Am J Transl Res 2025;17(3):2083-2093
www.ajtr.org /ISSN:1943-8141/AJTR0161841

Original Article

Prognostic value of mean platelet volume and platelet distribution width for the outcomes of patients with acute ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention

Baoqiang He, Qihan Sun

Department of Cardiology, Heilongjiang Province Hospital, Harbin 150036, Heilongjiang, China

Received November 7, 2024; Accepted January 24, 2025; Epub March 15, 2025; Published March 30, 2025

Abstract: Objective: To investigate the impact of mean platelet distribution width (PDW) and mean platelet volume (MPV) on the prognosis of patients with acute ST-segment elevation myocardial infarction (STEMI). Methods: A total of 435 patients with acute STEMI, admitted from November 2018 to March 2023, were included. Demographic and clinical data were collected from medical records. Patients were grouped as "High PDW" (n = 184), "Low PDW" (n = 251), "Low MPV" (n = 320) and "High MPV" (n = 115) based on the cut-off values. In addition, patients with elevated PDW and MPV were assigned a score of 2, those with elevation in either marker received a score of 1, and those without elevation in either marker received a score of 0. Based on these scores, patients were divided into three groups: group 0, group 1, and group 2. Clinical data of patients in different groups were compared. Multivariate logistic regression analysis and Cox proportional hazards models were used to identify independent predictors of in-hospital mortality and long-term major adverse cardiovascular events (MACEs), respectively. The Kaplan-Meier method was used to construct the cumulative survival curve for long-term MACEs. Results: Significant differences were observed between the High PDW and Low PDW groups in terms of gender distribution, diabetes, platelet count (PLT), PDW, MPV, D-dimer, left ventricular ejection fraction (LVEF), in-hospital mortality, MACEs, and follow-up duration (all $P < 0.05$). Similarly, significant differences were noted between the High MPV and Low MPV groups in gender distribution, PDW, MPV, D-dimer, SYNTAX score, in-hospital mortality, MACEs, and follow-up duration (all $P < 0.05$). Among the three groups with scores of 0, 1, and 2, significant differences were found in gender distribution, presence of diabetes, PLT, PDW, MPV, LVEF, SYNTAX score, in-hospital mortality, MACEs, and follow-up duration (all $P < 0.05$). Multivariate logistic regression analysis revealed that LVEF, SYNTAX score, D-dimer, creatinine, high PDW (in Model 1), and high MPV (in Model 2) were independent predictors of in-hospital death. In Model 3, LVEF, SYNTAX score, D-dimer, creatinine, GROUP(1), and GROUP(2) were independent predictors of in-hospital death. The Cox proportional hazards model showed that gender and high PDW value were independent predictors of MACEs in Model 1, high MPV value was an independent predictor of MACEs in Model 2, and GROUP(1) and GROUP(2) were independent predictors of MACEs in Model 3. Kaplan-Meier curves demonstrated significantly different survival rates based on PDW and MPV levels, as well as GROUP scores. Conclusion: PDW combined with MPV provides valuable prognostic information for the outcomes of patients with acute STEMI undergoing PCI.

Keywords: Mean platelet volume, platelet distribution width, mortality in hospital, long-term prognosis, acute ST-segment elevation myocardial infarction

Introduction

ST-segment elevation myocardial infarction (STEMI) remains a leading cause of morbidity and mortality worldwide [1]. Predictive factors for the extent and severity of coronary disease (CAD) and prognosis in acute coronary syndrome (ACS) contribute to a reduction in morbidity and mortality [2]. For this purpose, various scoring systems and laboratory parameters have been used in clinical practice. The SYNTAX score is one of the scoring systems used to assess the extent and severity of CAD. However, a significant limitation of these systems is the need for invasive methods such as coronary angiography for scoring [3]. Hence, there is a demand for an easily accessible, cost-effective, and non-invasive method to evaluate the sever-

https://doi.org/10.62347/0YHQ5176

ity of coronary disease and prognosis in ACS patients.

Platelets play a pivotal role in formation of intravascular thrombus, with dramatic changes in platelet activity being a major cause of ACS. Platelet reactivity is associated with the development and progression of atherosclerosis [4]. Following plaque rupture, platelet activation and thrombus formation lead to coronary artery occlusion [5]. Therefore, significant attention has been given to the pathogenesis of atherosclerosis and ACS. Mean platelet volume (MPV) is a potentially useful marker of platelet activity [6]. The circulating platelet population is heterogeneous: immature platelets are larger in size, contain more and denser granules, and are more active than mature platelets [7]. Previous studies have reported that MPV is elevated in patients with acute STEMI [8], and is associated with impaired angiographic reperfusion and poorer clinical outcome in STEMI patients [9].

