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ORIGINAL RESEARCH

Influencing Factors of New-Onset Atrial Fibrillation in Sepsis Patients and Construction of a Nomogram Prediction Model: Based on LASSO Regression

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Objective: To investigate the influencing factors of new-onset atrial fibrillation (AF) in patients with sepsis and to construct a nomogram prediction model.

Methods: A retrospective analysis of 245 sepsis patients admitted to our hospital from March 2021 to March 2024 was used as the training set. An additional 107 sepsis patients admitted to our hospital from April 2024 to April 2025 were included as the validation set. The training and validation sets were divided into an AF group and a non-AF group based on the occurrence of new-onset AF.

Results: In the training set, there were significant differences between the two groups in terms of age, mechanical ventilation, APACHE II score, acute kidney injury, metabolic disorders, theophylline medication, TNF-α, E/e′, and NT-proBNP ($P < 0.05$). LASSO regression analysis was used to screen for 7 predictive factors. Logistic regression analysis identified age, mechanical ventilation, APACHE II score, theophylline medication, TNF-α, E/e′, and NT-proBNP as risk factors for new-onset AF in sepsis patients ($P < 0.05$). The area under the curve (AUC) of the ROC curve for the training set was 0.869, and the Hosmer-Lemeshow test yielded $\chi2$=7.346 (P=0.713). The decision curve analysis (DCA) showed that the model has high clinical application value when the threshold probability is between 0.10 and 0.89. For external validation, the AUC of the ROC curve was 0.875, the Hosmer-Lemeshow test yielded $\chi2$=6.992 (P=0.703), and the DCA curve showed that the model has high clinical application value when the threshold probability is between 0.12 and 0.83.

Conclusion: Age, mechanical ventilation, APACHE II score, theophylline medication, TNF-α, E/e′, and NT-proBNP are influencing factors for new-onset AF in patients with sepsis. The nomogram prediction model constructed on the basis of these factors has good clinical applicability.

Keywords: sepsis, atrial fibrillation, influencing factors, nomogram model

Introduction

Sepsis is a life-threatening systemic inflammatory response syndrome caused by infection. After pyogenic bacteria invade the bloodstream, they multiply extensively, causing a relatively intense inflammatory response in the patient, which can increase the risk of death in severe cases.[1] Studies have shown that approximately 18 million people worldwide develop sepsis each year, with an annual increase of 1.5%–8%, and a mortality rate of about 30–70%.[2] The heart, as the core of the circulatory system, is a key target organ affected during the progression of sepsis, with nearly half of patients experiencing varying degrees of myocardial injury.[3] New-onset atrial fibrillation (AF) in sepsis patients is a serious complication that induces abnormal cardiac function. Studies have shown that new-onset AF occurs in about 40% of sepsis patients, which leads to unstable hemodynamics, increases the cost of medical treatment and hospitalization, and severely affects the patient's prognosis.[4,5] Therefore, early clinical identification of the influencing

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factors of new-onset AF can effectively improve patient prognosis. The nomogram model, as a visual graphical prediction tool, is a tool for clinical prognosis assessment. It is simple and fast to operate, can predict the risk value of a certain event, provides a reference for stratified management, and can assist clinicians in formulating corresponding measures to reduce the risk of morbidity.[6,7] Studies have shown that a nomogram for individualized prediction of delirium in patients with sepsis was constructed, identifying four independent predictive factors. The calibration curves of the predictive model were close to the ideal curve in both the training and validation cohorts, and decision curve analysis (DCA) indicated that the nomogram had clinical value. Its performance and clinical utility were satisfactory, helping clinicians to identify patients at risk of delirium in a timely manner, implement early interventions, and improve neurological outcomes.[6] Currently, there are few reports on new-onset AF in sepsis patients using nomogram prediction models. Therefore, this study aims to investigate the influencing factors of new-onset AF in sepsis patients and to construct a nomogram prediction model.

Materials and Methods

General Information

A retrospective collection of 245 sepsis patients admitted to our hospital from March 2021 to March 2024 was used as the training set. An additional 107 sepsis patients admitted to our hospital from April 2024 to April 2025 were collected as the validation set (for external validation). The training and validation sets were divided into an AF group (no prior history of atrial fibrillation, transferred to the ICU after the first detection of AF, or developed AF after entering the ICU) and a non-AF group. The case collection flowchart is shown in Figure 1. Inclusion criteria: (1) met the diagnostic criteria for sepsis;[8] (2) met the diagnostic criteria for AF,[9] with a duration >1 min, disappearance of P waves on the electrocardiogram, and irregular R-R intervals with normal atrioventricular conduction; (3) survival time >24 h after admission; (4) age >18 years; (5) complete clinical data. Exclusion criteria: (1) congenital heart disease; (2) pregnancy or lactation; (3) chronic or permanent atrial fibrillation; (4) immune dysfunction; (5) cognitive dysfunction. This study was approved by our hospital's ethics committee.

Clinical Data Collection

Data were collected from the electronic systems of hospitalized patients in our hospital, mainly including: age, gender, smoking history, alcohol consumption history, chronic obstructive pulmonary disease (COPD), heart failure, ischemic heart disease, cerebrovascular disease, malignant tumors, acute kidney injury, new-onset heart failure, metabolic disorders, coagulation dysfunction, respiratory failure, liver dysfunction, mechanical ventilation, APACHE II score, continuous renal replacement therapy (CRRT), cedilanide, anticholinergic drugs, theophylline drugs, amiodarone, white blood cell count (WBC), hemoglobin (HGB), fibrinogen (FIB), interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), procalcitonin (PCT), creatine kinase-MB (CK-MB), cardiac troponin I (cTnI), potassium ion (K+), left ventricular ejection fraction (LVEF), E/e′, and N-terminal pro-B-type natriuretic peptide (NT-proBNP).

