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A Study Of Serum Homocysteine & Lipid Profile In Patients With Vascular Dementia

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Abstract:
Background*: Vascular dementia is a type of cognitive impairment that arises from pathological disorders that inflict damage upon the blood vessels in the brain or disrupt the normal circulation of blood and oxygen to the brain. Homocysteine can be metabolized to S-adenosyl homocysteine, which may exhibit neurotoxicity by inhibition of methylation reactions and thus monoamine neurotransmitter metabolism and protein and phospholipid methylation.*
Materials and Methods: *The present study was conducted in the department of Biochemistry, Jawahar Lal Nehru (J. L. N.) Medical College and its Associated Hospitals Ajmer. The study encompassed a total of 95 patients diagnosed with Vascular dementia.*
Results: *Serum homocysteine & lipid profile was found to be considerably greater in participants with vascular dementia than controls.*
Conclusion: *Higher levels of total cholesterol, triglyceride, serum homocysteine are associated with vascular dementia.*
Key Word*: Vascular dementia, homocysteine, LDL, HDL, Lipid profile.*

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I. Introduction

According to the World Health Organization (2010), the term **"dementia"** encompasses a collection of illnesses characterized by cognitive decline, which arises from the loss or impairment of brain cells. It is a syndrome involving chronic disease of the brain usually of progressive nature resulting in disturbance of multiple higher cortical functions such as memory loss, orientation, in thinking skills, comprehension, learning capacity, language and judgement. The state of consciousness is not characterized by muddled cognition. The cognitive function impairments are frequently accompanied, and sometimes preceded, by a decline in emotional regulation, social conduct, or motivation. This syndrome is observed in individuals with Alzheimer's disease, cerebrovascular disease, and other conditions that primarily or secondarily impact the brain (World Health Organization, 2010). There exist five causes of dementia:

Alzheimer's disease (AD): It is the prevailing diagnosis for dementia among the elderly population. The etiology of this phenomenon can be attributed to neurobiological alterations, namely the presence of atypical accumulations of proteins, commonly referred to as amyloid plaques and tau tangles. Fronto-temporal dementia (FTD): is an infrequent manifestation of cognitive decline, typically observed in individuals under the age of 60. Lewy body dementia (DLB): Abnormal quantities or configurations of the proteins viz. tau and TDP-43 are linked to this phenomenon. Lewy body dementia is a variant of dementia that arises due to the presence of atypical accumulations of alpha-synuclein protein, commonly referred to as Lewy bodies.

Vascular dementia is a type of cognitive impairment that arises from pathological disorders that inflict damage upon the blood vessels in the brain or disrupt the normal circulation of blood and oxygen to the brain. Mixed dementia: It refers to the coexistence of two or more distinct kinds of dementia inside an individual. Vascular dementia is a general term describing problems with reasoning, planning, judgment, memory and other thought processes caused by brain damage from impaired blood flow to our brain. VD can be the result of ischemic or hemorrhagic tissue injury in a particular region of the brain which translates into clinically significant cognitive impairment. For example, a cerebral infarct in the speech area of the dominant hemisphere would translate into clinically significant impairment as would involvement of projection pathways such as the arcuate fasciculus.

Homocysteine is a thiol-containing amino acid that is generated during 1-carbon metabolism. Elevation of the plasma levels of this metabolite has been associated with increased risk for cardiovascular and

cerebrovascular disease in 2 studies. Homocysteine can also be metabolized to S-adenosyl homocysteine, which may exhibit neurotoxicity by inhibition of methylation reactions and thus monoamine neurotransmitter metabolism and protein and phospholipid methylation. Therefore, raised homocysteine levels have the potential to effect neurodegeneration.

II. Materials And Methods:

The present study was conducted in the department of Biochemistry, Jawahar Lal Nehru (J. L. N.) Medical College and its Associated Hospitals Ajmer. The study encompassed a total of 95 patients diagnosed with Vascular dementia. These individuals were either attending the outpatient clinics or were admitted to the wards of the Department of Psychiatry at J. L. N. Medical College and its Associated Hospitals in Ajmer. A total of 95 individuals who met the criteria of being healthy and within the same age group were chosen as control subjects for this study. The selection process involved recruiting volunteers from various backgrounds, including doctors, resident doctors, paramedical workers, and healthy attendants of patients. The study acquired consent from all participants involved.

