ORIGINAL ARTICLE

Atherogenic index of plasma as a stroke-subtype–independent predictor of acute cognitive dysfunction in ischemic stroke: A cross-sectional analytic study.

DAVID CHRISTIAN HAURISSA[1], MUHAMMAD YUNUS AMRAN[2,3,4], MUHAMMAD AKBAR[1,4], ABDUL MUIS[1,3], YUDY GOYSAL[1,3], ANDI ALFIAN ZAINUDDIN[5]

[1]Department of Neurology, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia; [2]Division of Interventional Neurology and Neuroendovascular Therapy, Department of Neurology, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia; [3]Brain Centre, Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia; [4]Hasanuddin University Teaching Hospital, Makassar, Indonesia; [5]Department of Public Health and Community Medicine, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia.

ABSTRACT

Background and aim: Cognitive impairment is a common complication in the acute phase of ischaemic stroke (IS). The Atherogenic Index of Plasma (AIP) has been proposed as a potential marker of vascular risk; however, its relationship with cognitive function across different stroke subtypes remains unclear. This study aimed to evaluate the association between AIP and cognitive performance, with consideration of IS subtypes, using the Montreal Cognitive Assessment Indonesian Version (MoCA-Ina).

Methods: A cross-sectional analytic study was conducted at Dr. Wahidin Sudirohusodo General Hospital, Makassar, from July to December 2025. Patients aged ≥40 years with first-ever acute ischaemic stroke (onset 24 hours–7 days) and preserved consciousness were included. Exclusion criteria comprised recurrent stroke, transient ischaemic attack, aphasia, pre-existing dementia, systemic or psychiatric conditions affecting cognition or lipid metabolism, and prior lipid-lowering therapy. AIP was calculated as $\log_{10}$ (triglyceride/HDL-C). Cognitive function was assessed using MoCA-Ina, and stroke subtype was classified as lacunar or non-lacunar. Statistical analyses included Spearman correlation, Mann–Whitney, and independent t-tests.

Results: Most patients showed cognitive impairment (74%; median MoCA-Ina 23.00) and high-risk AIP levels (65%; mean 0.285). AIP demonstrated a significant negative correlation with total MoCA-Ina score

Received: 12 March 2026 | Accepted: 12 April 2026

Correspondence: Muhammad Yunus Amran, M.D., Ph.D., FIPM, FINR, FINA / NEUROLOGIST & CONSULTANT OF NEURO-INTERVENTIONIST, Division of Interventional Neurology and Neuroendovascular Therapy, Department of Neurology, Faculty of Medicine, Hasanuddin University, Brain Centre, Dr. Wahidin Sudirohusodo General Hospital, and Hasanuddin University Teaching Hospital, Jl. Perintis Kemerdekaan KM 11, 90245, Makassar, South Sulawesi, Indonesia. / Email: muhyunusamran@med.unhas.ac.id ; yunusamran10@gmail.com
ORCID: 0000-0001-5079-7490

(r = -0.342; p = 0.001). Non-lacunar stroke patients showed lower cognitive scores compared with lacunar stroke (mean 19.02 vs 21.90; p = 0.028). In contrast, AIP values did not differ between subtypes (p = 0.991). **Conclusions:** Higher AIP is associated with worse cognitive performance in the acute phase of ischaemic stroke. This finding suggests that atherogenic dyslipidaemia may contribute to early cognitive decline irrespective of stroke subtype. (www.actabiomedica.it)

Key words: atherogenic index of plasma, cognitive function, ischemic stroke subtype, ischemic stroke

Introduction

Stroke is a leading cause of mortality and long-term disability worldwide, with post-stroke cognitive impairment (PSCI) emerging as a significant acute-phase complication that is often overlooked by clinicians (1,2). Cognitive deficits, which encompass the domains of memory, executive function, attention, and visuospatial abilities, significantly impair the independence and quality of life of stroke survivors (3–5). Although vascular cognitive impairment (VCI) can arise from various cerebrovascular etiologies, including large artery atherosclerosis (non-lacunar) and small vessel occlusion (lacunar), the pathophysiological mechanisms driving cognitive decline differ significantly between these two subtypes (6,7). Small vessel occlusion (SVO), which clinically manifests as lacunar stroke, reflects microvascular pathology that can strategically disrupt neural circuits despite having a minimal infarct volume of less than 20 mm (8,9). In contrast, non-lacunar strokes are dominated by macrovascular atherosclerosis, leading to extensive territorial infarcts (6,10). Dyslipidemia is a well-established risk factor for both ischemic stroke (IS) and associated cognitive impairment, primarily through the acceleration of intracranial and extracranial atherosclerotic processes (11,12). However, traditional lipid parameters often exhibit limited predictive value regarding stroke outcomes (13). Recent studies indicate that the Atherogenic Index of Plasma (AIP)—calculated as the logarithm of the triglyceride-to-high-density lipoprotein cholesterol ratio [log(TG/HDL-C)]—has emerged as a superior and more comprehensive biomarker for assessing cardiometabolic burden and systemic atherosclerosis (14–16). A high atherogenic burden and subclinical atherosclerosis can trigger chronic cerebral hypoperfusion, endothelial dysfunction, and white matter injury, which ultimately contribute to brain network dysfunction (17–19). Nevertheless, there remains very limited empirical evidence mapping the specific relationship between elevated AIP levels, stroke subtypes (lacunar vs. non-lacunar), and cognitive outcomes in the acute phase of IS.

