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Original Article

Risk factors for heart failure within one year after percutaneous coronary intervention in patients with acute coronary syndrome: development of a predictive model

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Abstract: Objective: To identify risk factors for heart failure (HF) within one year after percutaneous coronary intervention (PCI) in patients with acute coronary syndrome (ACS) and to develop a predictive nomogram model. Methods: A retrospective analysis was performed on 492 patients with ACS treated at Suzhou Municipal Hospital between January 2020 and October 2023. Patients were divided into the HF group and the non-HF group according to the occurrence of HF within one year after PCI. 70% of the cases were randomly assigned to the training set and 30% to the validation set. Univariate and multivariate logistic regression analyses were conducted to screen independent predictors, and a nomogram model was subsequently established. Model performance was evaluated using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA). Results: Among the 492 patients, the incidence of HF within one year after PCI was 26.42% (n = 130). Logistic regression identified type 2 diabetes mellitus (T2DM), left ventricular ejection fraction (LVEF), lipoprotein(a) [LP(a)], B-type natriuretic peptide (BNP), and high-sensitivity C-reactive protein (Hs-CRP) as independent predictors of HF, with odds ratios of 5.756, 0.904, 1.427, 1.012, and 1.666, respectively (all $P < 0.05$). The model demonstrated excellent discrimination, with areas under the ROC curve of 0.946 in the training set and 0.958 in the validation set. DCA indicated that the model provided greater net clinical benefit than the "treat-all" or "treat-none" strategies, and its predictive performance surpassed that of each individual factor ($P < 0.05$). Conclusion: The nomogram model incorporating T2DM, LVEF, LP(a), BNP and Hs-CRP provides an effective tool for predicting HF risk within one year after PCI in patients with ACS, offering valuable guidance for early clinical identification and risk stratification of high-risk individuals.

Keywords: Acute coronary syndrome, percutaneous coronary intervention, heart failure, risk factors, nomogram

Introduction

With the rapid development of the social economy and changes in lifestyle, particularly the acceleration of population aging and urbanization, unhealthy behaviors have become increasingly prevalent, leading to a continuous rise in the incidence of cardiovascular diseases (CVD) [1, 2]. According to data from the Global Burden of Disease Study, the age standardized incidence rate of CVD among Chinese individuals aged 1-79 years increased from 646.2 to 652.2 per 100,000 person-years between 1990 and 2019 [3]. Acute coronary syndrome (ACS) refers to a spectrum of clinical conditions characterized by acute myocardial ischemia caused by thrombosis secondary to the rupture or erosion of unstable atherosclerotic plaques in the coronary arteries. ACS represents an acute manifestation of coronary atherosclerotic heart disease [4-6]. Percutaneous coronary intervention (PCI) has become the preferred treatment for acute CVD because it can rapidly and effectively restore blood flow in the infarct-related artery.

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However, even when standard diagnostic and therapeutic strategies recommended by clinical guidelines are strictly followed, some patients with ACS still experience major adverse cardiovascular events after PCI, among which heart failure (HF) is one of the most common complications [7].

The main clinical manifestations of HF include dyspnea, exercise intolerance, and fluid retention. As the condition progresses, patients may develop pulmonary congestion, shortness of breath, chest tightness, bilateral lower-limb edema, fatigue, dizziness, and other related symptoms. The development of HF after PCI not only leads to a significant increase in hospital readmissions and medical expenses but also severely impairs patients' quality of life and reduces survival. Moreover, HF imposes substantial psychological and financial burdens on both patients and their families. Therefore, early identification and assessment of HF risk in patients with ACS after PCI are crucial for implementing timely and targeted interventions in high-risk individuals, thereby reducing the incidence of HF. Previous studies have indicated that the risk of post-PCI HF in ACS patients is influenced by multiple factors, including demographic characteristics, clinical features, biochemical markers, and structural as well as functional cardiac indices [8, 9]. Consequently, evaluating risk based solely on a single factor may not provide an accurate assessment of HF risk after PCI in patients with ACS.

A disease prediction model is a statistical or machine learning approach used to predict an individual's or population's future health status, disease risk, or disease progression. Machine learning algorithms commonly applied in clinical settings for prognostic modeling include linear regression, logistic regression, decision trees, support vector machines, gradient boosting, random forests, and neural networks [10, 11]. In the field of prognosis prediction for patients ACS, several studies have employed machine learning algorithms to establish predictive models. For instance, Sherazi et al. [12] developed random forest, decision tree, and gradient boosting models to predict in-hospital mortality risk in ACS patients. Similarly, Qin et al. [13] constructed a nomogram based on clinical data from ACS patients and reported that the model achieved an area under the curve (AUC) of 0.837 for predicting the risk of reperfusion arrhythmia during PCI. However, most existing studies have focused on short-term mortality or the risk of reinfarction after PCI, while predictive models for HF within one year post-PCI remain limited-particularly those integrating multidimensional variables such as clinical characteristics, biochemical markers, and imaging parameters. Therefore, this study aimed to develop a logistic regression-based nomogram model to predict the one-year risk of HF in patients with ACS after PCI, with the goal of facilitating early identification of high-risk individuals and providing a reference for clinical decision-making.

Materials and methods

Study population

A total of 492 patients with ACS who underwent treatment at Suzhou Municipal Hospital between January 2020 and October 2023 were included in this study. Inclusion criteria: (1) Diagnosis of ACS confirmed according to established clinical criteria; (2) Age ≥ 18 years; (3) First occurrence of ACS at the time of this admission; (4) Coronary angiography and PCI performed in accordance with current clinical guidelines; (5) Complete clinical data available, with a missing value rate ≤ 5%. Exclusion criteria: (1) Receipt of thrombolytic therapy before admission; (2) Presence of severe complications after ACS, such as cardiac rupture or cardiogenic shock, that could significantly affect survival; (3) Severe hepatic or renal dysfunction or ongoing dialysis treatment; (4) Severe systemic diseases such as sepsis or septic shock; (5) Congenital heart disease, myocarditis, or cardiomyopathy; (6) Malignant tumors, severe autoimmune diseases, or other serious comorbidities affecting survival time. This study was approved by the Ethics Committee of Suzhou Municipal Hospital.

