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Original Article

Establishment and validation of a risk prediction model for malignant ventricular arrhythmia in acute myocardial infarction patients based on 24-hour dynamic electrocardiogram parameters

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Abstract: Objectives: To develop and validate a nomogram for predicting malignant ventricular arrhythmias (MVA) risk in acute myocardial infarction (AMI), utilizing parameters derived from 24-hour dynamic electrocardiogram (Holter) monitoring. Methods: We enrolled 279 AMI patients with MVA diagnosed via Holter monitoring. Key parameters from 24-hour Holter monitoring, such as heart rate variability (HRV) and QT interval variability (QTV), were extracted for subsequent analysis. These parameters were subsequently incorporated with clinical indicators to develop a risk prediction model. Electrocardiographic parameters and clinical indicators associated with MVA in the multivariable logistic regression analysis were used to construct a predictive nomogram for risk visualization. The nomogram model underwent internal and external validation for discrimination, calibration, and clinical utility. SHAP was used for model interpretation. Results: Of the 279 AMI patients, 37 cases (13.3%) developed MVA. Multivariate analysis showed that SDNN, 24h-QTV, night-QTV, day-QTV, TnI and Killip classification were independent predictors. The AUC of the model was 0.95 (0.92-0.98). The nomogram demonstrated good calibration, with predicted probabilities aligning well with actual outcomes (Hosmer-Lemeshow test, P=0.781). Clinical net benefit of the model was observed over a wide threshold probability range of 0.10 to 0.80 in the decision curve analysis. Conclusions: The nomogram based on 24-hour Holter parameters can effectively identify a risk of MVA in AMI patients during hospitalization and provide an objective basis for clinical decision-making.

Keywords: Acute myocardial infarction, malignant ventricular arrhythmia, 24-hour Holter, prediction model

Introduction

Acute myocardial infarction (AMI) is a major cardiovascular disease, notable for its acute onset and rapid progression. This clinical profile contributes to its high mortality rate, making it a leading cause of death worldwide [1]. For example, with the significant advancement of medical standards today, among elderly American patients with AMI who survive 30 days after hospitalization, their mortality rate within 10 years is still as high as over 70%, and the rate of readmission due to myocardial infarction also reaches 25% [2]. In recent years, with the advancement of reperfusion therapy and drug therapy, the survival rate of patients with AMI has improved. However, malignant ventricular arrhythmia (MVA), as one of the most serious complications of AMI patients, primarily encompasses life-threatening arrhythmias, including sustained ventricular tachycardia (VT), ventricular flutter or ventricular fibrillation (VF) [3-5]. It is a major contributing factor to sudden cardiac death and significantly impairs patients' quality of life [6]. A study has shown that compared to patients without MVA, the 30-day survival rate of patients with MVA is significantly lower [7]. Hence, there is a need to develop a practical prediction model for assessing the risk of in-hospital MVA in AMI patient. This risk assess-

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ment can identify high-risk patients early upon admission and guide timely preventive measures to improve overall survival.

24-Hour dynamic electrocardiogram (Holter), as a non-invasive and continuous electrocardiographic monitoring technology, can record complete electrocardiographic activity information of patients during daily activities, providing abundant parameter information for risk assessment of MVA in AMI patients. Compared to conventional 12-lead electrocardiogram, dynamic electrocardiogram has the following advantages: higher sensitivity, specificity and accuracy; higher detection rates of ventricular premature beats (VPBs) in bigeminy and trigeminy, paired VPBs, atrial premature beats (APBs) in bigeminy and trigeminy, paired APBs, and atrioventricular block; higher patient satisfaction [8]. Heart rate variability (HRV) describes the physiologic variation in RR intervals between consecutive depolarizations of the sinoatrial node. It is one of the most important parameters in 24-hour dynamic electrocardiography, reflecting the regulatory function of the cardiac autonomic nervous system [9]. A study has shown that the AUC of SDNN, an indicator of HRV, which refers to the standard deviation of all normal RR intervals within 24 hours, is 0.711, indicating good predictive value [10]. Lower HRV serves as a marker for an increased risk of cardiovascular events and death, despite the precise extent of this relationship not being fully defined [11]. Dispersion of QT intervals (QTd) is defined as the difference between the maximum and minimum QT intervals measured in an electrocardiogram [12]. A study has found that an established association exists between abnormally increased QTd and a higher risk of arrhythmia [13]. This indicates that these electrocardiographic indicators have certain predictive value for the occurrence of MVA in patients with AMI.

Current research on predicting MVA in patients with AMI has focused primarily on single parameters such as HRV or QT QTV, often lacking external validation and model interpretability. This study integrated multi-dimensional Holter parameters with clinical indicators to construct a comprehensive prediction model and developed a visualized nomogram for intuitive risk quantification. Furthermore, SHAP analysis was supplemented based on internal validation (training/validation sets) and external validation in an independent cohort to elucidate the importance and impact direction of each feature. Therefore, this study provides a strong evidence-based medical basis for improving MVA prognosis in AMI patients.

