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Prevalence of Low HDL and High LDL Levels in Type II Diabetic Patients with Ischemic MCA Stroke

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ABSTRACT

Background: Type II diabetes mellitus is strongly associated with cerebrovascular disease, particularly ischemic stroke in the territory of the middle cerebral artery. Disorders of blood lipids are a frequent comorbidity in diabetic patients, with elevated low-density lipoprotein cholesterol and reduced high-density lipoprotein cholesterol playing central roles in endothelial damage, atherosclerosis, and plaque formation. **Objective:** To determine the frequency of high low density lipoproteins and low high density lipoproteins in stroke patients. **Study Design:** Descriptive cross-sectional study. **Duration and Place of Study:** This study was carried out from February to May 2025 in the Department of Medicine, Shaikh Zayed Federal Postgraduate Medical Institute, Lahore. **Methodology:** A total of 103 patients aged between 30 and 70 years with previously diagnosed type II diabetes mellitus and clinically suspected stroke were recruited through non-probability consecutive sampling. Diagnosis of ischemic stroke was confirmed on neuroimaging. Venous blood samples were obtained for assessment of lipid profile. Reduced high-density lipoprotein cholesterol was defined as values below 40 milligrams per deciliter in males and below 50 milligrams per deciliter in females. Elevated low-density lipoprotein cholesterol was defined as values above 130 milligrams per deciliter. **Results:** The mean age was 53.08±12.15 years, with males comprising 69.9% of participants. High LDL was observed in 59.2% of patients, while 29.1% demonstrated low HDL levels. No significant associations were found between lipid abnormalities and age, gender, BMI, hypertension, or family history of dyslipidemia. **Conclusion:** Elevated LDL and reduced HDL are common among type II diabetic patients with ischemic MCA stroke.

INTRODUCTION

Type II diabetes mellitus is a metabolic disease with a long course, manifested by the presence of persistent hyperglycemia because of decreased secretion of insulin, insulin resistance, or a combination of both mechanisms.[1] The disease is a strong pathogenic factor of cerebrovascular infarction, particularly ischemic stroke in the area of the middle cerebral artery (MCA), due to damage of endothelial cells of blood vessels, an acceleration of atherosclerosis of arteries, and an increase in thrombogenesis.[2] Besides having a high incidence of cerebral infarction, diabetes mellitus patients also have more severe neurological damage and unfavorable clinical results than non-diabetic patients.[3] For these reasons, the comorbidity of diabetes mellitus and ischemic infarction of the MCA is a critical clinical problem, and clarification of dyslipidemia and attention to treatment of the latter are required.[4]

Among the lipid abnormalities commonly seen in type II diabetic patients, reduced high-density lipoprotein (HDL) cholesterol is of great concern.[5] HDL, or anti-atherogenic cholesterol as it is otherwise referred to, has a critical role to play in reverse cholesterol transport, endothelial repair of blood vessels, and negative regulation of inflammation.[6] Insulin resistance and sustained hyperglycemia in the diabetic individual damage the biosynthesis and maturation of HDL particles, reducing their number as well as their functional capacity.[7] This deficiency of HDL results in an augmented accelerated atherogenesis, inflammation of blood vessels, and inadequate endothelial repair.[8] Consequently, low HDL cholesterol becomes an independent risk factor in diabetic patients who are predisposed to ischemic infarction, and its frequent presence in stroke patients with MCA attests to the fact that it is critical to target the defect of HDL in cerebrovascular prevention.[9]

Conversely, high low-density lipoprotein (LDL) cholesterol has been closely correlated with the development of ischemic cerebrovascular disease, particularly in patients with diabetes mellitus.[10] LDL, or pro-atherogenic cholesterol, deposited in the intima of arteries gathers lipid, causes endothelial damage, and accelerates oxidative stress and inflammatory cascades, thereby raising cerebrovascular atherosclerosis.[11] Glycation of LDL particles in type II diabetes mellitus also contributes to their atherogenic potential, producing active plaque growth and vascular stenosis.[12] High LDL cholesterol is thus closely associated with large-artery atherosclerotic stroke, particularly in the MCA territory, wherein occlusion of an artery could produce gross neurological disability.[11] High prevalence of high LDL cholesterol among diabetic patients presenting with ischemic infarct of the MCA draws attention to the importance of rigid lipid management with sugar control in the reduction of cerebrovascular disease risk.

