Article

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Association Analysis of Myocardial Injury Markers With Post-PCI Clinical Outcomes: A Single-Center Retrospective Study

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Background: Percutaneous coronary intervention (PCI) is a key revascularization strategy for coronary artery disease, but some patients still experience major adverse cardiovascular events (MACE) post-procedure. Myocardial injury biomarkers reflect cardiomyocyte damage, yet evidence on their combined analysis for post-PCI outcomes is limited. This study investigated the association between multiple myocardial injury biomarkers—including creatine kinase-MB (CK-MB), cardiac troponin I (cTnI), and aspartate aminotransferase (AST)—and post-PCI MACE, and evaluated their potential for risk identification.

Methods: This single-center retrospective study enrolled 266 consecutive patients who underwent PCI from January 2022 to January 2024. Patients were divided into MACE (n = 43) and non-MACE (n = 223) groups based on 12-month follow-up. Baseline data and post-procedural (within 24 h) levels of CK-MB, cTnI, AST, and alanine aminotransferase (ALT) were collected. Univariate and multivariate logistic regression identified independent factors for MACE, and receiver operating characteristic (ROC) curves assessed discriminative ability.

Results: Multivariate regression showed that CK-MB (odds ratio (OR) = 1.01, 95% confidence interval (CI): 1.01–1.02, $p < 0.001$), cTnI (OR = 1.04, 95% CI: 1.02–1.06, $p < 0.001$), and dyslipidemia (OR = 2.36, 95% CI: 1.14–4.91, $p = 0.021$) were independently associated with post-PCI MACE. The combination of these factors (dyslipidemia, CK-MB, cTnI) yielded an area under the curve (AUC) of 0.75 (95% CI: 0.66–0.84), sensitivity of 88.0% and specificity of 78.0%.

Conclusion: In post-PCI patients, CK-MB, cTnI, and dyslipidemia are independently associated with MACE risk. Combining these myocardial injury markers with clinical characteristics provides value for identifying adverse outcomes and may assist postoperative risk assessment.

Keywords: percutaneous coronary intervention; myocardial injury markers; CK-MB

Introduction

Percutaneous coronary intervention (PCI) has become a key revascularization strategy for the treatment of coronary artery disease (CAD), particularly in the setting of acute myocardial infarction [1]. With the continuous advancement of interventional techniques, devices, and perioperative management, PCI has significantly reduced the mortality rate and recurrent ischemic events in patients with acute coronary syndrome and improved their quality of life [2]. However, despite the continuous optimization of treatment strategies, a subset of patients undergoing PCI still experience major adverse cardiovascular events (MACE), including cardiac death, recurrent myocardial infarction, and revascularization. These events remain key determinants of long-term prognosis [3]. Therefore, how to effectively stratify the risk of PCI patients in clinical practice, so as to identify high-risk groups as early as possible and develop individualized treatment strategies, remains an important research direction in the current cardiovascular field.

In recent years, research on the prognosis after PCI has mainly focused on clinical characteristics, imaging indicators and angiographic parameters, such as patient age, diabetes status, left ventricular ejection fraction (LVEF) and the complexity of coronary artery lesions [4,5]. However, these indicators often require relatively complex examination or evaluation procedures in clinical practice, while serum biomarkers have gradually become a valuable tool for risk assessment due to their convenient acquisition and rapid detection. In particular, myocardial injury-related biomarkers are important for reflecting the extent of cardiomyocyte damage, and have therefore attracted increasing research attention. Among the various myocardial injury biomarkers, creatine kinase (CK), creatine kinase-MB (CK-MB) and cardiac troponin are the most frequently used indicators in clinical practice [6]. With the development of detection technology, cardiac troponin has gradually become the gold standard for diagnosing acute myocardial infarction, and has higher sensitivity and specificity in the de-

tection of myocardial injury [7]. Studies have shown that elevated myocardial markers detected after PCI are closely associated with adverse clinical outcomes in patients [8]. For example, a follow-up study of PCI patients found that elevated levels of CK-MB, cardiac troponin T (cTnT), and cardiac troponin I (cTnI) 6–24 hours after the procedure were significantly associated with the occurrence of MACE and demonstrated prognostic predictive value [9]. These studies suggest that perioperative myocardial injury markers can not only reflect myocardial injury that occurs during the procedure, but may also be associated with long-term prognosis. Specifically, myocardial injury detected within 24 hours post-PCI indicates the extent of perioperative cardiomyocyte damage, which can trigger subsequent adverse ventricular remodeling, microvascular dysfunction, and chronic inflammatory responses, thereby increasing the risk of MACE during long-term follow-up.

