

Original Article

Risk factors for new-onset atrial fibrillation in patients with acute myocardial infarction

Xiaoming Ge¹, Yaobing Tao¹, Qingli Wang²

¹Department of Cardiology, The First Hospital of Fangshan District, Beijing Municipality, Beijing 102400, China;

²Department of Cardiology, Sheyang County People's Hospital, Yancheng 224399, Jiangsu, China

Received May 14, 2025; Accepted August 29, 2025; Epub September 15, 2025; Published September 30, 2025

Abstract: Objective: To investigate the incidence of new-onset atrial fibrillation (NOAF) and its related risk factors in patients with acute myocardial infarction (AMI), providing a scientific basis for clinical prevention and treatment. Methods: Clinical data of 240 The First Hospital of Fangshan District between June 2022 and June 2024 were retrospectively analyzed. Patients were divided into a NOAF group (31 patients) and non-NOAF group (209 patients) according to whether NOAF occurred during hospitalization. Baseline data, laboratory test results, and imaging data of patients were collected. Independent risk factors for NOAF were screened by multifactorial logistic regression analysis and their predictive value was assessed by Receiver Operating Characteristic (ROC) curve. Model goodness of fit was evaluated using the calibration curve and Hosmer-Lemeshow test. The clinical efficacy of the model was evaluated by using the decision curve, accuracy, sensitivity, and specificity. Results: The incidence of NOAF in AMI patients was 12.92%. The NOAF group had significantly higher mean age, higher proportion of patients with a history of hypertension, higher Killip classification ≥ 2 , larger left atrial diameter (LAD), and elevated troponin I (cTnI) levels compared to the non-NOAF group ($P < 0.05$). Multifactorial logistic regression analysis identified age (OR = 1.10, 95% CI: 1.03-1.17), history of hypertension (OR = 8.29, 95% CI: 2.81-24.43), cTnI level (OR = 1.35, 95% CI: 1.18-1.54), and LAD (OR = 1.34, 95% CI: 1.15-1.56) as independent risk factors for the development of NOAF in patients with AMI ($P < 0.05$). ROC curve analysis showed a high predictive efficacy for combining these four indicators, with an AUC of 0.973 (95% CI: 0.951-0.994). In addition, patients in the NOAF group had a longer mean length of stay and a significantly higher rate of adverse events than those in the non-NOAF group ($P < 0.05$). Conclusion: The occurrence of NOAF in AMI patients is associated with a variety of factors. Identifying these high-risk factors may help clinicians to identify patients at high risk of NOAF early, optimize management, and thus reduce the incidence and adverse outcomes of NOAF.

Keywords: Acute myocardial infarction, new-onset atrial fibrillation, clinical characteristics, risk factors

Introduction

Acute Myocardial Infarction (AMI) is a serious cardiovascular event with persistently high morbidity and mortality rates worldwide [1]. The main pathophysiologic mechanism of AMI involves the rupture of coronary atherosclerotic plaque, which leads to thrombosis and acute coronary artery occlusion, leading to myocardial avascular necrosis [2]. In recent years, after in-depth exploration into AMI pathogenesis and advancements in diagnostic and therapeutic techniques, early identification and intervention of AMI have significantly improved [3, 4]. However, the incidence of AMI remains high globally, with a substantial number of new

cases each year. AMI not only brings great pain to patients, but also imposes a heavy burden on healthcare resources [5]. Following AMI, the normal structure and function of the heart are severely damaged, leading to changes in the electrophysiological properties of the ischemic and necrotic areas of the myocardium, dysfunction of the heart's conduction system, and pathophysiologic processes such as abnormal ventricular wall motion and left ventricular remodeling [6]. Collectively, these factors constitute the pathologic basis for various complications.

New-Onset Atrial Fibrillation (NOAF) is a common complication following AMI, with its inci-

Risk for new-onset atrial fibrillation

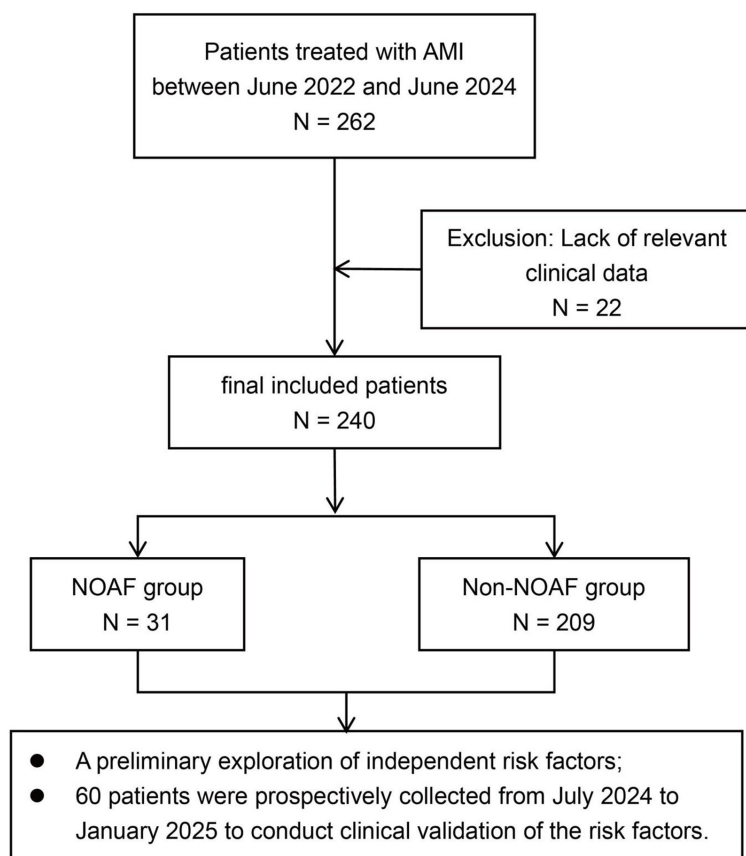


Figure 1. Flow chart of this study. Abbreviation: NOAF, new-onset atrial fibrillation.

dence reported between 5-20% [7, 8]. NOAF occurs when the atria lose their effective contractile function, leading to irregular ventricular rhythm and reduced cardiac output. Patients often experience symptoms such as palpitations, chest tightness, and shortness of breath, which significantly impair their quality of life [9]. More seriously, NOAF causes blood stagnation in the atria, significantly increasing the risk of thrombosis and subsequently ischemic stroke [10]. Given the current clinical practice, it is important to revisit the clinical characteristics of NOAF and its risk factors in AMI patients to optimize patient management and develop individualized prevention and treatment strategies.

