



ASSOCIATION BETWEEN A HISTORY OF DYSLIPIDEMIA AND CAROTID INTIMA-MEDIA THICKNESS IN PATIENTS WITH ISCHEMIC STROKE: A CROSS-SECTIONAL STUDY AT GATOT SOEBROTO ARMY HOSPITAL

¹Christian Hasudungan Nainggolan, ¹Muhammad Irvanie Rama Harahap, ¹Samuel Tandionugroho, ¹Vininta Fazharyasti, ¹Renny Farida Hutaauruk, ¹Terawan Agus Putranto

¹ Department of Radiology and Neurology, Faculty of Medicine, Universitas Pembangunan Nasional Veteran Jakarta, Jakarta, Indonesia
/ Gatot Soebroto Army Hospital, Jakarta, Indonesia

Corresponding E-mail: chyll_s@yahoo.com,
muhammad.irvanie.harahap@upnvj.ac.id

Article History :

Received date : 2026/06/21
Revised date : 2026/07/19
Accepted date : 2026/08/23
Published date : 2026/09/28



Copyright: © 2024 by the authors. Submitted for possible open access publication under the terms and conditions of the Creative Commons Attribution (BY NC) license (<https://creativecommons.org/licenses/by-nc/4.0/>).

E-ISSN :

ISSN 3048-1368



P-ISSN

ISSN 3048-1376



ABSTRACT

Background

Dyslipidemia is an important modifiable risk factor for atherosclerosis and ischemic stroke. Carotid intima-media thickness (CIMT), assessed using carotid Doppler ultrasonography, is a non-invasive marker of subclinical vascular wall changes and may reflect the burden of atherosclerotic disease. However, the association between a history of dyslipidemia and CIMT among patients with ischemic stroke remains inconsistent across populations.

Objective

This study aimed to evaluate the association between a history of dyslipidemia and carotid intima-media thickness assessed by Doppler ultrasonography in patients with ischemic stroke at Gatot Soebroto Army Hospital.

Methods

This observational analytic study used a cross-sectional design. Consecutive sampling was performed among patients with ischemic stroke who attended the Neurology outpatient clinic of Gatot Soebroto Army Hospital between July and August 2026. Patients aged ≥ 40 years with documented ischemic stroke, available lipid-profile data, and carotid Doppler ultrasonography were included. A history of dyslipidemia was determined from medical records and/or lipid-profile results. CIMT was assessed using carotid Doppler ultrasonography and categorized as ≤ 1 mm or > 1 mm. Because one cell in the 2×2 contingency table had an expected count below 5, Fisher's exact test was used. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated.

Results

A total of 38 patients were included. The mean age was 56.81 ± 8.35 years, and 21 patients (55.3%) were male. A history of dyslipidemia was identified in 27 patients (71.1%) according to the study's categorical definition. Increased LDL levels were found in 23 patients (65.8%). Thickened CIMT (> 1 mm) was observed in 17 of 27 patients (63.0%) with a history of dyslipidemia and in 8 of 11 patients (72.7%) without a history of dyslipidemia. Fisher's exact test demonstrated no statistically significant association between a history of dyslipidemia and CIMT category ($p = 0.714$). The OR was 0.638 (95% CI, 0.137–2.973).

Conclusion

In this study population of patients with ischemic stroke, no statistically significant association was found between a history of dyslipidemia and carotid intima-media thickness. The findings do not exclude the biological role of dyslipidemia in atherosclerosis,

but suggest that CIMT is influenced by multiple vascular and methodological factors. Larger studies using quantitative lipid measurements, standardized CIMT protocols, and adjustment for potential confounders are warranted.

Keywords: ischemic stroke; dyslipidemia; carotid intima-media thickness; carotid Doppler ultrasonography; atherosclerosis

INTRODUCTION

Stroke is one of the leading causes of death and long-term disability worldwide. Ischemic stroke accounts for the majority of stroke cases and results from cerebral arterial occlusion, leading to impaired cerebral perfusion and tissue injury. Atherosclerosis is an important underlying mechanism of ischemic stroke, particularly in large-artery atherosclerotic disease.

