e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(7); 1528-1537

Original Research Article

Association of Fasting Lipid Profile with Stroke Severity in Patients with Acute Ischemic Stroke: A Hospital-Based Observational Study

Shashikanth Patil[1], Sneha Bharathi S. Angadi[2], Lakkisetty Mounica[3]

[1](MD General Medicine), Senior resident General Medicine, Rajiv Gandhi Super specialty Hospital (OPEC), (A unit of Rims, Raichur), Raichur, Karnataka, India.

[2](MD Dermatology), Assistant Professor, Raichur Institute of Medical Sciences, Raichur, Karnataka, India

[3]Assistant Professor, Department of Respiratory Medicine, Navodaya Medical College and Research Centre, Raichur, Karnataka, India

Received: 01-04-2026 / Revised: 15-05-2026 / Accepted: 21-06-2026

Corresponding author: Dr. Shashikanth Patil

Conflict of interest: Nil

Abstract

Background: Dyslipidemia is a well-known vascular risk factor, but the relationship between single lipid fractions and the severity of acute ischemic stroke is complex. Total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) may relate to neurological impairment in different ways.

Objective: To study the association of fasting lipid profile parameters with neurological severity in patients with acute ischemic stroke.

Methods: This hospital-based prospective observational study included 100 adults with computed tomography confirmed acute cerebral infarction admitted to the Department of General Medicine, Navodaya Medical College Hospital and Research Center, Raichur from 1st January 2021 to 31st July 2022. Total cholesterol, triglycerides, HDL-C, and LDL-C were measured in the fasting state during hospitalization. Neurological severity was assessed by the National Institutes of Health Stroke Scale (NIHSS). Associations between lipid categories and NIHSS were evaluated using appropriate t-tests or analysis of variance. Statistical significance was set at $p<0.05$.

Results: The mean age of 100 participants was 64.57 ± 10.06 years, and 60% were males. Total cholesterol was >200 mg/dL in 53% of the study population, triglycerides >150 mg/dL in 51%, HDL-C <40 mg/dL in 67%, and LDL-C >130 mg/dL in 33%. The mean total cholesterol, triglycerides, HDL-C and LDL-C were 218.16 ± 57.87, 159.35 ± 60.24, 38.33 ± 16.84 and 140.45 ± 54.16 mg/dL respectively. Mean NIHSS in patients with total cholesterol >200 mg/dL was significantly higher than in those with <200 mg/dL (18.72 vs 11.85; $p<0.00001$). Mean NIHSS increased from 12.41 (<150 mg/dL) to 19.40 (>200 mg/dL) across triglyceride categories ($p=0.00030$). Mean NIHSS was higher in participants with HDL-C <40 mg/dL than in those with HDL-C 40–60 mg/dL or >60 mg/dL (17.76 vs 10.69 and 11.30; $p=0.00011$). Mean NIHSS also went up across LDL-C categories from 10.93 for <100 mg/dL to 19.30 for >130 mg/dL ($p=0.00009$).

Conclusion: Abnormalities in fasting lipid parameters were common in patients with acute ischemic stroke and significantly associated with neurological severity in this study. Higher NIHSS scores were associated with higher total cholesterol, triglycerides and LDL-C, and lower HDL-C These findings support the assessment of the lipid profile as part of the cardiovascular risk assessment of patients with acute ischemic stroke, but the observational design does not establish causality or independent prognostic significance.

Keywords: Acute Ischemic Stroke; Dyslipidemia; Total Cholesterol; Triglycerides; HDL Cholesterol; LDL Cholesterol; NIHSS; Stroke Severity.

DOI: 10.25258/ijcpr.18.7.231

Introduction

Stroke is a leading cause of death and long-term disability worldwide. Ischemic stroke comprises the majority of incident strokes. The global burden of ischemic stroke remains high, especially in low- and middle-income countries with rising vascular risk factors. [1] Dyslipidemia is a well-known modifiable risk factor of cardiovascular disease. Elevated atherogenic lipoproteins, particularly LDL-C, play a role in the development and progression of atherosclerotic plaque and are

consequently important targets for primary and secondary vascular prevention. However, the relationship between circulating lipids and the clinical severity of an established acute ischemic stroke is less clear-cut than their relationship with long-term vascular risk. [2,3] Different biological relations of acute cerebral ischemia are possible with different lipid fractions LDL-C and total cholesterol are contributors to atherosclerotic disease, whereas HDL-C has been historically thought to be cardioprotective. Triglycerides are closely associated with metabolic dysfunction and may interact with other vascular risk factors. Therefore, observational studies reported heterogeneous associations between individual lipid fractions, stroke severity and outcome. [4-6]

The association between lipid concentrations and NIHSS was investigated in several studies. Some groups have reported an association between low HDL-C and increased initial stroke severity, but other studies have not shown a reproducible association. [7,8] Similarly, some studies have reported associations of total cholesterol or LDL-C with poor outcomes, while other studies have reported apparently paradoxical associations of lower lipid concentrations with more severe disease. [5,9]

This apparent 'lipid paradox' may in part be a consequence of reverse causation, acute-phase changes in lipid metabolism, nutritional status, stroke subtype, pre-existing lipid-lowering therapy and differences in timing of blood sampling. [9,10] Thus, lipid measurements obtained acutely during stroke should be interpreted with caution.