In clinical practice, the management of NOAF in patients with AMI is challenging [11]. Currently, clinical management of new-onset AF in AMI patients relies on anticoagulation therapy and ventricular rate control, but there is still a lack of clear guidance on preventive measures for

NOAF [12, 13]. Therefore, an in-depth study of the clinical features and risk factors of NOAF in AMI patients is needed to identify high-risk patients at an early stage, enabling targeted preventive and therapeutic measures to reduce the incidence of NOAF and its associated adverse outcomes. This study aimed to systematically explore the clinical characteristics and independent risk factors of NOAF in AMI patients by retrospectively analyzing clinical data, providing a scientific basis for prevention and treatment, thus improving patient quality of life.

Patients and methods

Patient selection

Initially, a total of 262 patients with AMI who were treated at The First Hospital of Fangshan District between June 2022 and June 2024 were identified. Of these, 22 cases were excluded due to incomplete clinical data. As a result, 240 cases were included in the statistical analysis. Furthermore, to evaluate the clinical efficacy of the regression model, data from 60 patients were collected between July 2024 and January 2025. These patients met the same inclusion and exclusion criteria as the primary cohort. The validation cohort was independent of the primary retrospective cohort to avoid overlap and ensure objectivity in the validation results. This study was approved by the Ethics Committee of The First Hospital of Fangshan District. A flowchart of this study is shown in **Figure 1**.

Inclusion criteria: 1) Age ≥ 18 years; 2) Diagnosis of AMI by clinical examination [14]; 3) No history of atrial fibrillation on admission and no previous diagnosis of atrial fibrillation or atrial flutter; 4) At least one 12-lead electrocardiogram (ECG) or continuous ECG monitoring (≥ 24 hours) during hospitalization; 5) Complete clinical information. Exclusion criteria: 1) Comorbidities with other serious heart

conditions such as cardiomyopathy or heart valve disease; 2) Severe systemic diseases, such as severe hepatic or renal insufficiency, malignant tumor, or serious infection; 3) Comorbidities with other serious conditions that cause arrhythmias, such as hyperthyroidism or pheochromocytoma; 4) Treatment with positive inotropic drugs.

Data collection

Data collection was conducted independently by two trained cardiologists to ensure accuracy and consistency. The collected data included age, sex, admission heart rate, Killip classification, ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), cardiovascular risk factors including hypertension, diabetes mellitus, history of smoking, history of stroke, history of alcohol consumption (≥ 100 g of alcohol per day for at least one year consistently), electrocardiograms, cardiac function classifications, left atrial diameter (LAD), left intraventricular internal diameter, ejection fraction, and coronary angiography results. Cardiac function at admission was assessed according to the Killip classification criteria. Class 1: no heart failure; Class 2: heart failure with wet lung rales less than 50% of lung fields; Class 3: acute pulmonary oedema; and Class 4: cardiogenic shock [16]. Two-dimensional ultrasound and color Doppler were used to observe mitral regurgitation, with the degree of regurgitation quantitatively evaluated according to the ratio of regurgitant bundle area to the left atrium. Mild regurgitation: ratio $< 20\%$; moderate regurgitation: ratio of 21% - 40% ; severe regurgitation: ratio $> 40\%$ [17].

Outcome measurement

The primary outcome measure for this study was the occurrence of NOAF during hospitalization. Secondary outcomes included length of hospitalization and the incidence of adverse events. The diagnosis of NOAF was based on the physician's interpretation of the electrocardiogram, in accordance with current guidelines. All patients were monitored for a minimum of 72 hours during their hospitalization in the intensive care unit or general cardiac ward. Daily 12-lead electrocardiograms were performed during the hospital stay or if any new symptoms were detected.

Patients were classified as having NOAF if concomitant discordant atrial electrical excitation and ineffective atrial contractions were documented, or if typical ECG features consistent with a diagnosis of AF (P-wave absence, atrial activity represented by fibrillatory waves, and absolutely irregular RR intervals with a minimum duration of at least 30s or the entire 12-lead ECG) were documented during the acute hospitalization. NOAF was defined as a patient with no prior history of AF who was first diagnosed with AF during hospitalization for AMI [15]. According to the occurrence of NOAF during hospitalization, patients were divided into a NOAF group (31 cases) and NOAF group (209 cases).

Statistical analysis

Statistical analysis was conducted with SPSS 25.0. The Kolmogorov-Smirnov test was applied to assess the normality of continuous data. Normally distributed data were presented as mean $\pm$ standard deviation (SD), while non-normally distributed data were expressed as median (P25, P75). Comparisons between groups were conducted using the independent samples t-test for normally distributed data and the Mann-Whitney U test for non-normally distributed data. Categorical data were expressed as numbers (percentages), and comparisons between groups were performed using the chi-square (χ^2) test.

Univariate Logistic regression analysis was used to preliminarily screen for influencing factors. Based on the results of univariate Logistic regression analysis, variables with $P < 0.05$ were selected as potential risk factors and included in the multivariate Logistic regression analysis. Receiver operating characteristic (ROC) curves were generated to assess the predictive significance of risk factors for NOAF in AMI patients, and the area under the curve (AUC) was calculated. In addition, the degree of fit of the regression model was evaluated using the calibration curve and the Hosmer-Lemeshow goodness-of-fit test. The clinical decision curve, accuracy, sensitivity, and specificity were used to evaluate the clinical utility of the model. $P < 0.05$ was deemed significant.

