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ORIGINAL RESEARCH

Construction and Validation of a Nomogram to Identify the Risk of Cavitation in Pulmonary Tuberculosis

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Background: The present study aimed to construct and validate a nomogram based on clinical metrics to identify CPTB.

Patients and Methods: The present study retrospectively recruited pulmonary tuberculosis (PTB) patients admitted to Jiashan County First People's Hospital in China from November 2018 to September 2023. PTB patients were classified into the CPTB group and the non-CPTB group based on chest computed tomography findings, and were randomly allocated to the training set (70%) and the validation cohort (30%). The training set and validation set were used to establish and validate nomogram, respectively. Multivariate logistic regression analysis (MLSA) was used to identify the independent risk factors for CPTB in patients with PTB. Statistically significant variables in the MLSA were then used to construct a nomogram predicting CPTB in patients with PTB. The receiver operating characteristic (ROC) curve, calibration curve analysis (CCA), and decision curve analysis (DCA) were used for the evaluation of the nomogram.

Results: A total of 293 PTB patients, including 208 in the training set (85 CPTB) and 85 in the validation set (33 CPTB\), were included in this study. Stepwise MLSA showed that sputum smear (≥2+), smoking(yes), glycosylated hemoglobin A1c(HbA1c), hemoglobin (HB), and systemic inflammatory response index (SIRI) were independent risk factors for the development of cavitation in patients with PTB. The nomogram identifying the high-risk CPTB patients was successfully established and showed a strong predictive capacity, with area under the curves (AUCs) of 0.875 (95% CI:0.806–0.909) and 0.848 (95% CI:0.751–0.946) in the training set and validation set respectively. In addition, the CCA and DCA corroborated the nomogram's high level of accuracy and clinical applicability within both the training and validation sets.

Conclusion: The constructed nomogram, consisting of sputum smear positivity, smoking, HbA1C, HB, and SIRI, serves as a practical and effective tool for early identification and personalized management of CPTB.

Keywords: pulmonary tuberculosis, cavitation, nomogram

Introduction

Tuberculosis (TB) is a chronic infectious disease caused by Mycobacterium tuberculosis(Mtb) infection, most of which are pulmonary TB (PTB).[1] Before the pandemic of COVID-19, TB was an infectious disease causing the leading cause of death.[2] China is a country with a TB high burden, and TB cases account for 8.4% worldwide, second only to India (26%) and Indonesia (8.5%).[3]

Cavitation, one of pathological feature in PTB, occurs through a complex interplay between the host immune response and Mycobacterium tuberculosis (Mtb) pathogenicity. As Mtb infects lung tissue, it triggers a granulomatous reaction, leading to necrosis and caseation within the granuloma. If the immune response fails to contain the infection, the liquefied necrotic material can erode into adjacent airways, forming cavities that serve as reservoirs for persistent Mtb and facilitate transmission.[4] The incidence of cavitation in PTB ranges from 29% to 87%.[5–10] There are numerous clinical values for cavitation in PTB patients such as could be used for assessing the severity.[11] Several studies suggested that cavitation represented a large Mtb burden,[12,13] which led to more transmission events.[14] In addition, cavitation

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carries a poor prognosis in PTB patients. On the one hand, cavitary PTB(CPTB) had a high risk of treatment failure,[15] relapse,[16] and drug-resistance.[17] On the other hand, the presence of cavitation increases the coughing[18–20] or hemoptysis during anti-TB treatment.[21] In conclusion, cavitation has negative implications not only for the patient associated with poor treatment outcomes, including delayed sputum culture conversion, relapse after treatment, and development of drug resistance-but is also a public health threat, since cavitation greatly increases the risk of person-to-person transmission.[5,15] Hence, it is important to identify the high-risk population of cavitation in PTB patients, which reduces the impact of cavitation on patients, resulting in improved long-term survival in PTB patients.

Chest computer tomography (CT) plays a more and more important role in identifying cavitation in PTB patients. However, in some special situations, chest CT is prohibited or inconvenient. For example, chest CT is prohibited for pregnant women because of the impact of chest CT on fetal development.[22] In addition, Chest CT is not widely used, especially in some Countries with a high burden of TB.[23] Importantly, chest CT only recognizes existing cavitation, there is no method to identify high-risk populations that have not yet occurred. Hence, a simple and convenient method to identify cavitation in PTB patients is urgently needed.

Currently, an increasing number of studies are focused on exploring risk factors for CPTB and aim to improve the management and treatment of PTB patients. Previous studies have shown that smear-positive,[24–26] high levels of CRP,[27] diabetes mellitus,[14] and higher age,[28] et al were independent risk factors for CPTB. Although too many studies have investigated the risk factors for developing cavitation in PTB patients, it is not enough due to poor sensitivity and poor specificity of single indicators. In addition, there are no research built a nomogram or model to recognize CPTB.

In recent years, with the development of analytical approaches, the construction of mathematical models based on multiple markers has been increasingly applied in the field of medicine. In the present study, we aimed to develop a nomogram to identify CPTB in PTB patients and provide a reference for the formulation of personalized management plans for PTB patients, which would be an undeniable step toward precision medicine.

Material and Methods

Participants

The present study retrospectively recruited patients with PTB admitted to Jiashan County First People's Hospital in China from November 2018 to September 2023. According to the chest computed tomography, PTB patients were included in the CPTB group and non-CPTB (NCPTB) group. Additionally, the PTB patients were randomly included in the training set (70%) and validation set (30%) using a computer-generated randomization sequence to ensure unbiased distribution. Data from the training set and validation set were used to establish and validate the nomogram respectively. Inclusion criteria were as follows:①Positive results from smear microscopy, mycobacterium tuberculosis culture, or GeneXpert MTB/RIF; ②Aged 18 years or older. Exclusion criteria were as follows:①Nontuberculous mycobacterial infection; ②Existing missing value during data collection; ③Cavitation caused by other reasons such as pulmonary malignant tumor, pulmonary abscess, pulmonary fungal infection, and so on.

