

Evaluation of Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet Indices in Acute Ischemic Stroke: An Experience of a Tertiary Care Hospital

Anshu Gupta¹, Tanima Dwivedi²

¹Department of Emergency Laboratory, Institute of Human Behaviour and Allied Sciences, New Delhi, India

²Department of Onco-Hematopathology, National Cancer Institute, AIIMS, New Delhi, India

Email: dransh2002@yahoo.co.in

How to cite this paper: Gupta, A. and Dwivedi, T. (2026) Evaluation of Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet Indices in Acute Ischemic Stroke: An Experience of a Tertiary Care Hospital. *Open Journal of Pathology*, 16, 220-230. <https://doi.org/10.4236/ojpathology.2026.164023>

Received: July 18, 2026

Accepted: October 20, 2026

Published: October 23, 2026

Copyright © 2026 by author(s) and Scientific Research Publishing Inc. This work is licensed under the Creative Commons Attribution International License (CC BY 4.0). <http://creativecommons.org/licenses/by/4.0/>



Open Access

Abstract

Stroke is one of the leading causes of death and disability in India and across the world. Inflammatory cells, particularly neutrophils, play a major role in the pathogenesis of stroke by contributing to blood-brain barrier disruption, cerebral edema, and subsequent brain injury, and targeting these cells may help reduce infarct size and improve outcomes. In contrast, lymphocyte levels tend to decrease after an acute ischemic stroke (AIS), making the neutrophil-to-lymphocyte ratio (NLR) a simple, low-cost, and reliable inflammatory marker that reflects the body's response, with elevated levels commonly observed after a stroke. Platelets also participate in the development of intravascular thrombosis, and their size reflects their activity, which can be assessed through platelet indices (PI) such as mean platelet volume (MPV), platelet large cell ratio (P-LCR), platelet distribution width (PDW), and plateletcrit (PCT). With this background, the study aimed to evaluate the presence of an inflammatory state in stroke patients using NLR values and to assess the relationship between different platelet indices and AIS. This prospective study included 145 AIS patients admitted to the emergency department of a tertiary neurosciences hospital and compared them with an equally sized group of age- and sex-matched healthy individuals. The results showed that NLR and various platelet indices were significantly higher in AIS patients, indicating that these easily measurable parameters effectively reflect inflammation and platelet activity in individuals with acute ischemic stroke.

Keywords

Neutrophil to Lymphocyte Ratio, Platelet Indices, Stroke, Emergency Service

1. Introduction

Ischemic stroke is a leading cause of death and disability worldwide, including in India [1]. It occurs due to the sudden cessation of blood flow through an intracranial artery, caused by thrombosis or emboli, leading to oxygen and glucose deprivation, cell death, and ultimately liquefactive necrosis [1]. Clinically, ischemic stroke presents with acute neurological deficits, which vary depending on the affected brain region and may include speech disturbances, headache, and non-orthostatic dizziness [2]. Based on the duration of symptom onset, ischemic stroke can be classified as hyperacute (less than 6 hours), acute (6 - 24 hours), subacute (24 hours to 5 days), or chronic (5 days to weeks) [3] [4].

Inflammation, particularly involving neutrophils, plays a significant role in the pathogenesis of acute ischemic stroke (AIS). Neutrophils are the first cells to infiltrate the affected area, contributing to blood-brain barrier disruption, cerebral edema, and neuronal injury. Their activity can be quantified using the absolute neutrophil count (ANC). In contrast, lymphocytes decrease due to apoptosis after AIS, measured as the absolute lymphocyte count (ALC). The Neutrophil-to-Lymphocyte Ratio (NLR) reflects this balance between neutrophils and lymphocytes, providing a more accurate marker of systemic inflammation than ANC or ALC alone, and has been associated with increased infarct size and mortality in stroke patients [5] [6].

Platelets are also implicated in thromboembolic events underlying AIS, with larger, more active platelets promoting clot formation. Platelet activity can be assessed using platelet indices (PI), including mean platelet volume (MPV), platelet large cell ratio (P-LCR), platelet distribution width (PDW), and plateletcrit (PCT) [7]. The present study aimed to evaluate the utility of NLR and PIs as simple, low-cost markers for detecting inflammation and thromboembolic activity in stroke patients, potentially aiding in earlier intervention and reducing morbidity and healthcare costs.

