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ORIGINAL RESEARCH

Risk Factors for Cardiac Rupture After Acute Myocardial Infarction and Development of a Risk Prediction Model

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Aim: To investigate the influencing factors for acute myocardial infarction (AMI) complicated by cardiac rupture (CR),evaluate the predictive value of the systemic inflammation response index (SIRI), and construct a clinically practical risk prediction model.

Methods: A total of 53 AMI patients complicated with CR admitted to Tianshui First People's Hospital from January 2013 to December 2023 were enrolled as the CR group.During the same period, 159 AMI patients without CR were selected as the control group at a 1:3 ratio, matched for age and sex.Baseline data, clinical indicators, and laboratory test results of patients in both groups were collected, and SIRI was calculated. Lasso regression was used to screen core variables, multivariate Logistic regression analysis was performed to identify independent influencing factors, a nomogram prediction model was constructed based on key variables, and the receiver operating characteristic (ROC) curve was used to evaluate the model's efficacy.

Results: Multivariate Logistic regression analysis showed that admission heart rate (OR = 1.050,95% CI = 1.024–1.075, P < 0.001), Killip classification (OR = 2.092,95% CI = 1.460–2.997, P < 0.001) and SIRI (OR = 1.105,95% CI = 1.022–1.196, P = 0.012) were independent risk factors for CR in AMI patients. Primary PCI (OR = 0.239,95% CI = 0.097–0.589, P = 0.002) and taking ACEI / ARB drugs within 24 hours (OR = 0.173,95% CI = 0.060–0.500, P = 0.001) were protective factors. The ROC curve model constructed based on the above five indicators has an area under the curve (AUC) of 0.885.

Conclusion: Admission heart rate, Killip classification, and systemic inflammatory response index are independent risk factors for AMI with CR. Primary PCI and the administration of ACEI/ARB within 24 hours of admission were identified as protective factors against CR. The nomogram model demonstrated good predictive value for the occurrence of cardiac rupture in patients with AMI.

Keywords: acute myocardial infarction, cardiac rupture, systemic inflammation response index, predictive factors, nomogram

Introduction

With the acceleration of global population aging and the westernization of residents' lifestyles, cardiovascular diseases (CVDs) have emerged as one of the leading causes of mortality and disability both in China and worldwide.[1,2] Epidemiological studies by Du et al[3] have shown that the prevalence of CVDs in China continues to rise, posing a heavy burden on public health. The Global Burden of Disease study indicates that between 1990 and 2019,[4] the incidence and mortality rates of CVD in China increased by 93.75% and 57.39%, respectively. As the most critical subtype of cardiovascular disease,AMI is not only a leading cause of death among the Chinese population but also the primary cause of both out-of-hospital and in-hospital cardiac sudden death.[5] Although reperfusion therapy has become widely available, CR—one of the most lethal mechanical complications of AMI—remains the third leading cause of early in-hospital death among AMI patients.[6,7] The American Heart Association (AHA) 2021 scientific statement, which integrates data from multiple large-scale international AMI registries (eg., NRMI, ESC EORP-AMI, SWEDEHEART),[8] indicates that the incidence of CR after AMI is approximately 0.5%–2.0% in the contemporary era of widespread percutaneous coronary intervention (PCI). The situation regarding the prevention and treatment of cardiac rupture is particularly dire in northwestern China, where cardiovascular risk

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factors are poorly controlled and medical resources are relatively scarce.[9,10] Therefore, early identification of high-risk individuals is crucial for further reducing in-hospital mortality from AMI.

Inflammation plays a central role in cardiac remodeling and the development of CR following AMI.[11,12] The systemic inflammatory response triggered by myocardial ischemia and necrosis after AMI can,[13–15] through imbalances in the number and function of inflammatory cells, affect myocardial collagen metabolism and compromise ventricular wall stability, thereby indirectly increasing the risk of CR.[16] The Systemic Inflammation Response Index (SIRI) integrates neutrophil, lymphocyte, and monocyte counts,[17,18] compared to factors based on changes in only two indices—the neutrophil-to-lymphocyte ratio (NLR) which reflects the ratio of neutrophils to lymphocytes, and the platelet-to-lymphocyte ratio(PLR), which reflects the relative change in platelets to lymphocytes—it can simultaneously reflect the degree of pro-inflammatory cell accumulation and the suppression of anti-inflammatory cells, thereby more accurately assessing inflammation-immune imbalance. This index has demonstrated prognostic value in various malignant tumors,[19,20] but evidence regarding its predictive role in AMI complicated by CR remains lacking.[21]

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In view of this, this study retrospectively analyzed the clinical data of AMI patients admitted to Tianshui First People's Hospital (a single center) over a 10-year period (January 2013—December 2023). The aims were to explore the independent risk factors for AMI complicated by CR, focus on evaluating the predictive value of SIRI for CR in AMI patients, and construct a nomogram prediction model based on clinically accessible indicators. This study intends to provide a scientific basis for clinicians to early identify high-risk groups for CR and formulate individualized diagnosis and treatment strategies, thereby providing new ideas for reducing the incidence of CR and improving the long-term prognosis of AMI patients.

Materials and Methods

Study Population

This was a single-center retrospective observational analysis. The study subjects were patients with AMI admitted to Tianshui First People's Hospital from January 2013 to December 2023. During the study period, 53 patients diagnosed with AMI complicated by CR on echocardiography were enrolled as the CR group. Another 159 AMI patients without CR hospitalized during the same period were selected as the control group, matched 1:3 for age and sex.This study protocol was in compliance with the ethical principles of the Declaration of Helsinki and approved by the Ethics Committee of Tianshui First People's Hospital. As a retrospective observational study, the clinical data of patients were anonymized without additional medical burden, and informed consent was waived by the Ethics Committee after review.

