Journal of Society Medicine
Research & Review Articles on Diseases
Journal of Society Medicine. 2025; 4 (8)

Relationship Between Neutrophil-to-Lymphocyte Ratio and High-Density Lipoprotein with Major Cardiovascular Events in Acute Myocardial Infarction with ST-Segment Elevation Undergoing Primary Percutaneous Coronary Intervention at Adam Malik Hospital, Medan

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ARTICLE INFO

Article history:
Received
15 June 2025

Revised
29 July 2025

Accepted
31 August 2025

Manuscript ID:
JSOCMED-150625-48-3

Checked for Plagiarism: Yes

Language Editor: Rebecca

Editor-Chief:
Prof. AznanLelo, PhD

ABSTRACT

Introduction: Coronary artery calcium score (CACS) is a specific indicator of coronary atherosclerosis that plays a role in assessing the degree of calcification in atherosclerosis. Diastolic function is the first aspect of cardiac function to be impaired in ischemic heart disease. This study aims to determine the relationship between calcium scoring and diastolic dysfunction.

Methods: This analytical observational study with cross-sectional design evaluated the relationship between coronary artery calcium score (CACS) and left ventricular diastolic function in patients with stable CAD. Data were collected retrospectively from medical records at RSUP H. Adam Malik Medan during Nov 2023-Nov 2024. CACS was assessed using coronary CT scan, while left ventricular diastolic function was measured by echocardiography. Data analysis used chi-square test, Mann-Whitney U test, and ROC curve analysis to evaluate CACS threshold in predicting diastolic dysfunction.

Results: Among 158 analyzed samples, 113 patients had diastolic dysfunction. A calcium score ≥100 was found in 46.2% of patients, showing 1.318 times higher risk of diastolic dysfunction versus those with scores <100 (p = 0.006; 95% CI 1.083–1.605). ROC analysis showed CACS had moderate predictive ability for diastolic dysfunction with AUC of 0.647 (p = 0.004). A calcium score threshold of 45 had 65.5% sensitivity and 62.2% specificity in detecting diastolic dysfunction. Type 2 diabetes mellitus, urea, and creatinine levels were also significantly associated with diastolic dysfunction (p < 0.05).

Conclusion: Calcium score shows a significant relationship with diastolic dysfunction in stable CAD patients and can predict diastolic dysfunction in patients undergoing coronary CT scan.

Keywords

Coronary artery calcium score, Coronary computed tomography angiography, Diastolic dysfunction, Coronary artery disease, Echocardiography.

How to cite: Adam F, Hasan H, Haykal TB. Relationship Between Neutrophil-to-Lymphocyte Ratio and High-Density Lipoprotein with Major Cardiovascular Events in Acute Myocardial Infarction with ST-Segment Elevation Undergoing Primary Percutaneous Coronary Intervention at Adam Malik Hospital, Medan. *Journal of Society Medicine*. 2025; 4 (8): 253-259. **DOI: https://doi.org/10.71197/jsocmed.v4i8.231**

INTRODUCTION

Acute myocardial infarction (AMI) is a leading cause of morbidity and mortality worldwide. Globally, the prevalence of AMI is estimated to be 422.7 million cases, contributing to approximately 17.92 million deaths annually, with projections indicating a rise to 23.3 million by 2030 [1]. In 2018, an estimated 1.413 million patients were hospitalized for AMI [1]. In the Asia-Pacific region, AMI is the most prevalent form of coronary heart disease, with a mortality rate exceeding 5% [2]. In Indonesia, data from the Ministry of Health (2016) reported a 15 per 1,000 population increase in AMI prevalence in 2015, equating to approximately 2.784

million cases, making it the leading cardiovascular issue, with the highest prevalence in DKI Jakarta and West Java [3]. Furthermore, the Basic Health Research Survey (Riskesdas) in 2016 indicated that AMI accounted for 12.9% of the total mortality in Indonesia [3].

