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ORIGINAL RESEARCH

Development and Validation of a Nomogram for Predicting Chronic Kidney Disease in Older Patients with Type 2 Diabetes Mellitus and Cardiovascular Disease

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Background: Type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVD) are significant global health challenges, particularly in older adults where their coexistence exacerbates disease burden and leads to adverse outcomes. Chronic kidney disease (CKD), a severe complication of T2DM, is increasing in prevalence and significantly contributes to morbidity and mortality. Early detection of CKD in older patients with T2DM and CVD is crucial for timely intervention.

Methods: This study developed a predictive model using electronic health record data from 28,443 older inpatients with T2DM and CVD at the Affiliated Banan Hospital of Chongqing Medical University (2018–2025). The study population was divided into training (n=21,234) and validation (n=7,209) sets. Predictive variables were selected through univariate logistic regression, LASSO regression, and multivariate logistic regression analyses. The model was visualized using a nomogram and validated using ROC curves, calibration curves, and decision curve analysis (DCA).

Results: The model identified seven independent predictors of CKD: systolic blood pressure, uric acid (UA), hemoglobin, glycated hemoglobin, white blood cell count, triglyceride glucose, and UA/Cr ratio. The nomogram demonstrated good discriminative ability with AUCs of 0.845 (95% CI: 0.836–0.853) in the training set and 0.853 (95% CI: 0.838–0.867) in the validation set. Calibration curves showed good agreement between predicted and observed risks. DCA indicated that the model provided a net benefit across a range of threshold probabilities, highlighting its clinical utility.

Conclusion: The developed nomogram provides a practical tool for clinicians to predict CKD risk in older patients with T2DM and CVD using readily available clinical data. This model can facilitate early identification and intervention for high-risk patients, potentially improving outcomes.

Keywords: type 2 diabetes mellitus, cardiovascular disease, chronic kidney disease, predictive model, nomogram

Introduction

Type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVD) are chronic conditions that severely compromise patients' health.[1,2] Owing to their high incidence and mortality rates, both T2DM and CVD have emerged as significant global public health challenges.[3,4] Among the older population, the coexistence of T2DM and CVD is particularly prevalent, with the two diseases exacerbating each other's disease burden and leading to adverse outcomes. Studies have shown that from 1990 to 2021, the age-standardized prevalence and mortality rates of T2DM in individuals aged 65 years and older increased by 1.9% and 0.32% per year globally, respectively.[5] In 2021 alone, CVD resulted in approximately 19.4 million deaths and 428.3 million disability-adjusted life-years (DALYs) lost.[6]

Chronic kidney disease (CKD) is a severe complication of diabetes, and its global burden is progressively increasing.[7] Studies have indicated that more than half of patients with T2DM will develop CKD during their lifetime.[8,9] In the United States, nearly 800,000 patients underwent dialysis or transplantation due to renal failure from 2000 to 2019, with

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T2DM being the primary cause.[10] Between 1990 and 2017, the all-age mortality rate of CKD increased by 41.5% globally.[11] Moreover, the ranking of CKD in the list of causes of death continues to rise: it was ranked 13th in 2016 and 12th in 2017, and projections suggest that by 2040, it will become the fifth leading cause of life loss globally.[12] T2DM, CVD, and CKD are closely interconnected through bidirectional and synergistic pathways. Chronic hyperglycemia in T2DM drives both CKD and CVD via oxidative stress, advanced glycation end-products, and activation of the renin-angiotensin-aldosterone system (RAAS).[13,14] Conversely, CKD accelerates CVD through uremic toxins, systemic inflammation, and volume overload.[15,16] In older patients with coexisting T2DM and CVD, these pathways converge, creating a vicious cycle that accelerates renal function decline and increases cardiovascular mortality.[17] This complex interplay underscores the need for population-specific prediction models.

The present study aims to develop a predictive model based on electronic health record data to predict the risk of CKD in older patients with T2DM and CVD. This predictive model is intended to provide clinicians with a practical and user-friendly tool to identify older patients with T2DM and CVD who are at high risk of CKD, using common demographic, vital sign, and laboratory test indicators without the need for specialized instrument assessment.

Methods

Study Population and Data Source

Participants in this study were older inpatients with T2DM and CVD admitted to the Affiliated Banan Hospital of Chongqing Medical University from January 2018 to June 2025. The study protocol was approved by the Ethics Committee of the Affiliated Banan Hospital of Chongqing Medical University (approval number: BNLLKY2025086). Although informed consent was waived due to the retrospective observational design, the study strictly adhered to ethical guidelines and was reported in accordance with the Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis (TRIPOD) statement.[18] The inclusion criteria were: (1) inpatients diagnosed with T2DM and CVD; (2) hospital stay exceeding 48 hours. The exclusion criteria were: (1) age less than 65 years; (2) in-hospital death; (3) presence of malignant tumors.

Study Design

In accordance with the definition of CKD, the diagnostic criteria are an estimated Glomerular Filtration Rate (eGFR) of less than 60 mL/min/1.73 m^2 or a urinary albumin-to-creatinine ratio (ACR) of 30 mg/g or higher. In this study, eGFR was calculated using the 2009 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation based on serum creatinine values.[19] ACR was calculated using spot urine specimens collected at admission, with albumin measured by immunoturbidimetry and creatinine measured by the enzymatic method. The study population was divided into training and validation sets based on the admission date of January 1, 2024. The study subjects were further categorized into CKD and non-CKD groups according to the presence or absence of CKD. A variety of statistical methods were employed to identify risk factors for CKD and to construct a predictive model. The model was visualized using a nomogram, and its predictive ability and clinical utility were validated and evaluated. The study workflow is illustrated in Figure 1.

Data Collection and Data Quality Control

All data in this study were derived from the hospital's clinical data platform, which has anonymized all personal identifying information. Based on clinical practice and previous literature studies, the indicators we collected include demographic characteristics (age, gender, smoking history, drinking history), comorbidities (hyperlipidemia, arthritis), vital signs (systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse), laboratory indicators (total cholesterol (TC), triglycerides (TG), uric acid (UA), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), hemoglobin, glycated hemoglobin (HbA1c), fasting glucose (FG), white blood cell (WBC) count, platelet count), and a series of metabolic composite indicators (triglyceride glucose (TyG), total cholesterol to high-density lipoprotein cholesterol ratio (TC/HDL-C), uric acid to high-density lipoprotein cholesterol ratio (UHR), atherogenic index of plasma (AIP), non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (NHHR), glycated hemoglobin to high-density lipoprotein cholesterol ratio (GHR), uric acid to creatinine ratio (UA/Cr), stress

