



OPEN Analysis of risk factors and construction of nomogram model for nosocomial infection in patients with acute myocardial infarction after percutaneous coronary intervention

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To analyze the risk factors for hospital-acquired infections following percutaneous coronary intervention (PCI) in patients with acute myocardial infarction (AMI) and to develop a nomogram prediction model. Clinical data from 324 AMI patients who underwent PCI between July 2021 and June 2023 were retrospectively analyzed. Patients were categorized into an infection group ($n = 39$) and a non-infection group ($n = 285$) based on the occurrence of nosocomial infection postoperatively. Optimal cutoff values were determined using receiver operating characteristic (ROC) curve analysis. Independent risk factors for nosocomial infection after PCI were identified through multivariate logistic regression, and a nomogram model was constructed accordingly. The model underwent internal validation via calibration curves, and its predictive performance was assessed using decision curve analysis. No significant differences were observed between the two groups in terms of gender, drinking history, smoking history, hypertension, infarct location, or number of stents implanted (all $P > 0.05$). However, the infection group had significantly higher age, higher prevalence of diabetes, greater proportion of New York Heart Association (NYHA) class III/IV, more frequent invasive procedures, and longer hospital stays (all $P < 0.05$). ROC analysis identified optimal cutoff values of 60 years for age and 6 days for hospitalization time. Multivariate logistic regression confirmed that age > 60 years, diabetes, NYHA class III/IV, invasive procedures, and hospital stay > 6 days were independent risk factors for nosocomial infection after PCI. The nomogram model demonstrated excellent discrimination, with a C-index of 0.915 (95% CI 0.877–0.953). The calibration curve indicated good agreement between predicted and observed outcomes. The nomogram provided higher net clinical benefit beyond threshold probabilities of 0.24 compared to individual predictors. A nomogram incorporating age, diabetes, cardiac function classification, invasive procedures, and hospitalization time was developed to predict the risk of nosocomial infection in AMI patients after PCI. The model exhibits strong predictive performance and may assist clinicians in identifying high-risk patients for intensified monitoring and preventive strategies. However, as a prognostic tool, it does not directly mitigate infection risk and requires external validation before routine clinical implementation.

Keywords Acute myocardial infarction, Percutaneous coronary intervention, Nosocomial infection, Risk factors, Nomogram model

In recent years, the increasing prevalence of cardiovascular diseases has been attributed to societal development, accelerating population aging, and changes in dietary patterns and lifestyles¹. Acute myocardial infarction (AMI),

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the most prevalent and severe form of cardiovascular disease, is characterized pathologically by myocardial necrosis resulting from coronary artery occlusion, leading to acute and sustained ischemia and hypoxia of cardiomyocytes. AMI progresses rapidly and may further induce arrhythmias, heart failure, or cardiogenic shock, severely threatening patients' lives^{2,3}. In China, AMI accounts for approximately five-sevenths of annual cardiovascular disease-related deaths, making it a leading cause of mortality and disability⁴. Percutaneous coronary intervention (PCI) is widely used in clinical practice to restore coronary blood supply and improve myocardial perfusion in AMI patients, thereby preventing recurrent ischemic events⁵.

The abrupt necrosis of myocardial cells in AMI causes a sharp decline in cardiac output and function, resulting in systemic hypoperfusion of tissues and organs. This compromises the body's immune defenses and increases susceptibility to nosocomial infections^{6,7}. Furthermore, AMI patients requiring PCI are hospitalized for treatment, and prolonged stays in healthcare settings—coupled with postoperative immunosuppression—heighten the risk of hospital-acquired infections. Such infections can not impede recovery, prolong hospitalization, increase medical costs and family burden, but also potentially lead to fatal complications⁸. Therefore, early identification of high-risk AMI patients prone to nosocomial infection post-PCI and implementation of preventive strategies based on relevant risk factors are essential to reduce infection incidence.

