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Original Article

Risk factors for in-hospital heart failure in patients with acute myocardial infarction and construction of predictive models

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Abstract: Objective: To identify risk factors and develop a predictive model for heart failure in patients with acute myocardial infarction (AMI). Methods: Clinical data from 312 AMI patients were retrospectively collected. Patients were divided into a Heart failure group and a non-heart failure group based on the occurrence of heart failure during hospitalization. Comparative analyses were performed between the two groups. Multivariate logistic regression analysis was used to identify risk factors of in-hospital heart failure. A nomogram prediction model was constructed using R software. The model's performance was evaluated by receiver operating characteristic (*ROC*) curve analysis, 10-fold cross-validation (repeated 100 times), and decision curve analysis. Results: Among the 312 AMI patients, 94 (30.13%) developed heart failure during hospitalization. Multivariate logistic regression identified advanced age (OR = 2.158, P = 0.004), diabetes (OR = 1.964, P = 0.002), higher Gensini score (OR = 2.869, P = 0.001), left ventricular ejection fraction (LVEF) < 50% (OR = 2.581, P = 0.007), and elevated N-terminal pro B-type natriuretic peptide (NT-proBNP) levels (OR = 3.593, P < 0.001) as risk factors for heart failure in AMI patients. The constructed nomogram model demonstrated an area under the *ROC* curve (AUC) of 0.882, indicating good discriminative ability. The model demonstrated high stability through 100 repetitions of 10-fold cross-validation. Decision curve analysis confirmed its clinical utility. Conclusion: In-hospital heart failure in AMI patients is associated with older age, diabetes, elevated Gensini score, reduced LVEF, and increased NT-proBNP levels. The developed nomogram model effectively predicts the risk of heart failure in this population and may assist in early clinical risk stratification.

Keywords: Acute myocardial infarction, heart failure, risk factors, nomogram, prediction model

Introduction

Acute myocardial infarction (AMI) is characterized by coronary artery occlusion leading to acute myocardial necrosis due to transient ischemia and hypoxia. It presents with sudden onset, rapid progression, and high mortality, and may result in severe cardiovascular complications if not promptly managed [1]. Heart failure is one of the most common complications following AMI, with an incidence of up to 32% [2]. It can further impair coronary perfusion, exacerbate cardiac dysfunction, and negatively impact patient prognosis [3]. Emerging evidence reveals that post-AMI heart failure stems from multiple mechanisms, including pathological myocardial remodeling, neuroendocrine dysregulation and inflammatory cytokine activation [4]. Risk stratification studies have highlighted the importance of infarct characteristics (e.g., size and timing), pre-existing myocardial vulnerability, and mechanical complications in heart failure progression [5]. Early identification of high-risk individuals and implementation of precision interventions may substantially reduce the incidence of post-AMI heart failure. Therefore, this study retrospectively analyzed data from AMI patients to identify risk factors for in-hospital heart failure and to develop a predictive model to support clinical risk evaluation and personalized management strategies.

Material and methods

Study subjects

This retrospective study included 312 AMI patients admitted to Qingdao Municipal Hospital

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Table 1. Genini scoring criteria for coronary arteries

Degree of stenosis	Score (points)	Lesion location	Rating (points)
Complete occlusion	32	Small branches	0.5
91%-99%	16	Distal left anterior descending artery	1.0
76%-90%	8	Right coronary artery	1.0
51%-75%	4	Middle or distal left circumflex artery	1.0
26%-50%	2	Mid-segment of left anterior descending artery	1.5
1%-25%	1	Proximal left anterior descending or circumflex	2.5
-		Left main coronary artery	5

from January 2021 to December 2024. Patients were categorized into a heart failure group and a non-heart failure group based on whether heart failure occurred during hospitalization.

Inclusion criteria: (1) Diagnosis of AMI in accordance with the *Guidelines for the Diagnosis and Treatment of Acute Myocardial Infarction with Integrated Traditional Chinese and Western Medicine* [6], including evidence of arterial stenosis and infarction on coronary angiography, electrocardiographic findings of significant ST segment elevation or pathological Q waves, acute chest pain symptoms within 24 hours, and regional wall motion abnormalities of the interventricular septum on echocardiography; (2) Hospital admission within 24 hours of symptom onset; (3) Age ≥ 18 years; (4) Complete clinical data and follow-up records. Exclusion criteria: (1) History of old myocardial infarction; (2) Pre-existing heart failure; (3) Valvular or structural cardiac abnormalities; (4) Congenital heart disease; (5) Severe mental dysfunction; (6) Malignancies; (7) Severe hepatic, renal, or pulmonary dysfunction.