The management of a stroke today includes the evaluation and treatment of vascular risk factors such as dyslipidemia as part of secondary prevention. The 2026 American Heart Association/American Stroke Association guideline builds on the current evidence for management of acute ischemic stroke and early secondary prevention. Newer dyslipidemia guidance remains focused on lowering atherogenic lipoprotein burden in selected patients. [11,12] It is important not to automatically interpret the association between a lipid parameter and acute stroke severity as proof that changing that lipid concentration in the acute phase will improve neurological recovery. Hospital-based studies are needed to investigate the relationship between readily available lipid fractions and neurological severity, especially in populations with dyslipidemia and other vascular risk factors.

Objective: To determine the association between fasting total cholesterol, triglycerides, HDL-C and LDL-C and neurological severity, as measured by NIHSS, among patients with acute ischemic stroke.

Materials and Methods

Study design and setting: This was a hospital-based prospective observational study conducted in the Department of General Medicine, Navodaya Medical College Hospital and Research Centre, Raichur, Karnataka.

The study was a hospital-based prospective cross-sectional observational study.

Study Period: The study was conducted from January 1, 2021 to July 31, 2022.

Sample Size: The calculated minimum sample size was 66. This was based on a prevalence estimate of 21.9%, a 95% confidence level and an absolute allowable error of 10 percentage points. The final study included 100 participants.

The present manuscript therefore reports the actual final study population of 100, rather than artificially reducing the sample to the calculated minimum.

Sampling: Eligible patients were recruited until the planned sample size was attained during the study period, as described in the source thesis data-collection procedure.

Eligibility Criteria

Inclusion Criteria: Participants were eligible if they:

- were aged >18 years; and
- Had CT evidence of acute cerebral infarction.

Exclusion Criteria: Patients with the following were excluded:

- age <18 years;
- traumatic stroke;
- transient ischemic attack;
- cerebral venous thrombosis;
- intracerebral hemorrhage;
- space-occupying lesion;
- cerebellar or brainstem infarction;
- cardioembolic stroke; and
- Coagulation disorders.

Clinical and laboratory Assessment: Demographic and clinical information was obtained after enrollment. The clinical assessment included history of hypertension, diabetes mellitus, smoking and alcohol consumption.

Laboratory evaluation included:

- complete blood count;
- random blood glucose;
- fasting blood glucose;
- HbA1c;
- fasting lipid profile; and
- Serum creatinine.

CT brain imaging was performed to confirm acute cerebral infarction.

Lipid Profile Assessment: The fasting lipid profile included:

- total cholesterol;
- triglycerides;
- HDL-C; and
- LDL-C.

For analysis, lipid values were categorized according to the thresholds used in the original thesis:

Total cholesterol

- <200 mg/dL
- 200 mg/dL

Triglycerides

- <150 mg/dL
- 150–200 mg/dL
- 200 mg/dL

HDL-C

- <40 mg/dL
- 40–60 mg/dL
- 60 mg/dL

LDL-C

- <100 mg/dL
- 100–130 mg/dL
- 130 mg/dL.

Assessment of stroke severity: Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) at admission. NIHSS is a standardized measure of neurological impairment in acute stroke, with higher scores indicating greater neurological deficit. [13] For this manuscript, the primary outcome was NIHSS at admission, because the principal research question concerns the relationship between lipid status and initial neurological severity.

Statistical Analysis: Continuous variables were summarized as mean ± standard deviation and categorical variables as frequency and percentage.

Associations between lipid categories and NIHSS were evaluated using independent-sample t-tests for two-category variables and analysis of variance for variables with three categories, consistent with the statistical analyses reported in the thesis. A p value <0.05 was considered statistically significant. The original thesis reports use of Epi Info for statistical analysis. No multivariable regression analysis was performed for this manuscript because such analysis was not reported in the source thesis. Consequently, the findings are presented as unadjusted associations.

Ethical Approval: Institutional Ethics Committee approval was obtained from Navodaya Medical College Hospital and Research Centre, Raichur, on December 22, 2020. Written informed consent was obtained before enrollment.

