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Research Paper

Comparative Analysis of Various Serum Lipid Parameters in Patients with Ischemic Stroke

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ABSTRACT
Background: *Role of serum lipids, lipoproteins and lipoprotein related variables in the prediction of stroke is less clear. Abnormalities in plasma lipoproteins are the most firmly established and best understood risk factors for atherosclerosis. They are probable risk factors for ischemic stroke, largely by their link to atherosclerosis. Apo B reflects the concentration of potentially atherogenic particles (LDL), and Apo A1 reflects the corresponding concentration of anti-atherogenic particles (HDL), represent additional lipoprotein related variables that may indicate the vascular risk.*
Aim: *To study and compare serum concentration of Apolipoprotein A1, Apolipoprotein B, Apo B/Apo A1 ratio and Lipid profile in patients of acute ischemic stroke*
Design: *A total number of 128 subjects of Stroke (both clinically as well as computed tomographically proven cases) aged above 18, of both sexes, were taken for the study.*
Material and Methods: *Total cholesterol, HDL cholesterol and triglycerides are estimated by enzymatic method using autoanalyser, LDL cholesterol by Friedewald formula and Apo B and Apo A1 by nephlometry method using autoanalyser, in all the enrolled subjects as per inclusion criteria.*
Statistical Analysis: *Student 't' test and ANOVA were used to compare the data between subgroups.*
Results: *Patients with stroke had low HDL and low Apo AI, high Apo B levels, and high Apo B/Apo AI ratio. Though total cholesterol and high LDL levels are regarded are better predictors of atherosclerosis, this study showed that only a minority of the patients had total cholesterol on the higher side, and about 50% patients had high LDL levels, but more than 80 % patients had a high Apo B to Apo A1 ratio.*
Conclusion: *Low levels of Apo A1 and high Apo B / Apo A1 ratio can be used as predictors of stroke along with traditional lipid profile components.*
Keywords*: Apolipoprotein A1, Apolipoprotein B, Lipid profile, Stroke*

I. INTRODUCTION

Stroke or a Cerebrovascular Accident (CVA) is defined as the "Abrupt onset of a neurological deficit that is attributable to a focal vascular cause". If the neurological signs and symptoms last for < 24 hours, it is called as a Transient Ischaemic Attack (TIA) and if they exceed 24 hours, then it is called as stroke [1]. Stroke is a worldwide health problem. It makes an important contribution to the morbidity, mortality and the disability in the developed as well as the developing countries. A WHO collaborative study was done in 12 countries, showed that the incidence rates of stroke was 0.2 – 2.5 per 1000 population per year. It is the leading cause of adult disability and the second most common cause of death worldwide. It accounts for 10 – 12% of the total deaths in the developed countries [2].

"Little stroke, big trouble" the theme of the World Health Day, 2008, speaks about the importance of stroke as a critical warning sign of further more debilitating vascular events or death [3]. Disturbances in the cerebral function in stroke are caused by three morphological abnormalities, which include stenosis, occlusion or rupture of arteries, leading to ischaemia, infarction and cerebral haemorrhage respectively. Abnormal lipid parameters like Total Cholesterol (TC), Low Density Lipoprotein (LDL) cholesterol, High Density Lipoprotein (HDL) Cholesterol and Triglycerides (TG) are the probable risk factors for ischaemic stroke, largely due to their link to Atherosclerosis. The apolipoproteins are the protein components of the Lipoproteins. Apolipoprotein B (Apo B), which reflects the concentration of the potentially atherogenic Lipoprotein Particles (LDL) and Apolipoprotein A1(Apo A1), which reflects the concentration of the antiatherogenic particles (HDL), represent the additional lipoprotein related variables that may indicate a vascular risk [4].

Dyslipidaemia, low Apo A1 and high Apo B are widely accepted as the risk factors for coronary artery disease. In contrast, the correlation has not been well established for stroke [5]. So, the present study was

undertaken to evaluate the relationship between Apo A1, Apo B and the lipid profile in patients of ischaemic stroke..

II. MATERIAL AND METHODS

A prospective observational study was conducted over a period of one year, on 128 patients of clinically and tomographically proven acute ischaemic stroke(<7days of onset) above the age of 18 years, after obtaining written and informed consent from the patients and clearance from research and ethical committee of a tertiary care hospital of north India. The patients with hepatic disease, renal diseases, sepsis and malignancy and the patients who were on hypolipidaemic drugs were excluded from the study. Subjects were recruited from the inpatient and outpatient department, about 6 ml of blood was drawn under aseptic precautions in sterile bulbs after a period of overnight fasting for 12 hours for serum Apo A1, Apo B, lipid profile and other biochemical tests.

