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ORIGINAL RESEARCH

Machine Learning Models for Identifying the Risk of Chronic Kidney Disease in Patients with Coronary Heart Disease: A Retrospective Study

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Purpose: Coronary heart disease (CHD) has a significant co-morbid association with chronic kidney disease (CKD), but identification tools for the risk of concomitant CKD in patients with CHD are still lacking. The purpose of this research was to construct machine learning (ML) models for identifying undetected CKD in CHD patients.

Methods: 1786 CHD patients undergoing coronary intervention were retrospectively included. Lasso regression and multifactor logistic regression were used to screen feature variables. Five ML models, ie, logistic regression (LR), support vector machine (SVM), random forest (RF), gradient boosting machine (GBM), and extreme gradient boosting (XGBoost), were constructed. Participants were divided into the training set and validation set in a 7:3 ratio. The evaluation metrics included the area under the curve, calibration curve, and decision curve.

Results: Totally, 1786 CHD patients were enrolled and split into training (70%) and validation (30%) sets. The prevalence of CKD was 21.8% (390/1786). Multivariate logistic regression analysis showed that men, advanced age, hypertension, diabetes mellitus, history of atrial fibrillation (AF), high Gensini, low hemoglobin, low plateletcrit (PCT), high triglycerides (TG), high lipoprotein(a) (Lp(a)), hyperkalemia, high uric acid to albumin ratio (UAR), high systemic inflammation response index (SIRI), low lymphocyte to monocyte ratio (LMR), and high apolipoprotein B to apolipoprotein A1 (ApoB/ApoA1) ratio were the key clinical and laboratory test indicators of CKD. The XGBoost model performed optimally in the validation set (AUC=0.909, 95% CI 0.881 −0.937). SHapley Additive explanation analysis identified UAR, hypertension, Gensini score, age, and SIRI as the top 5 key features.

Conclusion: The XGBoost model constructed on routine clinical data was effective in identifying CKD risk in CHD patients, with UAR as a novel strong predictor. Decision curve analysis confirmed the clinical utility of the model, indicating that it may be used to guide decisions for enhanced monitoring and early intervention over a wide range of risk thresholds.

Keywords: coronary heart disease, chronic kidney disease, machine learning, XGBoost

Introduction

Cardiovascular diseases (CVD) remain the foremost contributor to mortality globally, accounting for approximately 17.9 million deaths annually (WHO, 2023). Coronary heart disease (CHD) is a major subtype of CVD, with a global prevalence of 113 million (GBD 2021). The prevalence of CVD in China continues to rise. There were 330 million CVD cases, of which 11.39 million were CHD cases, constituting a primary cause of death in the Chinese population.[1] Chronic kidney disease (CKD) now constitutes a major global disease burden, affects 13.4% of the global population (approximately 850 million people), and China has the largest number of CKD patients (approximately 150 million), accounting for nearly one-fifth of the global total.[2] CHD has a significant co-morbid association with CKD. On the one hand, patients with CKD exhibit substantially elevated incidence and mortality of CVD (including CHD) due to vascular

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calcification, chronic inflammation, and metabolic disorders.[3,4] On the other hand, CHD is often accompanied by renal impairment, and CKD is accorded equivalent risk status to CHD, with the two sharing the same risk factors of atherosclerosis (AS), hypertension, and diabetes mellitus.[5,6] Patients with CHD often exhibit an elevated risk of CKD owing to comorbid metabolic diseases such as hypertension and diabetes.[7,8] Early identification of CKD risk in patients with CHD is essential to optimize clinical management and reduce complications and mortality.

In incipient CKD, the awareness and diagnosis of CKD are low owing to the lack of clear clinical symptoms.[9] Moreover, identifying the risk of concurrent CKD in individuals with CHD still relies on traditional statistical models (eg, Cox regression) in current clinical practice, which are limited in their ability to select variables and capture interactions.[10] In recent years, machine learning (ML) has shown tremendous promise in the prediction of the risk of CKD. For example, by integrating variables such as biochemical indicators (eg, urinary proteins, serum creatinine), demographic characteristics (age, gender), and co-morbidities information (diabetes mellitus, hypertension), we can predict the progression of end-stage renal disease (ESKD) more accurately.[3,11,12] In addition, ML models have also made progress in the risk stratification of CVDs. For example, some models are constructed using electronic medical record data, and they can identify CHD patients at high risk for acute cardiovascular events.[7,11,13] However, existing studies have applied ML to predict the progression of CKD or cardiovascular events, but few studies focus on the risk of CKD in CHD patients. Traditional models (eg, Cox regression) have limitations in variable selection and capturing non-linear interactions, and do not integrate novel composite indicators (eg, UAR, SIRI) reflecting inflammation and nutrition, leading to insufficient predictive accuracy for this population. Due to the overlap in pathomechanisms between the two diseases (eg, arterial calcification, oxidative stress, inflammation),[7,14,15] the development of ML-based multimodal models (combining laboratory tests, imaging features, and dynamic physiological parameters) is expected to break through the limitations of the traditional methods.[16,17] This direction of research is not only in line with the needs of precision medicine, but also may improve the current situation of patients with comorbid CHD and CKD, who suffer from a high in-hospital mortality rate and a poor prognosis.[6,18]

This study aimed to construct five ML models (LR, SVM, RF, GBM, XGBoost) based on routine clinical data to identify preexisting CKD in patients with CHD, identify key risk factors via SHAP, especially UAR, and evaluate the optimal model's clinical utility to guide personalized renal monitoring.

Methods

Study Population

As per the Declaration of Helsinki, this study was approved by the Ethics Committee of the Central Hospital of Enshi Tujia and Miao Autonomous Prefecture. (Approval Number: [2025-110-01]; Date of Approval: [July 29, 2025]). Although the Ethics Committee granted exemption from informed consent for this retrospective medical record analysis, a certificate of approval from the Ethics Committee was provided to the editors to ensure ethical compliance. First, this cross-sectional study exclusively utilized clinical case data, ensuring no adverse consequences or harm to patients. Second, all patient data were anonymized before data access and analysis, enabling medical record review without informed consent. In this study, we retrospectively collected CHD patients who were hospitalized for the evaluation and treatment of CHD and subsequently underwent coronary intervention in the Department of Cardiology, Enshi Tujia and Miao Autonomous Prefecture Central Hospital from January 2022 to December 2024. This cohort included all patients with CHD, including scheduled elective surgery and urgent intervention after an acute coronary syndrome. The study was conducted at the Cardiovascular Disease Center of the Central Hospital of Enshi Tujia and Miao Autonomous Prefecture, a tertiary care center serving a large regional population. The center manages a high volume of CHD patients, with over 2000 CHD-related admissions annually. The inclusion criteria were (i) 18 years of age or older; (ii) with clinically evident CHD. The exclusion criteria were (i) non-atherosclerotic coronary artery disease; (ii) failure to adhere to standard postoperative regimens of statins and antiplatelet agents; (iii) patients with previous definite diagnosis of CKD or renal insufficiency; (iv) all included patients underwent coronary intervention (PCI), while those who used contrast agents within 1 month before admission were excluded to avoid the potential impact of contrast agents on the assessment of renal function; (v) occurrence of other postoperative cardiovascular adverse events; (vi) presence of other systemic

diseases, including immune system diseases, hematologic diseases, liver diseases, malignant tumors, and serious infections.

