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# Development and evaluation of the model for acute kidney injury in patients with cardiac arrest after successful resuscitation

Shanbing Hou[1], Lixiang Zhang[1,2], Hongzhi Ji[1], Tingting Zhao[1], Ming Hu[1], Ying Jiang[1], Quanquan Sun[1], Ming Zhang[3] and Min Dou[1*]

## Abstract

**Background** This study aims to construct a clinical prediction model and create a visual line chart depicting the risk of acute kidney injury (AKI) following resuscitation in cardiac arrest (CA) patients. Additionally, the study aims to validate the clinical predictive accuracy of the developed model.

**Methods** Data were retrieved from the Dryad database, and publicly shared data were downloaded. This retrospective cohort study included 347 successfully resuscitated patients post-cardiac arrest from the Dryad database. Demographic and clinical data of patients in the database, along with their renal function during hospitalization, were included. Through data analysis, the study aimed to explore the relevant influencing factors of acute kidney injury (AKI) in patients after cardiopulmonary resuscitation. The study constructed a line chart prediction model using multivariate logistic regression analysis with post-resuscitation shock status (Post-resuscitation shock refers to the condition where, following successful cardiopulmonary resuscitation after cardiac arrest, some patients develop cardiogenic shock.), C reactive protein (CRP), Lactate dehydrogenase (LDH), and Alkaline phosphatase (ALP) identified as predictive factors. The predictive efficiency of the fitted model was evaluated by the area under the curve (AUC) of the receiver operating characteristic (ROC) curve.

**Results** Multivariate logistic regression analysis showed that post-resuscitation shock status, CRP, LDH, and PAL were the influencing factors of AKI after resuscitation in CA patients. The calibration curve test indicated that the prediction model was well-calibrated, and the results of the Decision Curve Analysis (DCA) demonstrated the clinical utility of the model constructed in this study.

**Conclusion** Post-resuscitation shock status, CRP, LDH, and ALPare the influencing factors for AKI after resuscitation in CA patients. The clinical prediction model constructed based on the above indicators has good clinical discriminability and practicality.

**Keywords** Cardiac arrest, Acute kidney injury, Cardio pulmonary resuscitation, Nomogram, Influencing factors

Shanbing Hou, Lixiang Zhang and Hongzhi Ji are co-first authors of this work.

*Correspondence:
Min Dou
987960364@qq.com

[1]Department of Nursing, The First Affiliated Hospital of USTC, Division of Life Sciences and Medicine, University of Science and Technology of China, Hefei 230001, Anhui, China
[2]Department of Cardiology, The First Affiliated Hospital of USTC, Division of Life Sciences and Medicine, University of Science and Technology of China, Hefei, Anhui, 230001, China, Hefei 230001, Anhui, China
[3]School of Innovation and Entrepreneurship, Wannan Medicine College, Wuhu 241000, Anhui, China

Cardiac arrest (CA) is one of the major public health issues that seriously threatens people's lives and health [1]. Due to its sudden onset and the majority of patients experiencing CA outside of the hospital, the clinical death rate is high and the prognosis is poor. Therefore, CA is one of the most critical conditions in both in-hospital and out-of-hospital emergency medical care [2]. After experiencing cardiac arrest, patients lose their cardiac pumping function, leading to ischemia and hypoxia in vital organs such as the heart and brain. Without prompt and effective clinical intervention, irreversible damage occurs in these vital organs within minutes, eventually transitioning the body into biological death [3]. Studies have shown that only about 10.4% of patients who experience cardiac arrest and receive active cardiopulmonary resuscitation (CPR) are able to achieve the criteria for discharge and survive to be discharged from the hospital [4]. However, due to organ ischemia and hypoxia following cardiac arrest, resulting in multiple organ dysfunction, the occurrence of post-resuscitation complications in patients increases with the duration of return of spontaneous circulation (ROSC) [5]. Research has shown that the kidneys of patients after cardiac arrest remain in a state of continuous ischemia and hypoxia, and even effective CPR cannot meet the oxygen supply needs of the kidneys. With prolonged ischemia and hypoxia in the kidneys, coagulation function impairment occurs, leading to the release of various inflammatory substances, which in turn causes injury to the endothelial cells of blood vessels and impairs the reabsorption function of renal tubules for electrolytes [6, 7]. Heyman et al. [8] found that insufficient blood supply-induced ischemic injury can lead to disruption of cell polarity. In the kidneys, this reaction can increase the permeability of renal tubules, allowing large molecular substances to enter the tubular lumen and cause acute kidney injury [8]. Current research on acute kidney injury (AKI) in post-cardiopulmonary resuscitation (CPR) patients is somewhat fragmented both domestically and internationally. While some studies have preliminarily explored factors related to AKI and cardiorenal syndrome in post-arrest patients, the results remain incomplete and only serve as indirect references for clinical diagnosis and treatment, lacking direct clinical applicability. Therefore, this study aims to perform statistical analysis on clinical data from publicly available databases and construct a visual clinical prediction model to assist healthcare professionals in the early detection and preliminary screening of AKI in post-CPR patients. Furthermore, it aims to aid medical staff in providing early intervention to high-risk patients for AKI through scientifically effective clinical measures, thereby reducing the clinical incidence of AKI in post-CPR patients.

## Research objectives and methods

### Research data

Dyrad is a non-membership-based public data-sharing platform dedicated to enabling researchers to openly share data for public use. In this database, patient informed consent has been waived, as all data therein are retrospectively analyzed from existing data sources. In this study, the research data was obtained through registration and retrieval from the Dryad database (URL: https://DATADryad.org/). Relevant data related to the target research were searched and downloaded from the database. The downloaded data was then reviewed and subjected to secondary statistical analysis. The data used in this study were publicly available data downloaded from the Dryad database, with the data location specified as (https://doi.org/10.5061/Dryad.qv6fp83). Previous research literature [9] was cited as a reference for the analyzed data. The dataset contained a total of 374 patient records, out of which 347 patient records were included in this study after excluding cases based on the following criteria: ① patients who experienced clinical death within 24 h of Intensive care unit (ICU) admission; ② patients who were discharged automatically (After resuscitation, patients discontinued treatment before reaching the discharge or death, opting to abandon care midway through.) without subsequent treatment information ; ③ patient records with missing core research data such as age, gender, dose of adrenaline used, time of return of spontaneous circulation (ROSC), underlying comorbidities, and laboratory test results within 24 h after resuscitation. After applying the aforementioned selection criteria, a total of 347 valid samples were ultimately included in the analysis (Fig. 1). The study protocol was approved by the Medical Ethics Committee of The First Affiliated Hospital of the University of Science and Technology of China, Anhui, China (ID: 2024-RE-216) in 2024.

