Original Article

An Observational Study of Association of Extended Lipid Profile with Types of Stroke and Its Outcomes

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Abstract

Background: Alterations in the lipid profile can elevate the risk of ischemic stroke (IS). A connection exists between serum lipid concentrations and short-term mortality. Serum cholesterol levels in IS and hemorrhagic stroke (HS) should be assessed for prescribing lipid-lowering medication, which can improve outcomes. The primary objective was to assess the association between various parameters of lipid profile and stroke types (i.e. ischemic and hemorrhagic). The secondary objective was to determine risk factors of mortality following stroke, and the correlation of lipid profile with carotid intimal medial thickness (CIMT) among stroke patients. **Material and Methods:** An observational study was carried out from June 2019 to July 2021 on 200 stroke patients. Each patient's serum extended lipid profile was measured. MRI/MRI angiography, CT scan, as well as Carotid Doppler were performed. Outcomes were noted in terms of mortality. Determination of risk factors of mortality was done by performing logistic regression. A p-value below 0.05 was deemed to be significant. **Results:** Both HS and IS groups had comparable total cholesterol (180 vs. 204 mg/dL), triglycerides (146 vs. 173 mg/dL), VLDL (29.2 vs. 34.6 mg/dL), LDL (113.2 vs. 135.4 mg/dL), and HDL (36 vs. 36 mg/dL) (P>0.05 in all). Among patients with HS, CIMT showed a significant positive correlation with TG (r= 0.574, p=0.046) and a significant negative correlation with HDL (r=-0.512, p=0.046), while among patients with IS, CIMT showed a significant positive correlation with VLDL (r= 0.519, p=0.036). On performing univariate regression, diastolic blood pressure, apolipoprotein A1, and apolipoprotein B were significant risk factors of mortality with odds ratios of 1.053, 0.939 and 1.036, respectively. **Conclusion:** The monitoring of extended lipid profile seems of significance in patients with stroke since higher levels of apolipoprotein A1 and B increase the risk of mortality. Moreover, higher TG, higher VLDL and lower HDL were associated with higher CIMT and atherosclerosis.

Keywords: Cholesterol, Ischemic Stroke, Hemorrhagic Stroke, Dyslipidemia, Mortality.

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Introduction

Stroke is defined as "the sudden onset of clinical signs indicating a focal (or global) disruption of cerebral function, which occurs for above 24 hours or resulting in mortality, without any apparent cause other than a vascular origin".[1]

In India, stroke ranks as the fourth most common cause of death and the fifth most frequent cause of disability, marked by a significant incidence rate of 108-172 per lakh population per year and prevalence rate of 26-757 per lakh population per year. In addition, the 1-month mortality rate has been reported to be 18-42% in different states of India.[2]

Stroke is broadly classified as ischemic and hemorrhagic types. The predominant type of stroke is ischemic stroke (IS), as it constitutes 85% of total strokes; while hemorrhagic stroke (HS) is responsible for 15% cases.[3] Both HS and IS are devastating acute cerebrovascular accidents (CVA) with adverse outcomes. Thus, stroke demands acute critical care with long term management – to limit the adverse outcomes in stroke patients.[4] Factors that adversely affect stroke outcomes include higher age, female gender, existing comorbidities like diabetes mellitus, stroke severity, obesity, smoking, heart disease, atrial fibrillation, pre-stroke functional disability, as well as posterior circulation stroke syndrome.[5]

Although the association of risk factors like atrial fibrillation and hypertension are reported, the relation of lipid profile with stroke continues to be studies.[6] While some studies show that increased serum cholesterol levels are associated with elevated risk of IS,[7-10] while showing inverse relation to HS.[11] Additionally, elevated triglyceride levels are reported to elevate risk of both HS as well as IS in previous studies.[7,12]

Stroke is linked to alterations in lipid levels, possibly due to the stress and the overproduction of catecholamines that occur during an acute stroke. Serum lipid levels are also known to significantly affect short-term mortality in patients with stroke, and for these reasons, lipid profile to be the primary goal of prevention of primary and secondary stroke.[7]

We conducted this study to elaborate the findings of association

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between various parameters of lipid profile with stroke types and its outcomes. The results of the present study may allow us to have a better approach for management of lipid profile of the patients to lower the mortality and improve the outcomes.

