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Predicting Acute Myocardial Infarction Severity Using Inflammatory Markers Neutrophil-to-Lymphocyte Ratio and Platelet-to-Lymphocyte Ratio

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ABSTRACT

Introduction: Acute myocardial infarction (AMI) remains as one of major contributors to cardiovascular mortality worldwide. While Killip class and GRACE score are established tools for assessing AMI severity, evidence regarding prognostic potential of hematologic inflammatory markers, neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) remains limited, particularly in Southeast Asia. This study aimed to see the correlation between NLR and PLR with Killip classification, GRACE score, and in-hospital mortality in AMI patients.

Methods: This retrospective study included 47 AMI patients admitted to the Intensive Cardiovascular Care Unit of Universitas Sebelas Maret Hospital between September 2024 and June 2025. Data on clinical and laboratory parameters were extracted from medical records. NLR and PLR were calculated from complete blood count values. Killip class and GRACE score were assessed at admission. Statistical analyses included correlation tests, regression models, and outcome comparisons.

Results: Higher NLR and PLR values were significantly associated with increasing Killip class ($\rho = 0.897$ and $\rho = 0.921$, respectively) and GRACE score ($p < 0.001$). PLR was independent predictor of Killip class ($p = 0.020$), while only troponin I was independently associated with in-hospital mortality ($p = 0.040$). The GRACE score regression model showed excellent explanatory power ($R^2 = 0.919$), with NLR, PLR, and clinical variables contributing significantly.

Conclusion: NLR and PLR are significantly correlated with AMI severity and risk scores. PLR may serve as a simple adjunctive marker for early clinical stratification in AMI.

Keywords: acute myocardial infarction; NLR; PLR; Killip class; GRACE score

INTRODUCTION

Acute myocardial infarction (AMI) remains as one of leading causes of cardiovascular mortality worldwide, including in Indonesia[1]. Early and accurate risk stratification is important for optimizing clinical outcomes, with tools such as the Killip classification and the Global Registry of Acute Coronary Events (GRACE) score widely utilized to assess clinical severity and predict mortality[2,3].

Inflammation plays an essential role in pathophysiology of ACS, contributing to atherosclerotic plaque progression, rupture, and intracoronary thrombosis[4,5]. In this context, hematological inflammatory markers—particularly the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-

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lymphocyte ratio (PLR)—have emerged as simple, cost-effective indicators of systemic inflammation in cardiovascular disease, including AMI[4–7].

Elevated NLR and PLR values at admission have been associated with worse clinical outcomes, including heart failure, mechanical complications, and in-hospital mortality[6,7]. However, data linking these markers directly to validated clinical risk scores such as the Killip class and GRACE score remain limited, particularly within Southeast Asian populations. Given their accessibility, affordability, and potential prognostic value, it is important to explore whether NLR and PLR can serve as early markers of clinical severity in AMI. The study aims to evaluate the association between admission levels of NLR and PLR with Killip classification and GRACE score, and to assess their potential role as early predictors for risk stratification in patients with AMI.

METHODS

Study design and study participants

This retrospective cohort study was conducted at Universitas Sebelas Maret Hospital, Sukoharjo, Indonesia, using medical records from September 2024 to June 2025. Patients diagnosed with AMI (STEMI and NSTEMI) and admitted to the Intensive Cardiovascular Care Unit (ICVCU) were included. Diagnosis was based on clinical assessment, ECG findings, and elevated troponin I levels. Inclusion required complete data on laboratory parameters, Killip class, and GRACE score. Patients with conditions that could affect inflammatory markers—such as acute infections, autoimmune or hematologic diseases, malignancy, recent major surgery, or trauma—were excluded. A total sampling method was used. The research protocol was reviewed and approved by UNS Hospital Ethics Committee with approval number: No.077/UN27.46/TA.04.19/KEP/EC/2025. All participants provided informed consent prior to their participation.

Data collection

Data were collected from the hospital's electronic records before any treatment was initiated. Demographic details, vital signs, and laboratory results were recorded. NLR and PLR were calculated as the ratio of neutrophils or platelets to lymphocyte count, respectively. Killip classification at admission was used to categorize heart failure severity, while GRACE scores were calculated based on standard components including age, blood pressure, creatinine, heart rate, troponin I, ST changes, and cardiac arrest status. The primary outcome was in-hospital all-cause mortality. Variables with >50% missing data were excluded, and multiple imputation was applied for the remaining missing values.

