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Frequency of Multivessel Coronary Artery Disease and Its Association with Neutrophil-to-Lymphocyte and Platelet-to-Lymphocyte Ratios in Patients with ST-Segment Elevation Myocardial Infarction.

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Abstract

Background: To determine the frequency of multivessel coronary artery disease and evaluate its association with neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) in patients presenting with ST-segment elevation myocardial infarction (STEMI).

Methodology: This prospective observational cross-sectional study was conducted at Liaquat National Hospital and Medicare Cardiac and General Hospital, Karachi, from September 2024 to March 2025. A total of 150 consecutive patients aged 20–80 years presenting with STEMI and undergoing primary percutaneous coronary intervention (PCI) were enrolled. Baseline demographic characteristics, clinical variables, laboratory parameters, and angiographic findings were recorded. NLR and PLR were calculated from admission complete blood counts. The anatomical complexity of coronary artery disease was assessed using the SYNTAX score, which was calculated independently by two blinded interventional cardiologists and categorized into low (<22), intermediate (22–33), and high (>33) groups.

Results: Multivessel disease was highly prevalent, occurring in 91% of patients, with a median SYNTAX score of 27 (IQR 23–34). NLR and PLR did not differ significantly between patients with single-vessel and multivessel disease (p = 0.868 and p = 0.956, respectively). ROC curve analysis demonstrated poor discriminatory ability of NLR and PLR in predicting high SYNTAX scores, with areas under the curve of 0.542 and 0.533, respectively. In multivariable logistic regression analysis, increasing age (adjusted OR 1.06, 95% CI 1.01–1.11), elevated serum creatinine (adjusted OR 1.89, 95% CI 1.21–2.95), and higher troponin-I levels (adjusted OR 1.14, 95% CI 1.05–1.23) were independently associated with multivessel coronary artery disease.

Conclusion: NLR and PLR were not reliable predictors of multivessel coronary artery disease or high SYNTAX score in patients with STEMI. Conventional clinical and biochemical parameters, including age, serum creatinine, and troponin-I, demonstrated stronger predictive value.

Keywords

STEMI, Multivessel Disease, Neutrophil-to-Lymphocyte Ratio, Platelet-to-Lymphocyte Ratio, SYNTAX Score.

Introduction

Cardiovascular disease remains the leading cause of mortality worldwide despite substantial advances in preventive strategies, pharmacological therapies, and interventional cardiology. Among the spectrum of cardiovascular disorders, ischemic heart disease (IHD) accounts for the largest proportion of cardiovascular deaths and continues to represent a major global health burden[1].

Atherosclerosis, the underlying pathological process in IHD, is now widely recognized as a chronic immune-inflammatory disease rather than simply a disorder of lipid accumulation within the arterial wall. Increasing evidence indicates that inflammatory mechanisms play a central role in the initiation, progression, and destabilization of atherosclerotic plaques[2]. Experimental and clinical studies have demonstrated that activation of immune cells, particularly myeloid cells, contributes to endothelial dysfunction, plaque formation, and subsequent plaque rupture, ultimately leading to acute coronary events[3].

Chronic low-grade inflammation promotes a pro-atherogenic environment that interacts with traditional cardiovascular risk factors such as diabetes mellitus, metabolic syndrome, dyslipidemia, and endothelial dysfunction. These factors collectively accelerate vascular injury and increase susceptibility to thrombotic complications[4]. Acute coronary syndrome (ACS), including STEMI, represents the most severe clinical manifestation of ischemic heart disease and remains a major contributor to cardiovascular morbidity and mortality worldwide[5].

Although timely reperfusion therapy with primary PCI has substantially improved outcomes in STEMI patients, a considerable proportion of these individuals exhibit multivessel coronary artery disease at the time of presentation, indicating widespread atherosclerotic involvement of the coronary circulation[6]. Contemporary studies suggest that multivessel disease may occur in up to 50% of patients presenting with STEMI, depending on demographic characteristics and associated comorbidities[7]. The presence of multivessel coronary artery disease is clinically significant because it is associated with more complex revascularization procedures, higher risk of adverse cardiovascular events, and increased long-term mortality[8].

Inflammation occurring during acute myocardial infarction has been consistently associated with poor clinical outcomes. Elevated leukocyte counts and systemic inflammatory activation are linked with impaired coronary microvascular perfusion, no-reflow phenomena, cardiogenic shock, heart failure, and increased mortality[9]. In this context, simple hematological markers derived from routine blood tests have gained increasing attention as potential indicators of systemic inflammation and cardiovascular risk.

