Section: Radiodiagnosis

Original Research Article

A STUDY ON THE CORRELATION OF CAROTID INTIMA MEDIA THICKNESS WITH FASTING LIPID PROFILE AND STROKE SEVERITY IN ACUTE ISCHEMIC STROKE PATIENTS

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ABSTRACT

Background: Ischemic stroke represents approximately 87% of all strokes and is a primary cause of global mortality and chronic disability. Atherosclerosis is the main contributor to this condition, driven heavily by classic risk factors like dyslipidemia, diabetes mellitus, and hypertension. Carotid intima-media thickness (CIMT) acts as a validated, non-invasive surrogate marker for detecting subclinical atherosclerosis. This study evaluates the relationship between fasting lipid profiles, glycated haemoglobin (HbA1c), CIMT, and stroke severity in patients with acute ischemic stroke.

Materials and Methods: A descriptive cross-sectional observational study was conducted over 18 months, enrolling 50 adult patients admitted with confirmed acute ischemic stroke at GITAM Institute of Medical Sciences and Research Hospital. Stroke severity was evaluated utilizing the National Institutes of Health Stroke Scale (NIHSS) at admission. Fasting venous blood samples were analyzed for comprehensive lipid panels and HbA1c levels, while CIMT was bilaterally measured using high-resolution B-mode ultrasonography. Data were evaluated using statistical correlation analyses to assess metabolic and vascular relationships.

Results: The study cohort was predominantly male (64%), with the largest age demographic being individuals over 65 years (38%). Poor long-term glycemic control, defined as HbA1c ≥6.5%, was present in 56% of participants and was strongly linked to severe strokes (p=0.0001). Significant atherosclerotic changes (CIMT >1.5 mm) were identified in 34% of patients and positively correlated with both elevated HbA1c (r=0.38, p=0.012) and increased stroke severity (r=0.45, p=0.02). Lipid analysis revealed that low-density lipoprotein (LDL-C) and very-low-density lipoprotein (VLDL-C) had strong positive correlations with CIMT. Furthermore, total cholesterol, LDL-C, VLDL-C, and triglycerides showed statistically significant positive correlations with overall stroke severity, with triglycerides demonstrating the strongest association (r=0.45, p=0.048). Conversely, high-density lipoprotein cholesterol (HDL-C) showed a significant negative correlation with stroke severity (r = -0.27, p = 0.038).

Conclusion: Elevated atherogenic lipids and poor glycemic control are critically linked to increased carotid arterial thickening and more severe neurological deficits during acute ischemic strokes. Incorporating routine metabolic profiling and CIMT measurements provides valuable clinical insights into underlying vascular pathology, ultimately improving early risk stratification and the management of cerebrovascular disease.

Keywords: Carotid Intima Media Thickness, Lipid Profile, Dyslipidaemia, Acute Ischemic Stroke.