Dyslipidemia is an established modifiable vascular risk factor. Abnormal lipid metabolism, particularly elevated low-density lipoprotein cholesterol (LDL-C), elevated triglycerides, and reduced high-density lipoprotein cholesterol (HDL-C), contributes to endothelial dysfunction, lipid accumulation, vascular inflammation, and atherosclerotic plaque formation.

The carotid arteries are an important site for evaluating systemic atherosclerotic changes. Carotid intima-media thickness (CIMT) can be measured non-invasively using high-resolution ultrasonography. Increased CIMT has been widely investigated as a marker of subclinical atherosclerosis and has been associated with future cerebrovascular and cardiovascular events.

Carotid Doppler ultrasonography provides several advantages, including non-invasive assessment, lack of ionizing radiation, relatively low cost, and the ability to evaluate vascular morphology and hemodynamics. In addition to plaque and stenosis assessment, CIMT measurement may provide information regarding structural changes in the arterial wall.

Although dyslipidemia is biologically associated with atherosclerotic disease, previous studies have reported variable relationships between lipid abnormalities and CIMT. Differences in patient characteristics, age, concomitant vascular risk factors, lipid-lowering therapy, definitions of dyslipidemia, and CIMT measurement techniques may contribute to these differences.

In patients who have already experienced ischemic stroke, the burden of vascular risk factors may be substantial. Consequently, the independent contribution of a history of dyslipidemia to carotid wall thickening may be difficult to demonstrate. Furthermore, a history of dyslipidemia does not necessarily reflect the patient's current lipid exposure because patients may have received lipid-lowering therapy before CIMT assessment.

Therefore, this study evaluated the association between a history of dyslipidemia and carotid intima-media thickness in patients with ischemic stroke undergoing carotid Doppler ultrasonography at Gatot Soebroto Army Hospital.

MATERIAL AND METHODS

Study Design and Setting

This was an observational analytic study with a cross-sectional design. The study was conducted at the Neurology Outpatient Clinic and Department of Radiology of Gatot Soebroto Army Hospital, Jakarta, Indonesia, between July and August 2026.

The study was designed to evaluate whether patients with ischemic stroke and a history of dyslipidemia were more likely to have increased carotid intima-media thickness than patients without a history of dyslipidemia.

Study Population

The target population consisted of patients diagnosed with ischemic stroke who attended the Neurology outpatient clinic and underwent carotid Doppler ultrasonography.

Patients were selected using consecutive sampling. A total of 38 eligible subjects were included in the final analysis.

Eligibility Criteria

The inclusion criteria were:

1. Male or female patients.
2. Age ≥ 40 years.
3. Diagnosis of ischemic stroke.
4. Availability of clinical and radiological documentation supporting the diagnosis of ischemic stroke.
5. Availability of lipid-profile results in the medical record.
6. Willingness to participate and provision of informed consent.

Patients were excluded when medical-record data were incomplete, when they had hemorrhagic stroke, had no previous lipid-profile examination, had carotid Doppler findings that did not meet the study's imaging standards, or had died during the study period.

Study Variables

The primary independent variable was a history of dyslipidemia, categorized as present or absent.

The dependent variable was carotid intima-media thickness measured using carotid Doppler ultrasonography and categorized into:

- ≤ 1 mm
- 1 mm

Potential confounding variables identified in the study protocol included age, sex, family history of stroke, and lipid-lowering therapy.

Definition of Dyslipidemia

A history of dyslipidemia was defined as a previous diagnosis of lipid metabolism abnormality documented by a physician and/or lipid-profile findings meeting the study criteria.

The study criteria included:

- Total cholesterol ≥ 200 mg/dL
- LDL-C ≥ 100 mg/dL
- Triglycerides ≥ 160 mg/dL
- HDL-C < 35 mg/dL

Patients who did not meet any of these criteria were categorized as having no history of dyslipidemia according to the study definition.

Carotid Doppler Ultrasonography

Carotid ultrasonography was performed using a RESONA I9 ultrasound system (Mindray). CIMT measurements were obtained from longitudinal images of the carotid artery, with assessment of the far wall and visualization of the characteristic double-line appearance.

