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Original Article

Lipid Profile and Carotid Artery Plaques in Ischemic Stroke Patients: A Cross-Sectional Doppler Ultrasound Study

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ABSTRACT

Background: Ischemic stroke is strongly linked with atherosclerosis and modifiable vascular risk factors. Dyslipidemia and extracranial carotid plaques are clinically relevant because they identify patients who require intensified secondary prevention.

Objectives: To assess lipid profile abnormalities and carotid artery plaque distribution in patients with ischemic stroke, and to evaluate the relationship between lipid parameters and carotid plaque formation.

Methods: This prospective cross-sectional study included 30 computed tomography-confirmed ischemic stroke or transient ischemic attack patients aged 14 years or above at the Department of General Medicine, Konaseema Institute of Medical Sciences & Research Foundation, Amalapuram, Andhra Pradesh, India, from April 2025 to March 2026. Hemorrhagic stroke, recurrent stroke, cardioembolic sources, metabolic stroke mimics, and patients receiving lipid-lowering drugs were excluded. Clinical profile, blood pressure, fasting lipid profile, and extracranial carotid Doppler ultrasound findings were analysed.

Results: The mean age was 68.83 +/- 13.57 years, and 63.3% were aged 65 years or above. Females constituted 53.3%. Hypertension and diabetes were recorded in 30.0% and 13.3%, respectively; combined hypertension with diabetes was present in 10.0%. Total cholesterol above 200 mg/dL, LDL-C above 100 mg/dL, triglycerides above 150 mg/dL, and HDL-C below 40 mg/dL were observed in 43.3%, 56.7%, 46.7%, and 36.7%, respectively. Carotid plaques were detected in 46.7%. Plaque-positive patients had higher mean total cholesterol, LDL-C, and triglyceride levels than plaque-negative patients.

Conclusion: Dyslipidemia, particularly elevated LDL-C and triglycerides, showed a meaningful association with carotid plaque positivity in ischemic stroke patients. Carotid Doppler ultrasound combined with lipid assessment can strengthen vascular risk stratification and guide secondary prevention.

Keywords*: Ischemic stroke; lipid profile; carotid artery plaque; carotid Doppler ultrasound; LDL cholesterol; triglycerides; atherosclerosis.*

INTRODUCTION

Stroke is a major neurological emergency and a leading contributor to death and long-term disability worldwide. The updated American Heart Association/American Stroke Association definition recognizes stroke as central nervous system infarction or haemorrhage supported by clinical, neuroimaging, or pathological evidence [2]. Ischemic stroke forms the larger share of the global stroke burden, and recent global estimates show that incident stroke and stroke-related disability remain substantial across low- and middle-income regions [1]. In clinical practice, early identification of treatable vascular mechanisms is essential because recurrent events are common after ischemic stroke and transient ischemic attack [3,4].

Atherosclerosis is one of the central pathways linking traditional risk factors with cerebral ischemia. Hypertension, diabetes mellitus, smoking, dyslipidemia, obesity, and advancing age interact through endothelial dysfunction, inflammation, oxidative stress, and arterial wall lipid deposition. Among these factors, dyslipidemia is important because it is measurable, modifiable, and responsive to drug and lifestyle interventions. Low-density lipoprotein cholesterol (LDL-C) is directly involved in atherogenesis, while low high-density lipoprotein cholesterol (HDL-C) and increased triglycerides reflect a metabolic environment that favours plaque development and vascular events [5,6].

The extracranial carotid arteries are a frequent site of atherosclerotic plaque formation. Carotid intima-media thickness and plaque assessment by B-mode and Doppler ultrasound provide non-invasive information on subclinical and established atherosclerosis [7,8]. Population-based evidence has shown that carotid wall thickness predicts incident clinical stroke, and plaque morphology adds prognostic value beyond stenosis alone [9,10]. Therefore, carotid Doppler ultrasound has practical value in the evaluation of patients with ischemic stroke, especially where magnetic resonance angiography or computed tomography angiography is not readily available.