In one local study, 67% of participants demonstrated abnormal total cholesterol levels, with 53.13% exhibiting elevated low-density lipoprotein (LDL) cholesterol, 25.0% showing abnormal triglyceride concentrations, and 21.88% having reduced high-density lipoprotein (HDL) cholesterol.[13] In another regional study, the overall prevalence of dyslipidemia was reported at 92.3%, with hypercholesterolemia, hypertriglyceridemia, elevated LDL, and reduced HDL present in 47.8%, 50.8%, 33.8%, and 93.8% of cases, respectively.[14] Furthermore, in a study conducted on 79 patients with ischemic stroke, 42 individuals (53.16%; 95% Confidence Interval: 42.09–64.23) were found to have dyslipidemia. Among these, elevated total cholesterol was observed in 21 patients (50%), hypertriglyceridemia in 22 patients (52.38%), elevated LDL in 14 patients (33.33%), and reduced HDL in 20 patients (47.61%).[15]

The rationale behind conducting this study in Lahore lies in the increasing incidence of type II diabetes and its vascular complications there, with trends in lifestyle, dietary intake, and limited accessibility to early preventive care causing an enhanced prevalence of dyslipidemia and stroke. Although there has been an increasing number of ischemic MCA strokes among diabetic patients, there has been scant location-specific data providing evidence of the prevalence of low HDL and high LDL levels among diabetic stroke patients. As a significant urban center with a heterogeneous patient population, Lahore provides an ideal location from which to obtain locally applicable evidence capable of directing clinicians on the prevalence of lipid abnormalities among diabetic patients with stroke while informing development of targeted prevention as well as management strategies suited to the area community.

METHODOLOGY

This descriptive cross-sectional study was conducted in the Department of Medicine at Shaikh Zayed Federal Postgraduate Medical Institute, Lahore, over a four-month period from February to May 2025. Ethical clearance was obtained from the institutional review committee as well as from the College of Physicians and Surgeons Pakistan before the commencement of the study. The sample size was calculated using the WHO sample size calculator for a single proportion, with a 95% confidence level, 8% margin of error, and an anticipated frequency of low high-density lipoprotein cholesterol (HDL-C) in stroke patients of 21.88%.[13] A total of 103 patients were recruited through non-probability consecutive sampling. Patients of either sex between 30 and 70 years of age were included if they presented with clinical features of stroke lasting more than 24 hours, such as sudden weakness or numbness of a limb, slurred speech, headache, or visual disturbance. The diagnosis was confirmed by neuroimaging. On non-contrast CT scan, ischemic stroke was characterized by findings including hypodensity of basal ganglia or insular cortex, loss of gray-white matter differentiation, or a hyperdense arterial clot. Hemorrhagic stroke was confirmed by areas of hyper attenuation consistent with acute intracerebral bleeding. Exclusion criteria comprised pregnancy, malignant disease, psychiatric disorders, ongoing lipid-lowering therapy, and hypertensive emergency, defined as diastolic blood pressure exceeding 120 mmHg with acute target organ injury.

Written informed consent was obtained from all eligible participants. Demographic information was documented. Anthropometric measurements were recorded; weight was measured on a digital weighing scale, and height was measured on a wall-mounted stadiometer. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared, with values greater than 30 kg/m^2 categorized as obese. Medical history included diabetes mellitus, defined as fasting plasma glucose ≥126 mg/dl or random plasma glucose ≥200 mg/dl on two separate occasions, and hypertension, defined as systolic pressure ≥140 mmHg or diastolic pressure ≥90 mmHg at admission. For laboratory assessment, 3 ml of venous blood was drawn under aseptic conditions and analyzed in the institutional pathology laboratory. HDL-C levels below 40 mg/dl in males and below 50 mg/dl in females were regarded as low, whereas low-density lipoprotein cholesterol (LDL-C) concentrations greater than 130 mg/dl were considered high. Each patient's stroke subtype and lipid profile results were recorded on a structured proforma.

Data were analyzed using SPSS version 25. Continuous variables were tested for normality with the Shapiro–Wilk test, and results were expressed as mean ± standard deviation or median with interquartile range as appropriate. Categorical variables were described as frequencies and percentages. Stratification was carried out to control for potential effect modifiers, and associations between lipid abnormalities and categorical variables were assessed using the Chi-square test. A p-value of ≤0.05 was considered statistically significant.