Risk for new-onset atrial fibrillation

Table 1. Comparison of general clinical data between the NOAF and non-NOAF groups

Variable		NOAF group (n = 31)	Non-NOAF group (n = 209)	$\chi^2/t/F$	P
Age		67.32±6.50	61.44±9.47	3.344	< 0.001
Sex	Male	19 (61.29)	142 (67.94)	0.541	0.462
	Female	12 (38.71)	67 (32.06)		
History of smoking		17 (54.84)	140 (66.99)	1.761	0.185
History of alcohol consumption		13 (41.94)	96 (46.15)	0.174	0.677
History of hypertension		21 (67.74)	54 (25.84)	22.065	< 0.001
History of diabetes		9 (29.03)	60 (28.71)	0.001	0.970
BMI (Kg/m ²)		20.66±2.43	21.27±3.07	1.058	0.291
Killip class	1	14 (45.16)	143 (68.42)	6.456	0.011
	≥ 2	17 (54.84)	66 (31.58)		
Heart rate at admission (bpm)		82.61±12.09	79.88±10.25	1.351	0.178
Systolic blood pressure at admission		122.51±20.58	125.36±24.25	0.612	0.535
Diastolic blood pressure at admission		74.56±10.09	76.88±12.25	0.984	0.326
Mean arterial pressure		94.19±13.27	91.17±12.65	1.233	0.219
Classification of myocardial infarction	STEMI	20 (64.52)	138 (66.03)	0.028	0.868
	NSTEMI	11 (35.48)	71 (33.97)		

Abbreviations: NOAF, new-onset atrial fibrillation; AMI, acute myocardial infarction; BMI, body mass index; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction.

Table 2. Comparison of cardiac biomarkers and echocardiographic data between the NOAF and non-NOAF groups

Variable	NOAF group (n = 31)	Non-NOAF group (n = 209)	t	P
LAD (mm)	42.15±4.41	38.24±3.23	5.982	< 0.001
IVST (mm)	9.64±1.21	9.24±1.44	1.471	0.143
LVESd (mm)	36.34±4.98	35.22±5.34	1.099	0.273
LVEDd (mm)	50.79±4.93	51.36±5.84	0.517	0.606
LVEF (%)	53.58±10.39	56.55±9.41	1.612	0.108
cTnI (µg/L)	18.51±4.49	10.45±4.25	9.805	< 0.001
Moderate to severe mitral regurgitation	16 (48.39)	70 (19.67)	3.855	0.049

Abbreviations: NOAF, new-onset atrial fibrillation; AMI, acute myocardial infarction; LAD, left atrial diameter; IVST, interventricular septal thickness; LVESd, left ventricular end-systolic dimension; LVEDd, left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; cTnI, troponin I.

Results

Comparison of general information between the NOAF and non-NOAF groups

Of the 240 patients, 31 developed NOAF, with an incidence of 12.92% (**Table 1**). The NOAF group exhibited a significantly higher mean age (67.32±6.50 years) compared to the non-NOAF group (61.44±9.47 years) ($P < 0.05$). Additionally, the NOAF group had a greater proportion of patients with a history of hypertension (67.74%) and those classified as Killip

class ≥ 2 (54.84%) compared to the non-NOAF group (25.84% and 31.58%, respectively) ($P < 0.05$).

Comparison of cardiac biomarkers and echocardiography between the NOAF and non-NOAF groups

The LAD in the NOAF group was significantly larger than that of the non-NOAF group ($P < 0.001$) (**Table 2**). The cTnI level in the NOAF group (18.51±4.49 µg/L) was significantly higher than that of the non-NOAF group (10.45±4.25

Risk for new-onset atrial fibrillation

Table 3. Comparison of coronary angiographic findings between NOAF and non-NOAF groups

Variable		NOAF group (n = 31)	Non-NOAF group (n = 209)	$\chi^2/t/F$	P
IRA	LAD	15 (48.39)	102 (48.80)	0.200	0.841
	LCX	4 (12.90)	30 (14.35)		
	RCA	11 (35.48)	75 (35.89)		
	LM	1 (3.23)	2 (0.96)		
Number of diseased vessels	Single-vessel disease	13 (38.71)	128 (61.24)	4.153	0.042
	Multi-vessel disease	18 (61.90)	81 (38.76)		
Endotracheal Intubation		3 (9.68)	19 (9.09)	1.723	0.189

Abbreviations: NOAF, new-onset atrial fibrillation; AMI, acute myocardial infarction; IRA, infarct-related artery; LAD, left atrial diameter; LCX, left circumflex artery; RCA, right coronary artery; LM, left main coronary artery.

Table 4. Univariate logistic regression analysis of NOAF in AMI patients

Variable	β	S.E.	Z	P	OR (95% CI)
Age	0.07	0.02	3.19	0.001	1.08 (1.03-1.12)
History of hypertension					
No					1.00 (Reference)
Yes	1.80	0.42	4.32	< 0.001	6.03 (2.67-13.61)
Killip Class					
1					1.00 (Reference)
≥ 2	0.84	0.39	2.15	0.031	2.31 (1.08-4.95)
LAD (mm)	0.33	0.07	5.08	< 0.001	1.40 (1.23-1.59)
cTnI ($\mu\text{g/L}$)	0.28	0.05	5.13	< 0.001	1.32 (1.19-1.47)
Moderate to severe mitral regurgitation					
$\leq 40\%$					1.00 (Reference)
$> 40\%$	0.75	0.39	1.93	0.053	2.12 (0.99-4.53)
Number of diseased vessels					
Single-vessel disease					1.00 (Reference)
Multi-vessel disease	0.78	0.39	2.00	0.045	2.19 (1.02-4.17)

Abbreviations: NOAF, new-onset atrial fibrillation; AMI, acute myocardial infarction; SE, standard error; OR, odds ratio; CI, confidence interval; LAD, left atrial diameter; cTnI, troponin I.

$\mu\text{g/L}$) ($P < 0.05$). No significant differences were found in the other cardiac or echocardiographic data ($P > 0.05$).

Comparison of coronary angiography between the NOAF and non-NOAF groups

In AMI patients, coronary angiography results revealed a higher prevalence of multivessel disease in the NOAF group (61.90%) compared to the non-NOAF group (58.37%) ($P < 0.05$) (Table 3). However, the rates of endotracheal intubation were similar between the two groups ($P > 0.05$).