Inclusion and Exclusion Criteria

Inclusion Criteria

① Age ≥60 years; ② Diagnosed with ST-segment elevation myocardial infarction (STEMI) upon admission in accordance with the 2022 American College of Cardiology (ACC)/European Society of Cardiology (ESC) Guidelines for the Management of STEMI; ③ Patients in the CR group were confirmed to have CR by echocardiography (characteristic findings such as myocardial wall discontinuity and pericardial effusion) during hospitalization, while those in the non-CR group had no CR during hospitalization and 30-day follow-up after discharge; ④ Complete clinical and laboratory data.

Exclusion Criteria

① CR not caused by AMI (eg., trauma, infective endocarditis, aortic dissection involving the heart, etc).; ② Diseases or factors affecting neutrophil, lymphocyte, or monocyte counts (eg., hematological diseases, malignant tumors, autoimmune diseases, active infections, systemic inflammatory response syndrome, long-term (≥3 months) use of glucocorticoids or immunosuppressants, etc).; ③ Missing key clinical or laboratory indicators that affect the integrity of study data.

Data Collection and Processing

Relevant patient information was extracted from the hospital's electronic medical record system and laboratory information system, including:

Baseline Data

Age, gender, smoking history (defined as smoking ≥1 cigarette per day for ≥1 year), past stroke history.

Laboratory Indicators

Peripheral venous blood was collected within 24 hours after admission to detect white blood cell count (WBC), neutrophil count (N count), lymphocyte count (L count), monocyte count (M count), neutrophil percentage (N%), lymphocyte percentage (L%), hemoglobin (Hb), platelet count (PLT), and serum creatinine (SCr). The systemic inflammation response index (SIRI) was calculated as SIRI = (neutrophil count × monocyte count) / lymphocyte count (all cell count units in $\times 10^9$/L).

Comorbidities

Hypertension (HT), diabetes mellitus (DM), dyslipidemia, heart failure (HF), shock, prior myocardial infarction (prior-MI), and past stroke history.

Treatment Measures

Usage of angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker (ACEI/ARB), β-blocker, and other drugs, as well as reperfusion therapy such as percutaneous coronary intervention (PCI) and thrombolysis.

Information regarding infarct location, the number of diseased coronary vessels (single vessel or multivessel disease), and the specific site of coronary artery lesions was obtained from coronary angiography and medical records.

Statistical Analysis

Statistical analyses were performed using SPSS (version 22.0) and R software (version 4.0.0; "rms" for nomogram, "pROC" for ROC curve, "rmda" for DCA).Missing data. Variables with a missing rate >35% were excluded. For continuous variables with missing rate <5%, multiple imputation was applied; for categorical variables with missing rate <5%, mode imputation was used. Normally distributed variables were expressed as mean±SD ($\bar{x} \pm s$) (independent-samples *t*-test for intergroup comparison), and non-normal variables as median (IQR) [M (IQR)] (Mann–Whitney *U*-test). Categorical variables were presented as n (%) (Pearson's χ^2-test). Lasso regression was performed with 10-fold cross-validation. The optimal penalty parameter λ was selected using the lambda.1se criterion (λ=0.073), which balances model fit and parsimony. Variables with non zero coefficients were retained. These selected variables were then entered into a multivariate logistic regression model to identify independent risk factors for cardiac rupture.

Discrimination was assessed by the area under the ROC curve (AUC), sensitivity, and specificity. Calibration was evaluated using the Hosmer-Lemeshow test ($P > 0.05$ indicates good calibration) and calibration plots. Clinical utility was assessed by decision curve analysis (DCA). A two tailed $P < 0.05$ was considered statistically significant.

Results

Comparison of Key Indicators Between the Two Groups

Baseline clinical characteristics, laboratory parameters, and medication use were compared between the two groups (Table 1).Baseline clinical characteristics. Compared with the control group, patients in the CR group presented with higher admission heart rate, a higher proportion of Killip class ≥III, and lower systolic and diastolic blood pressure (all $P < 0.05$). Left ventricular ejection fraction (LVEF) was significantly lower in the CR group (44 (38–50) vs. 49 (41–58),

Table 1 Comparison of Various Indicators Between Groups with and without Cardiac Rupture

Indicators	Cardiac Rupture Group (n=53)	Non-Cardiac Rupture Group (n=159)	X^2/t/z	P
Male	33(62.26)	111(69.81)	1.039	0.308
Age (years)	73.91±6.09	74.67±7.06	0.708	0.480
First myocardial infarction (%)	51(96.23)	152(95.00)	0.000	>0.999
Smoking history (%)	23(43.40)	54(33.96)	1.503	0.216
Stroke History (%)	11(20.75)	17(10.69)	3.511	0.061
Dyslipidemia (%)	6(11.32)	14(8.81)	0.294	0.587
Coronary heart disease history (%)	7(13.21)	9(5.66)	2.253	0.133
PCI history (%)	1(1.89)	1(0.63)	-	0.438
Diabetes mellitus history (%)	17(32.08)	32(20.13)	3.194	0.074
Hypertension history (%)	17(32.08)	32(20.13)	3.194	0.074
Admission heart rate (beats/min)	95.83±23.42	77.88±16.55	−6.099	<0.001
Systolic blood pressure (mmHg)	119.25±24.41	128.42±2.57	2.509	0.013
Diastolic blood pressure (mmHg)	76.60±20.18	82.03±17.41	1.885	0.061
Killip Classification (%)			28.428	<0.001
Class 1	11(20.75)	96(60.38)		
Class 2	8(15.09)	18(11.32)		
Class 3	15(28.30)	26(16.35)		
Class 4	19(35.85)	19(11.95)		
Red blood cell count ($\times 10^9$/L)	4.40±0.66	4.41±0.62	0.094	0.925
Hemoglobin (g/L)	141(128,157)	140.5(128,148)	−0.978	0.328
PHR	220.29(144.25,251.14)	171.05(130.26,240.74)	−1.519	0.129
White blood cell count ($\times 10^9$/L)	11.27(9.5,14.54)	9.5(7.29,11.83)	−3.359	0.001
Neutrophil percentage (%)	82(78.2,87.8)	77.9(73.2,82.6)	−3.235	0.001
NLR (Neutrophil-to-Lymphocyte Ratio)	9.01(5.22,12.85)	5.42(3.8,7.4)	−4.521	<0.001

