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Original Article

Correlation of serum MAp44 protein concentration with degree of myocardial injury and 30-day prognosis in patients with acute myocardial infarction

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Abstract: Objective: To analyze the relationship between serum mannan-binding lectin-associated protein 44 (MAp44) level and myocardial injury severity in acute myocardial infarction (AMI) patients, and explore its value for 30-day prognostic assessment. Methods: A retrospective study included 128 AMI patients admitted to The First Hospital of Zhangjiakou from January 2023 to June 2024, divided into mild (n=57) and severe myocardial injury groups (n=71). General data and MAp44 levels were compared. For 30-day prognosis, patients were stratified into a major adverse cardiovascular event (MACE, n=37) and a non-MACE group (n=91). Univariate and multivariate analyses identified poor prognostic factors, and predictive values of indicators were compared. An independent cohort (82 AMI patients, February-October 2025) validated the model. Results: The severe group had significantly lower MAp44 and higher cardiac troponin T (cTnT) than the mild group (P<0.001). MAp44 was negatively correlated with cTnT (r=-0.666, P<0.001). The 30-day MACE incidence was 28.91%. Extensive infarction, ≥2 coronary lesions, elevated cTnT, and low MAp44 were MACE risk factors (P<0.05). The combined model (infarction size + coronary lesions + cTnT, and + MAp44) had the highest prognostic value (AUC=0.890, sensitivity =78.40%, specificity =83.50%), with 84.1% accuracy in the validation cohort. Conclusion: Serum MAp44 levels decrease with increasing myocardial injury severity in AMI patients; low MAp44 is an independent risk factor for 30-day MACE.

Keywords: Acute myocardial infarction, serum MAp44 protein, myocardial injury, prognosis

Introduction

The most common heart event in the world is acute myocardial infarction (AMI). The most common pathogenesis of AMI is the rupture or erosion of unstable coronary plaques, which leads to thrombus formation and persistent complete blockage of the coronary arteries [1]. This condition usually develops rapidly and deteriorates quickly. Blood clots in the blood vessels, plaque detachment, coronary artery stenosis, and vasospasm can all trigger the disease. If not treated in time, it can be life-threatening [2]. Global epidemiologic data estimate that there were approximately 197.2 million cases of AMI worldwide in 2019, of which 9.1 million died [3]. Trends show that the overall incidence and mortality of AMI in China are still rising, and the number of AMI patients in China will reach 23 million by 2030. Studies have shown that the 1-year, 3-year, and 5-year mortality rates of AMI patients are approximately 14.4%, 23.6%, and 30.0%, respectively [4]. Percutaneous coronary intervention is a common treatment for AMI patients. By clearing the blocked artery, blood can flow to the myocardium. Most patients recover well after the procedure, but in the short term, some patients will experience adverse cardiovascular events, affecting their prognosis [5]. Currently, the clinical methods used for prognostic assessment of AMI include the clinical trial score for thrombolytic therapy for myocardial infarction and the global acute coronary event registry score [6].

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However, these methods require a large number of examinations, are complex to operate and time-consuming, and have certain limitations. Therefore, the rapid and accurate determination of prognostic indicators for myocardial infarction has become a hot topic of clinical research. The main laboratory markers for clinical assessment of AMI are serum markers of myocardial injury, such as creatine kinase, creatine kinase isoenzymes, cardiac troponin and lactate dehydrogenase. Cardiac troponin has become the gold standard for diagnosing myocardial injury. It is an important serum regulatory protein complex in human myocardial contraction [7]. Mannose-binding lectin-associated protease 44 (MAP44) is a newly discovered non-enzymatic structural protein belonging to the mannose-binding lectin-associated serine protease (MASP) family [8]. It is expressed primarily in myocardial tissue, liver, skeletal muscle, and brain tissue. Serine protease inhibitors are key regulatory molecules that modulate and alleviate excessive inflammation and protease activity in tissues [9]. Studies have shown that plasma recombinant human MAp44 is involved in the development of cardiac tissue [10]. In many autoimmune diseases, MAp44 has been found to interact with complement components, leading to complement dysregulation and amplifying inflammatory responses [11]. These findings suggest that MAp44 may be a biomarker of disease states and a therapeutic target for alleviating complement-mediated diseases. Given the crucial role of the complement system and inflammation in the pathogenesis of myocardial ischemia-reperfusion injury following AMI [12, 13], we hypothesized that MAp44, as a key endogenous regulator of the lectin complement pathway, may reflect the extent of this injury and subsequent cardiac repair processes.

