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Correlation Between Uric Acid/High Density Lipoprotein Cholesterol Ratio and Postoperative AKI in Patients with CABG

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Objective: This study aims to investigate the association between preoperative serum uric acid to high-density lipoprotein cholesterol ratio (UHR) and the occurrence of postoperative acute kidney injury (AKI) in patients who underwent coronary artery bypass grafting (CABG).

Methods: A prospective observational study was conducted at Fujian Heart Medical Center between May 2022 and December 2023, recruiting patients scheduled for CABG. Participants were categorized into two groups based on AKI occurrence post-surgery. Univariate and multivariate logistic regression analyses were performed to identify predictor variables for AKI after CABG. A nomogram was constructed based on these predictors, and its calibration was evaluated using the receiver operating characteristic curve (ROC) and Hosmer-Lemeshow goodness of fit test. The diagnostic value of UHR in AKI after CABG was explored using the area under the curve (AUC).

Results: The study included 301 CABG patients, of whom 72 (23.92%) developed AKI. After adjusting for age, gender, body mass index, and extracorporeal circulation, binary logistic regression analysis revealed that a higher UHR value was an independent risk factor for developing AKI after CABG (OR=7.410, 95% CI: 3.829–14.855), P <0.05. The prediction nomogram demonstrated excellent discriminability, with an AUC of 0.87 and good calibration (Hosmer-Lemeshow test, P <0.05). Compared with other clinical indicators, ROC analysis indicated that UHR had the largest AUC (0.821), corresponding to 70.8% sensitivity and 79.0% specificity.

Conclusion: Higher UHR was associated with an increased risk of AKI after CABG and may serve as a prospective biomarker for predicting AKI.

Keywords: UHR, CABG, AKI, Nomogram

Introduction

Coronary atherosclerotic heart disease (CAD) stands as a significant cardiovascular ailment with global health implications. Accounting for one-third of all deaths in individuals over 35 years old, and up to 50% in Western countries.[1,2] Coronary Artery Bypass Grafting (CABG) emerges as the primary surgical intervention for CAD. However, CABG poses a notable risk of renal injury due to its impact on renal blood flow distribution, increased renal vascular resistance, and altered renal perfusion.[3] Reports indicate that the incidence of AKI following CABG varies from 6.7% to 45%, with 1% to 2% of patients necessitating renal-replacement therapy.[4–6] Besides prolonging ICU and total hospital stays, AKI elevates in-hospital mortality risk. The Kidney Disease: Improving Global Outcomes (KDIGO) criteria define AKI as a serum creatinine increase of 0.3 mg/dl above baseline or persistent oliguria (lasting over 6 hours).[7] However, the time required for diagnosis using these criteria impedes the development of effective mitigation strategies for AKI.[8] Early

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identification and warning of patients at high risk of AKI post-CABG assume paramount importance in guiding tailored treatment and enhancing patient prognosis.

The uric acid to high-density lipoprotein cholesterol ratio (UHR), a novel marker of inflammation and metabolism, reflects the balance between oxidative stress and antioxidant capacity in the body.[9,10] While UHR has been utilized in various studies, including incident ischemic heart disease,[11] sacroiliac arthritis[12] and diabetic renal injuries.[10] But its correlation with AKI post-CABG remains unexplored. Serum uric acid (UA), a byproduct of purine catabolism, inhibits nitric oxide generation, promoting endothelial dysfunction and vascular smooth muscle proliferation.[13] Conversely, High-Density Lipoprotein Cholesterol (HDL-C), a lipid parameter, plays a role in inhibiting blood oxidation and safeguarding endothelial cells.[14] Elevated UA levels and reduced HDL-C are closely linked to inflammation, endothelial dysfunction and oxidative stress, which are pivotal mechanisms in AKI development and progression.

Previous research has established the association of both UA and HDL-C with the development of AKI after cardiac surgery,[15,16] highlighting a synergistic effect between high UA and low HDL-C values. However, no study has yet delved into the comprehensive predictive value of their ratio (UHR) specifically for postoperative AKI after CABG. Additionally, the predictive efficacy of UHR might vary across different patient cohorts, warranting further investigations to ascertain its clinical utility as a dependable predictor of AKI in CABG patients. Therefore, we aimed to explore the correlation between UHR and AKI after CABG to identify the occurrence of AKI after CABG at an early stage, and provided a broader evidence-based basis for the diagnosis and treatment of AKI.