Screening for potential NOAF risk factors

Univariate Logistic regression was used to preliminarily screen for the risk factors for NOAF.

The results showed that older age (OR = 1.08, 95% CI: 1.03-1.12), history of hypertension (OR = 6.03, 95% CI: 2.67-13.61), Killip class ≥ 2 (OR = 2.31, 95% CI: 1.08-4.95), a higher number of diseased vessels (OR = 2.19, 95% CI: 1.02-4.17), LAD (OR = 1.40, 95% CI: 1.23-1.95), and higher cTnI levels (OR = 1.32, 95% CI: 1.19-1.47) were significantly associated with an increased risk of NOAF ($P < 0.05$) (Table 4).

Independent risk factors for NOAF

Variables with $P < 0.05$ in univariate Logistic regression were included in a multivariate Logistic regression model. The results of multivariate Logistic regression analysis revealed that age (OR = 1.10, 95% CI: 1.03-1.17), a history of hypertension (OR = 8.29, 95% CI: 2.81-

Risk for new-onset atrial fibrillation

Table 5. Multivariate logistic regression analysis of NOAF in AMI patients

Variable	β	S.E.	Z	P	OR (95% CI)	VIF
Age	0.09	0.03	2.87	0.004	1.10 (1.03-1.17)	1.043
History of Hypertension						
No					1.00 (Reference)	
Yes	2.11	0.55	3.83	< 0.001	8.29 (2.81-24.43)	1.040
LAD (mm)	0.29	0.08	3.75	< 0.001	1.34 (1.15-1.56)	1.107
cTnI ($\mu\text{g/L}$)	0.30	0.07	4.28	< 0.001	1.35 (1.18-1.54)	1.084

Abbreviations: NOAF, new-onset atrial fibrillation; AMI, acute myocardial infarction; SE, standard error; OR, odds ratio; CI, confidence interval; VIF, variance inflation factor; LAD, left atrial diameter; cTnI, troponin I.

Table 6. ROC curve analysis for independent risk factors in predicting NOAF in patients with AMI

Variable	AUC	P	95% CI	Sensibility	Specificity	Cutoff value
Age	0.705	< 0.001	0.622-0.788	0.968	0.407	58.50
History of hypertension	0.710	< 0.001	0.608-0.811	0.677	0.742	0.17
cTnI	0.791	< 0.001	0.705-0.878	0.613	0.885	15.19
LAD	0.744	< 0.001	0.632-0.856	0.613	0.861	41.47
Collaborative Forecasting	0.926	< 0.001	0.870-0.982	0.935	0.856	0.11

Abbreviations: NOAF, new-onset atrial fibrillation; AMI, acute myocardial infarction; ROC, receiver operating characteristic; AUC, area under the curve; CI, confidence interval; LAD, left atrial diameter; cTnI, troponin I.

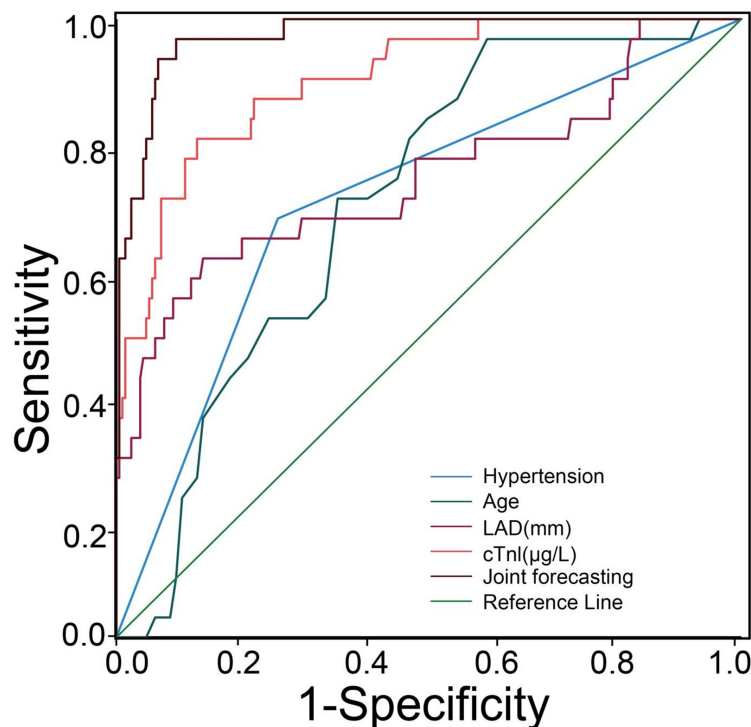


Figure 2. ROC curves for each risk factor and their combination in predicting NOAF in patients with AMI. NOAF, new-onset atrial fibrillation; AMI, acute myocardial infarction.

were independent risk factors for NOAF in AMI patients ($P < 0.05$) (**Table 5**). The individual predictive efficacy and combined predictive efficacy of these independent risk factors for NOAF are shown in **Table 6**.

ROC curve analysis

ROC curve analysis showed that the AUC for age, a history of hypertension, LAD, and cTnI levels in predicting NOAF in AMI patients were 0.705 (95% CI: 0.622-0.788), 0.710 (95% CI: 0.608-0.811), 0.744 (95% CI: 0.848-0.958), and 0.903 (95% CI: 0.848-0.958), respectively (**Figure 2**). The combined ACU for these four indicators was 0.973 (95% CI: 0.951-0.994).

