Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 22 July 2026
Revised 18 Aug 2026
Accepted 05 Sep 2026

Natural Resources for Human Health 2026, Vol. 6 (20s): 1136–1143

KEYWORDS:
Neutrophil-Lymphocyte Ratio (NLR), Platelet Count (PC), Ischemic Stroke, NIHSS.

Correlation Analysis of Neutrophil-Lymphocyte Ratio (NLR) and Platelet Count (PC) with Clinical Degrees of Ischemic Stroke Patients

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Abstract: This retrospective cross-sectional study examined associations of neutrophil-to-lymphocyte ratio (NLR) and platelet count (PC) with admission NIHSS scores at Bhayangkara Kendari Hospital. Total sampling included 32 ischemic stroke patients aged 18–80 years with onset within 72 hours before admission. Mean age was 59.41 ± 11.48 years; 62.5% were male. Median NLR was 3.35, PC was 262 × 10^3 cells/mm^3, and NIHSS was 6.50 (range 3–18). Study-defined NIHSS groups comprised 14 mild (43.8%; 0–4), 15 moderate (46.9%; 5–15), and 3 higher-severity cases (9.4%; >15). Spearman analysis showed a positive NLR–NIHSS correlation (r_s = 0.636, $p < 0.001$), whereas PC was not significantly correlated (r_s = 0.225, $p = 0.216$). Exact sampling intervals, assessor training, baseline risk factors, and pretreatment data were not available for evaluation. The small single-centre sample, retrospective selection, and lack of adjustment limit interpretation. NLR was associated with admission neurological impairment; independent effects and diagnostic, prognostic, or predictive performance were not established. NLR cannot replace clinical assessment on the basis of these findings.

1. INTRODUCTION

Stroke is one of the most significant global health problems and remains a leading cause of mortality and long-term disability worldwide [1]. According to the World Health Organization (WHO), stroke is defined as an acute neurological deficit lasting more than 24 hours or leading to death due to vascular causes [2]. In Indonesia, stroke is the leading cause of death among hospitalized patients, with a prevalence of approximately 12.1 per 1,000 population, and the burden continues to increase, particularly in regions such as Southeast Sulawesi [3].

Ischemic stroke occurs due to impaired cerebral blood flow caused by thrombosis, embolism, or hypoperfusion, leading to neuronal injury and infarction [4]. This condition results in damage not only to the infarct core but also to the surrounding penumbra region, which remains potentially salvageable with timely intervention [5,6]. One of the key mechanisms influencing the extent of brain injury is the inflammatory response triggered by ischemia [7]. This response involves the activation and infiltration of inflammatory cells such as neutrophils, monocytes, and lymphocytes, as well as the release of pro-inflammatory mediators including interleukins and tumor necrosis factor [8]. These processes contribute to secondary brain injury and are closely associated with the clinical severity of stroke [9].

The Neutrophil-to-Lymphocyte Ratio (NLR) is a hematological marker reflecting the balance between innate and adaptive immune responses, widely used to assess systemic inflammation [10]. NLR integrates neutrophils as acute inflammation mediators and lymphocytes as immune response regulators, making it a comprehensive indicator of inflammatory status. Studies show a positive correlation between elevated NLR and increased stroke severity, often measured using the NIH Stroke Scale (NIHSS) [11]. However, NLR values can be influenced by conditions like infection, autoimmune diseases, and malignancy, requiring careful clinical interpretation [12].

Besides NLR, platelet count (PC) is relevant to ischemic stroke pathophysiology through thrombosis and inflammation [13]. Platelets contribute to thrombus formation and inflammatory signaling through interactions with leukocytes and endothelial cells [14]. Previous studies have investigated hematological markers in relation to stroke severity and outcomes [15]. This study aimed to examine the associations of NLR and PC with admission NIHSS scores in ischemic stroke patients at Bhayangkara Kendari Hospital. Diagnostic accuracy, prediction of future outcomes, and the incremental value of these markers beyond clinical assessment were not evaluated.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This study employed an observational analytic design with a cross-sectional approach to determine the correlation between NLR, PC, and the clinical severity of ischemic stroke. The study was conducted at Bhayangkara Kendari Hospital, a secondary referral hospital in Southeast Sulawesi, Indonesia, using retrospective data derived from medical records.