Platelet activation is believed to induce morphologic changes, including alterations in shape and pseudopodia formation. Platelets with increased numbers and sizes of pseudopodia vary in size, possibly affecting platelet distribution width (PDW) [10]. PDW is the relative width of the platelet volume distribution and serves as an index of platelet heterogeneity [11]. In several studies examining the relationship between PDW and CAD [12-14], PDW was found to be an independent predictor of platelet activity. One study reported that PDW was associated with increased frequency of in-hospital stent thrombosis, long-term stent restenosis, and MACEs [15]. Moreover, a positive correlation was observed between PDW values and three-vessel disease. Similarly, PDW has been identified as an independent predictor of both in-hospital and long-term MACEs [16].

However, to date, no study has examined the combined effect of PDW and MPV on short-term mortality and long-term prognosis in STEMI patients undergoing PCI. Therefore, this study aims to investigate the correlation between the combined effect of PDW and MPV and STEMI severity in patients undergoing PCI. Furthermore, it aims to assess whether the combination of PDW and MPV provides a better predictive value than either marker alone for estimating short-term mortality or long-term prognosis in patients with acute STEMI undergoing PCI.

Methods

Patient selection

This retrospective cohort study involved 435 consecutively admitted patients diagnosed with acute STEMI between November 2018 and March 2023. All patients underwent percutaneous coronary intervention (PCI) within 12 hours of symptom onset. STEMI diagnosis was based on typical chest pain and new ST-segment elevation at the J point in two contiguous leads on electrocardiogram (≥0.2 mV in V1 through V3 and ≥0.1 mV in other leads) [17]. The study was conducted in compliance with the Declaration of Helsinki and was approved by the Human Ethics Committee of Heilongjiang Province Hospital. As a retrospective study involving no additional interventions or risks to patients, the Ethics Committee determined that informed consent was not required after anonymization of patient data.

Inclusion criteria: (1) Meeting the diagnostic criteria for STEMI; (2) Undergoing PCI within 12 hours of symptom onset; (3) No contraindications for PCI; (4) Availability of complete patient data. Exclusion criteria: (1) Hematological disorders (e.g., idiopathic thrombocytopenic purpura, aplastic anemia, or other diseases directly affecting platelet size and count); (2) End-stage renal disease; (3) Clinically diagnosed cancer; (4) Active or chronic inflammatory or autoimmune diseases; (5) Active infections; (6) Recent blood transfusion.

Intervention methods

All patients underwent emergency coronary angiography using the standard Judkins technique upon admission. A 300 mg chewable aspirin and a 600 mg loading dose of clopidogrel were administered before and at the time of coronary angiography. Angiography of the non-obstructed artery was performed first, and all patients received heparin (100 IU/kg) once the coronary anatomy was defined. The SYNTAX score was used to evaluate the severity of atherosclerosis, serving as an angiographic tool for grading the complexity of CAD. Each coronary lesion with a diameter stenosis of at least 50% in vessels ≥1.5 mm was scored using the SY-

NTAX score calculator 2.1 (http://www.syntaxscore.com) [18].

Data collection

Demographic and clinical data were collected from medical records, including age, gender, current smoking status, alcohol consumption, hypertension, and diabetes. Hypertension was defined as a history of hypertension and/or repeated systematic blood pressure measurements exceeding 140/90 mmHg. Diabetes was defined as a history of diabetes, a diagnosis of diabetes, or fasting blood glucose levels > 7.0 mmol/L (126 mg/dL) on two separate occasions, or at least one random blood glucose value exceeding 11.1 mmol/L (200 mg/dL) prior to the current admission.

Peripheral blood samples were collected from all participants at admission. The following parameters were measured: white blood cells (WBCs), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides, creatinine (CR), blood urea nitrogen (BUN), platelets (PLT), mean platelet volume (MPV), platelet distribution width (PDW), and D-dimer. Sample analysis was conducted using an automated hematology analyzer (Bayer ADVIA 2120, Bayer Diagnostics, Tarrytown, New York, USA) within 30 minutes of sampling, using complete blood count analysis to minimize time-dependent platelet swelling due to EDTA exposure.

Primary and secondary outcomes

The primary outcome was in-hospital mortality. Secondary outcomes included long-term MACEs, such as in-stent stenosis or stent thrombosis, non-fatal myocardial infarction, and cardiac-related death. Stent thrombosis was defined as complete occlusion confirmed by angiography. Non-fatal myocardial infarction was defined as recurrent chest pain and/or new electrocardiographic changes, accompanied by a 20% increase in cardiac biomarkers following the recurrent event.

Definition of group score

Receiver operating characteristic (ROC) curve analysis was used to determine the optimal cut-off values for PDW and MPV in predicting in-hospital mortality. Based on these cut-points, patients were classified as having either high or low values for each marker. A simplified scoring system was then developed using the data obtained upon admission, as detailed below: Patients with elevated PDW and MPV were assigned a score of 2, those with elevation in only one marker scored 1, and those without elevation in either marker scored 0. Based on these scores, patients were categorized into three groups: GROUP 0, GROUP 1, and GROUP 2.

Statistical analysis

All analyses were performed using SPSS 22.0 for Windows statistical software (SPSS Inc., Chicago, IL, USA). The following detailed methods were employed for each type of analysis:

1. Normality testing: The Kolmogorov-Smirnov test was utilized to assess the normality of distributions for continuous variables.