Currently, the risk factors for hospital-acquired infections following PCI in AMI patients have not been fully elucidated, hampering the development of targeted preventive measures. Thus, the analysis and prediction of nosocomial infections are of great clinical importance. However, there remains a lack of simple and effective methods for translating risk factors into practical clinical use. The nomogram prediction model integrates multiple variables to generate individualized probabilities for specific outcomes, allowing more accurate and intuitive prognostic assessment^{9,10}. This study aims to develop a nomogram model for predicting nosocomial infection in AMI patients after PCI by analyzing associated risk factors, thereby providing a basis for clinical infection prevention.

Materials and methods

Study population and design

A total of 324 patients diagnosed with AMI who underwent PCI at our hospital between July 2021 and June 2023 were retrospectively enrolled. Based on the occurrence of nosocomial infection following the procedure, patients were classified into two groups: an infection group (n = 39) and a non-infection group (n = 285).

Inclusion criteria

Patients were eligible for enrollment if they met all of the following conditions: (1) Confirmed diagnosis of AMI: Diagnosis established according to the universal definition of AMI, based on clinical presentation, elevated cardiac biomarkers (e.g., troponin), and electrocardiographic or imaging evidence. (2) Indication for PCI: Patients in whom percutaneous coronary intervention was clinically indicated and performed as part of their treatment strategy. (3) Age ≥ 18 years: Only adult patients were included to ensure applicability of clinical and ethical guidelines. (4) Availability of complete clinical records: Demographic information, laboratory findings, procedural details, and follow-up infection outcomes had to be complete and accessible for analysis.

Exclusion criteria

Patients were excluded if they met any of the following conditions: (1) Severe comorbid organ dysfunction: Advanced hepatic or renal insufficiency that could independently affect prognosis or confound the analysis of infection risk. (2) History of prior cardiac surgery: Such as coronary artery bypass grafting or valve replacement, because prior surgery could alter cardiac function and infection risk. (3) Chronic infectious disease: Patients with tuberculosis, chronic viral hepatitis, or HIV infection were excluded due to baseline altered immunity. (4) Immunologic or hematologic disorders: Autoimmune diseases, immunodeficiency syndromes, or blood dyscrasias that could bias infection susceptibility. (5) Malignancy: Any known active cancer, as both the disease and its treatment may alter immune response and infection risk. (6) Psychiatric or neurological conditions impairing cooperation: Patients unable to consent or comply with treatment and monitoring (e.g., severe dementia, psychosis, or advanced neurological disability). This study was approved by the Medical Ethics Committee of Jilin City Hospital of Chemical Industry (approval No. IEC-2020-010-01).

Data collection

Patient demographic and clinical data were collected, including: age, gender, smoking history, alcohol use history, presence of diabetes, hypertension, cardiac function classification, infarct location, duration of hospitalization, number of stents implanted, and whether any invasive procedures in addition to the interventional treatment were performed.

Invasive procedures were defined as any diagnostic or therapeutic intervention involving penetration of a body cavity or disruption of natural anatomical barriers, excluding the primary PCI procedure. These included: endotracheal intubation and mechanical ventilation; central venous catheterization; indwelling urinary catheterization (Foley catheter); nasogastric or enteral feeding tube placement; arterial line insertion; and other invasive bedside procedures required for intensive monitoring or supportive care.

Definition of nosocomial infection

The diagnostic criteria for infection were as follows:

- (1) Body temperature ≥ 38.0 °C, or sustained temperature ≥ 37.7 °C for at least 1 h;
- (2) Collection of secretion samples within 12 h of fever onset, with confirmed pathogenic infection by microbiological testing.

Nosocomial infections were identified based on the following criteria:

- (1) For infections without a clear incubation period, those manifesting more than 48 h after admission were considered hospital-acquired;
- (2) For infections with a defined incubation period, those occurring beyond the average incubation period after admission were classified as nosocomial.

