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ORIGINAL RESEARCH

Construction of a Column Chart Prediction Model for the Risk of Left Ventricular Thrombosis After PCI in Patients with Acute ST Segment Elevation Myocardial Infarction

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Objective: To explore the influencing factors of left ventricular thrombosis after percutaneous coronary intervention (PCI) in patients with acute ST segment elevation myocardial infarction (STEMI) and construct a column chart model.

Methods: A retrospective study was conducted on 331 STEMI patients who underwent PCI between July 2020 and January 2024. According to the principle of approximately 3:1, patients were randomly separated into 245 in the modeling group and 86 in the validation group, and clinical data of patients were collected. Multivariate logistic regression was applied to screen for risk factors. R software was applied to draw column charts. Bootstrap method was applied for internal validation. Hosmer-Lemeshow (H-L) was applied to test the fitting degree of the column chart model. Calibration curve and ROC curve were applied to verify calibration and discrimination, respectively. DCA curve was applied to analyze the clinical practicality of the column chart model.

Results: A history of angina pectoris, ventricular aneurysm, alcohol abuse, postoperative TIMI grade ≤ 2, LVEF, and total ischemic time were influencing factors for left ventricular thrombosis in STEMI patients after PCI (P<0.05). The predicted probabilities of the internal and external validation calibration curves were highly consistent with the actual probabilities, the concordance index of the ROC curve was 0.962 (95% CI: 0.931–0.994) and 0.958 (95% CI: 0.926–0.990), respectively, indicating high model calibration and discrimination; H-L inspection showed χ^2=11.977, 9.757 (P=0.152, 0.282). DCA curve showed that when the probability range of the high-risk threshold was 0.02~0.99, the column chart model performed better and had a higher net return.

Conclusion: The column chart model constructed by risk factors such as history of angina, ventricular aneurysm, history of alcohol abuse, postoperative TIMI ≤ grade 2, LVEF, and total ischemic time has high predictive value and can effectively predict left ventricular thrombosis in STEMI patients after PCI.

Keywords: acute ST-segment elevation myocardial infarction, percutaneous coronary intervention, left ventricular thrombosis, influencing factors, column chart

Introduction

Acute ST-segment elevation myocardial infarction (STEMI) is a disease with high incidence and mortality rates, and its incidence has been continuously rising in recent years, posing a serious threat to human life and health.[1] Despite improvements in the survival rate of STEMI patients due to percutaneous coronary intervention (PCI) and antiplatelet therapy over the past 20 years, post-infarction complications remain a significant cause of high mortality and morbidity.[2,3] The formation of left ventricular thrombus is one of the common complications of STEMI, with an incidence as high as 46%. Although surgical treatments have improved the situation, the severity remains concerning. If not treated promptly, it can lead to embolism and sudden death, increasing the total hospital stay and healthcare

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resource usage, thereby adding to the social, economic, and medical burden.[4,5] Although there are many studies on the risk factors of left ventricular thrombus in STEMI patients, the results vary and need further evaluation.[6,7] Currently, there is insufficient clinical evidence for the prevention of left ventricular thrombosis after PCI in STEMI patients, and it is imperative to develop effective strategies for its prevention. Nomograms are visual risk prediction tools constructed based on influencing factors. They integrate high-risk factors into the model and effectively predict the risk of disease occurrence for patients according to the weight of different factors. They have been widely used in clinical practice. For example, Li et al[8] developed and validated a nomogram for predicting the risk of left atrial thrombus in patients with non-valvular atrial fibrillation. However, there is still a lack of risk prediction models specifically for left ventricular thrombus in STEMI patients. Therefore, this study conducts a logistic regression analysis on the occurrence and risk factors of left ventricular thrombosis after PCI in STEMI patients, constructs a nomogram model, aiming to provide a reference for high-quality research on the prevention and treatment of left ventricular thrombosis.

Subjects and Methods

Subjects

Compared to the incidence of left ventricular thrombus in STEMI patients reported in previous literature, with 1-α set at 0.95 and β at 0.1, sample size calculation was performed using PASS 15.0 software. The results indicate that at least 264 samples are required for this study. Accounting for a follow-up loss rate of 15%, the final sample size needed is 304 cases.A retrospective selection of 331 STEMI(The infarct locations are all in the left ventricular lateral wall, corresponding to the area supplied by the left circumflex artery) patients who underwent PCI from July 2020 to January 2024 was made as the study subjects, randomly divided into a modeling group of 245 patients and a validation group of 86 patients, following an approximate 3:1 ratio. Simultaneously, the modeling group was divided into two subgroups based on the presence of left ventricular thrombus: 182 cases in the non-left ventricular thrombus group and 63 cases in the left ventricular thrombus group.

