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Short Communication

Neutrophil-to-lymphocyte ratio and stenosis severity in ischemic stroke: Digital subtraction angiography evaluation and implications for inflammation-based risk stratification in the Indonesian population

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Abstract

Neutrophil-to-lymphocyte ratio (NLR), an accessible biomarker derived from routine blood counts, has been associated with stroke severity and outcomes. However, its association with angiographically confirmed stenosis has not been fully established. The aim of this study was to investigate the correlation and association between the NLR and the severity of intracranial arterial stenosis assessed by digital subtraction angiography (DSA). An observational analytic study with a cross-sectional design was conducted. Patients with acute ischemic stroke who underwent DSA were included using a total sampling method. Eligible patients were aged >18 years, had ischemic stroke onset <14 days, and had no prior thrombolysis, endovascular treatment, or surgery for cerebral stenosis. Those with infection, hematological disorders, malignancy, or immunosuppression were excluded. NLR was calculated from complete blood count results at admission, while stenosis severity was quantified using the Warfarin–Aspirin Symptomatic Intracranial Disease (WASID) method. A total of 44 ischemic stroke patients who underwent DSA were included. Pearson correlation test revealed a strong positive correlation between NLR and the severity of intracranial arterial stenosis (r=0.671; p<0.001). In subgroup analysis, NLR showed a strong positive correlation with stenosis severity in the cerebral arteries (r=0.707; p<0.001), but not in the carotid arteries (r=0.434; p=0.182). One-way ANOVA revealed significant differences in NLR across stenosis severity groups (p<0.0001), with higher NLR in moderate and severe stenosis compared with mild stenosis (p=0.017 and p=0.0003, respectively). These findings suggest that NLR reflects the inflammatory burden contributing to vascular narrowing and may serve as a simple and widely available biomarker for identifying ischemic stroke patients with a higher burden of intracranial arterial stenosis, particularly in settings where access to advanced imaging is limited.

Keywords: Ischemic stroke, intracranial arterial stenosis, digital subtraction angiography, NLR, inflammation mediator

Introduction

Ischemic stroke remains a leading cause of mortality and long-term disability worldwide, with a particularly high burden in low- and middle-income countries [1,2]. There has been a 70 %

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increase in ischemic stroke incidence between 1990 and 2021; it now causes nearly five million deaths annually and accounts for a significant share of disability-adjusted life years (DALYs) [1,2]. In 2021, approximately 93.8 million individuals were living with ischemic stroke worldwide, and lower- and middle-income countries accounted for around 87 % of stroke-related deaths and 89% of DALYs [3]. The underlying pathophysiology of ischemic stroke is strongly associated with atherosclerotic changes in the cerebral vasculature, where luminal stenosis contributes to impaired cerebral perfusion and increased risk of recurrent stroke [4]. Identifying reliable markers that reflect stenosis severity is essential for risk stratification and optimizing management strategies [5].

Digital subtraction angiography (DSA) remains the gold standard for the precise characterization of cerebrovascular stenosis because of its high spatial resolution and ability to directly visualize vascular lumen narrowing [6]. While DSA provides unmatched spatial resolution for vascular imaging, its invasiveness, cost, technical challenges, and limited accessibility—especially in resource-constrained settings—drive the need for non-invasive, accessible biomarkers and imaging alternatives for cerebrovascular disease assessment [6,7]. Among the proposed markers, the neutrophil-to-lymphocyte ratio (NLR), which can be readily obtained from a routine complete blood count, has gained growing attention [8-11].

NLR reflects the balance between innate and adaptive immune responses, thereby serving as a simple yet robust marker of systemic inflammation [12,13]. In stroke research, elevated NLR has been consistently associated with increased stroke severity, higher rates of short-term complications, and greater mortality [8-11]. To date, no study in Indonesia has specifically explored the correlation between NLR and stenosis severity confirmed by DSA in ischemic stroke patients, leaving a critical gap in the understanding of inflammation-based prognostic markers within this high-burden population. Given the limited availability of DSA in resource-constrained settings across Indonesia, the use of simple and practical biomarkers such as NLR could serve as a valuable alternative for identifying patients at higher risk of severe cerebral arterial stenosis. Therefore, the aim of this study was to investigate the correlation between the NLR and the severity of intracranial arterial stenosis assessed by DSA.