This study was approved by the Medical Ethics Committee of Qingdao Municipal Hospital.

Methods

Data collection: Clinical data were obtained by reviewing the patients' electronic medical records, echocardiography reports, and laboratory test results upon admission. Collected variables included age, body mass index (BMI), sex, smoking and alcohol consumption history, presence of diabetes or hypertension, heart rate, number of diseased branches, treatment modality, left ventricular ejection fraction (LVEF), high-sensitivity C-reactive protein (hs-CRP), triacylglycerol (TG), cardiac troponin I (cTnI), and N-terminal pro B-type natriuretic peptide (NT-proBNP) levels.

Assessment of coronary artery disease severity: Coronary angiography reports of AMI patients upon admission were reviewed to evaluate the severity of coronary artery disease using the Gensini scoring system [7]. Each lesion was scored by multiplying a stenosis severity coefficient by a weighting factor based on the anatomical location. The total Gensini score was calculated by summing the scores of all lesions, with higher scores indicating more severe coronary artery disease. The specific scoring criteria are detailed in **Table 1**.

Diagnosis of heart failure: Heart failure was diagnosed based on the criteria outlined in the *2014 Chinese Guidelines for the Diagnosis and Treatment of Heart Failure* [8]. The major criteria included: widened pulse pressure (> 1.57 kPa), positive hepatojugular venous reflux, circulation time > 25 seconds, third heart sound (S3) gallop, acute pulmonary edema, jugular venous distension, cardiomegaly, pulmonary rales, and paroxysmal nocturnal dyspnea or orthopnea. The minor criteria included: heart rate ≥ 120 beats/min, weight loss > 4.5 kg after 5 days of treatment, exertional dyspnea, nocturnal cough, and ankle edema. A diagnosis of heart failure was established when either two major criteria, or one major plus two minor criteria, were met.

Statistical analysis

Statistical analyses were performed using SPSS version 27.0. Continuous variables were expressed as mean ± standard deviation (Mean ± SD) *and compared between* groups using the t-test. Categorical variables were expressed as frequencies and percentage [n (%)] and compared between two groups using X^2 test. Multivariate logistic regression was used to identify independent risk factors for in-hospital heart failure among AMI patients. A *P* value < 0.05 was considered statistically significant. A nomogram prediction model was constructed

Table 2. Comparison of clinical data between the two groups of patients

Clinical data	Heart failure group (n = 94)	No-heart failure group (n = 218)	t/X^2	P value
Age (years)	69.64 ± 8.52	61.81 ± 7.95	6.812	< 0.001
Body Mass Index (kg/m^2)	24.78 ± 3.93	24.42 ± 3.67	0.778	0.437
Sex			0.408	0.523
Males	59 (62.77)	145 (66.51)		
Female	35 (37.23)	73 (33.49)		
Smoking			1.975	0.160
Yes	31 (32.98)	55 (25.23)		
No	63 (67.02)	163 (74.77)		
Alcohol consumption			2.678	0.102
Yes	36 (38.30)	63 (28.90)		
No	58 (61.70)	155 (71.10)		
Combined diabetes			10.186	0.001
Yes	34 (36.17)	42 (19.27)		
No	60 (63.83)	176 (80.73)		
Combined hypertension			2.546	0.111
Yes	48 (51.06)	90 (41.28)		
No	46 (48.94)	128 (58.72)		
Heart rate (beats/min)	110.15 ± 17.93	107.92 ± 14.56	2.709	0.007
Gensini score (points)	76.73 ± 21.90	56.54 ± 15.81	9.163	< 0.001
Number of stenosed coronary vessel			1.820	0.403
Single vessel	39 (41.49)	83 (38.08)		
Double branch	45 (47.87)	99 (45.41)		
Triple vessel	10 (10.64)	36 (16.51)		
Treatment methods			1.932	0.165
PCI	45 (47.87)	123 (56.42)		
Intravenous thrombolysis	49 (52.13)	95 (43.58)		
LVEF			8.601	0.003
< 50%	54 (57.45)	86 (39.45)		
≥ 50%	40 (42.55)	132 (60.55)		
hs-CRP (mg/L)	4.13 ± 1.07	3.95 ± 1.02	1.409	0.159
TG (mmol/L)	1.42 ± 0.36	1.39 ± 0.34	0.702	0.483
cTnI (ng/mL)	5.58 ± 1.65	5.83 ± 1.86	1.126	0.261
NT-proBNP (pg/mL)	687.89 ± 180.72	440.15 ± 162.59	11.930	< 0.001

Note: PCI: Percutaneous coronary intervention; LVEF: Left ventricular ejection fraction; hs-CRP: High sensitivity C-reactive protein; TG: Triacylglycerol; cTnI: cardiac troponin I; NT-proBNP: N-terminal pro B-type natriuretic peptide.

using R 3.4.3 software. Receiver operating characteristic (*ROC*) curve analysis, repeated K-fold cross-validation, and decision curve were used to evaluate the model's predictive performance.