(*Continued*)

Table 1 (Continued).

Indicators	Cardiac Rupture Group (n=53)	Non-Cardiac Rupture Group (n=159)	X^2/t/z	P
Lymphocyte percentage (%)	15.8(11.3,20.6)	16.5(13.1,20.7)	−1.071	0.284
SIRI	5.67(3.82,8.53)	2.93(1.84,4.41)	−5.811	<0.001
Monocyte percentage (%)	5.5(3.9,7)	5.9(5.3,7.1)	−1.485	0.138
Serum potassium (mmol/L)	4.04(3.79,4.41)	4.19(3.76,4.32)	−0.141	0.888
Serum sodium (mmol/L)	138(134.9,140)	139(136,140.5)	−1.041	0.298
Serum calcium (mmol/L)	2.18(2.1,2.3)	2.18(2.11,2.25)	−0.602	0.547
CK-MB (ng/mL)	43.87(8.9,138.8)	28.86(4.4,100)	−2.038	0.042
Myoglobin (Myo,ng/mL)	400(113.96,848)	149(53.46,504)	−3.172	0.002
Creatine kinase (CK, U/L)	798(225,1902)	1151.2(304,2265)	−1.074	0.283
Lactate dehydrogenase (LDH, U/L)	780(432,1470)	526(342.5,872)	−2.754	0.006
Cardiac troponin I (cTnI, ng/mL)	3.45(0.28,14.34)	1.3(0.1,6.7)	−1.674	0.094
Prothrombin time (PT, s)	13.3(12.6,14.6)	12.8(12.2,13.7)	−2.533	0.011
Activated partial thromboplastin time (APTT, s)	31.2(27.3,33.9)	30.3(27.5,34.4)	−0.515	0.607
D-dimer (mg/L FEU)	1.02(0.67,3.11)	0.72(0.58,1.3)	−2.22	0.026
Fibrinogen (FIB, g/L)	3.98(2.65,5.13)	3.1(2.4,4.16)	−2.255	0.024
Thrombin time (TT, s)	18.1(16.4,19.4)	18.8(16.5,19.7)	−1.08	0.28
Blood glucose (Glu,mmol/L)	6.75(5.58,9.32)	6.14(5.3,8.17)	−1.394	0.163
Total cholesterol (TC,mmol/L)	3.38(2.63,4.33)	3.74(3.33,4.49)	−2.485	0.013
Triglycerides (TG,mmol/L)	1.13(0.88,1.52)	1.34(0.99,1.9)	−2.224	0.026
Low-density lipoprotein cholesterol (LDL-C, mmol/L)	2.09(1.42,2.61)	2.33(1.85,2.84)	−1.983	0.047
Alanine aminotransferase (ALT, U/L)	47.9(29.4,68.3)	40.3(25.5,61.6)	−0.936	0.349
Aspartate aminotransferase (AST, U/L)	121.9(41.9,246.5)	153.6(70.7,300.5)	−1.102	0.271
Albumin (Alb,g/L)	38.4(35.3,41.2)	39.5(37.1,42.4)	−2.047	0.041
Total protein (TP,g/L)	59.7(56.3,65.9)	62.9(60.2,66.5)	−2.987	0.003
Total bilirubin (TBil,μmol/L)	14(10.23,25.1)	15(9.8,18.1)	−0.78	0.436
Direct Bilirubin (DBil,μmol/L)	5.7(4.3,8.4)	5.3(3.8,7.3)	−1.289	0.197
Serum Creatinine (Scr,μmol/L)	74(64,95)	72(63,83)	−1.257	0.209
First Medical Contact Time (FMC, min)	960(360,2160)	172(103,418)	−7.023	<0.001
N-terminal Pro-B-type Natriuretic Peptide (NT-proBNP, pg/mL)	3349(1000,7100)	786(181,2490)	−4.658	<0.001
Left Ventricular Ejection Fraction (EF, %)	44(38,50)	49(41,57)	−2.647	0.008
Thrombolytic Therapy (%)	2(3.77)	17(10.69)	1.561	0.212
Oral Dual Antiplatelet Therapy (%)	52(98.11)	159(100.00)	-	0.250
ACEI/ARB (%)	6(11.32)	70(44.03)	18.487	<0.001
β-Blocker (%)	15(28.30)	75(47.17)	5.792	0.016
AMI location, n (%)				
Anterior wall	28(52.83)	71(44.65)	1.068	0.301
Anteroseptal wall	1(1.89)	3(1.89)	0.000	>0.999
Extensive anterior wall	17(32.08)	63(39.62)	0.964	0.326
Inferior wall	21(39.62)	61(38.36)	0.027	0.871
High lateral wall	3(5.66)	6(3.77)	0.039	0.844
Lateral wall	3(5.66)	8(5.03)	0.000	>0.999
Involving ≥2 walls	16(30.19)	43(27.04)	0.196	0.658
Left anterior descending artery (LAD), n (%)	41(77.36)	113(71.07)	0.791	0.374
Left circumflex artery (LCx), n (%)	17(32.79)	41(25.79)	0.791	0.374
Right coronary artery (RCA), n (%)	27(50.94)	87(54.72)	0.228	0.633
Left main coronary artery (LMCA), n (%)	6(11.32)	16(10.01)	0.068	0.795
Number of diseased vessels, n (%)			0.539	0.463
Single-vessel	18(33.96)	63(39.62)		
Multi-vessel	35(66.04)	96(60.38)		