The NLR, calculated from peripheral blood counts, has emerged as a readily available marker of inflammatory response. Elevated NLR values have been associated with increased mortality in STEMI patients, as well as with complications such as ventricular arrhythmias, stent thrombosis, and impaired myocardial recovery after reperfusion therapy. Furthermore, several studies have reported a positive association between higher NLR levels and greater angiographic severity of coronary artery disease, including higher SYNTAX scores[10].

Similarly, the PLR has been proposed as another composite inflammatory marker reflecting both platelet activation and immune system dysregulation. Elevated PLR levels have been linked with adverse cardiovascular outcomes in patients with acute coronary syndromes, including increased risk of mortality, recurrent myocardial infarction, stroke, and heart failure. Some studies also suggest that PLR may serve as a predictor of long-term clinical outcomes following coronary revascularization procedures[11].

Despite growing interest in inflammatory biomarkers, the relationship between admission levels of NLR and PLR and the angiographic severity of coronary artery disease, particularly the presence of complex multivessel disease in STEMI patients, remains incompletely understood. Identifying reliable and easily accessible biomarkers capable of predicting coronary disease burden at the time of presentation

could facilitate early risk stratification and assist clinicians in tailoring therapeutic strategies.

Therefore, the present study aimed to determine the frequency of multivessel coronary artery disease and evaluate the association between admission neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio with the severity of coronary artery disease as assessed by the SYNTAX score in patients presenting with ST-segment elevation myocardial infarction.

Methodology

Study Design

This investigation was designed as a prospective observational cross-sectional study aimed at determining the frequency of multivessel coronary artery disease and evaluating its association with inflammatory biomarkers, specifically the NLR and PLR, among patients presenting with STEMI. The study was conducted over a seven-month period from September 2024 to March 2025. All eligible patients presenting during this period and undergoing primary PCI were consecutively enrolled to minimize selection bias and ensure representation of routine clinical practice.

Ethics

Ethical approval for the study was obtained from the institutional ethics review board of the participating hospitals prior to the initiation of data collection. All procedures were conducted in accordance with the ethical standards of the institutional research committee and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment in the study. Patient confidentiality was strictly maintained throughout the research process by anonymizing all clinical and laboratory data used for analysis.

Setting

The study was carried out at two tertiary care cardiac centers in Karachi, Pakistan, Liaquat National Hospital and Medicare Cardiac and General Hospital. These institutions provide specialized cardiovascular care and receive a large number of patients presenting with acute coronary syndromes, including STEMI, thereby providing an appropriate clinical environment for studying angiographic disease severity and associated inflammatory biomarkers.

Participants

A total of 150 consecutive adult patients aged between 20 and 80 years presenting with ST-segment elevation myocardial infarction were included in the study. STEMI was diagnosed based on the 2015 American Heart Association criteria, defined as ST-segment elevation greater than 1 mm in at least two contiguous electrocardiographic leads accompanied by typical ischemic chest pain lasting more than 20 minutes. Patients undergoing primary PCI for STEMI were eligible for inclusion. Patients with a history of cardiomyopathy, chronic kidney disease, significant valvular heart disease, chronic inflammatory disorders, active infection, or hematological diseases were excluded to avoid confounding effects on inflammatory markers such as NLR and PLR. Patients with prior myocardial infarction were eligible for inclusion provided that they fulfilled other study criteria.

Variables

The primary outcome variable was the presence of multivessel coronary artery disease (MVD). MVD was defined as the presence of ≥50% stenosis in two or more major epicardial coronary arteries identified during diagnostic coronary angiography. The severity and complexity of coronary artery disease were additionally quantified using the SYNTAX score. The principal exposure variables included inflammatory biomarkers derived from admission blood tests, namely the NLR and PLR. Secondary clinical and biochemical variables assessed included age, sex, body mass index, comorbidities (hypertension, diabetes mellitus, dyslipidemia), troponin-I levels, and serum creatinine.

Data Sources / Measurement

Baseline clinical characteristics, laboratory findings, and angiographic data were collected using standardized data collection forms at the time of hospital admission. Peripheral blood samples were obtained prior to coronary angiography and analyzed for complete blood counts using standard laboratory procedures. From these measurements,

the neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio were calculated.