Among AMI subtypes, ST-segment elevation myocardial infarction (STEMI) is particularly associated with significant morbidity and mortality. The global prevalence of STEMI varies and is influenced by specific population factors. For patients with STEMI and symptom onset within 12 h, primary percutaneous coronary intervention (PPCI) is a class I recommendation for treatment [4]. However, STEMI can lead to complications, including major adverse cardiovascular events (MACE), which encompass all-cause mortality, non-fatal myocardial infarction, non-fatal ischemic stroke, and unplanned coronary revascularization within 30 days of diagnosis [5]. Early risk stratification is critical for predicting MACE in patients with STEMI undergoing PPCI.

The neutrophil-to-high-density lipoprotein (HDL) ratio is an emerging biomarker that has gained attention in medical research, particularly in the context of cardiovascular diseases and inflammatory conditions. Previous studies have demonstrated that the neutrophil-to-HDL-C ratio can serve as a risk stratification tool to predict long-term outcomes, such as heart failure or mortality, following STEMI, especially when combined with inflammatory markers such as troponin, creatine kinase, and other inflammatory indicators [6]. Additionally, a higher neutrophil-HDL ratio has been associated with a poorer prognosis in patients with STEMI, reflecting heightened inflammatory states and impaired protective lipid function, both of which may contribute to MACE [7]. The neutrophil-to-HDL-C ratio also represents the balance between inflammation and lipid-mediated protective effects, which are critical in the pathophysiology of STEMI. Monitoring this ratio may provide insights into the inflammatory response during the acute phase and recovery [8]. Based on this background, this study aimed to evaluate the relationship between the neutrophil-to-HDL ratio and MACE in patients with STEMI undergoing PPCI at Adam Malik Hospital, Medan.

METHODS

This observational analytical study employed a cross-sectional design to investigate the relationship between the neutrophil-to-high-density lipoprotein (HDL) ratio and major adverse cardiovascular events (MACE) during hospitalization in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PPCI) at our institution. Adam Malik Hospital, Medan, from February 2024 to May 2025. The study targeted patients diagnosed with STEMI, with the accessible population comprising those treated at the H. Adam Malik Hospital. Eligible participants were selected based on specific inclusion and exclusion criteria to ensure a homogeneous sample that reflected the study's objectives.

The sample size was determined using a formula for comparing two population proportions, accounting for the expected proportion of MACE in groups with high and low neutrophil-HDL ratios, a significance level of 0.05, and a power of 80 %. This calculation yielded a minimum of 90 patients per group (totaling 180 patients) to ensure adequate statistical power. The inclusion criteria encompassed patients with STEMI diagnosed within 12 h of symptom onset who underwent PPCI and had complete clinical and laboratory data in their medical records. The exclusion criteria included a history of stroke, prior myocardial infarction, cardiac arrest on admission, or previous revascularization (percutaneous coronary intervention or coronary artery bypass grafting) to minimize confounding factors.

Ethical clearance was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sumatera Utara, and research permission was granted by the Research and Development Unit of the H. Adam Malik Hospital. All participants provided written informed consent after receiving a detailed explanation of the study objectives, procedures, and protocols. Consecutive sampling was used to enroll all eligible STEMI patients undergoing PPCI until the required sample size was achieved. ST-Elevation Myocardial Infarction (STEMI) diagnosis adhered to the 2024 Indonesian Heart Association (PERKI) and European Society of Cardiology (ESC) guidelines, ensuring standardized case identification.

Data collection involved recording patient demographics (age, sex, body mass index), clinical history (hypertension, diabetes, smoking), and laboratory parameters (total cholesterol, triglyceride, low-density lipoprotein, HDL, and neutrophil counts). The neutrophil-to-HDL ratio was calculated by dividing the neutrophil count (%/mm³) by HDL (mg/dL) and categorized as ≥0.205 or <0.205 based on prior literature. For patients admitted before the study period, MACE and clinical outcomes were extracted from medical records, whereas those admitted during the study were monitored prospectively during hospitalization. MACE was defined as acute heart failure, recurrent myocardial infarction, cardiogenic shock, malignant arrhythmia, or cardiovascular-related death during hospitalization.

