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Differences in lipid profile between acute ischemic and hemorrhagic stroke patients admitted in a referral neuroscience hospital in Dhaka

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Abstract

Background: Blood lipid levels differently impact ischemic stroke and hemorrhagic stroke. The effect of acute illness on lipid metabolism may alter lipid levels after acute stroke.

Objectives: To measure the fasting lipid profile after acute stroke and to compare between ischemic and hemorrhagic stroke.

Methods: This cross-sectional study enrolled 247 acute stroke patients (112 hemorrhagic and 135 ischemic; age 59.7 (±13.4) years, 48.6% female) from a referral neuroscience hospital in Dhaka, during February-June 2021. Fasting lipid profile was measured by enzymatic [total cholesterol (TC)], accelerator selective detergent [both high-density lipoprotein cholesterol (HDL-c) and low-density lipoprotein cholesterol (LDL-c)], and glycerol phosphate oxidase method [triglyceride (TG)] by the Abbott Alinity system. Elevated TC, TG and LDL-c were defined as ≥200 mg/dL, ≥150 mg/dL and ≥130 mg/dL respectively while low HDL-c as <40 mg/dL. Presence of either elevated serum TC, TG, LDL-c or low HDL-c was regarded as dyslipidemia.

Results: Dyslipidemia was present in 70.9% (175 of 247) with similar frequency in stroke subtypes (p=0.221). Both TC and HDL-c were low in ischemic stroke in comparison to hemorrhagic stroke (p=0.017 and p=0.042 respectively). TG and LDL-c were similar between two groups (p=NS for both). Frequency of low HDL-c was higher in ischemic stroke (p=0.034) but no significant difference in frequency of elevated TC, TG, LDL-c between two groups. There was a higher TG/HDL-c ratio in ischemic stroke in comparison to hemorrhagic stroke (p=0.003), but TC/HDL-c and LDL-c/HDL-c ratio was similar. When the characteristics of the participants with or without dyslipidemia were compared within two stroke types, only the frequency of diabetes was higher in ischemic stroke with dyslipidemia in comparison to those who had no dyslipidemia (p=0.005).

Conclusions: Dyslipidemia is frequent in acute stroke. The fasting TC and HDL-c are low and TG/HDL-c is high in ischemic stroke in comparison to hemorrhagic stroke. *[J Assoc Clin Endocrinol Diabetol Bangladesh, July 2023; 2 (2):52-57]*

Keywords: Dyslipidemia, ischemic stroke, hemorrhagic stroke

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Introduction

The annual number of strokes and stroke-related deaths increased substantially worldwide over the last 30 years and currently stroke is the second leading cause of death and the third major cause of disability (as indicated by loss of disability-adjusted life-years or DALYs).[1] In Bangladesh, a population-based study involving both urban and rural people observed a stroke prevalence of 11.4 per thousand population which is higher than other low and middle-income countries (5.4 to 10.4 per thousand), but much lower than the reports (26–80 per thousand) from high-income countries.[2] To reduce the burden of stroke, its risk factors are needed to be addressed and well managed.

The relationship of dyslipidemia with different types of stroke is an area of interest for stroke researchers. A

positive association between ischemic stroke risk and low-density lipoprotein cholesterol (LDL-c) or non-high-density lipoprotein cholesterol (non-HDL-c) has been observed.[3] Lowering the lipid levels by statins is proven to be effective for both primary and secondary prevention of ischemic stroke especially for people at high cardiovascular risk.[4] However, the relationship is not similar for hemorrhagic stroke. Low LDL-c (<70 mg/dL) and low triglyceride (TG) levels are associated with an increased risk of hemorrhagic stroke.[5] A meta-analysis reported higher risk of hemorrhagic stroke in persons with lower total cholesterol (TC), lower LDL-c and higher HDL-c.[6] Low TG was also seen to increase the risk of hemorrhagic stroke while decrease the risk of ischemic stroke.[7] Therefore, it is possible that the blood lipid levels of those who experience an ischemic or hemorrhagic stroke differ, and that this difference may be seen after the acute occurrence of stroke.

