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ORIGINAL RESEARCH

Development and Internal Validation of a Nomogram for Predicting Acute Kidney Injury After Cardiac Valve Surgery Using the Serum Uric Acid-to-Albumin Ratio

Xiaoru Zhao[1,*], Juntao Wang[2,*], Wenxin Bai[1], Wenwen Zhang[1], Chunling Huang[1], Zengyuan Qin[1], Kaiyue Wei[1], Minghui Han[1,3], Lei Yan[1,3], Yue Gu[1,3], Fengmin Shao[1,3]

[1]Department of Nephrology, Zhengzhou University People's Hospital, Henan Provincial People's Hospital, Zhengzhou, Henan, People's Republic of China; [2]Department of Nephrology, The First People's Hospital of Shangqiu, Shangqiu, Henan, People's Republic of China; [3]Henan Provincial Clinical Research Center for Kidney Disease, Henan Provincial Key Laboratory of Kidney Disease and Immunology, Henan Provincial People's Hospital, Zhengzhou, Henan, People's Republic of China

*These authors contributed equally to this work

Correspondence: Fengmin Shao; Yue Gu, Email fengminshao@126.com; guyuesunny@zzu.edu.cn

Background: The serum Uric Acid-to-Albumin Ratio (sUAR) is a novel inflammatory indicator. We aimed to construct and validate a prediction model for acute kidney injury (AKI) following cardiac valve surgery (CVS) based on the preoperative sUAR.

Methods: We retrospectively collected clinical data from adult patients undergoing CVS with cardiopulmonary bypass at the Heart Center of Henan Provincial People's Hospital between December 2020 to December 2021. The primary outcome was postoperative AKI, defined according to the KDIGO creatinine criteria. Patients were categorized as either AKI or non-AKI based on this outcome. Multivariate logistic regression to identify independent risk factors. A nomogram model was developed. The receiver operating characteristic (ROC) curve assessed discrimination. The calibration curve and Hosmer-Lemeshow test evaluated calibration. Clinical practicability was assessed through decision curve analysis (DCA) and clinical impact curve (CIC). The Bootstrap method was used for internal verification.

Results: A total of 440 patients were enrolled, and the incidence of AKI was 33.4%. Multivariate analysis revealed that sUAR (per μmol/g, *OR*=1.467, 95% *CI* 1.311–1.642, *P*<0.001), age (per 10 years, *OR*=1.612, 95% *CI* 1.261–2.062, *P*<0.001), atrial fibrillation (*OR*=2.485, 95% *CI* 1.573–3.924, *P*<0.001), hemoglobin (per g/L, *OR*=0.985, 95% *CI* 0.973–0.998, *P*=0.025) were the independent risk factors. The nomogram based on sUAR achieved an area under the curve (AUC) of 0.779 (95% CI 0.734–0.824, *P*<0.001) for predicting AKI. The average AUC after internal validation of the nomogram model was 0.776 (95% CI 0.767–0.779). The calibration curve and Hosmer-Lemeshow test indicated that the predicted and observed results agreed well, while the DCA and CIC curves demonstrated favorable clinical applicability within a specific threshold range.

Conclusion: The prediction model incorporating sUAR provides reliable discrimination and clinical utility for assessing AKI risk following CVS.

Keywords: acute kidney injury, prediction model, cardiac valve surgery, serum uric acid-to-albumin ratio, nomogram

Introduction

Acute kidney injury (AKI) is a serious complication following cardiac surgery in the intensive care unit (ICU).[1,2] According to previous reports, the incidence of cardiac surgery-associated AKI (CSA-AKI) varies between 5% to 49%.[3,4] Increased postoperative mortality, longer ICU stays, and higher health care costs are all correlated with CSA-AKI.[4,5] Given the current dearth of clinically applicable interventions for CSA-AKI, early recognition of high-risk groups is essential for both CSA-AKI prevention and therapy. Several AKI-risk prediction models for cardiac surgery

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have been identified,[6–8] such as the Cleveland Clinic Score,[9] the Mehta score,[10] the AKICS Score,[11] and the Simplified Renal Index score.[12] These models provide an important foundation for clinical risk assessments. However, these models have not been widely used in clinical practice.[6,7] A nomogram is a graphical calculation tool that shows the contribution of each predictive factor to the overall risk visually, offering superior interpretability and ease of use compared to complex algorithmic scores.[13] This approach facilitates rapid, visual risk assessments at the bedside, which is crucial for timely clinical decision-making.[13,14]

Previous studies have indicated that preoperative high uric acid (UA) and low albumin (ALB) levels are significant risk factors.[15–17] The serum Uric Acid-to-Albumin Ratio (sUAR),[18] a composite indicator integrating both serum UA and ALB. It is linked to both AKI development and mortality in ICU patients, and has also been described as a novel inflammatory indicator for AKI.[18,19] Recent research has reported that elevated sUAR was a risk factor for post-contrast AKI.[18] AKI incidence is relatively high in cardiac valve surgery (CVS), which is a common cardiac surgical procedure performed under cardiopulmonary bypass (CPB).[20] The establishment of existing AKI clinical prediction models for cardiac surgery often requires a considerable number of variables, and these models have not incorporated sUAR. As a composite biomarker, the sUAR could provide a more integrative and robust risk assessment than single and conventional indicators, potentially leading to good predictive performance for AKI after CVS. Currently, the relationship between sUAR and AKI after CVS has not been investigated. It remains uncertain whether the sUAR can predict AKI following CVS. This study aims to explore the correlation between sUAR and postoperative AKI, and to build a nomogram model for AKI following CVS based on sUAR, with the expectation of providing a basis for the early intervention of AKI following CVS in clinical practice.