Methods

Protocol Recruitment of the Study Subjects

This was a prospective observational study. A total of 336 patients with CAD who planned to undergo CABG surgery were enrolled in the study at Fujian Heart Medical Center from May 1, 2022, to December 31, 2023. Ultimately, 301 subjects were enrolled in the study. The subject flowchart is shown in Figure 1. All patients were diagnosed with atherosclerosis by coronary angiography before operation; aged ≥18 years old; no history of malignant tumors; no previous history of cardiac surgery; no autoimmune disease; no chronic liver and kidney functional failure. All patients were anesthetized and operated on by the same anesthesia group and the same treatment group. The endpoint of the study was AKI within 7 days after surgery. This study was approved by the Ethics Committee of Fujian Medical University

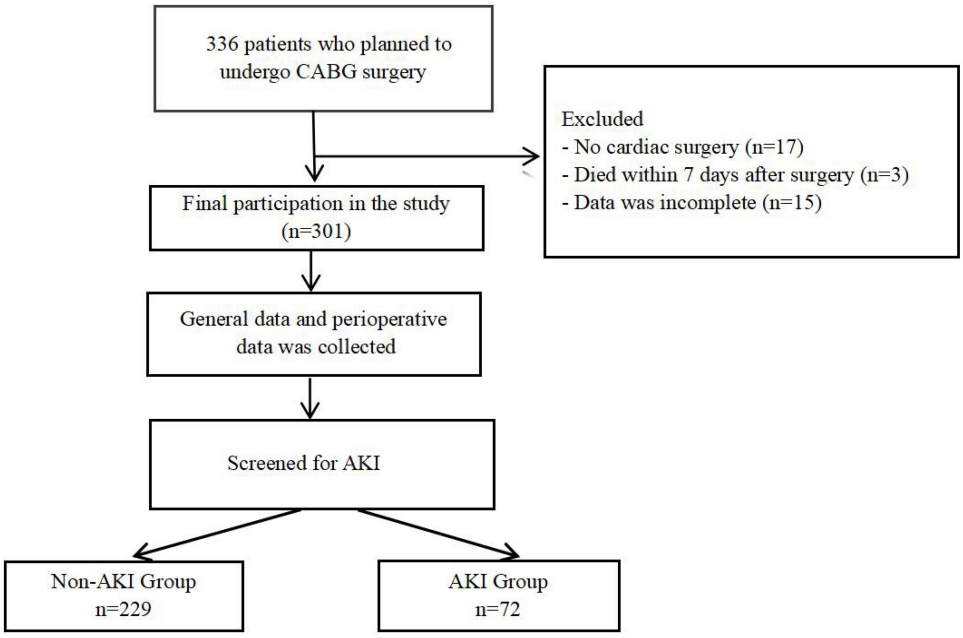

Figure 1 Patients' flowchart of the study.

Union Hospital (No. 2022KY082) and followed the tenets of the Declaration of Helsinki. Patients and family members already signed an informed consent form and volunteered to participate in the study.

Data Collection

A self-made questionnaire was used to collect general demographic information about the subjects, including age, gender, current medical history, past history, and personal history; Measurement of the height and weight of the study subjects. Venous blood was collected the following morning on an empty stomach after a 12-hour fast. The measurements included white blood cells (WBCs), Lymphocytes, Monocytes, glycated hemoglobin (HbA1c), brain natriuretic peptide (BNP), total cholesterol(TC), High Density Lipoprotein Cholesterol (HDL-C), Low Density Lipoprotein Cholesterol (LDL-C), uric nitrogen (UN), uric acid (UA), serum creatinine (SCr). Additionally, the patients' final postoperative outcome were tracked for AKI according to the medical record, along with the duration of mechanical ventilation, ICU stay, use of continuous renal replacement therapy (CRRT), secondary endotracheal intubation, secondary chest opening and in-hospital death.

