

Research Article

Association of Neutrophil-to-Lymphocyte Ratio with Severity and Prognosis of Acute Coronary Syndrome

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ABSTRACT

Background: Inflammation plays a critical role in the development and progression of atherosclerosis and acute coronary syndrome (ACS). The neutrophil-to-lymphocyte ratio (NLR), an easily available marker of systemic inflammation, has emerged as a potential predictor of coronary artery disease severity and adverse cardiovascular outcomes. This study aimed to evaluate the association of NLR with angiographic severity and prognosis among patients presenting with ACS.

Methods: This prospective observational cohort study included 150 patients with ACS. Baseline demographic characteristics, cardiovascular risk factors, clinical presentation, laboratory parameters, and NLR values were recorded at admission. Coronary angiographic findings were assessed to determine the extent of coronary artery disease. Patients were categorized into low NLR (<3.5) and high NLR (≥ 3.5) groups. The association of NLR with angiographic severity and in-hospital major adverse cardiovascular events (MACE) was analyzed. Multivariate logistic regression was performed to identify independent predictors of adverse outcomes.

Results: The mean age of patients was 58.6 ± 11.4 years, and 74.7% were males. The mean NLR value was 3.8 ± 1.9 . Triple vessel disease was associated with significantly higher NLR values compared with single and double vessel disease (5.1 ± 2.0 vs 2.6 ± 1.1 and 3.7 ± 1.5 ; $p < 0.001$). Patients with high NLR had increased prevalence of STEMI (59.7% vs 37.2%; $p = 0.006$) and lower LVEF ($43.2 \pm 9.4\%$ vs $50.1 \pm 8.7\%$; $p < 0.001$). High NLR was significantly associated with increased MACE (31.9% vs 10.3%; $p < 0.001$), mortality (12.5% vs 2.6%; $p = 0.03$), and heart failure (25.0% vs 7.7%; $p = 0.004$). On multivariate analysis, high NLR independently predicted MACE (OR: 3.6; 95% CI: 1.8–7.2; $p < 0.001$).

Conclusion: Elevated NLR is significantly associated with increased coronary disease severity and adverse in-hospital outcomes in patients with ACS. NLR may serve as a simple, cost-effective, and clinically useful biomarker for early risk stratification and prognostic assessment.

Keywords: Acute Coronary Syndrome, Neutrophil-To-Lymphocyte Ratio, Inflammation, Coronary Artery Disease, Prognosis, Major Adverse Cardiovascular Events.

INTRODUCTION

Acute coronary syndrome (ACS) is a spectrum of acute ischemic heart diseases resulting from sudden reduction or interruption of coronary blood flow, most commonly due to rupture or erosion of an atherosclerotic plaque followed by thrombus formation[1]. It includes unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). Despite significant advancements in early diagnosis, reperfusion therapy, antiplatelet agents, and secondary prevention strategies, ACS continues to be a major cause of cardiovascular morbidity and mortality worldwide[2]. Early identification of patients at increased risk of adverse

outcomes remains essential for timely intervention and improved clinical management[3,4]. Atherosclerosis, the fundamental pathological process underlying ACS, is a complex inflammatory disorder influenced by genetic, environmental, and metabolic factors. Conventional cardiovascular risk factors such as diabetes mellitus, hypertension, dyslipidemia, smoking, and metabolic syndrome contribute to endothelial dysfunction, oxidative stress, and chronic vascular inflammation, accelerating plaque formation and progression[5,6]. Inflammatory activation within the atherosclerotic plaque leads to endothelial injury, extracellular matrix degradation, plaque instability, and eventual

