

Comparative analysis of serum lipid profiles in ischemic and hemorrhagic stroke: A retrospective cross-sectional study

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Abstract. *Background and Aim:* Stroke continues to present a global burden of disability, commonly presenting with symptoms such as headache, nausea, vomiting, and altered motor function. Stroke is classified into two major types: Hemorrhagic Stroke (HS), when the brain is bleeding; and Ischemic Stroke (IS), when a blockage prevents blood from flowing in the brain. Lipid level is an established risk factor for stroke, so patients with poor lipid regulation have a higher risk of suffering stroke. This study aimed to compare the serum lipid profiles of patients diagnosed with IS and HS. *Methods:* This study was an analytical (observational) study with a retrospective cross-sectional design. Demographic data and lipid profiles were obtained from patient medical records to compare lipid profiles and lipid ratios of the two stroke types. *Results:* Comparison of lipid profiles between the two groups revealed no significant difference in terms of total cholesterol (TC) ($p=0.317$), high-density lipoprotein (HDL) ($p=0.94$), low-density lipoprotein (LDL) ($p=0.516$), and triglyceride (TG) ($p=0.356$). Similarly, comparisons of lipid ratios showed no statistically significant differences in TC/HDL ($p=0.926$), LDL/HDL ($p=0.788$), and TG/HDL ($p=0.166$) between IS and HS patients. *Conclusions:* Despite higher mean serum lipid values and lipid ratios (TC/HDL, LDL/HDL, TG/HDL) among IS patients in the clinical setting compared to HS patients, statistically significant differences were not detected. Further large-scale studies should be conducted to clarify the clinical significance of lipid profiles in different stroke subtypes. (www.actabiomedica.it)

Key words: stroke, lipid profile, comparative analysis, ischemic stroke, hemorrhagic stroke, lipid ratio

Introduction

Stroke remains a leading cause of disability and mortality worldwide. According to the Global Burden of Disease Study 2019, stroke is the second leading cause of death and the third leading cause of disability-adjusted life-years (DALYs) globally, accounting for over 12 million incident cases and 6.5 million deaths annually (1). Common symptoms include headache,

nausea, vomiting, and motor deficits. Stroke is broadly classified into two major types: hemorrhagic stroke (HS), which involves bleeding within the brain, and ischemic stroke (IS), which results from an obstruction of cerebral blood flow. In Indonesia, the burden of stroke is substantial, with an estimated 500,000 new cases annually. Alarmingly, approximately 2.5% of these patients—around 125,000 individuals—die from the disease, while many survivors experience

long-term disability to varying degrees (2). The pathogenesis of stroke involves a multifactorial interplay of modifiable and non-modifiable risk factors. Among the modifiable factors, dyslipidemia has emerged as a critical determinant. Dyslipidemia is abnormal lipid metabolism, including hypercholesterolemia, hypertriglyceridemia, and low high-density lipoprotein (HDL) cholesterol levels. The association between hypercholesterolemia and IS is well established. Elevated levels of low-density lipoprotein (LDL) cholesterol—commonly referred to as “bad” cholesterol—promote atherosclerosis, a progressive narrowing of cerebral vessels that reduces perfusion and increases the risk of IS (3). Conversely, the relationship between lipid abnormalities and HS remains less clear. While hypercholesterolemia is predominantly implicated in atherogenesis and ischemic events, some studies suggest that low cholesterol levels may increase the risk of HS by weakening the cerebrovascular endothelium, thereby promoting the formation and rupture of microaneurysms, which can lead to intracerebral hemorrhage (4). This dichotomy underscores the importance of differentiating preventive and therapeutic strategies based on stroke subtype. Dyslipidemia, as a modifiable risk factor, represents a key target for intervention. The quantitative effect of cholesterol levels on stroke risk has been demonstrated in several studies. For example, a 1 mmol/L increase in total cholesterol is associated with a 25% increased risk of IS due to accelerated atherogenesis (5). The total cholesterol to HDL cholesterol ratio is an important surrogate marker of cardiovascular risk, including IS. Optimal ratios are generally <4.6 for men and <4.0 for women; higher ratios indicate a predominance of atherogenic lipids over protective HDL cholesterol, elevating the risk of atherosclerotic complications (6). Given the significant morbidity and mortality associated with stroke and the differential influence of dyslipidemia on its subtypes, a detailed assessment of lipid profiles in HS and IS patients is warranted. This study compares the lipid profiles of IS and HS patients treated at Wahidin Sudirohusodo Hospital, a major tertiary referral center. The findings are expected to provide locally relevant data that could inform clinical decision-making and contribute to developing targeted stroke prevention and management strategies in this population.