Inclusion criteria: ① Patients conforming to the diagnostic and treatment standards,[9] confirmed by clinical, imaging, and laboratory examinations, and all patients with left ventricular thrombus were found to have thrombus through transthoracic echocardiography.; ② Age ≥18 years, with detailed and complete data without any loss; ③ Patients admitted within 12 hours of onset for PCI treatment; ④ The study has been approved by the hospital's ethics committee. Exclusion criteria: ① Patients with cardiovascular diseases such as dilated cardiomyopathy, hypertrophic cardiomyopathy, rheumatic heart disease, myocarditis, or pulmonary heart disease; ② Patients with contraindications for antiplatelet or anticoagulant therapy; ③ Patients with autoimmune diseases, blood disorders, or liver/kidney dysfunction; ④ Patients with a history of venous thrombosis; ⑤ Pregnant or breastfeeding women; ⑥ Patients who had major trauma or surgery within the last 3 months. See Figure 1 for the case collection flow chart.

Collection of Clinical Data

Review the patients' outpatient and inpatient records to collect their clinical data.The clinical data of the patients included gender, age, body mass index (BMI), systolic blood pressure, diastolic blood pressure, history of angina, old MI, ventricular aneurysm, smoking history, alcohol abuse history, chronic underlying diseases (hypertension, diabetes, hyperlipidemia), preoperative TIMI grade 0, postoperative TIMI grade ≤2, single-vessel disease, no collateral circulation, white blood cell count, creatinine, low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), total cholesterol (TC), peak cardiac troponin I (cTnI), peak creatine kinase-MB (CK-MB), blood potassium, left ventricular ejection fraction (LVEF), adjunct medications [statins, β-blockers, low molecular weight heparins, angiotensin-converting enzyme inhibitors (ACEI)], ≥4 leads ST-segment elevation, total ischemia time, mitral regurgitation, and pericardial effusion.

Statistical Analysis

Data processing was performed using IBM-SPSS 25.0 software. Continuous data (±) were analyzed using the independent sample *t*-test; categorical data [n (%)] were analyzed using the $\chi 2$ test. Multifactorial logistic regression analysis was

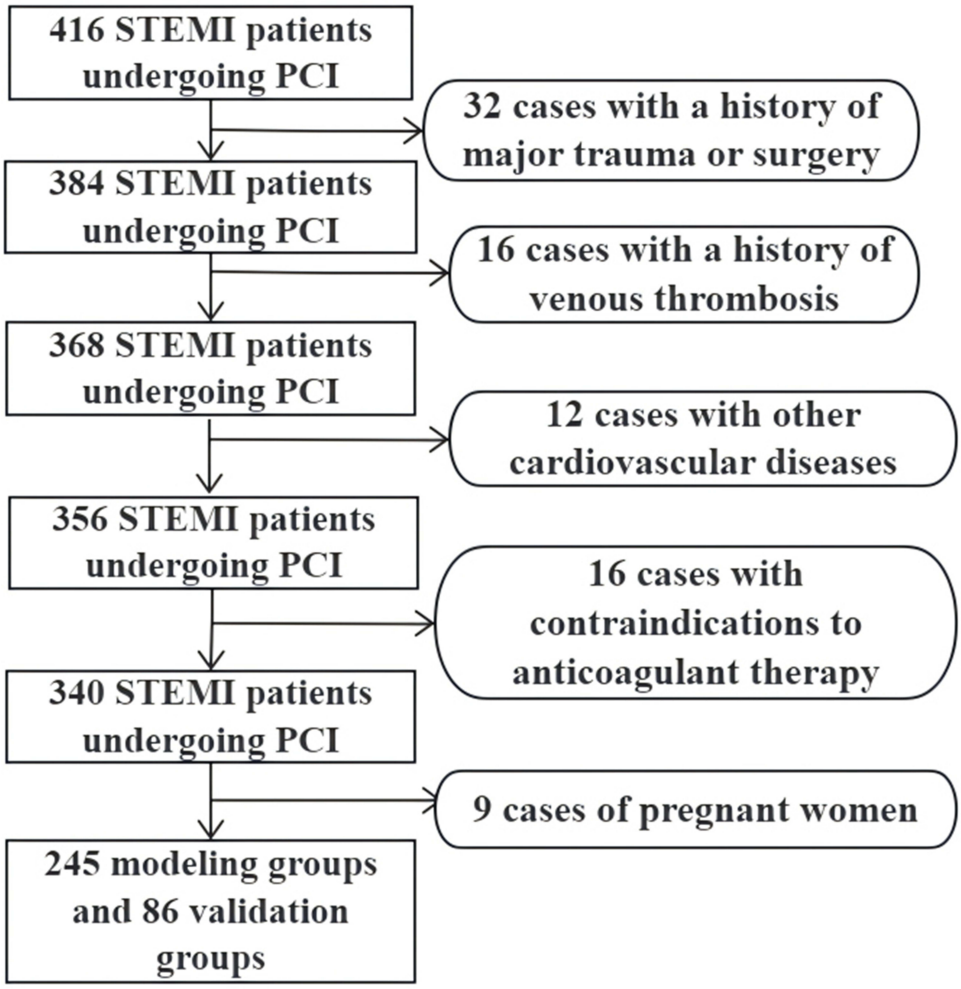

Figure 1 Case Collection Process Diagram.

used to screen for risk factors of left ventricular thrombus formation after PCI in STEMI patients. R 4.3.3 software was used to create nomograms for visualizing the results of the logistic regression analysis. Internal validation was conducted using the Bootstrap method (with 100 resamples), and the Hosmer-Lemeshow (H-L) test assessed the fit of the nomogram model. Calibration curves and ROC curves were used to verify the calibration and discriminative ability, respectively. Decision Curve Analysis (DCA) was employed to assess the clinical utility of the nomogram model. A P-value of <0.05 was considered statistically significant.