Total cholesterol was estimated by the enzymatic cholesterol oxidase phenol aminoantipyrine method. HDL cholesterol was estimated by the enzymatic cholesterol oxidase phenol amino-antipyrine method after the precipitation of LDL C, Very Low Density Lipoprotein (VLDL) cholesterol and chylomicrons. The triglycerides were estimated by the enzymatic glycerol phosphate oxidase – Phenol aminoantipyrine method. LDL cholesterol was estimated by using Friedewald's formula. ATP III guidelines were used as standard lipid guidelines to assess risk factor. Hypertension was defined according to the JNC VIII guidelines.Descriptive statistics such as mean and standard deviation has been used to describe normally distributed continuous variables such as Age, heart rate etc. Percentage and proportions have been used to describe categorical variables such as gender, presence of risk factors. Unpaired 't' test was used to test difference in means of continous normally distributed variables. Multivariate logistic regression analyses was performed to predict risk factors. Statistical significance was assessed at a p value of <0.05.

III. OBSERVATION AND RESULTS

Out of 128 patients, 66% were males, ranging from 24 years to 96 years. The mean age of patient diagnosed with thrombotic stroke in our hospital was 63.39 years. Maximum no. of patient with stroke were within 51 to 70 years of age. The mean systolic blood pressure was 140.01 while the mean diastolic blood pressure was 81.61mm of Hg. According to age groups, 13 patients belonged to young age group (<45 years), 58 patients belonged to middle age group while 57 patients were of older age group..

75% of the total patients were known cases of hypertension. Approximate 30% of the patients were previously diagnosed with diabetes mellitus, while 11.7% of the patients had previous history of stroke or TIA. 50.78% of the total patients were smokers, while 30% were alcoholic, and 24% were both smokers and alcoholics. Mean fasting blood glucose levels were 123mg/dl, with mean serum creatinine levels was 1.13 mg/dl. Mean Total Cholesterol levels were 162 mg/dl, which is below 200mg/dl(criteria for hypercholestremia), but the mean HDL was found to be less than 40 mg/dl, which favors at atherosclerosis. Mean LDL was levels were found to 98.75mg/dl which are below 120 (risk factor for atherosclerosis). Mean Apo AI level was 100.38 mg/dl, while mean Apo B level was 93.24 mg/dl

Only 20 patients (15.7%) out of 128 patients had total cholesterol levels above 200mg/dl, 97 patients (75.75%) HDL below 45mg/dl, while 65 patients (50.78%) had LDL above 100mg/dl. Significantly high Apo B/Apo AI ratio is seen in patients with low HDL levels and also a significantly high Apo B/Apo AI ratio in patients with high LDL and total cholesterol levels [Table No. 1].

Table No.1: Comparison of various lipid parameters (n=128)

T. Cholesterol(mg/dl)	**No. of Cases**	**Apo B/Apo AI ratio**	**p-level**
<200	108(84.3%)	0.89±0.27	0.001
≥ 200	20(15.7%)	1.36±0.29	
HDL(mg/dl)	**No. of Cases**	**Apo B/Apo AI ratio**	**p-level**
<45	97(75.7%)	1.02±0.32	0.0028
≥ 45	31(24.3%)	0.82±0.31	
LDL(mg/dl)	**No. of Cases**	**Apo B/Apo AI ratio**	**p-level**
<100	63(49.22%)	0.83±0.27	0.0008
≥ 100	65(50.78%)	1.01±0.32	
Non- HDL Cholesterol (mg/dl)	**No. of Cases**	**Apo B/Apo AI ratio**	**p-level**
<130	69(53.90%)	0.82±0.26	0.0001
≥130	59(46.09%)	1.14±0.31	

97 patients (75.75%) had Apo AI below 120mg/dl, 89 patients (69.6%) patients had Apo B above 80mg/dl, while approximate 81.25 % of the patients had an Apo B/Apo AI ratio of more than 0.65, which is considered as a risk for atherosclerosis [Table No. 2].