Statistical Analysis

SPSS 25.0 was used to process the data. Measurement data were analyzed using the *t*-test, expressed as (mean ± SD), while count data were analyzed using the χ^2 test, expressed as n (%). LASSO regression was used to screen variables. Logistic regression analysis was used to identify the influencing factors of new-onset AF in sepsis patients in the training set. R software was used to construct the nomogram prediction model. Bootstrap was used for internal validation (1000 resamples). The ROC and calibration curves were used to evaluate the discrimination and consistency of the nomogram model. DCA was used to assess the clinical application value. A P-value <0.05 was considered statistically significant.

Results

Comparison of Clinical Data Between AF and Non-AF Groups in the Training Set

Among the 245 patients in the training set, 72 developed AF, with an incidence rate of 29.39%. There were significant differences between the two groups in terms of age, mechanical ventilation, APACHE II score, acute kidney injury,

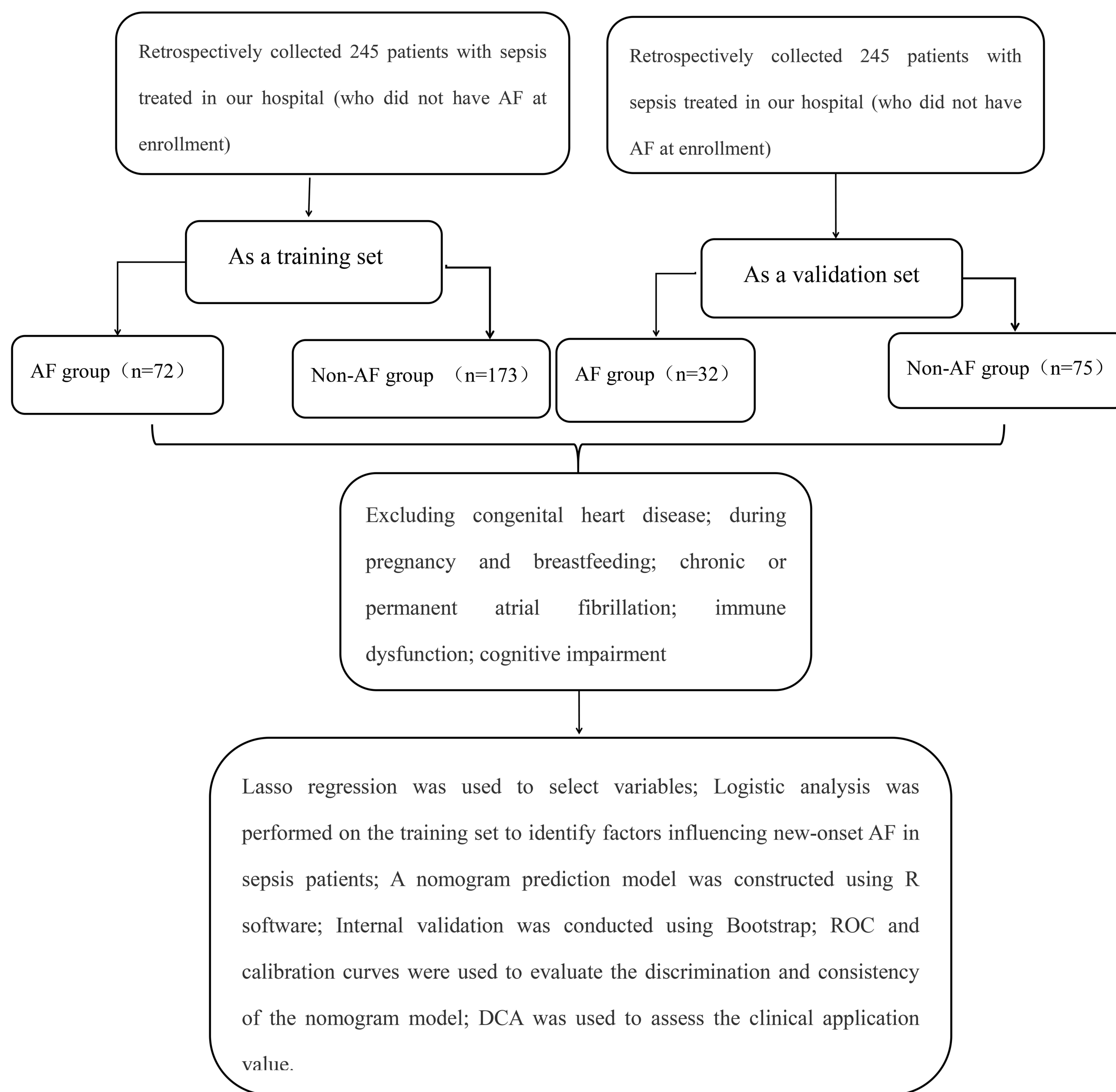

Figure 1 Case flow collection diagram.

metabolic disorders, theophylline medication, TNF-α, E/e′, and NT-proBNP ($P < 0.05$). There were no significant differences in other clinical data ($P>0.05$). See Table 1.

LASSO Regression Analysis

In the training set, with the occurrence of new-onset AF in sepsis patients as the dependent variable (Yes=1, No=0), and the aforementioned differing indicators—mechanical ventilation (Yes=1, No=0), acute kidney injury (Yes=1, No=0), metabolic disorders (Yes=1, No=0), theophylline medication (Yes=1, No=0), age, APACHE II score, TNF-α, E/e′, and NT-proBNP (all as measured values)—as independent variables, LASSO analysis was performed using R software (with 10-fold cross-validation to obtain the optimal λ value). The results showed that when the penalty coefficient

Table 1 Comparison of Clinical Data Between the AF Group and the Non-AF Group in the Training Set