Definition

According to KDIGO (2012) criteria, The diagnostic criteria for AKI were as follows: (1) a serum creatinine (Scr) level which is absolutely increased to ≥26.5 μmol/L (0.3 mg/dL) within 48 h; (2) an over 1.5-fold increase in Scr compared with the initial level within 7 days; (3) a urinary output that was less than 0.5 A mL/(kg·h) for 6 consecutive hours. AKI was diagnosed when any of the above points were met. The Scr value was based on the last serum Scr before surgery. AKI after CABG was defined as the AKI that occurred within 7 days after surgery. The occurrence of AKI was diagnosed by a specialized clinician with reference to this criterion. Smoking was defined as smoking more than one cigarette a day for at least 6 months. BMI= Weight/Height ^{2} (kg/m^2). UHR=UA (mg/dl) /HDL-C (mg/dl).

Sample Size Calculation

According to the overall rate estimation formula, the sample size calculation formula is $n = \frac{Z^2_{\alpha/2}\pi(1-\pi)}{\delta^2}$. According to the literature review, the incidence of postoperative AKI in patients with CABG was 26.5%.[17] The margin of error was controlled at δ = Π× 0.2, which resulted in δ = 0.053; and the α was set at 0.05. The sample size was expanded by 10% to take into account factors such as shedding. Therefore, the final calculated sample size was 293 cases.

Statistical Analysis

SPSS 26.0 and R version 3.5.1 were used for the analyses. The Kolmogorov–Smirnov test was used to determine if the parameters conformed to a normal distribution for continuous variables. Normally distributed data was expressed as mean±standard deviation; the median (interquartile range) was used to express the non-normal distribution data. The frequency (percentage) was used to represent the categorical variable. Student's *t*-test, Mann–Whitney test and χ^2 test or Fisher exact test were performed for comparative analysis between the groups for normally distributed data, non-normally distributed data and counting data, respectively. This study was a single factor analysis of all variables with the occurrence of AKI after CABG as the dependent variable. The statistical variables with $P < 0.2$ in the univariate analysis were further included in the multivariate Logistic regression analysis equation. The prediction model graph was established based on multivariate logistic regression analysis. ROC curve was drawn to calculate the AUC to evaluate the discriminant performance of the model. Calibration curves were drawn and the nomogram calibration was evaluated by Hosmer-Lemeshow goodness of fit test. Additionally, the diagnostic value of UHR was analyzed according to AUC. In the present study, a value of $P < 0.05$ was considered statistically significant.

Result

General Data and Laboratory Indicators

According to the diagnostic criteria for AKI, CABG patients were categorized into the non-AKI group (229) and the AKI group (72). Comparing the baseline characteristics of the two cohorts, there was no statistically significant difference

between the two groups in terms of baseline information such as age, BMI, gender, smoker, and hypertension ($p > 0.05$). There was a statistical difference in CPB, WBCs, Monocytes, BNP, HDL-C, UN, UA, SCr and UHR ($p < 0.05$) (Table 1).

The Risk Factors of AKI After CABG Were Analyzed by Logistic Regression

Clinical characteristics associated with AKI after CABG were identified by univariate analysis (Table 1). Clinical characteristics with $p<0.20$ were included in multifactor Logistic regression analysis. According to the best truncation value of Youden index, the continuous variable was transformed into categorical variable with AKI as the dependent variable and UHR as the independent variable. BNP, Scr, UN, WBCs, Monocytes and UHR were divided into high and low levels. With low value as a reference, MV ≥36H and high value UHR, Scr, and Monocytes were risk factors for AKI after CABG after correction for age, sex, BMI, MV, CPB, BNP, Scr, UN, WBCs, and Monocytes (Table 2, Model 1). Higher UHR, Scr and Monocytes remained the risk factors for AKI after CABG after further adjustment of CPB and MV (Table 2, Model 2).