Table No.2: Apolipoprotein Assessment (n=128)

Apo AI(mg/dl)	**No. of Cases**	**Percentage**
<120	108	84.3%
≥ 120	20	15.7%
Apo B(mg/dl)	**No. of Cases**	**Percentage**
<80	39	30.4%
≥ 80	89	69.6%
APO B/A1	**No. of Cases**	**Percentage**
<0.65	24	18.75%
> 0.65	104	81.25%

HDL levels were significantly low in males as compared to females, therefore a high Total cholesterol to HDL ratio. Apo AI levels were also significantly low in males, whereas the Apo B levels were significantly high in males as compared to females. High Apo B and low Apo AI levels in males led to an significantly higher Apo B to Apo AI ratio in males. The comparison of lipid parameters of patients with or without carotid artery stenosis showed Apo B levels were significantly higher in patients with carotid artery stenosis, along with high Apo B to Apo AI ratio.

Patients with history of smoking had higher LDL and higher Apo B as compared to the non smokers, and their Apo B to Apo AI ratio was also higher than compared to non smokers. Apo B/Apo AI ratio was more than 0.98 in smokers which favor atherosclerosis.

Multivariate logistic regression analysis for predictor of risk factor for stroke in patients with carotid artery stenosis revealed significantly high Apo B and LDL levels.

Table No. 3. Comparison of lipid parameters

	T.Chol.	**HDL**	**LDL**	**TG**	**T.Chol/HDL**	**APO AI**	**APO B**	**APO B/ APO A1**
Male	164.34±43.17	37.34±8.76	101.62±36.75	137.14±63.51	4.62±1.78	97.71±15.79	97.43±22.03	1.02±0.30
Female	158.48±45.01	44.4±10.52	92.39±36.51	131.16±60.91	3.72±1.25	105.65±25.87	84.94±21.58	0.86±0.34
p value	0.47	**0.0001**	0.1810	0.6109	**0.0036**	**0.0330**	**0.0028**	**0.0074**
Young	160.76±48.90	34.14±6.28	105.00±39.24	147.92±46.38	4.84±1.54	94.11±12.29	92.58±30.73	1.00±0.38
Middle	166.24±43.57	40.83±10.67	99.95±36.72	143.50±70.22	4.40±2.02	100.23±21.54	97.04±21.97	1.01±0.33
Old	158.81±43.12	39.86±9.56	95.60±36.70	123.70±55.94	4.13±1.23	101.97±19.70	89.52±20.86	0.92±0.32
p value	0.601	0.089	0.657	0.175	0.345	0.422	0.203	0.327
Smoker	167.71 ± 40.05	38.86 ± 9.02	105.47 ± 35.66	130.74 ± 52.59	4.48 ± 1.34	98.55 ± 15.49	98.68 ± 22.34	1.04 ± 0.31
Non Smoker	156.87 ± 76.85	40.60 ± 10.81	91.72 ± 37.03	139.67 ± 71.41	4.16 ± 1.95	102.28 ± 23.70	87.60 ± 21.61	0.91 ± 0.33
p value	0.3167	0.3241	0.0408	0.4210	0.2800	0.2925	0.0051	0.0232
Alcoholic	163.43 ± 43.90	39.23 ± 8.51	101.14 ± 36.18	131.57 ± 49.39	4.23 ± 1.08	97.90 ± 16.41	98.74 ± 20.68	1.04 ± 0.30
Non Alcoholic	161.95 ± 43.87	39.92 ± 10.50	97.81 ± 37.26	136.58 ± 67.27	4.36 ± 1.86	101.40 ± 21.24	90.98 ± 23.05	0.94 ± 0.34
p value	0.8629	0.7233	0.6448	0.6826	0.6909	0.3707	0.0780	0.1216
Hypertensive	163.19 ± 44.40	40.07 ± 9.93	100.02 ± 37.60	133.79 ± 59.67	4.2 ± 1.38	98.78 ± 19.84	93.46 ± 21.61	93.46 ± 21.61
Non Hypertensive	159.94 ± 42.18	38.67 ± 10.05	95.01 ± 34.80	139.16 ± 71.13	4.55 ± 2.33	105.21 ± 19.86	92.51 ± 25.68	0.92 ± 0.34

