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ORIGINAL RESEARCH

Pan-Immune-Inflammatory Value Predicts the Risk of Myocardial Infarction Among Patients with Unstable Angina Pectoris and the Outcomes After Percutaneous Coronary Intervention

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Objective: This study investigates the potential of the pan-immune-inflammatory value (PIV) as a predictive indicator for myocardial infarction (MI) risk in unstable angina pectoris (UAP) patients and its association with major adverse cardiovascular events (MACE) after percutaneous coronary intervention (PCI).

Methods: UAP patients diagnosed with MI underwent PCI and were monitored for MACE, including mortality, recurrent MI, revascularization, cerebrovascular accidents, and heart failure admissions. Clinical profiles and PIV levels were recorded. Multivariate logistic regression and receiver operating characteristic (ROC) analyses were conducted to identify variables associated with MI and MACE risk.

Results: MI patients had higher PIV (409.07 ± 127.63 vs 284.44 ± 126.96 × $10^{18}/L^2$, P < 0.001) and LDL-C (2.91 ± 1.04 vs 2.31 ± 1.06 mmol/L, P < 0.001) levels. Both PIV (OR = 1.008, P < 0.001) and LDL-C (OR = 1.694, P < 0.001) were significant predictors of MI. ROC analysis showed that PIV had stronger discriminatory capacity (AUC = 0.755) than LDL-C (AUC = 0.661), with their combined model improving predictive performance (AUC = 0.787). In PCI-treated MI patients, those developing MACE had higher PIV (452.66 ± 105.24 vs 378.45 ± 133.53 × $10^{18}/L^2$, P = 0.001) and TC levels (4.84 ± 0.39 vs 4.66 ± 0.42 mmol/L, P = 0.010). Both TC (OR = 3.337, P = 0.007) and PIV (OR = 1.005, P = 0.001) were independently associated with MACE. The combined model (AUC = 0.721) outperformed individual markers.

Conclusion: PIV is independently associated with MI risk in UAP patients and MACE following PCI. Combining PIV with lipid markers may enhance clinical risk assessment and inform management strategies.

Keywords: pan-immune-inflammatory value, unstable angina pectoris, myocardial infarction, percutaneous coronary intervention, major adverse cardiovascular events, predictive value

Introduction

Unstable angina pectoris (UAP) is a common and severe clinical manifestation within the spectrum of acute coronary syndrome (ACS), typically involving transient myocardial ischemia in the absence of sustained ST-segment elevation or conclusive biomarker indications of myocardial cell death.[1] A dynamic obstruction within the coronary artery may develop as a consequence of thrombus formation, which can occasionally follow the disruption, either by rupture or surface erosion, of an underlying atherosclerotic plaque.[2] Although UAP does not initially present with the same degree of myocardial injury as myocardial infarction (MI), it is inherently unstable and carries a substantial risk of progression to MI or sudden cardiac death if not promptly recognized and effectively managed.[3] Epidemiological studies have shown that, even in the contemporary era of evidence-based pharmacotherapy and early invasive strategies, a significant proportion of UAP patients will experience MI during the acute phase or within a short follow-up period, contributing to elevated morbidity and mortality rates worldwide.[4] Accordingly, recognizing individuals with UAP who may possess

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an elevated risk profile at an earlier stage could be helpful in informing treatment considerations and potentially minimizing the likelihood of subsequent cardiovascular complications.

The role of inflammation is increasingly viewed as contributory to various stages involved in atherosclerotic plaque development, including its onset, gradual progression, and eventual destabilization.[5,6] From the early stages of endothelial dysfunction to the terminal events of plaque rupture and thrombosis, immune-inflammatory responses orchestrate a cascade of cellular and molecular interactions involving neutrophils, monocytes, lymphocytes, and platelets.[5,6] The presence of a pro-inflammatory environment may be involved not only in facilitating the gradual development of atherosclerotic lesions but also in influencing plaque instability and possibly playing a role in the onset of acute coronary events. Conventional risk assessment models for ACS primarily rely on clinical characteristics, angiographic findings, and traditional biochemical markers such as lipid profiles or cardiac enzymes. However, these parameters often lack sufficient sensitivity and specificity for predicting short-term disease progression or post-intervention outcomes, particularly in heterogeneous populations such as UAP patients.[7] Over the past several years, a number of hematologic parameters obtained from routine complete blood counts, such as the monocyte-to-lymphocyte ratio (MLR),[8] platelet-to-lymphocyte ratio (PLR),[9] and neutrophil-to-lymphocyte ratio (NLR),[10] have been explored for their potential relevance as indirect indicators reflecting systemic inflammatory activity and alterations in immune function within the context of cardiovascular conditions. Although these hematologic indices are cost-effective and readily accessible in clinical settings, they offer only a limited snapshot of the multifaceted interactions between pro-inflammatory and anti-inflammatory mechanisms. In addition, variations in their prognostic utility have been observed across diverse patient populations and clinical contexts, thereby constraining their broader applicability for consistent risk assessment.