Results

Incidence and comparative analysis of in-hospital heart failure in AMI patients

Among the 312 patients included, 94 (30.13%) developed heart failure during hospitalization (Heart failure group), and the remaining 218 did not (No-heart failure group). Statistically significant differences were observed between the two groups in terms of age, prevalence of diabetes, heart rate, Gensini score, LVEF and NT-proBNP levels (all $P < 0.05$) (**Table 2**).

Multivariate logistic regression analysis of in-hospital heart failure

The occurrence of in-hospital heart failure was set as the dependent variable. Variables with

Table 3. Variable assignment

Variable		Assignment
Dependent variable	Heart failure occurs	No = 0; Yes = 1
Independent variable	Age	Enter the actual value
	Combined diabetes	No = 0; Yes = 1
	Heart rate	Enter the actual value
	Gensini score	Enter the actual value
	LVEF	≥ 50% = 0; < 50% = 1
	NT-proBNP	Enter the actual value

Note: LVEF: Left ventricular ejection fraction; NT-proBNP: N-terminal pro B-type natriuretic peptide.

Table 4. Multivariate logistic regression analysis for in-hospital heart failure in AMI patients

Variable	B	*SE*	Wald χ^2	*P* value	*OR* (95% *CI*)
Age	0.769	0.267	8.295	0.004	2.158 (1.279-3.640)
Combined diabetes	0.675	0.218	9.587	0.002	1.964 (1.281-3.011)
Heart rate	0.692	0.476	2.113	0.146	1.997 (0.786-5.078)
Gensini score	1.054	0.320	10.848	0.001	2.869 (1.533-5.371)
LVEF < 50%	0.948	0.352	7.253	0.007	2.581 (1.296-5.139)
NT-proBNP	1.279	0.354	13.054	< 0.001	3.593 (1.795-7.192)
Constant	6.743	1.527	19.499	< 0.001	-

Note: LVEF: Left ventricular ejection fraction; NT-proBNP: N-terminal pro B-type natriuretic peptide; AMI: Acute myocardial infarction.

statistical significance in the univariate analysis (age, diabetes prevalence, heart rate, Gensini score, LVEF and NT-proBNP) were included as independent variables in the logistic regression model. Variable coding criteria are provided in **Table 3**. Multivariate logistic regression analysis identified advanced age, diabetes, elevated Gensini score, LVEF < 50%, and high NT-proBNP levels as independent risk factors for in-hospital heart failure in AMI patients (all $P < 0.05$, **Table 4**).

Construction of a predictive model for in-hospital heart failure in AMI patients

The identified risk factors were incorporated into a nomogram prediction model using R software. The nomogram assigns a point value for each predictor based on its contribution to the overall risk. By aligning patient characteristics with the corresponding scales, individual scores can be determined. The total risk score is calculated by summing the points for all predictors, and the corresponding probability of heart failure in AMI patients is determined by projecting the total score vertically to the risk axis. For example, a 60-year-old AMI patient with diabetes mellitus, a Gensini score of 92, LVEF < 50%, NT-proBNP level of 690 pg/mL would have a total score of 37.5 + 19.5 + 80 + 21 + 65 = 223, corresponding to a predicted heart failure risk of 62% (**Figure 1**).

Model performance evaluation

The nomogram achieved an area under the *ROC* curve (AUC) of 0.882 (95% CI: 0.834-0.932) for predicting in-hospital heart failure risk in AMI patients, indicating moderate to high predictive performance (**Figure 2**). Furthermore, repeated 100 10-fold cross-validation confirmed the model's robustness and stability, with minimal variation across all original dataset metrics (**Table 5**). Decision curve analysis showed that the model provided a higher net clinical benefit than the "treat-all" or "treat-none" strategies across a wide range of threshold probabilities, supporting its clinical utility for clinical decision-making (**Figure 3**).