2. Materials & Methods

This was a prospective study conducted in the emergency laboratory of a tertiary neurosciences hospital over 6 months (July 2023 to December 2023), involving a total of 290 samples. Of these, 145 samples were taken from patients admitted to the emergency department with clinical and radiological suspicion of acute ischemic stroke (AIS), while another 145 samples were obtained from healthy volunteers who formed the control group. The control group was age- and sex-matched, engaged in full-time employment, and without any acute illness or chronic debilitating disease, including cancer and infectious diseases.

To clarify the prospective nature of the study, all AIS patient samples were collected in real-time at the point of care immediately upon patient arrival, and the corresponding clinical and radiological findings were recorded concurrently. Only after this prospective collection and documentation were the samples anonymized for analysis.

Patients aged ≥ 18 years who were suspected of having AIS clinically or radiologically within 24 hours of onset were included in the study. Exclusion criteria included patients admitted more than 24 hours post-onset of stroke, those with hemorrhagic stroke or transient ischemic attack (TIA), individuals with a history of prior stroke or who were already receiving treatment for stroke from another center, and patients with chronic inflammatory disease, active malignancy, trauma, surgery, fever, active infection before admission, or any systemic infection that occurred during the first 24 hours before or after admission.

Aseptic collection of 2 ml of venous blood was performed using a dipotassium ethylene diamine tetra-acetic acid (EDTA) blood collection tube, and the samples were immediately transported to the emergency laboratory for analysis. Complete blood counts (CBC) were processed on an automated hematology analyzer (Transasia Elite 580) within one hour of phlebotomy to minimize sample aging-related variability.

The Neutrophil-Lymphocyte Ratio (NLR) was calculated by dividing the absolute neutrophil count (ANC) by the absolute lymphocyte count (ALC). The parameters assessed in the study included ANC, ALC, Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), Platelet Large Cell Ratio (PLCR), Platelet Count (PC), and Plateletcrit (PCT), all recorded using the automated hematology analyzer. The normal range of parameters was: ANC: $2.0 - 7.0 \times 10^9/L$, ALC: $1.0 - 3.0 \times 10^9/L$, NLR: 0.78 - 3.53, MPV: 7.4 - 10.4 fL, PDW: 9 - 14 fL, PLCR: 15% - 35%, and PCT: 0.22% - 0.24% [8].

The adequate sample size for the study was calculated by following formula:

$$n = \left[Z^2 * P(1 - P) \right] / (d^2),$$

where n is the sample size, Z is the statistic corresponding to the confidence level (for 90%, it is 1.645), P is the expected prevalence from other studies (0.2%), and d is the precision (corresponding to the effect size), which was 4% in our study. According to the formula above, the calculated sample size was 270, which is close to our study sample size of 290.

This study was conducted solely on patient samples received in the emergency laboratory for routine testing. Although the samples were anonymized before analysis, they were collected prospectively at the time of patient presentation, and clinical details were recorded in real time, ensuring a truly prospective data collection process. Patient confidentiality was maintained. Since samples were pre-collected and treated anonymously, the Institutional Ethics Committee exempted the study from ethical review and provided a waiver for individual consent. Consequently, no written informed consent was obtained from participants.

3. Statistical Analysis

Statistical analysis was performed using SPSS software for Windows, version 16.0 (SPSS Inc., Chicago, IL, USA) and Microsoft Excel. The results of the study were presented as mean $\pm$ SD for continuous variables and as the number of cases and percentages for categorical data. Comparisons were performed with independent

t-tests for continuous variables and chi-square tests for categorical variables where appropriate. The correlation coefficient (r) was used to correlate categorical variables. The study endpoints were determined using logistic regression. Receiver operating characteristic (ROC) curve analysis and area under the curve (AUC) were used to evaluate the effectiveness of diagnostic tests (0.90 - 1: Excellent, 0.80 - 0.90: Good, 0.70 - 0.80: Fair, 0.60 - 0.70: Poor, 0.50 - 0.60: Fail). A p-value of < 0.05 was considered statistically significant.

4. Results

The present prospective study was conducted over a period of 6 months (July 2017 to December 2017). The mean ages of the study and control groups were 61.10 ± 12 and 60.34 ± 13.35 , respectively. Out of 145 AIS patients, 87 (60%) were males, and 58 (40%) were females.