Sample size calculation

This study employed multivariate logistic regression analysis to identify factors associated with the occurrence of HF within one year after PCI in patients with ACS. According to the general rule for logistic regression, the minimum required sample size should be at least 5-10 times the number of independent variables included in the model. In this study, 32 poten-

tial predictors were collected, indicating a minimum required sample size of 160-320 cases. Considering the available patient data in our hospital, a total of 492 patients were ultimately included in the analysis.

Data collection

Clinical data of all enrolled patients were collected, including the following categories: 1. General demographic information: sex, age, body mass index, smoking history, drinking history, type 2 diabetes mellitus (T2DM), hypertension, and hyperlipidemia. 2. Echocardiographic parameters: left atrial diameter, left ventricular end-systolic dimension, left ventricular end-diastolic dimension, left ventricular ejection fraction (LVEF), and the number of diseased coronary vessels. 3. Laboratory indicators: (a) Hematologic values: white blood cell count, neutrophil count, hemoglobin, red cell distribution width, hematocrit, mean platelet volume, and platelet count. (b) Biochemical indexes: serum creatinine, blood urea nitrogen, aspartate aminotransferase, serum potassium, creatine kinase-MB (CK-MB), lipoprotein(a) [LP(a)], direct bilirubin, indirect bilirubin, troponin T, and B-type natriuretic peptide (BNP). (c) Coagulation and inflammatory markers: prothrombin time and high-sensitivity C-reactive protein (Hs-CRP). 4. PCI-related parameters: operative duration and whether implantation was performed.

Grouping and model construction

Patients were categorized into the HF group and non-HF group according to the occurrence of HF within one year after PCI. Using the random sampling function in RStudio software (version 4.5.1X; R Foundation for Statistical Computing, Vienna, Austria), all 492 patients were randomly divided into two subsets: 70% (n = 344) were assigned to the training set for model development, and the remaining 30% (n = 148) were assigned to the validation set for model performance evaluation. Univariate analysis was first performed in the training set to identify variables that showed significant differences between the HF and non-HF groups. These significant variables were then entered into a multivariate logistic regression analysis, with the occurrence of HF as the dependent variable, to determine independent predictors. Based on the regression results, a nomogram was constructed to visualize the predictive model, displaying each variable, its corresponding score, total score, and predicted risk of HF. The model's performance was assessed using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA) to evaluate discrimination, calibration accuracy, and clinical net benefit, respectively. The predictive performance of the nomogram was further compared with that of each individual variable using ROC curve analysis.

Statistical analysis

All statistical analyses were performed using SPSS software (version 26.0; IBM Corp., Armonk, NY, USA) and R software (version 4.2.1; R Foundation for Statistical Computing, Vienna, Austria). Continuous variables with a normal distribution were expressed as mean ± standard deviation, whereas non-normally distributed variables were presented as median (interquartile range). Differences between two groups were compared using the independent-samples t test for normally distributed data and the Mann-Whitney U test for non-normally distributed data. Categorical variables were expressed as frequency (%) and compared using the chi-square test; ordinal categorical variables were analyzed using the rank-sum test. 70% of the total sample (n = 492) was randomly assigned to the training set for model construction, while the remaining 30% was used as the validation set to assess model performance. Model discrimination, calibration, and clinical utility were evaluated using ROC curve analysis, calibration curves, and DCA, respectively. A two-sided P value < 0.05 was considered significant.

Results

Baseline characteristics of the study population

The study participants were randomly divided into training and validation sets in a 7:3 ratio. The training set included 344 patients with ACS, among whom 94 (27.33%) developed HF within one year after PCI. The validation set comprised 148 patients, of whom 36 (24.32%) developed HF. The baseline clinical characteristics of patients in the two datasets are summarized in **Table 1**. Statistical analysis revealed no significant differences in baseline characteris-

Table 1. Baseline characteristics of the study population

Variable	Training set (n = 344)	Validation set (n = 148)	χ^2/t/Z	*P* value
Sex, n (%)			0.052	0.820
Male	206 (59.88)	87 (58.78)		
Female	138 (40.12)	61 (41.22)		
Age (years)			0.006	0.939
≥ 65	229 (66.57)	98 (66.22)		
< 65	115 (33.43)	50 (33.78)		
BMI (kg/m^2)	23.28 ± 2.28	23.21 ± 2.36	0.329	0.742
Smoking history, n (%)			2.191	0.139
Yes	143 (41.57)	51 (34.46)		
No	201 (58.43)	97 (65.54)		
Drinking history, n (%)			0.608	0.436
Yes	95 (27.62)	46 (31.08)		
No	249 (72.38)	102 (68.92)		
T2DM, n (%)			0.276	0.599
Yes	140 (40.70)	64 (43.24)		
No	204 (59.30)	84 (56.76)		
Hypertension, n (%)			1.558	0.212
Yes	151 (43.90)	56 (37.84)		
No	193 (56.10)	92 (62.16)		
Hyperlipidemia, n (%)			0.029	0.865
Yes	39 (11.34)	16 (10.81)		
No	305 (88.66)	132 (89.19)		
LA (mm)	38.03 ± 6.46	38.27 ± 6.40	0.380	0.704
LVESD (mm)	38.21 ± 6.00	38.28 ± 7.26	0.118	0.906
LVEDD (mm)	51.10 ± 5.35	50.92 ± 4.99	0.349	0.727
LVEF (%)	59.24 ± 7.97	58.99 ± 8.12	0.307	0.759
Diseased vessels, n	1 (1, 2)	1 (1, 2)	0.534	0.593
WBC (×10^9/L)	11.14 ± 2.54	11.04 ± 2.60	0.367	0.714
NEUT (×10^9/L)	6.12 ± 1.33	6.18 ± 1.31	0.466	0.641
HGB (g/L)	145.80 ± 17.61	145.82 ± 17.34	0.016	0.987
RDW (%)	13.07 ± 0.74	13.11 ± 0.80	0.594	0.553
MPV (fL)	10.16 ± 1.55	10.22 ± 1.67	0.430	0.667
HCT (%)	43.50 ± 4.78	43.26 ± 4.65	0.499	0.618
PLT (×10^9/L)	272.20 ± 39.89	275.17 ± 37.36	0.773	0.440
Scr (μmol/L)	81.76 ± 10.92	81.71 ± 10.63	0.053	0.958
BUN (mmol/L)	6.12 ± 2.04	6.49 ± 3.15	1.551	0.122
AST (U/L)	44.95 (31.03, 88.60)	49.45 (31.75, 91.08)	0.625	0.532
K^+ (mmol/L)	3.96 ± 0.55	3.97 ± 0.47	0.057	0.954
CK-MB (U/L)	31.09 (18.48, 66.38)	24.80 (15.50, 76.38)	0.649	0.516
LP(a) (mg/dL)	17.77 ± 4.04	17.83 ± 4.17	0.163	0.871
DBIL (μmol/L)	8.30 (6.60, 10.10)	8.3 (7.00, 10.48)	0.637	0.524
IBIL (μmol/L)	9.20 (6.80, 12.40)	9.10 (7.23, 11.38)	0.723	0.470
TnT (pg/mL)	41.25 ± 13.37	43.35 ± 12.80	1.621	0.106
BNP (pg/mL)	83.98 ± 12.43	82.21 ± 11.06	1.504	0.133
PT (s)	11.91 ± 2.27	11.76 ± 2.79	0.621	0.535
Hs-CRP (mg/L)	7.69 ± 1.85	7.39 ± 1.93	1.666	0.096
Procedure duration (min)	64.56 ± 11.70	64.95 ± 12.42	0.339	0.490