In addition to traditional myocardial-specific markers, some non-specific enzyme indicators may also reflect the degree of damage to the myocardium or other tissues. For example, aspartate aminotransferase (AST) is released into the bloodstream when cells are damaged and has been widely used in the early diagnosis of myocardial infarction [10]. Although its role in the diagnosis of myocardial injury has gradually declined with the emergence of more specific myocardial markers, AST may still reflect the overall degree of tissue ischemia or necrosis. Therefore, in patients with cardiovascular disease, changes in AST levels may reflect the severity of the disease and the state of damage to the body to some extent.

Although existing studies have confirmed a link between myocardial injury biomarkers and adverse events after PCI, several limitations remain. First, most studies primarily focus on the relationship between elevated postoperative biomarkers and perioperative complications, while research on the relationship between postoperative myocardial injury biomarker levels and short- to medium-term clinical outcomes is relatively limited. Second, the types of biomarkers included and the study endpoints vary considerably among different studies, leading to heterogeneity in the results. Furthermore, in clinical practice, multiple myocardial injury biomarkers are often measured simultaneously, and evidence remains insufficient to comprehensively analyze their potential value in prognostic evaluation.

This retrospective study analyzed clinical data from patients who underwent PCI at our center. The study aimed to explore the association between various myocardial injury-related biomarkers (including CK-MB, cTnI, and AST) and post-PCI clinical outcomes, and to assess their potential value in identifying the risk of MACE. The main innovation of this study lies in the comprehensive analysis of multiple conventional myocardial injury biomarkers, which comprehensively assessed their relationship with post-PCI prognosis. Through statistical analysis of various indicators, this study attempted to identify key biomarkers closely related to adverse outcomes after PCI, thereby providing a reference for early clinical identification of high-risk patients.

Therefore, this study aimed to analyze the association between the levels of myocardial injury markers and postoperative clinical outcomes after PCI, and to explore their potential value in risk stratification, thereby providing new evidence to support prognostic assessment and individualized management of PCI patients. In this study, biomarker levels measured at 24 hours post-PCI were analyzed in association with 12-month MACE.

Methods

Participants

This is a retrospective study conducted at Xianju People's Hospital. Patients who underwent PCI at our center between January 2022 and January 2024 were consecutively enrolled. PCI procedures were performed according to current clinical practice guidelines and our center's established protocols. This study protocol adheres to the principles outlined in the Declaration of Helsinki and was approved by the Ethics Committee of Xianju People's Hospital (NO.2026-35). As this is a retrospective observational study, data were collected only from patients during their routine clinical care. The research interventions were within the scope of routine clinical care and did not impose any additional risks or burdens on patients' physical, psychological, or social relationships. Furthermore, patient information has been anonymized to ensure that individuals cannot be identified, thus protecting patient privacy. This study was reviewed and approved by the Ethics Committee of Xianju People's Hospital and was deemed eligible for exemption from informed consent; therefore, we hereby apply for exemption from informed consent.

Inclusion criteria were: (1) successful PCI and complete clinical and laboratory data related to myocardial injury biomarkers; (2) 12-month follow-up after the procedure. Exclusion criteria were: (1) end-stage renal disease, defined as estimated glomerular filtration rate (eGFR) <15 mL/min/1.73 m^2 or requiring long-term dialysis [11]; (2) exposure to iodine contrast agent within 1 week before or 48 hours after PCI; (3) missing clinical data such as alanine aminotransferase (ALT) and AST ; (4) use of nephrotoxic drugs within 1 week before or 48 hours after PCI; (5) death within 24 hours after admission.