Nosocomial infection was determined using a combination of clinical and microbiological criteria. Fever was defined as body temperature $\geq 38.0^{\circ}\text{C}$, or $\geq 37.7^{\circ}\text{C}$ sustained for ≥ 1 h, with microbiological confirmation of pathogen infection from relevant secretions. In addition, we referred to the widely accepted definitions from the U.S. Centers for Disease Control and Prevention (CDC) and National Healthcare Safety Network (NHSN), which define nosocomial infection as an infection not present or incubating at the time of hospital admission, and occurring ≥ 48 h after admission¹¹.

In this study, all patients who developed clinical signs suggestive of infection (e.g., fever, leukocytosis, localized symptoms) underwent microbiological testing. Samples (such as sputum, urine, blood, or catheter-related specimens) were collected within 12 h of onset of fever or other suspected infection and processed for pathogen culture. Routine microbiological screening was not performed for all patients without symptoms; testing was targeted to those with suspected infection. Therefore, the diagnosis of nosocomial infection required both compatible clinical features and microbiological confirmation in symptomatic cases.

Statistical analysis

Statistical analyses were performed using SPSS version 22.0. Continuous variables are presented as mean \pm standard deviation, while categorical or ordinal data are summarized as frequencies and percentages. For normally distributed continuous variables, comparisons between groups were made using the Student's *t*-test. Receiver operating characteristic (ROC) curve analysis was employed to determine optimal cutoff values for relevant factors. Independent risk factors were identified using multivariate logistic regression analysis. A two-sided *P*-value < 0.05 was considered statistically significant.

Internal validation of the nomogram model was performed using bootstrap resampling (1000 iterations). The concordance index (C-index) was calculated to assess discrimination, and calibration was evaluated with a calibration plot comparing predicted versus observed probabilities. Decision curve analysis was also used to estimate the net clinical benefit.

Results

Comparison of clinical characteristics between the two groups

As shown in Table 1, baseline clinical characteristics were compared between the infection group ($n = 39$) and the non-infection group ($n = 285$). Patients who developed nosocomial infection were significantly older, had a higher prevalence of diabetes, exhibited higher NYHA functional class (III/IV), underwent more invasive procedures, and experienced longer hospital stays (all $P < 0.05$). In contrast, no significant differences were observed between the groups in terms of gender, smoking history, alcohol consumption, hypertension, infarct location, or number of stents implanted (all $P > 0.05$).

ROC curve analysis of predictive variables

ROC curve analysis (Table 2; Fig. 1) identified optimal cutoff values of 60 years for age and 6 days for length of hospital stay. Age greater than 60 years yielded an area under the curve (AUC) of 0.798, with a sensitivity of 74.36% and specificity of 77.89%. Hospital stay longer than 6 days resulted in an AUC of 0.759, with sensitivity of 76.92% and specificity of 75.79%. Both variables demonstrated moderate discriminative power, with cutoff values determined based on the Youden index.

Multivariate logistic regression analysis of nosocomial infection after PCI

Multivariate logistic regression analysis (Table 3) revealed that age > 60 years (OR = 1.084, 95% CI 1.033–1.212), diabetes (OR = 1.307, 95% CI 1.129–1.732), NYHA class III/IV (OR = 1.337, 95% CI 1.137–1.831), invasive procedures (OR = 1.318, 95% CI 1.126–1.803), and hospital stay > 6 days (OR = 1.081, 95% CI 1.032–1.206) were independent risk factors for nosocomial infection in AMI patients following PCI.

Development of a nomogram prediction model for hospital-acquired infection

A nomogram prediction model was constructed incorporating the following predictors: age, diabetes, cardiac function classification (NYHA class), invasive procedures, and duration of hospitalization. The nomogram is presented in Fig. 2.