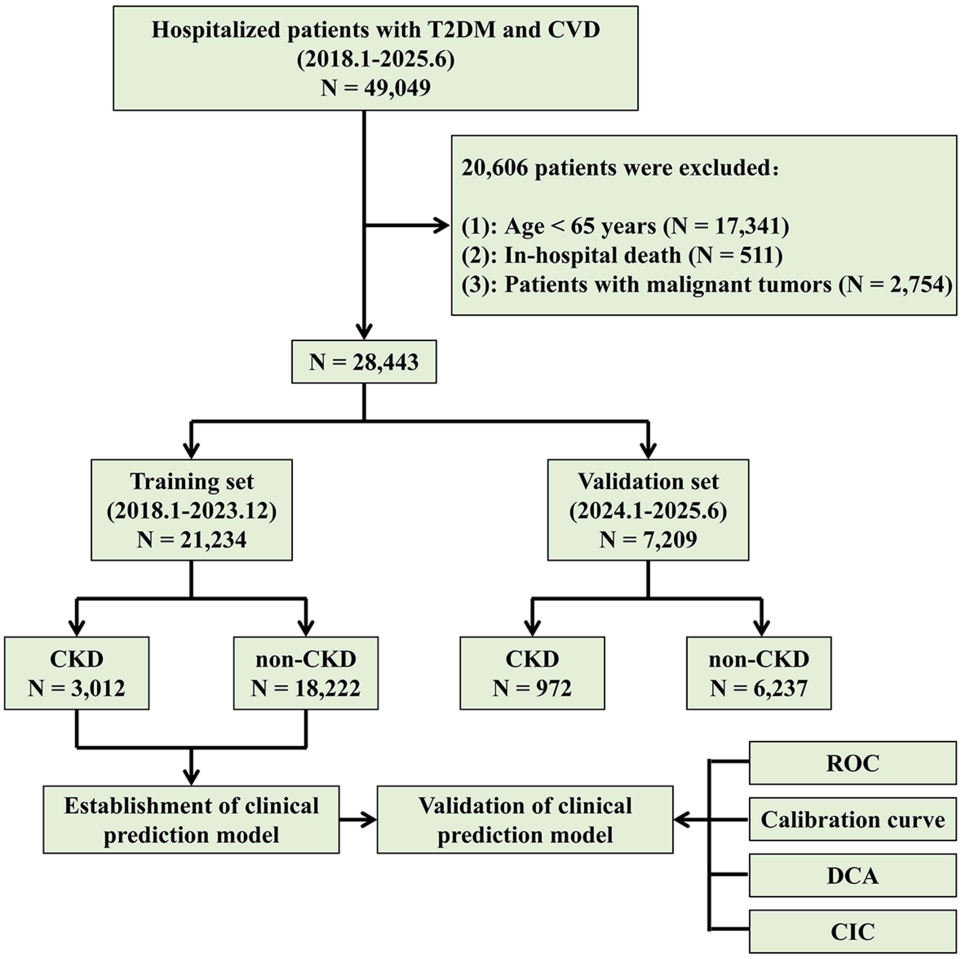

Figure 1 Flowchart of study participants.

hyperglycemia ratio (SHR)).[20–22] The calculation formulas for these composite indicators are detailed in Supplementary Table S1.

Considering that a laboratory indicator may have multiple test results for the same patient during hospitalization, in this study, we only selected the first test result after admission for statistical analysis. Given the long time span of this study, the same indicator may have different units; therefore, we standardized all laboratory indicators to the International System of Units (SI units). For example, UA was uniformly converted to μmol/L (1 mg/dL = 59.48 μmol/L) and FG to mmol/L (1 mg/dL = 0.0556 mmol/L). HbA1c was consistently expressed as a percentage (%). Standard conversion formulas were applied before any statistical analysis. In addition, we excluded indicators with a missing rate exceeding 30%, while indicators with a missing rate below 30% were handled using multiple imputation (MI) by chained equations. Continuous variables were imputed using linear regression models, and categorical variables were imputed using logistic regression models. The number of imputations was set to 5, and the final pooled estimates were calculated using Rubin's rules.

Feature Selection

The selection of predictive variables was conducted through three consecutive steps. Initially, a total of 27 clinical variables (including demographic characteristics, vital signs, laboratory indicators, and metabolic composite indicators) were considered as candidate predictors. In the training set, baseline variables were screened via univariate logistic regression analysis, with variables having $P < 0.05$ being retained as potentially CKD-related variables. This step identified 24 variables with significant associations. Subsequently, these retained variables were all incorporated into the

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least absolute shrinkage and selection operator (LASSO) regression model for automatic feature compression to further select predictive variables. LASSO regression was chosen because it effectively handles multicollinearity among correlated predictors, performs continuous variable selection, and reduces the risk of overfitting by shrinking regression coefficients, which is particularly advantageous when the number of candidate predictors is large relative to the number of events. Meanwhile, the multicollinearity of predictive variables was assessed using the variance inflation factor (VIF). Finally, variables screened by LASSO regression (n = 7) were input into a multivariate logistic regression model, and potential confounding factors were adjusted stepwise to determine the ultimate predictive variables.

Model Development and Evaluation

A nomogram was constructed to visualize the predictive model. Each independent predictor identified from the multivariate logistic regression analysis was assigned a point value proportional to its regression coefficient. To use the nomogram, the clinician locates the value of each predictor on its corresponding axis, draws a vertical line upward to the "Points" axis to obtain the points for that predictor, and sums the points across all predictors to obtain a total score. The total score is then mapped to the "Risk of CKD" axis to estimate the individual patient's probability of having CKD. The nomogram is designed for bedside risk stratification, with higher total scores indicating an increased risk of CKD. The model's discriminative ability was assessed using the Receiver Operating Characteristic (ROC) curve.[23] Calibration curves and the Hosmer-Lemeshaw test were employed to evaluate the model's calibration.[24] The clinical utility of the model was assessed using Decision Curve Analysis (DCA) and Clinical Impact Curves (CIC).[25] Additionally, the model's predictive performance was comprehensively evaluated using metrics from the confusion matrix, including the Area Under the Curve (AUC), sensitivity, specificity, Positive Likelihood Ratio (PLR), Negative Likelihood Ratio (NLR), Diagnostic Odds Ratio (DOR), and their 95% confidence intervals (CIs).

Statistical Analysis

Categorical variables are presented as counts (percentages) and compared using the chi-square (χ^2) test. Continuous variables are reported as mean ± standard deviation (Mean ± SD) if normally distributed and compared using the independent samples *t*-test; otherwise, they are summarized as median [interquartile range] (Median [IQR]) and compared using the Wilcoxon rank-sum test. All statistical analyses were performed using R software (version 4.3.3). The "mice" package was used for MI, the "glmnet" package for LASSO analysis, the "rms" package for calibration curve plotting, and the "dcurves" and "rmda" packages for DCA and CIC assessment, respectively. $P < 0.05$ was considered statistically significant.

Results

Demographic and Clinical Data Differences Analysis

A total of 28,443 older patients with T2DM and CVD who met the inclusion and exclusion criteria were ultimately included in this study, of whom 3,984 had CKD, accounting for 14.01% of the total. Based on the time of admission, the participants were divided into a training set (n=21,234) and a validation set (n=7,209). In the training set, there were 3,012 cases in the CKD group and 18,222 cases in the non-CKD group; in the validation set, there were 972 cases in the CKD group and 6,237 cases in the non-CKD group. In both the training and validation sets, significant differences were observed in age, smoking history, drinking history, hyperlipidemia, arthritis, SBP, DBP, TC, TG, LDL-C, HDL-C, WBC count, platelet count, TyG, TC/HDL-C, UHR, AIP, NHHR, GHR, UA/Cr, and SHR ($P < 0.05$) (Table 1). The imputation results for indicators with a missing rate of less than 30% in the training and validation sets are detailed in Supplementary Tables S2 and S3.