Blood proteins provide essential information about human health and can serve as biomarkers and drug targets. Compared to tissue-specific diseases, plasma proteins are directly exposed in blood vessels and therefore have important applications in the treatment of cardiovascular diseases [14]. Based on this, this study aimed to investigate the expression level of MAp44 in the serum of patients with AMI and analyze its correlation with the degree of myocardial injury and 30-day prognosis, so as to clarify the clinical value of MAp44 protein as a biomarker for assessing the severity of AMI and short-term prognosis. This will provide a new theoretical basis for optimizing risk stratification, early intervention, and improving prognosis of patients with AMI.

Materials and methods

Sample source

We conducted a retrospective study and selected 128 patients diagnosed with AMI and admitted to the First Hospital of Zhangjiakou from January 2023 to June 2024. These patients were divided into mild (n=57) and severe myocardial injury groups (n=71). To validate the predictive model, an additional 82 patients with AMI hospitalized between July 2024 and July 2025 were included as an external validation cohort. All included patients met the corresponding diagnostic criteria for AMI [15]. The clinical manifestations were recurrence and worsening of pre-existing angina symptoms, namely, sudden, severe, and persistent squeezing pain behind the sternum or in the precordial region, accompanied by a feeling of impending death, and reduced or ineffective nitroglycerin efficacy. The main imaging changes were the appearance of new Q waves, ST-segment elevation, and dynamic ST-T changes. This study has been approved by the Ethics Committee of the First Hospital of Zhangjiakou.

Sample size calculation

The sample size estimation formula was $N=Z^2\times[P\times(1-P)]/E^2$, where N is the total sample size, Z is the confidence interval (value 1.96), E is the sampling error range (value 0.1), and P is the probability value (value 0.3). Considering a sample loss rate of 20% to 30%, the sample size range is 97 to 105 cases. After comprehensive consideration, this study ultimately included 128 samples, which met the research criteria.

Inclusion and exclusion criteria

Inclusion criteria: (1) Meeting the diagnostic criteria for AMI and undergoing coronary angiography; (2) First-time diagnosis of AMI, with the time from onset to admission <24 hours; (3) Age between 18 and 80 years; (4) Complete clinical data. Exclusion criteria: (1) Mental disorder or cognitive impairment; (2) Congenital