Patients and methods

Study subjects

This was a retrospective cohort study that consecutively enrolled AMI patients admitted to the Tianyou Hospital Affiliated to Wuhan University of Science and Technology from January 2023 to June 2024. Inclusion criteria: (1) Diagnosis of AMI, defined as chest pain lasting >30 minutes, new ischemic ECG changes, and elevated cTnI (at least one value >99th percentile upper reference limit) [14]; (2) Hospital admission within 24 hours of symptom onset; (3) Age 18-80 years; (4) Undergoing 24-hour Holter electrocardiographic monitoring during hospitalization; (5) Availability of complete clinical data. Exclusion criteria: (1) Cardiogenic shock at admission; (2) Implanted cardiac pacemaker or defibrillator; (3) Complicated by severe liver and kidney insufficiency; (4) Previous history of MVA; (5) Missing clinical data. The sample size was calculated a priori using G*Power software. Setting an effect size=0.3, α=0.05, and a test power (1-β) of 0.9 yielded a minimum requirement of 183 subjects. To ensure adequate power, 279 participants were ultimately enrolled. According to the diagnostic criteria for MVA [15]: (1) Sustained VT: Wide QRS tachycardia with a ventricular rate ≥100 beats/min and duration ≥30 s; (2) VF: Irregular wide QRS rhythm with a ventricular rate ≥250 beats/min; (3) Hemodynamically unstable ventricular arrhythmia requiring electrical cardioversion or defibrillation. The subjects were divided into an MVA group (n=37) and a non-MVA group (n=242). We also enrolled 81 AMI patients between July 2024 and July 2025 as an independent external validation cohort to assess the performance of the developed clinical prediction model. This study was reviewed and approved by the Ethics Committee of Tianyou Hospital Affiliated to Wuhan University of Science and Technology (Approval No.: LL2025-07-28-01). As a retrospective study, all analyses were based on anonymized clinical data. Upon review, the Ethics Committee of Tianyou Hospital Affiliated to Wuhan University of Science and Technology granted a waiver of the requirement for obtaining written informed

consent from patients. Throughout the research process, we strictly adhered to the principles of the Declaration of Helsinki and implemented adequate measures, including the removal of all direct personal identifiers, to protect patient privacy and data security.

Collection of general data

Data on gender, age, hypertension status, and Killip classification were retrieved from the hospital information system. Cardiac function was assessed according to the Killip classification as follows [16]: I: No signs of heart failure with nearly normal cardiac output; II: Mild to moderate heart failure (rales <50% of lung fields), often with arrhythmia, etc.; III: Severe heart failure or acute pulmonary edema (rales >50% of of lung fields), with normal or decreased blood pressure; IV: Cardiogenic shock, systolic blood pressure <90 mmHg, blood pressure reduced to shock level.

Collection of laboratory indicators

Myocardial enzyme indicator (creatine kinase isoenzyme [CKMB]), troponin I (TnI) and biochemical indicators (blood urea nitrogen [BUN], creatinine [Cr], serum potassium ion level) of patients at admission were collected and sorted out. CKMB and TnI were detected using a Roche cobas e602 immunoanalyzer (Switzerland); BUN and Cr were analyzed using the VITROS 5600 Integrated Chemistry and Immunoassay System (USA). Serum potassium ion levels were measured using an HC-9884 electrolyte analyzer (China).

24-hour dynamic electrocardiogram indicators

A 24-hour dynamic electrocardiograph (Lepu SE-2012, China) was used, with customized electrode patches for each patient and a monitoring frequency set at 360 beats per minute. Continuous 24-hour monitoring and storage of cardiac electrophysiological signals were performed during patients' daily activities, and professional electrocardiogram analysis software (ECG Pro) was used for real-time analysis and interpretation of the signals.

Model construction

Based on the final multivariable logistic regression model, we included variables with $P<0.05$ to develop a visual nomogram for individualized risk prediction. Example using logistic regression: the regression equation: $\text{logit}(P)=\ln[P/(1-P)]=\beta_0+\beta_1X_1+\beta_2X_2+.....+\beta_KX_K$. Extract the absolute value of the regression coefficient (β) for all independent variables. Identify the maximum absolute coefficient (B). Assign the highest value level of this variable as 100 points (benchmark score). Calculate scores for other variables based on their "relative importance ratio" using the following formula: $(|\beta_i|/|\beta_{max}|)\times100$. Continuous Variables: Divide the score linearly based on the value range. For example, if Age has a coefficient B=0.5 and the maximum B=1.0, the maximum score for Age is 50 points. If the Age range is 20-80 years, 20 years corresponds to 0 points and 80 years corresponds to 50 points, with intermediate values calculated via linear interpolation. Categorical Variables: Allocate scores based on the B value differences across levels. For example, for "Tumor Stage", if Stage I has B=0.2, Stage II has B=0.3, and Stage III has B=0.5, they correspond to 20 points, 30 points, and 50 points, respectively (using the maximum B as the 100-point benchmark). The intercept represents the "baseline risk when all variables are zero". It is typically converted into a baseline score for the total points or directly integrated into the conversion from "total score to risk probability", and is not displayed as a separate item score.