Data Collection

General demographic data of all patients were drawn, encompassing age, gender, and smoking status; medical history, mainly including history of hypertension, diabetes mellitus, and cerebrovascular disease (stroke, cerebral infarction). Laboratory and clinical data were drawn from the first venous blood count of all patients after admission to the hospital, including leukocyte count, neutrophil count, monocyte count, lymphocyte count, platelet count, hemoglobin, albumin (ALB), lipid profile, blood glucose, bilirubin, creatinine, uric acid, and other auxiliary examination results. Patients' left main and multi-vessel coronary artery disease were recorded based on angiographic findings. The presence of significant coronary artery disease was adjudicated based on coronary angiography findings by two independent interventional cardiologists, with disagreements resolved by a third senior cardiologist (left main disease: stenosis $\geq$ 50% in left main artery; multi-vessel disease: stenosis $\geq$ 50% in at least 2 major arteries). The Gensini score was used to evaluate the severity of their coronary artery lesions. The patients were categorized into the CHD combined with CKD group and the CHD without CKD group. The inflammatory indices selected for this study were calculated based on relevant laboratory data, encompassing uric acid to albumin ratio (UAR), aggregate index of systemic inflammation (AISI, neutrophils×monocytes×platelets/lymphocytes), systemic inflammation response index (SIRI, neutrophils×monocytes/lymphocytes), systemic immune-inflammation index (SII, neutrophils×platelets/lymphocytes), neutrophil percentage to albumin ratio (NPAR), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), lymphocyte-to-monocyte ratio (LMR), and apolipoprotein B to apolipoprotein A1 (ApoB/ApoA1) ratio.

Definition of CKD

The outcome was the presence of undiagnosed CKD on admission. CKD was defined as serum creatinine calculated from the CKD-EPI formula according to the 2024 KDIGO Clinical Practice guidelines[19] for the Evaluation and Management of Chronic Kidney disease, based on laboratory data obtained at the time of this admission, in patients with no previous history of CKD or renal insufficiency. The estimated glomerular filtration rate (eGFR) was < 60 mL/min/1.73m^2.

Statistical Analysis

In terms of missing data, variables with > 20% missing values were excluded; those with < 20% missing values were imputed using multiple imputation with 5 iterations based on the chained equations method. For data preprocessing, outliers were handled via winsorization, where values exceeding the 99th percentile were capped at the 99th percentile and values below the 1st percentile were floored at the 1st percentile. To control data leakage, all preprocessing steps (including imputation, winsorization, and feature selection) were performed exclusively on the training set, and the same parameters derived from the training set were applied to the validation set to avoid information leakage between sets. For each ML model, patients were randomly divided into training and validation sets in a ratio of 7:3. To reduce the risk of overfitting, this study combined the least absolute shrinkage and selection operator (LASSO) with 10-fold cross-validation to screen characteristics. The screened key variables were further incorporated into a multifactor logistic regression. The hyperparameters were tuned using a grid search strategy, and the hyperparameter tuning of all models was performed by 10-fold cross-validation within the training set. Specifically, the training set was randomly divided into 10 mutually exclusive subsets, and 9 folds were used for training and the remaining 1 fold for validation in turn, cycling 10 times to ensure that each fold was used for validation. The evaluation index of this procedure was the average AUC value under the cross-validation curve. Based on these methods, five ML detection models were developed, including logistic regression, random forest (RF), gradient boosting machine (GBM), support vector machine (SVM), and extreme gradient boosting (XGBoost). Subsequently, discrimination power, accuracy, and clinical applicability were assessed in an internal validation cohort of the model, resulting in the selection of the best model. The discrimination ability was evaluated using the receiver operating characteristic (ROC) curve and the area under the curve (AUC). Model calibration was evaluated visually using a calibration curve and quantitatively using calibration slope, calibration intercept, and Brier score. The clinical utility was evaluated by decision curve analysis (DCA). SHapley Additive exPlanation (SHAP)

analysis was implemented to quantify the contributions of variables to identifications, enhancing the interpretability of the optimal model.

Data were processed and analyzed on R 4.5.0. Data distribution patterns determined analytical techniques. Normally distributed data were presented as mean ± standard deviation and analyzed using an independent samples *t*-test. Non-normally distributed data were presented as median (interquartile range) and analyzed leveraging the Mann–Whitney *U*-test. Categorical variables were expressed as frequencies and percentages, and group comparisons were made leveraging the chi-square (χ^2) test or Fisher's exact test. Results were considered statistically significant when $p < 0.05$.

Results

Demographic and Clinical Characteristics

In total, 1786 eligible patients were involved in this research. The study cohort comprised 390 subjects with CHD and CKD and 1396 non-CKD subjects. Their characteristic data are depicted in Table 1. Compared with the non-CKD group, patients in the CKD group were older, had a higher proportion of males, higher prevalence of hypertension, diabetes

