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Original Research Article

NEUTROPHIL-TO-LYMPHOCYTE RATIO IN ACUTE ISCHEMIC STROKE: ASSOCIATION WITH STROKE SEVERITY, FUNCTIONAL OUTCOME AND MORTALITY

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ABSTRACT

Background: Acute ischemic stroke (AIS) is a major cause of mortality and long-term disability worldwide. The inflammatory response that follows cerebral ischemia contributes substantially to secondary brain injury. Neutrophils are among the earliest inflammatory cells recruited to the ischemic brain, whereas lymphocyte responses may be associated with immunomodulatory and neuroprotective mechanisms. The neutrophil-to-lymphocyte ratio (NLR), calculated from routinely available complete blood count parameters, has therefore emerged as a simple marker of systemic inflammation. However, its relationship with stroke severity, functional outcome and mortality remains incompletely defined. **Objective:** To evaluate the association between NLR and stroke severity, neurological status, functional outcome and mortality among patients with acute ischemic stroke.

Materials and Methods: A prospective observational study was conducted among 100 adult patients with clinically and radiologically confirmed acute ischemic stroke admitted to the teaching hospitals attached to Sivagangai Medical College, Sivagangai, between August 2023 and July 2024. Patients presenting within 48 hours of symptom onset were enrolled after application of predefined inclusion and exclusion criteria. Clinical assessment included Glasgow Coma Scale (GCS), National Institutes of Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS). Complete blood count and biochemical investigations were performed. NLR was evaluated in relation to neurological severity, functional outcome and mortality. Patients were followed during hospitalization and for one month after stroke.

Results: Among the 100 patients included in the study, 70 were males and 30 were females. The overall mortality was 14%, with 86 patients surviving. NLR demonstrated a significant inverse association with GCS. Patients with GCS <9 had a mean NLR of 3.338 ± 1.042, compared with 1.923 ± 0.787 among those with GCS 13–15 ($p<0.001$). NLR increased with increasing NIHSS severity, although the association at admission was not statistically significant ($p=0.375$). At one-month follow-up, however, NLR was significantly associated with NIHSS severity ($p=0.004$). Similarly, higher NLR was associated with worse functional status at one month; patients with mRS 4–5 had a mean NLR of 3.30 ± 1.17 compared with 2.16 ± 0.90 among those with mRS 0–1 ($p<0.001$). Mortality was significantly associated with higher NLR. The mean NLR was 3.99 ± 0.25 among patients who died compared with 2.17 ± 0.99 among survivors ($p<0.001$). At one-month follow-up, the corresponding values were 3.50 ± 0.43 and 2.24 ± 1.06, respectively ($p=0.005$). NLR was positively associated with neutrophil levels and negatively associated with lymphocyte levels ($p<0.001$ for both).

Conclusion: NLR is a readily available and inexpensive inflammatory marker that demonstrated significant associations with neurological impairment, poor functional outcome and mortality in patients with acute ischemic stroke. Its

relationship with GCS, one-month NIHSS, one-month mRS and mortality suggests that NLR may have prognostic utility in the early assessment of AIS. Larger prospective studies incorporating serial NLR measurements and multivariable analysis are required to establish its independent predictive value.
Keywords: Acute ischemic stroke; neutrophil-to-lymphocyte ratio; NLR; inflammation; NIHSS; Glasgow Coma Scale; modified Rankin Scale; mortality; functional outcome.

