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Platelet-To-Lymphocyte Ratio Across Acute Coronary Syndrome Subtypes: Association and Discriminative Performance in Myocardial Infarction

Platelet-to-lymphocyte ratio across ACS subtypes

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ABSTRACT:

Background: The platelet-to-lymphocyte ratio (PLR) is an inexpensive haematological index that reflects both thrombotic and inflammatory activity, yet whether it differs across the clinical spectrum of acute coronary syndrome (ACS) remains uncertain.

Methods: This single-centre retrospective cross-sectional study included 235 adults admitted with ACS to a tertiary referral hospital in Makassar, Indonesia. PLR was calculated from the first complete blood count obtained on arrival. Differences among unstable angina pectoris (UAP), non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI) were assessed with the Kruskal-Wallis test and Dunn-Bonferroni post hoc comparisons, multivariable binary and multinomial logistic regression, and receiver operating characteristic analysis.

Results: Mean age was 58.49 ± 10.75 years and 77.0% of participants were male; STEMI accounted for 53.2% of cases, while UAP and NSTEMI each accounted for 23.4%. Median PLR differed significantly across subtypes (UAP 130.5, NSTEMI 170.0 and STEMI 162.9; $p = 0.002$), being higher in both infarction phenotypes than in UAP but not differing between NSTEMI and STEMI. Each 50-unit increase in PLR was independently associated with myocardial infarction rather than UAP (adjusted odds ratio 1.40, 95% confidence interval 1.12-1.76; $p = 0.004$), with adjusted odds ratios of 1.35 for NSTEMI versus UAP and 1.43 for STEMI versus UAP. Discrimination of myocardial

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infarction from UAP was modest (area under the curve 0.655, 95% confidence interval 0.574-0.733).

Conclusions: PLR is higher in myocardial infarction than in UAP but does not distinguish NSTEMI from STEMI, supporting its use as a low-cost adjunctive haematological index rather than a stand-alone diagnostic discriminator.

1. INTRODUCTION

Acute coronary syndrome (ACS) encompasses unstable angina pectoris (UAP), non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI), a clinical spectrum unified by acute myocardial ischaemia but differentiated by electrocardiographic findings, biomarker evidence of myocardial necrosis, coronary occlusion patterns and urgency of reperfusion. Contemporary guidelines emphasise early diagnosis and risk stratification because these distinctions directly affect invasive strategy, antithrombotic treatment and prognosis (Byrne et al., 2023; Thygesen et al., 2018; Bergmark et al., 2022).

The biological substrate of ACS is not limited to fixed coronary stenosis. Plaque rupture or erosion, platelet adhesion and aggregation, thrombus formation, and a coordinated innate and adaptive inflammatory response contribute to acute coronary instability (Ajoolabady et al., 2024; Stakos et al., 2012; Saigusa et al., 2020; Kounis et al., 2021). Platelets are central effectors of coronary thrombosis, whereas lymphocyte counts may fall during acute physiologic stress and systemic inflammation. A ratio incorporating both cell populations may therefore capture complementary thrombotic and inflammatory signals.

The platelet-to-lymphocyte ratio (PLR), calculated from routine complete blood count parameters, is inexpensive, rapidly available and requires no additional assay. Higher PLR has been associated with adverse cardiovascular outcomes, including mortality and subsequent symptomatic heart failure in ACS, and systematic reviews have supported its prognostic potential (Li et al., 2017; Oylumlu et al., 2020; Pruc et al., 2023; Intan et al., 2022). However, the ability of PLR to reflect differences across the clinical ACS spectrum is less established. Some studies have reported higher PLR in more severe coronary presentations or more complex angiographic disease, whereas the magnitude and consistency of subtype-specific differences remain variable (Kofos et al., 2025; Kurtul et al., 2014; Zhang et al., 2021; Yuksel et al., 2015; Fathurohim et al., 2024).