rupture, resulting in myocardial ischemia and acute coronary events[7]. Inflammatory cells, particularly neutrophils and lymphocytes, play contrasting but important roles in the pathogenesis and progression of coronary artery disease. Neutrophils contribute to acute inflammatory responses through the release of reactive oxygen species, proteolytic enzymes, cytokines, and matrix metalloproteinases, promoting endothelial damage, plaque destabilization, thrombus formation, and myocardial injury. Conversely, lymphocytes are involved in immune regulation, and reduced lymphocyte counts may reflect physiological stress, impaired immune modulation, and an exaggerated inflammatory response associated with adverse cardiovascular outcomes[8,9]. The neutrophil-to-lymphocyte ratio (NLR), derived from routine complete blood count parameters, has emerged as a simple, cost-effective, and easily accessible marker of systemic inflammation[10]. It represents the balance between neutrophil-mediated inflammatory activation and lymphocyte-mediated regulatory response, thereby providing a comprehensive assessment of inflammatory status. Unlike individual leukocyte parameters, NLR may better reflect the underlying inflammatory burden and has gained importance as a prognostic marker in cardiovascular diseases[11]. Previous studies have demonstrated that elevated NLR is associated with increased severity of coronary artery disease, higher thrombus burden, adverse angiographic characteristics, and poor clinical outcomes in patients with ACS. Increased NLR has been linked with higher incidence of major adverse cardiovascular events (MACE), including mortality, recurrent myocardial infarction, heart failure, and complications following percutaneous coronary intervention (PCI). Therefore, NLR may provide additional prognostic information beyond conventional clinical risk assessment tools[12,13]. Considering its biological significance, low cost, and routine availability, evaluation of NLR may help in early risk stratification and prognostic assessment of patients with ACS, particularly those undergoing PCI. Hence, the present study was undertaken to assess the association of neutrophil-to-lymphocyte ratio with disease severity and prognosis among patients presenting with acute coronary syndrome.

MATERIALS AND METHODS

This was a prospective observational cohort study conducted among patients presenting with acute coronary syndrome (ACS) at a tertiary care hospital. The study was designed to evaluate the association between neutrophil-to-lymphocyte ratio (NLR) and the severity of coronary artery disease, as well as its prognostic significance among patients with ACS undergoing evaluation and management. A total of 150 consecutive patients diagnosed with acute coronary syndrome were included in the study. Patients were recruited from the cardiology department during the study period based on predefined inclusion and exclusion criteria.

Inclusion Criteria

Patients were included if they fulfilled the following criteria:

- Age ≥ 18 years.
- Patients diagnosed with acute coronary syndrome, including:
 - ST-segment elevation myocardial infarction (STEMI).
 - Non-ST-segment elevation myocardial infarction (NSTEMI).
 - Unstable angina.
- Patients undergoing coronary angiographic evaluation.
- Patients willing to provide written informed consent.

Exclusion Criteria

Patients were excluded if they had:

- Active infection or systemic inflammatory disease.
- Known hematological disorders or malignancy.
- Chronic inflammatory or autoimmune disorders.
- Severe hepatic or renal dysfunction.
- Recent surgery, trauma, or major infection.
- Current use of immunosuppressive therapy.
- Incomplete clinical or laboratory data.

Clinical Assessment and Data Collection

Baseline demographic and clinical characteristics were recorded for all enrolled patients. Data regarding age, sex, cardiovascular risk factors, clinical presentation, previous history of coronary artery disease, diabetes mellitus, hypertension, dyslipidemia, and smoking status were collected. Patients were evaluated clinically at admission, and details regarding the type of ACS, electrocardiographic findings, cardiac

biomarkers, echocardiographic parameters, treatment received, and angiographic findings were documented.

Laboratory Investigations

Venous blood samples were collected at the time of hospital admission before initiation of definitive therapy. Complete blood count analysis was performed using an automated hematology analyzer. Absolute neutrophil count and absolute lymphocyte count were recorded, and the neutrophil-to-lymphocyte ratio (NLR) was calculated using the formula:

$$\text{NLR} = \frac{\text{Absolute neutrophil count}}{\text{Absolute lymphocyte count}}$$

Additional investigations including cardiac biomarkers (troponin levels), lipid profile, renal function tests, and other routine biochemical parameters were performed as per standard clinical protocols.

Assessment of Coronary Artery Disease Severity

All patients underwent coronary angiography, and the severity of coronary artery disease was assessed based on angiographic findings. The extent of coronary involvement was evaluated according to:

- Number of diseased coronary vessels (single-, double-, or triple-vessel disease).
- Degree of coronary artery stenosis.
- Presence of significant coronary lesions.

The angiographic severity was correlated with baseline NLR values to evaluate the association between inflammatory status and coronary disease burden.