Nomogram model calibration curve and clinical net benefit analysis

The nomogram model for predicting hospital infection after PCI in AMI patients showed good discrimination, with a C-index of 0.915 (95% CI: 0.877–0.953) after bootstrap validation. The calibration curve was close to the ideal 45-degree line, indicating good agreement between predicted and observed probabilities (Fig. 3). Decision curve analysis (DCA) demonstrated that the nomogram provided higher net benefit across a range of threshold probabilities compared with individual predictors such as age, diabetes, cardiac function classification, invasive procedures, and length of stay (Fig. 4). However, no additional reclassification statistics such as Net Reclassification Improvement (NRI) or Integrated Discrimination Improvement (IDI) were calculated, and therefore the incremental predictive value of the nomogram beyond individual predictors should be interpreted with caution.

Observation indicators	Infection group (n = 39)	Non-infectious group(n = 285)	t/ χ^2	P
Age (years)	61.79 ± 5.08	56.56 ± 4.56	6.619	< 0.001
Genders			0.058	0.810
Male	20 (51.28)	152 (53.33)		
Female	19 (48.72)	133 (46.67)		
Smoking history			1.924	0.165
Yes	21 (53.85)	120 (42.11)		
No	18 (46.15)	165 (57.89)		
Drinking history			1.998	0.157
Yes	24 (61.54)	141 (49.47)		
No	15 (38.46)	144 (50.53)		
Diabetes			12.381	< 0.001
Yes	23 (58.97)	87 (30.53)		
No	16 (41.03)	198 (69.47)		
Hypertensive			0.357	0.550
Yes	28 (71.79)	191 (67.02)		
No	11 (28.21)	94 (32.98)		
Infarct site			1.792	0.617
Anterior wall	12 (30.77)	99 (34.74)		
Anterior interstitial wall	16 (41.03)	87 (30.52)		
Inferior wall	6 (15.38)	56 (19.65)		
lateral wall	5 (12.82)	43 (15.09)		
Cardiac function classification			5.618	0.018
Grade I/II	20 (51.28)	200 (70.18)		
Grade III/VI_	19 (48.72)	85 (29.82)		
Intrusive operations			6.877	0.009
Yes	18 (46.15)	74 (25.96)		
No	21 (53.85)	211 (74.04)		
Number of stent implants (pcs)	2.10 ± 0.75	2.08 ± 0.81	0.184	0.854
Length of hospitalization (days)	7.36 ± 1.72	5.60 ± 1.57	6.478	< 0.001

Table 1. Comparison of the clinical data of the two groups of patients.

Variant	AUC	Optimal cutoff value	Youden index	SE	95% CI	Sensitivity (%)	Specificity (%)
Age	0.798	> 60 years old	0.523	0.041	0.750–0.841	74.36	77.89
Length of hospitalization	0.759	> 6 days	0.527	0.044	0.709–0.805	76.92	75.79

Table 2. Analysis of receiver operating characteristic (ROC) curve for relevant variables.

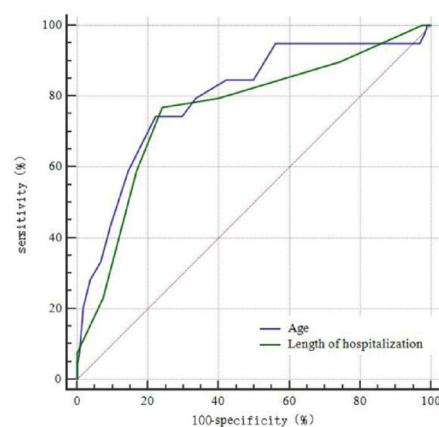


Fig. 1. Receiver operating characteristic (ROC) curves for the variables of interest. *Note:* Fig. 1 displays the flowchart of patient selection and grouping process.