Table 1 Clinical Characteristics of the Study Group

Characteristics	All Patients (N=1786)	Non CKD (N=1396)	CKD (N=390)	P-Value
Gender[n(%)]				<0.001
Female	545 (30.5%)	483 (34.6%)	62 (15.9%)	
Male	1241 (69.5%)	913 (65.4%)	328 (84.1%)	
Age(years)	65.0 [57.0;72.0]	64.0 [56.0;71.0]	69.0 [59.0;75.0]	<0.001
Hypertension[n(%)]				<0.001
No	855 (47.9%)	758 (54.3%)	97 (24.9%)	
Yes	931 (52.1%)	638 (45.7%)	293 (75.1%)	
Diabetes[n(%)]				<0.001
No	1388 (77.7%)	1131 (81.0%)	257 (65.9%)	
Yes	398 (22.3%)	265 (19.0%)	133 (34.1%)	
AF[n(%)]				<0.001
No	1714 (96.0%)	1363 (97.6%)	351 (90.0%)	
Yes	72 (4.03%)	33 (2.36%)	39 (10.0%)	
Stroke[n(%)]				<0.001
No	1557 (87.2%)	1239 (88.8%)	318 (81.5%)	
Yes	229 (12.8%)	157 (11.2%)	72 (18.5%)	
Smoking[n(%)]				<0.001
No	869 (48.7%)	718 (51.4%)	151 (38.7%)	
Yes	917 (51.3%)	678 (48.6%)	239 (61.3%)	
Drinking[n(%)]				<0.001
No	1316 (73.7%)	1058 (75.8%)	258 (66.2%)	
Yes	470 (26.3%)	338 (24.2%)	132 (33.8%)	
HR(bpm)	72.0 [70.0;80.0]	72.0 [70.0;79.0]	76.0 [70.0;84.0]	<0.001
LM[n(%)]				0.007
No	1641 (91.9%)	1296 (92.8%)	345 (88.5%)	
Yes	145 (8.12%)	100 (7.16%)	45 (11.5%)	
MVD[n(%)]				<0.001
No	822 (46.0%)	688 (49.3%)	134 (34.4%)	
Yes	964 (54.0%)	708 (50.7%)	256 (65.6%)	
Gensini	20.0 [8.00;42.0]	16.2 [7.00;37.6]	33.5 [15.0;62.0]	<0.001
WBC ($\times 10^9$/L)	6.43 [5.25;8.17]	6.31 [5.19;8.01]	6.91 [5.64;9.11]	<0.001
Hb (g/L)	140 [129;153]	142 [131;154]	134 [120;148]	<0.001
PCT (%)	0.20 [0.17;0.24]	0.21 [0.17;0.24]	0.20 [0.16;0.23]	<0.001

(*Continued*)

Table 1 (Continued).

Characteristics	All Patients (N=1786)	Non CKD (N=1396)	CKD (N=390)	P-Value
MPV(fL)	10.6 [9.8011.7]	10.6 [9.80;11.7]	10.5 [9.80;11.6]	0.275
RDW (%)	13.2 [12.8;13.7]	13.1 [12.7;13.6]	13.3 [12.9;14.1]	<0.001
ALT (U/L)	22.0 [15.0;32.0]	22.0 [15.0;32.0]	21.5 [15.0;34.0]	0.631
AST (U/L)	25.0 [20.0;36.0]	25.0 [20.0;34.2]	26.0 [21.0;41.0]	0.026
TBIL (umol/L)	12.1 [9.60;15.9]	12.2 [9.60;15.9]	12.0 [9.10;15.9]	0.290
DBIL (umol/L)	3.00 [2.20;3.90]	3.00 [2.20;4.00]	2.90 [2.10;3.90]	0.362
TC (mmol/L)	4.24 [3.48;5.19]	4.30 [3.54;5.19]	4.02 [3.32;5.07]	0.008
TG (mmol/L)	1.34 [0.97;1.99]	1.31 [0.96;1.95]	1.44 [1.00;2.19]	0.004
LDL (mmol/L)	2.62 [2.03;3.24]	2.66 [2.07;3.26]	2.48 [1.95;3.16]	0.017
HDL (mmol/L)	1.08 [0.91;1.28]	1.09 [0.92;1.30]	1.02 [0.86;1.19]	<0.001
LPa (g/L)	0.13 [0.05;0.32]	0.12 [0.05;0.31]	0.15 [0.07;0.35]	0.007
GLU (mmol/L)	5.52 [4.69;7.16]	5.39 [4.66;6.87]	6.00 [4.92;8.34]	<0.001
K(mmol/L)	4.02 [3.77;4.25]	4.00 [3.76;4.20]	4.16 [3.85;4.41]	<0.001
UAR	8.87 [7.23;11.0]	8.47 [6.91;10.1]	11.3 [9.05;13.4]	<0.001
AISI	205 [121;361]	190 [113;329]	282 [162;523]	<0.001
SIRI	1.08 [0.68;1.84]	0.98 [0.63;1.63]	1.52 [0.99;2.69]	<0.001
SII	532 [350;863]	499 [333;808]	666 [430;1113]	<0.001
NPAR	0.10 [0.08;0.15]	0.10 [0.08;0.14]	0.12 [0.09;0.18]	<0.001
NLR	2.76 [1.94;4.42]	2.57 [1.84;3.96]	3.61 [2.54;5.75]	<0.001
PLR	128 [97.1;174]	127 [94.9;169]	134 [103;190]	0.001
LMR	3.87 [2.85;5.12]	4.13 [3.10;5.39]	3.10 [2.24;4.16]	<0.001
ApoB/ApoA1	0.65[0.48;0.84]	0.63[0.47;0.82]	0.70[0.54;0.93]	<0.001

Notes: Data are given as median (IQR) or n (%). P < 0.001 were considered statistically significant.
Abbeviations: AF, atrial fibrillation; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; LM, left main coronary artery disease; MVD, multivessel coronary artery disease; WBC, white blood cell; Hb, hemoglobin; PCT, platelet crit; MPV, mean platelet volume; RDW, red blood cell distribution width; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TBIL, total bilirubin; DBIL, direct bilirubin; TC, total cholesterol; TG, triglycerides; LDL, low-density lipoprotein; HDL, high-density lipoprotein; LPa, lipoprotein a; GLU, blood glucose; K, blood potassium; UAR, uric acid-to-albumin ratio; AISI; systemic inflammation aggregation index; SIRI, systemic inflammatory response index; SII, systemic immune-inflammatory index; NPAR, neutrophil-to-albumin ratio; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; LMR, lymphocyte-to-monocyte ratio; ApoB/ApoA1, apolipoprotein B/apolipoprotein A1 ratio.

mellitus, atrial fibrillation, and stroke, and more severe coronary lesions (left main and multi-vessel disease, higher Gensini score) (all P<0.001 or P=0.007; Table 1). In terms of laboratory parameters, individuals in the CKD group had lower hemoglobin, plateletcrit (PCT), total cholesterol (TC), low-density lipoprotein (LDL) and high-density lipoprotein (HDL), higher white blood cell count, red blood cell distribution width (RDW), aspartate transaminase (AST), triglyceride (TG), lipoprotein (a) (Lp (a)), serum potassium, glucose and composite inflammatory/nutritional indicators (UAR, AISI, SIRI, SII, NPAR, NLR, PLR, ApoB/ApoA1 ratio), and lower lymphocyte-to-monocyte ratio (LMR) (all P<0.001; Table 1).