A direct comparison among UAP, NSTEMI and STEMI is clinically relevant because it may clarify whether PLR primarily separates ischaemia without biomarker-defined necrosis from myocardial infarction, or whether it also differentiates the two infarction phenotypes. In addition, PLR is influenced by systemic inflammation, renal dysfunction, malignancy, metabolic disease and smoking, which can complicate interpretation in acute cardiovascular illness (Brito et al., 2021; Umeres-Francia et al., 2022; Hudzik et al., 2015). This study therefore aimed to compare PLR values across UAP, NSTEMI and STEMI in patients admitted with ACS and to explore whether demographic characteristics, cardiovascular risk factors and selected comorbid conditions were associated with PLR. It was hypothesised that PLR would be higher in myocardial infarction than in UAP, reflecting greater acute thrombo-inflammatory activity.

2. MATERIALS AND METHODS

2.1. Study design, setting and population

This was a single-centre retrospective observational study with a cross-sectional analytic design conducted at a tertiary referral hospital in Makassar, Indonesia. Medical records of patients admitted beginning in December 2025 were reviewed retrospectively until the prespecified sample size was achieved. The analysis evaluated PLR at the index ACS presentation and compared values across UAP, NSTEMI and STEMI.

The source population consisted of adults admitted with ACS whose medical records contained the clinical, electrocardiographic, haematologic and cardiac biomarker data required for the analysis. Consecutive eligible records were included. Participants were aged 18 years or older and had a diagnosis of UAP, NSTEMI or STEMI based on the documented clinical presentation, electrocardiography and cardiac laboratory evaluation. High-sensitivity cardiac troponin I (hs-cTnI) was the cardiac biomarker used in routine care. UAP was defined as an ischaemic ACS presentation without biomarker evidence of acute myocardial necrosis; NSTEMI was defined by an ischaemic presentation with hs-cTnI evidence of myocardial injury or infarction without persistent ST-segment elevation; and STEMI was defined by a compatible ischaemic presentation with

persistent ST-segment elevation or an accepted STEMI-equivalent electrocardiographic pattern (Thygesen et al., 2018). Exclusion criteria were blood transfusion within the preceding 7 days, pregnancy, cytotoxic or immunosuppressive therapy, or incomplete records for the primary PLR analysis.

The planned minimum sample size was 117 participants, assuming a three-group comparison, a medium effect size (f = 0.25), alpha = 0.05 and 80% power. The final analytic sample included 235 patients. This calculation addressed the primary three-group comparison; exploratory subgroup analyses and multivariable models were not separately powered and were interpreted accordingly.

2.2. Variables and definitions

PLR was calculated as platelet count (cells/μL) divided by absolute lymphocyte count (cells/μL). The first complete blood count obtained on arrival at the cardiac emergency department, when ACS was clinically suspected and before final ACS subtype assignment, was used for the PLR calculation. ACS subtype was determined from the documented clinical presentation, electrocardiographic findings and hs-cTnI results as described above.

Demographic and clinical variables included age, sex, diabetes mellitus, hypertension, dyslipidaemia, smoking status, active infection, renal dysfunction on admission, malignancy and autoimmune disease. Diabetes mellitus was defined by a prior diagnosis, fasting plasma glucose ≥126 mg/dL or HbA1c ≥6.5%. Hypertension was defined as blood pressure ≥140/90 mmHg or a prior history of treated hypertension. Dyslipidaemia was recorded from the medical record on the basis of a documented diagnosis or lipid-lowering treatment, supported where available by total cholesterol >200 mg/dL, triglycerides >150 mg/dL or high-density lipoprotein cholesterol <40 mg/dL. Renal dysfunction on admission was defined as an estimated glomerular filtration rate <60 mL/min/1.73 m² or serum creatinine >1.5 mg/dL; because chronicity could not be established consistently from the retrospective records, this variable was not classified as chronic kidney disease. Active infection was defined by a documented clinical diagnosis of infection in the medical record around the ACS presentation, supported by clinical findings and/or relevant investigations; leucocytosis alone was not considered sufficient to define infection. Smoking, malignancy and autoimmune disease were recorded from the medical record.