Results

Demographic Characteristics: A total of 100 patients were included. The mean age was 64.57 ± 10.06 years, and 60% were male. Most participants were aged >50 years.

Table 1: Demographic characteristics

Characteristic	n (%)
Total participants	100 (100)
Male	60 (60)
Female	40 (40)
Age <40 years	1 (1)
Age 41–50 years	6 (6)
Age 51–60 years	24 (24)
Age 61–70 years	39 (39)
Age 71–80 years	20 (20)
Age >81 years	10 (10)
Mean age, years	**64.57 ± 10.06**

Fasting Lipid Profile: Total cholesterol was >200 mg/dL in 53% of participants. Triglycerides were ≥150 mg/dL in 51%, while 67% had HDL-C <40 mg/dL. LDL-C was >130 mg/dL in 33% and 100–130 mg/dL in 39%. The mean total cholesterol was 218.16 ± 57.87 mg/dL, mean triglycerides 159.35 ± 60.24 mg/dL, mean HDL-C 38.33 ± 16.84 mg/dL, and mean LDL-C 140.45 ± 54.16 mg/dL.

Table 2: Distribution of fasting lipid profile

Lipid parameter	Category	n (%)
Total cholesterol	<200 mg/dL	47 (47)
	>200 mg/dL	53 (53)
	Mean ± SD	**218.16 ± 57.87 mg/dL**
Triglycerides	<150 mg/dL	49 (49)
	150–200 mg/dL	31 (31)
	>200 mg/dL	20 (20)
	Mean ± SD	**159.35 ± 60.24 mg/dL**
HDL-C	<40 mg/dL	67 (67)
	40–60 mg/dL	23 (23)
	>60 mg/dL	10 (10)
	Mean ± SD	**38.33 ± 16.84 mg/dL**
LDL-C	<100 mg/dL	28 (28)
	100–130 mg/dL	39 (39)
	>130 mg/dL	33 (33)
	Mean ± SD	**140.45 ± 54.16 mg/dL**

Figure 1. Distribution of fasting lipid abnormalities

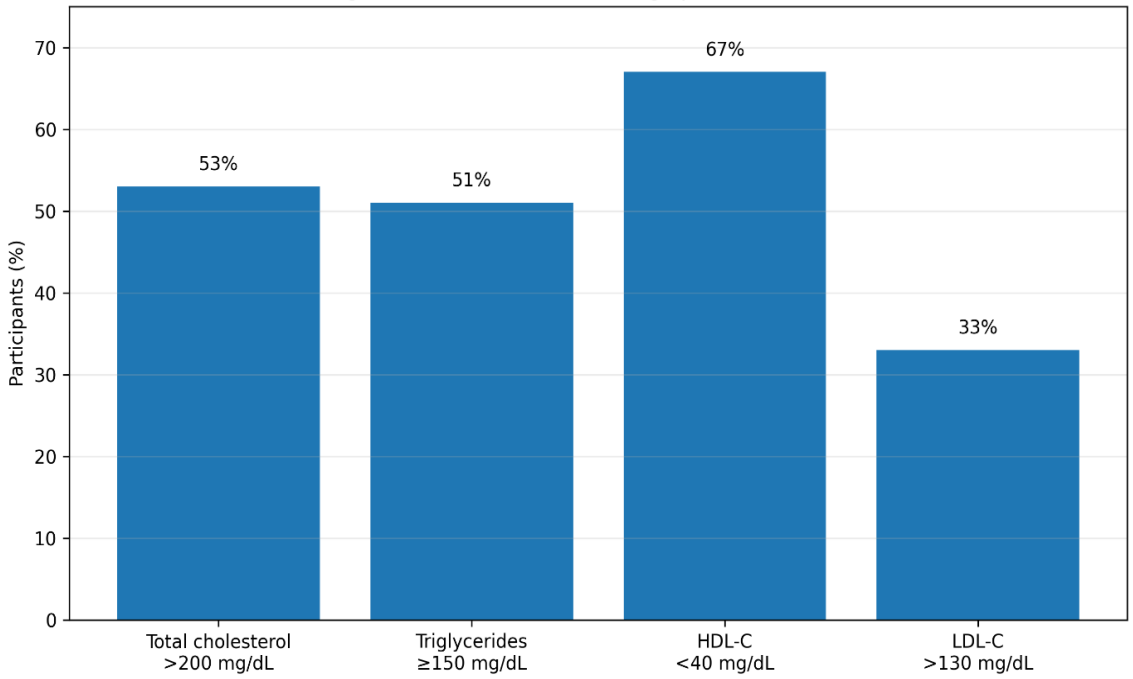

Figure 1: Distribution of fasting lipid abnormalities.

A grouped bar chart demonstrating the proportion of participants with elevated total cholesterol, elevated triglycerides, low HDL-C and elevated LDL-C.

