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Research article

Development of an acute kidney injury risk prediction model for patients undergoing extracorporeal membrane oxygenation

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ABSTRACT

Background: Some studies have reported to use some predictors before extracorporeal membrane oxygenation (ECMO) initiation to predict the acute kidney injury (AKI) risk. However, injury during the ECMO operation and the response of patients to ECMO may significantly influence the prognosis, and they are unpredictable before ECMO initiation. This study aims to develop a potential model based clinical characteristics at the 2-hour time point during ECMO for the early prediction of AKI in patients receiving ECMO.

Methods: 139 patients who underwent ECMO were enrolled in this study. The clinical characteristics and the laboratory examinations at 2-hour time point during ECMO were recorded. The least absolute shrinkage and selection operator (LASSO) regression method was performed to select predictors, and logistic regression and a nomogram were used to establish the prediction model. The area under curve (AUC) of the receiver operating characteristic and calibration curve were used to analyze the discrimination and calibration of the model. K-fold cross-validation method was performed to validate the accuracy of this model.

Results: Among the 139 patients receiving ECMO, 106 participants (76.26%) developed AKI. Four predictive variables including ECMO model, serum creatinine (Scr-2h), uric acid(UA-2h), and serum lactate (Lac-2h) at the 2-hour time point during ECMO were filtered from 39 clinical parameters by LASSO regression. These four predictors were incorporated to develop a model for predicting AKI risk using logistic regression. The AUC of the model was 0.905 (0.845–0.965), corresponding to 81.1% sensitivity, 90.9% specificity and 83.5% accuracy. Moreover, this model showed good consistency between observed and predicted probability based on the calibration curve ($P > 0.05$). The validation performed by K-fold cross-validation method showed that the accuracy was 0.874 ± 0.006 in training sets, 0.827 ± 0.053 in test sets, indicating a good capability for AKI risk prediction. Finally, a nomogram based on this model was constructed to facilitate its use in clinical practice.

Conclusion: The nomogram incorporating Scr-2h,Lac-2h, UA-2h, and ECMO model may facilitate the individualized prediction of the AKI risk among patients undergoing ECMO.

1. Introduction

Extracorporeal membrane oxygenation (ECMO) is a modified extracorporeal circulation technology that has been widely applied in support therapy for patients with severe acute cardiopulmonary function failure, such as cardiogenic shock due to acute myocardial infarction, acute pulmonary embolism, severe acute respiratory distress syndrome, and respiratory support for lung transplantation among others [1, 2]. However, ECMO can lead to serious and fatal complications [3]. Acute kidney injury (AKI), which is widely recognized as acute renal failure, is one of the most frequent and serious complications of ECMO [4]. Early diagnosis and treatment of AKI are important to prevent secondary multiple organ failure and to improve the prognosis of patients receiving ECMO [5, 6]. Therefore, a method for the early diagnosis of AKI in patients receiving ECMO is urgently needed.

According to the Kidney Disease: Improving Global Outcomes (KDIGO, 2012) [7], the diagnosis of AKI requires at least 6 h of continuous urinary output monitoring every hour after receiving ECMO. By this time, the patients may have already had overt AKI; however, up to 30% of hospital-acquired AKI cases can be prevented if predicted in time [8,

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9]. Additionally, patients receiving ECMO are critically ill, and the state of illness can change rapidly in a short time, which is why the early risk prediction of AKI is of great importance among patients receiving ECMO. In recent years, new biomarkers have been found to evaluate AKI [10, 11, 12, 13, 14]. However, most studies have focused on the early diagnosis of AKI caused by other diseases [10, 11, 12, 13, 14], and many of these new markers are not yet widely available in clinical practice. Therefore, in this retrospective study, we aimed to explore the independent risk factors for AKI, and then develop a potential model based on the clinical characteristics and commonly used clinical renal biomarkers at the 2-hour time point during ECMO.