To calculate the Total Score: Total Points=$S_1+S_2+.....S_K$. Sum the individual scores of all variables for a patient to obtain the Total Points. Use the inverse transformation of the regression equation to convert the total score back into a probability of occurrence. The formula is: $P=1/[1+e^{-(\beta_0+\beta_1X_1+\beta_2X_2+.....\beta_KX_K)}]$. The nomogram plots the correspondence between the "Total Score Scale" and the "Risk Probability Scale" (e.g., a total score of 100 corresponds to a 20% risk, and 200 corresponds to a 60% risk). The construction of the nomogram was implemented using the rms package in R 4.5.2 software.

Model validation and interpretation

In this study, we first presented the internal validation results, demonstrating that the model exhibits strong discriminative ability, good calibration, and clinical utility in the original cohort of 279 patients. Next, we provided the SHAP-based interpretation, thereby explaining the internal logic of the model's predictions. Finally, we presented the external validation results, confirming that the model maintains satisfac-

Table 1. Comparison of baseline characteristics of MVA and non-MVA groups

Variable	MCV n=37	non-MCV n=242	χ^2/t-value	*P*-value
Gender				
Male	20 (54.1)	129 (53.3)	0.007	0.932
Female	17 (45.9)	113 (46.7)		
Age (year)	65.30±9.94	65.47±9.81	0.103	0.918
Hypertension	21 (56.8)	125 (51.7)	0.335	0.563
CK-MB (U/L)	29.69±6.98	29.96±5.03	0.297	0.767
Cr (μmol/L)	43.85±7.55	45.25±8.15	0.982	0.327
BUN (mmol/L)	3.78±1.15	3.58±0.87	1.246	0.214
TnI (ng/L)	11.77±2.95	10.55±2.99	0.324	0.021
Killip			10.491	0.015
I	12 (32.4)	135 (55.8)		
II	10 (27.1)	56 (23.1)		
III	8 (21.6)	35 (14.5)		
IV	7 (18.9)	16 (6.6)		
Hypokalemia	11 (29.7)	44 (18.2)	2.704	0.1

CK-MB: Creatine Kinase MB Isoenzyme; Cr: Creatinine; BUN: Blood Urea Nitrogen; TnI: Troponin I.

Table 2. 24-hour electrocardiogram indicators in patients with or without MVA

Variable	MCV n=37	non-MCV n=242	t-value	*P*-value
SDNN (ms)	85.95±11.68	96.24±14.32	4.166	<0.001
RMSSD (ms)	2.54±1.02	2.82±0.75	2.023	0.044
PNN50 (%)	13.25±3.56	14.25±2.45	2.167	0.031
LF/HF	1.85±1.24	1.54±0.82	2.002	0.046
QTd (ms)	31.24±4.92	28.76±5.95	2.409	0.017
24h-QTV (ms)	24.82±2.09	23.86±2.20	2.498	0.013
night-QTV (ms)	23.84±2.19	22.86±1.24	3.983	<0.001
day-QTV (ms)	26.95±1.84	23.67±3.15	6.155	<0.001

SDNN: Standard Deviation of Normal-to-Normal Intervals; RMSSD: Root Mean Square of Successive Differences; PNN50: Percentage of Normal-to-Normal Intervals Exceeding 50 ms; LF/HF: Ratio of Low Frequency to High Frequency; QTd: QT Interval Dispersion; 24h-QTV: 24-Hour QT Interval Variability; night-QTV: Nighttime QT Interval Variability; day-QTV: Daytime QT Interval Variability.

tory performance in an independent cohort of 81 patients.

Statistical analysis

Data analysis was conducted with SPSS 27.0 and R 4.5.2. Normality-tested measured data were reported as mean ± standard deviation ($\bar{x}$ ±s) and compared by t-test; categorical variables as counts (percentages) and compared by χ^2 test. Following a 7:3 data split into training and validation sets, risk factors for MVA were identified by univariate (*P*<0.1 for inclusion) and subsequent multivariate logistic regression. Model performance was assessed by discrimination (ROC-AUC), calibration (calibration plot, Hosmer-Lemeshow test), and clinical utility (DCA), with model interpretation provided by SHAP. Statistical significance was set at *P*<0.05.

Results

Comparative analysis of baseline characteristics in AMI patients with and without MVA

Of the 279 AMI patients enrolled in the study, 149 (53.4%) were male and 130 (46.6%) were female. The overall mean age was 65.45±9.81 years. Hypertension was present in 146 patients (52.3%). MVA occurred in 37 patients, with an incidence rate of 13.3%. Patients with MVA had significantly higher TnI levels and Killip classification compared to those without MVA (*P*<0.05). The two groups were comparable with regard to age, gender, and other assessed laboratory values (**Table 1**).

