

# Association of platelet to lymphocyte ratio with in-hospital major adverse cardiovascular events and the severity of coronary artery disease assessed by the Gensini score in patients with acute myocardial infarction

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## Abstract

**Background:** The platelet to lymphocyte ratio (PLR) has recently emerged as a potential inflammatory biomarker and has been shown to be significantly associated with atherosclerotic coronary artery disease (CAD). Therefore, we aimed to explore the association of PLR with in-hospital major adverse cardiovascular events (MACEs) and the severity of CAD assessed by the Gensini score (GS) in patients with acute myocardial infarction (AMI) undergoing coronary angiography.

**Methods:** A total of 502 patients with AMI consecutively treated at the Affiliated Hospital of Qingdao University (Qingdao, China) and underwent coronary angiography from August 2017 to December 2018 were recruited in this study. The demographic, clinical, angiographic characteristics, and laboratory parameters were collected. According to the presence of in-hospital MACEs, the included patients were divided into the MACE group ( $n = 81$ ) and the non-MACE group ( $n = 421$ ). Further, according to tertiles of the GS, the patients were classified into three groups: the low GS group ( $GS \leq 32$  points,  $n = 173$ ), medium GS group ( $32 < GS \leq 60$  points,  $n = 169$ ), and high GS group ( $60 < GS \leq 180$  points,  $n = 160$ ). The main statistical methods included Chi-squared test, non-parametric Mann-Whitney  $U$  test, Kruskal-Wallis  $H$  test, logistic regression, and receiver operating characteristic curves.

**Results:** The PLR in the MACE group was significantly higher than that in the non-MACE group (179.43 [132.84, 239.74] *vs.* 116.11 [87.98, 145.45],  $Z = -8.109$ ,  $P < 0.001$ ). Further, there were significant differences in PLR among the tertiles of GS (110.05 [84.57, 139.06] *vs.* 119.78 [98.44, 157.98] *vs.* 140.00 [102.27, 191.83],  $H = 19.524$ ,  $P < 0.001$ ). PLR was demonstrated to be an independent risk factor of in-hospital MACEs (odds ratio [OR]: 1.012, 95% confidential interval [CI]: 1.006–1.018,  $P < 0.001$ ) and severe CAD assessed by the GS (OR: 1.004, 95% CI: 1.002–1.009,  $P = 0.042$ ). The cutoff value of PLR for predicting the development of in-hospital MACEs was 151.28 with a sensitivity of 66.7% and a specificity of 78.1% (area under the curve [AUC]: 0.786, 95% CI: 0.730–0.842,  $P < 0.001$ ), and a PLR of 139.31 was also identified to be an effective cutoff point for detecting a high GS ( $>60$  points) with a sensitivity of 49.4% and a specificity of 69.6% (AUC: 0.611, 95% CI: 0.556–0.666,  $P < 0.001$ ).

**Conclusions:** PLR as a novel inflammatory marker is significantly and independently associated with the occurrence of in-hospital MACEs and the severity of CAD assessed by the GS in patients with AMI. As an easily available and inexpensive inflammatory indicator, PLR could be widely used as an efficient inflammatory biomarker for identifying high-risk patients and for individualizing targeted therapy to improve the prognosis of AMI.

**Keywords:** Platelet to lymphocyte ratio; Major cardiovascular adverse event; Gensini score; Myocardial infarction

## Introduction

Acute myocardial infarction (AMI) is mainly caused by the rupture of a coronary atherosclerotic plaque or thrombosis, which leads to acute complete occlusion of the vascular cavity and necrosis of myocardial cells. As an important type

of coronary artery disease (CAD), AMI is a leading cause of morbidity and mortality worldwide. Despite the improvements in reperfusion strategies, the outcome of patients with AMI remains unsatisfactory. Thus, early risk stratification and timely intervention measures are of great significance in improving the prognosis of patients with AMI.

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Present studies have shown that inflammatory processes play an important role in atherosclerosis. As a novel inflammatory marker, the platelet to lymphocyte ratio (PLR) has been reported to be strongly associated with CAD. PLR has been showed distinct predictive value on mortality or major adverse cardiovascular events (MACEs)<sup>[1,2]</sup> and the severity of CAD was likewise demonstrated to be related to PLR.<sup>[3,4]</sup> The aim of this study was to investigate the potential association between PLR and in-hospital MACEs as well as CAD severity assessed by the Gensini score (GS) in patients with AMI.

## Methods

### Ethical approval

The study protocol complied with the principles of the *Declaration of Helsinki* and was approved by the Ethics Committee of the Affiliated Hospital of Qingdao University (No. QYFY WZLL 25627). Because this was a retrospective study and the data analysis was performed anonymously, this study was exempt from informed consent from patients.

### Study population

A total of 597 consecutive patients diagnosed with AMI and admitted to the Affiliated Hospital of Qingdao University from August 2017 to December 2018 were successively recruited in this retrospective study. All patients met the diagnostic criteria for AMI, which were based on the fourth global definition of myocardial infarction.<sup>[5]</sup> All patients underwent coronary angiography with or without percutaneous coronary intervention (PCI) and were simultaneously treated with effective expectation.

Patients who were already using fibrinolytic agents before being referred for primary PCI; those with a history of previous AMI or coronary revascularization (either coronary artery bypass graft surgery or PCI); those complicated with other cardiac diseases (including severe congenital heart disease, valvular heart disease, and cardiomyopathy); those with any hematological disease including anemia, any systemic inflammatory disease or autoimmune disorder, malignancy, severe renal and/or hepatic insufficiency, or recent infection; those with use of non-steroidal anti-inflammatory drugs in the previous week or steroids (including steroid creams) in the previous 6 months were excluded. After the elimination according to the exclusion criteria, the remaining 502 patients were finally recruited in the present study.

### Clinical information

The patients' demographic data (age, sex, body mass index [BMI]), information on risk factors for CAD (smoking, hypertension, and diabetes mellitus [DM]) and information on previous medications were collected after admission.