INTRODUCTION

Stroke remains one of the foremost causes of death and chronic disability worldwide. Ischemic stroke (IS) accounts for most cases, representing around 87% of all strokes. According to the Study on Global Burden of Disease in 2019, stroke.[1] is currently the second leading cause of death internationally, posing a major challenge for public health, mainly in economically developing countries like India.[2] In the Indian context, the impact of stroke has risen significantly in recent decades. Epidemiological studies report an incidence ranging from 105 to 152 cases for every 100,000 individuals/year. This increasing trend is particularly notable among populations with established cardiovascular risk factors, like diabetes mellitus, abnormal lipid profile, and hypertension. Among the multiple factors implicated in developing ischemic stroke, atherosclerosis plays the role of a main contributor. Atherosclerosis involves the progressive thickening and hardening of arterial walls, leading to luminal narrowing and, ultimately, compromised blood flow to critical brain regions. It is driven by a combination of classic risk factors like dyslipidaemia, smoking, hypertension, diabetes mellitus, and long-term inflammation.[3] Carotid intima-media thickness, commonly measured using ultrasonography (non-invasive B-mode), has emerged as a validated surrogate marker of subclinical atherosclerosis. An increased CIMT reflects early vascular changes that precede overt clinical events like myocardial infarction or stroke. Studies have consistently demonstrated a strong association between elevated CIMT and the probability of future cardiovascular and cerebrovascular disorders.[4] Large-scale international studies have emphasized the decisive contribution of dyslipidemia in the emergence of ischemic stroke. Increased levels of low-density lipoproteins and reduced levels of HDL cholesterol were found to separately increase the probability of ischemic stroke occurrence. These findings highlight the significance of lipid abnormalities not only in coronary artery disease but also in cerebrovascular pathology.[5-6] While triglycerides themselves may not be direct causal factors, their role in the overall lipid milieu and their relationship with components of metabolic syndrome position them as important contributors to cerebrovascular risk. Maintaining triglyceride levels within the normal range is therefore considered beneficial as part of a comprehensive strategy for stroke prevention. Stroke continues to be an important cause of death as well as disability in India, with metabolic risk factors playing a central role. While fasting lipid profile and HbA1c are routinely measured in clinical practice, their precise relationship with stroke severity and markers of subclinical atherosclerosis like CIMT remains inadequately explored in the Indian context. Understanding these associations could enhance risk stratification, guide preventive strategies, and improve clinical outcomes for stroke patients. The present study aimed to evaluate the relationship between fasting lipid profile and HbA1c with stroke severity and thickness of carotid intima-media (CIMT) among individuals experiencing acute ischemic stroke.

MATERIALS AND METHODS

Study Design: Descriptive (Cross-sectional) observational study

Study Duration: 18 months (August 2023 to February 2025)

Study Setting: GITAM Institute of Medical Sciences and Research Hospital.

Study Subjects: Patients admitted with acute ischemic stroke at GIMSR hospital

Sample Size and Patient Selection Criteria: A total sample of 50 patients with acute ischemic stroke was included.

Sample Size Justification: The sample size was determined to detect a moderate correlation among lipid profile parameters, HbA1c, stroke severity, and carotid intima-media thickness (CIMT), with adequate statistical power. For correlation-based studies, the formula used for the calculation of sample size is:

$n = (Z\alpha/2 + Z\beta)^2 / r^2 + 3$ Where: $Z\alpha/2 = 1.96$ (for a confidence interval of 95%); $Z\beta = 0.84$ (for 80% power)

r = assumed correlation coefficient (moderate effect size, $r \approx 0.4$). Substituting values: $n = (1.96 + 0.84)^2 / (0.4)^2 + 3 \approx 50$. Hence, a sample of approximately 50 patients was considered sufficient for statistical analysis.

Inclusion Criteria:

- Adults aged ≥18 years, irrespective of their gender
- Patients with confirmed acute ischemic stroke admitted at the study center
- Participant giving written informed consent

Exclusion Criteria:

- History of prior stroke or significant neurological disorders
- Current or recent use of lipid-lowering agents
- Inability or unwillingness to provide consent
- Pregnant or lactating women
- Known atherosclerotic cardiovascular conditions (CAD, CVD, PVD)
- Patients diagnosed with venous stroke
- Central nervous system tumours mimicking stroke
- Intracranial haemorrhages (extradural, subdural, subarachnoid)
- Presence of malignancies or other severe systemic illness

Methodology: Eligible participants were enrolled after obtaining informed consent. Demographic and clinical history was obtained through a structured interview, covering age, gender, lifestyle habits (smoking, alcohol), comorbidities (hypertension,