2.3. Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA), with the multivariable, multinomial and receiver operating characteristic (ROC) analyses independently verified from the original dataset using Python 3. Continuous variables are presented as mean ± standard deviation when approximately normally distributed and as median (interquartile range, IQR) when non-normally distributed; categorical variables are presented as counts and percentages. PLR differences across UAP, NSTEMI and STEMI were evaluated using the Kruskal-Wallis test, followed by Dunn pairwise comparisons with Bonferroni correction. Two-group PLR comparisons used the Mann-Whitney U test.

For the primary adjusted analysis, ACS was dichotomised as myocardial infarction (NSTEMI or STEMI) versus UAP, and binary logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs), with PLR modelled continuously per 50-unit increase. Age, sex, diabetes mellitus, hypertension, dyslipidaemia, smoking, renal dysfunction on admission and active infection were entered as prespecified covariates. To retain the original three-category ACS outcome, a secondary multivariable multinomial logistic regression was fitted using UAP as the reference category and the same covariates. A linear contrast of the PLR coefficients was used to compare STEMI with NSTEMI. Complete-case analysis was used for both multivariable models. ROC analysis quantified the ability of PLR to discriminate myocardial infarction from UAP; the area under the curve (AUC) 95% CI was estimated using 10,000 stratified bootstrap resamples, and the optimal PLR cutoff was selected by maximising the Youden index, with sensitivity and specificity calculated at this cutoff. A two-sided p value <0.05 was considered statistically significant.

2.4. Ethics statement

The study protocol and related documents were approved by the relevant institutional research ethics committee (approval details withheld for blinded peer review). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent to participate was obtained from all participants prior to inclusion in the study.

3. RESULTS AND DISCUSSION

3.1. Baseline and haematologic characteristics

A total of 235 patients with ACS were included. The mean age was 58.49 ± 10.75 years and 181 participants (77.0%) were male. STEMI was the most common ACS subtype (125 patients, 53.2%), while UAP and NSTEMI each occurred in 55 patients (23.4%). Across ACS subtypes there were no statistically significant differences in age, sex, diabetes mellitus, hypertension, dyslipidaemia, smoking, renal dysfunction on admission or active infection (all $p > 0.05$; Table 1). This baseline profile is consistent with commonly reported ACS cohorts: participants were predominantly male and in their late fifties, with high prevalences of smoking and hypertension. Ralapanawa et al. (2019) similarly reported a male-predominant ACS population in a tertiary hospital, while Pagidipati and Peterson (2016) highlighted important sex-related differences in ACS epidemiology and clinical presentation. The predominance of STEMI in the present cohort may reflect referral patterns at a tertiary centre, timing of presentation and local diagnostic practice, and differs from populations in which NSTEMI predominates, particularly in settings with widespread use of high-sensitivity cardiac troponin.

Table 1. Baseline demographic and clinical characteristics by acute coronary syndrome subtype.

Characteristic	UAP (n = 55)	NSTEMI (n = 55)	STEMI (n = 125)	p value
Age, years, mean ± SD	57.0 ± 10.1	59.4 ± 9.4	58.7 ± 11.6	0.469
Male sex, n (%)	38 (69.1)	44 (80.0)	99 (79.2)	0.277
Diabetes mellitus, n (%)	19 (34.5)	19 (34.5)	41 (32.8)	0.961
Hypertension, n (%)	40 (72.7)	34 (61.8)	71 (57.3)	0.144
Dyslipidaemia, n (%)	24 (43.6)	20 (36.4)	64 (51.6)	0.153
Smoking, n (%)	31 (56.4)	39 (70.9)	77 (62.1)	0.279
Renal dysfunction on admission, n (%)	8 (14.5)	12 (21.8)	18 (14.5)	0.439
Active infection, n (%)	5 (9.1)	7 (13.0)	13 (10.5)	0.801

Data are mean ± SD or n (%). The age comparison used one-way analysis of variance; categorical comparisons used the chi-square test on available observations. Percentages are based on non-missing values (missing: hypertension, dyslipidaemia and renal dysfunction, 1 each; active infection, 2). ACS, acute coronary syndrome; NSTEMI, non-ST-elevation myocardial infarction; SD, standard deviation; STEMI, ST-elevation myocardial infarction; UAP, unstable angina pectoris.

