Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Correlation between rewarming rate during cardiopulmonary bypass and postoperative acute kidney injury : a retrospective analysis

Shuang Xu
Nanjing Medical University
Danping Shen
Nanjing Medical University
LiYin Wang
Nanjing Medical University
ShaSha Zhou
Nanjing Medical University
Lei Zhang
Nanjing Medical University
Jie Shi
838096313@qq.com
Nanjing Medical University
Yun Wang
Nanjing Medical University

Research Article

Keywords: Extracorporeal circulation, Rewarming rate, Acute kidney injury, Cardiac surgery, Prediction model

Posted Date: November 11th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10314476/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Objective: To investigate the correlation between the rewarming rate during cardiopulmonary bypass (CPB) and the incidence of acute kidney injury (AKI) following open heart surgery.

Methods: Clinical data from 348 patients who underwent open heart surgery with CPB at the Department of Cardiac and Vascular Surgery, Nanjing Hospital Affiliated to Nanjing Medical University, between January 2020 and December 2023 were retrospectively analyzed. Patients were divided into slow rewarming (≤ 0.09 °C/min) and rapid rewarming (> 0.09 °C/min) groups based on the rewarming rate during CPB. The primary endpoint was the incidence and severity of AKI within 7 days postoperatively. Preoperative baseline characteristics, intraoperative variables, and postoperative outcomes were collected and compared between the two groups. Univariate analysis and multivariate logistic regression were performed to identify independent risk factors for postoperative AKI. A nomogram for predicting postoperative AKI risk was subsequently developed. Model discrimination was assessed using receiver operating characteristic (ROC) curves, calibration was evaluated via the Hosmer–Lemeshow test and calibration plots, and clinical net benefit was examined using decision curve analysis (DCA).

Results :A total of 348 patients were included, of whom 58 developed postoperative AKI, yielding an incidence of 16.7%. Compared with the slow rewarming group, the rapid rewarming group had a significantly higher incidence of AKI (23.1% vs. 10.8%, P<0.01) and more severe AKI clinical stages. Multivariate logistic regression revealed that rapid rewarming rate (OR=2.15, 95% CI:1.52–3.04), prolonged CPB time (per 10-minute increase, OR=1.18, 95% CI:1.05–1.33), decreased preoperative estimated glomerular filtration rate (per 10 mL/min/1.73 m² decrease, OR = 1.32, 95% CI: 1.12–1.56), and increased intraoperative red blood cell transfusion (per 1-unit increase, OR=1.09, 95% CI:1.02–1.17) were independent risk factors for postoperative AKI.

Conclusion :Rapid rewarming during CPB is an independent risk factor for AKI after cardiac surgery. The risk prediction model constructed from key indicators can effectively identify patients at high risk for postoperative AKI and enable early recognition of such patients.

Introduction

Acute kidney injury (AKI) is one of the most common and serious complications following open heart surgery with cardiopulmonary bypass (CPB). Characterized by a rapid postoperative decline in renal function, AKI not only prolongs hospital stays and increases healthcare costs but also substantially elevates the long-term risks of chronic kidney disease and mortality [1–2]. Epidemiological evidence indicates that the incidence of postoperative AKI in patients undergoing CPB-assisted cardiac surgery ranges from 15% to 30%, with approximately 5% to 10% of affected patients requiring renal replacement therapy. Moreover, once severe AKI develops, in-hospital mortality can exceed 50% [3–4]. Hence, early identification of at-risk individuals and timely implementation of targeted interventions are of considerable clinical importance for improving patient outcomes.

The non-physiological perfusion state during CPB has been consistently linked to the development of postoperative AKI. Among the modifiable intraoperative parameters, the rewarming rate has garnered increasing research interest in recent years [5]. Conventional wisdom holds that gradual rewarming helps preserve stable organ blood flow and attenuates ischemia-reperfusion injury; however, the precise relationship between rewarming rate and postoperative AKI remains inconclusive. Some studies suggest that rapid rewarming may disrupt the balance between tissue oxygen supply and demand, thereby activating inflammatory cascades and oxidative stress pathways, which could exacerbate renal injury [6–7]. In contrast, other investigators argue that the rewarming rate per se is not an independent risk factor, and that its effects are confounded by multiple factors, including CPB duration, transfusion volume, and patients' baseline renal function [8]. To date, few dedicated studies, either domestically or internationally, have specifically examined the association between rewarming rate and AKI after open heart surgery, and validated predictive models that quantify the risk attributable to rewarming rate remain scarce [9].

Against this backdrop, the present study retrospectively analyzed clinical data from patients who underwent open heart surgery with CPB. Patients were categorized into slow-rewarming and rapid-rewarming groups according to the rewarming rate during CPB. The primary objectives were to explore the correlation between rewarming rate and postoperative AKI, to identify independent risk factors, and to provide evidence-based support for optimizing CPB management strategies and reducing the risk of postoperative AKI in clinical practice.