diabetes mellitus, dyslipidemia), and familial history of cardiovascular disease or stroke. A detailed clinical examination was performed, including assessment of vital signs and neurological function. The severity of Stroke was evaluated at admission by a certified neurologist using the National Institutes of Health Stroke Scale (NIHSS). Diagnosis of acute ischemic stroke was confirmed using cranial CT and/or MRI. Neuroimaging was reviewed by experienced radiologists to exclude haemorrhagic stroke, venous infarction, and intracranial mass lesions. Following confirmation, venous blood samples were drawn after a minimum 10-hour overnight fast. Samples were processed for lipid panel analysis—total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol-using an automatic biochemical analyser. HbA1c was measured using high-performance liquid chromatography (HPLC) with a G8 HPLC Analyzer® (Tosoh Bioscience Inc., USA) in accordance with NGSP standards. CIMT measurement was performed using high-resolution ultrasonography (B- mode) with a 7 MHz linear transducer (Esaote MyLab™ Class C®, Italy) by a trained radiologist. CIMT was assessed bilaterally at the common carotid artery (CCA), bifurcation, and the internal carotid artery (ICA). An average maximum CIMT >0.8 mm was considered indicative of increased vascular risk. Atherosclerotic plaques were defined as focal protrusions >1.5 mm. Standardized quality control procedures were implemented throughout the study. CIMT readings were interpreted using blinded duplicate evaluations to decrease both inter- and intra-observer variability.

Statistical analysis: Data was analyzed using appropriate statistical tools. Tests such as ANOVA, Chi-square, and correlation analysis (Pearson or Spearman) were employed to assess relationships among metabolic parameters (lipid profile, HbA1c), NIHSS scores, and CIMT measurements. Results were shown as mean and standard deviation (SD), with significance set at 5% ($p < 0.05$) where applicable.

RESULTS

Table 1: Distribution of age among the study Participants

Age Group	Count (n)	Percentage (%)
<45	3	6%
45-55	12	24%
55-65	16	32%
>65	19	38%

The age group with the highest representation in the study was those over 65 years, comprising 38% of the total sample. This was followed by individuals aged 55–65 years (32%) and those aged 45–55 years (24%). Only 6% of participants were younger than 45 years. These findings highlight an age-related increase in stroke occurrence, consistent with known epidemiological patterns [Figure 1].

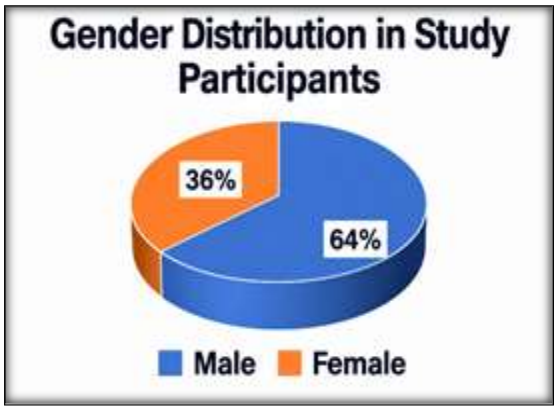


Figure 1: Gender Distribution Among Study Participants

Among the 50 patients, males accounted for 64% and females for 36%, indicating a male predominance. This aligns with broader population data showing higher stroke prevalence in males, possibly due to both behavioural and biological factors.

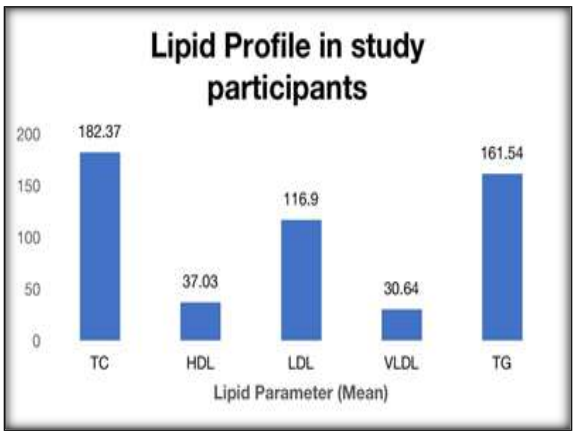


Figure 2: Lipid Profile of Study Participants

The mean total cholesterol (TC) was 182.37 mg/dL, with LDL-C averaging 116.90 mg/dL. HDL-C levels were low, averaging 37.03 mg/dL, which is below the threshold for cardiovascular protection. Mean triglyceride (TG) and VLDL levels were 161.54 mg/dL and 30.64 mg/dL, respectively, indicating dyslipidemia across the cohort [Figure 2].