Follow-up information was collected through multiple sources such as the hospital's electronic medical record system. The primary follow-up endpoint was the occurrence of major adverse cardiovascular events within 12 months post-surgery. MACE was defined as a composite endpoint of cardiovascular death, non-fatal myocardial infarction, heart failure requiring hospitalization, or repeat coronary revascularization [12]. For patients experiencing multiple events, only the first recorded event during the follow-up period was included in the analysis.

Table 1. Baseline characteristics of participants.

Variables	Non-MACE group (n = 223)	MACE group (n = 43)	Statistic	*p*
Age (years)	55.26 ± 7.88	58.45 ± 10.83	$t = -1.84$	0.072
HDL (mmol/L)	1.21 ± 0.28	1.26 ± 0.19	$t = -1.67$	0.100
LDL (mmol/L)	2.84 ± 0.73	2.76 ± 1.38	$t = 0.39$	0.700
AST (U/L)	37.96 (19.49, 52.19)	41.21 (27.02, 56.92)	Z = –0.84	0.400
ALT (U/L)	20.15 (14.00, 24.90)	21.74 (17.01, 26.97)	Z = –1.22	0.221
CK-MB (ng/mL)	65.15 (39.75, 94.46)	93.08 (54.95, 134.78)	Z = –3.30	<0.001
cTnI (ng/mL)	22.15 (13.68, 32.31)	33.94 (19.66, 45.95)	Z = –3.68	<0.001
WBC ($\times 10^9$/L)	7.50 (5.70, 9.25)	8.20 (5.70, 9.70)	Z = –1.34	0.180
GLU (mmol/L)	7.89 (5.81, 9.79)	8.14 (6.18, 9.35)	Z = –0.02	0.983
Hospital stay (days)	7.00 (5.00, 10.00)	8.00 (6.00, 9.00)	Z = –0.41	0.681
LVEF, n (%)			$\chi^2 = 1.29$	0.255
>50%	119 (53.36)	27 (62.79)		
≤50%	104 (46.64)	16 (37.21)		
Implanted stent number, n (%)			$\chi^2 = 2.25$	0.690
No stent	12 (5.38)	3 (6.98)		
I	93 (41.70)	16 (37.21)		
II	69 (30.94)	14 (32.56)		
III	34 (15.25)	9 (20.93)		
≥IV	15 (6.73)	1 (2.33)		
Gender, n (%)			$\chi^2 = 0.96$	0.326
Female	71 (31.84)	17 (39.53)		
Male	152 (68.16)	26 (60.47)		
Smoking, n (%)			$\chi^2 = 0.15$	0.700
No	172 (77.13)	32 (74.42)		
Yes	51 (22.87)	11 (25.58)		
Drinking, n (%)			$\chi^2 = 0.67$	0.414
No	183 (82.06)	33 (76.74)		
Yes	40 (17.94)	10 (23.26)		
Dyslipidemia, n (%)			$\chi^2 = 6.25$	0.012
No	139 (62.33)	18 (41.86)		
Yes	84 (37.67)	25 (58.14)		
Diabetes, n (%)			$\chi^2 = 0.10$	0.750
No	176 (78.92)	33 (76.74)		
Yes	47 (21.08)	10 (23.26)		
Hypertension, n (%)			$\chi^2 = 2.53$	0.112
No	132 (59.19)	31 (72.09)		
Yes	91 (40.81)	12 (27.91)		
Cardiac arrhythmia, n (%)			$\chi^2 = 0.00$	0.972
No	181 (81.17)	35 (81.40)		
Yes	42 (18.83)	8 (18.60)		

MACE, major adverse cardiovascular events; HDL, high-density lipoprotein; LDL, low-density lipoprotein; AST, aspartate aminotransferase; ALT, alanine aminotransferase; CK-MB, creatine kinase-MB; TnI, troponin I; WBC, white blood cell count; GLU, glucose; LVEF, left ventricular ejection fraction.

