

Research Article

Diagnostic Utility of Neutrophil-to-Lymphocyte Ratio and Platelet-to-Lymphocyte Ratio in Acute Inflammatory and Infectious Disorders

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ABSTRACT

Background: Rapid identification of infection and assessment of inflammatory severity are important for early clinical decision-making. Neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are inexpensive inflammatory indices derived from routine complete blood counts. This study evaluated their diagnostic utility in acute inflammatory and infectious disorders.

Methods: This prospective observational diagnostic study included 150 adults with acute infectious or noninfectious inflammatory disorders. NLR and PLR were calculated from initial complete blood counts and compared with clinical diagnosis, C-reactive protein (CRP), and disease severity. Receiver operating characteristic (ROC) analysis was used to determine diagnostic performance, and multivariate logistic regression identified predictors of severe disease.

Results: Infectious disorders were diagnosed in 96 (64.0%) patients, while 54 (36.0%) had noninfectious inflammatory disorders. Median NLR was significantly higher in infectious than noninfectious conditions [6.72 vs 3.61; $p < 0.001$], as was PLR [189.6 vs 139.2; $p < 0.001$]. Patients with severe disease had significantly higher NLR (9.18 vs 4.31) and PLR (229.4 vs 149.8; both $p < 0.001$). NLR correlated positively with CRP ($r = 0.56$; $p < 0.001$). For identifying infectious disorders, NLR > 4.8 demonstrated an AUC of 0.82, sensitivity of 79.2%, specificity of 72.2%, and accuracy of 76.7%, whereas PLR had an AUC of 0.73. On multivariate analysis, NLR > 7 independently predicted severe disease (AOR 3.26; 95% CI: 1.54-6.90; $p = 0.002$).

Conclusion: NLR demonstrated good diagnostic utility for identifying infectious disorders and was independently associated with disease severity. NLR may provide a rapid, inexpensive, and readily available adjunct to conventional inflammatory biomarkers.

Keywords: Neutrophil-To-Lymphocyte Ratio, Platelet-To-Lymphocyte Ratio, Nlr, Plr, Infection, Inflammation, C-Reactive Protein, Disease Severity.

INTRODUCTION

Acute inflammatory and infectious disorders constitute a major proportion of patients presenting to emergency departments and inpatient medical services and are associated with considerable morbidity and mortality.[1] Early recognition of clinically significant inflammation and infection is essential for timely investigation, appropriate antimicrobial therapy, assessment of disease severity, and prevention of complications. However, clinical manifestations such as fever, tachycardia, leukocytosis, and constitutional symptoms are often nonspecific, creating a need for reliable and readily available laboratory biomarkers.[2] The inflammatory response involves complex interactions between circulating leukocytes, platelets, endothelial cells, cytokines, and other mediators of innate and adaptive immunity.[3] Neutrophils represent an important component of the innate immune response and increase

rapidly during acute bacterial infection, tissue injury, and systemic inflammation. In contrast, lymphopenia may occur during severe physiological stress and systemic inflammatory responses as a consequence of redistribution, apoptosis, and altered immune regulation.[4] The neutrophil-to-lymphocyte ratio (NLR), calculated from the absolute neutrophil and lymphocyte counts, integrates these two opposing components of the inflammatory response. NLR has emerged as a simple, inexpensive, and readily available inflammatory marker that can be derived from a routine complete blood count without additional laboratory testing or expenditure.[5] Elevated NLR has been reported in bacterial infections, sepsis, pneumonia, appendicitis, pancreatitis, inflammatory diseases, cardiovascular disorders, and several other acute clinical conditions.[6]

NLR may provide additional information compared with the total leukocyte count because it reflects both neutrophilic inflammatory activation and relative lymphocyte suppression. Several studies have demonstrated associations between elevated NLR and disease severity, systemic inflammatory burden, intensive care requirement, and adverse clinical outcomes.[7] However, the optimal NLR threshold varies among clinical populations, and its diagnostic utility requires interpretation in conjunction with the underlying disease and other inflammatory parameters.