BMI (weight [kg]/height squared [m<sup>2</sup>]) was calculated for all patients. Those who smoked one or more cigarettes per day were categorized as smokers. Hypertension was diagnosed based on repeated blood pressure measurements

of systolic blood pressure  $\geq 140$  mmHg and/or diastolic blood pressure  $\geq 90$  mmHg (at least two times in different environments) or use of antihypertensive drugs. Patients using oral anti-diabetic agents or insulin injections or with a fasting serum glucose level of  $\geq 126$  mg/dL (7.0 mmol/L) were considered to have DM. MACE refers to the occurrence of acute cardiac failure, severe arrhythmias (ventricular tachycardia/ventricular fibrillation and severe conduction block), non-fatal myocardial infarction, and death.

Fasting peripheral venous blood was collected in the morning of the second day after admission, and all data were obtained from the same blood sample. The neutrophil to lymphocyte ratio (NLR) was calculated by dividing the neutrophil count by lymphocyte count and the PLR was computed using the absolute platelet count divided by the absolute lymphocyte count.

### Angiographic examinations

Selective coronary angiography was performed in all included patients using the standard Judkin's technique. The severity of CAD was assessed based on the GS.<sup>[6]</sup> The coronary angiograms were analyzed and the GS was determined by two interventional cardiologists who were blinded to the clinical and laboratory data of the patients. In the case of a disagreement, a third interventional cardiologist who was unaware of the laboratory results and the nature of the study evaluated the coronary angiograms and determined the GS. The number of diseased vessels was also recorded, and significant left main coronary artery lesion was considered the equivalent of three-vessel CAD.

### Statistical analysis

Statistical analyses were performed using SPSS software, version 25.0 (IBM, New York, NY, USA). The normality of the distribution of continuous variables was tested using the Kolmogorov-Smirnov test and Q-Q plots. Non-normally distributed continuous variables are expressed as medians with interquartile range (quartiles 1–3). Categorical variables are presented as counts and percentages. Mann-Whitney *U* test was used to compare non-normally distributed continuous variables between the MACE and non-MACE groups, whereas comparisons of non-normally distributed continuous variables among the GS tertiles were performed with Kruskal-Wallis *H* test. Categorical variables were compared using the Chi-squared test. Univariate and multivariate regression analyses were performed to analyze the risk factors of in-hospital MACEs and severe CAD assessed by the GS. Receiver-operating characteristic (ROC) curve analysis was also performed to determine the optimal cutoff values of PLR for predicting in-hospital MACEs and high GS. All *P*-values were two-tailed, and a probability of  $< 0.05$  was considered statistically significant.

## Results

### Baseline characteristics

A total of 502 consecutive patients with AMI referred for coronary angiography from August 2017 to December

2018 at the Affiliated Hospital of Qingdao University were finally included in the analysis.

The baseline demographic, clinical, and angiographic characteristics of the MACE and non-MACE groups are summarized in Table 1. As shown in Table 1, the two groups were similar in terms of age, sex, BMI, smoking habit, hypertension, DM, and previous medication (including calcium channel blocker, angiotensin-converting enzyme inhibitor or angiotensin-receptor blocker, and beta-blocker) (all  $P > 0.05$ ). The percentages of death, left main disease, and three-vessel disease were significantly higher in the MACE group than those in the non-MACE

group ( $P < 0.001$ ,  $P < 0.001$ , and  $P = 0.031$ , respectively). The length of hospital stay (7 [5, 9] days *vs.* 5 [4, 7] days,  $Z = -2.433$ ,  $P = 0.015$ ), admission systolic pressure (121.5 [107.8, 133.5] mmHg *vs.* 125.0 [112.8, 139.0] mmHg,  $Z = -2.265$ ,  $P = 0.023$ ), ejection fraction (53.0 [41.0, 58.0] % *vs.* 60.0 [57.0, 62.0] %,  $Z = -7.155$ ,  $P < 0.001$ ), and GS (73.0 [44.8, 92.0] *vs.* 40.0 [22.0, 65.0],  $Z = -7.130$ ,  $P < 0.001$ ) were significantly different between the two study groups.

The laboratory parameters of the two patient groups are summarized in Table 2. No significant differences were observed in white blood cell (WBC) count, monocyte

**Table 1: Baseline demographic, clinical, and angiographic characteristics of the study population according to in-hospital major adverse cardiovascular events.**

Variables	MACE group (n = 81)	Non-MACE group (n = 421)	Statistics	P
Age (years)	64.0 (52.0, 72.3)	61.5 (52.0, 69.0)	-1.284*	0.199
Male	57 (70.4)	315 (74.8)	0.701†	0.402
BMI (kg/m <sup>2</sup> )	24.86 (22.60, 26.91)	25.39 (23.10, 27.68)	-1.247*	0.212
Smoking	37 (45.7)	213 (50.6)	0.656†	0.418
Hypertension	41 (50.6)	236 (56.1)	0.813†	0.367
DM	26 (32.1)	110 (26.1)	1.226†	0.268
Length of hospital stay (days)	7 (5, 9)	5 (4, 7)	-2.433*	0.015
SP (mmHg)	121.5 (107.8, 133.5)	125.0 (112.8, 139.0)	-2.265*	0.023
DP (mmHg)	73.0 (65.0, 82.3)	75.0 (67.0, 84.0)	-0.814*	0.416
EF (%)	53.0 (41.0, 58.0)	60.0 (57.0, 62.0)	-7.155*	<0.001
Death	8 (9.9)	3 (0.7)	26.617†	<0.001
Prior medication				
CCB	9 (11.1)	55 (13.1)	0.233†	0.629
Beta-blocker	5 (6.2)	15 (3.6)	1.210†	0.271
ACE-I/ARB	4 (4.9)	42 (10.0)	2.071†	0.150
Angiographic data				
GS	73.0 (44.8, 92.0)	40.0 (22.0, 65.0)	-7.130*	<0.001
Left main disease	10 (12.3)	8 (1.9)	21.439†	<0.001
One-vessel disease	19 (23.5)	126 (29.9)	1.385†	0.239
Two-vessel disease	20 (24.7)	130 (30.9)	1.241†	0.265
Three-vessel disease	42 (51.9)	164 (39.0)	4.670†	0.031

The data are shown as median (Q1, Q3) or n (%). \* Mann-Whitney U test. † Chi-squared test. MACE: Major adverse cardiovascular event; BMI: Body mass index; DM: Diabetes mellitus; SP: Systolic pressure; DP: Diastolic pressure; EF: Ejection fraction; CCB: Calcium channel blocker; ACE-I: Angiotensin-converting enzyme inhibitor; ARB: Angiotensin receptor blocker; GS: Gensini score.