Collection of Biomarkers

Basic clinical data of enrolled patients were collected through the hospital's electronic medical record system, including age, gender, total length of hospital stay, smoking history, alcohol consumption history, history of hypertension, history of diabetes, history of dyslipidemia, and history of arrhythmia. Smoking and alcohol consumption history was defined as past or current regular smoking or drinking habits; the diagnosis of hypertension, diabetes, dyslipidemia, LVEF, implanted stent number and arrhythmia was based on relevant clinical guidelines and the patient's past medical history [13–15]. Fasting venous blood samples were collected from all patients within 24 hours post-surgery for testing. Serum AST, ALT, CK-MB, and cTnI levels were measured using a fully automated biochemical analyzer. Fasting blood glucose (GLU), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were also measured. White blood cell count (WBC) was determined using a fully automated hematology analyzer (Sysmex, Kobe Japan).

Table 2. Results of univariate logistic regression analysis.

Variables	β	SE	Z	p	OR (95% CI)
Gender					
Female					1.00 (Reference)
Male	–0.336	0.343	–0.98	0.327	0.71 (0.36~1.40)
LVEF, n (%)					
>50%					1.00 (Reference)
≤50%	0.126	0.397	0.319	0.750	1.135 (0.52~2.47)
Implanted stent number, n (%)					
≥IV					1.00 (Reference)
No stent	1.322	1.218	1.09	0.278	3.75 (0.34~40.81)
I	0.948	1.068	0.89	0.375	2.58 (0.32~20.92)
II	1.113	1.074	1.04	0.300	3.04 (0.37~24.96)
III	1.379	1.099	1.26	0.209	3.97 (0.46~34.20)
Smoking					
No					1.00 (Reference)
Yes	0.148	0.384	0.38	0.700	1.16 (0.55~2.46)
Drinking					
No					1.00 (Reference)
Yes	0.327	0.401	0.81	0.415	1.39 (0.63~3.04)
Dyslipidemia					
No					1.00 (Reference)
Yes	0.832	0.339	2.46	0.014	2.30 (1.18~4.46)
Diabetes					
No					1.00 (Reference)
Yes	0.126	0.397	0.32	0.750	1.13 (0.52~2.47)
Hypertension					
No					1.00 (Reference)
Yes	–0.577	0.366	–1.58	0.115	0.56 (0.27~1.15)
Cardiac arrhythmia					
No					1.00 (Reference)
Yes	–0.015	0.428	–0.04	0.972	0.99 (0.43~2.28)
Age	0.045	0.020	2.24	0.025	1.05 (1.01~1.09)
AST	0.004	0.007	0.60	0.550	1.00 (0.99~1.02)
ALT	0.022	0.022	1.00	0.320	1.02 (0.98~1.07)
CK-MB	0.015	0.004	3.91	<0.001	1.02 (1.01~1.02)
cTnI	0.045	0.011	4.19	<0.001	1.05 (1.02~1.07)
WBC	0.105	0.082	1.28	0.201	1.11 (0.95~1.30)
HDL	0.829	0.643	1.29	0.197	2.29 (0.65~8.08)
LDL	–0.113	0.193	–0.58	0.559	0.89 (0.61~1.30)
GLU	–0.008	0.063	–0.12	0.904	0.99 (0.88~1.12)
Hospital stay	0.021	0.066	0.32	0.747	1.02 (0.90~1.16)

OR, odds ratio; 95% CI, 95% confidence interval.

Data Processing and Analysis

Data processing and statistical analysis were performed using SPSS 24.0 (IBM Corp., Armonk, NY, USA) and R 4.2.2 (R Foundation for Statistical Computing, Vienna, Austria). The Shapiro-Wilk test was used to determine the normality of continuous variables. For normally distributed continuous variables, the data were presented as mean ± standard deviation. Levene's test was used to assess the homogeneity of variances, and the independent samples *t*-test (or Welch's *t*-test when variances were unequal) was used to compare differences between two groups. Skewed continuous variables were expressed as median [M (Q_1, Q_3)], and the Mann-Whitney U test was used for comparisons between groups. Categorical variables were described as frequencies (percentages). When the theoretical frequency T is greater than or equal to 5 and the sample size N is greater than or equal to 40, the chi-square test is used; when $1 \leq T < 5$ and N is greater than or equal to 40, the continuous correction chi-square test is employed; when T is less than 1, N is less than 40, or there