p value	0.7172	0.4926	0.5075	0.6754	0.3050	0.1136	0.8377	0.3043
Diabetic	163.13 ± 49.07	38.84 ± 10.25	96.81 ± 39.69	139.92 ± 61.03	4.41 ± 1.61	97.55 ± 20.43	91.44 ± 24.98	0.97 ± 0.33
Non Diabetic	162.06 ± 41.54	40.09 ± 9.84	99.59 ± 35.76	133.11 ± 63.31	4.29 ± 1.70	101.58 ± 19.76	93.98 ± 21.61	0.962± 0.31
p value	0.8999	0.5178	0.6981	0.5752	0.5530	0.2986	0.5755	0.8961
Carotid Stenosis	158.32 ± 45.00	37.43 ± 8.59	95.56 ± 36.57	133.32 ± 57.59	4.31 ± 1.13	94.55 ± 20.62	102.03 ± 22.89	1.12 ± 0.33
No Carotid Stenosis	163.67 ± 43.45	40.45 ± 10.27	99.74 ± 37.06	135.71 ± 64.24	4.33 ± 1.81	102.25 ± 19.49	90.41 ± 21.88	0.92 ± 0.32
p value	0.5551	0.1415	0.5844	0.8538	0.9539	0.613	0.01	0.0032

IV. DISCUSSION

In this study, we found that the majority of patients were above age of 60 years. This was similar to previous studies, where they found that almost two third of the patients of stroke are above the age group of 65 years (7). Male preponderance was observed which could be attributed to the co-morbidities such as hypertension, diabetes mellitus, smoking, alcohol consumption, sedentary lifestyle, which act as a risk factor the atherosclerosis and stroke per se.

Hypertension, the single most common modifiable risk factor for stroke was observed in 96 (75%) patients. 29% of patients were previously diagnosed case of diabetes mellitus. Diabetes is a major risk factor for stroke incidence as it causes lipid disturbances and progression of atherosclerosis. Prevalence of diabetes observed in our patients of strokes was similar to the previous studies which identified diabetes as a risk factor for stroke. They had prevalence of 33% of diabetes mellitus in patients of stroke while our study had prevalence of 29% (8). 11% of the patients had previous history of stroke or TIA.

50.7% of the patients were smokers. Previous studies show that smokers are three times more likely to have stroke than non smokers. A person who smokes 20 cigarettes per day, are six times more likely to have stroke compared to a non- smoker (9). 30% of the patients had consumption of alcohol intake in moderate amount. Studies shows that alcohol consumption when done in small amounts help to lower LDL levels and prevent atherosclerosis, but as the quantity of alcohol consumption increases, it acts as risk factor for stroke. Alcohol consumption in large amount leads to excessive subcutaneous fat deposition and derangement of lipid parameters.

Our study found that only 15% of the patients had total cholesterol levels of more than 200 mg/dl. This finding was similar to the study conducted by Per Harmsen, MD and co-workers in year 1990 which failed to reveal any significant relationship between serum total cholesterol and the risk of stroke (10). In the year 1994, a study conducted by Ming-Yao Chung and co-workers, also failed to reveal any significant difference in serum total cholesterol patients of ischemic stroke (11). In the year 1995, the association of blood cholesterol and diastolic blood pressure with subsequent stroke rates were investigated by a review of 45 prospective cohorts involving 4,50,000 individuals with 5 – 30 years of follow up by prospective studies collaboration. The study found no association between blood cholesterol levels and stroke except in those less than 45 years of age (12).

HDL levels of less than 45mg/dl are regarded as an independent risk factor for atherosclerosis (13). In our study we found that 97 patients (75%) out of the total 128 patients had HDL concentrations of less 45 mg/dl. Also, patients with low HDL levels had a significantly higher Apo B /Apo AI ratio as compared to the patients with HDL of more than 45 mg/dl. This finding was similar to study conducted by D.L. Trischwell, MD and co-workers in the year 2004 which showed that higher total cholesterol and lower HDL-C levels were associated with increased risk of ischemic stroke especially atherothrombotic and lacunar stroke (14). Contrary to our findings, Mark Woodward and co-workers failed to reveal any association between either HDL-C or total cholesterol with the risk of stroke (15). In the year 2000, study conducted by C. Vauthey, MD and coworkers showed, Ischemic stroke patients with high density cholesterol levels had 2.2 fold lower risk of death and lower risk of poor functional outcome (16). Tanne David, MD and Coworkers did a follow up study of 8586 men from Israeli Ischemic Heart Disease study in the year 1997. The study demonstrated an independent negative association between HDL-C levels and ischemic stroke mortality during a long term follow up (17).

