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ORIGINAL RESEARCH

Nomogram Model for Cardiac Surgery-Associated Acute Kidney Injury Based on Clinical Characteristics Combined with Plasma suPAR

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Objective: Analyze risk factors for cardiac surgery-associated acute kidney injury (CSA-AKI) in adults and establish a nomogram model for CSA-AKI based on plasma soluble urokinase-type plasminogen activator receptor (suPAR) and clinical characteristics.

Methods: In a study of 170 patients undergoing cardiac surgery with cardiopulmonary bypass, enzyme-linked immunosorbent assay (ELISA) measured plasma suPAR levels. Multivariable logistic regression analysis identified risk factors associated with CSA-AKI. Subsequently, the CSA-AKI nomogram model was developed using R software. Predictive performance was evaluated using a receiver operating characteristic (ROC) curve and the area under the curve (AUC). Internal validation was performed through the Bootstrap method with 1000 repeated samples. Additionally, decision curve analysis (DCA) assessed the clinical applicability of the model.

Results: Multivariable logistic regression analysis revealed that being male, age ≥ 50 years, operation time ≥ 290 minutes, postoperative plasma suPAR at 2 hours, and preoperative left ventricular ejection fraction (LVEF) were independent risk factors for CSA-AKI. Employing these variables as predictive factors, a nomogram model was constructed, an ROC curve was generated, and the AUC was computed as 0.817 (95% CI 0.726–0.907). The calibration curve indicated the accuracy of the model, and the results of DCA demonstrated that the model could benefit the majority of patients.

Conclusion: Being male, age ≥ 50 years, operation time ≥ 290 minutes, low preoperative LVEF, and elevated plasma suPAR at 2 hours are independent risk factors for CSA-AKI. The nomogram model established based on these risk factors has high accuracy and clinical value, serving as a predictive tool for assessing the risk of CSA-AKI.

Keywords: nomogram, acute kidney injury, prediction model, risk factors, cardiac surgery

Introduction

Acute Kidney Injury (AKI) is a prevalent complication following adult cardiac surgery.[1,2] Its incidence varies based on diagnostic criteria and surgical types, ranging from 5% to 42%, with 1% to 7% of patients requiring dialysis.[3] The rising number of cardiac surgeries and the accelerated aging of the population contribute to a continuous increase in the occurrence of Cardiac Surgery-Associated Acute Kidney Injury (CSA-AKI). Apart from patients undergoing aortic surgery, the incidence of dialysis-requiring AKI among patients undergoing other cardiac surgeries has been increasing year by year.[4] CSA-AKI not only extends patients' hospitalization periods and escalates healthcare resource utilization but also imposes a substantial economic burden. Furthermore, it has profound adverse effects on long-term patient prognosis,[5] resulting in a 10-fold increase in the risk of chronic kidney disease and a 2-fold increase in the risk of mortality.[6]

Currently, the diagnosis of AKI still relies on the Kidney Disease Improving Global Outcomes (KDIGO) standard, based on serum creatinine (Scr) and urine output.[7] However, Scr and urine output lack sensitivity to kidney damage and exhibit poor specificity. In an unstable condition, Scr does not always accurately reflect kidney function, leading to

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a delayed diagnosis.[8] Relying on changes in urine output to identify AKI is also not sufficiently sensitive. Insufficient blood volume often leads to decreased urine output, and the use of diuretics can mask the true level of urine output by increasing it. This makes it challenging for clinical practitioners to promptly recognize the occurrence of AKI. Therefore, diagnosing AKI solely based on changes in Scr and urine output may not allow for the early detection of AKI, potentially missing the optimal clinical intervention window. In recent years, an increasing number of studies have focused on finding serum biomarkers to predict AKI, Emerging biomarkers have been continuously validated for their potential value in early prediction.[9,10] Soluble urokinase-type plasminogen activator receptor (suPAR) is a protein released into circulation by multiple cells, participating in various physiological processes. Studies indicate a significant elevation of suPAR levels in the blood of AKI patients, and this increase correlates positively with the severity of the disease. This suggests that suPAR may promote the development of AKI by fostering renal inflammation, oxidative stress, and cell apoptosis,[11] providing strong evidence for suPAR to serve as a superior biomarker for predicting AKI. Unlike traditional indicators used to assess AKI, such as creatinine and blood urea nitrogen, suPAR's advantage lies in its elevation being unaffected by factors like age, muscle mass, or nutrition,[12,13] thus more reliably reflecting changes in kidney function. Additionally, suPAR elevates earlier in patients compared to increases in creatinine or blood urea nitrogen levels, indicating its predictive capability in the early stages of AKI.[11] Currently, numerous studies have substantiated the efficacy of suPAR in accurately predicting AKI across diverse clinical scenarios.[14–16] Existing CSA-AKI prediction models, such as the Cleveland score[17] and AKI following cardiac surgery (AKICS) score,[18] have not integrated any reliable biomarkers other than Scr. Combining patients' clinical features with novel kidney injury biomarkers to construct a clinical prediction model for CSA-AKI can significantly enhance the predictive efficiency. The study aims to assess the predictive value of plasma suPAR for CSA-AKI, explore the risk factors associated with CSA-AKI, and integrate plasma suPAR with patients' clinical characteristics to construct a nomogram model to visualize these results. This study endeavors to establish a more dependable foundation for early warning systems targeting CSA-AKI, aiming to offer robust support for personalized treatment and patient care.

