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ORIGINAL RESEARCH

Construction and Validation of a Nomogram-Based Predictive Model for Acute Kidney Injury Caused by Drug Resistance to Tuberculosis

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Objective: Acute kidney injury (AKI) is a common and serious adverse effect during tuberculosis (TB) treatment in clinical settings, particularly in patients with drug-resistant TB. AKI may lead to treatment interruption and poor prognosis. Early identification of patients at high risk for AKI is crucial to improve clinical outcomes.

Methods: We retrospectively enrolled 571 TB patients, divided into training and validation cohorts. LASSO and multivariate logistic regression were used to identify risk factors, and the nomogram was evaluated using AUC, calibration, and decision curve analysis (DCA).

Results: This study included 571 patients with TB. In this study, five variables (age, hypertension, diabetes, Scr, and ALB) were included to construct a nomogram for predicting AKI caused by drug resistance to TB. The AUC of the training set and validation set were 0.809 (95% CI: 0.7480–0.871, $P < 0.001$) and 0.841 (95% CI: 0.765–0.918, $P < 0.001$), respectively, indicating that the prediction model had good discriminative performance. The calibration curve shows that the predicted values of the model are basically consistent with the actual values, indicating good performance. DCA suggests that almost all ranges of TB patients can benefit from this new predictive model, indicating good clinical utility.

Conclusion: The nomogram model of AKI caused by drug resistance to TB established in this study has good predictive value and helps identify high-risk populations.

Keywords: anti tuberculosis drugs, acute renal injury, risk factors, nomogram

Introduction

Tuberculosis (TB) is a frequent disease of the respiratory system and an important social and public health problem worldwide.[1,2] According to the Global Tuberculosis Report, China ranked third among the 30 countries with the highest burden of TB in 2021.[1,2] Due to its high infectivity, timely and effective treatment is essential to control disease progression and prevent transmission.[1,2]

Pyrazinamide, ethambutol, rifampicin, isoniazid and other drugs are commonly used in clinical treatment of TB.[3,4] However, while these drugs have therapeutic effects, they can also cause certain damage to the body and trigger different types of adverse reactions.[4–6] AKI is a common type of adverse reaction in TB patients.[7] It not only damages the patient's physical health but also reduces their medication adherence and even leads to treatment interruption. This can affect treatment efficacy, leading to treatment failure and the formation of drug-resistant *Mycobacterium* TB.[7] Research has found that physiological, pathological, and environmental factors may cause abnormal absorption, metabolism, and distribution of anti TB drugs in the body, leading to an increase in nephrotoxic metabolites. This leads to AKI caused by

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drug resistance to TB.[7] Therefore, it is of great significance to clarify the risk factors related to AKI caused by drug resistance to TB, which can help guide the implementation of targeted interventions in clinical practice.

Nomogram-based predictive models have gained increasing popularity in clinical research due to their ability to transform complex regression equations into visual tools that are intuitive and user-friendly. These models integrate multiple predictors into a single graphical interface to estimate the probability of clinical outcomes. Nomograms have been widely used for individualized risk prediction in oncology, cardiovascular disease, and nephrology.[7,8] Their application in AKI prediction allows for early identification of high-risk patients and facilitates timely clinical decision-making.[9]

The early stage of AKI has no significant clinical symptoms or signs. If not detected in a timely manner, it may delay the optimal intervention time and lead to serious consequences such as chronic kidney disease. However, establishing a high-performance AKI risk prediction model will change this unfavorable situation.[10,11] Therefore, this study aims to establish a nomogram prediction model for AKI caused by drug resistance to TB based on the fusion of serum indicators and clinical data. Aiming to explore a convenient and reliable model for predicting the risk of AKI caused by drug resistance to TB patients, providing a scientific assessment tool for high-risk patients. This study aims to provide scientific references for exploring disease intervention and treatment strategies, reducing the occurrence of AKI caused by drug resistance to TB, and improving patient prognosis.

Materials And Methods

Research Design And Patients

The study was conducted at the Affiliated Hospital of Hebei University. We retrospectively selected TB patients admitted to the Affiliated Hospital of HeBei University from July 2021 to June 2024. This study was approved by the Ethics Review Board of the Affiliated Hospital of HeBei University (Approval No. HL2024122), and the requirement for informed consent was waived due to the retrospective design.

Inclusion criteria: 1) Meet the diagnostic criteria for pulmonary TB;[12] 2) Initial/recurrent treatment of pulmonary TB; 3) Age > 18 years old; 4) Complete clinical data.

Exclusion criteria: 1) AKI caused by other drugs; 2) Patients with organic lesions in other important organs; 3) Patients with malignant tumors; 4) Female patients during lactation and pregnancy; 5) Patients with autoimmune, endocrine, and metabolic system disorders.