The pan-immune-inflammatory value (PIV) has more recently been introduced as a potentially broader indicator aimed at capturing the cumulative activity of immune and inflammatory responses.[11,12] By integrating neutrophil, platelet, and monocyte counts, key cellular components involved in thrombosis, inflammation, and atherogenesis, and normalizing by lymphocyte count, PIV captures a broader spectrum of immune-inflammatory activity than any single ratio. Previous studies have primarily explored PIV in oncology, where it has been associated with tumor progression, treatment resistance, and poor prognosis.[13–15] Recent investigations in the cardiovascular field have preliminarily indicated that the PIV could be associated with certain aspects of ACS, including the extent of coronary artery involvement, complications occurring during hospitalization, and potential implications for longer-term prognosis.[16,17] Traditional cardiovascular risk markers, such as lipid profiles and inflammatory markers, provide valuable insights but often focus on isolated aspects of cardiovascular health. In contrast, PIV integrates multiple immune-inflammatory components, offering a more comprehensive assessment of the inflammation underlying atherosclerosis and cardiovascular events. This approach could enhance clinical decision-making by identifying high-risk patients who may not be adequately assessed by conventional markers. By offering a holistic view of immune activation, PIV may improve risk stratification and enable more tailored management, particularly in high-risk populations like those with unstable angina pectoris. Nevertheless, its role in predicting the transition from UAP to MI remains poorly defined.

Among individuals diagnosed with MI who undergo percutaneous coronary intervention (PCI), the period following the procedure may still represent a stage of increased clinical risk. Even in the context of successful revascularization and adherence to standard medical treatment protocols, a proportion of patients may subsequently experience major adverse cardiovascular events (MACE), which can include recurrent infarction, repeat revascularization of the target vessel, cerebrovascular events, hospital admissions due to heart failure, or mortality from any cause.[18] These post-infarction complications may contribute not only to a decline in patient well-being but also to increased utilization of healthcare resources. Although conventional prognostic indicators, such as residual myocardial ischemia, reduced left ventricular performance, and lipid abnormalities, remain clinically useful, they appear insufficient to fully explain the persistent risk observed in real-world practice. Considering the recognized involvement of inflammatory processes in myocardial remodeling and the recurrence of ischemic episodes following infarction, systemic immune-inflammatory markers, including the PIV, have been proposed as potentially supplementary tools for risk stratification. The present analysis was conducted to explore whether PIV holds predictive relevance for identifying MI among patients presenting with UAP, as well as its possible association with the incidence of MACE in individuals undergoing PCI following MI.

Methods

Study Design

This In this retrospective analysis, data were drawn from a cohort of 263 individuals presenting with UAP, consecutively admitted to the Department of Cardiovascular Medicine at Beijing Shijitan Hospital Affiliated to Capital Medical University between November 2023 and March 2025. Based on a combination of clinical symptoms, electrocardiographic findings, cardiac biomarker levels, and coronary angiographic assessment, aligned with standard diagnostic guidelines, 120 of these patients were classified as having UAP without MI, while 143 exhibited features consistent with MI in the setting of UAP. Those in the MI subgroup underwent PCI during their hospital stay and were monitored thereafter for the emergence of MACE, which encompassed a composite outcome including all-cause mortality, non-fatal reinfarction, target vessel revascularization, cerebrovascular events, and hospital readmissions for heart failure.[19] The follow-up duration for MACE events was a median of 12 months, during which patients were regularly monitored for cardiovascular outcomes. Based on follow-up data, the cohort of 143 patients with MI was subsequently divided into two subgroups for comparative purposes: those who did not experience major adverse cardiovascular events (non-MACE, n = 84) and those who did (MACE, n = 59). Since the study was retrospective without extra interventions, the protocol was waived by the Ethics Committee of Beijing Shijitan Hospital Affiliated to Capital Medical University, and informed consent was also waived. The study was carried out in alignment with the requirements outlined in the Declaration of Helsinki.

Inclusion and Exclusion Criteria

Inclusion criteria were: (1) diagnosis of UAP according to current guidelines, based on clinical symptoms, electrocardiographic findings, and/or coronary angiography; (2) age ≥18 years; (3) complete baseline demographic, clinical, and laboratory data, including lipid profile, hematologic indices, and PIV; and (4) for the MI group, diagnosis confirmed by elevated cardiac biomarkers with clinical and ECG evidence, and completion of PCI during hospitalization.

Participants were excluded from the study based on the following conditions: (1) presence of either acute or chronic infections, autoimmune disorders, or systemic inflammatory conditions; (2) diagnosis of hematological malignancies or other forms of cancer; (3) evidence of marked hepatic or renal impairment, defined as liver enzyme levels exceeding three times the upper reference limit or an estimated glomerular filtration rate below 30 mL/min/1.73 m^2; (4) history of major cardiovascular events occurring within the three months preceding admission; (5) ongoing treatment with corticosteroids or immunosuppressive medications; and (6) absence of complete clinical records, laboratory findings, or follow-up information necessary for analysis.