Study Outcome

The primary objective was to assess the association between admission NLR and PLR values with AMI severity, based on Killip classification and GRACE score. Secondary outcomes included evaluating the predictive value of these markers for in-hospital mortality and comparing clinical characteristics between survivors and non-survivors. Correlation strength and effect sizes (ORs, regression coefficients) were also analyzed.

Statictical analitycs

All analyses were carried out using SPSS version 27.0. Descriptive statistics summarized baseline characteristics. Normality was assessed with the Shapiro–Wilk test. Continuous variables were reported as mean ± SD or median (IQR), while categorical data were presented as frequencies and percentages. Group comparisons were investigated using one-way ANOVA or Kruskal–Wallis tests, as appropriate. Correlation analyses employed Pearson or Spearman coefficients. Multivariate linear and logistic regressions were used to identify independent predictors of Killip class, GRACE score, and

mortality, adjusting for confounders. Significant variables in bivariate analysis were included in multivariate models. P-value < 0.05 was considered statistically significant.

RESULTS

Baseline Characteristic

This study was conducted by reviewing the medical records of patients diagnosed with AMI (STEMI or NSTEMI) between September 2024 and June 2025. 47 patients were included, selected using a purposive sampling based on the predefined criteria.

As presented in Table 1, the majority of patients were male (68.1%), with a mean age of 60.98 ± 12.41 years. Most patients were diagnosed with STEMI (68.1%), while the remainder had NSTEMI (31.9%). Regarding Killip classification, the majority of patients were classified as Killip class I (42.6%), followed by class IV (23.4%), class II (19.1%), and class III (14.9%). A total of 25 patients (53.2%) died, while 22 patients (46.8%) survived. Kruskal-Wallis and ANOVA tests revealed statistically significant differences among the Killip class groups in several variables. These included respiratory rate (p = 0.017), oxygen saturation (p = 0.007), systolic blood pressure (p = 0.013), platelet count (p = 0.010), leukocyte count (p = 0.024), lymphocyte percentage (p < 0.001), neutrophil percentage (p < 0.001), potassium level (p = 0.033), urea (p = 0.004), creatinine (p = 0.006), as well as NLR (p < 0.001), PLR (p < 0.001), and troponin I (p < 0.001).

Post hoc analysis revealed the most notable differences were between Killip class I and classes II, III, and IV in terms of NLR, PLR, and troponin I. Significant differences were also observed between Killip class II and classes III and IV for these same parameters. These findings suggest that elevated NLR, PLR, and troponin I levels are associated with a higher Killip class, indicating a more severe clinical condition.

Table 1. Baseline Characteristics of the Research Sample (n = 47)

Characteristic	IMA (n = 47)	Killip Class (p-value)	Outcome (p-value)
Age (years)[1]	60.98 ± 12.41	0.645[a]	0.048[d]
Sex (n (%))		0.613[c]	
Male	32 (68.1%)		0.989[b]
Female	15 (31.9%)		
HR (bpm)[1]	82.36 ± 23.62	0.255[a]	0.225[d]
RR (x/mins)[2]	20 (20, 22)	0.017*[a]	0.019*[c]
SpO2 (%)[2]	97 (92, 98)	0.007*[a]	0.023*[c]
SBP (mmHg)[1]	123.72 ± 32.12	0.013*[a]	0.016*[d]
DBP (mmHg)[2]	85 (63, 98)	0.394[a]	0.114[c]
GRACE *Score*[1]	130.68 ± 43.22	0.000[e]	0.000[d]
Killip Class (n (%))		-	
I	20 (42.6%)		0.000*[c]
II	9 (19.1%)		
III	7 (14.9%)		
IV	11 (23.4%)		
Outcome (n (%))			-
Survived	22 (46.8%)	0.000*[c]	

