SERUM LIPOPROTEIN COMPONENTS IN ISCHEMIC CEREBROVASCULAR STROKE PATIENTS.

1° Dr. V. Bhagyalakshmi, 2° Dr. V. Bhavani, 3° Dr. G. Premkumar, 4° Dr. M. Jaiprakash Babu, 5° Dr. G. Rajeswari

1° Department of Biochemistry, Rangaraya Medical College, Kakinada, East Godavari Dist, Andhra Pradesh, India.
2° Department of Biochemistry, Siddardha Medical College, Vijayawada, Andhra Pradesh, India.
3° Department of Biochemistry, Rangaraya Medical College, Kakinada, East Godavari Dist, Andhra Pradesh, India.
4° Department of Biochemistry, Gitam Medical College, Visakhapatnam.
5° Department of Biochemistry, Rangaraya Medical College, Kakinada, East Godavari Dist, Andhra Pradesh, India.

ABSTRACT

Background: 1. To study the risk factors in patients of ischemic stroke. 2. To correlate the risk factors with reference to lipoprotein abnormalities. 3. To study the role of lipoprotein components in ischemic stroke patients. Method: A total number of 60 patients admitted in the medical wards under the department of general medicine of government general hospital, Rangaraya medical college, Kakinada, were included. 40 age & Sex matched healthy individuals were also included in the study as controls for comparison. Results: Serum triglycerides, total cholesterol, LDL Cholesterol showed positive association with ischemic stroke compared to controls. Serum HDL Cholesterol showed inverse association with ischemic stroke compared to controls. Conclusion: LDL cholesterol (oxidized) initiate inflammatory response which has been linked to atherogenesis. HDL cholesterol prevents oxidation of LDL cholesterol and is protective against ischemic stroke.
KEYWORDS: Ischemic stroke, Triglycerides, Total cholesterol, HDL, LDL, VLDL cholesterol.

INTRODUCTION
Stroke ranks foremost of all disorders of the nervous system. Stroke is a leading cause of death and disability. Stroke is a clinical syndrome characterized by loss of cerebral function, with symptoms lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin. Cerebral atherosclerosis with atheroma formation is the basic underlying pathophysiologic mechanism which increases the risk of vessel wall disease in ischemic stroke patients. Non modifiable risk factors for ischemic stroke include age & sex.\textsuperscript{[1,2]} Modifiable medical risk factors for ischemic stroke include hypertension, diabetes mellitus, hyperlipidemia.\textsuperscript{[1,2]} Smoking, alcohol consumption, obesity are the most important life style stroke risk factors.\textsuperscript{[13]} Different environmental factors, genetic influences and aging are playing a wide role in the prevalence of stroke among the old age group. Since hyperlipidemia is one of the important factor which contribute to the majority of cases of ischemic stroke\textsuperscript{[3]}, this study was undertaken to find out the association of lipid profile levels in ischemic stroke patients. Serum lipid levels have an established effect on short time mortality due to strokes. Hyperlipidemia is a major risk factor for atherosclerosis & ischemic stroke\textsuperscript{3} in old age around 60 years of age group. It is important to evaluate the difference in serum lipid levels in stroke patients to guide lipid lowering therapy which can reduce incidence of stroke and related mortality by adopting primary and secondary preventing measures. The management of risk factors for cerebral infarction as primary prevention is most important.\textsuperscript{[3]} Improved detection, modification or control of risk factors, life style modification, diet & physical activity could significantly reduce the incidence of stroke & reduce the impact of this disease.\textsuperscript{[13,14]}

MATERIALS AND METHODS
The present study was conducted on 60 patients of ischemic stroke patients admitted in the medical wards of government general hospital, Kakinada, East Godavari District, Andhra Pradesh, India.

The age distribution of the patients in the present study ranged from 21 to 65 years.

Peak incidence of stroke is at age 51 to 65 years.
INCLUSION CRITERIA
Should have developed a completed form of first ever stroke & that there is a neurological deficit of cerebrovascular cause which persists beyond 24 hours. Patients with cerebral infarction only are included in the present study. Diagnosis was done by CT brain Scan by the Department of Radiology, Government General Hospital, Kakinda, Eastgodavari District, Andhrapradesh, India were included in the present study. Diagnostic validity tests were conducted to discriminate those with stroke & those without stroke.

EXCLUSION CRITERIA
Patients with hemorrhagic stroke, TIA, DM, hepatic diseases, renal diseases, sepsis, malignancy& patients on hopolipidaemic drugs were excluded from the study. A signed informed consent was obtained from all patients of their family/Guardian. Venous blood samples were obtained after 12 hours of overnight fasting.

Blood samples were collected into plain tubes from patients. Samples were centrifused for 15 minutes after incubation of 20 minutes for extraction of serum. Serum was separated taking precautions to avoid hemolysis. The sera were analysed for lipid profile including total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, VLDL cholesterol by using Transasia Erba Chem 5 plus semi auto analyser. Statistical analysis was done. Mean values of all the parameters were determined in both groups of patients of ischemic stroke and control. The data was analysed and compared using proportion test for any significant difference taking P value of <0.05 as significant.


