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ORIGINAL RESEARCH

Investigation of Risk Factors and Development of Clinical Prediction Model for Nocardiosis in Lung Transplant Recipients

Hengyu Zhao[1,*], Zhibin Xu[1,*], Xiaohua Wang[1,*], Yu Xu[1], Yi Lu[1], Jiaqi Chen[1], Qiaoyu Ye[1], Xuan Li[1], Yanhua Wen[2], Chunrong Ju[1]

[1]State Key Laboratory of Respiratory Disease, National Clinical Research Center for Respiratory Disease, Guangzhou Institute of Respiratory Health, First Affiliated Hospital of Guangzhou Medical University, Guangzhou, People's Republic of China; [2]Department of Scientific Affaires, Hugobiotech Co., Ltd., Beijing, People's Republic of China

*These authors contributed equally to this work

Correspondence: Chunrong Ju, Email juchunrong@gzhmu.edu.cn

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Purpose: Nocardiosis is an opportunistic infection in lung transplant recipients but is often misdiagnosed or overlooked. This study aimed to identify risk factors and develop an effective predictive model for nocardiosis in this population.

Patients and Methods: This single-center retrospective study analyzed 679 lung transplant recipients from January 1, 2015, to July 9, 2024. Twenty patients with nocardiosis were compared with 40 matched controls. Feature selection was performed using LASSO regression, and logistic regression identified risk factors. Model performance was assessed via ROC curves, calibration curves, and decision curve analysis.

Results: Decreased $CD4^+$ T cells, elevated $CD8^+$ T cells, and reduced IgA levels were significantly associated with nocardiosis ($P < 0.05$). The model incorporating these factors demonstrated strong predictive ability with an area under the ROC curve of 0.955.

Conclusion: $CD4^+$ T cells, $CD8^+$ T cells, and IgA are independent risk factors for nocardiosis post-lung transplantation. The developed model effectively distinguishes nocardiosis cases, aiding early clinical identification.

Keywords: lung transplantation, organ transplantation, *Nocardia*, nocardiosis, prediction model

Introduction

Lung transplantation is the only effective treatment for end-stage lung disease, significantly extending patients' survival time and improving their quality of life.[1] However, post-transplant infectious complications significantly reduce recipients' survival rates and quality of life. Nocardiosis, an opportunistic infectious disease caused by *Nocardia* infection, affects immunocompromised individuals.[2] Lung transplant recipients are particularly susceptible to nocardiosis due to their long-term immunosuppressive therapy and compromised immune function.

Previous studies have shown that the incidence of nocardiosis in lung transplant recipients ranges from 1.9% to 3.5%.[3,4] Nocardiosis lacks specific clinical manifestations and biomarkers, requiring definitive pathogenic evidence for diagnosis.[5] Current pathogenic detection methods primarily include bacterial smears, bacterial cultures, and molecular biological detection methods such as metagenomic next-generation sequencing (mNGS).[6] In clinical practice, the low positive rate of *Nocardia* culture and delayed antimicrobial susceptibility testing often result in delayed diagnosis and non-standardized treatment, seriously affecting patient outcomes. A study showed that the mean diagnostic time in nocardiosis patients with poor prognosis was significantly longer than that in patients with favorable prognosis (19.7 days vs 7.3 days).[7] Currently, the six-month mortality rate for nocardiosis in solid organ transplant recipients is 14.3%.[8]

In recent years, the application of mNGS has improved the diagnosis rate of nocardiosis. Our team's previous research found that the prevalence of nocardiosis in lung transplant recipients is 4.2%,[9] with most cases occurring within