ANC, ALR, and NLR

The mean NLR, ANC, and ALC in AIS patients were $10.75 \times 10^9/L \pm 8.52 \times 10^9/L$, $9.49 \times 10^9/L \pm 3.40 \times 10^9/L$, and $1.34 \times 10^9/L \pm 1.11 \times 10^9/L$, respectively, with corresponding 95% confidence intervals (CIs) of 9.36 - 12.14 for NLR, 8.94 - 10.04 $\times 10^9/L$ for ANC, and 1.16 - 1.52 $\times 10^9/L$ for ALC. The ranges for these parameters were 2.43 - 33.97 $\times 10^9/L$ for ANC, 0.21 - 4.81 $\times 10^9/L$ for ALC, and 0.74 - 154 for NLR. In the control group, the mean NLR, ANC, and ALC were $2.42 \times 10^9/L \pm 1.05 \times 10^9/L$, $5.42 \times 10^9/L \pm 3.21 \times 10^9/L$, and $2.04 \times 10^9/L \pm 0.74 \times 10^9/L$, respectively, with 95% CIs of 2.25 - 2.59 for NLR, 4.90 - 5.94 $\times 10^9/L$ for ANC, and 1.92 - 2.16 $\times 10^9/L$ for ALC, and ranges of 0.69 - 4.43 for NLR, 2.43 - 33.97 $\times 10^9/L$ for ANC, and 0.21 - 4.81 $\times 10^9/L$ for ALC (Table 1).

Table 1. Comparison between the study and control groups according to NLR and platelet indices from laboratory data.

Parameter	Case Mean $\pm$ SD	Control Mean $\pm$ SD	p Value	AUC	S. Error	Sensitivity	Specificity	PPV	NPV	Cutoff/r Value
Age (years)	62	58	-	-	-					
Sex (no.)	M: 90 F: 55	M: 87 F: 58	-	-	-					
ANC ($\times 10^3/\mu L$)	9.49 ± 3.40	5.42 ± 3.21	<0.001	0.832	0.002	93.1	66.9	73.77	90.65	>5.24/0.5242
ALR ($\times 10^3/\mu L$)	1.34 ± 1.11	2.04 ± 0.74	<0.001	0.788	0.027	80.69	66.21	70.48	77.42	<1.75/0.3547
NLR	10.75 ± 8.52	2.42 ± 1.05	<0.001	0.975	0.009	91.03	96.55	96.34	91.45	>4.42/0.5671
MPV (fL)	13.24 ± 2.48	11.17 ± 1.47	<0.001	0.777	0.026	49.31	91.03	84.6	64.23	>12.9/0.1695
PDW (fL)	16.98 ± 3.99	14.35 ± 3.36	<0.001	0.696	0.031	51.03	77.24	51.03	77.24	>16.5/0.3369
PLCR (%)	44.59 ± 8.54	31.50 ± 9.04	<0.001	0.852	0.022	84.14	70.34	73.93	81.6	>36/0.5983
PCT (%)	0.66 ± 0.32	0.22 ± 0.068	0.05	0.904	0.017	77.93	84.83	83.71	79.35	>0.28/0.0969
PC ($\times 10^3/\mu L$)	228.19 ± 95.87	255.41 ± 169.07	0.099	0.593	0.033	64.83	55.17	59.12	61.07	<235/0.0899

NLR and ANC were significantly higher in AIS patients compared to controls, with positive correlations (NLR: 10.75 ± 8.52 vs. 2.42 ± 1.05 , $p < 0.001$, $r = 0.5671$; ANC: 9.49 ± 3.40 vs. $5.42 \pm 3.21 \times 10^9/L$, $p < 0.001$, $r = 0.5242$). The mean differences (effect sizes) were 8.33 for NLR and $4.07 \times 10^9/L$ for ANC. ALC was significantly lower in AIS patients, showing a negative correlation (1.34 ± 1.11 vs. $2.04 \pm 0.74 \times 10^9/L$, $p < 0.001$, $r = -0.3547$), with a mean difference of $-0.70 \times 10^9/L$. NLR exhibited high sensitivity (91.03%) and specificity (96.55%), while ANC and ALC demonstrated high sensitivity (ANC: 93.10%; ALC: 80.69%) but moderate specificity (ANC: 66.90%; ALC: 66.21%) (Figure 1, Table 1). The AUC for NLR (0.975) was higher than for ANC (0.832) and ALC (0.788) (Figure 1).