Development and Validation of the Prediction Nomogram

According to the results of univariate and multivariate analysis, the OR values of the indicators included in the model were plotted into forest maps (Figure 2A). We further developed a nomogram with predictive variables to predict postoperative AKI in patients with CABG (Figure 2B). Nomogram was used as follows. Firstly, draw a line up to the top point axis to get the point for that variable on the relevant axis where the patient's variable is located. Then repeat this process for each covariate and calculate the total score by summing all the points obtained from each covariate. The final sum was located on the Total Points axis, from which a straight line was drawn down to obtain the probability of AKI after CABG. The ROC showed that

Table 1 Baseline Characteristics and Laboratory Indicators in Non-AKI Vs AKI Group

Characteristics	Non-AKI (n=229)	AKI (n=72)	F/Z/χ2	P
Age (y)	62 [55, 68]	64 [56, 69]	0.752	0.452
Male, (n,%)	180(78.60)	51(70.83)	1.285	0.257
BMI, kg/m^2	23.88[21.88,26.02]	23.88[22.03,25.43]	0.383	0.703
Smoker, n(%)	115(50.22)	34(47.22)	0.197	0.657
Hypertension, n(%)	132(57.642)	47(65.278)	1.325	0.250
History of brain injury, n(%)	17(7.42)	6(8.33)	0.064	0.800
History of diabetes, n(%)	96(41.921)	29(40.278)	0.061	0.805
CPB, n(%)	38(16.594)	23(31.944)	7.988	**0.005**
WBCs, 10^9/L	6.71[5.85, 7.97]	7.36[6.19,9.01]	2.150	**0.032**
Lymphocytes, 10^9/L	1.85[1.45,2.31]	1.74[1.25,2.31]	0.870	0.385
Monocytes, 10^9/L	0.48[0.38,0.61]	0.57[0.45,0.69]	2.745	**0.006**
HbA1C, (%)	6.30[5.90,7.60]	6.40[5.90,7.50]	0.189	0.850
Hemoglobin	133[121,145]	131[117,146]	0.661	0.509
BNP, pg/mL	240[86,733]	571[151,1691]	3.219	**0.001**
TC, mmol/L	4.04[3.38,5.07]	4.02[3.42,5.00]	0.286	0.776
LDL-C, mmol/L	2.59[2.00,3.35]	2.57[2.06,3.33]	0.566	0.572
HDL-C, mmol/L	0.97[0.85,1.14]	0.84[0.75,1.01]	4.21	**<0.001**
UN, mmol/L	5.40[4.30,6.30]	6.40[5.00,8.60]	3.865	**<0.001**
UA, mmol/L	333[280,410]	469[373,525]	7.142	**<0.001**
Scr, mmol/L	76[65,87]	94[79,110]	5.843	**<0.001**
UHR	351.61 [265.09,434.26]	535.79[419.10,661.02]	8.219	**<0.001**

Notes: Continuous and categorical variables are expressed as mean (standard deviation) or median (interquartile range) and percentage, respectively. The classified data were tested using the chi-squared test. If they were in line with normal distribution and homogeneity of variance, an independent sample *t*-test was used for the comparison of quantitative data between groups. If the variance is not normal, the rank-sum test was performed. $p < 0.05$ indicated significance and bolded in the table.

Abbreviations: BMI, body mass index; WBCs, white blood cells; HbA1C, glycosylated hemoglobin; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; BNP, brain natriuretic peptide; UN, uric nitrogen; UA, uric acid; SCr, serum creatinine; CPB, cardiopulmonary bypass; UHR, uric acid/ high - density lipoprotein cholesterol ratio.

Table 2 Multivariate Analysis of AKI in Patients with CABG

Variables	Model 1		Model 2	
	OR [95% CI]	***P***	**OR [95% CI]**	***P***
Age, years	1.003[0.967,1.040]	0.867	0.995[0.963,1.029]	0.789
BMI, Kg/m^2	0.942[0.842,1.058]	0.313	0.959[0.868,1.062]	0.423
Male, n(%)	0.863[0.315,2.471]	0.777	0.743[0.298,1.921]	0.529
CPB	2.136[0.940,4.852]	0.069	–	–
MV≥36H	7.473[3.395,17.226]	**<0.0001**	–	–
BNP, pg/mL				
≤ 1227	**Reference**			
>1227	0.793[0.321,1.1883]	0.606	1.412[0.623,3.132]	0.400
Scr, mmol/L				
≤ 89	**Reference**			
> 89	2.879[1.253,6.640]	**0.012**	2.659[1.266,5.579]	**0.009**
UN, mmol/L				
≤ 7.8	**Reference**			
> 7.8	1.3685[0.663,4.254]	0.269	1.841[0.778,4.317]	0.161
WBCs, 10^9/L				
≤ 7.16	**Reference**			
> 7.16	1.55[0.714,3.367]	0.266	1.502[0.738,3.057]	0.260
Monocytes, 10^9/L				
≤ 0.62	**Reference**			
> 0.62	2.413[1.061,5.581]	**0.036**	2.233[1.042,4.847]	**0.039**
UHR				
≤ 454.1	**Reference**			
>454.1	8.403[4.030,18.500]	**<0.0001**	7.410[3.829,14.855]	**<0.0001**

