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Article

Predictive Performance of Serum Monomeric C-Reactive Protein for Major Adverse Cardiovascular Events in Patients With Acute ST-Segment Elevation Myocardial Infarction at One-Year Post-Discharge: A Retrospective Cohort Study

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Abstract

Aims/Background: Patients with acute ST-segment elevation myocardial infarction (STEMI) are at high risk of major adverse cardiovascular events (MACE) even after discharge, underscoring the need to identify reliable biomarkers. Therefore, this study aimed to investigate the predictive performance of serum monomeric C-reactive protein (mCRP) for MACE in patients with acute STEMI at one-year post-discharge. **Methods**: A retrospective analysis was conducted on 242 patients with acute STEMI who underwent emergency Percutaneous Coronary Intervention (PCI) in Liyang City People's Hospital between January 2021 and December 2023. Patients were divided into the MACE group (n = 58) and the non-MACE group (n = 184) based on major adverse cardiovascular events. Univariate and binary logistic regression analyses were performed to identify factors influencing MACE events in patients with acute STEMI treated with emergency PCI. Furthermore, predictive performance was assessed using receiver operating characteristic curve (ROC) analysis. **Results**: There were no statistically significant differences ($p > 0.05$) between the groups in terms of age, gender, body mass index (BMI), smoking history, alcohol consumption history, history of hypertension, left ventricular end-diastolic diameter (LVEDD), total cholesterol (TC), triglycerides (TG), B-type natriuretic peptide (BNP), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and left ventricular end-diastolic volume (LVEDV). Comparisons of mCRP (within 24 hours of admission), left ventricular ejection fraction (LVEF), and cardiac troponin I (cTnI) showed statistically significant differences ($p < 0.05$). Monomeric CRP demonstrated a positive correlation with cTnI levels ($r = 0.196$, $p < 0.05$). Binary logistic regression analysis identified mCRP, LVEF, and cTnI levels as independent predictors of one-year post-discharge MACE in patients with acute STEMI ($p < 0.05$). ROC analysis yielded an area under the curve of 0.840 for mCRP (standard error = 0.028; 95% CI, 0.785–0.896, $p < 0.001$). At the Youden index of 0.50, mCRP demonstrated a sensitivity of 77.59%, and a specificity of 72.83%. **Conclusion**: Monomeric CRP exhibits superior predictive performance for MACE in acute STEMI patients at one-year post-discharge.

Keywords: C-reactive protein; ST-segment; cardiovascular diseases

1. Introduction

Acute ST-segment elevation myocardial infarction (STEMI) is one of the leading causes of death globally [1]. Recent research has highlighted monomeric C-reactive protein (mCRP) as a potential biomarker in cardiovascular diseases [2]. During inflammation, the native CRP dissociates into monomeric form, mCRP, which exerts potent pro-inflammatory effects. In atherosclerosis, a cardiovascular disease primarily driven by chronic inflammation [3,4]. mCRP is closely associated with cardiovascular diseases, depositing abundantly in atherosclerotic plaques and infarcted myocardial and cerebral tissues, thereby aggravating lesion formation [5,6]. Functionally, mCRP activates the complement system and enhances the phagocytosis of pathogens and damaged cells, highlighting its crucial role in innate immunity [7]. However, a persistent increase in CRP contributes to endothelial cell dysfunction and promotes vascular inflammation, enhancing atherogenesis and elevating the risk of acute cardiovascular events. Additionally, systemic CRP increases have also been associated with intestinal inflammatory diseases, such as ulcerative colitis. During these pathological conditions, CRP disrupts mucosal immune homeostasis and microbial balance, further elevating systemic inflammation and endothelial damage, thereby increasing the risk of acute cardiovascular events.