INTRODUCTION

Acute ischemic stroke is characterized by sudden neurological dysfunction resulting from focal cerebral, spinal or retinal infarction due to vascular causes.[1] It represents one of the most important neurological emergencies and is a major contributor to death and disability worldwide.[2] Ischemic stroke occurs when blood flow to a region of the brain is interrupted, most commonly because of thrombotic or embolic occlusion of a cerebral artery.[3] The resulting deprivation of oxygen and metabolic substrates initiates a cascade of cellular injury, energy failure and neuronal death.[4] The clinical spectrum of acute ischemic stroke is broad. Patients may experience rapid neurological recovery, persistent neurological disability or death.[5] The burden of stroke is particularly important because a substantial proportion of survivors remain functionally impaired. The uploaded study notes that ischemic stroke accounts for approximately 70% of acute strokes and that more than half of survivors experience some degree of impairment.[6,7] The outcome following acute ischemic stroke is determined not only by the location and extent of the initial vascular occlusion but also by the complex secondary inflammatory response.[8] Cerebral ischemia induces activation of endothelial cells and inflammatory pathways, disruption of the blood-brain barrier and production of reactive and oxidant molecules. This inflammatory response begins within hours of ischemic injury and can contribute to progressive tissue damage.[9] Neutrophils are among the earliest inflammatory cells recruited to the ischemic brain. Their migration into ischemic tissue is accompanied by the release of inflammatory mediators and other substances capable of aggravating tissue injury.[10] Neutrophil accumulation can contribute to microvascular dysfunction, expansion of the infarct and impaired neurological recovery.[11] Conversely, stroke may induce a state of immunosuppression associated with lymphopenia.[12] Certain lymphocyte populations may exert immunomodulatory and potentially neuroprotective effects, and reduction in these populations may contribute to adverse neurological outcomes.[13] The neutrophil-to-lymphocyte ratio combines these two components of the systemic inflammatory response into a single readily available index.[14,15] It can be calculated from the routine complete blood count and requires no specialized laboratory equipment.[16] NLR has been investigated as an inflammatory and prognostic marker in cardiovascular and cerebrovascular diseases.[17] Previous studies have suggested associations between increased NLR and poor outcomes in ischemic stroke, including functional disability and mortality, although findings regarding its independent prognostic significance have not been entirely consistent.[18] The identification of simple and inexpensive prognostic markers is particularly valuable in acute stroke because early assessment of disease severity and prognosis can assist clinical decision-making, monitoring and allocation of healthcare resources.[19] Therefore, the present study was undertaken to evaluate the relationship between NLR and stroke severity, neurological status, functional outcome and mortality among patients with acute ischemic stroke.

MATERIALS AND METHODS

Study Design and Setting

A prospective observational study was conducted among patients diagnosed with acute ischemic stroke (AIS). The study was carried out in the teaching hospitals attached to Sivagangai Medical College, Sivagangai, over a period of one year from August 2023 to July 2024. A total of 100 patients with acute ischemic stroke were enrolled and evaluated clinically, biochemically, and radiologically.

Study Population

Consecutive adult patients admitted with a clinical diagnosis of acute ischemic stroke and presenting within 48 hours of symptom onset were considered for inclusion. The diagnosis of ischemic stroke was confirmed clinically and by computed tomography (CT) of the brain.

Inclusion Criteria

Patients were included if they fulfilled the following criteria:

1. Age ≥18 years.
2. Clinical diagnosis of acute ischemic stroke.
3. Presentation within 48 hours of onset of symptoms.
4. CT-confirmed ischemic stroke.
5. Provision of informed consent.

Exclusion Criteria

Patients were excluded if they were younger than 18 years or had intracranial hemorrhage, malignancy, a history of infection within two weeks preceding stroke onset, hematological disorders, liver

cirrhosis, atrial fibrillation, end-stage renal disease, or were receiving immunosuppressive therapy.

Clinical Assessment

A detailed history and clinical examination were performed for all enrolled patients. Relevant clinical characteristics and risk factors were recorded using a predesigned proforma. Neurological severity was assessed using the Glasgow Coma Scale (GCS) and National Institutes of Health Stroke Scale (NIHSS). GCS and NIHSS were assessed at admission and again at 72–96 hours after stroke onset. Functional disability was evaluated using the modified Rankin Scale (mRS) at discharge and during follow-up.

Assessment of Stroke Severity

Patients were categorized according to their admission NIHSS score as follows: mild stroke (NIHSS 1–4), moderate stroke (5–15), moderate-to-severe stroke (16–20), and severe stroke (21–40).