Data and Methods

Study Population

Patients who underwent open heart surgery with cardiopulmonary bypass (CPB) at the Department of Cardiac and Vascular Surgery, Nanjing Hospital Affiliated to Nanjing Medical University, between January 2020 and December 2023 were enrolled. A total of 348 patients were included in the final analysis. Inclusion criteria were: (1) first-time open heart surgery with intraoperative CPB support; (2) age ≥ 18 years; and (3) complete clinical data, including intraoperative rewarming rate and preoperative and postoperative renal function parameters. Exclusion criteria were: (1) preoperative end-stage renal disease or chronic renal replacement therapy; (2) acute kidney injury within 7 days prior to surgery; (3) intraoperative death or death within 24 hours postoperatively; (4) history of kidney transplantation or unilateral/bilateral nephrectomy; and (5) incomplete clinical data precluding calculation of rewarming rate or assessment of postoperative AKI.

Grouping Strategy

Patients were stratified according to the mean rewarming rate during CPB, defined as the average rate of rewarming from the lowest nasopharyngeal temperature to the time of weaning from CPB (unit: °C/min).

The optimal cut-off value (0.09°C/min) was determined using the Youden index. Accordingly, patients were divided into two groups: the slow rewarming group (rewarming rate ≤ 0.09°C/min) and the rapid rewarming group (rewarming rate > 0.09°C/min).

Data Collection

The following data were retrospectively extracted from the hospital's electronic medical record system: (1) Preoperative baseline characteristics: including age, sex, body mass index, comorbidities (hypertension, diabetes, coronary artery disease, heart failure), and laboratory parameters (preoperative serum creatinine, estimated glomerular filtration rate [eGFR], and blood urea nitrogen); (2) Intraoperative variables: including CPB time, aortic cross-clamp time, minimum nasopharyngeal temperature, rewarming rate, rewarming duration, intraoperative transfusion volumes (red blood cells, plasma, platelets), surgical procedure type, intraoperative minimum blood pressure, and use of vasoactive agents; and (3) Postoperative parameters: including daily serum creatinine levels, urine output, need for renal replacement therapy within 7 days postoperatively, intensive care unit (ICU) length of stay, total hospital stay, and 30-day all-cause mortality.

Outcome Measures

Primary outcome: The incidence and severity of acute kidney injury within 7 days postoperatively. AKI was diagnosed and staged according to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 criteria [10]: Stage 1 – serum creatinine rise to 1.5–1.9 times baseline, or an increase of ≥ 26.5 μmol/L, or urine output < 0.5 mL/kg/h for 6–12 hours; Stage 2 – serum creatinine rise to 2.0–2.9 times baseline, or urine output < 0.5 mL/kg/h for ≥ 12 hours; Stage 3 – serum creatinine rise to ≥ 3.0 times baseline, or an increase to ≥ 353.6 μmol/L, or initiation of renal replacement therapy, or urine output < 0.3 mL/kg/h for ≥ 24 hours.

Secondary outcomes

included the rate of postoperative renal replacement therapy, ICU length of stay, total hospital stay, and 30-day all-cause mortality.

Statistical Analysis

Statistical analyses were performed using SPSS version 26.0 and R language version 4.2. Normally distributed continuous variables were expressed as mean ± standard deviation ($\bar{x}\pm s$)and compared between groups using the independent-samples t-test. Non-normally distributed variables were presented as median with interquartile range [M (Q1, Q3)] and compared using the Mann–Whitney U test. Categorical variables were expressed as frequencies and percentages [n (%)] and compared using the chi-square test or Fisher's exact test, as appropriate. Variables with $P < 0.05$ in univariate analysis were entered into a multivariate logistic regression model to identify independent risk factors for

postoperative AKI. The predictive performance of the model was evaluated by the area under the receiver operating characteristic (ROC) curve (AUC) to assess discriminative ability. Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test and calibration plots, with a well-calibrated model indicated by a non-significant Hosmer–Lemeshow test($P > 0.05$). A two-sided $P < 0.05$ was considered statistically significant.

Results

Comparison of baseline data in this group of patients

A total of 348 patients undergoing open heart surgery with cardiopulmonary bypass were included in this study, including 178 patients in the slow rewarming group and 170 patients in the rapid rewarming group. The preoperative baseline data of the two groups were compared in detail in Table 1. The results showed that there was no significant difference in age, sex, BMI, history of hypertension, history of diabetes, NYHA cardiac function classification, preoperative LVEF, preoperative serum creatinine, preoperative eGFR and surgical type between the two groups (all $P > 0.05$), suggesting that there was good comparability between the two groups.