24-hour electrocardiogram indicators in patients with or without MVA

Patients with MVA had significantly lower SDNN, RMSSD, and PNN50 (*P*<0.05), and higher LF/HF ratio, QTd, 24h-QTV, night-QTV, and day-QTV compared to those without MVA (*P*<0.05) (**Table 2**).

Univariate analysis of early MVA occurrence in AMI patients

To explore the risk factors associated with MVA occurrence, univariate logistic regression analysis was performed. The results showed that patients with Hypokalemia, lower SDNN

Table 3. Univariate analysis of early MVA occurrence in AMI patients

Variable	β	S.E.	Z	*P*	*OR* (95% *CI*)
Gender					
Male					1.00 (Reference)
Female	-0.49	0.47	-1.02	0.306	0.62 (0.24-1.56)
Hypertension					
No					1.00 (Reference)
Yes	0.03	0.46	0.06	0.955	1.03 (0.41-2.54)
RMSSD (ms)	-0.93	0.34	-2.78	0.005	0.39 (0.20-0.76)
Killip					
I					1.00 (Reference)
II	0.82	0.61	1.35	0.177	2.27 (0.69-7.45)
III	1.05	0.64	1.64	0.101	2.87 (0.81-10.14)
IV	1.56	0.71	2.2	0.028	4.77 (1.19-19.18)
Hypokalemia					
No					1.00 (Reference)
Yes	1	0.49	2.05	0.040	2.73 (1.04-7.14)
Age	-0.01	0.02	-0.26	0.796	0.99 (0.95-1.04)
SDNN (ms)	-0.07	0.02	-3.37	<.001	0.93 (0.90-0.97)
PNN50 (%)	-0.12	0.09	-1.32	0.188	0.89 (0.75-1.06)
LF/HF	0.2	0.26	0.74	0.458	1.22 (0.73-2.03)
QTd (ms)	0.07	0.04	1.75	0.081	1.07 (0.99-1.15)
24h-QTV (ms)	0.3	0.11	2.69	0.007	1.35 (1.09-1.68)
Night-QTV (ms)	0.5	0.16	3.17	0.002	1.64 (1.21-2.24)
Day-QTV (ms)	0.35	0.09	3.89	<.001	1.43 (1.19-1.70)
CK-MB (U/L)	-0.05	0.04	-1.08	0.279	0.95 (0.88-1.04)
Cr (μmol/L)	-0.01	0.03	-0.21	0.833	0.99 (0.94-1.05)
BUN (mmol/L)	-0.11	0.25	-0.46	0.644	0.89 (0.55-1.45)
TnI (ng/L)	0.16	0.08	2.01	0.045	1.17 (1.01-1.37)

OR: Odds Ratio; *CI*: Confidence Interval; SDNN: Standard Deviation of Normal-to-Normal Intervals; RMSSD: Root Mean Square of Successive Differences; PNN50: Percentage of Normal-to-Normal Intervals Exceeding 50 ms; LF/HF: Ratio of Low Frequency to High Frequency; QTd: QT Interval Dispersion; 24h-QTV: 24-Hour QT Interval Variability; night-QTV: Nighttime QT Interval Variability; day-QTV: Daytime QT Interval Variability; CK-MB: Creatine Kinase MB Isoenzyme; Cr: Creatinine; BUN: Blood Urea Nitrogen; TnI: Troponin I.

and RMSSD as well as those with higher QTd, 24h-QTV, night-QTV, day-QTV, TnI levels, and Killip classification, had a higher risk of MVA (P<0.1) (**Table 3**).

Multivariate analysis of early MVA occurrence in AMI patients

We performed a multivariable logistic regression to identify factors associated with MVA, defined as variables that showed an association with P<0.1 in univariate analyses (**Table 3**) were advanced to this final model. The results indicated that low SDNN and elevated levels of 24h-QTV, night-QTV, day-QTV, and TnI, along with a higher Killip classification, were independent risk factors for MVA (P<0.05) (**Table 4**).

Construction of a risk prediction model for early MVA occurrence in AMI patients

A nomogram model for predicting the risk of MVA in AMI patients was constructed based on the risk factors screened by multivariate analysis (**Figure 1**). For each risk factor in AMI patients with MVA, projecting upward to the small scale can yield the score for each item of the patient. The total score is obtained by summing the scores of each item; the higher the total score, the greater the risk of early MVA in the patient.

Predictive performance with and without 24 h-Holter parameters

Predictive models were established by including and excluding the 24h-Holter parameters from the predictors in **Table 4**, respectively, to evaluate their performance. The results showed that incorporating the 24h-Holter parameters (AUC= 0.95) significantly improved the predictive ability compared to excluding them (AUC=0.72) (**Figure 2**).