Materials and Methods

Study Design

This study is a retrospective observational study. We enrolled adult patients who underwent cardiac surgery with extracorporeal circulation at the Department of Cardiac Surgery, First Hospital Affiliated to Army Military Medical University (Southwest Hospital), from March 2020 to February 2021. Plasma samples from all included patients were retrieved from the institution's Biobank, originating from a previous research project. Exclusion criteria included pre-existing conditions of AKI or chronic kidney insufficiency (Chronic kidney insufficiency is defined by an estimated glomerular filtration rate of less than 60 mL/min/1.73m^2),[19] preoperative proteinuria, postoperative mortality within 7 days, early discharge before the completion of 7 postoperative days, and absence of preserved plasma samples. Ethical approval for this study was granted by the Ethics Committee of the First Affiliated Hospital of the Army Medical University (Approval No. KY2022055), adhering to the principles outlined in the Helsinki Declaration.

Sample Collection and the Experimental Procedure

1. Sample collection: Venous blood samples were collected from patients before and 2 hours after extracorporeal circulation heart surgery. Within 1 hour of collection, the samples were centrifuged at 3000 r/min (centrifugation radius of 117 mm) for 10 minutes, and the upper plasma was aliquoted into cryogenic tubes and stored in the Biobank of the First Affiliated Hospital of the Army Medical University at −80 °C for future use.
2. ELISA experimental procedure: The enzyme-linked immunosorbent assay (ELISA) for plasma suPAR detection was conducted by experienced technicians without prior knowledge of the clinical data. The suPAR notics® assay kit was procured from Virogates, Denmark, with a detection limit of 0.1 ng/mL. All assays were carried out following the instructions provided in the assay kit.

Data Collection

Information on preoperative comorbidity and anthropometric data was obtained from the admission notes, with the diagnoses of diabetes and hypertension based on the patients' medical histories. Surgical-related information was extracted from the operative records, and biochemical data were sourced from the Hospital Information System.

Group

Obtain the nearest Scr from biochemical data within 7 days before surgery as the baseline level. Patients were categorized into non-AKI and AKI groups based on whether AKI occurred within 7 days after extracorporeal circulation cardiac surgery. AKI was defined according to the KDIGO criteria as follows: an increase in SCr of 26.5 μmol/L (≥0.3 mg/dL) within 48 hours, an increase in Scr to 1.5 times the baseline value within 7 days, or a urine output <0.5 $mL \cdot kg^{-1} \cdot h^{-1}$ sustained for 6 hours.

Statistical Analyses

The statistical analyses were conducted using SPSS software Version 23.0 (SPSS Inc., Chicago, IL, USA), MedCalc statistical software Version 19.6 (MedCalc Software Ltd., Ostend, Belgium), and R Version 4.3.1 (R Project for Statistical Computing, Vienna, Austria). Normally distributed continuous variables were described as mean ± SD and compared between groups by using Student's *t*-test; non-normally distributed continuous variables were presented as median (25th-75th percentiles) and compared by using the Mann–Whitney *U*-test. Percentage were used for categorical variables, and intergroup comparisons were performed by using the Chi-square test. We first used univariate logistic regression to perform single-variable analysis and selected variables with a $p < 0.05$. Then, we applied forward selection stepwise regression to further screen the selected variables and determine the variables that would ultimately enter the model. Finally, we conducted multivariable logistic regression analysis to construct the prediction model and plotted a nomogram. To evaluate the model's performance, we used the receiver operating characteristic (ROC) curve to calculate the area under the curve (AUC) and used Youden's index to determine the optimal threshold. Additionally, decision curve analysis (DCA) was employed to evaluate the clinical utility of the model. Bootstrap resampling (1000 iterations) was employed for internal validation, calculating the concordance index (C-Index) for assessing model discrimination, plotting a calibration curve to evaluate model calibration. A significance level of $P < 0.05$ was considered statistically significant.

Results

Comparison of Clinical Characteristics

In the present study, out of the initial 193 participants, 23 were excluded, resulting in a cohort of 170 patients for the conclusive analysis (refer to the CONSORT diagram, Figure 1). Among the included 170 patients, the AKI group comprised 34 cases, while the non-AKI group comprised 136 cases, yielding an AKI incidence rate of 20.0%. According to the KDIGO standard,[7] out of 34 cases of AKI, 31 were classified as stage 1 AKI, while the remaining 3 cases were stage 3 AKI, and there were no cases of stage 2 AKI. A comparative analysis of baseline characteristics and intraoperative variables between the two groups identified statistically significant differences in gender, age, preoperative albumin (Alb) levels, and operation time ($P < 0.05$). Conversely, no statistically significant disparities were observed in other measured parameters, as detailed in Table 1.