Categorical variables are reported as frequencies and percentages, while numerical variables are presented as mean ± standard deviation for normally distributed data or median (minimum–maximum) for non-normally distributed data. Normality was assessed using the Kolmogorov-Smirnov test for samples larger than 50 or the Shapiro-Wilk test for smaller ones. The association between the neutrophil-to-HDL-C ratio and MACE was evaluated using the chi-square test for normally distributed data and Fisher's exact test for non-normally distributed data. Significant variables from the bivariate analysis were further analyzed. All statistical analyses were conducted using SPSS version 19, with p-values <0.05 considered statistically significant.

RESULTS

This study enrolled 180 patients who met the inclusion and exclusion criteria. Data were analyzed and presented as frequencies and percentages for the categorical variables. Numerical variables are reported as mean ± standard deviation for normally distributed data or median (interquartile range) for non-normally distributed data. The demographic, clinical, echocardiographic, and laboratory characteristics of the study participants are presented in Table 1.

Table 1. Clinical Characteristics of Study Participants

Parameter	Total (n = 180)
Demographic and Clinical Data	
Male, n (%)	149 (82.8%)
Hypertension, n (%)	134 (74.4%)
Dyslipidemia, n (%)	7 (3.9%)
Smoking, n (%)	134 (74.4%)
Type 2 Diabetes Mellitus, n (%)	87 (48.3%)
Menopause, n (%)	25 (13.9%)
Age (years), mean ± SD	56.85 ± 8.44
GRACE Score, mean ± SD	101.23 ± 22.51
Body Weight (kg), median (range)	70.19 (43–104)
Height (cm), median (range)	165 (150–180)
BMI (kg/m²), median (range)	25.4 (17.3–37.78)
Systolic Blood Pressure (mmHg), median (range)	135.5 (62–230)
Diastolic Blood Pressure (mmHg), median (range)	80 (42–130)
Heart Rate (beats/min), mean ± SD	78.61 ± 19.83
Killip Class III–IV, n (%)	30 (16.7%)
Major Adverse Cardiovascular Events (MACE)	
Acute Heart Failure, n (%)	54 (30.0%)
Recurrent Myocardial Infarction, n (%)	12 (6.7%)
Cardiogenic Shock, n (%)	15 (8.3%)
Malignant Arrhythmia, n (%)	9 (5.0%)
Echocardiographic Parameters	
Normal Dimension, n (%)	35 (19.4%)
LV Concentric Remodeling, n (%)	34 (18.9%)
Concentric LVH, n (%)	85 (47.2%)
Eccentric LVH, n (%)	26 (14.4%)
Ejection Fraction (%), median (range)	55 (52–60)
TAPSE (mm), median (range)	20 (9–28)

Table 1. Continuous

Parameter	Total (n = 180)
Laboratory Parameters	
Hemoglobin (g/dL), mean ± SD	14.25 ± 1.44
Hematocrit (%), mean ± SD	41.55 ± 4.59
Leukocytes (cells/mm³), median (range)	12143 (8625–17260)
Platelets (cells/mm³), median (range)	222000 (158373–294000)
Urea (g/dL), median (range)	26 (13–103)
Creatinine (g/dL), mean ± SD	1.04 ± 0.28
Creatinine Clearance (mL/min), median (range)	77 (32–169)
Sodium (mEq/L), median (range)	143 (122–154)
Potassium (mEq/L), median (range)	4 (2.9–6.6)
Chloride (mEq/L), median (range)	105 (90–114)
Admission Blood Glucose (mg/dL), median (range)	151 (81–634)
Fasting Blood Glucose (mg/dL), median (range)	124 (37–424)
2-Hour Postprandial Glucose (mg/dL), median (range)	136.5 (79–439)
HbA1c (%), median (range)	6.05 (5.6–7.0)
Total Cholesterol (mg/dL), median (range)	248 (210–280)
HDL (mg/dL), median (range)	38.5 (25–68)
LDL (mg/dL), median (range)	170 (124–198)
Triglycerides (mg/dL), median (range)	189 (134–302)
Neutrophils (%/mm³), median (range)	10 (7–14.5)
Monocytes (%/mm³), median (range)	0.46 (0.1–0.72)
Lymphocytes (%/mm³), median (range)	1 (0.75–1.72)
Neutrophil-HDL Ratio, median (range)	0.19 (0.1–0.56)