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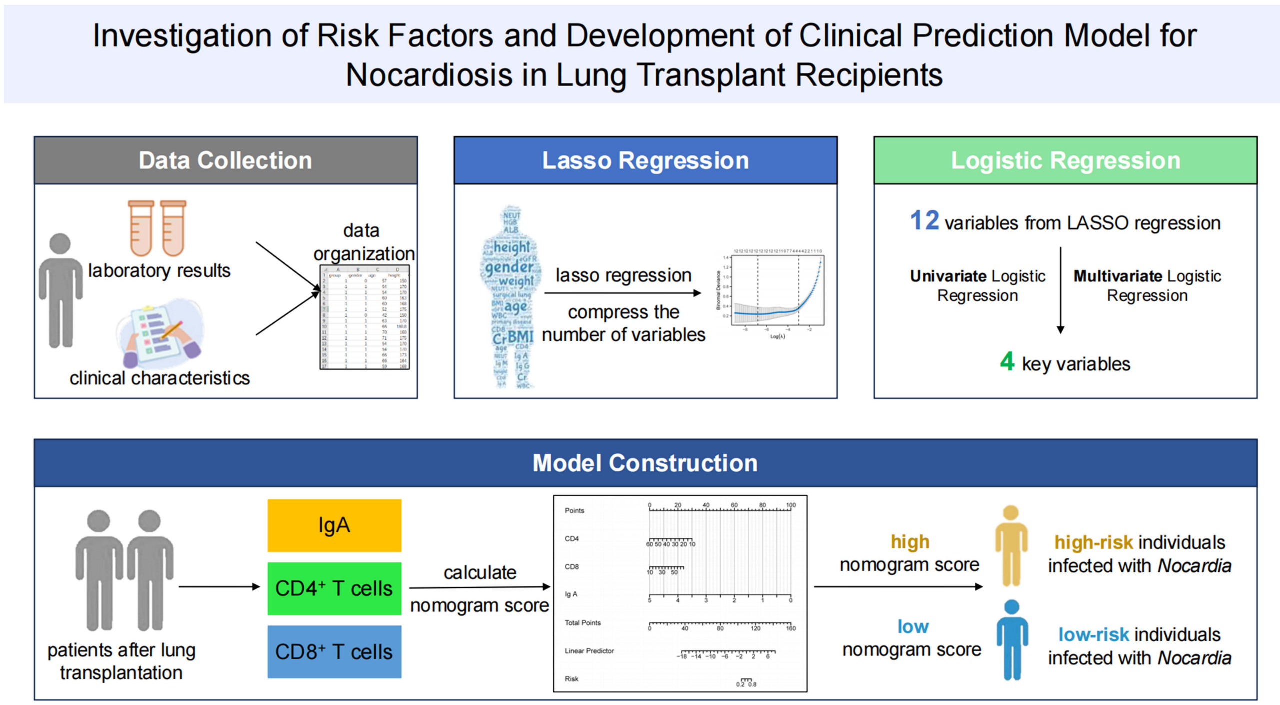
Graphical Abstract



one year after surgery, primarily presenting as pulmonary nocardiosis, diagnosed through mNGS detection of bronchoalveolar lavage fluid (BALF). However, bronchoalveolar lavage is an invasive procedure with significant trauma and certain contraindications, making it unsuitable for some patients. Furthermore, bronchoscopy carries iatrogenic risks such as pneumothorax and bleeding, which some patients cannot tolerate. Although mNGS is more sensitive and provides rapid detection compared to traditional methods, its cost is relatively high.[6] Therefore, identifying non-invasive, sensitive, convenient, and economical screening methods for nocardiosis to assist pathogenic diagnosis and guide empirical early treatment could improve clinical outcomes.

Materials and Methods

Study Population

This single-center retrospective observational study analyzed data from 679 patients who underwent lung transplantation at the First Affiliated Hospital of Guangzhou Medical University between January 1, 2015, and July 9, 2024. Twenty patients with nocardiosis were identified, with diagnoses occurring between January 1, 2022, and September 10, 2024. Using 1:2 matching, 40 lung transplant recipients without nocardiosis who were matched for age, sex, post-transplant duration, transplant type, and immunosuppressant use were selected as controls. This study was approved by the Ethics Committee of the First Affiliated Hospital of Guangzhou Medical University (Ethics number: ES-2024-K144-01).

Inclusion criteria for the nocardiosis group were: (1) age ≥18 years; (2) receipt of single lung, bilateral lung, or heart-lung transplantation; (3) diagnosis of nocardiosis meeting the criteria from “Nocardia infections in solid organ transplantation: Guidelines from the Infectious Diseases Community of Practice of the American Society of Transplantation”;[10] (4) respiratory-related symptoms; (5) pulmonary lesions on imaging; (6) pathogenic evidence confirming nocardiosis, including positive results from bronchoscopy samples (such as deep sputum, BALF) through smear, high-throughput sequencing, or culture. Exclusion criteria were: (1) incomplete clinical data or follow-up information; (2) patients who did not consent to the use of their clinical samples for research; (3) patients with acute rejection or CLAD.

Inclusion criteria for the control group were: (1) age ≥18 years; (2) receipt of single lung, bilateral lung, or heart-lung transplantation; (3) stable condition without nocardiosis after transplantation; (4) matched baseline characteristics with the nocardiosis group in terms of sex, age, and primary disease type. Exclusion criteria were: (1) the same exclusion criteria applied to the nocardiosis group; (2) patients with other types of pathogenic infections during the study period.