Material and methods

Study design

This study adopted an analytical observational study with a retrospective cross-sectional design conducted at Wahidin Sudirohusodo hospital, Makassar. Secondary data were extracted from the medical records of hospitalized stroke patients between January 2021 and December 2022. This single-center study aimed to evaluate and compare the serum lipid profiles of patients diagnosed with either HS or IS, based on radiological and clinical criteria. The retrospective cross-sectional design was selected due to the accessibility of well-documented, structured medical records within the specified study period, enabling the investigators to analyze pre-existing clinical and laboratory data by defined objectives (Figure 1.).

Sample criteria

The target population comprised all patients admitted with a definitive diagnosis of stroke—both HS and IS—within the stipulated timeframe. The accessible sample included all patients who fulfilled both the inclusion and exclusion criteria. The inclusion criteria were as follows: age ≥ 18 years at admission, clinical

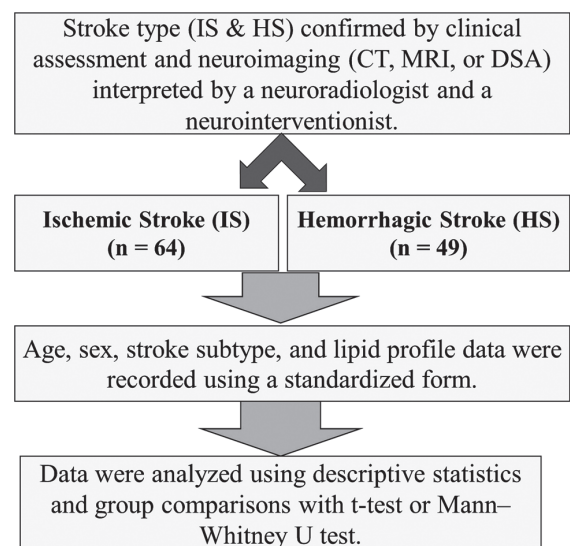


Figure 1. Overview of study flow.