Table 1
Comparison of preoperative baseline data between the two groups of patients

Variables	Slow rewarming group (n = 178)	Rapid rewarming group (n = 170)	Statistical value(t/χ^2/Z)	P value
Age (years)	58.4 ± 12.6	59.1 ± 13.2	-0.501	0.617
Male [n (%)]	102(57.3)	95(55.9)	0.073	0.787
BMI (kg / m2)	24.2 ± 3.5	23.9 ± 3.7	0.770	0.442
History of hypertension [n (%)]	76(42.7)	74(43.5)	0.024	0.877
History of diabetes [n (%)]	38(21.3)	35(20.6)	0.028	0.867
NYHA cardiac function grade ≥ III [n (%)].	45(25.3)	42(24.7)	0.016	0.900
Preoperative LVEF (%)	61.5 ± 8.2	60.9 ± 8.6	0.667	0.505
Preoperative serum creatinine (μmol / L).	82.5 ± 18.6	83.2 ± 19.4	-0.339	0.735
Preoperative eGFR (mL / min / 1.73m2).	86.3 ± 15.4	85.6 ± 16.1	0.409	0.683
Type of surgery [n (%)]			0.217	0.641
Coronary artery bypass grafting	82(46.1)	76(44.7)		
Valve surgery	71(39.9)	68(40.0)		
Other	25(14.0)	26(15.3)		

Note : BMI = body mass index ; nYHA = New York Heart Association ; lVEF = left ventricular ejection fraction ; eGFR = estimated glomerular filtration rate ; data are expressed as mean ± standard deviation or number of cases (percentage). **Comparison of postoperative AKI occurrence and staging**

A total of 58 patients developed AKI after surgery, with a total incidence of 16.7%. The incidence of postoperative AKI in the rapid rewarming group was significantly higher than that in the slow rewarming group [23.1% (39 / 170) vs.10.8% (19 / 178), P < 0.01]. In terms of AKI clinical stage, the rapid rewarming group was mainly KDIGO stage 2 and 3 (33.3% and 20.5%, respectively), while the slow rewarming group was mainly KDIGO stage 1 (73.7%). The difference in AKI stage distribution between the two groups was statistically significant (P < 0.05), suggesting that the severity of AKI in the rapid rewarming group was more severe. See Table 2 for details.

Table 2
Comparison of postoperative AKI occurrence and KDIGO staging between the two groups of patients

Variables	Slow rewarming group (n = 178)	Rapid rewarming group (n = 170)	Statistical value(χ^2/Z)	P value
Postoperative AKI occurred [n (%)].	19(10.8)	39(23.1)	9.152	0.002
Clinical stage of AKI [n (%)]			-2.462	0.014
Stage 1	14(73.7)	18(46.2)		
Stage 2	4(21.0)	13(33.3)		
Stage 3	1(5.3)	8(20.5)		

Note : AKI = acute kidney injury ; kDIGO = improve the prognosis of global kidney disease ; aKI staging was based on KDIGO criteria. The incidence of AKI was compared by χ2 test. Mann-Whitney U test was used to compare AKI staging. **Comparison of intraoperative data between the two groups of patients**

The intraoperative data of the two groups were compared in detail in Table 3. The results showed that the cardiopulmonary bypass time, aortic occlusion time and intraoperative red blood cell transfusion volume in the rapid rewarming group were higher than those in the slow rewarming group, and the differences were statistically significant ($P < 0.05$).

Table 3
Comparison of intraoperative data between the two groups of patients

Variables	Slow rewarming group (n = 178)	Rapid rewarming group (n = 170)	Statistical value($t/\chi^2/Z$)	P value
Operation time (min)	215.6 ± 45.3	223.8 ± 48.7	-1.630	0.104
Extracorporeal circulation time (min)	98.5 ± 28.6	115.2 ± 34.1	-4.982	< 0.001
Aortic occlusion time (min)	65.3 ± 21.4	74.8 ± 26.7	-3.640	< 0.001
The amount of crystal liquid (mL) was used during operation.	1520.4 ± 420.7	1560.8 ± 450.3	-0.862	0.389
The amount of colloidal fluid used during operation (mL)	850.6 ± 280.4	880.5 ± 295.6	-0.967	0.334
Intraoperative red blood cell transfusion (U)	1.0(0.0, 2.0)	2.0(1.0, 3.0)	-3.125	0.002
Intraoperative urine volume (mL)	1280.5 ± 420.8	1250.3 ± 440.6	0.650	0.516

Note : Normal distribution measurement data are expressed as mean ± standard deviation, and independent sample t test is used for comparison between groups ; the measurement data of non-normal distribution were expressed as median (interquartile range), and the Mann-Whitney U test was used for comparison between groups. The enumeration data were expressed as the number of cases (percentage), and the comparison between groups was performed by $\chi 2$ test or Fisher exact probability method.

Comparison of postoperative data between the two groups

The postoperative data of the two groups were compared in detail in Table 4. The results showed that the incidence of postoperative AKI, postoperative mechanical ventilation time, ICU stay time and postoperative hospital stay in the rapid rewarming group were higher than those in the slow rewarming group, and the differences were statistically significant ($P < 0.05$).