2. Materials and methods

2.1. Study population

This present study was a retrospective analysis of patients who underwent ECMO from January 2015 to April 2021 at the Department of Intensive Care Unit, Affiliated Jinhua Hospital, Zhejiang University School of Medicine. These patients had been diagnosed with acute myocardial infarction, fulminant myocarditis, severe acute respiratory distress syndrome, acute pulmonary embolism, or other conditions that required ECMO support. During the selection process of this study, the patients were excluded according to the following exclusion criteria: (1) patients who had medical history of renal insufficiency, (2) patients who received ECMO support for less than 6 h, (3) patients who passed away within 6 h of ECMO use, and (4) patients without any major clinical features and laboratory measurement results. Finally, A total of 139 patients underwent ECMO were recruited for the present study. In addition, patients suffered AKI after ECMO initiation were assigned to the AKI group, and patients without AKI after ECMO initiation were assigned to the non-AKI group.

In this study, the primary causes of ECMO were divided into four categories: (1) cardiogenic shock caused by cardiomyopathies, such as fulminant myocarditis, dilated cardiomyopathy, and rupture of papillary muscles and chordae tendineae; (2) cardiogenic shock caused by coronary artery diseases, such as coronary artery spasm, acute coronary syndrome, and anomaly of coronary artery; (3) cardiogenic shock caused by other diseases, such as malignant arrhythmia, cerebral hemorrhage, pulmonary embolism, pneumocranium, drug intoxication, and unexplained cardiac arrest; (4) acute respiratory distress syndrome (ARDS), such as severe viral pneumonia, severe bacterial pneumonia, and pneumocystic carinii pneumonia, pancreatitis and acute respiratory distress syndrome associated with crush injury and extrapulmonary disease. This study followed the tenets of the Declaration of Helsinki and was approved by the Ethics Committee of Affiliated Jinhua Hospital, Zhejiang University School of Medicine.

2.2. Data collection

The clinical characteristics of all participants in this study were collected from patient electronic medical records, including age, gender, acute Physiology and Chronic Health Evaluation II (APACHE II), vasopressor or not before ECMO initiation, ECMO model (veno-arterial ECMO or veno-venous ECMO), presence of AKI after ECMO initiation, primary causes of ECMO, time of ECMO, intensive care unit (ICU) hospital stay, total in-hospital stay. The important laboratory measurements at the 2-hour time point during ECMO collected in the study included serum creatinine (Scr), uric acid (UA), blood urea nitrogen, total bilirubin, total protein, albumin, serum amylase, alanine aminotransferase, aspartate aminotransferase, anion gap, lactic acid (Lac), blood glucose, hydrogen ion concentration, oxygen partial pressure, partial pressure of carbon dioxide, standard bicarbonate, actual bicarbonate, concentrations of potassium, calcium, sodium, chlorine and magnesium ions, white blood cell count, neutrophil count, hemoglobin, hematocrit, platelet count, troponin, lactate dehydrogenase, creatine kinase, creatine kinase isoenzymes, brain natriuretic peptide, c-reactive protein and procalcitonin.

2.3. Definition

According to the guidelines recommended by KDIGO (2012) [7], the diagnostic criteria of AKI were as follows: (1) a serum creatinine (Scr) level which is absolutely increased to $\geq$26.5 μmol/L (0.3 mg/dL) within 48 h; (2) an over 1.5-fold increase in Scr compared with the initial level within 7 days; or (3) a urinary output that was less than 0.5 Â ml/(kg·h) for 6 consecutive hours. Definition of basal creatinine level: (1) the Scr level at admission was considered as the basal Scr level; or (2) the lowest Scr value during hospitalization was used as the baseline Scr value; or (3) the lowest Scr value of the aforementioned three Scr values; or (4) the mean value of SCR recorded between 1 year before admission and 7 days before admission.