In the overall cohort, the median white blood cell count was 11,060 cells/μL (IQR 8,195-13,960), median platelet count was 260,000 cells/μL (IQR 216,000-325,000), median absolute lymphocyte count was 1,665.2 cells/μL (IQR 1,207.8-2,258.7) and median PLR was 152.2 (IQR 111.5-225.7). Mean haemoglobin was 13.48 ± 2.07 g/dL. White blood cell count, absolute lymphocyte count and PLR differed across ACS subtypes, whereas platelet count and haemoglobin did not (Table 2). The pattern indicates that the subtype difference in PLR was driven mainly by the lymphocyte denominator rather than by platelet number, which is consistent with stress-related lymphopenia during acute coronary events.

Table 2. Haematologic characteristics by acute coronary syndrome subtype.

Parameter	UAP (n = 55)	NSTEMI (n = 55)	STEMI (n = 125)	p value
White blood cell count, cells/μL	8,210 (6,910-9,685)	10,650 (7,940-14,325)	12,630 (10,650-15,600)	<0.001
Haemoglobin, g/dL	13.26 ± 1.96	13.09 ± 2.56	13.75 ± 1.83	0.088

Platelet count, cells/μL	272,000 (225,500-315,000)	255,000 (218,000-329,000)	259,000 (214,000-328,000)	0.849
Absolute lymphocyte count, cells/μL	2,214.8 (1,476.0-2,579.5)	1,573.0 (1,236.2-2,081.9)	1,584.4 (1,153.2-2,145.0)	0.003
Platelet-to-lymphocyte ratio	130.5 (96.2-178.4)	170.0 (114.4-233.7)	162.9 (116.1-242.1)	0.002

Data are median (interquartile range) except haemoglobin, which is presented as mean ± standard deviation. Kruskal-Wallis tests were used for non-normally distributed variables and one-way analysis of variance for haemoglobin. NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; UAP, unstable angina pectoris.

3.2. PLR across ACS subtypes

PLR differed significantly among UAP, NSTEMI and STEMI (Kruskal-Wallis p = 0.002). Dunn-Bonferroni post hoc testing showed significant differences between UAP and NSTEMI (p = 0.017) and between UAP and STEMI (p = 0.002), whereas PLR did not differ between NSTEMI and STEMI (p = 1.000). The overall difference across ACS subtypes was therefore principally driven by lower PLR in UAP compared with the two myocardial infarction groups (Table 3).

This finding is biologically plausible. ACS develops through a dynamic interaction among plaque disruption, platelet adhesion and aggregation, thrombin generation, coronary thrombosis, inflammatory signalling and myocardial injury (Ajoolabady et al., 2024; Stakos et al., 2012; Saigusa et al., 2020; Kounis et al., 2021). PLR combines two components of this response: platelets, which participate directly in coronary thrombus formation and inflammatory amplification, and lymphocytes, which may decline during acute stress because of neuroendocrine activation, apoptosis and redistribution. An increase in the numerator, a decrease in the denominator, or both can therefore raise PLR during a more intense acute thrombo-inflammatory state. The present subtype findings are broadly consistent with previous work linking higher PLR to more severe ACS or coronary disease. Kofos et al. (2025) reported subtype-related differences in PLR among hospitalised ACS patients; Kurtul et al. (2014) found that PLR was associated with the severity and complexity of coronary artery disease in ACS as measured by the SYNTAX score; Yuksel et al. (2015) reported a positive relationship between PLR and angiographic coronary severity; and Zhang et al. (2021) identified differing risk profiles across ACS subtypes.

The lack of a significant PLR difference between NSTEMI and STEMI is equally important. Although STEMI is often associated with abrupt epicardial occlusion and a large ischaemic burden, NSTEMI remains a myocardial infarction phenotype characterised by necrosis and substantial platelet-inflammatory activation. A biomarker such as PLR may therefore rise once the pathobiologic threshold of infarction is crossed without providing sufficient resolution to distinguish electrocardiographic infarction phenotypes. This interpretation is more cautious than treating PLR as a linear measure of ACS severity from UAP through NSTEMI to STEMI.

Table 3. Platelet-to-lymphocyte ratio across acute coronary syndrome subtypes and post hoc comparisons.