**Table 2: Laboratory parameters of the study population according to in-hospital major adverse cardiovascular events.**

Variables	MACE group (n = 81)	Non-MACE group (n = 421)	Statistics*	P
WBC ( $\times 10^9/L$ )	8.50 (6.11, 11.83)	7.82 (6.48, 9.73)	-1.596	0.110
Neutrophil ( $\times 10^9/L$ )	6.88 (4.48, 10.14)	4.98 (3.73, 6.88)	-3.731	<0.001
Lymphocyte ( $\times 10^9/L$ )	1.30 (0.99, 1.73)	1.78 (1.45, 2.34)	-6.750	<0.001
Monocyte ( $\times 10^9/L$ )	0.54 (0.39, 0.78)	0.63 (0.48, 0.80)	-1.429	0.153
Platelet ( $\times 10^9/L$ )	224.00 (195.00, 273.00)	209.00 (176.00, 244.00)	-3.033	0.002
NLR	5.31 (3.13, 8.01)	2.77 (1.96, 4.34)	-6.954	<0.001
PLR	179.43 (132.84, 239.74)	116.11 (87.98, 145.45)	-8.109	<0.001
TC (mmol/L)	4.49 (3.81, 5.31)	4.37 (3.70, 5.04)	-0.592	0.554
HDL-C (mmol/L)	1.11 (0.95, 1.21)	1.07 (0.93, 1.24)	-0.735	0.463
LDL-C (mmol/L)	2.82 (2.31, 3.32)	2.61 (2.10, 3.14)	-1.348	0.178
LDL-C/HDL-C	2.54 (2.06, 3.02)	2.48 (1.92, 3.03)	-0.818	0.413
UA ( $\mu\text{mol/L}$ )	357.2 (279.80, 458.00)	328.30 (279.00, 400.00)	-1.574	0.115

The data are shown as median (Q1, Q3). \* Mann-Whitney U test. MACE: Major adverse cardiovascular event; WBC: White blood cell; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio; TC: Total cholesterol; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; UA: Uric acid.

count, total cholesterol level, high-density lipoprotein cholesterol (HDL-C) level, low-density lipoprotein cholesterol (LDL-C) level, LDL-C/HDL-C ratio, and uric acid level between the two groups (all  $P > 0.05$ ). The NLR and PLR values were significantly higher in the MACE group than in the non-MACE group (5.31 [3.13, 8.01] *vs.* 2.77 [1.96, 4.34],  $Z = -6.954$ ,  $P < 0.001$  and 179.43 [132.84, 239.74] *vs.* 116.11 [87.98, 145.45],  $Z = -8.109$ ,  $P < 0.001$ , respectively), as expected. In addition, the neutrophil count (6.88 [4.48, 10.14]  $\times 10^9/L$  *vs.* 4.98 [3.73, 6.88]  $\times 10^9/L$ ,  $Z = -3.731$ ,  $P < 0.001$ ) and platelet count (224.0 [195.0, 273.0]  $\times 10^9/L$  *vs.* 209.0 [176.0, 244.0]  $\times 10^9/L$ ,  $Z = -3.033$ ,  $P = 0.002$ ) were significantly higher in the MACE group than in the non-MACE group, and the lymphocyte count (1.30 [0.99, 1.73]  $\times 10^9/L$  *vs.* 1.78 [1.45, 2.34]  $\times 10^9/L$ ,  $Z = -6.750$ ,  $P < 0.001$ ) was significantly lower in the MACE group than in the non-MACE group.

According to the GS tertiles, the included patients were classified into three groups: the low GS group (GS  $\leq 32$  points,  $n = 173$ ), medium GS group (32 points  $< GS \leq 60$  points,  $n = 169$ ), and high GS group (60 points  $< GS \leq 180$  points,  $n = 160$ ), the low GS group and medium GS group were defined as non-high GS group. The baseline demographic, clinical, and laboratory data of the study population are shown in Table 3. Age, sex, BMI, smoking habit, hypertension, length of hospital stay, systolic pressure, diastolic pressure, and laboratory parameters (including WBC count, neutrophil count, monocyte count, and uric acid level) were similar among the groups classified according to the GS (all  $P > 0.05$ ). There were significant differences in the prevalence of DM and the ejection fraction among the three groups ( $P = 0.001$  and  $P < 0.001$ , respectively).

As demonstrated in Table 3, among the three groups based on the GS, there were also significant differences in NLR, PLR, lymphocyte count, platelet count, total cholesterol level, HDL-C level, LDL-C level, and LDL-C/HDL-C ratio ( $P = 0.003$ ,  $P < 0.001$ ,  $P = 0.003$ ,  $P = 0.018$ ,  $P = 0.047$ ,  $P = 0.030$ ,  $P = 0.006$ , and  $P < 0.001$ , respectively). More importantly, the NLR, PLR, HDL-C level, and LDL-C/HDL-C ratio were significantly higher in the high GS group than in the low and medium GS groups ( $P = 0.011$ ,  $P < 0.001$ ,  $P = 0.008$ , and  $P = 0.001$ , respectively), as shown in Table 4. Further, the lymphocyte count was significantly lower in the high GS group than in the other two groups ( $P = 0.001$ ). In addition, DM is more prevalent in the high GS group than in the low and medium GS groups (35.6% *vs.* 23.1%,  $\chi^2 = 8.658$ ,  $P = 0.003$ ).