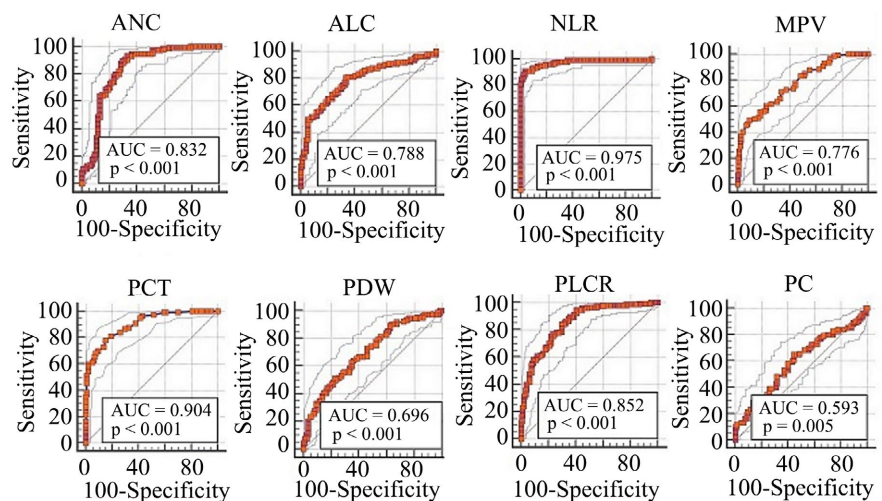


Figure 1. ROC curves of the parameters.

Previous studies have reported the normal range of NLR as 0.78 to 3.53 [9]-[12]. In our study, we found that only 1 patient (1%) had an NLR below this range, 7 patients (5%) had NLR within the normal range, while the vast majority, 137 patients (94%), had NLR values above the normal range (Figure 2).

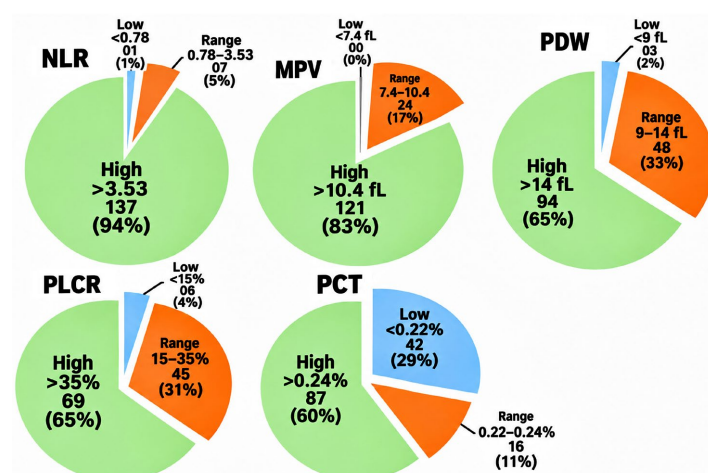


Figure 2. Variation of NLR and PIs in AIS patients.

Platelet Indices (MPV, PDW, PLCR, and PCT)

The mean platelet indices (PI) in AIS patients were, MPV $13.24 \text{ fL} \pm 2.48 \text{ fL}$ (95% CI: 12.78 - 13.70), PDW $16.98 \text{ fL} \pm 3.99 \text{ fL}$ (95% CI: 16.21 - 17.75), PLCR $44.59\% \pm 8.54\%$ (95% CI: 42.80 - 46.38), and PCT $0.66\% \pm 3.2\%$ (95% CI: 0.60 - 0.72). In the control group, the mean values were MPV $11.17 \text{ fL} \pm 1.47 \text{ fL}$ (95% CI: 10.89 - 11.45), PDW $14.35 \text{ fL} \pm 3.36 \text{ fL}$ (95% CI: 13.63 - 15.07), PLCR $31.50\% \pm 9.04\%$ (95% CI: 29.50 - 33.50), and PCT $0.22\% \pm 0.068\%$ (95% CI: 0.20 - 0.24). The ranges for AIS patients were 10.0 - 27.0 fL for MPV, 8.7 - 28.0 fL for PDW, 15.5% - 62% for PLCR, and 0.17% - 0.69% for PCT, while in controls the ranges were 8.0 - 14.6 fL, 7.5 - 18.8 fL, 10.5% - 34%, and 0.10% - 0.30%, respectively (**Table 1**).

MPV, PDW, PLCR, and PCT were significantly higher in AIS patients compared to controls, showing positive correlations (MPV: 13.24 ± 2.48 vs. 11.17 ± 1.47 , $p < 0.001$, $r = 0.1695$; PDW: 16.98 ± 3.99 vs. 14.35 ± 3.36 , $p < 0.001$, $r = 0.3369$; PLCR: 44.59 ± 8.54 vs. 31.50 ± 9.04 , $p < 0.001$, $r = 0.5983$; PCT: 0.66 ± 3.2 vs. 0.22 ± 0.068 , $p < 0.001$, $r = 0.0969$), with mean differences (effect sizes) of 2.07 fL for MPV, 2.63 fL for PDW, 13.09% for PLCR, and 0.44% for PCT. Platelet count (PC) was slightly lower in AIS patients compared to controls (228.19 ± 95.87 vs. 255.41 ± 169.07 ; mean difference -27.22 ; $r = -0.0989$; $p = 0.099$) (**Table 1**).