Predictor Selection

In the training set, univariate analysis revealed that gender, smoking history, hyperlipidemia, arthritis, SBP, DBP, pulse, TC, TG, UA, LDL-C, HDL-C, hemoglobin, HbA1c, FG, WBC count, platelet count, TyG, TC/HDL-C, UHR, AIP, NHHR, GHR, and UA/Cr were significantly associated with CKD ($P < 0.05$) (Table 2). LASSO regression identified the

Table 1 Baseline Characteristics in the Training and Validation Sets

Variables	Total (N=28,443)	Training Set (N=21,234)	Validation Set (N=7,209)	*P* value
Age (year)	75.00 (70.00, 80.00)	74.00 (70.00, 79.00)	75.00 (70.00, 80.00)	< 0.001
Gender, n (%)				0.570
Male	13094 (46.04)	9754 (45.94)	3340 (46.33)	
Female	15349 (53.96)	11,480 (54.06)	3869 (53.67)	
Smoking history, n (%)				< 0.001
No	20994 (73.81)	15,486 (72.93)	5508 (76.40)	
Yes	7449 (26.19)	5748 (27.07)	1701 (23.60)	
Drinking history, n (%)				< 0.001
No	22989 (80.82)	16,992 (80.02)	5997 (83.19)	
Yes	5454 (19.18)	4242 (19.98)	1212 (16.81)	
Hyperlipidemia, n (%)				< 0.001
No	10925 (38.41)	8350 (39.32)	2575 (35.72)	
Yes	17518 (61.59)	12,884 (60.68)	4634 (64.28)	
Arthritis, n (%)				< 0.001
No	26467 (93.05)	19,840 (93.44)	6627 (91.93)	
Yes	1976 (6.95)	1394 (6.56)	582 (8.07)	
SBP (mmHg)	138.00 (126.00, 153.00)	138.00 (126.00, 154.00)	137.00 (125.00, 152.00)	< 0.001
DBP (mmHg)	80.00 (71.00, 88.00)	80.00 (71.00, 88.00)	79.00 (71.00, 87.00)	< 0.001
Pulse (bpm)	80.00 (73.00, 91.00)	80.00 (73.00, 90.00)	81.00 (73.00, 92.00)	0.184
TC (mmol/l)	4.06 (3.32, 4.86)	4.10 (3.36, 4.91)	3.93 (3.21, 4.69)	< 0.001
TG (mmol/l)	1.37 (0.98, 1.99)	1.38 (0.98, 2.00)	1.35 (0.96, 1.97)	0.020
UA (umol/l)	328.00 (261.00, 409.00)	329.00 (262.00, 410.00)	326.00 (259.00, 407.00)	0.059
LDL-C (mmol/L)	2.38 (1.72, 3.11)	2.40 (1.73, 3.13)	2.33 (1.68, 3.06)	< 0.001
HDL-C (mmol/L)	1.02 (0.84, 1.25)	1.04 (0.86, 1.26)	0.99 (0.81, 1.19)	< 0.001
Hemoglobin (g/L)	126.00 (113.00, 138.00)	126.00 (113.00, 137.00)	126.00 (113.00, 138.00)	0.270
HbA1c (%)	7.40 (6.60, 8.80)	7.40 (6.60, 8.80)	7.50 (6.60, 8.80)	0.074
FG (mmol/l)	7.50 (6.20, 9.90)	7.50 (6.20, 9.90)	7.50 (6.20, 9.80)	0.625
WBC count (×10^9/L)	6.90 (5.50, 8.90)	6.90 (5.50, 8.80)	7.00 (5.60, 9.00)	0.001
Platelet count (×10^9/L)	185.00 (146.00, 229.00)	183.00 (145.00, 228.00)	189.00 (151.00, 233.00)	< 0.001
TyG	9.07 (8.61, 9.56)	9.08 (8.62, 9.56)	9.04 (8.61, 9.54)	0.028
TC/HDL-C	3.90 (3.20, 4.76)	3.89 (3.19, 4.75)	3.93 (3.22, 4.80)	0.036
UHR (%)	13.82 (9.94, 19.19)	13.67 (9.84, 18.96)	14.24 (10.26, 19.85)	< 0.001
AIP	0.49 (0.31, 0.69)	0.49 (0.30, 0.69)	0.50 (0.32, 0.71)	< 0.001
NHHR	2.90 (2.20, 3.76)	2.89 (2.19, 3.75)	2.93 (2.22, 3.80)	0.036
GHR	7.47 (5.82, 9.79)	7.36 (5.75, 9.64)	7.80 (6.09, 10.22)	< 0.001
UA/Cr	4.39 (3.39, 5.50)	4.41 (3.40, 5.54)	4.33 (3.36, 5.41)	0.002
SHR	14.88 (12.56, 17.60)	14.90 (12.59, 17.63)	14.78 (12.52, 17.55)	0.046

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triglycerides; UA, uric acid; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; HbA1c, glycated hemoglobin; FG, fasting glucose; WBC, white blood cell; TyG, triglyceride glucose; TG/HDL-C, total cholesterol to high-density lipoprotein cholesterol ratio; UHR, uric acid to high-density lipoprotein cholesterol ratio; AIP, atherogenic index of plasma; NHHR, non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio; GHR, glycated hemoglobin to high-density lipoprotein cholesterol ratio; UA/Cr, uric acid to creatinine ratio; SHR, stress hyperglycemia ratio.

Table 2 Univariate Analysis of Baseline Characteristics in the Training Set

Variables	Total (N=21,234)	Non-CKD Group (N=18,222)	CKD Group (N=3,012)	*P* value
Age (year)	74.00 (70.00, 79.00)	74.00 (70.00, 80.00)	74.00 (70.00, 79.00)	0.076
Gender, n (%)				< 0.001
Male	9754 (45.94)	8224 (45.13)	1530 (50.80)	
Female	11480 (54.06)	9998 (54.87)	1482 (49.20)	

(*Continued*)

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Table 2 (Continued).