The effect of critical illness is another factor that may change lipid levels in stroke. In patients with severe injury, hypocholesterolemia occurs and observed to be related to outcome of the patients.[8] Cardiovascular events like acute myocardial infarction (MI) also induces an acute fall in TC and LDL-c levels within 24 hours of event and persisting up to day 4.[9] However, change in HDL-c and TG was not significant. One of the independent predictors of decrease in LDL-c was diabetes mellitus. Such change in lipid profile may also be anticipated and has been observed in acute ischemic stroke.[10] Evidence on changes of lipid after acute hemorrhagic stroke is still inadequate.

To fully evaluate the impact and change of lipid on and after various types of stroke, a longitudinal study with periodic measurement of lipid profile would be ideal. However, this type of study is difficult to conduct in a low-resource setting. A cross-sectional study involving the acute stroke patients may provide a picture of lipid status in ischemic and hemorrhagic stroke. So, this study was carried out with a view to evaluate the lipid profile and to compare this between acute ischemic and hemorrhagic stroke.

Methods

Study design: This cross-sectional study was carried out in a referral neuroscience hospital in Dhaka during February 2021 to June 2021. Adult patients (aged >18 years) admitted within 48 hours of the acute event of stroke [both ischemic and hemorrhagic; diagnosed by computed tomography (CT) scan or magnetic resonance imaging (MRI) of brain] were included consecutively. Patients who were on lipid lowering drugs before the event and those with known liver disease, familial hypercholesterolemia or hypothyroid were excluded. The study also excluded individuals who had cerebral hemorrhage as a result of cerebral tumor, trauma, or pre-existing coagulation problems.

Study procedure: A semi-structured questionnaire was used to record sociodemographic, clinical, laboratory, and neuroradiological data. A 5-mL overnight fasting blood sample was collected from the stroke patients within 48 hours of the time of admission by laboratory personnel in accordance with standard operating procedures (SOPs). This was done after receiving the consent to participate in the study. After 30 minutes, samples were centrifuged at 3500 rpm for 5 minutes in order to extract serum. The serum from blood was measured for TC using the enzymatic method, HDL-c and LDL-c using accelerator selective detergent method and TG using glycerol phosphate oxidase method (Abbott Alinity system, Abbott Park, Illinois, USA).

Operational definition: TC, TG and LDL-c was considered elevated when they were ≥200 mg/dL, ≥150 mg/dL and ≥130 mg/dL respectively. Low HDL-c was defined as <40 mg/dL. Dyslipidemia was defined as the presence of either elevated serum TC, TG, LDL-c or low HDL-c.

Statistical analysis: Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp, Armonk, NY, USA). Results were described in frequencies and percentages for qualitative values. For quantitative values, the data distribution was assessed by Shapiro–Wilk test. Mean (±standard deviation; SD) or median with interquartile range (IQR) were used for normal or skewed data respectively. The unpaired t-test, Mann-Whitney U test, median test or chi-squared test were used to compare between two groups. $P<0.05$ was considered a significant difference.

Ethical aspects: Ethical approval was taken from the institutional review board (IRB) for the study. Patients were included in the study after taking prior written consent. The consent was taken from the legal guardian in case of non-communicable patient. Patients and/or their guardians were clearly briefed about the nature, purpose and procedure of the study before taking consent. Participant's right to refuse to participate or to withdraw from the study were reserved.