Comparison of Plasma suPAR Levels Between Two Groups of Patients

Preoperative plasma suPAR levels (P=0.001) and those measured 2 hours postoperatively (P<0.001) were markedly elevated in the AKI group relative to the non-AKI group, as evidenced in Table 2.

Univariate and Multivariable Logistic Regression Analysis of CSA-AKI

Univariate logistic analysis identified associations between gender (OR=0.461, 95% CI 0.215–0.990, P=0.047), age (OR=3.833, 95% CI 1.273–11.540, P=0.017), preoperative Scr (OR=1.029, 95% CI 1.002–1.057, P=0.034), postoperative 2-hour Scr (OR=1.028, 95% CI 1.004–1.054, P=0.023), preoperative left ventricular ejection fraction (LVEF)

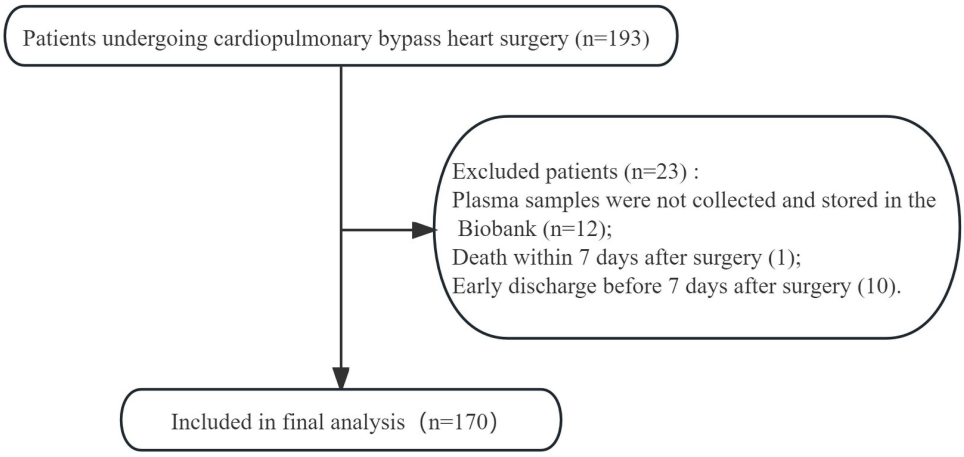

Figure 1 CONSORT diagram.

(OR=0.011, 95% CI 0.000–0.515, *P*=0.022), operation time (OR=2.705, 95% CI 1.254–5.837, *P*=0.011), preoperative suPAR (OR=1.425, 95% CI 1.153–1.760, *P*=0.001), postoperative 2-hour suPAR (OR=1.664, 95% CI 1.296–2.137, *P*<0.001), and CSA-AKI. In the multivariable logistic regression analysis, adjusting for the aforementioned influencing

Table 1 Baseline Characteristics of AKI and Non-AKI

Characteristics	AKI(n=34)	Non-AKI(n=136)	P
Gender, n (%)			0.044
Male	20(58.82%)	54(39.71%)	
Female	14(41.18%)	82(60.29%)	
Age, n (%)			0.021
≥50 years	30(88.24%)	90(66.18%)	
<50 years	4 (11.76%)	46(33.82%)	
BMI, kg/m^2	24.16 (22.77,26.66)	23.73 (21.63,26.09)	0.137
Medical histories, n (%)			
Diabetes	3 (8.82%)	6 (4.41%)	0.549
Hypertension	8 (23.53%)	23 (16.91%)	0.371
Heart failure	31 (91.18%)	127 (93.38%)	0.940
Biochemical examination			
Preoperative Src (umol/L)	75.30 (60.73,87.00)	69.25 (62.13,79.55)	0.080
Preoperative BUN (mmol/L)	6.32(5.08, 7.02)	5.78(4.71, 7.13)	0.600
Preoperative LVEF (%)	55.50(47.25, 63.00)	59.00(54.00, 64.75)	0.066
Preoperative WBC (x10^9/L)	6.35(5.15, 7.87)	5.82(4.83, 7.23)	0.124
Preoperative RBC (x10^{12}/L)	4.36±0.55	4.46±0.53	0.335
Preoperative Hb (x10^9/L)	137.50(112.75, 144.00)	135.00(122.00, 143.75)	0.612

(*Continued*)

Table 1 (Continued).