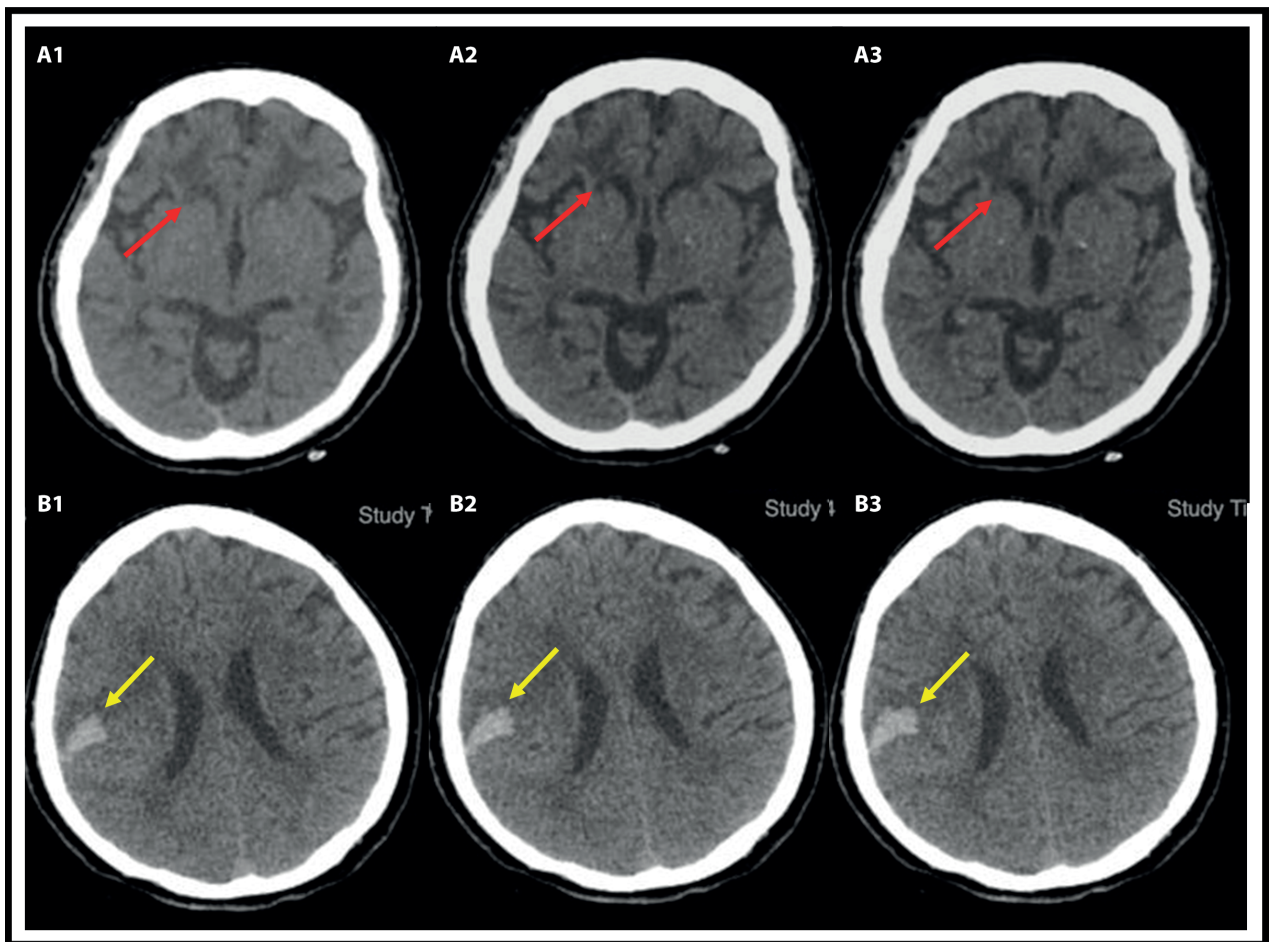


Figure 2. Non-contrast head CT scan of ischemic stroke and hemorrhagic stroke patients. a1, a2, a3). Showing a hypodense lesion with 17 Hounsfield Units (HU), a lacunar infarction in the right lentiform nucleus (indicated by the red arrow) in a patient with ischemic stroke; b1, b2, b3) Showing the presence of hyperdense lesions with 72 HU accompanied by perifocal edema around it with an estimated hemorrhage volume of \pm 2.8 cc in the right parietal lobe in a hemorrhagic stroke patient (indicated by the yellow arrow).

diagnosis of HS or IS, confirmed through imaging of neuroimaging such as head computed tomography (CT) scan or magnetic resonance imaging (MRI) or cerebral digital subtraction angiography (C-DSA) (Figure 2) and physician documentation, complete laboratory data available on lipid profile parameters, including total cholesterol, LDL, HDL, and triglycerides (TG). Exclusion criteria included incomplete or missing data related to lipid profiles in the medical records, history of concurrent acute infections, chronic kidney disease, or other metabolic conditions that may independently alter lipid metabolism and confound the interpretation of results. This rigorous inclusion

and exclusion process ensured a homogeneous and representative cohort for valid comparison between the two stroke subtypes.

Data collection procedure

Data was collected through a systematic review of electronic medical records, focusing on patients who met the eligibility criteria. Demographic variables (age, sex), clinical stroke subtype (HS or IS), and laboratory data, including serum lipid profiles, were extracted and recorded in a standardized data collection form. Data integrity and accuracy were verified

through independent cross-checking by two members of the research team.

Data classification and grouping

Once the data were compiled, subjects were categorized into two primary groups based on stroke classification: (1) hemorrhagic stroke group and (2) ischemic stroke group. Lipid profile variables and patient characteristics were compared between these two cohorts to identify any statistically significant differences.

Statistical analysis

All statistical analyses were carried out using the IBM Statistical Package for the Social Sciences (SPSS) software, version 25.0 for Windows (IBM Corp., Armonk, NY, USA). Demographic and clinical characteristics of the study population were described using descriptive statistics. Normal distribution of continuous variables was tested with the Kolmogorov–Smirnov test. For data with normal distribution, results were presented as mean \pm standard deviation (mean \pm SD), and for non-normal distribution outcomes, as medians and interquartile ranges (IQR). Categorical variables were described by absolute numbers and percentages. Independent samples t-test was done for between-group comparisons of normally distributed continuous data. In the situation of where the normality assumption was not satisfied, the non-parametric Mann–Whitney U test was used. Results were considered statistically significant when the two-tailed p-value was less than 0.05.