Therefore, this study aims to evaluate the impact of the AIP and IS classification—specifically the differences between lacunar and non-lacunar subtypes—on cognitive function in patients with acute ischemic stroke (AIS). By elucidating these metabolic and topographic determinants, this study seeks to identify early predictors of PSCI, thereby facilitating more targeted neurobehavioral management and secondary prevention strategies in acute-phase stroke care.

Materials and Methods

Study design and setting

This study was an analytic observational study using a cross-sectional design conducted at Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia, a tertiary referral center for neurological disorders. The study was carried out between July and December 2025 and included patients diagnosed with AIS. Clinical and laboratory data were obtained from medical records; while neuroimaging findings were reviewed through the hospital Picture Archiving and Communication System (PACS) to ensure diagnostic accuracy.

The primary objective was to evaluate the relationship between the atherogenic index of plasma (AIP), ischemic stroke subtypes (lacunar and non-lacunar), and cognitive function assessed during the acute phase of stroke. Ethical approval was obtained from the Health Research Ethics Committee of Dr. Wahidin Sudirohusodo General Hospital, Makassar (Approval No. 32/UN4.6.4.5.31/PP36/2025). As this study used anonymized clinical data, patient confidentiality was strictly maintained throughout the research process.

Sample criteria

Participants were recruited consecutively from patients admitted with AIS. Eligible subjects were adults aged ≥40 years with a first-ever IS confirmed clinically and radiologically, with onset between >24 hours and ≤7 days. Only patients with preserved consciousness Glasgow Coma Scale (GCS) score of 15 and the ability to communicate in Indonesian were included to allow reliable cognitive assessment. Patients were excluded if they had a history of transient ischemic attack (TIA), recurrent stroke, aphasia that interfered with cognitive testing, pre-existing dementia, psychiatric disorders, or systemic conditions known to affect cognition or lipid metabolism. In addition, patients who had received lipid-lowering therapy or cognitive-enhancing medications prior to admission were excluded to minimize confounding effects. Cases with incomplete clinical, laboratory, or cognitive assessment data were also excluded from the analysis.

Research procedure

Eligible patients were identified through hospital admission records and confirmed by clinical evaluation and neuroimaging findings. Baseline demographic data, including age and sex, as well as vascular risk factors and comorbidities, were recorded. Cognitive function was assessed during hospitalization using the Montreal Cognitive Assessment Indonesian version (MoCA-Ina), a validated screening tool for detecting mild cognitive impairment and vascular cognitive deficits (20). The total MoCA-Ina score was used to represent global cognitive performance, while individual domain scores were also documented for further analysis. Laboratory data, including triglyceride and high-density lipoprotein cholesterol (HDL-C) levels, were obtained from fasting blood samples collected at admission using standardized automated laboratory methods. The AIP was calculated using the formula $\log_{10}$ (triglyceride/HDL-C), which reflects the balance between atherogenic and protective lipoproteins and has been associated with vascular outcomes in IS (21). All collected data were coded and entered into a secure database prior to statistical analysis to ensure data integrity and confidentiality.

Stroke subtype classification

IS was classified into lacunar and non-lacunar subtypes based on clinical presentation and neuroimaging findings in accordance with established stroke classification principles (22). Lacunar stroke was defined as a small subcortical infarction consistent with small vessel occlusion, whereas non-lacunar stroke included infarctions related to large artery atherosclerosis or other non-small vessel mechanisms. Neuroimaging assessment was performed using CT or MRI as available, with lesion location and vascular territory taken into consideration are shown in figure 1.

(A, B) Brain CT (A) and MRI (B) show a large infarct area measuring >1.5 cm, involving both cortical and subcortical regions, consistent with a non-lacunar infarct subtype; (C, D) Brain CT (C) and diffusion-weighted MRI (DWI) (D) demonstrate small infarcts measuring <1.5 cm. The CT image (C) shows a lesion in the left thalamus, while the DWI image (D) reveals a lesion in the left periventricular region. These findings are consistent with the lacunar infarct subtype.

All statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Data distribution was assessed using the Kolmogorov–Smirnov test. Continuous variables were presented as mean ± standard deviation for normally distributed data and median (range) for non-normally distributed data, while categorical variables were expressed as frequencies and percentages. Because several variables, including MoCA-Ina scores and AIP values, were not normally distributed, non-parametric tests were primarily applied. The association between AIP and cognitive function was analysed using Spearman's