Factor	AF Group (n=72)	Non-AF Group (n=173)	t/χ^2	*P*
Age (years old)	69.35±5.81	61.10±5.64	10.338	<0.001
Gender			0.046	0.829
Male	46 (63.89)	108 (62.43)		
Female	26 (36.11)	65 (37.57)		
History of smoking			0.097	0.756
Yes	29 (40.28)	66 (38.15)		
No	43 (59.73)	107 (61.85)		
History of alcohol consumption			0.032	0.857
Yes	25 (34.72)	58 (33.53)		
No	47 (65.28)	115 (66.47)		
COPD			0.355	0.551
Yes	24 (33.33)	51 (29.48)		
No	48 (66.67)	122 (70.52)		
Heart failure			0.062	0.804
Yes	10 (13.89)	22 (12.72)		
No	62 (86.11)	151 (87.28)		
Hypertension			0.002	0.968
Yes	31 (43.06)	74 (42.77)		
No	41 (56.94)	99 (57.23)		
Diabetes			0.474	0.491
Yes	24 (33.33)	50 (28.90)		
No	48 (66.67)	123 (71.10)		
Ischemic heart disease			0.698	0.404
Yes	26 (36.11)	53 (30.64)		
No	46 (63.89)	120 (69.36)		
Cerebrovascular disease			0.044	0.833
Yes	15 (20.83)	34 (19.65)		
No	57 (79.17)	139 (80.35)		
Malignancy			0.543	0.461
Yes	6 (8.33)	10 (5.78)		
No	66 (91.67)	163 (94.22)		
Acute kidney injury			5.744	0.017
Yes	35 (48.61)	56 (32.37)		
No	37 (51.39)	117 (67.63)		
New-onset heart failure			2.225	0.136
Yes	18 (25.00)	29 (16.76)		
No	54 (75.00)	144 (83.24)		
Metabolic disorders			5.411	0.020
Yes	36 (50.00)	59 (34.10)		
No	36 (50.00)	114 (65.90)		
Coagulopathy			1.593	0.207
Yes	18 (25.00)	31 (17.92)		
No	54 (75.00)	142 (82.08)		
Respiratory failure			3.436	0.064
Yes	38 (52.78)	69 (39.88)		
No	34 (47.22)	104 (60.12)		
Liver dysfunction			0.894	0.344
Yes	12 (16.67)	21 (12.14)		
No	60 (83.33)	152 (87.86)		

(*Continued*)

Table 1 (Continued).

Factor	AF Group (n=72)	Non-AF Group (n=173)	t/χ^2	*P*
Mechanical ventilation			12.657	<0.001
Yes	39 (54.17)	52 (30.06)		
No	33 (45.83)	121 (69.94)		
APACHE IIscore (points)	25.26±3.41	23.41±2.74	4.469	<0.001
CRRT treatment			2.612	0.106
Yes	15 (20.83)	22 (12.72)		
No	57 (79.17)	151 (87.28)		
Cediran	14 (19.44)	32 (18.50)	0.030	0.863
Anticholinergic drugs	13 (18.06)	30 (17.34)	0.018	0.893
Theophylline	38 (52.78)	34 (32.37)	8.954	0.003
Amiodarone	9 (12.50)	16 (9.25)	0.587	0.444
WBC (×109/L)	18.96±5.02	18.02±5.01	1.337	0.182
HGB (g/L)	108.65±20.41	109.24±21.04	0.202	0.840
FIB (mg/dL)	4.92±1.03	4.83±1.08	0.602	0.548
IL-6 (ng/L)	2531.26±156.24	2498.62±146.28	1.559	0.120
TNF-α	132.54±21.02	89.65±16.24	17.210	<0.001
PCT (μg/L)	32.05±6.54	30.68±6.41	1.515	0.131
CK-MB (μg/L)	23.64±6.25	22.05±6.12	1.841	0.067
cTnI (μg/L)	5.94±1.21	6.13±1.24	1.100	0.272
K^+ (mmol/L)	4.31±0.97	4.26±0.98	0.365	0.716
LVEF (%)	51.43±10.32	51.67±10.26	0.167	0.868
E/e′	15.89±1.24	13.14±1.31	15.201	<0.001
NT-proBNP (ng/L)	3046.28±165.23	2491.68±156.26	24.881	<0.001

λ=0.03671194, the model performance was optimal with the fewest influencing factors. Ultimately, 7 predictive factors were selected: age, mechanical ventilation, APACHE II score, theophylline medication, TNF-α, E/e′, and NT-proBNP. See Figure 2.

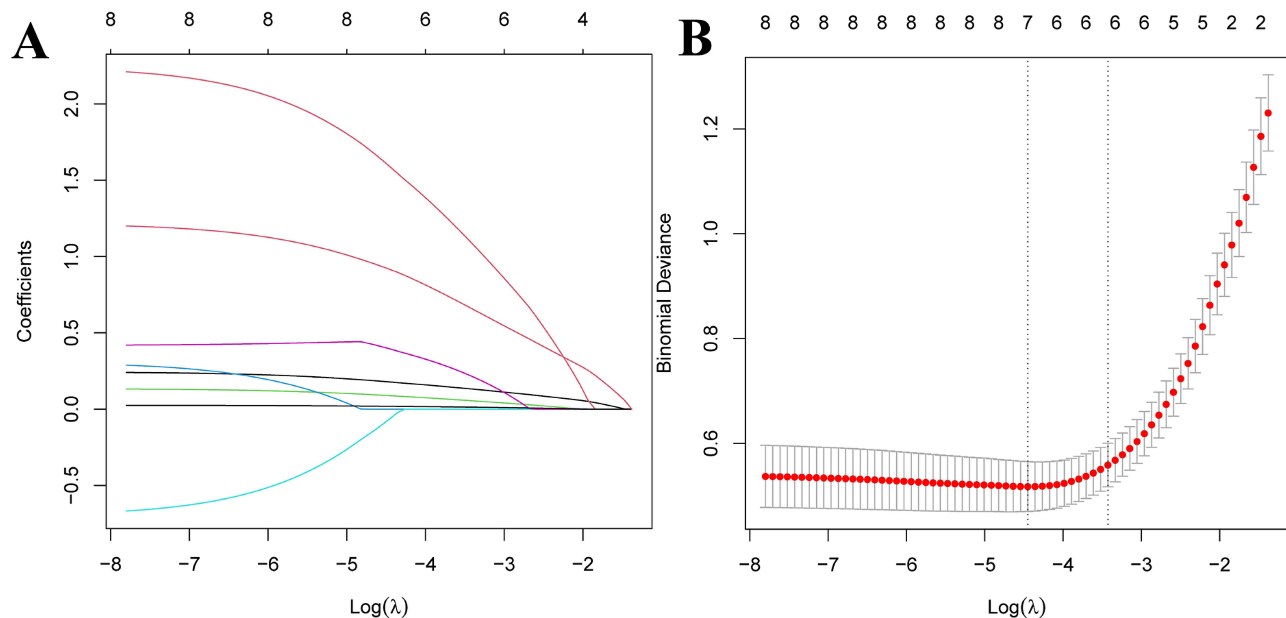

Figure 2 LASSO Regression Analysis LASSO. (**A**) Relationship plot of regression coefficients. (**B**) 10-fold cross-validation results of LASSO regression.