Methods

Study design and setting

An observational analytic study with a cross-sectional design was conducted in the Neurology Inpatient Ward of Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia, from February to April 2025. All ischemic stroke patients undergoing DSA during the study period were considered for inclusion based on predefined eligibility criteria. Data collection included demographic and clinical information, complete blood count for NLR, and DSA findings for intracranial arterial stenosis severity.

Patients and criteria

The study population consisted of patients who were clinically diagnosed with ischemic stroke and admitted to the Neurology Inpatient Ward of Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia. Only patients who underwent DSA as part of their diagnostic evaluation were considered for recruitment. A total sampling approach was employed, whereby all eligible patients meeting the predefined criteria during the study period were included. To ensure homogeneity of the study cohort, inclusion was restricted to patients presenting with acute ischemic stroke within 14 days of symptom onset, aged above 18 years, and with no prior history of thrombolytic therapy, mechanical endovascular intervention, or surgical procedures for cerebral arterial stenosis.

Patients with conditions that could potentially confound the interpretation of inflammatory markers were excluded, including those with active systemic infections, hematological disorders, or malignancies. Similarly, individuals who were immunocompromised, either due to underlying medical conditions or as a result of receiving immunosuppressive treatments such as corticosteroids or other immunotherapy agents, were also excluded.

Data collection and study variables

Demographic and clinical data, including age, sex, and comorbidities, were collected. All included patients underwent a structured clinical evaluation, which included detailed history taking and a complete neurological examination conducted by the attending neurologist upon admission.

Venous blood samples (3–5 mL) were collected via peripheral venipuncture under aseptic conditions within the first 24 hours of hospitalization, before DSA. Samples were processed immediately in the hospital's central laboratory using an automated hematology analyzer (Sysmex XN-Series, Sysmex Corporation, Kobe, Japan). Parameters obtained included absolute neutrophil and lymphocyte counts, from which the NLR was calculated by dividing the absolute neutrophil count (cells/µL) by the absolute lymphocyte count (cells/µL).

Assessment of intracranial arterial stenosis was performed using DSA by a single board-certified neurologist with expertise in neurointervention, who was blinded to all clinical and laboratory parameters to minimize assessment bias. As one experienced observer performed the evaluation, inter-observer reliability was not applicable in this study. DSA was performed in the Interventional Neurology Unit of Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia, using a Philips Allura Xper FD20 (Philips Healthcare, Eindhoven, The Netherlands) biplane angiography system. The procedure was conducted under local anesthesia with aseptic technique.

Femoral artery access was obtained using the Seldinger technique, followed by the insertion of a 5F or 6F vascular sheath. A nonionic contrast medium (iohexol 300 mg/mL) was administered through a diagnostic catheter to visualize the cerebral arteries. Standard projections were obtained for the internal carotid and vertebrobasilar systems to evaluate both anterior and posterior circulations. The severity of stenosis was calculated according to the Warfarin–Aspirin Symptomatic Intracranial Disease (WASID) method as the percentage reduction in the luminal diameter of the target vessel compared with the diameter of the most normal proximal arterial segment [14]. For patients with multiple intracranial arterial stenoses, only the single most severe lesion identified on DSA was included in the correlation analysis.

Data analysis

Data were analyzed using IBM SPSS Statistics version 25.0 (IBM Corp, Armonk, NY, USA) and GraphPad Prism version 10.2.0 (GraphPad Software, Boston, MA, USA). Categorical variables were summarized as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation (SD) or median with (min–max), depending on distribution. The Shapiro-Wilk test was applied to assess data normality. The correlation between NLR and severity of intracranial arterial stenosis was analyzed using Pearson correlation for non-normally distributed data. Comparison of mean NLR values across stenosis severity groups (mild, moderate, and severe) was performed using one-way ANOVA, followed by Dunnett's T3 post-hoc analysis to determine pairwise differences. A p-value ≤0.05 was considered statistically significant.