Materials and Methods

Study Design: The present observational study was carried out at a teaching hospital for a duration of 2 years, i.e. from June 2019 to July 2021. Ethical clearance for conducting the study was taken from the Institutional Ethical Committee prior to commencing the study.

Study setting: The study population included patients (>18 years) who attended the Medicine Outpatient Department (OPD) and indoor patients with suspected CVA. Any patients who received lipid-lowering agents; drugs that may modify lipid profile (such as thiazide diuretic, progestins, and anabolic steroids); or drugs which predispose to stroke (such as oral contraceptive pills and hormonal replacement therapy) were excluded.

Sample size: The sample size for the current study was determined on the basis of findings of Togha M et al,[8] who found that mean±SD of cholesterol was 223.53 ± 48.71 and TG was 152.88 ± 82.61 mg/dl. Consequently, 200 patients diagnosed with cerebrovascular disease, confirmed through radioimaging studies, were included who presented to the hospital during the study period. Every patient was asked to provide written informed consent, and their demographic as well as clinical characteristics were documented.

Methodology: Five ml of blood sample was collected in a plain yellow-topped vacutainer for extended lipid profile which comprised of total cholesterol, triglycerides, LDL, VLDL, HDL, Lipoprotein (a), apoLipoprotein A1 and apo Lipoprotein b. Before taking blood samples, all subjects had to fast for at least 12 hours. The serum total cholesterol levels were estimated using the enzymatic colorimetric method on the basis of CHOD-PAP technique. The glycerol phosphate oxidase/peroxidase enzyme technique was used to measure the level of triglycerides. The two stages in the determination of HDL cholesterol include precipitations and cholesterol estimation of HDL fraction by modification. Lipid profile determination of various parameters of LDL, VLDL, and chylomicrons was done by the principle of precipitation whereby the patient blood sample which was centrifugated to bring out the serum supernatant was added with magnesium ions and phosphotungstic acid. The addition of these precipitated the cholesterol subunits and chylomicrons. Then, further enzymatic analysis was done and Friedewald formula was used to estimate LDL cholesterol in which triglyceride, HDL, and total cholesterol levels were used as the basis for this standard WHO-approved method.[13] Philips MRI Achieva, 1.5 Tesla was used to perform MRI/MRI angiography, and Philips Brilliance, 16 slice CT to perform CT scan. Wipro G.E, Logic 400 MD Siemens Sonoline G60S was used to perform Carotid Doppler for CIMT.

The outcome measures noted in the present study were morality and its risk factors.

Statistical Analysis: The software used for performing data analysis were Microsoft Excel and Statistical Package for Social Sciences (SPSS). The categorical variables were presented as number (%), and quantitative data was presented in the form of median and mean with interquartile range. The Shapiro-Wilk test was employed to evaluate data normality; whereas for data not following normal distribution, the study used non-parametric tests. For determining the association, the test used was Mann-Whitney Test. To find correlation of CIMT(mm) with lipid profile, Spearman rank correlation coefficient was used. The risk factors of mortality were evaluated through univariate/multivariate logistic regression analysis. P-value below 0.05 was deemed statistically significant.

Ethical statement: The research complies with the guidelines for human studies and was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. The study was conducted after a written informed consent and that the study protocol was approved by the institute's ethical committee on human research of BVP, Pune (IEC/HR/2013/41/60), dated 14.10.2013.

Results

The patients' mean age was 64 ± 13.5 years, with 144 (72%) being males. Number of patients from urban area was 108 (54 %). Diabetes mellitus, hypertension, ischemic heart disease, and TIA/ CVA were present in 40(20.00%), 100(50.00%), 20(10.00%), and 44(22.00%) patients, respectively. 64 (32.00%) patients were smokers, 68 (34.00%) were taking alcohol, and 88 (44.00%) were taking tobacco. The mean BMI was 26.3±4.2 kg/m², and mean SBP and DBP were 174.2±32.01 and 103.4±18.03 mm Hg, respectively [Table 1].