Table 4
Comparison of postoperative data between the two groups

Variables	Slow rewarming group (n = 178)	Rapid rewarming group (n = 170)	Statistical value(t/χ^2/Z)	P value
Postoperative AKI occurred [n (%)].	19(10.8)	39(23.1)	9.152	0.002
Postoperative mechanical ventilation time (h)	14.5(10.0, 22.0)	20.0(14.0, 36.0)	-3.486	< 0.001
ICU stay time (d)	2.0(1.5, 3.5)	3.0(2.0, 5.0)	-3.742	< 0.001
Postoperative hospital stay (d)	10.5 ± 4.2	13.8 ± 5.6	-6.185	< 0.001
Postoperative new-onset atrial fibrillation [n (%)]	32(18.0)	38(22.4)	1.034	0.309
Second exploratory thoracotomy [n (%)]	4(2.2)	6(3.5)	0.520	0.471
30-day postoperative death [n (%)].	2(1.1)	5(2.9)	1.394	0.238

Note : Normal distribution measurement data are expressed as mean ± standard deviation, and independent sample t test is used for comparison between groups ; the measurement data of non-normal distribution were expressed as median (interquartile range), and the Mann-Whitney U test was used for comparison between groups. The enumeration data were expressed as the number of cases (percentage), and the comparison between groups was performed by $\chi 2$ test or Fisher exact probability method. AKI = acute kidney injury ; ICU = intensive care unit.

Analysis of independent risk factors for postoperative AKI

Variables with statistical significance in univariate analysis (rapid rewarming rate, cardiopulmonary bypass time, preoperative eGFR, intraoperative red blood cell transfusion) were included in the multivariate logistic regression model (forward stepwise method) to analyze the independent risk factors of AKI after open heart surgery. The results show. Fast rewarming rate (OR = 2.15,95% CI : 1.52–3.04, P < 0.001), prolonged cardiopulmonary bypass time (every 10 minutes increase, OR = 1.18,95% CI: 1.05–1.33,P = 0.005), decreased preoperative eGFR (every 10 mL / min / 1.73m2 decrease, OR = 1.32,95% CI: 1.12–1.56, P = 0.001) and increased intraoperative red blood cell transfusion (every 1 unit increase, OR = 1.09,95% CI: 1.02–1.17, P = 0.014) were independent risk factors for postoperative AKI. See Table 5 for details.

Table 5
Results of multivariate Logistic regression analysis of postoperative AKI

Variables	β value	Standard error	Waldχ^2 value	OR value	95%CI	P value
Rapid rewarming rate (> 0.09°C / min)	0.765	0.176	18.895	2.15	1.52–3.04	< 0.001
Extracorporeal circulation time (every 10 minutes)	0.166	0.059	7.917	1.18	1.05–1.33	0.005
Preoperative eGFR (decreased by 10 mL / min / 1.73 m2).	0.278	0.086	10.451	1.32	1.12–1.56	0.001
Intraoperative red blood cell transfusion volume (for each additional 1 unit)	0.086	0.035	6.041	1.09	1.02–1.17	0.014

Note : eGFR = estimated glomerular filtration rate ; oR = odds ratio ; cI = confidence interval. The forward stepwise method (Forward : LR) was used to include the independent variables, the inclusion criteria was 0.05, and the exclusion criteria was 0.10. Hosmer-Lemeshow test : $\chi 2 = 6.32$, P = 0.612.

Establishment of AKI prediction model after cardiopulmonary bypass open heart surgery

The results of this study showed that the AUC of the curve of rapid rewarming rate, prolonged cardiopulmonary bypass time, decreased preoperative eGFR and increased intraoperative red blood cell transfusion volume was 0.732,95CI % was 0.565-0.900, and the Hosmer-Lemeshow test chi-square value was 13.666, P = 0.091, the model was stable, see Fig. 1.

Internal validation of the performance of AKI prediction model after open heart surgery under cardiopulmonary bypass

In this study, the accuracy of the prediction model was verified by the calibration curve. As shown in Fig. 2, when compared with the observed probabilities, the probability distribution predicted by the model is very close to the diagonal, indicating a high degree of agreement between the predicted and actual values. The R2 of goodness-of-fit test was 0.892, indicating that the model explained 89.2% of the observed variation, confirming its good calibration accuracy. These results show that the prediction model established in this study not only has clinical identification ability, but also is accurate and reliable, and the predicted risk is highly consistent with the actual risk. See Fig. 2.

Discussion

Open heart surgery with cardiopulmonary bypass (CPB) is a cornerstone therapeutic modality for valvular heart disease, coronary artery disease, and complex congenital heart defects. However,

postoperative acute kidney injury (AKI), as a common and serious complication, significantly prolongs hospital stays, escalates medical costs, and increases long-term mortality, thereby critically impacting surgical efficacy and patient prognosis [11–12]. Previous literature has reported that the incidence of AKI following cardiac surgery ranges from 15% to 45%, with its pathophysiology involving multifactorial mechanisms, including ischemia-reperfusion injury, systemic inflammatory response syndrome, oxidative stress, and hemodynamic disturbances [13–14]. In recent years, advances in CPB technology have brought increasing attention to the role of perioperative temperature management strategies in modulating postoperative renal function. Nevertheless, studies specifically examining the association between CPB rewarming rate and postoperative AKI remain relatively scarce, and simple yet effective clinical prediction tools are still lacking [15]. Therefore, investigating the impact of rewarming rate on postoperative AKI and establishing a reliable predictive model are of considerable significance for early identification of high-risk patients and optimization of perioperative management strategies.