Diagnostic coronary angiography was performed for all participants as part of standard clinical management. The extent of coronary artery involvement was categorized into single-vessel, two-vessel, or three-vessel disease based on the number of significantly stenosed vessels. The anatomical complexity of coronary artery disease was quantified using the SYNTAX score, calculated through the online SYNTAX calculator based on the American Heart Association 16-segment coronary model. This scoring system incorporates lesion location, severity, and morphological complexity for lesions demonstrating greater than 50% luminal narrowing in vessels with a diameter of at least 1.5 mm. To reduce assessment bias, two experienced interventional cardiologists independently evaluated the angiograms while blinded to clinical and laboratory information.

Bias

Several methodological strategies were implemented to minimize potential sources of bias. Consecutive patient enrollment was adopted to reduce selection bias. Blinding of the interventional cardiologists performing SYNTAX score assessment minimized measurement bias during angiographic evaluation. Additionally, laboratory parameters were obtained using standardized automated techniques. Data entry was conducted using a double-entry method to reduce transcription errors and ensure data accuracy.

Study Size

The required sample size was calculated using the Wan Nor Arifin single-proportion sample size calculator. The calculation assumed a prevalence of multivessel coronary artery disease of approximately 54% among patients presenting with STEMI, with a desired margin of error of 8%. Based on these parameters, a minimum sample size of 150 participants was required. The primary aim of the sample size estimation was to determine the prevalence of multivessel disease within the study population. Multivariable regression and receiver operating characteristic (ROC) analyses were subsequently performed as exploratory analyses to identify potential predictors of multivessel disease[12].

Quantitative Variables

Continuous variables including age, body mass index, NLR, PLR, troponin-I levels, creatinine levels, and SYNTAX score were analyzed either as continuous measures or categorized into clinically meaningful groups where appropriate. For example, SYNTAX scores were categorized into low (<22), intermediate (22–33), and high (>33) groups to reflect increasing angiographic complexity. Similarly, laboratory variables such as troponin-I and creatinine were stratified based on clinically relevant thresholds.

Statistical Methods

Data analysis was performed using Statistical Package for Social Sciences (SPSS) version 27. Continuous variables were summarized as mean ± standard deviation (SD) for normally distributed data or as median with interquartile range (IQR) for non-normally distributed variables. Categorical variables were expressed as frequencies and percentages.

The Shapiro–Wilk test was applied to assess the normality of continuous variables. For group comparisons, the Mann–Whitney U test or Kruskal–Wallis test was used for non-parametric continuous variables, while the chi-square test or Fisher's exact test was applied for categorical variables.

To determine independent predictors of multivessel disease, multivariable logistic regression analysis was conducted. Variables initially considered for the model included age, sex, diabetes mellitus, hypertension, serum creatinine, troponin-I, NLR, and PLR. Due to the limited number of patients without multivessel disease, a parsimonious regression model was constructed including age, serum creatinine, and troponin-I to maintain model stability.

Model calibration was evaluated using the Hosmer–Lemeshow goodness-of-fit test, while model discrimination was assessed through receiver operating characteristic (ROC) curve analysis. A p-value less than 0.05 was considered statistically significant.

Results

Participants

A total of 150 patients presenting with ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (PCI) were included in the final analysis. All patients fulfilled the eligibility criteria and were enrolled consecutively during the study period. The study population comprised predominantly male patients, accounting for 66% (n = 99), while females represented 34% (n = 51). The median age of the participants was 64 years (interquartile range [IQR] 55–70 years). Most patients were older than 50 years (86.7%), reflecting the typical age distribution of STEMI presentations in clinical practice. The prevalence of cardiovascular risk factors was substantial in this cohort, with hypertension present in 80% of patients and diabetes mellitus in 67.3%. Additional baseline characteristics including body mass index, lipid abnormalities, family history of coronary artery disease, and tobacco use are presented in Table 1.

Descriptive Data

Baseline laboratory and angiographic findings of the study population are summarized in Table 1. The median neutrophil-to-lymphocyte ratio (NLR) was 5.0 (IQR 2.75–9.25), while the median platelet-to-lymphocyte ratio (PLR) was 113.5 (IQR 81.5–198.0). Median serum creatinine was 1.06 mg/dL (IQR 0.86–1.38), and the median troponin-I concentration was 1.94 ng/mL (IQR 0.80–7.33). Angiographic assessment revealed a median SYNTAX score of 27 (IQR 23–34), indicating moderate anatomical complexity of coronary artery disease. Based on SYNTAX score categorization, 23.3% of patients had low scores (<22), 50% had intermediate scores (22–33), and 26.7% had high scores (>33). Overall, multivessel coronary artery disease was highly prevalent in the study population, affecting 90.7% of patients (n = 136). These findings indicate that the majority of STEMI patients in this cohort had extensive coronary artery involvement. Detailed demographic, clinical, and laboratory characteristics are presented in Table 1.