The symptoms observed in the cases were diverse, with 80 (40.00%) experiencing right side weakness, an equal number presenting with left side weakness. In 28 (14.00%) cases, the primary symptom was an inability to speak, while 24 (12.00%) cases exhibited right facial deviation. Dizziness and loss of consciousness were reported in 10.00% of cases each. Other less common symptoms included generalized tonic-clonic seizures (GTCS) and slurring of speech, each occurring in 8.00% of cases. Loss of balance, vomiting was observed in 4.00% of cases each. Laboured breathing, loss of sensation and left facial deviation were rare, each occurring in 2.00% of cases [Figure 1].

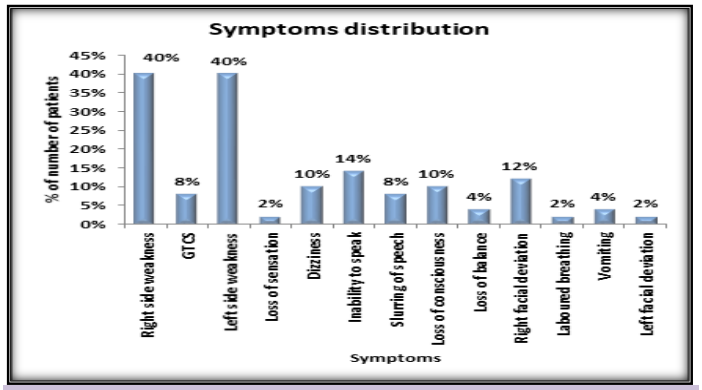

Figure 1: Symptoms distribution.

The observed signs in the cases were varied, with 72 (36.00%) showing left hemiplegia, 60 (30.00%) exhibiting right hemiplegia, and 52 (26.00%) displaying 7th nerve upper motor

neuron (UMN) palsy. Additionally, 32 (16.00%) cases presented with right hemiparesis, while 28 (14.00%) cases showed signs of aphasia. Other manifestations included dysarthria in 12.00% of cases, cerebellar signs in 8.00% of cases, left hemiparesis and sensory loss in 4.00% of cases each. Less frequently observed signs included being comatose, left monoplegia, and adopting decerebrating or decerebrate postures, each occurring in 2.00% of cases [Figure 2].

Among 200 patients of stroke, 180(90%) had IS, while 20(10%) had HS, and among those patients, during the follow-up 20(10%) died.

The HS patients were comparable with IS patients in terms of total cholesterol (mg/dL) (204 vs. 180, P=0.128), triglyceride (mg/dL) (173 vs. 146, P=0.348), VLDL (mg/dL) (34.6 vs. 29.2, P=0.348), LDL (mg/dL) (135.4 vs. 113.2, P=0.133) and HDL (mg/dL) (36 vs. 36, P=0.361) [Table 2].

Among patients with HS, CIMT showed a significant positive correlation with TG (r= 0.574, p=0.046) and a significant negative correlation with HDL (r=-0.512, p=0.046). Among patients with IS, CIMT showed a significant positive correlation with VLDL (r= 0.519, p=0.036) as shown in [Table 3].

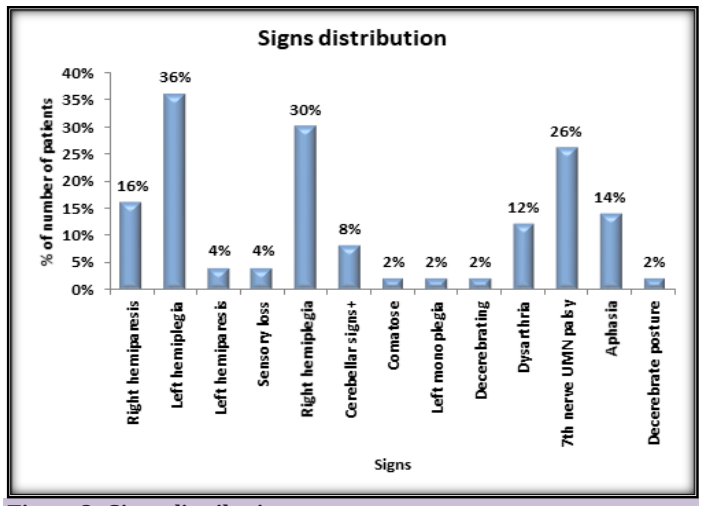

Figure 2: Signs distribution.