Table 2 Multivariate Analysis of New-Onset AF in Patients with Sepsis

Variable	B value	SE value	Wald χ^2 value	P value	OR value	95% CI
Age	0.199	0.053	13.901	<0.001	1.220	1.099~1.355
Mechanical ventilation	2.056	0.641	10.280	0.001	7.815	2.224~27.464
APACHE II score	0.111	0.039	8.092	0.004	1.117	1.035~1.206
Theophylline	1.278	0.345	13.718	<0.001	3.590	1.825~7.061
TNF-α	0.023	0.010	6.021	0.014	1.024	1.005~1.043
E/e′	1.329	0.267	24.698	<0.001	3.778	2.237~6.381
NT-proBNP	1.103	0.326	11.453	0.001	3.014	1.591~5.710
Constant	−44.700	7.191	38.644	<0.001	<0.001	–

Multivariate Analysis of New-Onset AF in Sepsis Patients

The 7 independent variables selected by LASSO regression were included in a Logistic regression analysis (forward stepwise method). The results showed that age, mechanical ventilation, APACHE II score, theophylline medication, TNF-α, E/e′, and NT-proBNP were risk factors for new-onset AF in sepsis patients ($P < 0.05$). See Table 2.

Construction of the Nomogram Model for New-Onset AF in Sepsis Patients

A nomogram model was constructed using the risk factors identified above, with $P=e^x/(1+e^x)$, where x = −44.700 + 0.199 × Age + 2.056 × Mechanical Ventilation + 0.111 × APACHE II Score + 1.278 × Theophylline Medication + 0.023 × TNF-α + 1.329 × E/e′ + 1.103 × NT-proBNP. In this model, E/e′ was identified as the strongest predictive factor. See Figure 3.

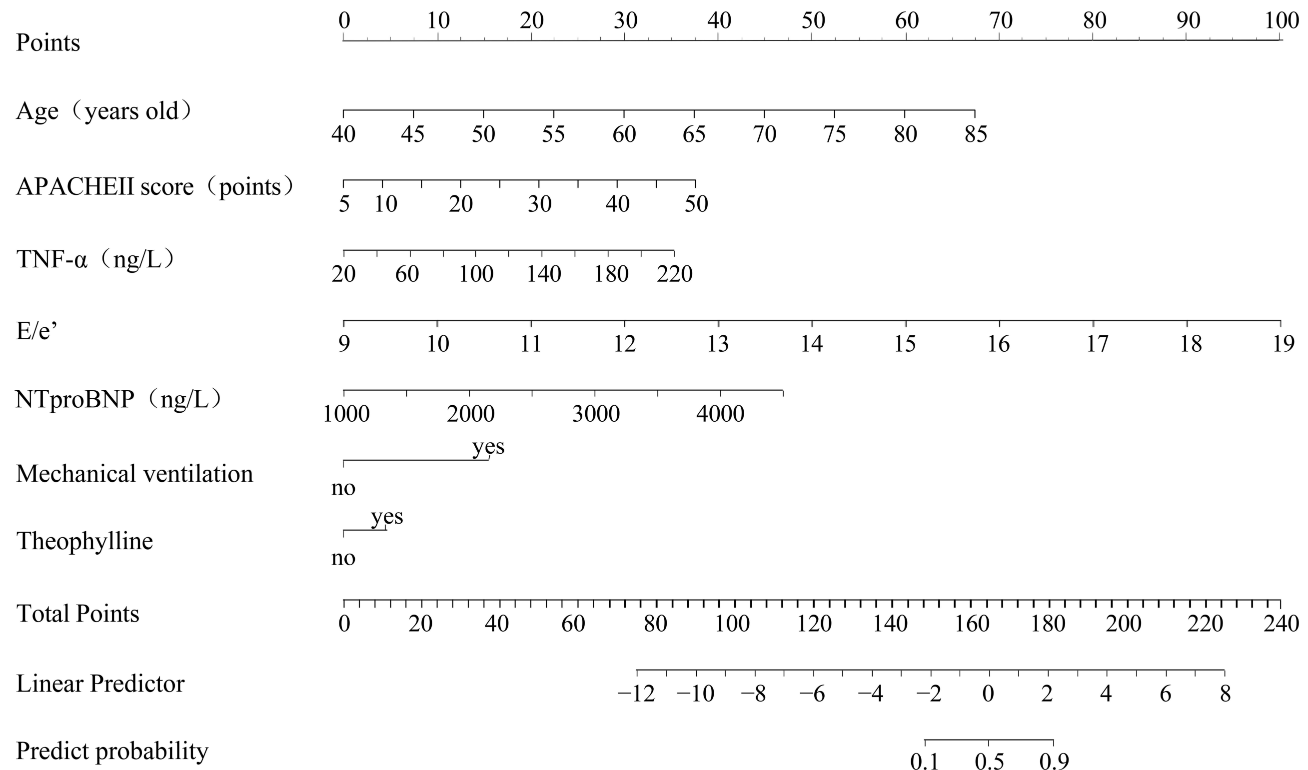

Figure 3 Construction of a nomogram model for new-onset AF in patients with sepsis.

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Internal Validation of the Nomogram Model for New-Onset AF in Sepsis Patients

The ROC curve was used to assess discrimination, yielding an AUC of 0.869 (95% CI = 0.823–0.915). The Hosmer-Lemeshow (H-L) test was used to validate consistency, yielding χ2=7.346 (P=0.713). See Figure 4.