Treatment Protocol

Newly diagnosed pulmonary TB received a standardized short course chemotherapy for 6 months according to the 2HREZ/4HR regimen. Resurrected pulmonary TB received standardized short-term chemotherapy for 8 months according to the 2SHRZE/1HRZE/5HRE regimen.[13]

Data Collection

Clinical data of patients were collected from electronic medical records: 1) General clinical characteristics: including gender, age, BMI, smoking, drinking, history of hypertension, history of diabetes, and whether to use gastric protective drugs. 2) Pre-treatment laboratory indicators include serum uric acid (UA), serum creatinine (Scr), urea nitrogen (Urea), cystatin C (Cys-C), and blood albumin (ALB) levels. Extract 3 mL of fasting venous blood and 5 mL of mid-morning urine from the patient in the morning; Measure UA and Scr levels using oxidase method; Measure the level of urea using urease UV method; Double antibody sandwich enzyme-linked immunosorbent assay was used to measure Cys-C levels, and bromocresol green assay was used to measure ALB levels. The relevant reagent kits were purchased from Roche Shanghai, China, and all of them were equipped with fully automatic biochemical analyzers such as Cobas ® 8000 (Roche, Basel, Switzerland) and supporting reagent kit for testing.

Diagnostic Criteria for AKI Caused by Drug Resistance to TB

Refer to the diagnostic criteria of the Global Kidney Disease Prognostic Organization's "Guidelines for Acute Renal Injury".[14] Within 48 hours of taking anti TB drugs, the Serum Scr increased by ≥26.5 μmol/L; or within 7 days of taking

anti TB drugs, the increase in Scr value is ≥1.5 times the baseline value; The definition of AKI caused by drug resistance to TB drugs is that the urine output is less than 0.5mL/(kg · hour) for 6 hours after taking anti TB drugs.

Quality Control

To ensure the authenticity and accuracy of the collected data and to minimize or avoid bias to the greatest extent possible, this study conducted quality control in all aspects of data collection, organization, and analysis. Organize necessary training for investigators in advance on the inclusion and exclusion criteria for selected medical records to reduce potential selection bias during the sample extraction process. All investigators have a background in medical knowledge and professional titles in health. They are required to strictly follow the pre-established standardized form content for data collection, treat each research subject equally without subjective judgment, and reduce potential information bias during the data collection process. After collecting the data, organize and review it to eliminate relevant materials with missing information and logical errors.

Statistical Analysis

We input the data into Microsoft Excel and analyze it using SPSS version 26.0 (IBM Corp, Armonk, NY, USA). We evaluate the normality of the distribution of continuous variables using the Shapiro Wilk test. Non-normally distributed data are represented by median and interquartile range (IQR), and Wilcoxon test is used to compare the differences between the training and validation cohorts. Count data is represented by n (%), and chi square test is used. To construct the nomogram, we first applied Least Absolute Shrinkage and Selection Operator (LASSO) logistic regression to reduce the dimensionality of predictor variables and address potential multicollinearity.[15] This method was implemented using the "glmnet" package in R (version 4.2.2), and ten-fold cross-validation was performed to identify the optimal penalization parameter (λ) that minimized mean cross-validated error. Variables with non-zero coefficients at the optimal λ were selected for subsequent multivariate logistic regression. The final nomogram was constructed using the "rms" package based on the regression coefficients, allowing graphical estimation of AKI risk in individual patients. Using the Hosmer Lemeshow goodness of fit, calibration curve and other tests to evaluate the goodness of fit between the predicted and observed values of the model (ie model consistency test); Calculate the C-index and the area under the working characteristic curve (AUC) of the subjects. Evaluate the discriminative power, ie accuracy, of the nomogram among patients. Using decision curve analysis (DCA), we evaluate the clinical application value of nomogram by calculating net benefits (NB) at different threshold probabilities. In this study, the threshold probability represents the risk of AKI caused by drug resistance to TB identified by the nomogram. Test level α=0.05.

Results

Patient Characteristics

A total of 571 anti TB patients were included in this study. It is then randomly divided into two cohorts in a 7:3 ratio. The training cohort included 400 patients, and the validation cohort included 171 patients. The screening process is shown in Figure 1. The clinical characteristics of the patients are shown in Table 1. After treatment, AKI incidence was 11.8% (47/400) in the training cohort and 15.2% (26/171) in the validation cohort (P=0.257), with no significant difference.

Construction and Validation of Nomogram Model

In the training cohort, we use LASSO regression algorithm for feature selection. This method helps to minimize the impact of multicollinearity and provides strong predictability and stability. We select features based on the minimum partial likelihood binomial bias, and LASSO regression retains 9 non-zero coefficient variables (Figure 2). These variables are considered to be significantly correlated with AKI. Determined variables include age, hypertension, diabetes, combination of gastric drugs, UA, Scr, Urea, Cys-C, and ALB.