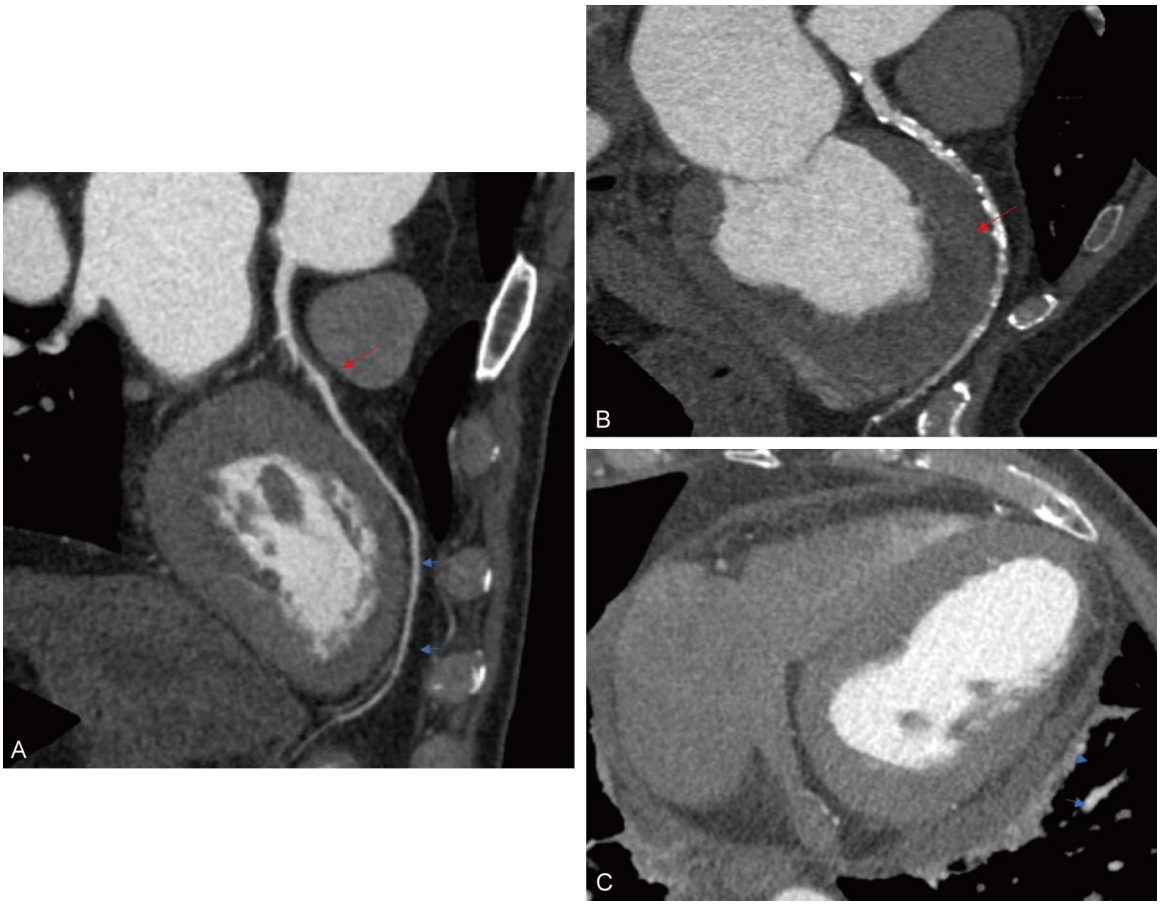

Figure 1. Representative coronary angiography and myocardial infarction imaging in AMI patients. A: 58-year-old male: Severe stenosis of proximal left anterior descending artery (LAD, red arrow) by non-calcified plaque; left ventricular subendocardial myocardial infarction (blue arrow, mild myocardial injury). B and C: 65-year-old male: Diffuse severe LAD stenosis (red arrow) by mixed plaques; left ventricular inferior wall transmural myocardial infarction (blue arrow, severe myocardial injury).

heart disease; (3) Malignant tumor; (4) Cerebrovascular disease; (5) Immune system disease; (6) Hematologic disease; (7) Severe liver and kidney dysfunction; (8) Refusal to participate in 30-day follow-up.

Determination of myocardial injury

Coronary angiography was performed on patients, and the criteria for evaluating the degree of myocardial injury were as follows: Mild myocardial injury was defined as infarct area <10% of left ventricular myocardium (assessed by quantitative coronary angiography [QCA]) and low density; contrast agent perfusion in the infarct area was significantly reduced (TIMI grade 2), but the area still retained a certain degree of systolic function (echocardiography showed left ventricular ejection fraction [LVEF] ≥50%). Severe myocardial injury was defined as infarct area ≥10% of left ventricular myocardium (QCA assessment) and high density; contrast agent perfusion in the infarct area was significantly reduced (TIMI perfusion grade 0-1) [16], or even completely absent, and ventricular wall motion was restricted or even completely absent (echocardiography showed LVEF<50%). A representative coronary angiography image and corresponding delayed gadolinium-enhanced cardiac MRI images showing the infarct size and transmural classification are shown in **Figure 1**.

Data collection

Patient information was collected, including gender, age, body mass index (BMI), hypertension, diabetes, hyperlipidemia, smoking history, alcohol consumption history, time from onset to admission, heart rate at admission, diastolic

Table 1. Comparison of general data of patients with different degrees of myocardial injury

Variable	Mild group (*n*=57)	Severe group (*n*=71)	Statistics	*P* value
Gender [n, (%)]			χ^2=0.019	0.891
Male	32 (56.14)	39 (54.93)		
Female	25 (43.86)	32 (45.07)		
Age (year)	52.46±9.39	54.27±9.00	*t*=-1.110	0.269
BMI (kg/m^2)	23.21±3.37	23.26±3.55	*t*=-0.072	0.943
Hypertension [n, (%)]	32 (56.14)	39 (54.93)	χ^2=0.019	0.891
Diabetes [n, (%)]	19 (33.33)	25 (35.21)	χ^2=0.049	0.824
Hyperlipidemia [n, (%)]	9 (15.79)	12 (16.90)	χ^2=0.029	0.866
Smoking [n, (%)]	21 (36.84)	18 (25.35)	χ^2=1.970	0.160
Drinking [n, (%)]	18 (31.58)	28 (39.44)	χ^2=0.848	0.357
MACE [n, (%)]	12 (21.05)	25 (35.21)	χ^2=3.08	0.079

Note: BMI, body mass index; MACE, major adverse cardiovascular events.

blood pressure at admission, systolic blood pressure at admission, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, infarct area, infarct location, number of coronary artery lesions, whether interventional treatment was received, cardiac troponin T (cTnT), and MAp44 levels.

30-day prognosis

All patients were followed up regularly every 30 days at the outpatient clinic or by telephone. The incidence of major adverse cardiovascular events (MACE) was recorded during the study period. MACE included recurrent myocardial infarction, severe heart failure, severe arrhythmia, cardiogenic shock, and death. Finally, 37 people were assigned to the MACE group, and 91 people were assigned to the non-MACE group.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation. Comparisons between groups were performed using t-tests or Mann-Whitney U tests. Categorical variables were described as frequencies and percentages. Differences between groups were analyzed using chi-square test and Fisher's exact test. Pearson correlation analysis was used to explore the correlation between MAp44 and cTnT levels, and logistic regression was used to analyze factors influencing poor patient prognosis. Relative operating characteristic curves (ROC) were used to evaluate the predictive value of each indicator, and Delong's tests were used to compare differences in predictive value. The performance difference between the predictive model and the gold standard was evaluated using McNemar's test. All statistical analyses were performed using SPSS 26.0 software. All statistical analyses were two-tailed tests. A *P* value <0.05 was considered significant.