Platelets also participate actively in inflammatory and immune responses in addition to their established role in hemostasis. Activated platelets interact with leukocytes and vascular endothelial cells and release cytokines, chemokines, and other mediators that contribute to inflammatory signaling.[8] Changes in platelet number and activity during acute inflammation have therefore led to increasing interest in platelet-based inflammatory indices.

The platelet-to-lymphocyte ratio (PLR), calculated by dividing the platelet count by the absolute lymphocyte count, has been proposed as another inexpensive marker of systemic inflammation. Elevated PLR has been associated with infectious diseases, systemic inflammatory conditions, cardiovascular disease, malignancy, and adverse clinical outcomes.[9] Because NLR and PLR can both be calculated from routinely available hematological parameters, they may be particularly useful in resource-limited clinical settings.

Traditional inflammatory biomarkers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and procalcitonin are commonly used for the evaluation of acute inflammation and infection. CRP is an acute-phase protein synthesized predominantly by hepatocytes in response to inflammatory cytokines and is widely used as a marker of systemic inflammatory activity.[10] Procalcitonin has gained importance as a biomarker of bacterial infection and may assist in differentiating bacterial infection from noninfectious inflammatory conditions, although its availability and cost may limit routine use in some settings.[11]

The relationship of NLR and PLR with established inflammatory biomarkers provides an opportunity to assess their potential diagnostic value. Significant correlations with

CRP or procalcitonin and reliable discrimination of clinically important infection or inflammation on receiver operating characteristic analysis could support their use as rapid adjunctive markers. However, neither NLR nor PLR is disease-specific, and elevations may occur in a wide variety of physiological and pathological conditions.[12]

Given their simplicity, availability, rapid calculation, and negligible additional cost, NLR and PLR may have practical value in the initial evaluation of patients with acute inflammatory and infectious disorders. The present study was therefore undertaken to assess the diagnostic utility of NLR and PLR and to determine their relationship with conventional inflammatory markers, disease severity, and short-term clinical outcomes.

MATERIALS AND METHODS

This was a prospective observational diagnostic study conducted in the Department of pathology at a tertiary care teaching hospital. Consecutive adult patients presenting to the emergency department or admitted to the medical wards with clinical features suggestive of an acute infectious or inflammatory disorder were screened for eligibility.

Study Population and Sample Size

A total of 150 patients fulfilling the eligibility criteria were enrolled during the study period. Patients were categorized into infectious and noninfectious inflammatory groups according to the final clinical diagnosis supported by relevant laboratory, microbiological, radiological, and other investigations.

Inclusion Criteria

Patients were included if they fulfilled the following criteria:

1. Age ≥ 18 years.
2. Acute onset of clinical features suggestive of infection or inflammation.
3. Presence of one or more features such as fever, leukocytosis, localized inflammatory symptoms, or elevated conventional inflammatory markers.
4. Availability of complete blood count with differential leukocyte count at initial evaluation.
5. Availability of relevant clinical and laboratory information necessary for establishing the final diagnosis.
6. Written informed consent for participation.

Exclusion Criteria

Patients with the following conditions were excluded:

1. Known hematological malignancy.
2. Active solid-organ malignancy receiving chemotherapy.
3. Recent chemotherapy or radiotherapy.
4. Chronic immunosuppressive therapy or systemic corticosteroid use.
5. Known primary bone marrow disorder.
6. Recent major surgery or significant trauma.
7. Pregnancy.
8. Recent blood or platelet transfusion.
9. Inadequate or incomplete laboratory data.

Clinical Assessment

A detailed clinical history and physical examination were performed at presentation. Demographic characteristics, duration of illness, fever, suspected source of infection or inflammation, comorbidities, medication history, heart rate, respiratory rate, blood pressure, oxygen saturation, and body temperature were recorded.

Patients were monitored for indicators of disease severity, including hypotension, hypoxemia, organ dysfunction, requirement for intensive care, prolonged hospitalization, and in-hospital mortality.

Laboratory Investigations

Venous blood samples were collected at the time of initial clinical evaluation, preferably before substantial therapeutic intervention. Laboratory investigations included:

- Complete blood count
- Total leukocyte count
- Differential leukocyte count
- Absolute neutrophil count
- Absolute lymphocyte count
- Platelet count
- C-reactive protein
- Procalcitonin, where indicated
- Renal function tests
- Liver function tests
- Serum electrolytes
- Blood glucose

Microbiological cultures, imaging, and other disease-specific investigations were performed according to clinical indications.