To further investigate their predictive significance, we conducted multiple logistic regression analysis using these seven variables. The results showed that age ≥60 years [odds ratio (OR)=2.667; 95% confidence interval (CI)=1.308–5.440; P=0.007], hypertension (OR=2.671; 95% CI=1.297–5.502; P=0.008), diabetes (OR=5.379; 95%

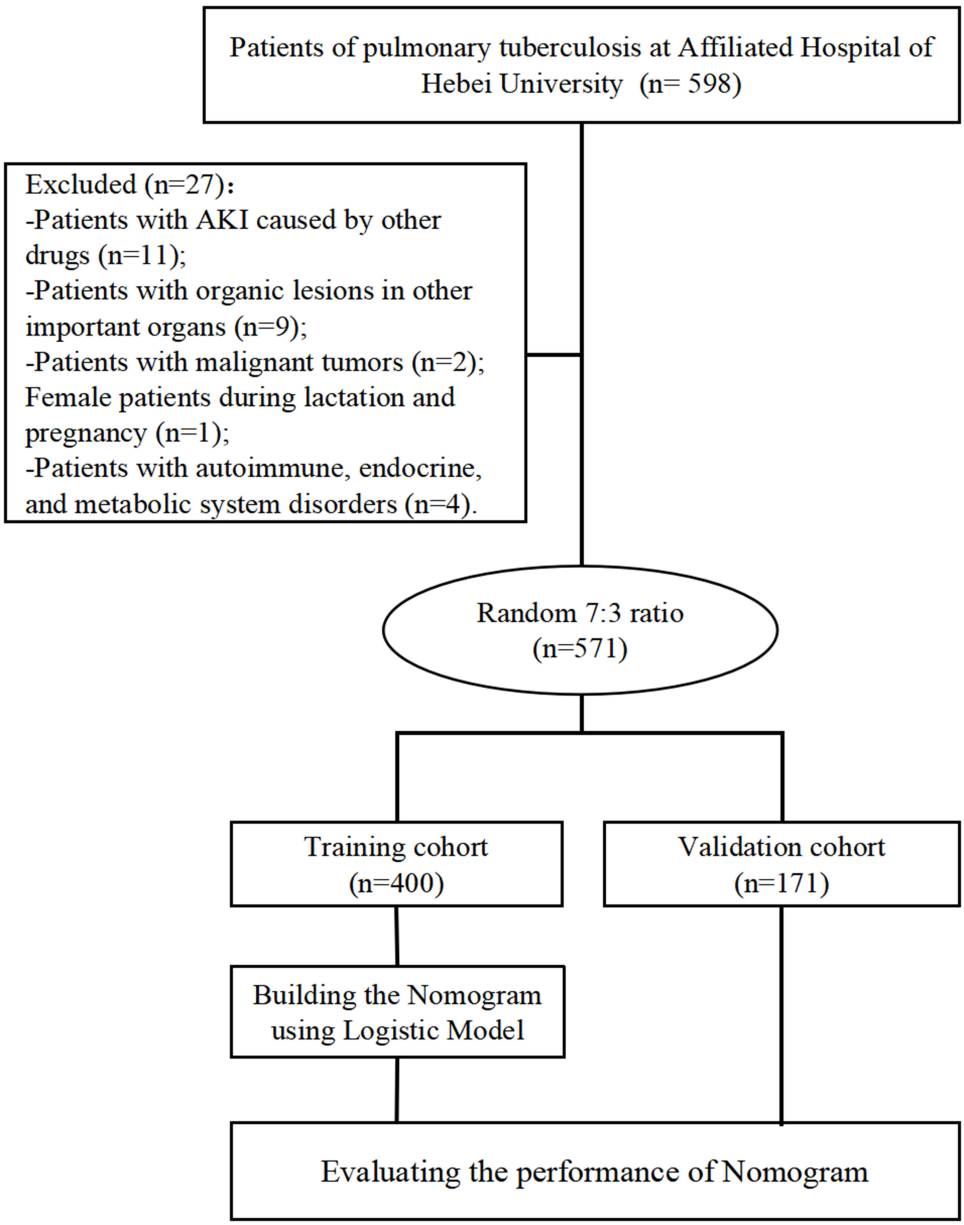

Figure 1 Participant screening process diagram.

CI=2.630–11.004; *P*<0.001), Scr (OR=1.032; 95% CI=1.011–1.053; *P*=0.003) and ALB (OR=0.908; 95% CI=0.842–0.979; *P*=0.012) are significant predictors of AKI in patients. Although albumin (ALB) was identified as a protective factor (OR < 1), it was retained in the final model due to its statistical significance and clinical relevance in improving predictive performance. The detailed results of multiple logistic regression analysis are shown in Table 2.

Construct a nomogram model for predicting the risk of AKI based on the five independent risk factors mentioned above (Figure 3). In this nomogram, each predictor is assigned a specific score according to its weight in the regression model. By summing these scores, a total score is calculated, which corresponds to a probability on the risk axis that estimates the likelihood of AKI occurrence.