Characteristics	AKI(n=34)	Non-AKI(n=136)	P
Preoperative Alb (g/L)	37.99±4.66	39.92±3.75	0.012
Post-Operative Scr at 2h (umol/L)	77.40 (56.68,89.72)	64.40 (57.49,74.39)	0.061
Post-Operative BUN at 2h (umol/L)	6.37 (5.12,8.00)	6.00 (5.09,6.78)	0.116
Intraoperative Parameters			
Operation time, n (%)			0.010
≥290min	20 (58.82%)	47 (34.56%)	
<290min	14 (41.18%)	89 (65.44%)	
Bypass time, n (%)			0.202
≥114min	29 (85.29%)	102 (75.00%)	
<114min	5 (14.71%)	34 (25.00%)	
Surgery type, n (%)			
CABG	1 (2.94%)	5 (3.68%)	1.000
CABG+Valve	2 (5.88%)	5 (3.68%)	0.923
Valve	26 (76.47%)	113 (83.09%)	0.371
Ascending aortic replacement	5 (14.71)	12 (8.82%)	0.482
Other cardiac surgery	0 (0.00%)	1 (0.74%)	1.000
Intraoperative Urine Output (mL)	2100.00(1500.00, 2800.00)	2200.00(1700.00, 2875.00)	0.489

Notes: Normally distributed continuous variables were displayed as mean±standard deviation; non-normally distributed continuous variables were displayed as median and interquartile ranges; categorical variable and rank variable were displayed as quantities and percentages.
Abbreviations: BMI, body mass index; Scr, serum creatinine; BUN, blood urea nitrogen; LVEF, left ventricular ejection fraction; Alb, albumin; WBC, white blood cell; RBC, red blood cell; Hb, hemoglobin; CABG, coronary artery bypass grafting.

Table 2 Comparison of Plasma suPAR Levels in AKI Vs Non-AKI

Variables	AKI (n=34)	Non-AKI(n=136)	P
Preoperative suPAR (ng/mL)	3.36(2.55, 5.47)	2.49(1.87, 3.44)	0.001
Post-Operative suPAR at 2h (ng/mL)	3.74(2.51, 5.36)	2.50(1.95, 3.48)	<0.001

Abbreviation: suPAR, soluble urokinase-type plasminogen activator receptor.

factors, gender (OR=3.323, 95% CI 1.361–8.682, *P*=0.010), age (OR=4.274, 95% CI 1.321–18.000, *P*=0.026), postoperative 2-hour suPAR (OR=1.786, 95% CI 1.373–2.430, *P*<0.001), preoperative LVEF (OR=0.006, 95% CI 0.000–0.432, *P*=0.021), and operation time (OR=3.319, 95% CI 1.381–8.410, *P*=0.009) emerged as independent risk factors for CSA-AKI, as depicted in Table 3 and Figure 2.

Construction and Evaluation of a Risk Nomogram for Adult CSA-AKI

Drawing of the nomogram: Based on the results of the multivariable logistic regression analysis, incorporating gender, age, postoperative 2-hour suPAR, preoperative LVEF, and operation time as the five variables, a predictive model was constructed: Logit(P) = −2.769 + 1.201 × gender (male=1, female=0) + 1.453 × age (≥50 years=1,<50 years=1) + 0.580 × postoperative 2-hour suPAR (ng/mL) - 5.201 × preoperative LVEF + 1.200 × operation time (≥290 min=1,<290 min=0),

Table 3 The Logistic Regression Analysis of CSA-AKI Risk Factors

Characteristic	β	Wald	P	OR	95% CI
Univariate					
Gender	−0.774	3.942	0.047	0.461	0.215–0.990
Age	1.344	5.711	0.017	3.833	1.273–11.540
Preoperative Src	0.029	4.474	0.034	1.029	1.002–1.057
Preoperative LVEF	−4.509	5.282	0.022	0.011	0.000–0.515
Post-Operative Scr at 2h	0.028	5.144	0.023	1.028	1.004–1.054
Post-Operative BUN at 2h	0.224	4.082	0.043	1.251	1.007–1.554
Operation time	0.995	6.433	0.011	2.705	1.254–5.837
Preoperative suPAR	0.354	10.743	0.001	1.425	1.153–1.760
Post-Operative suPAR at 2h	0.509	15.939	<0.001	1.664	1.296–2.137
Multivariable					
Gender	1.201	6.564	0.010	3.323	1.361–8.682
Age	1.453	4.937	0.026	4.274	1.321–18.000
Post-Operative suPAR at 2h	0.580	16.190	<0.001	1.786	1.373–2.430
Preoperative LVEF	−5.201	5.312	0.021	0.006	0.000–0.432
Operation time	1.200	6.849	0.009	3.319	1.381–8.410
Intercept	−2.769	3.323	0.068	0.063	

Logit(P) represents the natural logarithm of the odds. In this study, Logit(P) represents the logarithm of the probability of CSA-AKI occurrence. The visualized nomogram is shown in Figure 3.