Feature Selection

To avoid multicollinearity, component variables used to calculate composite indicators were excluded before the LASSO regression. The excluded component variables are shown in Supplementary Table S1. The characteristics of the variable coefficients are shown in Figure 1. A 10-fold cross-validation approach was adopted to conduct iterative analysis. The 17 variables that were strongly associated with CHD complicating CKD were gender, age, hypertension, diabetes mellitus, history of AF, smoking status, Gensini, hemoglobin, PCT, RDW, TG, Lp(a), serum potassium, UAR, SIRI, LMR, and ApoB/ApoA1 ratio. Multifactor logistic regression was used to further screen characteristic variables, and the final variables included in the model were gender, age, hypertension, diabetes mellitus, history of AF, Gensini, hemoglobin, PCT, RDW, TG, Lp(a), serum potassium, UAR, SIRI, LMR, and ApoB/ApoA1 ratio. (Table 2).

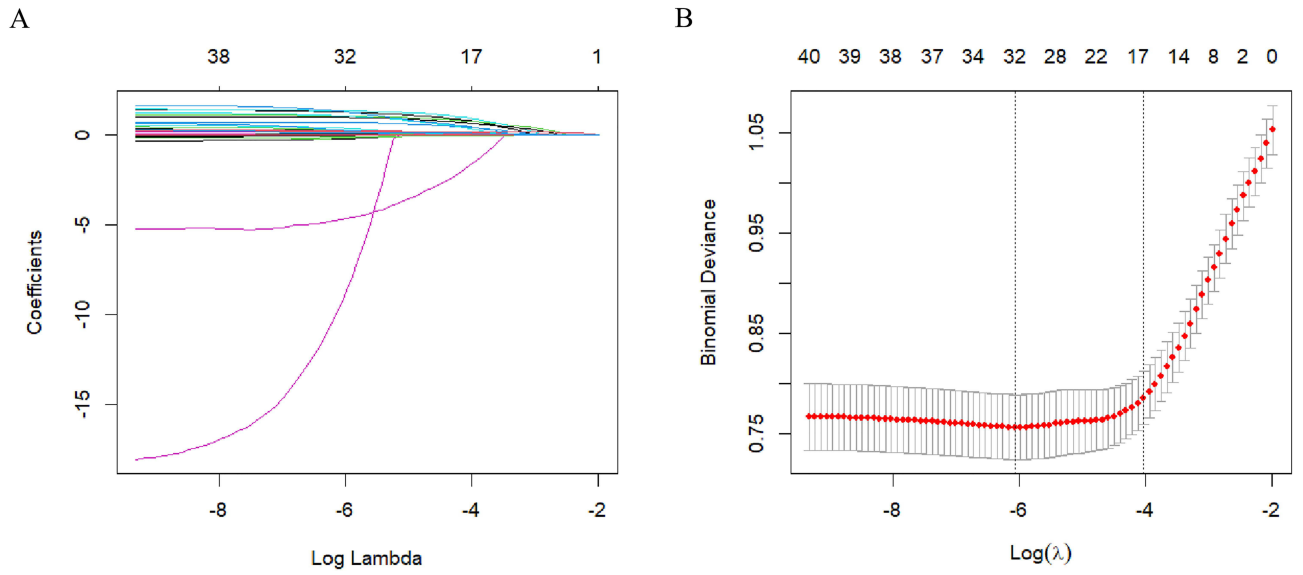

Figure 1 (**A**) Lasso regression coefficient path plot, the vertical axis represents the regression coefficients of different variables, and the horizontal axis Log (lambda) is the logarithm of the penalty coefficient. (**B**) The correlation between lamda with binomial deviance. There are two dashed lines in the graph. The left dashed line indicates the minimum mean squared error while the right one indicates one standard error away from the minimum mean squared error.

Comparisons of Model Performance

Five ML models were constructed to identify the risk of CKD in CHD patients. Table 3 and Figures 2–4 demonstrate the performance metrics of the five ML models. Specifically, Figure 2A shows the ROC curves of different ML models in the training set, and Figure 2B presents those in the validation set. In the validation set, the XGBoost model achieved the highest area under ROC (AUC) value of 0.909 (95% CI 0.881–0.937), followed by LR (AUC=0.905, 95% CI 0.877–0.933), GBM (AUC=0.897, 95% CI 0.867–0.928), SVM (AUC=0.878, 95% CI 0.843–0.914), and RF (AUC=0.873, 95% CI 0.837–0.909). Figure 3A shows the calibration plot for the training set, where most models'

Table 2 Multivariate Logistic Regression Analysis Was Performed

Characteristic	OR	95% CI	p-Value
Gender			
Female	—	—	
Male	3.89	2.24, 6.84	<0.001
Age	1.02	1.01, 1.04	0.013
Hypertension			
No	—	—	
Yes	3.01	2.10, 4.36	<0.001
Diabetes			
No	—	—	
Yes	2.11	1.43, 3.11	<0.001
AF			
No	—	—	
Yes	4.83	2.31, 10.2	<0.001
Smoking			
No	—	—	
Yes	1.32	0.87, 2.01	0.200

(*Continued*)

Table 2 (Continued).

Characteristic	OR	95% CI	p-Value
Gensini	1.01	1.01, 1.02	<0.001
Hb	0.97	0.96, 0.99	<0.001
PCT	0.01	0.00, 0.16	0.003
RDW	1.23	1.03, 1.47	0.022
TG	1.20	1.08, 1.35	0.001
LPa	2.91	1.44, 5.84	0.003
K	2.75	1.87, 4.10	<0.001
UAR	1.16	1.09, 1.23	<0.001
SIRI	1.12	1.01, 1.24	0.029
LMR	0.87	0.76, 0.99	0.038
ApoB/ApoA1	2.78	1.53, 5.05	<0.001

Abbreviations: CI, Confidence Interval; OR, Odds Ratio.