Assessment of Prognosis and Clinical Outcomes

Patients were followed during the hospital stay and evaluated for clinical outcomes. Prognostic assessment included documentation of major adverse cardiovascular events (MACE), including:

- In-hospital mortality.
- Recurrent myocardial infarction.
- Heart failure.
- Cardiogenic shock.
- Requirement for repeat revascularization.

The association between admission NLR levels and adverse clinical outcomes was analyzed.

Statistical Analysis

Data were entered into a Microsoft Excel spreadsheet and analyzed using SPSS.21 statistical software. Continuous variables were expressed as mean $\pm$ standard deviation (SD) or median with interquartile

range (IQR) depending on data distribution, while categorical variables were presented as frequencies and percentages. Normality of continuous variables was assessed using the Shapiro–Wilk test. Comparisons between groups were performed using the independent t-test or Mann–Whitney U test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables. The correlation between NLR and angiographic severity parameters was assessed using Pearson's or Spearman's correlation analysis. Receiver operating characteristic (ROC) curve analysis was performed to determine the predictive ability of NLR for adverse outcomes, including calculation of area under the curve (AUC), sensitivity, specificity, and optimal cut-off value. Multivariate logistic regression analysis was performed to identify independent predictors of adverse clinical outcomes. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 150 patients with acute coronary syndrome (ACS) were included in the present study. The mean age of the study population was 58.6 ± 11.4 years, with 78 (52.0%) patients aged ≥ 60 years. Males constituted the majority of the cohort (112; 74.7%), while females accounted for 38 (25.3%) patients. Among cardiovascular risk factors, hypertension was the most common comorbidity (86; 57.3%), followed by diabetes mellitus (62; 41.3%) and dyslipidemia (71; 47.3%). Current smoking was reported in 68 (45.3%) patients, while previous history of coronary artery disease was present in 34 (22.7%) patients. The mean BMI of the study population was 26.4 ± 3.8 kg/m² (Table 1).

Regarding clinical presentation, STEMI was the most common ACS subtype, observed in 72 (48.0%) patients, followed by NSTEMI in 56 (37.3%) and unstable angina in 22 (14.7%) patients. The mean hemoglobin level was 13.1 ± 1.8 g/dL, while the mean total leukocyte count was 9200 ± 2400 cells/mm³. The mean neutrophil and lymphocyte counts were 6400 ± 2100 cells/mm³ and 1800 ± 600 cells/mm³, respectively. The mean neutrophil-to-lymphocyte ratio (NLR) was 3.8 ± 1.9 , with a median value of 3.4 (IQR: 2.2–4.8). Troponin-I elevation was observed in 128 (85.3%) patients, and the mean left ventricular ejection fraction (LVEF) was $46.8 \pm 9.5\%$ (Table 2).

Coronary angiographic evaluation demonstrated that triple vessel disease was

present in 57 (38.0%) patients, followed by double vessel disease in 51 (34.0%) and single vessel disease in 42 (28.0%) patients. A progressive increase in NLR was observed with increasing severity of coronary artery involvement. Patients with triple vessel disease had significantly higher NLR values (5.1 ± 2.0) compared with double vessel disease (3.7 ± 1.5) and single vessel disease (2.6 ± 1.1) ($p < 0.001$), indicating a significant association between elevated NLR and angiographic severity of coronary artery disease (Table 3). Patients were categorized into low NLR (< 3.5) and high NLR (≥ 3.5) groups. The high NLR group had significantly higher mean age compared with the low NLR group (62.1 ± 11.3 vs 55.4 ± 10.6 years; $p < 0.001$). Diabetes mellitus was significantly more frequent among patients with high NLR (51.4% vs 32.1%; $p = 0.01$). STEMI presentation was also significantly higher in the high NLR group (59.7% vs 37.2%; $p = 0.006$). Patients with elevated NLR demonstrated higher frequency of troponin elevation (91.7% vs 79.5%; $p = 0.04$) and significantly reduced LVEF ($43.2 \pm 9.4\%$ vs $50.1 \pm 8.7\%$; $p < 0.001$) compared with patients with low NLR (Table 4). The comparison of clinical and laboratory parameters between the two NLR categories is illustrated in Figure 1. During the hospital stay, patients with high NLR experienced significantly greater adverse