Internal validation of the prediction model

The prediction model demonstrated strong validation performance across multiple metrics. It exhibited high discriminatory power, as evidenced by its consistent discriminatory power across both training (AUC=0.95; 95% *CI*: 0.82-0.98) and validation (AUC=0.82; 95% *CI*: 0.68-0.96) sets (**Figure 3A**). The model exhibited superb calibration, with curves for both the training and validation sets adhering closely to the ideal line. The non-significant Hosmer-Lemeshow test results (P=0.781 and P=0.347, respectively) statistically confirmed this strong

Table 4. Multivariate analysis of early MVA occurrence in AMI patients

Variable	β	S.E.	Z	*P*	*OR* (95% *CI*)
Killip					
1					1.000 (Reference)
2	1.352	0.848	1.595	0.111	3.865 (0.734-20.359)
3	0.978	0.950	1.03	0.303	2.660 (0.413-17.116)
4	2.265	1.060	2.138	0.033	9.635 (1.208-76.869)
SDNN (ms)	-0.115	0.034	-3.335	<.001	0.891 (0.833-0.954)
24h-QTV (ms)	0.442	0.179	2.461	0.014	1.555 (1.094-2.211)
Night-Qtv (ms)	0.722	0.253	2.852	0.004	2.059 (1.254-3.383)
Day-Qtv (ms)	0.473	0.126	3.763	<.001	1.604 (1.254-2.052)
TnI (ng/L)	0.278	0.120	2.325	0.02	1.320 (1.045-1.669)

OR: Odds Ratio; *CI*: Confidence Interval; SDNN: Standard Deviation of Normal-to-Normal Intervals; 24h-QTV: 24-Hour QT Interval Variability; night-QTV: Nighttime QT Interval Variability; day-QTV: Daytime QT Interval Variability; TnI: Troponin I.

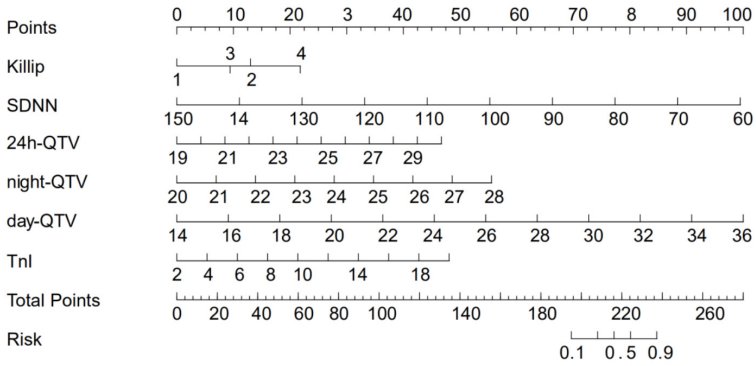

Figure 1. A nomogram model for predicting the risk of MVA in AMI patients. SDNN: Standard Deviation of Normal-to-Normal Intervals; RMSSD: Root Mean Square of Successive Differences; QTd: QT Interval Dispersion; 24h-QTV: 24-Hour QT Interval Variability; night-QTV: Nighttime QT Interval Variability; day-QTV: Daytime QT Interval Variability; TnI: Troponin I.

agreement (**Figure 3B**, **3C**). Additionally, decision curve analysis attested to its clinical utility, revealing a robust net benefit throughout the 0.1-0.8 probability threshold range in both sets (**Figure 3D**, **3E**).

Model interpretation through SHAP

The SHAP method was applied to interpret the model's predictions (**Figure 4**). SDNN, day-QTV, and 24h-QTV were identified as the top three risk factors based on mean absolute SHAP values (**Figure 4A**). The directional influence of each feature is displayed in **Figure 4B**, where the distribution of SHAP values shows that a decreased SDNN and elevated values of 24h-QTV, night-QTV, day-QTV, Killip classification, and TnI all contributed to a higher risk of MVA.

External validation of the predictive model

Among the external validation cohort, 19 patients were classified as MVA and 62 as non-MVA. We further validated the prediction model in the independent external cohort. The results revealed an AUC of 0.74 (95% CI: 0.62-0.87) for the external cohort, which was slightly lower than the AUC obtained from the training set (0.95 [95% CI: 0.82-0.98]) but still indicated satisfactory discriminative ability (**Figure 5A**). The calibration curve showed a good agreement between predicted and observed risks across all risk quantiles (Hosmer-Lemeshow test, *P*>0.05) (**Figure 5B**). Additionally, decision curve analysis confirmed the model's clinical utility, with significant net benefits observed when the probability threshold was set between 0.1 and 0.4 (**Figure 5C**).