Data Collection

The baseline data including demographic characteristics (age, gender, and BMI), clinical symptom (fever, cough, expectoration, hemoptysis, night sweat, and so on), smoking history, drinking history, treatment history, underlying disease[hypertension (HTN), diabetes mellitus (DM), chronic liver disease (CLD), chronic kidney disease (CKD) and chronic heart disease (CHD)], radiological features (cavitation or not), and laboratory parameters were retrospectively extracted from individual medical records. A cavity was defined as a gas-filled space within a nodule, mass, or area of consolidation, visible on chest CT scans. The Systemic Inflammatory Response Index (SIRI) was calculated using the formula: SIRI = (neutrophil count × monocyte count) / lymphocyte count. In addition, we calculated the combined indexes, and detailed information on them is shown in Supplementary Table 1.

Statistical Analysis

Categorical variables, represented by n%, were analyzed with the chi-square test or Fisher's exact test, and continuous variables, represented by median (Quartile range, IQR), were compared by the Mann–Whitney *U*-test. Continuous predictor variables were then scrutinized for the linearity assumption in the logit, utilizing the Box-Tidwell procedure. Necessary transformations were applied to variables that violated this assumption to conform to linearity requirements. Multivariate Logistic regression analysis (MLSA) was used to search the independent risk factors for CPTB in patients with PTB. Furthermore, we examined the presence of multicollinearity among continuous predictors to avoid biased estimates that could compromise the model. This was accomplished by evaluating the tolerance values and the Variance Inflation Factor (VIF) for each variable under consideration for inclusion in the multivariate logistic regression model. Variables with VIF values exceeding the threshold of 10, suggestive of significant multicollinearity, were subjected to further scrutiny and were potentially excluded from the model to ensure its robustness. And finally, the variables in MLSA with statistically significant differences were used to build a nomogram predicting CPTB in PTB patients. The receiver operating characteristic (ROC) curve, calibration curve analysis (CCA) and decision curve analysis (DCA) were used for the evaluation of the nomogram. Furthermore, the validation of the nomogram was conducted in the validation set.

All statistical analyses were performed with SPSS26.0 and R software (4.2.1 version). All P-values were based on a two-sided hypothesis, with $P<0.05$ indicating statistical significance.

Results

Patient Characteristics

A total of 293 patients with PTB were recruited in this study. The cohort was divided into a training set (N=208, including 85 patients with CPTB) and a validation set (N=85, including 33 CPTB patients), following a 7:3 ratio. As shown in Supplementary Tables 2 and 3, no significant differences were found between the training and validation sets in clinical characteristics and laboratory parameters ($P>0.05$), suggesting that the data from both sets are comparable and suitable for mutual validation.

Characteristics Selection in the Training Set

Table 1 demonstrates that there were no significant differences between the CPTB and NCPTB groups in the training set regarding age, gender, fever, cough, sputum, night sweats, chest pain, dyspnea, loss of appetite, fatigue, drinking history, hypertension (HTN), chronic liver disease (CLD), chronic kidney disease (CKD), coronary heart disease (CHD), T-spot, C-reactive protein (CRP), procalcitonin (PCT), erythrocyte sedimentation rate (ESR), total bilirubin (TBil), albumin (ALB), gamma-glutamyl transferase (r-GT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), uric acid, serum creatinine (Scr), total cholesterol (TC), triglycerides (TG), creatine kinase (CK), cystatin C (Cys), prothrombin time (PT), activated partial thromboplastin time (APTT), and derived neutrophil-to-lymphocyte ratio (dNLR) ($P>0.05$). However, significant differences were observed in BMI, hemoptysis, weight loss, smoking history, treatment history, diabetes mellitus (DM), sputum smear, GeneXpert MTB/RIF, white blood cell count (WBC), lymphocyte count, monocyte count, neutrophil count, hemoglobin (HB), platelet count (PLT), ESR, glycated hemoglobin (HbA1c), alanine aminotransferase (ALT), aspartate aminotransferase (AST), fasting blood glucose (FBG), fibrinogen (FIB), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), systemic inflammatory response index (SIRI), and hemoglobin-albumin-lymphocyte-platelet (HALP) index ($P<0.05$).

Risk Factors for CPTB in Patients with PTB in the Training Set

The 26 variables that were statistically significant in the analysis of differences between groups were further included in the multivariate analysis. Stepwise multivariate logistic regression analysis identified sputum smear ≥2+ (OR(95% CI): 1.124(1.058–1.264), $P<0.001$), smoking history (OR(95% CI): 1.240(1.113–1.510), $P<0.001$), SIRI (OR(95% CI): 1.176 (1.046–1.323), $P=0.007$), HB (OR(95% CI): 0.972(0.952–0.993), $P=0.009$), and HbA1c (OR(95% CI): 1.306(1.074–1.587), $P=0.007$) as independent risk factors for CPTB in patients with PTB (Table 2).