Prediction model performance: ROC curve analysis revealed that the AUC of the nomogram model for predicting CSA-AKI was 0.817, with a 95% CI of 0.726 to 0.907. The optimal threshold was 0.379, with sensitivity and specificity of 61.8% and 94.9%, respectively. This indicates that the model has good risk prediction capability for CSA-AKI, as shown in Figure 4.

Evaluation of discrimination and calibration in the prediction model: Employing the Bootstrap method with 1000 independent samples generated a new dataset. The corrected C-Index was computed, and a calibration curve was generated (as illustrated in Figure 5) to assess the model's discrimination and calibration. The corrected C-Index for this prediction model was 0.795, signifying excellent predictive classification. The degree of proximity between the calibration curve and the ideal curve positively correlates with the model's predictive capability.

Assessment of the clinical applicability of the prediction model: DCA confirmed the net benefit derived from employing this model for patients within the dataset. In this study, the incidence rate of CSA-AKI was 20%. At a threshold of 0.2, the DCA curve is situated above both the None line and the All line (as illustrated in Figure 6), signifying notable clinical utility of the model at this threshold. It can proficiently steer clinical decisions, leading to a heightened net benefit for patients.

Discussion

CSA-AKI stands out as a frequent complication following cardiac surgery, potentially leading to prolonged hospital stays, heightened treatment expenses, a notable decline in quality of life, and even mortality.[20] However, it is crucial to note that not every case of AKI results in severe adverse outcomes; some instances are reversible. The anticipation and early identification of AKI hold paramount significance in steering clinical practices and enhancing patient prognoses.[21]

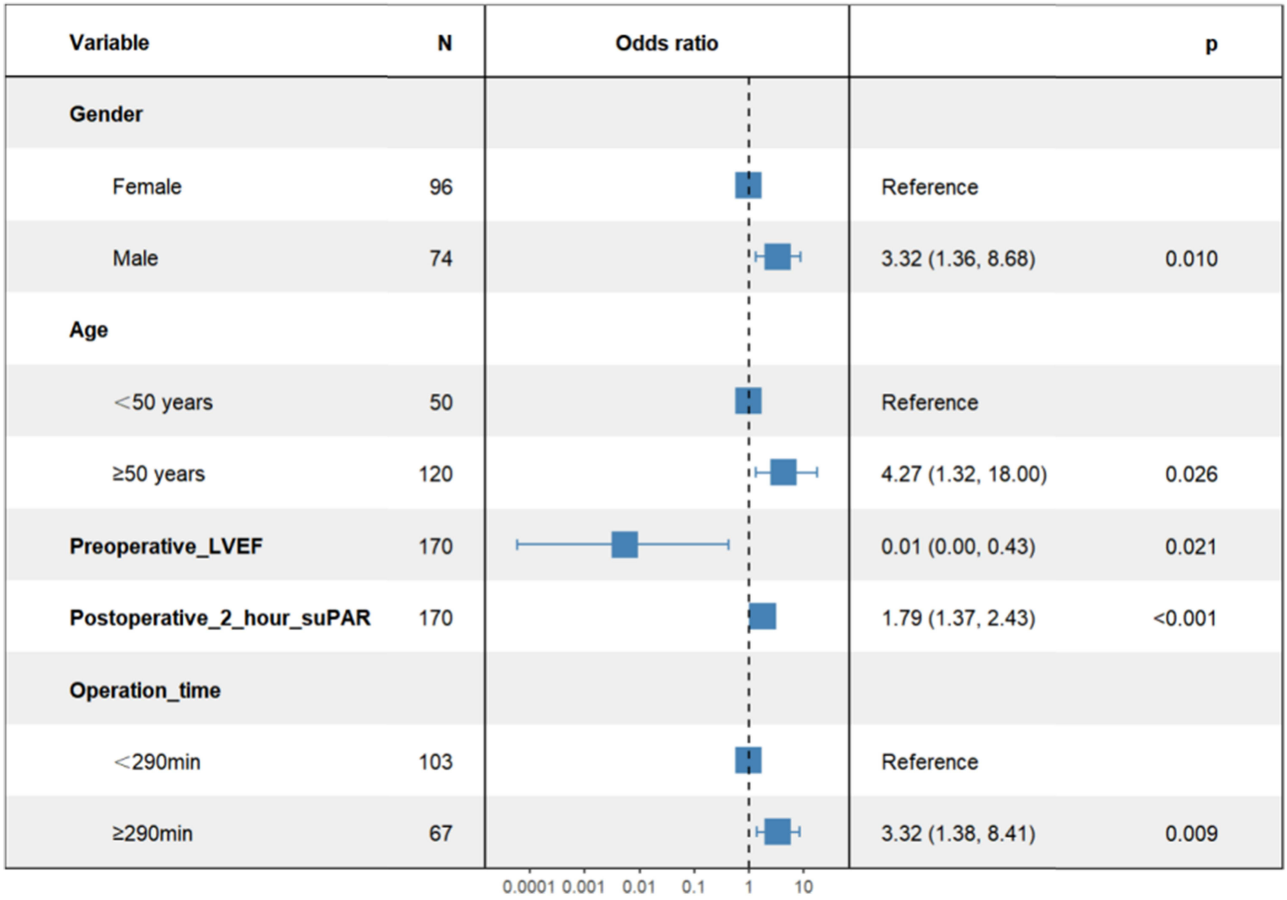

Figure 2 The multivariable logistic regression analysis of CSA-AKI risk factors.