Table 3 Comparison of the Performance of the ML Models

ML Models	Data Set	AUC (95% CI)	Specificity	Sensitivity	Accuracy	Precision	Recall	Youden Index	Calibration Slope	Calibration Intercept	Brier Score
LR	Train	0.862(0.838–0.886)	0.825	0.731	0.804	0.540	0.731	0.555	1.000	0.000	0.113
	Validation	0.905(0.877–0.933)	0.815	0.878	0.828	0.564	0.878	0.693	1.188	0.029	0.099
SVM	Train	0.930(0.911–0.949)	0.869	0.895	0.874	0.658	0.895	0.763	2.347	1.565	0.088
	Validation	0.878(0.843–0.914)	0.796	0.843	0.806	0.530	0.843	0.639	1.441	0.391	0.111
RF	Train	0.995(0.993–0.997)	0.955	0.978	0.960	0.859	0.978	0.933	5.164	5.355	0.072
	Validation	0.873(0.837–0.909)	0.817	0.774	0.808	0.536	0.774	0.591	1.664	1.231	0.119
GBM	Train	0.902(0.882–0.921)	0.861	0.785	0.844	0.614	0.785	0.646	2.222	1.326	0.109
	Validation	0.897(0.867–0.928)	0.886	0.774	0.862	0.650	0.774	0.660	2.099	1.057	0.111
XGBoost	Train	0.961(0.950–0.972)	0.911	0.873	0.902	0.734	0.873	0.783	2.115	0.897	0.070
	Validation	0.909(0.881–0.937)	0.891	0.791	0.869	0.664	0.791	0.682	1.358	0.326	0.097

Abbreviations: LR, Logistic regression; SVM, Support vector machine; RF, Random forest; GBM, Gradient boosting machine; XGBoost, Extreme gradient boosting.

calibration curves closely approach the ideal diagonal line. The calibration plots of the five models in the validation set (Figure 3B) showed satisfactory calibration performance, demonstrating their reliability in prediction. Table 3 lists the calibration slopes, calibration intercepts, and Brier scores for all five ML models in the training and validation sets. The XGBoost model showed an excellent fit. The calibration curves of the five ML models are shown in Supplementary Figures (LR: Figure S1A and B; SVM: Figure S2A and B; RF: Figure S3A and B; GBM: Figure S4A and B; XGBoost: Figure S5A and B). In addition, DCA curves indicated favorable clinical utility across all five models (Figure 4A and B), thus confirming their potential for future clinical decision-making. To further evaluate the classification performance at specific decision thresholds, we calculated the confusion matrix (Supplementary Table S2). The threshold was determined by maximizing the Youden index in the model to balance sensitivity and specificity. The XGBoost model demonstrated outstanding performance in most of these metrics.

Interpretability Analysis

To ascertain the contributions of the variables, the SHAP algorithm was applied to quantify the impact of each characteristic on predicting the risk of CHD with CKD within the XGBoost model. Figure 5A shows a swarm plot, where the horizontal axis denotes the SHAP values while the vertical axis denotes the characteristics ranked by their cumulative SHAP values, allowing us to visually evaluate the impact of each characteristic on the prediction results. Figure 5B depicts the importance of features in descending order. The top ten features were UAR, hypertension, Gensini score, gender, SIRI, serum potassium, hemoglobin, ApoB/ApoA1 ratio, PCT, and diabetes mellitus.

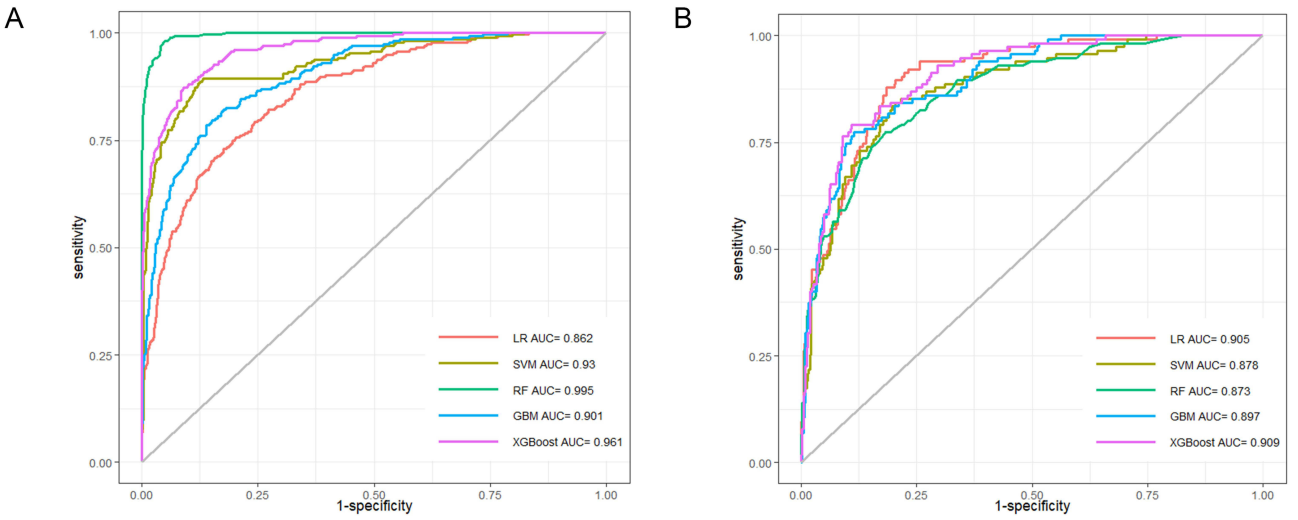

Figure 2 (**A**) Roc curves predicted by different ML models in the training set. (**B**) Roc curves predicted by different ML models in the validation set.
Abbreviations: AUC, area under curve; LR, Logistic regression; SVM, Support vector machine; RF, Random forest; GBM, Gradient boosting machine; XGBoost, Extreme gradient boosting.