Results

Comparison of general data of patients with different degrees of myocardial injury

General information was compared between patients with mild and severe myocardial injury. There were no statistically significant differences in the distribution of data regarding gender, age, body mass index (BMI), hypertension, diabetes, hyperlipidemia, smoking history, or alcohol consumption history between the two groups (all P>0.05) (**Table 1**). During the 30-day follow-up, the incidence of MACE in the severe injury group (35.21%) was higher than that in the mild injury group (21.05%), but the difference was not significant (P=0.079).

Comparison of MAp44 and cTnT levels among patients with different degrees of myocardial injury

The MAp44 level in the severe group (1.05±0.34 μg/ml) was lower than that of the mild group (1.71±0.67 μg/ml) (P<0.001) (**Figure 2**). The cTnT level in the severe group (3.10±0.53 ng/

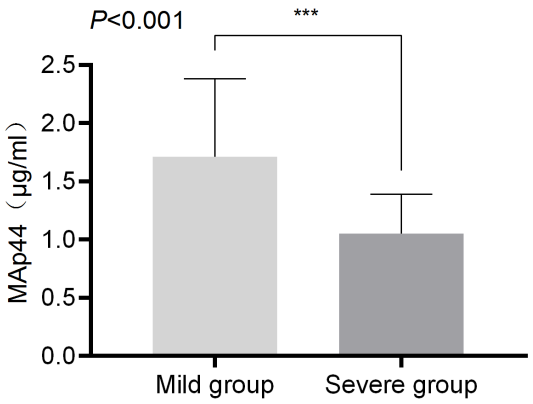

Figure 2. Comparison of Map44 between the mild group and severe group. Note: MAp44, Mannose-binding lectin-associated protease 44.

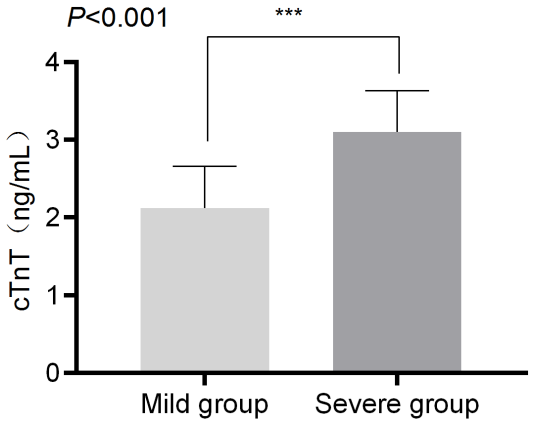

Figure 3. Comparison of cTnT between the mild group and severe group. Note: cTnT, cardiac troponin T. *** represents P<0.001.

mL) was higher than that of the mild group (2.12±0.54 ng/mL), (P<0.001) (**Figure 3**).

Comparison of MAp44 levels based on demographic and infarction-related factors

There were no significant differences in MAp-44 levels among patients stratified by sex, age, or BMI (all P>0.05), indicating that MAp44 expression is not influenced by these demographic and anthropometric factors (**Table 2**). MAp44 levels did not differ significantly between patients stratified by infarct size or infarct location (both P>0.05).

Correlation between MAp44 and cTnT levels

Pearson correlation analysis showed a significant negative correlation between MAp44 and cTnT (r=-0.666, P<0.001) (**Figure 4**).

MACE events

During a 30-day follow-up, 37 MACE events occurred in 128 patients with AMI, with an incidence rate of 28.91%. These included 8 cases of recurrent myocardial infarction, 12 cases of severe heart failure, 10 cases of severe arrhythmia, 5 cases of cardiogenic shock, and 2 deaths (**Figure 5**).

Univariate analysis of poor prognosis in patients

Comparing clinical data between the MACE group and the non-MACE group, the MACE group had a higher proportion of patients with large-area myocardial infarction (70.27%), a higher proportion of patients with coronary artery disease involving ≥2 vessels (37.84%), and a higher cTnT level (3.21 ±0.51) ng/mL, compared to (2.44±0.68) ng/mL in the non-MACE group. The MAP44 level in the MACE group (0.92±0.28) μg/ml was significantly lower than that in the non-MACE group (1.52±0.62) μg/ml (all P<0.05). However, there were no significant differences in the distribution of other data (all P>0.05) (**Table 3**).