Calculation of Neutrophil-to-Lymphocyte Ratio

NLR was calculated from the initial complete blood count as:

NLR = Absolute neutrophil count / Absolute lymphocyte count

The calculated NLR was recorded for each participant and evaluated in relation to final diagnosis, inflammatory biomarkers, and clinical severity.

Calculation of Platelet-to-Lymphocyte Ratio

PLR was calculated as:

PLR = Platelet count / Absolute lymphocyte count

PLR was similarly analyzed according to diagnostic category, inflammatory burden, and disease severity.

Diagnostic Classification

Based on the final clinical diagnosis, patients were broadly categorized into:

Infectious disorders

These included bacterial respiratory infections, urinary tract infections, gastrointestinal/intra-abdominal infections, skin and soft-tissue infections, bloodstream infections, and other clinically or microbiologically confirmed infections.

Noninfectious inflammatory disorders

These included acute pancreatitis, inflammatory/autoimmune exacerbations, acute inflammatory conditions without evidence of infection, and other appropriate noninfectious inflammatory disorders.

Final classification was established using the overall clinical picture together with biochemical, microbiological, radiological, and other relevant findings.

Assessment of Disease Severity

Patients were categorized into **non-severe** and **severe disease** groups based on the presence of clinically significant organ dysfunction and/or requirement for intensive monitoring or intensive care.

Indicators of severe disease included:

- Hemodynamic instability
- Hypoxemia requiring significant oxygen or ventilatory support
- Acute kidney injury
- Altered sensorium
- Multiple-organ dysfunction
- ICU admission

Assessment of Clinical Outcomes

Patients were followed during hospitalization. The following outcomes were recorded:

- Requirement for ICU admission
- Length of hospital stay
- Development of organ dysfunction

- Need for mechanical ventilation
- In-hospital mortality
- Composite adverse clinical outcome

Comparison with Conventional Inflammatory Markers

NLR and PLR were compared with CRP and, where available, procalcitonin. Their relationships with total leukocyte count and other markers of systemic inflammatory activity were also assessed.

Diagnostic Performance

Receiver operating characteristic (ROC) curve analysis was performed to assess the ability of NLR and PLR to discriminate infectious from noninfectious inflammatory disorders.

The optimal cut-off values were determined, and the following diagnostic parameters were calculated:

- Sensitivity
- Specificity
- Positive predictive value
- Negative predictive value
- Diagnostic accuracy
- Area under the ROC curve with 95% confidence interval

The diagnostic performance of NLR and PLR was compared with conventional inflammatory parameters where appropriate.

Outcome Measures

The **primary outcome** was the diagnostic performance of NLR and PLR for differentiating infectious from noninfectious acute inflammatory disorders.

Secondary outcomes included correlations of NLR and PLR with CRP and procalcitonin, their association with disease severity, and their ability to predict ICU requirement and other adverse in-hospital outcomes.

Statistical Analysis

Data were entered into a spreadsheet and analyzed using SPSS.17 statistical software. Continuous variables were expressed as mean $\pm$ standard deviation for normally distributed data and median with interquartile range for

non-normally distributed data. Categorical variables were expressed as frequencies and percentages.

Normality was assessed using the Shapiro–Wilk test. Continuous variables between two groups were compared using the independent-samples Student's t-test or Mann–Whitney U test, as appropriate. Comparisons among more than two groups were performed using ANOVA or the Kruskal–Wallis test. Categorical variables were analyzed using the Chi-square test or Fisher's exact test.

Correlations between NLR, PLR, CRP, procalcitonin, and other laboratory parameters were evaluated using Pearson or Spearman correlation analysis, as appropriate.

ROC curve analysis was used to determine the diagnostic performance of NLR and PLR. AUC with 95% confidence intervals, optimal cut-off values, sensitivity, specificity, PPV, NPV, and diagnostic accuracy were calculated.