2. Comparison of continuous variables: For comparisons between two groups involving continuous variables, Student's t-test was applied. A two-tailed *P*-value threshold of < 0.05 was deemed statistically significant. Continuous variables are presented as the mean ± standard deviation.

3. Cut-off value determination: Receiver Operating Characteristic (ROC) curve analysis was performed to determine the optimal cut-off values for PDW and MPV in predicting in-hospital mortality. The optimal cut-off values were determined by maximizing Youden's index (sensitivity + specificity - 1).

4. Comparison of patient characteristics: Patient characteristics were compared across groups based on their scores. Parametric characteristics were analyzed using one-way ANOVA, whereas nonparametric characteristics were compared using the Kruskal-Wallis test. Categorical variables, summarized as percentages, were compared using the chi-square (χ^2) test.

5. Multivariate logistic regression and Cox proportional hazards models: To identify independent predictors for in-hospital mortality and long-term MACEs, multivariate logistic regression analysis and Cox proportional hazards models were employed, respectively. Specifically, Cox proportional hazard models were constructed to investigate the association between

group scores and long-term MACEs during the study period. For this purpose, univariate Cox models were initially run for each predictor variable, with MACEs as the outcome. Variables found to be significant ($P < 0.05$) in these univariate models were then included in a multivariate Cox model. The odds ratios (ORs) and their corresponding 95% confidence intervals (CIs) were calculated.

6. Survival analysis: The Kaplan-Meier method was used to construct the cumulative survival curve for long-term MACEs. These curves were compared across groups using the log-rank test. A *P*-value of < 0.05 was considered statistically significant for all tests.

Results

Patient grouping

After the follow-up period, 24 out of 435 patients died. Based on the mortality status, patients were divided into a deceased group and a survival group. Statistical analysis was conducted to compare the values of PDW and MPV between the two groups. The cut-off values for PDW and MPV, obtained through ROC curve analysis, were 14.12% and 10.80 fl (femtoliters), respectively. The areas under the curve for PDW and MPV were 0.767 ($P < 0.001$) and 0.702 ($P < 0.001$), respectively. Therefore, patients were categorized as follows: High PDW group (> 14.12; n = 184), Low PDW group (≤ 14.12; n = 251), High MPV group (> 10.80; n = 115), and Low MPV group (≤ 10.80; n = 320).

Comparison of baseline characteristics and laboratory findings between patients in the low PDW/MPV and high PDW/MPV groups

The High PDW group had higher age, a greater number of patients with diabetes, higher PDW, D-dimer, in-hospital mortality rates, more MACEs, and longer follow-up duration compared to the Low PDW group (all $P < 0.05$). In contrast, the High PDW group had lower PLT and LVEF than the Low PDW group (all $P < 0.05$). The High MPV group had higher PDW, MPV, D-dimer, SYNTAX score, in-hospital mortality rates, and more MACEs compared to the Low MPV group (all $P < 0.05$). However, the High MPV group had a lower age and shorter follow-up duration than the Low MPV group ($P < 0.05$). Detailed information is provided in **Table 1**.

Comparison of baseline characteristics and laboratory findings in patients with GROUP scores (0, 1, 2)

In terms of age, the GROUP(2) patients were older than the GROUP(1) and GROUP(0) patients. Regarding gender, the number of male patients was highest in the GROUP(0), followed by the GROUP(1), and lowest in the GROUP(2). In terms of the number of patients with diabetes, the GROUP(1) had the highest proportion of diabetic patients, followed by GROUP(0) and GROUP(2). For PLT and LVEF, the GROUP(0) had higher PLT and LVEF than both the GROUP(1) and GROUP(2). In terms of PDW, MPV, SYNTAX score, and in-hospital mortalities, the GROUP(2) had significantly higher values for these parameters than the GROUP(1) and GROUP(0). Regarding follow-up duration, the GROUP(1) group had the longest follow-up duration, followed by the GROUP(0) and the GROUP(2). Detailed information is presented in **Table 2**.

Multivariate logistic regression analysis for in-hospital death

After adjusting for various clinical factors, including age, male gender, smoking, alcohol consumption, hypertension, diabetes, WBC, PLT, D-dimer, BUN, CR, TG, TC, HDL, LDL, LVEF, and SYNTAX score, multivariate logistic regression analysis revealed that LVEF, SYNTAX score, D-dimer, creatinine, and High PDW (in Model 1), as well as High MPV (in Model 2), were independent predictors of in-hospital death in Models 1 and 2, respectively. In Model 3, LVEF, SYNTAX score, D-dimer, creatinine, GROUP(1), and GROUP(2) were identified as independent predictors of in-hospital death (**Table 3**).

Effects of multiple variables on long-term MACEs in multivariate Cox regression analysis

After adjusting for various clinical factors, the Cox proportional hazards model revealed that gender and High PDW were independent predictors of MACEs in Model 1, High MPV was an independent predictor in Model 2, and GROUP(1) and GROUP(2) were independent predictors in Model 3 (**Table 4**). Kaplan-Meier curves indicated significantly different survival rates based on PDW and MPV levels, as well as the GROUP scores (**Figure 1**).