Variant	β	SE	Wald	Odds ratio (OR) (95% confidence interval)	P
Age (> 60 years old vs. \leq 60 years old)	0.081	0.029	7.801	1.084 (1.033–1.212)	< 0.001
Diabetes (yes vs. no)	0.268	0.101	7.041	1.307 (1.129–1.732)	0.008
Cardiac function classification (Grade III/VI vs. Grade I/II)	0.290	0.114	6.471	1.337 (1.137–1.831)	0.018
Intrusive operations (yes vs. no)	0.276	0.105	6.909	1.318 (1.126–1.803)	0.015
Length of hospitalization (> 6 days vs. \leq 6 days)	0.078	0.028	7.760	1.081 (1.032–1.206)	< 0.001

Table 3. Logistic multiple regression analysis of hospital-acquired infections in acute myocardial infarction patients undergoing percutaneous coronary intervention.

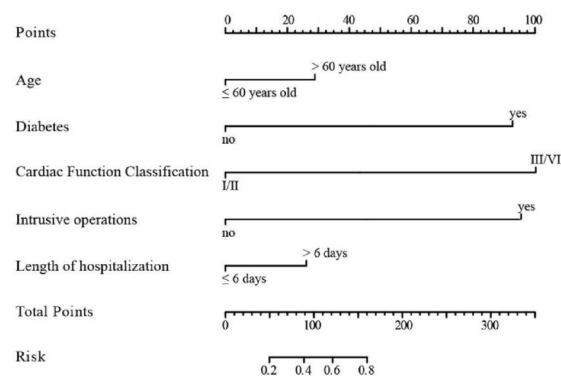


Fig. 2. A nomogram model for predicting hospital-acquired infections after percutaneous coronary intervention (PCI) in patients with acute myocardial infarction (AMI). *Note:* Fig. 2 illustrates the constructed nomogram for predicting nosocomial infection risk in AMI patients after PCI, incorporating five independent predictors (age, diabetes, New York Heart Association (NYHA) functional class, invasive procedures, and length of hospital stay).

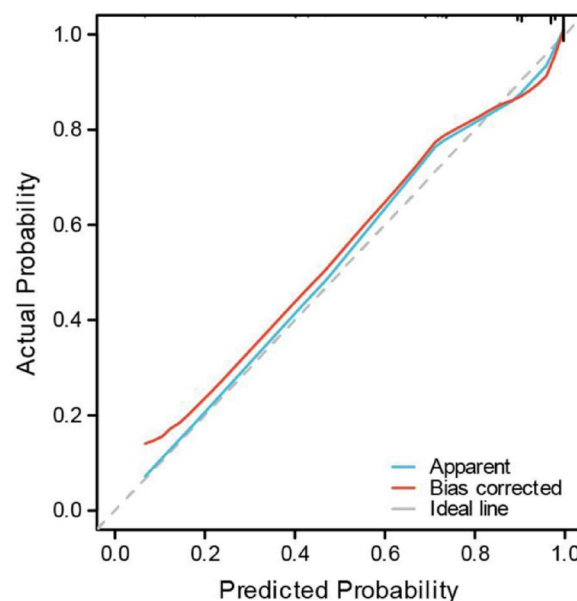


Fig. 3. Calibration curves for the nomogram model. *Note:* Fig. 3 shows the calibration plot of the nomogram, demonstrating good agreement between predicted and observed infection probabilities.

Discussion

AMI is a critical medical condition characterized by an unstable clinical course, rapid progression, and a high incidence of complications, all contributing to substantial mortality and a serious threat to patient safety. PCI plays a vital role in reestablishing coronary blood flow and improving cardiac function in AMI patients^{12,13}.