Characteristic	IMA (n = 47)	Killip Class (p-value)	Outcome (p-value)
Deceased	25 (53.2%)		
Diagnosis (n (%))		0.904[c]	0.215[b]
STEMI	32 (68.1%)		
NSTEMI	15 (31.9%)		
Laboratorium			
Hb (g/dL)[1]	13.86 ± 2.22	0.937[a]	0.422[d]
HCT (%)[1]	40.98 ± 6.8	0.996[a]	0.597[d]
Platelet (×10³/µL)[1]	263 (214, 383)	0.010*[a]	0.008[c]
Leukocyte (×10³/µL)[2]	12.59 (9.07, 15.15)	0.024*[a]	0.040*[c]
Erythrocyte (×10⁶/µL)[1]	4.79 ± 0.73	0.979[a]	0.714[d]
Lymphocyte (%)[1]	21.21 ± 10.91	<0.001*[a]	<0.001*[d]
Monocyte (%)[1]	5.56 ± 1.60	0.86[a]	0.920[d]
Neutrophil (%)[2]	71 (63.3, 82.2)	<0.001*[a]	0.000*[c]
Eosinophil (%)[2]	1.2 (0.2, 2.1)	0.079[a]	0.005*[c]
Basophil (%)[2]	0.2 (0.1, 0.4)	0.734[a]	0.386[c]
Sodium (mmol/L)[1]	136.76 ± 3.55	0.757[a]	0.393[d]
Potassium (mmol/L)[1]	3.80 ± 0.68	0.033*[a]	0.027*[d]
Chloride (mmol/L)[2]	103.9 (101.2, 105.6)	0.607[a]	0.488[c]
Calcium (mmol/L)[2]	1.09 (1.04, 1.15)	0.696[a]	0.890[c]
Ureum (mg/dL)[2]	38 (28, 62)	0.004*[a]	0.001*[c]
Creatinine (mg/dL)[2]	1.3 (0.81, 2.11)	0.006*[a]	0.006*[c]
Blood Glucose (mg/dL)[2]	156 (126, 242)	0.566[a]	0.898[c]
NLR[2]	3.48 (2.11, 6.96)	<0.001*[a]	<0.001*[c]
PLR[2]	128.24 (74.02, 194.21)	<0.001*[a]	<0.001*[c]
Troponin I[2]	1370 (169, 5182)	<0.001*[a]	<0.001*[c]

Description : *p <0.05 significant; 1, reported using Mean±SD, 2, reported using Median (Q1,Q3); a, Kruskall-Walis; b, Chi-Square; c, Mann-Whitney U; d, T-Test; e, ANOVA one-way

Correlation Analysis Results

Data normality was assessed using the Shapiro-Wilk test. Spearman correlation analysis demonstrated a very strong positive correlation between Killip class and several biomarkers: NLR ($\rho = 0.897$; $p < 0.001$), PLR ($\rho = 0.921$; $p < 0.001$), and troponin I ($\rho = 0.854$; $p < 0.001$). Positive correlations were also found with respiratory rate, platelet count, leukocyte count, neutrophil percentage, potassium, urea, and creatinine levels. In contrast, Killip class was negatively correlated with oxygen saturation ($\rho = –0.482$; $p < 0.001$), systolic blood pressure ($\rho = –0.407$; $p = 0.005$), lymphocyte percentage ($\rho = –0.908$; $p < 0.001$), and eosinophil count ($\rho = –0.298$; $p = 0.042$).

Analysis of correlations with the GRACE score revealed significant positive correlations with age, platelet count, neutrophil percentage, potassium, urea, creatinine, NLR, PLR, and troponin I. In contrast, negative correlations were observed between GRACE score and oxygen saturation ($\rho = -0.423$; $p = 0.003$), systolic blood pressure ($\rho = -0.653$; $p < 0.001$), and lymphocyte percentage ($\rho = -0.631$; $p < 0.001$). These findings indicate that higher GRACE scores are associated with worsening physiological and laboratory parameters.

Table 2. Correlation Test Results of Variables against Killip Class and GRACE Score