Outcome Data

The association between baseline clinical characteristics and the presence of multivessel coronary artery disease is presented in Table 2. Patients with multivessel disease tended to be older, with a median age of 65 years compared with 59 years in those without multivessel disease, although this difference did not reach statistical significance ($p = 0.078$). Similarly, other demographic and clinical variables including gender distribution, body mass index, diabetes mellitus, hypertension, tobacco use, and dyslipidemia did not differ significantly between the multivessel and single-vessel disease groups. Laboratory parameters including NLR and PLR also showed no significant differences between the groups ($p = 0.868$ and $p = 0.956$, respectively).

However, the SYNTAX score demonstrated a strong association with multivessel disease. Patients with multivessel disease had significantly higher SYNTAX scores compared with those with single-vessel disease ($p < 0.001$). These findings suggest that angiographic severity of coronary artery disease is strongly linked with the presence of multivessel involvement. Detailed comparisons are shown in Table 2.

Further analysis examined the relationship between baseline characteristics and SYNTAX score categories. As presented in Table 3, increasing age was significantly associated with higher SYNTAX scores ($p = 0.042$). Patients with high SYNTAX scores (>33) had a median age of 66 years compared with 58 years among those with low SYNTAX scores (<22). Other clinical variables including gender, obesity, hypertension, diabetes mellitus, and tobacco use did not demonstrate statistically significant differences across SYNTAX score categories. Likewise, inflammatory biomarkers NLR and PLR did not differ significantly between low, intermediate, and high SYNTAX score groups ($p = 0.942$ and $p = 0.497$, respectively). These results suggest that systemic inflammatory ratios measured at admission may not reflect the anatomical complexity of coronary artery disease in STEMI patients. Full comparisons are shown in Table 3.

Main Results

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of NLR and PLR in identifying patients with high SYNTAX scores (>33). As illustrated in Figure 1 and summarized in Table 4, both biomarkers demonstrated poor discriminatory ability.

For NLR, the area under the ROC curve (AUC) was 0.542 (95% CI: 0.440–0.643), indicating minimal predictive accuracy. The optimal cut-off value identified was 9.5, which yielded a sensitivity of 35% and specificity of 79.1%. For PLR, the AUC was similarly low at 0.533 (95% CI: 0.428–0.637). The optimal PLR cut-off value of 95.85 produced a sensitivity of 75% and specificity of 40.9%. These results demonstrate that both NLR and PLR had limited diagnostic utility in predicting severe coronary anatomical complexity among STEMI patients. Detailed diagnostic performance metrics are presented in Table 4, and ROC curves are shown in Figure 1.

Multivariable logistic regression analysis was subsequently conducted to identify independent predictors of multivessel coronary artery disease. Due to the high prevalence of multivessel disease and the limited number of patients without it, a parsimonious model including age, serum creatinine, and troponin-I was constructed.

As shown in Table 5, increasing age was independently associated with multivessel disease (adjusted OR 1.06, 95% CI 1.01–1.11, p = 0.018). Elevated serum creatinine levels also demonstrated a significant association (adjusted OR 1.89, 95% CI 1.21–2.95, p = 0.005), as did higher troponin-I concentrations (adjusted OR 1.14, 95% CI 1.05–1.23, p = 0.002).

The regression model demonstrated good calibration with a Hosmer–Lemeshow p value of 0.64 and acceptable discriminatory performance with an AUC of 0.78 (95% CI 0.71–0.85). Variance inflation factor (VIF) values for all variables were below 2, indicating no evidence of multicollinearity. Overall, these findings suggest that traditional clinical and biochemical parameters rather than inflammatory ratios are more reliable predictors of multivessel coronary artery disease in STEMI patients. Detailed regression results and model performance indicators are presented in Table 5.

Table 1: Baseline Clinical and Demographic Characteristics of the Study Population (n=150).