### Research methods

The Dryad database software (URL: https://DATADryad.org/) was first accessed. The search term "cardiac arrest" was entered in the search box to retrieve the research data. The final report by Iesu E [9] and the associated research data were obtained. The original research report indicated that cardiac arrest can lead to liver and kidney dysfunction in patients and analyzed the relevant factors contributing to acute liver dysfunction caused by hypoxic hepatitis. However, there has been no systematic analysis and summary of acute kidney dysfunction occurring after resuscitation in patients with cardiac arrest. Therefore, this study extracted data from the research data by Iesu E [9] to obtain clinical data such as patient age, gender, dose of adrenaline used, ROSC time, underlying comorbidities, laboratory test indicators within 24 h after

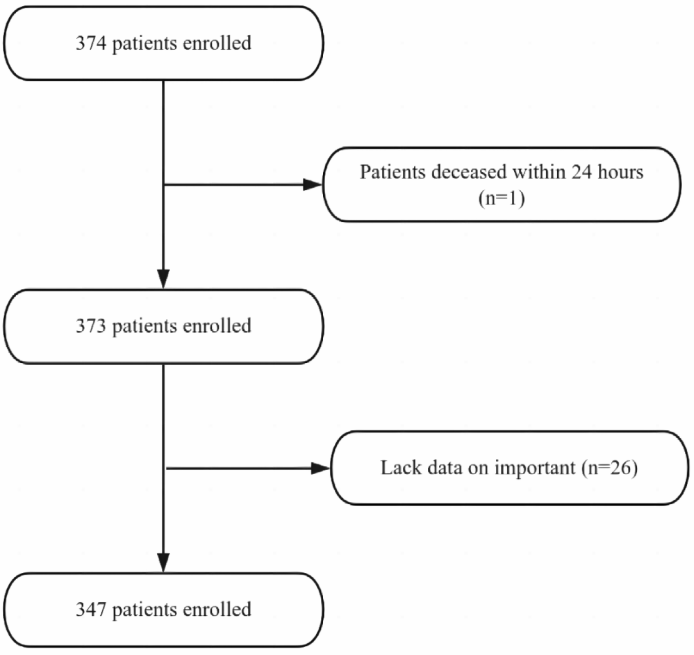


**Fig. 1** Study fow chart

resuscitation, Sepsis-related organ failure assessment (SOFA) score, Acute Physiology and Chronic Health Evaluation (APACHE) II score, and the incidence of acute kidney dysfunction. Based on the occurrence of acute kidney dysfunction (AKI) during the recovery stage after cardiac arrest, patients were divided into AKI group and non-AKI group, and the baseline data of the two groups were compared. The clinical diagnostic criteria for acute kidney dysfunction [10] are as follows: ① an increase in serum creatinine (SCr) by ≥0.3 mg/dl (≥26.5 μmol/L) within 48 h; ② SCr increases to >1.5 times the baseline value within 7 days, either known or presumed to be due to renal injury; ③ urine output<0.5 ml/(kg·h) for a duration of 6 h.

### Statistical methods

Data analysis was performed using SPSS 22.0 software and R Studio 4.3.2 software in this study. For continuous variables, the data were expressed as mean±standard deviation or median (interquartile range) depending on whether the data variables followed a normal distribution. Independent sample t-test or Mann-Whitney U test was used for between-group comparisons based on the normality of the continuous variables. Categorical variables were presented as counts and percentages, and Pearson's chi-squared test was used for between-group comparisons.Variables with statistical significance in the univariate analysis were included in the multivariable logistic regression model to explore the independent factors associated with acute kidney dysfunction after resuscitation in cardiac arrest patients. The independent factors identified by the multivariable logistic regression model were then imported into R Studio 4.3.2 software, and the "rms" package was used to plot the calibration curve. To prevent overfitting of the model, the established calibration curve model was validated according to the statement of the TRIPOD prediction model [11]. Bootstrap resampling was performed 1000 times for internal validation of the calibration curve model, and the predictive ability of the model was evaluated using the C-Index and the area under the receiver operating characteristic (ROC) curve (AUC) after internal validation. The goodness-of-fit of the model was assessed using the Hosmer-Lemeshow test and the calibration curve to evaluate the prediction consistency and calibration of the calibration curve model. In this study, a significance level of $P<0.05$ was considered statistically significant for all analyses.

## Result

### Univariate analysis of acute kidney dysfunction after resuscitation in cardiac arrest patients

The results of this study showed that among the 347 cardiac arrest patients who were successfully resuscitated, 197 patients developed acute kidney dysfunction during the recovery process, while 150 patients did not. The results of the univariate analysis revealed statistically significant differences (all $P<0.05$) in the following factors: presence of bystander witness, use of extracorporeal membrane oxygenation (ECMO), post-resuscitation shock, return of spontaneous circulation (ROSC), adrenaline dosage, C-reactive protein (CRP), lactate

dehydrogenase (LDH), alkaline phosphatase (ALP), total bilirubin (BilTOT), activated partial thromboplastin time (aPTT), prothrombin time (PT), international normalized ratio (INR), and mean arterial pressure (MAP). Please refer to Table 1 for detailed information.

### Multivariate logistic regression analysis of acute kidney injury occurrence in patients after cardiac arrest resuscitation

In the multivariate logistic regression analysis, 13 variables that showed statistical significance in the univariate analysis were included as independent variables, and the occurrence of acute kidney injury (AKI) during the treatment and recovery stage after resuscitation was included as the dependent variable. The results of the multivariate regression analysis revealed that shock status, CRP, LDH, and ALP were independent factors that significantly influenced the occurrence of acute kidney injury in patients after cardiac arrest resuscitation (all with $P<0.05$). Please refer to Table 2 for detailed results.