Previous studies from South Asian and hospital-based settings have reported variable associations between lipid parameters, carotid plaque, and ischemic stroke [12-14]. These variations reflect differences in age distribution, vascular risk profiles, diagnostic methods, lipid thresholds, and sample size. In tertiary care hospital settings, combined evaluation of lipid profile and carotid Doppler findings is especially useful because it can be performed at relatively low cost and can support individualized prevention strategies. Such evidence also helps clinicians separate broad stroke risk from plaque-related atherosclerotic risk during early follow-up.

The present study was conducted with the objective of assessing lipid profile abnormalities and carotid artery plaque distribution among patients with ischemic stroke. The study also aimed to evaluate the relationship between hyperlipidemia and carotid plaque formation, with particular emphasis on the association of total cholesterol, LDL-C, HDL-C, triglycerides, and Doppler-detected extracranial carotid plaques.

METHODOLOGY

Study design and setting: This prospective cross-sectional study was conducted in the Department of General Medicine, Konaseema Institute of Medical Sciences & Research Foundation, Amalapuram, Andhra Pradesh, India, from April 2025 to March 2026 The study was designed to assess clinical profile, lipid profile, and carotid artery Doppler findings among patients presenting with ischemic stroke or transient ischemic attack. The reporting approach followed an observational design, while standard definitions of stroke and transient ischemic attack were aligned with accepted clinical and neuroimaging concepts [2,3].

Study population and eligibility criteria: A total of 30 patients aged 14 years or above with clinical features of acute focal neurological deficit and computed tomography-confirmed cerebral infarction or transient ischemic attack were enrolled. Patients with haemorrhagic stroke, recurrent stroke, clinically or electrocardiographically suspected cardiac embolic source, hypoglycemia, diabetic ketoacidosis, hyperosmolar coma, uraemia, and other stroke mimics were excluded. Patients already receiving lipid-lowering therapy, beta-blockers, or thiazide diuretics were excluded as far as practicable because these drugs can influence lipid parameters or haemodynamic interpretation.

Sampling and data collection: Eligible patients attending the inpatient or outpatient services during the study period were recruited after written informed consent. A predesigned proforma was used to record demographic data, presenting symptoms, comorbidities, blood pressure, pulse rate, neurological findings, and clinical evidence of carotid artery pulsation abnormality. History of hypertension, diabetes mellitus, dyslipidemia, transient ischemic attack, ischemic heart disease, smoking, alcohol use, and drug intake was elicited. Neurological examination included motor power, laterality of weakness, plantar response, cranial nerve assessment, and Glasgow Coma Scale at admission.

Investigations and operational definitions: All patients underwent routine laboratory evaluation, lipid profile estimation, computed tomography of the brain, and extracranial carotid Doppler ultrasound. Lipid parameters included total cholesterol, HDL-C, LDL-C, very-low-density lipoprotein cholesterol (VLDL-C), and triglycerides. Dyslipidemia thresholds were total cholesterol greater than 200 mg/dL, LDL-C greater than 100 mg/dL, HDL-C less than 40 mg/dL, and triglycerides greater than 150 mg/dL. Carotid Doppler ultrasound was used to detect plaque location in the right common carotid artery, left common carotid artery, or bilateral common carotid arteries, consistent with the clinical utility of carotid ultrasound in stroke risk assessment [7-10].

Statistical analysis and ethics: Data were analysed using Epi Info software. Categorical variables were expressed as frequency and percentage, while continuous variables were expressed as mean, standard deviation, median, and range. Chi-square test was applied for categorical associations, and independent t-test was used for comparison of continuous variables between plaque-positive and plaque-negative groups. Odds ratios were calculated with 95% confidence intervals where appropriate. A p-value below 0.05 was considered statistically significant. Permission was obtained from

the Head of Department and Head of Institution, and ethical clearance was obtained from the Institutional Ethics Committee, Konaseema Institute of Medical Sciences & Research Foundation, Amalapuram, Andhra Pradesh, India. Written informed consent was obtained from patients or legally acceptable representatives.

RESULTS

A total of 30 patients with ischemic stroke or transient ischemic attack were analysed. The mean age was 68.83 +/- 13.57 years, with a median of 70.5 years and a range of 35 to 89 years. Patients aged 65 years or above constituted 63.3% of the study population. Females were slightly more common than males, although the difference was not statistically significant. Hypertension was the most frequent comorbidity, and raised systolic blood pressure at admission was common. The baseline demographic and vascular risk profile is presented in Table 1.