Regarding diagnostic performance, MPV and PDW had low sensitivity (MPV: 49.31%, PDW: 51.03%) but high specificity (MPV: 91.03%, PDW: 77.24%). PLCR showed high sensitivity (84.14%) and specificity (70.34%), whereas PCT demonstrated high sensitivity (77.93%) and specificity (84.83%) (**Table 1**). The AUC for PCT (0.904) indicated excellent diagnostic accuracy, while PLCR (AUC: 0.852) and MPV (AUC: 0.777) were good, and PDW (AUC: 0.696) and PC (AUC: 0.593) were poor for identifying thrombogenic states in AIS patients (**Figure 1**). Most AIS patients had elevated platelet indices [MPV: 121 (83%), PDW: 94 (65%), PLCR: 94 (65%), and PCT: 87 (60%)] (**Figure 2**).

5. Discussion

Acute ischemic stroke (AIS) is defined as a reduction or interruption in cerebral blood flow due to thrombosis, embolism, or focal hypoperfusion, resulting in permanent neurologic deficits [9]. It is the second most common cause of death after coronary artery disease and is a leading cause of disability both globally and in India. AIS is the most common type of stroke seen in emergency departments. Based on the duration of onset, AIS can be classified into hyperacute (less than 6 hours), acute (6 to 24 hours), subacute (24 hours to 5 days), and chronic (5 days to weeks) [3] [4]. Neuroimaging techniques such as non-contrast computed tomography (CT) and magnetic resonance imaging (MRI) are the primary diagnostic tools for the initial evaluation of suspected ischemic stroke [9]. On a CT scan, AIS typically presents as loss of grey-white matter differentiation, hypoattenuation of deep nuclei, cortical hypodensity, and parenchymal swelling with sulcal-gyral effacement [2]-[4].

The pathophysiology of stroke is multifactorial, involving inflammatory pathways, excitotoxicity, oxidative damage, ionic imbalances, apoptosis, angiogenesis, and neuroprotection [10]. In the acute ischemic area, stagnant blood in occluded and hypoperfused vessels triggers an inflammatory cascade, activating intravascular leukocytes and releasing pro-inflammatory mediators from the ischemic brain parenchyma and endothelium, leading to neuronal death and irreversible loss of neuronal function. This cascade results in systemic inflammation [11].

Neutrophils are the first cells to infiltrate the affected area after AIS, starting to accumulate within 6 - 12 hours, increasing over 24 hours, peaking at 48 - 72 hours, and being replaced by mononuclear cells after 4 - 6 days [10]. Neutrophils also play a significant role in thrombogenesis and atherosclerosis, as shown in **Figure 3** [5] [12]. Elevated total leukocyte counts, higher neutrophil counts, and lower lymphocyte counts have been associated with increased incidence of cerebral infarction, larger infarct volumes, and higher mortality in AIS patients, as reported in previous studies [12]. The neutrophil-to-lymphocyte ratio (NLR) reflects the balance between neutrophils and lymphocytes, and its association with inflammation has been extensively studied [11] [12]. Studies conducted by Jager *et al.* also concluded that NLR is a better predictor of bacteremia than routine parameters like C-reactive protein (CRP), total leukocyte count, and neutrophil count [13]. In contrast, studies by Smith *et al.* and Li *et al.* directly compared NLR values in AIS patients versus controls and reported mean differences similar to our findings (AIS NLR 10.3 - 11.2 vs controls 2.0 - 2.5), confirming our observed effect sizes and highlighting the reproducibility of NLR as an inflammatory marker in stroke.

In our study, we measured ANC, ALC, and NLR in suspected AIS patients. ANC and ALC were found to be elevated and reduced, respectively, in AIS patients, and both were weak parameters as determined by AUC. However, the ANC/ALC ratio (NLR) is a stronger indicator of inflammation as it reflects not just the absolute neutrophil and lymphocyte counts but also the overactivation of neutrophils and depletion of lymphocytes, leading to a wider gap between the two leukocyte types [13]. NLR was also found to be higher in AIS patients. In our study, the superiority of NLR over ANC and ALC was supported by its AUC, high sensitivity, and high specificity. The high predictive value of NLR suggests that neutrophils play a significant role in the pathogenesis of AIS. Our findings are consistent with other studies [14], which also concluded that NLR was significantly higher in AIS patients. These studies compared NLR values between mortality and survival groups and found higher NLR values in the mortality group than in the survival group [14].