### Construction of a nomogram prediction model for acute kidney injury occurrence in patients after cardiac arrest resuscitation

In the study, the logistic regression analysis model was used to identify four independent factors that showed statistical significance. These factors were then selected as predictive variables. The occurrence of acute kidney injury during the treatment and recovery stage after resuscitation was considered as the outcome variable. The regression coefficients of the independent factors from the logistic regression analysis were incorporated into the R Studio 4.3.2 software using the "rms" package for visual data analysis and construction of a nomogram prediction model for acute kidney injury occurrence in patients after cardiac arrest resuscitation (Fig. 2). The clinical interpretation of this nomogram is as follows: Locate the corresponding target values on the coordinates of each influencing factor in the nomogram prediction model and draw a vertical line upwards to obtain the specific score on the "Point" axis at the top. Add up the specific score values from all the influencing factors in the "Point" axis to obtain the total score ("Total Point"). Find the corresponding value on the "Total Point" axis and draw a vertical line downwards to intersect with the "Risk" axis, which yields the risk probability of acute kidney injury occurrence in patients after cardiac arrest resuscitation.For example, if a female patient has a CRP value of 400, LDH value of 400, ADL value of 150 in blood tests, and did not experience shock symptoms during the process of cardiac arrest resuscitation, then the total score ("Total Point") for this patient would be $40+12+22+0=74$. The corresponding risk value ("Risk") would be 0.82, indicating that the clinical incidence rate of acute kidney injury occurrence after cardiac arrest resuscitation in this patient is 82%. Please refer to Fig. 1 for detailed results.

### Clinical evaluation of the clinical effect of the nomogram prediction model for acute kidney injury in patients who experience cardiac arrest and are successfully resuscitated

In this study, the ROC curve was used to evaluate and analyze the constructed Nomogram prediction model. To prevent overfitting of the nomogram model, bootstrap resampling (1000 times) was employed for internal validation to mitigate the impact of overfitting on its predictive stability. The ROC curves of the nomogram model before and after internal validation were plotted using the "pROC" package (Figs. 3 and 4). According to the ROC curves, the AUC of the nomogram model before and after internal validation were 0.713 and 0.712, respectively, indicating that the nomogram model possesses good discriminative ability. See Fig. 2 for details. The calibration curve showed that the predictive model constructed in this study has good fit and internal consistency. See Fig. 5 for details. The clinical applicability of the Nomogram prediction model was statistically analyzed using DCA to construct decision curves. The results showed that when the threshold for acute kidney injury in cardiac arrest patients after successful resuscitation was between 0.17 and 0.95, the net benefit of the Nomogram prediction model constructed in this study was positive, suggesting that it has good clinical adaptability. See Fig. 6 for details.

## Discussion

Cardiac arrest (CA) is one of the major public health concerns that threaten people's life and health [1]. Early respiratory and circulatory support, as well as the use of relevant medications, are among the key factors influencing the success of patient resuscitation [12]. According to Gomes et al. [12], for each minute of delay in early respiratory and circulatory support after the onset of CA, the patient's survival rate decreases by 7–10%. Additionally, Hajbaghery et al. [13] found that patients who received respiratory and circulatory support within 6 min of CA accounted for 97.5% of all surviving patients. After experiencing cardiac arrest, patients suffer from the loss of cardiac pumping function, leading to ischemia and hypoxia in vital organs such as the heart and brain. Without prompt and effective intervention, irreversible damage may occur in these vital organs within minutes [3]. Morigi et al. [7] demonstrated that post-cardiac arrest organ ischemia and hypoxia can result in multiple organ dysfunction, with the kidneys being subjected to continuous ischemia and hypoxia. Even effective CPR cannot meet the renal blood supply demand. Prolonged renal ischemia and hypoxia can induce the release of various inflammatory substances, causing endothelial cell injury

**Table 1** Univariate analysis results of acute kidney injury occurrence in patients after cardiac arrest resuscitation

| Variable | Total (*n* = 347) | No-AKI (*n* = 150) | AKI (*n* = 197) | Statistic | *P* |
|---|---|---|---|---|---|
| **Sex** | | | | | |
| Male | 94(27.089) | 45(30.000) | 49(24.873) | 1.133 | 0.287 |
| Female | 253(72.911) | 105(70.000) | 148(75.127) | | |
| **Witnessed event** | | | | | |
| NO | 49(14.121) | 28(18.667) | 21(10.660) | 4.502 | 0.034 |
| YES | 298(85.879) | 122(81.333) | 176(89.340) | | |
| **Immediate CPR** | | | | | |
| NO | 111(31.988) | 52(34.667) | 59(29.949) | 0.871 | 0.351 |
| YES | 236(68.012) | 98(65.333) | 138(70.051) | | |
| **Non-cardiac cause** | | | | | |
| NO | 207(59.654) | 85(56.667) | 122(61.929) | 0.980 | 0.322 |
| YES | 140(40.346) | 65(43.333) | 75(38.071) | | |
| **Non-shockable rhythm** | | | | | |
| NO | 147(42.363) | 69(46.000) | 78(39.594) | 1.431 | 0.232 |
| YES | 200(57.637) | 81(54.000) | 119(60.406) | | |
| **Chronic heart failure** | | | | | |
| NO | 271(78.098) | 124(82.667) | 147(74.619) | 3.224 | 0.073 |
| YES | 76(21.902) | 26(17.333) | 50(25.381) | | |
| **Hypertension** | | | | | |
| NO | 198(57.061) | 90(60.000) | 108(54.822) | 0.932 | 0.334 |
| YES | 149(42.939) | 60(40.000) | 89(45.178) | | |
| **Coronary artery disease** | | | | | |
| NO | 208(59.942) | 95(63.333) | 113(57.360) | 1.265 | 0.261 |
| YES | 139(40.058) | 55(36.667) | 84(42.640) | | |
| **Diabetes** | | | | | |
| NO | 262(75.504) | 117(78.000) | 145(73.604) | 0.890 | 0.346 |
| YES | 85(24.496) | 33(22.000) | 52(26.396) | | |
| **COPD** | | | | | |
| NO | 290(83.573) | 123(82.000) | 167(84.772) | 0.477 | 0.490 |
| YES | 57(16.427) | 27(18.000) | 30(15.228) | | |
| **Previous neurological disease** | | | | | |
| NO | 295(85.014) | 127(84.667) | 168(85.279) | 0.025 | 0.874 |
| YES | 52(14.986) | 23(15.333) | 29(14.721) | | |
| **Liver cirrhosis** | | | | | |
| NO | 331(95.389) | 145(96.667) | 186(94.416) | 0.981 | 0.322 |
| YES | 16(4.611) | 5(3.333) | 11(5.584) | | |
| **IABP** | | | | | |
| NO | 325(93.660) | 142(94.667) | 183(92.893) | 0.451 | 0.502 |
| YES | 22(6.340) | 8(5.333) | 14(7.107) | | |
| **ECMO** | | | | | |
| NO | 310(89.337) | 141(94.000) | 169(85.787) | 6.030 | 0.014 |
| YES | 37(10.663) | 9(6.000) | 28(14.213) | | |
| **Shock** | | | | | |
| NO | 172(49.568) | 99(66.000) | 73(37.056) | 28.539 | < 0.001 |
| YES | 175(50.432) | 51(34.000) | 124(62.944) | | |
| **Acute liver failure** | | | | | |
| NO | 163(46.974) | 79(52.667) | 84(42.640) | 3.437 | 0.064 |
| YES | 184(53.026) | 71(47.333) | 113(57.360) | | |
| **Age** | 62.000[52.000,75.000] | 61.000[52.000,73.000] | 65.000[51.000,75.000] | -1.103 | 0.270 |
| **Weight** | 77.000[68.000,85.000] | 75.000[65.000,85.000] | 77.000[70.000,85.000] | -1.027 | 0.303 |
| **ICU Day** | 4.000[3.000,9.000] | 4.000[3.000,8.000] | 5.000[3.000,10.000] | -1.401 | 0.157 |
| **ROSC** | 15.000[7.000,25.000] | 10.000[5.000,21.000] | 15.000[8.000,25.000] | -2.171 | 0.030 |
| **Epinephrine** | 3.000[1.000,5.000] | 2.000[1.000,5.000] | 4.000[2.000,6.000] | -2.768 | 0.005 |