Results

Characteristics of the included patients

A total of 44 patients with ischemic stroke were included in this study, consisting of 29 males (65.9%) and 15 females (34.1%), with a mean age of 56.16±10.15 years (**Table 1**). The most prevalent comorbid was hypertension, present in all patients (100%), followed by type 2 diabetes mellitus in 34 patients (77.3%), dyslipidemia in 16 patients (36.4%), and heart disease in 2 patients (4.5%). Regarding the distribution of intracranial arterial stenosis, the most frequently affected site was the right middle cerebral artery (MCA) in 14 patients (31.8%), followed by the right internal carotid artery (ICA) in 8 patients (18.2%) and the left MCA in 7 patients (15.9%). Stenosis of the right anterior cerebral artery (ACA) was observed in 5 patients (11.4%) and of the left ACA in 4 patients (9.1%). Less common sites included the left ICA in 3 patients (6.8%), the basilar artery in 2 patients (4.5%), and the right posterior cerebral artery (PCA) in 1 patient (2.3%). The mean intracranial arterial stenosis severity was 56.20±27.40%, with nearly half of the patients classified as having mild stenosis (<50%) (47.7%), followed by severe (>70%) (31.8%) and moderate (50–69%) (20.5%) stenosis. The median neutrophil percentage was 67.50 (range:

43.00–92.00), the mean lymphocyte percentage was 19.40±9.65, and the mean NLR was 4.90±2.71 (**Table 1**).

Table 1. Characteristics of the included ischemic stroke patients in the study (n=44)

Variables	Frequency (%)
Sex	
Male	29 (65.9)
Female	15 (34.1)
Age (years), mean±SD	56.16±10.15
Risk factor	
Hypertension	44 (100)
Type 2 diabetes mellitus	34 (77.3)
Dyslipidemia	16 (36.4)
Heart disease	2 (4.5)
Location of stenosis	
Right anterior cerebral artery (ACA)	5 (11.4)
Left ACA	4 (9.1)
Right middle cerebral artery (MCA)	14 (31.8)
Left MCA	7 (15.9)
Right posterior cerebral artery (PCA)	1 (2.3)
Right internal carotid artery (ICA)	8 (18.2)
Left ICA	3 (6.8)
Basilar artery	2 (4.5)
Intracranial arterial stenosis severity (%), mean±SD	56.20±27.40
Mild (<50%)	21 (47.7)
Moderate (50–69%)	9 (20.5)
Severe (>70%)	14 (31.8)
Neutrophil count (%), median (min-max)	67.50 (43.00–92.00)
Lymphocyte count (%), mean±SD	19.40±9.65
Neutrophile-to-lymphocyte ratio (NLR), mean±SD	4.90±2.71

Correlation and association between neutrophil-to-lymphocyte ratio and severity of intracranial arterial stenosis in ischemic stroke patients

Pearson correlation test revealed a significant, strong positive correlation was observed between NLR and severity of intracranial arterial stenosis (r=0.671; p<0.001) (**Figure 1**). Patients with higher NLR values demonstrated a greater severity of intracranial arterial stenosis on DSA.

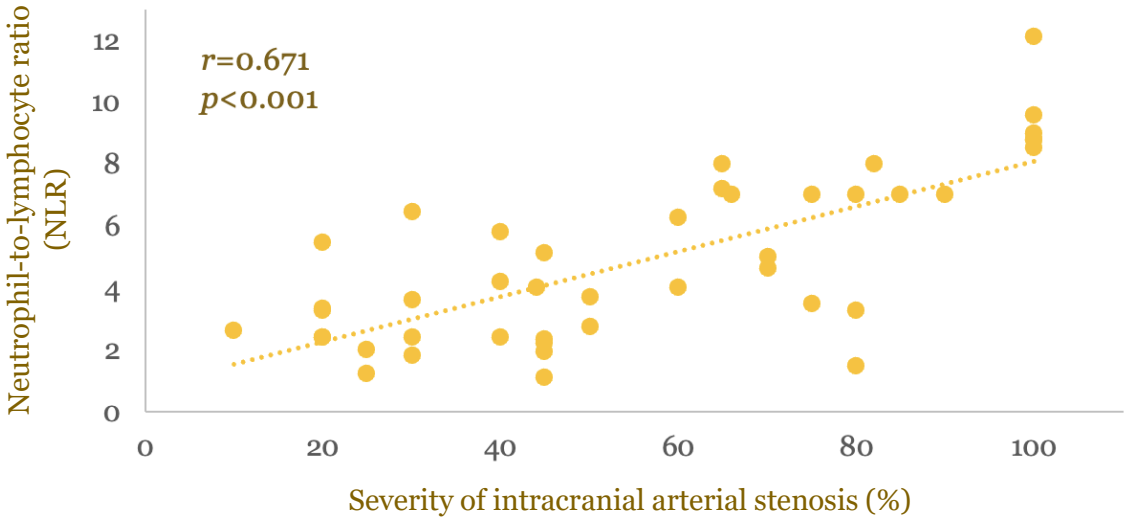

Figure 1. Correlation between neutrophil-to-lymphocyte ratio (NLR) and severity of intracranial arterial stenosis as measured by digital subtraction angiography (DSA) in ischemic stroke patients.