2.4. Statistical analysis

R software (version 3.6.4) and SPSS 26.0 statistical software were used to perform the data analyses for this study. The data with normal distribution were presented as mean ± standard deviation, the non-normally distributed variables were presented as median (interquartile range, Q1–Q3), and the categorical variables were expressed as percentages. Student's t-test, Mann-Whitney test (skewed distributed data), and Pearson's chi-squared test (categorical variables) were performed for comparative analysis between the groups for normally distributed data, non-normally distributed data, and categorical data, respectively. The least absolute shrinkage and selection operator (LASSO) is a regression method especially valuable in datasets with numerous variables and support unbiased parameter selection. Compared to traditional regression approaches, LASSO regression can handle a larger set of potential predictors, picking out the variables most associated with the disease, and avoid overfitting of the model [15]. Due to the sample size (139 subjects) and a larger number of clinical variables (39 variables) in this study, by the principle of simplifying model and avoiding over-fitting, firstly, LASSO regression was used to strictly filtrate the potential prediction variables, and then the logistic regression model was established using the potential prediction variables. A nomogram based on the model was constructed to facilitate its use in clinical practice. The area under the curve (AUC) of the receiver operating characteristic (ROC) curve was used to estimate the discriminative ability of this model. Z test was used to compared the AUC of different models or variables for AKI risk prediction. The goodness-of-fit of the model was assessed using the calibration curve. K-fold cross-validation method was performed to validate the accuracy of this model. In the present study, a value of $P < 0.05$ was considered statistically significant.

3. Results

Among the 139 participants with a mean age of 48.86 ± 17.30 years, 72.66% were male, 82.73% received veno-arterial ECMO. The average APACHE II score was 17.41 ± 4.94, the average ECMO time was 5.71 ± 4.64 days, the median hospital stay was 13 (3–21.5) days. Additionally, 106 of them (76.26%) suffered AKI after ECMO initiation. Patients suffered AKI after ECMO initiation were assigned to the AKI group, and patients without AKI after ECMO initiation were assigned to the non-AKI group. The comparisons of the clinical characteristics and the laboratory examinations at the 2-hour time point after ECMO initiation between these two groups were listed in Table 1.

In the present study, 39 clinical parameters of participants were analyzed by LASSO regression to filter potential predictive factors for AKI risk (Figure 1). Finally, four variables were selected as potential predictors based on non-zero coefficients, including the levels of Scr-2h, Lac-2h, UA-2h and ECMO model. A predictive model for AKI risk of patients undergoing ECMO was established based on the combined analysis of

Table 1. Clinical characteristics of ECMO-treated patients.