Internal Validation of the Nomogram Model's DCA Curve in the Training Set

The results of the decision curve analysis (DCA) showed that when the threshold probability was between 0.10 and 0.89, the model had high clinical application value for assessing new-onset AF in sepsis patients. See Figure 5.

Comparison of Clinical Data Between AF and Non-AF Groups in the Validation Set

Among the 107 patients in the validation set, 32 developed AF, with an incidence rate of 29.91%. There were significant differences between the two groups in age, mechanical ventilation, APACHE II score, theophylline medication, TNF-α, E/e′, and NT-proBNP ($P < 0.05$). There were no significant differences in other variables ($P>0.05$). See Table 3.

External Validation of the Nomogram Model for New-Onset AF in Sepsis Patients in the Validation Set

The AUC of the ROC curve for external validation was 0.875 (95% CI=0.811–0.939). The Hosmer-Lemeshow (H-L) test was used to validate consistency, yielding χ2=6.992 (P=0.703). See Figure 6.

External Validation of the Nomogram Model's DCA Curve in the Validation Set

The results of the decision curve analysis (DCA) showed that when the threshold probability was between 0.12 and 0.83, the model had high clinical application value for assessing new-onset AF in sepsis patients. See Figure 7.

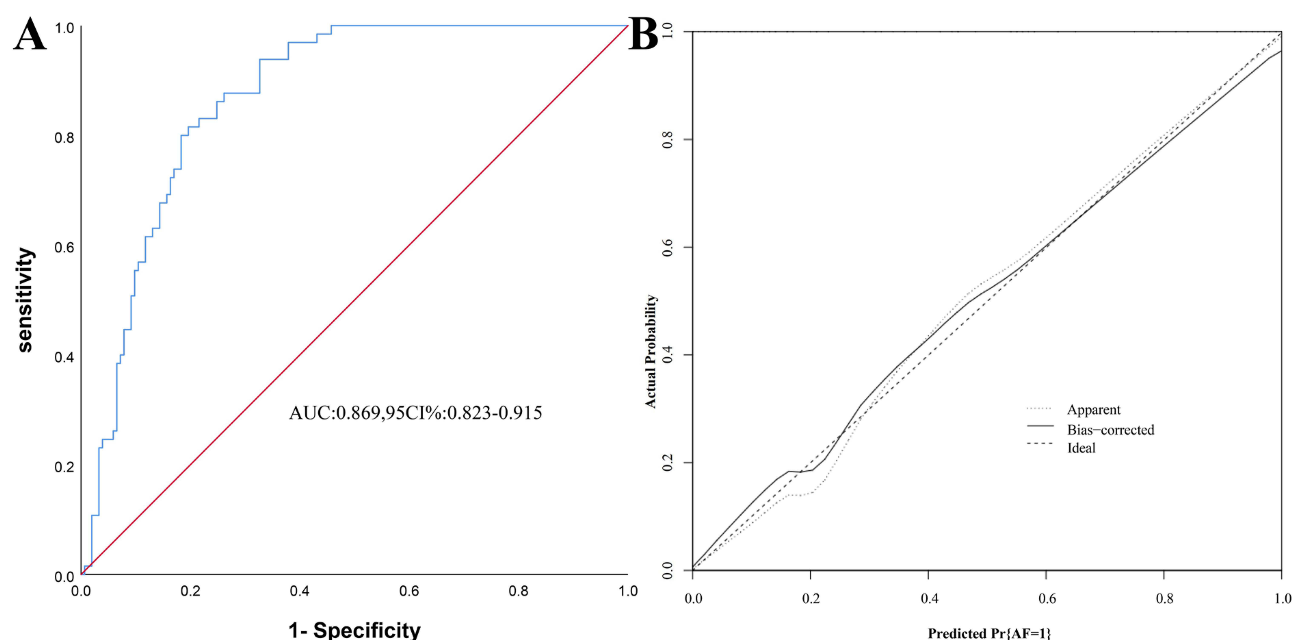

Figure 4 Internal validation of the nomogram model for new-onset atrial fibrillation (AF) in sepsis patients in the training set. (**A**) ROC curves — the larger the area under the curve (AUC), the better the model's discriminative ability. (**B**) Calibration curves — the closer the curve is to the 45° diagonal line, the better the agreement between predicted and observed outcomes.

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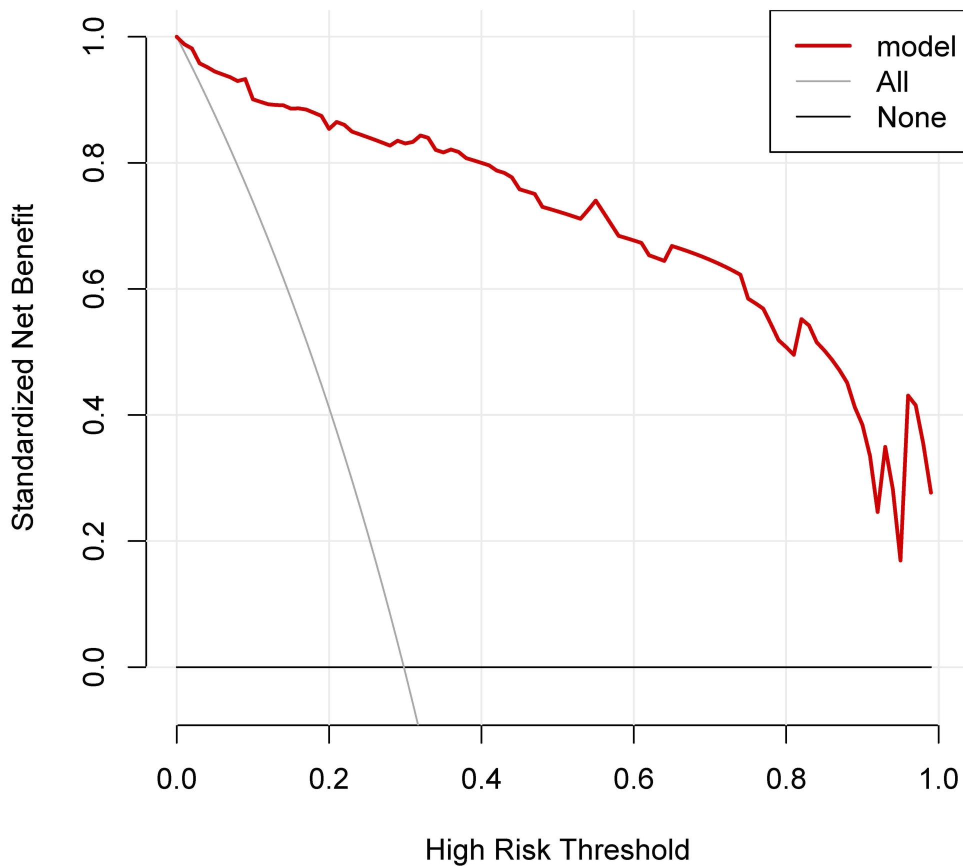