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P = 0.008), and the rate of primary percutaneous coronary intervention (PCI) was lower (P < 0.05). These findings suggest that patients who developed CR had more severe hemodynamic compromise and less frequent timely revascularization. No significant differences were observed between the two groups in terms of culprit vessel or number of diseased vessels (all P > 0.05).(see Table 1).

Laboratory parameters. Regarding cardiac biomarkers, the CR group showed significantly elevated levels of Myo, CK-MB, and NT-proBNP (all P < 0.05). Coagulation parameters including PT, D-dimer, and fibrinogen were also significantly higher in the CR group (all P < 0.05), indicating a more pronounced hypercoagulable state. In contrast, metabolic parameters such as total cholesterol, triglycerides, albumin, total protein, and LDL-C were significantly lower in the CR group (all P < 0.05). (see Table 1).

Inflammatory markers and blood cell counts. Patients in the CR group had higher white blood cell count, neutrophil percentage, neutrophil-to-lymphocyte ratio (NLR), hemoglobin, and SIRI compared with the control group (all P < 0.05). Among these,SIRI demonstrated the most pronounced elevation (5.67 [IQR:3.82–8.53] vs. 2.93 [IQR:1.84–4.41],*P* < 0.001), suggesting systemic inflammation may play a prominent role in cardiac rupture. (see Table 1).

Medication use. The proportion of patients receiving ACEI/ARB or β-blockers within 24 hours of admission was significantly lower in the CR group than in the control group (both P < 0.05), highlighting a potential protective role of early neurohormonal modulation against cardiac rupture. (see Table 1).

Multivariate Logistic Regression Analysis of AMI Complicated with CR

Lasso regression and multivariate logistic regression. To eliminate multicollinearity and screen key predictive variables, Lasso regression was performed on all potential factors using 10-fold cross-validation with the lambda.1se criterion (λ = 0.073). Six variables with non-zero coefficients were identified: admission heart rate, Killip classification, SIRI, total protein, primary PCI, and ACEI/ARB use within 24 hours (Figures 1 and 2).These six variables were then entered into a multivariate logistic regression model. The results showed that admission heart rate, Killip classification, SIRI, primary PCI, and ACEI/ARB use were independent factors associated with cardiac rupture (all P < 0.05), whereas total protein was not statistically significant (P > 0.05) and was not retained in the final model (see Table 2).Among these, admission

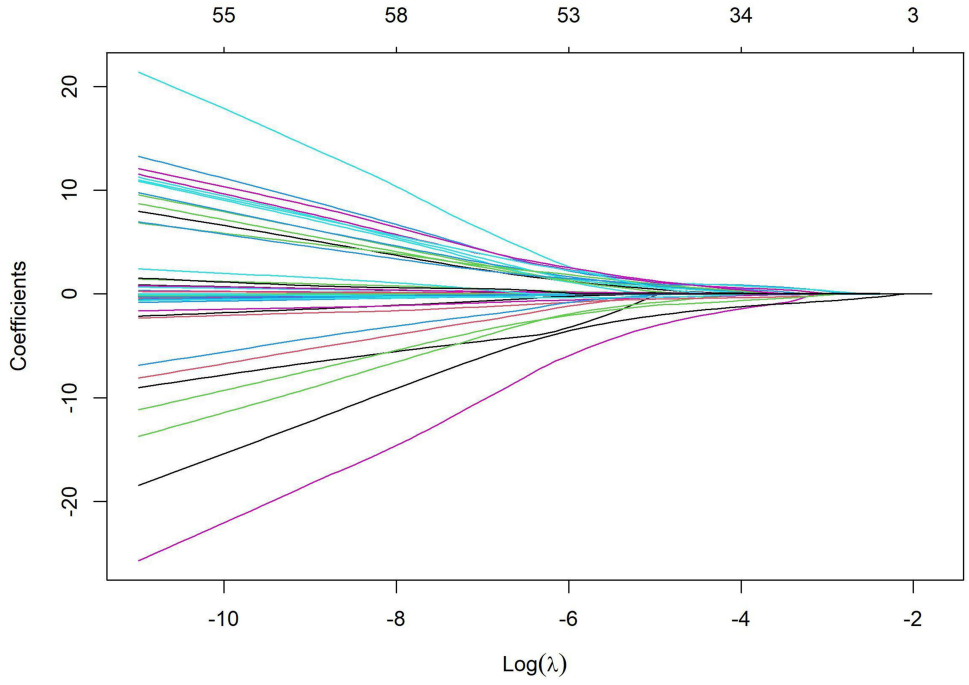

Figure 1 Lasso regression coefficient paths for variable selection. Each colored line represents the coefficient trajectory of a candidate predictor as a function of the logarithm of the regularization parameter (Log(λ)). As λ increases (moving rightward), coefficients are shrunk toward zero,facilitating variable selection. The top axis displays the number of non-zero coefficients retained at each λ value.