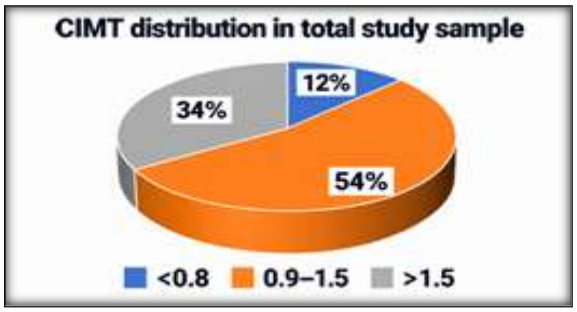


Figure 3: CIMT Distribution within the Entire Study Sample

Most participants (54%) had CIMT values between 0.9 and 1.5 mm. A further 34% had CIMT >1.5 mm, indicating advanced atherosclerosis, while only 12% had CIMT <0.8 mm. CIMT findings reflect a significant burden of subclinical vascular disease among the study population.

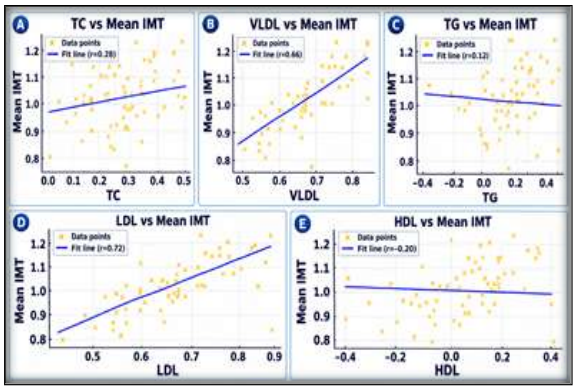


Figure 4: Scattered plots showing the association between Lipid Profile and CIMT

The scatter plot for total cholesterol (TC) versus mean IMT showed a weak positive correlation (r = 0.28). However, the relationship was not statistically significant (p = 0.15). Triglycerides (TG) showed a very weak positive correlation with mean IMT (r = 0.12), which was not statistically significant (p = 0.56). LDL cholesterol showed a strong positive correlation with mean IMT (r = 0.72), which was statistically significant (p = 0.021). The scatter plot of VLDL versus mean IMT showed a strong positive correlation (r = 0.66) that was statistically significant (p = 0.008). A weak negative correlation was observed between HDL cholesterol and mean IMT (r = –0.20), but this association was statistically insignificant (p = 0.32) [Figure 4].

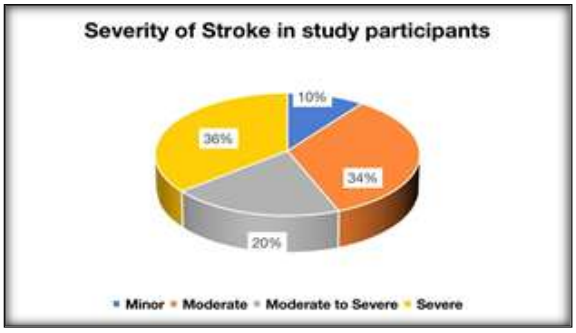


Figure 5: Stroke Severity Among Study Participants

Severity analysis showed that 36% of patients experienced severe strokes (NIHSS 21–42), while 34% had moderate severity (NIHSS 5–15), and 20% fell into the moderate to severe category (NIHSS 16–20). Only 10% had minor strokes (NIHSS 0–4) [Figure 5].

Table 2: Association between HbA1c with Stroke Severity

HbA1C Category	Minor Stroke (0-4)	Moderate Stroke (5-15)	Moderate To Severe (16-20)	Severe Stroke (21-42)	Total (%)
<6.5	5 (22.7%)	14 (63.6%)	3 (13.6%)	0 (0.0%)	22 (44%)
≥6.5	0 (0.0%)	3 (10.7%)	7 (25.0%)	18 (64.3%)	28 (56%)
Total	5 (10%)	17 (34%)	10 (20%)	18 (36%)	50 (100%)
χ^2 = 19.58, df = 3, p = 0.0001) P=0.0001					

Patients with HbA1c ≥6.5% were more likely to experience severe strokes, with 18 out of 28 falling in the severe category. In contrast, those with HbA1c <6.5% were mostly in the minor- or moderate-severity groups. The association was statistically significant (χ^2 = 19.58, p = 0.0001) (Table 2).