Body weight was assessed while wearing lightweight clothing and without shoes or a cap. A calibrated digital weighing scale with an accuracy of 0.1kg was used for the measurements. The measurement of height was conducted in the absence of shoes and headwear, use a wall-mounted stadiometer with an accuracy of 0.5 cm. The body mass index (BMI) was calculated by dividing an individual's weight by the square of their height (kg/m²). (Garrow and Webster,1985)
Weight (kg)

The study subject is divided into three groups:
1. Group 1: Healthy Control subject (n=95)
Mean Age: 66±7 years
Mean BMI value: (26.44 ±5.32) kg/m²

Group 2: Vascular dementia subject (n= 95):
Mean Age: 66±7 years
Mean BMI value: (26.39 ±4.93) kg/m²

Following biochemical parameters was analyzed with plasma:
Plasma Homocysteine: by ELISA method

Following Biochemical parameters was analyzed with serum:
1. Serum total cholesterol: by Enzymatic CHOD-POD, end point method (Allian CC; 1974).
2. Serum HDL-cholesterol: Phoshphotungstic acid, end point method (Finley PR; 1978).
3. Serum TG: by Enzymatic GPO-POD Enzymatic, end point method (Fossati P; 1982).
4. VLDL-cholesterol & LDL –cholesterol calculated by Friedwald's equation

The variables were reported in the form of mean ± standard deviation (SD). The parameters within the groups were examined using an analysis of variance (ANOVA) test, followed by a Tukey honestly significant difference (HSD) post hoc analysis. The study employed the Pearson's rho (r: correlation coefficient) correlation test to examine the associations between variables. The study employed a two-tailed P value with a significance level of $P < 0.05$ for all statistical analyses conducted.

III. Results:

Table 1: Correlation between homocysteine and age in patients with vascular dementia (VD)

Homocysteine (µmol/L) Mean (SD)	Age (years) Mean (SD)	R	p-value
22.63 (1.64)	66.44 (7.14)	0.073	0.481

r= correlation coefficient

Table 2: Correlation between total cholesterol and age in patients with vascular dementia (VD)

Total cholesterol (mg/dl) Mean (SD)	Age (years) Mean (SD)	R	p-value
254.96 (30.12)	66.44 (7.14)	0.076	0.466

r= correlation coefficient

In the present study we found that older age was associated with vascular dementia (VD) dementia compared to younger age (Table 1). De Bruijn and Ikram, (2014) , Jackson and Sudlow, (2006), and Kalaria, (2010) all lend credence to our research as well.

Dementia is estimated to affect 6.4% of the population in Western nations, but 7.4% of the population in India.

Total cholesterol was found to be considerably greater in participants with vascular dementia (VD) (254.96 ± 30.12 mg/ dl) compared to controls (165.57± 10.38 mg/dl) in our investigation (Table 6). Total cholesterol levels differed significantly ($p<0.01$) between healthy controls and people with Vascular dementia (Table 6). Our findings corroborate those of Nafija et al. (2013) and other research that found elevated total cholesterol in those with Vascular dementia.

Serum triglyceride levels in persons with Vascular dementia were substantially greater than in controls (203.02 ± 34.24 mg/ dl vs. 117.35± 15.38 mg/dl; (Table 5). Serum Triglyceride levels were significantly different between people with Vascular dementia and healthy controls ($p<0.01$) [Table 5]. Nafija et al. (2013) and other research corroborate our findings that patients with Vascular dementia had significantly elevated serum Triglyceride levels.

Reitz et al (2004) said that there are different pathways in which plasma lipids could be associated with the risk of VD. High concentrations of LDL and low levels of HDL are known to be independent risk factors for coronary heart disease and carotid artery atherosclerosis, which in turn may lead to cognitive impairment through cerebral hypoperfusion or embolism. Particles of HDL might also be linked with small-vessel disease by playing a role in the removal of excess cholesterol from the brain by interaction with ApoE and heparan sulfate proteoglycans in the sub-endothelian space of cerebral microvessels. Second, the brain appears to be particularly vulnerable to oxidative lipid damage because of its high content of polyunsaturated fatty acids.

In the present study serum levels of HDL-Cholesterol were significantly lower in VD dementia subjects (31.92 ± 2.27 mg/ dl) compared to control subjects (48.92± 4.68 mg/dl). A highly significant difference ($p<0.01$) in the level of serum HDL-Cholesterol was found between control subjects and VD subjects. Our findings is compatible with the study conducted by Nafija et al (2013), Reitz et al (2004) and numerous studies which indicated a significant drop in the amount of blood HDL-Cholesterol in patients with VD dementia.

Patients with VD dementia had serum levels of VLDL-Cholesterol that were considerably higher than those of control patients.