In the Hosmer Lemeshow test, the training cohort had $\chi 2$=6.482, *P*=0.593, and the internal validation cohort had $\chi 2$=6.852, *P*=0.553. This result indicates that the predicted results are close to the observed results. The ROC curve in the training cohort showed good discriminability (AUC: 0.809; 95% CI: 0.7480–0.871); The discriminative performance of the model was validated in the validation cohort (0.841; 0.765–0.918) (Figure 4). In addition, calibration curve analysis

Table 1 Clinical Characteristics of Patients

Characteristics	Training Cohort (n=400)	Validation Cohort (n=171)	χ2/t	P
Sex, n(%)				
Male	167 (41.8)	78 (45.6)	0.73	0.393
Female	233 (58.2)	93 (54.4)		
Age (year), n (%)			0.316	0.574
<60	264 (66.0)	117 (68.4)		
≥60	136 (34.0)	54 (31.6)		
BMI (kg/m^2), n (%)			0.588	0.443
<28	300 (75.0)	123 (71.9)		
≥	100 (25.0)	48 (28.1)		
Smoking (Yes),n(%)	129 (32.3)	65 (38.0)	1.773	0.183
Drinking alcohol (yes), n(%)	120 (30.0)	59 (34.5)	1.129	0.288
History of hypertension (yes), n(%)	110 (27.5)	54 (31.6)	0.974	0.324
History of diabetes (Yes), n(%)	83 (20.8)	39 (22.8)	0.302	0.583
Combination stomach medication (Yes), n(%)	238 (59.5)	106 (62.0)	0.310	0.578
UA (umol/L), M(P25/P75)	347 (295–379)	340 (294–365)	−0.893	0.372
Scr (umol/L), M(P25/P75)	87 (81–100)	87 (78–97)	−0.466	0.641
Urea (umol/L), M(P25/P75)	3.55 (3.2–4.2)	3.5 (3.2–4.1)	−0.224	0.823
Cys-C (mg/L), M(P25/P75)	1.54 (1.21–2.15)	1.59 (1.11–1.94)	−1.665	0.096
ALB (g/L), M(P25/P75)	32 (27–35)	34 (27–36)	−1.412	0.158
AKI (yes), n (%)	47 (11.8)	26 (15.2)	1.282	0.257

Notes: For binary variables, only the "Yes" group is displayed for simplicity and space-saving purposes.

shows that there is a good consistency between the predicted probability and the observed incidence of AKI in both the training cohort and validation cohort (Figure 5). Draw DCA curves using training cohort data and validation cohort data separately (Figure 6). The DCA curves of the training cohort and validation cohort indicate that patients can benefit from this novel prediction model almost entirely within the range of 0% to 100%. This indicates that the prediction model has good clinical practicality.

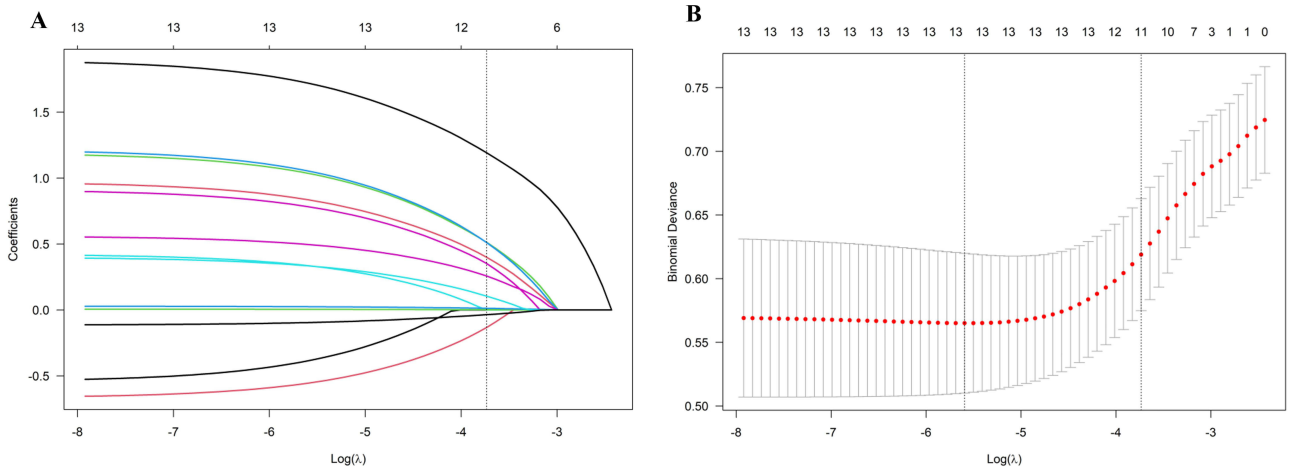

Figure 2 LASSO coefficient curve of AKI caused by drug resistance to TB. (**A**) Each curve in the graph represents the coefficient variation of each variable; The vertical axis represents the coefficient values, the lower horizontal axis represents log (λ), and the upper horizontal axis represents the number of non-zero coefficients in the model at each time point. (**B**) 10-fold cross-validation fitting.