Results

This study is a retrospective observational study conducted at Wahidin Sudirohusodo Hospital Makassar, utilizing secondary data obtained from inpatient medical records of patients diagnosed with stroke during the 2021 and 2022 treatment periods. A total of 113 subjects met the inclusion and exclusion criteria and were categorized into two groups: IS (n = 64) and HS (n = 49). Data analysis was performed using SPSS

version 25. The Kolmogorov–Smirnov test was used to assess normality (7). Variables with normal distribution were expressed as mean \pm standard deviation (SD) and compared using the Independent Samples t-test. Non-normally distributed variables were analyzed using the Mann–Whitney U test. Categorical data—including gender, comorbid hypertension and diabetes mellitus (DM), lipid levels, and lipid profile ratios—were assessed using the Chi-square test (8). Results are summarized in Table 1, Table 2, and Table 3. The sample included 62 male and 51 female patients, with an overall mean age of 60 ± 13 years. IS patients were significantly older than HS patients, while gender distribution was similar across both groups. Most patients in both groups presented with acute stroke, and the difference in acute versus recurrent stroke distribution was not statistically significant. DM was significantly more common in the IS group, whereas the prevalence of hypertension was high in both groups without a significant difference. Systolic and diastolic blood pressure values were generally higher in the HS group. However, due to the absence of p-values for these variables in the table, statistical significance could not be determined.

This table summarizes and compares demographic and clinical variables between IS and HS groups. Categorical variables are presented as counts and percentages, and continuous variables are reported as mean \pm SD. Group differences were evaluated using the appropriate statistical tests, with p-values indicating statistical significance. Significant differences were observed in age and DM comorbidity, while other variables such as gender, stroke type, and hypertension did not differ significantly between groups.

This study compared three primary serum lipid ratios—total cholesterol to HDL (TC/HDL), LDL to HDL (LDL/HDL), and triglycerides to HDL (TG/HDL)—between patients with IS and HS, involving 113 eligible patients (64 IS, 49 HS). The analysis showed no statistically significant differences in lipid ratios between the two groups ($p > 0.05$ for all comparisons). Although the mean values varied slightly—TC/HDL and LDL/HDL ratios were higher in IS, and TG/HDL was marginally higher in HS—these differences were not significant. Importantly, both IS and HS groups demonstrated mean TG/HDL ratios

Table 1. Comparative analysis of demographic and clinical characteristics between Ischemic Stroke (IS) and Hemorrhagic Stroke (HS) patients

Variable	Total (n=113)	IS (n=64)	HS (n=49)	Difference	p-value
Age (years), n (%)	60 ± 13	62 ± 13	57 ± 12	5	0.046**
Gender, n (%):					
Male	62 (54.9)	35 (54.7)	27 (55.1)	NA	0.965
Female	51 (45.1)	29 (45.6)	22 (44.9)	NA	
Stroke Type, n (%)					
Acute	93 (82.3)	50 (78.1)	43 (87.8)	NA	0.184
Recurrent	20 (17.7)	14 (21.9)	6 (12.2)	NA	
DM Comorbidity, n (%)					
DM	25 (22.1)	20 (31.3)	5 (10.2)	NA	0.008**
Without DM	93 (82.3)	44 (68.8)	44 (89.8)	NA	
Hypertension Comorbidity, n (%)					
Hypertension	93 (82.3)	53 (82.8)	40 (81.6)	NA	0.073
Normotension	20 (17.7)	11 (17.2)	9 (18.4)	NA	
SBP, mmHg (mean±SD)	153 ± 29	147 ± 28	161 ± 30	-14	0,116
DBP, mmHg (mean±SD)	90 ± 18	87 ± 16	93 ± 20	-6	

**P < 0.05

Table 2. Differences in serum lipid ratios between Ischemic Stroke (IS) and Hemorrhagic Stroke (HS) Patients

Ration Serum Lipids	IS (n=64) mean±SD	HS (n=49) mean±SD	Mean Difference	p-value
TC/HDL Ratio	6.13 ± 7.75	5.38 ± 2.55	0.75	0.926*
LDL/HDL Ratio	3.67 ± 4.00	3.35 ± 1.54	0.33	0.788*
TG/HDL Ratio	4.23 ± 2.90	4.35 ± 5.81	-0.12	0.166*