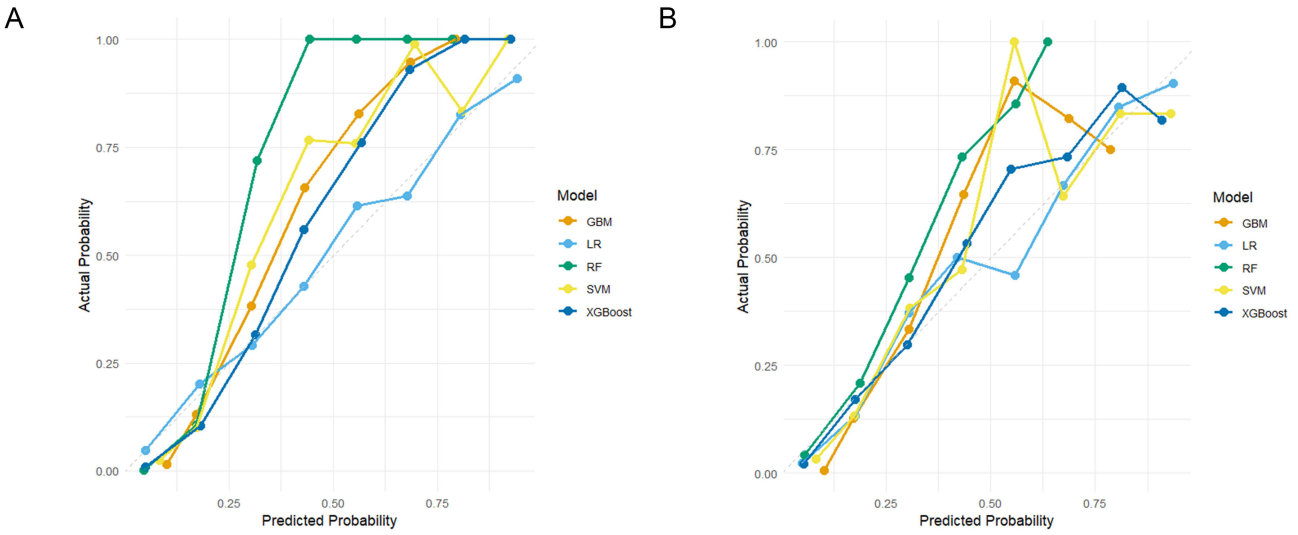

Figure 3 (**A**) Calibration curves predicted by different ML models in the training set. (**B**) Calibration curves predicted by different ML models in the validation set.
Abbreviations: LR, Logistic regression; SVM, Support vector machine; RF, Random forest; GBM, Gradient boosting machine; XGBoost, Extreme gradient boosting.

Discussion

This study constructed 5 ML models for the risk of CKD in 1786 CHD patients. In this study, XGBoost demonstrated the highest overall performance (AUC=0.909) while maintaining good specificity (0.891) and sensitivity (0.791). Its AUC was significantly higher than that of RF and SVM, comparable to that of GBM, and slightly higher than that of LR. RF showed obvious overfitting (training set: AUC=0.945 vs validation set: AUC=0.873), with a decay of 7.6%, which was higher than that of XGBoost (decay = 5.4%). Considering its excellent performance in the training set (AUC = 0.961) and smaller performance decay in the validation set, XGBoost had the best resistance to overfitting and strong generalizability. The calibration curves of the XGBoost model were close to the ideal diagonal in both the training and validation sets, indicating that the predicted probability matched the actual probability well. The DCA revealed that over a wide range of threshold probabilities (approximately 0.1 to 0.7), the XGBoost model achieved a good net benefit for clinical

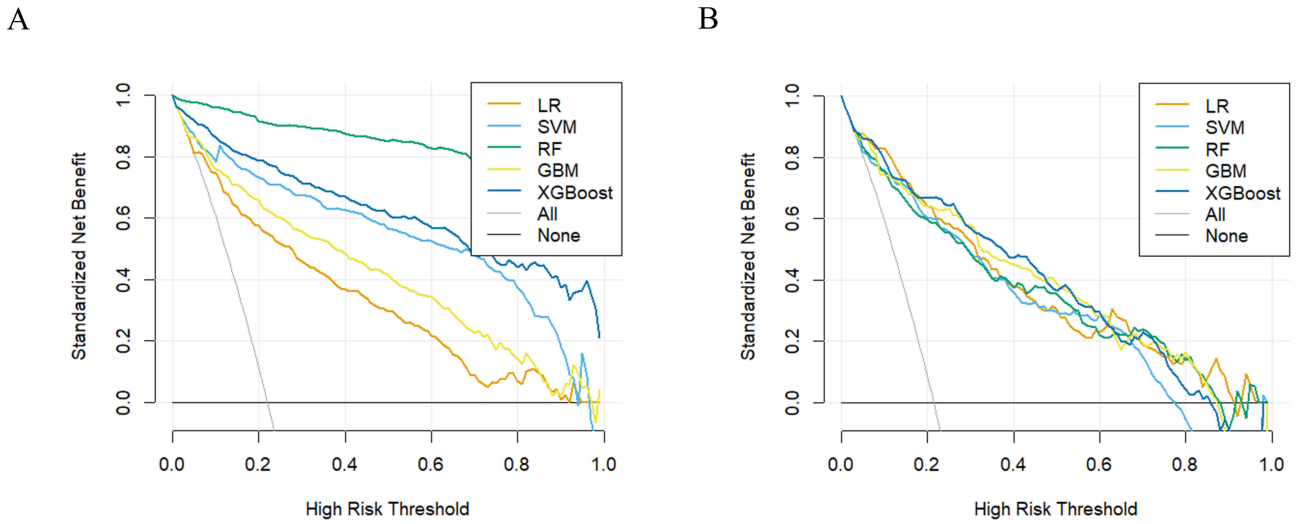

Figure 4 (A) Decision curves predicted by different ML models in the training set. (**B**) Decision curves predicted by different ML models in the validation set.
Abbreviations: LR, Logistic regression; SVM, Support vector machine; RF, Random forest; GBM, Gradient boosting machine; XGBoost, Extreme gradient boosting.