2.2 Study Population and Sampling

The study population comprised patients admitted to Bhayangkara Kendari Hospital with a recorded diagnosis of ischemic stroke during the study period. Total sampling was used, and 32 patients meeting the stated eligibility criteria were included in the analysis. The available study documentation does not specify the calendar dates of admission, the number of records initially screened, or the number excluded for each criterion. Consequently, a complete screening flow and the proportion of otherwise eligible patients excluded because of missing records cannot be reconstructed.

2.3 Inclusion and Exclusion Criteria

Inclusion criteria were a recorded ischemic stroke diagnosis confirmed clinically and/or radiologically, age 18–80 years, and stroke symptom onset within 72 hours before admission. Exclusion criteria were incomplete medical records; acute infection, autoimmune disease, malignancy, or hematological disorders that could affect inflammatory markers; and use of corticosteroids or immunosuppressive agents that could alter leukocyte or platelet counts. Diagnostic ascertainment relied on the clinical and/or radiological diagnosis documented in the medical records. The available documentation does not specify a uniform diagnostic case definition, the imaging modality, or whether imaging confirmation was obtained in every participant. The onset criterion referred to the recorded onset-to-admission interval; the source of onset information, the handling of unwitnessed or wake-up stroke, and whether last-known-well time was used were not documented. These limitations prevent verification of diagnostic and onset-time consistency across participants.

2.4 Data Collection Procedures

Data were obtained retrospectively from hospital medical records and included age, sex, neutrophil and lymphocyte values, platelet count, and admission NIHSS scores. NLR was calculated as the neutrophil-to-lymphocyte ratio. The descriptive data report leukocyte differential percentages; because paired differential percentages share the same total leukocyte denominator, their ratio is equivalent to the ratio of paired absolute counts from the same sample. PC was obtained from routine hematological examination. The available documentation does not identify the specific blood sample selected when multiple results existed or provide the exact interval from symptom onset to venipuncture, the interval between venipuncture and NIHSS assessment, or their sequence relative to treatment. Admission within 72 hours of onset should therefore not be interpreted as evidence of a standardized blood-sampling interval.

Admission NIHSS scores were obtained retrospectively from the medical records, rather than through a new examination for this study. The available documentation does not establish whether scores were transcribed as recorded totals or reconstructed from individual examination items, nor does it identify assessor profession, NIHSS training or certification, or inter-rater reliability. The analysis used three study-defined groups: mild (0–4), moderate (5–15), and higher severity (>15), comprising 14,

15, and 3 participants, respectively. These cutoffs preserve the original three-group tabulation and distinguish lower, intermediate, and higher recorded neurological impairment; an a priori rationale or validation of these thresholds in this sample was not documented. The >15 group is termed higher severity rather than severe to avoid implying a uniform clinical classification. Observed scores ranged from 3 to 18; thus, no participant had a score of 0 or a score above 18. Continuous NIHSS scores were retained for the primary correlation analysis.

2.5 Statistical Analysis

Descriptive statistics summarized participant characteristics. Because the variables were not normally distributed, Spearman rank correlation was used to examine the associations of NLR and PC with continuous NIHSS scores. The reported Chi-square analyses of categorized variables were treated as exploratory. Several contingency-table cells contained small or zero counts, limiting the reliability of the asymptotic Chi-square p-values. The NLR threshold of 4.29 and the platelet-count categories reproduce the original analysis; their derivation and independent validation were not documented. They should not be interpreted as validated clinical decision thresholds. Statistical significance was defined as $p < 0.05$; values originally displayed as $p = 0.000$ are reported as $p < 0.001$. No multivariable adjustment, diagnostic-accuracy analysis, predictive model, or longitudinal outcome analysis was performed.

3. RESULTS AND DISCUSSION

3.1 Baseline Characteristics of Study Participants

The 32 participants had a mean age of 59.41 ± 11.48 years; 9 (28.1%) were aged 61–70 years and 1 (3.1%) was aged 31–40 years. There were 20 men (62.5%) and 12 women (37.5%). The median NLR was 3.35 (range 0.69–14.63), and median PC was 262 × 10^3 cells/mm^3 (range 138–662 × 10^3 cells/mm^3). The median lymphocyte percentage was 20.25%, with a reported minimum of 6.20%; the originally tabulated upper value of 120% could not be verified and is not presented as a valid percentage. Admission NIHSS had a median of 6.50 (range 3–18). There were 14 participants with mild impairment (43.8%), 15 with moderate impairment (46.9%), and 3 in the higher-severity group (9.4%). Percentages may not sum to 100% because of rounding.