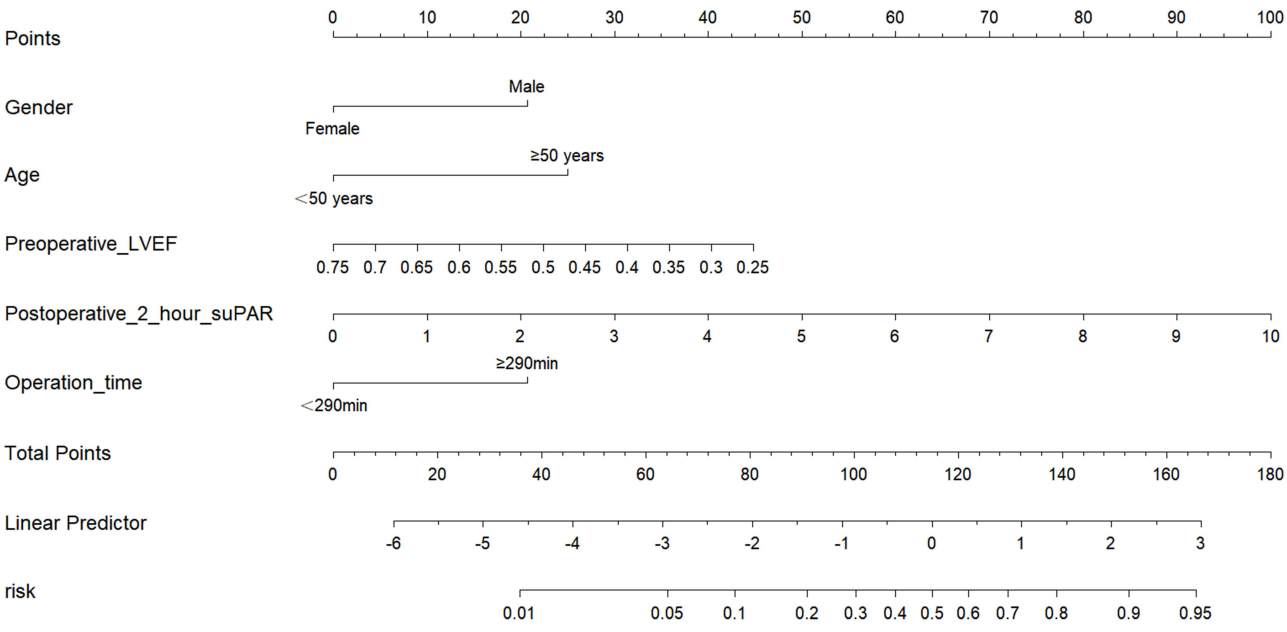

Figure 3 A nomogram predicting the risk of CSA-AKI.

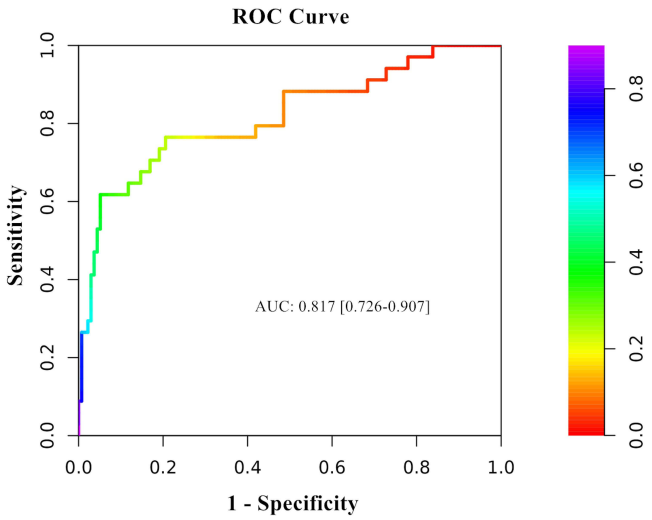

Figure 4 The ROC curve depicts the predictive accuracy of the nomogram model for CSA-AKI diagnosis.