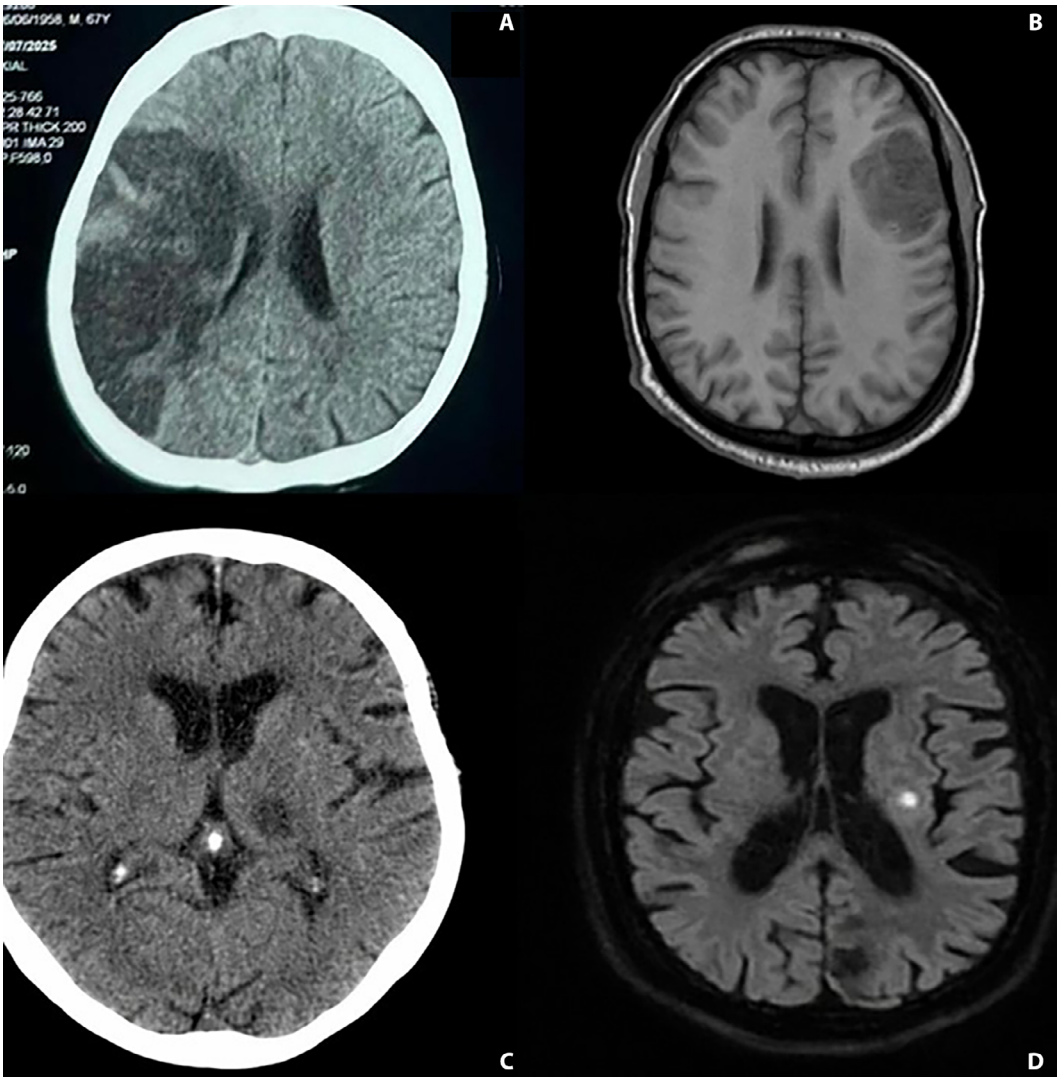

Figure 1. Brain CT and MRI findings demonstrating lacunar and non-lacunar infarct subtypes.

rank correlation. Differences in cognitive scores between stroke subtypes were assessed using the Mann–Whitney test. The independent t-test was used for comparisons of normally distributed variables where appropriate. A two-tailed p-value <0.05 was considered statistically significant.

Results

Baseline characteristics of the study population

This study included 131 patients with AIS who fulfilled all inclusion criteria. The baseline demographic and clinical characteristics of the study population are presented in Table 1. The majority of patients were male (57.3%), with a mean age of 59.5 ± 9.82 years. Regarding vascular risk factors, hypertension combined with dyslipidemia was the most common comorbidity (48.1%), followed by diabetes mellitus with hypertension (9.2%) and diabetes mellitus with dyslipidemia (7.6%). In terms of stroke characteristics, lacunar stroke was more frequent than non-lacunar stroke (63.4% vs. 36.6%). Lesions were predominantly located in the anterior circulation (85.0%), while posterior circulation involvement was observed in 15.0% of cases. Cognitive assessment using MoCA-Ina showed that 97 patients (74.0%) had cognitive impairment,

Table 1. Baseline demographic, clinical, and laboratory characteristics of patients with acute ischemic stroke (n = 131)

Variable	n (%) or mean ± SD / Median (Range)
Age (years), Mean ± SD	59.5 ± 9.82
Sex	
Male	75 (57.3)
Female	56 (42.7)
Major comorbidities	
Hypertension & dyslipidemia	63 (48.1)
Diabetes mellitus & hypertension	12 (9.2)
Diabetes mellitus & dyslipidemia	10 (7.6)
Stroke subtype	
Lacunar	83 (63.4)
Non-lacunar	48 (36.6)
Lesion location	
Anterior circulation	111 (85.0)
Posterior circulation	20 (15.0)
Cognitive status (MoCA-Ina)	
Cognitive impairment	97 (74.0)
Normal	34 (26.0)
MoCA-Ina score, Median (Range)	23.00 (0–30)
Lipid profile & atherogenic index	
Triglyceride (mg/dL), Median (Range)	132 (59–631)
HDL (mg/dL), Median (Range)	39.2 (10–110)
LDL (mg/dL), Mean ± SD	144 ± 53.75
Total cholesterol (mg/dL), Mean ± SD	209 ± 52.5
AIP (high risk), n (%)	85 (65.0)

Data are presented as mean ± standard deviation (SD) for normally distributed variables and median (range) for non-normally distributed variables. Categorical variables are presented as frequencies and percentages. *Abbreviations:* MoCA-Ina = Montreal Cognitive Assessment Indonesian version. AIP (atherogenic index of plasma) was calculated as $\log 1_0$ (triglyceride/HDL-C).

while 34 patients (26.0%) had normal cognitive function. The median MoCA-Ina score was 23.00 (range 0–30). From a metabolic perspective, the median triglyceride level was 132 mg/dL (range 59–631), and the median HDL level was 39.2 mg/dL (range 10–110). The mean LDL and total cholesterol levels were 144 ± 53.75 mg/dL and 209 ± 52.5 mg/dL, respectively. Based on the AIP, 65.0% of patients were classified as having high atherogenic risk is shown in Table 1 (23).