Techniques like enzyme-linked immunosorbent assay (ELISA) and immunofluorescence (IF) are commonly used for quantifying plasma mCRP levels, but chemiluminescence assays have recently become the method of choice, demonstrating excellent stability, sensitivity and specificity [8,9]. Moreover, changes in mCRP levels after myocardial infarction are complex and dynamic, with various temporal

patterns observed across patient subgroups and influenced by individual variability and disease severity [10].

We hypothesize that mCRP, due to its direct involvement in plaque instability, demonstrates superior predictive potential for major adverse cardiovascular events (MACE) compared with conventional biomarkers (e.g., CRP). To test this assumption, we conducted a comprehensive analysis of serum mCRP dynamics in STEMI patients, compared these changes with established clinical indicators, and explore their diagnostic potential in acute myocardial infarction. Furthermore, we correlated mCRP levels with echocardiography, cardiac magnetic resonance imaging, and other detection methods, as well as with long-term clinical outcomes. By monitoring patients for one year after discharge, this analysis aims to elucidate the prognostic utility of mCRP in STEMI and to provide enhanced approaches for risk stratification and patient management.

2. Methods

2.1 Research Subjects

For the modeling group, the sample size was determined using the equation $N = Z_{\alpha/2}{}^2\pi(1\text{-}\pi)/\delta^2$, where the incidence ($\pi$) of MACE after STEMI was 8.3% [11], the significance level (α) was 0.05, and the allowable error (δ) was 0.06. This yielded a minimum required sample size of 82. Considering a 30% rate of invalid samples or missing data, the sample size increased to 99. Thus, data from 300 patients were collected for screening.

Out of the total samples, 242 patients with acute STEMI who underwent emergency Percutaneous Coronary Intervention (PCI) at Liyang City People's Hospital between January 2021 and December 2023 were included in the retrospective analysis. Patients were divided into two groups based on major adverse cardiovascular events during follow-up: the MACE group (n = 58) and the non-MACE group (n = 184).

This study followed the principles the Declaration of Helsinki and was approved by the Medical Ethics Committee of Liyang City People's Hospital (2025027). Informed consent was obtained from all participants or their families throughout the study. A flow chart of patient selection and screening is illustrated in Fig. 1.

2.1.1 Inclusion Criteria

The diagnosis of STEMI followed the American College of Cardiology/American Heart Association criteria [12]. Eligible patients presented within 12 hours of symptom onset, were between 18 and 80 years, underwent emergency PCI, and had complete clinical data.

2.1.2 Exclusion Criteria

Patient exclusion criteria included (1) diagnosis of rheumatic or other autoimmune diseases; (2) presence of malignant tumors; (3) severe illnesses with an expected survival of less than 1 year; (4) complications resulting from other diseases; (5) refusal to participate in the study.

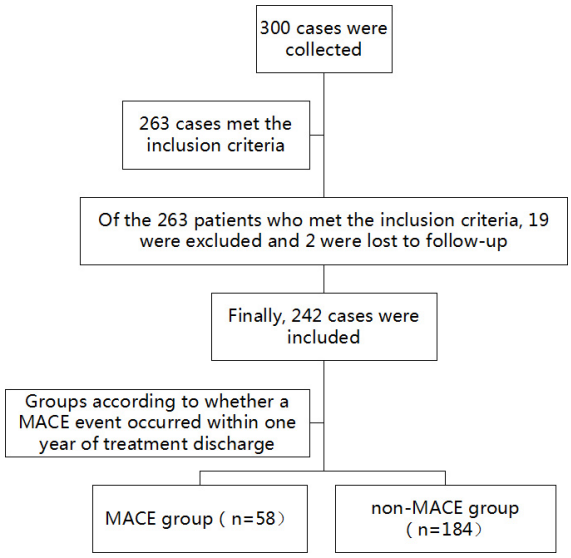

Fig. 1. A flow chart of patient selection. Note: After screening, 242 patients were finally included and divided into two groups: the MACE group (n = 58) and the non-MACE group (n = 184). MACE, major adverse cardiovascular events.