Results

Comparison of Demographic and Disease-Related Data Between the Modeling and Validation Groups

No significant differences were found between the modeling and validation groups in terms of gender, age, BMI, systolic blood pressure, diastolic blood pressure, history of angina, old MI, ventricular aneurysm, smoking history, alcohol abuse history, chronic underlying diseases (hypertension, diabetes, hyperlipidemia), preoperative TIMI grade 0, postoperative TIMI grade ≤2, single-vessel disease, no collateral circulation, white blood cell count, creatinine, LDL-C, TG, HDL-C, TC, peak cTnI, peak CK-MB, blood potassium, LVEF, adjunct medications (statins, β-blockers, low molecular weight heparins, ACEI), ≥4 leads ST-segment elevation, total ischemia time, mitral regurgitation, and pericardial effusion (P>0.05). See Table 1.

Table 1 Comparison of Demographic and Disease Related Data Between the Modeling and Validation Groups [($\bar{x} \pm S$) /n (%)]

Index	*n*	Modeling Group (*n*=245)	Validation group (*n*=86)	χ^2/t	*P*
Male[*n* (%)]	268	197 (80.41)	71 (82.56)	0.191	0.662
Age (years)		60.73±10.11	60.75±10.24	0.016	0.987
BMI (kg/m^2)		22.98±3.01	22.94±2.84	0.108	0.914
Systolic pressure (mmHg)		127.24±12.87	128.01±12.55	0.480	0.631
Diastolic pressure (mmHg)		80.87±8.20	80.42±8.16	0.438	0.661
History of angina pectoris[*n* (%)]	85	66 (26.94)	19 (22.09)	0.783	0.376
Old MI[*n* (%)]	50	39 (15.92)	11 (12.79)	0.486	0.486
Ventricular aneurysm[*n* (%)]	77	55 (22.45)	22 (25.58)	0.350	0.554
Smoking history[*n* (%)]	245	179 (73.06)	66 (76.74)	0.449	0.503
History of alcoholism[*n* (%)]	47	35 (14.29)	12 (13.95)	0.006	0.939
Chronic underlying diseases[*n* (%)]					
Hypertension	229	167 (68.16)	62 (72.09)	0.461	0.497
Diabetes	46	31 (12.65)	15 (17.44)	1.220	0.269
Hyperlipidemia	42	31 (12.65)	11 (12.79)	0.001	0.974
Preoperative TIMI grading 0[*n* (%)]	106	73 (29.80)	33 (38.37)	2.151	0.142
Postoperative TIMI grading ≤ Level 2[*n* (%)]	40	31 (12.65)	9 (10.47)	0.287	0.592
Single vessel disease[*n* (%)]	100	70 (28.57)	30 (34.88)	1.203	0.273
No collateral circulation[*n* (%)]	320	237 (96.73)	83 (96.51)	0.010	0.921
White blood cell count (×10^9/L)		10.09±2.54	9.94±2.31	0.482	0.630
Creatinine (μmol/L)		67.95±14.05	67.74±13.94	0.119	0.905
LDL-C (mmol/L)		2.23±0.52	2.22±0.53	0.153	0.879
TG (mmol/L)		1.39±0.24	1.41±0.22	0.679	0.498
HDL-C (mmol/L)		1.02±0.18	1.01±0.20	0.430	0.667
TC (mmol/L)		4.80±0.59	4.85±0.60	0.673	0.501
cTnI peak value (μg/L)		13.88±2.45	13.79±2.62	0.288	0.774
CK-MB peak value (U/L)		129.61±24.84	125.94±26.71	1.156	0.249
Blood potassium (mmol/L)		4.20±0.37	4.25±0.42	1.040	0.299
LVEF (%)		46.57±7.30	45.43±7.80	1.224	0.222
Adjuvant medication[*n* (%)]					
Statins	325	241 (98.37)	84 (97.67)	0.172	0.679
Beta-blocker	245	176 (71.84)	69 (80.23)	2.333	0.127
Low molecular weight heparin	288	213 (86.94)	75 (87.21)	0.004	0.949
ACEI	317	233 (95.10)	84 (97.67)	1.040	0.308
≥ 4 leads with ST segment elevation[*n* (%)]	142	103 (42.04)	39 (45.35)	0.284	0.594
Total ischemic time (h)		7.57±2.08	7.82±2.10	0.957	0.339
Mitral regurgitation[*n* (%)]	158	118 (48.16)	40 (46.51)	0.070	0.792
Pericardial effusion[*n* (%)]	80	62 (25.31)	18 (20.93)	0.665	0.415