Note: $p < 0.05$ indicated significance and bolded in the table.
Abbreviations: BMI, body mass index; CPB, cardiopulmonary bypass; MV, mechanical ventilation; BNP, brain natriuretic peptide; SCr, serum creatinine; UN, uric nitrogen; WBCs, white blood cells; UHR, uric acid/ high - density lipoprotein cholesterol ratio; OR, odds ratio; CI, confidence interval.

the model had good discriminatory power with AUC at 0.87 (95% Cl = 0.82–0.92)(Figure 2C). The Hosmer-Lemeshow test difference was not statistically significant ($p > 0.05$), indicating a good model fit. In addition, the calibration charts graphically showed that there was consistency between the risks predicted by the model and actual observations (Figure 2D).

Predictive Value of UHR for Postoperative AKI

ROC curve analysis was used and AUC was calculated to assess the value of UHR for diagnosing AKI after CABG. The AUC of UHR was 0.821, with a diagnostic sensitivity of 70.8% and a specificity of 79.0%; the AUC of Scr was 0.728, with a diagnostic sensitivity of 59.7% and a specificity of 78.6%; the AUC of Monocytes was 0.607, with a diagnostic sensitivity of 44.4% and specificity of 77.3%. UHR has better predictive value in identifying AKI after CABG. The results of the ROC analysis are shown in Figure 3.

In-Hospital Events of CABG Patients in Different UHR Level Groups

We found that the threshold point for UHR to diagnose AKI was 454.10 in ROC analysis. The UHR was categorized into a low UHR (<454.10) group and a high UHR (≥454.10) group by utilizing this threshold. Table 3 shows the in-hospital