ACS subtype	**PLR, median (IQR)**	**Minimum**	**Maximum**	**Overall p**
UAP	130.5 (96.2-178.4)	49.4	376.7	0.002
NSTEMI	170.0 (114.4-233.7)	49.5	519.1	
STEMI	162.9 (116.1-242.1)	58.8	498.5	
Pairwise comparison	**Mean rank, group 1**	**Mean rank, group 2**	**Z**	**Bonferroni p**
UAP vs NSTEMI	UAP 90.07	NSTEMI 125.84	-2.759	0.017
UAP vs STEMI	UAP 90.07	STEMI 126.84	-3.342	0.002
NSTEMI vs STEMI	NSTEMI 125.84	STEMI 126.84	-0.091	1.000

Overall comparison by Kruskal-Wallis test; pairwise comparisons by Dunn test with Bonferroni correction. ACS, acute coronary syndrome; IQR, interquartile range; PLR, platelet-to-lymphocyte ratio.

3.3. PLR according to demographic and clinical characteristics

In the overall cohort, PLR did not differ significantly by sex, age group, diabetes mellitus, hypertension, dyslipidaemia, smoking status, active infection, renal dysfunction on admission or malignancy (all $p > 0.05$; Table 4). PLR was numerically higher among patients with active infection and renal dysfunction on admission, but these differences were not statistically significant, and the malignancy comparison was based on only three affected patients. These unadjusted comparisons were considered descriptive because the primary adjusted analysis focused on myocardial infarction versus UAP.

This should not be interpreted as evidence that these conditions do not influence PLR. Rather, the multivariable models indicate that the association between PLR and myocardial infarction was not explained by the measured covariates available in this dataset. The comorbidities were recorded categorically and therefore did not capture disease duration, treatment status, metabolic control, lipid concentrations, blood-pressure severity or cumulative tobacco exposure. Chronic cardiovascular risk factors contribute to atherosclerosis over years, whereas PLR measured during an acute admission may be more sensitive to contemporaneous inflammation, thrombosis and physiologic stress. In diabetes, for example, Hudzik et al. (2015) found PLR to be prognostically relevant in patients with STEMI, a different question from whether PLR differs among ACS subtypes within a diabetic subgroup. Age may likewise modify the prognostic significance of PLR after ACS, as age-specific prognostic associations have been reported previously (Kazem et al., 2022).

Table 4. Platelet-to-lymphocyte ratio according to demographic and clinical characteristics.

Characteristic	Category	PLR, median (IQR)	p value
Sex	Male	162.9 (111.6-225.9)	0.199
	Female	144.7 (111.7-192.6)	
Age	≤50 years	137.8 (94.6-215.9)	0.163
	>50 years	155.6 (113.8-229.5)	
Diabetes mellitus	Yes	176.8 (113.9-225.8)	0.473
	No	151.4 (109.9-219.3)	
Hypertension	Yes	149.1 (111.0-213.4)	0.675
	No	163.8 (111.7-242.1)	
Dyslipidaemia	Yes	150.7 (113.3-220.8)	0.855
	No	156.3 (106.2-231.4)	
Smoking	Yes	157.1 (111.5-224.0)	0.688
	No	145.7 (111.4-231.0)	
Active infection	Yes	176.8 (101.6-349.4)	0.205
	No	151.4 (111.6-215.7)	
Renal dysfunction on admission	Yes	177.0 (112.0-263.0)	0.253
	No	151.7 (111.4-213.9)	
Malignancy	Yes	132.4 (106.0-157.3)	0.425
	No	152.4 (111.4-225.8)	

Two-group comparisons were performed with the Mann-Whitney U test. Autoimmune disease was not analysed because no participant had a recorded autoimmune condition. IQR, interquartile range; PLR, platelet-to-lymphocyte ratio.

