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A Prospective Study of Role of Neutrophil-Lymphocyte Ratio in Short-Term Mortality in Patients with Stroke

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KEYWORDS

Neutrophil-lymphocyte ratio (NLR), Stroke, Short-term mortality, Inflammation, Risk stratification, Acute ischemic stroke.

ABSTRACT:

The Neutrophil-Lymphocyte Ratio (NLR) has emerged as a promising biomarker for predicting the onset and severity of stroke, one of the leading causes of morbidity and mortality worldwide. As an easily obtainable and cost-effective parameter derived from routine blood tests, NLR reflects the balance between systemic inflammation (neutrophils) and immune regulation (lymphocytes). This ratio has been shown to correlate with the severity of stroke, particularly ischemic stroke, where inflammation plays a critical role in pathogenesis. However, its specific role in predicting short-term mortality in stroke patients remains fully underexplored. This study aims to evaluate the utility of NLR effectiveness as an early predictor of 30-day mortality in patients presenting with acute stroke, potentially aiding in better risk assessment and clinical decision-making.

MATERIALS AND METHODS: This prospective cohort study was conducted at a tertiary care hospital, enrolling patients aged 18 years and older who were admitted with a diagnosis of acute ischemic or transient ischemic stroke. Exclusion criteria included patients with pre-existing inflammatory diseases, recent infections, or those on immunosuppressive therapy.

RESULTS: Eighty stroke patients were monitored throughout their hospital stay in this prospective cohort study. NLR was calculated soon after admission. Elevated NLR on admission was significantly associated with increased 30-day mortality. The findings indicate that NLR is an independent predictor of short-term mortality in stroke patients. This suggests that systemic inflammation, as reflected by NLR, plays a significant role in the early outcomes of stroke. Given the ease of obtaining NLR from routine blood tests, it could be readily incorporated into clinical practice for early risk stratification.

CONCLUSION: The neutrophil-lymphocyte ratio is a valuable predictor of short-term mortality in patients with acute stroke. Elevated NLR at admission is associated with a higher risk of death within 30 days, underscoring its potential as an early, accessible biomarker for guiding clinical management. Further studies are recommended to validate these findings in larger, more diverse populations and to explore the underlying mechanisms linking NLR to stroke prognosis.

INTRODUCTION

Stroke or Cerebrovascular accident is the sudden onset of a neurological deficit. It is the third leading cause of death and a major cause of disability worldwide. The global incidence of stroke has increased over the last few decades, with the most common subtype being ischemic stroke[1,2]. Studies have therefore assessed the ability of various biological markers to predict stroke prognosis. Inflammatory markers have been reported to be predictors of stroke[3,4,5]. Inflammation occurs when stagnant blood flow due to ischemic or hemorrhagic lesions results in the release and accumulation of pro-inflammatory mediators, which lead to the migration of neutrophils to the area of stroke. Various types of inflammatory cells, including neutrophils, lymphocytes, and monocytes, are recruited to ischemic brain tissues, where they produce several types of inflammatory

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mediators. Both proinflammatory and anti-inflammatory mediators are involved in the pathogenesis of ischemic stroke, an imbalance of which leads to inflammation[6]. Thus, the combined increase in neutrophils and decrease in lymphocytes during inflammation manifests as an increased neutrophil-to-lymphocyte ratio (NLR). NLR is an emerging biomarker for assessing the systemic inflammatory status of an individual. In this study, the association between NLR and stroke will be evaluated, which helps to assess the morbidity and mortality of stroke, as NLR is an easy and cost-effective investigation[7, 8].