RESULTS

The patient population had a mean age of 53.08±12.15 years, with males comprising 69.9% (n=72) and females 30.1% (n=31) of the cohort. The mean duration was 72.91±29.66 hours, with patients having a mean height of 165.57±7.89 cm, weight of 69.71±11.95 kg, and BMI of 25.61±5.07 kg/m^2. Laboratory values showed mean LDL levels of 140.84±33.56 mg/dL and HDL levels of 51.49±13.68 mg/dL. Hypertension was present in 65.0% (n=67) of patients, while 61.2% (n=63) resided in urban areas compared to 38.8% (n=40) in rural areas. A family history of dyslipidemia was reported in 40.8% (n=42) of patients. Stroke types included 86.4% (n=89) ischemic strokes and 13.6% (n=14) hemorrhagic strokes (as shown in Table 1).

Table 1

Patient Demographics

Demographics		Mean ± SD
Age (years)		53.08±12.15
Duration (hours)		72.91±29.66
Height (cm)		165.57±7.89
Weight (kg)		69.71±11.95
BMI		25.61±5.07
LDL (mg/dL)		140.84±33.56
HDL (mg/dL)		51.49±13.68
Gender	Male n (%)	72 (69.9%)
	Female n (%)	31 (30.1%)
Hypertension	Yes n (%)	67 (65.0%)
	No n (%)	36 (35.0%)
Residence	Rural n (%)	40 (38.8%)
	Urban n (%)	63 (61.2%)
Family History of Dyslipidemia	Yes n (%)	42 (40.8%)
	No n (%)	61 (59.2%)
Stroke Type	Hemorrhagic stroke n (%)	14 (13.6%)
	Ischemic stroke n (%)	89 (86.4%)

The prevalence of lipid abnormalities was substantial among the study population. High LDL levels were observed in 59.20% (n=61) of patients, while 40.80% (n=42) had normal LDL levels. Low HDL levels were present in 29.10% (n=30) of patients, with 70.90% (n=73) maintaining normal HDL levels (as shown in Table 2).

Table 2

Frequency of Lipid Abnormalities Among Type II Diabetic Patients with Ischemic MCA Stroke

Lipid Parameter		Frequency	% age
High LDL	Yes	61	59.20%
	No	42	40.80%
	Total	103	100%
Low HDL	Yes	30	29.10%
	No	73	70.90%
	Total	103	100%

Analysis of high LDL association with demographic factors revealed no statistically significant relationships across all examined variables. Among age groups, 54.3% (n=19) of patients ≤50 years and 61.8% (n=42) of patients >50 years had high LDL (p=0.464). Gender distribution showed 58.3% (n=42) of males and 61.3% (n=19) of females with high LDL (p=0.779). BMI stratification revealed 62.5% (n=30) of patients with BMI ≤25 kg/m^2 and 56.4% (n=31) with BMI >25 kg/m^2 had high LDL (p=0.527). Hypertensive patients showed 59.7% (n=40) prevalence versus 58.3% (n=21) in normotensive patients (p=0.893). Family history of dyslipidemia was associated with 66.7% (n=28) high LDL prevalence compared to 54.1% (n=33) without family history (p=0.202). Stroke type analysis showed 57.1% (n=8) of hemorrhagic stroke patients and 59.6% (n=53) of ischemic stroke patients had high LDL (p=0.865) (as shown in Table 3).

Table 3

Association of High LDL with Demographic Factors

Demographic Factors		High LDL		p-value
		Yes n(%)	No n(%)	
Age (years)	≤50	19 (54.3%)	16 (45.7%)	0.464
	>50	42 (61.8%)	26 (38.2%)	
Gender	Male	42 (58.3%)	30 (41.7%)	0.779
	Female	19 (61.3%)	12 (38.7%)	
BMI (Kg/m^2)	≤25	30 (62.5%)	18 (37.5%)	0.527
	>25	31 (56.4%)	24 (43.6%)	
Hypertension	Yes	40 (59.7%)	27 (40.3%)	0.893
	No	21 (58.3%)	15 (41.7%)	
Family History of Dyslipidemia	Yes	28 (66.7%)	14 (33.3%)	0.202
	No	33 (54.1%)	28 (45.9%)	
Stroke Type	Hemorrhagic stroke	8 (57.1%)	6 (42.9%)	0.865
	Ischemic stroke	53 (59.6%)	36 (40.4%)	