Evaluation of the ROC curve

The calibration curve is shown in **Figure 3**. The P value of the Hosmer-Lemeshow test was

24.43), cTnI levels (OR = 1.35, 95% CI: 1.18-1.54), and LAD (OR = 1.34, 95% CI: 1.15-1.56)

0.988, indicating no significant difference between the observed and predicted values of

Risk for new-onset atrial fibrillation

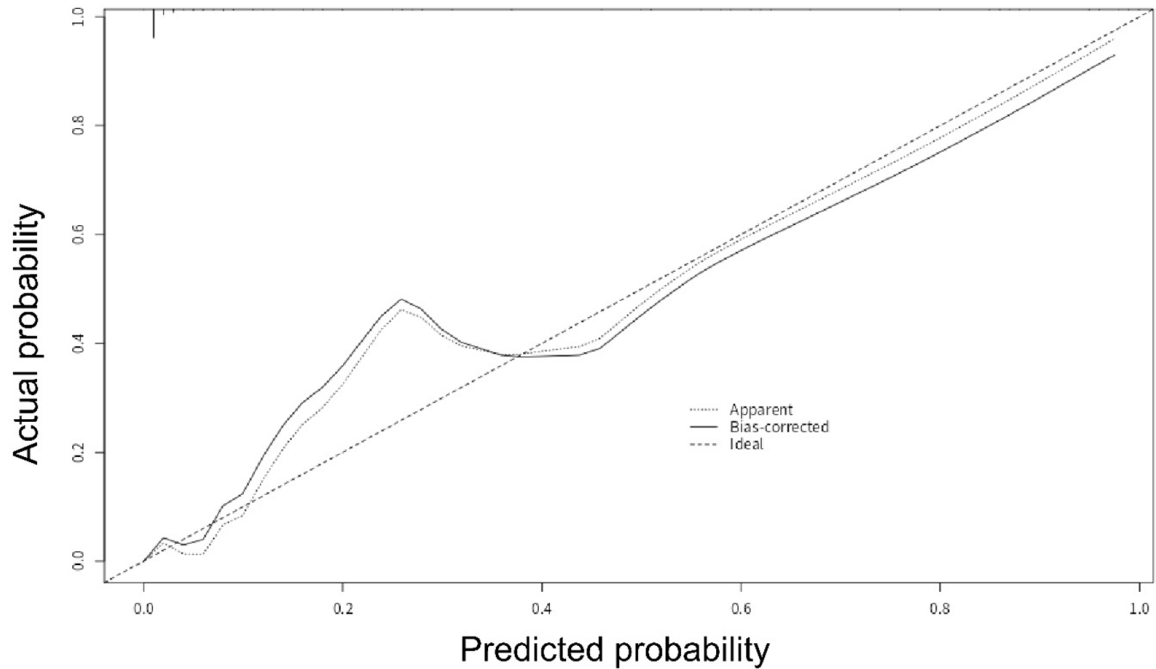


Figure 3. Calibration curve analysis.

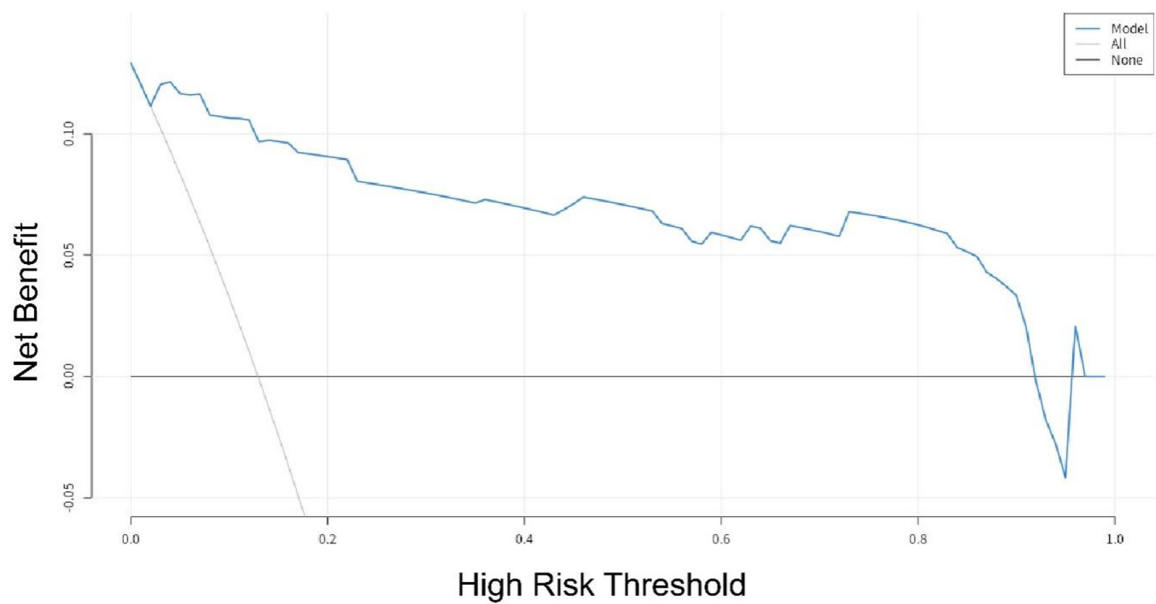


Figure 4. Clinical decision curve.

the model, confirming a good fit. The clinical decision curve is shown in **Figure 4**. The results demonstrated that when the risk probability falls within the range of 0.05 to 0.90, this model provides positive net benefits and has good clinical value.

Clinical value verification

To further verify the clinical efficacy of the Logistic regression model, we collected data of 60 patients from different periods for clinical validation. The prediction results for these

Table 7. Clinical efficacy evaluation

Prediction result	Gold standard		Total
	NOAF	Non-NOAF	
NOAF	45	2	47
Non-NOAF	6	7	13
Total	51	9	60

Abbreviation: NOAF, new-onset atrial fibrillation.

patients were compared with the gold standard, and the results are shown in **Table 7**. The accuracy, sensitivity, and specificity of this model were 86.67%, 88.24% and 77.78% respectively.

Comparison of adverse events

The mean length of hospital stay in the NOAF group was 10.15 days, which was significantly higher than that in the non-NOAF group ($P = 0.002$) (**Table 8**). A comparison of adverse events between the two groups revealed that both groups experienced recurrent myocardial infarction, repeat coronary artery reconstruction, heart failure, and cerebral infarction. The proportion of adverse events in the NOAF group (32.26%) was significantly higher than in the non-NOAF group (15.31%) ($P = 0.021$).