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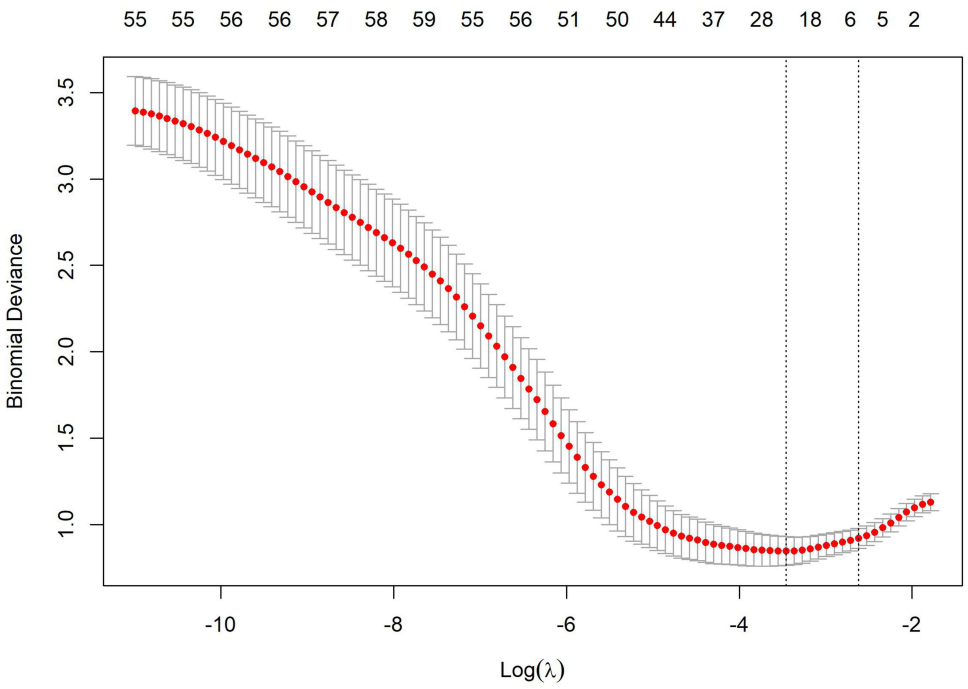

Figure 2 Lasso regression cross validation curves. The red dots represent the mean binomial deviance across cross-validation folds at each value of Log(λ). The vertical gray error bars indicate the standard error of the mean deviance. The vertical dashed lines mark the optimal Log(λ) values (minimum deviance and 1-standard-error rule) selected for final variable selection. The top axis shows the number of non-zero coefficients at each λ.

heart rate (OR = 1.050, 95% CI: 1.024–1.075, P < 0.001), Killip classification (OR = 2.092, 95% CI: 1.460–2.997, P < 0.001), and SIRI (OR = 1.105, 95% CI: 1.022–1.196, P = 0.012) were identified as independent risk factors for cardiac rupture, while emergency PCI (OR = 0.239, 95% CI: 0.097–0.589, P = 0.002) and ACEI/ARB use within 24 hours (OR = 0.173, 95% CI: 0.060–0.500, P = 0.001) were independently associated with a lower risk of cardiac rupture.

Construction and Efficacy Evaluation of the Prediction Model

Based on the five independent variables identified by multivariate logistic regression (admission heart rate, Killip classification, SIRI, primary PCI, and ACEI/ARB use), a nomogram prediction model for AMI complicated with CR was constructed (Figure 3). ROC curve analysis demonstrated good discriminative ability, with an AUC of 0.885 (95% CI: 0.835–0.936) (Figure 4). At the optimal cutoff value determined by the Youden index, the model achieved a sensitivity of 84.9% and a specificity of 76.7%. Regarding calibration, the Hosmer Lemeshow test showed no significant lack of fit (χ^2 = 12.694, P = 0.123), and the calibration plot (Figure 5) demonstrated acceptable agreement between observed outcomes and predicted probabilities. Decision curve analysis (Figure 6) demonstrated that the nomogram provided a higher net benefit than the "treat

Table 2 Multivariate Logistic Regression Analysis

Variables	*B*	*S.E.*	*Wald*	*P*	*OR*	*95% CI*	
						Lower Bound	Upper Bound
Admission Heart Rate	0.048	0.012	15.165	<0.001	1.050	1.024	1.075
Killip Classification	0.738	0.183	16.206	<0.001	2.092	1.460	2.997
SIRI	0.100	0.040	6.247	0.012	1.105	1.022	1.196
Total Protein	−0.041	0.031	1.766	0.184	0.960	0.904	1.019
ACEI/ARB	−1.756	0.542	10.494	0.001	0.173	0.060	0.500
Primary PCI	−1.432	0.461	9.657	0.002	0.239	0.097	0.589

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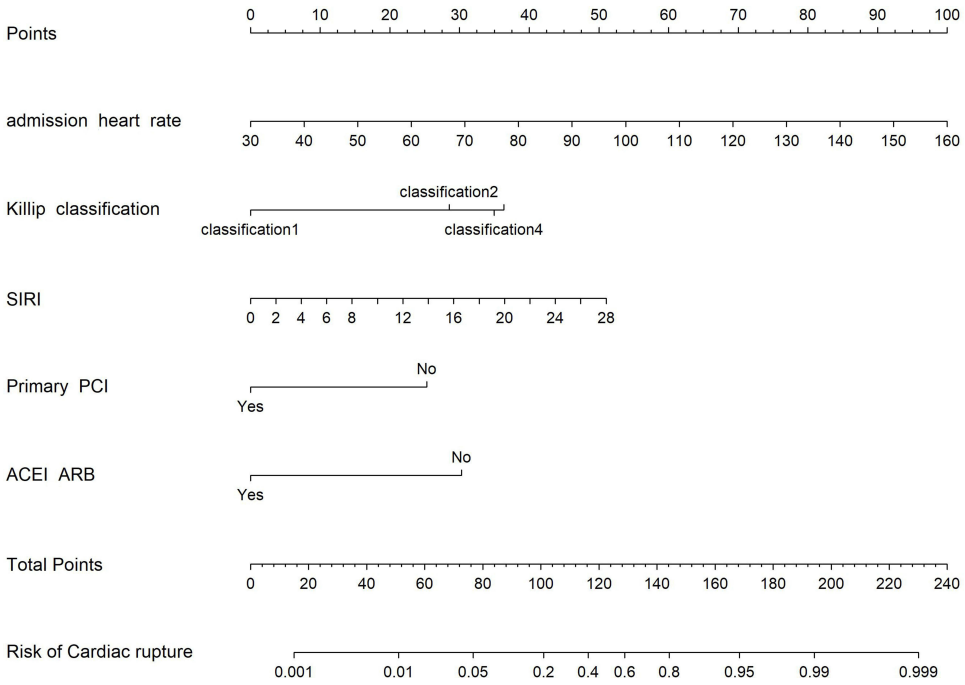