Variables	Total (N=21,234)	Non-CKD Group (N=18,222)	CKD Group (N=3,012)	*P* value
Smoking history, n (%)				0.015
No	15486 (72.93)	13,345 (73.24)	2141 (71.08)	
Yes	5748 (27.07)	4877 (26.76)	871 (28.92)	
Drinking history, n (%)				0.165
No	16992 (80.02)	14,553 (79.86)	2439 (80.98)	
Yes	4242 (19.98)	3669 (20.14)	573 (19.02)	
Hyperlipidemia, n (%)				< 0.001
No	8350 (39.32)	7378 (40.49)	972 (32.27)	
Yes	12884 (60.68)	10,844 (59.51)	2040 (67.73)	
Arthritis, n (%)				< 0.001
No	19840 (93.44)	17,082 (93.74)	2758 (91.57)	
Yes	1394 (6.56)	1140 (6.26)	254 (8.43)	
SBP (mmHg)	138.00 (126.00, 154.00)	138.00 (126.00, 153.00)	143.00 (129.00, 160.00)	< 0.001
DBP (mmHg)	80.00 (71.00, 88.00)	80.00 (72.00, 88.00)	79.00 (70.00, 88.00)	0.025
Pulse (bpm)	80.00 (73.00, 90.00)	80.00 (73.00, 90.00)	82.00 (75.00, 91.00)	< 0.001
TC (mmol/l)	4.10 (3.36, 4.91)	4.12 (3.39, 4.92)	3.96 (3.19, 4.88)	< 0.001
TG (mmol/l)	1.38 (0.98, 2.00)	1.37 (0.98, 1.98)	1.42 (1.01, 2.11)	< 0.001
UA (umol/l)	329.00 (262.00, 410.00)	322.00 (257.00, 398.00)	390.00 (308.00, 483.00)	< 0.001
LDL-C (mmol/L)	2.40 (1.73, 3.13)	2.43 (1.76, 3.15)	2.17 (1.54, 2.95)	< 0.001
HDL-C (mmol/L)	1.04 (0.86, 1.26)	1.05 (0.87, 1.27)	0.97 (0.79, 1.19)	< 0.001
Hemoglobin (g/L)	126.00 (113.00, 137.00)	127.00 (115.00, 138.00)	113.00 (94.00, 128.00)	< 0.001
HbA1c (%)	7.40 (6.60, 8.80)	7.40 (6.60, 8.70)	7.70 (6.70, 9.50)	< 0.001
FG (mmol/l)	7.50 (6.20, 9.90)	7.50 (6.20, 9.80)	7.90 (6.10, 10.80)	< 0.001
WBC count (×10^9/L)	6.90 (5.50, 8.80)	6.80 (5.50, 8.80)	7.10 (5.70, 9.00)	< 0.001
Platelet count (×10^9/L)	183.00 (145.00, 228.00)	184.00 (146.00, 228.00)	182.00 (140.00, 228.00)	0.008
TyG	9.08 (8.62, 9.56)	9.07 (8.61, 9.54)	9.15 (8.66, 9.68)	< 0.001
TC/HDL-C	3.89 (3.19, 4.75)	3.88 (3.18, 4.72)	4.01 (3.27, 4.96)	< 0.001
UHR (%)	13.67 (9.84, 18.96)	13.19 (9.59, 18.18)	17.02 (12.29, 24.03)	< 0.001
AIP	0.49 (0.30, 0.69)	0.48 (0.30, 0.68)	0.53 (0.34, 0.74)	< 0.001
NHHR	2.89 (2.19, 3.75)	2.88 (2.18, 3.72)	3.01 (2.27, 3.96)	< 0.001
GHR	7.36 (5.75, 9.64)	7.23 (5.67, 9.45)	8.24 (6.31, 10.78)	< 0.001
UA/Cr	4.41 (3.40, 5.54)	4.59 (3.65, 5.69)	2.86 (1.51, 4.13)	< 0.001
SHR	14.90 (12.59, 17.63)	14.90 (12.66, 17.57)	14.91 (12.08, 18.06)	0.316

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triglycerides; UA, uric acid; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; HbA1c, glycated hemoglobin; FG, fasting glucose; WBC, white blood cell; TyG, triglyceride glucose; TG/HDL-C, total cholesterol to high-density lipoprotein cholesterol ratio; UHR, uric acid to high-density lipoprotein cholesterol ratio; AIP, atherogenic index of plasma; NHHR, non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio; GHR, glycated hemoglobin to high-density lipoprotein cholesterol ratio; UA/Cr, uric acid to creatinine ratio; SHR, stress hyperglycemia ratio; CKD, chronic kidney disease.

following seven predictive variables at λ.1se = 0.008532179 (the most regularized parameter within one standard error of the minimum deviation): SBP, UA, hemoglobin, HbA1c, WBC count, TyG, and UA/Cr (Figure 2). These seven predictive factors were then entered into a multivariate logistic regression model using forward stepwise selection. The results showed that SBP [odds ratio (OR) = 1.014, 95% CI: 1.012–1.016, $P < 0.001$], UA (OR = 1.007, 95% CI: 1.006–1.007, $P < 0.001$), hemoglobin (OR = 0.985, 95% CI: 0.983–0.987, $P < 0.001$), HbA1c (OR = 1.170, 95% CI: 1.143–1.198, $P < 0.001$), WBC count (OR = 0.951, 95% CI: 0.939–0.964, $P < 0.001$), TyG (OR = 1.127, 95% CI: 1.054–1.205, $P = 0.001$), and UA/Cr (OR = 0.441, 95% CI: 0.426–0.457, $P < 0.001$) were independently associated with CKD (Figure 3). Supplementary Table S4 presents the results of multicollinearity diagnostics for these candidate variables. All VIF were < 5, and tolerance values were > 0.1, indicating the absence of significant multicollinearity in the model.