Laboratory Investigations and NLR Estimation

Peripheral venous blood samples were collected for routine laboratory investigations, including complete blood count, erythrocyte sedimentation rate, serum urea, and creatinine. Serum calcium and fasting blood sugar were also assessed as part of the biochemical evaluation.

The neutrophil-to-lymphocyte ratio (NLR) was calculated from the complete blood count parameters using the following formula:

NLR = Absolute neutrophil count / Absolute lymphocyte count

Neurological and Functional Outcome Assessment

Neurological severity was evaluated using the NIHSS and GCS. Functional outcome was assessed using the modified Rankin Scale, ranging from 0 (no symptoms) to 6 (death). The study assessed functional status during hospitalization and at follow-up.

Radiological Assessment

All patients underwent CT imaging of the brain using a high-speed helical CT scanner. Cerebral infarct volume was estimated from the largest lesion slice. The longest lesion axis was measured as A, and a perpendicular measurement representing the widest dimension was recorded as B. The third dimension (C) was calculated using the number of slices multiplied by slice thickness. Cerebral infarct volume was subsequently estimated using the formula ABC/2. The CT slice thickness was 5 mm.

Follow-up

Patients were followed throughout their hospital stay and during subsequent hospital visits. Follow-up was performed for up to three months according to the study protocol, with the reported outcome analysis including one-month neurological and functional outcomes.

Study Outcomes

The primary outcomes evaluated were the association of NLR with:

- Neurological severity at admission.
- GCS at admission.
- NIHSS at admission and follow-up.
- Functional status assessed using mRS.
- In-hospital mortality.
- Mortality at one-month follow-up.

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS; IBM Analytics). Descriptive and comparative statistical analyses were performed as appropriate for the variables studied. Tables and graphs were prepared using Microsoft Excel. A p-value <0.05 was considered statistically significant.

Ethical Considerations

Informed consent was obtained from patients who fulfilled the eligibility criteria before enrolment. Patient information was collected using a predesigned proforma. The ethics committee approval number and date were not available in the uploaded draft and should be added from the original institutional ethics approval document before journal submission.

RESULTS

A total of 100 patients with acute ischemic stroke were included in the prospective study. Of these, 70 (70%) were males and 30 (30%) were females. During the study period, 86 patients survived and 14 patients died, giving an overall mortality of 14%. The majority of patients were aged >60 years (56%), followed by those aged 51–60 years (22%), 41–50 years (12%), and <40 years (10%). There was no statistically significant association between age and mortality (p=0.855). Similarly, gender was not significantly associated with mortality (p=0.345). [Figure 1]

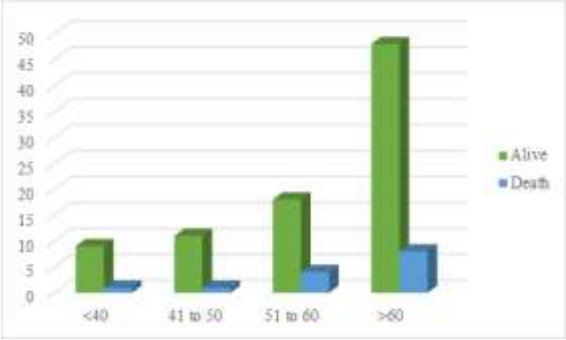

Figure 1: Age and gender distribution and mortality outcomes among patients with acute ischemic stroke

Association of NLR with Clinical Characteristics

The mean NLR was higher among patients with hemiplegia (2.34 ± 1.09) than among those without hemiplegia (1.95 ± 0.68); however, this difference was not statistically significant (p=0.392) (Figure 2). The mean NLR increased with increasing admission NIHSS score. Patients with NIHSS <10 had a mean NLR of 1.83 ± 0.41, compared with 2.33 ± 1.13 among those with NIHSS 11–30 and 2.42 ± 1.08 among those with NIHSS >30. Although there was

an increasing trend, the association was not statistically significant (p=0.375). Similarly, patients with higher mRS scores had higher mean NLR values. The mean NLR was 2.12 ± 0.97 among patients with mRS 2–3 and 2.41 ± 1.12 among those with mRS 4–5. This difference was not statistically significant (p=0.205).