Association between Total Cholesterol and NIHSS: Participants with total cholesterol >200 mg/dL had a substantially higher mean NIHSS than those with total cholesterol <200 mg/dL:

- <200 mg/dL: 11.85 ± 1.07
- 200 mg/dL: 18.72 ± 0.95

The difference was statistically significant ($p<0.00001$).

Table 3: Association between total cholesterol and NIHSS

Total cholesterol	n	Mean NIHSS	SD
<200 mg/dL	47	11.85	1.07
>200 mg/dL	53	18.72	0.95
p value			**<0.00001**

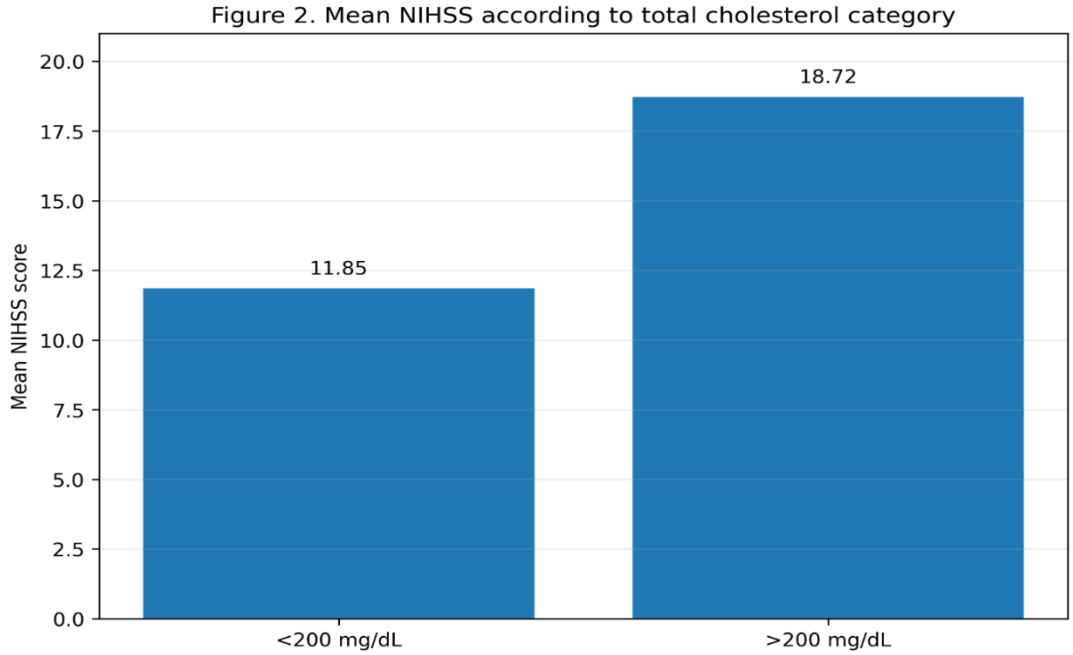

Figure 2: Mean NIHSS according to total cholesterol category.

Association between Triglycerides and NIHSS: Mean NIHSS increased progressively with increasing triglyceride category:

- <150 mg/dL: 12.41 ± 7.31
- 150–200 mg/dL: 17.84 ± 8.24
- 200 mg/dL: 19.40 ± 5.51

The difference between groups was statistically significant (F=8.8166, p=0.00030).

Table 4: Association between triglycerides and NIHSS

Triglycerides	n	Mean NIHSS	SD
<150 mg/dL	49	12.41	7.31
150–200 mg/dL	31	17.84	8.24
>200 mg/dL	20	19.40	5.51
p value			**0.00030**