Methods

Study Population

During the period from December 2020 to December 2021, adult patients undergoing CVS at the Heart Center of Henan Provincial People's Hospital were enrolled in this retrospective study. The inclusion criteria were: (1) elective CVS under CPB; (2) age ≥18 years old. The exclusion criteria were: (1) estimated glomerular filtration rate (eGFR)<60 $mL \cdot min^{-1} \cdot (1.73m^2)^{-1}$, chronic kidney disease (CKD), dialysis before the operation, renal transplant patients, patients with preoperative AKI, or kidney stone patients; (2) patients with chronic liver disease, gout, malignant tumors, severe infection, infective endocarditis, and autoimmune disease; (3) intraoperative or within the first 24 hours postoperative death; (4) underwent coronary artery bypass graft (CABG) surgery during the same period; (5) patients with missing preoperative serum creatinine (SCr), UA, ALB or postoperative SCr.

Data Collection

All of the enrolled patients' clinical data were collected. Preoperative clinical data included age, gender, comorbidities, the latest preoperative laboratory blood biochemical parameters, and so on. Intraoperative clinical data included CPB time, type of valve surgery (valvuloplasty, single valve replacement, double valve replacement). Based on the latest preoperative SCr, the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula was used to calculate the eGFR.[21] The sUAR was calculated as the serum UA/ALB. To decrease the influence of extreme values and reduce skewness, the concentration of N-terminal pro-B-type natriuretic peptide (NT-proBNP) was natural log transformed.[22]

Definition of Postoperative AKI

Postoperative AKI was defined as a ≥26.5 μmol/L increase in SCr within 48h, or an increase in SCr to ≥1.5 times baseline within 7 days, based on the KDIGO guidelines in 2012.[23] As the urine output data were not precisely recorded for patients, the urine output criteria were not used.

Grouping

Patients were assigned to the AKI and non-AKI groups based on the occurrence of AKI during the seven days after CVS. The latest preoperative SCr was used as the baseline SCr.

Sample Size

A priori sample size justification: We performed a sample size calculation using the method proposed by Riley et al and implemented in the pmsampsize R package.[24] According to a previous study,[25] we assumed that the incidence of AKI following CVS was 0.346, the target C-statistic for the model was set to 0.814, and six parameters were intended to be included. The calculation indicated that the minimum sample size required for new model development was 348. Therefore, our final sample size of 440 met this requirement.

Events-Per-Parameter (EPP) evaluation: The primary binary outcome (postoperative AKI) occurred in 147 patients (events, E), and the total number of parameters (P) was four in our final model (the continuous variables sUAR, age, and HGB contributed three parameters, and the categorical variable AF with two levels contributed one parameter). Consequently, the EPP ratio in our study was 147/4=36.75, which met the conventional criterion of EPP≥10.[24]

Study Protocol

This study was conducted in accordance with the Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis (TRIPOD) and Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.[26,27]

Statistical Analysis

SPSS 26.0, MedCalc 20.022, and R 4.5.0 were used for statistical analyses. A P value <0.05 was considered statistically significant (two-tailed). We used the mean value imputation or median imputation to handle missing values based on the distribution of the variables, since only five continuous variables with less than 1% missing data in this study. Categorical variables were compared using the Chi-square test and presented as frequencies with percentages. According to the Shapiro–Wilk test, continuous variables were expressed as mean ± standard deviation or median (25th and 75th percentiles) and compared using the independent samples t-test or Mann–Whitney U-test. Variables with P<0.05 in the univariate analysis of the study were selected for univariate logistic regression analysis. Variables with $P < 0.05$ and no evidence of collinearity in the univariate logistic regression analysis were then added to the multivariate stepwise logistic regression model (forward: conditional) to identify independent risk factors of AKI following CVS and build a nomogram prediction model. Variance inflation factor (VIF) was used to quantify multicollinearity, and thresholds were set at VIF<5, which indicated weak collinearity. The restricted cubic spline (RCS) analysis was applied to assess the potential relationships between the continuous predictors and AKI risk. To enhance the clinical meaningfulness of odds ratios (ORs), the effect estimates for sUAR, age, and hemoglobin (HGB) were expressed in increments of 1 μmol/g,10 years, and 1 g/L, respectively, in multivariate analysis. These increments were set according to the previous literature.[18,28,29]

The predictive power was assessed by the area under the receiver operating characteristic (ROC) curve (AUC). The difference in AUC was compared by the DeLong test. The calibration curve and the Hosmer-Lemeshow test were applied to evaluate the calibration. The clinical practicability was assessed by the decision curve analysis (DCA) and clinical impact curve (CIC). The model was internally validated with 1000 bootstrap resamplings.

Results

Clinical Data Comparison

The analysis included 440 eligible patients in total (Figure 1). Of these, 147 patients (33.4%) developed postoperative AKI, 87 (19.8%) stage I AKI, 42 (9.5%) stage II AKI, and 18 (4.1%) stage III AKI. AKI patients were older, had higher preoperative levels of urea, SCr, UA, blood glucose, LnNT-proBNP, and sUAR, and had a larger percentage of atrial fibrillation (AF) than non-AKI patients, while the preoperative levels of eGFR, lymphocyte (LY), HGB, and serum ALB were lower (all P<0.05) (Table 1).

Variable Screening

The univariate logistic regression analysis included variables in Table 1 with P<0.05, and the results showed that advanced age, AF, preoperative LnNT-proBNP, HGB, urea, SCr, eGFR, UA, blood glucose, ALB, and sUAR were