Study Parameters

Clinical information collected included sex, age, BMI, primary disease, transplanted lung, and Postoperative Disease-Free Interval. Laboratory indicators included white blood cell count (WBC), neutrophil count (NEUT), lymphocyte count (LYMPH), estimated Glomerular Filtration Rate (eGFR), albumin (ALB), hemoglobin (HGB), T lymphocyte subsets ($CD4^+$ T cells, $CD8^+$ T cells), immunoglobulin A (IgA), immunoglobulin G (IgG), and immunoglobulin M (IgM).

For patients with nocardiosis, all indicators were taken from examination results at the time of diagnosis.

Research Methods

Patient Characteristics and Baseline Analysis

Clinical information and laboratory indicators were organized, and preliminary variable screening was performed.

Feature Variable Selection

LASSO regression incorporated 6 clinical information indicators and 12 laboratory indicators mentioned in the Study Parameters. LASSO regression was conducted to select variables with important predictive value through penalty terms. Variables corresponding to the minimum λ value were selected and designated as core variables for subsequent analysis. Variables selected by LASSO regression underwent univariate logistic regression analysis, with variables showing $P < 0.05$ in univariate analysis included in multivariate logistic regression. The enter method was used for multivariate logistic regression analysis. Variables causing complete separation were excluded from multivariate modeling to ensure convergence and validity.

Construction and Validation of Nomogram

Variables with $P < 0.05$ in multivariate logistic regression were used to construct a nomogram model for predicting nocardiosis risk. Patient risk was assessed using comprehensive risk scores calculated from individual variable scores.

Model sensitivity and specificity were evaluated using receiver operating characteristic (ROC) curves. Model fit was assessed using calibration curves to examine agreement between predicted and actual values. Clinical utility was evaluated using decision curves by calculating net benefits at different probability thresholds.

Statistical Analysis

Statistical analysis was performed using R software (version 4.2.1) and SPSS (version 25), with $\alpha = 0.05$ as the significance level. For normally distributed continuous variables, data are expressed as mean ± standard deviation and compared using independent-samples t-tests. For skewed continuous variables, data are presented as median (first quartile, third quartile) and analyzed using the Mann–Whitney *U*-test. Categorical variables are reported as number (percentage) and compared using either the chi-square test or Fisher's exact test, as appropriate.

LASSO regression was performed using the glmnet package in R software. Univariate and multivariate logistic regression analyses were performed using SPSS to calculate odds ratios (OR), and the rms package was used to construct the nomogram model.

Results

Patient Characteristics

Among 679 patients, 20 cases of nocardiosis were identified. All 20 patients exhibited clinical characteristics of pulmonary nocardiosis, with diagnoses confirmed through detection of *Nocardia* in BALF, including bacterial smears, mNGS, and bacterial cultures. Additionally, 5 patients had concurrent *Nocardia* bloodstream infections confirmed by positive peripheral blood cultures, and 3 patients had infections in other organs (2 intracranial infections, 1 gallbladder infection) confirmed by positive mNGS results from cerebrospinal fluid and gallbladder drainage fluid, respectively. Based on the characteristics of nocardiosis patients, 40 lung transplant recipients without nocardiosis were selected as controls through 1:2 matching, with similar age, sex, and post-transplant duration. The final analysis included 60 lung transplant recipients: 20 (33.33%) with nocardiosis and 40 (66.67%) controls.

Baseline Variables Analysis

There were no significant differences between the two groups in baseline variables including age, sex, BMI, primary disease, and transplanted lung ($P > 0.05$) (Table 1). Significant differences were observed between the nocardiosis and control groups in lymphocyte count, albumin, hemoglobin, $CD4^+$ T cells, $CD8^+$ T cells, and IgA levels ($P < 0.05$) (Table 2).

Table 1 Baseline Clinical Characteristics

Variables	Control Group (n=40)	Nocardiosis Group (n=20)	Statistics	*P*
Age, M[a] (Q_1, Q_3)[b]	59.00 (53.75, 66.00)	60.00 (54.00, 66.00)	Z=−0.35 [c]	0.73
BMI, Mean ± SD[d]	20.98 ± 3.06	20.27 ± 4.06	t=0.76[e]	0.451
Sex, n (%)			χ^2=0.00[f]	1
Male	36 (90.00)	18 (90.00)		
Female	4 (10.00)	2 (10.00)		
Primary Disease, n (%)			- [g]	0.131
ILD	22 (55.00)	15 (75.00)		
COPD	11 (27.50)	5 (25.00)		
Other	7 (17.50)	0 (0.00)		
Transplant type, n (%)			- [g]	0.519
Single Lung	20 (50.00)	7 (35.00)		
Bilateral Lung	19 (47.50)	13 (65.00)		
Heart-lung Transplantation	1 (2.50)	0 (0)		
Postoperative Disease-Free Interval (Days), M[a] (Q_1, Q_3)[b]	631.50 (328.50, 1214.75)	407.00 (155.50, 807.00)	Z=−1.05 [c]	0.293

Abbreviations: [a]M, mean value; [b]Q_1, first quartile, Q_3, third quartile; [c]Z, Mann–Whitney test; [d]SD, standard deviation; [e] t, *t*-test; [f] χ^2, Chi-square test; [g] -, Fisher's exact test.