Multivariate logistic regression analysis was performed to determine whether elevated NLR and PLR were independently associated with severe disease or adverse clinical outcomes after adjustment for relevant clinical and laboratory variables. Adjusted odds ratios with 95% confidence intervals were reported. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 150 patients with acute inflammatory or infectious disorders were included in the study. The mean age was 47.8 ± 16.2 years, and 86 (57.3%) patients were male. Infectious disorders were diagnosed in 96 (64.0%) patients, while 54 (36.0%) had noninfectious inflammatory disorders. Among infectious conditions, respiratory tract infections were the most frequent (26.0%), followed by urinary tract infections (14.0%) and gastrointestinal/intra-abdominal infections (10.7%). Hypertension and diabetes mellitus were present in 34.7% and 30.7% of patients, respectively (Table 1).

Table 1. Baseline demographic, clinical, and diagnostic characteristics of the study population (N=150)

Variable	n (%) / Mean $\pm$ SD
Age (years), mean $\pm$ SD	47.8 $\pm$ 16.2
Age ≥ 60 years	42 (28.0)
Male sex	86 (57.3)
Female sex	64 (42.7)
Diabetes mellitus	46 (30.7)

Hypertension	52 (34.7)
Diagnostic category	
Infectious disorders	96 (64.0)
Noninfectious inflammatory disorders	54 (36.0)
Infectious disorders	
Respiratory tract infection	39 (26.0)
Urinary tract infection	21 (14.0)
Gastrointestinal/intra-abdominal infection	16 (10.7)
Skin/soft-tissue infection	10 (6.7)
Bloodstream/other infection	10 (6.7)
Noninfectious inflammatory disorders	
Acute pancreatitis	21 (14.0)
Autoimmune/inflammatory exacerbation	17 (11.3)
Other acute inflammatory disorders	16 (10.7)

The median NLR and PLR in the overall study population were 5.42 (IQR: 3.31–8.76) and 168.5 (IQR: 126.4–231.7), respectively. Median CRP was 46.8 mg/L (IQR: 21.5–96.4), while the

median procalcitonin among patients in whom it was measured was 0.58 ng/mL (IQR: 0.18–2.16). The overall hematological and inflammatory profile is summarized in Table 2.

Table 2. Hematological and inflammatory parameters of the study population (N=150)

Parameter	Value
Hemoglobin (g/dL), mean $\pm$ SD	11.8 $\pm$ 2.1
Total leukocyte count (/ μ L), median (IQR)	12,480 (9,620–16,850)
Absolute neutrophil count (/ μ L), median (IQR)	9,240 (6,520–13,160)
Absolute lymphocyte count (/ μ L), median (IQR)	1,720 (1,180–2,240)
Platelet count ($\times 10^3$ / μ L), mean $\pm$ SD	276.4 $\pm$ 98.6
NLR, median (IQR)	5.42 (3.31–8.76)
PLR, median (IQR)	168.5 (126.4–231.7)
CRP (mg/L), median (IQR)	46.8 (21.5–96.4)
Procalcitonin (ng/mL), median (IQR)*	0.58 (0.18–2.16)
Serum creatinine (mg/dL), mean $\pm$ SD	1.18 $\pm$ 0.64

*Procalcitonin was assessed where clinically indicated.

Patients with infectious disorders demonstrated significantly greater inflammatory responses than those with noninfectious inflammatory disorders. Median NLR was significantly higher in the infectious group [6.72 (4.41–10.34)] compared with the noninfectious group [3.61

(2.48–5.26); $p < 0.001$]. Similarly, median PLR was significantly higher among patients with infection [189.6 (143.8–256.4) vs 139.2 (105.7–181.3); $p < 0.001$]. CRP and total leukocyte count were also significantly elevated in the infectious group (Table 3).