Screening for Risk Factors of Left Ventricular Thrombus Formation After PCI in STEMI Patients in the Modeling Group

No significant differences were found between the non-left ventricular thrombus group and the left ventricular thrombus group in gender, age, BMI, systolic blood pressure, diastolic blood pressure, old MI, smoking history, chronic underlying diseases (hypertension, diabetes, hyperlipidemia), preoperative TIMI grade 0, single-vessel disease, no collateral circulation, white blood cell count, creatinine, LDL-C, TG, HDL-C, TC, peak cTnI, peak CK-MB, blood potassium, adjunct medications (statins, β-blockers, low molecular weight heparins, ACEI), ≥4 leads ST-segment elevation, mitral regurgitation, and pericardial effusion (P>0.05). The left ventricular thrombus group had a higher incidence of angina history, ventricular aneurysm, alcohol abuse history, postoperative TIMI grade ≤2, and total ischemia time, and a lower LVEF than the non-left ventricular thrombus group (P<0.05). See Table 2.

Table 2 Screening of Risk Factors for Left Ventricular Thrombosis After PCI in Modeling Group STEMI Patients[($\bar{x} \pm S$) /n (%)]

Index	*n*	Non Left Ventricular Thrombus Group (*n*=182)	Left Ventricular Thrombus Group (*n*=63)	χ^2/t	*P*
Male[*n* (%)]	197	145 (79.67)	52 (82.54)	0.245	0.621
Age (years)		60.78±10.14	60.59±10.09	0.128	0.898
BMI (kg/m^2)		22.94±3.05	23.08±2.97	0.316	0.752
Systolic pressure (mmHg)		126.75±12.84	128.66±12.91	1.016	0.311
Diastolic pressure (mmHg)		80.45±8.15	82.07±8.24	1.356	0.176
History of angina pectoris[*n* (%)]	66	31 (17.03)	35 (55.56)	35.287	0.000
Old MI[*n* (%)]	39	27 (14.84)	12 (19.05)	0.620	0.431
Ventricular aneurysm[*n* (%)]	55	20 (10.99)	35 (55.56)	53.392	0.000
Smoking history[*n* (%)]	179	134 (73.63)	45 (71.43)	0.115	0.735
History of alcoholism[*n* (%)]	35	10 (5.49)	25 (39.68)	44.672	0.000
Chronic underlying diseases[*n* (%)]					
Hypertension	167	119 (65.38)	48 (76.19)	2.518	0.113
Diabetes	31	20 (10.99)	11 (17.46)	1.773	0.183
Hyperlipidemia	31	19 (10.44)	12 (19.05)	3.138	0.077
Preoperative TIMI grading 0[*n* (%)]	73	49 (26.92)	24 (38.10)	2.793	0.095
Postoperative TIMI grading ≤ Level 2[*n* (%)]	31	10 (5.49)	21 (33.33)	32.817	0.000
Single vessel disease[*n* (%)]	70	48 (26.37)	22 (34.92)	1.675	0.196
No collateral circulation[*n* (%)]	237	178 (97.80)	59 (93.65)	2.553	0.110
White blood cell count (×10^9/L)		10.16±2.75	9.90±2.25	0.676	0.500
Creatinine (μmol/L)		67.20±13.35	70.10±15.04	1.438	0.152
LDL-C (mmol/L)		2.22±0.54	2.27±0.50	0.645	0.519
TG (mmol/L)		1.38±0.20	1.42±0.26	1.262	0.208
HDL-C (mmol/L)		1.01±0.17	1.05±0.20	1.536	0.126
TC (mmol/L)		4.76±0.56	4.90±0.61	1.671	0.096
cTnI peak value (μg/L)		13.80±2.32	14.10±2.64	0.853	0.394
CK-MB peak value (U/L)		128.76±25.41	132.08±24.15	0.905	0.366
Blood potassium (mmol/L)		4.18±0.35	4.24±0.38	1.147	0.253
LVEF (%)		48.76±8.15	40.24±6.43	7.523	0.000
Adjuvant medication[*n* (%)]					
Statins	241	179 (98.35)	62 (98.41)	0.001	0.974
Beta-blocker	176	135 (74.18)	41 (65.08)	1.914	0.167
Low molecular weight heparin	213	162 (89.01)	51 (80.95)	2.677	0.102
ACEI	233	174 (95.60)	59 (93.65)	0.383	0.536
≥ 4 leads with ST segment elevation[*n* (%)]	103	76 (41.76)	27 (42.86)	0.023	0.879
Total ischemic time (h)		6.95±2.01	9.38±2.12	8.154	0.000
Mitral regurgitation[*n* (%)]	118	84 (46.15)	34 (53.97)	1.145	0.285
Pericardial effusion[*n* (%)]	62	42 (23.08)	20 (31.75)	1.861	0.173

Multifactorial Logistic Regression Analysis of Left Ventricular Thrombus Formation After PCI in STEMI Patients in the Modeling Group

Based on the statistically significant factors identified in the results of 2.2, history of angina (yes=1, no=0), ventricular aneurysm (yes=1, no=0), alcohol abuse history (yes=1, no=0), postoperative TIMI grade ≤2 (yes=1, no=0), LVEF, and total ischemia time were included as independent variables in the multifactorial logistic analysis. The results showed that history of angina, ventricular aneurysm, alcohol abuse history, postoperative TIMI grade ≤2, LVEF, and total ischemia time were influencing factors for the formation of left ventricular thrombus after PCI in STEMI patients (P<0.05). See Table 3.