Figure 5 Internal validation of the nomogram model in the training set using the decision curve analysis (DCA). The horizontal axis represents the threshold probability, ranging from 0 to 1, corresponding to the probability threshold used in clinical practice to determine whether a patient will experience a disease or an adverse outcome. The vertical axis represents net benefit, with higher values indicating greater net clinical benefit at that threshold.

Discussion

The pathogenesis of sepsis is related to the pathophysiological changes in multiple organs and systems of the body, including systemic inflammation, tissue injury, etc. Clinically, most patients exhibit a systemic inflammatory response, increased respiratory rate, and decreased blood oxygen, which can also lead to shock, threatening human life.[10] Previous

Table 3 The Clinical Data of the AF Group and the Non-AF Group in the Validation Set

Factor	AF Group (n=32)	Non-AF Group (n=75)	t/χ^2	*P*
Age (years old)	68.65±5.24	60.89±5.13	7.119	<0.001
Gender			0.247	0.619
Male	20 (62.50)	43 (57.33)		
Female	12 (37.50)	32 (42.67)		
History of smoking			0.036	0.849
Yes	13 (40.63)	29 (38.67)		
No	19 (59.38)	46 (61.33)		
History of alcohol consumption			0.011	0.917
Yes	11 (34.38)	25 (33.33)		
No	21 (65.63)	50 (66.67)		
COPD			0.039	0.843
Yes	10 (31.25)	22 (29.33)		
No	22 (68.75)	53 (70.67)		

(*Continued*)

Table 3 (Continued).

Factor	AF Group (n=32)	Non-AF Group (n=75)	t/χ^2	*P*
Heart failure			0.076	0.783
Yes	4 (12.50)	8 (10.67)		
No	28 (87.50)	67 (89.33)		
Hypertension			0.791	0.374
Yes	14 (43.75)	26 (34.67)		
No	18 (56.25)	49 (65.33)		
Diabetes			0.267	0.605
Yes	11 (34.38)	22 (29.33)		
No	21 (65.63)	53 (70.67)		
Ischemic heart disease			0.172	0.678
Yes	12 (37.50)	25 (33.33)		
No	20 (62.50)	50 (66.67)		
Cerebrovascular disease			0.048	0.826
Yes	7 (21.08)	15 (20.00)		
No	25 (78.13)	60 (80.00)		
Malignancy			0.238	0.626
Yes	3 (9.38)	5 (6.67)		
No	29 (90.63)	70 (93.33)		
Acute kidney injury			1.414	0.234
Yes	15 (46.08)	26 (34.67)		
No	17 (53.13)	49 (65.33)		
New-onset heart failure			0.173	0.677
Yes	8 (25.00)	16 (21.33)		
No	24 (75.00)	59 (78.67)		
Metabolic disorders			3.020	0.082
Yes	14 (43.75)	20 (26.67)		
No	18 (56.25)	55 (73.33)		
Coagulopathy			0.146	0.702
Yes	7 (21.88)	14 (18.67)		
No	25 (78.13)	61 (81.33)		
Respiratory failure			0.850	0.357
Yes	15 (46.88)	28 (37.33)		
No	17 (53.13)	47 (62.67)		
Liver dysfunction			0.130	0.718
Yes	14 (43.75)	30 (40.00)		
No	18 (56.25)	45 (60.00)		
Mechanical ventilation			10.914	0.001
Yes	16 (50.00)	14 (18.67)		
No	16 (50.00)	61 (81.33)		
APACHE IIscore (points)	24.98±3.21	22.10±2.34	5.192	<0.001
CRRT treatment			0.280	0.597
Yes	6 (18.75)	11 (14.67)		
No	26 (81.25)	64 (85.33)		
Cediran	5 (15.63)	10 (13.33)	0.098	0.755
Anticholinergic drugs	6 (18.75)	12 (16.00)	0.121	0.728
Theophylline	17 (53.13)	18 (24.00)	8.644	0.003
Amiodarone	4 (12.50)	6 (8.00)	0.536	0.464
WBC (×109/L)	17.59±4.15	17.11±4.06	0.556	0.579
HGB (g/L)	106.38±19.86	107.69±20.24	0.308	0.759
FIB (mg/dL)	4.89±0.99	4.85±1.01	0.189	0.851

(*Continued*)

Table 3 (Continued).

Factor	AF Group (n=32)	Non-AF Group (n=75)	t/χ^2	P
IL-6 (ng/L)	2530.68±143.25	2510.68±136.58	0.683	0.496
TNF-α	128.62±20.11	96.58±17.21	8.377	<0.001
PCT (μg/L)	31.09±6.12	30.72±6.21	0.283	0.777
CK-MB (μg/L)	22.68±6.11	22.34±6.04	0.266	0.791
cTnI (μg/L)	5.96±1.06	6.02±1.03	0.274	0.785
K^+ (mmol/L)	4.29±0.99	4.27±0.85	0.106	0.916
LVEF (%)	50.96±9.86	50.72±9.62	0.117	0.907
E/e′	15.43±1.11	12.34±1.05	13.702	<0.001
NT-proBNP (ng/L)	3001.69±156.38	2476.58±154.65	16.028	<0.001

research has found that sepsis-induced cardiac dysfunction increases patient mortality; patients experience changes in cardiac structure, which ultimately induce cardiovascular events such as AF, impair cardiac function, and in severe cases, lead to death.[11] Some studies have confirmed that patients undergoing sepsis-related treatment are prone to tachycardia and AF. Under these circumstances, treatment for rapid arrhythmias is generally ineffective, leading to a poor prognosis.[12] Therefore, clinically identifying the factors that can influence AF for early prevention is crucial to maximally improve patient outcomes.

