Section: General Medicine

Original Research Article

A STUDY OF ROLE OF PLATELET TO LYMPHOCYTE RATIO [PLR] AND ITS CORRELATION WITH NATIONAL INSTITUTE OF HEALTH STROKE SCALE [NIHSS] FOR PREDICTION OF SEVERITY IN PATIENTS OF ACUTE ISCHEMIC STROKE

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Received *: 12/03/2026*
Received in revised form *: 05/04/2026*
Accepted *: 09/04/2026*

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DOI: 10.70034/ijmedph.2026.2.91

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 543-547

ABSTRACT

Background: Despite of advances in clinical management, there is no robustic prognostic marker in acute stroke. The Platelet-to-Lymphocyte Ratio (PLR) is considered to be important marker for predicting stroke severity and its outcomes. This study aims to find out correlation between PLR and the National Institute of Health Stroke Scale (NIHSS) for predicting stroke severity and prognosis.

Materials and Methods: This prospective observational study was conducted at Koppal Institute of Medical Sciences, Koppal, over one year (May 2022 to April 2023). Fifty patients with acute ischemic stroke, presenting within seven days of symptom onset, were enrolled. Detailed clinical and laboratory data, including platelet and lymphocyte counts, were collected at admission, at day3 /at time of discharge. Stroke severity was assessed using the NIHSS at both time points. Statistical analysis was performed using SPSS version 21, with Pearson's correlation coefficient used to analyze the relationship between PLR and NIHSS.

Results: The study found a significant positive correlation between PLR and NIHSS at both admission ($r = 0.874$, $p < 0.001$) and day3 /discharge ($r = 0.907$, $p < 0.001$). Changes in PLR from admission to discharge were also strongly correlated with changes in NIHSS ($r = 0.938$, $p < 0.001$). These findings suggest that higher PLR values are associated with greater stroke severity and that changes in PLR reflect changes in stroke severity.

Conclusion: PLR is a simple inflammatory marker for predicting stroke severity and helps in prognosis of patients with acute ischemic stroke. The significant correlations of PLR with NIHSS at both admission and day3 /discharge underscore the potential of PLR to enhance clinical assessment and guide therapeutic decisions. Further research is required to validate these findings in larger, multi-centre cohorts and to find out the mechanisms of the relationship between PLR and stroke outcomes.

Keywords: Acute ischemic stroke, Platelet to Lymphocyte Ratio, National Institute of Health Stroke Scale, Stroke severity.

INTRODUCTION

Stroke is a leading cause of morbidity and mortality worldwide, with ischemic stroke accounting for approximately 87% of all stroke cases.[1,2] Despite advances in acute stroke management, predicting stroke severity and prognosis remains a clinical challenge. The National Institute of Health Stroke Scale (NIHSS) is a widely used tool for assessing stroke severity and predicting outcomes.[3,4] However, there is a growing interest in identifying

additional biomarkers that can enhance the predictive accuracy of existing clinical scales.

The Platelet-to-Lymphocyte Ratio (PLR) has emerged as a potential biomarker of inflammation and thrombosis, both of which play critical roles in the pathophysiology of acute ischemic stroke.[5,6]

Higher platelet counts may increase thrombocyte activation and aggravate the release of inflammatory mediators, prompting a harmful inflammatory process.[7] Recently, lymphopenia was associated with the increased risk for developing adverse outcome in terms of morbidity and mortality in cardiovascular diseases particularly MI.[8]

Platelets are involved in the formation of thrombi, while lymphocytes are key players in the immune response. An elevated PLR has been associated with poor outcomes in various cardiovascular diseases, including acute coronary syndromes and peripheral artery disease.[9]

Recent studies have suggested that PLR may also be a valuable prognostic marker in acute ischemic stroke.[9] For instance, several observational studies have reported a significant association between elevated PLR and increased stroke severity, as well as worse functional outcomes at discharge. Moreover, a higher PLR has been linked to increased mortality and recurrent stroke risk in patients with acute ischemic stroke.[10]

Recently, interest in the study of PLR has grown because this ratio has been found to be predictor of prognosis in patients with diverse inflammatory and ischemic conditions.[11] High PLR as an inflammatory marker has been correlated with the poor prognosis in various diseases like Myocardial infarction, critical limb ischemia, end-stage renal failure, pulmonary embolism and various malignancies including breast, ovarian, pancreatic, hepatobiliary carcinoma and other solid tumours.[12]

The advantage of PLR is that it reflects the condition of both inflammation and thrombosis pathways and is more valuable than either platelet or lymphocyte counts alone. This emerging marker has not been frequently studied with acute ischemic stroke; hence present study was done to find out the role of PLR (Platelet to lymphocyte ratio) in patients of acute ischemic stroke and correlating with NIHSS for predicting the prognosis.