Variables		**N (%)**
Gender	Male	99 (66)
	Female	51 (34)
Age (years); Median (IQR)		64 (55-70)
Age Groups	≤50 years	20 (13.3)
	>50 years	130 (86.7)
BMI (kg/m²); Median (IQR)		28.04 (25.96-30.05)
Obesity		40 (26.7)
Dominance	Left	20 (13.3)
	Right	130 (86.7)
Comorbidities	Dyslipidaemia	27 (18)
	Hypertension	120 (80)
	Family History of Premature CAD	8 (5.3)
	Prior MI	14 (9.3)
	Diabetes mellitus	101 (67.3)
	Tobacco use	35 (23.3)
	Heart Failure	9 (6)

	Cardiogenic shock at start of PCI	7 (4.7)
Troponin-I (ng/mL); Median (IQR)		1.94 (0.80-7.33)
Trop-I groups	≤10 ng/mL	123 (82)
	>10 ng/mL	27 (18)
Creatinine (mg/dL); Median (IQR)		1.06 (0.86-1.38)
Creatinine groups	<1.3 mg/dL	106 (70.7)
	≥1.3 mg/dL	44 (29.3)
NLR; Median (IQR)		5.00 (2.75-9.25)
NLR Groups	<2	11 (7.3)
	2-3	48 (32)
	>3	91 (60.7)
PLR; Median (IQR)		113.50 (81.50-198.01)
PLR Groups	≤100	64 (42.7)
	101-171	38 (25.3)
	>171	48 (32)
SYNTAX score; Median (IQR)		27.00 (23.00-34.00)
SYNTAX score categories	<22	35 (23.3)
	22-33	75 (50)
	>33	40 (26.7)
Multivessel disease		136 (90.7)

Table 2: Association and comparison of multi-vessel disease.

Variables		**Multi-vessel disease [N(%)]**		**p-value**
		Yes	**No**	
Gender	Male	93(68.4)	6(42.9)	0.075
	Female	43(31.6)	8(57.1)	
Age (years); Median (IQR)		65.00(56.00-70.00)	59.00(49.75-65.00)	0.078
Age Groups	≤50 years	16(11.8)	4(28.6)	0.095
	>50 years	120(88.2)	10(71.4)	
BMI (kg/m²); Median (IQR)		28.21(26.08-30.18)	27.19(25.89-29.26)	0.456
Obesity		38(27.9)	2(14.3)	0.355
Dominance	Left	20(14.7)	0(0)	0.217
	Right	116(85.3)	14(100)	
Comorbidities	Dyslipidaemia	22(16.2)	5(35.7)	0.134
	Hypertension	109(80.1)	11(78.6)	1.000
	Family History of Premature CAD	8(5.9)	0(0)	1.000
	Prior MI	13(9.6)	1(7.1)	1.000
	Diabetes mellitus	92(67.6)	9(64.3)	0.773
	Tobacco use	32(23.5)	3(21.4)	1.000
	Heart Failure	9(6.6)	0(0)	1.000
	Cardiogenic shock at start of PCI	6(4.4)	1(7.1)	0.504
Troponin-I (ng/mL); Median (IQR)		1.99(0.82-8.45)	1.26(0.66-2.03)	0.080

Trop-I groups	≤10 ng/mL	109(80.1)	14(100)	0.075
	>10 ng/mL	27(19.9)	0(0)	
Creatinine (mg/dL); Median (IQR)		1.07(0.86-1.40)	0.92(0.78-1.22)	0.093
Creatinine groups	<1.3 mg/dL	94(69.1)	12(85.7)	0.235
	≥1.3 mg/dL	42(30.9)	2(14.3)	
NLR; Median (IQR)		5.00(2.25-9.75)	5.50(2.75-10.00)	0.868
NLR Groups	<2	10(7.4)	1(7.1)	0.672
	2-3	45(33.1)	3(21.4)	
	>3	81(59.6)	10(71.4)	
PLR; Median (IQR)		112.12(82.15-198.00)	115.00(69.92-228.27)	0.956
PLR Group	≤100	58(42.6)	6(42.9)	0.561
	101-171	36(26.5)	2(14.3)	
	>171	42(30.9)	6(42.9)	
SYNTAX score; Median (IQR)		28.00(24.00-34.00)	18.00(17.00-19.00)	<0.001*
SYNTAX score group	<22	21(15.4)	14(100)	<0.001*
	22-33	75(55.1)	0(0)	
	>33	40(29.4)	0(0)	

Continuous variables are presented as median (interquartile range) and categorical variables are presented as n (%).
Mann–Whitney U test was used for continuous variables.
Chi-square or Fisher's exact test was used for categorical variables.
*p < 0.05 was considered statistically significant.

Table 3: Association of Baseline Characteristics with SYNTAX Score (n = 150).