The study protocol included assessment of the distal common carotid artery approximately 1 cm proximal to the carotid bulb and the proximal internal carotid artery approximately 1 cm distal to the bifurcation.

CIMT was categorized as ≤ 1 mm or > 1 mm according to the operational definition used in the study.

All CIMT evaluations were performed by a radiologist with more than five years of experience. Lipid-profile data were reviewed by an experienced neurologist.

Statistical Analysis

Descriptive analysis was performed to summarize demographic and clinical characteristics. Categorical variables were presented as frequencies and percentages, whereas continuous variables were summarized using means and standard deviations.

The association between a history of dyslipidemia and CIMT category was initially considered using the chi-square test. Because one cell in the 2×2 contingency table had an expected count below 5, Fisher's exact test was used for the primary analysis.

The level of statistical significance was set at $\alpha = 0.05$. An OR and 95% CI were calculated to estimate the magnitude of association. All statistical analyses were performed using IBM SPSS Statistics version 25.

Ethical Considerations

The study received institutional permission and ethical approval in accordance with the requirements of Gatot Soebroto Army Hospital and Universitas Pembangunan Nasional Veteran Jakarta. Patient confidentiality was maintained by using coded research data rather than directly identifying information.

RESULTS

Participant Characteristics

During the study period, 46 patients with available lipid-profile data were initially identified. After application of the study criteria, 38 subjects were included in the final analysis.

The mean age was 56.81 ± 8.35 years, with an age range of 40–76 years. The largest age group was 51–60 years, comprising 17 patients (44.7%). Twenty-one patients (55.3%) were male and 17 (44.7%) were female.

Table 1. Demographic Characteristics of the Study Population

Characteristic	n (%) or Mean $\pm$ SD
Age, years	56.81 ± 8.35
40–50 years	8 (21.1)
51–60 years	17 (44.7)
61–70 years	11 (28.9)
71–80 years	2 (5.2)
Male	21 (55.3)
Female	17 (44.7)

Lipid Profile

The mean total cholesterol level was 195.31 ± 46.69 mg/dL. The mean LDL-C level was 114.23 ± 36.74 mg/dL. The mean triglyceride level was 160.00 ± 217.70 mg/dL, while the mean HDL-C level was 45.89 ± 8.65 mg/dL.

Increased LDL-C was identified in 23 patients (65.8%), whereas 15 patients (34.2%) had LDL-C within the study-defined normal range.

Table 2. Lipid Profile of the Study Population

Lipid parameter	Mean $\pm$ SD (mg/dL)	Range (mg/dL)
Total cholesterol	195.31 ± 46.69	132–390
LDL-C	114.23 ± 36.74	64–207
Triglycerides	160.00 ± 217.70	50–1426
HDL-C	45.89 ± 8.65	32–66

Other Vascular Risk Characteristics

A family history of ischemic stroke was reported in 18 patients (47.4%), whereas 20 patients (52.6%) had no reported family history.

Twenty-seven patients (71.1%) had a history of lipid-lowering therapy, while 11 patients (28.9%) had no documented history of dyslipidemia therapy.

Carotid Intima-Media Thickness

The mean CIMT reported in the study was 0.94 ± 0.52 mm. When categorized according to the study cutoff, CIMT >1 mm was found in 17 of 27 patients (63.0%) with a history of dyslipidemia and in 8 of 11 patients (72.7%) without a history of dyslipidemia.

Table 3. Association Between History of Dyslipidemia and CIMT Category

History of dyslipidemia	CIMT ≤ 1 mm, n (%)	CIMT >1 mm, n (%)	Total
Present	10 (37.0)	17 (63.0)	27
Absent	3 (27.3)	8 (72.7)	11
Total	13	25	38

Fisher's exact test demonstrated no statistically significant association between a history of dyslipidemia and CIMT category ($p = 0.714$).

The estimated OR was 0.638 (95% CI, 0.137–2.973). Because the 95% CI included 1.0, the estimated association was not statistically significant.