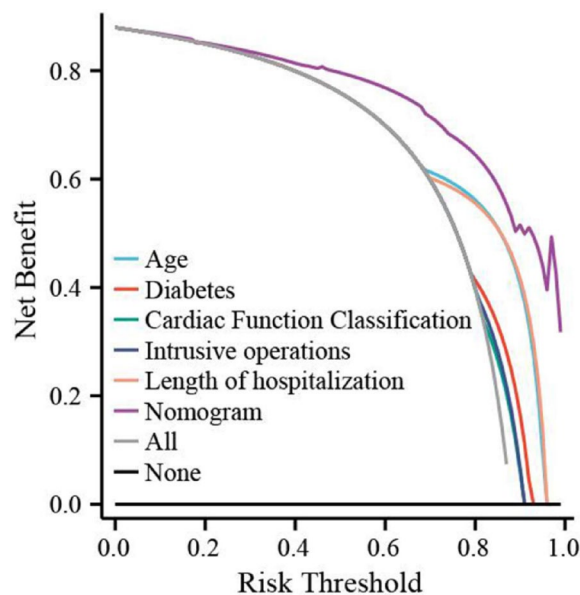


Fig. 4. Decision curve of the nomogram model. *Note:* Fig. 4 presents the decision curve analysis (DCA) comparing net clinical benefit of the nomogram versus individual predictors across a range of threshold probabilities. The nomogram consistently provided greater net benefit.

It has become a cornerstone in the management of acute coronary syndromes and is widely employed during acute episodes of coronary heart disease. Although continuous advancements have been made in PCI techniques and devices, it remains an invasive procedure and is consequently associated with a inherent risk of infection. The occurrence of infection can not only compromise the immediate therapeutic outcome but may also lead to severe consequences, including mortality. With the evolution of modern healthcare, medical disputes pertaining to nosocomial infections have become increasingly prominent, undermining trust between patients, their families, and healthcare providers, and disrupting normal clinical operations¹⁴. Hence, the prevention, control, and management of hospital-acquired infections have emerged as major priorities in clinical practice. While nomogram models are well-established for predicting survival and prognosis in oncology^{15,16}, their application in other medical domains remains limited. To our knowledge, no specific nomogram exists for predicting the risk of hospital-acquired infections following PCI in AMI patients. This study aimed to identify independent risk factors for nosocomial infection in this population and to integrate them into a nomogram model for individualized risk prediction. The resulting tool may facilitate early identification of high-risk patients and enable targeted interventions to mitigate infection incidence.

The results of this study identified age > 60 years, diabetes, NYHA class III/IV, hospital stay > 6 days, and invasive procedures as independent risk factors for hospital-acquired infections in AMI patients undergoing PCI. Advanced age may contribute to increased susceptibility to nosocomial infections, likely attributable to immunosenescence and a higher prevalence of comorbidities, which collectively lead to progressive organ dysfunction and impaired infection control¹⁷. In this study, the optimal age cut-off of 60 years was determined using the Youden index derived from ROC analysis. This threshold is consistent with commonly applied definitions of elderly status in cardiovascular risk stratification, where patients over 60 years are recognized to exhibit reduced immune competence, greater comorbidity burden, and elevated risk of infectious complications. Similarly, a hospital stay exceeding 6 days represents a clinically significant duration associated with a marked increase in cross-infection risk. Although the Youden index offers a statistically optimal balance between sensitivity and specificity, the clinical relevance of these cut-offs is further supported by their alignment with well-established risk patterns observed in AMI and critical care cohorts^{18,19}. These findings indicate that the selected thresholds are not only statistically sound but also clinically meaningful, enhancing their utility in routine clinical practice.

Age is a central variable in several bedside risk scores and remains a powerful marker of adverse outcomes in the critically ill. The Intermountain Risk Score (IMRS)—which combines routine laboratory values with age and sex—has demonstrated prognostic utility in cardiogenic shock: in STEMI patients complicated by shock, IMRS independently predicted both short- and long-term mortality and outperformed the Synergy-STEMI Score by ROC analysis²⁰. In acute coronary syndrome patients with cardiogenic shock supported by intra-aortic balloon pump in the intensive cardiac care unit, advancing age was significantly associated with in-hospital mortality on univariable analysis (odds ratio per year increase 1.079, $p < 0.001$), underscoring the contribution of age to risk stratification in this population²¹. Moreover, in a larger cohort of STEMI with cardiogenic shock treated with primary PCI, multiple non-hemodynamic factors independently predicted death, highlighting the value of composite scores that incorporate age alongside clinical and laboratory parameters to refine prognostication²².