Stent implantation, n (%)			0.002	0.965
Yes	254 (73.84)	109 (73.65)		
No	90 (26.16)	39 (26.35)		

Abbreviations: BMI, body mass index; T2DM, type 2 diabetes mellitus; LA, left atrial diameter; LVESD, left ventricular end-systolic dimension; LVEDD, left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; WBC, white blood cell; NEUT, neutrophil; HGB, hemoglobin; RDW, red cell distribution width; HCT, hematocrit; MPV, mean platelet volume; PLT, platelet count; Scr, serum creatinine; BUN, blood urea nitrogen; AST, aspartate aminotransferase; K^+, serum potassium; CK-MB, creatine kinase-MB; LP(a), lipoprotein(a); DBIL, direct bilirubin; IBIL, indirect bilirubin; TnT, troponin T; BNP, B-type natriuretic peptide; PT, prothrombin time; Hs-CRP, high-sensitivity C-reactive protein.

tics between the two sets (all P > 0.05), indicating good comparability.

Characteristics of patients with and without HF in the training set

In the training set, significant differences were observed between patients with and without HF in several variables, including T2DM, hyperlipidemia, LVEF, LP(a), BNP, and Hs-CRP (all P < 0.05). The detailed clinical characteristics of the HF and non-HF groups are presented in **Table 2**.

Univariate and multivariate analyses

Variables that showed significant differences between the HF and non-HF groups were included as independent variables, with the occurrence of HF serving as the dependent variable. Univariate and multivariate logistic regression analyses were performed to identify independent predictors of HF (variable assignments are presented in **Table 3**). Multivariate logistic regression analysis revealed that T2DM, LVEF, LP(a), BNP and Hs-CRP were independent risk factors for HF within one year after PCI in patients with ACS. The corresponding odds ratios were 1.506, 0.917, 1.438, 1.082, and 1.735, respectively (all P < 0.05), as shown in **Table 4**.

Construction of the nomogram model

The results from the multivariate logistic regression analysis were visualized to develop the nomogram model, as shown in **Figure 1**. The nomogram consists of each independent factor and its corresponding scale, with each scale representing the score for that factor. Researchers can determine the score of an individual factor based on the patient's specific characteristics. The total score for a patient is obtained by summing the individual scores of all relevant factors. The risk HF within one year after PCI can be calculated by drawing a vertical line downward from the total score to the corresponding risk value on the risk axis. For example, for a patient with diabetes, an LVEF of 90%, an LP(a) level of 22 mg/dL, a BNP level of 350 pg/mL, and an Hs-CRP level of 7 mg/L, the total score is calculated as follows: 18 + 0 + 53 + 35 + 22.5 = 128.5, resulting in a risk value of less than 0.1.

Inspection of the nomogram model

ROC curve analysis was performed to assess the discrimination ability of the nomogram model in both the training set and validation set, as shown in **Figure 2A**, **2B**. The AUC for the model in the training set was 0.946 (95% confidence interval [CI]: 0.925-0.967), and in the validation set, it was 0.958 (95% CI: 0.930-0.986), both of which are > 0.9, indicating excellent discriminatory power. The calibration curves shown in **Figure 3A**, **3B** demonstrate that the predictive accuracy of the model in both the training set and validation set aligns closely with the corrected prediction accuracy, indicating good calibration. As depicted in the DCAs in **Figure 4A**, **4B**, based on the incidence rate of HF of 27.33%, the model demonstrated a higher net benefit than the "all" and "none" strategies across a range of threshold probabilities. In the training set, the net benefit of the model exceeded that of the "all" curve and the "none" curve across threshold probabilities ranging from 1% to 100%. In the validation set, the model's net benefit remained higher than the "all" and "none" curves within threshold probabilities of 1% to 95%. The "all" curve refers to the intervention strategy in which all patients receive treatment, regardless of HF risk, leading to a decrease in net benefit as the threshold increases due to overtreatment. The "none" curve refers to a strate-

Table 2. Baseline characteristics of patients with and without HF in the training set