clinical outcomes. The incidence of major adverse cardiovascular events (MACE) was significantly higher among patients with NLR ≥ 3.5 compared with those with NLR < 3.5 (31.9% vs 10.3%; $p < 0.001$). High NLR was also associated with increased mortality (12.5% vs 2.6%; $p = 0.03$), heart failure (25.0% vs 7.7%; $p = 0.004$), and cardiogenic shock (9.7% vs 1.3%; $p = 0.04$). Although recurrent myocardial infarction was more frequent in the high NLR group, the difference did not reach statistical significance (11.1% vs 3.8%; $p = 0.08$) (Table 5). The relationship between NLR category and in-hospital outcomes is depicted in Figure 2. On multivariate logistic regression analysis, high NLR (≥ 3.5) was identified as an independent predictor of adverse cardiovascular outcomes. Patients with elevated NLR had 3.6-fold higher odds of developing MACE (adjusted OR: 3.6; 95% CI: 1.8–7.2; $p < 0.001$). Other independent predictors included triple vessel disease (OR: 3.2; 95% CI: 1.7–6.1; $p < 0.001$), reduced LVEF $< 40\%$ (OR: 2.7; 95% CI: 1.4–5.2; $p = 0.003$), STEMI presentation (OR: 2.4; 95% CI: 1.3–4.6; $p = 0.006$), diabetes mellitus (OR: 1.8; 95% CI: 1.0–3.4; $p = 0.04$), and age ≥ 60 years (OR: 1.9; 95% CI: 1.1–3.6; $p = 0.03$) (Table 6). The independent predictors of adverse outcomes are presented graphically in the forest plot (Figure 3).

Table 1. Baseline Demographic and Clinical Characteristics of Study Population (N=150)

Variable	n (%) / Mean $\pm$ SD
Age (years), mean $\pm$ SD	58.6 $\pm$ 11.4
Age ≥ 60 years	78 (52.0)
Male sex	112 (74.7)
Female sex	38 (25.3)
Diabetes mellitus	62 (41.3)
Hypertension	86 (57.3)
Dyslipidemia	71 (47.3)
Current smoking	68 (45.3)
Previous CAD history	34 (22.7)
Family history of CAD	39 (26.0)
BMI (kg/m ²), mean $\pm$ SD	26.4 $\pm$ 3.8

Table 2. Clinical Presentation, Laboratory Parameters and NLR Distribution Among Study Participants (N=150)

Parameter	Value
Type of ACS	
STEMI	72 (48.0)
NSTEMI	56 (37.3)
Unstable angina	22 (14.7)
Hemoglobin (g/dL), mean $\pm$ SD	13.1 $\pm$ 1.8
Total leukocyte count (cells/mm ³), mean $\pm$ SD	9200 $\pm$ 2400

Neutrophil count (cells/mm ³), mean $\pm$ SD	6400 $\pm$ 2100
Lymphocyte count (cells/mm ³), mean $\pm$ SD	1800 $\pm$ 600
NLR, mean $\pm$ SD	3.8 $\pm$ 1.9
Median NLR (IQR)	3.4 (2.2–4.8)
Troponin-I elevation	128 (85.3)
Mean LVEF (%)	46.8 $\pm$ 9.5

Table 3. Association of NLR with Severity of Coronary Artery Disease on Angiography

Angiographic Severity	Number of Patients (n=150)	NLR (Mean $\pm$ SD)	p-value
Single vessel disease	42 (28.0)	2.6 $\pm$ 1.1	
Double vessel disease	51 (34.0)	3.7 $\pm$ 1.5	
Triple vessel disease	57 (38.0)	5.1 $\pm$ 2.0	<0.001

Table 4. Comparison of Clinical and Laboratory Parameters According to NLR Category

Parameter	Low NLR (<3.5) (n=78)	High NLR ($\geq$ 3.5) (n=72)	p-value
Age (years), mean $\pm$ SD	55.4 $\pm$ 10.6	62.1 $\pm$ 11.3	<0.001
Male sex, n (%)	55 (70.5)	57 (79.2)	0.21
Diabetes mellitus, n (%)	25 (32.1)	37 (51.4)	0.01
Hypertension, n (%)	39 (50.0)	47 (65.3)	0.06
STEMI presentation, n (%)	29 (37.2)	43 (59.7)	0.006
Troponin elevation, n (%)	62 (79.5)	66 (91.7)	0.04
Mean LVEF (%)	50.1 $\pm$ 8.7	43.2 $\pm$ 9.4	<0.001