2.1.3 Withdrawal Criteria

As an observational study assessing the relationship between mCRP levels and cardiac function in STEMI patients, this analysis poses no risk of harm to patients. No withdrawal criteria were established, except for loss to follow-up or voluntary withdrawal by the patient.

2.1.4 Research Termination Criteria

This observational study poses no intervention-related risk nor effects the patient's health, thus no predetermined termination criteria were required.

2.1.5 Research Intervention Measures

This study is purely observational and involves no interventions or drug therapies; thus, there were no restrictions on participant's medication regimens.

2.2 Evaluation Indicators

Patients were scheduled for at least two follow-up visits after discharge, at 1 month and 1 year after discharge. Patient baseline characteristics, including age, gender, and medical history, were collected from the hospital's electronic medical records system.

2.2.1 Measuring mCRP Levels

Within 24 hours of admission, 5 mL of venous blood was collected from each patient. After centrifugation at 3000 r/min for 10 minutes, the resulting supernatant was collected and stored at –80 °C until analysis. mCRP levels

were assessed using an ELISA kit (Kit No. HBP36523R, Shanghai Huabang, Shanghai, China) following the manufacturer's instructions. Frozen samples were equilibrated to room temperature for 1 hour, and all kit reagents (stored at 4 °C) were equilibrated at room temperature for 30 minutes. Standards were prepared by serial two-fold dilution of 400 µg/mL stock to form 200, 100, 50, 25, and 12.5 µg/mL. In each reaction plate, the blank well received only substrate (developer A and B solutions) and stop solutions, the standard well received 50 µL of each standard and the sample well received 40 µL of sample diluent and 10 µL of the sample. Plates were sealed and incubated at 37 °C for 30 minutes. Distilled water was used to dilute the concentrated washing solution 30 times for later use. Then the sealing film of plates was removed, the liquid was discarded, and the sample well was spined dry. Each hole was filled with diluted washing solution and kept still for half a minute, with discarding reaction plate, which was repeated 5 times and then patted dry. After that, 50 µL of enzyme conjugate was added to all wells except the blank wells and incubated at 37 °C for 30 minutes. After washing, each 50 µL of developer A and developer B were added and incubated again at 37 °C in the dark for 15 minutes. The reaction was terminated with 50 µL stop solution per well, and absorbance at 450 nm was determined using a microplate reader within 15 minutes. Based on the concentration and optical density (OD) value of the standard, the linear regression equation was calculated. The OD value of the sample was measured and subsequently applied to the standard regression equation to calculate the preliminary concentration, which was then multiplied by the corresponding dilution factor to determine the final analyte concentration.

2.2.2 Cardiac Function Indicators

Cardiac function was assessed before surgery using echocardiography and cardiac magnetic resonance imaging (MRI). Key parameters, such as left ventricular end-diastolic diameter (LVEDD), left ventricular ejection fraction (LVEF), and left ventricular end-diastolic volume (LVEDV), were determined to evaluate myocardial recovery after infarction.

2.2.3 Follow-Up for MACE Events at One-Year Post-Discharge

Major adverse cardiovascular events (MACE) were categorized into primary and secondary endpoint events. The primary endpoint included cardiac death, non-fatal myocardial infarction, non-fatal stroke, and unplanned revascularization procedures such as PCI or coronary artery bypass grafting (CABG). The secondary endpoint events consisted of recurrent myocardial infarction, repeat emergency revascularization procedures, sudden cardiac arrest, heart failure manifestations, cardiogenic shock, malignant arrhythmias (such as tachycardia/ventricular fibrillation, sinus arrest, high-grade or third-degree atrioventricular block), mechanical complications arising from myocardial infarction, stroke, and severe bleeding characterized as a hemoglobin drop of less than 3 g/L.

2.2.4 Secondary Indicators

At each follow-up, patients were assessed for additional inflammatory and cardiac markers like B-type natriuretic peptide (BNP) and cardiac troponin I (cTnI). Furthermore, data on other relevant clinical and laboratory indicators, such as triglyceride levels, were also recorded.