Variable	HF group (n = 94)	Non-HF group (n = 250)	χ^2/t/Z	*P* value
Sex, n (%)			1.112	0.290
Male	52 (55.32)	154 (61.60)		
Female	42 (44.68)	96 (38.40)		
Age (years)			0.771	0.380
≥ 65	66 (70.21)	163 (65.20)		
< 65	28 (29.79)	87 (34.80)		
BMI (kg/m^2)	23.48 ± 2.42	23.20 ± 2.22	0.996	0.320
Smoking history, n (%)			0.515	0.473
Yes	42 (44.68)	101 (40.40)		
No	52 (55.32)	149 (59.60)		
Drinking history, n (%)			0.281	0.596
Yes	24 (25.53)	71 (28.40)		
No	70 (74.47)	179 (71.60)		
T2DM, n (%)			26.100	< 0.001
Yes	59 (62.77)	81 (32.40)		
No	35 (37.23)	169 (67.60)		
Hypertension, n (%)			0.304	0.581
Yes	39 (41.49)	112 (44.80)		
No	55 (58.51)	138 (55.20)		
Hyperlipidemia, n (%)			5.859	0.015
Yes	17 (18.09)	22 (8.80)		
No	77 (81.91)	228 (91.20)		
LA (mm)	38.33 ± 6.37	38.12 ± 6.49	0.277	0.782
LVESD (mm)	37.96 ± 5.28	38.30 ± 6.26	0.477	0.634
LVEDD (mm)	51.03 ± 5.72	51.12 ± 5.21	0.142	0.887
LVEF (%)	55.17 ± 5.68	60.76 ± 8.19	6.099	< 0.001
Diseased vessels, n	1 (1, 2)	1 (1, 2)	0.452	0.651
WBC (×10^9/L)	11.21 ± 2.48	11.11 ± 2.56	0.342	0.733
NEUT (×10^9/L)	5.93 ± 1.37	6.19 ± 1.31	1.664	0.097
HGB (g/L)	145.01 ± 20.05	146.09 ± 16.63	0.507	0.612
RDW (%)	12.99 ± 0.67	13.10 ± 0.67	1.185	0.237
MPV (fL)	10.22 ± 1.55	10.13 ± 1.55	0.476	0.634
HCT (%)	43.16 ± 4.75	43.62 ± 4.79	0.810	0.421
PLT (×10^9/L)	270.26 ± 39.20	272.92 ± 40.20	0.551	0.582
Scr (μmol/L)	82.58 ± 11.97	81.45 ± 10.51	0.856	0.393
BUN (mmol/L)	6.12 ± 1.91	6.11 ± 2.09	0.002	0.998
AST (U/L)	49.85 (30.53, 112.60)	43.25 (31.23, 79.93)	0.765	0.444
K^+ (mmol/L)	3.91 ± 0.42	3.98 ± 0.59	1.147	0.252
CK-MB (U/L)	29.99 (18.55, 71.74)	31.74 (18.35, 64.27)	0.035	0.972
LP(a) (mg/dL)	21.29 ± 4.28	16.44±3.03	10.079	< 0.001
DBIL (μmol/L)	8.95 (6.80, 10.23)	8.20 (6.50, 10.03)	0.984	0.325
IBIL (μmol/L)	9.10 (6.58, 12.60)	9.25 (6.86, 12.40)	0.182	0.856
TnT (pg/mL)	42.47 ± 13.11	40.79 ± 13.46	1.035	0.302
BNP (pg/mL)	92.31 ± 12.14	80.86 ± 11.03	8.342	< 0.001
PT (s)	11.96 ± 1.62	11.88 ± 2.47	0.294	0.769
Hs-CRP (mg/L)	9.19 ± 2.13	7.13 ± 1.37	8.733	< 0.001
Procedure duration (min)	64.51 ± 10.88	64.57 ± 12.01	0.043	0.965

Stent implantation, n (%)			0.150	0.699
Yes	68 (72.34)	186 (74.40)		
No	26 (27.66)	64 (25.60)		

Abbreviations: HF, heart failure; BMI, body mass index; T2DM, type 2 diabetes mellitus; LA, left atrial diameter; LVESD, left ventricular end-systolic dimension; LVEDD, left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; WBC, white blood cells; NEUT, neutrophil; HGB, hemoglobin; RDW, red cell distribution width; HCT, hematocrit; MPV, mean platelet volume; PLT, platelet count; Scr, serum creatinine; BUN, blood urea nitrogen; AST, aspartate aminotransferase; K^+, serum potassium; CK-MB, creatine kinase-MB; LP(a), lipoprotein(a); DBIL, direct bilirubin; IBIL, indirect bilirubin; TnT, troponin T; BNP, B-type natriuretic peptide; PT, prothrombin time; Hs-CRP, high-sensitivity C-reactive protein.

Table 3. Variable assignment table

Variable	Assignment
Dependent variable	
HF	0 = HF, 1 = Non-HF
Independent variables	
T2DM	0 = "No", 1 = "Yes"
Hyperlipidemia	0 = "No", 1 = "Yes"
LVEF	Enter actual value
LP(a)	Enter actual value
BNP	Enter actual value
Hs-CRP	Enter actual value
TnT	Enter actual value

Abbreviations: HF, heart failure; T2DM, type 2 diabetes mellitus; LVEF, left ventricular ejection fraction; LP(a), lipoprotein(a); BNP, B-type natriuretic peptide; Hs-CRP, high-sensitivity C-reactive protein; TnT, troponin T.

gy in which no patients are treated, resulting in a constant net benefit of zero.

Comparison of ROC curves between the model and individual factors

As shown in **Figure 5** and **Table 5**, the AUC of the nomogram model was 0.946, whereas the AUC values for individual predictors-T2DM, LVEF, LP(a), BNP, and Hs-CRP-were 0.652, 0.706, 0.832, 0.750, and 0.782, respectively. Statistical comparison showed that the AUC of the nomogram model was significantly higher than that of T2DM, LVEF, LP(a), BNP, and Hs-CRP ($Z = 10.152$, 8.311, 5.633, 7.236, and 5.650, respectively; all $P < 0.05$), indicating that the model demonstrated superior predictive performance compared to any single variable.

Discussion

With advances in imaging technology, clinicians can now more accurately evaluate coronary artery lesions, thereby enhancing the precision and safety of PCI and improving surgical outcomes. However, although PCI effectively resolves vascular occlusion in patients with ACS, not all patients derive equivalent long-term benefits from the procedure. The incidence of HF following PCI remains high and is closely associated with adverse long-term prognosis [14]. HF not only impairs patients' quality of life but also significantly increases the risk of rehospitalization and mortality [15]. In this study, clinical data from 492 patients with ACS were analyzed, and T2DM, LVEF, LP(a), BNP, and Hs-CRP were identified as independent factors associated with the development of HF within one year after PCI.