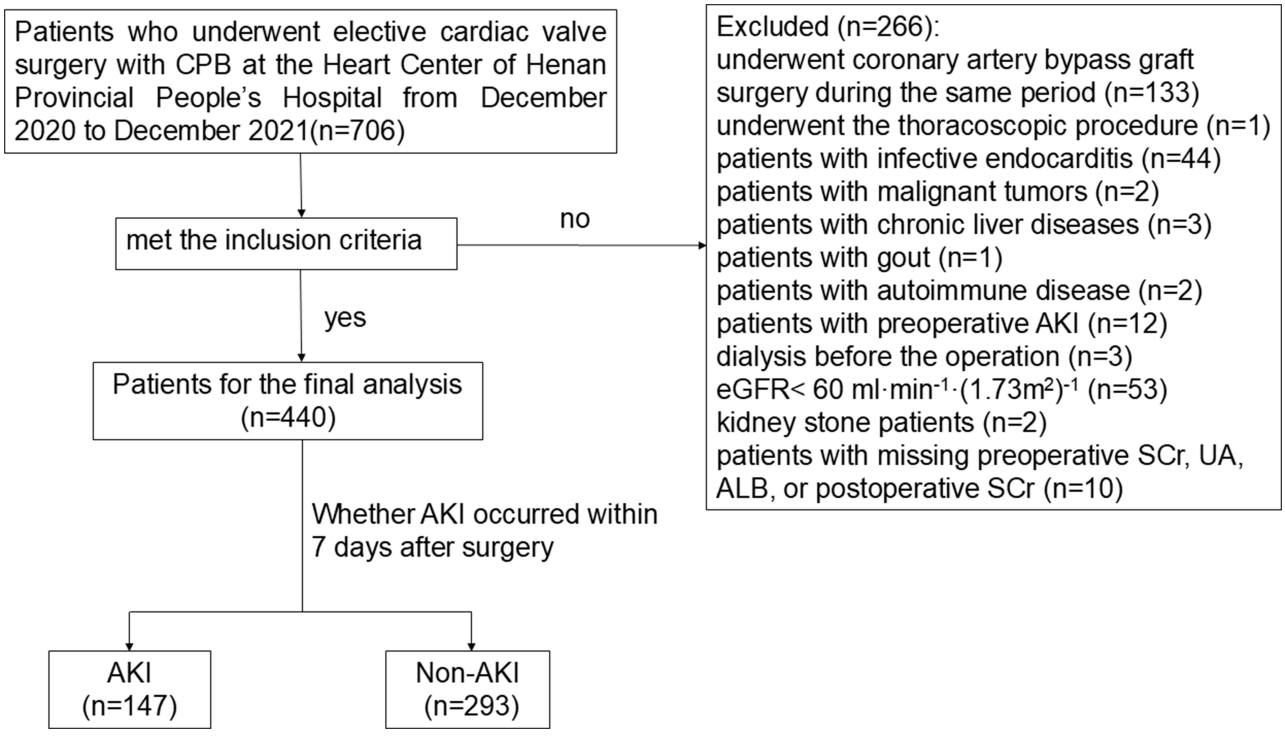

Figure 1 Study enrollment flowchart.

influencing factors for AKI following CVS (Table 2). Based on the results displayed in Table 2 and the collinearity analysis (Table S1), the variables of age, AF, preoperative LnNT-proBNP, HGB, urea, SCr, eGFR, blood glucose, and sUAR were included in the multivariate analysis, indicating that the high preoperative sUAR (per μmol/g, odds ratio

Table 1 Comparison of the Variables Between the AKI and Non-AKI Groups

Variables	Non-AKI (n=293)	AKI (n=147)	t/Z /χ^2	*P* Value
Age (years)	55.00(48.00,63.00)	61.00(55.00,67.00)	−5.480	<0.001
Female (n, %)	124(42.32)	75(51.02)	2.991	0.084
Weight (kg)	65.40(57.70,72.75)	63.50(55.50,73.00)	−1.017	0.309
Body mass index (kg/m^2)	24.25±3.25	24.80±3.86	−1.461	0.145
Smoking history (n, %)	97(33.11)	42(28.57)	0.931	0.335
Drinking history (n, %)	69(23.55)	27(18.37)	1.541	0.214
Hypertension (n, %)	91(31.06)	59(40.14)	3.590	0.058
Diabetes (n, %)	23(7.85)	14(9.52)	0.356	0.551
Coronary heart disease (n, %)	47(16.04)	34(23.13)	3.274	0.070
AF (n, %)	93(31.74)	82(55.78)	23.620	<0.001
NYHA class III/IV (n, %)	235(80.20)	128(87.07)	3.200	0.074
Preoperative LVEF (%)	59.00(54.50,64.00)	59.00(55.00,62.00)	−0.863	0.388
NT-proBNP (pg/mL)	529.00(153.40,1060.50)	989.50(408.30,1742.00)	−4.973	<0.001
lnNT-proBNP (pg/mL)	6.27(5.03,6.97)	6.90(6.01,7.46)	−4.976	<0.001
WBC (×10^9/L)	5.90(4.99,6.89)	5.63(4.68,7.27)	−0.575	0.565
NE (×10^9/L)	3.45(2.82,4.34)	3.46(2.70,4.57)	−0.186	0.852
LY (×10^9/L)	1.72(1.38,2.06)	1.58(1.17,1.98)	−2.285	0.022
MO (×10^9/L)	0.40(0.32,0.49)	0.38(0.31,0.50)	−0.699	0.484
HGB (g/L)	137.00(127.00,149.00)	133.00(121.00,144.00)	−2.479	0.013

(*Continued*)

Table 1 (Continued).

Variables	Non-AKI (n=293)	AKI (n=147)	t/Z /χ^2	*P* Value
PLT (×10^9/L)	191.00(157.50,231.50)	179.00(150.00,221.00)	−1.443	0.149
Urea (mmol/L)	5.90(5.00,7.00)	6.50(5.40,8.10)	−3.665	<0.001
SCr (μmol/L)	70.00(61.00,82.00)	75.00(67.00,86.00)	−3.725	<0.001
eGFR [mL·min^{-1}·$(1.73m^2)^{-1}$]	95.40(85.65,102.95)	83.60(74.30,93.30)	−7.131	<0.001
UA (μmol/L)	322.00(261.00,385.00)	376.00(320.00,435.00)	−6.389	<0.001
Blood glucose (mmol/L)	4.57(4.15,5.12)	4.78(4.25,5.52)	−2.383	0.017
ALB (g/L)	42.37±3.29	41.37±3.33	2.996	0.003
sUAR (μmol/g)	7.46(6.15,8.98)	8.96(7.71,10.53)	−7.166	<0.001
Total cholesterol (mmol/L)	3.84±0.83	3.83±0.92	0.045	0.964
Triglyceride (mmol/L)	1.13(0.88,1.52)	1.13(0.80,1.43)	−0.669	0.503
HDL-C(mmol/L)	1.12(0.94,1.34)	1.12(0.98,1.33)	−0.525	0.600
LDL-C(mmol/L)	2.37(1.79,2.84)	2.34(1.75,2.88)	−0.127	0.899
CPB duration (min)	151.00(122.50,192.50)	158.00(125.00,192.00)	−0.829	0.407
Type of surgery (n, %)			1.490	0.475
Valvuloplasty	57(19.45)	27(18.37)		
Single valve replacement	204(69.63)	98(66.67)		
Double valve replacement	32(10.92)	22(14.96)		