Ischemic brain damage is linked to post-stroke inflammation. Stroke patients' prognosis and clinical results may be impacted by their inflammatory condition [9]. Even while research has assessed the processes behind immunological and inflammatory reactions following acute ischemic stroke, the specific pathogenic pathways are still not fully understood. After ischemic brain injury, the pathophysiology of the inflammatory response depends on the activation of many kinds of inflammatory cells, such as neutrophils, macrophages, lymphocytes, and microglia [10]. The release of inflammatory mediators from ischemic brain tissues encourages endothelial cells to express adhesion molecules, which in turn attract leukocytes to injured brain lesions [11, 12]. Leukocyte infiltration can be accelerated by the cytokines produced by microglial cells during the acute phase of ischemic brain damage [13, 14]. After a stroke, inflammation is linked to the development of ischemic brain injury. During the subacute phase of ischemic brain injury, neutrophils first type of leukocytes to penetrate damaged brain tissue-produced toxic amounts of reactive oxygen species and inflammatory mediators, which further accelerated the inflammatory process. A key player in hemorrhagic transformation and blood-brain barrier disruption, matrix metalloproteinase-9 (MMP-9) is one of these inflammatory mediators [15]. Following an acute ischemic stroke, the inflammatory process progresses and involves various types of lymphocytes. Specifically, by generating anti-inflammatory cytokines and inhibiting additional inflammatory processes, regulatory T cells have neuroprotective benefits on injured ischemic brain lesions.Interleukin-10 (IL-10), which is produced by regulatory T cells, inhibits post-ischemic inflammatory pathways by downregulating pro-inflammatory cytokines [16]. NLR is a systemic inflammatory biomarker that reflects the balance between circulating neutrophils and lymphocytes. NLR at hospital admission has been shown to correlate positively with the NIHSS scores of acute ischemic stroke patients, with a higher NLR being associated with an increased risk of 60-day mortality [17]. An increased NLR was also shown to be associated with unfavorable outcomes on the modified Rankin Scale 3 months after stroke onset, as well as with unfavorable outcomes on the modified Barthel Index at discharge [18,19,20]. Cognitive impairment 3 months after stroke onset was found to correlate with higher NLR levels [21]. A recent systematic review and meta-analysis revealed that early neurological deterioration in stroke patients was associated with a higher NLR[22]. A recent systematic review and meta-analysis revealed that early neurological deterioration in stroke patients was associated with a higher NLR [23]. To our knowledge, the present study is the first to demonstrate the relationship between NLR and physical performance in patients with acute ischemic stroke by incorporating the Berg Balance Scale (BBS) and the Manual Function Test (MFT). Several studies have shown a relationship between physical function and inflammatory biomarkers in older people. Higher levels of C-reactive protein (CRP) and IL-6 in people aged over 65 years were associated with poor physical performance, as determined by walking speed, chair-stand tests, and standing balance tests, along with decreased hand-grip strength (HGS) [24]. Higher CRP levels were linked to worse chair-stand performance and HGS loss in hospitalized older adults, indicating that inflammation is linked to a decline in hand power and physical function. This result is in line with the current findings that indicate correlations between NLR and MFT and BBS scores. Lower muscle mass and strength have been linked to greater levels of inflammation in adults, which may affect hand function and general balance. The present study found negative relationships between NLR and BBS and MFT scores, which may help explain. A connection between inflammation and cognitive performance has also been shown in other clinical investigations.[25]

The present study found negative relationships between NLR and BBS and MFT scores, which may help explain. A connection between inflammation and cognitive performance has also been shown in other clinical investigations. Interleukin-12 (IL-12) levels were shown

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to be raised in ischemic stroke patients with cognitive loss, and greater IL-6 levels were linked to worse cognitive performance as measured by the MMSE in the Northern Manhattan Study. All of these findings point to a possible relationship between inflammation level and both cognitive and physical function[26].

MATERIALS AND METHODS

This is a prospective study of 80 patients, which included all patients who had been diagnosed with acute ischemic stroke and transient ischemic stroke and admitted to the Malla Reddy hospital, Suraram, Hyderabad, Telangana, India, from December 2023 to June 2024. All Patients who were above 18 years and had a clinical and CT or MRI confirmed diagnosis of stroke who were admitted to the hospital were included in this study. Patients with ischemic stroke were included in this study. Patients below 18 years; patients with stroke due to trauma/neoplasm/active infection/ immunosuppressive agents/hematological disease; patients with previous history of cerebrovascular accident or stroke; patients with incomplete data; patients who did not consent to participate in the study; patients with incomplete or lacking medical, demographic, clinical laboratory, and radiological data. Institutional ethical clearance was obtained from the Ethics Committee of the Malla Reddy Hospital, Suraram, Hyderabad, Telangana. All patients participating in this study were informed in detail about the purpose of the study undertaken, and informed consent was obtained from all patients.