**P < 0.05

Table 3. Lipid profile parameters between Ischemic Stroke (IS) and Hemorrhagic Stroke (HS) patients

Profile Lipid	IS (n=64)		HS (n=49)		Mean Difference	p
	Mean	SD	Mean	SD		
TC (mg/dL)	205.52	140.96	186.06	55.48	19,46 0	0.317
TG (mg/dL)	141.80	74.29	127.49	86.23	13,31	0.356
LDL (mg/dL)	124.44	74.29	117.00	46.46	7,44	0.516
HDL (mg/dL)	38.27	12.76	38.08	12.92	0,19	0.94

**P < 0.05

above the commonly accepted atherogenic threshold (>3.0), suggesting a shared underlying lipid abnormality that may contribute to cerebrovascular risk. Elevated TG/HDL levels have been associated with

small dense LDL particles, which are particularly atherogenic and may contribute to endothelial dysfunction and atherosclerosis. These findings are consistent with prior research, including studies by Alkhaneen

et al. and Hasibuan & Thristy et al. (9,10), which reported no significant associations between stroke subtypes and lipid ratios. The lack of discriminative power for lipid ratios in distinguishing IS from HS in this cohort highlights the need for further research using larger sample sizes and prospective designs. Additional factors such as hypertension, smoking, metabolic syndrome, and lifestyle may need to be considered better to understand the complex role of dyslipidemia in stroke pathogenesis.

This section evaluated lipid profile parameters—including total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL)—in patients with IS and HS. While descriptive analysis showed slightly higher mean values for TC, TG, and LDL in the IS group and nearly identical HDL values between groups, none of these differences reached statistical significance ($p > 0.05$ for all). Detailed values and standard deviations are presented in Table 3. Of note, the high standard deviation in TC and LDL values among IS patients suggests a wide inter-individual variability. This variability and overlapping ranges complicate the potential for lipid parameters to distinguish between stroke subtypes. These results align with previous literature indicating that, although dyslipidemia is common among stroke patients, individual lipid markers—especially TG and LDL—may have limited discriminatory value between IS and HS. Overall, the lack of significant difference in serum lipid profiles between the two groups suggests that while dyslipidemia remains an important vascular risk factor, conventional lipid parameters alone may not serve as reliable biomarkers for distinguishing stroke subtypes. Nevertheless, consistently elevated lipid levels across both groups highlight the need for routine lipid monitoring and therapeutic management in stroke prevention strategies.

Discussion

This study compared serum lipid profiles and ratios in IS and HS patients. Parameters included TC, TG, LDL, HDL, and ratios such as TC/HDL, LDL/HDL, and TG/HDL. Dyslipidemia—characterized by elevated TC, TG, LDL, and low HDL—is a

well-established modifiable risk factor for stroke, and its pattern across different stroke subtypes may have implications for prevention and management (11). Although significant progress has been made in stroke prevention and management, stroke remains the second leading cause of death globally and a major contributor to long-term disability (11). Elevated LDL is a known contributor to atherosclerosis, which underlies most cases of IS (12). At the same time, emerging evidence suggests that extremely low LDL levels may increase the risk of HS in specific populations (13). TG and HDL also play roles in stroke risk (14), and statins, though effective for IS, must be used cautiously in HS due to bleeding concerns (15). Novel agents like PCSK9 inhibitors and other lipid-modifying therapies (16), alongside lifestyle modifications, are under investigation for broader stroke prevention (17). In our study, lipid profiles and ratios were slightly higher in the IS group than in the HS group; however, none of the differences were statistically significant ($p > 0.05$). These findings are consistent with previous research by Alkhaneen et al. and Hasibuan and Thristy et al., which reported similar trends (9,10). HDL cholesterol has anti-atherogenic characteristics that have a high ability to transport cholesterol from outside to the liver, reducing the atherosclerosis risk, which, in our IS patients, is an IS risk factor (18). Notably, both groups had TG/HDL ratios above the atherogenic threshold 3.0, suggesting a shared atherogenic profile. Elevated TG/HDL is associated with the small dense LDL particles, which are highly atherogenic and may contribute to endothelial dysfunction and vascular inflammation (19). The slightly higher LDL/HDL ratio observed in IS patients, though insignificant in the present study, aligns with previous studies identifying this ratio as a marker of pro-atherogenic lipid profiles (20). Atherosclerosis, the accumulation of plaque inside the arteries, is the primary cause of IS, which occurs when the blood flow to the brain is impaired. Although the original paper by Hasibuan and Thristy et al. appropriately focused on lipid profiles, the overall paradigm of risk for stroke comprises a constellation of factors (10, 21). New evidence reiterates what we already know; that the LDL/HDL ratio is crucial in overall cardiovascular health and the risk of stroke. For example, a report in the *Journal of Atherosclerosis and*