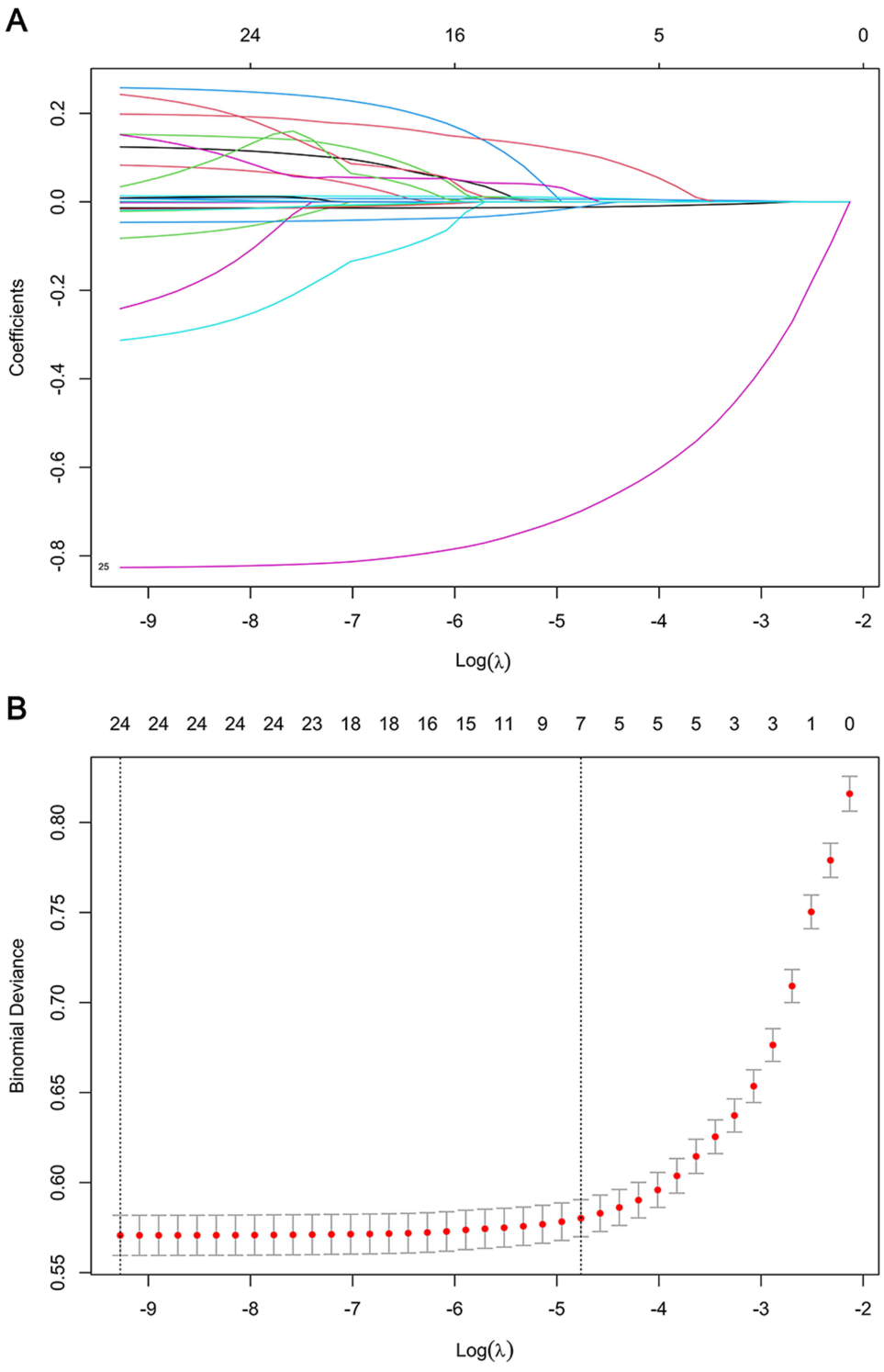

Figure 2 Variable selection via LASSO regression model. (**A**) Visualization of cross-validation error versus logarithmically transformed penalty parameter (λ) in the LASSO regularized regression framework. The lower X-axis represents Log(λ), and the upper X-axis represents the corresponding number of nonzero coefficients. Vertical dashed lines demarcate critical thresholds: the λ value yielding minimum cross-validation error (left) and λ value plus one standard error (right). (**B**) LASSO coefficient shrinkage paths delineating feature selection dynamics, demonstrating convergence trajectories of retained predictors across λ values. This visualization captures variable importance hierarchy and elucidates how regularization intensity modulates model architecture.

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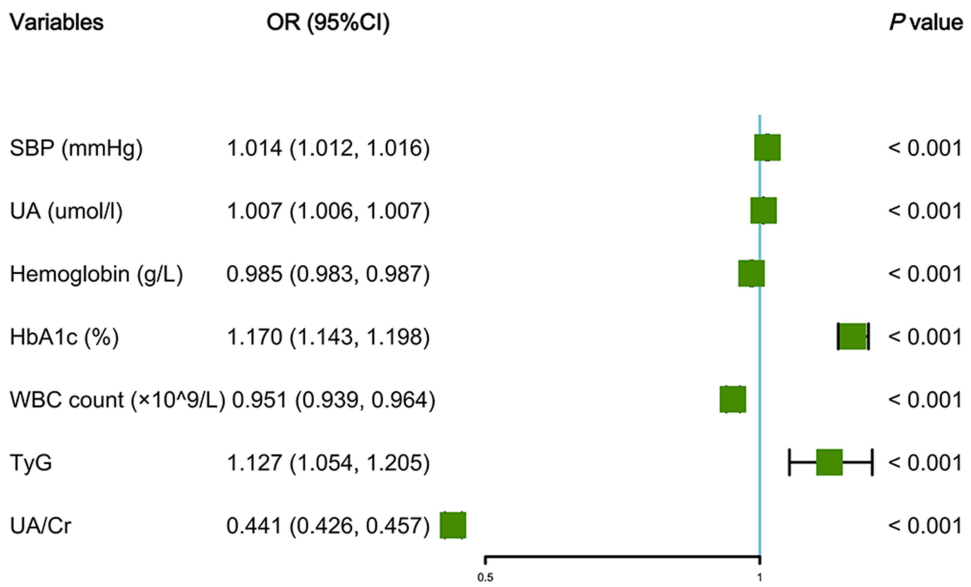

Figure 3 Forest plot showing the results of multivariable analysis.

Construction of the Nomogram Prediction Model

Based on the results of multivariate logistic regression analysis, a nomogram was constructed using the seven independent risk factors identified: SBP, UA, hemoglobin, HbA1c, WBC count, TyG, and UA/Cr (Figure 4). The direction of association was as follows: higher SBP, higher UA, higher HbA1c, and higher TyG were associated with increased CKD risk, while lower hemoglobin, lower WBC count, and lower UA/Cr were associated with increased CKD risk. By summing the scores corresponding to each predictive indicator, a total score is obtained, and the risk value corresponding to this total score represents the probability of the patient having CKD. The higher the total score, the greater the risk of CKD in older patients with T2DM and CVD. For example, a patient with SBP of 156 mmHg, UA level of 342 μmol/L, hemoglobin level of 97 g/L, HbA1c level of 6.6%, WBC count of 4.7×10^9/L, TyG of 8.87, and UA/Cr of 1.49 accumulates a total score of 460, corresponding to a predicted probability of having CKD of 60.90% (Supplementary Figure S1).

Evaluation and Validation of the Prediction Model

Following the construction of the nomogram, the discriminative ability of the model was quantified using ROC curves. In both the training set (Figure 5A) and the validation set (Figure 5B), the AUCs were 0.845 (95% CI: 0.836–0.853) and 0.853 (95% CI: 0.838–0.867), respectively, indicative of a high degree of predictive accuracy. Supplementary Figure S2 presents the ROC curves for each predictive variable, with UA/Cr exhibiting the highest AUC value of 0.773, followed by hemoglobin at 0.685. Calibration curves demonstrated a close agreement between predicted and observed risks. The Brier scores for the training and validation cohorts were 0.081 (95% CI: 0.078–0.083) and 0.078 (95% CI: 0.073–0.082), respectively (Figure 5C and D), with both calibration curves closely approximating the ideal line, thereby confirming the model's reliable probability estimation.

DCA revealed the net benefit of the model across a broad range of risk thresholds. At a threshold probability of 0.12, the model-guided intervention strategy yielded a higher net benefit compared to treating all or no patients (Figure 6A, training set; Figure 6B, validation set), highlighting the clinical value of the model. CIC showed a sharp decline in the number of individuals classified as high risk and a gradual decrease in the number of true CKD events among those labeled as high risk as the threshold increased (Figure 6C and D). This pattern indicates that as the threshold rises, the model increasingly focuses on the target population, thereby facilitating the rational allocation of resources. Table 3 summarizes the model's performance in terms of sensitivity, specificity, PLR, NLR, and DOR, providing a comprehensive evaluation.