METHODOLOGY

The National Institutes of Health Stroke Scale (NIHSS) score was calculated at the time of admission. An NIHSS score of ≤ 5 was determined to be a mild stroke, while an NIHSS score of >21 was determined to be a severe stroke. A sample of whole blood (2 ml) was collected at the time of admission or within 24 hours of admission from each patient and analyzed within one hour of collection. A complete blood count was obtained from which NLR was determined and correlated with NIHSS to assess the inflammatory status and pathogenesis of brain damage. The assessment of brain damage pathogenesis in stroke commonly involves the utilization of Computed Tomography (CT-scan) or Magnetic Resonance Imaging (MRI). The patients were observed for the entire duration of their hospital stay and followed up for 30 days to assess the short-term mortality rate.

Ethical Approval: The study was carried out according to the Declaration of Helsinki and approved by the Institutional Ethics Committee of the Malla Reddy Institute of Medical Sciences -MRIMS (2024/172)

Informed Consent Form: The written Informed Consent form was obtained from the patients.

Comprehensive Data Collection: Our study collects a variety of patient information, including age, gender, comorbidities, and clinical parameters such as NLR. This comprehensive approach can provide a more holistic understanding of the factors influencing stroke.

Location of Study: Conducting the study at a tertiary care teaching hospital adds credibility to the research, as such institutions often deal with a diverse range of cases and have access to advanced medical expertise.

Data Analysis: Statistical analysis was carried out with Microsoft Excel. By using IBM SPSS software results were analyzed to conclude. Thereafter, stratification of the results was done, and the detailed report was prepared and submitted.

LIMITATIONS

Sample Size and Generalizability: A sample size of 80 patients might not fully represent the diversity and complexity of stroke cases. The findings might not be directly generalizable to broader populations or other healthcare settings.

Single-Centre Study: Conducting the study in a single hospital might limit the diversity of patient demographics and disease severity, potentially impacting the external validity of the results.

Blood sample collection: NLR was measured only at the time of hospital admission; dynamic changes in NLR reflecting the time course of neuroinflammation were not evaluated.

Comorbidity Confounding: The presence of comorbidities in patients could complicate the analysis of factors influencing stroke. Controlling for the impact of various comorbidities on study outcomes might be challenging.

Limited Follow-up Period: A study period of only 6 months might not capture long-term outcomes, such as cognitive function and mortality rate, that could result from acute ischemic stroke.

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Selection Bias: The exclusion criteria might unintentionally exclude certain subsets of patients, potentially affecting the study's external validity.

Limited Intervention Information: The study does not provide information about interventions, treatments, or therapies given to patients. This could impact the understanding of patient outcomes and the effectiveness of different interventions.

Ethnic and Socioeconomic Diversity: The study might lack representation from diverse ethnic and socioeconomic backgrounds, which could limit the generalizability of findings to a broader population.

RESULTS

This prospective cohort study involved a total of 80 stroke patients whose diagnosis was confirmed by CT or MRI. The results of the present study show that age is also a confounding factor of stroke. The distribution of patients based on age groups revealed that most of the patients, n= 95% were older than 41 years, with 46% of the whole sample being older than 60 years of age, where the mean (±SD) age group was found to be 59.7 (±11.9). Although it shows an insignificant p-value, age plays a major role in cognitive performance and functional outcomes post-stroke. Other risk factors, including history of smoking, alcohol, diabetes, hypertension, dyslipidaemia, and coronary artery disease, were also compared with the functional outcome of stroke, but they were observed to be statistically significant. The mean (±SD) systolic and diastolic blood pressure was found to be 150.5 (±26.1) and 89.9 (±16.7) respectively with the mean (±SD) systolic blood pressure being 148.0 (±24.9) among survivors, 163.2 (±29.3) among non survivors and diastolic blood pressure being 88.5 (±16.4) among survivors, 96.9 (±17.5) among non survivors. The mean time duration of hospital admission and duration of hospital stay also showed a significant difference between survivors and non-survivors. However, the statistical analysis showed a significant p-value. It is observed that GCS also has a strong correlation with short-term mortality in patients with stroke. The lower the GCS, the higher the risk. The mean (±SD) GCS of survivors is observed to be 13.3(±2.3), which is greater than that of non-survivors of 10.3(±3.4) with a statistically significant p value. As neutrophils and lymphocytes are part of the immune system, the WBC count is also considered to be statistically significant. The mean (±SD) WBC count of survivors is 10461.8 (±3373.8) and non non-survivors is 16130.8 (±3292.0), whereas the overall mean (±SD) WBC count is 11383.0 (±3947.8).