Table 1 Characteristics Selection in Training Set

Variables	CPTB (n=85)	NCPTB (n=123)	*P*
Age (IQR)	56.00 (32.00–73.00)	62.00 (43.00–74.00)	0.294
Gender (Male)	66 (77.6)	88 (71.5)	0.324
BMI (IQR)	18.939 (17.439–21.563)	20.069 (18.026–22.985)	0.048
Clinical symptoms			
Fever	24 (28.2)	43 (35.0)	0.308
Cough	75 (88.2)	112 (91.1)	0.507
Sputum	67 (78.8)	90 (73.2)	0.352
Hemoptysis	18 (21.2)	12 (9.8)	0.021
Night sweats	10 (11.8)	10 (8.1)	0.382
Weight loss	32 (37.6)	28 (22.8)	0.020
Chest pain	11 (12.9)	20 (16.3)	0.509
Dyspnea	17 (20.0)	30 (24.4)	0.457
Loss of appetite	15 (17.6)	19 (15.4)	0.673
Fatigue	18 (21.2)	22 (17.9)	0.554
Smoking history	47 (55.3)	40 (32.5)	0.001
Drinking history	23 (27.1)	27 (22.0)	0.397
Treatment history			0.003
Initial treatment	69 (81.2)	116 (94.3)	
Re-treatment	16 (18.8)	7 (5.7)	
Underlying disease			
DM	24 (28.2)	15 (12.2)	0.004
HTN	18 (21.2)	36 (29.3)	0.191
CLD	16 (18.8)	36 (29.3)	0.087
CKD	5 (5.9)	5 (4.1)	0.743
CHD	7 (8.2)	17 (13.8)	0.215
HIV	0 (0.0)	0 (0.0)	-
HBV	7 (8.2)	12 (9.8)	0.708
HCV	1 (0.8)	1 (1.2)	0.792
Sputum smear			
≤1+	29 (34.1)	100 (81.3)	<0.001
≥2+	56 (65.9)	23 (18.7)	
GeneXpert MTB/RIF			
Negative or weakly positive	34 (40.0)	84 (68.3)	<0.001
>weakly positive	51 (60.0)	39 (31.7)	
T-spot	147.150 (58.290–350.600)	128.690 (47.040–368.640)	0.605
WBC (IQR)	7.100 (5.200–9.900)	6.300 (4.300–8.300)	0.008
Lymphocyte count (IQR)	1.000 (0.700–1.300)	1.200 (0.800–1.600)	0.018
Monocyte count (IQR)	0.580 (0.365–0.750)	0.470 (0.300–0.620)	0.001
Neutrophil count (IQR)	5.000 (3.600–7.100)	4.000 (2.800–5.900)	0.002
HB (IQR)	122.00 (107.00–131.00)	126.00 (118.00–138.00)	0.001
PLT (IQR)	253.00 (195.00–318.00)	215.00 (140.00–271.00)	0.000
CRP (IQR)	28.790 (11.115–64.505)	17.520 (2.860–76.020)	0.098
PCT (IQR)	0.050 (0.020–0.100)	0.050 (0.020–0.100)	0.581
ESR (IQR)	60.00 (35.50–79.00)	46.00 (19.00–64.00)	0.001
HbA1c (IQR)	5.800 (5.600–7.700)	5.700 (5.500–6.000)	0.030
TBil (IQR)	11.200 (8.900–15.250)	10.400 (7.88–15.100)	0.464
ALB (IQR)	33.700 (31.000–37.700)	35.800 (31.900–39.800)	0.169
ALT (IQR)	11.00 (8.00–18.50)	13.00 (10.00–22.00)	0.035
AST (IQR)	17.00 (13.00–22.00)	19.00 (15.00–25.00)	0.029
r-GT (IQR)	28.00 (18.50–50.00)	26.00 (17.00–49.00)	0.372

(*Continued*)

Table 1 (Continued).

Variables	CPTB (n=85)	NCPTB (n=123)	*P*
ALP (IQR)	80.00 (70.00–95.50)	76.00 (61.00–93.00)	0.074
LDH (IQR)	202.00 (172.50–262.00)	199.00 (169.00–241.00)	0.525
Uric acid (IQR)	314.00 (226.50–410.00)	271.00 (211.00–351.00)	0.138
FBG (IQR)	5.110 (4.350–7.690)	4.700 (4.150–5.570)	0.019
Scr (IQR)	66.00 (57.50–71.50)	65.00 (58.00–74.00)	0.875
TC (IQR)	3.740 (3.150–4.175)	3.760 (3.200–4.550)	0.250
TG (IQR)	0.820 (0.625–1.165)	0.860 (0.680–1.170)	0.640
CK (IQR)	47.00 (29.50–75.00)	51.00 (35.00–78.00)	0.422
Cys (IQR)	12.200 (9.000–15.350)	12.000 (8.500–15.000)	0.609
PT (IQR)	11.800 (11.250–12.650)	11.800 (11.100–12.700)	0.946
FIB (IQR)	4.233 (3.395–5.353)	3.711 (2.765–5.060)	0.022
APTT (IQR)	30.200 (27.700–32.850)	30.200 (27.300–32.100)	0.497
Combined indexes			
dNLR (IQR)	2.706 (2.071–4.180)	2.571 (1.625–3.643)	0.077
NLR (IQR)	5.330 (3.535–7.525)	3.600 (2.080–5.630)	0.000
MLR (IQR)	0.570 (0.350–0.795)	0.370 (0.230–0.550)	0.000
PLR (IQR)	260.000 (176.915–409.210)	171.430 (110.000–278.330)	0.000
SII (IQR)	1322.250 (726.820–2239.500)	641.450 (390.920–1427.620)	0.000
SIRI (IQR)	2.810 (1.720–5.330)	1.290 (0.800–2.950)	0.000
HALP (IQR)	14.660 (8.500–25.135)	27.490 (14.490–45.770)	0.000

Abbreviations: CLD, chronic liver disease; CKD, chronic kidney disease; CHD, chronic heart disease; HTN, hypertension; DM, diabetes mellitus; CRP, c-reactive protein; PCT, procalcitonin; ESR, erythrocyte sedimentation rate; TBil, total bilirubin; ALB, albumin; r-GT, gamma-glutamyl transferase; ALP, alkaline phosphatase; LDH, lactate dehydrogenase; Uric acid, uric acid; Scr, serum creatinine; TC, total cholesterol; TG, triglycerides; CK, creatine kinase; Cys, cystatin C; PT, prothrombin time; APTT, activated partial thromboplastin time; dNLR, derived neutrophil to lymphocyte ratio; BMI, body mass index; Hemoptysis, hemoptysis; WBC, white blood cell count; HB, hemoglobin; PLT, platelet count; HbA1c, glycated haemoglobin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; FBG, fasting blood glucose; FIB, fibrinogen; NLR, neutrophil to lymphocyte ratio; MLR, monocyte to lymphocyte ratio; PLR, platelet to lymphocyte ratio; SII, systemic immune-inflammation index; SIRI, systemic inflammatory response index; HALP, hemoglobin-albumin-lymphocyte-platelet score.

Table 2 Stepwise Multivariate Logistic Analysis in the Training Set

Variable	β	OR (95% CI)	P
Sputum smear (≥2+)	−2.085	1.124 (1.058–1.264)	<0.001
Smoking (Yes)	−1.427	1.240 (1.113–1.510)	<0.001
SIRI	0.162	1.176 (1.046–1.323)	0.007
HB	−0.028	0.972 (0.952–0.993)	0.009
HbA1c	0.267	1.306 (1.074–1.587)	0.007

Abbreviations: SIRI, systemic inflammatory response index; HB, hemoglobin; HbA1c, glycated haemoglobin.