Discussion

Heart failure following AMI is a major contributor to in-hospital mortality [9, 10]. Therefore, identifying factors associated with heart failure after AMI is essential for early risk assessment and timely intervention.

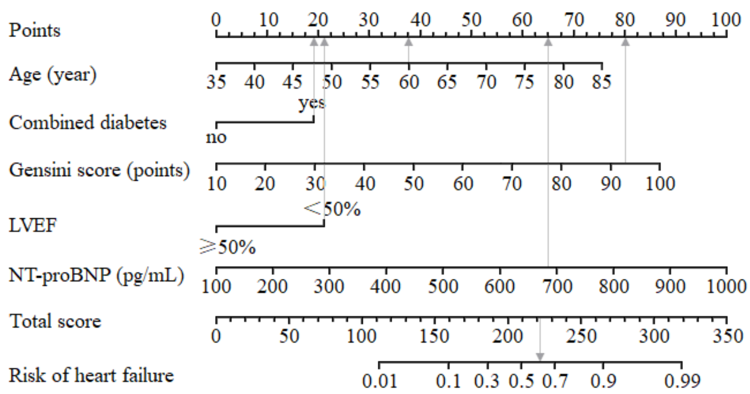

Figure 1. Nomogram prediction model for in-hospital heart failure risk in patients with AMI. Note: LVEF: Left ventricular ejection fraction; NT-proBNP: N-terminal pro B-type natriuretic peptide; AMI: Acute myocardial infarction.

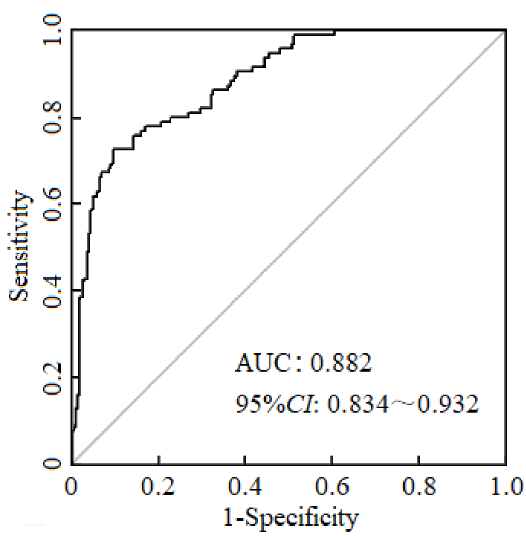

Figure 2. *ROC* curve analysis of nomogram prediction model. Note: *ROC*: Receiver Operating Characteristic.

AMI can lead to myocardial cell necrosis and apoptosis, leading to impaired myocardial contractility and ventricular remodeling, which are key mechanisms in the development of heart failure. Previous studies have demonstrated that advanced age is a significant risk factor for heart failure following AMI [11], consistent with our findings. Aging is associated with reduced cardiac reserve and diminished compensatory capacity, which decreases myocardial compliance and predisposes patients to heart failure. Additionally, elderly AMI patients often have underlying diseases such as hypertension, diabetes, further raising the risk [12].

Chronic dysregulation of glucose metabolism contributes to coronary atherosclerosis and vascular stenosis, which in turn lead to myocardial ischemia and eventual heart failure. Berezina et al. [13] also reported a higher susceptibility to heart failure in diabetic patients, which is supported by others [14]. In this study, diabetes mellitus emerged as an independent risk factor for in-hospital heart failure in AMI patients, consistent with previous reports [15]. This may be attributed to long-term glucose metabolic dysfunction in diabetic patients, which reduces myocardial contractility and cardiac output, ultimately resulting in impaired tissue perfusion. Consequently, this results in circulatory dysfunction primarily characterized by impaired myocardial relaxation and/or contraction, ultimately progressing to heart failure.

The Gensini score provides a quantitative assessment of the severity of coronary artery lesions. Sinning et al. [16] demonstrated that the Gensini score, when combined with clinical indicators, could predict cardiovascular prognosis in patients with coronary artery disease over an 8-year period. Similarly, Qin et al. [17] showed that the Gensini score could predict short-term prognosis in patients with acute ST-segment elevation myocardial infarction (STEMI). The research by Yokokawa et al. [18] indicated that the Gensini score was associated with heart failure in patients with coronary artery lesions, aligning with our findings that the Gensini score serves as a risk factor for heart failure in AMI patients. This correlation might be attributed to the fact that patients with severe coronary lesions often have poor collateral circulation, resulting in extensive myocardial ischemia and compromised cardiac function, thereby increasing the risk of heart failure. In the early phase of AMI, myocardial cell necrosis and apoptosis can lead to weakened contractile coordination, as evidenced by a significant decrease in LVEF observed on echocardiography. Park et al. [19] reported that decreased LVEF levels were associated with an increased risk of heart failure, a finding corrob-