Table 1. Baseline demographic and vascular risk profile of the study population

Variable	Category / statistic	Value
Age, years	Mean +/- SD	68.83 +/- 13.57
Age, years	Median (range)	70.5 (35-89)
Age group	>=65 years	19 (63.3%)
Age group	<65 years	11 (36.7%)
Sex	Male	14 (46.7%)
Sex	Female	16 (53.3%)
Comorbidity	Diabetes mellitus	4 (13.3%)
Comorbidity	Hypertension	9 (30.0%)
Comorbidity	Hypertension + diabetes mellitus	3 (10.0%)
Comorbidity	None	14 (46.7%)
Systolic blood pressure	>140 mmHg	19 (63.3%)
Systolic blood pressure	Mean +/- SD	150.87 +/- 23.64 mmHg
Diastolic blood pressure	>90 mmHg	6 (20.0%)
Diastolic blood pressure	Mean +/- SD	84.20 +/- 13.41 mmHg
Pulse rate	>100/minute	9 (30.0%)
Pulse rate	Mean +/- SD	91.87 +/- 15.98/minute

The neurological profile showed right-sided upper and lower limb paralysis in 50.0% and left-sided paralysis in 40.0%, while 10.0% had no limb paralysis at assessment. Extensor plantar response was more frequently recorded on the right side. Clinically diminished carotid artery pulsation was uncommon and was documented in four patients. These findings are summarised in Table 2.

Table 2. Clinical and neurological findings

Clinical variable	Category	n (%)
Laterality of paralysis	Both upper and lower limbs, right	15 (50.0%)
Laterality of paralysis	Both upper and lower limbs, left	12 (40.0%)
Laterality of paralysis	Normal	3 (10.0%)
Motor power of limbs	RE, BL, LT	16 (53.3%)
Motor power of limbs	RE, BL, RT	12 (40.0%)
Motor power of limbs	Normal	2 (6.7%)
Plantar response	E, RT	15 (50.0%)
Plantar response	E, LT	12 (40.0%)
Plantar response	Flexor	3 (10.0%)
Carotid artery pulsation	Normal	26 (86.7%)
Carotid artery pulsation	Diminished, right	3 (10.0%)
Carotid artery pulsation	Diminished, left	1 (3.3%)

Abbreviations in Table 2: E, extensor; LT, left; RT, right; RE, reduced; BL, bilateral.

Lipid profile analysis showed that LDL-C elevation was the most common lipid abnormality. LDL-C above 100 mg/dL was observed in 56.7%, triglycerides above 150 mg/dL in 46.7%, total cholesterol above 200 mg/dL in 43.3%, and HDL-C below 40 mg/dL in 36.7%. The lipid distribution is shown in Table 3.

Table 3. Lipid profile distribution among ischemic stroke patients

Lipid parameter	Category / statistic	Value
Total cholesterol	>200 mg/dL	13 (43.3%)
Total cholesterol	<=200 mg/dL	17 (56.7%)
Total cholesterol	Mean +/- SD	202.90 +/- 58.81 mg/dL
HDL-C	<40 mg/dL	11 (36.7%)
HDL-C	>=40 mg/dL	19 (63.3%)
HDL-C	Mean +/- SD	44.40 +/- 11.26 mg/dL
LDL-C	>100 mg/dL	17 (56.7%)
LDL-C	<=100 mg/dL	13 (43.3%)
LDL-C	Mean +/- SD	122.50 +/- 53.64 mg/dL
VLDL-C	Mean +/- SD	34.40 +/- 16.60 mg/dL
Triglycerides	>150 mg/dL	14 (46.7%)
Triglycerides	<=150 mg/dL	16 (53.3%)
Triglycerides	Mean +/- SD	170.23 +/- 82.93 mg/dL

Carotid Doppler ultrasound detected plaque in 14 patients, giving an overall plaque frequency of 46.7%. Left common carotid artery plaque was the most frequent isolated plaque location, followed by bilateral common carotid artery plaque and right common carotid artery plaque. No plaque was detected in 53.3% of the patients. The Doppler findings are given in Table 4.