Several parameters differed significantly between patients with and without MACE. Hypertension ($p = 0.045$), leukocyte count ($p < 0.0001$), platelet count ($p < 0.0001$), high-density lipoprotein (HDL) ($p < 0.0001$), low-density lipoprotein (LDL) ($p < 0.0001$), triglycerides ($p < 0.0001$), neutrophils ($p = 0.001$), monocytes ($p < 0.0001$), lymphocytes ($p < 0.0001$), and the neutrophil-HDL ratio ($p < 0.0001$) were significantly associated with These findings are detailed in Table 2.

Table 2. Comparison of Clinical Characteristics by MACE

Parameter	MACE (n = 90)	No MACE (n = 90)	p-value
Demographic and Clinical Data			
Male, n (%)	78 (86.7%)	71 (78.9%)	0.167a
Hypertension, n (%)	48 (53.4%)	42 (46.7%)	0.045a
Dyslipidemia, n (%)	1 (1.1%)	6 (6.7%)	0.118b
Smoking, n (%)	72 (80.0%)	62 (68.9%)	0.087a
Type 2 Diabetes Mellitus, n (%)	45 (50.0%)	42 (46.7%)	0.655a
Menopause, n (%)	11 (12.2%)	14 (15.6%)	0.518a
Age (years), mean ± SD	57.84 ± 4.3	56.4 ± 10.79	0.239c
GRACE Score, mean ± SD	99.22 ± 21.79	103.23 ± 23.18	0.233c
Body Weight (kg), median (range)	70 (50–94)	70 (43–104)	0.702d
Height (cm), median (range)	165 (150–180)	165 (150–170)	0.529d
BMI (kg/m²), median (range)	25.71 (17.3–37.79)	25.30 (18.61–37.29)	0.520d
Systolic BP (mmHg), median (range)	136 (73–227)	135 (62–230)	0.796d
Diastolic BP (mmHg), median (range)	80 (42–130)	80 (49–130)	0.660d
Heart Rate (beats/min), mean ± SD	78.01 ± 18.84	79.2 ± 20.87	0.689c
Laboratory Parameters			
Hemoglobin (g/dL), mean ± SD	14.39 ± 1.44	14.12 ± 1.44	0.212c
Hematocrit (%), mean ± SD	41.79 ± 4.35	41.31 ± 4.83	0.482c
Leukocytes (cells/mm³), median (range)	14800 (12300–17260)	9781 (8625–11987)	<0.0001d
Platelets (cells/mm³), median (range)	256000 (189000–294000)	189737 (158373–225000)	<0.0001d
Urea (g/dL), median (range)	27 (9–84)	28 (13–105)	0.804d
Creatinine (g/dL), mean ± SD	1.07 ± 0.26	1.00 ± 0.29	0.075c
Creatinine Clearance (mL/min),	75 (30–169)	75 (30–169)	0.709d

Note: a Fisher's exact test, b Mann-Whitney U test, c Independent T-test, d Mann-Whitney U test.