Abbreviations: AKI, acute kidney injury; AF, atrial fibrillation; NYHA, New York Heart Association; LVEF, left ventricular ejection fractions; NT-proBNP, N-terminal pro-B-type natriuretic peptide; WBC, white blood cell; NE, neutrophil; LY, lymphocyte; MO, monocyte; HGB, hemoglobin; PLT, platelet; SCr, serum creatinine; eGFR, estimated glomerular filtration rate; UA, uric acid; ALB, albumin; sUAR, serum uric acid-to-albumin ratio; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; CPB, cardiopulmonary bypass.

Table 2 Univariate Logistic Regression Analysis

Variables	*OR* (95% CI)	*P* Value
Age (years)	1.057(1.034–1.080)	<0.001
AF	2.713(1.804–4.080)	<0.001
lnNT-proBNP (pg/mL)	1.516(1.285–1.789)	<0.001
LY (×10^9/L)	0.752(0.525–1.079)	0.121
HGB (g/L)	0.988(0.977–0.998)	0.026
Urea (mmol/L)	1.197(1.089–1.316)	<0.001
SCr (μmol/L)	1.028(1.013–1.043)	<0.001
eGFR [mL·min^{-1}·$(1.73m^2)^{-1}$]	0.951(0.937–0.966)	<0.001
UA (μmol/L)	1.008(1.006–1.011)	<0.001
Blood glucose (mmol/L)	1.199(1.037–1.386)	0.014
ALB (g/L)	0.913(0.859–0.970)	0.003
sUAR (μmol/g)	1.449(1.307–1.607)	<0.001

Abbreviations: AF, atrial fibrillation; NT-proBNP, N-terminal pro-B-type natriuretic peptide; LY, lymphocyte; HGB, hemoglobin; SCr, serum creatinine; eGFR, estimated glomerular filtration rate; UA, uric acid; ALB, albumin; sUAR, serum uric acid-to-albumin ratio.

[*OR*]=1.467, 95% confidence interval [*CI*] 1.311–1.642, *P*<0.001), advanced age (per 10 years, *OR*=1.612, 95% *CI* 1.261–2.062, *P*<0.001), AF (per g/L, *OR*=2.485, 95% *CI* 1.573–3.924, *P*<0.001), and low HGB (*OR*=0.985, 95% *CI* 0.973–0.998, *P*=0.025) were independent risk factors for AKI following CVS (Table 3). As shown in Figure S1, the RCS curve demonstrated that there were no non-linear relationships between sUAR (*P* for nonlinear=0.191), age (*P* for nonlinear=0.516), HGB (*P* for nonlinear=0.432), and AKI risk.

Table 3 Multivariate Logistic Regression Analysis

Variables	β (SE)	*Wald*	*P* Value	*OR* (95% CI)
Age (per 10 years)	0.478(0.125)	14.518	<0.001	1.612 (1.261–2.062)
AF (Yes/No)	0.910(0.233)	15.242	<0.001	2.485 (1.573–3.924)
HGB (per g/L)	−0.015(0.007)	5.029	0.025	0.985 (0.973–0.998)
sUAR (per μmol/g)	0.383(0.058)	44.376	<0.001	1.467 (1.311–1.642)
Constant	−5.060(1.314)	14.827	<0.001	0.006

Abbreviations: AF, atrial fibrillation; HGB, hemoglobin; sUAR, serum uric acid-to-albumin ratio.

Development and Validation of Nomogram Prediction Model

Predictive Ability

The AUCs of age, AF, HGB, and sUAR were 0.660 (95% *CI* 0.607–0.713, *P*<0.001), 0.620 (95% *CI* 0.572–0.669, *P*<0.001), 0.572 (95% *CI* 0.515–0.603, *P*=0.013), and 0.709 (95% *CI* 0.660–0.759, *P*<0.001), respectively. The evaluation metrics were summarized in Table 4.

In addition, we compared the sUAR with UA and ALB. The results showed that the AUC of the sUAR was greater than that of UA (AUC=0.687, 95% *CI* 0.636–0.737) and ALB (AUC=0.588, 95% *CI* 0.532–0.644) alone (Figure S2 and Table S2).

We constructed model 1 combined with age, AF, and HGB, and then added the sUAR to model 1 to construct the final model. Comparative analysis showed that the AUC of the final model (AUC=0.779, 95% *CI* 0.734–0.824) was superior to that of model 1(AUC=0.707, 95% *CI* 0.656–0.759), and the difference is statistically significant (*P*<0.001), indicating that the sUAR enhanced the model's discriminatory ability. Using the DeLong test, we further compared the model's predictive ability with that of sUAR, age, AF, and HGB. The results demonstrated that the model's AUC was also superior to those of sUAR, age, AF, and HGB (Figure 2 and Table 4).

Therefore, we built a nomogram model based on the sUAR, age, AF, and HGB (Figure 3). ROC analysis revealed that the model's AUC was 0.779 (95% *CI* 0.734–0.824, *P*<0.001) (Figure 2 and Table 4). And the model's average AUC was 0.776 (95% *CI* 0.767–0.779) following internal validation (Figure 4), suggesting that the model had good discrimination capacity.

Calibration Degree

The model's calibration and ideal curve were found to be reasonably consistent in the calibration plot (Bootstrap method, n=1000) (Figure 5). Combined with the result of the Hosmer-Lemeshow test (χ^2=6.795, *P*=0.559) and Figure S3 (intercept: −0.00, slope: 1.00), suggesting that the predicted and actual probabilities agreed well.