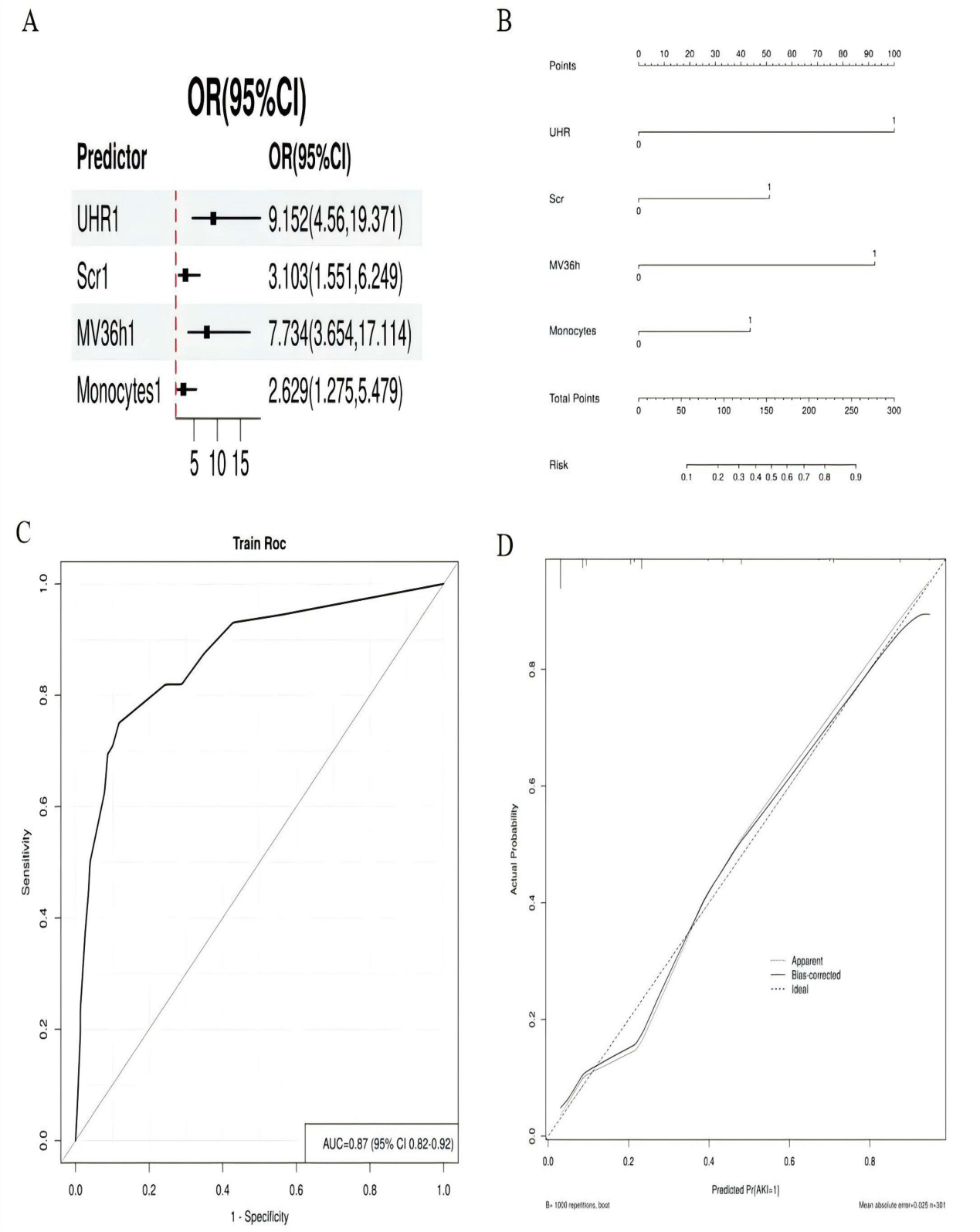

Figure 2 Development and validation of the prediction nomogram (**A**) Forest plot for assessing the risk of developing postoperative AKI in patients with CABG. (**B**) Nomogram for assessing the risk of developing postoperative AKI in patients with CABG. (**C**) ROC curve for evaluating the model's discrimination performance. The area under the ROC curve was 0.87. (**D**) Calibration curves for the nomogram. The x-axis represents the nomogram-predicted probability, and the y-axis represents the actual probability of the nomogram. The dashed line represents the entire cohort (n=301), and the solid line is bias-corrected by bootstrapping (B=1000 repetitions), indicating observed nomogram performance. The mean absolute error = 0.025(2.5%).