is a sample size of 0, Fisher's exact test is adopted. To avoid model overfitting, the widely adopted "one in ten" rule was followed, which recommends a minimum of 10 outcome events per independent variable in logistic regression analysis. To analyze the association between myocardial injury markers and adverse outcomes after PCI, a univariate logistic regression model was first used to assess the correlation between each variable and the outcome event. Variables with p-values < 0.05 in the univariate analysis were then entered into a multivariate logistic regression model using the backward stepwise selection method (likelihood ratio-based, with removal criterion set at $p > 0.10$) to correct for confounding factors and identify independent association factors. Before fitting the multivariate model, collinearity among the independent variables was assessed using the variance inflation factor (VIF). A VIF value <5 was considered no significant multicollinearity. The odds ratio (OR) and its 95% confidence interval (95% CI) were calculated. Receiver operating characteristic (ROC) curves were used to analyze the ability of each myocardial injury marker and their combination to identify adverse outcomes after PCI, and the area under the curve (AUC) was calculated to evaluate their discriminative performance. Statistical inferences were performed using two-tailed tests, with $p < 0.05$ considered statistically significant.

Results

Baseline Data Analysis

This study ultimately included 266 patients, with 223 (83.83%) in the non-MACE group and 43 (16.17%) in the MACE group. There were no statistically significant differences between the two groups in terms of age, sex, smoking history, alcohol consumption history, history of diabetes, history of hypertension, history of arrhythmia, or levels of HDL, LDL, AST, ALT, WBC, GLU, LVEF, implanted stent number and length of hospital stay ($p > 0.05$). However, regarding myocardial injury markers and comorbidities, the MACE group had significantly higher levels of CK-MB and cTnI than the non-MACE group, and the proportion of patients with dyslipidemia was also significantly higher in the MACE group ($p < 0.05$). Detailed baseline characteristics are shown in Table 1.

Univariate Logistic Regression Analysis

Using the occurrence of MACE within 12 months after PCI as the dependent variable, various clinical characteristics and laboratory indicators were analyzed using a univariate logistic regression model. Results showed that age (OR = 1.05, 95% CI: 1.01–1.09, $p = 0.025$), dyslipidemia (OR = 2.30, 95% CI: 1.18–4.46, $p = 0.014$), CK-MB (OR = 1.02, 95% CI: 1.01–1.02, $p < 0.001$), and cTnI (OR = 1.05, 95% CI: 1.02–1.07, $p < 0.001$) were significantly associated with the risk of MACE after PCI. However, gender, smoking history, alcohol consumption history, history of diabetes, history of hypertension, history of arrhythmia, as well as AST, ALT, WBC, HDL, LDL, GLU, LVEF, implanted stent number and length of hospital stay were not significantly associated with the risk of MACE ($p > 0.05$). The results of the univariate logistic regression analysis are detailed in Table 2.

Table 3. Results of multivariate logistic regression analysis.

Variables	β	SE	Z	p	VIF	OR (95% CI)
Dyslipidemia						
No						1.00 (Reference)
Yes	0.859	0.373	2.30	0.021	1.014	2.36 (1.14~4.91)
Age	0.038	0.022	1.70	0.089	1.019	1.04 (0.99~1.08)
CK-MB	0.015	0.004	3.39	<0.001	1.034	1.01 (1.01~1.02)
cTnI	0.040	0.011	3.49	<0.001	1.025	1.04 (1.02~1.06)

VIF, variance inflation factor.