Construction, Evaluation, and Validation of the Nomogram

A nomogram for predicting CPTB in patients with PTB was developed based on five independent risk factors (Figure 1). Each factor was assigned a specific score, contributing to the overall prediction model. The nomogram's predictive performance was evaluated using the receiver operating characteristic (ROC) curve, which demonstrated high accuracy with an area under the curve (AUC) of 0.875 (95% CI: 0.806–0.909) in the training set (Figure 2A) and 0.848 (95% CI: 0.751–0.946) in the validation set (Figure 2B), respectively, which manifested the favorable predictive ability of the CPTB nomogram. In addition, the CCA showed a high consistency between predicted and actual CPTB occurrences in both sets (Figure 3). The nomogram was constructed using multivariate logistic regression analysis, identifying five independent risk factors: sputum smear (≥2+), smoking history, SIRI, Hb, and HbA1c. The superiority and clinical net benefits of the CPTB nomogram were evaluated by

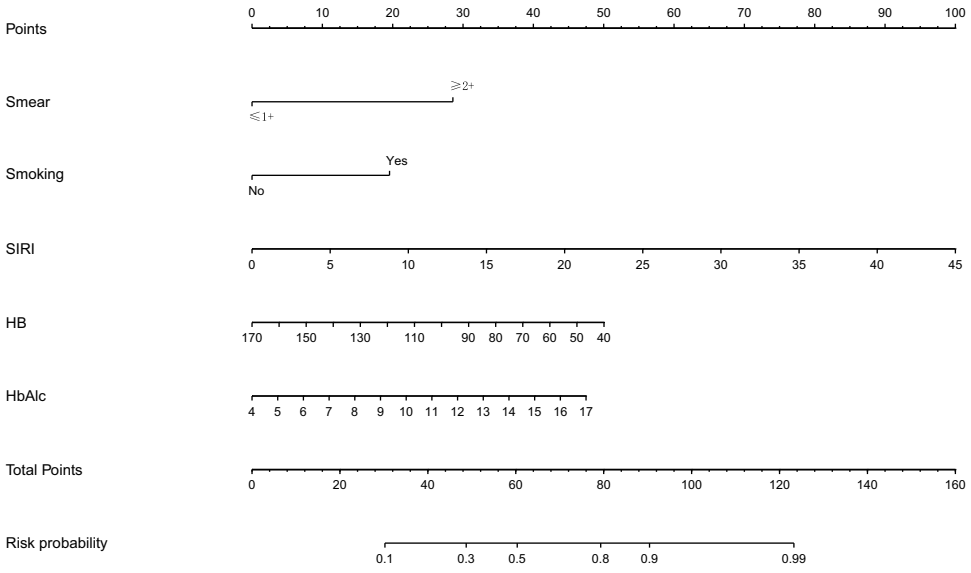

Figure 1 Nomogram to predict CPTB in patients with PTB.

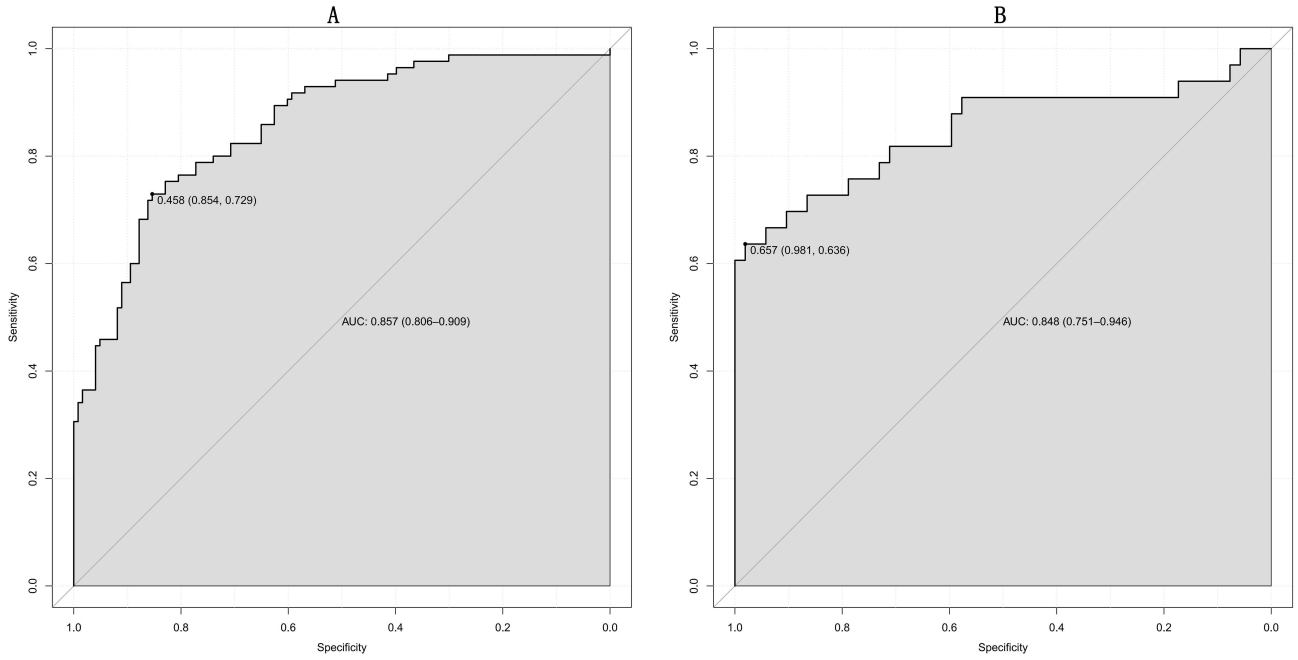

Figure 2 The AUCs of nomogram in the training set (**A**) and validation set (**B**).

DCA. Compared to the "None" strategy, the CPTB nomogram exhibited a wide range of threshold probabilities in both sets, which suggested that the CPTB nomogram provides a better prediction of CPTB in patients with PTB (Figure 4).