Discussion

AMI, as a serious cardiovascular disease, continues to exhibit a high morbidity and mortality rate globally, placing a heavy burden on patients and society [1, 18]. With in-depth research on the pathogenesis of AMI and continuous advancement of diagnostic and therapeutic techniques, the early recognition and intervention of AMI have notably improved. However, the severe damage of cardiac structure and function in AMI patients often leads to a variety of complications, of which NOAF is quite prevalent. This study aimed to provide a scientific basis for optimizing patient management and developing individualized preventive treatment by retrospectively analyzing the clinical characteristics and risk factors of NOAF in AMI patients. A total of 240 AMI patients were included in this study, 31 of whom developed NOAF during hospitalization, with an incidence rate of 12.92%. The results showed that age, history of hypertension, cTnI level, and LAD were independent risk factors for NOAF in AMI patients. ROC curve analysis further confirmed

the predictive value of these factors, especially the cTnI level, which demonstrated a high AUC of 0.903. In addition, the duration of hospitalization and the incidence of adverse events were significantly higher in the NOAF compared to the non-NOAF group.

Age, as an independent risk factor for NOAF, is consistent with the findings of previous studies [19]. Zhang et al. conducted a retrospective analysis of 397 patients and identified advanced age as one of the independent risk factors for NOAF [20]. The underlying mechanism may be related to the electrophysiological characteristics and structural changes of atrial myocytes. With advancing age, the electrophysiological properties and structure of atrial myocytes undergo changes, such as fibrosis of atrial myocytes and reduced conduction velocities, which increase the risk of atrial fibrillation [21]. Among AMI patients, the elderly population often has relatively poorer cardiac reserve, lower tolerance to myocardial infarction, and is more susceptible to atrial remodeling and electrophysiological disturbances that can induce atrial fibrillation. In addition, it has been shown that chronic low-grade inflammation in the elderly leads to elevated levels of pro-inflammatory factors (e.g., IL-6, TNF- α), which promote atrial fibrosis and electrical remodeling [22]. Aging also exacerbates redox reactions in the body, producing more reactive oxygen species (ROS), the accumulation of which can damage cardiomyocytes, affect calcium regulation, and increase susceptibility to atrial fibrillation [23, 24]. This study also observed that hypertension was associated with an increased risk of NOAF. As a common chronic disease, hypertension affects the cardiovascular system in many ways. Chronic hypertension increases the pressure load on the left ventricle, leading to compensatory hypertrophy of cardiomyocytes and resulting in left ventricular hypertrophy (LVH) [25]. LVH not only increases myocardial oxygen consumption, but also leads to abnormalities in the electrical activity of the cardiomyocytes, increasing the risk of arrhythmias [26]. Hypertrophied cardiomyocytes contribute to myocardial fibrosis and interstitial hyperplasia, further altering the electrical conduction properties of the myocardium and promoting the development of arrhythmia [27]. Hypertension causes abnormal electrical activity in atrial myocytes, primarily manifested as altered

Table 8. Comparison of hospitalization time and adverse event rates between patients in the NOAF and non-NOAF groups

Variable	NOAF group (n = 31)	Non-NOAF group (n = 209)	x ² /t	P
Length of hospital stay	10.15±3.88	8.15±3.20	3.155	0.002
Adverse event				
Recurrent myocardial infarction	2 (6.45)	8 (3.83)		
Revascularization	1 (3.23)	5 (2.39)		
Heart Failure	5 (16.13)	13 (6.22)		
Cerebral Infarction	2 (6.45)	6 (2.87)		
Total	10 (32.26)	32 (15.31)	5.370	0.021

Abbreviation: NOAF, new-onset atrial fibrillation.

action potentials and folding potentials [28]. These electrophysiological alterations increase the autoregulation of atrial myocytes and promote the formation of atrial ectopic rhythmic points, thereby increasing the risk of atrial fibrillation. In addition, hypertension leads to delayed intra-atrial conduction and foldback phenomena, further promoting the development of NOAF [29].

This study also observed an association between cTnI and NOAF. Elevated levels of cTnI, a myocardium-specific protein, usually reflect damage or necrosis of cardiomyocytes [30]. Damage to cardiomyocytes leads to impaired membrane integrity and ion channel dysfunction, which leads to intracellular calcium overload and membrane potential instability [31]. These changes increase the automaticity of cardiomyocytes, promoting the formation of ectopic rhythm points and increasing the risk of arrhythmia. Myocardial ischemia and reperfusion injury caused by AMI trigger oxidative stress, leading to excessive generation of ROS [32]. Oxidative stress induces damage to cardiomyocytes, further exacerbating abnormalities in myocardial electrical activity and fibrosis, thus increasing the risk of arrhythmia [33]. Elevated cTnI levels suggest a more severe degree of myocardial injury, which may be accompanied by higher oxidative stress, further increasing the risk of NOAF. In this study, there was also an association between LAD and NOAF risk. Atrial dilatation increases mechanical stress on cardiomyocytes, which in turn activates myocardial fibroblasts and promotes myocardial fibrosis [34]. Fibrosis alters the electrical conduction properties of the myocardium, increases electrical conduction heterogeneity, and further promotes arrhythmogenesis [19, 35]. LAD enlargement indicates

the presence of cardiac insufficiency, which can lead to elevated atrial pressure and further aggravate atrial dilation [36]. Elevated atrial pressure can alter the electrical conduction characteristics of the atrium, thereby increasing the risk of arrhythmia [37]. This mechanism might partially explain the association between LAD and NOAF in this study, but its specific mechanism of action requires further research for verification.

Despite the achievements of this study, there are some limitations. First, this study was a single-center retrospective study with a relatively limited sample size, which may have introduced certain selection bias and confounding factors. While multivariate adjustments were performed, unmeasured confounders (e.g., undiagnosed sleep apnea) may influence NOAF risk. Future prospective studies with multi-center and large samples should be conducted to validate the findings of this study. Second, this study mainly focused on the occurrence of NOAF during the acute phase of AMI, and did not explore the long-term incidence of NOAF and its risk factors post-AMI. Future research should strengthen long-term follow-up to better understand the pattern of NOAF occurrence and its risk factors. Furthermore, this study mainly relied on clinical data and statistical analyses. It lacked in-depth molecular biology and histology studies. Future studies should incorporate molecular and cellular techniques to explore the molecular mechanisms and cellular basis for the development of NOAF after AMI. Attention should focus on developing individualized prevention and treatment strategies for AMI patients with different risk factors to reduce the risk of NOAF. For example, in older AMI patients with a history of hypertension, enhanced blood pressure management could

reduce atrial remodeling. For patients with severe myocardial injury, improving myocardial perfusion should be prioritized to reduce the degree of myocardial injury. For patients who have developed NOAF, anticoagulants and ventricular rate control drugs should be reasonably applied to reduce the occurrence of adverse events.