Table 4. Carotid artery Doppler ultrasound findings

Doppler finding	n (%)
Bilateral common carotid artery plaque	5 (16.7%)
Left common carotid artery plaque	6 (20.0%)
Right common carotid artery plaque	3 (10.0%)
No plaque	16 (53.3%)
Any carotid plaque	14 (46.7%)
Total	30 (100.0%)

Clinical factors were analysed according to the presence or absence of carotid plaque. Plaque was more frequent among patients aged above 65 years, males, and those with comorbidity, but these associations did not reach statistical significance. The clinical association analysis is shown in Table 5.

Table 5. Association of selected clinical variables with carotid artery plaque

Variable	Plaque present n (%)	Plaque absent n (%)	Total n (%)	Odds ratio (95% CI)	p-value
Age >65 years	10 (52.6%)	9 (47.4%)	19 (100.0%)	1.94 (0.42-8.39)	0.38
Age <=65 years	4 (36.4%)	7 (63.6%)	11 (100.0%)	Reference	-
Male	9 (64.3%)	5 (35.7%)	14 (100.0%)	3.96 (0.86-18.11)	0.07
Female	5 (31.3%)	11 (68.8%)	16 (100.0%)	Reference	-
Comorbidity present	9 (56.3%)	7 (43.8%)	16 (100.0%)	2.31 (0.53-10.09)	0.26
Comorbidity absent	5 (35.7%)	9 (64.3%)	14 (100.0%)	Reference	-

Categorical lipid analysis demonstrated a strong association between LDL-C above 100 mg/dL and carotid plaque. Triglycerides above 150 mg/dL were also associated with a higher frequency of plaque positivity. HDL-C below 40 mg/dL showed a higher odds value but was not statistically significant in this cohort. These findings are presented in Table 6.

Table 6. Association of lipid categories with carotid artery plaque

Lipid category	Plaque present n (%)	Plaque absent n (%)	Total n (%)	Odds ratio	p-value
LDL-C >100 mg/dL	14 (82.4%)	3 (17.6%)	17 (100.0%)	Not estimable*	<0.001
LDL-C <=100 mg/dL	0 (0.0%)	13 (100.0%)	13 (100.0%)	Reference	-
Triglycerides >150 mg/dL	10 (71.4%)	4 (28.6%)	14 (100.0%)	7.50	0.026
Triglycerides <=150 mg/dL	4 (25.0%)	12 (75.0%)	16 (100.0%)	Reference	-
HDL-C <40 mg/dL	6 (54.5%)	5 (45.5%)	11 (100.0%)	1.65	0.707
HDL-C >=40 mg/dL	8 (42.1%)	11 (57.9%)	19 (100.0%)	Reference	-

*Odds ratio was not estimable because no plaque-positive patient had LDL-C <=100 mg/dL.
Mean values of different clinical and biochemical parameters were compared between plaque-positive and plaque-negative patients. Total cholesterol, LDL-C, and triglycerides were significantly higher in patients with carotid plaque. Age, systolic blood pressure, diastolic blood pressure, pulse rate, HDL-C, and VLDL-C were not significantly different between the two groups. The comparative analysis is provided in Table 7.

Table 7. Comparison of continuous variables between patients with and without carotid artery plaque

Parameter	With plaque Mean +/- SD	Without plaque Mean +/- SD	t value	p-value
Age, years	70.57 +/- 15.639	67.31 +/- 11.780	0.637	0.530
DBP, mmHg	83.71 +/- 12.350	84.63 +/- 14.674	0.185	0.855
SBP, mmHg	154.43 +/- 24.384	147.75 +/- 23.311	0.764	0.452
Pulse rate/minute	92.71 +/- 17.813	91.13 +/- 14.751	0.264	0.794
Total cholesterol, mg/dL	240.71 +/- 56.746	169.81 +/- 37.330	3.982	0.001
HDL-C, mg/dL	42.57 +/- 10.286	46.00 +/- 12.144	0.837	0.410
LDL-C, mg/dL	162.00 +/- 50.859	87.94 +/- 24.288	4.975	<0.001
VLDL-C, mg/dL	36.93 +/- 14.840	32.19 +/- 18.181	0.786	0.439
Triglycerides, mg/dL	201.86 +/- 90.550	142.56 +/- 66.627	2.118	0.044