In this study, T2DM was identified as an independent risk factor for HF in patients with ACS within one year after PCI (odds ratio: 4.510). Long-term hyperglycemia in diabetic patients can lead to insulin resistance and metabolic abnormalities. Moreover, hyperglycemia promotes the formation of advanced glycation end products, which cause damage to cardiomyocytes and vascular endothelial cells, leading to myocardial fibrosis and increased vascular stiffness. These pathologic changes subsequently impair both systolic and diastolic cardiac function [16]. In addition, acute occlusion of the coronary artery in ACS patients combined with diabetes further aggravates coronary lesions, making post-interventional recanalization less effective than in non-diabetic patients. LVEF is a key indicator of left ventricular systolic function. A reduced LVEF reflects weakened myocardial contractility and impaired cardiac pump function. In this study, the risk of HF after PCI increased with decreasing LVEF. During ACS, acute coronary occlusion leads to myocardial ischemia and necrosis, directly compromising systolic function. LVEF effectively reflects the extent and severity of myocardial infarction; a lower LVEF indicates a larger infarct size and more severe impairment of ventricular contraction [17]. LP(a) is a unique

Table 4. Univariate and multivariate analyses of factors influencing heart failure in ACS patients after PCI

Variable	Univariate analysis			Multivariate analysis		
	β	OR (95% CI)	*P* value	β	OR (95% CI)	*P* value
T2DM	1.258	3.517 (2.144-5.770)	< 0.001	1.506	4.510 (2.051-9.918)	< 0.001
Hyperlipidemia	0.828	2.288 (1.155-4.533)	0.018	0.388	1.474 (0.454-4.782)	0.518
LVEF	-0.108	0.897 (0.864-0.932)	< 0.001	-0.087	0.917 (0.864-0.973)	0.004
LP(a)	0.415	1.514 (1.367-1.677)	< 0.001	0.363	1.438 (1.267-1.632)	< 0.001
BNP	0.089	1.093 (1.066-1.121)	< 0.001	0.079	1.082 (1.047-1.118)	< 0.001
Hs-CRP	0.708	2.030 (1.701-2.423)	< 0.001	0.551	1.735 (1.396-2.155)	< 0.001
Constant	-	-	-	-14.903	-	-

Abbreviations: OR, odds ratio; CI, confidence interval; ACS, acute coronary syndrome; PCI, percutaneous coronary intervention; T2DM, type 2 diabetes mellitus; LVEF, left ventricular ejection fraction; LP(a), lipoprotein(a); BNP, B-type natriuretic peptide; Hs-CRP, high-sensitivity C-reactive protein.

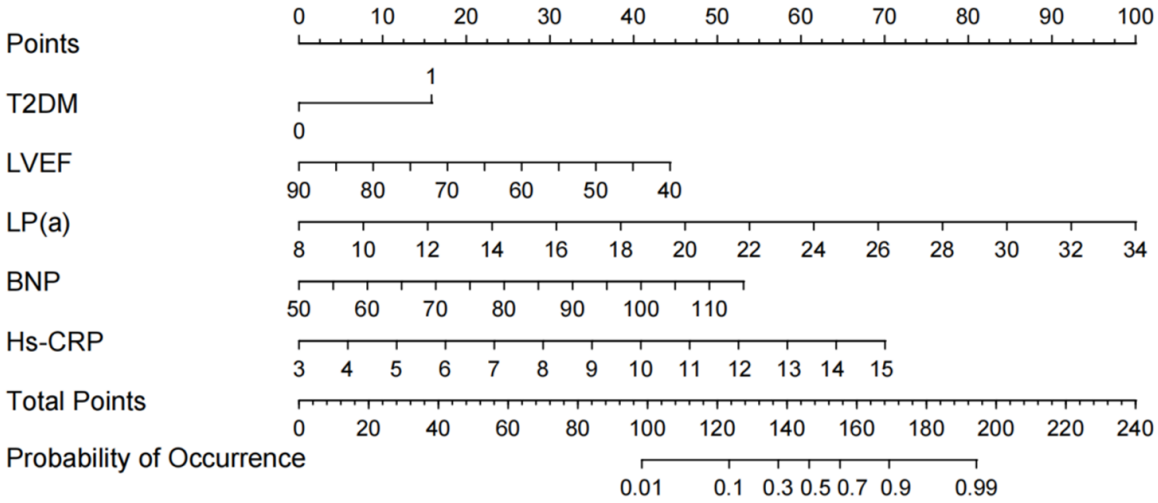

Figure 1. Nomogram for predicting HF risk in patients with ACS after PCI. Abbreviations: HF, heart failure; ACS, acute coronary syndrome; PCI, percutaneous coronary intervention; T2DM, type 2 diabetes mellitus; LVEF, left ventricular ejection fraction; LP(a), lipoprotein(a); BNP, B-type natriuretic peptide; Hs-CRP, high-sensitivity C-reactive protein.

cholesterol-rich macromolecular lipoprotein complex, consisting of cholesterol and phospholipids encapsulated within hydrophilic carriers [18]. LP(a) readily penetrates and deposits within the vascular wall, promoting the progression of atherosclerosis and thereby increasing the risk of HF after PCI. Moreover, LP(a) has been demonstrated to exert pro-inflammatory, pro-oxidative, pro-thrombotic, and pro-atherogenic effects, further contributing to cardiovascular dysfunction [19].