The available baseline data were limited to the variables shown in Table 1. Frequencies of hypertension, diabetes mellitus, dyslipidemia, smoking, atrial fibrillation, other vascular or systemic comorbidities, previous stroke, and prestroke disability were not available for this analysis. Receipt and timing of thrombolysis, thrombectomy, antiplatelets, anticoagulants, or other treatment before laboratory assessment were likewise not available. Absence of these data should not be interpreted as absence of these conditions or treatments.

Table 1. Characteristics of Research Subjects

Characteristics			**n (%)**	***Mean* ± SD/ Median (Min-Max)***
Age	31-40		1 (3.1)	59.41± 11.48
	41-50		8 (25.0)	
	51-60		8 (25.0)	
	61-70		9 (28.1)	
	71-80		6 (18.8)	
Gender	Male		20 (62.5)	
	Female		12 (37.5)	
Inflammatory Biomarkers	Neutrophils (%)			67.29±12.8
	Lymphocytes (%)			20.25

	NLR			3.35 (0.69-14.63)
	PC(x10³cells/mm)			262 (138-662)
Clinical Degree	Mild (0-4)		14 (43.8)	6.50 (3 - 18)
	Moderate (5-15)		15 (46.9)	
	Higher severity (>15)		3 (9.4)	

*Data presented as Mean ± SD if normally distributed or Median (Min - Max) if not normally distributed. Source: Secondary Data, 2024 The lymphocyte upper limit is unverified because the source table reported 120%, which is not a valid differential percentage. Severity groups are study-defined; see Section 2.4. Vascular risk factors, comorbidities, prior stroke, and treatment before testing were not available for analysis.

3.2 Correlation of Neutrophil-Lymphocyte Ratio with Clinical Degrees of Ischemic Stroke

The chi-square test in table 2 was performed to assess the relationship between NLR and the clinical degree of ischemic stroke based on the National Institutes of Health Stroke Scale. There were 17 (53.1%) subjects with non-increased NLR and 15 (46.9%) with increased NLR. Furthermore, 14 (43.8%) subjects had a mild NIHSS score, 15 (46.9%) had a moderate NIHSS score and 3 (9.4%) had a higher-severity NIHSS score when first admitted to the emergency department. Subjects with NLR did not increase, 14 (43.8%) had mild NIHSS scores, 3 (9.4%) had moderate NIHSS scores, and no subjects had higher-severity NIHSS scores.

Table 2. Relationship between Neutrophil-Lymphocyte Ratio (NLR) and Clinical Degree of Ischemic Stroke

NLR	**NIHSS**			**Total**	**p-value**
	Mild	**Moderate**	**>15***	**n (%)**	
NLR <4.29	14 (43.8)	3 (9.4)	0 (0)	17 (53.1)	<0.001
NLR ≥4.29	0 (0)	12 (37.5)	3 (9.4)	15 (46.9)	
Total n (%)	14 (43.8)	15 (46.9)	3 (9.4)	32 (100)	

NIHSS: National Institutes of Health Stroke Scale; NLR: Neutrophil-Lymphocyte Ratio; *Chi-Square Test. Higher severity (>15) is the study-defined category marked >15* in the table.

Among participants with NLR ≥4.29, 12 (37.5% of the total sample) had moderate NIHSS scores and 3 (9.4%) had higher-severity scores. The exploratory Chi-square analysis yielded $p < 0.001$, subject to the sparse-cell limitation described above. The primary Spearman analysis showed a positive association between NLR and continuous NIHSS scores ($r_s = 0.636$, $p < 0.001$). Higher NLR values were associated with greater neurological impairment at admission; this unadjusted association does not establish causality or independent predictive value.

Table 3. Correlation Test of Neutrophil-Lymphocyte Ratio (NLR) with Clinical Degrees of Ischemic Stroke

Variables	P-value	r-value
NLR	<0.001	0.636
NIHSS		

NIHSS: National Institutes of Health Stroke Scale; NLR: Neutrophil-Lymphocyte Ratio; *Spearman Correlation Test

3.3 Correlation of Platelet Count with Clinical Severity of Ischemic Stroke

The chi-square test in Table 4 was performed to assess the relationship between PC and the clinical degree of ischemic stroke based on the National Institutes of Health Stroke Scale. There were 29 (90.6%) subjects with PC in the normal range, while 3 (9.4%) subjects had increased PC. There were no subjects with decreased PC. A total of 14 (43.8%) subjects with normal PC had mild NIHSS scores, 12 (37.5%) had moderate NIHSS scores, and 3 (9.4%) had higher-severity NIHSS scores. Subjects with increased PC, namely 3 (9.4%) subjects, all had moderate NIHSS scores. None of the subjects with increased PC had mild or higher-severity NIHSS scores. The chi-square test in table 4 showed a p value of 0.153, indicating that there was no statistically significant relationship between platelet count and the clinical degree of ischemic stroke based on NIHSS.