Clinical Practicability

DCA demonstrated that when the threshold probability fell between 0.09 and 0.99, the model had a good net benefit in predicting AKI following CVS (Figure 6). In a similar vein, CIC demonstrated that the nomogram was clinically useful within certain threshold probabilities (Figure 7).

Table 4 Predictive Value of Age, AF, HGB, sUAR, Model 1, and Model

Variables	AUC	95% CI	*P* Value	Cut-Off Value	Sn	Sp
Age (years)	0.660	0.607–0.713	<0.001	57	65.31%	60.41%
AF	0.620	0.572–0.669	<0.001	Yes	55.78%	68.26%
HGB (g/L)	0.572	0.515–0.603	0.013	136	61.90%	50.17%
sUAR (μmol/g)	0.709	0.660–0.759	<0.001	7.29	84.35%	45.73%
Model 1	0.707	0.656–0.759	<0.001	0.347	64.63%	68.60%
Model	0.779	0.734–0.824	<0.001	0.326	71.43%	72.35%

Notes: Delong test: Model vs Age (*P*<0.001); Model vs AF (*P*<0.001); Model vs HGB (*P*<0.001); Model vs sUAR (*P*<0.001); Model vs Model 1 (*P*<0.001); Model 1: Age, AF, and HGB; Model: add sUAR to Model 1.
Abbreviations: AUC, area under the ROC curve; AF, atrial fibrillation; HGB, hemoglobin; sUAR, serum uric acid-to-albumin ratio; ROC, receiver operating characteristic; Sn, sensitivity; Sp, specificity.

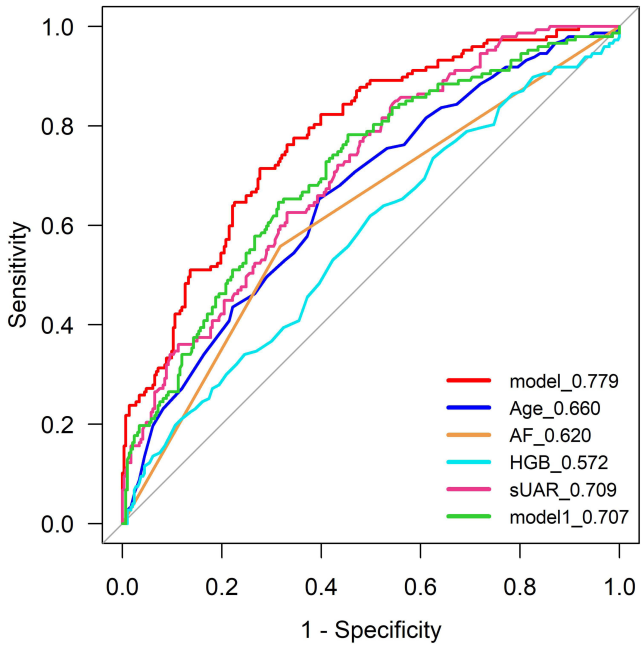

Figure 2 ROC curve for predicting AKI.
Notes: model1: Age, AF, and HGB; model: add sUAR to model 1.

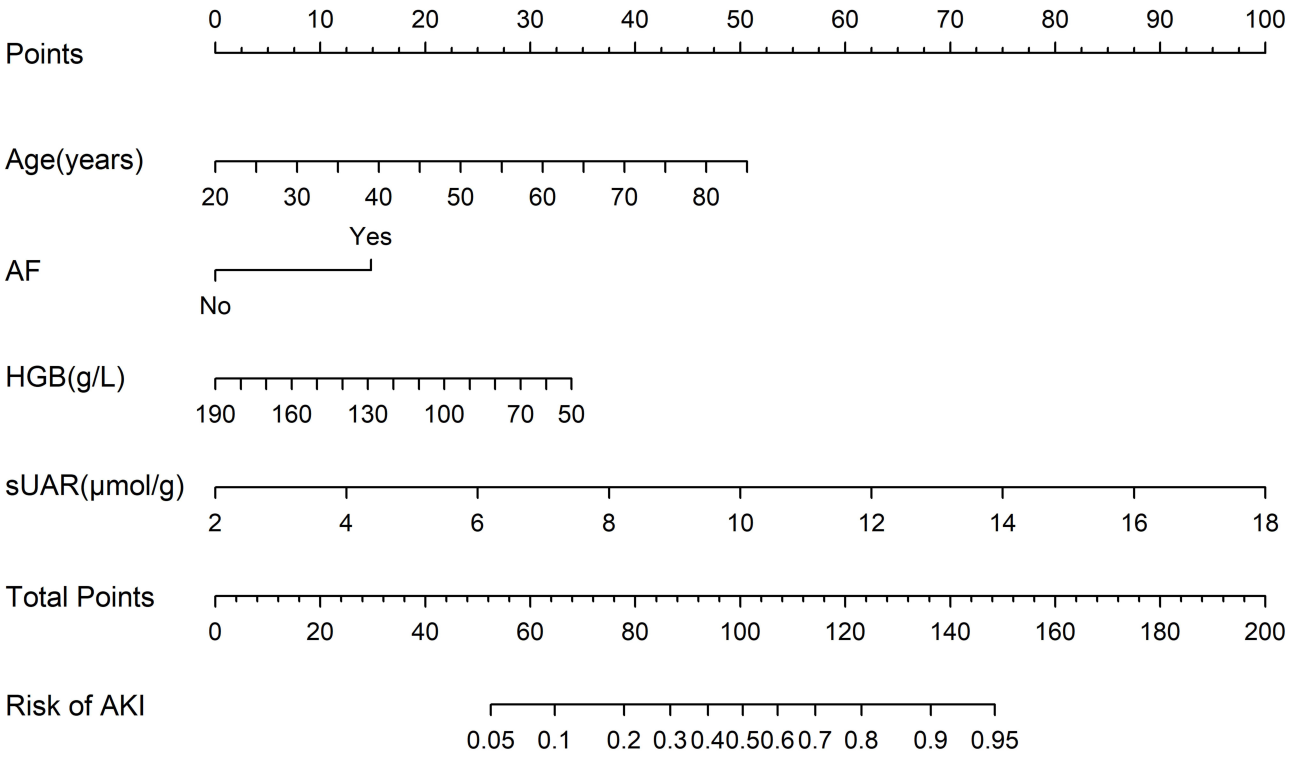

Figure 3 Nomogram for predicting AKI following cardiac valve surgery.