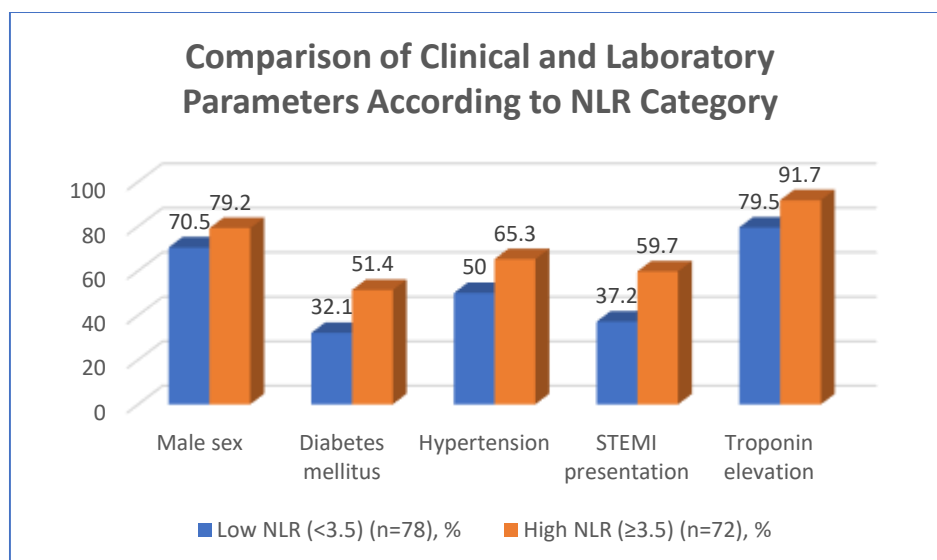
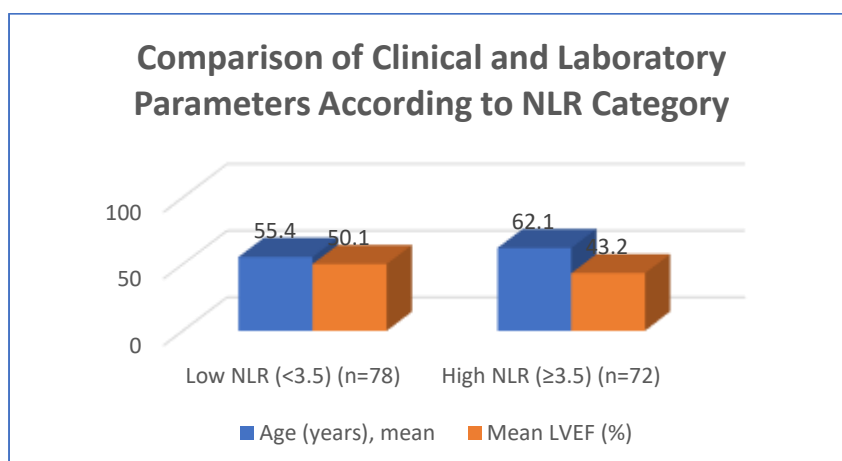


Figure 1 Comparison of Clinical and Laboratory Parameters According to NLR Category

Table 5. Association of NLR with In-Hospital Clinical Outcomes

Clinical Outcome	Low NLR (<3.5) (n=78)	High NLR (≥3.5) (n=72)	p-value
Major adverse cardiovascular events (MACE)	8 (10.3)	23 (31.9)	<0.001
Mortality	2 (2.6)	9 (12.5)	0.03
Heart failure	6 (7.7)	18 (25.0)	0.004
Cardiogenic shock	1 (1.3)	7 (9.7)	0.04
Recurrent MI	3 (3.8)	8 (11.1)	0.08