When PLR was compared across ACS subtypes within demographic and clinical strata, significant between-subtype differences were observed among men, both age categories, patients without diabetes, patients without hypertension, patients without dyslipidaemia, smokers and non-smokers, patients without active infection, patients without renal dysfunction on admission and patients without malignancy. Differences were not significant among women or among patients with diabetes, hypertension, dyslipidaemia, active infection or renal dysfunction on admission. These patterns may reflect biological effect modification, but they may equally arise from reduced subgroup sample sizes, unequal distributions and multiple testing; they are therefore best viewed as hypothesis-generating, and future studies should formally test interactions in multivariable models rather than infer subgroup effects from separate unadjusted comparisons.

3.4. Multivariable association with myocardial infarction

When NSTEMI and STEMI were combined as the myocardial infarction group and compared with UAP, PLR remained independently associated with myocardial infarction after adjustment for age, sex, diabetes mellitus, hypertension, dyslipidaemia, smoking, renal dysfunction on admission and active infection. The complete-case multivariable model included 233 patients. Each 50-unit increase in PLR was associated with a 40% increase in the adjusted odds of myocardial infarction rather than UAP (adjusted OR 1.40, 95% CI 1.12-1.76; p = 0.004), and none of the other measured covariates reached conventional statistical significance in the adjusted model (Table 5). The persistence of the association after adjustment indicates that the subtype difference was not merely a reflection of the risk-factor profile of the infarction groups.

Table 5. Univariable and multivariable logistic regression for myocardial infarction (NSTEMI or STEMI) versus unstable angina pectoris.

Variable	Unadjusted OR (95% CI)	p value	Adjusted OR (95% CI)	p value
PLR, per 50-unit increase	1.43 (1.15-1.78)	0.002	1.40 (1.12-1.76)	0.004
Age, per year	1.02 (0.99-1.05)	0.241	1.02 (0.99-1.05)	0.236
Male sex (vs female)	1.73 (0.88-3.40)	0.113	1.41 (0.60-3.30)	0.427
Diabetes mellitus	0.95 (0.50-1.79)	0.868	0.88 (0.43-1.80)	0.728
Hypertension	0.53 (0.27-1.03)	0.062	0.50 (0.25-1.01)	0.054
Dyslipidaemia	1.14 (0.62-2.10)	0.669	1.26 (0.65-2.44)	0.492
Smoking	1.43 (0.77-2.64)	0.258	1.27 (0.59-2.72)	0.537
Renal dysfunction on admission	1.18 (0.51-2.76)	0.697	0.99 (0.39-2.53)	0.984
Active infection	1.27 (0.45-3.55)	0.654	1.08 (0.36-3.25)	0.898

Adjusted model n = 233 (complete cases). PLR was modelled continuously per 50-unit increase. CI, confidence interval; NSTEMI, non-ST-elevation myocardial infarction; OR, odds ratio; PLR, platelet-to-lymphocyte ratio; STEMI, ST-elevation myocardial infarction.

3.5. Multinomial analysis across ACS subtypes

In the secondary multinomial model with UAP as the reference category, the complete-case analysis included 233 patients (UAP n = 55, NSTEMI n = 54, STEMI n = 124). After adjustment for the same covariates, each 50-unit increase in PLR was associated with higher odds of NSTEMI versus UAP (adjusted OR 1.35, 95% CI 1.04-1.74; p = 0.024) and of STEMI versus UAP (adjusted OR 1.43, 95% CI 1.13-1.81; p = 0.003). A direct contrast showed no adjusted difference in the PLR association between STEMI and NSTEMI (adjusted OR 1.06, 95% CI 0.89-1.26; p = 0.500), supporting the interpretation that the principal PLR separation was between UAP and the myocardial infarction phenotypes (Table 6).

Table 6. Multivariable multinomial logistic regression for acute coronary syndrome subtype (reference category: unstable angina pectoris).