Discussion

Following cardiac surgery, CSA-AKI is a common complication that is related to longer hospital stays, worse short and long-term prognoses, and increased treatment costs.[3–5] In patients undergoing CVS, postoperative AKI is more likely to

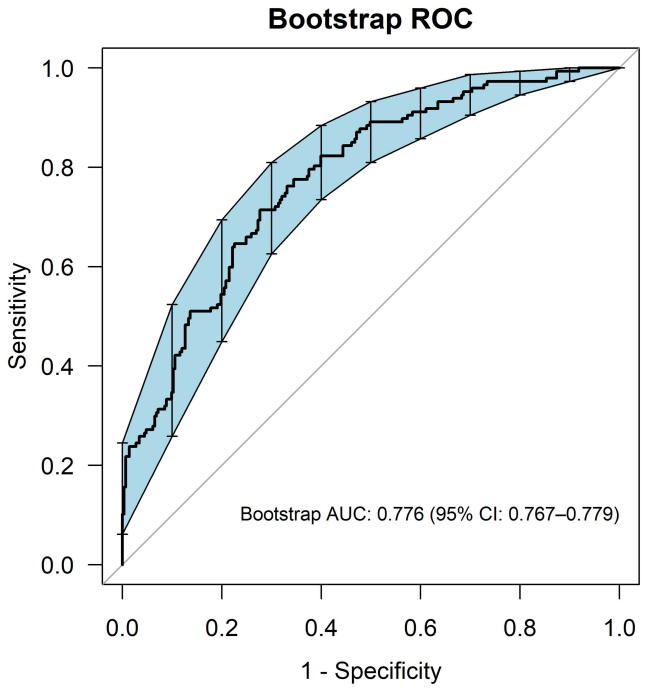

Figure 4 ROC curve for bootstrap validation.

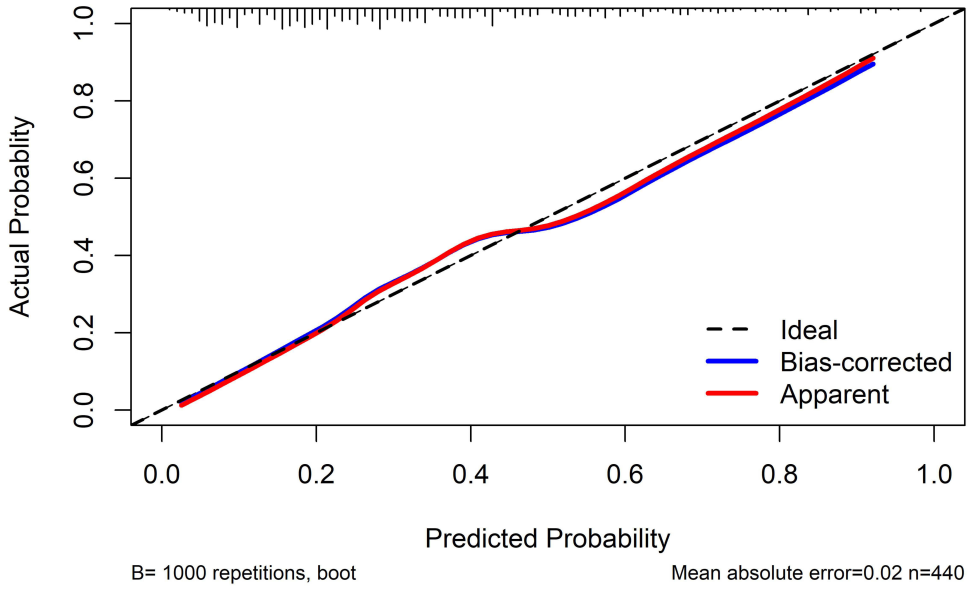

Figure 5 Calibration curve of the nomogram prediction model for AKI.

occur.[30] Thereby, it is especially crucial to screen AKI risk factors following CVS, recognize high-risk groups early, and carry out reasonable intervention to minimize postoperative AKI incidence.[31]

It demonstrates that the sUAR was an independent indicator of AKI after CVS in this study. In addition, age, AF, and HGB were also identified as independent influencing factors of postoperative AKI in this study. Internal validation demonstrated that the prediction model established based on sUAR had good discrimination and calibration, while DCA and CIC indicated that the model was clinically useful. This model demonstrated a good predictive ability in predicting AKI following CVS, which could assist medical staff in recognizing high-risk patients of AKI and formulating personalized treatment plans.