2.2.5 Quality Control

To minimize confounding factors, relevant baseline data were collected at admission before and after any medication started. For analytical consistency and quality assurance, ELISA procedures were performed following the manufacturer's instructions, instruments were regularly calibrated, and each sample was tested in duplicate. To minimize potential biases in follow-up data collection, researchers received standardized training, a well-structured questionnaire was used, and all information was cross-verified.

2.3 Statistical Analysis

All statistical analyses were performed using SPSS 27.0 (International Business Machines Corporation, Armonk, NY, USA). Normality in the data was evaluated using the Shapiro-Wilk test. Continuous variables following a normal distribution were expressed as mean ± standard deviation ($\bar{X} \pm S$) and compared using independent sample *t*-tests. Moreover, the skewed variables were reported as median and interquartile range (IQR), and analyzed using the Mann-Whitney U. Categorical data was presented as counts and evaluated with chi-square tests. Multicollinearity was assessed through linear regression variance inflation factors, and the correlation between variables was examined using Spearman's rank test. Univariate and binary logistic regression analyses were conducted to identify factors that influenced the occurrence of MACE events in patients with acute STEMI treated with emergency PCI at one-year post-discharge. Furthermore, receiver operating characteristic curve (ROC) curves were generated to compare the diagnostic performance of mCRP versus other indicators. Missing data were excluded listwise deletion, and statistical significance was determined at a p-value of <0.05.

3. Results

3.1 Univariate Analysis and Comparison of Factors Influencing MACE

No significant differences were found between the two groups regarding age, gender, body mass index (BMI), smoking history, alcohol consumption history, LVEDD, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), BNP, high-density lipoprotein cholesterol (HDL-C), hypertension his-

Table 1. Univariate analysis of factors affecting MACE.

Indicator	MACE group (n = 58)	Non-MACE group (n = 184)	$Z/t/\chi^2$ value	*p*-value
Age (years)	55.50 (41.00, 65.25)	55.00 (46.00, 64.00)	–0.515	0.606
Gender			0.159	0.690
Male	32	96		
Female	26	88		
BMI (kg/m^2)	21.53 (20.28, 22.82)	21.52 (20.19, 22.80)	–0.044	0.965
Hypertension history			2.883	0.090
Yes	16	32		
No	42	152		
Smoking history			0.865	0.352
Yes	15	37		
No	43	147		
Alcohol consumption history			1.006	0.316
Yes	17	42		
No	41	142		
LVEDD (mm)	52.99 (51.81, 54.67)	53.13 (51.62, 54.55)	–0.122	0.903
mCRP (μg/mL)	338.38 (315.54, 348.85)	299.01 (277.62, 319.69)	–7.814	<0.001
LVEF (%)	52.65 (44.82, 58.29)	55.48 (50.70, 59.20)	–3.158	0.002
BNP (ng/L)	120.01 (113.73, 130.60)	118.85 (114.72, 123.29)	–1.873	0.061
cTnI (μg/L)	40.06 ± 2.53	36.06 ± 3.87	7.384	<0.001
TC (mmol/L)	4.38 (3.95, 5.01)	4.26 (3.86, 4.72)	–1.239	0.215
TG (mmol/L)	1.53 ± 0.17	1.50 ± 0.17	1.172	0.242
LDL-C (mmol/L)	1.45 (1.33, 1.59)	1.48 (1.34, 1.62)	–0.827	0.408
HDL-C (mmol/L)	2.12 (1.85, 2.41)	2.15 (1.93, 2.45)	–1.107	0.268
LVEDV (mL)	47.89 (46.62, 49.57)	47.50 (45.96, 49.27)	–1.150	0.250

Note: BMI, body mass index; mCRP, monomeric C-reactive protein; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; BNP, B-type natriuretic peptide; cTnI, cardiac troponin I; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; LVEDV, left ventricular end-diastolic volume; MACE, major adverse cardiovascular events.