Table 2 Baseline Laboratory Data

Variables	Control Group (n=40)	Nocardiosis Group (n=20)	Statistics	*P*
WBC ($\times 10^9$/L), Mean ± SD[a]	6.97 ± 1.79	7.67 ± 3.38	t=−0.86[b]	0.399
NEUT ($\times 10^9$/L), M [c] (Q_1, Q_3)[d]	4.92 (3.73, 5.78)	4.53 (3.54, 8.22)	Z=−0.49[e]	0.621
LYMPH ($\times 10^9$/L), M [c] (Q_1, Q_3)[d]	1.15 (0.74, 1.80)	0.74 (0.47, 1.07)	Z=−2.37[e]	0.018
eGFR (mL/min/1.73m^2), M [c] (Q_1, Q_3)[d]	65.84 (54.60, 77.59)	56.25 (46.75, 78.12)	Z=−1.14[e]	0.256
ALB (g/L), M [c] (Q_1, Q_3)[d]	39.90 (37.80, 40.60)	30.30 (29.67, 32.90)	Z=−6.02[e]	<0.001
HGB (g/L), M [c] (Q_1, Q_3)[d]	119.00 (106.00, 132.50)	95.50 (76.00, 111.25)	Z=−3.77[e]	<0.001
T-lymphocyte (%), Mean ± SD[a]	69.30 ± 12.39	70.95 ± 15.03	t=−0.45[b]	0.651
$CD4^+$ T cells (%), Mean ± SD[a]	37.39 ± 10.97	26.09 ± 7.76	t=4.11[b]	<0.001
$CD8^+$ T cells (%), M [c] (Q_1, Q_3)[d]	27.20 (20.83, 32.25)	38.80 (31.68, 50.33)	Z=−3.62[e]	<0.001
IgA (g/L), M [c] (Q_1, Q_3)[d]	1.47 (1.09, 1.90)	0.80 (0.50, 0.99)	Z=−4.10[e]	<0.001
IgG (g/L), Mean ± SD[a]	8.46 ± 2.78	7.18 ± 2.71	t=1.69[b]	0.096
IgM (g/L), M [c] (Q_1, Q_3)[d]	0.78 (0.47, 1.05)	0.67 (0.55, 0.88)	Z=−0.64[e]	0.52

Abbreviations: [a]SD, standard deviation; [b]t, *t*-test; [c]M, mean value; [d]Q_1, first quartile, Q_3, third quartile; [e]Z, Mann–Whitney test.

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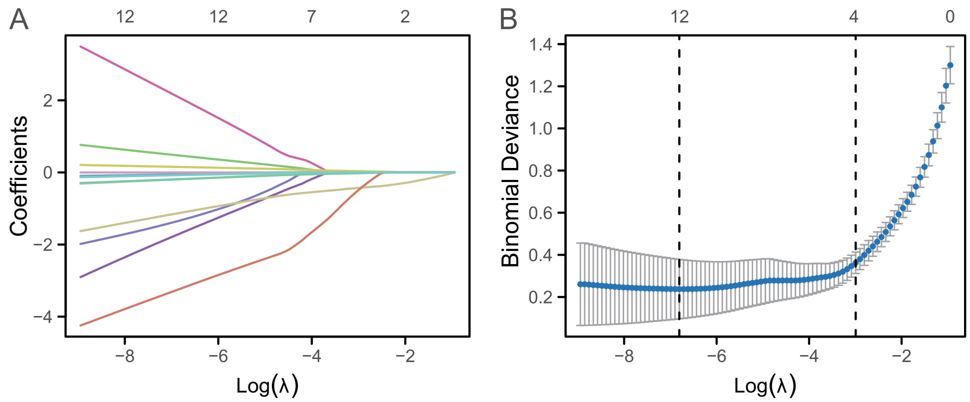

Figure 1 LASSO Regression Analysis. (**A**) Coefficient paths for predictors across log(λ) values. (**B**) Cross validation of the model.

