



A STUDY ON ROLE OF HDL IN CEREBROVASCULAR ACCIDENT AND COMPARISON OF HDL-LEVEL IN ISCHEMIC AND HEMORRHAGIC STROKE

General Medicine

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ABSTRACT

Objectives: To study serum HDL level in patient with cerebrovascular accident and to compare the level of serum HDL level between ischemic & hemorrhagic categories of stroke. **Methods:** This study was a retrospective cross sectional study conducted in the department of general medicine in Alluri Sitarama Raju Academy of Medical sciences, Eluru, Andhra Pradesh. **Results:** The Mean serum HDL of ischemic stroke is 32.36 & the mean serum HDL of haemorrhagic stroke is 39.36. On comparing them both Mean HDL of ischemic stroke is lower than Hemorrhagic stroke, the P value <0.05 which is statistically significant. **Conclusion:** This study shows that there is a significant decrease in HDL in ischemic stroke than Hemorrhagic stroke. Based on the results obtained from the present study HDL is used as an early predictor of atherosclerosis and ischemic stroke. By measuring the HDL earlier, early intervention measures by pharmaceutical means or by dietary means can be done to increase the HDL level, to decrease the morbidity and mortality of stroke.

KEYWORDS

INTRODUCTION:

A stroke or cerebrovascular accident (CVA) is the most common neurological cause of morbidity and mortality all over the world.⁽¹⁾ It involves the rapid loss of brain function caused by a disruption of blood supply to the brain triggered by ischemia (lack of blood flow), blockage (thrombosis, arterial embolism), or haemorrhage.⁽¹⁾ There are various risk factors, such as age, gender, family history, and other modifiable risk factors like lifestyle habits such as tobacco, alcohol use, sedentary life, obesity, high blood pressure, hyperlipidemia, diabetes, and atherosclerosis.⁽¹⁾

Dyslipidemia is a major risk factor for stroke, following hypertension, diabetes, and smoking. Dyslipidemia is also an important risk factor for the prevention and treatment of other vascular diseases such as coronary artery disease and peripheral vascular disease, including stroke.⁽²⁾ The relationship between blood lipids and stroke has been extensively investigated. Total cholesterol (TC) mainly consists of low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C); $TC = LDL-C + HDL-C + triglyceride$.⁽²⁾

High density lipoprotein (HDL) particles are heterogeneous in terms of size, shape, density, composition, and surface charge. HDL subclasses differ in their ability to promote cholesterol efflux, which is the first step in reverse cholesterol transport. Furthermore, the effectiveness of the anti-atherogenic functionality of HDL may differ based on particle number and/or size. Interestingly, HDL efflux varies between individuals and is independently associated with CVD events.⁽³⁾

HDL Structure And Function:

Lipoproteins are complexes of proteins, phospholipids and cholesterol. HDL is the smallest and densest of the lipoproteins because of its high protein content. Proteins make up about 50% of its mass and apolipoprotein A-I (apoA-I) accounts for 70% of that protein component.

ApoA-I is a 243 amino acid protein synthesized in the liver (70%) and intestine (30%) and secreted into the serum in a lipid-free state. Individual apoA-I molecules associate with other apoA-I molecules, membrane phospholipids and cholesterol to form lipid-poor pre- β -HDL. Lipid-poor HDL assumes a double-belt-like structure which becomes spherical with maturation and addition of cholesterol.

Lipid-poor β -HDL is able to take up cholesterol from artery wall macrophages via the ATP-binding cassette A-1 (ABCA-1) receptor. Cholesterol is transferred into lipid poor HDL and is then esterified by the enzyme lecithin-cholesterol acyltransferase. As cholesterol is

esterified it is packaged into the core of the HDL leading to formation of the mature spherical lipoprotein.

Mature spherical HDL can then unload its cholesterol by 2 main mechanisms. Transfer of cholesterol back to the liver is facilitated by interaction with the scavenger receptor-B1 (SR-B1) receptor on hepatocytes and leads to formation of bile and secretion into the gut. Alternatively, cholesterol can be transferred from mature HDL to low-density lipoprotein (LDL) or very-low-density lipoprotein (VLDL), a process dependent on cholesteryl ester transfer protein (CETP). This leads to a recycling of cholesterol, potentially back into the artery wall.⁽⁴⁾

HDL mechanism of action: There are several mechanisms by which HDL protects against atherosclerosis, including reverse cholesterol transport, antioxidant effects, anti-inflammatory effects, antithrombotic effects, and modification of endothelial function. In reverse cholesterol transport, cholesterol is transported by HDL from the artery wall to the liver for excretion. Cholesterol is removed from macrophages in the subintima of the vessel wall by the interaction of HDL with ABCA-1, SR-B1, or by passive diffusion. Once esterified, cholesterol in the HDL is then transported to the liver for excretion.

In contrast to Vitamin E, a far less potent antioxidant, HDL and apoA-I are able to prevent lipid oxidation, a key mechanism in atherosclerosis in both cell-free and artery wall co-culture studies.⁴ HDL is a major carrier of lipid hydroperoxidases and paraoxonase, an enzyme involved in preventing and reversing oxidative damage.