Variables		**Syntax score**			**p-value**
		<22 (n=35)	**22–33 (n=75)**	**>33 (n=40)**	
Gender	Male	20 (57.1)	49 (65.3)	30 (75.0)	0.262
	Female	15 (42.9)	26 (34.7)	10 (25.0)	
Age (years); Median (IQR)		58.00 (50.00–66.00)	64.00 (57.00–70.00)	66.00 (60.00–72.00)	0.042*
Age Groups	≤50 years	7 (20.0)	9 (12.0)	4 (10.0)	0.442
	>50 years	28 (80.0)	66 (88.0)	36 (90.0)	
BMI (kg/m²); Median (IQR)		27.80 (25.40–29.90)	28.30 (26.10–30.50)	27.60 (25.80–29.70)	0.755
Obesity		6 (17.1)	22 (29.3)	12 (30.0)	0.346
Dominance	Left	2 (5.7)	11 (14.7)	7 (17.5)	0.277
	Right	33 (94.3)	64 (85.3)	33 (82.5)	
Comorbidities	Dyslipidaemia	5 (14.3)	13 (17.3)	9 (22.5)	0.638
	Hypertension	27 (77.1)	62 (82.7)	31 (77.5)	0.716
	Family History of Premature CAD	2 (5.7)	3 (4.0)	3 (7.5)	0.645
	Prior MI	1 (2.9)	9 (12.0)	4 (10.0)	0.327
	Diabetes mellitus	22 (62.9)	50 (66.7)	29 (72.5)	0.664
	Tobacco use	8 (22.9)	18 (24.0)	9 (22.5)	0.961
	Heart Failure	2 (5.7)	2 (5.7)	5 (12.5)	0.103
	Lesion Vessel	13 (37.1)	39 (52.0)	17 (42.5)	0.303

	Cardiogenic shock at start of PCI	2 (5.7)	2 (5.7)	3 (7.5)	0.461
Troponin-I (ng/mL); Median (IQR)		1.60 (0.75–4.50)	1.95 (0.85–7.10)	2.40 (0.90–8.90)	0.584
Trop-I groups	≤10 ng/mL	29 (82.9)	62 (82.7)	32 (80.0)	0.928
	>10 ng/mL	6 (17.1)	13 (17.3)	8 (20.0)	
Creatinine (mg/dL); Median (IQR)		0.95 (0.80–1.20)	1.10 (0.88–1.45)	1.20 (0.90–1.50)	0.291
Creatinine groups	<1.3 mg/dL	28 (80.0)	52 (69.3)	26 (65.0)	0.340
	≥1.3 mg/dL	7 (20.0)	23 (30.7)	14 (35.0)	
NLR; Median (IQR)		4.50 (2.50–8.75)	5.25 (2.75–9.50)	5.75 (3.00–10.25)	0.942
NLR Groups	<2	3 (8.6)	7 (9.3)	1 (2.5)	0.686
	2–3	10 (28.6)	23 (30.7)	15 (37.5)	
	>3	22 (62.9)	45 (60.0)	24 (60.0)	
PLR; Median (IQR)		100.50 (78.00–170.00)	118.00 (85.00–205.00)	125.00 (88.00–210.00)	0.497
PLR Groups	≤100	16 (45.7)	34 (45.3)	14 (35.0)	0.697
	101–171	9 (25.7)	16 (21.3)	13 (32.5)	
	>171	10 (28.6)	25 (33.3)	13 (32.5)	
SYNTAX score; Median (IQR)		18.00 (16.00–20.00)	27.00 (25.00–30.00)	38.00 (35.00–41.00)	<0.001*

Continuous variables are presented as median (interquartile range) and categorical variables are presented as n (%).
Kruskal–Wallis test was used for continuous variables.
Chi-square or Fisher's exact test was used for categorical variables.
*p < 0.05 was considered statistically significant.