Discussion

Acute myocardial infarction (AMI) is the most dangerous clinical type of acute coronary syndrome. Its main pathologic basis is acute and persistent occlusion or severe stenosis of the coronary arteries, which leads to a sharp reduction or interruption of blood flow in myocardial tissue, and then causes myocardial cell ischemia, injury, and necrosis. Characterized by acute onset and critical condition, this disease is one of the major cardiovascular emergencies causing disability and death worldwide [17]. AMI is prone to be complicated by ventricular or supraventricular arrhythmias, among which MVA is the most common. Driven by the clinical imperative of MVA, a well-established major contributor to mortality in AMI [4], this study leveraged data from 279 patients to construct and validate a predictive model based on clinical and 24-hour dynamic electrocardiogram parameters. The results showed that MVA occurred in 37

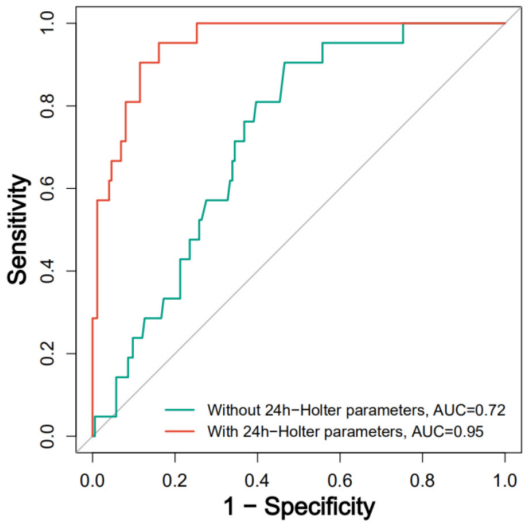

Figure 2. The ROC with and without 24h-Holter parameters. The green line represents the model including factors (TnI and Killip classification), and the red line represents the model including factors (TnI, Killip classification, SDNN, 24h-QTV, night-QTV and day-QTV).

out of 279 AMI patients, with an incidence rate of 13.3%. Low levels of SDNN as well as high levels of 24h-QTV, night-QTV, day-QTV, TnI and Killip classification, were independent risk factors for MVA. The prediction model exhibited strong overall performance, as evidenced by outstanding discriminatory power (AUC=0.95), satisfactory calibration (Hosmer-Lemeshow test, *P*=0.781), and promising clinical utility.

In recent years, several studies have focused on screening predictors of MVA in AMI patients. A study published in 2024 enrolled 1493 AMI patients and found that a set of factors - including Killip classification ≥3, STEMI, LVEF<50%, and others - were independent predictors. The AUC of the constructed model was 0.815 [18]. The incidence of ventricular arrhythmia in this study was 4.7%, which was lower than 13.3% in our study, possibly due to differences in the study population. In a dedicated cohort of 285 ST-segment elevation myocardial infarction (STEMI) patients (2023), age, magnitude of ST-segment elevation, and symptom-to-balloon time were established as independent risk factors for MVA [19]. Compared to our study, this study focused on STEMI patients and did not include dynamic electrocardiogram parameters. Regarding dynamic electrocardiogram parameters, a study by Curione et al. explored the guiding significance of 24-hour dynamic electrocardiogram parameters for the occurrence of MVA in AMI patients. It was found that parameters such as SDNN, SDNN-index, and PNN50 in the MVA group were lower than those in the non-MVA group, which is conducive to predicting the occurrence of MVA after AMI [20]. This is consistent with the findings of this study.

Logistic regression analysis in this study showed that the risk factors for MVA in AMI patients were low levels of SDNN as well as high levels of 24h-QTV, night-QTV, day-QTV, TnI, and Killip classification. Severe pain and anxiety caused by AMI can activate the sympathetic nervous system, release a large amount of catecholamines, leading to calcium overload in myocardial cells, increased myocardial excitability, and induced MVA. Therefore, the higher the Killip classification, the stronger the sympathetic nerve activation, the more severe the impairment of the patient's cardiac function, the higher the probability of ventricular electrical remodeling and increased myocardial cell charge, and the higher the risk of arrhythmia [21, 22]. Wang et al.'s study found that patients with Killip classification ≥ Grade III had a significantly increased risk of MVA (*OR*=5.034, *P*=0.005) [23], which is similar to the results of this study. Elevated TnI levels reflect extensive myocardial injury, exacerbate electrical instability, and provide a pathologic basis for MVA. Persistently high TnI levels may also indicate severe impairment of cardiac function, such as decreased cardiac contractility and ventricular remodeling [24, 25]. HRV has been recognized as a predictor of arrhythmias [26]. SDNN reflect parasympathetic nerve activity in HRV [27], which is indicative of vagal nerve activity [28], while LF/HF is an indicator representing sympathetic nerve activity [29]. Increased sympathetic nervous system activity can trigger arrhythmias, while enhanced parasympathetic nervous system activity appears to inhibit such electrical activity [30, 31]. Sufficient evidence has shown that the average QT interval of the electrocardiogram is regulated by the autonomic nervous system [32]. Increased sympathetic nerve activity can elevate QTV, which may be caused by the spatial dispersion of the action potential duration of ventricular