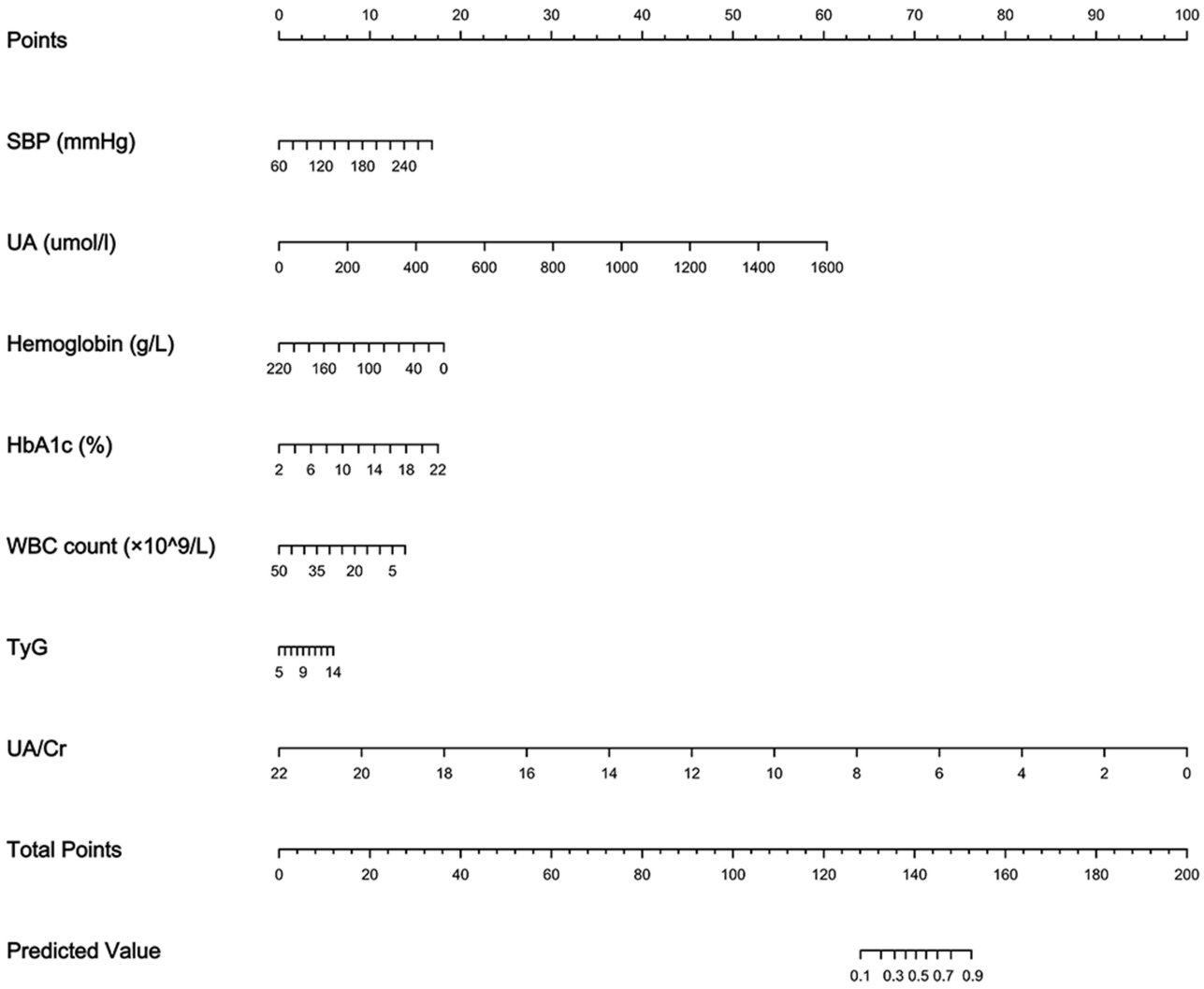

Figure 4 Prediction nomogram model for risk of CKD.

Discussion

Given the complex interplay and shared risk factors among these diseases, the comorbidity of T2DM, CVD, and CKD poses significant challenges. Our study addresses this challenge by developing a predictive model that can identify individuals at high risk of CKD among older patients with T2DM and CVD, thereby enabling targeted interventions. This approach not only enhances the precision of clinical management but also has the potential to alleviate the societal and economic burdens associated with these chronic conditions.

The global prevalence of CKD is estimated to range from 11% to 13%, and the number of patients with end - stage renal disease (ESRD) requiring renal replacement therapy is estimated to be between 4.902 million and 7.083 million.[26,27] A longitudinal cohort study based on primary care in the United Kingdom showed that the prevalence of CKD was 18.2% (95% CI: 16.9–19.6%).[28] Within Asia, there is a significant variation in the prevalence of CKD, which ranges from approximately 7.0% to 34.3%.[29] Among Chinese adults, the prevalence of CKD is about 10.8% (95% CI: 10.2–11.3%).[30] CKD often lacks obvious clinical symptoms in its early stages, and patients are typically not diagnosed until significant renal impairment occurs.[31] Moreover, given that some patients have comorbidities such as hypertension, diabetes, severe electrolyte disturbances, or renal structural abnormalities, patients with CKD are usually at high risk of adverse prognosis and mortality.[32] To date, appropriate therapeutic approaches for older patients with CKD remain unclear. Given the high prevalence of CKD in the older population, it is particularly important to identify and

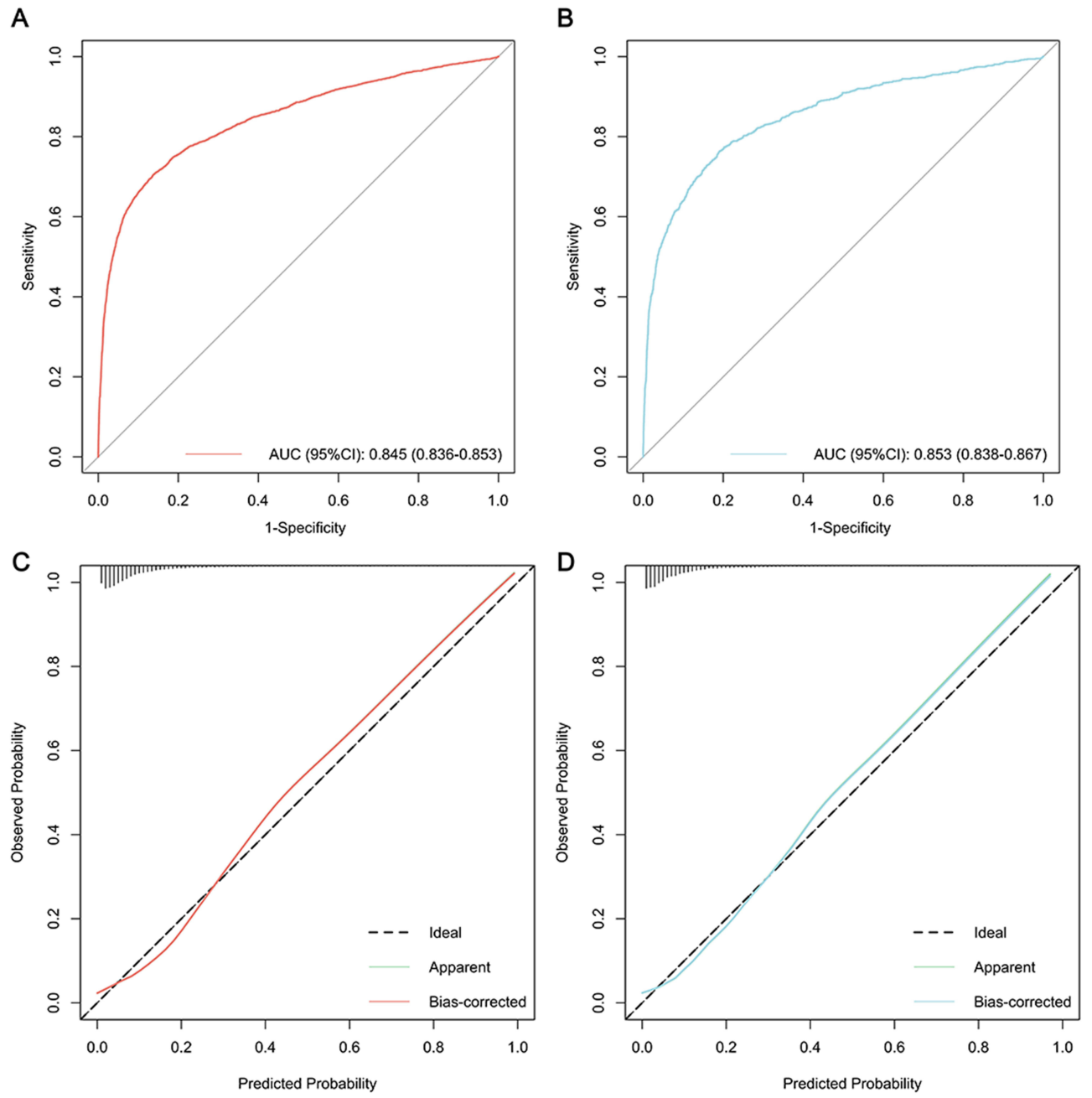

Figure 5 The performance of the nomogram model to predict the probability of CKD. (**A**) ROC curve and AUC to evaluate the prediction accuracy in the training set. (**B**) ROC curve and AUC to evaluate the prediction accuracy in the validation set. (**C**) Calibration curve to assess the agreement of actual probabilities and predicted probabilities for prediction accuracy in the training set. (**D**) Calibration curve to assess the agreement of actual probabilities and predicted probabilities for prediction accuracy in the validation set. The black line segments at the top of panels (**C** and **D**) represent the density distribution of predicted probabilities, with higher density indicated by longer line segments. The diagonal dashed line represents perfect calibration, and the solid line represents the observed calibration of the model.