Clinical Data Collection

Demographic characteristics, clinical profiles, and laboratory parameters were retrospectively retrieved from electronic hospital records at the time of admission. Demographic information encompassed age, sex, and body mass index (BMI), while clinical history included smoking behavior and prior diagnoses of hypertension (HTN) and diabetes mellitus (DM). In patients diagnosed with MI, details related to PCI procedures and subsequent follow-up for MACE were also reviewed. Within 24 hours of admission, fasting venous blood samples were obtained and assessed for a range of biomarkers, including hemoglobin (Hb), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and serum creatinine (SCr). The PIV was calculated using the following formula: neutrophil count × platelet count × monocyte count divided by lymphocyte count. All laboratory analyses were conducted in the hospital's central facility under standardized operational protocols.

Statistical Analysis

Statistical analyses were carried out using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed for normality using the Shapiro–Wilk test. Variables following a normal distribution were summarized as mean ± standard deviation (SD) and compared between groups through the independent samples *t*-test. For variables not conforming to normality assumptions, values were described as

median with interquartile range (IQR), which were analyzed by the Mann–Whitney *U*-test. Categorical data were reported as percentages or frequencies, and either the Fisher's exact test or chi-squared (χ^2) test was applied, depending on the distribution and sample size. To explore associations with MI among patients with UAP, as well as with MACE in the MI subgroup following PCI, univariate logistic regression analyses were initially performed. Variables demonstrating a P-value below 0.05 in univariate analysis were subsequently entered into multivariate logistic regression models to identify potential independent predictors. Outcomes were expressed as odds ratios (ORs) along with corresponding 95% confidence intervals (CIs). Receiver operating characteristic (ROC) curve analysis was employed to evaluate the ability of selected predictors—both individually and in combination—to discriminate between outcomes related to MI and MACE. The area under the curve (AUC) and its 95% CI were calculated for each model. Optimal thresholds were determined using the Youden index, with corresponding sensitivity and specificity also reported. All statistical tests were two-sided, and significance was defined by a P-value less than 0.05.

Results

Comparison of Baseline Characteristics Between UAP Patients with and without MI

The study population consisted of 263 individuals diagnosed with UAP, including 120 patients without MI who were categorized as the control group, and 143 patients with MI, referred to as the MI group. Clinical and demographic characteristics at baseline for the two groups are summarized in Table 1. There were no significant differences in body mass index (25.21 ± 2.62 vs 25.83 ± 3.11 kg/m^2, $P = 0.083$), age (61.13 ± 8.67 vs 63.10 ± 7.67 years, $P = 0.052$), history of hypertension ($P = 0.800$), or diabetes mellitus ($P = 0.262$) between the 2 groups. However, a higher proportion of males (74.83% vs 58.33%, $P = 0.006$) and current smokers (63.64% vs 40.00%, $P < 0.001$) was noted in the MI group. Most routine laboratory markers, including Hb, TC, HDL-C, TG, and SCr, did not differ meaningfully between groups (P values > 0.05). In

Table 1 Comparison of Baseline Characteristics Between UAP Patients with and without MI

Indices	Con Group (n=120)	MI Group (n=143)	P value
Age	61.13±8.67	63.10±7.67	0.052
Gender [n(%)]			0.006
Female	50 (41.67)	36 (25.17)	
Male	70 (58.33)	107 (74.83)	
BMI (kg/m2)	25.21±2.62	25.83±3.11	0.083
Smoking [n(%)]			<0.001
Yes	48 (40.00)	91 (63.64)	
No	72 (60.00)	52 (36.36)	
HTN [n(%)]			0.8
Yes	74 (61.67)	85 (59.44)	
No	46 (38.33)	58 (40.56)	
DM [n(%)]			0.262
Yes	18 (15.00)	30 (20.98)	
No	102 (85.00)	113 (79.02)	

(*Continued*)

Table 1 (Continued).

Indices	Con Group (n=120)	MI Group (n=143)	P value
Laboratory testing			
Hb (g/L)	137.60±10.11	135.49±10.03	0.092
TC (mmol/L)	4.64±0.44	4.74±0.42	0.067
HDL (mmol/L)	1.14±0.09	1.12±0.11	0.218
LDL (mmol/L)	2.31±1.06	2.91±1.04	<0.001
TG (mmol/L)	4.71±0.66	4.85±0.61	0.067
SCr (umol/L)	75.68±8.72	76.43±10.35	0.531
PIV ($10^{18}/L^2$)	284.44±126.96	409.07±127.63	<0.001

Abbreviations: UAP, Unstable Angina Pectoris; MI, Myocardial Infarction; BMI, Body Mass Index; HTN, Hypertension; DM, Diabetes Mellitus; Hb, Hemoglobin; TC, Total Cholesterol; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; TG, Triglyceride; SCr, Serum Creatinine; PIV, Pan-Immune-Inflammatory Value.

contrast, LDL-C levels were elevated in the MI group (2.91 ± 1.04 vs 2.31 ± 1.06 mmol/L, $P < 0.001$). Additionally, the PIV was higher among MI patients compared to controls (409.07 ± 127.63 vs 284.44 ± 126.96 × $10^{18}/L^2$, $P < 0.001$).