Table 1. The baseline characteristics and laboratory findings of patients in the low and high PDW/MPV groups

	Low PDW ≤ 14.12 (*n* = 251)	High PDW > 14.12 (*n* = 184)	*P* value	Low MPV ≤ 10.80 (*n* = 320)	High MPV > 10.80 (*n* = 115)	*P* value
Age (years)	57.00±12.00	60.00±12.00	0.013	57.00±12.00	61.00±12.00	0.002
Male, n (%)	215 (85.66)	146 (79.34)	0.084	271 (84.69)	90 (78.26)	0.116
Smoking, n (%)	114 (45.42)	86 (46.74)	0.785	147 (45.94)	53 (46.09)	0.978
Alcohol consumption, n (%)	113 (45.02)	87 (47.28)	0.640	149 (46.56)	51 (44.35)	0.683
Hypertension, n (%)	116 (46.22)	97 (52.72)	0.180	148 (46.25)	65 (56.52)	0.059
Diabetes, n (%)	40 (15.94)	52 (28.26)	0.002	67 (20.94)	25 (21.74)	0.857
Admission blood parameters						
WBC (×10^9/L)	11.0±3.6	10.9±3.6	0.764	10.9±3.5	11.1±3.7	0.605
PLT (×10^9/L)	228±66	200±53	< 0.001	219±64	206±54	0.053
PDW (%)	11.54±1.36	17.63±2.59	< 0.001	13.26±3.13	16.50±3.76	< 0.001
MPV (fl)	10.04±0.80	10.36±1.36	0.002	9.72±0.84	11.44±0.52	< 0.001
BUN (mmol/L)	5.34±1.76	5.19±2.12	0.421	5.32±1.93	5.16±1.89	0.444
Creatinine (mmol/L)	75.35±18.60	77.16±22.35	0.358	75.91±19.30	76.67±22.80	0.731
TG (mmol/L)	2.17±1.45	2.43±1.68	0.085	2.58±0.91	2.61±1.25	0.785
TC (mmol/L)	4.55±1.16	4.46±0.91	0.383	4.54±1.09	4.43±0.97	0.340
HDL (mmol/L)	0.97±0.23	0.93±0.21	0.064	0.95±0.23	0.96±0.20	0.680
LDL (mmol/L)	2.89±0.77	2.87±0.70	0.781	2.91±0.76	2.83±0.70	0.324
D-dimer (ug/l)	127 (77,210)	147 (102,328)	0.001	130 (78,218)	147 (102,351)	0.019
Medication in hospital						
Aspirin, n (%)	243 (96.81)	182 (98.91)	0.149	312 (97.50)	113 (98.26)	0.641
Clopidogrel, n (%)	237 (94.42)	179 (97.28)	0.149	304 (95.00)	112 (97.39)	0.282
Statins, n (%)	241 (96.02)	181 (98.37)	0.154	309 (96.56)	113 (98.26)	0.359
ACEI, n (%)	157 (62.55)	125 (67.93)	0.245	204 (63.75)	78 (67.83)	0.432
ARB, n (%)	53 (21.12)	35 (19.02)	0.591	67 (20.94)	21 (18.26)	0.540
β-Blockers, n (%)	211 (84.06)	158 (85.87)	0.604	267 (83.44)	102 (88.70)	0.178
LVEF (%)	60.00±6.00	58.00±7.00	0.002	59.00±6.00	58.00±7.00	0.856
SYNTAX score	17.20±7.90	18.30±8.60	0.168	16.70±7.80	20.20±8.70	< 0.001
Mortalities In-hospital, n (%)	4 (1.59)	20 (10.87)	< 0.001	9 (2.81)	15 (13.04)	< 0.001
MACEs, n (%)	8 (3.18)	15 (8.15)	0.022	10 (3.13)	13 (11.30)	0.001
Follow-up duration (days)	540.00±360.00	858.00±565.00	< 0.001	677.00±463.00	567.00±340.00	0.020

Notes: WBC, white blood cell; PLT, platelet; PDW, platelet distribution width; MPV, mean platelet volume; BUN, blood urea nitrogen; TG, triglyceride; TC, total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; LVEF, left ventricular ejection fraction.

Discussion

Acute STEMI is a critical cardiovascular event that typically requires immediate intervention. Platelet activation markers, such as PDW and MPV, are gaining increased recognition for their potential role in risk stratification of STEMI patients. This study aimed to assess the predictive value of PDW, MPV, and the GROUP score in a cohort of 435 STEMI patients undergoing PCI.