Variables	AKI (n = 106)	Non-AKI (n = 33)	*P* value
Age (year)	49.42 ± 16.843	47.09 ± 18.867	0.502
Male(n, %)	78 , 73.6 %	23 , 69.7%	0.662
APACHE II score before ECMO initiation	27.36 ± 4.87	19.51 ± 7.37	0.536
Vassopressor before ECMO (n,%)	65 , 74.7%	11 , 45.8%	0.809
VA-ECMO (n,%)	96 , 90.6%	19 , 57.6%	< 0.001
Death (n,%)	70 , 66.0 %	12 , 36.4%	0.002
The primary causes of ECMO			
Cardiogenic shock caused by cardiomyopathy (n, %)	21 , 19.8 %	4 , 12.1 %	0.438
Cardiogenic shock caused by coronary artery disease (n, %)	33 , 31.1%	8 , 24.2%	0.449
Cardiogenic shock caused by other diseases (n, %)	40 , 37.7%	7 , 21.2%	0.080
ARDS caused by diseases (n, %)	12 , 11.3%	14 , 42.4%	< 0.001
The time of ECMO (day)	5.47 ± 4.67	8.52 ± 11.99	0.033
ICU hospital stay (day)	13.61 ± 17.63	18.24 ± 22.60	0.221
Total hospital stay (day)	16.33 ± 23.41	22.97 ± 22.95	0.154
Lac-2h (mmol/L)	11.99 ± 6.46	5.80 ± 5.26	< 0.001
Anion gap-2h (mmol/L)	15.73 ± 8.12	8.22 ± 8.16	< 0.001
Glucose-2h (mmol/L)	10.95 ± 5.57	12.42 ± 5.55	0.189
PH2h	7.49 ± 1.10	7.62 ± 1.03	0.561
Oxygen partial pressure-2h (mmHg)	306.80 ± 171.23	203.05 ± 140.37	0.001
partial pressure of carbon dioxide-2h (mmHg)	29.47 ± 10.73	27.07 ± 7.32	0.150
Standard bicarbonate-2h (mmol/L)	15.51 ± 5.54	19.55 ± 4.66	< 0.001
Actual bicarbonate-2h (mmol/L)	13.98 ± 5.64	17.24 ± 4.79	0.003
Potassium-2h (mmol/L)	4.14 ± 1.43	4.00 ± 0.67	0.570
Sodium-2h (mmol/L)	144.69 ± 7.95	140.37 ± 5.95	0.005
Chlorine-2h (mmol/L)	114.26 ± 7.72	114.20 ± 6.10	0.965
Calcium-2h (mmol/L)	1.47 ± 2.38	1.65 ± 2.43	0.703
Magnesium-2h (mmol/L)	2.08 ± 3.09	2.39 ± 3.57	0.633
Alanine aminotransferase-2h (U/L)	585.75 ± 812.74	125.73 ± 168.63	< 0.001
aspartate aminotransferase-2h (U/L)	1497.59 ± 2023.12	253.67 ± 267.17	< 0.001
Total bilirubin-2h (U/L)	20.61 ± 19.14	16.09 ± 7.52	0.049
Albumin-2h (g/L)	23.90 ± 6.36	25.59 ± 5.94	0.178
Total protein-2h (g/L)	41.70 ± 12.67	45.76 ± 14.14	0.120
Serum amylase-2h (g/L)	452.94 ± 707.85	139.88 ± 206.69	< 0.001
Scr-2h (umol/L)	166.62 ± 64.64	97.27 ± 36.69	< 0.001
Blood urea nitrogen-2h (mmol/L)	9.57 ± 5.84	7.32 ± 4.21	0.042
UA-2h (umol/L)	453.39 ± 248.64	237.27 ± 128.79	< 0.001
White blood cell-2h (*10^9/L)	17.33 ± 9.23	14.33 ± 6.82	0.086
Neutrophils count-2h (*10^9/L)	14.30 ± 8.07	12.52 ± 5.86	0.243
Hemoglobin-2h (g/L)	106.91 ± 31.51	114.85 ± 24.10	0.131
Hematocrit (%)	32.73 ± 10.85	34.27 ± 7.15	0.447
Platelet-2h (*10^9/L)	160.91 ± 81.43	183.82 ± 120.51	0.214
Troponin T-2h (ng/mL)	290.27 ± 883.77	40.04 ± 55.25	0.005
Lactate dehydrogenase-2h (U/L)	3864.25 ± 5588.09	901.80 ± 1102.02	< 0.001
Creatine Kinase-2h (U/L)	4444.41 ± 5942.21	1076.83 ± 1409.05	< 0.001
Creatine Kinase isoenzymes-2h (U/L)	632.79 ± 723.73	134.65 ± 161.38	< 0.001
B-type natriuretic peptide-2h (pg/mL)	7617.95 ± 14666.76	2922.51 ± 4670.96	0.005
C-reactive protein-2h (mg/L)	37.56 ± 52.63	58.68 ± 64.55	0.094
Procalcitonin-2h (ng/mL)	13.99 ± 25.77	2.89 ± 3.99	< 0.001

ECMO: extracorporeal membrane oxygenation; APACHE II： acute Physiology and Chronic Health Evaluation II; VA-ECMO: vein-artery ECMO; AKI: acute kidney injury; ICU: intensive care unit; ARDS: acute respiratory distress syndrome; Lac: lactic acid; Scr: serum creatinine; UA: uric acid.

these four potential predictors using multivariate logistic regression method. The results of the logistic regression model were listed in Table 2. ROC analysis was performed to assess the discriminative ability of this model (Figure 2A). The optimal cut-off value was 0.782 based on the maximum principle of the Youden index, while the AUC was 0.905 (0.845–0.965), corresponding to 81.1% sensitivity, 90.9% specificity and 83.5% accuracy. The AUC of Scr-2h, UA-2h and Lac-2h for AKI risk prediction were 0.87 (0.80–0.94), 0.76 (0.68–0.84) and 0.80 (0.71–0.89), respectively. The predictive performance of the constructed model was relatively higher than each of the three variables. Especially the AUC of this model was significantly greater than that of UA-2h and Lac-2h ($P < 0.05$). The calibration curve of the model indicated a good consistency between observed and predicted outcomes (Figure 2B). Moreover, the proposed model showed the similar accuracy for prediction of AKI risk for both male group and female group. For female group, the AUC was 0.893 (0.788–0.998), corresponding to 78.6% sensitivity,