Spearman analysis showed a weak positive correlation between PC and NIHSS ($r_s = 0.225$, $p = 0.216$), which was not statistically significant. These data do not establish an association, and the small sample does not exclude a clinically relevant relationship.

Table 4. Relationship of Platelet Count (PC) with Clinical Degrees of Ischemic Stroke

PC (cells/mm³)	NIHSS			Total n (%)	p-value
	Mild	Moderate	>15*		
Low PC (<100 × 10³)	0 (0)	0 (0)	0 (0)	0 (0)	0.153
Normal PC (100 - 450x10³)	14 (43.8)	12 (37.5)	3 (9.4)	29 (90.6)	
PC increased (>450x10³)	0 (0)	3 (9.4)	0 (0)	3 (9.4)	
Total n (%)	14 (43.8)	15 (46.9)	3 (9.4)	32 (100)	

NIHSS: National Institutes of Health Stroke Scale; PC: Platelet Count;*Chi-Square Test. Higher severity (>15) is the study-defined category marked >15* in the table.

Table 5. Correlation Test between Platelet Count (PC) and Clinical Degree of Ischemic Stroke

Variables	P-value	r-value
PC	0.216	0.225
NIHSS		

NIHSS: National Institutes of Health Stroke Scale; PC: Platelet Count; *Spearman Correlation Test

3.4 Discussion

This study showed that the majority of ischemic stroke patients were in the age group of 61–70 years. This finding is consistent with previous evidence indicating that increasing age is a major non-modifiable risk factor for stroke. Age-related vascular changes, including endothelial dysfunction, arterial stiffness, and impaired cerebral autoregulation, contribute significantly to both the incidence and severity of ischemic stroke. Inline with a study by Zhang et al. (2021), older age is strongly associated with worse clinical outcomes and higher NIHSS scores due to reduced neuroplasticity and limited compensatory mechanisms following brain injury [16].

In terms of sex distribution, this study found a predominance of male patients. This finding aligns with previous studies suggesting that men have a higher risk of stroke during productive years. Inline with a study by Bushnell et al. (2018), hormonal

factors, particularly the neuroprotective effects of estrogen in women, play a significant role in reducing inflammation, improving cerebral perfusion, and delaying atherosclerosis [17]. However, after menopause, this protective effect declines, increasing stroke susceptibility in women. In addition, lifestyle-related risk factors such as smoking, dyslipidemia, and physical inactivity are more prevalent in males and further contribute to stroke incidence.

NLR was positively associated with admission NIHSS scores in this sample ($r_s = 0.636$, $p < 0.001$). This observation is compatible with a relationship between systemic inflammation and neurological impairment. Infarct size and subsequent clinical outcomes were not evaluated here, so the observed correlation cannot be extended to those endpoints.

From a pathophysiological perspective, neutrophils are rapidly recruited to ischemic brain tissue and play a central role in secondary brain injury. They release reactive oxygen species (ROS), matrix metalloproteinases (MMPs), and pro-inflammatory cytokines such as TNF-α and IL-6, which damage the blood–brain barrier and exacerbate cerebral edema. This inflammatory cascade amplifies neuronal injury and worsens clinical severity.

Conversely, lymphocyte levels tend to decrease in acute stroke due to stroke-induced immunosuppression (SII). This reduction weakens the anti-inflammatory and neuroprotective response, further contributing to disease severity. Inline with Gill et al. (2018), lymphopenia is associated with poorer clinical outcomes and increased risk of complications in stroke patients [19].

An increased neutrophil-to-lymphocyte ratio may reflect an imbalance between inflammatory and regulatory immune responses. This provides a biological context for the observed association, but the cross-sectional analysis cannot determine whether elevated NLR contributes to neurological injury, reflects the systemic response to a more severe stroke, or shares other underlying determinants with NIHSS.