Variable	NSTEMI vs UAP, adjusted OR (95% CI)	p value	STEMI vs UAP, adjusted OR (95% CI)	p value
PLR, per 50-unit increase	1.35 (1.04-1.74)	0.024	1.43 (1.13-1.81)	0.003
Age, per year	1.02 (0.99-1.06)	0.230	1.02 (0.98-1.05)	0.322
Male sex (vs female)	1.18 (0.40-3.48)	0.767	1.52 (0.62-3.75)	0.361
Diabetes mellitus	0.95 (0.40-2.26)	0.907	0.86 (0.41-1.83)	0.698
Hypertension	0.55 (0.24-1.29)	0.171	0.48 (0.23-1.00)	0.051
Dyslipidaemia	0.81 (0.36-1.82)	0.605	1.53 (0.76-3.05)	0.230
Smoking	1.72 (0.66-4.51)	0.271	1.12 (0.50-2.48)	0.789
Renal dysfunction on admission	1.38 (0.47-4.08)	0.556	0.83 (0.30-2.24)	0.709
Active infection	1.19 (0.33-4.28)	0.794	1.02 (0.32-3.29)	0.972

Adjusted model n = 233 (complete cases; UAP n = 55, NSTEMI n = 54, STEMI n = 124). PLR was modelled continuously per 50-unit increase. The adjusted PLR coefficient contrast for STEMI versus NSTEMI corresponded to an odds ratio of 1.06 (95% CI 0.89-1.26; p = 0.500). CI, confidence interval; NSTEMI, non-ST-elevation myocardial infarction; OR, odds ratio; PLR, platelet-to-lymphocyte ratio; STEMI, ST-elevation myocardial infarction; UAP, unstable angina pectoris.

3.6. Discriminative performance and clinical implications

ROC analysis showed that PLR had modest ability to discriminate myocardial infarction from UAP, with an AUC of 0.655 (95% CI 0.574-0.733; $p < 0.001$). The Youden-optimal cutoff was 155.6, providing 56.1% sensitivity and 72.7% specificity (Youden index 0.288). These values indicate statistically significant but limited stand-alone discrimination (Table 7 and Figure 1).

Table 7. Receiver operating characteristic performance of the platelet-to-lymphocyte ratio for myocardial infarction (NSTEMI or STEMI) versus unstable angina pectoris.

Marker	AUC (95% CI)	Optimal cutoff	Sensitivity	Specificity	Youden index
PLR	0.655 (0.574-0.733)	155.6	56.1%	72.7%	0.288

The AUC confidence interval was estimated with 10,000 stratified bootstrap resamples, and the cutoff maximised the Youden index. AUC, area under the receiver operating characteristic curve; CI, confidence interval; NSTEMI, non-ST-elevation myocardial infarction; PLR, platelet-to-lymphocyte ratio; STEMI, ST-elevation myocardial infarction.

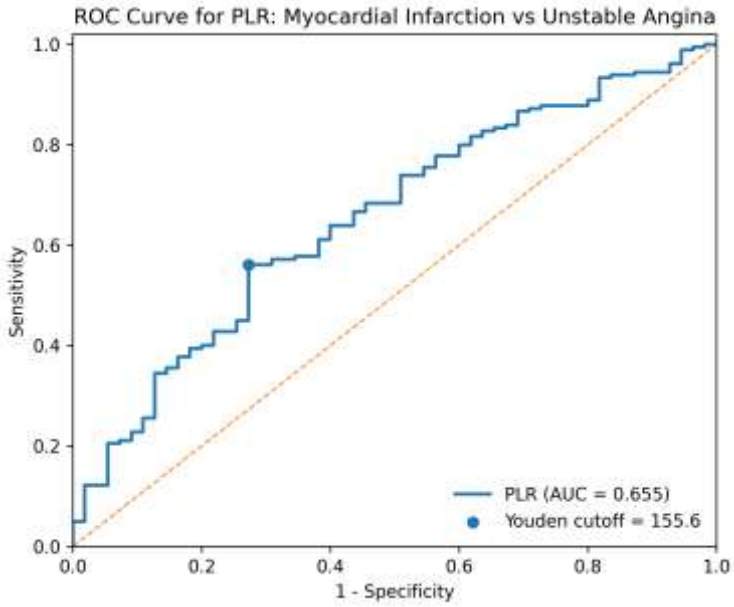

Figure 1. Receiver operating characteristic curve of the platelet-to-lymphocyte ratio for discriminating myocardial infarction (NSTEMI or STEMI) from unstable angina pectoris. The area under the curve was 0.655 (95% confidence interval 0.574-0.733). NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction.