In the present study, the overall incidence of AKI within 7 days postoperatively among 348 patients undergoing open heart surgery with CPB was 16.7%, which is broadly consistent with previous reports [16]. Notably, the rapid rewarming group exhibited a significantly higher incidence of AKI and more severe AKI stages compared to the slow rewarming group, suggesting that rapid rewarming not only increases the overall risk of AKI but also exacerbates the severity of renal injury. Several mechanisms may underlie this association. During the rewarming phase of CPB, rapid restoration of normothermia can precipitate sudden peripheral vasodilation, leading to relative hypovolemia and reduced renal perfusion pressure, which in turn diminishes glomerular filtration rate and exacerbates renal ischemia–hypoxia injury. Concurrently, rapid rewarming may amplify the systemic inflammatory response, activating neutrophil and complement cascades and triggering the release of large quantities of pro-inflammatory cytokines, such as tumor necrosis factor-α and interleukin-6, which directly damage renal tubular epithelial cells and induce apoptosis and necrosis [17]. Furthermore, excessively rapid rewarming can provoke post-ischemic oxidative stress and mitochondrial respiratory chain dysfunction, generating abundant reactive oxygen species that attack the lipid bilayer of tubular cell membranes, initiate lipid peroxidation chain reactions, and further aggravate cellular injury [18]. In addition, the renal medulla is exquisitely sensitive to hypoxia and is already in a hypoxic state during CPB. The pronounced hemodynamic fluctuations accompanying rapid rewarming exacerbate the oxygen supply–demand mismatch in the renal medulla, leading to aggravated energy metabolism disturbances in tubular epithelial cells and culminating in acute tubular necrosis. These findings are corroborated by previous studies, which have also confirmed a positive correlation between CPB rewarming rate and the incidence of postoperative AKI, indicating that rapid rewarming may impair renal function through multiple converging pathways [19].

Consistent with prior literature establishing the close relationship between intraoperative variables and postoperative AKI [20], our multivariate analysis identified prolonged CPB time, decreased preoperative estimated glomerular filtration rate (eGFR), and increased intraoperative red blood cell transfusion as independent risk factors for AKI after CPB surgery. The underlying mechanisms are as follows: prolonged CPB extends renal exposure to non-physiological perfusion, with continuous activation of

complement and inflammatory cells on the surface of non-biological circuit materials, along with microembolization and hemolysis, all of which may aggravate renal microcirculatory disturbances. Intraoperative red blood cell transfusion increases the risk of postoperative AKI, possibly due to the infusion of free hemoglobin, inflammatory mediators, and micropolymers present in stored blood, which can induce tubular injury and microcirculatory embolization. Moreover, blood transfusion itself often serves as a surrogate marker of heightened systemic inflammation and generally indicates more intraoperative bleeding or greater hemodynamic instability, indirectly contributing to an elevated AKI incidence [21]. Finally, reduced preoperative eGFR reflects an underlying decline in renal functional reserve, with impaired autoregulatory capacity. Such patients have a compromised ability to compensate for the non-pulsatile perfusion, hypothermia, and hemodynamic fluctuations during CPB rewarming, rendering them more susceptible to ischemia-reperfusion injury and thus significantly increasing the risk of postoperative AKI—a finding consistent with earlier research [22].

Currently, a growing consensus supports the utility of intraoperative data in predicting postoperative AKI with satisfactory performance. In this study, a nomogram model for postoperative AKI risk prediction was constructed incorporating four variables: rapid rewarming rate, CPB time, preoperative eGFR, and intraoperative red blood cell transfusion. The model achieved an AUC of 0.732, indicating good discriminatory ability. A major advantage of this prediction model lies in its practicality: all four included indicators are routinely recorded before or during surgery (the rewarming rate can be directly calculated from CPB records without additional testing), making the model cost-effective, operationally simple, and readily applicable in routine clinical practice. Clinicians can utilize this model to rapidly assess a patient's risk of AKI early postoperatively and implement targeted interventions in advance for high-risk individuals, such as controlled rewarming, optimized perfusion management, judicious transfusion strategies, and enhanced perioperative renal protection, which may help reduce the incidence of postoperative AKI and improve patient outcomes [23]. Furthermore, we explored the impact of rapid rewarming on early postoperative recovery. Our results demonstrated that patients in the rapid rewarming group had significantly longer durations of postoperative mechanical ventilation, ICU stay, and total hospital stay compared to those in the slow rewarming group, suggesting that AKI and other potential complications attributable to rapid rewarming adversely affect the recovery trajectory. These findings align with previous scholarly reports. However, no significant intergroup differences were observed in postoperative new-onset atrial fibrillation, re-exploration for bleeding, or mortality, which may be attributable to the relatively limited sample size and the low overall incidence of these events in our cohort, potentially leading to false-negative results due to insufficient statistical power.