Similarly, low HDL associations with demographic factors showed no statistically significant correlations. Age-based analysis revealed 25.7% (n=9) of patients ≤50 years and 30.9% (n=21) of patients >50 years had low HDL (p=0.585). Gender distribution showed 29.2% (n=21) of males and 29.0% (n=9) of females with low HDL (p=0.989). BMI stratification demonstrated 29.2% (n=14) of patients with BMI ≤25 kg/m^2 and 29.1% (n=16) with BMI >25 kg/m^2 had low HDL (p=0.993). Hypertensive patients showed 23.9% (n=16) prevalence versus 38.9% (n=14) in normotensive patients (p=0.110). Family history of dyslipidemia was associated with 28.6% (n=12) low HDL prevalence compared to 29.5% (n=18) without family history (p=0.918). Stroke type analysis revealed 42.9% (n=6) of hemorrhagic stroke patients and 27.0% (n=24) of ischemic stroke patients had low HDL (p=0.224) (as shown in Table 4).

Table 4

Association of Low HDL with Demographic Factors

Demographic Factors		Low HDL		p-value
		Yes n(%)	No n(%)	
Age (years)	≤50	9 (25.7%)	26 (74.3%)	0.585
	>50	21 (30.9%)	47 (69.1%)	
Gender	Male	21 (29.2%)	51 (70.8%)	0.989
	Female	9 (29.0%)	22 (71.0%)	
BMI (Kg/m^2)	≤25	14 (29.2%)	34 (70.8%)	0.993
	>25	16 (29.1%)	39 (70.9%)	
Hypertension	Yes	16 (23.9%)	51 (76.1%)	0.110
	No	14 (38.9%)	22 (61.1%)	
Family History of Dyslipidemia	Yes	12 (28.6%)	30 (71.4%)	0.918
	No	18 (29.5%)	43 (70.5%)	
Stroke Type	Hemorrhagic stroke	6 (42.9%)	8 (57.1%)	0.224
	Ischemic stroke	24 (27.0%)	65 (73.0%)	

DISCUSSION

The present study demonstrates a notably high prevalence of dyslipidemia among type II diabetic patients with stroke, with 59.20% exhibiting elevated LDL levels and 29.10% presenting with low HDL levels, highlighting the significant burden of atherogenic lipid profiles in this high-risk population. The predominance of elevated LDL cholesterol can be attributed to the complex interplay between diabetic metabolic dysfunction and cerebrovascular pathophysiology, where chronic hyperglycemia promotes oxidative stress and glycation of lipoproteins, leading to enhanced atherogenicity and accelerated formation of atherosclerotic plaques in cerebral vessels. The concurrent presence of low HDL levels in nearly one-third of patients reflects the characteristic diabetic dyslipidemia pattern, where insulin resistance and hyperinsulinemia accelerate the catabolism of HDL particles while reducing the synthesis of apolipoprotein A-I, thereby compromising the reverse cholesterol transport mechanism that normally protects against atherothrombosis. The present study's findings of high LDL prevalence (59.20%) and low HDL prevalence (29.10%) in type II diabetic stroke patients align with several international studies while demonstrating some notable variations. Our LDL elevation rate is comparable to Nirmala A.C. et al. [16] who reported elevated LDL in 58.6% of ischemic stroke patients, though their study included both diabetic and non-diabetic patients, suggesting that our exclusively diabetic cohort exhibits similar atherogenic lipid patterns. The consistency between studies indicates that LDL cholesterol remains a predominant lipid abnormality in cerebrovascular disease regardless of diabetic status, likely reflecting the shared pathophysiological mechanisms of atherosclerosis and endothelial dysfunction.