Table 3. Comparison of inflammatory and hematological parameters between infectious and noninfectious inflammatory disorders

Parameter	Infectious (n=96)	Noninfectious inflammatory (n=54)	p-value
Total leukocyte count (/ μ L), median (IQR)	13,860 (10,720–18,140)	10,320 (8,410–13,760)	<0.001
Absolute neutrophil count (/ μ L), median (IQR)	10,580 (7,840–14,630)	6,820 (5,180–9,210)	<0.001
Absolute lymphocyte count (/ μ L), median (IQR)	1,510 (1,020–2,010)	1,920 (1,450–2,480)	0.002
NLR, median (IQR)	6.72 (4.41–10.34)	3.61 (2.48–5.26)	<0.001
PLR, median (IQR)	189.6 (143.8–256.4)	139.2 (105.7–181.3)	<0.001
CRP (mg/L), median (IQR)	64.2 (32.8–122.5)	28.7 (14.4–59.6)	<0.001

Disease severity was evaluated in all 150 patients. Severe disease was identified in 48 (32.0%) patients, while 102 (68.0%) had non-severe disease. Patients with severe disease demonstrated markedly higher NLR [9.18 vs

4.31; $p < 0.001$] and PLR [229.4 vs 149.8; $p < 0.001$]. CRP was also significantly elevated in severe disease. ICU admission was required in 31 (20.7%) patients, while 8 (5.3%) patients died during hospitalization (Table 4).

Table 4. Association of NLR and PLR with disease severity and clinical outcomes

Parameter	Non-severe (n=102)	Severe (n=48)	p-value
NLR, median (IQR)	4.31 (2.91–6.24)	9.18 (6.54–13.72)	<0.001
PLR, median (IQR)	149.8 (113.6–194.7)	229.4 (171.5–304.8)	<0.001
CRP (mg/L), median (IQR)	34.5 (17.2–68.6)	103.8 (61.4–164.2)	<0.001
TLC (/ μ L), median (IQR)	11,320 (8,940–14,720)	16,580 (12,610–21,460)	<0.001
ICU admission, n (%)	3 (2.9)	28 (58.3)	<0.001
Mechanical ventilation, n (%)	1 (1.0)	15 (31.3)	<0.001
In-hospital mortality, n (%)	0 (0.0)	8 (16.7)	<0.001
Hospital stay (days), median (IQR)	5 (3–7)	9 (6–13)	<0.001

ROC curve analysis demonstrated good diagnostic discrimination of NLR for differentiating infectious from noninfectious inflammatory disorders. At an optimal cut-off of >4.8, NLR had a sensitivity of 79.2%, specificity of 72.2%, and AUC of 0.82 (95% CI: 0.75–

0.89; $p < 0.001$). PLR >160 demonstrated a sensitivity of 70.8%, specificity of 66.7%, and AUC of 0.73. CRP showed an AUC of 0.80. Thus, NLR demonstrated better diagnostic discrimination than PLR and comparable performance to CRP (Table 5).

Table 5. Diagnostic performance of NLR, PLR, and CRP for identifying infectious disorders

Parameter	NLR	PLR	CRP
Optimal cut-off	>4.8	>160	>40 mg/L
Sensitivity (%)	79.2	70.8	76.0
Specificity (%)	72.2	66.7	72.2
PPV (%)	83.5	79.1	82.0
NPV (%)	66.1	56.3	62.9
Accuracy (%)	76.7	69.3	74.7
AUC	0.82	0.73	0.80
95% CI	0.75–0.89	0.65–0.81	0.73–0.87
p-value	<0.001	<0.001	<0.001

Correlation analysis further demonstrated a significant positive relationship between NLR and CRP (Spearman $r = 0.56$; $p < 0.001$). PLR was also positively correlated with CRP, although the relationship was weaker ($r = 0.39$; $p < 0.001$). NLR and PLR were positively correlated with each other ($r = 0.47$; $p < 0.001$), supporting their relationship with systemic inflammatory activity. Multivariate logistic regression analysis was performed to determine independent predictors

of severe disease. After adjustment for age, infection status, CRP, and other relevant variables, NLR >7 remained independently associated with severe disease (AOR 3.26; 95% CI: 1.54–6.90; $p = 0.002$). CRP >75 mg/L was also an independent predictor (AOR 2.72; $p = 0.008$). PLR >200 demonstrated an increased risk of severe disease but did not retain statistical significance after adjustment (AOR 1.78; $p = 0.14$) (Table 6).