**Table 1** (continued)

| Variable | Total ($n=347$) | No-AKI ($n=150$) | AKI ($n=197$) | Statistic | *P* |
|---|---|---|---|---|---|
| **CRP** | 36.000[12.000,80.000] | 26.000[6.000,52.000] | 50.000[20.000,100.000] | -4.821 | < 0.001 |
| **$ScvO_2/SvO_2$** | 69.000[63.800,74.400] | 69.400[64.500,74.500] | 68.500[63.600,74.300] | 0.920 | 0.358 |
| **GOT** | 86.000[44.000,169.000] | 87.000[45.000,178.000] | 81.000[42.000,164.000] | 0.210 | 0.834 |
| **GPT** | 62.000[31.000,128.000] | 62.000[32.000,123.000] | 59.000[31.000,128.000] | 0.303 | 0.762 |
| **LDH** | 326.000[234.000,451.000] | 295.000[222.000,407.000] | 352.000[244.000,505.000] | -2.737 | 0.006 |
| **ALP** | 76.000[58.000,104.000] | 71.000[54.000,89.000] | 82.000[62.000,114.000] | -3.202 | 0.001 |
| **GGT** | 68.000[43.000,103.000] | 73.000[44.000,101.000] | 66.000[41.000,103.000] | 0.613 | 0.540 |
| **BilTOT** | 0.510[0.330,0.860] | 0.480[0.300,0.730] | 0.570[0.350,0.970] | -2.397 | 0.017 |
| **aPTT** | 32.100[27.200,42.800] | 30.600[25.800,37.500] | 33.800[28.600,46.500] | -3.279 | 0.001 |
| **PT** | 64.386 ± 22.597 | 68.733 ± 22.599 | 61.076 ± 22.027 | 3.163 | 0.002 |
| **INR** | 1.250[1.110,1.500] | 1.210[1.070,1.410] | 1.300[1.150,1.590] | -3.112 | 0.002 |
| **PLT** | 202.000[141.000,269.000] | 208.000[160.000,285.000] | 188.000[122.000,259.000] | 2.411 | 0.016 |
| **Glu** | 202.000[157.000,291.000] | 196.000[162.000,268.000] | 210.000[152.000,300.000] | -0.927 | 0.354 |
| **pH** | 7.300[7.220,7.380] | 7.300[7.220,7.390] | 7.300[7.200,7.380] | 0.834 | 0.404 |
| **$PO_2$** | 111.000[85.000,178.000] | 114.000[89.000,173.000] | 109.000[83.000,183.000] | 0.730 | 0.466 |
| **$PCO_2$** | 38.000[33.000,44.000] | 38.000[34.000,44.000] | 37.000[32.000,44.000] | 1.944 | 0.052 |
| **MAP** | 87.000[76.000,105.000] | 93.000[80.000,107.000] | 83.000[74.000,103.000] | 2.699 | 0.007 |
| **APACHE II** | 24.000[20.000,29.000] | 24.000[19.000,29.000] | 25.000[21.000,29.000] | -0.905 | 0.365 |
| **SOFA** | 11.098 ± 3.493 | 10.840 ± 3.595 | 11.294 ± 3.400 | -1.200 | 0.231 |

IABP: Intra-aortic balloon pump; ECMO: Extracorporeal membrane oxygenation; ICU: Intensive care unit; ROSC: Restoration of spontaneous circulation; CRP: C reactive protein ; GOT: Glutamic-oxal(o)acetic transaminase; GPT: Glutamic-pyruvic transaminase; LDH: Lactate dehydrogenase; ALP: Alkaline phosphatase; GGT: gamma-glutamyl transferase ; BilTOT: otal bilirubin; aPTT: activated partial thromboplastin time; PT: prothrombin time; INR: international normalized ratio; MAP: mean arterial pressure; PLT: Platelet; APACHE II: Acute Physiology and Chronic Health Evaluation II; SOFA: Sepsis-related organ failure assessment