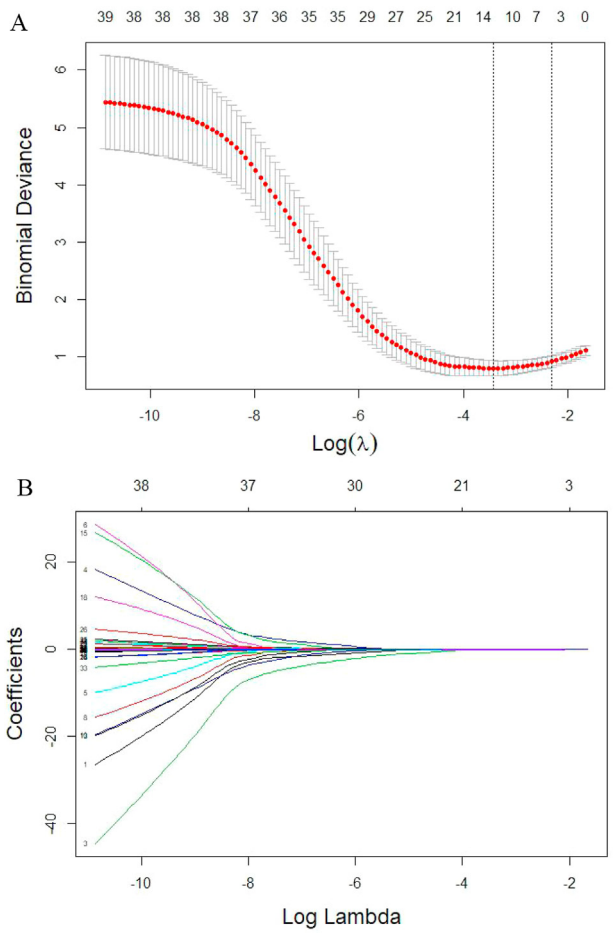

Figure 1. Predictive variables selection using the LASSO regression method. (A) Tuning parameter selection in the LASSO regression used 10-fold cross-validation. (B) LASSO regression coefficient profiles of variables.

90.0% specificity. For male group, the AUC was 0.907 (0.834–0.981), corresponding to 84.6% sensitivity, 91.3% specificity.

K-fold cross-validation method (K = 5) was performed to validate the accuracy of the aforementioned model for AKI risk among the patients underwent ECMO. The results showed that the accuracy was 0.874 ± 0.006 in training sets, 0.827 ± 0.053 in test sets, indicating a good capability for AKI risk prediction. Finally, a nomogram based on this model was constructed to provide a quantitative tool to estimate the AKI risk of patients receiving ECMO in clinical practice (Figure 3).

4. Discussion

In this retrospective study, we identified four predictive factors by LASSO regression method, which consisted of ECMO model, Scr-2h, Lac-2h, and UA-2h. A nomogram based on these four predictors was constructed to facilitate individualized prediction for AKI risk in the early stage of ECMO. This predictive model showed good discriminative ability in AKI risk among patients underwent ECMO, and had a high predictive accuracy of 83.5%.

The study of AKI is important as it can significantly increase the mortality rate, prolong the length of hospital stay, and increase the cost of hospitalization in the short term; in the long term, it can also lead to the recurrence of AKI, development of chronic kidney disease or end stage renal disease, and cause adverse cardiovascular events [16, 17, 18]. In the clinical setting, AKI is the most common complication of ECMO [19]. Although the timing of continuous renal replacement therapy (CRRT) for patients receiving ECMO is still inconclusive, it is generally believed that early diagnosis of AKI and CRRT treatment can prevent secondary multiple organ failure [20]. Kon et al. [6] reported that among patients receiving ECMO, those receiving early CRRT for AKI had a better prognosis, and the renal function of most of those patients could be reversible. Additionally, a retrospective study by Antonucci showed that early application of CRRT after ECMO surgery did not increase the mortality rate of patients [5]. Therefore, risk prediction and early detection of AKI are vitally important for improving prognosis because of its high incidence among patients receiving ECMO.

