

ORIGINAL ARTICLE

A Cross-Sectional Study of Frequency of Dyslipidemia in Stroke Patients at Benazir Bhutto Hospital, Rawalpindi

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ABSTRACT

Objective: To determine the frequency of dyslipidemia and its associations with stroke type and other risk factors in patients at a tertiary care hospital.

Study Design: A cross-sectional study.

Place and Duration of Study: This study was conducted at the Department of Neurology at Benazir Bhutto Hospital, Rawalpindi, Pakistan, from July 2024 to November 2024.

Methods: The study included 95 consecutive patients with CT-confirmed stroke (both ischemic and hemorrhagic). A predefined form was used to collect information on demographics, stroke subtype, vascular risk factors (hypertension, diabetes, smoking), and fasting lipid profiles. Dyslipidemia was defined as any of the following: TC \geq 200 mg/dL, TG \geq 150 mg/dL, LDL \geq 130 mg/dL, or HDL $<$ 40 mg/dL. Data was analyzed using SPSS, with means \pm SD and proportions, and group comparisons used t-tests and Chi-Square tests.

Results: The average age was 59.4 \pm 12.8 years (range 25-75), with 60.0% being male. Overall, dyslipidemia was present in 57 out of 95 patients (60.0%). Dyslipidemia was more common in ischemic stroke (40/60, 66.7%) than hemorrhagic stroke (17/35, 48.6%), but the difference was not statistically significant (χ^2 =3.02, P =0.08). The prevalence of dyslipidemia increased with age (40% in \leq 45 years vs. 68% in $>$ 65 years; χ^2 =5.66, P =0.02). Patients with diabetes (75% vs. 54%) or hypertension (68% vs. 41%) had significantly higher dyslipidemia rates (P \leq 0.05). The dyslipidemia group had significantly higher mean lipid levels (TG 198 \pm 72 vs. 132 \pm 45 mg/dL, LDL 145 \pm 32 vs. 105 \pm 28 mg/dL, P $<$ 0.001).

Conclusion: More than half of stroke patients had dyslipidemia, particularly those with ischemic stroke and comorbid diabetes or hypertension. These findings are comparable to other reports, such as ~56-70% dyslipidemia in stroke patients. Routine lipid screening and management should be emphasized in stroke care to address this common modifiable risk factor.

Keywords: Diabetes, Dyslipidemia, Risk factors, Stroke.

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Introduction

Stroke is a major global health problem, ranking as the second leading cause of death and a leading cause of disability worldwide.¹ The global burden of

stroke continues to rise; for example, in 2021, there were approximately 11.9 million incident strokes and 93.8 million prevalent cases, with substantial mortality and disability-adjusted life years (DALYs) lost.² Low- and middle-income countries bear most of this burden, reflecting the impact of modifiable risk factors in these regions.³ In South Asia, including Pakistan and India, stroke incidence and mortality are particularly high, often affecting younger populations than in Western countries.^{4,5} Known modifiable risk factors include hypertension, diabetes mellitus, smoking, obesity, atrial fibrillation, and dyslipidaemia.^{4,6} Among these, dyslipidaemia, defined by abnormalities of

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cholesterol and triglyceride levels, is recognised as a contributor to atherosclerosis and ischaemic stroke. Indeed, guidelines from major stroke associations list dyslipidaemia as a risk factor for stroke.⁷ Large clinical studies have found that approximately 45–60% of ischaemic stroke patients have concomitant hypercholesterolaemia.⁸

Despite this, the frequency of dyslipidaemia among stroke patients varies widely by population and is under-reported in many developing-country settings. Previous single-centre studies in Pakistan and neighbouring regions have found dyslipidaemia prevalence ranging from about 40% to 80% in stroke patients. Notably, dyslipidaemia tends to be more common in ischaemic stroke compared to haemorrhagic stroke, although some analyses have found minimal differences between stroke types. Variations in study methods, lipid cut-off definitions, and population characteristics contribute to this wide range of findings.

