Bone Mineral Density, Adiposity, and Cognitive Functions

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Abstract

Alzheimer's Disease (AD)-related cognitive impairment and dementia have been linked to a variety of genetic, environmental, and lifestyle variables. When investigating preventative or ameliorative interventions aimed at dementia and its preclinical stages, a variety of potentially modifiable risk variables should be taken into account. This study looked into the relationship between two such potentially modifiable risk factors −Bone Mineral Density (BMD) and body composition−and cognitive impairment. For this longitudinal study, 164 patients were enrolled, who were tested cognitively and clinically at baseline and three years later. Their ages ranged from 34 to 87 .

Dual Energy X-Ray Absorptiometry (DXA) was performed on the same day as the cognitive assessment, and blood samples were taken for Apolipoprotein E (APOE) genotyping. We discovered through hierarchical regression analysis that BMD and lean body mass, as determined by DXA, were important predictors of episodic memory. Premorbid IQ, APOE status, age, and gender were taken into account. Particularly, BMD and lean mass were substantially correlated with List A learning from the California Verbal Learning Test at both the baseline and follow-up assessments. Our results show a substantial relationship between BMD, lean body mass, and episodic linguistic learning.

Keywords: Dual Energy X-Ray Absorptiometry •Bone Mineral Density• Adiposity

Introduction

The contemporary aging population is greatly concerned about dementia because it is a seriously debilitating condition. More than 35.6 million people worldwide had dementia diagnoses in 2010. By 2050, dementia cases will have substantially increased worldwide, according to prevalence forecasts. In particular, according to a prediction from the Australian Institute of Health and Welfare, by the year 2031, there would be 465,000 more dementia sufferers in Australia than there are currently (175,000). To address the financial and societal effects of this disorder, preventative research to lessen the burden connected to dementia is crucial.

According to recent modeling cited by Alzheimer's Australia, if dementia was delayed by two years, the number of new cases would be reduced by 13%, or 398,000 cumulatively, by 2050. Furthermore, a 5-year wait would result in a cumulative reduction in new cases of 30%, or 935,000 people, by 2050. Programs to prevent dementia would have a big economic impact and raise the standard of living for those who have it and their families. A possible approach to assisting dementia incidence decreases is the identification of potentially modifiable risk factors, including lifestyle factors [1-2].

The most prevalent type of dementia in the world is dementia brought on by Alzheimer's Disease (AD). The formation of extracellular amyloid deposits is thought to be caused by the cerebral accumulation of a small peptide called beta-amyloid (A), which is caused by a complex combination of genetic, lifestyle, and hormonal factors. The underlying causes of the late-onset form of the disease are still unknown. According to research, 30% of cases of AD are avoidable.

Cognitive impairment and dementia have been linked to osteoporosis and low BMD (osteopenia) .BMD is controlled by the brain, which may help to partially explain the connection between BMD, dementia, and cognitive decline. The areas of the hypothalamus, which are involved in obesity, also control bone remodelling through laborious and sluggish processes involving hormones like leptin. According to researchers, leptin is hypothesized to mediate BMD via binding to specific receptors in the ventromedial hypothalamus. This suggests that osteoporosis may be a neuro-skeletal disorder [3].

Noteworthy is the negative correlation between plasma leptin levels and the risk of dementia and AD. Additionally, the association between lower BMD and dementia may be influenced by cumulative oestrogen exposure, as the Framingham Study discovered that lower femoral neck BMD was linked to a two-fold increase in the risk of AD in women, possibly as a result of estrogen exposure.

Another potentially modifiable risk factor for dementia and cognitive decline is body fat or adiposity. However, research on this topic has yielded rather contradictory results. While the majority of studies have shown a significant correlation between obesity and cognitive decline, some studies have not been able to find a significant correlation between these two factors and some of the cognitive functions linked to AD, such as verbal memory. For instance, it has been proposed that obesity, which increases the risk of insulin resistance and hyperinsulinemia, may increase amyloid deposits in the brain, causing AD. In conclusion, the evidence indicates that midlife central obesity plays a significant role in age-related cognitive decline and greatly raises the risk of dementia.

Increasing obesity and osteoporosis have both been linked to Cardiovascular Disease (CVD), which has been linked to AD-plasma amyloid-protein and has been found to raise the risk of cognitive decline and dementia. Surprisingly, BMD is negatively correlated with CVD, and subclinical CVD has been shown to increase the risk of bone loss and fracture. Additionally, there is a link between osteoporosis and cardiovascular issues, and lipid-related issues may contribute to an increased risk of osteoporosis. The link between osteoporosis and atherosclerosis has been demonstrated in animal models. Higher atherogenic lipid profiles and lipoproteins are inversely related to bone density, according to observational research, although the precise processes behind this association are yet unknown. [4].

Clinical And Mental Tests

Between 1.5 and 2.5 hours of thorough clinical and neuropsychological evaluations were conducted with breaks available as needed. Using the GDS, depression was assessed at baseline. The Cambridge Contextual Reading Test (CCRT) was used to measure premorbid cognitive performance. With the CAMCOG-R, general cognitive abilities were evaluated. The CVLT, or California Verbal Learning Test, was used to measure verbal episodic memory. List Learning, Short Delay Free Recall (SDFR), Short Delay Cued Recall (SDCR), Long Delay Free Recall (LDFR), Long Delay Cued Recall (LDCR), and recognition discriminability are the CVLT's baseline and 3-year follow-up scores (RecDisc).

Genetic and Biochemical Analysis

A fasting venous blood sample was taken and placed in serum, EDTA (containing prostaglandin E to stop platelet activation), and heparin blood collection tubes on the same day as the DXA scan and cognitive/clinical evaluation. Then, using customary centrifugation methods, the whole blood was divided into its various components.DNA was extracted from leukocytes, and APOE genotype was identified using Polymerase Chain Reaction (PCR) amplification and restriction enzyme digestion [5].

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