Table 3: Correlation between HbA1c and CIMT

HbA1C Category	CIMT			Total n (%)
	<0.8 mm	0.9-1.5 mm	>1.51 mm	
<6.5	4 (18.2%)	13 (59.1%)	5 (22.7%)	22 (100%)
≥6.5	2 (7.1%)	14 (50.0%)	12 (42.9%)	28 (100%)
Total	6 (12.0%)	27 (54.0%)	17 (34.0%)	50 (100%)
χ^2 =5.207; p-value: 0.074				

A higher percentage of patients with HbA1c ≥6.5% had CIMT >1.5 mm, suggesting a relationship between poor glycemic control and increased vascular thickening. Although the association did not reach conventional levels of statistical significance (p = 0.074), the trend was clear [Table 3].

Triglycerides exhibited the strongest positive correlation among all lipid parameters with stroke severity (r = 0.45, p = 0.004). LDL cholesterol showed a moderate positive correlation (r = 0.35) with stroke severity, which was statistically significant (p = 0.018). The scatter plot shows a moderate positive correlation (r = 0.32) between total

cholesterol (TC) and stroke severity. This indicates that higher TC levels are associated with increased severity of stroke presentations. The association is statistically significant (p = 0.015). VLDL was found to have a moderately strong positive correlation (r = 0.42) with stroke severity (p = 0.008), indicating that higher VLDL levels may be linked to greater neurological deficits. HDL cholesterol showed a negative correlation with stroke severity (r = –0.27), which was statistically significant (p = 0.038) [Figure 6].

Table 4: Carotid Intima-Media Thickness (CIMT) Correlated with Stroke Severity

CIMT Category (mm)	Stroke severity				Total
	Minor	Moderate	Moderate to severe	Severe	
<0.8	4 (66.7%)	2 (33.3%)	0 (0%)	0 (0%)	6 (12%)
0.9-1.5	1 (3.7%)	7 (25.9%)	7 (25.9%)	12(44.4%)	27 (54%)
>1.5	0 (0%)	8 (47.1%)	3 (17.6%)	6 (35.3%)	17 (34%)
Total	5 (10%)	17 (34%)	10 (20%)	18 (36%)	50 (100%)
Chi square: 27.8; p-value: 0.0001*					

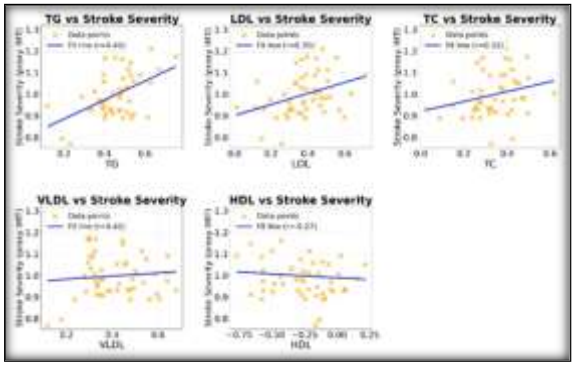


Figure 6: Scattered Plots Showing the Association between Lipid Profile and Severity of Stroke

Lower CIMT values (<0.8 mm) were seen in patients with minor strokes, while higher CIMT values (>1.5 mm) were associated with more severe strokes. This pattern indicates a strong relationship between arterial thickening and neurological impairment (χ^2 = 27.8; p = 0.0001) [Table 4].