Given the impact of dyslipidaemia on cerebrovascular disease, understanding its frequency and correlates in local stroke populations is important for both prevention and management. In Pakistan, data on dyslipidaemia in stroke patients remain limited. The current study aimed to fill this gap by determining the frequency of dyslipidaemia and its association with stroke subtypes and other clinical factors in a series of patients at a tertiary care hospital. By following a predefined protocol and standardised criteria, we provide detailed data on lipid abnormalities in acute stroke, thereby informing clinicians and guiding secondary prevention strategies.

The goal of this study was to determine the frequency of dyslipidemia and its associations with stroke type and selected vascular risk factors in patients at a tertiary care hospital, with particular focus on routinely screened metabolic comorbidities

Methods

This study was conducted at the Department of Neurology at Benazir Bhutto Hospital, Rawalpindi, Pakistan, from July 2024 to November 2024, after obtaining approval from the hospital's Ethical Review Committee vide letter no: 300/IREF/RMU/2024, dated 28th May 2024. A sample size of 95 was calculated to estimate the prevalence of

dyslipidemia with reasonable precision, based on an expected frequency of approximately 57% in prior studies. Consecutive sampling was utilized. Neuroimaging confirmed acute stroke in adult patients aged 18 or older were included. The study included both ischemic and hemorrhagic strokes. The operational definition of stroke was a sudden onset of focal neurological deficit lasting more than 24 hours (or resulting in death), with supporting CT/MRI findings. Patients with transient ischemic attack (TIA), stroke mimics, chronic kidney or liver disease, active malignancy, or those taking lipid-lowering medication before stroke were excluded. Patients on anticoagulants were also excluded due to the risk of intracranial bleeding without prior imaging, as were pregnant women. Chronic kidney disease was not analyzed as a study variable due to incomplete baseline renal profiling in all admitted stroke patients. The institutional ethics committee approved the study protocol, and patients and guardians provided informed consent.

Data were collected prospectively using a structured case proforma. Demographics (age, gender, body mass index), stroke characteristics (type, onset), and vascular risk factors (history of hypertension, diabetes, smoking) were all documented. CT/MRI classified strokes as either ischemic (infarction) or hemorrhagic (intracerebral or subarachnoid hemorrhage). Risk factors were defined using standard criteria, including hypertension (BP $\geq 140/90$ mmHg), diabetes (fasting glucose ≥ 126 mg/dL), and smoking (current or former). Smoking was recorded but not emphasized in the analysis because of its lower prevalence in this sample.

Within 48 hours of admission, each patient provided a fasting blood sample (at 8-12 hours). The central laboratory used enzymatic colorimetric methods to measure serum total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C). Quality control procedures were followed. Dyslipidemia was operationally defined according to the NCEP ATP III criteria. TC ≥ 200 mg/dL, TG ≥ 150 mg/dL, LDL-C ≥ 130 mg/dL, or HDL-C < 40 mg/dL. Patients who met at least one criterion were classified as having dyslipidemia.

Data were entered into SPSS version 23.0.

Continuous variables (age, BMI, lipid levels) were presented as mean \pm SD. Categorical variables (gender, stroke type, risk factors, dyslipidemia, yes/no) were presented as frequencies and percentages. For group comparisons (e.g., dyslipidemia vs no dyslipidemia, ischemic vs hemorrhagic), the Student's t-test was used for continuous measures and the chi-square test for proportions. A two-sided P -value <0.05 indicated statistical significance. We also calculated 95% confidence intervals around key prevalence

estimates. Results are reported in accordance with STROBE guidelines

Results

Ninety-five patients were analyzed. The mean age was 59.4 ± 12.8 years (range 25–75); 57 (60.0%) were male and 38 (40.0%) female. Overall, 60 patients (63.2%) had ischemic stroke and 35 (36.8%) had hemorrhagic stroke. The mean BMI was 26.8 ± 4.3 kg/m². Hypertension was present in 66 (69.5%) patients, and diabetes mellitus in 28 (29.5%). Smoking history was noted in 19 (20.0%). (Table 1).