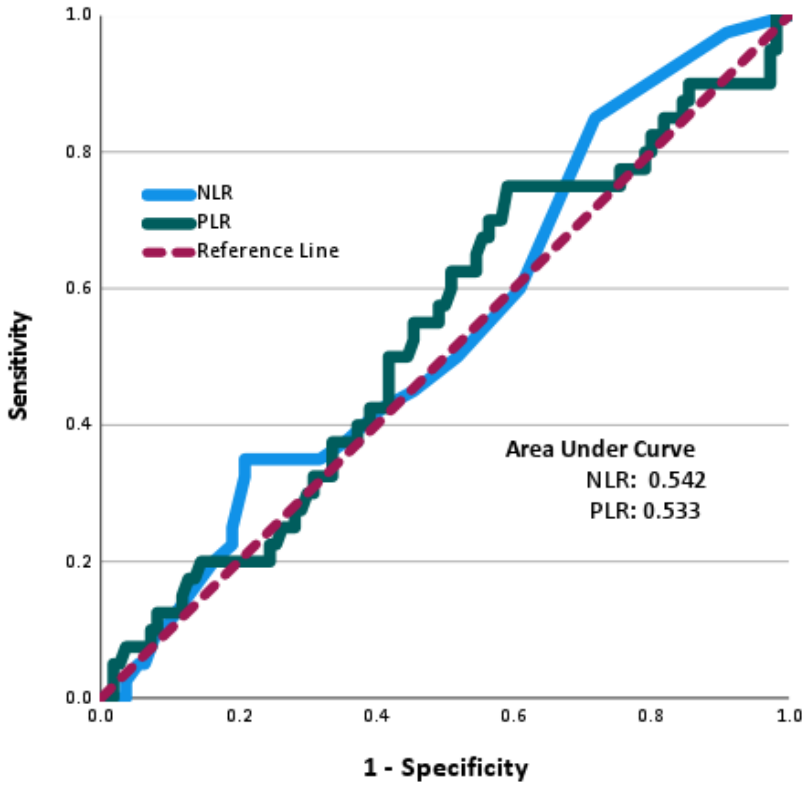

Figure 1: ROC curves for PLR and NLR in identifying syntax values above 33.

Table 4: Diagnostic Performance of NLR and PLR for Identifying High SYNTAX Score (>33).

Values		Sensitivity	Specificity	Youden Index
NLR	9.5	35%	79.1%	0.141
	2.5	85%	28.2%	0.132
	10.5	32.5%	79.1%	0.116
PLR	95.85	75%	40.9%	0.159
	95.35	75%	40%	0.15
	94.5	75%	39.1%	0.141
	97.295	70%	43.6%	0.136
	93.8	75%	38.2%	0.132

Table 5: Multivariable Logistic Regression Analysis of Independent Predictors of Multi-vessel Coronary Artery Disease.

Variables	Adjusted OR	95% CI	p-value
Age (years)	1.06	1.01–1.11	0.018
Serum creatinine	1.89	1.21–2.95	0.005
Troponin-I	1.14	1.05–1.23	0.002

Hosmer–Lemeshow goodness-of-fit test: p = 0.64 (indicating good calibration). Area under the ROC curve (AUC): 0.78 (95% CI: 0.71–0.85), showing acceptable discriminatory ability. Variance inflation factors (VIF) for all variables <2, indicating no multicollinearity

Logistic regression included age, serum creatinine, troponin-I.

Statistical significance was set at $p < 0.05$.

Discussion

In this study of patients presenting with STEMI undergoing primary PCI, we investigated the relationship between systemic inflammatory biomarkers NLR and PLR and the severity of coronary artery disease as assessed by the SYNTAX score and the presence of multivessel disease. The principal findings of this study demonstrate that although multivessel coronary artery disease was highly prevalent in the study population, neither NLR nor PLR showed a significant association with multivessel disease or higher SYNTAX scores. In contrast, conventional clinical and biochemical parameters, including age, serum creatinine, and troponin-I levels, were independently associated with the presence of multivessel coronary artery disease.

The high prevalence of multivessel disease observed in our study population is consistent with previously reported findings in STEMI cohorts, where a substantial proportion of patients demonstrate widespread coronary atherosclerosis at the time of presentation. Multivessel involvement reflects the systemic and progressive nature of atherosclerosis and is associated with more complex coronary anatomy, increased procedural challenges during revascularization, and poorer long-term cardiovascular outcomes[13,14]. These findings emphasize the importance of identifying reliable predictors of disease burden that may assist in early risk stratification and treatment planning.

Inflammation plays a central role in the development and progression of atherosclerosis and contributes to plaque instability and thrombosis during acute coronary syndromes. Elevated leukocyte counts and systemic inflammatory activation have been associated with adverse clinical outcomes, including impaired myocardial perfusion, microvascular obstruction, heart failure, and increased mortality following myocardial infarction[15]. Consequently, hematological inflammatory indices such as NLR and PLR have attracted attention as potential

biomarkers reflecting both inflammatory and thrombotic processes in cardiovascular disease.

Several previous studies have reported a positive association between elevated NLR or PLR levels and greater angiographic severity of coronary artery disease, including higher SYNTAX scores and increased plaque burden[16]. These findings suggest that systemic inflammatory status may reflect the underlying atherosclerotic disease burden and contribute to the progression of coronary artery disease. However, in the present study, neither NLR nor PLR demonstrated a statistically significant relationship with multivessel disease or SYNTAX score categories.