Figure 3 Nomogram for predicting cardiac rupture after acute myocardial infarction. The nomogram integrates clinical predictors to estimate the individual risk of cardiac rupture. For each variable,the corresponding point value is determined by drawing a vertical line to the top "Points" axis. The sum of all points is located on the "Total Points" axis, and a final vertical line to the "Risk of Cardiac rupture" axis yields the predicted probability.

all" and "treat none" strategies across threshold probabilities ranging from 0.02 to 1.0, suggesting favorable clinical utility in identifying patients at high risk for cardiac rupture following acute myocardial infarction.

Discussion

AMI complicated by CR remains a clinically challenging and frequently fatal complication.[22–24] Although the widespread adoption of reperfusion therapies such as PCI has reduced its incidence, mortality remains high.[25] Structural rupture within the cardiovascular system, whether involving the myocardium or major vessels, is frequently fatal and often progresses too rapidly for successful intervention.[26].

Among the 53 patients with cardiac rupture included in this study, 48 cases were diagnosed as free wall rupture and 5 cases as ventricular septal perforation, while no case of papillary muscle rupture was detected.The absence of papillary muscle rupture in the present study may be attributed to the inherent low incidence of this lesion, limitations of clinical diagnosis, and the relatively small sample size. Further validation is warranted in future large-scale multicenter studies.

The pathogenesis of CR involves myocardial necrosis, inflammatory imbalance, extracellular matrix degradation, and ventricular remodeling.[27–29] However, the lack of practical early warning tools often leads to missed intervention opportunities. Thus, identifying key risk factors and developing an accurate predictive model are of great clinical value for risk stratification and improving outcomes.[30] In this study, we used Lasso regression to select core variables and developed a nomogram for CR based on admission heart rate, Killip classification, SIRI, primary PCI, and ACEI/ARB use. The model showed good discrimination (AUC = 0.885), providing a clinically useful tool, though prospective multicenter validation is needed.

The role of inflammation in CR is well established, but previous studies have largely relied on single indicators such as neutrophil-to-lymphocyte ratio (NLR) or C-reactive protein (CRP), which incompletely capture the complex interplay between systemic inflammation and immune function.[31,32] SIRI, which integrates neutrophil, lymphocyte, and monocyte counts, more comprehensively reflects inflammatory status and has been linked to major adverse cardiovascular events after AMI.[33,34] Our study identified SIRI as an independent risk factor for CR, consistent with mechanistic insights: neutrophil-derived proteases degrade myocardial collagen, monocyte-mediated chronic inflammation promotes