Table 1: Demographic and clinical features by stroke subtype

Characteristic	Ischemic (N=60)	Hemorrhagic (N=35)	Test of Significance value	P-value
Age, mean \pm SD (yr)	58.1 ± 12.2	61.8 ± 13.7	$t=1.45$	0.15
Male sex, N (%)	36 (60.0%)	21 (60.0%)	$\chi^2=0.00$	1.00
Diabetes, N (%)	18 (30.0%)	10 (28.6%)	$\chi^2=0.02$	0.89
Hypertension, N (%)	42 (70.0%)	24 (68.6%)	$\chi^2=0.02$	0.88
BMI, mean \pm SD	27.0 ± 4.2	26.5 ± 4.6	$t=0.530$	0.60
Dyslipidemia, N (%)	40 (66.7%)	17 (48.6%)	$\chi^2=3.02$	0.08

Table 2: Lipid profile by stroke subtype

Lipid parameter	Ischemic (N=60)	Hemorrhagic (N=35)	t-value	P-value
Total cholesterol	205.4 ± 29.8 mg/dL	191.2 ± 36.4 mg/dL	2.58	0.01
Triglycerides	175.2 ± 65.3 mg/dL	158.1 ± 81.0 mg/dL	1.18	0.24
LDL-C	130.8 ± 30.1 mg/dL	122.3 ± 39.2 mg/dL	1.16	0.15
HDL-C	38.1 ± 9.5 mg/dL	41.3 ± 12.1 mg/dL	1.47	0.08

Table 3: Lipid profile by dyslipidemia status

Parameter	Dyslipidemia (N=57)	No Dyslipidemia (N=38)	t-value	P-value
TC (mg/dL)	220.5 ± 24.3	178.6 ± 22.1	8.97	<0.001
TG (mg/dL)	198.3 ± 71.6	132.2 ± 44.8	5.54	<0.001
LDL-C (mg/dL)	145.7 ± 32.4	104.5 ± 27.9	6.61	<0.001
HDL-C (mg/dL)	34.2 ± 8.5	47.3 ± 10.2	6.55	<0.001

There were no significant differences in age, gender, BMI, diabetes, or hypertension rates between ischemic and hemorrhagic groups (all $P>0.1$). Dyslipidemia was more common in ischemic stroke (66.7%) than hemorrhagic stroke (48.6%), but this difference was not statistically significant ($\chi^2=3.02$, $P=0.08$). In total, 57 out of 95 patients (60.0%) had dyslipidemia.

Overall mean lipid levels were: TC 200.3 ± 33.5 mg/dL, TG 170.4 ± 72.1 mg/dL, LDL-C 128.7 ± 34.1 mg/dL, HDL-C 39.2 ± 10.5 mg/dL. Table 2 compares lipid values by stroke subtype. Ischemic stroke patients

had higher mean TC and TG and lower HDL-C than hemorrhagic patients, though differences were modest.

Ischemic patients had significantly higher TC (205 vs. 191 mg/dL, $P=0.01$) and lower HDL (38.1 vs. 41.3 mg/dL, $P=0.02$) than hemorrhagic patients. However, the differences in mean TG and LDL did not reach statistical significance.

When patients were grouped by dyslipidemia status, marked differences emerged (Table 3). Those with dyslipidemia had much higher mean TC, TG, and LDL levels and lower HDL levels than those without. For

example, mean TG was 198.3 ± 71.6 mg/dL in the dyslipidemia group versus 132.2 ± 44.8 mg/dL in the no-dyslipidemia group ($P < 0.001$). Similar significant differences were seen for LDL (145.7 ± 32.4 vs. 104.5 ± 27.9 mg/dL, $P < 0.001$) and TC (220.5 ± 24.3 vs. 178.6 ± 22.1 mg/dL, $P < 0.001$).