Severity and HbA1c Versus Mean CIMT

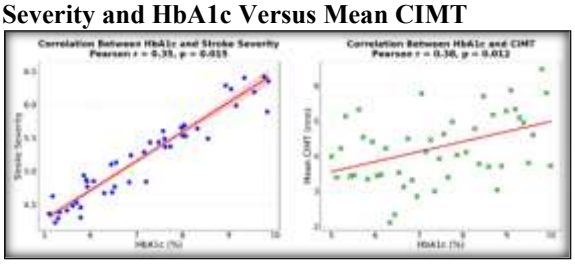


Figure 7: Scattered Plots Showing Relationship between HbA1c Versus Stroke Severity and HbA1c Versus Mean CIMT

The relationship between HbA1c and mean CIMT exhibited a weak to moderate positive correlation (Pearson r = 0.38, p = 0.012), suggesting that elevated HbA1c levels correlate with increased carotid intima-media thickness, reflecting greater arterial thickening (CIMT) and worsening vascular health. HbA1c vs Stroke severity – Moderate positive correlation (r = 0.35, p = 0.015), suggesting that higher HbA1c levels, reflecting chronic hyperglycaemia, are associated with more severe cerebrovascular presentations [Figure 7].

DISCUSSION

The onset of the acute type of ischemic stroke initiates a cascade of cellular and molecular disturbances that ultimately result in neuronal injury and death. A key early event in this process is the disturbance of intracellular calcium homeostasis. When cerebral blood flow is compromised due to vascular occlusion, the oxygen and glucose supply to neurons is abruptly halted. This leads to energy failure, as ATP synthesis—heavily reliant on oxidative phosphorylation in mitochondria - comes to a halt.[7] In the absence of sufficient ATP, ion pumps like the Na^+/K^+-ATPase and Ca^{2+}-ATPase fail to function efficiently. This ionic imbalance permits calcium ions to accumulate excessively within neurons, particularly in mitochondria and cytosol. The sustained calcium overload is not only cytotoxic but also activates several downstream destructive pathways. Lipids play a very important role in maintaining the structure and function of the central nervous system (CNS). Impaired lipid metabolism has been involved in several neurological conditions, including neurodegenerative diseases like Alzheimer's, Parkinson's, Huntington's disease, and various psychiatric disorders like bipolar disorder and schizophrenia.[8] Altered lipid metabolism is also thought to contribute significantly to CNS injuries, including those caused by traumatic brain injury, injury to the spinal cord, and stroke. Several studies suggest that disruptions in lipid pathways can increase the vulnerability of neural tissues to oxidative stress, inflammation, and excitotoxic damage. Understanding the role of lipids in CNS physiology and pathology offers important therapeutic insights, especially for managing conditions such as ischemic stroke, where lipid metabolism is closely linked to disease progression and recovery.[9] Atherosclerosis is a major contributor to ischemic stroke and is primarily an inflammatory disease affecting medium- and large-sized arteries. In the early stages, low-density lipoprotein particles infiltrate the endothelial lining and accumulate within the arterial intima. Plaque rupture is a crucial phenomenon leading to thrombus