On performing univariate regression, diastolic blood pressure, apolipoprotein A1, apolipoprotein B were significant risk factors of mortality. With the increase in apolipoprotein A1, risk of mortality significantly decreases with odds ratio of 0.939. With the increase in diastolic blood pressure, apolipoprotein B, risk of mortality significantly increases with odds ratio of 1.053, 1.036, respectively [Table 4]. However, on performing multivariate regression, none of the parameter was independent significant risk factor of mortality [Table 5].

Table 1: Demographic characteristics distribution

Demographic characteristics	n(%)	Mean ± SD	Median(25th-75th percentile)	Range
Gender				
Female	56 (28.00%)	-	-	-
Male	144 (72.00%)			
Area of residence				
Rural	92 (46.00%)	-	-	-
Urban	108 (54.00%)			
Past history of any illness				
Diabetes mellitus	40 (20.00%)	-	-	-
Hypertension	100 (50.00%)	-	-	-
Ischemic heart disease	20 (10.00%)	-	-	-
TIA/ CVA	44 (22.00%)	-	-	-
Personal history				
Alcohol	68 (34.00%)	-	-	-
Tobacco	88 (44.00%)	-	-	-
Smoking	64 (32.00%)	-	-	-
Body mass index(kg/m²)				
18.5 to 24.99 kg/m² {Normal BMI}	56 (35.90%)	26.3 ± 4.2	26.8(23.4-28.55)	18.7-39.1
25 to 29.99 kg/m² {Overweight}	76 (48.72%)			
>=30 kg/m² {Obese}	24 (15.38%)			
Age(years)	-	64 ± 13.5	65(50.5-75)	46-95
Systolic blood pressure(mmHg)	-	174.2 ± 32.01	180(160-197.5)	110-240
Diastolic blood pressure(mmHg)	-	103.4 ± 18.03	100(92.5-110)	70-150

BMI: body mass index; CVA: cerebrovascular accident; TIA: transient ischemic attack.

Table 2: Association of lipid profile with type of stroke

Lipid profile	HS (n=20)	IS (n=180)	Total	P value
Total cholesterol(mg/dL)	180(172-192)	204(192-220)	203(189.5-219.75)	0.128*
Triglyceride(mg/dL)	146(136-208)	173(148-214)	172.5(146.5-212.5)	0.348*
VLDL(mg/dL)	29.2(27.2-41.6)	34.6(29.6-42.8)	34.5(29.3-42.5)	0.348*
LDL(mg/dL)	113.2(96.8-121.8)	135.4(128-155.8)	134.3(122.85-155.05)	0.133*
HDL(mg/dL)	36(36-42)	36(30-37)	36(30.5-37)	0.361*

* Mann Whitney test

HDL: high-density lipoprotein; LDL: low-density lipoprotein; VLDL: very low-density lipoprotein; HS: hemorrhagic stroke; IS: Ischemic stroke.

Table 3: Correlation of CIMT with lipid profile in different stroke types

CIMT	HS		IS	
Lipid profile	**r-value**	**p-value**	**r-value**	**p-value**
TC	0.319	0.213	0.298	0.376
TG	0.574	0.046	0.392	0.096
VLDL	0.387	0.081	0.519	0.036
LDL	0.164	0.213	0.328	0.218
HDL	-0.512	0.046	-0.418	0.059

HDL: high-density lipoprotein; LDL: low-density lipoprotein; VLDL: very low-density lipoprotein; HS: hemorrhagic stroke; IS: Ischemic stroke; CIMT: carotid intimal medial thickness.
Spearman rank correlation coefficient, R= correlation coefficient, p= level of significance.