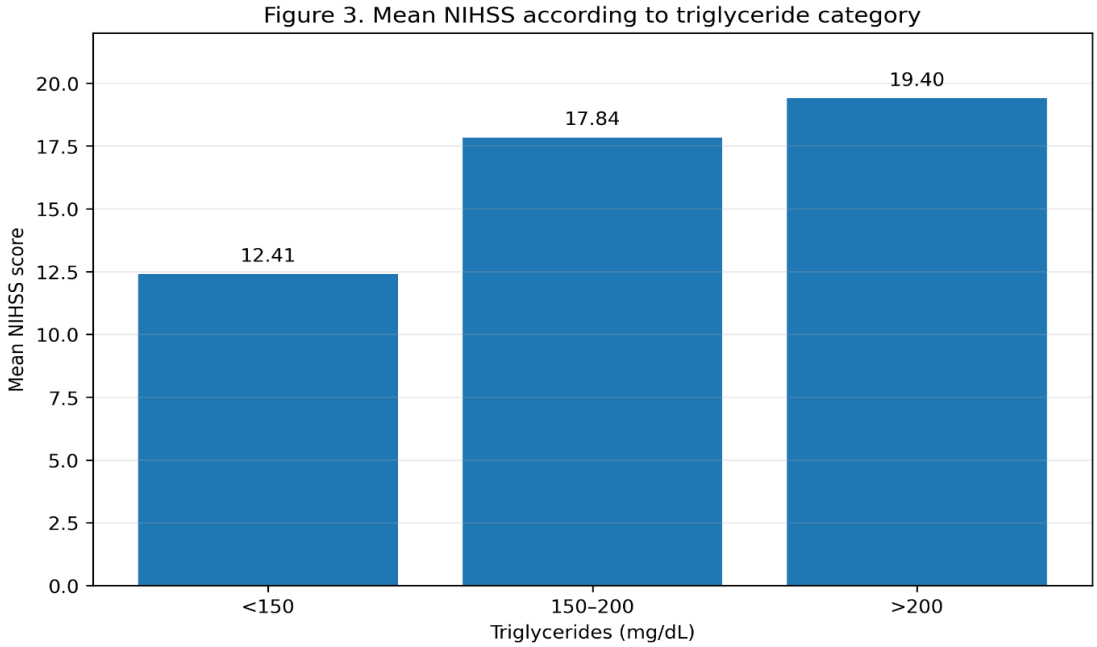

Figure 3: Mean NIHSS according to triglyceride category.

Association between HDL-C and NIHSS: Participants with HDL-C <40 mg/dL had a mean NIHSS of 17.76 ± 8.21, compared with 10.69 ± 4.61 among those with HDL-C 40–60 mg/dL and 11.30 ± 4.27 among those with HDL-C >60 mg/dL. The difference was statistically significant (F=10.0327, p=0.00011).

Table 5: Association between HDL-C and NIHSS

HDL-C	n	Mean NIHSS	SD
<40 mg/dL	67	17.76	8.21
40–60 mg/dL	23	10.69	4.61
>60 mg/dL	10	11.30	4.27
p value			**0.00011**

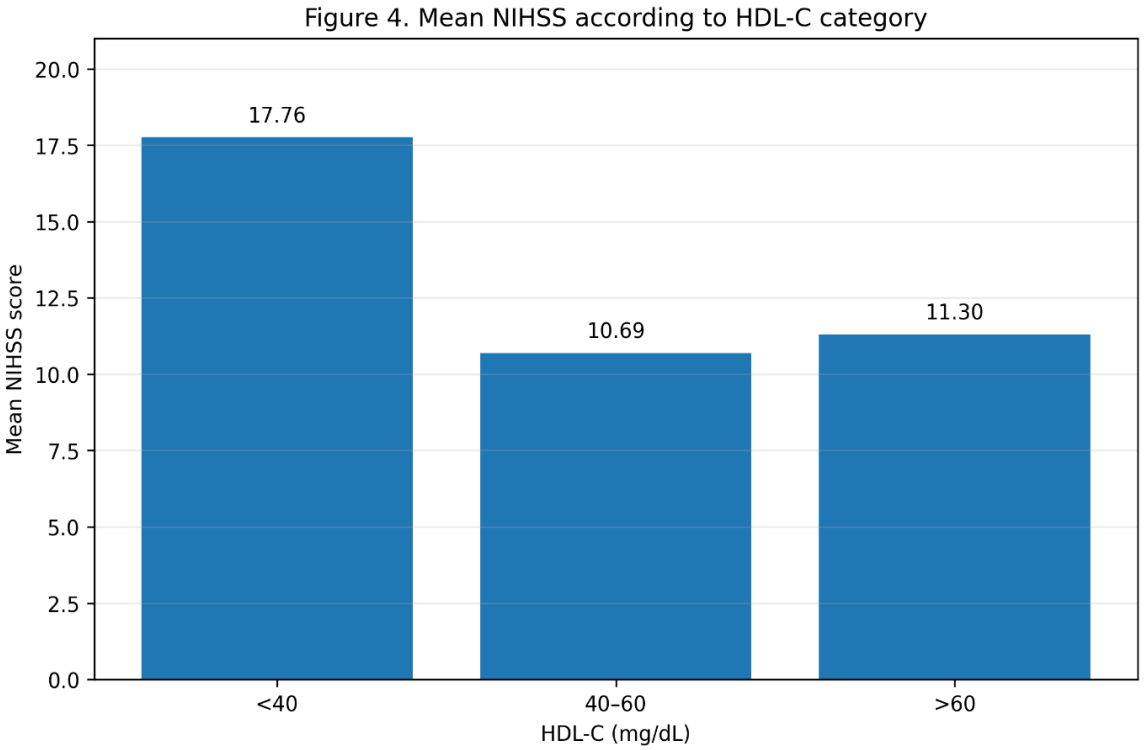

Figure 4: Mean NIHSS according to HDL-C category.