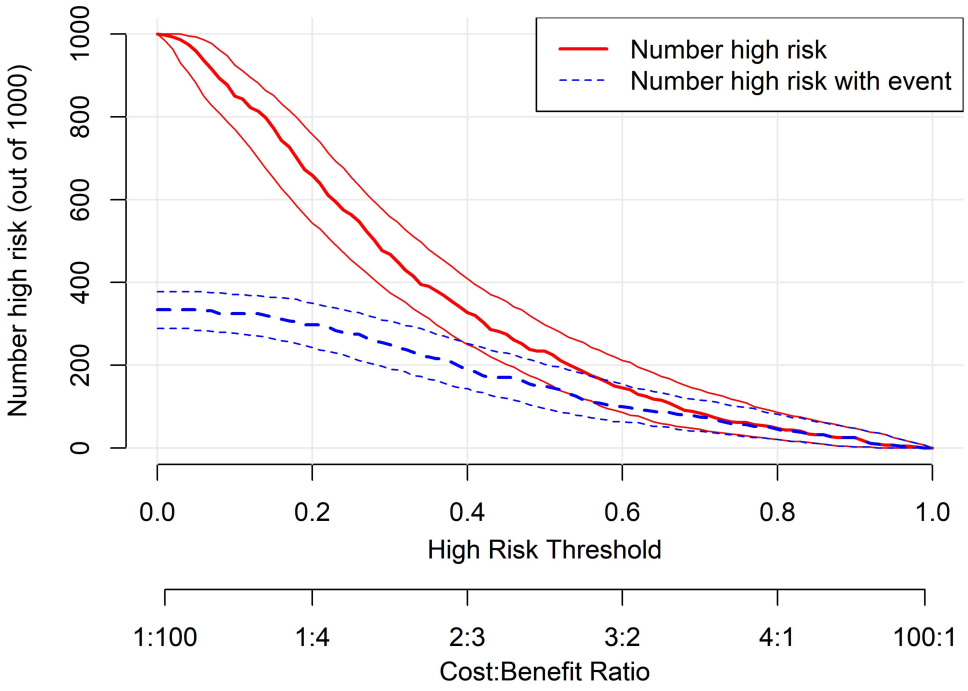

Figure 6 Decision curve analysis (DCA) of the Nomogram prediction model.

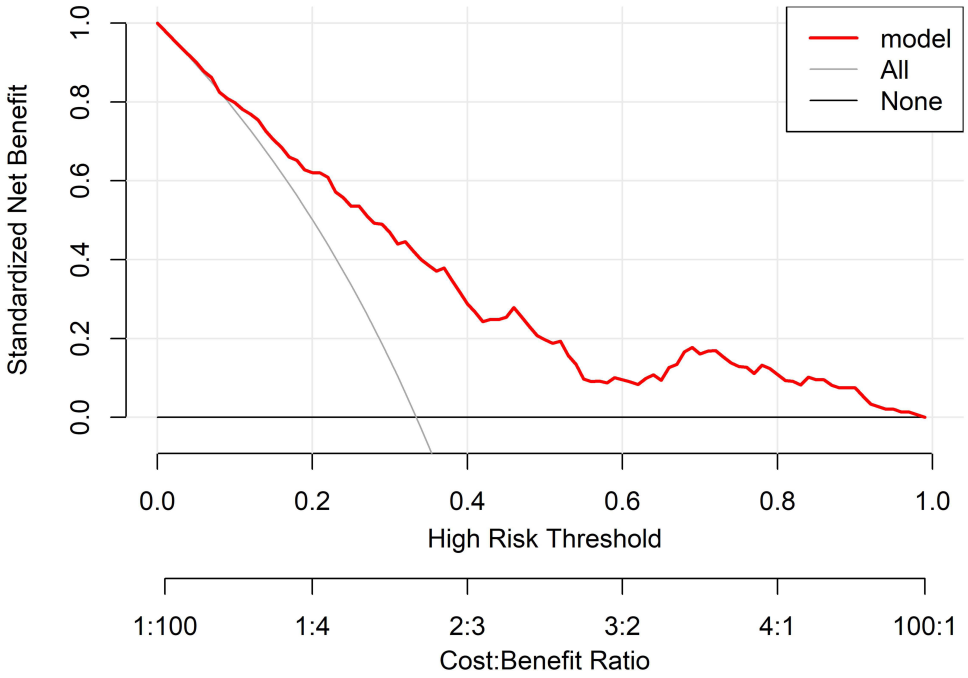

Figure 7 Clinical impact curve (CIC) of the Nomogram prediction model.

Some studies have found that serum UA and serum ALB were independently associated indicators of AKI.[32–34] According to earlier research, decreased serum ALB and elevated serum UA were risk factors for AKI following cardiac surgery.[35–39] In AKI patients after non-cardiac surgery, the preoperative serum ALB levels were low.[40] In this study, we discovered that preoperative serum UA and ALB were the influencing factors of postoperative AKI. sUAR is calculated from serum UA and ALB. In ICU patients, sUAR was related to AKI.[19] Recent studies have shown that sUAR is a good

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predictor of post-contrast AKI.[18,41] In our study, the AUC for sUAR was 0.709 (P<0.001), indicating that sUAR had a moderate predictive value for AKI following CVS. The exact mechanisms by which elevated sUAR levels affect the development of AKI after CVS are still unknown. The functions that serum UA and ALB may help explain why. Firstly, UA has characteristics of both an antioxidant and a pro-oxidant agent. Elevated UA levels are linked to inflammation and oxidative stress, as well as renin-angiotensin system activation, nitric oxide system inhibition, endothelial dysfunction, and renal dysfunction.[36,38,42–44] Secondly, serum ALB has antioxidant activity and can reduce the necrosis of renal tubular cells; its elevated metabolism and decreased synthesis indicate an inflammatory state.[34,45] Finally, the pathogenesis of AKI following CVS is complex, and many factors play significant roles in its development and occurrence, such as inflammation, ischemia-reperfusion damage, hemolysis, hypoperfusion, oxidative stress, and so on.[4,46] Therefore, sUAR is a composite indicator of inflammation and oxidative stress, which could be the possible mechanism for the correlation between sUAR and AKI after CVS.

This study found that AF was an independent risk factor. According to a previous report, patients with AF at admission had a 2.6 times greater incidence of AKI during hospitalization compared to those with sinus rhythm.[47] Because AF reduces the cardiac output, it leads to systemic congestion,[48,49] which, in turn, causes the renal venous pressure to increase and then results in the renal perfusion pressure decreasing. Since GFR is primarily determined by renal perfusion pressure, AF's damage to glomerular perfusion can lead to renal function impairment.[50] Additionally, AF causes activation of the neurohormonal system, such as the renin-angiotensin-aldosterone pathway,[51] which might negatively impact renal function if sustained. Furthermore, AF may result in thromboembolism, which can lead to renal infarction.[50] Other independent risk factors in this study included age and preoperative HGB, which were consistent with previous studies.[4,28,46] According to previous studies, prolonged CPB time was considered a risk factor for AKI.[52] It could be due to the relatively small sample size that we were unable to find a statistically significant difference in CPB time between AKI patients and non-AKI patients after surgery.