**Univariate and multivariate analyses**

The risk factors of in-hospital MACEs and high GS were studied using univariate and multivariate logistic regression analyses. According to the univariate and multivariate logistic regression analysis [Tables 5 and 6], NLR, PLR, and GS were independent risk factors of in-hospital MACEs (odds ratio [OR]: 1.174, 95% confidential interval [CI]: 1.049–1.315,  $P = 0.005$ ; OR: 1.012, 95% CI: 1.006–1.018,  $P < 0.001$ ; and OR: 1.017, 95% CI: 1.009–1.025,  $P < 0.001$ , respectively). Further, the left main disease was also identified to be an independent and significant predictor of in-hospital MACEs (OR: 4.727, 95% CI: 1.514–14.759,  $P = 0.008$ ). In addition, as shown in Table 7, the results of the multivariate logistic regression analysis of variables predicting a high GS ( $> 60$  points) suggested that NLR and PLR were also independent risk factors of severe CAD, assessed using the GS after

**Table 3: Comparison of demographic, clinical, and laboratory characteristics among the low Gensini score tertile (Group 1), medium Gensini score tertile (Group 2), and high Gensini score tertile (Group 3).**

Variables	Group 1 (n = 173)	Group 2 (n = 169)	Group 3 (n = 160)	P (sta)	P1 (sta)	P2 (sta)	P3 (sta)
Age (years)	62.0 (52.0, 68.0)	62.0 (52.0, 70.0)	61.0 (52.0, 71.0)	0.644 (0.880 <sup>*</sup> )	0.358 (-0.919 <sup>‡</sup> )	0.518 (-0.646 <sup>‡</sup> )	0.837 (-0.205 <sup>‡</sup> )
Male	138 (79.8)	120 (71.0)	114 (71.3)	0.110 (4.417 <sup>†</sup> )	0.060 (3.543 <sup>†</sup> )	0.070 (3.277 <sup>†</sup> )	0.961 (0.002 <sup>†</sup> )
BMI (kg/m <sup>2</sup> )	24.80 (22.49, 27.34)	25.47 (22.49, 27.76)	25.39 (23.48, 27.76)	0.241 (2.849 <sup>†</sup> )	0.387 (-0.865 <sup>‡</sup> )	0.078 (-1.760 <sup>‡</sup> )	0.496 (-0.681 <sup>‡</sup> )
Smoking	94 (54.3)	84 (49.7)	72 (45.0)	0.235 (2.899 <sup>†</sup> )	0.391 (0.735 <sup>†</sup> )	0.089 (2.898 <sup>†</sup> )	0.393 (0.729 <sup>†</sup> )
Hypertension	87 (50.3)	96 (56.8)	94 (58.8)	0.262 (2.678 <sup>†</sup> )	0.227 (1.459 <sup>†</sup> )	0.121 (2.398 <sup>†</sup> )	0.721 (0.127 <sup>†</sup> )
DM	31 (17.9)	48 (28.4)	57 (35.6)	0.001 (13.415 <sup>†</sup> )	0.021 (5.289 <sup>†</sup> )	< 0.001 (13.403 <sup>†</sup> )	0.160 (1.973 <sup>†</sup> )
Length of hospital stay (days)	5 (4, 7)	6 (5, 7)	6 (5, 8)	0.075 (5.190 <sup>†</sup> )	0.414 (-0.817 <sup>‡</sup> )	0.024 (-2.258 <sup>‡</sup> )	0.150 (-1.440 <sup>‡</sup> )
SP (mmHg)	126.0 (115.0, 141.0)	124.0 (109.5, 136.0)	123.0 (111.3, 138.0)	0.296 (2.432 <sup>*</sup> )	0.177 (-1.351 <sup>‡</sup> )	0.181 (-1.337 <sup>‡</sup> )	0.987 (-0.016 <sup>‡</sup> )
DP (mmHg)	75.0 (68.0, 85.0)	75.0 (66.5, 83.0)	73.0 (65.3, 83.0)	0.222 (3.009 <sup>*</sup> )	0.340 (-0.955 <sup>‡</sup> )	0.081 (-1.745 <sup>‡</sup> )	0.455 (-0.747 <sup>‡</sup> )
EF (%)	60.0 (58.5, 62.0)	59.0 (56.0, 62.0)	58 (52.0, 60.0)	< 0.001 (49.862 <sup>†</sup> )	0.001 (-3.248 <sup>‡</sup> )	< 0.001 (-7.064 <sup>‡</sup> )	< 0.001 (-3.775 <sup>‡</sup> )
Laboratory data							
WBC ( $\times 10^9/L$ )	7.67 (6.18, 10.20)	8.16 (6.68, 10.45)	8.21 (6.69, 10.52)	0.255 (2.732 <sup>*</sup> )	0.104 (-1.624 <sup>‡</sup> )	0.282 (-1.077 <sup>‡</sup> )	0.617 (-0.500 <sup>‡</sup> )
Neutrophil ( $\times 10^9/L$ )	4.78 (3.70, 7.45)	5.61 (4.12, 7.76)	5.66 (4.08, 7.39)	0.071 (5.288 <sup>*</sup> )	0.027 (-2.207 <sup>‡</sup> )	0.097 (-1.658 <sup>‡</sup> )	0.641 (-0.467 <sup>‡</sup> )
Lymphocyte ( $\times 10^9/L$ )	1.79 (1.45, 2.40)	1.77 (1.43, 2.30)	1.63 (1.14, 1.99)	0.003 (11.720 <sup>†</sup> )	0.407 (-0.830 <sup>‡</sup> )	0.001 (-3.180 <sup>‡</sup> )	0.008 (-2.634 <sup>‡</sup> )
Monocyte ( $\times 10^9/L$ )	0.59 (0.46, 0.79)	0.58 (0.47, 0.77)	0.59 (0.42, 0.79)	0.874 (0.270 <sup>*</sup> )	0.835 (-0.208 <sup>‡</sup> )	0.613 (-0.506 <sup>‡</sup> )	0.743 (-0.328 <sup>‡</sup> )
Platelet ( $\times 10^9/L$ )	197.00 (174.50, 230.50)	209.50 (179.00, 243.50)	220.00 (184.00, 256.50)	0.018 (7.992 <sup>†</sup> )	0.016 (-2.409 <sup>‡</sup> )	0.014 (-2.463 <sup>‡</sup> )	0.923 (-0.097 <sup>†</sup> )
NLR	2.61 (1.93, 4.18)	3.16 (2.13, 5.01)	3.41 (2.15, 6.00)	0.003 (11.951 <sup>†</sup> )	0.016 (-2.419 <sup>‡</sup> )	0.001 (-3.267 <sup>‡</sup> )	0.246 (-1.159 <sup>‡</sup> )
PLR	110.05 (84.57, 139.06)	119.78 (98.44, 157.98)	140.00 (102.27, 191.83)	< 0.001 (19.524 <sup>†</sup> )	0.012 (-2.512 <sup>‡</sup> )	< 0.001 (-4.265 <sup>‡</sup> )	0.028 (-2.203 <sup>‡</sup> )
TC (mmol/L)	4.41 (3.67, 4.94)	4.62 (3.85, 5.23)	4.29 (3.65, 5.08)	0.047 (6.122 <sup>*</sup> )	0.013 (-2.479 <sup>‡</sup> )	0.135 (-1.495 <sup>‡</sup> )	0.415 (-0.816 <sup>‡</sup> )
HDL-C (mmol/L)	1.12 (0.95, 1.25)	1.12 (0.94, 1.29)	1.03 (0.93, 1.19)	0.030 (6.981 <sup>*</sup> )	0.863 (6.981 <sup>*</sup> )	0.023 (-2.273 <sup>‡</sup> )	0.020 (-2.320 <sup>‡</sup> )
LDL-C (mmol/L)	2.49 (2.09, 3.06)	2.82 (2.29, 3.22)	2.58 (2.13, 3.21)	0.006 (10.082 <sup>†</sup> )	0.003 (-2.930 <sup>‡</sup> )	0.014 (-2.465 <sup>‡</sup> )	0.618 (-0.498 <sup>‡</sup> )
LDL-C/HDL-C	2.23 (1.85, 2.81)	2.50 (2.01, 3.13)	2.59 (2.08, 3.12)	< 0.001 (18.116 <sup>†</sup> )	0.009 (-2.614 <sup>‡</sup> )	< 0.001 (-4.205 <sup>‡</sup> )	0.114 (-1.579 <sup>‡</sup> )
UA ( $\mu\text{mol/L}$ )	327.30 (277.60, 386.60)	329.00 (274.50, 402.50)	339.00 (282.00, 414.00)	0.357 (2.059 <sup>*</sup> )	0.700 (-0.386 <sup>‡</sup> )	0.154 (-1.427 <sup>‡</sup> )	0.342 (-0.949 <sup>‡</sup> )