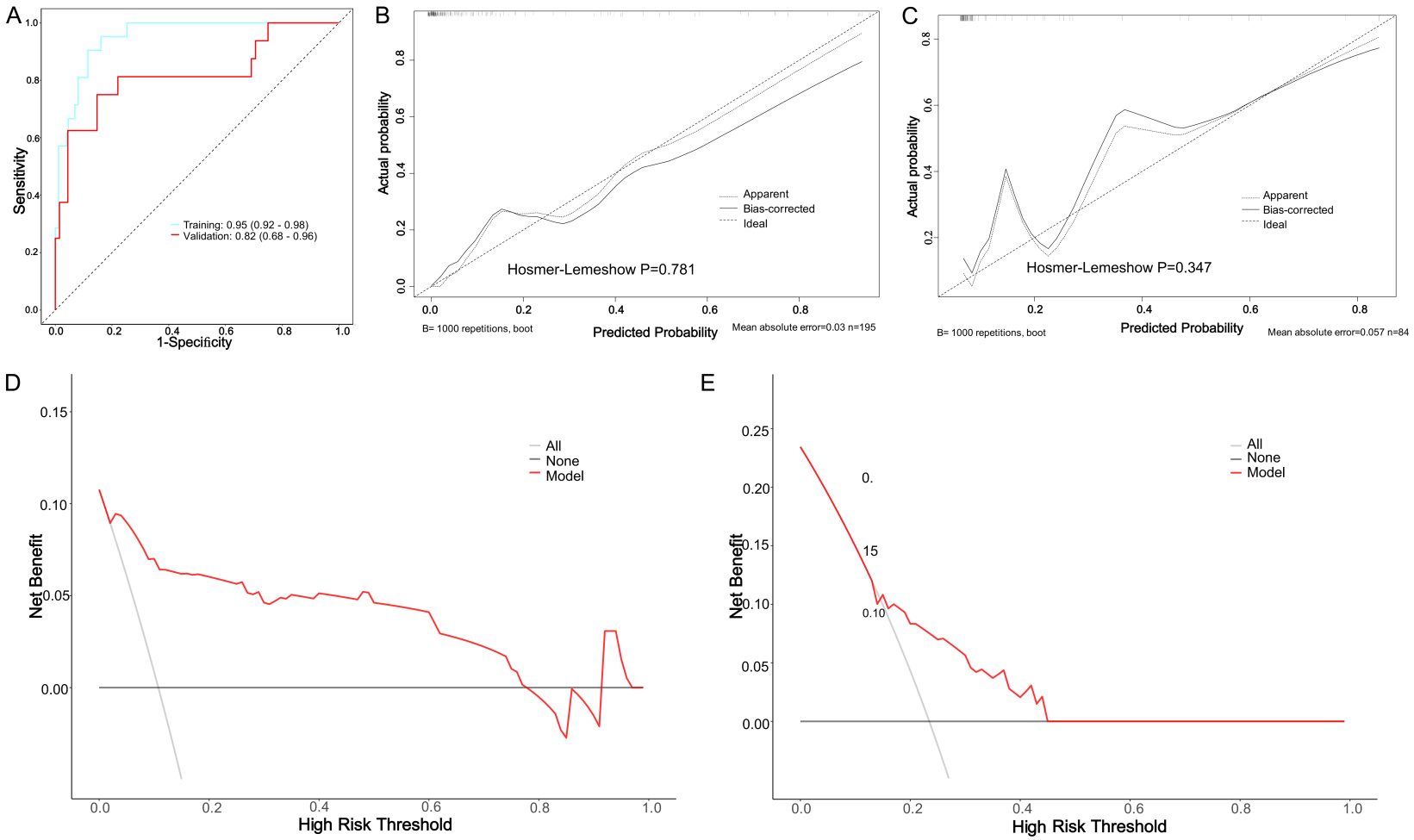

Figure 3. Validation of the Prediction Model. A. The ROC curve evaluating discrimination. Light green indicates the training set; red indicates the validation set. B, C. The calibration plots present the agreement between predicted and observed outcomes in the training and validation sets, respectively. D, E. Decision curve analysis depicts the net clinical benefit of the model in the training and validation sets, respectively.

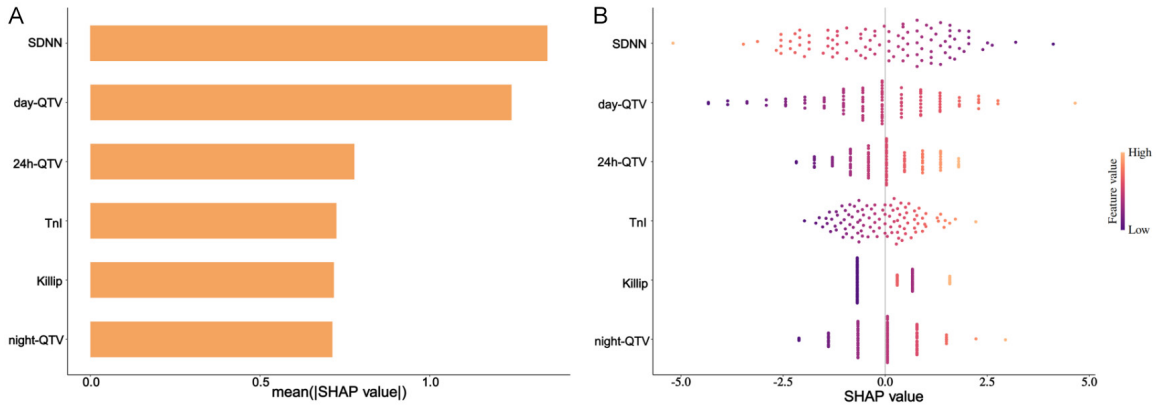

Figure 4. Model interpretation using SHAP. A. The importance of each feature as determined by mean absolute SHAP values. B. Effect of each feature on the model output. Each dot represents an individual patient; its horizontal position corresponds to the SHAP value (indicating the feature's effect on the prediction), and the color corresponds to the original feature value (orange for high, purple for low).