Discussion

Cavitary pulmonary tuberculosis (CPTB) presents a significant clinical challenge due to its association with treatment failure, relapse, drug-resistance, and frequent clinical symptoms.[29] Hence, constructing a nomogram for the early identification of CPTB is crucial for the personalized management of PTB patients.

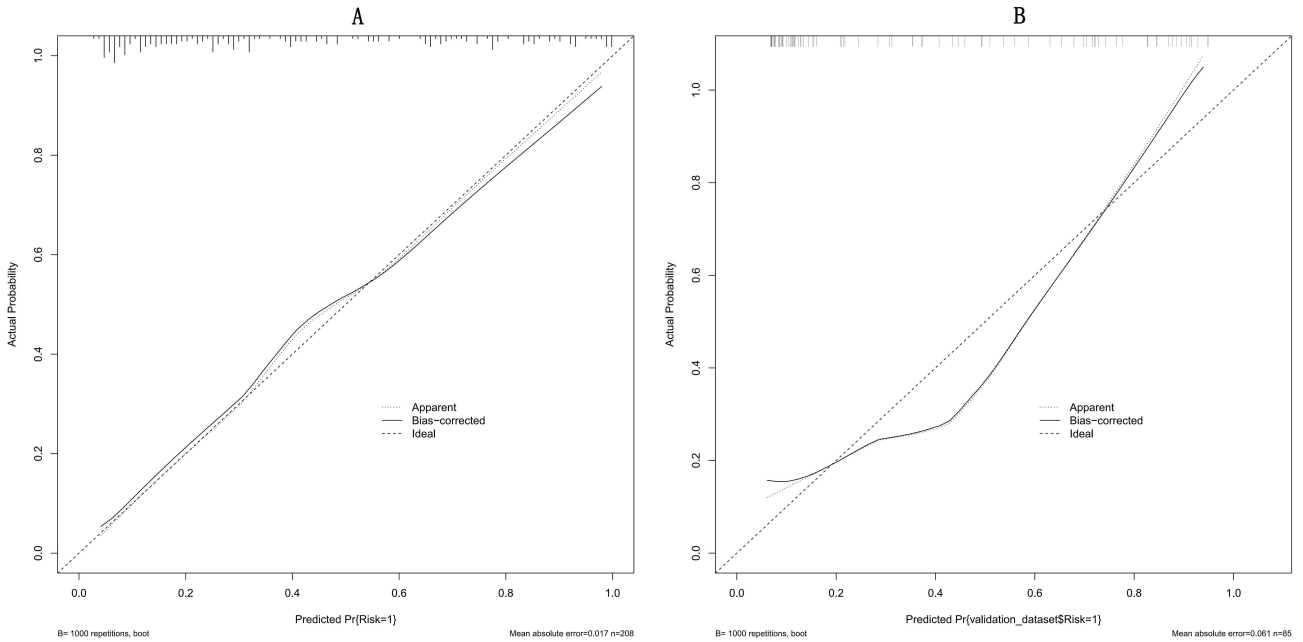

Figure 3 Calibration curves of the nomogram in the training set (**A**) and validation set (**B**).

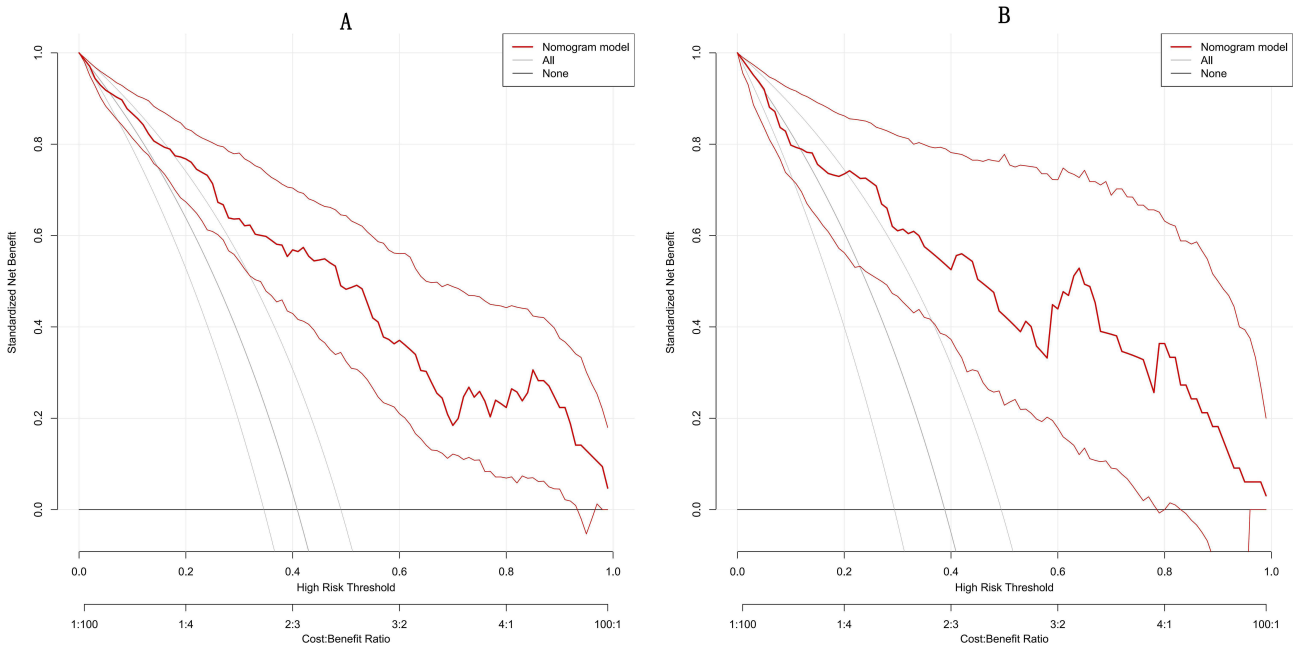

Figure 4 DCA of the nomogram in the training set (**A**) and validation set (**B**).