Logistic Regression Analysis of Risk Factors for MI in Patients with UAP

In the univariate logistic regression analysis, both LDL-C and the PIV demonstrated statistically significant associations with the occurrence of MI in patients presenting with UAP, as shown in Table 2. An increase in LDL levels was linked to a higher likelihood of MI (B = 0.551, SE = 0.127, Wald $\chi^2 = 18.824$, $P < 0.001$), corresponding to an OR of 1.735 (95% CI: 1.353–2.225). A similar trend was observed for PIV, which also showed a notable association with MI risk (B = 0.008, SE = 0.001, Wald $\chi^2 = 41.227$, $P < 0.001$; OR = 1.008, 95% CI: 1.005–1.010). Following adjustment for potential confounding variables in the multivariate logistic regression model, both LDL (B = 0.527, SE = 0.140, Wald $\chi^2 = 14.219$, $P < 0.001$; OR = 1.694, 95% CI: 1.288–2.228) and PIV (B = 0.008, SE = 0.001, Wald $\chi^2 = 38.381$, $P < 0.001$; OR = 1.008, 95% CI: 1.005–1.010) remained independently associated with MI occurrence. These findings suggest that elevated levels of both parameters may be linked to an increased probability of infarction in this clinical context, although further investigation is warranted to confirm their prognostic utility.

ROC Analysis for Predicting MI in Patients with UAP

Table 3 and Figure 1 summarize the diagnostic characteristics of LDL-C, the PIV, and their combination in relation to MI detection among patients with UAP. For LDL, calculation of the AUC was performed at 0.661 (95% CI: 0.595–0.726, $P < 0.001$). At an optimal threshold of 2.865 mmol/L, this measure yielded a sensitivity of 55.9% and specificity of

Table 2 Univariate and Multivariate Logistic Regression Analyses Identifying Independent Predictors of MI in Patients with UAP

Variables	Univariate Logistic Analysis						Multivariate Logistic Analysis					
	B	SE	Wald X2	P	OR	95% CI	B	SE	Wald X2	P	OR	95% CI
LDL	0.551	0.127	18.824	<0.001	1.735	1.353~2.225	0.527	0.14	14.219	<0.001	1.694	1.288~2.228
PIV	0.008	0.001	41.227	<0.001	1.008	1.005~1.010	0.008	0.001	38.381	<0.001	1.008	1.005~1.010

Abbreviations: UAP, Unstable Angina Pectoris; MI, Myocardial Infarction; LDL, Low-Density Lipoprotein; PIV, Pan-Immune-Inflammatory Value; SE, Standard Error; OR, Odds Ratio; CI, Confidence Interval.

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Table 3 ROC Analysis of PIV for Predicting MI in Patients with UAP

Variables	AUC	95% CI	Best Cut-off Value	Sensitivity (%)	Specificity (%)	P value
LDL	0.661	0.595~0.726	2.865	55.9	72.5	<0.001
PIV	0.755	0.697~0.813	344.125	69.9	70.8	<0.001
Combined	0.787	0.732~0.841		62.9	84.2	<0.001

Abbreviations: UAP, Unstable Angina Pectoris; MI, Myocardial Infarction; AUC, Area Under the Curve; CI, Confidence Interval; LDL, Low-Density Lipoprotein; PIV, Pan-Immune-Inflammatory Value.

72.5%. PIV demonstrated comparatively greater discriminative capacity, with an AUC of 0.755 (95% CI: 0.697–0.813, $P < 0.001$), and a derived cut-off point of $344.125 \times 10^{18}/L^2$, corresponding to sensitivity and specificity values of 69.9% and 70.8%, respectively. When LDL and PIV were integrated into a combined model, the overall performance improved modestly, achieving an AUC of 0.787 (95% CI: 0.732–0.841, $P < 0.001$), with specificity and sensitivity of 84.2% and 62.9%, respectively. Taken together, these results suggest that while both LDL and PIV, when considered individually, demonstrate moderate discriminative value in identifying MI among patients with UAP, their combined application may offer a more refined risk estimation approach. Nonetheless, further prospective studies would be beneficial to validate the clinical relevance of these findings across broader patient populations.

Baseline Features Between MI Patients with and without MACE After PCI

In an effort to explore baseline variables that might be associated with the development of MACE following PCI in patients with MI, demographic, clinical, and laboratory data were compared between those who did (n = 59) and did not (n = 84) experience MACE, as detailed in Table 4. There were no significant differences between the groups in sex distribution ($P = 0.559$), age (63.29 ± 7.48 vs 62.83 ± 7.99 years, $P = 0.728$), body mass index (25.76 ± 3.24 vs 25.93 ± 2.92 kg/m^2, $P = 0.737$), smoking behavior ($P = 1.000$), presence of HTN ($P = 0.387$), or history of DM ($P = 0.678$). Similarly, no group-level differences were detected for a range of laboratory indicators, including Hb concentration, HDL-C, LDL-C, TG, or SCr values (all $P > 0.05$). However, the subgroup that experienced MACE demonstrated modestly higher total cholesterol concentrations (4.84 ± 0.39 vs 4.66 ± 0.42 mmol/L, $P = 0.010$), along with elevated PIV measurements (452.66 ± 105.24 vs $378.45 \pm 133.53 \times 10^{18}/L^2$, $P = 0.001$), when compared with those who remained event-free during follow-up.

