diabetes and Alzheimer's disease. Specifically, cognitive deficits, inflammation, oxidative stress, glucose intolerance, and impaired insulin signaling/resistance appear to link Alzheimer's disease with type 2 diabetes (Walker & Harrison, 2015). Impaired insulin signaling and obesity are linked to pro-inflammatory cytokines, some of which (tumor necrosis factor and interleukins) can cross the blood brain barrier and affect the neural tissue. Obesity is also associated with an increased number of macrophages and decreased grey matter density(Walker & Harrison, 2015). By decreasing the amount of dietary fat consumed, inflammation is greatly decreased.

While dietary changes and decreasing inflammatory biomarkers will not stop the pathological processes involved in development of Alzheimer's disease, risk factor modification is a viable method of slowing cognitive decline and improving quality of life.

Implications for Nursing Care

- As an advanced practice nurse, holistic care is pivotal. Frequently, patients with dementia are unaware of the implications of their diagnosis and are left out of medical decisions (Lin, 2017). Ensuring that access to care, treatment choices, coordination of care, and diagnostic tests are available to patients and families dealing with dementia allows care providers to support the emotional and cognitive well-being of their patients.

- Primary care providers must be proficient in recognizing and evaluating cognitive decline, as patients will likely have more contact with a primary care provider than any other care provider (Falk et al., 2018).

- Involving patients and caregivers in formulating a care plan allows patients to be in control to the extent of their cognitive abilities (Lin, 2017).

- Counseling/educating patients and caregivers about modifiable risk factors and supporting healthy changes, while prescribing pharmacological agents as appropriate allows families to actively embrace treatment.
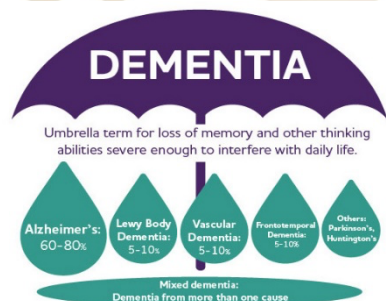
Conclusions

Currently, there is no cure for dementia. Treatment is not aimed at reversing or halting damage to neuronal tissue. Goals of treatment include maximizing quality of life for the patient and caregiver, slowing disease progression, and modifying risk factors as possible. Advanced practice nurses are poised to change lives through early diagnosis and holistic dementia care.

References

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