Several clinical prediction models for AKI risk assessment following cardiac surgery have been identified.[6,8–12] The Cleveland Clinic Score,[9] a classic renal risk score for cardiac surgery, was established to predict AKI requiring dialysis following cardiac surgery.[8,15] These prediction models provide an important foundation for AKI risk assessments. However, these risk scores incorporate multiple clinical risk factors that do not include the sUAR, and the collection and calculation processes are rather complex. Accordingly, they have not been widely used in clinical practice.[7,15] In addition, the [TIMP2] × [IGFBP7],[53] comprising urine biomarkers reflecting early renal tubular cell injury, has been widely studied to identify severe AKI risk.[46,54] However, the widespread adoption of these markers could be limited by costs and the need for specialized testing platforms. In our study, the AUC of the sUAR was superior to that of UA and ALB alone. Additionally, ROC curve analysis showed that the predictive performance of the model containing the sUAR was improved compared with that of the model without the sUAR. These results indicated that the sUAR provides a practical tool for clinicians to assess the risk of postoperative AKI in CVS patients before surgery. The AUC for the nomogram model based on the sUAR was 0.779 (95% CI 0.734–0.824), and the average AUC after internal validation of the nomogram model was 0.776 (95% CI 0.767–0.779), indicating that the nomogram model had a moderate predictive value for AKI following CVS. The UA and ALB tests required for the sUAR are routine laboratory items in most hospitals worldwide, which is highly conducive to the sUAR's promotion and popularization in clinical environments with diverse resources. In addition, the sUAR, as a novel composite predictor, uniquely links two critical pathways involved in the development of AKI—systemic stress and inflammation. It further integrates systemic information that is often overlooked by existing risk scores, thereby providing pathophysiological insights beyond SCr and eGFR. Moreover, it also offers powerful clinical interpretability. An increase in the sUAR directly informs the clinician of either elevated UA (implying oxidative stress and inflammation) or reduced ALB (reflecting poor nutrition or inflammation), providing direct evidence for personalized patient management. Of course, there are some scenarios where the sUAR may be misleading; therefore, sUAR must be interpreted with caution in clinical practice. For instance, in patients with severe hypoalbuminemia due to acute systemic infection, an elevated sUAR may be primarily driven by a sharp decline in ALB levels, which may reflect the severity of the acute inflammatory response, rather than direct kidney injury. Consequently, a comprehensive assessment based on the overall clinical context is essential.

Limitations and Future Directions

The results of this study provide warning signs for patients at a high risk of AKI after CVS. In the future, the prediction model can be embedded into the electronic medical record system to achieve automatic calculations. For high-risk patients identified by the model before the operation, the system will automatically issue an alarm and trigger a standardized bundle of postoperative nephroprotective measures, such as optimizing hemodynamics, avoiding nephrotoxic drugs, and closely monitoring SCr and urine output.[46] This series of active renal protection intervention measures would reduce the incidence of postoperative AKI and improve patient outcomes. Most importantly, a prospective, interventional randomized controlled trial will be essential for validating the clinical utility of our model.

It has some limitations in this study. First, it was a single-center retrospective study. As such, it was unable to completely rule out the impact of some unmeasured confounding variables. Second, some cases were excluded due to missing variables and the sample size's relative limitations, which could result in a certain degree of bias. Exclusion of CKD, combined CABG, and other patients may introduce bias, and therefore, our results may not apply to these higher-risk groups. External validation in multicenter studies involving more diverse populations is required in the future. Third, as patients' urine output data were not recorded, AKI was diagnosed only using the SCr criteria, and the urine output criteria were not used. Future prospective validation using the full KDIGO criteria is necessary. Fourth, it was unable to compare our model directly against established renal risk scores for cardiac surgery due to missing data for several key variables. A future prospective study, designed to collect all necessary data for a head-to-head comparison against risk scores such as the Cleveland Clinic Score, is essential to definitively establish the clinical utility and incremental benefit of sUAR. Besides, it is also necessary to prospectively study whether the combination of sUAR and other biomarkers (such as [TIMP2] × [IGFBP7]) can improve the prediction of AKI after cardiac surgery. Finally, there is a lack of external validation of the prediction model, which requires further verification from other centers.

Conclusion

The elevated preoperative sUAR was independently associated with a high risk of AKI following CVS. The prediction model constructed based on sUAR has good predictive value and clinical applicability, which can assist medical professionals in recognizing patients at a greater risk of AKI early during the perioperative period and take corresponding intervention measures to reduce postoperative AKI risk and improve prognosis. Before being put into clinical application, prospective and multi-center studies are still needed to externally validate this model.

Data Sharing Statement

The data supporting the research results of this study can be obtained from the corresponding author upon reasonable request.

Ethics Approval and Informed Consent

The Medical Ethics Committee of Henan Provincial People's Hospital approved the study (approval number: 2020-Lunshen-206). The ethics committee waived the informed consent, since the study was retrospective in nature. The data were anonymous, and all personal identifiers were removed before analysis to ensure patient confidentiality. This study was conducted according to the Declaration of Helsinki.

Author Contributions

Xiaoru Zhao: Conceptualization, Formal analysis, Investigation, Writing–Original Draft. Juntao Wang: Conceptualization, Formal analysis, Investigation, Writing–Original Draft. Wenxin Bai: Investigation, Formal analysis, Data Curation. Wenwen Zhang: Methodology, Data Curation. Chunling Huang: Formal analysis, Investigation. Zengyuan Qin: Investigation, Formal analysis. Kaiyue Wei: Methodology, Investigation. Minghui Han: Validation, Data Curation, Formal analysis, Software, Visualization. Lei Yan: Methodology, Writing–Review & Editing. Yue Gu: Supervision, Resources, Conceptualization. Fengmin Shao: Supervision, Resources, Conceptualization, Project administration, Funding Acquisition. 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. All authors gave final approval of the version to be published.

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Disclosure

The authors report no conflicts of interest in this work.

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