Table 3 Multivariate Logistic Regression Analysis of Left Ventricular Thrombosis After PCI in Modeling Group STEMI Patients

Index	*β*	*SE*	*Wald*χ^2	*OR*	*95% CI*	*P*
History of angina pectoris	1.350	0.556	5.889	3.859	1.297~11.485	0.015
Ventricular aneurysm	2.578	0.615	17.580	13.169	3.946~43.945	0.000
History of alcoholism	2.488	0.696	12.792	12.035	3.079~47.047	0.000
Postoperative TIMI grading ≤ Level 2	2.157	0.766	7.920	8.645	1.925~38.929	0.005
LVEF	−0.220	0.047	21.923	0.846	0.736~0.966	0.000
Total ischemic time	0.758	0.173	19.079	2.133	1.518~2.997	0.000
Constant	−19.186	3.097	38.370	0.000	–	0.000

Development and Evaluation of the Nomogram Model for the Risk of Left Ventricular Thrombus Formation After PCI in STEMI Patients

Including the aforementioned six indicators: history of angina, ventricular aneurysm, alcohol abuse history, postoperative TIMI grade ≤2, LVEF, and total ischemia time, the nomogram model for the risk of left ventricular thrombus formation after PCI in STEMI patients was constructed using the rms package in R 4.3.3 software, as shown in Figure 2. In internal validation, the calibration curve showed high consistency between predicted and actual probabilities, with an concordance index of the ROC curve being 0.962 (95% CI: 0.9310.994), indicating high model calibration and discrimination; the H-L test $\chi2$=11.977 (P=0.152). See Figure 3A and B. In external validation, the calibration curve also showed high consistency between predicted and actual probabilities, with an concordance index of the ROC curve being 0.958 (95% CI: 0.9260.990), indicating high model discrimination and calibration; the H-L test $\chi2$=9.757 (P=0.282). See Figure 4A and B.

Clinical Utility Evaluation of the Nomogram Model for the Risk of Left Ventricular Thrombus Formation After PCI in STEMI Patients

The All line represents the assumption that all STEMI patients develop left ventricular thrombus after PCI, while the None line represents the assumption that no STEMI patients develop left ventricular thrombus early after PCI. The DCA

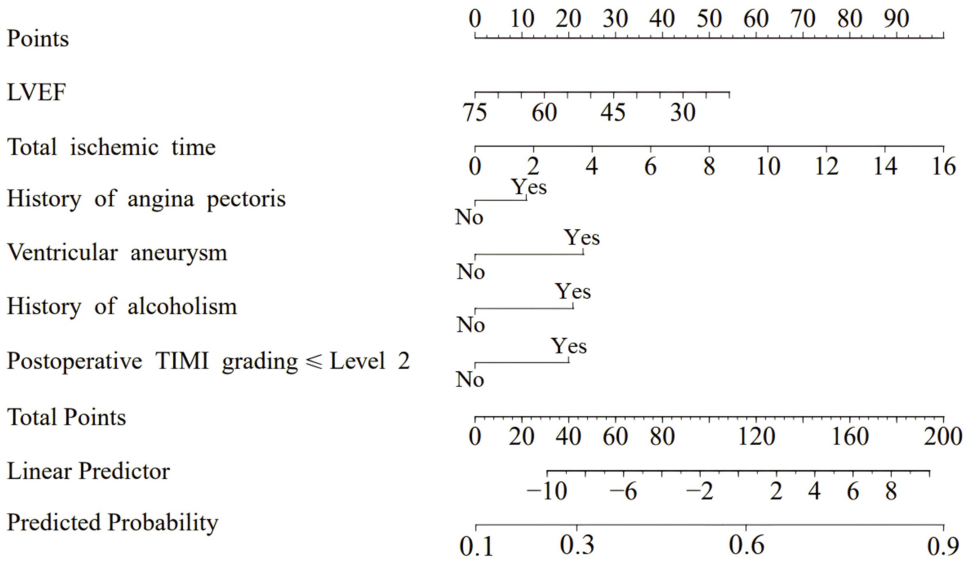

Figure 2 A risk nomogram for left ventricular thrombosis after PCI surgery.
Notes: Draw vertical on the "Points" axis from the corresponding points of each variable in the nomogram, and scale score is the score of that variable. The point corresponding to the "Predicted Probability" axis is the corresponding early left ventricular thrombosis formation risk in STEMI patients after PCI.