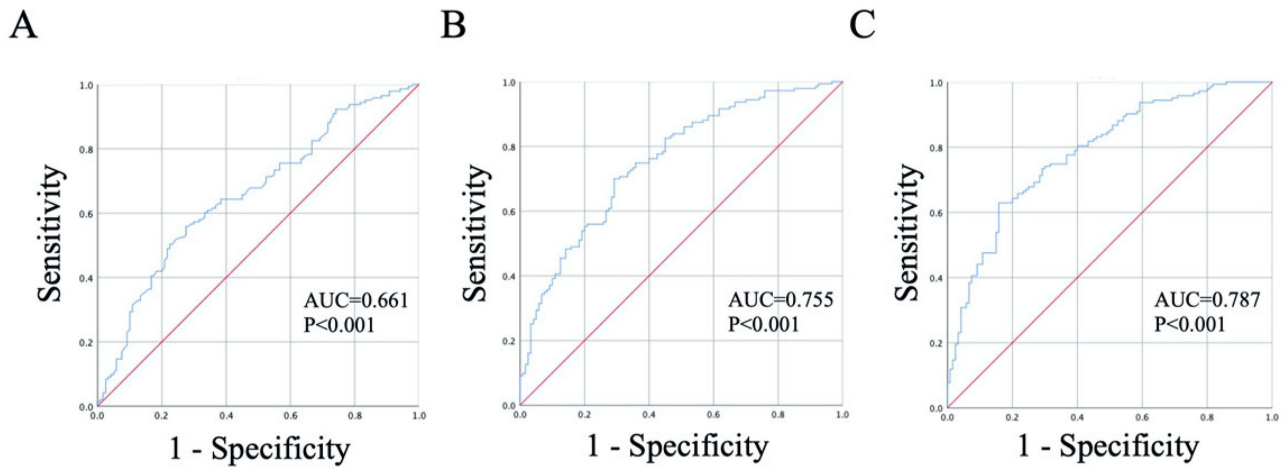

Figure 1 ROC Analysis of (**A**) LDL, (**B**) PIV, and (**C**) Their Combination for Predicting MI in Patients with UAP.
Abbreviations: LDL, Low-Density Lipoprotein; PIV, Pan-Immune-Inflammatory Value; AUC, Area Under the Curve; ROC, Receiver Operating Characteristic; MI, Myocardial Infarction.

Table 4 Comparison of Baseline Characteristics Between MI Patients with and without MACE After PCI

Indices	Non-MACE Group (n=84)	MACE Group (n=59)	P value
Age	63.29±7.48	62.83±7.99	0.728
Gender [n(%)]			0.559
Female	23 (27.38)	13 (22.03)	
Male	61 (72.62)	46 (77.97)	
BMI (kg/m^2)	25.76±3.24	25.93±2.92	0.737
Smoking [n(%)]			1
Yes	31 (36.90)	21 (35.59)	
No	53 (63.10)	38 (64.41)	
HTN [n(%)]			0.387
Yes	47 (55.95)	38 (64.41)	
No	37 (44.05)	21 (35.59)	
DM [n(%)]			0.678
Yes	19 (22.62)	11 (18.64)	
No	65 (77.38)	48 (81.36)	
Laboratory testing			
Hb (g/L)	136.69±9.64	133.78±10.42	0.088
TC (mmol/L)	4.66±0.42	4.84±0.39	0.01
HDL (mmol/L)	1.12±0.11	1.12±0.11	0.845
LDL (mmol/L)	3.02±1.03	2.76±1.03	0.15
TG (mmol/L)	4.83±0.59	4.88±0.63	0.64
SCr (umol/L)	76.19±10.81	76.78±9.75	0.74
PIV ($10^{18}/L^2$)	378.45±133.53	452.66±105.24	0.001

Abbreviations: PCI, Percutaneous Coronary Intervention; MACE, Major Adverse Cardiovascular Events; BMI, Body Mass Index; HTN, Hypertension; DM, Diabetes Mellitus; Hb, Hemoglobin; TC, Total Cholesterol; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; TG, Triglyceride; SCr, Serum Creatinine; PIV, Pan-Immune-Inflammatory Value.

Logistic Regression Analysis of Predictors of MACE in MI Patients After PCI

To explore factors that may be independently associated with the occurrence of MACE in patients with MI undergoing PCI, both univariate and multivariate logistic regression analyses were conducted (Table 5). In the initial univariate analysis, TC and the PIV each showed a statistically significant association with MACE. Elevated TC levels were related to a greater probability of experiencing MACE (B = 1.079, SE = 0.430, Wald $\chi^2 = 6.300$, P = 0.012), corresponding to an OR of 2.942 (95% CI: 1.267–6.833). Likewise, increased PIV values were linked to higher event rates (B = 0.005, SE = 0.002, Wald $\chi^2 = 10.743$, P = 0.001), with an OR of 1.005 (95% CI: 1.002–1.008). After accounting for potential confounders, results from multivariate analysis indicated that both TC (B = 1.205, SE = 0.450, Wald $\chi^2 = 7.170$, P = 0.007; OR = 3.337, 95% CI: 1.381–8.060) and PIV (B = 0.005, SE = 0.002, Wald $\chi^2 = 11.656$, P = 0.001; OR = 1.005, 95% CI: 1.002–1.008) remained statistically significant variables associated with MACE in this particular cohort.