Multivariate Logistic Regression Analysis

Variables with p-values < 0.05 in univariate analysis (age, dyslipidemia, CK-MB, cTnI) were included in a multivariate logistic regression model for analysis. The collinearity analysis showed that the variance inflation factor (VIF) for each variable was less than 5, indicating no significant multicollinearity among the independent variables (results are shown in Table 3). After adjusting for confounding factors, CK-MB (OR = 1.01, 95% CI: 1.01–1.02, $p < 0.001$), cTnI (OR = 1.04, 95% CI: 1.02–1.06, $p < 0.001$), and comorbid dyslipidemia (OR = 2.36, 95% CI: 1.14–4.91, $p = 0.021$) remained independently associated with the risk of MACE after PCI. Age was not statistically significant in the multivariate analysis (OR = 1.04, 95% CI: 0.99–1.08, $p = 0.089$). The results of the multivariate logistic regression analysis are detailed in Table 3.

Discriminative Ability of the Combined Factors

The results showed that the combined AUC of dyslipidemia, CK-MB, and cTnI was 0.75 (95% CI: 0.66–0.84), suggesting a moderate discriminatory ability for the occurrence of MACE. Based on the principle of maximizing Youden's index, the combination showed a sensitivity of 88.0% and a specificity of 78.0% in identifying MACE. The ROC curve is shown in Fig. 1.

Discussion

This study, based on single-center retrospective data, systematically analyzed the relationship between various myocardial injury-related biomarkers and postoperative clinical outcomes after PCI. The results showed that in patients undergoing PCI, postoperative myocardial injury-related marker levels were significantly associated with the occurrence of MACE within 12 months of follow-up. These results suggest that the extent of perioperative myocardial injury not only reflects the potential myocardial damage

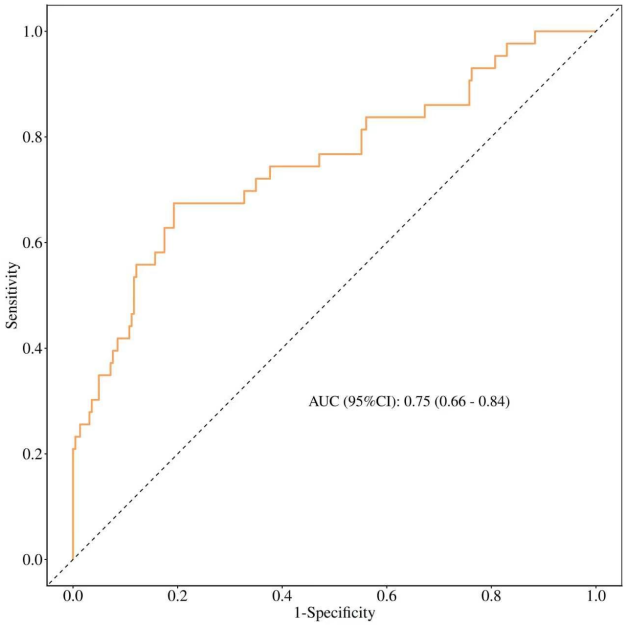

Fig. 1. Receiver operating characteristic (ROC) curve of the combination of dyslipidemia, creatine kinase-MB (CK-MB), and cardiac troponin I (cTnI).

during the procedure but may also, to some extent, reflect the patient's overall postoperative cardiovascular risk.

PCI has become an important treatment for coronary artery disease, particularly acute coronary syndrome; however, a percentage of patients still experience adverse cardiovascular events during follow-up after PCI [16–18]. These events not only affect the long-term prognosis but also increase the burden on medical resources. Therefore, it is of great significance to identify patients who may be at high risk and implement targeted management in clinical practice. Among the various biomarkers that can be used to assess myocardial injury, CK-MB and cardiac troponin are currently the most frequently used indicators in clinical practice [19,20]. Cardiac troponin has high myocardial specificity and sensitivity, and is widely regarded as an important biomarker for the detection of myocardial injury. Previous studies have shown that elevated cardiac troponin levels detected after PCI often indicate perioperative myocardial injury, and there is a correlation between the degree of elevation and subsequent clinical events in patients to some extent [21]. Some large cohort studies have also shown that even mild elevations in myocardial markers may indicate a broader range of cardiomyocyte damage or impaired microcirculation perfusion, which may have a potential impact on the recovery of cardiac function in patients [22–24]. The results of this study are generally consistent with previous studies, namely that patients with higher levels of myocardial injury markers after PCI had a relatively higher rate of MACE during follow-up, and that CK-MB levels were associated with adverse postoperative outcomes. This may be explained by the fact that increased CK-MB is correlated with the extent of myocardial necrosis, and its changes may reflect the disruption of cardiomyocyte membrane integrity [25].