Despite these promising findings, the utility of PLR as a prognostic marker in acute ischemic stroke remains underexplored, particularly about its correlation with established clinical scales like the NIHSS. This prospective observational study aims to evaluate the role of PLR in patients with acute ischemic stroke and to determine its correlation with NIHSS for predicting stroke severity and prognosis.

We hypothesize that PLR, both at admission and discharge, is strongly correlated with NIHSS scores and that changes in PLR reflect changes in stroke severity as measured by NIHSS. This study will provide insights into the potential of PLR as an important inflammatory biomarker for assessing the prognosis in patients with acute ischemic stroke.

MATERIALS AND METHODS

This prospective observational study was conducted at Department of General Medicine, Koppal Institute of Medical Sciences, Koppal, from May 1, 2022, to April 30, 2023. The study aimed to evaluate the role of Platelet to Lymphocyte Ratio (PLR) in predicting the severity and prognosis of acute ischemic stroke, correlating it with the National Institute of Health Stroke Scale (NIHSS). A total of 50 patients with acute ischemic stroke who presented within seven days of symptom onset and provided written informed consent were enrolled in the study. Patients with hemorrhagic stroke, cerebral venous sinus thrombosis, or those unwilling to participate were excluded.

This study was obtained Ethical Clearance Approval from Institutional Ethical Committee No. KIMS-Koppal/IEC/115/2022-23.

Upon admission, the diagnosis of acute ischemic stroke was confirmed through clinical evaluation and imaging studies, including a CT scan or MRI of the brain. Detailed patient histories were taken, and clinical examinations were performed to identify risk factors for ischemic stroke. Baseline demographic data, including age, sex, occupation, smoking status, alcohol consumption, hypertension (HTN), diabetes mellitus (DM), obesity, ischemic heart disease (IHD), were recorded.

Laboratory investigations were conducted at admission to measure platelet count, lymphocyte count, urea, creatinine, total cholesterol, triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). The PLR was calculated by dividing the platelet count by the lymphocyte count. The severity of the stroke was assessed using the NIHSS at the initial presentation and again at discharge or on the third day of hospitalization.

Statistical Analysis: Statistical analyses were performed using SPSS version 21. Data were entered into Microsoft Excel and analyzed for frequencies, percentages, means, and standard deviations. Pearson's correlation coefficients were used to calculate correlation coefficients between PLR and NIHSS scores at admission and discharge. The changes in PLR and NIHSS from admission to discharge were also computed and correlated to evaluate the predictive value of PLR for stroke severity and prognosis.

RESULTS

Table 1: Baseline Demographic, Clinical & laboratory data of study population

Variable		Value(%)
Age (Mean±SD)		67.3 ± 9.4
Sex		Male: 31 (62%), Female: 19 (38%)
Occupation		Sedentary: 21 (42%), Active: 29 (58%)
Habits	a) Smoking	29(58%)
	b) Alcohol	18(36%)
Comorbidities	a)Hypertension (HTN)	30(60%)
	b)Diabetes Mellitus(DM)	13(26%)
	c)Dyslipidemia	20(40%)
	d)Ischemic Heart Disease(IHD)	15(30%)
	e)Obesity	8(16%)
	f)Others	5(10%)
Hemiparesis	a)Right side	40(80%)
	b)Left side	10(20%)
Blood pressure mmHg		143/88±12/9g
Haemoglobin (Hb)gm/dl		13.7±1.3
Infarct territory		MCA 30(60%) ACA 11(22%) PCA 6(12%) MCA+ACA 3(6%)
NIHSS at admission		13.2±4.3

The study included 50 participants, with an average age of 67.3± 9.4 years. The gender distribution showed males (62%) more than females (38%).

In terms of habits, 58% were smokers and Alcohol consumption 36% of the study population. In underlying comorbidities Hypertension was prevalent in 60% of the total, followed by 40% had dyslipidemia.

The initial clinical presentation and stroke characteristics of the study participants revealed that 80% experienced Right sided hemiparesis, while 20% had left sided hemiparesis. The average blood pressure recorded at admission was 143/88 mmHg, with SD±12/9 mmHg.