Table 6. Multivariate logistic regression analysis of predictors of severe disease

Predictor	Adjusted OR	95% CI	p-value
Age ≥ 60 years	1.84	0.86–3.94	0.12
Infectious etiology	2.18	1.02–4.66	0.044
NLR >7	3.26	1.54–6.90	0.002
PLR >200	1.78	0.83–3.82	0.14
CRP >75 mg/L	2.72	1.30–5.71	0.008

Overall, NLR and PLR were significantly elevated in infectious compared with noninfectious inflammatory disorders and increased further with increasing disease severity. NLR demonstrated stronger diagnostic discrimination than PLR, with an AUC of 0.82 for identifying infectious disorders. Furthermore, NLR >7 remained an independent predictor of severe disease after adjustment for relevant clinical and inflammatory variables, suggesting that NLR may serve as a simple and readily available adjunctive marker for both diagnostic assessment and early risk stratification.

DISCUSSION

The present study evaluated the diagnostic utility of neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) in 150 patients with acute inflammatory and infectious disorders. Infectious disorders accounted for 64.0% of cases, while 36.0% had noninfectious inflammatory conditions. The principal finding was that both NLR and PLR were significantly elevated in patients with infectious disorders and increased further with disease severity. NLR demonstrated better diagnostic discrimination than PLR and remained independently associated with severe disease after adjustment for other relevant factors.

The mean age of the study population was 47.8 ± 16.2 years, with a slight male predominance (57.3%). Respiratory tract infections were the most frequent infectious disorders, followed by urinary tract and gastrointestinal/intra-abdominal infections. The inflammatory response to infection is characterized by activation of innate immunity, increased neutrophil production and recruitment, and alterations in lymphocyte number and function. These hematological changes provide the physiological basis for using leukocyte-derived inflammatory indices in acute infection.[13]

The median NLR in the overall study population was 5.42, while the median PLR was 168.5. Patients with infectious disorders had a significantly higher NLR than those with noninfectious inflammatory conditions [6.72 vs 3.61; $p < 0.001$]. This observation is consistent with previous studies showing that NLR increases during bacterial infection and systemic inflammation. Zahorec described the ratio of neutrophils to lymphocytes as a simple parameter reflecting the intensity of systemic inflammation and physiological stress.[14]

The increased NLR in infectious disorders results from two complementary changes:

neutrophilia and relative lymphopenia. Neutrophilia reflects activation of innate immunity and mobilization of neutrophils from bone marrow and marginal pools, whereas lymphopenia may result from stress-mediated redistribution and apoptosis of lymphocytes during severe inflammation.[15] Consequently, NLR may amplify information contained within the differential leukocyte count and provide greater discriminatory ability than either neutrophil or lymphocyte counts alone.

de Jager et al. evaluated NLR in patients with community-acquired pneumonia and demonstrated that increased NLR was associated with disease severity and mortality and provided useful prognostic information.[16] Similarly, Gürol et al. reported that NLR could assist in predicting bacteremia and showed a relationship with systemic inflammatory activity.[17] These observations support the significantly higher NLR found among patients with infectious disorders in the present study.

PLR was also significantly elevated in the infectious group compared with the noninfectious inflammatory group [189.6 vs 139.2; $p < 0.001$]. Platelets have important functions beyond hemostasis and actively participate in inflammation through interactions with leukocytes, endothelial cells, and inflammatory mediators. Platelet activation may contribute to immune responses, microvascular inflammation, and thrombo-inflammatory processes during infection.[18] PLR therefore provides an integrated measure of platelet-related inflammatory activation and alterations in lymphocyte count.

Previous studies have demonstrated associations between elevated PLR and systemic inflammation, infectious disease severity, and adverse outcomes.[19] However, PLR is influenced by several factors, including reactive thrombocytosis, thrombocytopenia during severe sepsis, hematological disorders, and medication use. These factors may partly explain why PLR generally showed weaker diagnostic and prognostic performance than NLR in the present study.

A particularly important observation was the relationship between these hematological indices and disease severity. Patients with severe disease had a median NLR of 9.18 compared with 4.31 among those with non-severe disease ($p < 0.001$). Similarly, median PLR increased from 149.8 to 229.4 ($p < 0.001$). Severe cases also demonstrated significantly higher CRP concentrations, leukocyte counts,

ICU admission, mechanical ventilation requirement, and in-hospital mortality. These findings indicate that increasing systemic inflammatory burden is reflected in both NLR and PLR.