**Table 2** Multivariate logistic regression analysis of acute kidney injury occurrence in patients after cardiac arrest resuscitation

| Variable | B | Beta | Wald | *P* | Odds ratio | 95%CI |
|---|---|---|---|---|---|---|
| Shock | 0.957 | 0.238 | 16.211 | <0.001 | 2.605 | 1.634 ~ 4.151 |
| CRP | 0.004 | 0.002 | 4.196 | 0.041 | 1.004 | 1.000 ~ 1.008 |
| LDH | 0.001 | 0.001 | 4.298 | 0.038 | 1.001 | 1.000 ~ 1.003 |
| ALP | 0.007 | 0.003 | 5.811 | 0.016 | 1.007 | 1.001 ~ 1.012 |
| Constant (quantity) | -1.506 | 0.337 | 19.965 | 0.000 | 0.222 | |

CRP: C reactive protein; LDH: Lactate dehydrogenase; ALP: Alkaline phosphatase

and impairing the reabsorption function of renal tubules. In this study, clinical data of cardiac arrest patients obtained from the Dryad database were retrieved for secondary statistical analysis. Logistic regression analysis was conducted to explore the relevant factors influencing the occurrence of acute kidney injury in post-cardiac arrest patients and to construct a clinical prediction model. The results of the study analysis showed that post-resuscitation shock status, CRP, LDH, and PAL were clinical predictors of acute kidney injury in post-cardiac arrest patients. The AUC value indicated that the clinical prediction model constructed in this study had good clinical predictive performance.

The analysis results of this study indicate that CRP levels are an independent factor influencing the occurrence of acute kidney injury in patients after cardiac arrest. CRP is a protein synthesized by the liver that can induce an inflammatory response in the body [14]. Zhu et al. [15] found that serum inflammatory markers can reflect the occurrence probability of various clinical complications and adverse outcomes after cardiopulmonary resuscitation (CPR) in cardiac arrest patients to some extent, which is consistent with the analysis results of this study. Ramesh et al. [16] showed that CRP levels increase progressively in myocardial infarction, cardiac arrest, and various organ injuries, promoting the further development of the inflammatory response. Serum CRP levels begin to rise within approximately 4–6 h after tissue or organ injury and exhibit an exponential increase trend within 48 h. Studies by Tanveer et al. and Liu D et al. [17, 18] have shown a correlation between serum CRP levels and left ventricular ejection fraction in patients with myocardial injury. After sudden cardiac arrest, the body activates inflammatory responses due to ischemia and hypoxia, causing further damage to myocardial cells. With prolonged ischemia and hypoxia, the secretion of inflammatory substances increases, exacerbating the damage to myocardial cells and gradually impeding cardiac ejection function. Studies indicate that patients with lower left ventricular ejection fraction exhibit

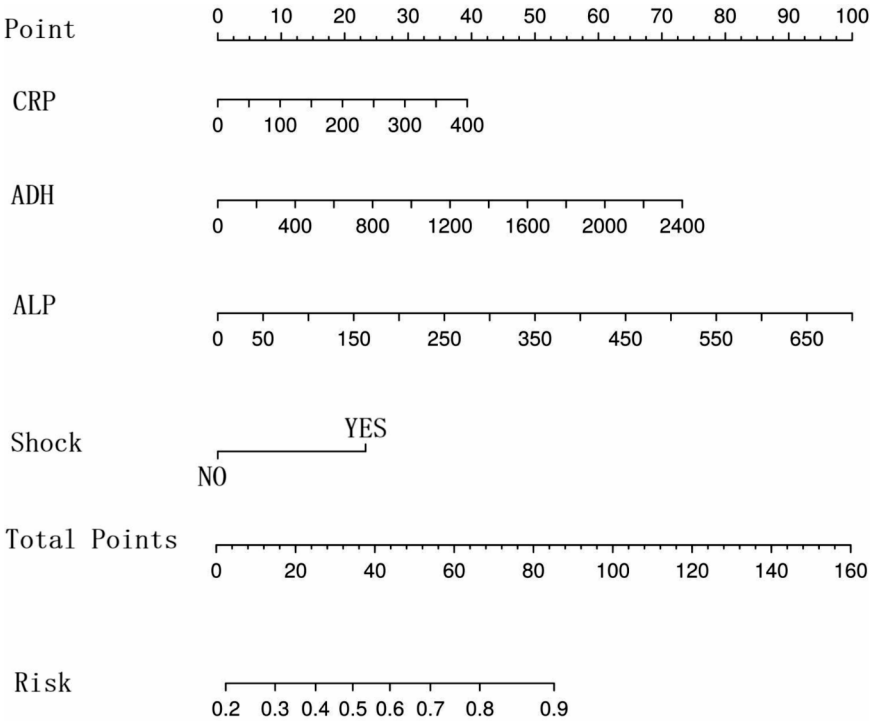


**Fig. 2** Predicts the Nomogram prediction model of acute renal function injury after resuscitation in patients undergoing cardiac arrest. *CRP (C reactive protein ); LDH (Lactate dehydrogenase); ALP (Alkaline phosphatase)

significantly elevated serum CRP levels, which directly reflect cardiac output and myocardial function [17, 18]. Patients with low left ventricular ejection fraction exhibit significantly elevated serum CRP levels. Left ventricular ejection fraction directly reflects cardiac output and myocardial function. When the left ventricular ejection fraction decreases, the myocardial function and cardiac output per beat decrease, resulting in inadequate systemic perfusion, continuous ischemia and hypoxia in organs such as the liver and kidneys, and subsequently leading to acute liver and kidney dysfunction. Therefore, in clinical practice, early anti-inflammatory therapy tailored to the patient's condition is crucial to control and reduce the secretion and release of inflammatory substances. This alleviates the damage to myocardial cells caused by inflammatory substances and further improves myocardial function, indirectly safeguarding the blood perfusion levels of vital organs in patients. Lactate dehydrogenase (LDH) is a cytoplasmic enzyme widely expressed in tissues. It is associated with the body's gluconeogenesis response. Studies have shown that when tissues or organs are in a hypoxic state, LDH reacts chemically with acetoacetate in the body to convert it into lactate [19]. After cardiac arrest, the heart loses its pumping function, and systemic circulation cannot be maintained. The kidneys are in a state of continuous ischemia and hypoxia, even if effective CPR cannot meet the kidney's blood supply needs. As ischemia and hypoxia persist, a series of inflammatory mediators are released in the kidneys, causing damage to the endothelial cells of the renal vasculature and impairing the reabsorption function of renal tubules [6, 7]. Due to the continued ischemia and reperfusion injury in the kidneys after successful resuscitation, LDH is secreted in large quantities in the kidneys, inducing the further conversion of acetoacetate into lactate, leading to aggravated renal damage [20]. Hence, in clinical practice, it is essential to monitor lactate dehydrogenase (LDH) levels to assess the accumulation of lactate in the kidneys and employ measures such as urine alkalinization to neutralize the accumulated lactate, thereby improving the renal microenvironment. Alkaline phosphatase (ALP) is an isoenzyme that hydrolyzes into phosphate in an alkaline environment. Recent studies have shown that under certain conditions, ALP can prevent endothelial dysfunction, oxidative stress, inflammation, vascular damage, calcification, and other conditions, and may be involved in the development of cardiovascular and cerebrovascular diseases. There is a certain degree of association between ALP and multiple kidney diseases such as glomerulonephritis, nephrotic syndrome, and renal dysfunction [21–24]. Research results have shown that damaged renal tubular cells can increase serum ALP levels, indicating that this characteristic may serve as an indicator for kidney disease and acute kidney injury. Under physiological conditions, ALP is also expressed on the endothelial cells,