Variable	Parameter	Correlation	p-value
Killip Class	Age	0.163	0.273[b]
	HR	0.241	0.103[b]
	RR	0.373	0.010*[b]
	SpO2	-0.482	<0.001*[b]
	SBP	-0.407	0.005*[b]
	DBP	-0.245	0.097[b]
	Hb	-0.073	0.626[b]
	HCT	-0.028	0.852[b]
	Platelet	0.479	<0.001*[b]
	Leukocyte	0.447	0.002*[b]
	Erythrocyte	0.027	0.859[b]
	Lymphocyte	-0.908	<0.001*[b]
	Monocyte	0.066	0.658[b]
	Neutrophil	0.874	<0.001*[b]
	Eosinophil	-0.298	0.042*[b]
	Basophil	0.027	0.855[b]
	Sodium	0.115	0.442[b]
	Potassium	0.310	0.034*[b]
	Chloride	0.137	0.359[b]
	Calcium	-0.064	0.667[b]
	Ureum	0.462	<0.001*[b]
	Creatinine	0.451	<0.001*[b]
	Blood Glucose	-0.022	0.882[b]
	NLR	0.897	<0.001*[b]
	PLR	0.921	<0.001*[b]
	Troponin I	0.854	<0.001*[b]
GRACE Score	Age	0.720	<0.001*[a]
	HR	0.137	0.359[a]
	RR	0.243	0.1[b]
	SpO2	-0.423	0.003*[b]
	SBP	-0.653	<0.001*[a]
	DBP	-0.464	0.001*[b]
	Hb	-0.286	0.051[a]
	HCT	-0.242	0.102[a]
	Platelet	0.385	0.007*[b]
	Leukocyte	0.262	0.075[b]
	Erythrocyte	-0.235	0.111[a]
	Lymphocyte	-0.631	<0.001*[a]
	Monocyte	0.119	0.425[a]
	Neutrophil	0.656	<0.001*[b]

Variable	Parameter	Correlation	p-value
	Eosinophil	-0.344	0.018*[b]
	Basophil	-0.038	0.799[b]
	Sodium	0.042	0.78[a]
	Potassium	0.463	0.001*[a]
	Chloride	0.156	0.296[b]
	Calcium	-0.069	0.646[b]
	Ureum	0.583	<0.001*[b]
	Creatinine	0.407	0.005*[b]
	Blood Glucose	0.084	0.576[b]
	NLR	0.635	<0.001*[b]
	PLR	0.700	<0.001*[b]
	Troponin I	0.713	<0.001*[b]

Description: *p < 0,05 significant; a, Pearson; b, Spearman

Multivariate Analysis of Killip Class

Table 3. Multivariate Analysis of Ordinal Logistic Regression on Killip Class

Variable	Estimate	(95% CI)		p
		Lower	Upper	
NLR	0.469	-1.799	2.737	0.685
PLR	0.221	0.035	0.408	0.020*
Troponin I	0.002	0.000	0.005	0.091
SBP	-0.075	-0.191	0.040	0.201
RR	0.427	-0.726	1.580	0.468
SpO2	-0.032	-0.858	0.795	0.940
Eosinophil	1.853	-0.190	5.186	0.276
Ureum	-0.27	-3.350	0.136	0.745
Creatinine	1.962	-9.705	7.275	0.469
Potassium	-4.010	-3.914	1.684	0.168
Sex	4.406	-3.914	12.727	0.299
Diagnosis	0.061	-6.190	6.312	0.985

To assess the influence of NLR, PLR, and troponin I, along with potential confounding variables, on the Killip classification of patients, an ordinal logistic regression analysis was conducted. The overall model was significant (Chi-square = 122.538; $p < 0.001$), indicating that the set of predictors reliably distinguished between the Killip class categories. Based on the parameter estimates, only PLR demonstrated a statistically significant association with Killip class ($p = 0.020$). The regression coefficient for PLR was 0.221, with a 95% CI ranging from 0.035 to 0.408, suggesting that an increase

in PLR is associated with higher odds of a patient being classified into a more severe Killip class. This finding implies that elevated PLR values may reflect more severe clinical manifestations of heart failure. In contrast, troponin I (p = 0.091) and NLR (p = 0.685) were not significantly associated with Killip class in the multivariate model.

Multivariate Analysis of Patient Outcomes

To evaluate the effects of NLR, PLR, and troponin I on in-hospital mortality, a binary logistic regression analysis was conducted, as the dependent variable (survival status) was dichotomous (survived vs. deceased). Among the three predictors, only troponin I was found to have a statistically significant association with patient outcomes (p = 0.040). The regression coefficient for troponin I was 0.002, with an odds ratio (Exp(B)) of 1.002, indicating that each 1-unit increase in troponin I level was associated with a 0.2% increase in the odds of in-hospital mortality. In contrast, NLR (p = 0.805) and PLR (p = 0.770) did not show statistically significant associations with mortality in this model. These findings suggest that among the three biomarkers evaluated, troponin I was the only significant independent predictor of in-hospital death, whereas NLR and PLR were not significant predictors in this multivariate analysis.