The distribution of infarct territory showed that the majority of the strokes occurred in the Middle Cerebral Artery (MCA) territory, with 30 cases (60%). This was followed by the Anterior Cerebral Artery (ACA) territory, which accounted for 11 cases (22%).

The severity of the stroke at admission, as assessed by the National Institute of Health Stroke Scale (NIHSS), had an average score of 13.2, with SD±4.3. This comprehensive clinical profile underscores the extent and severity of the initial stroke presentation among the participants in the study.

Table 2: Haematological and biochemical profile of study patients at Admission

Variable	Mean ± SD
Platelet Count (x10^3/cu.mm)	253 ± 48
Lymphocyte Count (x10^3/cu.mm)	1.7 ± 0.4
Platelet to Lymphocyte Ratio (PLR)	148.8 ± 52.1
Urea (mg/dL)	39 ± 11
Creatinine (mg/dL)	1.3 ± 0.2
Random blood sugar	196.33±92.26
Total Cholesterol (mg/dL)	198 ± 37
Triglycerides (TG) (mg/dL)	147 ± 47
Low-Density Lipoprotein (LDL) (mg/dL)	123 ± 29
High-Density Lipoprotein (HDL) (mg/dL)	47 ± 9

The laboratory investigations conducted at admission provided key insights into the haematological and biochemical status of the study participants. The platelet count averaged 253 x 10^3/cu.mm with SD± 48, indicating a relatively normal platelet level across the group. The lymphocyte count had a mean value of 1.7 x 10^3/cu.mm, SD±0.4.

The Platelet to Lymphocyte Ratio (PLR), a crucial marker in this study, averaged 148.8, with a standard deviation of 52.1, suggesting significant variability in this inflammatory marker among patients.

Lipid profile analysis showed that the total cholesterol levels averaged 198 mg/dL with a standard deviation of 37 mg/dL. Triglyceride levels were found to have a mean of 147 mg/Dl SD±47mg/dL. The low-density lipoprotein (LDL) levels averaged 123 mg/dL SD±29 mg/dL, and high-density lipoprotein (HDL) levels had a mean value of 47 mg/dL SD±9 mg/dL.

Table 3: Comparison of PLR & NIHSS in study patients

Variable	Admission (Mean ± SD)	Discharge/Day 3 (Mean ± SD)
NIHSS	13.2 ± 4.3	7.9 ± 3.1
Platelet Count (x10^3/cu.mm)	253 ± 48	246 ± 43
Lymphocyte Count (x10^3/cu.mm)	1.7 ± 0.4	1.6 ± 0.3
PLR	148.8 ± 52.1	153.8 ± 48.7

The clinical and laboratory findings at discharge or on Day 3 showed notable changes compared to admission values, reflecting the progression and treatment response in the patients. The National Institute of Health Stroke Scale (NIHSS) score, which measures stroke severity, showed a significant improvement from an average of 13.2 (± 4.3) at admission to 7.9 (± 3.1) at discharge, indicating a reduction in stroke severity. The platelet counts lightly decreased, with the mean value changing from 253 x 10^3/cu.mm (± 48) at admission to 246 x 10^3/cu.mm (± 43) at discharge. This small decrease is within the expected range and reflects a stable haematological status.

The lymphocyte count also exhibited a minor decline from an average of 1.7 x 10^3/cu.mm (± 0.4) at admission to 1.6 x 10^3/cu.mm (± 0.3) at discharge. This change indicates a slight reduction in the immune cell count during the hospital stay. Interestingly, the platelet-tolymphocyte ratio (PLR) showed a slight increase from 148.8 (± 52.1) at admission to 153.8 (± 48.7) at discharge. This increase in PLR could suggest ongoing inflammatory processes or other clinical factors influencing platelet and lymphocyte dynamics during the acute phase of stroke recovery.

Table 4: Correlation between PLR and Stroke Severity (NIHSS)

Variable	Correlation Coefficient (r)	p-value
PLR at Admission vs NIHSS	0.874	<0.001
PLR at Discharge vs NIHSS	0.907	<0.001
Change in PLR vs Change in NIHSS	0.938	<0.001

The correlation analysis between Platelet to Lymphocyte Ratio (PLR) and stroke severity, as measured by the National Institute of Health Stroke Scale (NIHSS), yielded significant results. At admission, the PLR demonstrated a strong positive correlation with the NIHSS score, with a correlation coefficient (r) of 0.874 and a p-value of less than 0.001. This indicates that higher PLR values at admission were strongly associated with more severe strokes. At discharge or on Day 3, the correlation between PLR and NIHSS was even stronger, with a coefficient of 0.907 and a p-value of less than 0.001. This further underscores the robustness of PLR as a predictor of stroke severity, suggesting that PLR remains a relevant marker throughout the acute phase of stroke recovery.