Distribution of cognitive domains

The distribution of MoCA-Ina scores across cognitive domains is summarized in Table 2. Performance was relatively preserved in the naming, language, and orientation domains. In the naming domain, 70.2% of patients achieved the maximum score, while in the language domain, 55.0% reached the highest score. Similarly, in the orientation domain, the majority of patients (60.3%) obtained full scores, indicating relatively intact performance in these domains. In contrast, impairment was more apparent in the executive function and delayed recall domains. In the executive domain, scores were widely distributed, with a substantial proportion of patients falling within the lower score range (0–3), reflecting varying degrees of executive dysfunction. A similar pattern was observed in the delayed recall domain, where scores were distributed across lower and intermediate ranges, indicating reduced memory performance in a considerable number of patients. These findings suggest that executive function and memory are among the most affected cognitive domains in the acute phase of IS, consistent with disruption of fronto-subcortical networks (24).

Correlation between Atherogenic Index of Plasma (AIP) and cognitive function

Spearman correlation analysis demonstrated a significant negative association between AIP and total MoCA-Ina score ($r = -0.342$; $p = 0.001$) is shown in Table 3. This indicates that higher atherogenic burden is associated with poorer global cognitive performance. Further domain-specific analysis showed that this relationship was consistent across all cognitive domains. The strongest correlations were observed in language ($r = -0.317$; $p = 0.001$), attention ($r = -0.313$; $p = 0.001$), and executive function ($r = -0.308$; $p = 0.001$). Other domains, including abstraction ($r = -0.261$; $p = 0.003$), naming ($r = -0.224$; $p = 0.010$), orientation ($r = -0.187$; $p = 0.033$), and delayed recall ($r = -0.179$; $p = 0.040$), also showed significant but weaker correlations. The coefficient of determination ($R^2 = 0.098$) suggests that AIP explains approximately 9.8% of the

Table 2. Distribution of MoCA-Ina scores across cognitive domains (n = 131)

	N (%)						
Domains	**0**	**1**	**2**	**3**	**4**	**5**	**6**
Executive (0–5)	12 (9.2)	14 (10.7)	29 (22.1)	26 (19.8)	14 (10.7)	36 (27.5)	–
Naming (0–3)	5 (3.8)	18 (13.7)	16 (12.2)	92 (70.2)	–	–	–
Attention (0–6)	9 (6.9)	14 (10.7)	20 (15.3)	13 (9.9)	14 (10.7)	19 (14.5)	42 (32.1)
Language (0–3)	6 (4.6)	23 (17.6)	30 (22.9)	72 (55.0)	–	–	–
Abstraction (0–2)	21 (16.0)	28 (21.4)	82 (62.6)	–	–	–	–
Delayed Recall (0–5)	18 (13.7)	13 (9.9)	27 (20.6)	28 (21.4)	32 (24.4)	13 (10.0)	–
Orientation (0–6)	5 (3.8)	2 (1.5)	0 (0.0)	6 (4.6)	11 (8.4)	28 (21.4)	79 (60.3)

Data are presented as frequency (percentage). Maximum scores for each domain are shown in parentheses. *Abbreviations:* MoCA-Ina = Montreal Cognitive Assessment Indonesian version.

Table 3. Correlation between AIP and cognitive function (MoCA-Ina)

Variable	**r**	**95% CI**	***p*-value**	**Strength**
Total MoCA-Ina	−0.342	−0.481 to −0.184	0.001**	Moderate
Executive function	−0.308	−0.450 to −0.147	0.001**	Weak
Attention	−0.313	−0.454 to −0.153	0.001**	Weak
Language	−0.317	−0.458 to −0.157	0.001**	Weak
Abstraction	−0.261	−0.408 to −0.095	0.003**	Weak
Naming	−0.224	−0.374 to −0.054	0.010*	Weak
Orientation	−0.187	−0.342 to −0.017	0.033*	Very weak
Delayed recall	−0.179	−0.335 to −0.008	0.040*	Very weak

Spearman rank correlation test. *$p < 0.05$, ** $p < 0.01$ indicates statistical significance. 95% confidence intervals (CI) were calculated using Fisher's z-transformation. Strength of correlation was interpreted as: 0.00–0.19 = very weak; 0.20–0.39 = weak; 0.40–0.59 = moderate.

Table 4. Comparison of cognitive function (MoCA-Ina) between stroke subtypes

Variable	**Non-lacunar (n = 48)**	**Lacunar (n = 83)**	**Total (n = 131)**	***p*-value**
MoCA-Ina score, Mean (SD)	19.02 (7.58)	21.90 (6.03)	20.86 (6.74)	0.028*

Data are presented as mean (standard deviation). Comparison between groups was performed using the Mann–Whitney test. $p < 0.05$ indicates statistical significance.

variability in cognitive scores, indicating that cognitive impairment in stroke is influenced by multiple factors beyond lipid-related mechanisms.