Table 5 Univariate and Multivariate Logistic Regression Analyses Identifying Independent Predictors of MACE in MI Patients After PCI

Variables	Univariate Logistic Analysis						Multivariate Logistic Analysis					
	B	SE	Wald X2	P	OR	95% CI	B	SE	Wald X2	P	OR	95% CI
TC	1.079	0.43	6.3	0.012	2.942	1.267~6.833	1.205	0.45	7.17	0.007	3.337	1.381~8.060
PIV	0.005	0.002	10.743	0.001	1.005	1.002~1.008	0.005	0.002	11.656	0.001	1.005	1.002~1.008

Abbreviations: MACE, Major Adverse Cardiovascular Events; MI, Myocardial Infarction; PCI, Percutaneous Coronary Intervention; TC, Total Cholesterol; PIV, Pan-Immune-Inflammation Value; CI, Confidence Interval; OR, Odds Ratio.

Table 6 ROC Analysis of PIV for Predicting MACE After PCI

Variables	AUC	95% CI	Best Cut-off Value	Sensitivity (%)	Specificity (%)	P value
TC	0.648	0.556~0.740	4.565	86.4	51.2	0.003
PIV	0.682	0.595~0.770	389.37	72.9	58.3	<0.001
Combined	0.721	0.637~0.804		78	64.3	<0.001

Abbreviations: MACE, Major Adverse Cardiovascular Events; PCI, Percutaneous Coronary Intervention; TC, Total Cholesterol; PIV, Pan-Immune-Inflammatory Value; AUC, Area Under the Curve; CI, Confidence Interval.

ROC Analysis for Predicting MACE in MI After PCI

To assess the potential prognostic utility of TC, the PIV, and their combined use in relation to MACE following PCI, ROC curve analysis was conducted (Table 6 and Figure 2). The AUC for TC was 0.648 (95% CI: 0.556–0.740, P = 0.003), with a corresponding threshold of 4.565 mmol/L, yielding a sensitivity of 86.4% and a specificity of 51.2%. PIV alone showed a modestly improved discriminatory capacity relative to TC, with an AUC of 0.682 (95% CI: 0.595–0.770, P < 0.001). The optimal cut-off value identified for PIV was $389.37 \times 10^{18}/L^2$, providing a sensitivity of 72.9% and specificity of 58.3%. When TC and PIV were analyzed together as part of a combined model, the overall performance showed a further incremental increase, with an AUC of 0.721 (95% CI: 0.637–0.804, P < 0.001), a sensitivity of 78.0%, and specificity of 64.3%.

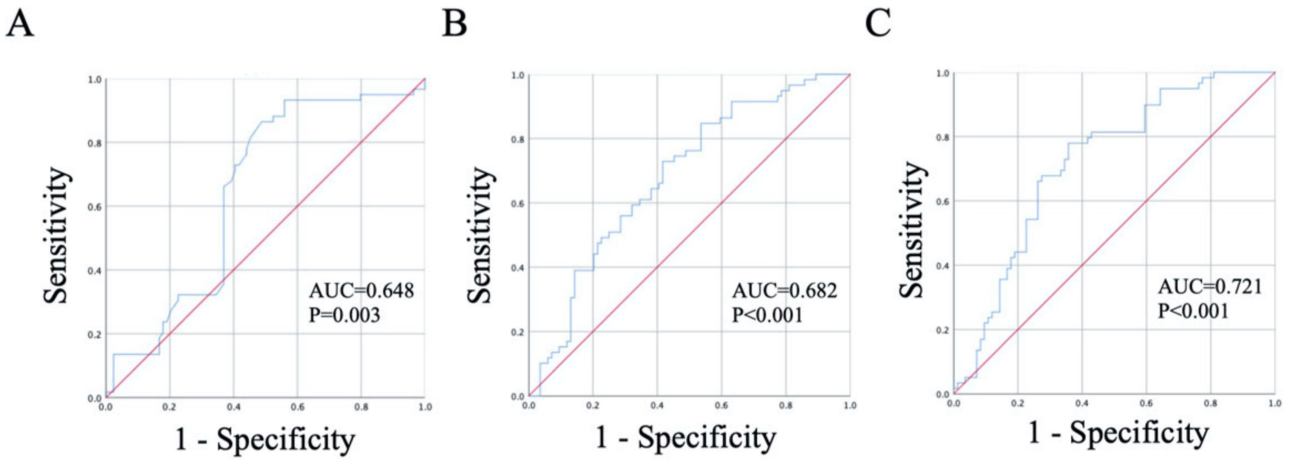

Figure 2 ROC Analysis of (**A**) Total Cholesterol, (**B**) Pan-Immune-Inflammatory Value, and (**C**) Their Combination for Predicting MACE in MI Patients After PCI.
Abbreviations: ROC, Receiver Operating Characteristic; TC, Total Cholesterol; PIV, Pan-Immune-Inflammatory Value; MACE, Major Adverse Cardiovascular Events; MI, Myocardial Infarction; PCI, Percutaneous Coronary Intervention; AUC, Area Under the Curve.