In the subgroup analysis based on vascular location, the NLR demonstrated a strong positive correlation with the severity of intracranial arterial stenosis in the cerebral arteries (r=0.707; p<0.001) (**Table 2**). In contrast, the correlation between NLR and intracranial arterial stenosis in the carotid arteries was not statistically significant (r=0.434; p=0.182).

Table 2. Correlation between neutrophil-to-lymphocyte ratio (NLR) and severity of arterial stenosis based on vascular location

Vascular location	Neutrophil-to-lymphocyte ratio (NLR)	
	r	*p*-value [a]
Cerebral arteries	0.707	<0.001*
Carotid arteries	0.434	0.182

[a] Analyzed using Pearson correlation test
* Statistically significant at $p<0.05$

Comparison between neutrophil-to-lymphocyte ratio and severity of intracranial arterial stenosis in ischemic stroke patients

One-way ANOVA test revealed a significant difference in NLR among the three groups of intracranial arterial stenosis severity ($p<0.0001$) (**Figure 2**). Post-hoc analysis using Dunnett's T3 test indicated that patients with moderate stenosis had a significantly higher mean NLR than those with mild stenosis ($p=0.017$). Likewise, the severe stenosis group demonstrated a significantly higher NLR compared with the mild group ($p=0.0003$). In contrast, no significant difference was observed between the moderate and severe groups ($p=0.195$).

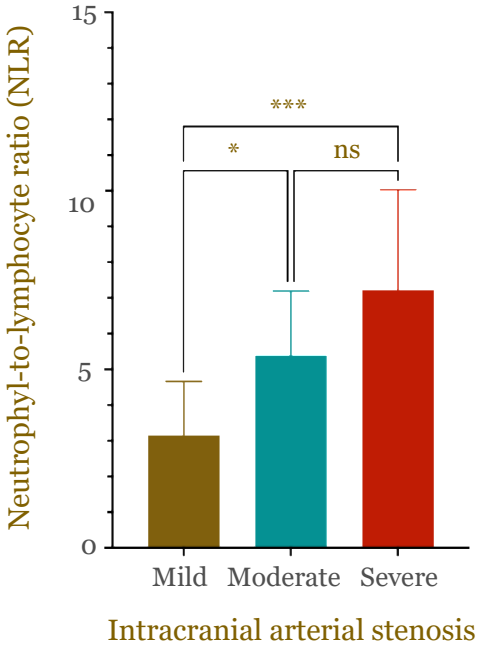

Figure 2. Comparison of neutrophil-to-lymphocyte ratio (NLR) across intracranial arterial stenosis severity groups. *$p<0.05$; ***$p<0.0001$; ns: not significant.

Discussion

This study demonstrated a strong positive correlation between NLR and the severity of intracranial arterial stenosis assessed by DSA in patients with ischemic stroke. Higher NLR values were consistently associated with more severe stenosis, highlighting the potential utility of NLR as an accessible inflammatory biomarker to complement angiographic evaluation. The findings align with the established role of inflammation in the pathophysiology of atherosclerosis and cerebrovascular disease [15-17]. Neutrophils contribute to plaque development and instability through the release of proteolytic enzymes, reactive oxygen species, and pro-inflammatory cytokines. In contrast, lymphopenia reflects an impaired adaptive immune response under systemic stress [18-21]. The resulting elevation of NLR therefore serves as a surrogate marker of heightened inflammatory activity, which accelerates endothelial dysfunction, vascular remodeling, and luminal narrowing [9,22,23]. This mechanistic link explains the observed correlation between elevated NLR and more advanced stenosis.