DISCUSSION

This cross-sectional study examined the lipid profile and carotid Doppler findings of 30 patients with ischemic stroke. The study population was predominantly elderly, with a mean age of 68.83 years and nearly two-thirds aged 65 years or above. This finding is biologically plausible because vascular aging, arterial stiffness, cumulative exposure to hypertension, and progressive atherosclerosis increase ischemic stroke susceptibility. The observed female predominance was modest and statistically non-significant; therefore, it should be interpreted cautiously because sex distribution in small hospital samples is strongly influenced by referral patterns and survival to hospital presentation.

Hypertension remained the most frequent recorded comorbidity, followed by diabetes mellitus. Raised systolic blood pressure at admission was common, reflecting either pre-existing hypertension or the acute haemodynamic response to cerebral ischemia. Current secondary prevention guidelines emphasize aggressive control of blood pressure, diabetes, lipids, smoking, and other modifiable factors after ischemic stroke or transient ischemic attack [4]. In this setting, the present findings support the need for systematic vascular risk screening even in tertiary care hospitals.

The lipid profile showed a considerable burden of dyslipidemia. LDL-C above 100 mg/dL was present in 56.7%, triglycerides above 150 mg/dL in 46.7%, and total cholesterol above 200 mg/dL in 43.3%. Plaque-positive patients had substantially higher mean total cholesterol, LDL-C, and triglyceride levels than patients without plaque. The difference was strongest for LDL-C, consistent with the established role of LDL-C in atherosclerotic plaque initiation and progression. Evidence from statin trials and meta-analyses confirms that lipid-lowering treatment reduces recurrent stroke and other vascular events, particularly in non-cardioembolic ischemic stroke [5,6].

Carotid Doppler ultrasound detected plaques in 46.7% of patients, with left common carotid artery plaques being slightly more frequent than right-sided or bilateral plaques. Hospital-based studies from South Asia have similarly reported carotid stenosis or plaque in a clinically important proportion of ischemic stroke patients [13,14]. The finding is also supported by longitudinal data showing that carotid wall thickness and plaque features predict future cerebrovascular events [8-11]. Thus, carotid Doppler can serve as a practical bedside extension of vascular assessment, especially when advanced vascular imaging is limited.

The association analysis suggested that age, sex, and comorbidity were not statistically significant predictors of carotid plaque in this small cohort, although plaque odds were numerically higher among older, male, and comorbid patients. In contrast, LDL-C categories and triglyceride categories showed clearer plaque association, and mean comparisons confirmed higher total cholesterol, LDL-C, and triglycerides in plaque-positive patients. These findings indicate that lipid profile assessment should not be viewed as a routine laboratory formality; it provides actionable information for secondary prevention, carotid atherosclerosis identification, and risk communication in ischemic stroke care.

Limitations

The study had a small sample size and was conducted at a single center study, which limits statistical power and external validity. Lipid levels recorded after acute stroke can decline temporarily and influence classification. Doppler ultrasound interpretation is operator-dependent, and plaque morphology or stenosis severity was not analysed in depth. Long-term outcomes, recurrent vascular events, medication adherence, and plaque progression were not assessed.

CONCLUSION

In this cross-sectional study of ischemic stroke patients, dyslipidemia and carotid atherosclerotic plaques were frequent findings. LDL-C elevation was the most common lipid abnormality, while higher mean total cholesterol, LDL-C, and triglyceride levels were observed among patients with Doppler-detected carotid plaques. Carotid Doppler ultrasound provided useful, non-invasive evidence of extracranial carotid disease and helped identify patients requiring intensified vascular risk reduction. The findings support routine lipid profile estimation and carotid Doppler evaluation in suitable ischemic stroke patients. Early detection of dyslipidemia and carotid plaque can improve secondary prevention planning, especially in resource-limited hospital settings. Larger multicentric studies with longitudinal follow-up are required to confirm the strength of these associations and define locally relevant screening pathways.

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