Table 2. Continuous

Parameter	MACE (n = 90)	No MACE (n = 90)	p-value
Sodium (mEq/L), median (range)	144 (129–149)	143 (122–154)	0.784d
Potassium (mEq/L), median (range)	4.0 (3.0–5.3)	4.1 (2.52–6.6)	0.486d
Chloride (mEq/L), median (range)	105 (98–114)	105 (90–114)	0.824d
Admission Blood Glucose (mg/dL)	154 (99–420)	151 (81–634)	0.661d
Fasting Blood Glucose (mg/dL)	120 (70–323)	123 (37–424)	0.222d
2-Hour Postprandial Glucose (mg/dL)	128 (82–339)	138 (79–439)	0.364d
HbA1c (%), median (range)	6.2 (5.6–7.0)	6.0 (5.6–7.0)	0.468d
Total Cholesterol (mg/dL)	248 (220–274)	247 (210–280)	<0.0001d
HDL (mg/dL), median (range)	32 (25–63)	48 (40–68)	<0.0001d
LDL (mg/dL), median (range)	156 (123–189)	153 (105–190)	<0.0001d
Triglycerides (mg/dL), median (range)	178 (152–320)	165 (152–320)	<0.0001d
Neutrophils (%/mm³), median (range)	12.8 (7–14.5)	8 (6–12)	0.001d
Monocytes (%/mm³), median (range)	0.47 (0.1–0.72)	0.45 (0.01–0.72)	<0.0001d
Lymphocytes (%/mm³), median (range)	1 (0.75–1.72)	0.98 (0.75–1.72)	<0.0001d
Neutrophil-HDL Ratio, median (range)	0.4 (0.12–0.56)	0.15 (0.1–0.19)	<0.0001d

Note: a Fisher's exact test, b Mann-Whitney U test, c Independent T-test, d Mann-Whitney U test.

Bivariate analysis was conducted to assess the association between the neutrophil-HDL ratio and MACE in STEMI patients undergoing PPCI. Among patients with a neutrophil-HDL ratio <0.205, 87 (96.7%) experienced MACE, whereas none (0%) were free of MACE. Conversely, among those with a neutrophil-HDL ratio ≥0.205, three (3.3%) experienced MACE, and 90 (100%) did not. This difference was statistically significant ($p < 0.0001$; OR = 31.00; 95% CI: 10.184–94.365; chi-square test), as shown in Table 3.

Table 3. Bivariate Analysis of Neutrophil-HDL Ratio and MACE

Neutrophil-HDL Ratio	MACE	No MACE	p-value	OR (95% CI)
<0.205	87 (96.7%)	0 (0%)	<0.0001*	31.00 (10.184–94.365)
≥0.205	3 (3.3%)	90 (100%)		

Noted: *Chi-square test

DISCUSSION

This study enrolled 180 patients with ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (PPCI), of whom 90 (50%) experienced major adverse cardiovascular events (MACE) during hospitalization. The observed MACE incidence aligns with previous reports, which range from 36.7% to 51.7% in hospital settings and are influenced by treatment modalities and follow-up duration [9-12]. Unlike prior studies that identified diabetes and reduced left ventricular ejection fraction (LVEF) as significant MACE predictors [2], our findings showed no statistically significant association with type 2 diabetes mellitus ($p = 0.655$) or LVEF ($p > 0.05$). This may be attributed to the relatively uniform blood glucose levels across patients with and without MACE and the preserved LVEF (median, 55%) in our cohort, suggesting less severe baseline cardiac dysfunction.