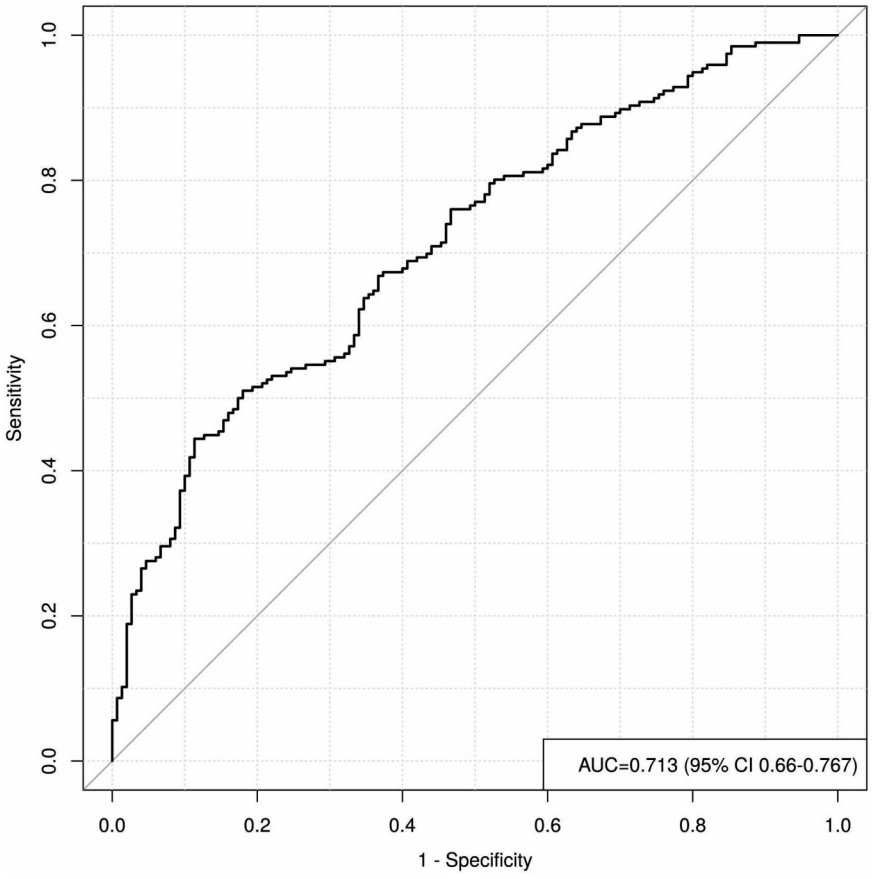


**Fig. 3** ROC curves of the nomogram mode. *AUC(area under the curve )

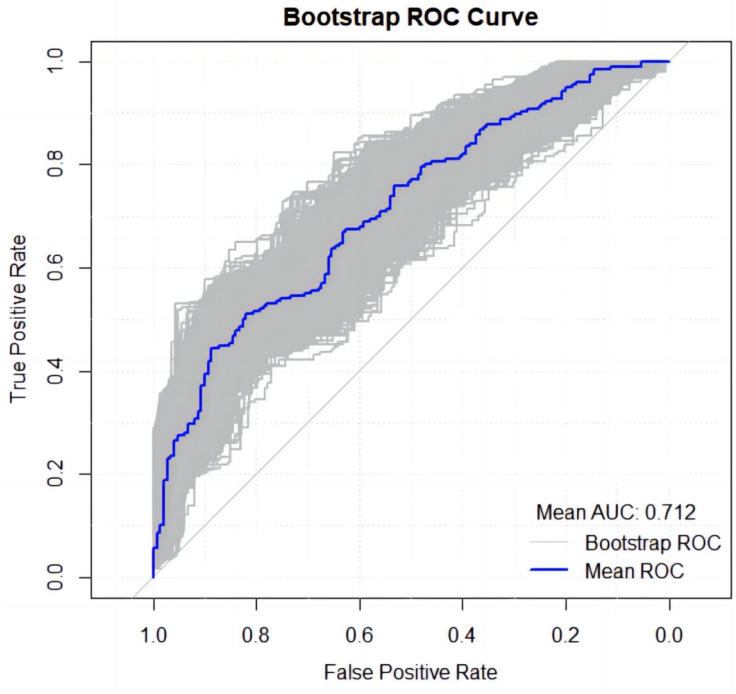


**Fig. 4** ROC curves after internal validation of the nomogram mode. *AUC(area under the curve )

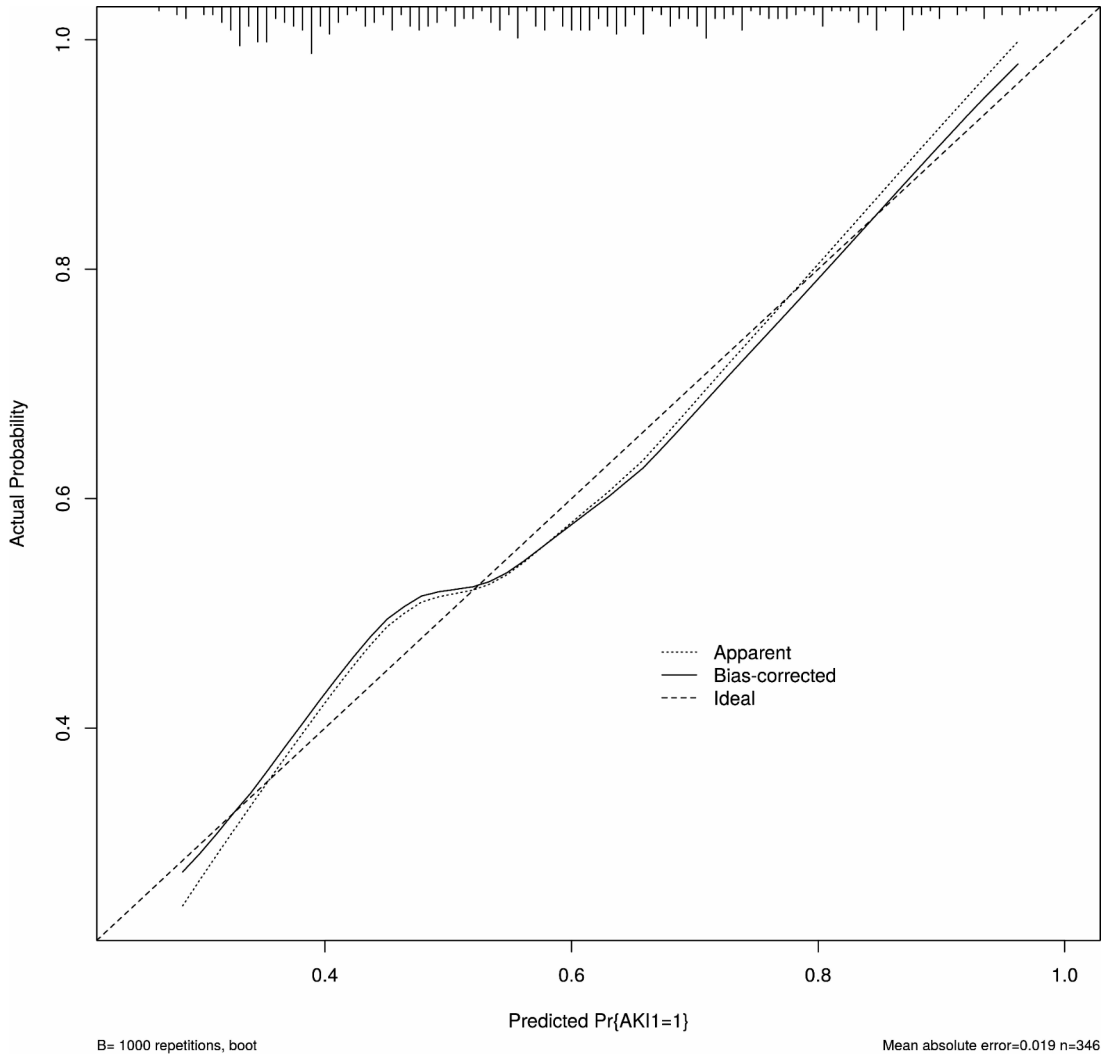


**Fig. 5** Calibration curve of the Nomogram prediction model

mesangial cells, and epithelial cells of Bowman's capsule, which may increase the excretion of ALP in high filtration. However, the specific mechanisms are not yet clear and require continuous exploration and verification through future basic research projects [25–28]. Post-resuscitation shock refers to the condition where, following successful cardiopulmonary resuscitation (CPR) after cardiac arrest, some patients develop cardiogenic shock. Its mechanism involves reduced cardiac output, systemic vasoconstriction, cardiac ischemia, inadequate perfusion of vital organs, and tissue hypoxia. Clinically, it manifests as hypotension, ineffectiveness of fluid resuscitation, and is characterized by inadequate perfusion of end-organ tissues, requiring pharmacological or mechanical intervention [29, 30]. The analysis of this study shows that the sustained shock state after successful resuscitation in cardiac arrest patients is also a major influencing factor for the occurrence of acute kidney injury. This is because the continuous shock state of the body leads to sustained ischemia and hypoxia in vital organs such as the heart, brain, liver, and kidneys, inducing oxidative stress and the synthesis and release of inflammatory mediators, resulting in disruption of cellular polarity. This reaction occurring in the kidneys increases tubular permeability, allowing large molecules to enter the tubules and cause acute kidney injury [8]. Therefore, in clinical practice, after cardiopulmonary resuscitation, it is crucial to focus on managing organ perfusion levels and to relieve shock status in patients through appropriate and proactive volume expansion and vasopressor therapy, thereby improving the blood perfusion levels of vital organs.

The nomogram prediction model is a popular visual prediction model and clinical screening tool in recent years. It visualizes data based on the parameters of a conventional regression model, resulting in a concise and easy-to-understand visual assessment tool that facilitates personalized dynamic assessment of patients by clinical healthcare workers [31]. Relevant research