Moreover, the changes in PLR and NIHSS from admission to discharge were also highly correlated, with a correlation coefficient of 0.938 and a p-value of less than 0.001. This finding highlights that the variation in PLR is closely aligned with changes in stroke severity, making it a potentially valuable dynamic marker for monitoring patient progress and prognosis.

DISCUSSION

The findings of this study underscore the significant role of the Platelet-to-Lymphocyte Ratio (PLR) as a prognostic biomarker in acute ischemic stroke. The strong positive correlations between PLR and the National Institute of Health Stroke Scale (NIHSS) at admission and discharge indicate that higher PLR values are associated with greater stroke severity. This is consistent with previous research highlighting,[13,14,15,16] the prognostic value of PLR in cardiovascular and cerebrovascular diseases.

At admission, the correlation coefficient between PLR and NIHSS was 0.874, with a p-value of less than 0.001. This strong correlation suggests that PLR can serve as an early indicator of stroke severity. The literature supports this finding; for instance, a study by Oylumlu et al. (2015),[13] found that elevated PLR levels were significantly associated with increased severity of ischemic stroke and worse clinical outcomes. Similarly, the work by Kim et al. (2012),[14] demonstrated that inflammatory markers like PLR are closely related to stroke severity and can predict adverse outcomes.

The correlation between PLR and NIHSS at discharge was even stronger ($r = 0.907$, $p < 0.001$), suggesting that PLR remains a relevant marker throughout the acute phase of stroke recovery. This finding is corroborated by Kurtul and Ornek (2019),[15] who reported that persistent elevation of PLR was associated with poor prognosis and higher mortality in patients with ischemic stroke. The significant correlation observed at discharge underscores the potential of PLR as a diagnostic marker and a continuous monitoring tool for assessing patient progress and treatment efficacy.

The study also found a robust correlation between the PLR and NIHSS changes from admission to discharge ($r = 0.938$, $p < 0.001$). This indicates that changes in PLR reflect changes in stroke severity, making PLR a valuable dynamic marker. This aligns with the findings of Adams et al. (1999),[16] who noted that serial measurements of inflammatory markers could provide insights into disease progression and response to therapy.

Our study supports the finding of Pei-Hsun Sung et al,[17] Stella Bouziana et al,[18] Andres Perez et al,[19] and Ozge Altintas et al,[20] which demonstrated that patients of acute ischemic stroke with higher PLR had poor outcome as compared to patients with lower PLR values.

The clinical and laboratory findings at admission and discharge highlighted the stability of haematological parameters such as platelet and lymphocyte counts while noting a slight increase in PLR at discharge. This increase in PLR, despite overall clinical improvement, suggests ongoing

inflammatory or stress responses, which are common in the acute recovery phase of stroke. The role of inflammation in stroke pathophysiology has been welldocumented, with studies indicating that persistent inflammation can hinder recovery and contribute to secondary complications.

Limitations of the study

1. The sample size of our study was small
2. Only single center patients of acute ischemic stroke were included
3. Future research should focus on validating these findings in exploring the mechanisms underlying the relationship between PLR and stroke outcomes

CONCLUSION

In conclusion, this study's results support the utility of PLR as a robust inflammatory biomarker for predicting stroke severity and monitoring patient progress. The significant correlations with NIHSS at both admission and discharge, along with the strong association with changes in stroke severity, highlight the potential of PLR to enhance clinical assessment and guide therapeutic decisions. Basic health care units can acquire this ratio and use it to make decisions about immediate referral of patients for better prognosis.

Author contributions

Srinivasa J, Gavishiddesh Vishwanath Ronad, Umesh G Rajoor, Krishnakumar Naik, Shivaprasada T, Mohd Naveed Khan, conceptualized the study and were responsible for resources and data collection, data analysis, manuscript preparation, review, and revisions.

All the authors reviewed and accepted the final version of the manuscript.

Data availability statement: The datasets generated and analysed during the current study are available with the corresponding author and can be shared upon reasonable request.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

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