At present, according to the guidelines recommended by the KDIGO (2012) [7], the diagnostic criteria for AKI are mainly based on the changed Scr level within 48 h or the urinary output for 6 consecutive hours. However, this becomes problematic for many patients receiving ECMO as they may already have overt AKI by then. In recent years, research on the early diagnosis of AKI has developed rapidly, and a number of new renal damage biomarkers have been found for the evaluation of AKI, such as serum kidney injury molecule 1, insulin-like growth factor binding protein 1, and neutrophil gelatinase-associated lipocalin, etc [10, 11, 12, 13, 14]. However, most studies have focused on the early diagnosis of AKI caused by other diseases, while the early risk prediction of AKI in patients receiving ECMO has rarely been reported. Additionally, the measurements of these new biomarkers are not yet widely available in clinical laboratories. Therefore, at present, a method based on routine clinical parameters is urgently needed for early risk prediction of AKI among patients receiving ECMO.

Serum Lac is closely related with tissue hypoperfusion, abnormal carbohydrate metabolism, and the use of special drugs [21]. Lac level can be used both as a risk indicator for critically ill patients and as a therapeutic target, the higher the Serum lactate level, the greater the risk of death [21]. In the present study, Lac level at the 2-hour time point during ECMO was one of the strong predictors for AKI risk. The results was consistent with other studies, an elevated Lac level had predictive value for AKI in septic patients or patients heart failure [22, 23].

Previous studies had reported that the levels of Scr and UA significantly associated with renal impairment [24, 25]. Scr is a metabolite of creatine, and its serum level can reflect the glomerular filtration rate. As a traditional indicator, Scr has been widely applied in clinical practise to evaluate the renal function. Additionally, UA might promote inflammation, oxidative stress, mitochondrial dysfunction, and activate renin-angiotensin system to induce renal vasoconstriction and the impairment of renal vascular endothelium [26, 27]. In this study, both these two typical biomarkers were the potential predictors for AKI risk. The increased levels of them at the 2-hour time point during ECMO significantly associated with the elevated AKI risk.

Additionally, the selected patient population for this study comprised over 70% male and so only 30% female patients. In order to estimate

Table 2. Logistic regression for the four predictive variable selected out by LASSO regression.

Variables	Partial regression coefficients	Standard error	Wald value	OR (95%CI)	*P*
Lac-2h	0.132	0.055	5.855	1.14 (1.035–1.27)	0.016
Scr-2h	0.027	0.008	12.398	1.03 (1.01–1.05)	< 0.001
UA-2h	0.003	0.002	2.760	1.01 (0.99–1.01)	0.097
ECMO model	−0.241	0.737	0.107	0.79 (0.19–3.33)	0.743

Lac: lactic acid; Scr: serum creatinine; UA: uric acid; ECMO: extracorporeal membrane oxygenation.