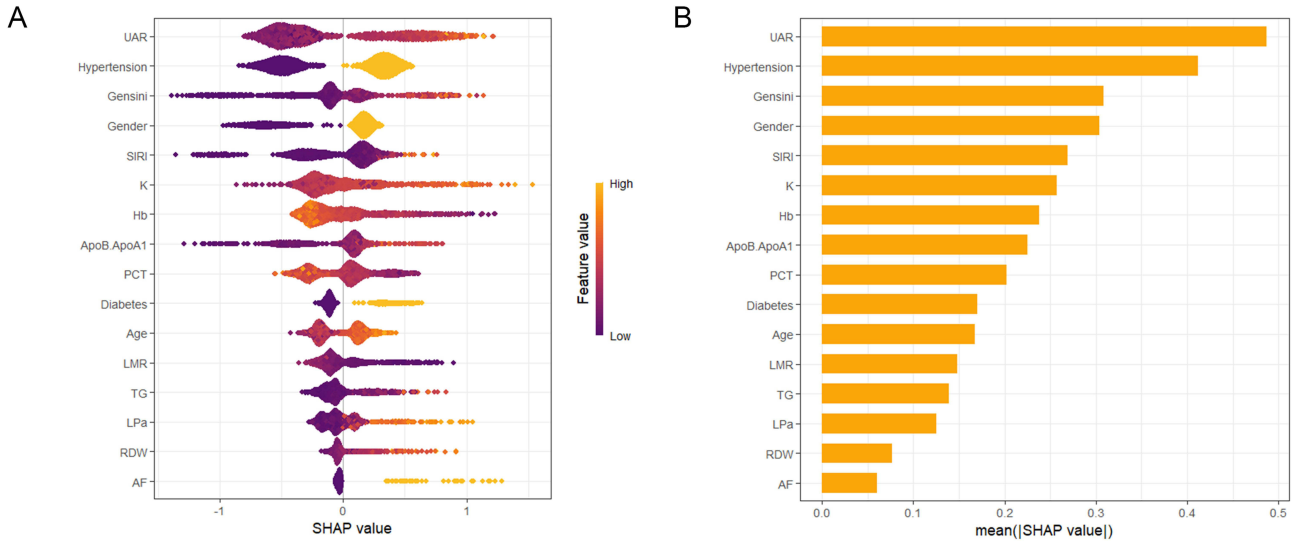

Figure 5 Interpretability analysis of the optimal XGBoost model using SHapley Additive exPlanations (SHAP). (**A**) Beeswarm plot summarizing the feature impacts on the model output. Each point represents a patient from the validation set. The position on the x-axis is the SHAP value, which indicates the impact of that feature on the prediction for that individual (positive SHAP values increase the predicted risk of CKD). The color represents the actual value of the feature for that patient, from low (blue) to high (red). Features are ordered by their overall importance; (**B**) Beeswarm plot summarizing the feature impacts on the model output. Each point represents a patient from the validation set. The position on the x-axis is the SHAP value, which indicates the impact of that feature on the prediction for that individual (positive SHAP values increase the predicted risk of CKD). The color represents the actual value of the feature for that patient, from low (blue) to high (red). Features are ordered by their overall importance.
Abbreviations: UAR, uric acid-to-albumin ratio; SIRI,systemic inflammatory response index; K,serum potassium; Hb,hemoglobin; ApoB/ApoA1,apolipoprotein B/apolipoprotein A1 ratio; PCT, platelet crit; LMR,lymphocyte-to-monocyte ratio; TG,triglycerides; LPa,lipoprotein a; RDW,red blood cell distribution width; AF,atrial fibrillation.

decision-making. This implies that using the test results of this model to guide clinical decision-making (eg, enhanced surveillance, early intervention) can provide higher clinical benefit for patients. Furthermore, SHAP analysis was employed to detect high-contributing characteristics, revealing the substantial link between the risk of CKD and variables such as UAR, hypertension, Gensini score, gender, SIRI, serum potassium, hemoglobin, ApoB/ApoA1 ratio, PCT, and diabetes mellitus.

Previously, Zhang et al[20] developed a nomogram prediction model for the occurrence of CKD in patients with CHD based on the results of multivariate logistic regression analysis (AUC = 0.867). The main variables incorporated into this

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model were age, uric acid, and a history of diabetes mellitus. In contrast, our XGBoost model (AUC = 0.909) exhibited superior performance, possibly since our model incorporated CHD-specific indicators (eg, Gensini score) and new composite indicators (eg, UAR, SIRI), making it more suitable for patients with CHD. Notably, the XGBoost model demonstrated excellent performance, especially in handling high-dimensional and complex data. Its generalizability and predictive performance far surpassed those of traditional models.

UA participates in various pathological processes, hyperuricemia drives urate crystallization and its deposition in the renal tubulointersitium, leading to chronic inflammation in the kidney (eg, NLRP3 inflammasome activation, release of IL-1β, IL-6 and other cytokines), triggering oxidative stress (reactive oxygen species(ROS)), activating the renin-angiotensin system, all of which contribute to further endothelial dysfunction and vascular smooth muscle cell proliferation, promoting glomerulosclerosis and interstitial fibrosis, and thus accelerating renal damage.[21,22] A prospective study involving 755 patients with CKD demonstrated that the rs734553 polymorphism of the GLUT9 gene (associated with abnormal urate transport) was associated with a 2.35-fold increase in the risk of CKD. Furthermore, this association was independent of traditional indicators such as proteinuria and estimated glomerular filtration rate (eGFR), directly confirming the causal relationship between abnormal urate transport and renal injury.[23] Hypoalbuminemia, on the other hand, is often associated with inflammation and malnutrition, and it is demonstrated by multiple studies that hypoalbuminemia correlates with renal function deterioration.[24,25] UAR is a novel composite index that integrates nutritional status, inflammation, and metabolic syndromes. It comprehensively represents uric acid excretion load (frequently elevated in renal impairment) and nutritional and inflammatory status (indicated by ALB levels), making it a more sensitive and specific predictor than single indicators like uric acid or albumin alone. Previous studies have investigated its accuracy in predicting contrast nephropathy,[26] the extent of coronary artery disease in individuals with non-ST-segment elevation myocardial infarction,[27] and the prognosis of some diseases (eg, acute kidney injury[28,29] and ST-segment elevation myocardial infarction[30]). In addition, a study by Qin et al has disclosed that elevated UAR is an independent prognostic biomarker for IgA nephropathy (IgAN).[31] Although Qin et al have demonstrated the predictive value of UAR for IgA nephropathy, which supports our finding regarding the role of UAR in predicting CKD, UAR as the primary predictor has not been reported in the prediction of CKD in patients with CHD. The current research showed that elevated levels of UAR (OR=1.16, 95% CI 1.09–1.23, P<0.001) were an independent risk factor for CKD in CHD individuals and ranked first in importance among the variables. This suggests that UAR can be a good predictor of CHD complicating CKD. By combining these two markers, ie, uric acid and ALB, a convincing and feasible predictor of kidney disease risk was created.

Elevation of the Gensini score (OR=1.01, 95% CI 1.01–1.02, P<0.001), a quantitative index of the severity of coronary artery lesion, significantly increases the risk of CKD. Its core value lies in (a) early warning: Gensini score combined with markers such as cystatin C and cardiac troponin I (cTnI) to identify high-risk patients before a significant decline in epidermal growth factor receptor (eGF);[32,33] (b) risk stratification: scores > 40 require intensive anti-inflammatory and calcium-phosphorus management, and >60 initiates combined cardiorenal and renal followup;[34,35] (c) treatment orientation: guiding individualized interventions (eg, lipid-lowering target selection, intensity of blood phosphorus control) to improve long-term prognosis of CKD patients.[36] This strongly suggests that systemic AS affects both the coronary arteries and renal arteries/intrarenal microvasculature, with endothelial dysfunction and ischemia serving as the shared underlying pathophysiology. An extensive atherosclerotic load may directly reflect the health of the renal vasculature.