Several limitations of this study should be acknowledged. First, as a single-center retrospective investigation, the study population may be subject to selection bias. Second, although 348 patients were enrolled, the number of AKI cases (n = 58) was relatively modest, which may compromise the stability and statistical power of the predictive model. Future multicenter, large-scale, prospective cohort studies are warranted to validate and refine our findings. Third, the follow-up period was relatively short, and we did not analyze long-term renal function recovery, progression to chronic kidney disease, or long-term

survival outcomes in patients with postoperative AKI. Extended follow-up is necessary to consolidate the conclusion that intraoperative rewarming rate increases postoperative AKI risk.

In summary, the incidence of AKI after open heart surgery with CPB is substantial. A rapid rewarming rate significantly increases the risk of postoperative AKI. In addition, prolonged CPB time, reduced preoperative eGFR, and intraoperative red blood cell transfusion were identified as independent risk factors. The prediction model constructed from these indicators demonstrates satisfactory discriminatory ability and calibration accuracy, offering valuable utility for early clinical identification of high-risk patients.

Funding Declaration

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Declarations

Author Contribution

Shuang Xu;Danping Shen;LiYinWang;ShaShaZhou;Lei Zhang wrote the main manuscript text and JieShi;YunWang prepared figures 1-2. All authors reviewed the manuscript

References

1. 1. Leballo G, Chakane PM. Cardiac surgery-associated acute kidney injury: pathophysiology and diagnostic modalities and management. Cardiovasc J Afr. 2020 Jul/Aug;31(4):205–212.
2. 2. Wysham C, Bindal A, Levrat-Guillen F, Kostadinova D, Poon Y. A systematic literature review on the burden of diabetic ketoacidosis in type 2 diabetes mellitus. Diabetes Obes Metab. 2025 May;27(5):2750–2767.
3. 3. Sun M, Yang L, Zong Q, Ying L, Liu X, Lin R. Serum soluble triggering receptor levels expressed on myeloid cells2 identify early acute kidney injury in infants and young children after pediatric cardiopulmonary bypass. Front Pediatr. 2023 Jun 26;11:1185151.
4. 4. Grigore AM, Murray CF, Ramakrishna H, Djaiani G. A core review of temperature regimens and neuroprotection during cardiopulmonary bypass: does rewarming rate matter? Anesth Analg. 2009 Dec;109(6):1741-51.
5. 5. Newland RF, Tully PJ, Baker RA. Hyperthermic perfusion during cardiopulmonary bypass and postoperative temperature are independent predictors of acute kidney injury following cardiac surgery. Perfusion. 2013 May;28(3):223 – 31.
6. 6. Boodhwani M, Rubens FD, Wozny D, Nathan HJ. Effects of mild hypothermia and rewarming on renal function after coronary artery bypass grafting. Ann Thorac Surg. 2009 Feb;87(2):489 – 95.