Significant differences were observed between the high PDW/MPV group and the low PDW/MPV group in terms of the incidence of diabetes, LVEF, and D-dimer levels. These differences may be driven by several factors. First, high PDW/MPV may reflect heightened platelet activation and inflammatory response, which are commonly observed in both diabetes and cardiovascular diseases. Second, increased PDW and MPV may be associated with endothelial dysfunction, leading to thrombus formation in the vasculature and a subsequent decline in LVEF [19, 20]. In addition, the rise in D-dimer levels, often associated with a hypercoagulable state, further corroborates these findings and serves as a known adverse prognostic factor in STEMI patients. Our results align with previous studies that have linked elevated PDW and MPV to poor outcomes in STEMI patients [21]. Furthermore, the GROUP score, which combines PDW and MPV, was also found to be a significant predictor of in-hospital mortality and MACEs. The utility of the GROUP score may lie in its ability to provide a more comprehensive

Table 2. The baseline characteristics and laboratory findings of patients with difference GROUP scores

Variable	GROUP(0) (*n* = 211)	GROUP(1) (*n* = 149)	GROUP(2) (*n* = 75)	*P*-Value
Age (years)	57.00±12.00	58.00±12.00	63.00±12.00	0.001
Male, n (%)	180 (85.31)	126 (84.56)	55 (73.33)	0.049
Smoking, n (%)	95 (45.02)	71 (47.65)	34 (45.33)	0.880
Alcohol consumption, n (%)	95 (45.02)	72 (48.32)	33 (44.00)	0.770
Hypertension, n (%)	95 (45.02)	74 (49.66)	44 (58.67)	0.125
Diabetes, n (%)	34 (16.11)	39 (26.17)	19 (25.33)	0.044
Admission blood parameters				
WBC (×10^9/l)	11.00±3.6	10.8±3.40	11.20±4.00	0.325
PLT (×10^9/l)	229.00±69.00	207.00±52.00	198.00±55.00	0.028
PDW (%)	11.35±1.26	15.77±2.82	18.61±2.79	< 0.001
MPV (fL)	9.81±0.64	10.00±1.24	11.19±3.96	< 0.001
BUN (mmol/L)	5.40±1.75	5.13±2.13	5.23±1.94	0.411
Creatinine (mmol/L)	75.48±19.11	76.18±18.75	77.76±25.79	0.705
TG (mmol/L)	2.18±1.52	2.33±1.95	1.82±1.39	0.096
TC (mmol/L)	4.57±1.18	4.47±0.93	4.43±0.94	0.519
HDL (mmol/L)	0.97±0.24	0.93±0.20	0.95±0.22	0.810
LDL (mmol/L)	2.90±0.78	2.89±0.73	2.81±0.67	0.657
D-dimer (ug/L)	125 (76,205)	136 (90,258)	162 (105,440)	0.064
Medication in hospital				
Aspirin, n (%)	204 (96.68)	147 (98.66)	74 (98.67)	0.390
Clopidogrel, n (%)	198 (93.84)	145 (97.32)	73 (97.33)	0.208
Statins, n (%)	203 (96.21)	144 (96.64)	75 (100)	0.242
ACEI, n (%)	132 (62.56)	97 (65.10)	53 (70.67)	0.451
ARB, n (%)	44 (20.85)	32 (21.48)	12 (16.00)	0.601
β-Blockers, n (%)	173 (81.99)	132 (88.59)	64 (85.33)	0.227
LVEF (%)	60.00±6.00	59.00±6.00	58.00±7.00	0.042
SYNTAX score	16.50±7.90	18.1±7.70	19.90±9.50	0.006
Mortalities In-hospital, n (%)	2 (0.95)	9 (6.04)	13 (17.33)	< 0.001
MACEs, n (%)	4 (1.90)	10 (6.71)	9 (12.00)	0.002
Follow-up duration (days)	541±362	831±515	739±606	< 0.001

Notes: WBC, white blood cell; PLT, platelet; PDW, platelet distribution width; MPV, mean platelet volume; BUN, blood urea nitrogen; TG, Triglyceride; TC, total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; LVEF, left ventricular ejection fraction.

evaluation of platelet activation and size, offering a more accurate identification of high-risk patients [22]. By combining multiple risk factors, the GROUP score offers clinicians a clearer understanding of patient risk, facilitating the development of more personalized and targeted treatment plans.

After adjusting for various clinical factors, LVEF, SYNTAX score, D-dimer, creatinine, as well as PDW and MPV, were confirmed as independent predictors for in-hospital mortality. The rationale for this may lie in the following: a reduced LVEF typically reflects impaired cardiac pumping function, while an increased SYNTAX score is associated with the complexity of CAD [23]. Elevated D-dimer and creatinine may indicate thrombosis and renal insufficiency, respectively, both of which are established adverse prognostic factors in STEMI patients [24]. Furthermore, the Cox proportional hazards model reinforced the significance of gender, PDW, MPV, and the GROUP score as independent predictors for long-term MACEs. These findings highlight the role of these factors in long-term cardiovascular health following STEMI [25]. Gender differences may be related to genetic factors, hormonal levels, and physiological differences,