Correlation of stroke subtype and cognitive function

Cognitive outcomes differed significantly between stroke subtypes are shown in Table 4 and boxplot Figure 2. Patients with non-lacunar stroke exhibited *lower cognitive scores* compared with lacunar stroke patients (mean MoCA-Ina 19.02 ± 7.58 vs. 21.90; $p = 0.028$). This finding suggests that larger vessel involvement and more extensive infarction patterns may contribute to greater disruption of cognitive networks (25).

Correlation of atherogenic index of plasma and stroke subtype

Despite the difference in cognitive outcomes, no significant difference in AIP values was observed between lacunar and non-lacunar stroke groups (mean

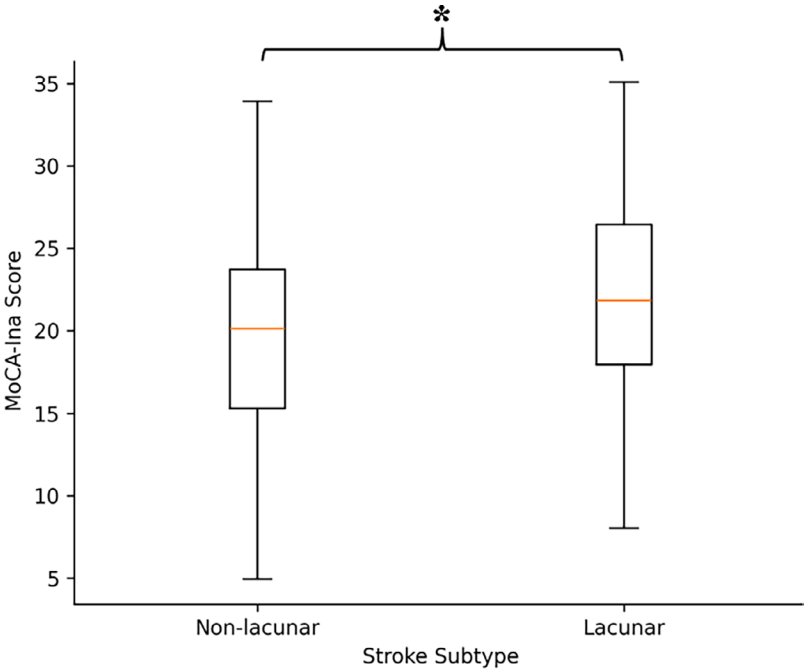

Figure 2. Boxplot of MoCA-Ina scores according to stroke subtype (*p < 0.05).

Table 5. Comparison of Atherogenic Index of Plasma (AIP) between stroke subtypes

Variable	Non-lacunar (n = 48)	Lacunar (n = 83)	Total (n = 131)	p-value
AIP, Mean (SD)	0.285 (0.19)	0.285 (0.24)	0.285 (0.22)	0.991

Data are presented as mean (standard deviation). Comparison between groups was performed using the independent t-test. A p-value < 0.05 was considered statistically significant.

AIP 0.285 in both groups; p = 0.991) are shown in Table 5 and boxplot Figure 3. This indicates that atherogenic burden is similarly distributed across different IS mechanisms, supporting the concept that systemic vascular risk factors contribute to both large- and small-vessel disease (26).

Correlation between AIP and cognitive function across stroke subtypes

Subgroup analysis demonstrated that the negative relationship between AIP and cognitive function remained consistent across both stroke subtypes are shown in table 6 and figure 4. In the lacunar group, AIP was significantly correlated with MoCA-Ina score (r = −0.350; p = 0.001). Similarly, in the non-lacunar group, a significant negative correlation was also observed (r = −0.338; p = 0.019).

To further assess whether this association differed between stroke subtypes, an interaction analysis was performed. The interaction term between AIP and stroke subtype was not statistically significant (p for interaction > 0.05), indicating that the strength of the association between AIP and cognitive function did not differ meaningfully between lacunar and non-lacunar groups. Taken together, these findings suggest that higher AIP is associated with poorer cognitive performance regardless of stroke subtype, supporting the notion of a shared, systemic vascular mechanism underlying cognitive dysfunction in acute ischemic stroke.

Discussion

The present study shows that the Atherogenic Index of Plasma (AIP) is associated with

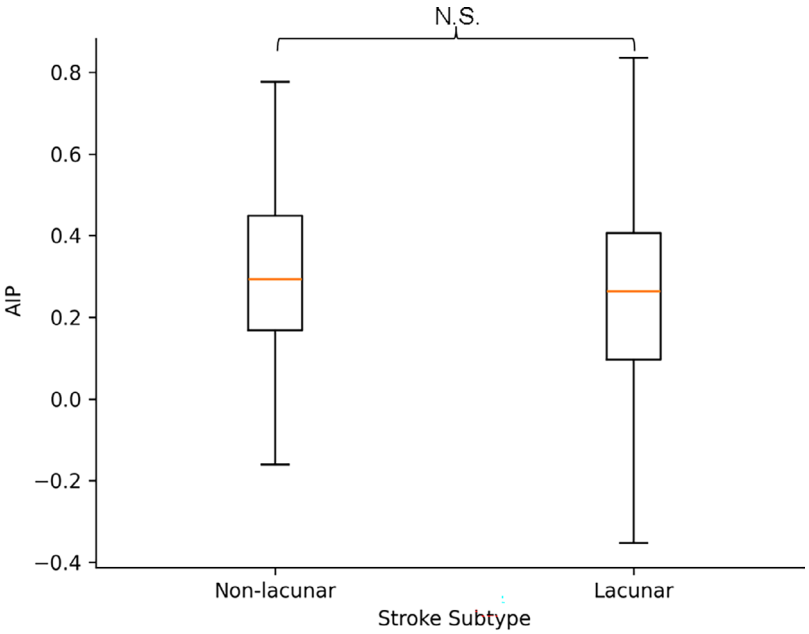

Figure 3. Boxplot of Atherogenic Index of Plasma (AIP) according to stroke subtype (N.S.= Not Significant).