Association between LDL-C and NIHSS:

Mean NIHSS increased across LDL-C categories:

- <100 mg/dL: 10.93 ± 6.42
- 100–130 mg/dL: 15.54 ± 8.10
- 130 mg/dL: 19.30 ± 6.74

The association was statistically significant (F=10.1787, p=0.00009).

Table 6: Association between LDL-C and NIHSS

LDL-C	n	Mean NIHSS	SD
<100 mg/dL	28	10.93	6.42
100–130 mg/dL	39	15.54	8.10
>130 mg/dL	33	19.30	6.74
p value			**0.00009**

Figure 5. Mean NIHSS according to LDL-C category

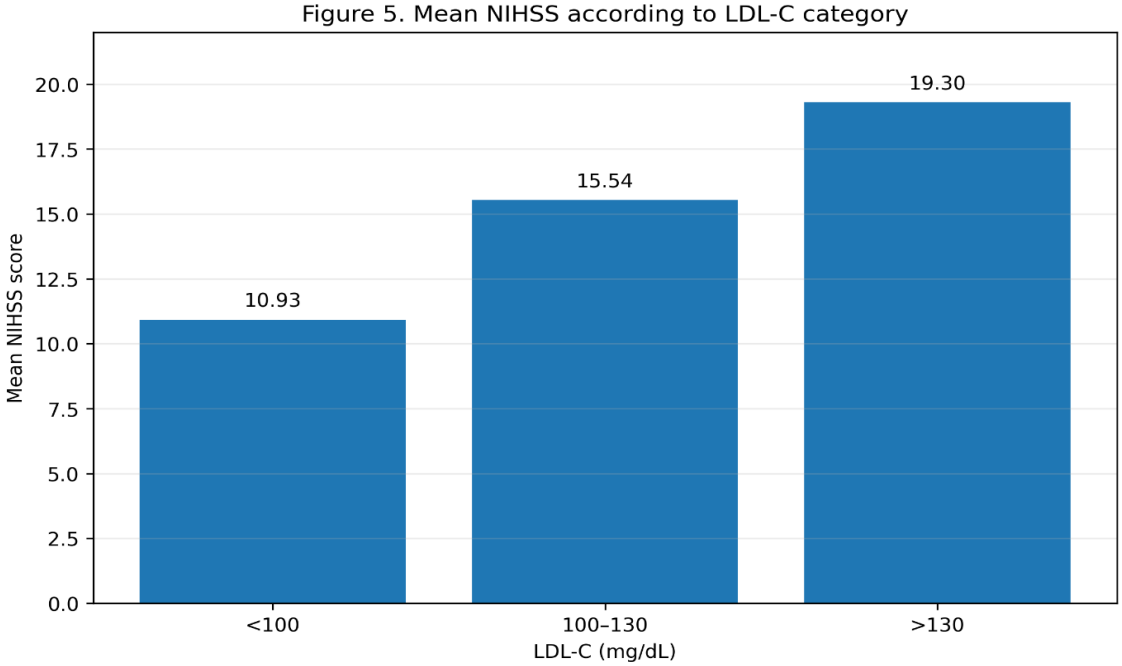

Figure 5: Mean NIHSS according to LDL-C category.

Overall Findings: Across all four lipid parameters, the thesis analysis demonstrated statistically significant differences in mean NIHSS between lipid categories.

Table 7: Summary of lipid parameters and NIHSS

Lipid parameter	Lowest category: mean NIHSS	Highest category: mean NIHSS	Overall p value
Total cholesterol	11.85	18.72	<0.00001
Triglycerides	12.41	19.40	0.00030
HDL-C*	17.76 (<40 mg/dL)	11.30 (>60 mg/dL)	0.00011
LDL-C	10.93	19.30	0.00009