This study found that in the training set, there were differences between the two groups in age, mechanical ventilation, APACHE II score, acute kidney injury, metabolic disorders, theophylline medication, TNF-α, E/e′, and NT-proBNP. After screening with LASSO regression, 7 predictive factors were identified. Logistic analysis confirmed that age, mechanical ventilation, APACHE II score, theophylline medication, TNF-α, E/e′, and NT-proBNP are risk factors for new-onset AF in sepsis patients. The reasons are analyzed as follows: (1) For older patients, due to lower muscle mass, their immune function is correspondingly reduced, and the function of various organs also declines. The inflammatory response produced by sepsis can rapidly invade myocardial tissue, causing myocardial injury and inducing AF. Furthermore, elderly individuals have a high prevalence of cardiovascular diseases and poorer physical constitution,

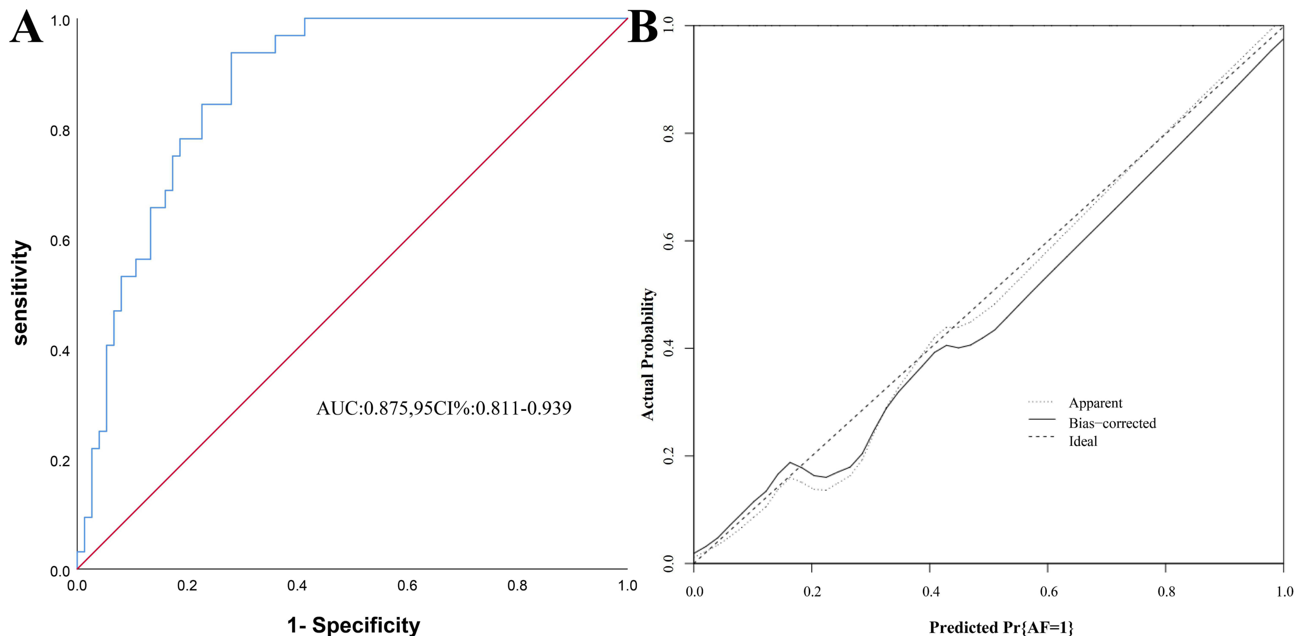

Figure 6 External validation of the nomogram model for new-onset atrial fibrillation (AF) in septic patients in the validation cohort. (**A**) ROC curves — the larger the area under the curve (AUC), the better the model's discriminative ability. (**B**) Calibration curves — the closer the curve is to the 45° diagonal line, the better the agreement between predicted and observed outcomes.