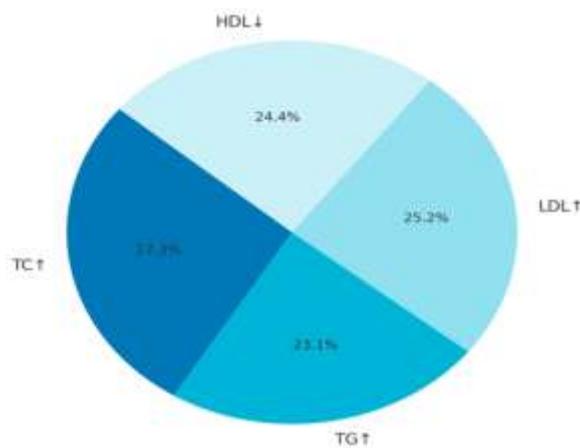


Fig.1: Distribution of lipid parameter abnormalities among ischemic stroke patients (N=60). Values indicate the proportion of patients with deranged levels of each lipid fraction. Total cholesterol (TC) and very low-density lipoprotein (VLDL, not tabulated above) were most commonly elevated

Figure 1 illustrates the high prevalence of deranged

lipids: over half the ischemic stroke patients had elevated TC or VLDL levels. This supports the notion that LDL and TC are key lipid fractions implicated in stroke pathogenesis.

Overall, 57 patients (60.0%) met criteria for dyslipidemia. The frequency was 66.7% in ischemic stroke versus 48.6% in hemorrhagic stroke. Although this difference trended higher for ischemic stroke, it did not reach statistical significance ($P = 0.08$).

We examined the frequency of dyslipidemia across demographic and risk-factor subgroups (Table 4). Dyslipidemia was more common in older patients: only 10 of 25 patients aged ≤ 45 years had dyslipidemia (40%), whereas 47 of 70 patients aged > 45 years had dyslipidemia (67.1%) ($\chi^2 = 5.66$, $P = 0.02$). There was no significant gender difference (59.6% of males vs. 60.5% of females; $P = 0.94$). However, comorbid conditions were strongly related: 21 of 28 (75.0%) diabetic patients had dyslipidemia versus 36 of 67 (53.7%) non-diabetics ($P = 0.05$). Hypertensive patients had an even higher rate: 45 of 66 (68.2%) vs. 12 of 29 (41.4%) among non-hypertensives ($\chi^2 = 6.03$, $P = 0.01$). Thus, both diabetes and hypertension were significantly associated with dyslipidemia (Figure 1, Table 4).

Table 4: Dyslipidemia status by subgroups

Subgroup	With Dyslipidemia (%)	Without Dyslipidemia (%)	Chi-Square (χ^2)	P-value
Age ≤ 45 (N=25)	10 (40.0%)	15 (60.0%)		
Age 46–65 (N=45)	30 (66.7%)	15 (33.3%)	5.66	0.02
Age > 65 (N=25)	17 (68.0%)	8 (32.0%)		
Male (N=57)	34 (59.6%)	23 (40.4%)		
Female (N=38)	23 (60.5%)	15 (39.5%)	0.01	0.94
Diabetes (N=28)	21 (75.0%)	7 (25.0%)		
No Diabetes (N=67)	36 (53.7%)	31 (46.3%)	3.72	0.05
Hypertension (N=66)	45 (68.2%)	21 (31.8%)		
No Hypertension (N=29)	12 (41.4%)	17 (58.6%)	6.03	0.01

Key findings: older age, diabetes, and hypertension were significantly associated with higher dyslipidemia prevalence. Gender had no effect. These patterns underscore the interplay of metabolic risk factors: diabetics and hypertensives were much more likely to have lipid abnormalities

This graph highlights that dyslipidemia occurred in 66.7% of ischemic versus 48.6% of hemorrhagic strokes, 75% of diabetics versus 53.7% of non-diabetics, and 68.2% of hypertensives versus 41.4% of non-hypertensives. The bar chart demonstrates clear clustering of dyslipidemia with vascular comorbidities, reinforcing the metabolic

interdependence of these risk factors.