Therefore, among elderly patients undergoing emergency coronary intervention, enhanced nutritional support and meticulous care are essential to mitigate the risk of nosocomial infections. In patients with diabetes,

dysregulated glucose metabolism contributes to a hyperglycemic environment that impairs cellular defense mechanisms and immune function, thereby diminishing host resistance²³. Diabetes-related microvascular damage, autonomic neuropathy, and diabetic cardiomyopathy can alter cardiac structure and function, resulting in impaired left ventricular relaxation, reduced compliance, and an increased propensity for heart failure and cardiogenic shock following AMI, ultimately elevating both complication rates and mortality²⁴. Moreover, diabetic patients with coronary artery disease often exhibit more extensive and severe coronary lesions. Evidence consistently indicates a strong association between diabetes and infection²⁵. Diabetes not only increases susceptibility to infections but also disrupts adaptive immune responses and neutrophil function. Furthermore, hyperglycemia promotes microbial colonization and pathogen proliferation. Thus, in diabetic AMI patients post-PCI, vigilant monitoring for hospital-acquired infections, strict glycemic control, and early rehabilitation—when clinically appropriate—are recommended to reduce infection risk. Myocardial ischemia activates chemokine release, promoting leukocyte adhesion to necrotic tissue and facilitating clearance. This process, however, also generates abundant inflammatory mediators—such as free radicals and reactive oxygen species—that can exacerbate local injury and predispose patients to secondary infections^{26,27}. The NYHA functional classification reflects the severity of underlying cardiac impairment and vascular disease. Higher NYHA classes are associated with more advanced cardiac dysfunction and compromised systemic perfusion, which may impair organ function and reduce overall resistance to infection. Consequently, these patients are at increased risk of nosocomial infections, which can in turn worsen heart failure symptoms, creating a vicious cycle²⁸. It is therefore crucial to prioritize cardioprotective strategies and optimize cardiac function in clinical management.

Cardiac function status, commonly assessed by the NYHA classification, provides a practical and widely adopted measure of symptom burden and functional capacity in heart failure patients. Higher NYHA classes (III/IV) indicate advanced ventricular dysfunction, poor exercise tolerance, and hemodynamic compromise. In our study, patients with NYHA class III/IV were significantly more susceptible to nosocomial infection. Several mechanisms may explain this finding: impaired cardiac output and systemic perfusion weaken immune defense; pulmonary congestion and edema predispose to respiratory infections; and patients with severe heart failure often require invasive procedures and prolonged hospitalization, further increasing infection risk. Previous studies have confirmed that NYHA class independently predicts morbidity and mortality in cardiovascular disease cohorts^{29,30}. More recently, infections themselves have been recognized as both a trigger and a complication of acute heart failure, reinforcing the bidirectional relationship between advanced NYHA class and infection burden²⁸. These findings emphasize that careful monitoring, early mobilization, and proactive infection-prevention strategies should be prioritized in AMI patients with NYHA class III/IV.