The nomogram developed in this study exhibited robust predictive performance, with area under the curve (AUC) values of 0.875 in the training set and 0.848 in the validation set. Calibration curve analysis and decision curve analysis further validated the nomogram's accuracy and clinical utility, demonstrating its potential as a practical tool for early identification of CPTB. Previous models have used clinical, radiological, and laboratory data to predict multidrug-resistant TB (MDR-TB), TB relapse and treatment outcome.[30–32] hese models, while useful, did not specifically address the risk of cavitation, which is critical for understanding TB progression and management.

The present study showed that Sputum smear ≥2+, Smoking history, SIRI, HB, and HbA1c were the independent influence factors for CPTB in patients with PTB. These findings are consistent with previous research, which has demonstrated that higher sputum smear levels correlate with increased cavitation risk in PTB patients.[26] A histological study in PTB patients undergoing surgery found a large number of Mycobacterium tuberculosis (Mtb) organisms on the surface of cavitation, further supporting this association.[33] The selective loss of CD4+ and CD8+ T cells in CPTB patients may prevent direct interaction between T cells and macrophages, facilitating Mtb growth in cavitation.[34] Additionally, the correlation between smoking and PTB has been reported.[35] Several studies found that PTB patients with smoking history had a higher risk of cavitation,[36,37] which were consistent with the present study. Cigarette smoke leads to documented ciliary dysfunction, diminished immune response, and a defective macrophage immune response could explain the Results.[38]

Our nomogram incorporates SIRI, a novel marker of systemic inflammation, which has been used in the diagnosis and prognosis of various cancers and infectious diseases.[39] The inclusion of SIRI reflects the broader immune response to Mtb infection, providing a more comprehensive picture of the inflammatory state that contributes to cavitation. To our knowledge, there are no reports about the relationship between cavitation and SIRI in PTB patients. Elevated SIRI levels indicate a heightened inflammatory state, which our study found to be significantly associated with cavitation. This finding aligns with research in other inflammatory conditions, where SIRI has been shown to predict outcomes.[40] The novel association between SIRI and CPTB underscores the importance of systemic inflammation in the pathogenesis of cavitary lesions in PTB.

Anemia in TB is often multifactorial, resulting from chronic disease inflammation, nutritional deficiencies, and direct bone marrow suppression by Mtb.[41] According to several survey reports, the prevalence of anemia among TB patients is between 32% and 86%.[42] Retrospective studies have shown that anemia is positively correlated with Mtb burden, and hence, PTB patients with anemia are more likely to form cavitation.[43] Our study identified low hemoglobin levels as an independent risk factor for CPTB, which was consistent with the previous findings.[44] Several previous studies have shown that DM is associated with the pathogenesis of pulmonary tuberculosis, possibly mediated by multiple pro-inflammatory cytokine mechanisms.[45] The role of HbA1c, a marker of long-term glycemic control, in predicting CPTB highlights the need for rigorous blood sugar management in TB patients. Studies have shown that poor glycemic control is associated with more severe radiographic manifestations and slower sputum culture conversion.[46–49] Our findings reinforce the necessity of integrated diabetes management in TB care to reduce cavitation risk.

The nomogram developed in this study offers several advantages. First, the prediction nomogram model not only explains the mechanism of cavitation occurrence but also reflects the clinical characteristics of CPTB, thus providing targeted guidance to PTB patients. For instance, we can persuade smoking cessation, optimize hyper-inflammatory states, improve anemia status, and control blood sugar, thereby minimizing the incidence rate of cavitation in PTB patients. Secondly, variables in our prediction nomogram model are accessible in most hospitals, even primary hospitals, and it's of great significance for poor areas, especially areas with high TB burden. Last but not least, our prediction nomogram model has a remarkable performance power in predicting CPTB. When patients are at greater risk of cavitation, they should be advised to go to a high-level hospital for further treatment, which will reduce the incidence rate of cavitation, time cost, and financial loss, and improve the prognosis.

However, our study has limitations, including its retrospective design and single-center setting, which may limit the generalizability of the findings. Additionally, the relatively small sample size necessitates further research with larger, multicenter cohorts to validate and refine the nomogram. Future studies should explore the nomogram's applicability to special populations, such as pregnant women, and investigate the integration of additional biomarkers or advanced imaging techniques to enhance predictive accuracy.

Conclusion

Our study is the first to establish a nomogram for predicting cavitation risk in PTB patients, incorporating novel and traditional risk factors such as SIRI, smoking history, HbA1c, hemoglobin, and sputum smear positivity. This tool provides valuable insights for early identification and personalized management of CPTB, potentially improving patient outcomes and reducing the public health impact of TB.

Ethics Statement

The Declaration of Helsinki was followed in the conduct of this study. The Ethics Committee of Jiashan County First People's Hospital approved the study protocol (2023-013). To maintain privacy, no personally identifiable information was provided. Due to the retrospective nature of this study and all patient identities were anonymous Participants, the present study obtained the exemption of informed consent from The Ethics Committee of Jiashan County First People's Hospital. In addition, during the implementation of this study, the present study was conducted according to the national legislation and institutional requirements.

Author Contributions

All authors made a significant contribution to the work reported, whether 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.

Disclosure

The authors declare that they have no competing interests in this work.