Table 2. Correlation analysis of clinical variables.

		LVEDD	mCRP	LVEF	cTnI	LVEDV	BNP
LVEDD	*r*	1.000	–0.004	–0.058	–0.010	0.000	0.067
	p	-	0.953	0.369	0.873	0.998	0.301
mCRP	*r*	–0.004	1.000	–0.112	0.196	0.083	0.070
	p	0.953	-	0.082	0.002	0.197	0.275
LVEF	*r*	–0.058	–0.112	1.000	0.020	0.054	–0.075
	p	0.369	0.082	-	0.760	0.400	0.244
cTnI	*r*	–0.010	0.196	0.020	1.000	0.111	0.025
	p	0.873	0.002	0.760	-	0.086	0.703
LVEDV	*r*	0.000	0.083	0.054	0.111	1.000	–0.039
	p	0.998	0.197	0.400	0.086	-	0.542
BNP	*r*	0.067	0.070	–0.075	0.025	–0.039	1
	p	0.301	0.275	0.244	0.703	0.542	-

tory, or LVEDV (all $p > 0.05$). Conversely, the two groups demonstrated substantial differences in mCRP, LVEF, and cTnI levels ($p < 0.05$, Table 1).

3.2 Variable Correlation Analysis

Spearman's correlation analysis revealed that mCRP was positively correlated with cTnI (r = 0.196, $p < 0.05$). Spearman's correlation analysis is detailed in Table 2.

Table 3. Variable assignment.

Influencing factor	Assignment
mCRP	Original value
LVEF	Original value
cTnI	Original value

3.3 Binary Logistic Regression Analysis of Factors Affecting MACE

Significant variables from univariate analysis (Table 1) were introduced into a multivariate binary logistic regression analysis. mCRP, LVEF, and cTnI were included as independent variables, and the occurrence of MACE at one-year post-discharge as the dependent variable (occurred = 1, not occurred = 0). Findings demonstrated mCRP, LVEF, and cTnI as significant predictors of MACE in acute STEMI patients at one-year post-discharge ($p < 0.05$, Tables 3,4).

3.4 ROC Analysis to Assess Predictive Performance of Indicators

ROC analysis yielded an area under the curve of 0.840 for mCRP (standard error = 0.028; 95% CI, 0.785–0.896, $p < 0.001$). At the optimal cutoff identified by a Youden

Table 4. Binary logistic regression analysis of factors influencing MACE.

Factor	β	SE	Wald	p-value	Exp (β)	95% CI	
						Lower limit	Upper limit
mCRP	0.068	0.012	32.485	<0.001	1.071	1.046	1.096
LVEF	–0.171	0.049	12.409	<0.001	0.843	0.766	0.927
cTnI	0.543	0.107	25.582	<0.001	1.722	1.395	2.125
Constant	–46.079	7.605	36.714	<0.001	0.000	-	-

Table 5. ROC analysis of predictive performance evaluation.

Indicator	Area under curve	Standard error	95% CI	Youden	Sensitivity	Specificity	p-value	Cutoff value
mCRP	0.840	0.028	0.785–0.896	0.50	77.59	72.83	<0.001	314.02 (μg/mL)
LVEF	0.638	0.045	0.547–0.726	0.36	68.97	67.39	0.002	57.86 (%)
cTnI	0.780	0.030	0.721–0.840	0.31	31.03	99.50	<0.001	36.20 (μg/L)

Note: ROC, receiver operating characteristic.

index of 0.50, mCRP demonstrated 77.59% sensitivity and 72.83% specificity. ROC analyses are presented in Table 5 and Fig. 2.