Table 4. Multivariate Analysis of Binary Logistic Regression on Patient Outcome

	B	SE	p-value	OR (Exp (B))
Age	0.085	0.063	0.177	1.088
NLR	0.201	0.813	0.805	1.223
PLR	0.008	0.028	0.770	1.008
Troponin I	0.002	0.001	0.040	1.002
SBP	0.006	0.022	0.779	1.006
SpO2	0.234	0.140	0.095	1.263
RR	0.208	0.148	0.159	1.231
Constant	-36.920	18.742	0.049	0.000

Multivariate Analysis of GRACE Score

To assess the effect of NLR, PLR, and troponin I on GRACE scores, a multiple linear regression analysis was conducted. Prior to the regression, classical assumption tests including assessments of normality, linearity, homoscedasticity, and multicollinearity were performed and all assumptions were met, ensuring the validity of the regression results. The F-test in the ANOVA table showed that the overall regression model was statistically significant ($F = 46.846$; $p < 0.001$), indicating that the set of independent variables collectively influenced the GRACE score. The model's R^2 value was 0.919, suggesting that approximately 91.9% of the variation in GRACE scores could be explained by the combined effects of the predictor variables, which included NLR, PLR, troponin I, and several confounding factors such as age, systolic blood pressure, and oxygen saturation.

The t-test results indicated that both NLR (p = 0.029) and PLR (p = 0.025) had a statistically significant positive effect on GRACE scores, with regression coefficients of 2.497 and 0.092, respectively. These findings suggest that increases in NLR and PLR are associated with higher GRACE scores, reflecting greater clinical risk. In contrast, troponin I did not exhibit a significant association

with GRACE score in the multivariate model (p = 0.454), despite its significant predictive value for patient outcomes observed in prior analyses.

Among the confounding variables, several demonstrated significant associations with GRACE scores. Age was a strong predictor (p < 0.001), with a regression coefficient of 1.520, indicating that older age is associated with higher clinical risk. Systolic blood pressure (SBP) was inversely associated with GRACE scores (p = 0.001; B = –0.347), suggesting that lower SBP corresponds to increased risk. Similarly, oxygen saturation (SpO_2) at admission showed a significant inverse relationship (p = 0.020; B = –0.641), with lower SpO_2 levels linked to higher GRACE scores.In conclusion, both inflammatory markers (NLR and PLR) and conventional clinical parameters (age, SBP, and oxygen saturation) significantly contribute to the estimation of clinical risk as reflected by GRACE scores in patients with ACS.

Table 5. Linear Regression Multivariate Analysis of GRACE Score

Model	Variable	B	(95% CI)		p
			Lower	Upper	
1	(*Constant*)	121.231	47.188	195.274	0.002
	Age	1.520	1.120	1.919	<0.001
	NLR	2.497	0.263	4.731	0.029
	PLR	0.092	0.012	0.173	0.025
	Troponin I	0.000	0.000	0.001	0.454
	SBP	-0.347	0.546	-0.148	0.001
	DBP	-0.151	-0.429	-0.126	0.277
	SpO2	-0.641	-1.177	-0.106	0.020
	Eosinophil	-2.807	0.395	0.395	0.084
	Potassium	2.336	9.750	9.750	0.527

Description: CI, confidence interval; p, significant
R *Square* = 0,919; p *value* F test = 0.000

DISCUSSION

This study investigated the role of NLR and PLR as inflammatory biomarkers in assessing the severity and prognosis of AMI, particularly in relation to Killip class, GRACE score, and in-hospital mortality. Our findings demonstrate that both NLR and PLR were significantly elevated in patients with higher Killip classes and higher GRACE scores, reflecting more severe clinical presentations. These results support the notion that systemic inflammation, as represented by elevated NLR and PLR, plays a significant role in the pathophysiology and progression of AMI.[4,5,8,9]

The strong positive correlations between NLR, PLR, and Killip class ($\rho = 0.897$ and $\rho = 0.921$, respectively) underscore the relationship between heightened inflammatory states and worsening heart failure classification. These are consistent with prior studies indicating that elevated NLR and PLR values are associated with poorer cardiac function, increased myocardial injury, and higher rates of complications.[3–5,7] Previous studies found that patients with STEMI and higher NLR levels were more likely to present with Killip class II–IV and had worse outcomes.[10] Other studies demonstrated that PLR is a useful marker in predicting short- and long-term mortality in patients with ACS.[4,7,11,12] Our results

further reinforce these observations by providing robust correlation coefficients and statistically significant associations across multiple parameters of disease severity.