BNP is a key biomarker for the clinical diagnosis of HF. It is mainly secreted by ventricular myocytes in response to increased ventricular wall stress [20]. In patients with acute myocardial infarction, myocardial ischemia and necrosis lead to impaired systolic function and elevated left ventricular end-diastolic pressure, which in turn stimulate BNP secretion by cardiomyocytes. As a bioactive hormone, BNP promotes vasodilation and diuresis while inhibiting activation of the renin-angiotensin-aldosterone system [21]. Elevated BNP levels not only reflect the severity of myocardial ischemia but may also indicate the presence of reperfusion injury. Reperfusion injury can result in cardiomyocyte edema, mitochondrial dysfunction, and microvascular impairment, thereby aggravating myocardial injury, reducing cardiac pump function, and increasing the risk of HF. Stewart et al. [22] also reported that plasma BNP concentration remains an independent predictor of cardiovascular mortality even more

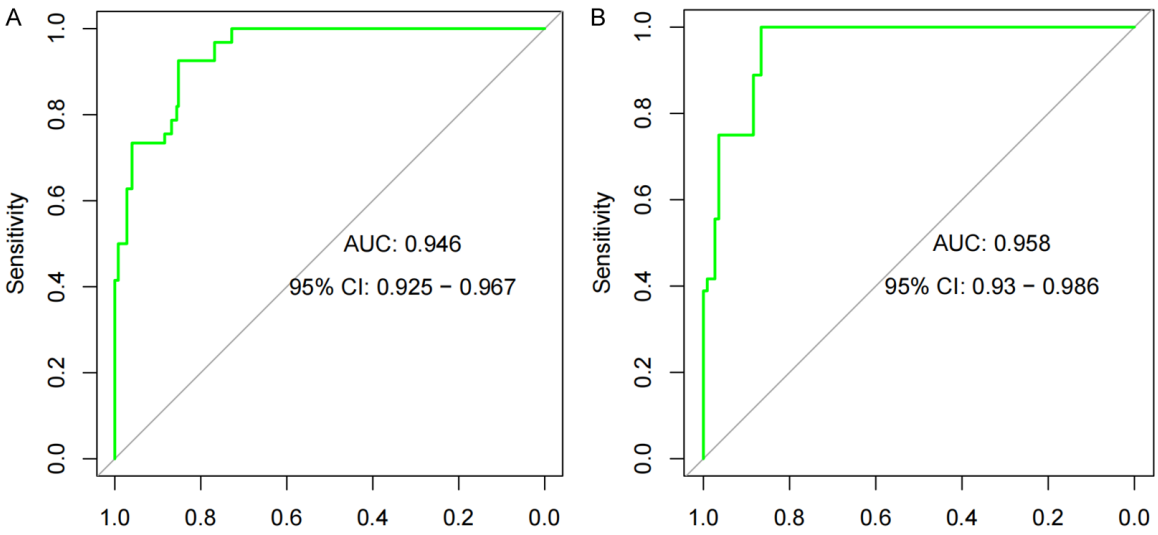

Figure 2. ROC curves of the nomogram model in the training and validation sets. A. Training set; B. Validation set. Abbreviations: ROC, receiver operating characteristic; AUC, area under the curve; CI, confidence interval.

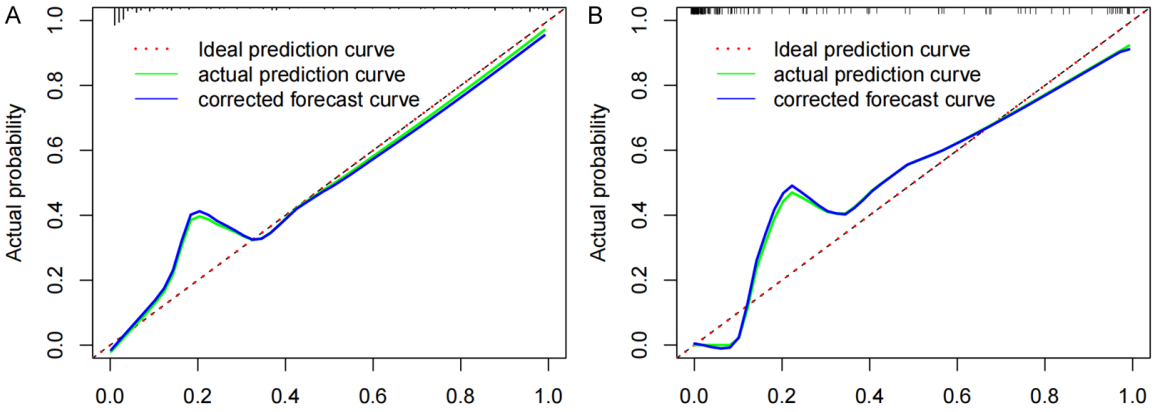

Figure 3. Calibration curves of the nomogram model in the training and validation sets. A. Training set; B. Validation set.

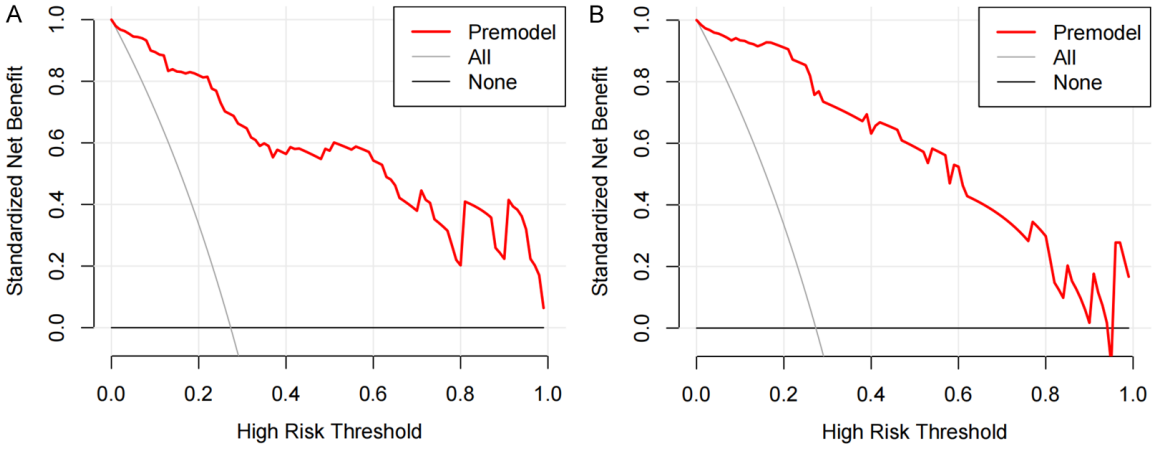

Figure 4. Decision curve analyses of the nomogram model in the training and validation sets. A. Training set; B. Validation set.