Table 4: Univariate logistic regression to find out significant risk factors of mortality

Variable	Beta coefficient	Standard error	P value	Odds ratio	Odds ratio Lower bound (95%)	Odds ratio Upper bound (95%)
Age(years)	0.000	0.035	1.000	1.000	0.933	1.072
Systolic blood pressure(mm of Hg)	0.030	0.018	0.093	1.030	0.995	1.067
Diastolic blood pressure(mm of Hg)	0.052	0.026	0.049	1.053	1.000	1.109
Lipoprotein A (mg/dL)	0.009	0.014	0.515	1.009	0.981	1.038
Apolipoprotein A1 (mg/dL)	-0.063	0.025	0.011	0.939	0.894	0.986
Apolipoprotein B (mg/dL)	0.036	0.015	0.014	1.036	1.007	1.067
Gender						
Female				1.000		
Male	-1.451	0.919	0.114	0.234	0.039	1.419
Diabetes mellitus	-1.187	1.596	0.457	0.305	0.013	6.964
Hypertension	2.617	1.537	0.089	13.692	0.673	278.421
Ischemic heart disease	1.207	1.130	0.285	3.345	0.366	30.610
TIA/ CVA	0.000	1.138	1.000	1.000	0.107	9.304
Alcohol	-0.525	1.018	0.606	0.591	0.080	4.349
Tobacco	-0.023	0.940	0.981	0.977	0.155	6.165
Smoking	-0.415	1.019	0.684	0.661	0.090	4.866
Dyslipidemia	0.596	1.652	0.718	1.814	0.071	46.226

CVA: cerebrovascular accident; TIA: transient ischemic attack.

Table 5: Multivariate logistic regression to find out significant independent risk factors of mortality

Variable	Beta coefficient	Standard error	P value	Odds ratio	Odds ratio Lower bound (95%)	Odds ratio Upper bound (95%)
Diastolic blood pressure (mm of Hg)	0.023	0.030	0.441	1.024	0.965	1.086
Apolipoprotein A1 (mg/dL)	-0.041	0.024	0.092	0.960	0.915	1.007
Apolipoprotein B (mg/dL)	0.022	0.015	0.155	1.022	0.992	1.054

Discussion

In the present study, most of the cases, that is, 90%, were ischemic -type stroke, and 10% were hemorrhagic -type stroke. We found that lipid profile was found to be more deranged in ischemic stroke as compared to hemorrhagic stroke; however statistically it failed to cross the boundaries of significance. As for CIMT, it showed a significant positive correlation with TG and negative correlation with HDL in HS patients; and positive correlation with VLDL in IS patients.

In comparison, Alemayehu et al,[14] found that type of stroke showed significant association with lipid profile. When compared to patients with HS and controls, those with IS had significantly higher total cholesterol (mg/dl) (168.5 vs. 159 vs. 158, P=0.01) and higher LDL-c (mg/dl) (119 vs. 104 vs. 105, P<0.001), whereas controls had significantly higher HDL-c (mg/dl) (52 vs. 48 vs. 48, P<0.001) and triglycerides (mg/dl) (127 vs. 113 vs. 88, P<0.001) than patients with IS and HS. Thus, HDL-c and stroke were inversely related in their investigation, which was attributed to HDL-C in line with its mechanism of decreasing the levels of cholesterol in peripheral cells as it successfully transfers it from peripheral parts of the body (cells) to the liver and also prevents the peroxidation of lipids, and the higher levels of overall total cholesterol and the low-density part of the lipoproteins in the patients with IS was explained by the fact that elevations in these parameters seem to be a marker of atherosclerotic as well as prothrombotic alterations and have a direct atherogenic effect. Increased total cholesterol levels have been connected to fibrinolysis and coagulation disorders, which are both associated with IS. Similarly, in contrast with the mechanism of HDL, LDL promotes the peroxidation of the lipids and these products that deposited in blood vessels increasing the atherogenecity and further the chances of IS.[14]

Gong X et al,[15] in a systematic review including 50 studies (n=3,301,613) found that elevated total cholesterol and triglyceride levels showed association with increased risk of IS and decreased HS risk (P < 0.001). On the contrary, HDL-C showed positive correlation with decreased IS risk, however no such association was seen with HS risk. Also, increased LDL-C or non–HDL-C were not significantly associated with IS and HS risk.