LDL-C and CIMT

In the analysis of LDL-C and CIMT, 16 of 23 patients (69.6%) with increased LDL-C had CIMT >1 mm, compared with 9 of 15 patients (60.0%) with normal LDL-C.

The prevalence ratio was 1.16, while the Pearson chi-square test demonstrated no statistically significant association between LDL-C category and CIMT ($p = 0.544$).

Table 4. Association Between LDL-C Category and CIMT

LDL-C category	CIMT ≤ 1 mm, n (%)	CIMT >1 mm, n (%)	PR	p-value
Increased	7	16 (69.6)	1.16	0.544
Normal	6	9 (60.0)	—	—

DISCUSSION

This study evaluated the association between a history of dyslipidemia and carotid intima-media thickness in patients with ischemic stroke. The main finding was that no statistically significant association was identified between a history of dyslipidemia and CIMT category based on Fisher's exact test ($p = 0.714$).

The absence of statistical significance should not be interpreted as evidence that dyslipidemia has no biological role in atherosclerosis. Dyslipidemia remains an important component of the atherosclerotic process. Elevated LDL-C and other lipid abnormalities can promote endothelial dysfunction, lipid deposition, inflammation, and vascular remodeling. These mechanisms provide a biological basis for an association between lipid abnormalities and carotid wall changes.

The findings of this study may instead reflect the way dyslipidemia and CIMT were defined and measured. In this study, dyslipidemia was represented by a categorical history of dyslipidemia rather than by the magnitude or duration of lipid exposure. Patients with a previous diagnosis of dyslipidemia may have subsequently achieved controlled lipid levels following lifestyle modification or pharmacological treatment. Therefore, a binary classification of "history present" versus "history absent" may not adequately represent cumulative lipid exposure.

The high proportion of patients receiving lipid-lowering therapy is particularly relevant. In this study, 71.1% of patients had a documented history of dyslipidemia therapy. Lipid-lowering treatment, particularly statin therapy, may modify the progression of atherosclerotic disease and vascular wall changes. Consequently, patients with a history of dyslipidemia who have received treatment may demonstrate different CIMT characteristics compared with untreated patients.

Age is another important consideration. The study population had a mean age of 56.81 years, and vascular wall remodeling generally progresses with increasing age. Other vascular risk factors, including hypertension, diabetes mellitus, smoking, obesity, and genetic susceptibility, may also influence CIMT. If several of these factors are prevalent in patients with ischemic stroke, the

specific contribution of dyslipidemia may be difficult to distinguish using a small cross-sectional sample.

The secondary analysis of LDL-C showed a similar pattern. CIMT >1 mm was observed in 69.6% of patients with increased LDL-C compared with 60.0% of those with normal LDL-C. The prevalence ratio was 1.16, suggesting a higher observed prevalence of increased CIMT in the elevated-LDL group. However, the difference was not statistically significant ($p = 0.544$).

This distinction between descriptive differences and statistical significance is important. A higher proportion of increased CIMT in the elevated-LDL group may indicate a possible biological trend, but the current sample does not provide sufficient statistical evidence to establish an association.

The OR for the primary analysis was 0.638, with a wide 95% CI of 0.137–2.973. Although the point estimate was below 1, the confidence interval crossed 1. Therefore, the OR should not be interpreted as demonstrating a protective effect of dyslipidemia. Rather, the estimate was imprecise and compatible with different directions and magnitudes of association.

The wide confidence interval is likely related, at least in part, to the relatively small sample size. With only 38 subjects, the statistical power to detect a modest association is limited. A larger study population would provide narrower confidence intervals and a greater ability to distinguish between a true absence of association and a failure to detect an existing association.

Another important methodological consideration is the categorization of CIMT. Converting a continuous measurement into two categories, ≤ 1 mm and >1 mm, results in loss of information. For example, individuals with CIMT values just above the cutoff and those with substantially greater CIMT may be placed into the same category despite potentially meaningful biological differences. Future studies may therefore benefit from analyzing CIMT as a continuous variable.