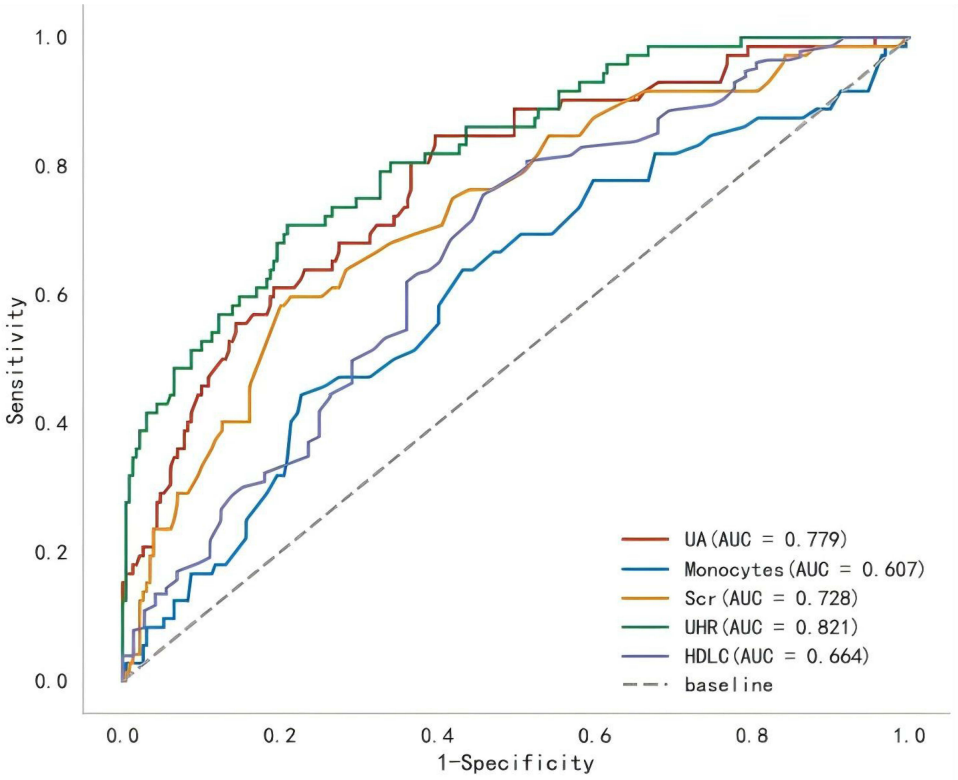

Figure 3 The receiver operating characteristic curves of predict AKI of patients with CABG.

outcomes of CABG patients in both groups. The difference between the two groups in terms of MV ≥36H, AKI, CRRT, and 30-day all-cause mortality was statistically significant ($P<0.05$). There was no statistically significant difference in the comparison of the two groups in terms of secondary tracheal intubation, secondary thoracotomy, secondary ICU transfer, ICU stay, LOS ($P>0.05$).

Discussion

The study revealed a 23.92% incidence of AKI in CABG patients, aligning with findings from prior research.[4,6,18] Additionally, it conducted a comparative analysis of Uric Acid to High-Density Lipoprotein Cholesterol Ratio (UHR)

Table 3 The in-Hospital Events in Patients with CABG According UHR

Variables	Low UHR; <454.10 (n=202)	High UHR; ≥454.10 (n = 99)	F/Z/χ2	*p*
Secondary tracheal intubation, n (%)	8(3.96)	5(5.05)	0.191	0.662
Secondary thoracotomy, n (%)	7(3.47)	4(4.04)	0.062	0.803
Secondary ICU transfer, n (%)	10(4.95)	7(7.07)	0.56	0.454
MV≥36H, n (%)	34[16,83]	27[27,27]	4.482	**0.034**
ICU stay (h)	44[38.0,68.5]	47[38.5,93.5]	1.396	0.163
LOS, Day	20[16,28]	21[18,28]	0.672	0.501
AKI, n (%)	21 (10.40)	51(51.52)	61.727	**<0.001**
CRRT, n (%)	3(1.49)	6(6.06)	4.795	**0.029**
30 days all-cause mortality, n (%)	6 (2.97)	8(8.08)	3.913	**0.048**

Note: $p < 0.05$ indicated significance and bolded in the table.
Abbreviations: MV, mechanical ventilation; ICU, intensive care unit; LOS, Length of stay; AKI, Acute kidney injury; CRRT, continuous renal replacement therapy.

differences between AKI and non-AKI patients post-CABG. Logistic regression analysis indicated that elevated UHR was an independent risk factor for AKI post-CABG (OR=7.410, 95% CI: 3.829~14.855), with a significant *p*-value ($p<0.05$) after adjusting for confounding variables. The constructed prediction nomogram exhibited excellent discriminability, with an area under the ROC curve of 0.880, indicating good predictive accuracy. Notably, UHR demonstrated the largest area under the ROC curve (AUC=0.821), with corresponding sensitivity of 70.8% and specificity of 79.0%, surpassing other clinical indicators. These findings underscore the potential utility of UHR as a predictive marker for AKI development post-CABG.

Currently, the pathogenesis of AKI remains elusive, with inflammation and oxidative stress emerging as early pivotal events in its progression.[19] Guarda NS's study highlighted UHR as a novel inflammatory and metabolic marker with significant predictive value in early diabetic nephropathy,[20] which was consistent with the results of this study. UHR, derived from serum UA divided by HDL-C, has garnered attention due to its association with kidney damage. Elevated UA levels, as demonstrated by Zhou F's six-year cohort study, are linked to a rapid decline in eGFR and kidney impairment.[21] Additionally, research by Lu J suggests that higher UA levels correlate with the risk of contrast media-induced AKI following coronary angiography.[22] UA, functioning as a weak organic acid, can precipitate into monosodium urate crystals in serum under specific pH and temperature conditions. Exceeding 6.5 mg/dL, UA can lead to tissue damage through oversaturated monosodium urate crystal deposition in the kidneys.[23] Furthermore, intracellular UA conversion to a pro-oxidant fosters reactive oxygen species (ROS) production, a crucial mechanism in kidney ischemia and reperfusion injury.[24] The increase in ROS is precisely an important mechanism for kidney ischemia and re -injection. Research indicates that reduced LDL-C levels are strongly associated with poor cardiovascular outcomes, whereas HDL-C was known for its cardioprotective effects due to its antioxidant and anti-inflammatory properties.[16] An observational study found that higher preoperative HDL-C levels were independently correlated with a lower risk of AKI following cardiac surgery.[25] Consistent with these findings, our study revealed that the AKI group had lower HDL-C levels compared to the non-AKI group. HDL-C plays a crucial role in mitigating the inflammatory response by regulating monocyte activation, adhesion, and the proliferation of progenitor cells that can differentiate into monocytes.[26] Additionally, HDL-C protects endothelial cells from LDL-C and prevents LDL-C oxidation.[27] In combination, high UA concentration and low HDL-C concentration create synergistic effects that exacerbate inflammation and oxidative stress.