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Table 2 Analysis of Adverse Risk Factors for AKI Caused by Drug Resistance to TB

Independent variables	B	95% CI	P
Age ≥ 60 years	0.981	2.667(1.308–5.440)	0.007
Hypertension	0.983	2.671(1.297–5.502)	0.008
Diabetes	1.683	5.379(2.630–11.004)	<0.001
Scr	0.031	1.032(1.011–1.053)	0.003
ALB	−0.097	0.908(0.842–0.979)	0.012

Note: B is the regression coefficient.

Discussion

The selected demographic variables—age, hypertension, and diabetes—are established clinical risk factors for kidney injury.[14] Older individuals often have reduced renal reserve, and both hypertension and diabetes contribute to renal microvascular damage, making patients more susceptible to AKI during intensive anti-TB treatment.[8,9,16]

The results of this study showed that the incidence of AKI caused by drug resistance to TB was 12.8% (73/571). Chang et al[17] found that out of 1394 patients receiving anti TB treatment, 99 (7.1%) had AKI. In addition, Huerga et al[16] observed 472 patients receiving bedaquiline and delamanid treatment in 14 countries/regions and found that 40 patients (8.5%) developed AKI. It can be seen that our hospital has a high incidence of AKI caused by drug resistance to TB.

Recent evidence from TB-endemic regions underlines the need for effective AKI screening tools. Bagcchi called attention to frequent underdiagnosis of AKI in high-burden areas and emphasized early detection as critical for patient prognosis.[1] Our nomogram directly responds to this demand by integrating five routine clinical variables into an accessible risk stratification tool.

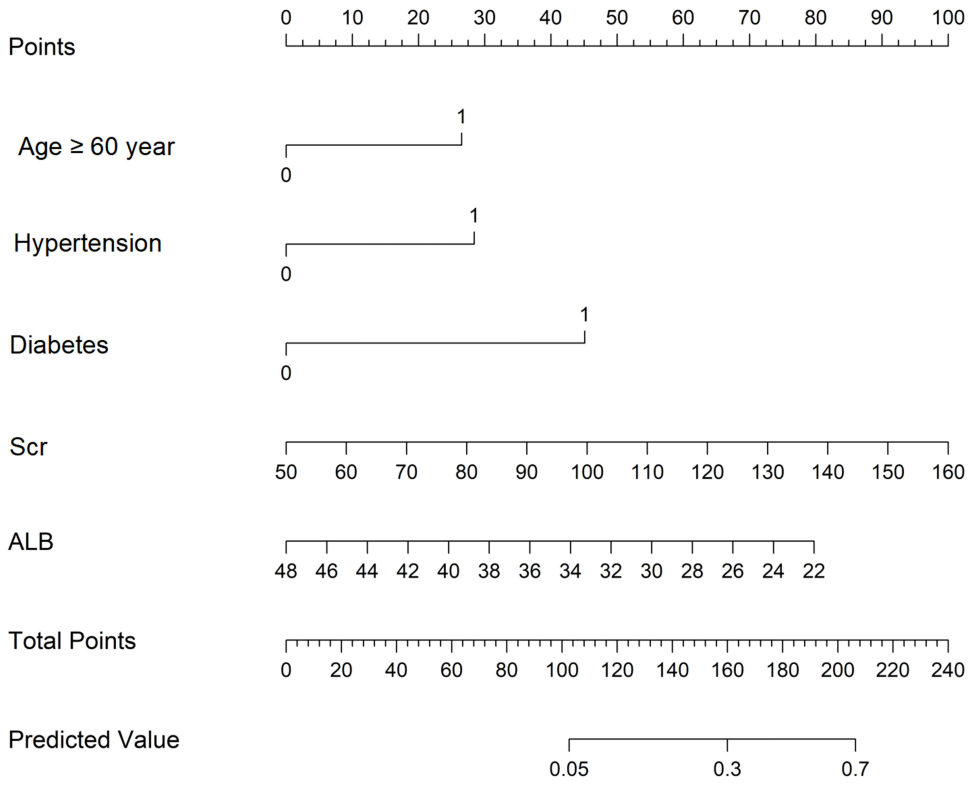

Figure 3 Nomogram of the risk prediction model for AKI caused by drug resistance to TB. In the prediction model, indicates no AKI and indicates presence of AKI. Each level of the predictor variable represents a specific score. The total score is generated by summarizing the scores of each predictor variable. The total score corresponds to the AKI caused by drug resistance to TB probability.