Quan *et al.* also studied NLR and found that elevated NLR within the first 24 hours was associated with an increased risk of both short- and long-term adverse clinical outcomes in patients with ischemic stroke, regardless of etiology [15]. A recent study by Ahmed *et al.* (2022) concluded that there was a positive correlation between NLR and length of hospital stay. They also observed that patients

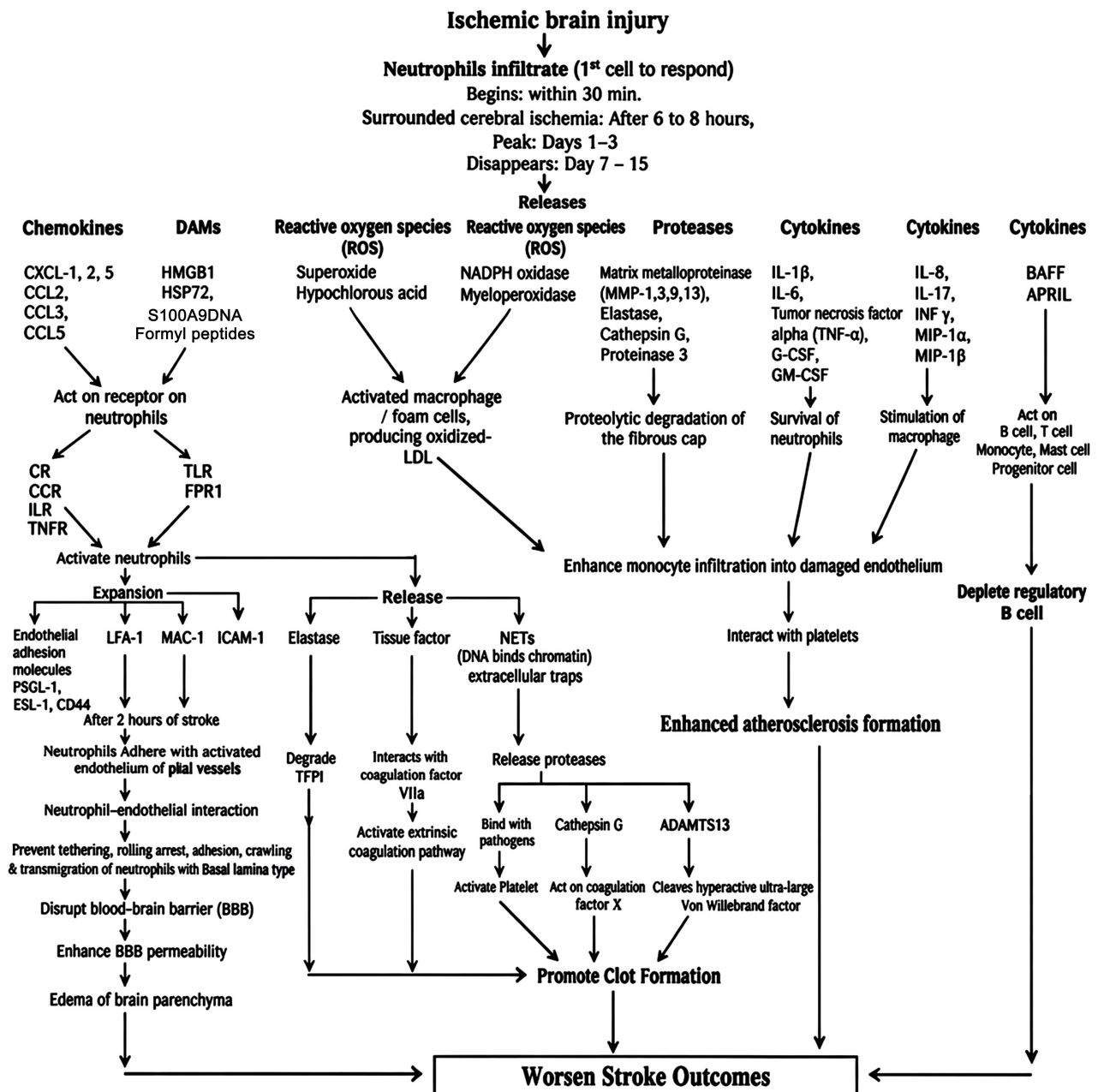


Figure 3. Flow chart depicting the role of neutrophils in thrombogenesis and atherosclerosis in ischemic brain injury.

with high NLR had complications such as aspiration pneumonia, seizures, and urinary tract infections [16]. These findings provide context to our results, reinforcing the clinical relevance of NLR as a marker of systemic inflammation in AIS. However, in our study, prognosis was not determined as we included only AIS patients admitted to the emergency department, and serial measurements and follow-up were not feasible due to the referral of most patients to higher medical centers.