The data are shown as median (Q1, Q3) or  $n$  (%). <sup>\*</sup> Kruskal-Wallis  $H$  test. <sup>†</sup> Chi-squared test. <sup>‡</sup> Mann-Whitney  $U$  test.  $P$ :  $P$  value for trend; P1: Group 1 *vs.* group 2; P2: Group 1 *vs.* group 3; P3: Group 2 *vs.* group 3; sta: Statistics; BMI: Body mass index; DM: Diabetes mellitus; SP: Systolic pressure; DP: Diastolic pressure; EF: Ejection fraction; WBC: White blood cell; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio; TC: Total cholesterol; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; UA: Uric acid.

**Table 4: Comparison of diabetes mellitus and laboratory parameters between the high Gensini score group (GS > 60 points) and non-high Gensini score group (GS ≤ 60 points).**

Variables	High GS group (n = 160)	Non-high GS group (n = 342)	Statistics	P
DM	57 (35.6)	79 (23.1)	8.658*	0.003
Lymphocyte (×10 <sup>9</sup> /L)	1.63 (1.14, 1.99)	1.76 (1.44, 2.33)	-3.343 <sup>†</sup>	0.001
Platelet (×10 <sup>9</sup> /L)	220.0 (184.0, 256.5)	206.0 (178.0, 244.0)	-1.483 <sup>†</sup>	0.138
NLR	3.41 (2.15, 6.00)	2.81 (2.00, 4.73)	-2.557 <sup>†</sup>	0.011
PLR	140.0 (102.27, 191.83)	119.08 (90.40, 151.19)	-3.726 <sup>†</sup>	<0.001
TC (mmol/L)	4.29 (3.65, 5.08)	4.44 (3.74, 5.04)	-0.411 <sup>†</sup>	0.681
HDL-C (mmol/L)	1.03 (0.93, 1.19)	1.12 (0.95, 1.28)	-2.638 <sup>†</sup>	0.008
LDL-C (mmol/L)	2.58 (2.13, 3.21)	2.65 (2.16, 3.17)	-1.156 <sup>†</sup>	0.248
LDL-C/HDL-C	2.59 (2.08, 3.12)	2.43 (1.87, 2.99)	-3.354 <sup>†</sup>	0.001

The data are shown as *n* (%) or median (Q1, Q3). \* Chi-squared test. <sup>†</sup> Mann-Whitney *U* test. GS: Gensini score; DM: Diabetes mellitus; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio; TC: Total cholesterol; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol.