Interestingly, while both NLR and PLR were strongly correlated with Killip class and GRACE score, multivariate analysis revealed that only PLR remained a significant independent predictor of Killip class ($p = 0.020$). This suggests that PLR may be a more robust inflammatory marker in capturing the severity of heart failure in AMI patients, potentially due to the combined effects of thrombocytosis and lymphopenia in response to systemic stress and endothelial injury.[12,13] Meanwhile, although NLR also correlated with Killip and GRACE scores, its predictive power diminished in multivariate modeling, which may reflect its susceptibility to confounding by other clinical and hematological variables.

When in-hospital mortality was analyzed, only troponin I emerged as a statistically significant independent predictor ($p = 0.040$), despite NLR and PLR being significantly higher in the deceased group in bivariate analysis. These findings highlight troponin I's central role as a direct biomarker of myocardial injury and confirms its superior prognostic value in predicting short-term outcomes.[14,15] The diminished significance of NLR and PLR in multivariate analysis may reflect the complex interplay of inflammation with other determinants of mortality, such as age, hemodynamic instability, and renal dysfunction. Nevertheless, their significant associations in bivariate analysis suggest that these inflammatory markers should not be overlooked, especially as part of a broader clinical risk stratification model.

Furthermore, our regression model for GRACE score demonstrated a high explanatory power ($R^2 = 0.919$), affirming that the combination of inflammatory markers (NLR, PLR), myocardial damage markers (troponin I), and vital clinical parameters (age, systolic blood pressure, oxygen saturation) can collectively provide a comprehensive risk profile.[9,16] These findings align with the growing body of literature emphasizing the integration of hematological and clinical variables in AMI prognostication. For example, a study emphasized the role of NLR and PLR in augmenting traditional risk scores for AMI, advocating for their use as low-cost and readily available tools in emergency settings.[17]

However, this study is not without limitations. First, the relatively small sample size ($n = 47$) may limit the generalizability of our findings and reduce the statistical power, particularly in multivariate analyses. This is attributed to the purposive sampling approach, which only included patients with AMI who had no major comorbidities—such as diabetes mellitus, chronic kidney disease, or active infections—that could influence inflammatory marker levels. Second, our study did not include high-sensitivity C-reactive protein (hs-CRP) or other established inflammatory biomarkers that could provide a comparative reference, nor did it evaluate dynamic changes in NLR and PLR over the course of hospitalization. Future studies with larger, more diverse populations and prospective designs are warranted to validate and expand upon these findings.

CONCLUSIONS

Our study supports the utility of NLR and PLR as reflective markers of AMI severity and systemic inflammation. While PLR demonstrated stronger predictive value for Killip classification, both markers were significantly associated with higher GRACE scores and adverse clinical features. Although not independent predictors of mortality in multivariate models, their significant bivariate associations with death suggest potential value in risk stratification when used alongside established biomarkers like troponin I. Given their affordability and ease of measurement, incorporating NLR and PLR into routine clinical assessment may enhance early identification of high-risk AMI patients and guide more personalized therapeutic strategies.

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CONFLICT OF INTEREST

None.