References

1. Li H, Ge M, Zhang M. Spatio-temporal distribution of tuberculosis and the effects of environmental factors in China. *BMC Infect Dis*. 2022;22(1):565. doi:10.1186/s12879-022-07539-4
2. Yu Q, Yan J, Tian S, et al. A scoring system developed from a nomogram to differentiate active pulmonary tuberculosis from inactive pulmonary tuberculosis. *Front Cell Infect Microbiol*. 2022;12:947954. doi:10.3389/fcimb.2022.947954
3. Zhang Q, Song W, Liu S, et al. An Ecological Study of Tuberculosis Incidence in China, From 2002 to 2018. *Front Public Health*. 2021;9:766362. doi:10.3389/fpubh.2021.766362
4. Furin J, Cox H, Pai M. Tuberculosis. *Lancet*. 2019;393(10181):1642–1656. doi:10.1016/S0140-6736(19)30308-3
5. Palaci M, Dietze R, Hadad DJ, et al. Cavitary disease and quantitative sputum bacillary load in cases of pulmonary tuberculosis. *J Clin Microbiol*. 2007;45(12):4064–4066. doi:10.1128/JCM.01780-07
6. Wilcke JT, Askgaard DS, Nybo Jensen B, Døssing M. Radiographic spectrum of adult pulmonary tuberculosis in a developed country. *Respir Med*. 1998;92(3):493–497. doi:10.1016/s0954-6111(98)90297-9
7. Gomes M, Saad Júnior R, Stirbulov R. Pulmonary tuberculosis: relationship between sputum bacilloscopy and radiological lesions. *Rev Inst Med Trop Sao Paulo*. 2003;45(5):275–281. doi:10.1590/s0036-46652003000500007
8. Andreu J, Cáceres J, Pallisa E, Martinez-Rodriguez M. Radiological manifestations of pulmonary tuberculosis. *Eur J Radiol*. 2004;51(2):139–149. doi:10.1016/j.ejrad.2004.03.009
9. Woodring JH, Vandiviere HM, Fried AM, Dillon ML, Williams TD, Melvin IG. Update: the radiographic features of pulmonary tuberculosis. *AJR Am J Roentgenol*. 1986;146(3):497–506. doi:10.2214/ajr.146.3.497
10. Miller WT, Miller WT. Tuberculosis in the normal host: radiological findings. *Semin Roentgenol*. 1993;28(2):109–118. doi:10.1016/s0037-198x(05)80100-2
11. Li Y, Wang B, Wen L, et al. Machine learning and radiomics for the prediction of multidrug resistance in cavitary pulmonary tuberculosis: a multicentre study. *Eur Radiol*. 2023;33(1). doi:10.1007/s00330-022-08997-9
12. Murthy SE, Chatterjee F, Crook A, et al. Pretreatment chest x-ray severity and its relation to bacterial burden in smear positive pulmonary tuberculosis. *BMC Med*. 2018;16(1):73. doi:10.1186/s12916-018-1053-3
13. Kim HW, Shin AY, Ha JH, Ahn JH, Kang HS, Kim JS. Effect of serum isoniazid level on treatment outcomes among tuberculosis patients with slow response - A retrospective cohort study. *J Infect Chemother*. 2021;27(11):1555–1561. doi:10.1016/j.jiac.2021.06.016
14. Tong X, Wang D, Wang H, et al. Clinical features in pulmonary tuberculosis patients combined with diabetes mellitus in China: an observational study. *Clin Respir J*. 2021;15(9):1012–1018. doi:10.1111/crj.13405
15. Benator D, Bhattacharya M, Bozeman L, et al. Rifapentine and isoniazid once a week versus rifampicin and isoniazid twice a week for treatment of drug-susceptible pulmonary tuberculosis in HIV-negative patients: a randomised clinical trial. *Lancet*. 2002;360(9332):528–534. doi:10.1016/s0140-6736(02)09742-8
16. Kriel M, Lotz JW, Kidd M, Walzl G. Evaluation of a radiological severity score to predict treatment outcome in adults with pulmonary tuberculosis. *Int J Tuberc Lung Dis*. 2015;19(11):1354–1360. doi:10.5588/ijtld.15.0098
17. Anley DT, Akalu TY, Merid MW, et al. Development and validation of a nomogram for the prediction of late culture conversion among multi-drug resistant tuberculosis patients in North West Ethiopia: an application of prediction modelling. *PLoS One*. 2022;17(8):e0272877. doi:10.1371/journal.pone.0272877
18. Tao NN, Li YF, Song WM, et al. Risk factors for drug-resistant tuberculosis, the association between comorbidity status and drug-resistant patterns: a retrospective study of previously treated pulmonary tuberculosis in Shandong, China, during 2004–2019. *BMJ Open*. 2021;11(6):e044349. doi:10.1136/bmjopen-2020-044349
19. Proaño A, Bui DP, López JW, et al. Cough frequency during treatment associated with baseline cavitary volume and proximity to the airway in pulmonary TB. *Chest*. 2018;153(6):1358–1367. doi:10.1016/j.chest.2018.03.006