**Table 5: Univariate logistic regression analysis of variables on in-hospital major adverse cardiovascular events.**

Variables	OR (95% CI)	P
Age (years)	1.015 (0.994–1.035)	0.158
Male	0.799 (0.473–1.351)	0.403
DM	1.337 (0.799–2.236)	0.269
GS	1.022 (1.015–1.029)	<0.001
Left main disease	7.271 (2.775–19.051)	<0.001
Three-vessel disease	1.688 (1.047–2.721)	0.032
Neutrophil (×10 <sup>9</sup> /L)	1.072 (1.004–1.145)	0.038
Lymphocyte (×10 <sup>9</sup> /L)	0.213 (0.127–0.357)	<0.001
Platelet (×10 <sup>9</sup> /L)	1.006 (1.002–1.009)	0.002
NLR	1.377 (1.260–1.505)	<0.001
PLR	1.017 (1.012–1.021)	<0.001

OR: Odds ratio; CI: Confidence interval; DM: Diabetes mellitus; GS: Gensini score; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio.

**Table 6: Multivariate logistic regression analysis of selected variables on in-hospital major adverse cardiovascular events.**

Variables	OR (95% CI)	P
GS	1.017 (1.009–1.025)	<0.001
Left main disease	4.727 (1.514–14.759)	0.008
NLR	1.174 (1.049–1.315)	0.005
PLR	1.012 (1.006–1.018)	<0.001

OR: Odds ratio; CI: Confidence interval; GS: Gensini score; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio.

adjusting for other factors, in patients with AMI (OR: 1.102, 95% CI: 1.005–1.208, *P* = 0.039 and OR: 1.004, 95% CI: 1.002–1.009, *P* = 0.042, respectively). Moreover, DM and LDL-C/HDL-C ratio were also identified to be independent risk factors of a high GS (OR: 1.819, 95% CI: 1.180–2.802, *P* = 0.007 and OR: 1.344, 95% CI: 1.052–1.717, *P* = 0.018, respectively).

### ROC curve analysis

Figures 1 and 2 present the findings of the ROC curve analysis of NLR and PLR for predicting in-hospital MACEs and a high GS. The cutoff value of NLR for

predicting the development of in-hospital MACEs was 5.05 with a sensitivity of 54.3% and a specificity of 82.4% (area under the curve [AUC]: 0.744, 95% CI: 0.685–0.803, *P* < 0.001). The cutoff value of PLR for predicting the occurrence of in-hospital MACEs was 151.28 with a sensitivity of 66.7% and specificity of 78.1% (AUC: 0.786, 95% CI: 0.730–0.842, *P* < 0.001). As shown in Table 8, an NLR of 3.32 was identified to be an effective cutoff point for detecting a high GS (>60 points) with a sensitivity of 54.5% and a specificity of 59.9% (AUC: 0.571, 95% CI: 0.516–0.627, *P* = 0.011), and a PLR of 139.31 was also identified to be a sufficient cutoff point for detecting a high GS (>60 points) with a sensitivity of 49.4% and a specificity of 69.6% (AUC: 0.611, 95% CI: 0.556–0.666, *P* < 0.001). Further, the cutoff value of LDL-C/HDL-C for predicting a high GS (>60 points) was 2.01 with a sensitivity of 84.0% and a specificity of 31.8% (AUC: 0.594, 95% CI: 0.541–0.647, *P* < 0.001).

### Discussion

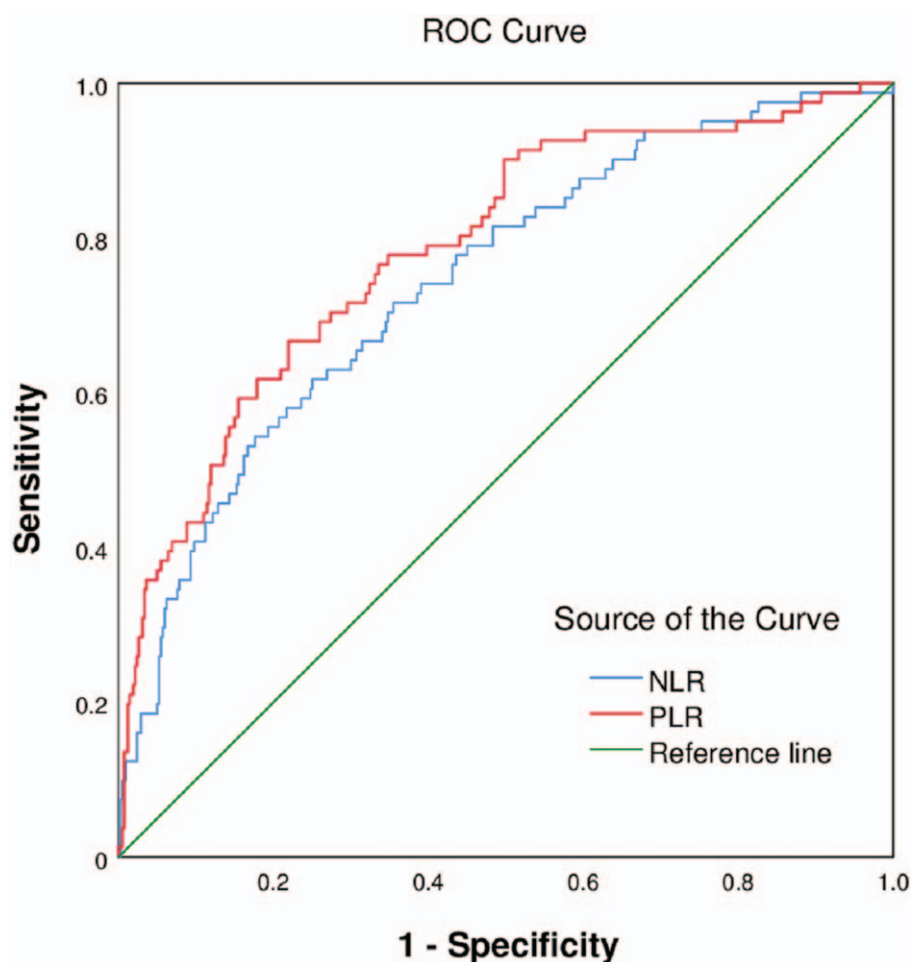
Atherosclerotic CAD is the leading cause of mortality and morbidity worldwide. Atherosclerosis is a systemic, lipid-driven immune-inflammatory disease.<sup>[7]</sup> The inflammatory process plays a pivotal role in the initiation and development of atherosclerosis.<sup>[8]</sup> Furthermore, inflammation has also been reported to be one of the main causes of DM, hyperlipidemia, and metabolic syndrome.<sup>[9]</sup> Thus, various inflammatory biomarkers have emerged as potential predictors for identifying individuals at high risk for the unsatisfactory outcomes of CAD.