Table 3. Multivariate logistic regression analysis for in-hospital death

Variables	Unadjusted		Adjusted					
	OR (95% CI)	*p* value	Model 1		Model 2		Model 3	
Male	3.194 (1.341, 7.607)	0.009	2.363 (0.531, 10.526)	0.259	2.46 (0.563, 10.800)	0.231	2.409 (0.541, 10.723)	0.248
Age	1.067 (1.027, 1.108)	0.001	1.040 (0.978, 1.105)	0.208	1.040 (0.979, 1.104)	0.204	1.037 (0.973, 1.105)	0.262
Smoking	1.416 (0.620, 3.234)	0.410	3.742 (0.480, 29.188)	0.208	2.677 (0.340, 20.916)	0.351	3.746 (0.477, 29.430)	0.209
Alcohol consumption	1.186 (0.521, 2.702)	0.684	0.596 (0.069, 5.175)	0.639	0.831 (0.099, 6.970)	0.865	0.612 (0.072, 5.189)	0.652
Hypertension	1.247 (0.546, 2.848)	0.601	1.083 (0.295, 3.979)	0.904	0.899 (0.243, 3.327)	0.873	1.001 (0.266, 3.769)	0.999
Diabetes	2.866 (1.229, 6.684)	0.015	2.208 (0.547, 8.904)	0.266	2.641 (0.680, 10.252)	0.160	2.087 (0.507, 8.591)	0.308
LVEF	0.861 (0.818, 0.906)	< 0.001	0.892 (0.825, 0.964)	0.004	0.890 (0.824, 0.961)	0.003	0.886 (0.817, 0.961)	0.003
SYNTAX	1.179 (1.109, 1.253)	< 0.001	1.103 (1.022, 1.191)	0.012	1.099 (1.020, 1.185)	0.013	1.097 (1.016, 1.184)	0.018
WBC	1.158 (1.050, 1.277)	0.003	1.104 (0.938, 1.298)	0.234	1.075 (0.907, 1.274)	0.405	1.102 (0.925, 1.312)	0.276
PLT	0.999 (0.992, 1.006)	0.726	0.999 (0.986, 1.012)	0.829	0.998 (0.985, 1.010)	0.703	0.999 (0.985, 1.014)	0.935
D-dimer	1.001 (1.000, 1.001)	0.031	1.001 (1.000, 1.002)	0.025	1.001 (1.000, 1.002)	0.034	1.001 (1.000, 1.002)	0.026
BUN	1.142 (0.960, 1.358)	0.133	0.824 (0.618, 1.098)	0.186	0.851 (0.629, 1.152)	0.296	0.852 (0.631, 1.150)	0.295
Creatinine	1.036 (1.019, 1.053)	< 0.001	1.028 (1.002, 1.054)	0.034	1.032 (1.006, 1.059)	0.016	1.029 (1.002, 1.056)	0.032
TG	1.031 (0.994, 1.069)	0.105	1.016 (0.963, 1.073)	0.560	1.020 (0.970, 1.074)	0.439	1.016 (0.962, 1.072)	0.570
TC	0.841 (0.562, 1.259)	0.400	0.852 (0.396, 1.832)	0.681	0.858 (0.470, 1.806)	0.686	0.794 (0.354, 1.783)	0.577
HDL	0.138 (0.015, 1.239)	0.077	0.135 (0.005, 3.674)	0.235	0.070 (0.002, 2.208)	0.131	0.113 (0.004, 3.537)	0.250
LDL	0.902 (0.513, 1.585)	0.719	1.494 (0.465, 4.793)	0.500	1.893 (0.609, 5.888)	0.270	1.658 (0.515, 5.339)	0.397
High PDW			4.160 (1.031, 16.792)	0.045				
High MPV					3.858 (1.125, 13.235)	0.032		
GROUP(0)							-	-
GROUP(1)							7.866 (1.147, 53.925)	0.036
GROUP(2)							12.734 (1.714, 94.581)	0.013

Notes: CI, confidence interval; OR, odds ratio; WBC, white blood cell; PLT, platelet; PDW, platelet distribution width; MPV, mean platelet volume; BUN, blood urea nitrogen; TG, triglyceride; TC, total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein. Model 1, incorporating high PDW. Model 2, incorporating high MPV. Model 3, incorporating GROUP score.

Table 4. Effects of multiple variables on long-term MACEs in multivariate Cox regression analysis