***For HDL-C, the direction of association was inverse: the lowest HDL-C category had the highest mean NIHSS.**

Figure 6. Comparative mean NIHSS across lipid categories

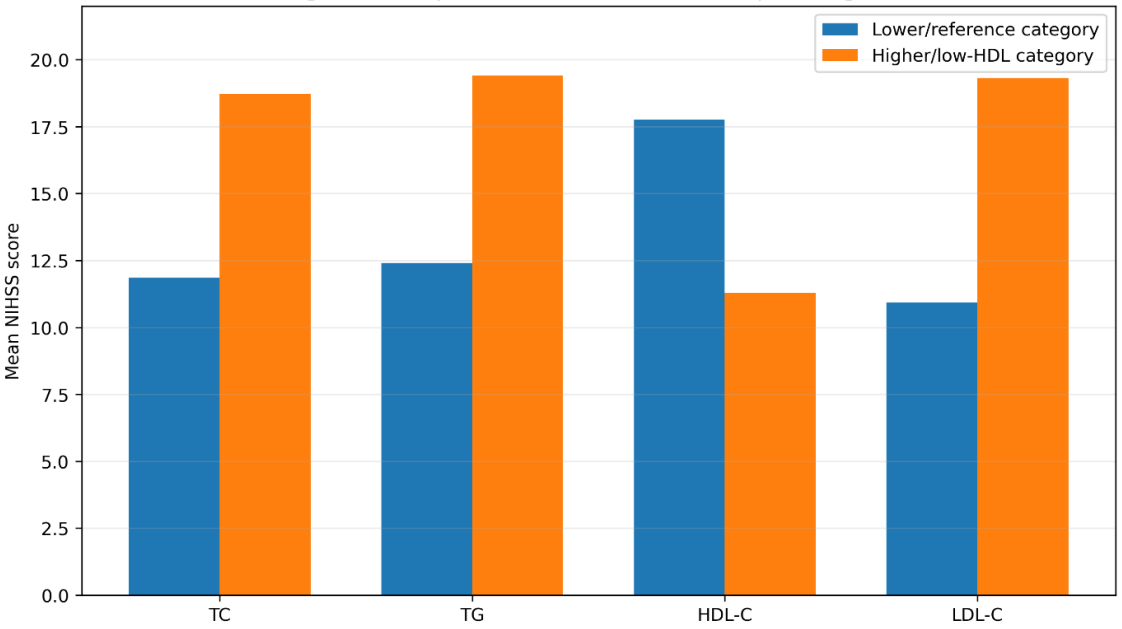

Figure 6: Comparative mean NIHSS across lipid categories.

A multi-panel figure showing the relationship of total cholesterol, triglycerides, HDL-C and LDL-C categories with mean NIHSS.

Discussion

The main finding of the study was that all four conventional fasting lipid parameters evaluated, total cholesterol, triglycerides, HDL-C and LDL-C, and were significantly associated with NIHSS in patients with acute ischemic stroke. Higher mean NIHSS was associated with higher total cholesterol, triglyceride and LDL-C categories, while the lowest HDL-C category was associated with significantly higher neurological severity.

More than half of the participants had total cholesterol >200 mg/dL and the mean total cholesterol was 218.16 mg/dL. Participants with total cholesterol >200 mg/dL had a mean NIHSS of 18.72 compared to 11.85 among those with lower total cholesterol. This finding is in line with observational studies reporting associations between dyslipidemia and adverse neurological outcomes. A study on dyslipidemia in acute ischemic stroke found that serum total cholesterol, LDL-C and HDL-C were associated with outcomes defined by NIHSS >10 at discharge or death. [5] The present cohort had a similar pattern for LDL-C. Mean NIHSS increased from 10.93 in subjects with LDL-C <100mg/dL to 19.30 in those with LDL-C >130 mg/dL. Biologic plausibility for this association is provided by the known role of LDL-containing lipoproteins in atherosclerosis and large artery disease in the cerebrovascular circulation. However, circulating LDL-C measured at the time of an acute vascular event may not necessarily reflect the patient's long-term lipid exposure. Measured lipid concentrations may be influenced by an acute illness, previous statin therapy and changes in nutritional or metabolic status. [2,3]

The relationship between triglycerides and NIHSS was also statistically significant. For participants with triglycerides <150 mg/dL mean NIHSS was 12.41 which increased to 19.40 for those with triglycerides >200 mg/dL. This finding is noteworthy since previous studies have shown inconsistent associations between triglycerides and stroke severity. In one cohort, lower triglyceride levels relative to higher levels were independently associated with more severe ischemic stroke and higher in-hospital mortality, demonstrating the heterogeneous nature of lipid-outcome relationships. [6] Therefore, the present finding should be interpreted as an association in this particular cohort, rather than as evidence that elevated triglycerides directly caused greater neurological injury. The results for HDL-C were directionally consistent with several studies showing an inverse relationship between HDL-C and stroke severity. In this cohort, 67% of participants had HDL-C <40 mg/dL and this group had a mean NIHSS of 17.76 compared with 10.69 in participants with HDL-C 40–60 mg/dL. Low HDL-C was independently associated with increased stroke severity and adverse clinical outcomes in a large cohort of patients with atherosclerotic ischemic stroke. [7] However, the literature is not consistent. In a previous study of 241 patients with ischemic stroke no overall association between low HDL-C and NIHSS was found, but an association was found in younger patients. [8] These differences indicate that HDL-C concentration may not completely represent HDL function or its association with cerebral ischemia.