This study found a strong correlation between NLR and NIHSS with the short-term mortality, which is observed to be statistically significant with a p-value of <0.001. The mean (±SD) NLR among survivors is found to be 5.0 (±2.7), and in non-survivors it is 13.0 (±4.0). The mean (±SD) NIHSS among survivors is found to be 7.8 (±4.7), and in non-survivors it is 27.9 (±7.0).

The study included a total of 80 stroke patients, all of whom had their diagnoses confirmed via CT or MRI imaging. The analysis focused on the distribution of patients across different age groups, revealing that a significant majority (95%) were older than 41 years, with 46% ofthe sample being over 60 years of age. The mean age of the cohort was calculated to be 59.7 years (±11.9).

Table 1: Comparison of different factors with mortality at the hospital

Hospital Mortality	Dead	Alive	p-value
	Mean ± SD	Mean ± SD	
NLR	13.0 ± 5.42	5.7 ± 3.42	0.00002
NIHSS	27.7 ± 9.11	9.45 ± 7.3	0.0000002
GCS	12.1 ± 3.34	12.89 ±2.65	0.488
Time duration	58.0 ± 58.6	31.9 ± 42.9	0.141
Duration of hospital stay	10.7 ± 8.69	8.81 ± 5.5	0.41

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Table 1 Inference: NLR and NIHSS show a strong positive correlation with the mortality rate at the hospital, with an average value of 13.0 ± 5.42 and 27.7 ± 9.11, and a highly significant p value of 0.00002 and 0.0000002, respectively.

Table 2: Comparison of different factors with mortality at 1 month

Mortality at 1 Month	Dead	Alive	p-value
	Mean ± SD	Mean ± SD	
NLR	13.0 ± 1.6	5.79 ± 3.8	0.00002
NIHSS	28.2 ± 4.3	9.7 ± 7.82	0.0000002
GCS	8.2 ± 1.94	13.2 ± 2.4	0.000003
Time duration	22 ± 25.4	35.2 ± 45.8	0.491
Duration of hospital stay	12.2 ± 3.9	8.7 ± 5.9	0.162

Table 2 Inference: NLR and NIHSS show a strong positive correlation with the mortality rate at one month, with an average value of 13.0 ± 1.6 and 28.2 ± 4.3, and a highly significant p value of 0.00002 and 0.0000002 respectively.

Table 3: overall comparison

Variable	OUTCOME (n=80)				p-value
	ALIVE (n=67)		DEAD (n=13)		
	Mean	SD	Mean	SD	
Age	58.72	11.27	64.5	14.42	0.135
Time (hours)	32.78	44.2	41.4	48.3	0.284
SBP	148.03	24.9	163.2	29.3	0.058
DBP	88.51	16.35	96.9	17.5	0.097
Hospital stays	8.51	5.54	11.4	6.68	0.119
WBC	10462	3374	16130.8	3292	<0.000*
NLR	5.04	2.7	13	3.96	<0.000*
NIHSS	7.78	4.67	27.9	7.01	<0.000*
GCS	13.31	2.28	10.3	3.38	0.004*

Table 3 Inference: NLR and NHSS show a significant p-value, indicating that NLR is higher in patients with ischemic stroke compared to transient stroke.