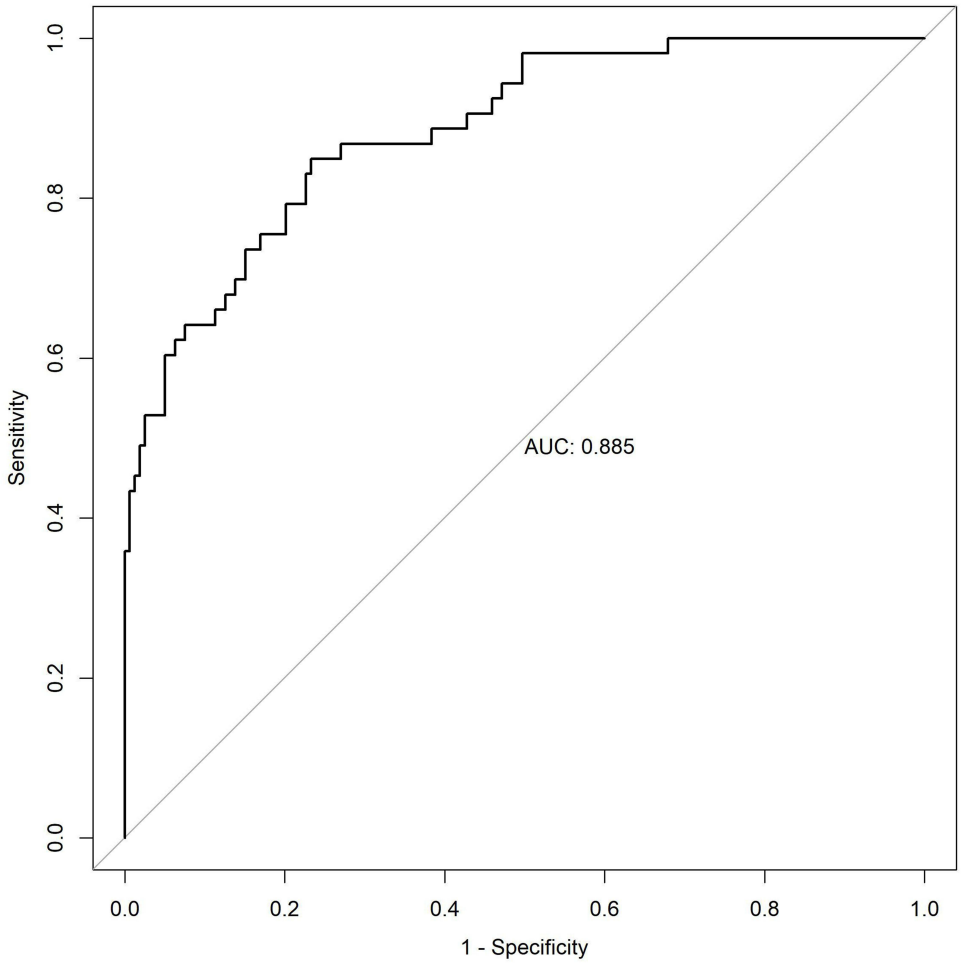

Figure 4 Receiver operating characteristic (ROC) curve of the nomogram. The ROC curve illustrates the diagnostic performance of the nomogram for predicting cardiac rupture. The area under the curve (AUC) is 0.885,indicating good discriminatory ability of the model. The diagonal line represents the null hypothesis of no predictive value (AUC = 0.5).

ventricular remodeling, and lymphocyte reduction impairs tissue repair.[35] Together, these processes compromise the mechanical stability of the infarcted ventricular wall. Importantly, SIRI can be calculated from routine blood tests without additional cost, making it suitable for rapid use in emergency and intensive care settings.

Elevated admission heart rate and Killip class are established risk factors for CR.[36–38]Tachycardia increases myocardial oxygen consumption and wall stress, exacerbating mechanical load on the infarcted myocardium, while higher Killip class reflects larger infarct size and more severe ventricular dysfunction—both contributing to mechanical instability and increased rupture risk. Our findings are consistent with these previous observations.

Consistent with prior studies,[39] primary PCI was independently associated with a lower risk of CR in our cohort. In the era of primary PCI, the incidence of CR has declined substantially.[40,41] Mechanistically, timely reperfusion limits infarct size and prevents transmural necrosis, reducing ventricular wall thinning and rupture risk.[42] Successful revascularization also mitigates adverse remodeling, reduces border zone stress, and creates a favorable microenvironment for wound healing. However, PCI may occasionally induce intramyocardial hemorrhage,[43] nevertheless, the net clinical benefit in reducing CR risk is well established. These findings underscore that timely revascularization remains a cornerstone of CR prevention.

Our study observed that ACEI/ARB use was independently associated with a lower risk of CR, supporting the potential role of renin angiotensin aldosterone system (RAAS) inhibition in CR prevention.[44] Mechanistically, ACEI/ARB may reduce myocardial interstitial collagen degradation, attenuate ventricular remodeling, and lower ventricular

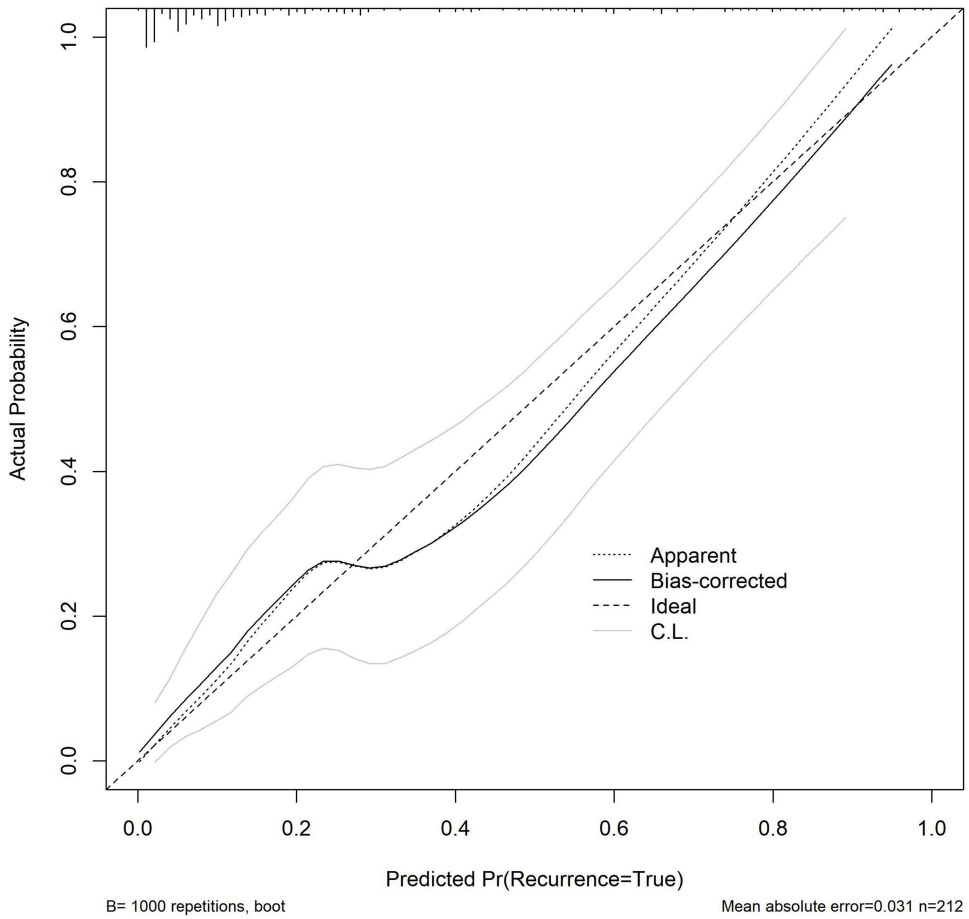

Figure 5 Calibration curve of the nomogram. The dashed line (Ideal)represents perfect calibration (predicted probability= actual probability). The solid line (Bias-corrected) shows the nomogram's calibration after bootstrapping bias correction (B= 1000repetitions). The dotted line (Apparent) shows calibration in the original training dataset. The gray shaded area (C.L.) denotes the 95% confidence interval of the bias-corrected curve. The mean absolute error(MAE) between predicted and actual probabilities is 0.031 (n = 212).