Measurement methodology may also contribute to variability. CIMT can vary according to the carotid segment examined, right or left side, measurement location, acquisition technique, and whether mean or maximum CIMT is used. Standardized acquisition and measurement protocols are therefore important for improving reproducibility.

The present findings are consistent with the concept that CIMT is a multifactorial marker of vascular remodeling rather than a specific marker of dyslipidemia. The structural characteristics of the carotid arterial wall may reflect the cumulative effects of age, lipid exposure, blood pressure, glucose metabolism, smoking, genetic factors, medication, and other vascular conditions.

The study also has clinical implications. Carotid Doppler ultrasonography remains a useful non-invasive modality for evaluating carotid vascular morphology, plaque, stenosis, and CIMT. However, CIMT should not be interpreted in isolation as evidence of dyslipidemia or as a direct surrogate for a single cardiovascular risk factor. Rather, it should be considered within the broader clinical and vascular risk profile of the patient.

LIMITATIONS

This study has several limitations.

First, the sample size was relatively small, with only 38 participants. This may have limited the statistical power to detect modest associations and contributed to the wide confidence interval.

Second, the cross-sectional design limits the ability to establish temporal relationships between dyslipidemia and CIMT. Therefore, causal conclusions cannot be drawn.

Third, dyslipidemia was evaluated as a categorical history rather than using quantitative measures of cumulative lipid exposure or duration of dyslipidemia.

Fourth, a substantial proportion of participants had a history of lipid-lowering therapy, which may have modified their current lipid levels and vascular characteristics.

Fifth, several potential confounding factors, including smoking, physical activity, obesity, diabetes mellitus, and hypertension, were not fully evaluated in the final analysis.

Finally, CIMT was categorized into two groups, which may have resulted in loss of information compared with analysis of CIMT as a continuous variable.

CONCLUSION

In this cross-sectional study of patients with ischemic stroke at Gatot Soebroto Army Hospital, no statistically significant association was found between a history of dyslipidemia and carotid intima-media thickness assessed using Doppler ultrasonography.

Although increased CIMT was observed in a substantial proportion of patients with and without a history of dyslipidemia, the difference did not reach statistical significance. Similarly, increased LDL-C was associated with a descriptively higher prevalence of CIMT >1 mm, but this association was not statistically significant.

These findings do not indicate that dyslipidemia is unrelated to atherosclerosis. Rather, they suggest that the relationship between lipid abnormalities and CIMT may be influenced by age, other vascular risk factors, lipid-lowering therapy, duration of lipid exposure, sample size, and CIMT measurement methodology.

Future studies should include larger sample sizes, standardized CIMT measurement protocols, quantitative lipid measurements, assessment of treatment duration, and multivariable adjustment for relevant cardiovascular risk factors.

DECLARATIONS

Ethics Approval and Consent to Participate

The study was conducted following approval from the relevant institutional ethics committee and hospital authorities. Written informed consent was obtained from participants according to the study protocol.

Consent for Publication

Not applicable.

Competing Interests

The author declares no competing interests.

Funding

No external funding was reported for this study.

Author Contributions

The author was responsible for study conception, data collection, data analysis, interpretation of findings, and preparation of the manuscript.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request and subject to applicable institutional and ethical restrictions.

Acknowledgments

The author would like to acknowledge the supervisors, Department of Radiology, Department of Neurology, and staff of Gatot Soebroto Army Hospital and Universitas Pembangunan Nasional Veteran Jakarta for their support during the study.

REFERENCES

1. Salonen R, Salonen JT. Determinants of carotid intima-media thickness: A population-based ultrasonography study in eastern Finnish men. *J Intern Med*, 1991; 229: 225-231
2. Burke GL, Evans GW, Riley WA, Sharrett AR, Howard G, Barnes RW, Rosamond W, Crow RS, Rautaharju PM, Heiss G. Arterial wall thickness is associated with prevalent cardiovascular disease in middle-aged adults. The Atherosclerosis Risk in Communities (ARIC) study. *Stroke*, 1995; 26: 386-391
3. Grobbee DE, Bots ML. Carotid artery intima-media thickness as an indicator of generalized atherosclerosis. *J Intern Med*, 1994; 236: 567-573
4. Howard G, Sharrett AR, Heiss G, Evans GW, Chambless LE, Riley WA, Burke GL. Carotid artery intima-media thickness distribution in general populations as evaluated by B-mode ultrasound. ARIC investigators. *Stroke*, 1993; 24: 1297-1304
5. Cao JJ, Arnold AM, Manolio TA, Polak JF, Psaty BM, Hirsch CH, Kuller LH, Cushman M. Association of carotid artery intima-media thickness, plaques, and C-reactive protein with future cardiovascular disease and all-cause mortality: The Cardiovascular Health Study. *Circulation*, 2007; 116: 32-38