Multivariate analysis of poor prognosis in patients

Using the occurrence of MACE within 30 days as the dependent variable (1: occurrence, 0: no occurrence), the significant factors by univariate analysis were used as independent variables. Definitions of categorical independent variables: (1) 1: large-area myocardial infarction; 0: small-area myocardial infarction; (2) 1: number of coronary artery lesions ≥2; 0: single-vessel lesion. cTnT and MAp44 were included in the multivariate logistic regression analysis with their raw values. The results showed that large-area infarction, coronary artery lesions ≥2, high cTnT levels, and low MAp44 levels were risk factors for MACE within 30 days (all P<0.05) (**Table 4**).

Predictive model for poor prognosis

The predictive model was constructed using multivariate logistic regression, with the following formula: Logit(P) = 1.878 × (infarct size) + 1.659 × (number of coronary artery lesions) + 1.553 × (cTnT) - 2.279 × (MAp44) - 4.26. Where

Table 2. Differences in MAp44 levels stratified by demographic characteristics, infarct size, and infarct location

Grouping		MAp44 (μg/ml)	T/F value	*P* value
Demographic characteristics				
Gender	Male (n=71)	1.41±0.61	1.322	0.189
	Female (n=57)	1.26±0.60		
Age (year)	<50 (n=40)	1.36±0.56	0.240	0.811
	≥50 (n=88)	1.34±0.63		
BMI (kg/m^2)	<24 (n=79)	1.36±0.60	0.371	0.711
	≥24 (n=49)	1.32±0.63		
Infarction situation				
Infarct area	Big (n=62)	1.34±0.55	0.03	0.976
	Small (n=66)	1.35±0.67		
Infarction location	Anterior interwall (n=62)	1.46±0.64	2.27	0.107
	Anterior side wall (n=26)	1.21±0.51		
	Extensive front wall (n=40)	1.25±0.60		

Note: MAp44, Mannose-binding lectin-associated protease 44; BMI, body mass index.

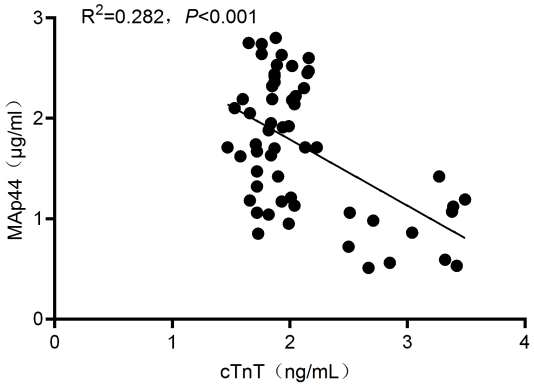

Figure 4. Correlation analysis between MAp44 and cTnT. Note: cTnT, cardiac troponin T; MAp44, Mannose-binding lectin-associated protease 44.

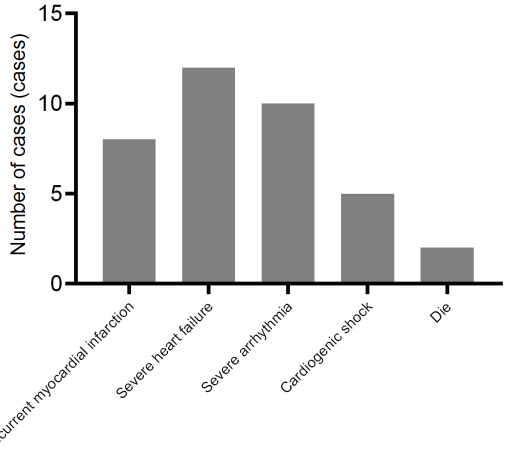

Figure 5. MACE incident situation. Note: MACE, major adverse cardiovascular events.

P is the probability of MACE, infarct area is coded as 1 for large area (left ventricle ≥10%) and 0 for small area (left ventricle <10%); lesions ≥2 are coded as 1 for presence and 0 for absence; cTnT is input in ng/mL and MAp44 in μg/mL. Comparing the prognostic predictive value of infarct area, number of coronary artery lesions, cTnT, MAp44, and the combined value of these four indicators, the combined prediction had the highest AUC of 0.890 (0.830-0.951), with a sensitivity of 78.40% and a specificity of 83.50%. Further Delong's testing was performed on the AUC values, comparing the combined value of the four indicators with each individual indicator (infarct area, number of coronary artery lesions, cTnT, MAP44) (all P<0.05) (**Tables 5** and **6**; **Figure 6**).

