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Original Research Article

ROLE OF HDL IN CEREBROVASCULAR ACCIDENT AND COMPARISON OF HDL LEVEL IN ISCHEMIC AND HAEMORRHAGIC STROKE: A COMPARATIVE STUDY

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ABSTRACT

Background: Stroke is a leading cause of death and disability worldwide, classified broadly into ischemic and haemorrhagic subtypes. The role of serum lipid profile — particularly high-density lipoprotein cholesterol (HDL-C) — in modulating stroke risk and differentiating stroke subtypes remains an active area of investigation. HDL exerts atheroprotective effects through reverse cholesterol transport, anti-inflammatory action, inhibition of platelet aggregation, and endothelial protection. Low HDL-C is an established risk factor for ischemic stroke, yet paradoxically, higher HDL-C levels have been documented in haemorrhagic stroke, suggesting divergent lipid pathophysiology across stroke subtypes. Population-based data from South Indian tertiary care settings are sparse, warranting comparative studies to delineate the lipid profile differences between these two stroke categories. The objective is to study serum HDL levels in cerebrovascular accident (CVA) patients and compare HDL and full lipid profile (total cholesterol, triglycerides, LDL) between ischemic and haemorrhagic stroke; to compare blood pressure, age, and random blood sugar between stroke subtypes; and to determine site involvement in haemorrhagic and artery involvement in ischemic stroke.

Materials and Methods: This prospective comparative study enrolled 100 CVA patients (aged 35–70 years) admitted to Government General Hospital, Kurnool, over two years. Patients were diagnosed and classified by CT/MRI brain as ischemic (n=56) or haemorrhagic (n=44). Biochemical parameters (total cholesterol, HDL, TG, LDL, RBS) and clinical parameters (age, SBP, DBP) were recorded. Statistical analysis used Student's t-test for continuous variables and chi-square for categorical data; $p < 0.05$ was considered significant. ROC curve analysis was performed for HDL's diagnostic role in stroke.

Results: Ischemic stroke constituted 56% and haemorrhagic stroke 44%. Mean age for haemorrhagic stroke (58.7 ± 8.59 years) was significantly higher than ischemic stroke (51.6 ± 9.35 years; $p < 0.001$). SBP and DBP were significantly elevated in haemorrhagic stroke ($p < 0.001$ and $p = 0.032$ respectively). Mean HDL was significantly higher in haemorrhagic stroke (42.8 ± 5.43 mg/dL) compared to ischemic stroke (37.8 ± 5.58 mg/dL; $p < 0.001$). Total cholesterol was also significantly higher in haemorrhagic stroke (211 ± 13.19 vs 200.4 ± 16.98 mg/dL; $p < 0.001$). TG and LDL differences were not statistically significant. ROC curve analysis showed HDL had fair discriminatory capacity for stroke (AUC = 0.7). Putamen was the most common haemorrhagic site (34.1%); Middle Cerebral Artery was most involved in ischemic stroke (46.4%).

Conclusion: Serum HDL levels differ significantly between ischemic and haemorrhagic stroke, with haemorrhagic stroke patients exhibiting paradoxically higher HDL. Low HDL levels in ischemic stroke reflect the

atheroprotective deficit, while the elevated HDL in haemorrhagic stroke may represent dysfunctional HDL with impaired anti-inflammatory properties. Lipid profiling including HDL-C should be incorporated in CVA risk assessment and management protocols.
Keywords: HDL cholesterol; ischemic stroke; haemorrhagic stroke; cerebrovascular accident; lipid profile; reverse cholesterol transport; atherosclerosis.

INTRODUCTION

Stroke is defined as rapidly developing clinical signs of focal or global cerebral dysfunction lasting at least 24 hours or leading to death, with no apparent cause other than vascular origin.[1] It is the second leading cause of death globally and a major contributor to long-term disability. One in eight deaths worldwide is attributed to stroke, imposing an enormous burden on families and healthcare systems.[2] Strokes are broadly classified as ischemic (approximately 80%) and haemorrhagic (approximately 20%), with fundamentally different pathophysiological mechanisms, risk factor profiles, and outcomes.[3,4] Among modifiable risk factors, dyslipidaemia — particularly perturbations in HDL cholesterol — has garnered significant interest. HDL-C is known to exert atheroprotective effects through reverse cholesterol transport, anti-oxidant activity, and inhibition of platelet aggregation.[5] While elevated total cholesterol and low HDL-C are well-established risk factors for ischemic stroke,[6,7] their relationship with haemorrhagic stroke is less well defined and sometimes paradoxical. Some prospective studies suggest that high HDL-C may be associated with an increased risk of haemorrhagic stroke.[8] The TC/HDL-C ratio has been proposed as a superior cardiovascular risk predictor compared to either lipid parameter alone.[9] Despite global data, comparative lipid studies from South Indian tertiary care hospitals are limited, and there is a clinical need to characterise lipid differences across stroke subtypes to guide preventive strategies.[10] This study was undertaken to compare serum HDL levels and full lipid profiles between ischemic and haemorrhagic stroke patients admitted to Government General Hospital, Kurnool.