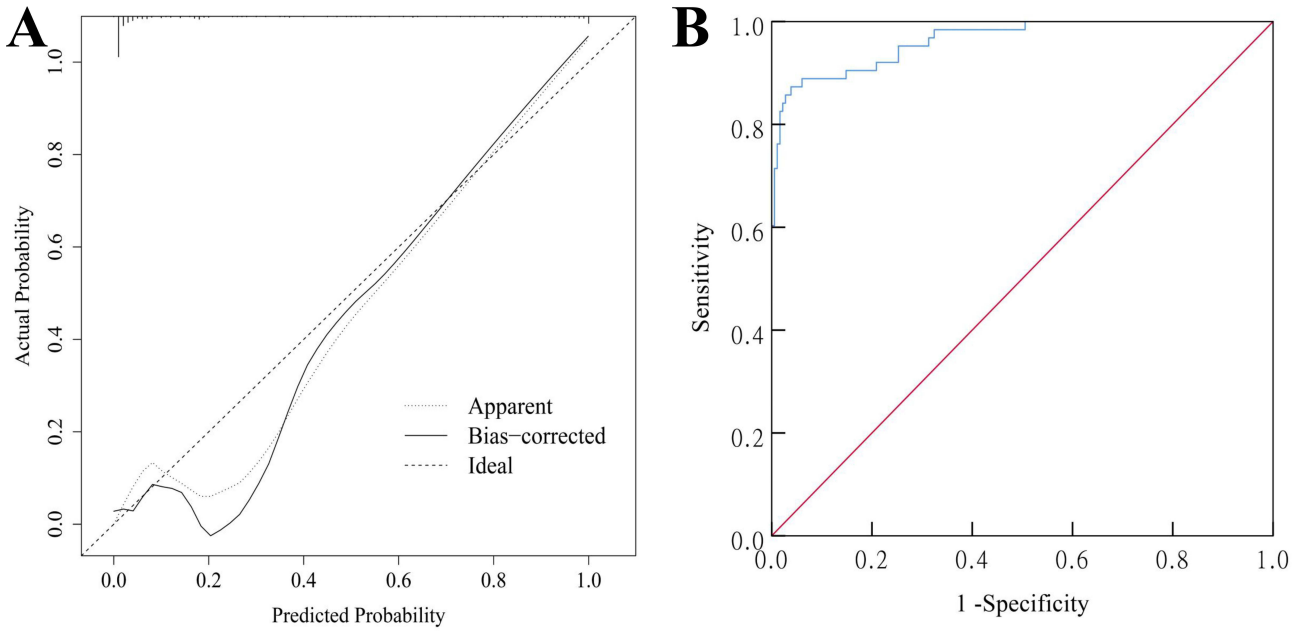

Figure 3 Correction curves (**A**) and ROC curves (**B**) of the risk nomogram for left ventricular thrombosis after PCI in the modeling group.