DISCUSSION

The results of the present study can be explained by the underlying inflammatory processes that occur after acute ischemic stroke. Neuroinflammation after stroke can have a detrimental effect on the early-stage progression of ischemic brain injury.[27] This neuroinflammation is mediated by the disruption of the blood-brain barrier, which leads to brain oedema and secondary ischemic brain damage. NLR, which reflects the combined activities of neutrophils and lymphocytes, is an indicator of the inflammatory status of ischemic stroke patients. A higher NLR is associated with a more severe degree of inflammation, which may result in poorer functional outcomes.[28,29]

Ultimately, this combined increase in neutrophils and decrease in lymphocytes during inflammation manifests

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as an increased NLR. NLR has been established to be higher in patients with ischemic stroke compared to patients with transient ischemic attacks. Elevated NLR values in patients with ischemic stroke have been linked to a higher 30-day mortality rate and an extended duration of hospital stays.[29]

CONCLUSION

The neutrophil to lymphocyte ratio at hospital admission was able to predict the mortality at one month. This study has found that the mean NLR calculated within 24 hours of hospitalization was higher among patients who died than among those who survived. If calculating NLR at the early stages of stroke-induced inflammation can help predict a poor prognosis, patients presenting with a high NLR at admission can be prioritized for targeted treatment, thereby potentially reducing mortality and post-stroke complications. It is concluded that there's a strong association between NLR and NIHSS, which makes NLR a potential biomarker in predicting the severity of stroke.

REFERENCES:

1. Donkor ES. Stroke in the21stCentury: A snapshot of the burden, epidemiology, and quality of life. Stroke Res Treat [Internet]. 2018;2018:1–10
2. Pandian JD, Sudhan P. Stroke epidemiology and stroke care services in India. J Stroke [Internet]. 2013;15(3):128.
3. Zhang X-G, Xue J, Yang W-H, Xu X-S, Sun H-X, Hu L, et al. Inflammatory markers as independent predictors for stroke outcomes. Brain Behav [Internet]. 2021;11(1).
4. Jin R, Yang G, Li G. Inflammatory mechanisms in ischemic stroke: role of inflammatory cells. J Leukoc Biol [Internet]. 2010;87(5):779–89.
5. Quan K, Wang A, Zhang X, Wang Y. Leukocyte count and adverse clinical outcomes in acute ischemic stroke patients. Front Neurol [Internet]. 2019;10.
6. Kim M-S, Heo MY, Joo HJ, Shim GY, Chon J, Chung SJ, et al. Neutrophil-to-lymphocyte ratio as a predictor of short-term functional outcomes in acute ischemic stroke patients. Int J Environ Res Public Health [Internet]. 2023;20(2):898.
7. Sharath M, Siddegowda RB, Lonimath A, Cheluvaiah JD. An elevated neutrophil-to-lymphocyte ratio predicts in-hospital mortality in stroke patients: A prospective cohort study. Cureus [Internet]. 2022;
8. Tokgoz S, Kayrak M, Akpinar Z, Seyithanoğlu A, Güney F, Yürüten B. Neutrophil lymphocyte ratio as a predictor of stroke. J Stroke Cerebrovasc Dis [Internet]. 2013;22(7):1169–74.
9. Liu C-C, Ko H-J, Liu W-S, Hung C-L, Hu K-C, Yu L-Y, et al. Neutrophil-to-lymphocyte ratio as a predictive marker of metabolic syndrome. Medicine (Baltimore) [Internet]. 2019;98(43):e17537.
10. Buonacera A, Stancanelli B, Colaci M, Malatino L. Neutrophil to lymphocyte ratio: An emerging marker of the relationships between the immune system and diseases. Int J Mol Sci [Internet]. 2022;23(7):3636.
11. McColl BW, Allan SM, Rothwell NJ. Systemic infection, inflammation and acute ischemic stroke. Neuroscience [Internet]. 2009;158(3):1049–61.
12. Amantea D, Nappi G, Bernardi G, Bagetta G, Corasaniti MT. Post-ischemic brain damage: pathophysiology and role of inflammatory mediators: Neuroinflammatory mediators in brain ischemia. FEBS J [Internet]. 2009;276(1):13–26.
13. Yilmaz G, Granger DN. Cell adhesion molecules and ischemic stroke. Neurol Res [Internet]. 2008;30(8):783–93.
14. del Zoppo GJ, Milner R, Mabuchi T, Hung S, Wang X, Berg GI, et al. Microglial activation and matrix protease generation during focal cerebral ischemia. Stroke [Internet]. 2007;38(2):646–51
15. da Fonseca ACC, Matias D, Garcia C, Amaral R, Geraldo LH, Freitas C, et al. The impact of microglial activation on blood-brain barrier in brain diseases. Front Cell Neurosci [Internet]. 2014;8.
16. Kriz J. Inflammation in ischemic brain injury: Timing is important. Crit Rev Neurobiol [Internet]. 2006;18(1–2):145–57.
17. Castellanos M, Leira R, Serena J, Pumar JM, Lizasoain I, Castillo J, et al. Plasma metalloproteinase-9 concentration predicts hemorrhagic transformation in acute ischemic stroke. Stroke [Internet]. 2003;34(1):40–6.
18. Liesz A, Suri-Payer E, Veltkamp C, Doerr H, Sommer C, Rivest S, et al. Regulatory T cells are key cerebroprotective immunomodulators in acute