Clinical validation

External validation of the combined predictive model (including infarct area, number of coronary artery lesions, cTnT, and MAp44) was performed in an independent cohort of 82 patients. Of these, 25 were clinically diagnosed with MACE, and 57 were not MACE. The model identified 24 MACE-positive patients and 58 MACE-negative patients. The confusion matrix (**Table 7**) showed that the model correctly predicted 18 true positives and 51 true negatives,

Table 3. Univariate analysis of poor prognosis

Variable	MACE group (*n*=37)	Non-MACE group (*n*=91)	Statistics	*P* value
Gender [n, (%)]			χ^2=0.336	0.562
Male	22 (59.46)	49 (53.85)		
Female	15 (40.54)	42 (46.15)		
Age (year)	52.84±9.16	53.71±9.24	*t*=-0.488	0.626
BMI (kg/m^2)	23.16±3.51	23.27±3.45	*t*=-0.148	0.883
Hypertension [n, (%)]	18 (48.65)	53 (58.24)	χ^2=0.980	0.322
Diabetes [n, (%)]	13 (35.14)	31 (34.07)	χ^2=0.013	0.908
Hyperlipidemia [n, (%)]	6 (16.22)	15 (16.48)	χ^2=0.001	0.970
Smoking [n, (%)]	10 (27.03)	29 (31.87)	χ^2=0.291	0.590
Drinking [n, (%)]	12 (32.43)	34 (37.36)	χ^2=0.278	0.598
Time from onset to admission (h)	3.24±1.38	3.27±1.41	*t*=-0.115	0.908
Heart rate (times/min)	92.14±15.08	91.84±15.78	*t*=0.099	0.922
DBP (mmHg)	70.81±13.24	71.05±13.20	*t*=-0.095	0.925
SBP (mmHg)	107.35±17.52	106.54±17.45	*t*=0.239	0.812
TC (mmol/L)	3.46±0.62	3.44±0.65	*t*=0.161	0.872
TG (mmol/L)	1.09±0.21	1.11±0.23	*t*=-0.464	0.644
HDL-C (mmol/L)	0.82±0.23	0.84±0.21	*t*=-0.333	0.740
LDL-C (mmol/L)	2.43±0.64	2.41±0.64	*t*=0.164	0.870
Infarct area [n, (%)]			χ^2=9.933	0.002
Big	26 (70.27)	36 (39.56)		
Small	11 (29.73)	55 (60.44)		
Infarction location [n, (%)]			χ^2=1.437	0.488
Anterior interwall	15 (40.54)	47 (51.65)		
Anterior side wall	8 (21.62)	18 (19.78)		
Extensive front wall	14 (37.84)	26 (28.57)		
Number of coronary artery lesions [n, (%)]			χ^2=9.875	0.002
1	23 (62.16)	79 (86.81)		
≥2	14 (37.84)	12 (13.19)		
Interventional therapy [n, (%)]	22 (59.46)	61 (67.03)	χ^2=0.662	0.416
cTnT (ng/mL)	3.21±0.51	2.44±0.68	*t*=6.289	<0.001
MAp44 (μg/ml)	0.92±0.28	1.52±0.62	*t*=-5.674	<0.001

Note: MACE, major adverse cardiovascular events; BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; cTnT, cardiac troponin T; MAp44, Mannose-binding lectin-associated protease 44.

Table 4. Multifactorial analysis of poor prognosis

Variable	*β* value	*SE*	*Wald* χ^2	*P* value	*OR* (95%*CI*)
Infarct area	1.878	0.568	10.930	0.001	6.539 (2.148-19.907)
Number of coronary artery lesions	1.659	0.644	6.632	0.010	5.252 (1.486-18.556)
cTnT	1.553	0.526	8.708	0.003	4.727 (1.685-13.262)
MAp44	-2.279	0.898	6.448	0.011	0.102 (0.018-0.595)

Note: SE, standard error; OR, odds ratio; CI, confidence interval; cTnT, cardiac troponin T; MAp44, Mannose-binding lectin-associated protease 44.

yielding a sensitivity of 72.0% and a specificity of 89.5%. The overall accuracy of the model was 84.1%, demonstrating its robust clinical applicability.

Table 5. The value of different indicators in prognosis prediction

Variable	AUC	95% CI	*P* value	Cut off	Youden index	Sensitivity (%)	Specificity (%)
Infarct area	0.654	0.550-0.757	0.007		0.307	70.30	60.40
Number of coronary artery lesions	0.623	0.510-0.736	0.029		0.246	37.80	86.80
cTnT	0.803	0.729-0.878	<0.001	2.48 ng/mL	0.528	94.60	58.20
MAp44	0.802	0.725-0.880	<0.001	1.205 μg/ml	0.446	91.90	52.70
Joint indicator	0.890	0.830-0.951	<0.001		0.784	78.40	83.50

Note: AUC, area under curve; CI, confidence interval; cTnT, cardiac troponin T; MAp44, Mannose-binding lectin-associated protease 44.