wall stress—key pathways in CR pathogenesis.[45] However, several limitations should be considered. As with any observational study, treatment indication bias cannot be fully excluded. Moreover, detailed data on ACEI/ARB dosage and timing of initiation were not consistently available, precluding a more nuanced analysis. Thus, the observed association should be interpreted as hypothesis generating rather than causal. Prospective studies with standardized protocols are needed to confirm these findings.

Research on CR prediction models remains at an exploratory stage. Some studies have employed machine learning with high predictive performance but rely on complex analyses that are difficult to implement in routine practice. Others have developed simpler tools, but limitations in indicator availability or discriminative power restrict their clinical utility.[46] Our nomogram incorporates five easily obtainable routine parameters, enabling rapid risk assessment in emergency or intensive care settings. The nomogram model,which exhibited an AUC of 0.885, may offer useful clinical insights for identifying patients at risk of cardiac rupture, although these preliminary findings require validation in prospective multicenter studies.

Several limitations should be acknowledged. First, this was a single center retrospective study, which limits causal inference and may introduce selection bias. Second, the 10 year study period may introduce temporal bias due to evolving clinical practices, although matching by admission period partially mitigated this. Third, data on echocardiographic details, door-to-needle/balloon times, and certain clinical presentations were incomplete, precluding a more comprehensive analysis.Fourth, the study was restricted to patients aged ≥60 years, limiting generalizability to younger

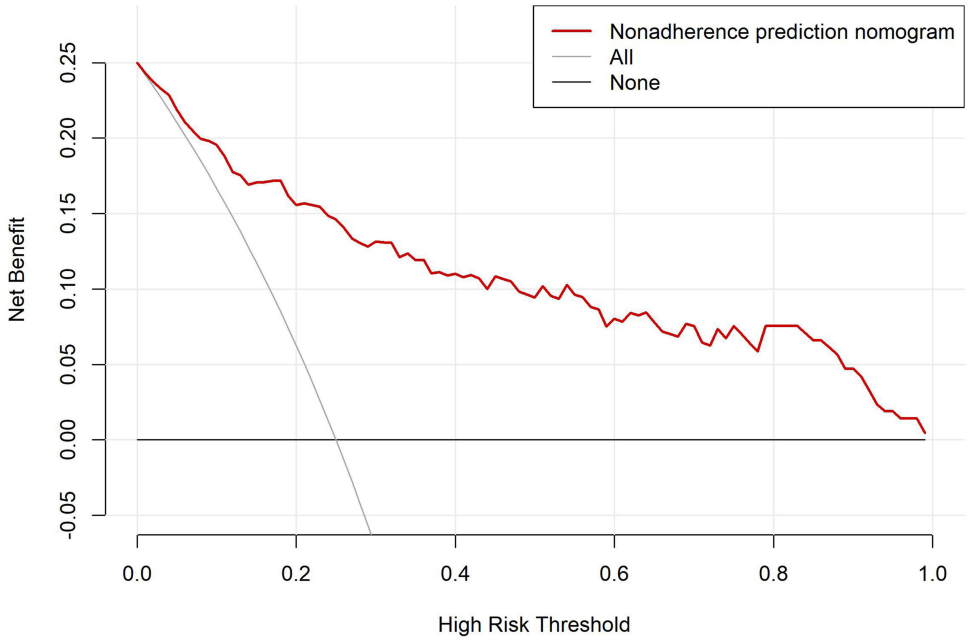

Figure 6 Decision curve analysis (DCA) of the nomogram. The red line represents the net benefit of the nomogram for predicting nonadherence across different high-risk thresholds. The gray line ("All") assumes all patients are classified as high-risk, and the black line ("None") assumes no patients are high-risk. The nomogram yields a higher net benefit than the "All" and "None"strategies across a wide range of threshold probabilities, demonstrating its clinical utility.

populations. Fifth, the small number of CR events raises potential overfitting risk despite Lasso regression. Finally, external validation in prospective multicenter cohorts is needed to confirm the robustness of the nomogram.

Conclusion

In this study, increased admission heart rate, elevated Killip classification, and higher SIRI were identified as independent risk factors for CR after AMI, whereas primary PCI was an independent protective factor and ACEI/ARB use was independently associated with a lower risk of CR. A nomogram incorporating these five variables demonstrated good discriminative ability and may serve as a practical tool for early risk stratification in clinical practice. Given the retrospective, single-center design, external validation in prospective multicenter cohorts is warranted to confirm the generalizability of these findings.

Data Sharing Statement

All data generated during this study have been analyzed, and the results are included in this manuscript. The data supporting the findings of this study are available upon reasonable request from the corresponding author.

Ethics Approval and Informed Consent

This study was approved by the Ethics Committee of Tianshui First People's Hospital (Approval ID: 2025-055; Amendment approved on October 28, 2025) for research involving human participants. Informed consent was waived by the Ethics Committee due to the retrospective nature of the study, and all patient data were anonymized to protect privacy, in compliance with the Declaration of Helsinki.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

All authors declare no conflicts of interest in this work.

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