In contrast, this study did not find a statistically significant association between PC and stroke severity, although a weak positive correlation was observed. This finding is consistent with previous studies suggesting that platelet count alone may not adequately represent platelet activity. Inline with Yang et al. (2021), platelet count is a quantitative parameter and does not reflect functional aspects such as platelet activation, aggregation, and inflammatory mediator release [15].

Platelets are known to play a role in thrombus formation and neuroinflammation through interactions with leukocytes and endothelial cells. However, their contribution to stroke severity is more closely related to functional activity rather than absolute count. Inline with Rawish et al. (2020), platelet-leukocyte interactions and inflammatory signaling pathways are critical in stroke pathophysiology, but these mechanisms cannot be fully captured by platelet count alone [14].

NLR is obtainable from routine blood testing, making its association with admission NIHSS relevant for further investigation. However, a statistically significant NLR correlation and a nonsignificant PC correlation do not demonstrate that NLR is a superior biomarker; the correlations were not formally compared. No sensitivity, specificity, discrimination, calibration, or added clinical value was evaluated. NLR should therefore not replace NIHSS or be used on the basis of this study alone to diagnose stroke, predict prognosis, or guide treatment.

Several limitations affect interpretation. The small sample (n = 32), single-centre setting, and only three participants in the higher-severity group restrict precision and generalizability. Retrospective selection of complete medical records may introduce selection bias if excluded patients differed in illness severity or access to testing; the unavailable screening counts prevent quantification of this bias. Diagnostic confirmation, symptom-onset ascertainment, and assessor training could not be verified uniformly, creating potential misclassification and measurement bias. Blood-sampling times and their temporal relationship to admission NIHSS were not standardized in the available documentation. Because inflammatory parameters and neurological deficits can change during the acute phase, differences in onset-to-sampling and onset-to-assessment intervals may influence the observed association.

Baseline vascular risk factors, comorbidities, previous stroke, prestroke function, and treatments before blood collection were not available for adjustment. Differences in these factors could affect both blood counts and NIHSS, leaving residual confounding. Excluding selected inflammatory conditions does not eliminate all potential confounding. The cross-sectional design does not establish temporal direction or causality, and unadjusted correlations do not show that NLR independently determines severity. Sparse cells limit the categorized Chi-square analyses, and the category thresholds were not validated. Other inflammatory and platelet-function markers were not evaluated. Prospective multicentre studies with larger samples, standardized sampling and trained NIHSS assessors, detailed treatment and baseline data, and multivariable analyses are needed;

longitudinal follow-up and explicit performance evaluation would be required before prognostic or predictive use could be proposed.

4. CONCLUSION

In this retrospective cross-sectional study of 32 ischemic stroke patients, NLR was positively associated with admission NIHSS scores ($r_s = 0.636$, $p < 0.001$). PC was not significantly associated with NIHSS ($r_s = 0.225$, $p = 0.216$). These findings describe unadjusted associations and do not establish that NLR independently determines stroke severity or provides validated diagnostic, prognostic, or predictive information. Clinical neurological assessment remains essential. Larger prospective studies with standardized assessment timing and adjustment for baseline clinical characteristics and treatment are needed to determine the reproducibility and clinical relevance of the association.

ACKNOWLEDGEMENTS

The authors would like to thank Bhayangkara Kendari Hospital for providing access to medical record data and the Faculty of Medicine, Halu Oleo University, for supporting this research.

AUTHOR CONTRIBUTIONS

AS: Conception and design of the study; supervision; critical revision of the manuscript.

MN: Data collection; statistical analysis; drafting of the manuscript.

NIN: Data interpretation; manuscript review.

SW: Methodological guidance; data validation; final approval of the manuscript.

MK: Methodological guidance; data validation; final approval of the manuscript.

IR: Methodological guidance; data validation; final approval of the manuscript.

All authors have read and approved the final version of the manuscript.

CONFLICT OF INTEREST

The authors declare no competing interest.

ETHICS STATEMENT

This study utilized secondary data and ensured patient confidentiality by anonymizing all personal identifiers. Ethical approval was obtained from the Health Research Ethics Committee, Faculty of Medicine, Halu Oleo University, with Ethical Clearance Number 133/UN29.17.1.3/ETIK/2024 prior to data collection.

FUNDING STATEMENT

This work received no specific grant from any funding agency in the public, commercial, or non-profit sectors.

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