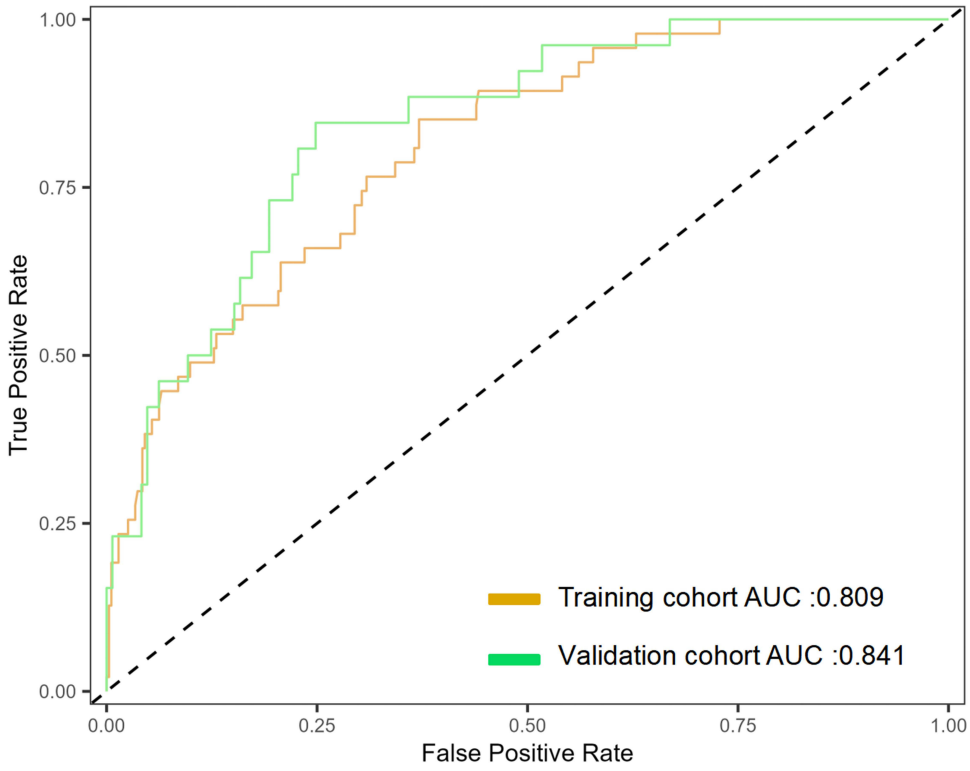

Figure 4 Shows the ROC curve and AUC of the prediction model. ROC:receiver operating characteristic; AUC:under the curve.

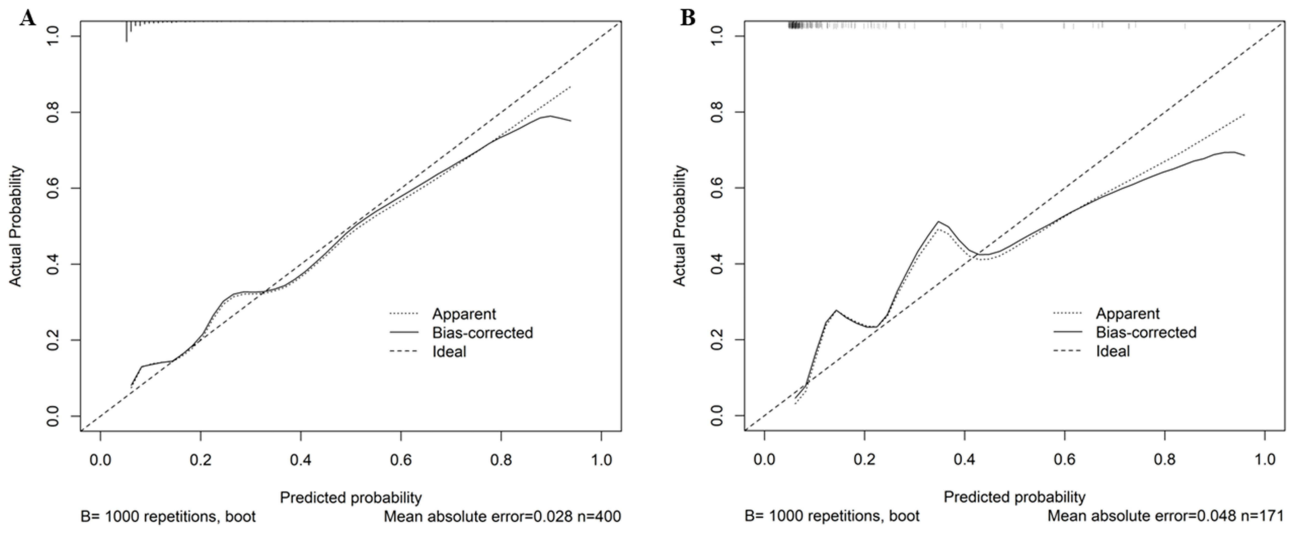

Figure 5 Calibration diagram of the prediction model. (**A**). Calibration chart of the training cohort. (**B**). Calibration chart in the internal validation cohort. The x-axis represents the predicted probability of AKI caused by drug resistance to TB. The y-axis represents the observed AKI caused by drug resistance to TB. The diagonal dashed line represents the perfect prediction of the ideal model. The solid line represents the performance of the nomogram. It indicates that solid lines are closer to diagonal dashed lines for better prediction, indicating that the model has good predictive ability.