Results

The study included 247 acute stoke patients (112 hemorrhagic and 135 ischemic) admitted within 48 hours of acute event. The mean age of the participants

Table-I: Clinical characteristics of study participants (n=247)

Variables		Total n = 247	Hemorrhagic n = 112	Ischemic n = 135	*p
Age; (mean±SD)		59.7±13.4	59.5±13.5	59.9±13.3	0.831
Gender	Male	127 (51.4)	53 (47.3)	74 (54.8)	0.241
	Female	120 (48.6)	59 (52.7)	61 (45.2)	0.275
History of hypertension		174 (70.4)	75 (67.0)	99 (73.3)	0.001
History of Diabetes		105 (42.5)	35 (31.3)	70 (51.9)	0.925
GCS score; median (interquartile range)		10 (9-12)	10 (8-13)	10 (9-12)	0.057
NIHSS score; median (interquartile range)		16 (12-19)	17.5 (12-20)	16 (10-19)	

*stands for comparison between hemorrhagic and ischemic stroke by unpaired t-test, chi-squared test or Mann-Whitney U test as appropriate
Within parentheses are percentages over column total if not mentioned otherwise
GCS: Glasgow coma scale NIHSS: National Institute of Health Stroke Scale

Table-II: Comparison of lipid levels between hemorrhagic and ischemic stroke patients (n=247)

Variables	Total n = 247	Hemorrhagic n = 112	Ischemic n = 135	*p
TC (mean±SD)	197.95±52.45	206.54±46.67	190.60±55.82	**0.017***
TG (median, IQR)	95.5 (69.8-133.0)	94.0 (69.3-118.0)	98.5 (69.8-147.3)	**For median 0.317; for distribution 0.119
LDL-c (mean±SD)	128.38±47.99	132.74±43.48	124.63±51.24	0.186*
HDL-c (median, IQR)	46.0 (39.0-57.0)	49.0 (40.3-59.8)	45.0 (36.8-55.0)	**For median **0.042; for** distribution **0.002**

* stands for comparison between hemorrhagic and ischemic stroke by unpaired T-test
** stands for comparison between hemorrhagic and ischemic stroke by Median test (for median) and Mann-Whitney U test (for distribution)
TC: total cholesterol TG: Triglyceride LDL-c: low density lipoprotein cholesterol
HDL-c: high density lipoprotein cholesterol

Table-III: Comparison of dyslipidemia between hemorrhagic and ischemic stroke patients (n=247)

Variables	Total n = 247	Hemorrhagic n = 112	Ischemic n = 135	*p
Elevated TC	116 (47.0%)	58 (51.8%)	58 (43.0%)	0.167
Elevated TG	50 (20.2%)	17 (15.2%)	33 (24.4%)	0.071
Elevated LDL-c	116 (47.0%)	56 (50.0%)	60 (44.4%)	0.384
Low HDL-c	67 (27.1%)	23 (20.5%)	44 (32.6%)	**0.034**
Presence of dyslipidemia	175 (70.9%)	75 (67.0%)	100 (74.1%)	0.221

* stands for comparison between hemorrhagic and ischemic stroke by chi-squared test
TC: total cholesterol TG: Triglyceride LDL-c: low density lipoprotein cholesterol
HDL-c: high density lipoprotein cholesterol Elevated TC ≥200 mg/dL
Elevated TG ≥150 mg/dL Elevated LDL-c ≥130 mg/dL Low HDL-c <40 mg/dL
Dyslipidemia defined as either serum TC levels of ≥200 mg/d, serum HDL-c levels of <40 mg/dl, and serum LDL-c levels of ≥130 mg/dl or serum TG levels of ≥150 mg/dl

was 59.7 (±13.4) years, 48.6% were female. There was no significant difference between hemorrhagic and ischemic stroke patients in relation to age, gender, history of hypertension, Glasgow coma scale (GCS) score or National Institute of Health Stroke Scale (NIHSS) score. Diabetes was present in 42.5% participants, which was higher in frequency in ischemic stroke in comparison to hemorrhagic stroke (ischemic vs. hemorrhagic: 51.9% vs. 31.3%; p=0.001) (Table-I).