myocytes resulting from the inhomogeneity of β-adrenergic receptors and the dendritic variation of sympathetic nerves [33]. In addition, AMI can lead to myocardial cell necrosis and inflammatory response, releasing a large number of inflammatory mediators such as C-reactive protein and interleukin-6 [34]. These inflammatory mediators not only aggravate myocardial injury but also affect the electrophysiologic characteristics of the myocardium, increasing the susceptibility to arrhythmias.

By constructing a prediction model, this study provides a basis for identifying high-risk patients with MVA, which has certain predictive value and clinical applicability. This tool can guide clinical efforts in early identification of high-risk AMI patients, enabling prompt monitoring, rescue, and referral. Completing 24-hour dynamic electrocardiogram monitoring and risk assessment within 24 hours after AMI patients are admitted to the hospital can help clinicians quickly formulate diagnosis and treatment plans. The high sensitivity of the model reduces the missed diagnosis rate of high-risk patients, and the high specificity reduces unnecessary interventions.

It is important to acknowledge the limitations of this study. Primarily, the single-center design may have selection bias. All subjects were from the same medical center and may not represent all AMI patient populations. Second, the study adopted a retrospective design, which could not fully control for confounding factors. This study only used 24-hour dynamic electrocardiogram data and did not integrate other types of electrocardiogram information, such as 12-lead electrocardiogram, which may have reduced the accuracy of arrhythmia prediction [35]. Finally, the external validation cohort of this study had a relatively small sample size, which thus limited the accurate assessment of model performance, particularly calibration, and increased the uncertainty in the estimation of results. Therefore, the model of this study may not be applicable for long-term risk prediction.

Conclusion

This study successfully constructed and validated an MVA risk prediction model for AMI patients based on 24-hour dynamic electrocardiogram parameters. The study found that low levels of SDNN and PNN50, as well as high levels of LF/HF ratio, QTd, night-QTV, day-QTV, TnI, and Killip classification, were independent predictors of in-hospital MVA in AMI patients. The prediction model performed well in terms of discrimination, calibration, and clinical utility.

Disclosure of conflict of interest

None.

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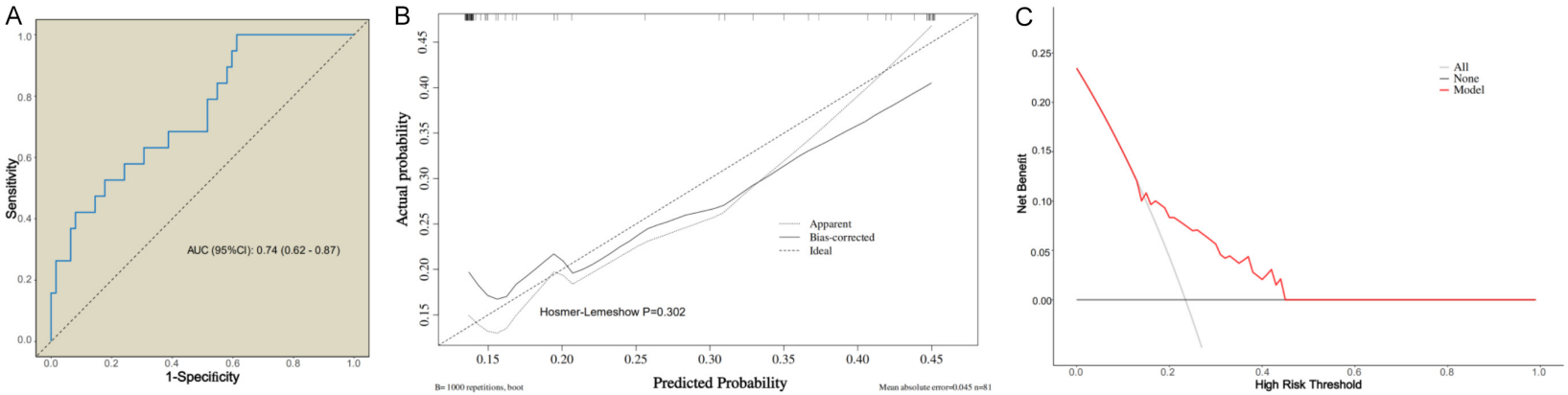

Figure 5. External validation of the predictive model. A. The ROC curve evaluating discrimination. B. The calibration plots present the agreement between predicted and observed outcomes. C. Decision curve analysis depicts the net clinical benefit of the model.

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