Moreover, recent nomogram-based studies in other patient populations further validate this methodological approach. For example, Parsons et al developed an AKI risk model for vancomycin-treated ICU patients with strong discrimination (AUC ≈0.79) using LASSO regression—a similar statistical framework to ours.[8,9] Additionally, Zhang et al noted that low albumin levels significantly correlated with AKI incidence in patients with acute non-variceal upper gastrointestinal bleeding, supporting our finding that ALB acts as a protective factor.[8]

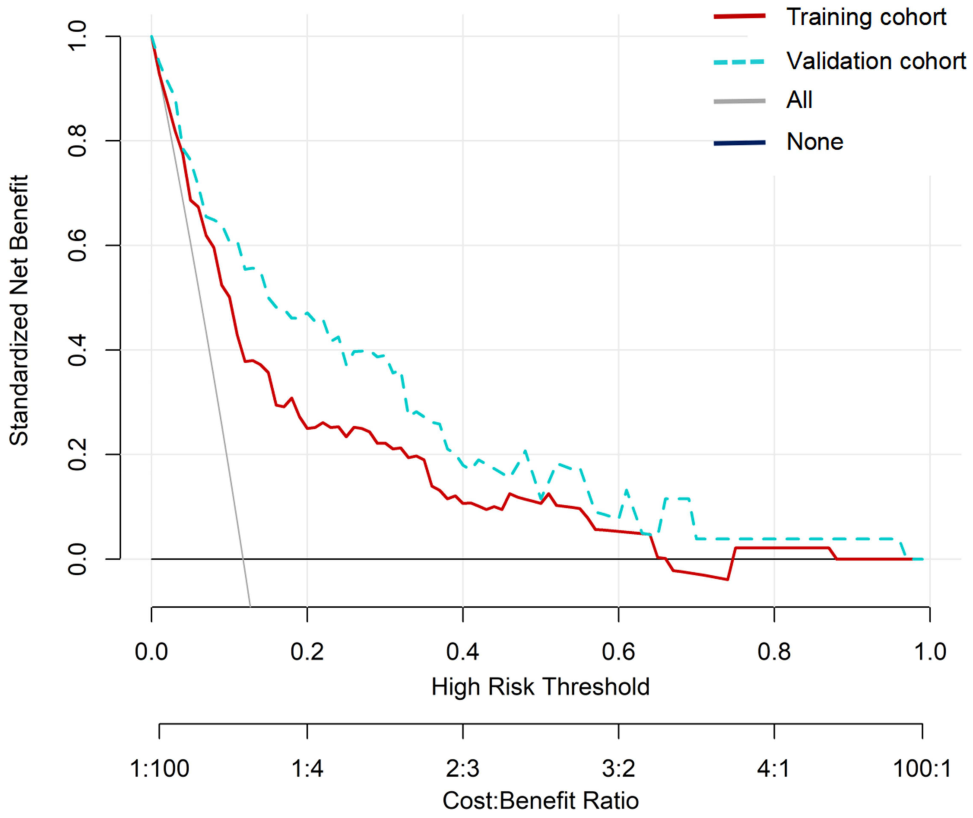

Figure 6 DCA of the nomogram. DCA: decision curve analysis.

To predict AKI caused by drug resistance to TB, we developed a nomogram model. Intended to accurately identify high-risk individuals for AKI using easily accessible examination results. The nomogram prediction model demonstrates excellent discrimination and calibration capabilities, providing net benefits almost across the entire threshold range. To our knowledge, this model is the first of its kind to accurately identify AKI caused by drug resistance to TB.

Nomogram includes five clinically relevant variables: age, hypertension, diabetes, Scr and ALB levels. The data for these variables can be easily obtained from the medical records of anti TB patients. Age is the first factor we consider. Due to changes in the structure and function of the kidneys, the rate of drug excretion through the kidneys also slows down in elderly people, resulting in a prolonged half-life of the drug. The drug accumulates in the body for a long time, leading to increasing toxicity, and the susceptibility of the kidneys to adverse drug reactions also increases accordingly.[17] At present, advanced age has been confirmed as the main risk factor for AKI and has been included in many AKI risk models.[17,18] It is reported that the incidence of renal damage in TB patients with hypertension and diabetes is high.[19,20] This is because patients with long-term hypertension are prone to ischemia, hypoxia, and hyalinization in the intima of their renal arterioles when blood pressure increases. The lumen of the affected blood vessels gradually narrows, renal blood flow decreases, glomerular and interstitial fibrosis occurs, leading to renal dysfunction.[20] In diabetes patients, hyperglycemia, advanced glycation end products and sorbitol metabolites can activate hormones or cytokines including angiotensin.[21,22] These substances can promote thickening of the glomerular basement membrane, accumulation of mesangial matrix, and abnormal changes in renal hemodynamics.[22] Therefore, anti TB treatment in the case of hypertension and diabetes will undoubtedly further aggravate kidney damage and easily induce AKI.[18–22] AKI is particularly concerning in TB patients due to its potential to interrupt prolonged drug regimens, reduce medication adherence, and increase the likelihood of treatment failure or emergence of drug-resistant strains. These factors significantly worsen prognosis and create challenges for public health control of TB.[1,22]