6. Chambless LE, Folsom AR, Clegg LX, Sharrett AR, Shahar E, Nieto FJ, Rosamond WD, Evans G. Carotid wall thickness is predictive of incident clinical stroke: The Atherosclerosis Risk in Communities (ARIC) study. *Am J Epidemiol*, 2000; 151: 478-487
7. Iglesias del Sol A, Bots ML, Grobbee DE, Hofman A, Witteman JC. Carotid intima-media thickness at different sites: Relation to incident myocardial infarction; The Rotterdam study. *Eur Heart J*, 2002; 23: 934-940
8. Rosvall M, Janzon L, Berglund G, Engström G, Hedblad B. Incidence of stroke is related to carotid IMT even in the absence of plaque. *Atherosclerosis*, 2005; 179: 325-331
9. Lorenz MW, von Kegler S, Steinmetz H, Markus HS, Sitzer M. Carotid intima-media thickening indicates a higher vascular risk across a wide age range: Prospective data from the Carotid Atherosclerosis Progression Study (CAPS). *Stroke*, 2006; 37: 87-92
10. van den Oord SC, Sijbrands EJ, ten Kate GL, van Klaveren D, van Domburg RT, van der Steen AF, Schinkel AF. Carotid intima-media thickness for cardiovascular risk assessment: Systematic review and meta-analysis. *Atherosclerosis*, 2013; 228: 1-11
11. Den Ruijter HM, Peters SA, Anderson TJ, Britton AR, Dekker JM, Eijkemans MJ, Engström G, Evans GW, de Graaf J, Grobbee DE, Hedblad B, Hofman A, Holewijn S, Ikeda A, Kavousi M, Kitagawa K, Kitamura A, Koffijberg H, Lonn EM, Lorenz MW, Mathiesen EB, Nijpels G, Okazaki S, O'Leary DH, Polak JF, Price JF, Robertson C, Rembold CM, Rosvall M, Rundek T, Salonen JT, Sitzer M, Stehouwer CD, Witteman JC, Moons KG, Bots ML. Common carotid intima-media thickness measurements in cardiovascular risk prediction: A metaanalysis. *JAMA*, 2012; 308: 796-803
12. Goff DC, Jr, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Sr, Gibbons R, Greenland P, Lackland DT, Levy D, O'Donnell CJ, Robinson JG, Schwartz JS, Shero ST, Smith SC, Jr, Sorlie P, Stone NJ, Wilson PWF. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*, 2014; 63: 2935-2959

13. LeFevre ML, U.S. Preventive Services Task Force. Screening for asymptomatic carotid artery stenosis : U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*, 2014; 161: 356-362
14. Onut R, Balanescu AP, Constantinescu D, Calmac L, Marinescu M, Dorobantu PM. Imaging Atherosclerosis by Carotid Intima-media Thickness in vivo: How to, Where and in Whom? *Maedica (Buchar)*, 2012; 7: 153-162
15. Stein JH, Korcarz CE, Hurst RT, Lonn E, Kendall CB, Mohler ER, Najjar SS, Rembold CM, Post WS, American Society of Echocardiography Carotid Intima-Media Thickness Task Force Use of carotid ultrasound to identify subclinical vascular disease and evaluate cardiovascular disease risk: A consensus statement from the American Society of Echocardiography Carotid Intima-Media Thickness Task Force. Endorsed by the Society for Vascular Medicine. *J Am Soc Echocardiogr*, 2008; 21: 93-111; quiz 189–190