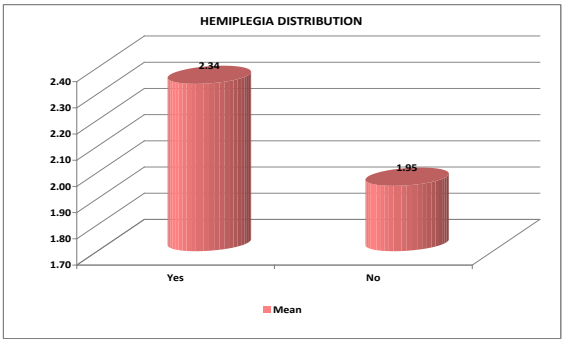

Figure 2: Association of neutrophil-to-lymphocyte ratio (NLR) with clinical characteristics

Association of NLR with Glasgow Coma Scale

A statistically significant inverse association was observed between GCS and NLR (p<0.001). Patients with GCS <9 had a mean NLR of 3.338 ± 1.042, while those with GCS 9–12 and 13–15 had mean NLR values of 2.068 ± 0.937 and 1.923 ± 0.787, respectively. Thus, NLR progressively increased as GCS decreased (Table 1). The highest NLR was observed among patients with severe impairment of consciousness, whereas patients with preserved consciousness had lower NLR values.

Table 1: Association between GCS and NLR

GCS category	n	Mean NLR	SD	p-value
<9	21	3.338	1.042	**<0.001**
9–12	66	2.068	0.937	
13–15	13	1.923	0.787	
Total	**100**			

Association of NLR with Neutrophil and Lymphocyte Counts

A statistically significant positive association was observed between neutrophil count and NLR (p<0.001). Mean NLR increased from 1.42 ± 0.25 among patients with neutrophil values <3.0 to 2.35 ± 1.05 among those with values 3.1–5.0 and 3.25 ± 0.83 among those with values >5.0 (Table 2). Conversely, a significant inverse association was observed between lymphocyte count and NLR (p<0.001). Patients with lymphocyte values <1.5 had a mean NLR of 3.07 ± 1.07, whereas patients with lymphocyte values >2.5 had a lower mean NLR of 1.58 ± 0.38.

Table 2: Neutrophil and lymphocyte counts in relation to NLR

Parameter	Category	Mean NLR ± SD	p-value
Neutrophil	<3.0	1.42 ± 0.25	<0.001
	3.1–5.0	2.35 ± 1.05	
	>5.0	3.25 ± 0.83	
Lymphocyte	<1.5	3.07 ± 1.07	<0.001
	1.6–2.5	2.08 ± 0.96	
	>2.5	1.58 ± 0.38	

NLR and One-Month Neurological Outcome

At one-month follow-up, a statistically significant association was observed between NIHSS category and NLR (p=0.004). The mean NLR was 2.008 ± 0.887 in patients with mild stroke (NIHSS 1–4), 2.064 ± 0.945 in those with moderate stroke (NIHSS 5–15), 2.343 ± 1.112 among patients with NIHSS 16–20, and 3.019 ± 1.147 among patients with NIHSS 21–40 (Table 3). The findings demonstrate a progressive increase in NLR with increasing neurological severity at one-month follow-up.

Table 3: NLR according to NIHSS at one-month follow-up

NIHSS category	Severity	n	Mean NLR	SD
1–4	Mild	13	2.008	0.887
5–15	Moderate	45	2.064	0.945
16–20	Severe	21	2.343	1.112
21–40	Very severe	21	3.019	1.147
Total		**100**		

NLR and Functional Outcome at One Month

A significant association was observed between NLR and functional disability at one-month follow-up (p<0.001). Patients with mRS 0–1 had a mean NLR of 2.16 ± 0.90, while those with mRS 2–3 had a similar mean NLR of 2.16 ± 1.03. In contrast,

patients with severe functional disability (mRS 4–5) had a markedly higher mean NLR of 3.30 ± 1.17 (Table 4). Thus, higher NLR was associated with poorer functional outcome at one month.