Estimation of serum triglycerides: Dynamic extended stability with lipid clearing agent GPO-Trinder method. Triglycerides were estimated by the enzymatic Glycerol phosphate oxidase-phenol aminoantipyrine method. Estimation of VLDL cholesterol & LDL cholesterol. VLDL cholesterol & LDL cholesterol were calculated by friedewald formula. VLDL cholesterol = Triglycerides/5 LDL cholesterol = Total Cholesterol – (HDL Cholesterol + VLDL cholesterol).
RESULTS AND OBSERVATIONS

In the present study it was found that Serum triglyceride levels were increased in cases with 187±69.7 mg/dl compared to controls with 134.8±36.8 mg/dl with a statistically significant ‘P’ value of <0.001. Serum total cholesterol levels were increased in ischemic stroke patients 217±62.8 mg/dl compared to controls with 183±35mg/dl with a statistically significant ‘P’ value of <0.0001. Serum HDL cholesterol decreased in cases with 35.6±14.2 mg/dl compared to controls with 43.7±11.6 mg/dl with a statistically significant ‘P’ value of <0.01. Serum LDL cholesterol levels were increased in cases with 143.8±57.2mg/dl compared to controls with 113±32.3mg/dl with a statistically significant ‘P’ value of <0.001. Serum VLDL cholesterol levels were increased in cases with 37.6±13.8 mg/dl compared to controls with 27±7.3 mg/dl with a statistically significant ‘P’ value of <0.05.

DISCUSSION

60 Ischemic stroke patients aged 21 to 65 yrs of both sexes were studied for serum lipid profile – serum total cholesterol, Triglycerides, HDL C, VLDL C, LDL C. The age distribution of the patients in the present study ranged from 21 to 65 years with a peak incidence of 51 to 65 years. Out of 60 patients 49 were males, 11 were females. There was male preponderance in the present study, with male and female ratio being 5:1. 40 age & sex matched healthy controls were taken for comparison. In all the cases & controls serum Lipid profile was done.

Smoking was the most common risk factor seen in 42 patients (72%), followed by hypertension in 29 out of 60 patients (47%) NIDDM in 18 patients (30%) and Carotid artery disease in 12 patients (20%) Multiple risk factors, three or more than three were present in 27 patients (45%) in the study group.

These risk factors can damage the inner lining of the arterial endothelium making them susceptible to atherosclerosis.[16,17] Because of the damage fats & other cellular debris deposit in the artery walls forming plaque. Atherosclerosis is an inflammatory response of the intima to injury.[17] The injury is initiated by lipids particularly LDL- Cholesterol. Oxidised lipid (LDL Cholesterol) is chemotactic for monocytes, initiate inflammatory response & monocyte becomes stuffed with lipid to become foam cell.[17] Atherosclerotic lesion is made up of focal collection of lipid filled foam cells within the intima.[17] Smooth muscle cells migrate into & proliferate within the plaque, more collagen is produced and plaque size increases.[17] Plaque necrosis leads to development of thrombosis which blocks blood vessels to brain leading to
ischemic stroke.\cite{17} LDL cholesterol (oxidized) initiate inflammatory response which has been linked to atherogenesis.\cite{17} HDL cholesterol prevents oxidation of LDL Cholesterol & has antiatherogenic properties with ability to trigger the flux of cholesterol from peripheral cells to the liver, thus having a protective effect.\cite{17}

In the above study it was found that Serum triglycerides. Total cholesterol, LDL Cholesterol were significantly increased in cases compared to controls The above study is in consistent with the study of Hachinski V, Graffagnino C et al.\cite{14,5,6,7,8,11,12,13} Serum HDL cholesterol was decreased significantly in cases compared to controls. The above study is in consistent with the study of Soyama Y\cite{1}, Miura K, Morikawa Y et al.\cite{2,5,8,9,10,12,13} Elevated Total cholesterol,\cite{5,6,7,12,13} LDL Cholesterol,\cite{4,7,8,11} Triglycerides,\cite{5,8,11} & decreased HDL Cholesterol\cite{5,8,10,12,13} are significant independent risk factors in patients with atherothrombotic cerebrovascular disease manifesting as stroke.

**CONCLUSION**

Now that many risk factors are established, greater emphasis should be placed on identifying high stroke-risk patient populations for intensive risk factor modification. Better understanding & management of stroke risk factors will undoubtedly improve our ability to prevent first & recurrent ischemic stroke.\cite{1} The ability to detect or modify the risk factors for ischemic stroke should lead to a substantial reduction in the mortality & morbidity by stroke.\cite{2} Regular monitoring of lipid profile among stroke patients may decrease the risk of atherosclerosis & cardiovascular disease among the stroke patients. Life style modification has been recommended by the national cholesterol education program.\cite{15} Correction of life style as a non-drug therapy may clearly improve hyperlipidemia or hypo HDL Cholesterolemia.\cite{14} Diet & exercise in adults can reduce serum lipid values, its risk for atherosclerosis, its clinical sequelae so that this approach should be employed as part of the prevention & treatment for hyperlipidemias.\cite{14,15}

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