In the present study we found that 50.7% patients of stroke had LDL levels of more than 100mg/dl. Patients with high LDL levels also had higher Apo B/Apo AI ratio as compared to the patients with normal LDL levels. This finding of LDL levels are similar to study conducted by Ming-Yao Chung and coworkers (11). In the year 2002, Kio-LiongChien, MD and coworkers conducted a community based cohort study in Taiwan,

where they also found that stroke patients had significantly higher concentrations of LDL-C levels compared to the controls (18).

In our study we found 84.3% patients had a low Apolipoprotien AI levels, <120mg/dl. Low Apolipoprotein levels of less than 120mg/dl has been considered as a risk factor for atherosclerosis and stroke as proved by previous studies. In the year 1991, a study conducted by J. Woo, MD et. al., low apolipoprotein A-I ,hypertension, and a high serum lipoprotein (a) concentration, were found to be independent risk factors for all strokes (19).Similar study done by G. Walliduset. al., showed a low Apo A-I value was the single strongest contributing factor to an increased Apo B/Apo A-I ratio and to risk as it was significant in different subtypes of stroke including also haemorrhagic and subarachnoidal strokes. They also found that low HDL cholesterol was as common as a low Apo A-I value (20).

We also found that more than 80% of the patients of stroke had Apo B levels of more 80 mg/dl, associated with increased risk of atherosclerosis, similar to the study done in Taiwan on 3602 adults aged ≥ 35 years. They found that after Apo B levels were significantly higher as compared to controls (18). Whereas a study conducted by M. Bhatia and co- workers in 2006, concluded that Apo B and Apo B / Apo A-1 ratio are strongest independent predictors of ischemic stroke in patients with TIA (21).

81.25% of our patients had a Apo B to Apo AI ratio of more than 0.65, similar to the study conducted by Jong-Ho Park and et al., they concluded a higher Apo B/Apo AI ratio is a predictor of intracranial atherosclerosis, and concluded Apo B/Apo AI ratio may act as a biomarker for intracranial atherosclerosis in Asian patients with stroke (22). In 2006, G. Walliduset. al., showed strong and direct relationship between increasing values of the Apo B/Apo A-I ratio and the risk of fatal stroke (20).

Patients with carotid artery stenosis on carotid doppler, had significantly higher Apo B levels with higher Apo B/Apo AI ratio. Jyoti Jain and et. al., found Carotid Intima Media Thickness (CIMT) in computerized tomography-proven ischemic stroke was significantly higher than expected for the age of the study population. The relation of decreased Apo A-I and increased Apo B with CIMT > 0.7 mm was also statistically significant (23).

We found that patient with large size stroke had significantly lower HDL levels however no significant difference was seen when comparing the lipid parameters in relation to number of vessels involved.

Our study, found that that patients who had history of smoking or were current smokers, had significantly higher LDL and Apo B levels. Apo B/Apo AI ratio was also significantly raised in smokers as compared to the non smokers. In year, 2010, Ramachandran Meenakshi Sundaram and co-workers studied the effect of smoking on lipid parameters. They concluded that Number of pack years was directly proportional to abnormal lipid profile. They concluded that changes in Apo-AI and Apo-B were more significant when compared to HDL and LDL cholesterol among smokers (24).

V. CONCLUSION

Our study showed that low HDL and Low Apo AI, high Apo B levels, and High Apo B/Apo AI ratio, are independent risk factors for stroke. Though total cholesterol and high LDL levels are regarded are better predictors of atherosclerosis, we found that only a minority of the patients had total cholesterol on the higher side, and about 50% patients with high LDL levels, but when comparing their Apo B to Apo AI ratio, more than 80 % had a high ratio. Thus high Apo B to Apo AI ratio can be considered as a better predictor of atherosclerosis, and should be considered a risk factor of thrombotic stroke.High Total cholesterol/HDL ratio is also a significant predictor of atherosclerosis. High Apo B/Apo AI ratio could be correlated high large artery atherosclerosis as smoking has adverse effect on lipid parameters as it significantly increases LDL levels, Apo B levels and Apo B/Apo AI ratio.Total cholesterol, LDL and Apo B levels alone cannot be considered as a good predictors of stroke.Lipid parameters as a predictor of stroke incidence have been challenging topic. Regular lipid parameter assessment and maintain a High HDL levels, High Apo AI levels should be the target of lipid management. Smokers are at higher risk for stroke incidence as compared to the non smokers. All the efforts to increase HDL levels should be the mainstay of stroke prevention in patients which have higher risk of stroke.

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