7. 7. Delbridge MS, Shrestha BM, Raftery AT, El Nahas AM, Haylor JL. The effect of body temperature in a rat model of renal ischemia-reperfusion injury. Transplant Proc. 2007 Dec;39(10):2983-5.
8. 8. Burke-Gaffney A, Svermova T, Mumby S, Finney SJ, Evans TW. Raised plasma Robo4 and cardiac surgery-associated acute kidney injury. PLoS One. 2014 Oct 31;9(10):e111459.
9. 9. Jain R, Yadav S, Bukke SPN, Chettupalli AK, Thalluri C. Post-operative infection treatment in cardiac surgery: current practices and future directions. Perioper Med (Lond). 2025 Oct 15;14(1):110.
10. 10. Levey AS. Defining AKD: The Spectrum of AKI, AKD, and CKD. Nephron. 2022;146(3):302–305.
11. 11. Brown JK, Shaw AD, Mythen MG, Guzzi L, Reddy VS, Crisafi C, Engelman DT; PeriOperative Quality Initiative and the Enhanced Recovery After Surgery Cardiac Workgroup. Adult Cardiac Surgery-Associated Acute Kidney Injury: Joint Consensus Report. J Cardiothorac Vasc Anesth. 2023 Sep;37(9):1579–1590.
12. 12. Hobson CE, Yavas S, Segal MS, Schold JD, Tribble CG, Layon AJ, Bihorac A. Acute kidney injury is associated with increased long-term mortality after cardiothoracic surgery. Circulation. 2009 May 12;119(18):2444-53.
13. 13. Lannemyr L, Lundin E, Reinsfelt B, Bragadottir G, Redfors B, Oras J, Ricksten SE. Renal tubular injury during cardiopulmonary bypass as assessed by urinary release of N-acetyl-ß-D-glucosaminidase. Acta Anaesthesiol Scand. 2017 Oct;61(9):1075–1083.
14. 14. Bai YX, Wang ZH, Lv Y, Liu J, Xu ZZ, Feng YQ, Liu GY, Yin P, Wang YT, Dong NG, Wu QP. Association between frailty and acute kidney injury after cardiac surgery: unraveling the moderation effect of body fat through an international, retrospective, multicohort study. Int J Surg. 2025 Jan 1;111(1):761–770.
15. 15. Engelman R, Baker RA, Likosky DS, Grigore A, Dickinson TA, Shore-Lesserson L, Hammon JW. The Society of Thoracic Surgeons, The Society of Cardiovascular Anesthesiologists, and The American Society of ExtraCorporeal Technology: Clinical Practice Guidelines for Cardiopulmonary Bypass–Temperature Management during Cardiopulmonary Bypass. J Extra Corpor Technol. 2015 Sep;47(3):145 – 54.
16. 16. Mangano CM, Diamondstone LS, Ramsay JG, Aggarwal A, Herskowitz A, Mangano DT. Renal dysfunction after myocardial revascularization: risk factors, adverse outcomes, and hospital resource utilization. The Multicenter Study of Perioperative Ischemia Research Group. Ann Intern Med. 1998 Feb 1;128(3):194–203.
17. 17. Qing M, Vazquez-Jimenez JF, Klosterhalfen B, Sigler M, Schumacher K, Duchateau J, Messmer BJ, von Bernuth G, Seghaye MC. Influence of temperature during cardiopulmonary bypass on leukocyte activation, cytokine balance, and post-operative organ damage. Shock. 2001 May;15(5):372-7.
18. 18. Tassani P, Barankay A, Haas F, Paek SU, Heilmaier M, Hess J, Lange R, Richter JA. Cardiac surgery with deep hypothermic circulatory arrest produces less systemic inflammatory response

than low-flow cardiopulmonary bypass in newborns. J Thorac Cardiovasc Surg. 2002 Apr;123(4):648 – 54.

19. 19. Sgouralis I, Evans RG, Layton AT. Renal medullary and urinary oxygen tension during cardiopulmonary bypass in the rat. Math Med Biol. 2017 Sep 1;34(3):313–333.
20. 20. Hendrix RHJ, Ganushchak YM, Weerwind PW. Oxygen delivery, oxygen consumption and decreased kidney function after cardiopulmonary bypass. PLoS One. 2019 Nov 22;14(11):e0225541.
21. 21. Ansart F, Pirenne F. Patient blood management and surgery: Impact of new surgical techniques on transfusion needs. Vox Sang. 2026 May;121(5):540–545.
22. 22. Kheterpal S, Tremper KK, Englesbe MJ, O'Reilly M, Shanks AM, Fetterman DM, Rosenberg AL, Swartz RD. Predictors of postoperative acute renal failure after noncardiac surgery in patients with previously normal renal function. Anesthesiology. 2007 Dec;107(6):892–902.
23. 23. Demirjian S, Bashour CA, Shaw A, Schold JD, Simon J, Anthony D, Soltesz E, Gadegbeku CA. Predictive Accuracy of a Perioperative Laboratory Test-Based Prediction Model for Moderate to Severe Acute Kidney Injury After Cardiac Surgery. JAMA. 2022 Mar 8;327(10):956–964.