SIRI, as a novel inflammatory marker, combines three complete blood count markers to more systematically and comprehensively reflect the inflammatory state of the body, and is highly correlated with diseases affecting multiple organ systems. In CKD patients, a persistent microinflammatory state accelerates the decline of renal function through oxidative stress, endothelial injury, and fibrosis. A study has shown that elevated SIRI constitutes an independent risk factor for all-cause mortality and cardiovascular mortality in individuals with CKD, and may be of higher significance in the early stages of CKD (stage I to III).[37] SIRI may be considered as a valid predictor for assessing the morbidity of CKD, as well as the mortality risk of CKD patients in the general US population.[38] The present study demonstrated that high SIRI (OR=1.12, 95% CI 1.01–1.24, P=0.029) played an important role in the diagnosis of CKD in CHD patients.

Hyperkalemia (OR=2.75, 95% CI 1.87–4.10, P<0.001) is a marker of advanced renal insufficiency, which is primarily due to the decreased ability of the kidneys to excrete potassium. Even a mild elevation of serum potassium levels suggests a decrease in glomerular filtration rate (GFR).[39] Low hemoglobin (OR=0.97, 95% CI 0.96–0.99, P<0.001) is an early marker and outcome of CKD. The kidney is the main organ for erythropoietin (EPO) synthesis. When renal function is impaired (especially destruction of renal paratubular mesangial cells), EPO synthesis is significantly reduced, causing anemia.[40] At the same time, anemia exacerbates renal hypoxia and oxidative stress. Anemia-induced hypoxia further promotes CKD progression by triggering inflammation, fibrosis, and dysregulated angiogenesis.[41]

HDLs are anti-inflammatory, antioxidant, antithrombotic, and antiapoptotic. They inhibit the progression of AS by removing excess cholesterol through the reverse cholesterol transport (RCT) pathway, making it the only plasma lipoprotein with anti-AS effects.[42,43] ApoA1 is a major structural protein, accounting for about 70% of the protein content of HDL.[44] ApoB is a notable structural protein in LDL, TG-rich lipoproteins, and lipoprotein(a), critical for their formation, metabolism, and atherogenic properties.[45] A high ApoB/ApoA1 ratio is a strong predictor of cardiovascular risk.[46] This study confirms that it is also a risk factor for CKD (ApoB/ApoA1, OR=2.78, 95% CI 1.53–5.05, P<0.001), and that high ratios reflect the imbalance between atherogenic lipoprotein particles (represented by ApoB) and protective lipoprotein particles (represented by ApoA1 for HDL). The imbalance also damages the renal microvasculature.

CHEN et al explored the link between four systemic inflammatory markers and all-cause mortality of individuals with CKD in a prospective study. NLR and LMR showed the strongest ability in predicting all-cause mortality in individuals with CKD. High NLR values indicated an increase in chronic inflammation, which was positively linked to all-cause mortality in CKD. In comparison, higher LMR levels conferred protection against adverse clinical outcomes in the CKD population.[47] This study yielded similar results: a lower LMR (OR=0.87, 95% CI 0.76–0.99, P=0.038) indicated an increased risk of CKD. Mechanistically, low LMR reflects relative lymphocytopenia (indicating immunosuppression or consumption) and/or monocytosis (suggesting heightened inflammatory states). They collectively suggest that immunological dysregulation contributes to the pathogenesis of CKD.

Multifactor logistic regression clearly showed that male (OR=3.89, 95% CI 2.24–6.84, P<0.001), advanced age (OR=1.02, 95% CI 1.01–1.04, P=0.013), hypertension (OR=3.01, 95% CI 2.10–4.36, P<0.001), diabetes mellitus (OR=2.11, 95% CI 1.43–3.11, P<0.001), and AF (OR=4.83, 95% CI 2.31–10.2, P<0.001) were strong independent predictors of CKD. This is highly consistent with established epidemiologic evidence. Male sex, advanced age, hypertension, and diabetes are recognized risk factors for CKD.[48,49] They contribute to the decline in renal function by promoting glomerulosclerosis, tubulointerstitial fibrosis, and vascular injury.[50,51] Reduced PCT (OR=0.01, 95% CI 0.00–0.16, P=0.003) was related to an elevated risk of CKD. Mechanistically, inflammation states and immune disorders cause platelet destruction, and thus, PCT decreases. In addition, high TG (OR=1.20, 95% CI 1.08–1.35, P=0.001) and high Lp(a) (OR=2.91, 95% CI 1.44–5.84, P=0.003) were significant risk factors for CKD. Hypertriglyceridemia is associated with insulin resistance and renal lipid deposition.[52] Lp(a) has pro-atherogenic and pro-thrombotic properties and may directly damage the glomerular endothelium.[53]

This study constructed diagnostic models specifically for CKD in patients with CHD was established. It fills the gap of targeted prediction tools in the field, and the results will be of direct guidance to cardiovascular physicians in identifying patients with CHD who may have comorbid or impending CKD. The study incorporates and validates the value of a variety of emerging and readily available hematologic markers (eg, UAR, SIRI, LMR, ApoB/ApoA1) in predicting CKD risk. These composite inflammatory, immune, or nutritional metrics provide early risk signals beyond traditional markers of renal function (eg, creatinine).

This study has several limitations. Its single-center, retrospective design may limit the generalizability of the findings and introduce selection bias. Although the Events Per Variable (EPV ≈ 23) was adequate, and measures were taken to mitigate overfitting, the sample size and the lack of external validation may still affect the stability and applicability of the model. Furthermore, given the cross-sectional design of this study, it is infeasible to infer causal relationships. Finally, while stringent procedures were applied during data preprocessing, the possibility of undetected data leakage in a complex pipeline cannot be fully excluded. Future prospective, multi-center studies with external validation are needed to confirm and extend our findings.

Conclusions

In this study, a series of ML models were successfully constructed and validated for identifying preexisting CKD in CHD patients. Among multiple ML models, XGBoost demonstrated the optimal overall detection performance (validation set AUC=0.909) and a good generalization ability. Decision curve analysis further confirmed the potential net benefit of the XGBoost model in guiding clinical decisions.

Data Sharing Statement

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

Author Contributions

Ting He and Jinbo Zhao contributed equally to this work and should be considered as co-first authors. 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 they have no competing interests.

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