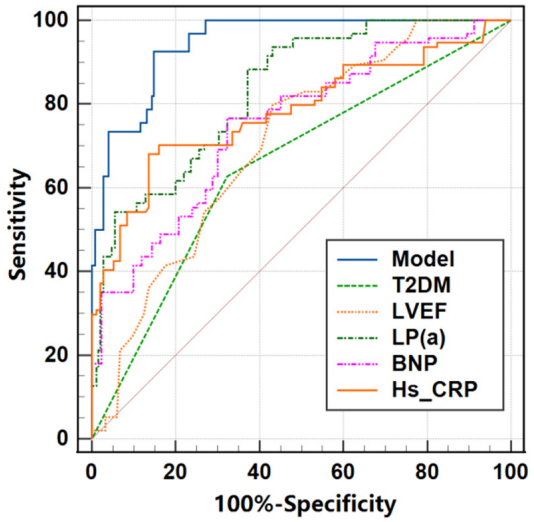

Figure 5. Comparison of ROC curves between the nomogram model and individual predictors. Abbreviations: ROC, receiver operating characteristic; T2DM, type 2 diabetes mellitus; LVEF, left ventricular ejection fraction; LP(a), lipoprotein(a); BNP, B-type natriuretic peptide; Hs-CRP, high-sensitivity C-reactive protein.

Table 5. Comparison of the area under the ROC curve between the model and individual predictors

Variable	AUC	95% CI	Z	*P* value
Model	0.946	0.925-0.967	-	-
T2DM	0.652	0.599-0.702	10.152	< 0.001
LVEF	0.706	0.655-0.754	8.311	< 0.001
LP(a)	0.832	0.788-0.870	5.633	< 0.001
BNP	0.750	0.700-0.795	7.236	< 0.001
Hs-CRP	0.782	0.734-0.824	5.650	< 0.001

Abbreviations: ROC, area under the curve; AUC, area under the curve; CI, confidence interval; T2DM, type 2 diabetes mellitus; LVEF, left ventricular ejection fraction; LP(a), lipoprotein(a); BNP, B-type natriuretic peptide; Hs-CRP, high-sensitivity C-reactive protein.

than ten years after measurement. Hs-CRP is acute-phase reactant and an important marker of inflammatory activation that plays a regulatory role in the body's innate immune response [23, 24]. During an episode of ACS, serum Hs-CRP levels rise sharply. Elevated concentrations of Hs-CRP may not only participate directly in local and systemic inflammatory reactions and exacerbate myocardial injury, but also damage vascular endothelial cells, cause vasoconstriction, and consequently increase the risk of postoperative HF in patients [25, 26].

The univariate analysis in this study suggested that a history of hyperlipidemia was associated with the occurrence of HF. However, the multivariate analysis indicated that the influence of hyperlipidemia on HF risk was not significant after adjustment for other factors. These findings further highlight that a single variable may not accurately predict the risk of HF after PCI in patients with ACS. To address this limitation, a nomogram model was developed based on the independent predictors identified through multivariate logistic regression-T2DM, LVEF, LP(a), BNP, and Hs-CRP. The model demonstrated good discriminatory ability, calibration, and clinical applicability, suggesting its utility as a decision-support tool for risk assessment in clinical practice. Nevertheless, several limitations should be acknowledged. First, this was a single-center retrospective study, and the data available for analysis were relatively limited. Second, there may be other potential factors influencing the risk of HF in ACS patients that were not included in the current model. Therefore, further prospective, multicenter studies with larger sample sizes are warranted to validate and optimize the predictive performance of this nomogram.

Conclusion

This study identified T2DM, LVEF, LP(a), BNP, and Hs-CRP as independent factors associated with the development of HF in patients with ACS after PCI. Based on these variables, a nomogram model was constructed, which demonstrated favorable predictive performance and may serve as a useful tool for identifying high-risk patients with HF and guiding individualized clinical management.

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Disclosure of conflict of interest

None.

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References

[1] Yang M, Liu J, Shen Q, Chen H, Liu Y, Wang N, Yang Z, Zhu X, Zhang S, Li X and Qian Y. Body roundness index trajectories and the incidence of cardiovascular disease: evidence from the China health and retirement longitudinal study. J Am Heart Assoc 2024; 13: e034768.

[2] Joynt Maddox KE, Elkind MSV, Aparicio HJ, Commodore-Mensah Y, de Ferranti SD, Dowd WN, Hernandez AF, Khavjou O, Michos ED, Palaniappan L, Penko J, Poudel R, Roger VL and Kazi DS; American Heart Association. Forecasting the burden of cardiovascular disease and stroke in the united states through 2050-prevalence of risk factors and disease: a presidential advisory from the American heart association. Circulation 2024; 150: e65-e88.

[3] Wang H, Zhang H and Zou Z. Changing profiles of cardiovascular disease and risk factors in China: a secondary analysis for the Global Burden of Disease study 2019. Chin Med J (Engl) 2023; 136: 2431-2441.

[4] Atwood J. Management of acute coronary syndrome. Emerg Med Clin North Am 2022; 40: 693-706.

[5] Damluji AA, Forman DE, Wang TY, Chikwe J, Kunadian V, Rich MW, Young BA, Page RL 2nd, DeVon HA and Alexander KP; American Heart Association Cardiovascular Disease in Older Populations Committee of the Council on Clinical Cardiology and Council on Cardiovascular and Stroke Nursing; Council on Cardiovascular Radiology and Intervention; and Council on Lifestyle and Cardiometabolic Health. Management of acute coronary syndrome in the older adult population: a scientific statement from the American Heart Association. Circulation 2023; 147: e32-e62.

[6] Latif A, Tran A, Ahsan J, Lateef N, Abusina W, Kapoor V, Ahsan Z, Ahmad S and Mirza M. Coronary artery aneurysms as a cause of acute coronary syndrome presentation-a focused review. Curr Cardiol Rev 2023; 19: 68-72.

[7] Xiong S, Luo Y, Chen Q, Chen Y, Su H, Long Y, Chen X, Yang S, Qi L, Huang W, Hou J, Liu H and Cai L. Adjustment of the grace score by the stress hyperglycemia ratio improves the prediction of long-term major adverse cardiac events in patients with acute coronary syndrome undergoing percutaneous coronary intervention: a multicenter retrospective study. Diabetes Res Clin Pract 2023; 198: 110601.