Table-II shows the comparison of lipid levels between hemorrhagic and ischemic strokes. Both TC and HDL-c were modestly but significantly lower in ischemic stroke

Table-IV: Comparison of lipid ratios between hemorrhagic and ischemic stroke patients (n=247)

Variables	Total n = 247	Hemorrhagic n = 112	Ischemic n = 135	*p
TC/HDL-c	4.1 (3.4-5.2)	3.9 (3.3-4.9)	4.4 (3.4-5.4)	*For median 0.096; for distribution 0.101
TG/HDL-c	2.0 (1.4-3.2)	1.9 (1.3-2.4)	2.2 (1.5-4.0)	*For median 0.055; for **distribution 0.003**
LDL-c/HDL-c	2.7 (2.0-3.6)	2.6 (2.0-3.4)	2.8 (2.1-3.8)	*For median 0.055; for distribution 0.163

* stands for comparison between hemorrhagic and ischemic stroke by Median test (for median) and Mann-Whitney U test (for distribution)
TC: total cholesterol TG: Triglyceride LDL-c: low density lipoprotein cholesterol
HDL-c: high density lipoprotein cholesterol

Table-V: Comparison of clinical characteristics in participants with or without dyslipidemia within each stroke group (n=247)

Variables	Hemorrhagic			Ischemic		
	With dyslipidemia (n=75)	Without dyslipidemia (n=37)	*p	With dyslipidemia (n=100)	Without dyslipidemia (n=35)	*p
Age	58.9±12.8	60.8±15.1	0.831	59.2±13.2	61.7±13.5	0.342
Gender						
Male	30 (40.0)	23 (62.2)	0.241	54 (54.0)	20 (57.1)	0.748
Female	45 (60.0)	14 (37.8)		46 (46.0)	15 (42.9)	
Hypertension	54 (72.0)	21 (56.8)	0.275	73 (73.0)	26 (74.3)	0.882
Diabetes	24 (32.0)	11 (29.7)	0.001	59 (59.0)	11 (31.4)	**0.005**
GCS; median (interquartile range)	10 (8-13)	10 (9-14)	0.925	10 (9-12)	10 (9-12)	0.895
NIHSS; median (interquartile range)	18 (12-20)	17 (11-22)	0.057	16 (11-19)	14 (10-18)	.473

* by unpaired t-test, chi-squared test or Mann-Whitney U test as appropriate
GCS: Glasgow coma scale NIHSS: National Institute of Health Stroke Scale

group in comparison to hemorrhagic stroke (p=0.017 and p=0.042 respectively). The level of TG and LDL-c was statistically similar between two groups (p=NS for both). When the cholesterol level was dichotomized by using specific cut-offs (Table-III), there was no significant difference in frequency of elevated TC, TG, LDL-c between two groups, but frequency of low HDL-c was significantly higher in ischemic stroke patients (ischemic vs. hemorrhagic: 32.6% vs. 20.5%; p=0.034). The overall presence of dyslipidemia in the participants was 70.9% (175 out of 247), with statistical similar frequency in stroke subtypes. Comparison of lipid ratios between hemorrhagic and ischemic stroke revealed a higher TG/HDL-c ration in ischemic stroke patients (p=0.003 for distribution), although TC/HDL-c and LDL-c/HDL-c ration was statistically similar (Table-IV).

When the characteristics of the participants with or without dyslipidemia was compared within two stroke types, all the variables like age, gender, hypertension, GCS and NIHSS score was similar. However, frequency of diabetes was higher in ischemic stroke patients with dyslipidemia (p=0.005) (Table-V).

Discussion

This study evaluated the fasting lipid profile of hospitalized acute stroke patients soon after the acute incidence and compared the level between ischemic and hemorrhagic stroke. Near about 70% of stroke patients had some form of dyslipidemia. In contrast to usual observation of higher TC in ischemic stroke, the study observed a lower TC in this group in comparison to hemorrhagic stroke. However, HDL-c was also lower in ischemic stroke while LDL-c and TG were statistically

similar between two stroke types. A higher TG/HDL ration was observed in ischemic stroke patients. Frequency of diabetes was higher in ischemic stroke group, especially in those who had dyslipidemia.