Table 6. Delong test of the ROC curve

Variable	AUC difference	*SE*	95%*CI*	*Z*	*P* value
Infarct area-Joint indicator	-0.237	0.275	-0.323- -0.151	-5.383	<0.001
Number of coronary artery lesions-Joint indicator	-0.267	0.272	-0.354- -0.181	-6.049	<0.001
cTnT-Joint indicator	-0.087	0.260	-0.148- -0.026	-2.796	0.005
MAp44-Joint indicator	-0.088	0.264	-0.167- -0.009	-2.190	0.029

Note: AUC, area under curve; SE, standard error; CI, confidence interval; cTnT, cardiac troponin T; MAp44, Mannose-binding lectin-associated protease 44.

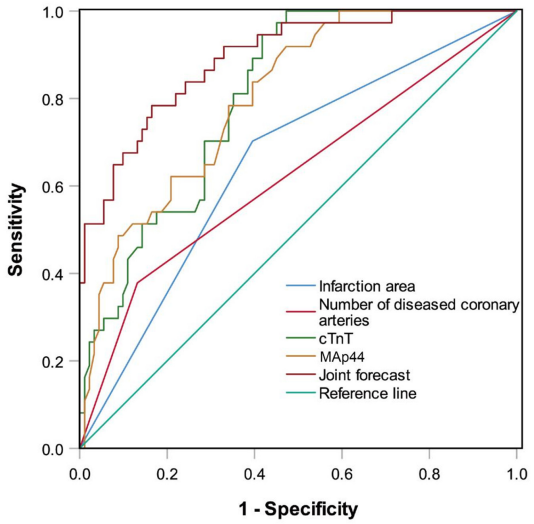

Figure 6. ROC curve analysis of predictive efficacy. Note: ROC, receiver operating characteristic; cTnT, cardiac troponin T; MAp44, Mannose-binding lectin-associated protease 44.

Table 7. Clinical validation of the predictive model

Predictive model	Clinical diagnosis		Total
	MACE	Non-MACE	
MACE	18	6	24
Non-MACE	7	51	58
Total	25	57	
P value[a]			1.00

Note: MACE, major adverse cardiovascular events. [a]P: McNemar's test.

Discussion

AMI has a complex pathologic mechanism and poses a serious threat to patients' lives [17]. Early risk stratification of AMI is crucial for guiding treatment intensity and follow-up strategies to prevent MACE. In blood indicators, this manifests as abnormalities in myocardial enzyme components. However, in the clinical diagnosis of myocardial injury, myocardial enzymes have limitations in assessing clinical condition due to their long half-lives. MAp44 is an endogenous inhibitor of the lectin complement pathway and is highly expressed in cardiac tissue [18, 19]. Its potential role in cardiovascular disease stems from its ability to modulate complement-mediated inflammation, a key driver of ischemia-reperfusion injury [20]. We therefore hypothesized that circulating MAp44 levels might reflect the degree of myocardial damage and repair capacity post-AMI.

This study compared the levels of MAp44 and cTnT in patients with mild and severe myocardial injury. The results showed that the severe group had lower MAp44 levels and higher cTnT levels. When AMI occurs, myocardial ischemia-reperfusion triggers complement system activation, releasing a large number of inflammatory factors and oxygen free radicals, leading to myocardial cell necrosis [20]. MAp44 can inhibit excessive activation of the complement system by competing with MASP-2 to bind lectins,

thereby reducing the damage to myocardial cells caused by the inflammatory response. Hertle et al. [21] found that the expression level of recombinant human MAp44 in plasma was positively correlated with endothelial dysfunction, and that it had a function in endothelial activation independent of the lectin pathway. cTnT is a specific marker of myocardial cell necrosis. That is, the lower the MAp44 level, the more severe the myocardial cell necrosis, and the more cTnT is released. The results of this study showed that there was no significant difference in MAp44 levels between male and female subgroups in different gender, age, or BMI subgroups. This may be because the core function of MAp44 is to inhibit excessive activation of the complement system, and its synthesis and release are primarily regulated by pathological signals such as myocardial ischemia and inflammatory factors, rather than directly influenced by gender-related hormones or metabolic indicators [22]. In addition, studies have shown that MAp44 concentrations do not differ between sexes or exhibit significant diurnal variations. The study also found that capillary concentrations of MAp44 were highly consistent with those in venous blood samples [23]. These findings may be helpful for future research on the lectin pathway in infants and young children.