REFERENCES

1. Naveed MA, Neppala S, Chigurupati HD, Ali A, Rehan MO, Fath A, et al. Acute myocardial infarction-related mortality among older adults (≥65 years) with malignancy in the U.S. from 1999 to 2020. International Journal of Cardiology Cardiovascular Risk and Prevention. 2025 Jun;25:200392.
2. De Luca L, Maggioni A Pietro, Cavallini C, Leonardi S, Lucci D, Sacco A, et al. Clinical profile and management of patients with acute myocardial infarction admitted to cardiac care units: The EYESHOT-2 registry. Int J Cardiol. 2025 Jan;418:132601.
3. Martha JW, Sihite TA, Listina D. The Difference in Accuracy Between Global Registry of Acute Coronary Events Score and Thrombolysis in Myocardial Infarction Score in Predicting In-Hospital Mortality of Acute ST-Elevation Myocardial Infarction Patients. Cardiol Res. 2021 Jun;12(3):177–85.
4. Liyis BG de, Ciaves AF, Intizam MH, Jusuf PJ, Artha IMJR. Hematological Biomarkers of Troponin, Neutrophil-to-Lymphocyte Ratio, and Monocyte-toLymphocyte Ratio Serve as Effective Predictive Indicators of High-Risk Mortality in Acute. Biomedicine (Taipei). 2023 Nov 27;13(4):32–43.
5. Pashapour P, Ghaffarii S, Separham A. The relationship of Neutrophil-to-Lymphocyte Ratio and Platelet-to-Lymphocyte Ratio in peripheral blood with ST-segment resolution and the clinical outcomes of STEMI patients receiving thrombolytic therapy. Romanian Journal of Internal Medicine. 2019 Mar 1;57(1):47–54.
6. Xin J, Liu Y, Zhang C, Wang Q. Predictive value of novel inflammatory markers combined with GRACE score for in-hospital outcome in patients with ST-segment elevation myocardial infarction: a retrospective observational study. BMJ Open. 2025 Apr 3;15(4):e096621.
7. Karadeniz F, Karadeniz Y, Altuntaş E. Systemic immune–inflammation index, and neutrophilto-lymphocyte and platelet-to-lymphocyte ratios can predict clinical outcomes in patients with acute coronary syndrome. Cardiovasc J Afr. 2024 Sep 1;35(2):18–24.
8. Tudurachi BS, Anghel L, Tudurachi A, Sascău RA, Stătescu C. Assessment of Inflammatory Hematological Ratios (NLR, PLR, MLR, LMR and Monocyte/HDL–Cholesterol Ratio) in Acute Myocardial Infarction and Particularities in Young Patients. Int J Mol Sci. 2023 Sep 21;24(18):14378.
9. Willim HA, Pakpahan SP, Suprihatin E, Agustyana A, Arilaksono DG, Suhardi S. Association Between Neutrophil-to-Lymphocyte Ratio and Platelet-to-Lymphocyte Ratio with Global Registry of Acute Coronary Events Risk Score in Acute Myocardial Infarction. Cermin Dunia Kedokteran. 2021 Jun 2;48(6):299–302.
10. Karaca G, Ekmekci A, Kimiaei A, Safaei S, Özer N, Tayyareci G. The Impact of the Neutrophil-to-Lymphocyte Ratio on In-Hospital Outcomes in Patients With Acute ST-Segment Elevation Myocardial Infarction. Cureus. 2024 Feb 18;
11. Wang H, Zulikaier T, Yumaierjiang B, Lyu S, He P. Platelet-To-Lymphocyte Ratio Efficiency in Predicting Major Adverse Cardiovascular Events After Percutaneous Coronary Intervention in Acute Coronary Syndromes: A Meta-Analysis. Rev Cardiovasc Med. 2025 May 21;26(5).

12. Oylumlu M, Oylumlu M, Arslan B, Polat N, Özbek M, Demir M, et al. Platelet-to-lymphocyte ratio is a predictor of long-term mortality in patients with acute coronary syndrome. Advances in Interventional Cardiology. 2020;16(2):170–6.
13. Wang H, Li L, Ma Y. Platelet-to-lymphocyte ratio a potential prognosticator in acute myocardial infarction: A prospective longitudinal study. Clin Cardiol. 2023 Jun 14;46(6):632–8.
14. Antman EM, Tanasijevic MJ, Thompson B, Schactman M, McCabe CH, Cannon CP, et al. Cardiac-Specific Troponin I Levels to Predict the Risk of Mortality in Patients with Acute Coronary Syndromes. New England Journal of Medicine. 1996 Oct 31;335(18):1342–9.
15. Firani NK, Prisilla J. Procalcitonin and Troponin-I as Predictor of Mortality in Acute Myocardial Infarction Patients. INDONESIAN JOURNAL OF CLINICAL PATHOLOGY AND MEDICAL LABORATORY. 2022 Jun 3;28(2):170–4.
16. Wikananda GDD, Diartoputra AYR. Association of Neutrophil-lymphocyte ratio and platelet lymphocyte ratio with GRACE risk score and high-sensitivity Troponin T in acute coronary syndrome patient. Intisari Sains Medis. 2023 Jun 7;14(2):489–92.
17. Liu J, Ao W, Zhou J, Luo P, Wang Q, Xiang D. The correlation between PLR-NLR and prognosis in acute myocardial infarction. Am J Transl Res. 2021;13(5):4892–9.