Table 6. Correlation between Atherogenic Index of Plasma (AIP) and cognitive function (MoCA-Ina) across stroke subtypes

Subgroup	r	95% CI	*p*-value	Strength
Lacunar	−0.350	−0.517 to −0.158	0.001**	Moderate
Non-lacunar	−0.338	−0.571 to −0.047	0.019*	Weak–moderate

*Spearman rank correlation test. *p < 0.05, ** p < 0.01 indicates statistical significance.* 95% confidence intervals (CI) were calculated using Fisher's z-transformation. Strength of correlation was interpreted as: 0.20–0.39 = weak; 0.40–0.59 = moderate.

cognitive function in patients with acute ischaemic stroke. Higher AIP was consistently linked to lower global cognitive performance, with a more evident effect in attention, language, and executive domains. Although cognitive impairment was more pronounced in non-lacunar stroke, AIP levels were similar between subtypes, and its association with cognition remained consistent. From a biological standpoint, AIP reflects atherogenic dyslipidaemia characterised by elevated triglycerides and reduced HDL cholesterol. This profile promotes endothelial dysfunction, oxidative stress, and formation of small dense lipoproteins, all of which may impair cerebral perfusion and disrupt neural networks (27-30). Such mechanisms are well aligned with vascular cognitive impairment, where fronto-subcortical circuits are particularly vulnerable. The domain-specific pattern observed in this study further supports this mechanism. Deficits in executive and attentional functions are commonly linked to subcortical and white matter injury, while language impairment may reflect network disconnection rather than focal cortical damage (31). Memory, in contrast, appeared less affected, which is in keeping with typical vascular cognitive profiles. An important observation is that AIP did not differ between lacunar and non-lacunar stroke, yet remained associated with cognitive impairment in both groups. This suggests that atherogenic burden acts as a systemic factor influencing both

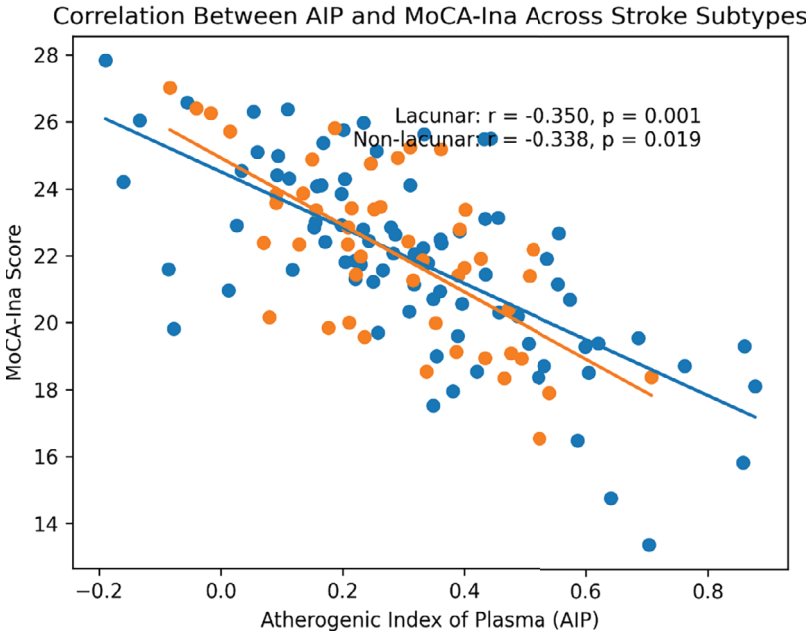

Figure 4. Scatter plot showing the relationship between Atherogenic Index of Plasma (AIP) and cognitive function (MoCA-Ina score) across stroke subtypes. A consistent negative association is observed in both lacunar and non-lacunar groups. The correlation remained significant in the lacunar subgroup (r = −0.350; p = 0.001) and in the non-lacunar subgroup (r = −0.338; p = 0.019).

large- and small-vessel disease (32,33). The absence of interaction between AIP and stroke subtype further indicates that its effect on cognition is not dependent on stroke mechanism. Cognitive assessment was performed in the acute phase, where performance may be influenced by transient factors such as hypoperfusion, inflammation, and fatigue. Therefore, the observed association likely reflects a combination of acute and chronic vascular influences rather than structural injury alone (34). Taken together, this study provides novel evidence that AIP is linked to early cognitive impairment in acute stroke, independent of stroke subtype. This supports the view that metabolic factors contribute to cognitive vulnerability beyond conventional neuroimaging classification.

Clinical implications

AIP may serve as a simple and accessible biomarker to identify patients at risk of early cognitive impairment. Its consistent association across stroke subtypes suggests that metabolic profiling should complement neurological and imaging evaluation. Early recognition of high-risk patients may allow more targeted interventions, including lipid control and closer cognitive monitoring.

Limitations

This study has several limitations. The cross-sectional design does not allow causal inference. The single-centre setting may limit generalisability. Cognitive assessment was limited to the acute phase and may not reflect long-term outcomes. AIP was measured at a single time point, and unmeasured factors such as infarct volume, lesion location, and cognitive reserve may have influenced the results.