In recent years, with the advancement of treatment technology, the mortality rate of AMI has decreased, but long-term observation has found that some patients still experience MACE. It has been reported that MACE increases the instability of atherosclerotic plaques, aggravates myocardial damage, and increases the risk of adverse outcomes [24]. Therefore, focusing on screening high-risk groups for MACE and taking active preventive measures is an important way to reduce the incidence of MACE and improve patient prognosis. This study found that large infarction, ≥2 coronary artery lesions, high cTnT levels, and low MAp44 levels were risk factors for MACE within 30 days. The infarct area and the number of diseased vessels are positively correlated with the severity of the patient's condition; as the infarct area expands and the number of diseased vessels increases, the patient's cardiac contractility decreases, and the condition becomes more severe, making MACE events more likely to occur [25]. In the study by Alusik et al. [26], the cardiac compensatory capacity of patients with large myocardial infarction decreased and the cardiac pumping function was insufficient. cTnT is a specific structural protein within cardiomyocytes, that is released into the bloodstream in large quantities only when cardiomyocytes die or their cell membrane integrity is compromised. Numerous studies have suggested that high cTnT levels increase the risk of MACE in patients with AMI through mechanisms such as expanding the extent of myocardial necrosis, triggering inflammation and oxidative stress, suggesting the severity of coronary artery disease, and disrupting myocardial electrophysiological stability [27, 28]. Previous studies have shown that MASP-1 and MASP-2 contribute to the formation of fibrin clots in vitro, and MASP-1 is crucial for obstructive thrombosis in a mouse model of arterial injury [29]. In contrast, MAp44 may have a protective effect against cardiovascular disease because it can reduce infarct size and prevent ischemia/reperfusion injury [30]. Furthermore, preliminary explorations have been conducted on the expression changes and clinical significance of MAp44 in other diseases, and its role as a regulator of inflammation and complement systems may have a universality across diseases. Patients with variant immunodeficiency had MAp44 levels 0.87 times lower than normal [31]. The lectin pathway is altered in patients with head and neck cancer, and MAp44 levels in cancer patients are lower than in healthy individuals [32]. Michalski et al. [33] investigated factors related to the initiation and regulation of the complement lectin pathway influencing the prognosis of pediatric cardiopulmonary bypass surgery and found that patients with low MAp44 levels had a higher risk of complications such as postoperative low cardiac output syndrome, renal insufficiency, and systemic inflammatory response syndrome. However, in the study by Frauenknecht et al. [34], the levels of MASP-1 and MASP-2 in circulating plasma of patients with cardiovascular disease were altered, while the levels of MASP-3 and MAp44 did not show differences, which contradicts the results of this study. This may be related to differences in disease type and pathologic stage, sample selection, confounding factors, and the sensitivity of the detection methods.

The results of this study have several potential clinical implications. First, combining serum

MAp44 detection with established biomarkers (such as cTnT) may allow for a more nuanced assessment of myocardial injury severity, moving beyond infarct size assessment alone. The significant negative correlation between the two suggests that reduced MAp44 may indicate excessive complement activation and impaired intrinsic protective function, thus identifying high-risk pathophysiologic phenotypes. Second, and more importantly, MAp44 has become an independent predictor of 30-day MACE. Incorporating it into a multifactor model (including infarct size, multivessel disease, and cTnT) significantly improved the accuracy of prognostic assessment (AUC of 0.890). This model was validated in independent cohorts, providing a practical tool for early risk stratification. In clinical practice, patients identified as high-risk through such models can be targeted for more intensive surveillance, earlier and more aggressive secondary prevention, or consideration of novel therapies designed to modulate complement pathways. The stability of MAp44 levels across different demographic characteristics (sex, age, BMI) further supports its practicality as a reliable biomarker, unaffected by these common variables.

However, this study had limitations. It was a single-center retrospective study with a relatively small sample size, potentially leading to selection bias. Furthermore, it only followed up on short-term prognosis over 30 days, failing to reflect its association with long-term prognosis. Future research should conduct multi-center, large-sample prospective studies, including AMI patients from different regions and with diverse population characteristics, to validate the universality of MAp44 as a prognostic biomarker and extend the follow-up period to one year or longer to clarify the association between MAp44 and the long-term risk of MACE and long-term prognosis in AMI patients.

Conclusion

The results of this study indicate that serum MAp44 levels in AMI patients are associated with the severity of myocardial injury. Patients with severe myocardial injury have lower MAp44 levels and higher cTnT levels, and MAp44 is negatively correlated with cTnT. Multivariate analysis showed that large infarct area, ≥2 coronary artery lesions, high cTnT levels, and low MAp44 levels were independent risk factors for MACE within 30 days in AMI patients. In addition, the combined predictive model of infarct area, number of coronary artery lesions, cTnT, and MAp44 achieved an AUC of 0.890, demonstrating a high predictive value. External validation in an independent patient cohort confirmed the model's high accuracy, supporting the clinical application of MAp44 as part of a multi-biomarker strategy for risk stratification in AMI patients. Combined detection of multiple indicators helps optimize clinical risk stratification and individualized intervention strategies.

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Disclosure of conflict of interest

None.

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