Table 5. Cross-validation for model performance evaluation

Change	AUC/C-index	R^2	D	*U* value	Brier score	E_{max}	E_{avg}
Modeling data values	0.882	0.705	0.687	-0.008	0.136	0.057	0.015
Corrected value	0.857	0.654	0.572	0.027	0.142	0.207	0.086
Change (Δ)	-0.025	-0.051	-0.115	0.035	0.006	0.150	0.071

Note: AUC/C-index: Ratio of area under curve to consistency index; D: Discriminant index; E_{max}: Maximum calibration deviation; E_{avg}: Average calibration deviation.

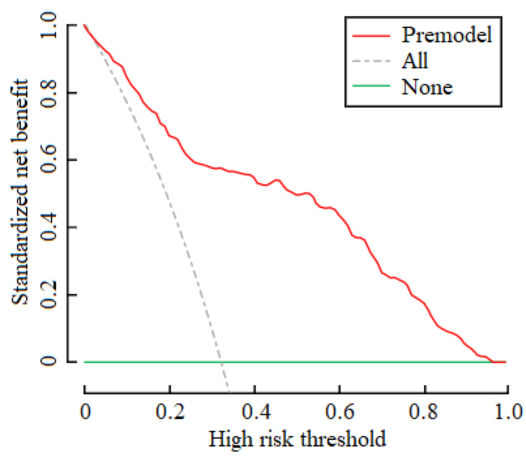

Figure 3. Decision curve analysis of nomogram prediction model.

orated by our results. This phenomenon might be explained by the fact that a lower LVEF indicates diminished cardiac systolic function and inadequate systemic blood perfusion, which promotes ventricular remodeling and accelerates cardiac deterioration, ultimately contributing to heart failure progression [20].

NT-proBNP is a biologically inactive protein with relatively stable biological mechanisms [21]. Studies have found a significant correlation between NT-proBNP and LVEF [22], suggesting that NT-proBNP is a reliable indicator of cardiac function. Kristensen et al. [23] demonstrated that dynamic changes in NT-proBNP concentrations could predict the risk of developing heart failure. Similarly, Lam et al. [24] further validated the diagnostic value of NT-proBNP as a biomarker for heart failure, supporting the findings of this study. The underlying mechanism may be explained by the pathophysiological changes induced by myocardial ischemia and hypoxia in AMI, which can lead to coronary spasm, vascular remodeling, increased blood viscosity, and elevated aldosterone levels. These alterations contribute to increased atrial wall tension and myocardial stress, ultimately impairing cardiac function and elevating the risk of heart failure.

Heart failure following AMI increases the risk of death [25]. Establishing simple and intuitive risk prediction models to assess the risk of heart failure in AMI patients enhances doctor-patient communication, facilitates early preventive strategies, and ultimately improves patient prognosis. In this study, the identified independent risk factors were incorporated into a nomogram prediction model using R software. The nomogram clearly visualizes the relative contribution of each factor, enabling quantification of individual risk and supporting personalized clinical decision-making. *ROC* curve analysis showed that the model demonstrated strong discriminative ability, with an AUC of 0.882. The cross-validation method showed good stability (generalization ability) of the model. Decision curve analysis further confirmed the model's clinical utility by showing a favorable net benefit across a range of threshold probabilities. The indicators used in this model are common clinical indicators, which facilitates the application and potential promotion of the model in other hospitals.

This study does have limitations: ① Retrospective analysis of patient data from a single center may limit the generalizability of the findings. ② Although logistic regression was used to identify predictors and construct the nomogram, the relatively small number of included variables may limit the model's ability to capture complex interactions in high-dimensional data, potentially affecting its predictive accuracy. Therefore, future research should prioritize prospective, multicenter designs and consider additional variables to enhance the robustness and comprehensiveness of the risk prediction model for heart failure in AMI patients.

In summary, age, diabetes, Gensini score, LVEF, and NT-proBNP are independent risk factors for in-hospital heart failure in AMI patients. The nomogram model constructed based on these variables demonstrated good predictive performance in assessing the risk of heart failure in patients with AMI.

Disclosure of conflict of interest

None.

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