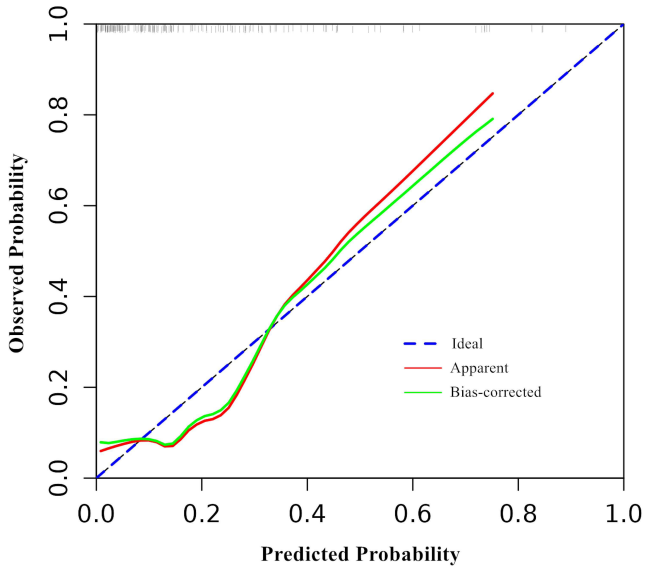

Figure 5 The calibration curve of the nomogram model for predicting CSA-AKI.

This study meticulously documented and analyzed baseline data, intraoperative conditions, as well as preoperative and postoperative biochemical indicators in patients undergoing cardiopulmonary bypass cardiac surgery. Notably, we pioneered the development of a clinical prediction model for CSA-AKI, incorporating plasma suPAR and clinical characteristics. The aim is to furnish guidance for predicting and intervening in CSA-AKI within the realm of clinical practice.

In our study, the incidence rate of CSA-AKI was 20.0%, lower than the results of other similar studies.[3,22–24] This difference may be attributed to our study excluding patients with pre-existing AKI or chronic kidney dysfunction. We analyzed factors associated with CSA-AKI occurrence and identified male, age ≥ 50 years, preoperative LVEF level, and operation time ≥ 290 minutes as independent risk factors for CSA-AKI. These findings align with previous research,[25,26] further emphasizing the significance of these factors in predicting CSA-AKI. In clinical practice, it is essential to consider these risk factors and implement corresponding intervention strategies to reduce the occurrence of CSA-AKI.

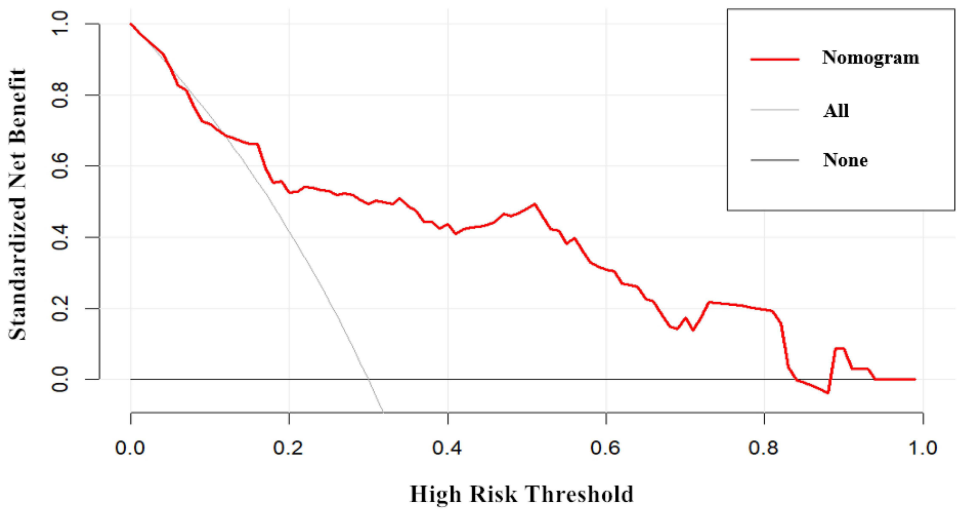

Figure 6 The nomogram model predicts the DCA for CSA-AKI.

An aspect worthy of further exploration is that univariate logistic analysis suggests postoperative 2-hour Scr as a potential risk factor for the occurrence of CSA-AKI. However, after adjusting for other risk factors through multivariable logistic regression analysis, postoperative 2-hour Scr does not emerge as an independent risk factor for the occurrence of CSA-AKI. Consequently, it was excluded from the CSA-AKI risk prediction model. Creatinine, a byproduct of muscle metabolism, is generated in the body at a relatively constant rate and is primarily filtered and excreted through the renal glomerular filtration membrane.[27] Thus, Scr serves as a commonly utilized and crucial clinical indicator for assessing kidney function, providing a simple, cost-effective, and efficient method for evaluating kidney function. However, Scr can be influenced by various factors, including patients' dietary habits, age, and medication use.[28] Therefore, when utilizing Scr to assess kidney function, the potential impact of these variables must be taken into consideration. Furthermore, as indicated by our study, Scr exhibits a certain degree of lag in reflecting declining kidney function, and its timeliness is not as sensitive as plasma suPAR.