Discussion

In this cross-sectional study of 95 stroke patients, dyslipidaemia was found in 60.0%, which is consistent with other recent studies in South Asia. For example, a study in Pakistan found that dyslipidemia was prevalent in 57.7% of ischaemic

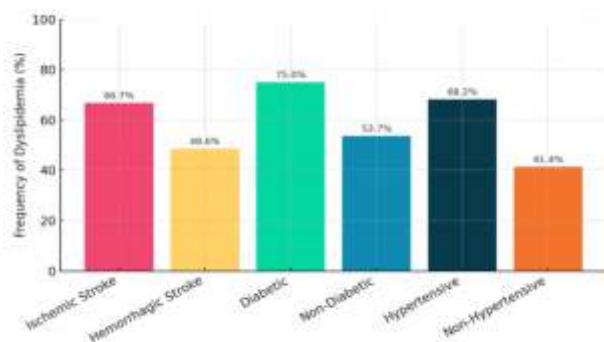


Fig.2: Bar chart showing the frequency of dyslipidemia across stroke type, diabetes, and hypertension status (N = 95)

stroke patients.^{9,10} A study from Peshawar discovered dyslipidemia in 51.6% of first-episode ischaemic stroke patients.¹¹ These figures are comparable to ours, indicating that dyslipidemia is a common comorbidity in these populations. Internationally, dyslipidaemia rates in stroke vary greatly: one Ethiopian hospital-based series discovered dyslipidaemia in 40.5% of stroke patients, whereas a larger meta-analysis of general adult populations in Ethiopia reported a pooled dyslipidaemia prevalence of 56.6%.^{12,13} Although not limited to stroke, the high prevalence highlights the underlying lipid burden in low- and middle-income countries.

We discovered that dyslipidemia was more prevalent in ischaemic stroke (66.7%) than in haemorrhagic stroke (48.6%). This trend is consistent with known pathophysiology: ischaemic strokes are more closely associated with atherosclerotic changes, which are influenced by high cholesterol and triglyceride levels. Several studies have also found higher lipid abnormalities in ischaemic strokes than in haemorrhagic strokes, though the difference is not always statistically significant. One Indian case-control study found no significant difference in lipid profiles between infarction and haemorrhage, but it did identify high total and LDL cholesterol as risk factors for both types of stroke.¹⁴ The potential explanation is that, while elevated lipids clearly promote large-artery atherosclerosis (thereby increasing the risk of ischaemic stroke), the relationship with haemorrhagic stroke is more complicated; some evidence even suggests that elevated cholesterol may reduce the risk of intracerebral haemorrhage.¹⁵ Given the small sample

size, we cannot definitively resolve subtype lipid differences; larger multicenter studies are required. Importantly, in our sample, dyslipidaemia was strongly linked to other vascular risks. Diabetics had 75% dyslipidaemia compared to 53.7% in non-diabetics ($P=0.05$), while hypertensive patients had 68.2% versus 41.4% in non-hypertensives ($P=0.01$). This is consistent with the findings of SeetlaniNket al. in Pakistan, who reported dyslipidaemia in approximately 74% of diabetic stroke patients and 81% of hypertensives.² These associations highlight the clustering of metabolic risk factors: diabetes, hypertension, and dyslipidemia frequently coexist, exacerbating cerebrovascular risk. Furthermore, we found that older age (>45 years) was significantly associated with a higher prevalence of dyslipidaemia (67.0% vs 40.0% in ≤ 45 years). This age trend is consistent with large epidemiological datasets, which show that lipid abnormalities increase with age. We found no gender difference (59.6% male vs 60.5% female), which is consistent with the Peshawar study, which found no significant gender difference in dyslipidemia prevalence among stroke patients.¹²

Although this study focused on commonly documented metabolic risk factors such as diabetes and hypertension, other emerging contributors to stroke risk, particularly chronic kidney disease (CKD), were not evaluated. CKD is increasingly recognized as an independent risk factor for both ischemic and hemorrhagic stroke due to shared mechanisms of endothelial dysfunction, inflammation, and dyslipidemia. The absence of CKD analysis in the present study may partly explain the overlap of dyslipidemia with other metabolic comorbidities and represents an important area for future investigation.