From a clinical perspective, PLR has practical appeal because it is derived from a complete blood count routinely obtained at the initial ACS evaluation. In this study, PLR remained independently associated with myocardial infarction phenotype, but an AUC of 0.655 and a sensitivity of 56.1% at the Youden-optimal cutoff indicate that it cannot reliably classify individual patients by itself. Electrocardiography, hs-cTnI, clinical assessment and established risk-stratification tools remain fundamental (Byrne et al., 2023; Thygesen et al., 2018). PLR may therefore be most useful as a low-cost complementary index of the inflammatory-thrombotic response, particularly in settings where additional biomarkers are not routinely available.

3.7. Strengths and limitations

This study included a relatively large single-centre cohort, directly compared all three conventional ACS subtypes and used post hoc pairwise testing to identify which groups drove the overall difference. It also evaluated a range of demographic characteristics, cardiovascular risk factors and clinical comorbidities that could influence PLR. In addition, both binary and multinomial multivariable models were used, allowing the principal myocardial-infarction-versus-UAP finding to be checked while retaining the original three-category ACS outcome.

Several limitations should be acknowledged. First, the retrospective cross-sectional design relies on information recorded during routine care and cannot establish temporal or causal relationships. Second, the study was conducted at a single tertiary hospital, limiting generalisability. Third, PLR was based on a single complete blood count obtained at the index emergency presentation; serial changes during treatment were not assessed. Fourth, inflammatory markers such as C-reactive protein and specific markers of platelet activation were not systematically available, preventing direct comparison of PLR with more pathway-specific biomarkers. Fifth, some clinically important subgroups, particularly active infection, renal dysfunction on admission and malignancy, were small, and no participant had a recorded autoimmune disease. Sixth, although the multivariable models adjusted for several prespecified covariates, residual and unmeasured confounding remains possible; the original sample-size calculation was based on the primary three-group comparison rather than on individual subgroup or multivariable parameters, so estimates from smaller strata and some covariate categories remain imprecise. Seventh, the ROC analysis was internally derived from the same cohort and was not externally validated, so the cutoff of 155.6 should not be considered a clinical decision threshold. Finally, the study did not evaluate clinical outcomes such as mortality, major adverse cardiovascular events, heart failure, length of stay, reperfusion success or no-reflow. These limitations are important when translating the observed PLR differences into clinical use.

4. CONCLUSION

The platelet-to-lymphocyte ratio differed significantly across acute coronary syndrome subtypes in this 235-patient retrospective cross-sectional study. PLR was lower in unstable angina pectoris than in NSTEMI and STEMI, while the two myocardial infarction phenotypes had comparable values. Each 50-unit increase in PLR remained independently associated with myocardial infarction versus unstable angina pectoris after adjustment for measured clinical covariates (adjusted OR 1.40, 95% CI 1.12-1.76), and the secondary multinomial model gave adjusted ORs of 1.35 for NSTEMI and 1.43 for STEMI versus unstable angina pectoris, with a non-significant STEMI-versus-NSTEMI contrast. Nevertheless, PLR provided only modest discrimination (AUC 0.655) and should not be used as a stand-alone diagnostic test. Prospective multicentre studies incorporating serial PLR measurements, inflammatory biomarkers, clinical outcomes and external validation are warranted before PLR can be recommended for routine subtype-oriented decision-making.

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AUTHOR CONTRIBUTIONS

Conceptualisation: M.R. Juni, I. Mappangara. Methodology: M.R. Juni, A.A. Zainuddin. Formal analysis: M.R. Juni, A.A. Zainuddin. Investigation and data curation: M.R. Juni. Supervision: I. Mappangara, S. Saleh, S. Bakri, H. Kasim. Writing – original draft: M.R. Juni. Writing – review and editing: all authors. All authors have read and approved the final version of the manuscript.

CONFLICT OF INTEREST

The authors declare that they have no competing interests relevant to this article.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol and related documents were approved by the relevant institutional research ethics committee. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki, and written informed consent to participate was obtained from all participants prior to inclusion.

DATA AVAILABILITY

The de-identified dataset supporting the findings of this study is available from the corresponding author on reasonable request.

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