In this study, we measured plasma suPAR levels and found that the plasma suPAR levels in the AKI group were significantly higher than those in the non-AKI group both preoperatively and 2 hours postoperatively. suPAR is the soluble form of urokinase-type plasminogen activator receptor (uPAR), expressed on the cell membrane of various cells through glycosyl-phosphatidylinositol (GPI).[12] When the body is subjected to various stimuli, GPI anchors are cleaved by various proteinases, leading to the release of uPAR from the membrane, forming suPAR.[12,29] Previous studies have indicated that elevated plasma suPAR levels are associated with the occurrence of AKI in different clinical cohorts.[14,15,30,31] Our study further confirms this conclusion and supports its potential as a predictive biomarker for AKI. The mechanistic role of suPAR in kidney injury is not fully understood, but some possible causal relationships have been proposed. Studies have shown that exposure to suPAR can upregulate and activate αvβ3 integrin expressed on podocytes, leading to cell detachment and proteinuria.[32–34] suPAR also increases the energy demand of renal tubular epithelial cells and induces oxidative stress, making them particularly sensitive to ischemia-reperfusion injury.[14] These findings suggest that suPAR plays a critical role in reshaping the renal filtration barrier and influencing processes in renal tubular epithelial cells.

There are now many studies reporting clinical prediction models for CSA-AKI, but most studies focus on AKI requiring dialysis as the endpoint.[17,35,36] Mild AKI is associated with long-term adverse outcomes in patients, and its occurrence rate is higher, also deserving attention. Furthermore, existing CSA-AKI clinical prediction models mainly concentrate on the risk of AKI after cardiac surgery in patients with kidney dysfunction, with a significant emphasis on the weight of kidney dysfunction in the model. This is not applicable to CSA-AKI risk prediction in populations with

normal kidney function. This study excluded patients with pre-existing kidney dysfunction, demonstrating broader clinical utility.

suPAR, as a novel biomarker for AKI, exhibits excellent predictive efficacy. In order to enhance the predictive ability for CSA-AKI and address the shortcomings of current prediction models, we constructed a risk prediction model based on multivariable logistic regression analysis. This model ultimately incorporates five variables: gender, age, postoperative 2-hour plasma suPAR level, preoperative LVEF level, and operation time. It is the first time that plasma suPAR levels have been included in the CSA-AKI prediction model. The results indicate that the model has a high predictive accuracy, with an AUC of 0.817 as suggested by ROC curve analysis. Furthermore, at the optimal cutoff point, the model demonstrates high sensitivity and specificity, further supporting its potential application value in clinical practice.

In the realm of clinical practice, our CSA-AKI prediction model, which relies on the analysis of plasma suPAR and clinical features, adeptly evaluates the risk of CSA-AKI within the initial 2 hours following cardiac surgery. This model furnishes clinicians with a pivotal temporal window to proactively implement personalized intervention measures, thereby markedly mitigating the likelihood of CSA-AKI and enhancing overall patient prognosis. In instances where the model signals heightened risk, clinicians are advised to vigilantly oversee fluid balance and blood pressure, abstain from nephrotoxic drugs, and minimize the occurrence of CSA-AKI.

Nevertheless, it is crucial to acknowledge several limitations in our study that may impact the interpretation of the findings. Firstly, the single-center nature of our investigation introduces the possibility of selection bias, limiting the generalizability of our results. Validation through multicenter studies is imperative to ensure the robustness and external applicability of our findings. Second, due to limitations in the implementation of the clinical study and ethical considerations, we were unable to collect the hemodynamic status and use of vasopressors in the two groups of patients postoperatively, which may impact the comprehensiveness and accuracy of the CSA-AKI prediction model. Third, the relatively small sample size in our study may affect the stability of the prediction model. Future research endeavors should focus on larger cohort studies to validate and refine our predictive model. Additionally, despite the satisfactory performance of our risk prediction model in internal validation, further exploration is needed to assess its applicability across diverse patient populations, including individuals of different ethnicities and those undergoing various types of cardiac surgeries.

Conclusions

In summary, we have constructed a highly accurate CSA-AKI prediction model based on gender, age, postoperative 2-hour plasma suPAR level, preoperative LVEF level, and operation time. This model will contribute to an improved understanding and prediction of CSA-AKI. Through early prediction and intervention, clinicians can take measures to reduce the occurrence of CSA-AKI and improve patient outcomes. Future research should further explore the pathogenesis and predictive factors of AKI, as well as enhance the accuracy and practical value of prediction models. Additionally, emphasis should be placed on conducting intervention studies to identify optimal strategies for reducing AKI occurrence and, consequently, improving patient outcomes.

Ethics Statement

This study was conducted according to the principles of the Declaration of Helsinki; informed consent was obtained from all subjects. This study was approved by the Ethics Committee of the First Affiliated Hospital of the Army Medical University (Approval No. KY2022055).

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report there are no competing interests to declare in this work.

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