Variables	Unadjusted		Adjusted					
	OR (95% CI)	*p* value	Model 1		Model 2		Model 3	
Male	3.592 (1.554, 8.299)	0.003	3.436 (1.060, 11.136)	0.040	2.938 (0.927, 9.304)	0.067	3.109 (0.940, 10.289)	0.063
Age	1.011 (0.976, 1.047)	0.538	0.990 (0.947, 1.035)	0.662	0.998 (0.946, 1.032)	0.578	0.987 (0.944, 1.032)	0.564
Smoking	0.907 (0.398, 2.068)	0.816	2.376 (0.160, 35.199)	0.529	1.869 (0.153, 22.821)	0.624	2.355 (0.213, 26.002)	0.485
Alcohol consumption	0.894 (0.392, 2.039)	0.790	0.512 (0.034, 7.594)	0.626	0.604 (0.051, 7.210)	0.691	0.470 (0.043, 5.144)	0.537
Hypertension	1.137 (0.502, 2.578)	0.758	0.871 (0.351, 2.164)	0.767	0.982 (0.920, 1.047)	0.573	0.982 (0.919, 1.049)	0.581
Diabetes	1.377 (0.543, 3.493)	0.501	1.101 (0.381, 3.188)	0.859	1.478 (0.490, 4.455)	0.488	1.172 (0.387, 3.545)	0.779
LVEF	0.970 (0.914, 1.030)	0.321	0.981 (0.920, 1.046)	0.557	0.785 (0.309, 1.996)	0.612	0.742 (0.288, 1.912)	0.537
SYNTAX	1.037 (0.984, 1.092)	0.175	1.039 (0.980, 1.103)	0.199	1.021 (0.963, 1.083)	0.480	1.032 (0.972, 1.095)	0.302
WBC	0.989 (0.878, 1.114)	0.856	0.978 (0.850, 1.126)	0.760	0.966 (0.838, 1.112)	0.628	0.958 (0.829, 1.108)	0.564
PLT	1.003 (0.997, 1.009)	0.299	1.005 (0.996, 1.013)	0.270	1.004 (0.996, 1.012)	0.385	1.006 (0.997, 1.014)	0.178
D-dimer	1.000 (1.000, 1.001)	0.558	1.000 (1.000, 1.001)	0.237	1.000 (1.000, 1.001)	0.273	1.001 (1.000, 1.001)	0.182
BUN	1.001 (0.793, 1.262)	0.995	1.036 (0.822, 1.306)	0.762	1.020 (0.810, 1.285)	0.867	1.043 (0.835, 1.303)	0.790
Creatinine	0.982 (0.958, 1.006)	0.133	0.990 (0.966, 1.015)	0.429	0.991 (0.966, 1.016)	0.474	0.990 (0.965, 1.016)	0.454
TG	0.881 (0.641, 1.210)	0.434	0.898 (0.633, 1.274)	0.546	0.878 (0.612, 1.259)	0.480	0.896 (0.629, 1.075)	0.542
Cholesterol	1.010 (0.677, 1.506)	0.961	0.841 (0.416, 1.699)	0.629	0.848 (0.421, 1.709)	0.645	0.827 (0.395, 1.733)	0.615
HDL	1.012 (0.166, 6.177)	0.989	0.908 (0.124, 6.620)	0.924	0.734 (0.089, 6.082)	0.775	0.927 (0.119, 7.217)	0.942
LDL	1.212 (0.701, 2.095)	0.491	1.471 (0.609, 3.554)	0.391	1.583 (0.648, 3.866)	0.313	1.618 (0.650, 4.032)	0.301
Aspirin	0.384 (0.051, 2.874)	0.351	0.667 (0.023, 19.445)	0.814	0.882 (0.027, 28.774)	0.944	0.728 (0.021, 24.714)	0.860
Clopidogrel	0.572 (0.075, 4.332)	0.588	0.393 (0.035, 4.440)	0.450	0.357 (0.027, 4.775)	0.436	0.305 (0.021, 4.359)	0.381
Statins	0.532 (0.070, 3.910)	0.528	1.145 (0.046, 28.318)	0.934	0.970 (0.037, 25.781)	0.986	0.952 (0.040, 22.411)	0.975
ACEI	1.335 (0.549, 3.245)	0.524	0.818 (0.274, 2.439)	0.719	0.799 (0.255, 2.498)	0.699	0.844 (0.275, 2.597)	0.768
ARB	0.327 (0.077, 1.396)	0.131	0.315 (0.057, 1.736)	0.185	0.347 (0.062, 1.933)	0.227	0.342 (0.060, 1.945)	0.226
High PDW			3.249 (1.248, 8.458)	0.016				
High MPV					4.097 (1.663, 10.094)	0.002		
GROUP(0)							-	-
GROUP(1)							4.818 (1.351, 17.181)	0.015
GROUP(2)							9.357 (2.450, 35.736)	0.001

Notes: CI, confidence interval; OR, odds ratio; WBC, white blood cell; PLT, platelet; PDW, platelet distribution width; MPV, mean platelet volume; BUN, blood urea nitrogen; TG, triglyceride; TC, total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; LVEF, Left ventricular ejection fraction. Model 1, incorporating high PDW. Model 2, incorporating high MPV. Model 3, incorporating GROUP score.