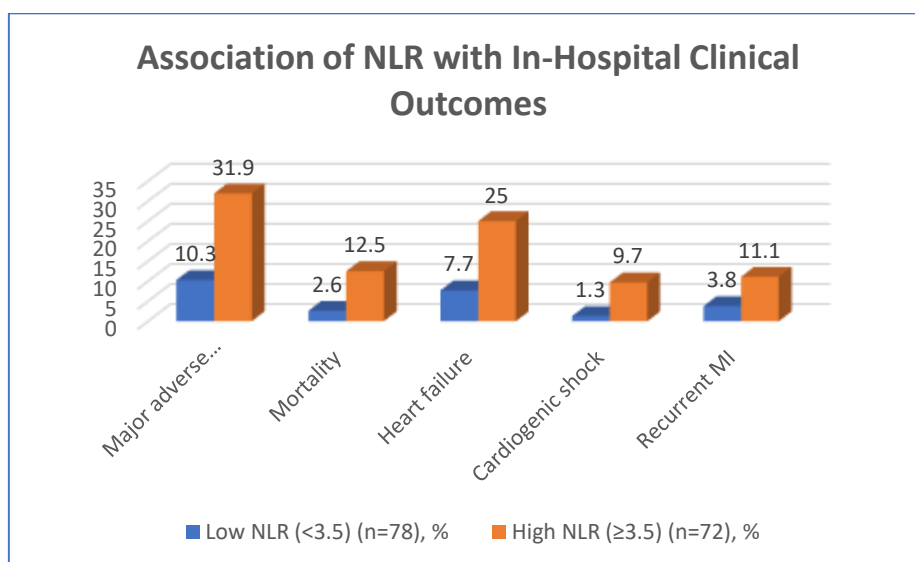


Figure 2 Association of NLR with In-Hospital Clinical Outcomes

Table 6. Multivariate Logistic Regression Analysis of Predictors of Adverse Outcomes (MACE)

Predictor	Adjusted OR	95% CI	p-value
Age ≥60 years	1.9	1.1–3.6	0.03
Diabetes mellitus	1.8	1.0–3.4	0.04
STEMI presentation	2.4	1.3–4.6	0.006
Triple vessel disease	3.2	1.7–6.1	<0.001
Reduced LVEF (<40%)	2.7	1.4–5.2	0.003
High NLR (≥3.5)	3.6	1.8–7.2	<0.001

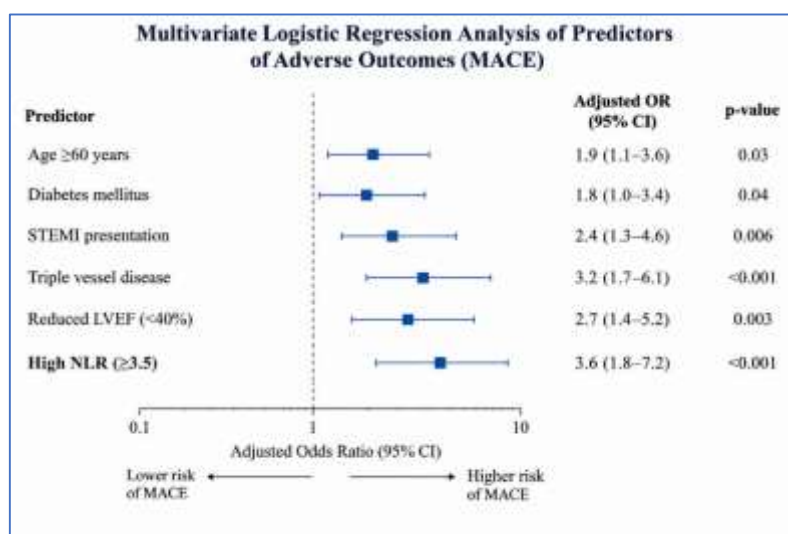


Figure 3 Multivariate Logistic Regression Analysis of Predictors of Adverse Outcomes (MACE)