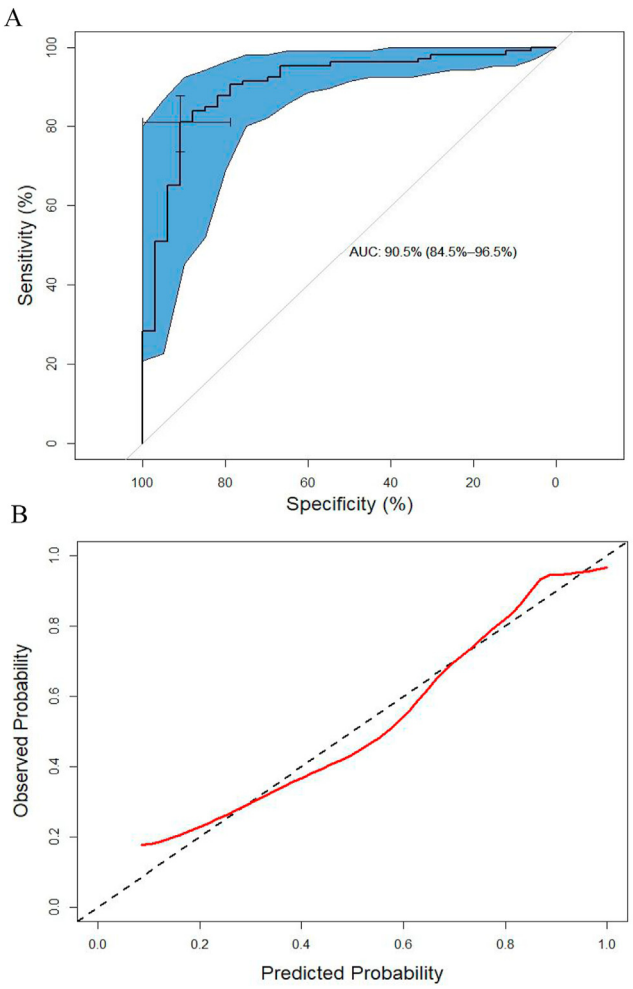

Figure 2. The ROC curve (A) and calibration curve (B) of this predictive model. The blue area presents 95% confidence interval. The intersection of a cross is the optimal cut-off value of the ROC curve.

whether the proposed model would be equally valid for prediction of AKI risk for male and female patients, the data was separated and the ROC analyses were performed for male and female groups, respectively. Finally, male group had the similar AUC with female group (0.907 vs 0.893), indicating that the model had the same accuracy for prediction of AKI risk for both male and female patients.

The present study has several limitations. First, this was a retrospective study, and the blood samples that were collected from patients receiving ECMO were not sufficient. Hence, this study did not include the novel biomarkers, such as kidney injury molecule 1 and/or neutrophil gelatinase-associated lipocalin. Second, the results of this study were observed in a relatively small population from a single center. Third, we did not analyze subsequent laboratory parameters, the optimal time point to predict the AKI risk of patients underwent ECMO needed more researches. Forth, it was challenging to assess if the AKI secondary to the severity of patients condition and shock or to the ECMO therapy. A well-designed, large-scale study is necessary to establish a more effective early prediction model for AKI risk in patients receiving ECMO.

5. Conclusion

In conclusion, nearly three-quarters of ECMO-treated patients suffered AKI in this retrospective study. We constructed a nomogram for AKI risk based on four predictive factors, which consisted of ECMO model, the levels of Scr, UA, and Lac at the 2-hour time point during ECMO. This predictive model demonstrated potential application in clinical practice due to the high accuracy for assessing the AKI risk of the patients receiving ECMO. However, this model should be validated in a larger external cohort before widespread utilization.

Declarations

Author contribution statement

Liming Wang: Conceived and designed the experiments; Performed the experiments; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Lin Chen: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data.

Hongying Ni; Hongsheng Deng: Performed the experiments; Analyzed and interpreted the data.

Kun Chen: Conceived and designed the experiments.

Huabin Wang: Contributed reagents, materials, analysis tools or data; Wrote the paper.

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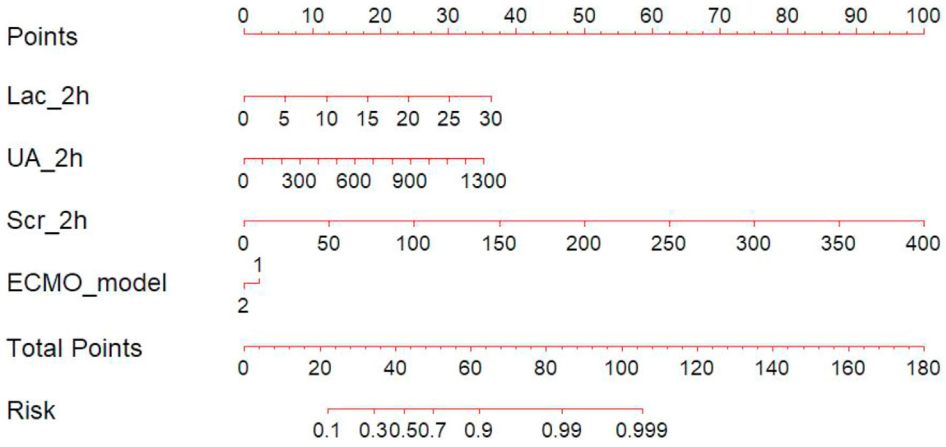

Figure 3. A nomogram for predicting AKI risk of patients receiving ECMO. Lac: lactic acid; Scr: serum creatinine; UA: uric acid; ECMO: extracorporeal membrane oxygenation; ECMO model 1: VA-ECMO; ECMO model 2: VV-ECMO.

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Data availability statement

Data will be made available on request.

Declaration of interest's statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

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