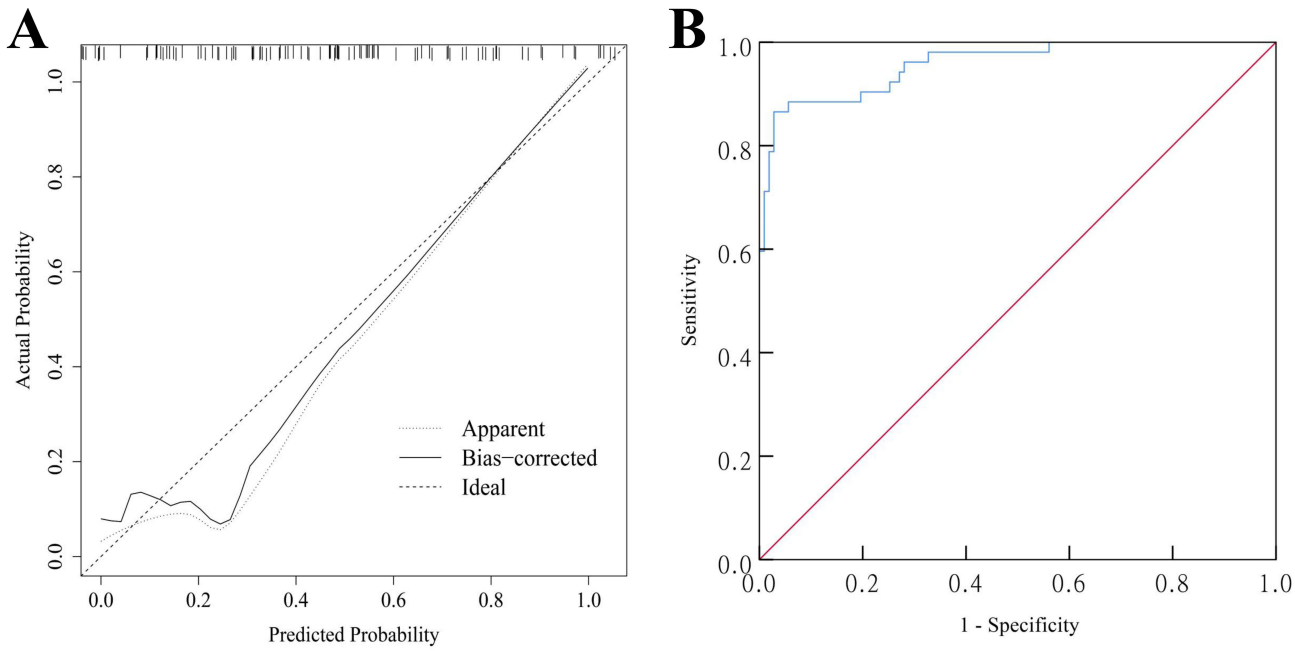

Figure 4 Correction curves (**A**) and ROC curves (**B**) of the risk nomogram for left ventricular thrombosis after PCI in the validation group.

curve shows that the nomogram model performs better in the high-risk threshold probability range of 0.02~0.99, yielding a higher net benefit. See Figure 5.

Discussion

The clinical hazard of left ventricular thrombosis mainly lies in the rupture and detachment of thrombi, leading to embolism in peripheral arteries and critical organs, presenting a high risk of disability or death to patients.[10] Therefore, identifying the incidence and risk factors of left ventricular thrombosis in STEMI patients and providing early treatment

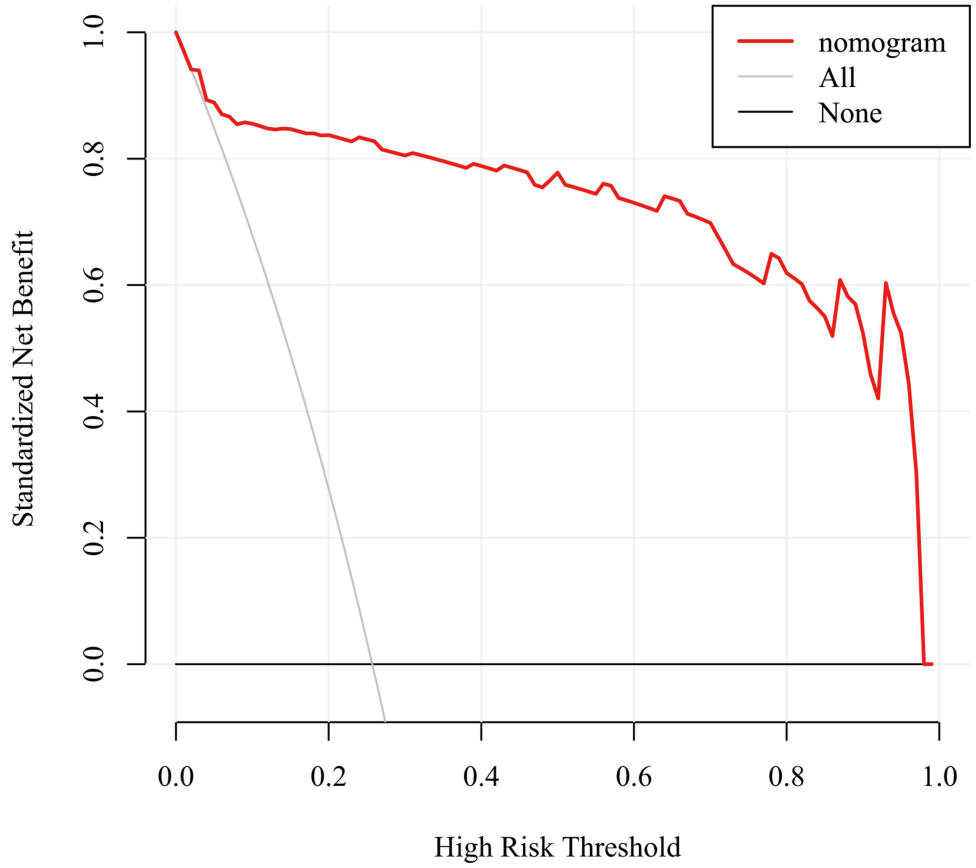

Figure 5 DCA curve of nomogram for left ventricular thrombosis after PCI.

for high-risk patients to prevent severe embolic events are of great significance. Cardiac magnetic resonance imaging is the gold standard for diagnosing left ventricular thrombosis, but it is costly and not widely available.[11,12] Moreover, given that left ventricular thrombosis is a complex disease involving various pathophysiological mechanisms, binary diagnostic and predictive methods, such as cardiac magnetic resonance imaging, cannot reflect the complexity of the patient's condition.[13] Thus, a quantitative model based on multiple factors to determine the risk of left ventricular thrombosis is needed.