Aims and Objectives: To study serum HDL levels in patients with cerebrovascular accident and to compare HDL-C and full lipid profile (total cholesterol, TG, LDL), blood pressure, age, and RBS between ischemic and haemorrhagic stroke subtypes, and to determine site and artery involvement in each stroke category.

MATERIALS AND METHODS

This prospective comparative study was conducted at the Department of General Medicine, Government General Hospital (GGH), Kurnool, over a period of two years. A total of 100 patients admitted with CVA and neurological weakness, aged 35–70 years of both sexes, were enrolled. Patients with pre-existing cardiac disease, no CT evidence of stroke, CVA secondary to tumour or trauma, and liver disease were excluded. All patients provided written informed consent. CT/MRI brain plain was performed on all subjects to classify stroke as ischemic or haemorrhagic and to document lesion site and artery involvement.[11] Biochemical investigations — total cholesterol (enzymatic colorimetric), HDL-C (direct enzymatic), triglycerides (enzymatic GPO-POD), LDL-C (Friedewald formula), and RBS (hexokinase method) — were estimated on an automated biochemistry analyser.[12] Clinical parameters including systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded at admission. Data were entered in MS Excel and analysed using SPSS v20. Continuous variables were compared using Student's independent t-test and categorical variables using chi-square test. Receiver operating characteristic (ROC) curve analysis was performed to assess HDL's discriminatory ability for stroke type. Statistical significance was set at $p < 0.05$.

RESULTS

Of 100 CVA patients, 56 (56%) had ischemic and 44 (44%) had haemorrhagic stroke. Majority were aged 51–55 years (19%), with overall mean age 54.7 ± 9.66 years. The mean age of haemorrhagic stroke patients (58.7 ± 8.59 years) was significantly greater than ischemic stroke patients (51.6 ± 9.35 years; $p < 0.001$). Female patients (51%) slightly outnumbered males (49%), though gender distribution was not significantly different between stroke types ($p = 0.859$). Mean RBS was 149.1 ± 31.2 mg/dL in ischemic and 138.8 ± 28.7 mg/dL in haemorrhagic stroke ($p = 0.094$, NS). SBP was significantly higher in haemorrhagic stroke (164.4 ± 14.85 vs 154.4 ± 14.19 mmHg; $p < 0.001$) as was DBP (102 ± 8.32 vs 98.5 ± 6.66 mmHg; $p = 0.032$). All results are summarised in [Table 1]. Serum HDL was significantly higher in haemorrhagic stroke (42.8 ± 5.43 mg/dL) compared to ischemic stroke (37.8 ± 5.58 mg/dL; $p < 0.001$; [Figure 1]). Total cholesterol was also significantly higher in haemorrhagic stroke (211 ± 13.19 vs 200.4 ± 16.98 mg/dL; $p < 0.001$; Figure 1). Triglycerides (174.7 ± 17.3 vs 171.3 ± 14.5 mg/dL; $p = 0.279$) and LDL (125.7 ± 19.36 vs 120.4 ± 17.2 mg/dL; $p = 0.152$) did not differ significantly between stroke types (Figure 2). ROC curve analysis demonstrated HDL had fair discriminatory capacity for stroke type (AUC = 0.7). Site and artery involvement are summarised in [Table 2]. Putamen was the most common haemorrhagic site (34.1%), while MCA was most involved in ischemic stroke (46.4%).