Discussion

In this study, patients with UAP who subsequently experienced MI were observed to have higher PIV levels at baseline compared to those who did not progress to MI. Elevated PIV was found to be statistically associated with MI occurrence, even after adjusting for potential confounders. LDL-C also showed an independent association with MI, and when combined with PIV, the integrated model provided improved, though still moderate, discriminative capability for identifying individuals at increased risk. Furthermore, within the subgroup of MI patients who underwent PCI, both PIV and TC appeared to be independently linked to the development of MACE during follow-up. The combination of these two markers demonstrated enhanced predictive performance relative to either variable alone, though further validation in larger populations is warranted. These findings highlight the potential of PIV, either alone or combined with lipid parameters, as a practical biomarker for integrated cardiovascular risk assessment.

The higher PIV observed in UAP patients with MI is consistent with the pathophysiological role of inflammation in plaque destabilization and rupture. Neutrophils release proteolytic enzymes and reactive oxygen species that weaken the fibrous cap, facilitating thrombus formation.[20–22] Platelets participate in thrombus growth and release pro-inflammatory mediators that amplify vascular injury.[23,24] Monocytes infiltrate the intima and differentiate into macrophages, further driving the inflammatory cascade.[25,26] Conversely, lymphopenia reflects impaired adaptive immune function and heightened stress responses.[27] The PIV represents a derived hematologic marker, formulated by multiplying neutrophil, platelet, and monocyte counts, and subsequently dividing the result by the lymphocyte count.[28] Unlike single-ratio indices such as the NLR or PLR, PIV simultaneously captures the contributions of three pro-inflammatory and pro-thrombotic cellular populations while accounting for the relative depletion of lymphocytes, which reflect immunoregulatory capacity. Emerging evidence has suggested that the PIV may hold potential prognostic and diagnostic relevance in various clinical settings outside the cardiovascular domain, with a growing body of literature focusing particularly on its applications in oncology. In the context of esophageal cancer, higher levels of the PIV have been observed in association with poorer overall survival and lower densities of tumor-infiltrating lymphocytes. These findings imply that systemic immune status could potentially influence clinical outcomes, at least in part, by affecting localized immune activity within the tumor microenvironment.[29] Similarly, in colorectal cancer, higher PIV values correlate with advanced tumor stage and adverse clinicopathological characteristics.[30] Findings from studies on epithelial ovarian cancer suggest that higher PIV levels may be related to less favorable outcomes, including reduced overall and progression-free survival. Additionally, the use of a nomogram incorporating PIV has been explored as a supplementary tool that may improve prognostic assessment and aid in refining clinical risk categorization.[31] In pancreatic cancer, elevated levels of the PIV have been associated with less favorable overall and progression-free survival outcomes. These observations suggest that PIV may serve as a potentially informative, though indirect, marker of prognosis within this clinical setting.[32] Similar patterns have been observed in cases of inflammatory breast cancer, in which higher PIV levels appear to be associated with shorter durations of disease-free and overall survival. These associations indicate a potential relationship between systemic inflammatory status and clinical outcomes in this malignancy, although the underlying mechanisms are still not much clear.[33] In addition to its possible relevance in prognostic assessment, the PIV has been explored for its potential diagnostic application. Among individuals with PSA levels ranging from 4 to 20 ng/mL, elevated PIV levels have been associated with the presence of prostate cancer, including cases considered clinically significant. Moreover, combining PIV with PSA measurements may contribute to enhanced diagnostic performance, although further validation is needed to clarify its added value in routine clinical practice.[34]

Research on PIV in cardiovascular medicine has expanded considerably in recent years, yielding growing evidence of its value as a biomarker across a spectrum of cardiac conditions. Current findings suggest that higher PIV levels may be linked to more extensive coronary artery involvement, a greater likelihood of in-hospital complications among individuals with acute coronary syndrome, and an elevated risk of mortality in patients with heart failure. However, these associations should be interpreted with caution, as underlying mechanisms and causality remain to be fully established.[16,17,35–37] Among individuals presenting with ST-segment elevation myocardial infarction, elevated PIV levels have been observed in association with greater thrombotic burden, suboptimal microvascular reperfusion, and less favorable short-term outcomes following PCI. These findings appear to align with the proposed involvement of

systemic inflammatory processes in the pathophysiology of atherothrombosis, myocardial tissue damage, and subsequent ventricular remodeling, although further investigation is warranted to clarify the extent and mechanisms of these relationships.[38] Recent studies have further expanded these observations across various cardiovascular contexts. In coronary heart disease, particularly among elderly populations, PIV has shown a positive association with disease presence, and its diagnostic accuracy improves when combined with the controlling nutritional status score.[11] In the context of evaluating coronary artery disease burden, increased PIV levels have been reported to show an independent association with elevated SYNTAX scores among patients presenting with non-ST-elevation myocardial infarction.[16] Additionally, PIV has demonstrated a modest correlation with the presence of more advanced atherosclerotic findings on coronary computed tomographic angiography, suggesting a potential link with overall disease severity.[39] Beyond ischemic heart disease, PIV demonstrates prognostic relevance in heart failure, where elevated values are independently and non-linearly linked to increased risk, underscoring its utility for clinical monitoring.[40] Moreover, in ST-elevation myocardial infarction, PIV independently predicts the occurrence of no-reflow after PCI and outperforms the systemic immune-inflammatory index in predictive accuracy.[41] Collectively, these findings support PIV as a cost-effective, accessible, and clinically informative biomarker with broad potential applications in cardiovascular risk stratification and prognosis.