The lack of association observed in our study may be explained by several factors. First, STEMI itself represents an acute inflammatory event characterized by rapid activation of immune pathways and significant systemic inflammatory responses. In this context, inflammatory biomarkers measured at a single time point during the acute phase may primarily reflect the inflammatory response to myocardial injury rather than the chronic inflammatory processes underlying atherosclerotic plaque development[17]. This acute inflammatory surge may therefore reduce the discriminatory capacity of markers such as NLR and PLR in identifying differences in underlying coronary anatomical complexity.

Second, the pathophysiological mechanisms contributing to elevated inflammatory ratios are complex and influenced by multiple factors. Neutrophils contribute to plaque destabilization through oxidative stress, proteolytic enzyme release, and inflammatory signaling pathways, while lymphopenia may reflect neurohormonal stress responses and immune dysregulation during acute cardiovascular events[18]. Platelets further contribute to thrombo-inflammatory responses through platelet activation, aggregation, and interaction with leukocytes and endothelial cells[19]. Although these mechanisms support a theoretical link between inflammatory ratios and coronary disease severity, the dynamic nature of inflammatory responses during acute myocardial infarction may limit their reliability as predictors of angiographic complexity.

In contrast, our findings demonstrated that traditional clinical and biochemical variables, particularly age, serum creatinine, and troponin-I levels, were independently associated with multivessel coronary artery disease. Increasing age is widely recognized as a marker of cumulative exposure to cardiovascular risk factors and progressive atherosclerotic burden. Similarly, impaired renal function, reflected by elevated serum creatinine levels, has been associated with endothelial dysfunction, vascular calcification, and accelerated atherosclerosis[20]. Elevated troponin-I concentrations may also indicate greater myocardial injury and more extensive ischemic territory, which may indirectly reflect more widespread coronary artery involvement.

Taken together, the results of this study suggest that while NLR and PLR remain inexpensive and easily obtainable inflammatory markers, their ability to predict the anatomical severity of coronary artery disease in the setting of acute STEMI may be limited. Conventional clinical parameters and biochemical markers appear to provide more reliable information for assessing disease burden in this population. Future investigations incorporating multimarker inflammatory profiles and longitudinal biomarker assessment may provide further insights into the complex relationship between inflammation and coronary artery disease severity.

Limitations

Several limitations of this study should be acknowledged. First, the observational cross-sectional design limits the ability to establish causal relationships between inflammatory biomarkers and the severity of coronary artery disease. Second, the study was conducted in a relatively moderate sample size from two centers, which may limit the generalizability of the findings to broader populations. Third, the distribution of patients with multivessel disease and single-vessel disease was highly imbalanced, with a predominance of multivessel involvement in the study cohort. This

imbalance resulted in a relatively low number of events per variable in the logistic regression analysis, which may have affected the stability of the regression model and increased the possibility of overfitting. Therefore, the results of the regression analysis should be interpreted cautiously and considered exploratory. Fourth, inflammatory biomarkers were measured only at a single time point at admission. Serial measurements of inflammatory markers might provide more accurate insights into the dynamic inflammatory responses occurring during acute myocardial infarction. Additionally, other inflammatory biomarkers such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6, or systemic immune-inflammatory index were not assessed in this study and may provide additional prognostic information. Finally, residual confounding cannot be completely excluded, as certain clinical factors such as prior medication use, baseline inflammatory status, and lifestyle factors were not comprehensively evaluated. Future studies incorporating larger multicenter cohorts and broader biomarker panels may help clarify the role of inflammatory indices in assessing coronary disease severity.

Conclusion

This study demonstrated a high prevalence of multivessel coronary artery disease among patients presenting with ST-segment elevation myocardial infarction undergoing primary PCI. However, commonly used inflammatory biomarkers such as neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio were not significantly associated with multivessel disease or higher SYNTAX scores in this population. In contrast, traditional clinical and biochemical variables including age, serum creatinine, and troponin-I emerged as independent predictors of multivessel coronary artery disease. These findings suggest that conventional clinical parameters may remain more reliable indicators of coronary disease burden in the acute STEMI setting. Further large-scale multicenter studies incorporating longitudinal inflammatory marker assessment and multimarker risk models are needed to better define the potential role of inflammatory biomarkers in predicting coronary artery disease severity and guiding risk stratification strategies in patients with acute myocardial infarction.

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