NLR showed a significant positive correlation with CRP ($r=0.56$; $p<0.001$), while PLR demonstrated a weaker but significant correlation ($r=0.39$; $p<0.001$). CRP is an established acute-phase reactant synthesized predominantly in the liver in response to pro-inflammatory cytokines, particularly interleukin-6.[20] The correlations observed in the present study suggest that CBC-derived inflammatory indices reflect, at least partly, the same systemic inflammatory processes represented by conventional acute-phase biomarkers.

ROC curve analysis demonstrated an AUC of 0.82 for NLR in differentiating infectious from noninfectious inflammatory disorders. At a cut-off of >4.8 , NLR demonstrated 79.2% sensitivity, 72.2% specificity, and 76.7% diagnostic accuracy. PLR >160 showed lower diagnostic performance, with an AUC of 0.73, sensitivity of 70.8%, and specificity of 66.7%. NLR also demonstrated diagnostic performance comparable to CRP, which had an AUC of 0.80. Previous investigations have similarly demonstrated moderate-to-good discriminatory performance of NLR in infectious diseases and sepsis, although reported cut-off values vary considerably.[21] Differences in patient populations, infection sites, disease severity, underlying comorbidities, laboratory methods, and definitions of infection may account for variability in optimal thresholds. Therefore, a universal NLR cut-off is unlikely to be appropriate across all clinical conditions.

In the present study, NLR >7 remained independently associated with severe disease on multivariate analysis (AOR 3.26; 95% CI: 1.54–6.90; $p=0.002$). CRP >75 mg/L was also an independent predictor of severe disease. In contrast, PLR >200 did not retain statistical significance after adjustment. These findings suggest that NLR may be a more robust marker of systemic inflammatory severity than PLR. Similar associations between elevated NLR and adverse outcomes have been demonstrated in patients with sepsis and other critical illnesses.[22]

The relationship between elevated NLR and poor outcomes is biologically plausible. Excessive neutrophil activation may contribute to tissue injury through the release of reactive oxygen species, proteolytic enzymes, cytokines, and neutrophil extracellular traps. At the same

time, lymphopenia may reflect impaired adaptive immune function and immune dysregulation, particularly in severe infection and sepsis.[23] A high NLR therefore potentially represents the combined effects of exaggerated innate inflammation and relative adaptive immune suppression. The present findings have practical clinical implications. Both NLR and PLR can be calculated rapidly from a routine complete blood count without requiring additional blood sampling, specialized assays, or additional expenditure. NLR in particular may therefore be useful as an adjunctive marker in emergency departments, general medical wards, and resource-limited healthcare settings. It could assist clinicians in identifying patients who warrant more intensive evaluation or monitoring.

Nevertheless, neither NLR nor PLR should be regarded as disease-specific diagnostic tests. Both parameters may be altered by age, physiological stress, medications, malignancy, hematological conditions, cardiovascular disease, and other inflammatory states.[23] Their greatest clinical value is therefore likely to be as complementary markers interpreted alongside history, physical examination, microbiological findings, imaging, and established inflammatory biomarkers rather than as standalone diagnostic tests.

Overall, the present study demonstrates that NLR and PLR are useful indicators of systemic inflammatory activity in acute infectious and inflammatory disorders. NLR showed superior diagnostic discrimination compared with PLR, correlated more strongly with CRP, and independently predicted severe disease. These findings support the potential incorporation of NLR into routine initial laboratory assessment for rapid and inexpensive adjunctive risk stratification.

CONCLUSION

NLR and PLR were significantly elevated in infectious disorders and increased with greater disease severity. NLR demonstrated better diagnostic performance than PLR and was independently associated with severe disease. As a simple and inexpensive CBC-derived parameter, NLR may serve as a useful adjunct to conventional inflammatory markers for early diagnostic assessment and risk stratification.

Limitations

The study was limited by its single-centre design, heterogeneous spectrum of infectious and inflammatory disorders, and relatively

modest sample size of 150 patients. Microbiological confirmation was not available in every infectious case, and procalcitonin was not uniformly measured in all participants. Serial NLR and PLR measurements were also not performed, limiting assessment of dynamic changes during treatment.

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