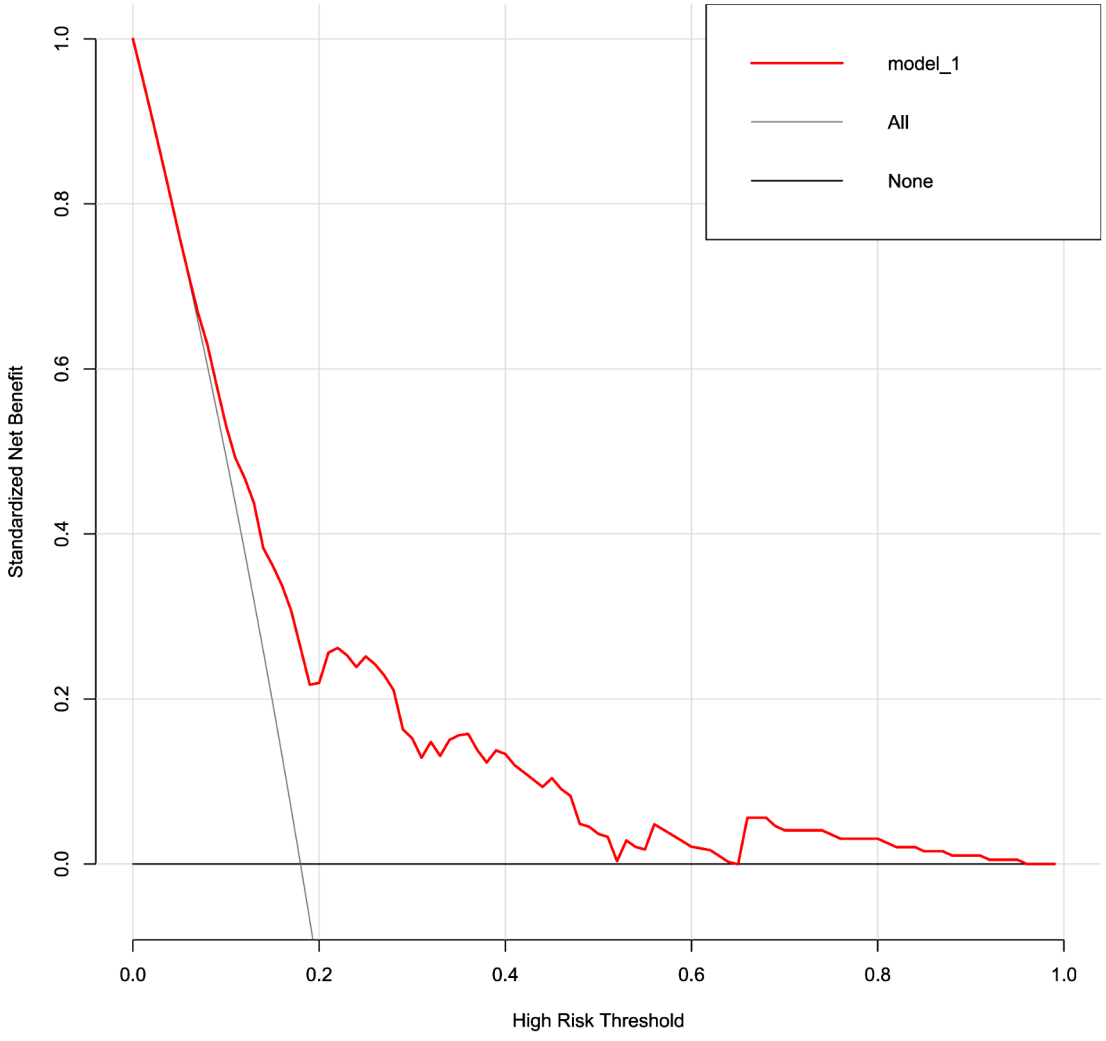


**Fig. 6** The DCA curve of the Nomogram prediction model

results have shown that the nomogram prediction model can effectively evaluate and predict short-term neurological prognosis in successfully resuscitated cardiac arrest patients [32]. Additionally, this model also demonstrates good clinical value in predicting the impact of vasopressor doses on long-term neurological prognosis in cardiac arrest patients [33]. However, there are currently no reported studies on risk prediction assessment tools for acute kidney injury after successful resuscitation in cardiac arrest patients, both domestically and internationally. Therefore, this study conducted a search and downloaded relevant data from the Dryad database software, and successfully constructed a risk prediction model for acute kidney injury after resuscitation in cardiac arrest patients through secondary data analysis. The constructed prediction model has good clinical applicability, accuracy, and internal consistency. The predictive indicators in this model are common clinical data indicators that are relatively easy to obtain. Thus, this prediction model can effectively assist clinical staff in individualized assessment and judgment of patient conditions.

## Conclusions

The nomogram model integrates multiple clinical variables to provide personalized risk assessment for each patient. This capability for personalized prediction enables doctors to more accurately assess the risk of developing specific diseases in patients, thereby facilitating the adoption of tailored intervention measures. Post-resuscitation shock status, CRP, LDH, and ALP are the influencing factors for AKI after resuscitation in CA patients. The clinical prediction model constructed based on the above indicators has good clinical discriminability and practicality. Clinicians can utilize this predictive model in clinical practice to estimate the probability of AKI occurring post-resuscitation in cardiac arrest patients. High-risk patients for AKI can

be identified through this model, enabling early intervention strategies such as real-time assessment of monitoring indicators, adjustment of pharmacological interventions, optimization of fluid management strategies, and early application of CRRT. These interventions aim to promptly identify and intervene, thereby reducing the incidence of AKI or alleviating its symptoms post-resuscitation, promoting early recovery and improving clinical outcomes for patients.

## Limitations of the study

The study has certain limitations that need to be further addressed and improved in subsequent research. 1. As this is a retrospective study, it lacks clinical data on patient lifestyles and habits. Additionally, being a single-center retrospective study, it is constrained by factors such as geographical location, cultural differences, and lifestyle habits, and thus cannot fully represent the treatment processes of patients nationwide. However, it can serve as clinical reference material to guide clinical practice. Additionally, due to limitations of publicly available database data, factors such as patient stress response index, body mass index, and triglyceride glucose product index were not included in this study. 2.This study involves a secondary analysis of public databases, thus it is limited by the content available within these databases. The study did not account for the relationship between each stage of acute kidney injury and various baseline indices. In future research endeavors, we aim to overcome these limitations by establishing a comprehensive, large-scale public database through dedicated funding and administrative interventions, encompassing multiple regions, centers, samples, comprehensive follow-ups, and diverse patient populations. Collaborations with other healthcare institutions will be sought to gather additional data and apply the model across various regions, healthcare settings, and patient cohorts to ensure robustness and generalizability of its performance.

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-024-04110-8.

Supplementary Material 1

Supplementary Material 2

### Author contributions

SBH, LXZ, and HZJ were responsible for drafting the manuscript, designing the study, and analyzing the data. TTZ, MH, YJ, QQS, and MZ conducted data verification and cross-checking of the manuscript. MD provided support in human resources and contributed to the study design. All authors have read and approved the final manuscript.

### Funding

The study was conducted spontaneously by the investigator without any official funding.

### Data availability

Data is provided within the manuscript or supplementary information files.

## Declarations

### Ethical approval

This study has obtained ethical exemption review from the First Affiliated Hospital of the University of Science and Technology of China (ID: 2024-RE-25), and this study is a retrospective study of a public database and therefore does not require additional participants to provide written consent.

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

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## Publisher's Note