Table 1: Comparison of Clinical and Biochemical Parameters between Ischemic and Haemorrhagic Stroke (n=100)

Parameter	Ischemic (n=56) Mean ± SD	Hemorrhagic (n=44) Mean ± SD	p value
Age (years)	51.6 ± 9.35	58.7 ± 8.59	< 0.001 (S)
Gender (F:M)	29F : 27M (51.8% F)	22F : 22M (50% F)	p=0.859 (NS)
RBS (mg/dL)	149.1 ± 31.2	138.8 ± 28.7	p=0.094 (NS)
SBP (mmHg)	154.4 ± 14.19	164.4 ± 14.85	< 0.001 (S)
DBP (mmHg)	98.5 ± 6.66	102 ± 8.32	p=0.032 (S)
Total Cholesterol (mg/dL)	200.4 ± 16.98	211 ± 13.19	< 0.001 (S)
Triglycerides (mg/dL)	171.3 ± 14.5	174.7 ± 17.3	p=0.279 (NS)
HDL (mg/dL)	37.8 ± 5.58	42.8 ± 5.43	< 0.001 (S)
LDL (mg/dL)	120.4 ± 17.2	125.7 ± 19.36	p=0.152 (NS)

S = significant; NS = not significant; SBP = systolic blood pressure; DBP = diastolic blood pressure; RBS = random blood sugar; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglycerides

Table 2: Site Involvement in Haemorrhagic and Artery Involvement in Ischemic Stroke

Hemorrhagic Stroke – Site (n=44)	n (%)	Ischemic Stroke – Artery (n=56)	n (%)
Putamen	15 (34.1%)	Middle Cerebral Artery	26 (46.4%)
Thalamus	8 (18.2%)	Vertebrobasilar Artery	10 (17.8%)
Temporo-parietal	6 (13.6%)	Anterior Cerebral Artery	8 (14.3%)
Multiple sites	6 (13.6%)	Internal Carotid Artery	7 (12.5%)
Brain Stem	4 (9.1%)	Multiple Arteries	5 (9.0%)
Fronto-parietal	3 (6.8%)	—	—
Cerebellum	2 (4.5%)	—	—

MCA = Middle Cerebral Artery; ACA = Anterior Cerebral Artery; VBA = Vertebrobasilar Artery; ICA = Internal Carotid Artery.

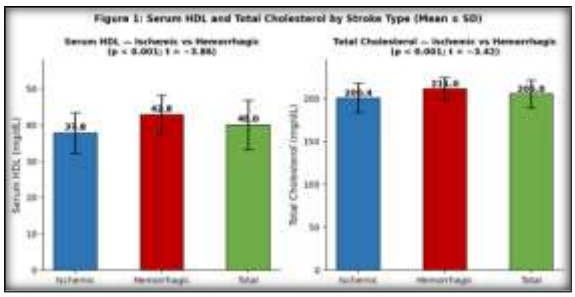

Figure 1: Serum HDL and Total Cholesterol — Ischemic vs Haemorrhagic Stroke (Mean ± SD)

HDL significantly higher in haemorrhagic stroke ($p < 0.001$); Total cholesterol significantly higher in haemorrhagic stroke ($p < 0.001$)

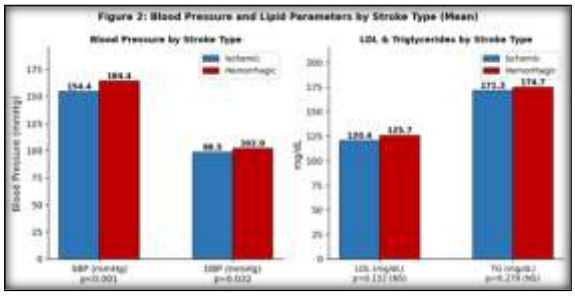

Figure 2: Blood Pressure and Lipid Parameters (LDL, TG) by Stroke Type

SBP ($p<0.001$) and DBP ($p=0.032$) were significantly higher in haemorrhagic stroke; LDL and TG differences were not statistically significant ($p=0.152$ and $p=0.279$ respectively)

DISCUSSION

The present comparative study of 100 CVA patients at GGH Kurnool found ischemic stroke to be more prevalent (56%) than haemorrhagic stroke (44%), consistent with global epidemiology where ischemic stroke constitutes approximately 80% of all strokes.[13] The higher prevalence of ischemic stroke reflects the predominance of large-artery atherosclerosis and small-vessel disease as the dominant mechanisms of cerebral infarction in the Indian population, where cardiovascular risk factors such as hypertension, dyslipidaemia, and diabetes are highly prevalent.[14] Saproo N et al. in a comparable study from Gurugram similarly reported ischemic predominance, with mean age of 57.82 years aligning closely with our findings.[15]