However, our LDL findings contrast with Jan S.S. et al. [17] who found no significant LDL elevation in diabetic stroke patients (129.94±45.63 mg/dL vs 114.44±42.79 mg/dL in non-diabetics, p=0.083), despite our mean LDL of 140.84±33.56 mg/dL being comparable to their diabetic group. This discrepancy may be attributed to different population characteristics, with our study focusing specifically on MCA territory strokes, which may represent a more severe atherosclerotic phenotype requiring higher LDL thresholds for plaque rupture and thrombosis formation. Additionally, regional dietary patterns and genetic predispositions in different populations may influence lipid metabolism and cardiovascular risk profiles. Our HDL findings show interesting parallels and contrasts with existing literature. The 29.10% prevalence of low HDL in our study is substantially lower than the 78% reported by Khan S. et al. [18] in acute ischemic stroke patients, where diabetic patients had significantly lower HDL levels (0.77±0.24 mmol/L) compared to non-diabetics. Similarly, Rafique S. et al. [19] reported low HDL in 64% of ischemic stroke patients, considerably higher than our findings. These differences may reflect variations in HDL cutoff criteria, with our study potentially using more stringent definitions, or may indicate that MCA territory strokes have different lipid associations compared to all-territory ischemic strokes. The lower prevalence in our study could also suggest better baseline HDL levels in our population or different metabolic control among our diabetic patients.

The protective role of HDL demonstrated by Shen Y. et al. [20] in their large prospective study of 67,544 diabetics supports our findings, where they showed an inverse dose-dependent relationship between HDL levels and stroke risk (HR 0.89 per 15 mg/dL increase). This reinforces the clinical significance of our 29.10% low HDL prevalence, as these patients represent a particularly high-risk subset. The mechanistic basis for this relationship involves HDL's role in reverse cholesterol transport, anti-inflammatory properties, and endothelial protection, which become particularly crucial in diabetic patients who already face increased oxidative stress and endothelial dysfunction. The comparison between ischemic and hemorrhagic stroke subtypes in our study, where both showed similar lipid abnormality patterns, differs from Amna Rauf & Rauf S. [21] and Mahmood A. et al. [22] who found significantly higher total cholesterol and LDL in ischemic compared to hemorrhagic stroke. Rauf A. et al. [21] reported higher LDL-C in ischemic stroke (3.1±1.08 vs 2.5±0.9 mmol/L), while Mahmood A. et al. [22] found elevated total cholesterol in 42% of ischemic versus only 5% of hemorrhagic cases. Our findings suggest that in diabetic patients, the metabolic dysfunction may override the traditional lipid-stroke subtype associations, as diabetes creates a pro-atherogenic environment that affects both large vessel atherosclerosis and small vessel disease, potentially leading to similar lipid profiles across stroke subtypes. The absence of significant demographic associations with lipid abnormalities in our study contrasts with some previous findings. Luo Y. et al. [23] demonstrated age-dependent associations between low HDL and ischemic stroke risk in diabetics (OR 6.82 in patients ≤70 years), while our study showed no significant age-related differences. This inequality may point to the differences in study designs, with Luo's case-control study possibly having different risk correlations from our cross-sectional study of patients with established stroke. There being an absence of demographic associations in our series verifies the fact that diabetic dyslipidemia is an independent pathophysiologic entity, crossing all traditional stratifications of risk factors, with all diabetic patients, regardless of their age, gender, or BMI, requiring tight lipid monitoring and management to avert stroke.

There are also several limitations to this study that must be kept in mind while interpreting the results. Single-center study design may limit the generalizability of results to larger populations since institutional processes, patient groups, and referral patterns vary significantly across healthcare facilities. The sample size is fairly small with 103 patients, and it may not have enough statistical power to establish significant correlations between lipid abnormalities and demographic indices, which may explain the finding of nonsignificant correlations in the present study. The cross-sectional study design does not help us define causative relationships between lipid indices and stroke outcome so we cannot define with precision whether the association of stroke-dyslipidemia occurred prior to the stroke or due to acute metabolic derangements secondary to stroke events. In addition, confounding factors such as history of medication, duration of diabetes, current status of glycemic control, dietary intake, etc., were also not taken into consideration in the study and may have significantly influenced lipid profiles as well as predisposition to stroke.

CONCLUSION

Our study has concluded that dyslipidemia represents a significant metabolic burden among type II diabetic patients with ischemic MCA stroke, with elevated LDL cholesterol being the predominant lipid abnormality affecting the majority of patients, while low HDL cholesterol affects approximately one-third of this high-risk population.

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