Conclusion

By analyzing the clinical characteristics of NOAF in AMI patients, this study identified age, history of hypertension, cTnI level, and LAD as independent risk factors for NOAF in AMI patients. This study provides a scientific foundation for optimizing patient management and developing individualized preventive strategies. However, due to the study's limitations, further in-depth research is needed to elucidate the mechanisms and preventive measures for NOAF after AMI.

Disclosure of conflict of interest

None.

Address correspondence to: Qingli Wang, Department of Cardiology, Sheyang County People's Hospital, No. 129, Xingfu Avenue, Sheyang County, Yancheng 224399, Jiangsu, China. Tel: +86-153-96736316; E-mail: wangqingli1907@163.com

References

- [1] Krittanawong C, Khawaja M, Tamis-Holland JE, Girotra S and Rao SV. Acute myocardial infarction: etiologies and mimickers in young patients. *J Am Heart Assoc* 2023; 12: e029971.
- [2] Galli M, Niccoli G, De Maria G, Brugaletta S, Montone RA, Vergallo R, Benenati S, Magnani G, D'Amario D, Porto I, Burzotta F, Abbate A, Angiolillo DJ and Crea F. Coronary microvascular obstruction and dysfunction in patients with acute myocardial infarction. *Nat Rev Cardiol* 2024; 21: 283-298.
- [3] Zhu D, Zhang X, Fang Y, Xu Z, Yu Y, Zhang L, Yang Y, Li S, Wang Y, Jiang C and Huang D. Identification of a lactylation-related gene signature as the novel biomarkers for early diagnosis of acute myocardial infarction. *Int J Biol Macromol* 2024; 282: 137431.
- [4] Scarsini R, Portolan L, Della Mora F, Marin F, Mainardi A, Ruzzarin A, Levine MB, Banning AP, Ribichini F, Garcia Garcia HM and De Maria GL. Angiography-derived and sensor-wire methods to assess coronary microvascular dys-

- function in patients with acute myocardial infarction. *JACC Cardiovasc Imaging* 2023; 16: 965-981.
- [5] Murakami T, Sakakura K, Jinnouchi H, Taniguchi Y, Tsukui T, Watanabe Y, Yamamoto K, Seguchi M, Wada H and Fujita H. Comparison of medical resource use and total admission cost in patients with acute myocardial infarction between on-hours visit versus off-hours visit. *Cardiovasc Interv Ther* 2022; 37: 651-659.
- [6] Reddy RK, Howard JP, Jamil Y, Madhavan MV, Nanna MG, Lansky AJ, Leon MB and Ahmad Y. Percutaneous coronary revascularization strategies after myocardial infarction: a systematic review and network meta-analysis. *J Am Coll Cardiol* 2024; 84: 276-294.
- [7] Li Z, Liu Q, Liu F, Hidru TH, Yang Y, Wang S, Bai L, Chen J, Yang X and Xia Y. Atrial cardiomyopathy markers and new-onset atrial fibrillation risk in patients with acute myocardial infarction. *Eur J Intern Med* 2022; 102: 72-79.
- [8] Sadat B, Al Taii H, Sabayon M and Narayanan CA. Atrial fibrillation complicating acute myocardial infarction: prevalence, impact, and management considerations. *Curr Cardiol Rep* 2024; 26: 313-323.
- [9] Johnston BW, Chean CS, Duarte R, Hill R, Blackwood B, McAuley DF and Welters ID. Management of new onset atrial fibrillation in critically unwell adult patients: a systematic review and narrative synthesis. *Br J Anaesth* 2022; 128: 759-771.
- [10] Alhatemi G, Zghouzi M, Sattar Y, Ahmad B, Ullah W and Alraies MC. Anticoagulation management of post-cardiac surgery new-onset atrial fibrillation. *Cleve Clin J Med* 2022; 89: 329-335.
- [11] Yue B, Hou Q, Bredehorst J, Han Q, Zhang B, Zhang C, Zhang J, Chen S, Wu S and Li K. Atrial fibrillation increases the risk of new-onset myocardial infarction amongst working-age population: a propensity-matched study. *Herz* 2023; 48: 408-412.
- [12] Lasica R, Djukanovic L, Popovic D, Savic L, Mrdovic I, Radovanovic N, Radovanovic MR, Polovina M, Stojanovic R, Matic D, Uscumlic A and Asanin M. Use of anticoagulant therapy in patients with acute myocardial infarction and atrial fibrillation. *Medicina (Kaunas)* 2022; 58: 338.
- [13] Lancini D, Prasad A, Thomas L, Atherton J, Martin P and Prasad S. Predicting new onset atrial fibrillation post acute myocardial infarction: echocardiographic assessment of left atrial size. *Echocardiography* 2023; 40: 456-463.
- [14] Lindahl B and Mills NL. A new clinical classification of acute myocardial infarction. *Nat Med* 2023; 29: 2200-2205.