[8] Huang M, Li D, Li L, Wang Y, Zhang L, Chen Y and Wang DW. Factors predicting the occurrence of net adverse clinical and cerebral events in patients with acute coronary syndrome treated with clopidogrel or ticagrelor in combination with aspirin: a real-world study. Ann Transl Med 2022; 10: 98.

[9] Tromp J, Paniagua SMA, Lau ES, Allen NB, Blaha MJ, Gansevoort RT, Hillege HL, Lee DE, Levy D, Vasan RS, van der Harst P, van Gilst WH, Larson MG, Shah SJ, de Boer RA, Lam CSP and Ho JE. Age dependent associations of risk factors with heart failure: pooled population based cohort study. BMJ 2021; 372: n461.

[10] Zuo D, Yang L, Jin Y, Qi H, Liu Y and Ren L. Machine learning-based models for the prediction of breast cancer recurrence risk. BMC Med Inform Decis Mak 2023; 23: 276.

[11] Geng Z, Yang C, Zhao Z, Yan Y, Guo T, Liu C, Wu A, Wu X, Wei L, Tian Y, Hu P and Wang K. Development and validation of a machine learning-based predictive model for assessing the 90-day prognostic outcome of patients with spontaneous intracerebral hemorrhage. J Transl Med 2024; 22: 236.

[12] Sherazi SWA, Zheng H and Lee JY. A machine learning-based applied prediction model for identification of acute coronary syndrome (ACS) outcomes and mortality in patients during the hospital stay. Sensors 2023; 23: 1351.

[13] Qin Z, Fu L, Gou Q, Yang Y and He L. Establishment and application of an intraoperative reperfusion arrhythmia prediction model for PCI in elderly patients with acute coronary syndrome. Am J Transl Res 2024; 16: 6447-6456.

[14] Delmas C, Vandenbriele C and Pappalardo F. Persistent high mortality in acute myocardial infarction-associated cardiogenic shock despite early mechanical circulatory support: need for stepwise and integrated approach of care. Eur J Heart Fail 2023; 25: 2034-2036.

[15] Hernandez AF, Udell JA, Jones WS, Anker SD, Petrie MC, Harrington J, Mattheus M, Seide S, Zwiener I, Amir O, Bahit MC, Bauersachs J, Bayes-Genis A, Chen Y, Chopra VK, G AF, Ge J, S GG, Gotcheva N, Goto S, Gasior T, Jamal W, Januzzi JL, Jeong MH, Lopatin Y, Lopes RD, Merkely B, Parikh PB, Parkhomenko A, Ponikowski P, Rossello X, Schou M, Simic D, Steg PG, Szachniewicz J, van der Meer P, Vinereanu D, Zieroth S, Brueckmann M, Sumin M, Bhatt DL and Butler J. Effect of Empagliflozin on heart failure outcomes after acute myocardial infarction: insights from the EMPACT-MI trial. Circulation 2024; 149: 1627-1638.

[16] Tian H, Zhao X, Zhang Y and Xia Z. Abnormalities of glucose and lipid metabolism in myocardial ischemia-reperfusion injury. Biomed Pharmacother 2023; 163: 114827.

[17] Omote K, Verbrugge FH and Borlaug BA. Heart failure with preserved ejection fraction: mechanisms and treatment strategies. Annu Rev Med 2022; 73: 321-337.

[18] Szarek M, Reijnders E, Jukema JW, Bhatt DL, Bittner VA, Diaz R, Fazio S, Garon G, Goodman SG, Harrington RA, Ruhaak LR, Schwertfeger M, Tsimikas S, White HD, Steg PG, Cobbaert C and Schwartz GG; ODYSSEY OUTCOMES Investigators. Relating lipoprotein(a) concentrations to cardiovascular event risk after acute coronary syndrome: a comparison of 3 Tests. Circulation 2024; 149: 192-203.

[19] Dimitroglou Y, Aggeli C, Theofilis P, Tsioufis P, Oikonomou E, Tsioufis K and Tousoulis D. Lipoprotein(a) as a predictive biomarker and therapeutic target for acute coronary syndromes. Curr Pharm Des 2023; 29: 1835-1843.

[20] Harpaz D, Seet RCS, Marks RS and Tok AIY. B-type natriuretic peptide as a significant brain biomarker for stroke triaging using a bedside point-of-care monitoring biosensor. Biosensors 2020; 10: 107.

[21] Nishikimi T and Nakagawa Y. B-type Natriuretic Peptide (BNP) Revisited-Is BNP still a biomarker for heart failure in the angiotensin receptor/neprilysin inhibitor Era? Biology 2022; 11: 1034.

[22] Stewart RAH, Kirby A, White HD, Marschner SL, West M, Thompson PL, Sullivan D, Janus E, Hunt D, Kritharides L, Keech A, Simes J and Tonkin AM. B-type natriuretic peptide and long-term cardiovascular mortality in patients with coronary heart disease. J Am Heart Assoc 2022; 11: e024616.

[23] Gao Y, Wang M, Wang R, Jiang J, Hu Y, Wang W, Wang Y and Li H. The predictive value of the hs-CRP/HDL-C ratio, an inflammation-lipid composite marker, for cardiovascular disease in middle-aged and elderly people: evidence from a large national cohort study. Lipids Health Dis 2024; 23: 66.

[24] Cui C, Liu L, Qi Y, Han N, Xu H, Wang Z, Shang X, Han T, Zha Y, Wei X and Wu Z. Joint association of TyG index and high sensitivity C-reactive protein with cardiovascular disease: a national cohort study. Cardiovasc Diabetol 2024; 23: 156.

[25] Burger PM, Koudstaal S, Mosterd A, Fiolet ATL, Teraa M, van der Meer MG, Cramer MJ, Visseren FLJ, Ridker PM and Dorresteijn JAN; UCC-SMART study group. C-reactive protein and risk of incident heart failure in patients with cardiovascular disease. J Am Coll Cardiol 2023; 82: 414-426.

[26] Ferreira JP, Claggett BL, Liu J, Sharma A, Desai AS, Anand IS, O'Meara E, Rouleau JL, De Denus S, Pitt B, Pfeffer MA, Zannad F and Solomon SD. High-sensitivity C-reactive protein in heart failure with preserved ejection fraction: findings from topcat. Int J Cardiol 2024; 402: 131818.