The normal range of NLR was 0.78 to 3.53, as reported in previous studies. In our study, the normal NLR in the control group ranged from 1.30 to 2.78 (mean:

2.04 ± 0.74), consistent with other studies [9]-[12].

Platelets also play a crucial role in the pathogenesis of AIS [17]. Stagnant blood in occluded vessels causes trauma to the vascular endothelium and platelets, releasing P-selectin. This P-selectin binds to leukocytes, forming clusters that lead to intravascular blockages. In AIS, platelets are hyperactive, releasing more granules and increasing platelet turnover, resulting in larger, more active platelets that are prone to forming clots. This contributes to worsening stroke outcomes [13]. These larger platelets can be measured using platelet indices (PI) such as MPV, PDW, PLCR, and PCT. MPV refers to the average volume of platelets in the bloodstream, measured by analyzing the platelet distribution curve. The normal range of MPV is 7.4 - 10.4 fL. PDW is calculated by measuring the width of the platelets histogram at the 20% height level, with a normal range of 9 - 14 fL. PLCR indicates platelets larger than 12 fL, with a normal range of 15% - 35%. PCT represents the volume occupied by platelets in the blood, with a normal range of 0.22% - 0.24% [13].

In our study, all PIs were significantly higher and positively correlated in the AIS group compared to controls, suggesting that larger platelets play a significant role in clot formation and AIS pathogenesis [15]. Among the PIs, PCT was the most effective diagnostic test for detecting large, active platelets in AIS patients, as it had the highest AUC, sensitivity, specificity, and predictive values. PLCR also showed high sensitivity, specificity, and predictive values. However, both MPV and PDW were less effective diagnostic tests for detecting large platelets, as they had low sensitivity but high specificity. Platelet counts (PC) were lower in the AIS group compared to the control group and showed a negative correlation. This may suggest that reactive platelets are released into circulation in response to ischemic site mediators, with platelets being consumed during thrombosis. These findings align with previous literature, such as studies, which reported elevated MPV and PLCR in AIS patients and linked them to thrombotic risk and stroke severity [6] [15].

The advantages of this study include that both NLR and PIs are simple, low-cost, and easy-to-measure diagnostic tests for AIS. Potential confounders were minimized by excluding patients with infections, but the study has important limitations, such as the relatively small sample size, which reduces the statistical power, and the lack of serial measurements and follow-up data limits the ability to assess prognostic value and long-term outcomes. These limitations should be considered when interpreting the findings and planning future studies.

6. Conclusion

In conclusion, our study found that both NLR and platelet indices (PIs) were elevated in AIS patients, indicating the roles of systemic inflammation and thrombogenesis by larger, reactive platelets in the pathogenesis of AIS. These parameters (NLR, MPV, PDW, PLCR, and PCT) are simple, cost-effective, readily available, and useful diagnostic tools that may aid in the early identification of high-risk pa-