Togha M et al,[8] mentioned that only triglycerides level was

significantly associated with type of stroke, as levels were significantly higher in IS than HS (164.9 ± 86.1 vs. 117.2 ± 71.1,P<0.001). IS and HS stroke groups were similar in terms of HDL (46.8 ± 16.2 vs. 48.4 ±14.1 P=0.577) and LDL (146.8 ± 42.5 vs. 146.1 ± 57.6, P=0.927).

Vakilian A et al,[16] reported that when compared with patients of HS, those with IS had significantly higher triglycerides (130.62 ± 59.25 vs. 101.32 ± 35.63 mg/dl, P=0.007) and lower HDL-C (36.86 ± 10.22 vs. 42.55 ± 11.47 mg/dl, P=0.007). There was comparable total cholesterol as well as LDL-C levels between both groups.

Variations in study results may stem from differences in the racial, lifestyle, environmental, sociocultural, and socioeconomic characteristics of the populations studied. Nonetheless, dyslipidemia is widely recognized as a key factor in the onset of stroke. Because of atherosclerosis, it becomes an especially important risk factor of stroke. As a result, post-stroke patients may improve from cholesterol-lowering treatments such as statins, which may decrease the likelihood of recurrence and enhanced functional results.[14]

As for outcomes, in the present study, 20(10%) patients expired. Like present study, the mortality rate was 10.6% Lisk DR et al study.[17] The rate of mortality in the study by Gidey K et al,[18] was 13.6%. Russell JB et al,[19] reported higher in-hospital mortality rate, i.e. 34.8%. Similarly, Viderman et al,[20] reported higher mortality rate of 37.2%. The higher mortality rate in these studies was due to sample population only severely ill patients were included. Kamabu LA et al,[21] reported mortality rate of 31.7% in the study on 180 patients with stroke.

The variation in mortality rate may be due to heterogeneous population and varied follow-up periods, and when we determined the risk factors that affected the mortality, we found that diastolic blood pressure, apolipoprotein A1, and apolipoprotein B were significant risk factors of mortality indicating the role of extended lipid profile estimation for outcomes of stroke.

Consistent with the current study, previous research has indicated that various factors are linked to mortality in stroke patients. Gidey K et al,[18] reported that presence of complications (such as aspiration pneumonia, hospital-acquired pneumonia, electrolyte abnormalities, acute kidney injury, and complications related to hospital stay and progression of disease) significantly predicted increased in-hospital mortality. Russell JB et al,[19] mentioned that the factors that predicted in-hospital mortality were hypertension, prior stroke, Glasgow Coma Scale score below 8, clinical diagnosis in the absence of imaging, HS, and aspiration pneumonia. Viderman et al,[20] reported that the factors that predicted mortality in stroke patients were male sex. Moreover, HS showed association with lesser mortality, whereas cerebral edema showed association with increased mortality risk (P<0.05). Kamabu LA et al,[21] reported that the factors that showed positive correlation with mortality were HS, reaching hospital (late than 24 hours), poor adherence towards antidiabetics and antihypertensive, recurrent stroke, and advanced age more than 75 years.

Conclusion

In conclusion, lipid profile was found to be more deranged in ischemic stroke as compared to hemorrhagic stroke, though statistically it was comparable. Diastolic blood pressure, apolipoprotein A1, and apolipoprotein B were significant risk factors of mortality indicating the role of extended lipid profile estimation for outcomes of stroke. Thus, lipid profile must be monitored in patients with stroke and higher levels must be managed with lipid lowering medications.

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Conflicts of interest

There are no conflicts of interest.

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