Dyslipidaemic stroke patients had significantly higher mean LDL (~ 146 mg/dL) and TG (~ 198 mg/dL) levels compared to non-dyslipidaemic patients (LDL ~ 105 mg/dL). These clinically significant differences ($P < 0.001$) indicate poor lipid control in this cohort, highlighting the potential benefit of aggressive lipid-lowering interventions in the stroke population. Evidence suggests that statin therapy and LDL-lowering reduce the risk of recurrent stroke and atherosclerotic progression in stroke survivors.¹⁶

Our findings are consistent with global findings that dyslipidemia is a significant modifiable risk factor for stroke and other cardiovascular diseases. A systematic review of lipid profiles in stroke risk, for example, found that high LDL-C and TC levels continue to be significant predictors of ischaemic stroke across diverse populations.¹⁷ While the landmark INTERSTROKE study (2010) quantified the population-attributable risk of various modifiable factors for stroke, newer data show that rising lipid levels, particularly in developing countries, continue to drive cerebrovascular disease progression. Our finding of ~67% dyslipidemia prevalence in ischaemic stroke patients is broadly consistent with the often-quoted 45-60% range for hypercholesterolemia among ischaemic stroke patients.¹⁸⁻²⁰ Our work adds a local context: dyslipidaemia is still extremely common among acute stroke patients in a Pakistani tertiary-care hospital, highlighting the urgent need for screening and secondary prevention.

There are several limitations to consider. First, our cross-sectional design prevents us from determining whether lipid abnormalities predate or postdate the stroke. Second, the small sample size (N = 95) and single-center recruitment reduce generalizability; the stroke population may differ in other settings or regions. Third, although we excluded patients already receiving lipid-lowering therapy, we did not measure pre-stroke lipid levels, so residual confounding is possible. Fourth, stroke itself can cause acute changes in lipid metabolism (for example, stress-induced changes), so fasting lipids collected within 48 hours of admission may not accurately reflect baseline. We tried to mitigate this with early sampling, but the possibility remains. Our study followed a predefined protocol, used standard dyslipidaemia definitions (TC \geq 200 mg/dL, TG \geq 150 mg/dL, LDL \geq 130 mg/dL, HDL $<$ 40 mg/dL), and collected detailed subgroup data (by stroke type, age, and comorbidity), which improves the findings' quality.

Additionally, other vascular risk factors, such as chronic kidney disease, could not be assessed, which may have limited a more comprehensive evaluation of dyslipidemia-related stroke risk. Despite these limitations, the study provides valuable local data

from a public-sector tertiary care hospital, highlighting the persistent burden of dyslipidemia in acute stroke patients and reinforcing the need for aggressive lipid screening and secondary prevention strategies in resource-limited settings

Conclusion

More than half of stroke patients had dyslipidemia, particularly those with ischemic stroke and comorbid diabetes or hypertension. These findings are comparable to other reports, such as ~56-70% dyslipidemia in stroke patients. Routine lipid screening and management should be emphasized in stroke care to address this common modifiable risk factor.

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Author Contributions

ZE: Conception and design of the work, writing the original draft, proofreading, and approval for final submission

MRM: Manuscript writing for methodology design and investigation

MA: Validation of data, interpretation, and write-up of results

NK: Data acquisition, curation, and statistical analysis

HG: Revising, editing, and supervising for intellectual content