LDL-C also remained a robust predictor of MI in UAP patients. Elevated LDL-C promotes lipid deposition within the arterial wall, triggers endothelial dysfunction, and enhances oxidative modification of lipoproteins, all of which accelerate atherosclerosis.[42–44] Oxidized LDL further activates innate immune pathways, augmenting vascular inflammation and plaque vulnerability.[45,46] The enhanced predictive value observed when combining LDL-C with PIV likely reflects the integration of atherogenic lipid burden and inflammatory status, providing a more complete assessment of cardiovascular risk. Among MI patients treated with PCI, elevated TC and PIV were both independently associated with subsequent MACE. Persistently elevated TC may indicate residual lipid-driven atherothrombotic activity, which predisposes patients to recurrent ischemic events despite revascularization.[47,48] Elevated PIV after PCI may reflect a sustained inflammatory state triggered by vascular injury, microvascular obstruction, and reperfusion injury, which in turn contributes to adverse ventricular remodeling, restenosis, and recurrent thrombotic events. Patients with higher PIV may therefore have an amplified inflammatory and thrombotic response that places them at greater risk for post-procedural complications.

ROC analysis confirmed that while TC and PIV each provided moderate discriminatory ability for predicting MACE, their combination yielded greater accuracy. This finding underscores the multifactorial nature of residual risk after PCI, which involves both metabolic and inflammatory components. Incorporating PIV alongside traditional lipid measures could therefore improve risk stratification and support more targeted secondary prevention strategies, including intensified lipid-lowering therapy and anti-inflammatory interventions. These results emphasize the central role of systemic inflammation, as reflected by PIV, in both the acute progression from UAP to MI and the long-term prognosis following PCI in MI patients. The capacity of PIV to capture multiple dimensions of the inflammatory and thrombotic response likely explains its consistent predictive value in both settings. Given that PIV can be derived from routine complete blood counts, it represents an accessible and cost-effective biomarker for clinical practice.

Limitations

Several considerations should be noted in interpreting the results of this study. As a retrospective analysis conducted at a single institution, the findings may have limited external applicability. The sample size was relatively small, which may affect statistical power. Although a formal power calculation was not performed, the sample size may limit the robustness of subgroup analyses, and future studies with larger cohorts are needed to validate the findings. Although adjustments were made for critical confounding factors, the possibility of residual bias due to unmeasured variables cannot be entirely ruled out. Moreover, commonly used inflammatory markers such as high-sensitivity C-reactive protein and interleukin-6 were not assessed, which prevented direct comparison with the PIV. Further validation through prospective, multicenter investigations involving larger and more diverse patient populations would be beneficial to confirm these observations and to explore whether strategies incorporating PIV could guide treatment decisions.

Conclusion

In summary, PIV was independently associated with MI occurrence in UAP patients and MACE development in those undergoing PCI. Combining PIV with conventional lipid parameters enhances predictive performance, highlighting the roles of inflammation and lipid metabolism in coronary artery disease. These findings support the integration of PIV into clinical risk models for personalized management in high-risk populations. However, given the single-center retrospective design, caution is needed when generalizing results, and further validation in larger, multi-center studies is necessary. PIV may offer a cost-effective biomarker for improved risk stratification and treatment decisions, especially in resource-limited settings. Further studies are needed to confirm its broader clinical applicability.

Abbreviations

PIV, pan-immune-inflammatory value; MI, myocardial infarction; UAP, unstable angina pectoris; MACE, major adverse cardiovascular events; PCI, percutaneous coronary intervention; ROC, receiver operating characteristic; AUC, area under the curve; TC, total cholesterol; ACS, acute coronary syndrome; MLR, monocyte-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio.

Data Sharing Statement

All data generated or analyzed during this study are included in this manuscript. Further inquiries should be directed to the corresponding author.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was waived by the Ethics Committee of Beijing Shijitan Hospital Affiliated to Capital Medical University since the study was retrospective and no additional interventions were given. Given the retrospective design, the requirement for individual informed consent was waived.

Author Contributions

JP: Conceptualization, Formal analysis, Methodology, Writing – review & editing; CC: Software, Validation, Writing – original draft; BZ: Investigation, Supervision, Writing – review & editing; YF: Investigation, Supervision, Writing – review & editing; All authors made significant contributions to the work, reviewed and approved all versions of the manuscript, including the final version, and agreed on the journal for submission. They take responsibility for all aspects of the work, ensuring its accuracy and integrity.

Disclosure

The authors declare that there is no conflicts of interest.

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