Haemorrhagic stroke patients were significantly older (mean 58.7 vs 51.6 years; $p < 0.001$). This age difference reflects the distinct pathophysiological mechanisms of the two stroke subtypes.16 Ischemic stroke predominantly results from atherosclerotic plaque formation and thromboembolism — processes that begin earlier in life driven by endothelial dysfunction, lipid oxidation, and foam cell formation from the third decade.17 Haemorrhagic stroke, in contrast, is primarily driven by hypertension-induced lipohyalinosis and fibrinoid necrosis of small penetrating arteries — a process that accelerates with increasing duration of uncontrolled hypertension and structural arterial remodelling seen more commonly in older patients.18 Charcot-Bouchard microaneurysm formation in the basal ganglia perforators, predisposing to putaminal haemorrhage — the most common site in our study (34.1%) — is a direct consequence of chronic hypertensive arteriolar injury.[19] AizazAli et al. reported an overall mean stroke age of 62 ± 14 years in a Qatari cohort, slightly higher than ours, possibly reflecting greater longevity in that population.[20]

SBP (164.4 vs 154.4 mmHg, p < 0.001) and DBP (102 vs 98.5 mmHg, p = 0.032) were significantly elevated in haemorrhagic stroke, confirming hypertension as its dominant risk factor. The pathophysiological basis for this difference is the direct mechanical effect of elevated systemic blood pressure on cerebrovascular integrity.[21] Sustained hypertension causes structural changes in small cerebral perforating arteries — medial hypertrophy, replacement of smooth muscle by collagen, lipohyalinosis — progressively weakening the arterial wall and predisposing to spontaneous rupture.[22] In ischemic stroke, while hypertension also plays a role through acceleration of large-vessel atherosclerosis, the acute blood pressure at presentation is often reactive (Cushing response) rather than the primary cause of the insult, explaining the relatively lower admission blood pressures in ischemic compared to haemorrhagic stroke patients.[23] AizazAli et al. reported 80% hypertension prevalence in stroke patients, underscoring hypertension's pre-eminent role across both stroke subtypes.[20]

The pivotal finding of this study is the significantly higher mean HDL in haemorrhagic stroke (42.8 ± 5.43 mg/dL) compared to ischemic stroke (37.8 ± 5.58 mg/dL; p < 0.001). This paradox — higher HDL in the stroke associated with arterial rupture rather than occlusion — has important mechanistic implications and has been documented in prior literature.[24] In ischemic stroke, the low HDL reflects the failure of the reverse cholesterol transport pathway. HDL removes excess cholesterol from arterial wall macrophage-derived foam cells and transports it to the liver via the ATP-binding cassette transporter A1 (ABCA1) pathway, impeding atherosclerotic plaque development.[25] When HDL-C is low, this efflux capacity is reduced, cholesterol accumulates in macrophages promoting foam cell formation, oxidised LDL accelerates endothelial injury, and vulnerable plaque progresses — ultimately leading to thromboembolic occlusion of cerebral arteries. HDL also exerts potent anti-inflammatory actions by suppressing endothelial expression of VCAM-1, ICAM-1, and E-selectin, and by scavenging oxidised phospholipids. Furthermore, Apolipoprotein A-1 (ApoA-1), the major HDL apoprotein, reduces platelet activation and inhibits thrombin generation, reducing thrombotic risk. In the ischemic brain, HDL has been shown to reduce neuronal damage in middle cerebral artery occlusion models by maintaining eNOS activity and cerebrovascular autoregulation. ApoA-1 infusion reduced brain lesion size by 64% in experimental MCA occlusion models.28 Deficiency of these protective mechanisms in ischemic stroke patients — reflected by the lower HDL (37.8 mg/dL) — is therefore mechanistically consistent with their atherothrombotic disease.