Subjects with VD dementia had higher plasma homocysteine levels (22.63± 1.64 mol/L) compared to controls (16.51± 2.31 mol/L). Compared to healthy controls, people with VD dementia had significantly higher levels of homocysteine, as seen in [Table 12] ($p<0.01$). Consistent with previous research, we found that the plasma homocysteine levels of individuals with VD dementia were significantly higher than those of healthy controls.

Feligioni M et al (2019) said that Hyperhomocysteinemia is a well known vascular risk factor and a higher level of serum homocysteine in VD subjects is probably causal to the vascular lesions underlying this disorder .On the other hand, while the deficiency of folate, or vitamin B 12 or pyridoxine is responsible for hyperhomocysteinemia in many cases, It is interesting to notice in our present results that the VD subjects who presumably have greater degrees of cerebrovascular pathologies than AD patients have correspondingly higher serum homocysteine level than the latter. Similar higher values of serum homocysteine in VD compared to AD have also been reported earlier.

IV. Conclusion:

Vascular dementia (VD) is a neurocognitive illness that accounts for 20% of all occurrences of dementia, making it the second most common kind of dementia after Alzheimer's disease (AD). The predicted prevalence is 1.0 between the ages of 71 and 79, and rises to 4.1% between the ages of 80 and 89. The most prevalent symptoms of VD include cognitive deterioration, early gait disorder, sadness and personality abnormalities. Brain hypoperfusion caused by cerebrovascular diseases (CVD) is responsible for the cognitive impairment seen in this condition. VD is characterised by harm to perforating arterioles and capillaries, which causes damage to deep grey and white matter; the importance of venular damage is less known. Arteriosclerosis, fibrinoid necrosis, and lipohyalinosis are histologically recognised outcomes of plasma components and inflammatory cells infiltrating arteriolar walls and perivascular tissue. Total Cholesterol, low-density lipoprotein cholesterol (LDL), triglyceride, and high-density lipoprotein cholesterol (HDL) imbalances are commonly referred to as dyslipidemia. In addition to hypertension, this is increasingly recognised as the single most important risk factor for coronary artery disease (CAD) and other cardiovascular disease (CVD). Despite an enormous quantity of epidemiological and medical information in support of the use of plasma lipid and cholesterol levels as biomarkers of prediction and pharmaceutical targets in all vascular pathologies, there has been an enough and increasing awareness that standard lipid profiles lack the capacity to fully explain CVD and CAD risk. Plasma LDL levels continue to be utilised as the primary biomarker for coronary artery disease and cardiovascular disease risk. In our study level of LDL-cholesterol level were considerably raised in vascular dementia group in comparing to control group. LDL-

cholesterol levels and age are correlated in a way that is not statistically significant among those with vascular dementia. Patients in the study's vascular dementia group showed a weak association between LDL-cholesterol and body mass index.Lipids play an important part in many of the pathogenic processes that contribute to the onset of vascular dementia (VD). Cognitive impairment due to cerebral hypoperfusion or embolism can be caused by either high levels of low-density lipoprotein (LDL) cholesterol or low levels of high-density lipoprotein (HDL) cholesterol, both of which are known risk factors for carotid atherosclerosis and coronary artery disease. In the subendothelial area of cerebral microvessels, APOE and heparin sulphate proteoglycans remove excess cholesterol. HDL cholesterol may play a role in this process. The effects of oxidised LDL particles on endothelium-dependent arterial relaxation are antagonised by HDL particles, and HDL particles also block the expression of endothelial cell adhesion molecules that are triggered by cytokines. Both of these are plausible pathways leading to VD.In the present study, people with vascular dementia had considerably higher triglyceride levels than those without the condition. Triglyceride levels and age are correlated, but not significantly, in those with vascular dementia. In the study Triglyceride exhibited a non significant connection with BMI in vascular dementia group participants. Homocysteine (Hcy) is an amino acid that contains sulphur but is not found in food and does not get incorporated into proteins. Methionine, an amino acid found in high concentrations in both plant and animal proteins and the primary source of sulphur atoms in proteins, is the only known source of Hcy.

Hyperhomocysteinemia (HHcy) refers to an increase in plasma homocysteine levels and is a substantial, yet often overlooked, risk factor for vascular contributions to cognitive impairment dementia (VCID). The elderly often suffer from B vitamin deficiency, the most common cause of HHcy. Homocysteine (Hcy) in the aetiology of numerous mental illnesses. Hcy has been shown to promote atherogenesis and thrombosis.