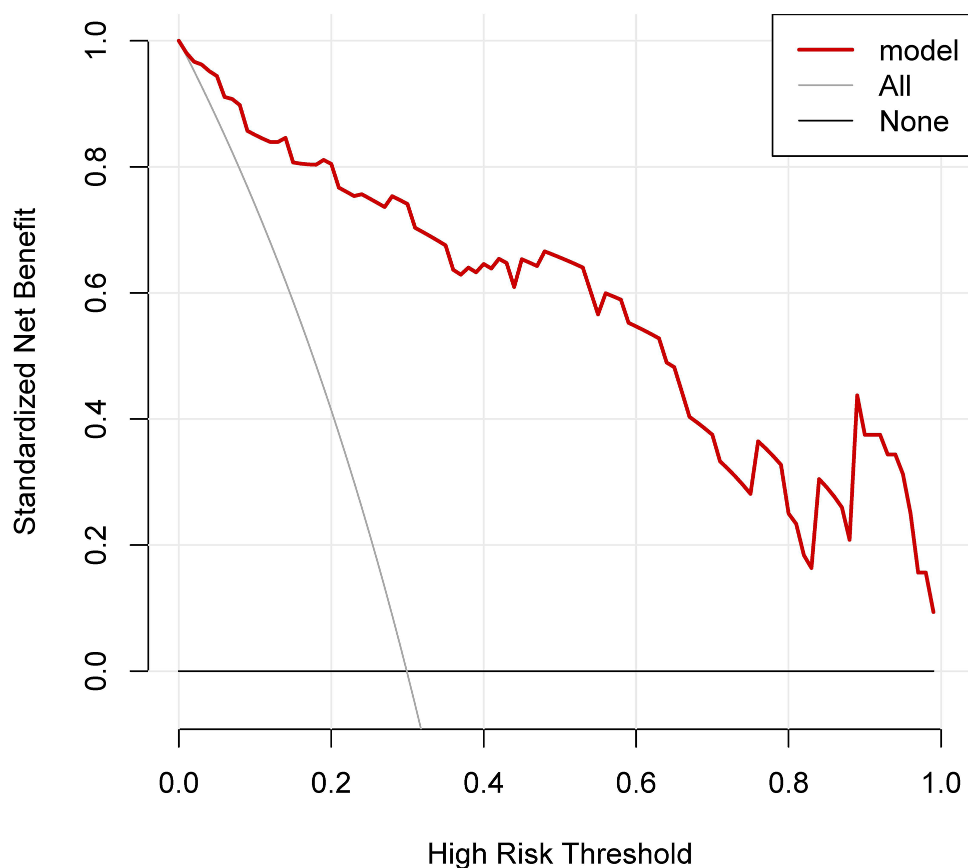

Figure 7 External validation of the nomogram model using decision curve analysis (DCA) in the validation set. The horizontal axis represents the threshold probability, ranging from 0 to 1, corresponding to the probability threshold used in clinical practice to determine whether a patient will experience a disease or an adverse outcome. The vertical axis represents net benefit, with higher values indicating greater net clinical benefit at that threshold.

which reduces myocardial fiber compliance and cardiac reserve function. When sepsis occurs, the severity of the condition increases the risk of new-onset AF.[13,14] Studies have shown that sepsis is more common in the elderly population mainly due to advanced age and reduced physical fitness, which predisposes them to AF,[15] consistent with the findings of this study. (2) Mechanical ventilation can improve a patient's ventilation and oxygenation, but during treatment, improper operation by medical staff can cause the patient to resist violently, leading to a rapid increase in heart rate. Additionally, excessive ventilation pressure during treatment can cause excessive intrathoracic pressure, reducing the patient's cardiac output, stimulating the sympathetic nervous system and increasing myocardial excitability, which can easily trigger AF, thereby lowering blood pressure, increasing heart rate, and impairing cardiac function, thus increasing the risk of AF.[16,17] (3) One study found that the APACHE II score is a factor for AF in ICU sepsis patients, which is similar to our study's findings, indicating that the more severe the patient's condition, the higher the likelihood of developing AF.[18] Therefore, regular clinical monitoring of changes in the APACHE II score can be used to assess disease progression and thus predict the occurrence of AF. (4) Theophylline drugs can increase myocardial blood supply and cardiac contractility when treating sepsis patients. However, due to the significant stimulatory nature of these drugs, studies have found that infusing theophylline in sepsis patients may cause AF,[19] which is consistent with the results of this study. Therefore, close monitoring of the patient's heart rate is necessary when using this drug clinically, as this class of drugs may also impair cardiac function while improving the patient's condition, and attention should be paid to the dosage used. (5) The pathogenesis of AF is closely related to cardiac structural remodeling caused by the inflammatory response. TNF-α plays a role in the progression of inflammation. Studies have found that TNF-α is significantly elevated in patients with atrial fibrillation, and the use of tumor necrosis factor inhibitors can alleviate atrial remodeling, suggesting that it may induce AF by participating in the structural remodeling of the atria, mainly because inflammatory

factors can impair cardiac function, leading to an acute increase in left ventricular end-diastolic pressure and excessive stretching of the left atrium, thereby promoting the occurrence of AF.[20–22] (6) E/e′ is an indicator for assessing left ventricular diastolic function. Sepsis-induced cardiomyopathy mainly manifests as left atrial enlargement and diastolic dysfunction. An elevated E/e′ indicates possible diastolic dysfunction, which can easily lead to AF. This is because when the body's inflammation damages cardiac function, it causes an acute increase in left ventricular end-diastolic pressure, leading to a subsequent rise in E/e′, which causes excessive stretching of the left atrium and increases the risk of AF.[23,24] (7) NT-proBNP is a hormone secreted by ventricular myocytes in response to heart failure and increased ventricular wall tension. When ventricular volume load and ventricular wall tension increase, the secretion of NT-proBNP is promoted. Studies have found that inflammatory factors and endotoxins released in sepsis patients can cause AF, leading to elevated NT-proBNP levels,[25,26] which is consistent with this study, indicating that its elevated level can be used as an indicator to assess AF in patients.

Nomograms can transform complex regression equations into graphical representations, making the results of predictive models more intuitive and facilitating the assessment of patients by clinicians. Based on LASSO regression and Logistic analysis, this study constructed a nomogram model. The AUCs for the training set and external validation were 0.869 and 0.875, respectively, and the H-L test showed good consistency. The DCA curve showed high clinical application value when the threshold probability was 0.10–0.89 for the training set and 0.12–0.83 for the external validation set. This indicates that the nomogram model has high clinical utility and can assist medical personnel in predicting the risk of AF and implementing timely prevention, thereby effectively improving patient prognosis. Therefore, in clinical practice, high-risk patients should be closely monitored, and patients with inflammatory responses should also receive heightened attention, in order to minimize the occurrence of AF as much as possible. This study has certain limitations. As a retrospective study with a relatively small sample size, there is a potential risk of model overfitting. Moreover, being a single-center study, it may be subject to bias. Future studies with larger sample sizes are needed for further validation.

Conclusion

In summary, age, mechanical ventilation, APACHE II score, theophylline medication, TNF-α, E/e′, and NT-proBNP are influencing factors for new-onset AF in sepsis patients. The nomogram prediction model constructed based on these factors has good clinical applicability and can serve as an assessment tool for screening patients at risk of new-onset AF.

Data Sharing Statement

The datasets used during the present study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study involving human participants was in accordance with the ethical standards of The Guangdong Medical University Affiliated Hospital Research Committee (No. PJ2025134) and with the 1964 Helsinki Declaration. Written informed consent was obtained from all participants. For patients who were unable to provide informed consent themselves due to severe sepsis or impaired consciousness, consent was obtained from their legal guardians in accordance with ethical requirements.

Disclosure

The authors declare no conflicts of interest in this work.

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