The results of this study indicate that Scr is a risk factor for AKI caused by drug resistance to TB, while ALB is a protective factor. Scr is one of the main methods for detecting serum creatinine in clinical practice, which is an

important indicator of kidney function. Elevated serum creatinine indicates damage to kidney function.[23] TB can lead to malnutrition. ALB is a direct indicator reflecting the nutritional status of patients.[24] Malnutrition is an important risk factor for abnormal immune system in TB patients. Low ALB is closely related to AKI, and anti TB drugs usually need to bind to ALB before they can be transported to the target site to exert their effects. If the ALB level is low, the binding between the drug and ALB is reduced. This leads to an increase in the concentration of free drugs, which in turn increases the accumulation and toxicity of drugs in the kidneys, thereby increasing the risk of AKI.[24–26] Li et al[26] also showed that malnutrition increases the risk of AKI, and both AKI and malnutrition worsen prognosis. Elevated serum creatinine reflects a reduced glomerular filtration rate, indicating pre-existing renal stress that may be exacerbated by nephrotoxic anti-TB drugs.[25] Meanwhile, hypoalbuminemia not only reflects malnutrition but also alters drug pharmacokinetics by reducing protein binding, resulting in increased levels of free, active drug molecules that can intensify renal toxicity.[23,24,26]

This study synthesized the above 5 independent risk factors and constructed a nomogram model for predicting AKI caused by drug resistance to TB. These 5 factors are clinically easy to measure and routinely available, and these predictive factors were established in a training cohort of well-characterized TB patients. By applying individual clinical indicators of patients, we can intuitively obtain scores for various influencing factors of patients, and calculate the total score through a nomogram, thereby obtaining the probability of AKI occurrence in patients and identifying high-risk patients. The nomogram model has good discriminability, with ROC values of 0.809 and 0.841 for the training cohort and validation cohort, respectively. The completion of this study will also provide a new approach for the prevention, treatment, and prediction of AKI caused by drug resistance to TB, which has important theoretical significance and practical value. This not only reduces the consumption of medical resources but also helps to avoid medical disputes, which has certain social value and clinical significance.

The nomogram model constructed in this study has good accuracy in predicting the probability of AKI caused by drug resistance to TB, but it also has certain limitations. This study is a single-center retrospective analysis with limited patient data included. If a larger sample size can be used to incorporate more case data such as drug dosage, treatment duration, and drug interactions to improve the model and validate it, it can be more practical for clinical work. 2. Our study lacks some biomarkers that may be related to AKI caused by drug resistance to TB and reflect glomerular damage, such as urinary glucose and neutrophil gelatinase-related lipocalin, including these indicators may help improve the predictive accuracy of the model. This is a single-center study. To validate the prediction model, we randomly divided the entire cohort into a training cohort and an internal validation cohort in a 7:3 ratio. However, we still lack an external validation cohort. In the future, another prospective multicenter study with a larger sample size is needed to further confirm the predictive performance of the predictive model.

Conclusion

The incidence of AKI caused by drug resistance to TB is closely related to ≥60 years old, history of hypertension, history of diabetes, and Scr and ALB levels. The nomogram model constructed based on the above can effectively predict the occurrence of AKI, providing guidance for clinical prevention and control plans. This predictive tool may assist clinicians in identifying high-risk individuals at an early stage, allowing for personalized adjustments to anti-TB therapy and enhanced renal monitoring strategies to reduce the incidence and severity of AKI.

Abbreviations

AKI, Acute kidney injury; TB, Tuberculosis; LASSO, Least Absolute Shrinkage and Selection Operator; ROC, Receiver operating characteristic; DCA, Decision curve analysis; AUC, under the curve; BMI, body mass index; IQR, interquartile range; OR, odds ratio; CI confidence interval; UA, uric acid; Scr, serum creatinine; Urea, urea nitrogen; Cys-C, cystatin C; ALB, albumin.

Data Access Statement

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics Statement

All procedures performed in the study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee(s) and the Helsinki Declaration (as revised in 2013). The requirement for informed consent was waived by the ethics committee due to the observational and retrospective nature of the study. Our study was approved by the Ethics Review Board of AFFiliated Hospital of HeBei University.

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. Mo Deng and Hui Wang have contributed equally to this work.

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Disclosure

The authors declare that there are no conflicts of interest that could be perceived as prejudicing the impartiality of the research reported.

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