Elevated NLR is not only associated with stenosis severity but also with worse clinical outcomes in acute ischemic stroke with intracranial atherosclerotic stenosis (ICAS), including

greater initial stroke severity and poorer short-term prognosis [22,24–28]. NLR is also associated with poorer collateral circulation in patients with severe intracranial stenosis or occlusion [28]. Multiple studies consistently demonstrate that higher NLR values are independently associated with both the presence and severity of ICAS [22,24-28]. In a large DSA-based study, increased NLR was independently associated with both symptomatic stenosis and greater stenosis severity [27]. Patients in higher NLR quartiles had significantly higher odds ratios (OR) of severe intracranial stenosis, with OR for highest vs. lowest quartile of 2.23; $p<0.001$ [27]. This association holds true across diverse populations and is observed even after adjusting for traditional vascular risk factors. Studies using non-invasive imaging such as magnetic resonance angiography (MRA) and computed tomography angiography (CTA) similarly report that higher NLR is associated with both the presence of ICAS and increased risk of ischemic stroke, with ICAS partially mediating the NLR-stroke relationship [22,25]. Furthermore, NLR also correlates with the number and burden of ICAS lesions, showing a dose-response relationship [26]. Because NLR can be influenced by systemic inflammation, diabetes mellitus, and dyslipidemia, these factors were recognized as potential confounders in interpreting the association between NLR and stenosis severity. Although multivariate regression analysis was not conducted to adjust for these variables in the present study, such confounding influences were acknowledged as limitations. They should be considered in future research to better delineate the predictive value of NLR in populations with varying metabolic and inflammatory profiles.

The clinical implications of these findings are particularly relevant for the Indonesian population, where ischemic stroke represents a major health burden and access to advanced imaging modalities such as DSA is often limited to tertiary referral centers. Compared with other populations, Indonesian patients with cerebral arterial stenosis tend to present with a higher proportion of intracranial rather than extracranial lesions, resembling patterns observed in other East and Southeast Asian cohorts [29]. The condition often develops at a relatively younger age, frequently associated with multiple metabolic risk factors such as hypertension, diabetes mellitus, and dyslipidemia [29]. The invasive nature, high cost, and limited distribution of DSA across the archipelago create significant barriers for routine use in early diagnostic evaluation. By contrast, the NLR can be obtained from a standard complete blood count, a test that is widely available even in primary and secondary healthcare facilities throughout Indonesia. Integrating NLR into routine stroke assessment could enhance risk stratification at the point of first contact, allowing clinicians to identify patients with a higher likelihood of severe intracranial stenosis. Such an approach may guide timely referral for advanced imaging, when necessary, support targeted allocation of limited resources, and enable earlier initiation of intensive secondary prevention strategies. In the broader context of Indonesia's healthcare system, where disparities in infrastructure and specialist availability remain pronounced, NLR offers a practical and scalable biomarker that could bridge the gap between sophisticated diagnostic standards and real-world clinical practice.

This study has several limitations. The cross-sectional design precludes the determination of causal relationships, and the modest sample size limits generalizability to broader populations. The absence of longitudinal follow-up data, such as stroke recurrence or mortality, restricts the ability to evaluate prognostic implications of elevated NLR. Moreover, inflammatory biomarkers such as C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR) were not measured, preventing direct comparison between NLR and established systemic inflammatory markers. The study was conducted at a single tertiary center, which may introduce referral bias and limit external validity. Although exclusion criteria were applied to minimize confounding factors, unrecognized systemic or subclinical inflammation may still have affected NLR values. Additionally, inter-observer variability in DSA interpretation, while minimized through standardized assessment and review by an experienced neurologist, remains an inherent limitation of angiographic evaluation. Future research should aim to validate these findings in larger, multicenter cohorts and explore the predictive value of NLR for long-term outcomes in patients with intracranial atherosclerotic disease. Prospective studies combining NLR with other inflammatory and imaging biomarkers could further refine its role in risk stratification and management of ischemic stroke.

Conclusion

Higher NLR values were significantly correlated with greater stenosis severity among ischemic stroke patients. NLR may serve as a simple, inexpensive, and widely accessible biomarker for identifying ischemic stroke patients with a greater burden of intracranial arterial stenosis. By reflecting the underlying inflammatory state that contributes to atherosclerosis progression, NLR has the potential to complement imaging modalities in risk stratification and clinical decision-making. This role is particularly relevant in resource-limited settings where access to advanced imaging techniques, such as digital subtraction angiography, may be restricted.

Ethics approval

The study protocol was reviewed and approved by the Ethics Committee for Health Research, Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia (No. 025/ETIK-RSUDZA/2025), in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from the patient or patients' legal guardians prior to enrollment.

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Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

This study used artificial intelligence (AI) tool and methodology of which AI-based language model ChatGPT was employed in the language refinement (improving grammar, sentence structure, and readability of the manuscript). We confirm that all AI-assisted processes were critically reviewed by the authors to ensure the integrity and reliability of the results. The final decisions and interpretations presented in this article were solely made by the authors.

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