The comparison of lipid profile involving stroke patients resulted in variable observations by different groups of researchers. A retrospective study compared pre-event lipid profile of 114 hemorrhagic and 87 ischemic stroke patients and observed no significant difference in lipid levels between two groups.[11] Another study measured immediate post-event lipid profile in acute stroke (60 hemorrhagic, 60 ischemic) and again observed no significant difference between two stroke types.[12] However, in a study involving 258 patients with acute stroke, there was a higher post-stroke TG value in ischemic group in comparison to hemorrhagic group but TC, LDL-c and HDL-c was statistically similar.[13] We did not observe any difference of TG levels between two types of stroke, but presence of low HDL-c and high TG/HDL-c ration in ischemic stroke reflects the classical pattern of metabolic syndrome that leads to cardiovascular events like ischemic stroke.[14]

There was a lower TC in ischemic stroke in comparison to hemorrhagic stroke in the present study. As dyslipidemia is related more to ischemic stroke, the finding seems to be paradoxical. However, it may be explained by the acute changes in lipid levels following ischemic stroke. It is evident that there is a fall of TC after a cardiovascular event.10 This change is more pronounced and found to be associated with mortality in ischemic stroke as like it occurs after MI.[15,16] As a result, the expected higher TC in ischemic stroke may be nullified under the influence of acute event. However, it is not clear why the same change does not take place after hemorrhagic stroke.

Low HDL-c had been observed to be the most important component of dyslipidemia related to acute ischemic stroke risk.[17] In contrast, HDL-c had no significant linear relationship with hemorrhagic stroke.[18] The finding of low HDL-c in acute ischemic stroke in the current study supports this observation. TG/HDL-c ration is good risk marker for metabolic syndrome and cardiovascular disease.[19] Our observation of higher TG/HDL-c in ischemic stroke in comparison to hemorrhagic group supports this notion.

Having no healthy control group is a limitation of the current study. The changes of lipid profile over time after stroke was not evaluated due to the cross-sectional design of the study. Pre-stroke event lipid profile measurement was also beyond the scope of the present study. The future studies should overcome these limitations. The current study, by measuring the post-event lipid profile in a considerable number of stroke patients, highlighted the fact that lipid metabolism may be differently affect the pathogenesis of ischemic and hemorrhagic stroke. The immediate effects of acute stroke on blood lipids may also differ according to stroke type.

Conclusions

In summary, dyslipidemia is present in a considerable number of acute stroke patients. The fasting TC and HDL-c are low and TG/HDL-c is high in ischemic stroke in comparison to hemorrhagic stroke.

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Conflict of Interest

The authors have no conflicts of interest to disclose.

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Data Availability

Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

This study was approved by the Institutional Review Board (IRB) of NINS. Informed written consent was obtained from each of the participants included in the study.

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References

1. GBD 2019 Stroke Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Neurol 2021;20(10):795-820. DOI:10.1016/S1474-4422 (21)00252-0.
2. Mondal MBA, Hasan ATMH, Khan N, Mohammad QD. Prevalence and risk factors of stroke in Bangladesh: A