Future studies with longitudinal design, repeated metabolic assessment, and advanced neuroimaging are needed to better define the temporal and mechanistic relationship between atherogenic burden and cognitive outcomes.

Conclusions

Higher AIP is associated with poorer cognitive function in the acute phase of ischaemic stroke. This relationship is consistent across lacunar and non-lacunar subtypes, suggesting that atherogenic dyslipidaemia acts as a systemic contributor to cognitive impairment rather than a subtype-specific factor. AIP may therefore serve as a practical biomarker for early identification of patients at risk of post-stroke cognitive decline.

Ethic Approval: All research designs were reviewed and approved by the Health Research Ethics Committee of Dr. Wahidin Sudirohusodo Hospital, Makassar– Faculty of Medicine, Hasanuddin University. (Approval No. 32/UN4.6.4.5.31/PP36/2025)

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

Authors Contribution: Conceptualization, DCH, MYA, AM and MA; Methodology, DCH, MYA and MA; Software, MYA; Validation, MYA, and AAZ; Formal analysis, MYA, DCH, MA and AAZ; Investigation, MYA and DCH; Resources, DCH, MYA, MA and AAZ; Data Curation, DCH, and MYA; Writing—Original Draft Preparation, DCH, and MYA; Writing Review and Editing, MYA; Visualization, DCH, MYA, MA, AAZ, AM and YG; Supervision, MYA, AM and MA; Project Administration, DCH. All authors have read and agreed to the published version of the manuscript.

Declaration on the use of AI: None.

Consent for Publication: All subjects have provided consent for publication.

Acknowledgments: The authors would like to express their sincere gratitude to Dr. Wahidin Sudirohusodo Hospital and its affiliated hospitals in Makassar for providing the facilities and institutional support that made this study possible. We are also grateful to the medical staff and colleagues in the Department of Neurology, Faculty of Medicine, Universitas Hasanuddin, for their valuable assistance during patient recruitment and data collection, as well as for their guidance and academic mentorship throughout the research process and the preparation of this manuscript.

Our deepest appreciation is extended to the patients and their families for their participation, cooperation, and trust, without whom this research would not have been possible. We hope that this study will contribute to the advancement of neurological science, particularly in the field of haemorrhagic stroke and clinical neuroimaging.

Funding: This research received no external funding.

Data Availability Statement: All the data are available from the corresponding author upon a reasonable request (MYA).

Abbreviations

PSCI Post-stroke cognitive impairment
VCI Vascular cognitive impairment
SVO Small vessel occlusion
IS Ischemic stroke
AIP Atherogenic index of plasma
AIS Acute ischemic stroke
PACS Picture archiving and communication system
GCS Glasgow coma scale
TIA Transient ischemic attack
MoCA-Ina Montreal cognitive assessment Indonesian version

References

1. Donkor ES. Stroke in the 21st century: a snapshot of the burden, epidemiology, and quality of life. Stroke Res Treat. 2018;2018:3238165. doi: 10.1155/2018/3238165.
2. Dichgans M, Leys D. Vascular cognitive impairment. Circ Res. 2017;120(3):573-91. doi: 10.1161/CIRCRESAHA.116.308426.
3. Nys GM, van Zandvoort MJ, de Kort PL, et al. Cognitive disorders in acute stroke: prevalence and clinical determinants. Cerebrovasc Dis. 2007;23(5-6):408-16. doi: 10.1159/000101464.
4. Pendlebury ST, Rothwell PM. Prevalence, incidence, and factors associated with pre-stroke and post-stroke dementia: a systematic review and meta-analysis. Lancet Neurol. 2009;8(11):1006-18. doi: 10.1016/S1474-4422(09)70236-4.