prevent risk factors for CKD in the older, especially in older patients with T2DM and CVD, which can help to better understand, prevent, and treat this disease.

Cheru et al conducted a retrospective cohort study involving 520 patients with diabetes in the Harari region of Ethiopia.[33] The results indicated that the risk of CKD was tripled in patients aged 60 years and older [adjusted hazard ratio (AHR): 3.09, 95% CI: 1.56–6.14], highlighting the necessity for focused follow - up of older patients with T2DM to prevent further progression to CKD. Cai et al developed a CKD prediction model based on 210 patients with T2DM using logistic regression analysis, which included five risk factors (FG, Cr, HbA1c, diabetic retinopathy, and duration of

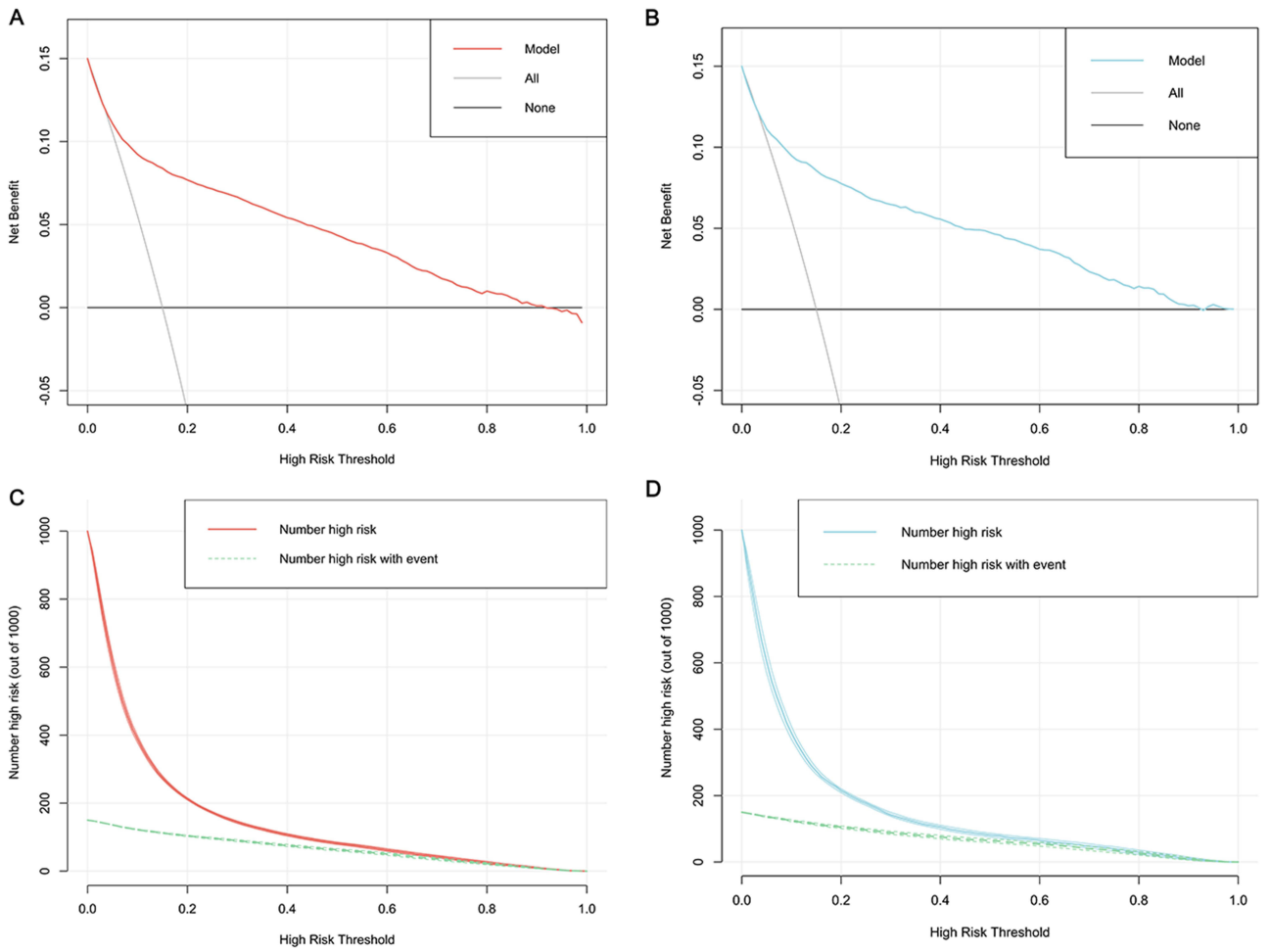

Figure 6 Clinical decision curves and impact curves for the nomogram model. (**A**) Decision curve analysis of the nomogram in the training set. (**B**) Decision curve analysis of the nomogram in the validation set. The "all" line indicates the benefit when all patients are treated, and the "none" line when no patients are treated. (**C**) Clinical impact curve of the nomogram in the training set. (**D**) Clinical impact curve of the nomogram in the validation set. The solid lines depict the total number of individuals identified as high risk, and the dashed lines represent those at high risk who experienced the true event.

diabetes), with an AUC of 0.811.[34] In contrast, our model exhibited a higher AUC (0.854), and our cohort generally consisted of older patients with higher levels of Cr and HbA1c. In our study, 14.01% of patients had CKD, compared with 19.09% in the cohort of Zhang et al[35] Compared with previous models targeting general T2DM populations or those at high CVD risk, our model has several specificities: (1) it includes SBP and TyG to capture the combined effects of hypertension and insulin resistance, which are particularly pronounced in older patients with both T2DM and CVD; (2) it incorporates UA/Cr ratio as a predictor of early tubular injury, a feature less emphasized in general populations; (3) it

Table 3 Predictive Performances of the Risk Prediction Model for CKD

Performance Indicators	Training Set	Validation Set
AUC (95% CI)	0.845 (0.836–0.853)	0.853 (0.838–0.867)
Sensitivity (95% CI)	0.705 (0.689–0.721)	0.763 (0.737–0.790)
Specificity (95% CI)	0.862 (0.857–0.867)	0.810 (0.800–0.819)
PLR (95% CI)	5.121 (4.906–5.347)	4.011 (3.770–4.268)
NLR (95% CI)	0.342 (0.323–0.361)	0.292 (0.261–0.327)
DOR (95% CI)	14.960 (13.688–16.351)	13.676 (11.646–16.060)

Abbreviations: AUC area under the ROC curve, PLR positive likelihood ratio, NLR negative likelihood ratio, DOR diagnostic odds ratio, CI confidence interval.