It was worth noting that our research focuses on the value of early UHR in predicting AKI after CABG. A well-differentiated and calibrated prediction model was constructed by further combining Scr, MV≥36H, and Monocytes. While numerous established risk factors for AKI after CABG were identified, including MV, CPB, WBCs, Monocytes, BNP, SCr, UA, and UN, our study revealed that WBCs, BNP, and UN were not independent risk factors in multivariate analysis, possibly due to the dilution effect of other risk factors.

Higher preoperative baseline Scr level was also a risk factor for AKI after CABG in this study, matching the findings of Chen SW[28] and Xu J.[29] Higher Scr was generally a sign of decreased glomerular filtration rate. And higher preoperative Scr indicates that patients undergoing elective surgery may have hidden kidney injury preoperatively. Secondly, certain surgical procedures, such as extracorporeal circulation, can be stressful for the patient and have an impact on the kidneys. Renal hypoperfusion during CPB, inflammation and oxidative stress both increase the risk of AKI.[25,30] The results of this study also indicate that mechanical ventilation time greater than 36H is an independent risk factor for AKI after CABG. On the one hand, MV affects the function of glomerular filtration and can easily lead to AKI by altering renal hemodynamics, neurohumoral and inflammatory transmitter release. On the other hand, AKI can lead to pulmonary edema and thus prolong mechanical ventilation. This causes a vicious circle. Monocytes have also been shown to be highly predictive of AKI after CABG. Monocytes were also associated with AKI in the present study. However, it was not superior to UHR in predicting AKI based on the AUC of ROC.

The classification of CABG patients into high and low UHR groups based on a cutoff value allowed for statistical analysis of short-term postoperative outcomes. Patients in the high UHR group exhibited a higher incidence of post-operative morbidities, including MV≥36H, AKI, CRRT, and 30-day all-cause mortality. These findings underscore the significant impact of UHR on CABG-associated AKI and emphasize its importance as a reliable and readily available biomarker for predicting AKI in CABG patients. In summary, UHR, an inexpensive and widely available parameter, is a reliable biomarker for predicting AKI in patients with CABG.

Limitation

The present study has several limitations. Firstly, the study was a single-center observational study. The findings have not been verified in external study populations. Larger sample sizes and multi-center trials are needed in the future to determine the value of UHR in predicting AKI after CABG. Secondly, our study dealt only with in-hospital complications and short-term survival, and did not follow up the long-term prognosis of the patients.

Conclusion

The identification of higher UHR as an independent risk factor for AKI after CABG highlights its significance in perioperative care. Its superior predictive value compared to other factors emphasizes the importance of monitoring UHR indicators and managing UA and HDL-C levels during the perioperative period for CABG patients. Integrating UHR assessment into clinical practice can enhance risk assessment and facilitate proactive interventions to mitigate AKI risk, ultimately improving patient outcomes and postoperative care efficacy.

Data Sharing Statement

The data for this study were available by contacting the corresponding author upon reasonable request.

Ethics Statement

The investigation conformed with the principles outlined in the Declaration of Helsinki and was approved by the hospital's Research Ethics Committee (Ethics Approve No. 2022KY082). Informed consent was obtained from patients before this study.

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Author Contributions

Fei Jiang, Yanchun Peng and Yuezhen Hong contributed equally to this work and share first authorship. 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 declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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