formation. Exposure of thrombogenic material, including tissue factor from apoptotic macrophages, activates the coagulation cascade, resulting in clot formation. If the thrombus occludes a cerebral artery, it can precipitate an acute ischemic stroke. Alternatively, thrombus fragments may embolize distally, causing cerebral embolism.[10] The National Institutes of Health (NIH) Stroke Scale is a standard tool for assessing stroke severity. It evaluates neurological functions such as motor strength, sensory loss, language ability, level of consciousness, visual fields, facial symmetry, and coordination. The NIHSS is quick to administer, widely validated, and can be reliably used by a range of healthcare professionals. A baseline NIHSS score below 6 is generally associated with better outcomes, while scores above 16 suggest a higher likelihood of severe disability or death. Importantly, the NIHSS also helps distinguish true strokes from stroke mimics.[11] Lipid abnormalities have been closely associated with both ischemic and haemorrhagic strokes, though their relationships differ depending on the stroke subtype. Most observational studies have demonstrated that elevated low- density lipoproteins and total cholesterol levels are significant risk factors for ischemic stroke. In addition to total cholesterol and LDL-C, high-density lipoproteins demonstrated a protective effect against stroke. Several studies have reported that increased HDL-C levels are inversely related to the risk of ischemic stroke. A pooled analysis of 10 prospective cohort studies indicated that for every 10 mg/dL increase in HDL cholesterol, the risk of ischemic stroke decreased by approximately 11%. The link between triglyceride levels and risk of stroke was more inconsistent compared to cholesterol parameters. Thickness of the Carotid intima-media (CIMT) is a minimally invasive marker widely used to detect subclinical atherosclerosis. It reflects the combined thickness of the intimal and medial layers of the carotid artery wall and can be measured reliably using high-resolution B-mode ultrasonography.[12] Numerous studies have demonstrated that elevated CIMT is linked with an increased risk of future cardiovascular and cerebrovascular events, including ischemic stroke. One meta-analysis of 15 cohort investigations indicated that for every 0.1 mm increase in CIM thickness, there was an estimated 13–18% rise in the occurrence of myocardial infarction and stroke. The present study aimed to investigate the associations between these metabolic parameters and stroke severity, assessed using the National Institutes of Health Stroke Scale (NIHSS), as well as carotid intima-media thickness (CIMT), a known surrogate marker of subclinical atherosclerosis. By investigating these relationships, we sought to determine whether commonly available clinical tests could provide meaningful insight into the extent of underlying vascular pathology and the severity of neurological impairment. Many of the stroke patients in our cohort were aged above 55 years, affirming age as a well-documented non-modifiable risk factor for the occurrence of ischemic stroke. We also observed a male predominance among stroke cases. This observation is consistent with previous research, which suggests that men are generally at higher risk for stroke. Among all risk factors, hypertension emerged as the most prevalent in our study population, followed by diabetes mellitus and dyslipidemia. This finding echoes the results of the INTERSTROKE study.[13] which identified elevated blood pressure as the most impactful modifiable contributor to stroke risk across diverse populations. Dyslipidemia was also frequently observed, with elevated LDL-C and low HDL-C being the most common abnormalities. These lipid changes are known to enhance atherogenesis and promote plaque formation in large arteries, increasing the likelihood of ischemic events.[14-15] Our analysis showed a positive correlation between total cholesterol (TC) and NIH Stroke Scale (NIHSS) scores, suggesting that higher TC levels are linked with more severe strokes.[16-17] Similarly, elevated LDL-C levels were significantly associated with greater neurological deficit, likely due to their role in atheroma formation and arterial narrowing. In contrast, HDL-C levels demonstrated a negative correlation with stroke severity. Higher HDL-C is protective due to its anti-inflammatory, antioxidant, and endothelium-stabilising functions.[18-20] These effects may mitigate the extent of ischemic injury and improve clinical outcomes. Triglycerides (TG) and very-low-density lipoproteins also demonstrated a mild to moderate positive correlation with stroke severity. While their impact is less pronounced than that of LDL-C, elevated TG and VLDL contribute to the formation of small, dense LDL particles, which are more atherogenic and thrombogenic.[21-22] Thickness of Carotid intima-media (CIMT) was observed to be increased in patients with severe strokes. As a surrogate marker of subclinical atherosclerosis, CIMT provides insight into early vascular changes that precede clinical events.[23-24] Our findings revealed that higher CIMT values were associated with elevated lipid levels and HbA1c, reinforcing the interconnected nature of metabolic risk factors and vascular health. Additionally, CIMT was higher in patients with traditional risk factors like hypertension and diabetes, aligning with previous studies.[25-26] The non-invasive nature and predictive value of CIMT make it a valuable tool for early detection of individuals at heightened risk for cerebrovascular events. Its incorporation into routine screening protocols could improve risk stratification, particularly in asymptomatic individuals.[27-30]

CONCLUSION

The findings of this study validate CIMT as a reliable, non-invasive, and accessible tool for early risk stratification and outcome prediction in stroke patients. Timely identification of individuals with subclinical vascular changes can facilitate prompt

intervention and targeted management strategies. Additionally, the results emphasize the crucial role of metabolic disturbances such as dyslipidemia and hyperglycemia in promoting vascular pathology, which subsequently impacts clinical outcomes following stroke. Addressing these metabolic risk factors through preventive and therapeutic approaches remains essential for improving stroke prognosis.

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