Various risk factors of coronary atherosclerosis can lead to injury of arterial intima and vascular endothelium and then result in the adhesion, aggregation, and activation of platelets. The present study has confirmed that platelets play a pivotal role in the pathophysiology of atherosclerotic CAD. Platelets compounded with fibrin lead to the formation of a coronary thrombus.<sup>[7]</sup> Furthermore, activated platelets could initiate and promote the development of atherosclerotic lesions by inducing endothelial cells and leukocytes to release inflammatory substances that cause monocyte adhesion and transmigration, and accelerating the recruitment of leukocytes in circulation to injured vascular endothelial cells.<sup>[10,11]</sup> These interactions

**Table 7: Univariate and multivariate logistic regression analysis of selected variables on a high Gensini score (>60 points).**

Variables	Univariate logistic regression analysis		Multivariate logistic regression analysis	
	OR (95% CI)	P	OR (95% CI)	P
DM	1.842 (1.223–2.775)	0.003	1.819 (1.180–2.802)	0.007
NLR	1.173 (1.094–1.258)	<0.001	1.102 (1.005–1.208)	0.039
PLR	1.007 (1.004–1.010)	<0.001	1.004 (1.002–1.009)	0.042
LDL-C/HDL-C	1.434 (1.138–1.807)	0.002	1.344 (1.052–1.717)	0.018

OR: Odds ratio; CI: Confidence interval; DM: Diabetes mellitus; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio; HDL-C: High-density lipoprotein cholesterol level; LDL-C: Low-density lipoprotein cholesterol.

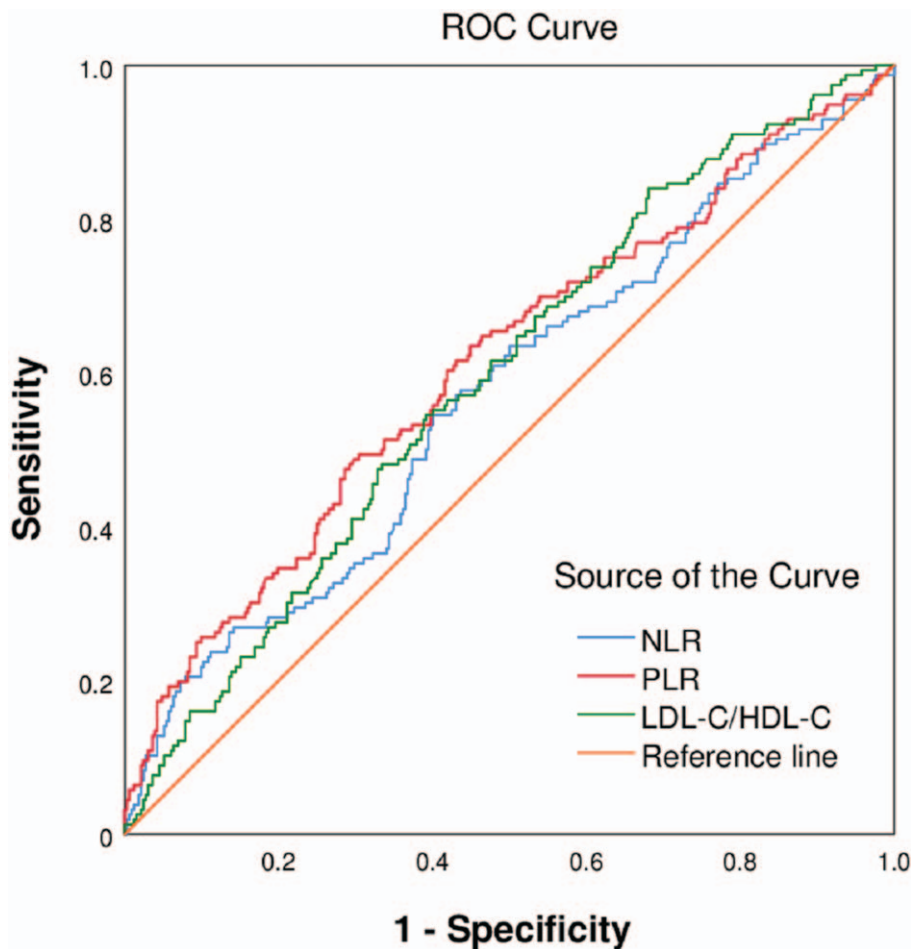


**Figure 1:** Receiver-operating characteristic (ROC) curve of neutrophil to lymphocyte ratio and platelet to lymphocyte ratio for predicting in-hospital major adverse cardiovascular events. The ROC curve could assess neutrophil to lymphocyte ratio as a satisfying marker of in-hospital MACEs, yielding a cutoff of 5.05, with a sensitivity of 0.543 and a specificity of 0.824 (AUC: 0.744, 95% CI: 0.685–0.803,  $P < 0.001$ ). Platelet to lymphocyte ratio yielding a cutoff of 151.28, with a sensitivity of 0.667 and a specificity of 0.781 (AUC: 0.786, 95% CI: 0.730–0.842,  $P < 0.001$ ). MACEs: Major adverse cardiovascular events; AUC: Area under the curve; CI: Confidential interval.