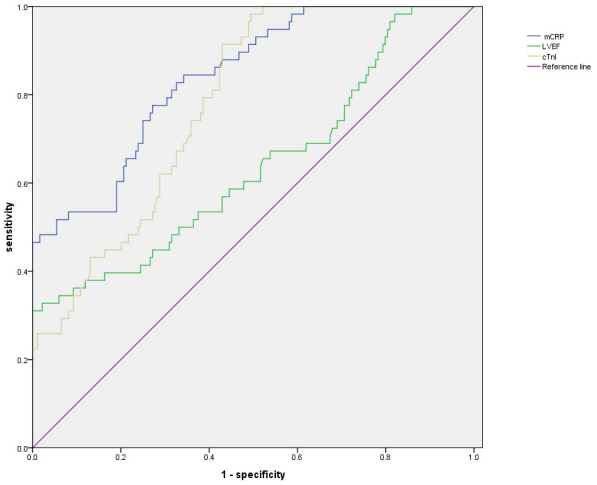

Fig. 2. ROC curve analysis of predictive performance. Note: mCRP showed the highest area under the curve (AUC), indicating its best predictive performance.

4. Discussion

STEMI is a high-mortality disease. Although emergency PCI has significantly reduced in-hospital deaths, long-term post-discharge outcomes continued to be undermined by MACE, which potentially impacts quality of life. Therefore, identifying reliable biomarkers to predict MACE is crucial for guiding treatment and improving patient outcomes.

We observed that mCRP, left ventricular ejection fraction (LVEF), and cardiac troponin I (cTnI) levels differed substantially between patients with and without MACE, and binary logistic regression analysis confirmed these three variables (mCRP, LVEF, and cTnI) as independent predictors of one-year MACE after STEMI. These observations highlight the prognostic significance of mCRP and underscore the role of LVEF and cTnI in this condition. As indicators of cardiac function and injury, decreased LVEF and increased BNP are associated with a poor prognosis in heart failure [13]. Conversely, persistently elevated cTnI indicates ongoing myocardial injury and reflects elevated MACE risk [14]. A study by Huang *et al.* [15] identified LVEF and cTnI as independent predictors of in-hospital MACE after initial PCI in acute STEMI patients, aligning closely with our findings, further substantiating this study. Furthermore, Spearman's correlation analysis demonstrated a modest positive correlation between mCRP and cTnI ($r = 0.196$), suggesting that elevated mCRP levels coincide with lower cardiac function and higher myocardial damage [10]. Given cTnI's sensitivity and specificity for cardiomyocyte injury, the correlation between mCRP and cTnI further supports the mCRP's potential as a cardiovascular biomarker [16].

Previously, Wang *et al.* [10] reported circulating mCRP as a potential biomarker for diagnosing acute myocardial infarction (AMI) and assessing disease severity in a mouse model [10]; however, this study lacked clinical validation. To evaluate the predictive performance of mCRP for MACE, this study conducted ROC curve analysis and achieved an area under the curve (AUC) of 0.840 (95% CI: 0.785–0.896), indicating good predictive accuracy. At a maximum Youden index of 0.50, mCRP demonstrated a sensitivity of 77.59% and a specificity of 72.83%. These findings suggest that mCRP exhibits high sensitivity and specificity in predicting MACE events in acute STEMI patients at one-year post-discharge. These results further indicate that assessing mCRP levels can accurately identify STEMI patients at higher risk of MACE and inform more precise management. Mechanistically, mCRP en-

hances inflammation, accelerates lipid accumulation, and disrupts endothelial integrity during antherogenesis [17]. Furthermore, another study suggested mCRP's predictive performance in viascular dysfunction [2], further supporting the results of our study. Additionally, mCRP may exacerbate myocardial damage and heart failure through mechanisms such as complement system activation, promoting thrombus formation, and inducing cell apoptosis. Therefore, treatment approaches aimed to reduce serum mCRP levels could offer novel avenues for preventing and treating cardiovascular diseases.