Figures

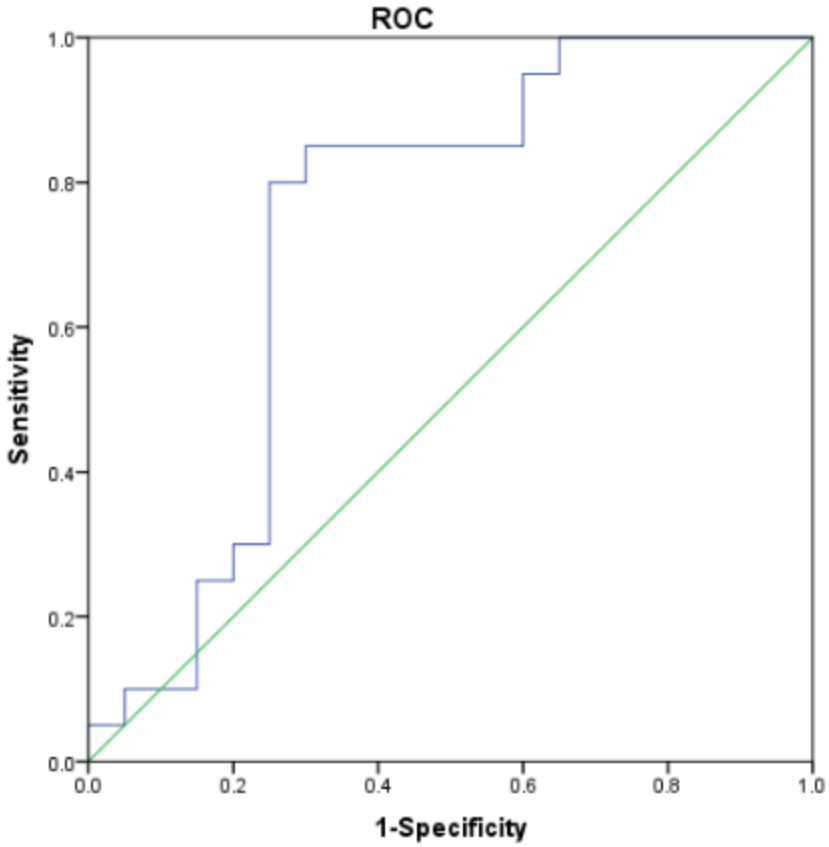


Figure 1

Receiver operating characteristic (ROC) curve of the nomogram model for predicting postoperative AKI

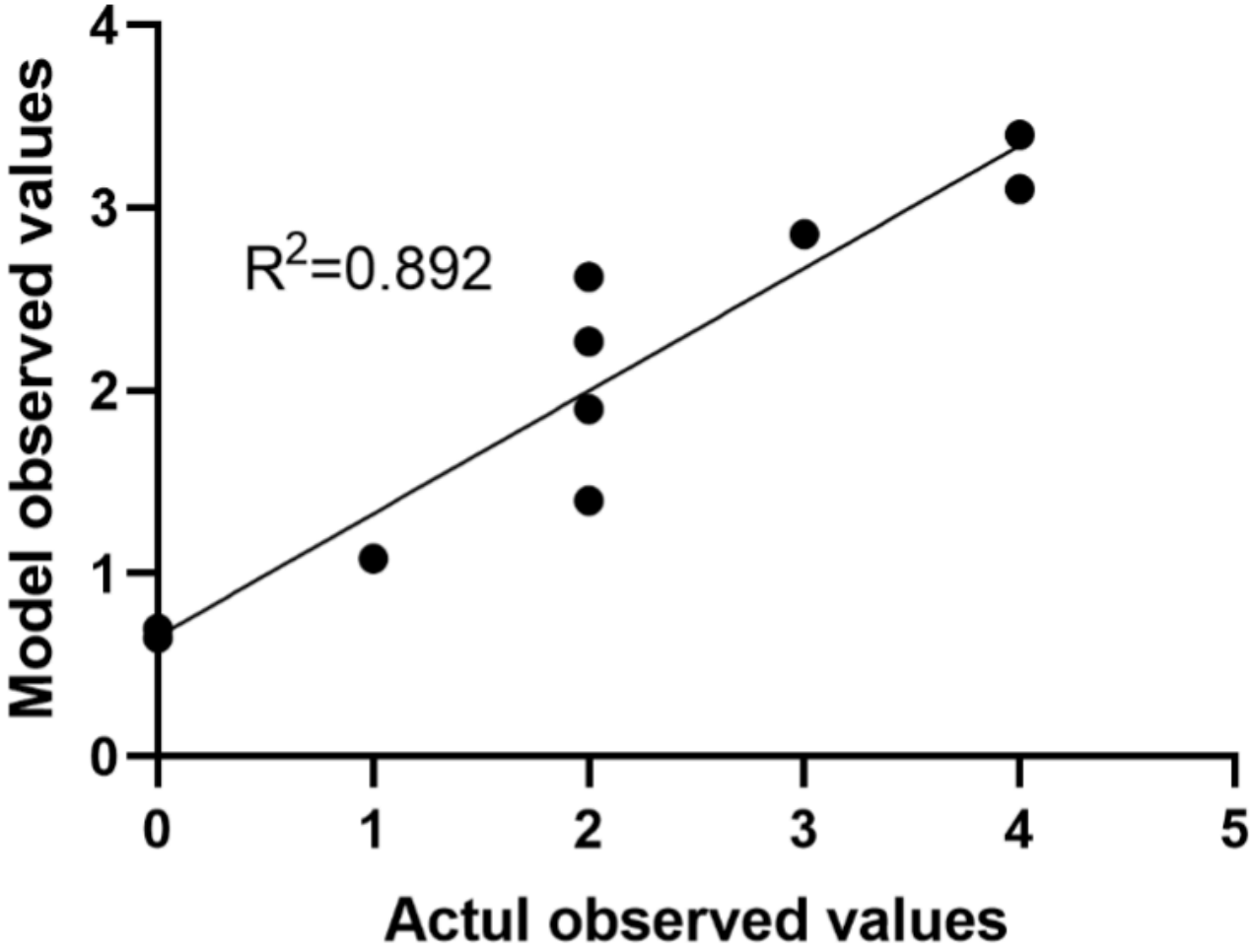


Figure 2

Calibration plot of the nomogram model for predicting postoperative AKI