Table 4: NLR according to mRS at one-month follow-up

mRS category	Mean NLR	SD	p-value
0–1	2.16	0.90	<0.001
2–3	2.16	1.03	
4–5	3.30	1.17	

NLR and Mortality

A strong statistically significant association was observed between NLR and mortality. Among the 86 patients who survived, the mean NLR was 2.17 ± 0.99, whereas the 14 patients who died had a substantially higher mean NLR of 3.99 ± 0.25 (p<0.001) (Table 5). The mean NLR among patients who died was considerably higher than that among survivors, suggesting an association between increased systemic inflammatory response and mortality in acute ischemic stroke.

Table 5: NLR according to in-hospital outcome

Outcome	n	Mean NLR	SD	p-value
Alive	86	2.17	0.99	<0.001
Death	14	3.99	0.25	

NLR and Mortality at One-Month Follow-up

The association between NLR and mortality remained statistically significant at one-month follow-up. The mean NLR was 2.24 ± 1.06 among survivors compared with 3.50 ± 0.43 among patients who died (p=0.005) (Table 6). Overall, the study demonstrated that higher NLR was associated with impaired consciousness, greater neurological severity at one-month, poorer functional outcome, and increased mortality. The strongest statistically significant associations were observed for GCS, one-month NIHSS, one-month mRS, neutrophil count, lymphocyte count, and mortality.

Table 6: NLR according to outcome at one-month follow-up

Clinical Outcome Measured	Mean ± SD of Outcome	Correlation Coefficient (*r*)	Statistical Significance (*p*-value)	Clinical Interpretation
Duration of ICU Stay	2.64 ± 1.75 days	**+0.504**	**< 0.001**	Moderate positive correlation; highly significant
Duration of Hospital Stay	6.54 ± 1.51 days	+0.124	0.212	Weak positive correlation; statistically non-significant

DISCUSSION

Acute ischemic stroke (AIS) is associated with a complex inflammatory response that contributes to secondary neuronal injury. The neutrophil-to-lymphocyte ratio (NLR), calculated from routinely available complete blood count parameters, reflects the balance between neutrophil-mediated inflammation and lymphocyte-mediated immune responses. In the present study of 100 patients with AIS, higher NLR was significantly associated with impaired consciousness, poor neurological outcome at one month, severe functional disability, and mortality. These findings support the potential role of NLR as a simple and inexpensive prognostic biomarker in patients with acute ischemic stroke. In the present study, a significant inverse association was observed between NLR and Glasgow Coma Scale (GCS). Patients with GCS <9 had a mean NLR of 3.338 ± 1.042, whereas patients with GCS 13–15 had a mean NLR of 1.923 ± 0.787 (p<0.001). This finding indicates that patients with more severe impairment of consciousness had greater systemic inflammatory activation. Following cerebral ischemia, inflammatory mediators promote leukocyte recruitment to the ischemic region. Activated neutrophils can release reactive oxygen species, proteolytic enzymes and inflammatory mediators, thereby contributing to endothelial dysfunction, blood-brain barrier disruption and secondary cerebral injury.[20,21] The association between NLR and neurological severity was also evident from the NIHSS analysis. Although NLR increased progressively with increasing admission NIHSS, from 1.83 ± 0.41 in patients with NIHSS <10 to 2.42 ± 1.08 in those with NIHSS >30, the difference was not statistically significant (p=0.375). In contrast, at one-month follow-up, NLR was significantly associated with NIHSS severity (p=0.004). Mean NLR increased from 2.008 ± 0.887 among patients with mild neurological impairment to 3.019 ± 1.147 among those with very severe impairment. This finding suggests that NLR may have greater prognostic relevance for subsequent neurological recovery than for initial stroke severity. Our findings are consistent with previous research. Wang et al. reported that higher NLR was associated with poor outcomes after acute ischemic stroke and that elevated NLR predicted three-month death or disability.[12] Similarly, Zhu et al. demonstrated that NLR was associated with early clinical outcomes in patients with acute ischemic stroke and suggested