HDL blocks inflammation by acting as an antioxidant but also by limiting the expression of cytokines such as tumor necrosis factor- α and interleukin-1 that mediate upregulation of leukocyte endothelial adhesion molecules. This has been demonstrated in cell cultures where monocyte chemotactic activity can be blocked with the addition of normal functioning HDL.⁵

Finally, HDL may also reduce thrombotic risk through the inhibition of platelet activation and aggregation⁶ and may improve endothelial function by stimulating prostacyclin release.

MATERIALS AND METHODS:

Study Place:

The present study was conducted in the Alluri sitarama raju academy of medical sciences hospital, Eluru.

Study Period:

January 2023 to January 2024

Study Design:

The present study is one year retrospective cross sectional study on the patients with cerebro vascular accident.

Source of DATA:

The patients with both ischemic and haemorrhagic stroke admitted in department of General medicine and Neurology of Alluri sitaramaraju academy of medical sciences hospital, Eluru were enrolled in the study. Sample size: the study is conducted in 100 patients of cerebro vascular accident of which 50 were ischemic stroke and 50 were haemorrhagic stroke.

Inclusion Criteria:

Patients with cerebro vascular accident admitted with neurological weakness.

Exclusion Criteria:

1) Pre-existing cardiac diseases 2) Presumptive diagnosis of stroke with no evidence of CT 3) cerebro vascular accident with tumor or trauma 4) Preexisting liver disease.

Methodology:

Case record data collected of all the patients with cerebro vascular accident fulfilling inclusion and exclusion criteria. Descriptive data of participant name, age, sex, chief complaints, personal history and fasting lipid profile with parameters including HDL, total cholesterol, VLDL and LDL reports were collected as per the case record form, data was analysed in excel sheet by SPSS version 26.0, data presented in graphs and tables and mean difference was analysed using unpaired 't' test, a p value <0.05 is considered statistically significant.

RESULTS

The Mean serum HDL of ischemic stroke is 32.36 & the mean serum HDL of hemorrhagic stroke is 39.36. On comparing them both, mean HDL of ischemic stroke is lower than Hemorrhagic stroke, whose p value <0.05 which is statistically significant.

Table 1: Comparison Of Mean HDL Between Ischemic And Hemorrhagic Stroke

PARAMETER	ISCHEMIC STROKE	HAEMORRHAGIC STROKE
HDL	32.36	39.36

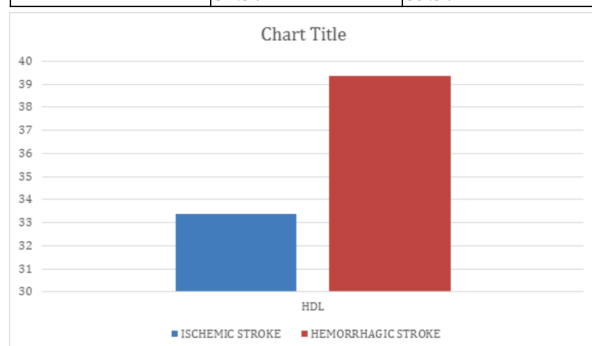


Figure 1: Comparison of Mean HDL between Ischemic and Hemorrhagic stroke

On comparison of mean lipid profile between ischemic stroke and haemorrhagic stroke, the mean Total Cholesterol, triglycerides, VLDL and LDL values are higher in ischemic stroke than hemorrhagic stroke.

Table 2 Comparison Of Mean Lipid Profile Between Ischemic And Hemorrhagic Stroke

LIPID PROFILE	ISCHEMIC STROKE	HEMORRHAGIC STROKE
TOTAL CHOLESTROL	161.2	159.70
TRIGLYCERIDES	159.56	157.14
VLDL	31.90	31.46
LDL	96.96	88.88

DISCUSSION:

The present study done with 100 subjects (50 were ischemic stroke and 50 were Hemorrhagic stroke) supports the fact that measurement of Lipid profile was useful in predicting the risk factors for stroke.

The Mean value of HDL of Hemorrhagic stroke (39.36) was higher when compared to the Mean Ischemic Stroke (32.36), which is statistically significant with p value <0.05.

The mean values of Total Cholesterol (161.22), Triglycerides (159.56), VLDL (31.90) and LDL (96.96) of ischemic stroke were higher than Hemorrhagic Stroke, whose mean values are Total Cholesterol (159.56), Triglycerides (157.14), VLDL (31.46) and LDL (88.88).

This study shows HDL was significantly decreased in ischemic stroke than hemorrhagic stroke, whereas HDL was high in hemorrhagic stroke. Total cholesterol, Triglycerides, LDL, VLDL were more in ischemic stroke than hemorrhagic stroke.

CONCLUSION:

This study shows that there is a significant decrease in HDL in ischemic stroke than Hemorrhagic stroke. As the Reverse Cholesterol Transport, Anti-inflammatory activity, Anti-oxidative activity, Anti-apoptotic activity, Endothelial repair, Anti-thrombotic activity, Anti-infectious activity of HDL are reduced due to decreased HDL leading to ischemic stroke than Hemorrhagic stroke. Based on the results obtained from the present study, HDL is used as an early predictor of atherosclerosis and ischemic stroke. By measuring the HDL earlier, early intervention measures by pharmaceutical means or by dietary means can be done to increase the HDL level to decrease the morbidity and mortality of stroke.

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