DISCUSSION

Acute coronary syndrome (ACS) remains a major cause of cardiovascular morbidity and mortality worldwide. Inflammation plays a crucial role in atherosclerotic plaque formation, progression, and rupture. The neutrophil-to-lymphocyte ratio (NLR), derived from routine complete blood count parameters, has emerged as a simple marker reflecting systemic inflammation and cardiovascular risk[14]. The present study evaluated the association of NLR with coronary artery disease severity and in-hospital outcomes among 150 patients with ACS. In this study, the mean age of patients was 58.6 ± 11.4 years, with 52.0% aged ≥ 60 years, and males constituted 74.7% of the cohort. Hypertension (57.3%) was the most common risk factor, followed by dyslipidemia (47.3%) and diabetes mellitus (41.3%) (Table 1). Similar demographic patterns have been reported in previous ACS studies, reflecting the predominance of older males with multiple cardiovascular risk factors. STEMI was the most common ACS presentation (48.0%), followed by NSTEMI (37.3%) and unstable angina (14.7%) (Table 2). The mean NLR was 3.8 ± 1.9 with a median value of 3.4 (IQR: 2.2–4.8)[15]. Increased NLR in ACS reflects enhanced inflammatory activation, where neutrophils contribute to plaque instability through cytokine release and oxidative injury, while lymphopenia represents stress-related immune suppression. A significant association was observed between NLR and angiographic severity[16]. Patients with triple vessel disease had significantly higher NLR values (5.1 ± 2.0) compared with double vessel (3.7 ± 1.5) and single vessel disease (2.6 ± 1.1) ($p < 0.001$) (Table 3). Similar findings were reported by Previous study ., who demonstrated an association between elevated NLR and higher SYNTAX scores, suggesting that NLR reflects coronary disease complexity. Gibson et al. also reported that inflammatory markers were associated with increased coronary plaque burden. Patients with high NLR (≥ 3.5) showed significantly adverse clinical characteristics compared with those with low NLR (< 3.5). High NLR patients were older (62.1 ± 11.3 vs 55.4 ± 10.6 years; $p < 0.001$), had higher prevalence of diabetes (51.4% vs 32.1%; $p = 0.01$), more frequent STEMI presentation (59.7% vs 37.2%; $p = 0.006$), and lower LVEF ($43.2 \pm 9.4\%$ vs $50.1 \pm 8.7\%$; $p < 0.001$) (Table 4). These findings indicate that elevated NLR is associated with greater myocardial injury and impaired cardiac function. High NLR was also

significantly associated with adverse in-hospital outcomes. Patients with $\text{NLR} \geq 3.5$ had higher incidence of MACE (31.9% vs 10.3%; $p < 0.001$), mortality (12.5% vs 2.6%; $p = 0.03$), heart failure (25.0% vs 7.7%; $p = 0.004$), and cardiogenic shock (9.7% vs 1.3%; $p = 0.04$) (Table 5). Similar results were reported in previous studies., who demonstrated that elevated NLR predicted mortality and cardiovascular events following myocardial infarction[15,16]. Multivariate logistic regression showed that high NLR remained an independent predictor of MACE (adjusted OR 3.6; 95% CI: 1.8–7.2; $p < 0.001$) after adjustment for conventional risk factors. Other significant predictors included triple vessel disease, reduced LVEF, STEMI presentation, diabetes mellitus, and advanced age (Table 6). Previous studies similarly demonstrated the prognostic value of NLR in ACS patients. The clinical importance of NLR lies in its simplicity, low cost, and easy availability at admission[16,17]. Unlike complex risk prediction models, NLR can provide rapid assessment of inflammatory burden and help identify high-risk ACS patients, particularly in resource-limited settings.

CONCLUSION

The present study demonstrated that an elevated neutrophil-to-lymphocyte ratio (NLR) was significantly associated with greater coronary artery disease severity and adverse clinical outcomes among patients with acute coronary syndrome. Higher NLR values correlated with triple vessel disease, reduced left ventricular function, and increased incidence of major adverse cardiovascular events. $\text{NLR} \geq 3.5$ emerged as an independent predictor of in-hospital adverse outcomes. Being a simple, inexpensive, and routinely available marker, NLR may serve as a useful tool for early risk stratification and prognostic assessment in patients with ACS. Further large-scale studies are warranted to validate its clinical utility.

Limitations

The study was conducted at a single tertiary care centre with a relatively small sample size of 150 patients, which may limit the generalizability of the findings. The observational design restricted the ability to establish causal relationships between NLR and adverse outcomes. Long-term follow-up outcomes were not assessed, and only in-hospital clinical events were evaluated.

Additionally, NLR may be influenced by other unrecognized inflammatory conditions despite exclusion criteria.

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