tients and support timely clinical decision-making. However, the findings should be interpreted with caution due to the study's relatively small sample size, lack of longitudinal follow-up, and absence of prognostic outcome data. While these indices can currently serve as adjuncts alongside radiological findings for acute management, further large-scale, prospective, and randomized studies are warranted to investigate their predictive and therapeutic potential, particularly regarding interventions targeting neutrophil-mediated inflammation and platelet hyperactivity in AIS.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Pandian, J.D. and Sudhan, P. (2013) Stroke Epidemiology and Stroke Care Services in India. *Journal of Stroke*, **15**, 128-134. <https://doi.org/10.5853/jos.2013.15.3.128>
- [2] Yew, K.S. and Cheng, E. (2009) Acute Stroke Diagnosis. *American Family Physician*, **80**, 33-40.
- [3] Birenbaum, D., Bancroft, L.W. and Felsberg, G.J. (2011) Imaging in Acute Stroke. *Western Journal of Emergency Medicine*, **12**, 67-76.
- [4] Nour, M. and Liebeskind, D.S. (2014) Imaging of Cerebral Ischemia: From Acute Stroke to Chronic Disorders. *Neurologic Clinics*, **32**, 193-209. <https://doi.org/10.1016/j.ncl.2013.07.005>
- [5] Jin, R., Yang, G. and Li, G. (2010) Inflammatory Mechanisms in Ischemic Stroke: Role of Inflammatory Cells. *Journal of Leukocyte Biology*, **87**, 779-789. <https://doi.org/10.1189/jlb.1109766>
- [6] Kim, J.Y., Park, J., Chang, J.Y., Kim, S. and Lee, J.E. (2016) Inflammation after Ischemic Stroke: The Role of Leukocytes and Glial Cells. *Experimental Neurobiology*, **25**, 241-251. <https://doi.org/10.5607/en.2016.25.5.241>
- [7] Meena, P., Khare, M., Airun, A., *et al.* (2017) A Study of Platelet Indices in Patients of Acute Ischemic Stroke: A Prospective Study. *IOSR Journal of Dental and Medical Sciences*, **16**, 26-29.
- [8] Bates, I. (2017) Reference Ranges and Normal Values. In: Bain, B.J., Bates, I. and Laffan, M.A., Eds., *Dacie and Lewis Practical Haematology*, Elsevier, 8-17. <https://doi.org/10.1016/b978-0-7020-6696-2.00002-3>
- [9] Guo, Y., Li, P., Guo, Q., Shang, K., Yan, D., Du, S., *et al.* (2014) Pathophysiology and Biomarkers in Acute Ischemic Stroke—A Review. *Tropical Journal of Pharmaceutical Research*, **12**, 1097-1105. <https://doi.org/10.4314/tjpr.v12i6.35>
- [10] Deb, P., Sharma, S. and Hassan, K.M. (2010) Pathophysiologic Mechanisms of Acute Ischemic Stroke: An Overview with Emphasis on Therapeutic Significance beyond Thrombolysis. *Pathophysiology*, **17**, 197-218. <https://doi.org/10.1016/j.pathophys.2009.12.001>
- [11] Anrather, J. and Iadecola, C. (2016) Inflammation and Stroke: An Overview. *Neurotherapeutics*, **13**, 661-670. <https://doi.org/10.1007/s13311-016-0483-x>
- [12] Jickling, G.C., Liu, D., Ander, B.P., Stamova, B., Zhan, X. and Sharp, F.R. (2015) Targeting Neutrophils in Ischemic Stroke: Translational Insights from Experimental Studies. *Journal of Cerebral Blood Flow & Metabolism*, **35**, 888-901.

- <https://doi.org/10.1038/jcbfm.2015.45>
- [13] de Jager, C.P., van Wijk, P.T., Mathoera, R.B., de Jongh-Leuvenink, J., van der Poll, T. and Wever, P.C. (2010) Lymphocytopenia and Neutrophil-Lymphocyte Count Ratio Predict Bacteremia Better than Conventional Infection Markers in an Emergency Care Unit. *Critical Care*, **14**, 192-200. <https://doi.org/10.1186/cc9309>
 - [14] Xue, J., Huang, W., Chen, X., Li, Q., Cai, Z., Yu, T., *et al.* (2017) Neutrophil-to-Lymphocyte Ratio Is a Prognostic Marker in Acute Ischemic Stroke. *Journal of Stroke and Cerebrovascular Diseases*, **26**, 650-657. <https://doi.org/10.1016/j.jstrokecerebrovasdis.2016.11.010>
 - [15] Quan, K., Wang, A., Zhang, X., Meng, X., Chen, P., Li, H., *et al.* (2021) Neutrophil to Lymphocyte Ratio and Adverse Clinical Outcomes in Patients with Ischemic Stroke. *Annals of Translational Medicine*, **9**, 1047-1047. <https://doi.org/10.21037/atm-21-710>
 - [16] Ahmed, M., Green, S.R., Pugalendhi, S. and Sanikommu, D.R. (2022) Neutrophil to Lymphocyte Ratio among Section Acute Ischaemic Stroke Patients. *Journal of Clinical and Diagnostic Research*, **16**, OC28-OC30. <https://doi.org/10.7860/jcdr/2022/56872.17144>
 - [17] Elsayed, A.M. and Mohamed, G.A. (2017) Mean Platelet Volume and Mean Platelet Volume/Platelet Count Ratio as a Risk Stratification Tool in the Assessment of Severity of Acute Ischemic Stroke. *Alexandria Journal of Medicine*, **53**, 67-70. <https://doi.org/10.1016/j.ajme.2016.03.003>