nationwide population-based survey. eNeurologicalSci 2022;28: 100414. DOI:10.1016/j.ensci.2022.100414.
3. Glasser SP, Mosher A, Howard G, Banach M. What is the association of lipid levels and incident stroke? Int J Cardiol 2016;220:890-94. DOI:10.1016/j.ijcard.2016.06.091.
4. Milionis H, Ntaios G, Korompoki E, Vemmos K, Michel P. Statin-based therapy for primary and secondary prevention of ischemic stroke: A meta-analysis and critical overview. Int J Stroke 2020;15(4):377-384. DOI: 10.1177/1747493019873594.
5. Rist PM, Buring JE, Ridker PM, Kase CS, Kurth T, Rexrode KM. Lipid levels and the risk of hemorrhagic stroke among women. Neurology 2019;92(19):e2286-e2294. DOI:10.1212/ WNL.0000000000007454.
6. Wang X, Dong Y, Qi X, Huang C, Hou L. Cholesterol levels and risk of hemorrhagic stroke: a systematic review and meta-analysis. Stroke 2013;44(7):1833-39. DOI:10.1161/ STROKEAHA.113.001326.
7. Bonaventure A, Kurth T, Pico F, Barberger-Gateau P, Ritchie K, Stapf C, et al. Triglycerides and risk of hemorrhagic stroke vs. ischemic vascular events: The Three-City Study. Atherosclerosis 2010 May;210(1):243-8. DOI: 10.1016/j.atherosclerosis. 2009.10.043.
8. Dunham CM, Fealk MH, Sever WE. Following severe injury, hypocholesterolemia improves with convalescence but persists with organ failure or onset of infection. Crit Care 2003;7(6):145-53. DOI:10.1186/cc2382.
9. Rott D, Klempfner R, Goldenberg I, Leibowitz D. Cholesterol levels decrease soon after acute myocardial infarction. Isr Med Assoc J 2015;17(6):370-3.
10. Yan B, Parsons M, McKay S, Campbell D, Infeld B, Czajko R, et al. When to measure lipid profile after stroke? Cerebrovasc Dis 2005;19(4):234-38. DOI: 10.1159/000084086.
11. Alkhaneen H, Alsadoun D, Almojel L, Alotaibi A, Akkam A. Differences of lipid profile among ischemic and hemorrhagic stroke patients in a tertiary hospital in Riyadh, Saudi Arabia: A retrospective cohort study. Cureus 2022;14(5):e25540. DOI:10.7759/cureus.25540.
12. Grace M, Jacob KJ, Kumar AV, K SV. Role of dyslipidemia in stroke and comparison of lipid profile in ischemic and hemorrhagic stroke -a case-control study. Int J of Adv Med 2016;3(3):694-698. DOI:10.18203/2349-3933.ijam20162520.
13. Togha M, Gheini MR, Ahmadi B, Khashaiar P, Razeghi S. Lipid profile in cerebrovascular accidents. Iran J Neurol 2011; 10(1-2):1-4.
14. Nie G, Hou S, Zhang M, Peng W. High TG/HDL ratio suggests a higher risk of metabolic syndrome among an elderly Chinese population: A cross-sectional study. BMJ Open. 2021;11(3): e041519. DOI:10.1136/bmjopen-2020-041519.
15. Kumar N, Kumar S, Kumar A, Shakoor T, Rizwan A. Lipid profile of patients with acute myocardial infarction (AMI). Cureus 2019;11(3):e4265. DOI: 10.7759/cureus.4265.
16. Cheng KH, Lin JR, Anderson CS, Lai WT, Lee TH. Lipid Paradox in statin-naïve acute ischemic stroke but not hemorrhagic stroke. Front Neurol 2018;9:541. DOI:10.3389/ fneur.2018.00541.
17. Ramírez-Moreno JM, Rebollo B, Macías-Sedas P, Valverde N, Parejo A, Felix-Redondo FJ, et al. Strength of association of classical vascular risk factors in young patients with ischaemic stroke: a case-control study. Neurologia 2022: S2173-5808(22) 00142-0. DOI: 10.1016/j.nrleng.2022.07.006.
18. Jin X, Chen H, Shi H, Fu K, Li J, Tian L, et al. Lipid levels and the risk of hemorrhagic stroke: A dose-response meta-analysis. Nutr Metab Cardiovasc Dis 2021;31(1):23-35. DOI: 10.1016/ j.numecd.2020.10.014.
19. Kosmas CE, Rodriguez Polanco S, Bousvarou MD, Papakonstantinou EJ, Peña Genao E, et al. The triglyceride/ high-density lipoprotein cholesterol (TG/HDL-C) ratio as a risk marker for metabolic syndrome and cardiovascular disease. Diagnostics 2023;13(5):929. DOI: 10.3390/ diagnostics 13050929.