that inflammatory markers may provide additional prognostic information beyond conventional clinical assessment.[22] The present study demonstrated a significant relationship between NLR and functional outcome. At one-month follow-up, patients with mRS 0–1 had a mean NLR of 2.16 ± 0.90, whereas patients with severe functional disability (mRS 4–5) had a substantially higher mean NLR of 3.30 ± 1.17 ($p<0.001$). This finding indicates that increased inflammatory activity is associated with poorer functional recovery. These observations are supported by the meta-analysis conducted by Song et al., which included 37 studies involving 43,979 participants. The authors reported that higher baseline NLR was significantly associated with unfavorable functional outcomes in patients with ischemic stroke.[2] Zhang et al. also reported a significant association between elevated NLR and death or disability at three months following acute ischemic stroke.[7] Thus, the findings of the present study are consistent with the existing literature demonstrating the prognostic importance of NLR in functional recovery after AIS.

The strongest association observed in our study was between NLR and mortality. The mean NLR was 3.99 ± 0.25 among patients who died compared with 2.17 ± 0.99 among survivors ($p<0.001$). At one-month follow-up, NLR remained significantly higher among patients who died than among survivors (3.50 ± 0.43 vs. 2.24 ± 1.06; $p=0.005$). These findings suggest that marked systemic inflammatory activation may be associated with increased risk of death following AIS. Previous meta-analyses have also demonstrated an association between elevated NLR and mortality after ischemic stroke. Song et al. reported that increased baseline NLR was significantly associated with mortality in patients with ischemic stroke.[2] A further systematic review and meta-analysis similarly demonstrated that elevated NLR was associated with increased mortality and poor clinical outcomes following stroke.[7] These findings strengthen the observation in the present study that NLR may have prognostic value in identifying patients at increased risk of mortality. The relationship between NLR and outcome may be explained by the individual contributions of neutrophils and lymphocytes. In our study, NLR demonstrated a significant positive association with neutrophil count ($p<0.001$). Mean NLR increased from 1.42 ± 0.25 among patients with lower neutrophil values to 3.25 ± 0.83 among those with higher neutrophil values. Neutrophils are rapidly recruited following cerebral ischemia and may contribute to microvascular obstruction, endothelial injury, oxidative stress and infarct expansion.[20,21] Conversely, NLR demonstrated a significant inverse association with lymphocyte count ($p<0.001$). Patients with lymphocyte values <1.5 had a mean NLR of 3.07 ± 1.07, whereas those with lymphocyte values >2.5 had a mean NLR of 1.58 ± 0.38. Lymphopenia is commonly observed following acute stroke and may reflect stroke-associated immune dysregulation. Therefore, elevation of NLR may reflect both increased neutrophil-mediated inflammation and reduced lymphocyte-mediated immune regulation.[21,12] The prognostic importance of NLR has also been demonstrated among patients receiving reperfusion therapy. Sharma et al. performed a systematic review and meta-analysis and reported that NLR was associated with outcomes following reperfusion therapy in acute ischemic stroke.[8] Similarly, a systematic review of patients receiving intravenous thrombolysis found that elevated NLR was associated with poor functional outcome and hemorrhagic complications.[23] These findings indicate that NLR may provide additional prognostic information even in patients receiving active reperfusion treatment.