Monomeric CRP is superior to conventional CRP by more accurately reflecting the local inflammatory microenvironment and atherosclerotic plaque stability. While conventional CRP serve as a non-specific marker of inflammation, mCRP, a cleavage product of CRP, directly participates in atherosclerosis and is associated closely with MACE risk factors such as plaque rupture and thrombosis. Previous research has shown that inflammation accelerates the progression of atherosclerosis and increases the risk of MACE through endothelial dysfunction, oxidative stress, and immune cell infiltration [13]. As a crucial mediator linking inflammation to atherosclerosis, mCRP helps identify high-risk individuals earlier. Therefore, we recommend incorporating mCRP into existing cardiovascular risk scores to improve risk assessment and formulate personalized treatment plans. Intervention strategies for mCRP include anti-inflammatory approaches, such as statin therapy to lower blood lipids and reduce inflammation, or novel agents targeting specific inflammatory pathways. Additionally, lifestyle modifications such as diet optimization, regular exercise, smoking cessation, and alcohol restriction can reduce mCRP levels and hence, alleviate MACE risk. By targeting mCRP using these integrated approaches, we aim to reduce the burden of inflammation-driven cardiovascular diseases and improve long-term patient prognosis.

Although this study highlights mCRP's predictive performance for MACE, it is not the only influencing factor. In clinical practice, each patients' clinical presentations, laboratory results, imaging findings, and other relevant information are crucial to developing personalized treatment plans. Furthermore, as a retrospective study with a limited sample size, the findings may be subject to selection bias and residual confounding; prospective studies are needed to validate these observations. Currently, there is no definitive research evidence on how to effectively reduce serum mCRP levels and improve outcomes. While preliminary reports suggest that anti-inflammatory drugs, statins, and lifestyle interventions may help minimize mCRP levels, their precise efficacy and mechanisms remain unexplored. Future large-scale, multicenter, randomized controlled trials are needed to evaluate the impact of different interventions on mCRP reduction and their effect on cardiovascular prognosis.

5. Conclusion

In conclusion, this retrospective analysis of acute STEMI patients treated with emergency PCI confirms that serum mCRP levels reliably predict the occurrence of MACE at 1-year after discharge. As a novel cardiovascular biomarker, elevated mCRP levels indicate impaired cardiac function and worsening myocardial damage. Incorporating serum mCRP levels into acute STEMI risk-scoring system could enhance prognostic accuracy and better inform treatment decisions. However, its optimal threshold and weight need validation in prospective, multicenter investigations. Future randomized trials should assess targeted anti-inflammatory approaches (such as statins and colchicine) and combine them with antioxidant therapies to examine whether decreasing mCRP levels can be translated into clinical advantages. Furthermore, mechanistic investigations are required to elucidate how mCRP contributes to disease progression and how its levels can be effectively reduced. Additionally, the predictive performance and therapeutic potential of mCRP in cardiovascular diseases should be validated through prospective study designs.

Key Points

- This study investigated mCRP's predictive performance on MACE in STEMI patients at one-year post-discharge.
- Univariate and binary logistic regression analyses identified mCRP, LVEF, and cTnI levels as independent predictors affecting MACE progression in patients with acute STEMI.
- We observed a positive correlation between modified mCRP and cTnI levels ($r = 0.196$, $p < 0.05$), indicating a potential link between inflammation and myocardial damage.
- Monomeric CRP demonstrated superior predictive performance for MACE, with an area under the curve of 0.840, a sensitivity of 77.59%, and a specificity of 72.83%, underscoring its potential as a reliable biomarker for risk stratification in STEMI patients.

Availability of Data and Materials

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

Author Contributions

YZ and JHW designed the research study. CYD, YYS and ZYF performed the research. SW and YZ analyzed the data. YZ drafted the manuscript. All authors contributed to important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study is in accordance with the Declaration of Helsinki. The study was approved by the Medical Ethics Committee of Liyang City People's Hospital (2025027). The principle of informed consent was followed throughout the experiment, and information about the study was provided to patients or their families, and consent was obtained.

Acknowledgment

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

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