Journal of Chemical Health Risks
www.jchr.org
JCHR (2024) 14(6), 2823-2829 | ISSN:2251-6727

experimental stroke. Nat Med [Internet]. 2009;15(2):192–9.
19. Piepke M, Clausen BH, Ludewig P, Vienhues JH, Bedke T, Javidi E, et al. Interleukin-10 improves stroke outcome by controlling the detrimental Interleukin-17A response. J Neuroinflammation [Internet]. 2021;18(1).
20. Tokgoz S, Kayrak M, Akpinar Z, Seyithanoğlu A, Güney F, Yürüten B. Neutrophil lymphocyte ratio as a predictor of stroke. J Stroke Cerebrovasc Dis [Internet]. 2013;22(7):1169–74.
21. Xue J, Huang W, Chen X, Li Q, Cai Z, Yu T, et al. Neutrophil-to-lymphocyte ratio is a prognostic marker in acute ischemic stroke. J Stroke Cerebrovasc Dis [Internet]. 2017;26(3):650–7.
22. Giede-Jeppe A, Madžar D, Sembill JA, Sprügel MI, Atay S, Hoelter P, et al. Increased neutrophil-to-lymphocyte ratio is associated with unfavorable functional outcome in acute ischemic stroke. Neurocrit Care [Internet]. 2020;33(1):97–104.
23. Xue J, Zhang X-G, Jiang H-Y, Cui X-K, Zhang D, Yao Z-W, et al. An increase in neutrophil-to-lymphocyte ratio predicts poor functional outcomes in older patients with acute ischemic stroke: a retrospective study. J Integr Neurosci [Internet]. 2021;20(2).
24. Lee M, Lim J-S, Kim C-H, Lee S-H, Kim Y, Hun Lee J, et al. High neutrophil–lymphocyte ratio predicts post-stroke cognitive impairment in acute ischemic stroke patients. Front Neurol [Internet]. 2021;12.
25. Sarejloo S, Kheradjoo H, Es Haghi S, Hosseini S, Gargari MK, Azarhomayoun A, et al. Neutrophil-to-lymphocyte ratio and early neurological deterioration in stroke patients: A systematic review and meta-analysis. Biomed Res Int [Internet]. 2022;2022(1).
26. Cesari M, Penninx BWJH, Pahor M, Lauretani F, Corsi AM, Williams GR, et al. Inflammatory markers and physical performance in older persons: The InCHIANTI study. J Gerontol A Biol Sci Med Sci [Internet]. 2004;59(3):M242–8.
27. Rossi AP, Zanandrea V, Zoico E, Zanardo M, Caliari C, Confente S, et al. Inflammation and nutritional status as predictors of physical performance and strength loss during hospitalization. Eur J Clin Nutr [Internet]. 2016;70(12):1439–42.
28. Miyamoto S, Kondo T, Suzukamo Y, Michimata A, Izumi S-I. Reliability and validity of the manual function test in patients with stroke. Am J Phys Med Rehabil [Internet]. 2009;88(3):247–55.30.
29. Lee D-K, Kang M-H, Lee T-S, Oh J-S. Relationships among the Y balance test, Berg Balance Scale, and lower limb strength in middle-aged and older females. Braz J Phys Ther [Internet]. 2015;19(3):227–34.