The significant predictors of MACE in this study included hypertension ($p = 0.045$), leukocyte count ($p < 0.0001$), platelet count ($p < 0.0001$), HDL ($p < 0.0001$), LDL ($p < 0.0001$), triglycerides ($p < 0.0001$), neutrophils ($p = 0.001$), monocytes ($p < 0.0001$), lymphocytes ($p < 0.0001$), and the neutrophil-HDL ratio ($p < 0.0001$). Hypertension was a notable risk factor, consistent with a study of 792 STEMI patients, in which antecedent hypertension was associated with a higher MACE incidence (8% vs. 3%, $p < 0.01$) and remained an independent predictor after multivariate adjustment (HR 3.42, 95% CI: 1.45–8.08, $p < 0.01$) [13-15]. The absence of significant differences in LVEF or myocardial salvage indices in our study mirrors these findings, indicating that the role of hypertension in MACE may be driven by systemic vascular stress rather than direct myocardial damage.

The neutrophil-to-HDL-C ratio emerged as a robust predictor of MACE, with a median value of 0.4 (0.12–0.56) in the MACE group compared to 0.15 (0.1–0.19) in the non-MACE group ($p < 0.0001$). Bivariate analysis revealed that 96.7% of patients with a ratio <0.205 experienced MACE, compared to only 3.3% of those with a ratio ≥0.205 ($p < 0.0001$, OR 31.00, 95% CI: 10.184–94.365). This aligns with a retrospective

analysis of 532 STEMI patients, where the MACE group had a significantly higher neutrophil-HDL ratio (10.93 vs. 8.13, p = 0.001), and a ratio >11.28 was associated with a 24.8% MACE incidence compared to 9.6% for lower ratios (p < 0.001) [4]. The predictive value of the ratio is further supported by a study reporting a 43% increased MACE risk per standard deviation increase in the ratio (HR 1.43, 95% CI: 1.25–1.64, p < 0.001) and an AUC of 0.722 for MACE prediction [5]. These findings underscore the role of the ratio as a marker of inflammation and lipid dysfunction, which are critical drivers of adverse outcomes in STEMI.

Lower HDL levels were significantly associated with MACE (median 32 mg/dL in MACE vs. 48 mg/dL in non-MACE, p < 0.0001), reinforcing HDL's protective role of HDL-C. A study of 1,109 STEMI patients with low HDL (<40 mg/dL) reported higher MACE and mortality rates than in 306 patients with normal HDL (p = 0.03 and p = 0.01, respectively) [16-18]. Similarly, elevated LDL and triglyceride levels in our MACE group (p < 0.0001) corroborate the findings from a cohort of 47,884 post-PCI patients, where LDL ≥100 mg/dL was associated with a 1.78-fold higher cardiovascular event risk (95% CI: 1.64–1.94) compared to LDL <70 mg/dL [7]. The heightened inflammatory state, marked by elevated leukocytes, neutrophils, monocytes, and lymphocytes, further supports the pro-inflammatory milieu in MACE, consistent with a meta-analysis reporting increased neutrophil counts and decreased lymphocytes post-ST elevation myocardial infarction as mediators of MACE (30-day MACE: 14.9%, 95% CI: 5.3–24.4, p < 0.001) [18-20]. The neutrophil-to-HDL ratio, hypertension, and lipid profile abnormalities are significant predictors of MACE in patients with STEMI undergoing PPCI. The strong association of the neutrophil-to-HDL-C ratio with MACE highlights its potential as a practical biomarker for risk stratification, enabling the early identification and targeted management of high-risk patients to reduce in-hospital morbidity and mortality.

CONCLUSION

This study identified a 50% incidence of major adverse cardiovascular events (MACE) in STEMI patients undergoing PPCI, with hypertension, leukocyte and platelet counts, lipid profile (HDL, LDL, and triglycerides), and inflammatory markers (neutrophils, monocytes, and lymphocytes) significantly associated with MACE. The neutrophil-HDL ratio was a strong predictor, with a significantly higher MACE rate in patients with a ratio <0.205 compared to those with a ratio ≥0.205, highlighting its potential as a valuable biomarker for risk stratification in clinical practice

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

FUNDING

None

COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

All authors contributed to the work, including data analysis, drafting, and reviewing the article. They approved the final version and were accountable for all aspects.

ACKNOWLEDGMENTS

None

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