20. Hales CM, Heilig CM, Chaisson R, et al. The association between symptoms and microbiologically defined response to tuberculosis treatment. *Ann Am Thorac Soc*. 2013;10(1):18–25. doi:10.1513/AnnalsATS.201207-038OC
21. Brik A, Salem AM, Shoukry A, Shouman W. Surgery for hemoptysis in various pulmonary tuberculous lesions: a prospective study. *Interact Cardiovasc Thorac Surg*. 2011;13(3):276–279. doi:10.1510/icvts.2011.270991
22. Raveendran A, Keepanasseril A, Balu RK, Shetty A, Chetty M. Tuberculosis in pregnancy. *Obstet Gynaecol*. 2023;25(3):175–185. doi:10.1111/tog.12888
23. Ruperez M, Shanaube K, Mureithi L, et al. Use of point-of-care C-reactive protein testing for screening of tuberculosis in the community in high-burden settings: a prospective, cross-sectional study in Zambia and South Africa. *Lancet Glob Health*. 2023;11(5):e704–e714. doi:10.1016/S2214-109X(23)00113-4
24. Kim JH, Kim MJ, Ham SY. Clinical characteristics and chest computed tomography findings of smear-positive and smear-negative pulmonary tuberculosis in hospitalized adult patients. *Medicine*. 2019;98(34):e16921. doi:10.1097/MD.0000000000016921
25. Carlesi E, Orlandi M, Mencarini J, et al. How radiology can help pulmonary tuberculosis diagnosis: analysis of 49 patients. *Radiol Med*. 2019;124(9):838–845. doi:10.1007/s11547-019-01040-w
26. Gopalan N, Srinivasalu VA, Chinnayan P, et al. Predictors of unfavorable responses to therapy in rifampicin-sensitive pulmonary tuberculosis using an integrated approach of radiological presentation and sputum mycobacterial burden. *PLoS One*. 2021;16(9):e0257647. doi:10.1371/journal.pone.0257647
27. Goto A, Komiya K, Kan T, et al. Factors associated with atypical radiological findings of pulmonary tuberculosis. *PLoS One*. 2019;14(7):e0220346. doi:10.1371/journal.pone.0220346
28. Heo EY, Chun EJ, Lee CH, et al. Radiographic improvement and its predictors in patients with pulmonary tuberculosis. *Int J Infect Dis*. 2009;13(6):e371–e376. doi:10.1016/j.ijid.2009.01.007
29. Urbanowski ME, Ordonez AA, Ruiz-Bedoya CA, Jain SK, Bishai WR. Cavitary tuberculosis: the gateway of disease transmission. *Lancet Infect Dis*. 2020;20(6):e117–e128. doi:10.1016/S1473-3099(20)30148-1
30. Wang S, Tu J. Nomogram to predict multidrug-resistant tuberculosis. *Ann Clin Microbiol Antimicrob*. 2020;19(1):27. doi:10.1186/s12941-020-00369-9
31. Yan J, Luo H, Nie Q, Hu S, Yu Q, Wang X. A scoring system based on laboratory parameters and clinical features to predict unfavorable treatment outcomes in multidrug- and rifampicin-resistant tuberculosis patients. *Infect Drug Resist*. 2023;16:225–237. doi:10.2147/IDR.S397304
32. Li D, Tang SY, Lei S, Xie HB, Li LQ. A nomogram for predicting mortality of patients initially diagnosed with primary pulmonary tuberculosis in Hunan province, China: a retrospective study. *Front Cell Infect Microbiol*. 2023;13:1179369. doi:10.3389/fcimb.2023.1179369
33. Liu X, Hou XF, Gao L, et al. Indicators for prediction of Mycobacterium tuberculosis positivity detected with bronchoalveolar lavage fluid. *Infect Dis Poverty*. 2018;7(1):22. doi:10.1186/s40249-018-0403-x
34. Kaplan G, Post FA, Moreira AL, et al. Mycobacterium tuberculosis growth at the cavity surface: a microenvironment with failed immunity. *Infect Immun*. 2003;71(12):7099–7108. doi:10.1128/IAI.71.12.7099-7108.2003
35. Bai KJ, Lee JJ, Chien ST, Suk CW, Chiang CY. The influence of smoking on pulmonary tuberculosis in diabetic and non-diabetic patients. *PLoS One*. 2016;11(6):e0156677. doi:10.1371/journal.pone.0156677
36. Leung CC, Li T, Lam TH, et al. Smoking and tuberculosis among the elderly in Hong Kong. *Am J Respir Crit Care Med*. 2004;170(9):1027–1033. doi:10.1164/rccm.200404-512OC
37. Altet-Gômez MN, Alcaide J, Godoy P, Romero MA, Hernández Del Rey I. Clinical and epidemiological aspects of smoking and tuberculosis: a study of 13,038 cases. *Int J Tuberc Lung Dis*. 2005;9(4):430–436.
38. Silva DR, Muñoz-Torrico M, Duarte R, et al. Risk factors for tuberculosis: diabetes, smoking, alcohol use, and the use of other drugs. *J Bras Pneumol*. 2018;44(2):145–152. doi:10.1590/s1806-37562017000000443
39. Chai B, Wu D, Fu N, et al. Evaluation of prognostic inflammatory and systemic inflammatory response indices in auxiliary diagnosis of bacteria-negative pulmonary tuberculosis: a diagnostic accuracy study. *Medicine*. 2023;102(12). doi:10.1097/MD.0000000000033372
40. Suryana K, Dharmesti NWW, Rai IBN. High pretreatment level of neutrophil to lymphocyte ratio, monocyte to lymphocyte ratio and other factors associated with delayed sputum conversion in patients with pulmonary tuberculosis. *Infect Drug Resist*. 2022;15:5455–5462. doi:10.2147/IDR.S380166
41. Calderon RI, Arriaga MB, Lopez K, et al. High prevalence and heterogeneity of Dysglycemia in patients with tuberculosis from Peru: a prospective cohort study. *BMC Infect Dis*. 2019;19(1):799. doi:10.1186/s12879-019-4416-2
42. Gil-Santana L, Cruz LAB, Arriaga MB, et al. Tuberculosis-associated anemia is linked to a distinct inflammatory profile that persists after initiation of antitubercular therapy. *Sci Rep*. 2019;9(1):1381. doi:10.1038/s41598-018-37860-5
43. Luo M, Liu M, Wu X, et al. Impact of anemia on prognosis in tuberculosis patients. *Ann Transl Med*. 2022;10(6):329. doi:10.21037/atm-22-679
44. Ashenafi S, Bekele A, Aseffa G, et al. Anemia is a strong predictor of wasting, disease severity, and progression, in Clinical Tuberculosis (TB). *Nutrients*. 2022;14(16):3318. doi:10.3390/nu14163318
45. Koo HK, Min J, Kim HW, et al. Clinical profiles and prediction of treatment failure in patients with tuberculosis. *BMC Infect Dis*. 2020;20:1–7. doi:10.21203/rs.3.rs-27791/v1
46. Yoon YS, Jung JW, Jeon EJ, et al. The effect of diabetes control status on treatment response in pulmonary tuberculosis: a prospective study. *Thorax*. 2017;72(3):263–270. doi:10.1136/thoraxjnl-2015-207686
47. Bezerra AL, Moreira A da SR, Isidoro-Gonçalves L, et al. Clinical, laboratory, and radiographic aspects of patients with pulmonary tuberculosis and dysglycemia and tuberculosis treatment outcomes. *J Bras Pneumol*. 2022;48(6):e20210505. doi:10.36416/1806-3756/e20210505
48. Xia LL, Li SF, Shao K, Zhang X, Huang S. The correlation between CT features and glycosylated hemoglobin level in patients with T2DM complicated with primary pulmonary tuberculosis. *Infect Drug Resist*. 2018;11:187–193. doi:10.2147/IDR.S146741
49. Chiang CY, Lee JJ, Chien ST, et al. Glycemic control and radiographic manifestations of tuberculosis in diabetic patients. *PLoS One*. 2014;9(4):e93397. doi:10.1371/journal.pone.0093397