The timing of NLR measurement may also influence its prognostic performance. NLR is a dynamic inflammatory marker that can change during the acute and subacute phases of stroke. A meta-analysis evaluating dynamic NLR demonstrated that changes in NLR after reperfusion therapy were associated with clinical outcomes.[24] Therefore, serial assessment of NLR may potentially provide more prognostic information than a single measurement at admission. The clinical importance of NLR lies in its simplicity and availability. NLR can be calculated from a routine complete blood count without specialized laboratory equipment or additional expenditure. This may be particularly useful in resource-limited healthcare settings. However, NLR should be regarded as an adjunctive biomarker rather than a replacement for established clinical assessment tools such as GCS, NIHSS and mRS. The present study has several limitations. The sample size was relatively small and the study was conducted at a single centre. Multivariable regression analysis was not performed; therefore, it cannot be established whether NLR was an independent predictor of mortality or functional outcome after adjustment for age, baseline NIHSS, diabetes, hypertension and other potential confounding factors. In addition, receiver operating characteristic analysis was not performed to determine an optimal NLR cutoff value. Serial changes in NLR were also not comprehensively evaluated. Overall, the findings of the present study are consistent with previous cohort studies and systematic reviews demonstrating that elevated NLR is associated with poor functional outcome and mortality following AIS. The significant associations observed between NLR and GCS, one-month NIHSS, one-month mRS and mortality further support its potential value as a readily available prognostic marker. Larger multicentre prospective studies incorporating serial NLR measurements, standardized cutoff values and multivariable prediction models are required to establish its independent prognostic utility.

Limitations

Several limitations should be considered when interpreting the findings. First, the study included only 100 patients and was conducted at a single medical college-associated hospital setting. Therefore, the findings may not be generalizable to all populations. Second, although significant associations were observed, the available analysis does not demonstrate whether NLR is an independent predictor of mortality or functional outcome after adjustment for potential confounding factors such as age, baseline NIHSS, diabetes, hypertension, infarct volume and other clinical variables. Third, the available manuscript does not provide a multivariable regression analysis, receiver operating characteristic curve, area under the curve, sensitivity, specificity or an optimal NLR cut-off value. Therefore, it is not possible from the present dataset description to establish a clinically validated NLR threshold for predicting mortality. Fourth, the study primarily reports NLR measurements and their associations with outcomes; serial changes in NLR are not sufficiently detailed to determine whether dynamic changes provide additional prognostic information. Finally, an internal discrepancy exists in the source document concerning the statistical association between diabetes and mortality. One table reports p=0.035, whereas the subsequent results summary reports p=0.346. This should be verified against the original statistical output before submission.

Clinical Implications

NLR has several characteristics that make it attractive as a potential adjunctive prognostic marker in acute ischemic stroke. It is inexpensive, rapidly available and based on parameters routinely obtained during the initial evaluation of hospitalized patients. In this study, higher NLR was associated with lower GCS, worse neurological status at one-month, greater functional disability and increased mortality. Therefore, patients presenting with elevated NLR may warrant particularly careful neurological monitoring and assessment for complications. Nevertheless, NLR should be interpreted within the clinical context. Because the present study does not establish an independent predictive threshold, treatment decisions should not be based on NLR alone.

CONCLUSION

The present prospective study demonstrates that the neutrophil-to-lymphocyte ratio is associated with important clinical outcomes in patients with acute ischemic stroke. Higher NLR was significantly associated with poorer neurological status as reflected by lower GCS, greater neurological severity at one-month follow-up, poorer functional outcome according to mRS and increased mortality. The mean NLR was substantially higher among patients who died than among survivors, both during hospitalization and at one-month follow-up. These findings suggest that NLR may serve as a simple, inexpensive and readily available inflammatory marker for risk stratification in acute ischemic stroke. However, larger multicentre prospective studies with serial NLR measurements and appropriately adjusted multivariable analyses are required to determine whether NLR independently predicts mortality and functional outcome and to establish clinically useful cut-off values.

Ethics Approval

The study was conducted after obtaining informed consent from eligible participants. The ethics committee approval number and date were not provided in the available draft and should be inserted from the original institutional approval documentation.

Consent for Participation

Informed consent was obtained from the study participants before enrolment, as stated in the study protocol.

Author Contributions

All authors contributed equally to the conception and design of the study, data collection, analysis and interpretation of data, drafting and revision of the manuscript, and approval of the final version for publication. All authors agree to be accountable for all aspects of the work and have approved the final manuscript.

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