Thrombosis demonstrated a significant relationship between increased LDL/HDL ratio and first stroke incidence in a large group of middle-aged adults (22). The present study, however, further clarified the potential mechanisms and showed that LDL was associated with endothelial dysfunction and plaque development. In contrast, HDL was protective with reverse cholesterol transport and anti-inflammatory activities. In addition, studies over the past several years have highlighted specific lipid subfractions and stroke risk. The role of a subfraction of this lipoprotein in stroke risk has been addressed, for example, by Lee & Park et al., to other additional atherogenic factors possibly induce increased risk levels even when the total concentration of LDL would seem within the normal value (23). In the functional domain, aspects of the functionality of HDL, including cholesterol efflux, have recently been proposed as more informative for assessing CVD risk beyond HDL cholesterol levels alone (24). This study has several limitations. Its retrospective, single-center design and relatively small sample size may limit generalizability and reduce statistical power. The analysis relied on secondary data also did not capture key confounding variables such as smoking status, physical activity, diet, body mass index, or genetic risk factors. In addition, lipid subfractions and functional assays (e.g., HDL efflux capacity or LDL particle size) were not assessed, which could provide deeper insight into the pathophysiological mechanisms of stroke. Future studies with larger cohorts, prospective designs, and broader clinical profiling are needed to determine better the potential diagnostic and prognostic value of lipid markers in stroke classification.

Conclusion

This retrospective cross-sectional study demonstrated no statistically significant differences in serum lipid levels (TC, LDL, HDL, TG) and lipid profile ratios (TC/HDL, LDL/HDL, TG/HDL) between ischemic stroke (IS) and hemorrhagic stroke (HS) groups. Although all lipid parameters and ratios exhibited higher mean values in IS patients, these differences did not reach statistical significance. These findings suggest that conventional lipid markers have

limited discriminative utility for differentiating between stroke subtypes in clinical practice. However, the consistently elevated TG/HDL ratios observed in both groups may indicate a potential role in the atherogenic process, warranting further investigation. Future research should employ larger sample sizes, prospective cohort designs with extended follow-up, and inclusion of comprehensive vascular risk factors to better elucidate the involvement of dyslipidemia in stroke pathogenesis and its relevance in stroke classification and prevention.

Ethical Approval: This study was approved by the institutional review board and conducted in accordance with the principles of the Declaration of Helsinki. Ethical clearance was obtained from the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University, under protocol number UH23080679 and approval letter number 668/UN4.6.4.5.31/PP36/2023 and the approval year 2023. Additional site-specific approval was granted by RSUP Dr. Wahidin Sudirohusodo for the use of institutional data.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted article.

Authors' Contribution: MYA was involved in the conception, statistical analyses, and review, supervised the study, and reviewed the manuscript draft; MMP, ARMN, DSHP were involved in the collected data and prepared the initial draft and statistical analyses; SGAG was involved in the collected data, prepared the initial draft, and reviewed the manuscript draft.

Declaration on the Use of AI: None.

Data Availability Statement: The data presented in this study are available on request from the corresponding author [MYA].

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Abbreviations

DALYs: Disability-adjusted life-years
HS: Hemorrhagic stroke

IS: Ischemic stroke
HDL: High-density lipoprotein
LDL: Low-density lipoprotein
CT: Computed tomography
MRI: Magnetic resonance imaging
C-DSA: Cerebral digital subtraction angiography
TG: Triglycerides
SPSS: Statistical Package for the Social Sciences
SBP: Systolic blood pressure
DBP: Diastolic blood pressure
TC/HDL: Total Cholesterol to High-Density Lipoprotein ratio
LDL/HDL: Low-Density Lipoprotein to High-Density Lipoprotein ratio
TG/HDL: Triglycerides to High-Density Lipoprotein ratio

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