Multifactorial analysis revealed that history of angina, ventricular aneurysm, alcohol abuse history, postoperative TIMI grade ≤2, LVEF, and total ischemia time are influencing factors for left ventricular thrombosis after PCI in STEMI patients. (1) History of angina. Angina is mainly caused by coronary artery stenosis due to arteriosclerosis leading to myocardial ischemia.[14,15] It is hypothesized that STEMI patients with a history of angina have relatively severe coronary stenosis, increasing the risk of left ventricular thrombosis. (2) Ventricular aneurysm. The presence of ventricular aneurysms can lead to weakened or absent myocardial motion in the affected area, slowing blood flow within the aneurysm, causing stasis, and increasing the risk of left ventricular thrombosis.[16] (3) Alcohol abuse history. Excessive alcohol intake can lead to various complications in the liver, myocardium, pancreas, etc., causing reduced blood flow and a hypercoagulable state, which can trigger thrombosis.[17] Furthermore, excessive alcohol consumption can directly increase the risk of venous thrombosis. Excessive alcohol intake can cause oxidative stress damage, stimulate platelet aggregation, and activate various inflammatory cytokines, thereby increasing the risk of left ventricular dilation and thrombosis. (4) Postoperative TIMI grade ≤2 and total ischemia time. In this study, a postoperative TIMI grade ≤2 added an impact weight of 17.21 points, and each additional 2 hours of total ischemia time increased the impact weight on left ventricular thrombosis by 12.5 points. Previous studies have shown that a postoperative TIMI grade ≤2 indicates continuous myocardial ischemia, decreased left ventricular contractile function, slowed left ventricular blood flow, and

a significant increase in platelet activation factors and inflammatory cytokines,[18] all of which can lead to the formation of left ventricular thrombosis. (5) LVEF. Studies by Wang et al[19] found that STEMI patients with lower LVEF after PCI had larger MI areas, reduced myocardial contractility, significant pump dysfunction, and left ventricular blood stasis, tending towards left ventricular thrombosis. A similar conclusion was drawn in this study: lower LVEF after PCI in STEMI patients increased the risk of left ventricular thrombosis, with each 5% decrease in LVEF adding 8.42 points to the impact weight in the nomogram. However, due to the limitations of the data included in this study, LVEF was not stratified for analysis, and therefore its threshold value could not be determined.

The nomogram model, based on the results of logistic proportional hazards or logistic regression analysis, graphically and visually predicts individual disease risks, making it more intuitive and easier to implement in clinical practice.[20] Compared with traditional risk scoring systems, the nomogram model integrates more risk factors and provides numerical probabilities of the target event, quantifying risks more accurately and offering more flexible application.[21,22] This study developed an easy-to-use nomogram model, integrating six clinically accessible and routinely collected indicators, providing an accurate and effective tool for predicting left ventricular thrombosis after PCI. Both internal and external validations showed that the nomogram had good predictive performance, high discrimination and calibration, with concordance index of 0.962 and 0.958, respectively, and the H-L test results indicated no bias between the predicted and actual values of the nomogram model. In the study by Li et al[23] the nomogram demonstrated strong predictive accuracy for left ventricular thrombus in patients with non-valvular atrial fibrillation, with C-index values of 0.836 in the training group and 0.794 in the validation group. Another study by Li et al[24] showed a C-index of 0.92 for predicting ventricular thrombus in patients with dilated cardiomyopathy, based on internal validation. The results of the present study exceed those reported in the previous studies. Clinicians can utilize the nomogram developed in this research to identify high-risk STEMI patients who may develop left ventricular thrombus, enabling closer monitoring, early diagnosis, and targeted prevention and treatment. Furthermore, the newly constructed nomogram model may serve as a potential alternative to transthoracic echocardiography for STEMI patients who are unable to tolerate the procedure.

In conclusion, the incidence of left ventricular thrombosis is high in STEMI patients after PCI treatment, with history of angina, ventricular aneurysm, alcohol abuse history, postoperative TIMI grade ≤2, LVEF, and total ischemia time being its risk factors. The nomogram model constructed from these risk factors has high predictive value for left ventricular thrombosis after PCI in STEMI patients. Clinicians should pay close attention to patients at high risk for left ventricular thrombosis predicted by the nomogram model, actively provide appropriate treatment, and increase the frequency of examinations and follow-ups to reduce the occurrence of further complications.

Limitations

This study still has some limitations. First, as a cross-sectional retrospective study, the argument is not comprehensive enough, and whether the identified risk factors are merely markers of left ventricular thrombosis or have a causal relationship still needs further determination. Second, due to the limitations of small sample analysis and variability in follow-up time, the results may exhibit high heterogeneity. Third, the collinearity of related risk factors, such as history of angina and LVEF, which can both cause myocardial damage and exacerbate each other, may affect the analysis results of this study. Fourth, as a retrospective analysis, this study may have selection bias in the sample selection process. Lastly, all patients included in this study were from the same hospital and race, so factors like medical standards and race might influence the outcomes.

Data Sharing Statement

The original contributions presented in the study are included in the article.

Ethics Approval

This study involving human participants were reviewed and approved by the ethical standards of the Medical Ethics Committee of The Affiliated Nanhua Hospital, Hengyang Medical School, University of South China and with the 1964 helsinki Declaration. And obtain the informed consent form of the patient or their guardian, and sign on the informed consent form.

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Disclosure

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