- [15] Tzeis S, Gerstenfeld EP, Kalman J, Saad EB, Sepehri Shamloo A, Andrade JG, Barbhuiya CR, Baykaner T, Boveda S, Calkins H, Chan NY, Chen M, Chen SA, Dagues N, Damiano RJ, De Potter T, Deisenhofer I, Derval N, Di Biase L, Duytschaever M, Dyrda K, Hindricks G, Hocini M, Kim YH, la Meir M, Merino JL, Michaud GF, Natale A, Nault I, Nava S, Nitta T, O'Neill M, Pak HN, Piccini JP, Pürerfellner H, Reichlin T, Saenz LC, Sanders P, Schilling R, Schmidt B, Supple GE, Thomas KL, Tondo C, Verma A, Wan EY, Steven D, Agbayani MJ, Jared Bunch T, Chugh A, Díaz JC, Freeman JV, Hardy CA, Heidebuchel H, Johar S, Linz D, Maesen B, Noseworthy PA, Oh S, Porta-Sanchez A, Potpara T, Rodriguez-Diez G, Sacher F, Suwalski P and Trines SA. 2024 European Heart Rhythm Association/Heart Rhythm Society/Asia Pacific Heart Rhythm Society/Latin American Heart Rhythm Society expert consensus statement on catheter and surgical ablation of atrial fibrillation. *Europace* 2024; 26: euae043.
- [16] Milwidsky A, Greidinger D, Frydman S, Hochstadt A, Ifrach-Kashtan N, Mizrahi M and Topilsky Y. Echocardiographic killip classification. *J Am Soc Echocardiogr* 2022; 35: 287-294.
- [17] Maher T, Vegh A and Uretsky S. Mitral regurgitation: advanced imaging parameters and changing treatment landscape. *Heart Fail Clin* 2023; 19: 525-530.
- [18] Danchin N. Improved long-term survival after acute myocardial infarction: the success of comprehensive care from the acute stage to the long term. *Eur Heart J* 2023; 44: 499-501.
- [19] Bizhanov KA, Abzaliev KB, Baimbetov AK, Sarsenbayeva AB and Lyan E. Atrial fibrillation: epidemiology, pathophysiology, and clinical complications (literature review). *J Cardiovasc Electrophysiol* 2023; 34: 153-165.
- [20] Zhang LX, Cao JY and Zhou XJ. Construction and validation of a nomogram prediction model for the risk of new-onset atrial fibrillation following percutaneous coronary intervention in acute myocardial infarction patients. *BMC Cardiovasc Disord* 2024; 24: 642.
- [21] Laredo M, Waldmann V, Khairy P and Nattel S. Age as a critical determinant of atrial fibrillation: a two-sided relationship. *Can J Cardiol* 2018; 34: 1396-1406.
- [22] Zhang H and Dhalla NS. The role of pro-inflammatory cytokines in the pathogenesis of cardiovascular disease. *Int J Mol Sci* 2024; 25: 1082.
- [23] Chaudhary MR, Chaudhary S, Sharma Y, Singh TA, Mishra AK, Sharma S and Mehdi MM. Aging, oxidative stress and degenerative diseases: mechanisms, complications and emerging therapeutic strategies. *Biogerontology* 2023; 24: 609-662.
- [24] Wang L, Qiu S, Li X, Zhang Y, Huo M and Shi J. Myocardial-targeting tannic cerium nanocatalyst attenuates ischemia/reperfusion injury. *Angew Chem Int Ed Engl* 2023; 62: e202305576.
- [25] Đorđević DB, Koračević GP, Đorđević AD and Lović DB. Hypertension and left ventricular hypertrophy. *J Hypertens* 2024; 42: 1505-1515.
- [26] Bacharova L, Kollarova M, Bezak B and Bohm A. Left ventricular hypertrophy and ventricular tachyarrhythmia: the role of biomarkers. *Int J Mol Sci* 2023; 24: 3881.
- [27] Schlittler M, Pramstaller PP, Rossini A and De Bortoli M. Myocardial fibrosis in hypertrophic cardiomyopathy: a perspective from fibroblasts. *Int J Mol Sci* 2023; 24: 14845.
- [28] Aggarwal K, Valleru PS, Anamika FNU, Aggarwal P, Gupta I, Gupta V, Garg N and Jain R. Unraveling the complex relationship-atrial fibrillation and pulmonary hypertension. *Curr Cardiol Rep* 2024; 26: 885-891.
- [29] Antoun I, Layton GR, Nizam A, Barker J, Abdelrazik A, Eldesouky M, Koya A, Lau EYM, Zakkar M, Somani R and Ng GA. Hypertension and atrial fibrillation: bridging the gap between mechanisms, risk, and therapy. *Medicina (Kaunas)* 2025; 61: 362.
- [30] Canty JM Jr. Myocardial injury, troponin release, and cardiomyocyte death in brief ischemia, failure, and ventricular remodeling. *Am J Physiol Heart Circ Physiol* 2022; 323: H1-H15.
- [31] Varró A, Tomek J, Nagy N, Virág L, Passini E, Rodriguez B and Baczkó I. Cardiac transmembrane ion channels and action potentials: cellular physiology and arrhythmogenic behavior. *Physiol Rev* 2021; 101: 1083-1176.
- [32] Peng Y, Tao Y, Liu L, Zhang J and Wei B. Cross-talk among reactive oxygen species, autophagy and metabolism in myocardial ischemia and reperfusion stages. *Aging Dis* 2024; 15: 1075-1107.
- [33] Tang SP, Mao XL, Chen YH, Yan LL, Ye LP and Li SW. Reactive oxygen species induce fatty liver and ischemia-reperfusion injury by promoting inflammation and cell death. *Front Immunol* 2022; 13: 870239.
- [34] Marques-Alves P, Ferreira JA, Freitas AA, Almeida JP, Baptista R, Castro G, Martins R, Donato P, Ferreira MJ and Gonçalves L. Atrial mechanics in hypertrophic cardiomyopathy: discriminating between ventricular hypertrophy and fibrosis. *Arq Bras Cardiol* 2022; 118: 77-87.
- [35] Boyle PM, Del Álamo JC and Akoum N. Fibrosis, atrial fibrillation and stroke: clinical updates and emerging mechanistic models. *Heart* 2021; 107: 99-105.
- [36] Debonnaire P, Joyce E, Hiemstra Y, Mertens BJ, Atsma DE, Schalij MJ, Bax JJ, Delgado V and

Risk for new-onset atrial fibrillation

Marsan NA. Left atrial size and function in hypertrophic cardiomyopathy patients and risk of new-onset atrial fibrillation. *Circ Arrhythm Electrophysiol* 2017; 10: e004052.

[37] Pozios I, Vouliotis AI, Dilaveris P and Tsioufis C. Electro-mechanical alterations in atrial fibrillation: structural, electrical, and functional correlates. *J Cardiovasc Dev Dis* 2023; 10: 149.