In addition to traditional myocardial-specific markers, this study also included routine biochemical indicators such as AST for analysis. AST is widely present in various tissues such as myocardium, liver and skeletal muscle, and can be released into the bloodstream when cells are damaged or necrotic [26]. The results of this study showed that there was no statistically significant difference in AST levels between the MACE group and the non-MACE group, and no significant association was found between AST and the risk of MACE within 12 months after PCI in the logistic regression analysis. This result suggests that compared with myocardial-specific markers such as CK-MB and cTnI, AST may have relatively limited value in reflecting PCI-related myocardial injury and long-term clinical outcomes. This may be because the sensitivity and specificity of non-specific enzyme indicators in the assessment of myocardial injury are lower than those of myocardial-specific biomarkers.

In addition to laboratory indicators, this study also analyzed the relationship between various individual baseline characteristics and postoperative clinical outcomes after PCI. The results suggested that a history of dyslipidemia was significantly associated with the occurrence of MACE after PCI. Dyslipidemia is one of the important risk factors for the formation and development of coronary atherosclerosis. Long-term lipid metabolism disorders can lead to the deposition of low-density lipoprotein in the vascular wall, promote inflammatory response and plaque formation, and increase plaque instability [27,28]. Even after revascularization with PCI, the underlying pathological process of atherosclerosis may persist, thereby increasing the risk of future cardiovascular events. Therefore, in patients with dyslipidemia, long-term lipid management and lifestyle modification remain necessary after PCI to reduce the risk of atherosclerosis progression and adverse cardiovascular events.

From a clinical perspective, the results of this study have practical significance. First, CK-MB and cTnI are both routine tests after PCI, and their detection is convenient and results are obtained quickly. Analyzing the levels of these indicators helps to better understand the perioperative myocardial injury in patients. Second, this study found an association between elevated levels of myocardial injury markers and the occurrence of MACE after PCI, suggesting that patients with significantly elevated myocardial enzyme levels may require closer observation and management during follow-up. Furthermore, the association between dyslipidemia and adverse outcomes also suggests that continuous control of blood lipid levels remains crucial in the long-term management of PCI patients.

Of course, this study still has certain limitations. First, this is a single-center retrospective study with a relatively

limited sample size, and the results may be affected by selection bias. Second, the variables included in this study are relatively limited; some factors that may affect the prognosis after PCI, such as the complexity of coronary artery lesions, stent type, and perioperative drug therapy, could not be further analyzed in this study. Furthermore, this study primarily explores the association between myocardial injury markers and clinical outcomes; future multi-center, large-sample studies are needed to further validate the relevant results.

Conclusion

In conclusion, this study indicates that in patients undergoing PCI, the levels of postoperative myocardial injury-related biomarkers are associated with the occurrence of major adverse cardiovascular events within 12 months of follow-up. Routine indicators such as CK-MB and cTnI may reflect perioperative myocardial injury and overall stress status to some extent, and may be of reference value for understanding the postoperative clinical outcomes of PCI patients. Further multicenter prospective studies will help to elucidate the clinical significance of these indicators in the long-term management of PCI patients.

Availability of Data and Materials

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

YQL and XQW designed the research study. XQW and LPW performed the research. YQL and HYL analyzed the data. XQW and LPW drafted the article. All authors contributed to the important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

This retrospective study was approved by the Ethics Committee of Xianju People's Hospital (NO.2026-35). The research was carried out in compliance with principles outlined in the Declaration of Helsinki. Due to the retrospective nature of the study and the use of anonymized clinical data, the requirement for informed consent was waived by the ethics committee.

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Conflict of Interest

The authors declare no conflict of interest.

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