During active coronary reperfusion therapy, timely measures to enhance cardiac function and promote diuresis should be implemented to optimize hemodynamic status and proactively prevent infections. Due to disease severity or associated complications, some AMI patients require invasive supportive procedures such as mechanical ventilation and indwelling catheterization. Previous studies^{31–33} have demonstrated that invasive interventions—including vascular catheterization, nasogastric tube placement, and tracheal intubation—elevate infection risk when mucosal barriers are compromised, aseptic techniques are inadequately followed, or equipment sterilization is insufficient. Indwelling urinary catheters may cause urethral mucosal injury, disrupt local immune barriers, and facilitate pathogenic bacterial colonization and ascending infection^{34,35}. Similarly, mechanical ventilation can compromise respiratory tract immunity, increasing susceptibility to respiratory pathogens and raising the risk of ventilator-associated pneumonia³⁶. Therefore, minimizing the use of invasive procedures is strongly recommended. When such interventions are necessary, strict adherence to indication guidelines, use of disposable medical devices, and meticulous aseptic techniques in sterile environments are essential to reducing hospital-acquired infections. The hospital environment itself represents a reservoir of pathogenic microorganisms, and prolonged hospitalization has been consistently associated with increased risk of nosocomial infections³⁷. Previous research has confirmed a correlation between length of stay and infection incidence in cardiovascular patients³⁸, a finding supported by the present study. Our results further indicate that extended hospitalization following PCI is linked to higher rates of infection, likely due to increased exposure to nosocomial pathogens. Thus, early discharge should be considered for AMI patients once their clinical condition has stabilized post-PCI.

In this study, continuous variables such as age and length of hospital stay were dichotomized using ROC curve-derived cut-off values (> 60 years and > 6 days, respectively). This approach facilitates clinical interpretation and enables easier application in bedside decision-making. However, dichotomization of continuous predictors can lead to loss of information, reduced statistical power, and potential instability of the cut-off points across different populations. Alternative modeling strategies, such as using continuous forms of predictors or restricted cubic splines, may better preserve prognostic information. Nevertheless, we selected cut-off values guided by ROC analysis to improve clinical applicability of the nomogram in routine practice.

This study identified risk factors for nosocomial infection following PCI in AMI patients and incorporated them into a nomogram prediction model. The model demonstrated excellent discriminative ability, with a C-index of 0.915 (95% CI 0.877–0.953). The calibration curve indicated close agreement between predicted and observed outcomes, aligning well with the ideal curve. Using a risk threshold of > 0.24, the nomogram provided greater clinical net benefit than models based solely on individual predictors such as age, diabetes, cardiac function class, invasive procedures, or hospitalization time. These results suggest that the nomogram can effectively identify high-risk AMI patients for nosocomial infection after PCI, thereby supporting clinical decision-making and infection prevention strategies.

However, several limitations should be acknowledged. The model was developed and validated using a single-center dataset without external validation. Given the multitude of factors influencing post-PCI infection, it is possible that not all relevant variables were included in the analysis. Furthermore, the sample was drawn from

a specific geographical region, which may limit generalizability. The relatively low number of infection events ($n = 39$) also raises concern for potential model overfitting, despite internal validation using bootstrapping. This may have led to overly optimistic performance estimates. Therefore, further validation in larger, multi-center cohorts is essential to confirm the robustness and general applicability of the model.

In conclusion, this study developed and internally validated a nomogram incorporating age, diabetes, cardiac function classification, invasive procedures, and length of stay to predict the risk of nosocomial infection in AMI patients after PCI. The model shows promising predictive accuracy and may serve as a practical tool for stratifying infection risk and guiding preventive measures in clinical practice.

Data availability

The simulation experiment data used to support the findings of this study are available from the corresponding author upon request.

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Author contributions

Shuo Huang and Yahui Zhou carried out study concepts and design, and manuscript editing; Shuo Huang, Yahui Zhou and Dandan Han helped to clinical studies, data acquisition; Shuo Huang, Yahui Zhou and Ran Zhou helped to data and statistical analysis and manuscript preparation; Dandan Han and Ran Zhou were the guarantor of integrity of the entire study, helped to literature research and manuscript review.

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Declarations

Competing interests

The authors declare no competing interests.

Ethical approval and informed consent

All procedures performed in studies involving human participants were in accordance with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This study was granted an exemption from ethics approval approved by the Ethics Committee of Jilin City Hospital of Chemical Industry (approval No. IEC-2020-010-01), and written informed consent was obtained. Informed consent was obtained from all individual participants included in the study.

Additional information

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