The paradoxically elevated HDL in haemorrhagic stroke patients (42.8 mg/dL) is best explained by the concept of dysfunctional HDL. In patients with metabolic syndrome, hypertension, chronic kidney disease, or severe systemic inflammation, HDL particles undergo structural and compositional changes — loss of ApoA-1, enrichment with serum amyloid A (SAA) and myeloperoxidase (MPO)-oxidised components — that convert them from cardioprotective to proinflammatory particles. Dysfunctional HDL fails to suppress macrophage activation, loses reverse cholesterol transport capacity, and may paradoxically activate endothelial inflammatory pathways. Schwedhelm proposed calculating 'biologically effective' HDL to differentiate functional from dysfunctional HDL. Additionally, in hypertensive individuals — who predominate among haemorrhagic stroke patients — the renin-angiotensin-aldosterone system (RAAS) activation and endothelin-1 overexpression cause systemic vasospasm and oxidative stress that can paradoxically elevate measured HDL-C while the particle is functionally inert. This is further supported by the failure of pharmacological HDL-C raising strategies — AIM-HIGH (niacin), ACCORD (fenofibrate), and ILLUMINATE (torcetrapib) — to reduce cardiovascular outcomes, suggesting that quantity of HDL-C alone does not confer benefit if the particles are dysfunctional. Zhang et al. demonstrated a 9% decrease in ischemic stroke likelihood per SD increase in HDL-C (aOR 0.91, 95% CI 0.85–0.98), confirming HDL's protective role specifically against ischemic but not haemorrhagic stroke mechanisms. ROC curve analysis in our study yielded AUC = 0.7 for HDL in discriminating stroke type, indicating fair diagnostic utility.

Total cholesterol was significantly higher in haemorrhagic stroke (211 vs 200.4 mg/dL; p < 0.001). Elevated total cholesterol in haemorrhagic stroke, in the context of a higher HDL contribution, suggests a predominance of large-particle, cholesterol-rich HDL and VLDL remnants in this group. Hypercholesterolaemia has not been consistently linked with haemorrhagic stroke in the literature; some studies suggest that very low cholesterol levels may actually increase the risk of intracerebral haemorrhage by weakening vascular wall integrity through impaired phospholipid membrane stability in cerebral arterioles. The non-significant difference in triglycerides (p = 0.279) and LDL (p = 0.152) between stroke subtypes aligns with observations by Saproo N et al. and Damayanthi et al., who also found that these parameters did not significantly discriminate stroke type.[15] Ischemic stroke is predominantly driven by LDL-dependent atherosclerosis and thromboembolism. However, the LDL difference in our study was non-significant, possibly because both ischemic and haemorrhagic patients shared a background of chronic hypertension and mixed dyslipidaemia common in the Kurnool population. The MCA was the most commonly involved artery in ischemic stroke (46.4%), reflecting its broad territory coverage and susceptibility to embolic and atherothrombotic occlusion.

Taken together, the pathophysiological basis for the observed differences between ischemic and haemorrhagic stroke in our study can be summarised as follows: ischemic stroke arises predominantly through HDL deficiency-driven atherosclerosis and thromboembolism, with low HDL failing to clear endothelial cholesterol, suppress inflammation, and prevent platelet aggregation. Haemorrhagic stroke, by contrast, occurs through hypertension-driven arteriolar injury, where elevated HDL may represent a dysfunctional, structurally altered particle that has lost its protective function, contributing to rather than preventing vascular damage. Perovic et al. demonstrated that lower HDL at discharge was associated with worse disability (Barthel index < 60) and higher 90-day mortality in ischemic stroke, while Tian et al. confirmed in 1568 Chinese ischemic stroke patients that low admission HDL predicted adverse outcomes (NIHSS > 10 or death). These findings reinforce the conclusion that quantitative HDL-C measurement, while informative as a population-level risk marker, must be interpreted in the context of stroke subtype and the functional quality of HDL particles.

CONCLUSION

Ischemic stroke was more prevalent (56%) than haemorrhagic stroke (44%) in this series. Haemorrhagic stroke patients were significantly older and had higher SBP and DBP, reflecting the central role of chronic hypertension-induced arteriolar injury in its pathogenesis. Serum HDL was paradoxically and significantly higher in haemorrhagic stroke (42.8 vs 37.8 mg/dL; $p < 0.001$), reflecting dysfunctional HDL particles with impaired atheroprotective capacity in hypertensive individuals, while low HDL in ischemic stroke underscores the failure of reverse cholesterol transport and anti-inflammatory mechanisms. Total cholesterol was also significantly higher in haemorrhagic stroke. Lipid profiling including quantitative and functional HDL-C assessment, alongside blood pressure monitoring, should be integral to CVA risk stratification and management in clinical practice.

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