between platelets, leukocytes, and endothelial cells together lead to the destabilization of atherosclerotic plaques. Gary *et al*<sup>[12]</sup> reported that higher platelet counts may lead to the elevation of blood viscosity and promote inflammation. Moreover, increased platelet count was also found to be involved in the formation of atherosclerotic plaques.<sup>[13]</sup> Kaplan *et al*<sup>[14]</sup> demonstrated that an elevated platelet count is associated with cardiovascular adverse events. The present study has shown that the platelet count is significantly related to the incidence of coronary restenosis

and stent thrombosis.<sup>[15]</sup> In addition, platelet count has also been shown to be associated with short-term and long-term mortality in patients with ST-segment elevation myocardial infarction (STEMI), non-STEMI (NSTEMI), and unstable angina pectoris.<sup>[16,17]</sup>

Lymphocytes also play an important role in the atherosclerotic process,<sup>[18]</sup> which represents the regulatory pathway of the immune system. In AMI, lymphocytes infiltrate to the ischemic and reperfused myocardium and



**Figure 2:** Receiver-operating characteristic (ROC) curve of neutrophil to lymphocyte ratio, platelet to lymphocyte ratio, and LDL-C to HDL-C ratio for predicting a high Gensini score (>60 points). HDL-C: High-density lipoprotein cholesterol level; LDL-C: Low-density lipoprotein cholesterol.

**Table 8: Receiver-operating characteristic curve analysis of platelet to lymphocyte ratio and platelet to lymphocyte ratio and LDL-C to HDL-C ratio for predicting a high Gensini score (>60 points).**

Variable	AUC	95% CI	P	Sensitivity	Specificity	Cutoff
NLR	0.571	0.516–0.627	0.011	0.545	0.599	3.32
PLR	0.611	0.556–0.666	< 0.001	0.494	0.696	139.31
LDL-C/HDL-C	0.594	0.541–0.647	0.001	0.840	0.318	2.01

AUC: Area under the curve; CI: Confidence interval; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio; HDL-C: High-density lipoprotein cholesterol level; LDL-C: Low-density lipoprotein cholesterol.

express interleukin-10, which may play an important role in the transmigration of mononuclear cells and induce the expression of tissue inhibitor of metalloproteinase-1.<sup>[19]</sup> Owing to the decrease of the total number of circulating plasma lymphocytes and the CD4/CD8 ratio in patients with the acute coronary syndrome (ACS), the body’s anti-inflammatory ability declines significantly, leading to a short-term inflammatory status.<sup>[20]</sup> Ommen *et al*<sup>[21,22]</sup> reported that decreased lymphocyte count has been shown to be related to adverse events after AMI and advanced heart failure. In addition, low lymphocyte count has been reported to be associated with mechanical complications after myocardial infarction.<sup>[23]</sup>

Azab *et al*<sup>[2]</sup> found that PLR is associated with short-term and long-term mortality in patients after NSTEMI, and a similar relationship has also been reported for STEMI.<sup>[24]</sup> Thus, PLR is an important predictor of clinical cardiovascular events after AMI. Furthermore, PLR has also been demonstrated to be an independent risk factor for no-reflow, high SYNTAX scores, insufficient myocardial reperfusion, and in-hospital adverse events in patients with AMI undergoing primary PCI.<sup>[2,25]</sup> Kurtul *et al*<sup>[3]</sup> reported that in patients with ACS, PLR at admission is significantly associated with the severity and complexity of coronary atherosclerosis. And a high level of PLR may also be related to vulnerable plaque features of non-culprit lesions

in patients with ACS.<sup>[26]</sup> PLR seems to be a significant predictor of the long-term outcomes of percutaneous interventions, and is of great importance in early risk stratification and providing timely intervention in patients with AMI.<sup>[27,28]</sup> In a prospective study performed by Lee *et al*<sup>[29]</sup> elevated PLR was verified to be associated with long-term all-cause mortality in patients at high risk of CAD who undergo coronary angiography. Li *et al*<sup>[30]</sup> revealed that high PLR was an independent factor associated with all-cause mortality and cardiovascular events in patients with ACS in a meta-analysis, and PLR was proved to be a promising biomarker in predicting worse prognosis in patients with ACS.<sup>[31]</sup>

The present study further demonstrated the role of high PLR as an independent risk predictor of in-hospital MACEs and CAD severity in AMI patients undergoing coronary angiography, suggesting that PLR could be used as a predictive marker for the assessment of high-risk AMI and CAD severity assessed by the GS. Moreover, owing to its characteristics of being inexpensive and broadly available in daily clinical practice, PLR could be useful as a supplemental marker to traditional risk factors for identifying high-risk patients with AMI and may contribute to guiding the evaluation and individualized targeted therapy of patients with AMI.

This study has several limitations. First, this was a single-center retrospective study with a small study population. Second, we did not strictly restrict the interval between the occurrence of AMI or the timing of admission, and we analyzed only the admission PLR, PLR may change dynamically with different clinical outcomes during the course of the disease, the lack of follow-up data of PLR is a notable drawback. Third, the severity of CAD should be evaluated by other clinical characteristics such as the plaque vulnerability at the same time instead of basing on the GS only. Multi-center prospective studies are warranted to assess the relationship between the dynamic changes of this biomarker and the long-term prognosis of AMI. Further, the combined effects of PLR with other traditional predictors should also be studied.

In summary, the present study demonstrated that, in patients with AMI, PLR is associated with in-hospital MACEs and CAD severity assessed according to the GS. Complete blood count analysis is a routine, inexpensive, and broadly available method that may be useful for the timely identification of high-risk patients. The combination of PLR and other traditional markers might be of great significance in identifying high-risk patients and providing timely intervention strategies to improve the prognosis of AMI.

### Conflicts of interest

None.

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