5. Sachdev P, Kalaria R, O'Brien J, et al. Diagnostic criteria for vascular cognitive disorders: a VASCOG statement. Alzheimer Dis Assoc Disord. 2014;28(3):206-18. doi: 10.1097/WAD.0000000000000034.
6. Adams HP Jr, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. definitions for use in a multicenter clinical trial. Stroke. 1993;24(1):35-41. doi: 10.1161/01.str.24.1.35.
7. Rundek T, Tolea M, Ariko T, et al. Vascular cognitive impairment (VCI). Neurotherapeutics. 2022;19(1):68-88. doi: 10.1007/s13311-021-01170-y.
8. Wardlaw JM, Smith EE, Biessels GJ, et al. Neuroimaging standards for research into small vessel disease and its contribution to ageing and neurodegeneration. Lancet Neurol. 2013;12(8):822-38. doi: 10.1016/S1474-4422(13)70124-8.
9. Pantoni L. Cerebral small vessel disease: from pathogenesis and clinical characteristics to therapeutic challenges. Lancet Neurol. 2010;9(7):689-701. doi: 10.1016/S1474-4422(10)70104-6.
10. Markus HS, de Leeuw FE. Cerebral small vessel disease: recent advances and future directions. Int J Stroke. 2023;18(1):4-14. doi: 10.1177/17474930221144911.
11. Jebari-Benslaiman S, Galicia-García U, Larrea-Sebal A, et al. Pathophysiology of atherosclerosis. Int J Mol Sci. 2022;23(6):3346. doi: 10.3390/ijms23063346.
12. Anbar R, Jones S, Chaturvedi N, et al. Associations of carotid atherosclerosis with cognitive function and brain health: findings from a UK tri-ethnic cohort study (Southall and Brent revisited). Atheroscler Plus. 2024;55:39-46. doi: 10.1016/j.athplu.2024.01.002.
13. Eslami MH, Sedokani A, Panahi N, et al. The association between atherogenic index of plasma and cognitive impairment among older adults in Southern Iran: findings from the Bushehr elderly health program. BMC Geriatr. 2025;25(1):614. doi: 10.1186/s12877-025-06224-w.
14. Liu H, Liu K, Pei L, et al. Atherogenic index of plasma predicts outcomes in acute ischemic stroke. Front Neurol. 2021;12:741754. doi: 10.3389/fneur.2021.741754.
15. Zheng H, Wu K, Wu W, et al. Relationship between the cumulative exposure to atherogenic index of plasma and ischemic stroke: a retrospective cohort study. Cardiovasc Diabetol. 2023;22(1):313. doi: 10.1186/s12933-023-02044-7.
16. Zhou J, Han H, Bai W. Association between atherogenic index of plasma and cognitive impairment in middle-aged and older adults: results from CHARLS. Front Aging Neurosci. 2025;17:1506973. doi: 10.3389/fnagi.2025.1506973.
17. Gao Y, Li D, Lin J, et al. Cerebral small vessel disease: pathological mechanisms and potential therapeutic targets. Front Aging Neurosci. 2022;14:961661. doi: 10.3389/fnagi.2022.961661.
18. Wardlaw JM, Smith C, Dichgans M. Small vessel disease: mechanisms and clinical implications. Lancet Neurol. 2019;18(7):684-96. doi: 10.1016/S1474-4422(19)30079-1.
19. Iadecola C. The pathobiology of vascular dementia. Neuron. 2013;80(4):844-66. doi: 10.1016/j.neuron.2013.10.008.
20. Dong Y, Lee WY, Basri NA, et al. The Montreal cognitive assessment is superior to the Mini-Mental State Examination in detecting patients at higher risk of dementia. Int Psychogeriatr. 2012;24(11):1749-55. doi: 10.1017/S1041610212001068.
21. Wang Q, Jiang G, Yan L, et al. Association of atherogenic index of plasma with early neurological deterioration in patients with acute ischemic stroke. Clin Neurol Neurosurg. 2023;234:108014. doi: 10.1016/j.clineuro.2023.108014.
22. Gordon DL, Bendixen BH, Adams HP Jr, et al. Interphysician agreement in the diagnosis of subtypes of acute ischemic stroke: implications for clinical trials. Neurology. 1993;43(5):1021-7. doi: 10.1212/wnl.43.5.1021.
23. Akivis Y, Alkaissi H, McFarlane SI, et al. The role of triglycerides in atherosclerosis: recent pathophysiologic insights and therapeutic implications. Curr Cardiol Rev. 2024;20(2):39-49. doi: 10.2174/011573403X272750240109052319.
24. O'Brien JT, Thomas A. Vascular dementia. Lancet. 2015;386(10004):1698-706. doi: 10.1016/S0140-6736(15)00463-8.
25. Zhao L, Biesbroek JM, Shi L, et al. Strategic infarct location for post-stroke cognitive impairment: a multivariate lesion-symptom mapping study. J Cereb Blood Flow Metab. 2018;38(8):1299-1311. doi: 10.1177/0271678X17728162.
26. Cannistraro RJ, Badi M, Eidelman BH, et al. CNS small vessel disease: a clinical review. Neurology. 2019;92(24):1146-56. doi: 10.1212/WNL.0000000000007654.
27. Padro T, Muñoz-Garcia N, Badimon L. The role of triglycerides in the origin and progression of atherosclerosis. Clin Investig Arterioscler. 2021;33 Suppl 2:20-28. doi: 10.1016/j.arteri.2021.02.007.
28. Blagov AV, Churov AV, Golovyuk AL, et al. The role of metabolic disorders in the development of atherosclerosis. Cell Mol Biol (Noisy-le-grand). 2024;70(9):148-55. doi: 10.14715/cmb/2024.70.9.21.
29. Frazier DT, Seider T, Bettcher BM, et al. The role of carotid intima-media thickness in predicting longitudinal cognitive function in an older adult cohort. Cerebrovasc Dis. 2014;38(6):441-7. doi: 10.1159/000366469.
30. Nam KW, Kwon HM, Park JH, Kwon H. The atherogenic index of plasma is associated with cerebral small vessel disease: a cross-sectional study. J Lipid Atheroscler. 2022;11(3):262-71. doi: 10.12997/jla.2022.11.3.262.
31. Inoue Y, Shue F, Bu G, et al. Pathophysiology and probable etiology of cerebral small vessel disease in vascular dementia and Alzheimer's disease. Mol Neurodegener. 2023;18(1):46. doi: 10.1186/s13024-023-00640-5.

32. Onkenhout L, Appelmans N, Kappelle LJ, et al. Cerebral perfusion and the burden of small vessel disease in patients referred to a memory clinic. Cerebrovasc Dis. 2020;49(5):481-6. doi: 10.1159/000510969.
33. Cuadrado-Godia E, Dwivedi P, Sharma S, et al. Cerebral small vessel disease: a review focusing on pathophysiology, biomarkers, and machine learning strategies. J Stroke. 2018;20(3):302-20. doi: 10.5853/jos.2017.02922.
34. Sun JH, Tan L, Yu JT. Post-stroke cognitive impairment: epidemiology, mechanisms and management. Ann Transl Med. 2014;2(8):80. doi: 10.3978/j.issn.2305-5839.2014.08.05.