Indeed, emerging evidence suggests that HDL function may be more informative than HDL concentration. In a prospective study of patients with acute ischemic stroke, measures of HDL cholesterol efflux capacity and HDL composition were correlated with stroke severity and outcome, suggesting that the biological function of HDL may not be completely reflected in its circulating concentration. [10]

The so-called lipid paradox is a major problem in the interpretation of lipid levels during acute stroke. Some of the observational studies have shown that lower concentrations of some lipid fractions are associated with more severe stroke or hemorrhagic transformation. A systematic review found evidence for associations between lipid profile and hemorrhagic transformation, but heterogeneity between studies was large. [9] Similarly, studies of acute ischemic stroke have reported seemingly paradoxical associations between lipid levels and outcomes. [6,9]

Several mechanisms can be proposed for these seemingly conflicting findings. First, acute systemic illness can change lipid metabolism. Secondly, severe stroke may be associated with inadequate nutritional intake and systemic inflammatory responses. Third, the patients with known dyslipidemia may be on statin therapy. Fourth, different mechanisms of stroke, such as large-artery atherosclerosis, small-vessel disease, and cardioembolism, may have different relationships with lipid concentrations.

Therefore, the clinical implications should be carefully worded. Dyslipidemia is an important modifiable vascular risk factor and management of lipids is an important component of secondary stroke prevention. Current cardiovascular guidance recommends reduction of atherogenic lipoprotein burden according to individual cardiovascular risk. Modern stroke guidance includes early secondary prevention within acute stroke care. (The American Heart Association/American Stroke Association guideline) [11,12] However, this study does not demonstrate that lowering cholesterol or

triglycerides during acute hospitalization will decrease NIHSS or improve neurological recovery. The majority of the evidence on statin therapy in acute ischemic stroke has been extrapolated from observational studies, and systematic reviews have highlighted the shortcomings of such evidence. [13,14] Thus, the lipid associations in this study should primarily be interpreted as markers of vascular/metabolic phenotype rather than as evidence for acute lipid-targeted treatment based on NIHSS alone.

Nevertheless, the results of the study are of clinical utility as fasting lipid profile is cheap, routinely available and relevant to long-term secondary prevention. Detection of dyslipidemia during stroke hospitalization provides an opportunity for assessment of cardiovascular risk and for ensuring adequate long-term lipid management.

Strengths and Limitations

Strengths

1. The study evaluated all four conventional fasting lipid fractions.
2. The cohort consisted exclusively of patients with CT-confirmed acute cerebral infarction.
3. NIHSS was used as a standardized measure of neurological severity.
4. The analysis examined lipid parameters individually rather than combining them into a nonspecific dyslipidemia category.
5. The study provides hospital-based data from an Indian clinical setting.

Limitations

1. This was a single-center study with a relatively small sample of 100 patients.
2. The observational design prevents determination of causality.
3. The analysis was unadjusted; therefore, residual confounding by age, diabetes, hypertension, smoking, stroke mechanism and other vascular risk factors cannot be excluded.
4. The study did not provide multivariable regression analyses to establish independent associations.
5. The timing of lipid measurement relative to symptom onset was not sufficiently detailed in the available thesis material.
6. Pre-stroke statin or other lipid-lowering therapy was not incorporated into the analysis.
7. Stroke subtypes based on a standardized etiological classification were not analyzed separately.
8. Long-term outcomes such as 90-day modified Rankin Scale were not available.
9. HDL functionality, apolipoproteins, lipoprotein (a), non-HDL cholesterol and small dense LDL were not measured.
10. The study contains an inconsistency in the description of sampling methodology; this was not artificially reconciled.
11. Because the study excluded cardioembolic stroke and posterior fossa infarctions, the findings may not be generalizable to all patients with acute ischemic stroke.

Conclusions

In this hospital-based cohort of 100 patients with acute ischemic stroke, abnormalities of fasting lipid profile were common. More than half had total cholesterol or triglycerides >200 mg/dL, two-thirds had HDL-C <40 mg/dL, and one-third had LDL-C >130 mg/dL.

Higher categories of total cholesterol, triglycerides and LDL-C were also significantly associated with higher NIHSS scores. Low HDL-C was associated with greater neurological severity. These findings imply that typical lipid parameters could provide useful information on the metabolic and vascular profile of patients presenting with acute ischemic stroke.

However, the results are unadjusted associations and should not be interpreted as evidence that lipid abnormalities independently determine stroke severity or that acute lipid lowering will improve neurological recovery. Larger prospective multicenter studies that include stroke mechanism, pre-stroke lipid lowering therapy, serial lipid measurements, and long-term functional outcomes are needed to clarify these relationships.

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