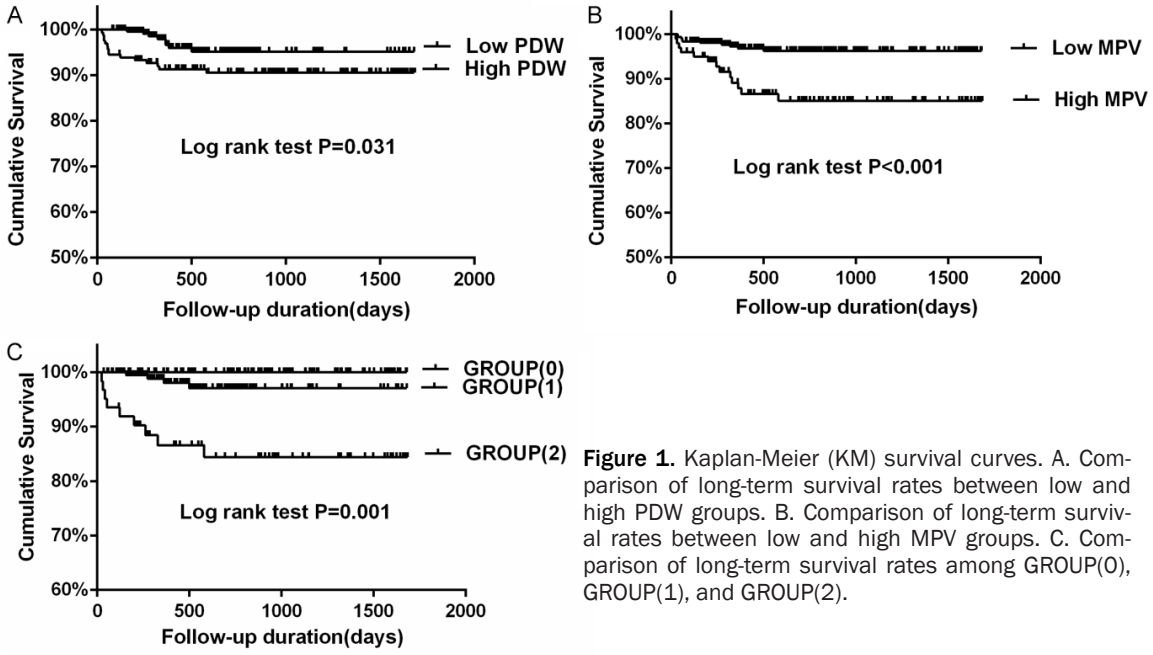

Figure 1. Kaplan-Meier (KM) survival curves. A. Comparison of long-term survival rates between low and high PDW groups. B. Comparison of long-term survival rates between low and high MPV groups. C. Comparison of long-term survival rates among GROUP(0), GROUP(1), and GROUP(2).

all of which can influence both the development and prognosis of cardiovascular diseases [26]. The prognostic significance of PDW and MPV over the long term may be associated with sustained platelet activation and an ongoing inflammatory state, which could predispose patients to recurrent cardiovascular events [27].

The Kaplan-Meier curves in this study further supported the association between PDW, MPV, and GROUP score with survival rates. Elevated PDW, MPV, and GROUP scores may indicate ongoing platelet activation and inflammation, which can contribute to recurrent cardiovascular events and increased mortality. Upon further analysis, larger platelets are more metabolically and enzymatically active, possess heightened thrombogenic properties. These larger platelets can exacerbate microvascular dysfunction, trigger inflammatory responses, and induce myocardial damage through the release of inflammatory mediators. These changes can lead to unsuccessful microcirculatory reperfusion, unfavorable left ventricular remodeling, larger infarct size, and deterioration in heart function, all of which may explain the mechanistic link between MPV and increased mortality [28, 29]. Additionally, PDW is a marker of platelet activation [30]. Larger platelets are more adhesive and more prone to aggregation. Collectively, these factors contribute to an increased incidence of cardiovascular events [31].

Conclusion

This study demonstrates that platelet activation markers, such as PDW and MPV, are significant predictors of in-hospital mortality and long-term MACEs in STEMI patients who underwent PCI. The GROUP score, which integrates these markers, provides a comprehensive risk assessment tool that may enhance clinical decision-making. Our findings align with previous research, reaffirming the predictive value of PDW and MPV for adverse outcomes. The novel contribution of this study lies in the development and validation of the GROUP score, offering a potential advancement in prognostication for STEMI patients. Future research should focus on validating these markers' utility in diverse populations and exploring their mechanistic links with cardiovascular outcomes to further refine risk stratification and improve personalized treatment strategies.

Limitations

This study has several limitations. First, it is based on a relatively small cohort of patients, which may limit the generalizability of the findings. Second, the absence of intravascular ultrasonography (IVUS) limits the ability to accurately assess the extent of atherosclerotic plaque and severity of CAD. Future studies may benefit from incorporating IVUS for a more detailed evaluation. Third, the blood sample col-

lection and testing methods represent a significant limitation. Ideally, platelet function tests should be performed with fresh samples collected in a fasting and resting state from patients who are not smoking or using caffeine or medications known to affect platelet function, such as nonsteroidal anti-inflammatory drugs (NSAIDs).

Disclosure of conflict of interest

The author(s) declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Address correspondence to: Qihan Sun, Department of Cardiology, Heilongjiang Province Hospital, Harbin 150036, Heilongjiang, China. E-mail: sunqihanhrb@gmail.com

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