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includes hemoglobin and WBC count to reflect the specific vulnerabilities of older patients to anemia and inflammation; and (4) it relies solely on routine clinical data, ensuring practical applicability in older patients with multimorbidity. Moreover, predictive models for early CKD have been developed for various populations, such as patients with liver cirrhosis, stone formers, and renal cell cancer patients, incorporating a range of clinical and laboratory variables.[36–38] However, studies focusing on older patients with T2DM and CVD are relatively scarce. The model proposed in this study, which is based on common laboratory indicators to predict early CKD in older patients with T2DM and CVD, is particularly useful in routine clinical settings. This is crucial because predictive models involving rare variables, complex calculations, or expensive tests are less likely to be used in practice.

Numerous large-scale epidemiological studies have thoroughly investigated the association between UA levels and the prevalence or progression of kidney disease. In the United States Renal Data System (USRDS) database, a prospective study involving 177,570 patients with a follow-up period exceeding 25 years revealed that patients in the highest quartile of UA had a HR of 2.14 for end-stage renal disease (ESRD).[39] Obermayr et al reported on the Vienna Health Screening Project, which assessed 21,475 patients and found that a mild elevation in UA levels (7.0–8.9 mg/dL) was almost doubly associated with the risk of CKD (OR = 1.74; 95% CI: 1.45–2.09), while a significant increase in UA levels (≥9.0 mg/dL) was associated with a threefold risk of CKD (OR = 3.12; 95% CI: 2.29–4.25), consistent with the findings of our study.[40] Moreover, patients with hyperuricemia often exhibit varying degrees of renal lesions, including small artery sclerosis, glomerulosclerosis, and interstitial fibrosis, frequently accompanied by extrarenal UA crystal deposition. UA levels have also been identified as an independent predictor of IgA nephropathy progression and are closely related to the development of renal insufficiency in patients with T2DM.[41] Notably, as a derivative index of UA, UA/Cr was identified as an important predictor of CKD in our study. UA/Cr reflects net UA production, and we observed that UA levels did not entirely correlate with Cr levels. This phenomenon may be attributed to tubular damage. Therefore, we hypothesize that interstitial damage is associated with the occurrence of CKD. Moreover, in the context of extensive tubulointerstitial damage, decreased reabsorption of UA leads to increased creatinine levels and a decreased UA/Cr ratio.[42,43] These findings may elucidate the potential utility of UA/Cr as a biomarker and diagnostic indicator for CKD.

In our study, hemoglobin levels emerged as a protective factor against CKD, with higher levels associated with a lower risk of CKD development. Xie et al discovered that anemia (defined as Hb < 120 g/L in women and Hb < 130 g/L in men) is a significant predictor of renal function decline in patients with T2DM.[44] A prospective study based on 439 patients with CKD stage G3 demonstrated that, compared to non-anemic patients, anemic patients experienced a more rapid decline in eGFR, reached CKD stages G4 or G5 more quickly, and had a higher mortality rate.[45] The primary mechanism is that anemia leads to tissue hypoxia and/or increased oxidative stress, which may accelerate renal function deterioration. Another study based on the China Health and Retirement Longitudinal Study (CHARLS) reached a conclusion consistent with ours. After adjusting for potential confounding factors, hemoglobin levels were independently associated with rapid renal function decline (OR=0.90, 95% CI: 0.87–0.94).[46]

In addition, our study identified elevated SBP, elevated HbA1c levels, elevated TyG levels, and low WBC count as significant predictive variables for CKD. Elevated SBP indicates potential hypertension risk, and a study has shown that the relationship between hypertension and CKD is bidirectional: hypertension can be both a cause and a consequence of CKD.[47] In CKD patients, elevated blood pressure is typically caused by sodium retention, activation of the renin-angiotensin-aldosterone system, excessive activity of the sympathetic nervous system, and increased vascular stiffness.[48] Long-term hyperglycemia can lead to glomerular hyperfiltration and tubulointerstitial damage, thereby triggering CKD.[49] Moreover, elevated HbA1c levels are also closely related to the risk of cardiovascular disease, which may further exacerbate renal damage through pathways such as activating oxidative stress and impairing vascular endothelial function.[50] Notably, even in non-diabetic populations, elevated HbA1c levels are significantly associated with the risk of developing CKD.[51] Multiple studies have also confirmed that elevated TyG levels are an independent risk factor for CKD.[52–54] WBC count is an indicator of systemic inflammation, which plays a key role in the development and progression of CKD. One study found that low WBC count is independently associated with the progression of CKD, suggesting that changes in WBC count may reflect changes in immune system function, thereby affecting the development of renal disease.[55] Additionally, low WBC count may also be related to the overall health status and decreased

immune function in older patients, which further increases the risk of CKD.[56] In summary, the above indicators hold significant potential for predicting CKD.

To further illustrate the novelty and advantages of our model over existing approaches, we summarized the key characteristics of previously published CKD prediction models for patients with T2DM alongside our present model in Supplementary Table S5. As shown, our model features a larger sample size, a more specific target population (older T2DM with comorbid CVD), a broader set of predictors (including novel ones such as UA/Cr, TyG, and WBC count), a user-friendly nomogram, higher discriminative performance (AUC > 0.85), and formal clinical utility assessment (DCA and CIC). Notably, to our knowledge, few prior models have been specifically developed for this high-risk population, and all predictors are routinely collected variables, making our model easily implementable at bedside without additional cost.

It should be acknowledged that several limitations exist in the present study. First, as a retrospective study, issues of information bias and selection bias are inevitable Second, being a single-center study, although the training and validation sets were separated, our findings still require external validation datasets for further corroboration. Lastly, due to resource constraints, we were unable to include more relevant factors, and some variables may lack universally accepted definitions. Therefore, further studies are needed to confirm and expand upon our findings.

Conclusions

This study identified seven independent predictors of CKD in older patients with T2DM and CVD: SBP, UA, hemoglobin, HbA1c, WBC count, TyG, and UA/Cr. The nomogram developed from these predictors demonstrated good discriminative ability (AUC 0.845–0.853) and satisfactory calibration, with formal clinical utility confirmed by decision curve analysis. Compared with previous models, our nomogram offers a more targeted tool for this specific high-risk population, relies solely on routine clinical data, and provides a user-friendly format for bedside risk stratification. By enabling early identification of high-risk individuals, this model can facilitate timely interventions, potentially delaying CKD progression, reducing cardiovascular complications, and improving overall outcomes in this vulnerable population. External validation in diverse cohorts is warranted to further confirm its generalizability.

Data Sharing Statement

The datasets used and/or analyzed in the current study are available from the corresponding authors upon reasonable request.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Affiliated Banan Hospital of Chongqing Medical University (Approval No. BNLLKY2025086). Written informed consent for participation was not required for this study due to its retrospective design. This study was conducted in full compliance with the ethical principles outlined in the Declaration of Helsinki (1964) and its subsequent amendments. All procedures and methodologies adhered strictly to the relevant ethical guidelines and regulatory requirements.

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Author Contributions

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

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Disclosure

The authors declare no competing interests in this work.

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