Feature Variable Selection

LASSO regression was used to generate λ-coefficient correlation curves (Figure 1A) and deviation-λ correlation curves (Figure 1B). Twelve feature variables corresponding to the minimum λ value were selected. Univariate logistic regression analysis identified lymphocyte count, albumin, $CD4^+$ T cells, $CD8^+$ T cells, and IgA levels as variables significantly associated with nocardiosis status (vs controls; $P < 0.05$) (Table 3). Multivariate logistic regression analysis of variables with $P < 0.05$ in univariate analysis identified $CD4^+$ T cells, $CD8^+$ T cells, and IgA as independent risk factors for nocardiosis in lung transplant recipients (Table 4).

Table 3 Univariate Logistic Regression

Variables	*P*	OR[a]	95% CI[b]
Primary Disease	0.061	0.39	0.15, 1.04
Surgical Method	0.274	1.86	0.61, 5.63
Age	0.734	1.01	0.96, 1.05
BMI	0.444	0.94	0.80, 1.10
Postoperative Disease-Free Interval	0.890	1.00	1.00, 1.00
WBC	0.300	1.13	0.90, 1.41
LYMPH	0.049	0.37	0.14, 0.99
ALB	0.008	0.31	0.13, 0.74
$CD4^+$ T cells	<0.001	0.90	0.84, 0.96
$CD8^+$ T cells	<0.001	1.11	1.04, 1.18
IgA	<0.001	0.06	0.01, 0.28
IgG	0.100	0.84	0.68, 1.03

Abbreviations: [a]OR, odd ratio; [b]CI, confidence interval.

Table 4 Multivariate Logistic Regression

Variables	*P*	OR[a]	95% CI[b]
LYMPH	0.674	0.756	0.204, 2.793
$CD4^+$ T cells	0.009	0.865	0.776, 0.964
$CD8^+$ T cells	0.020	1.113	1.017, 1.218
IgA	0.004	0.007	0, 0.207

Abbreviations: [a]OR, odd ratio; [b]CI, confidence interval.

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Construction and Validation of Nomogram

A prediction model was constructed using CD4$^+$ T cells, CD8$^+$ T cells, and IgA levels and visualized through a nomogram (Figure 2). ROC curves were plotted for individual indicators and their combination. The areas under the curve (AUC) for CD4$^+$ T cells, CD8$^+$ T cells, and IgA were 0.791, 0.789, and 0.828, respectively (Figure 3A). The combined model showed an AUC of 0.955 (Figure 3B), with a cutoff value of 0.64353, sensitivity of 0.95, and specificity of 0.875. The calibration curve indicated a strong correlation between the anticipated risk and the actual risk represented by the model (Figure 4A). The decision curve suggested that the model provides superior net benefit versus "treat-all" or "treat-none" strategies across risk thresholds of 0.2 to 0.8, confirming its value for clinical decision making (Figure 4B).

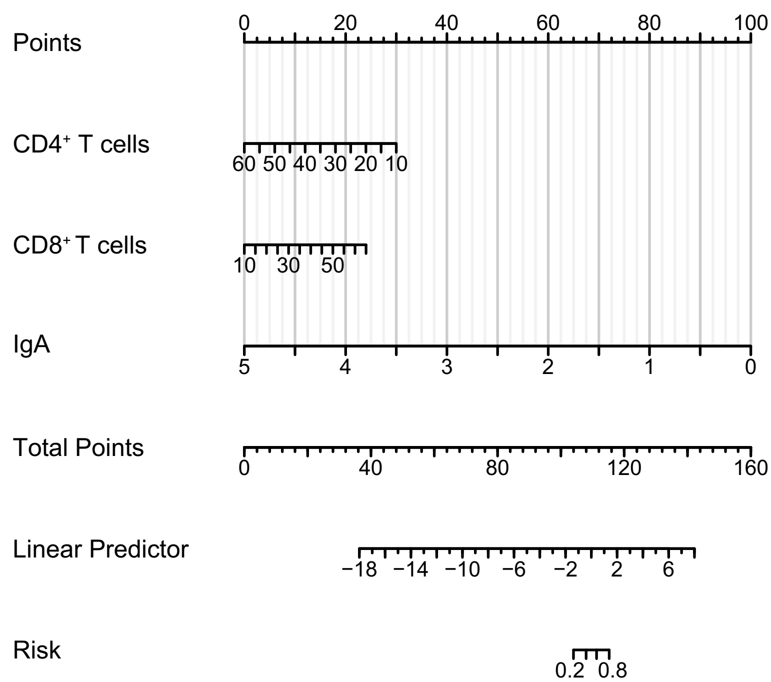

Figure 2 Nomogram model for predicting nocardiosis in lung transplant recipients.

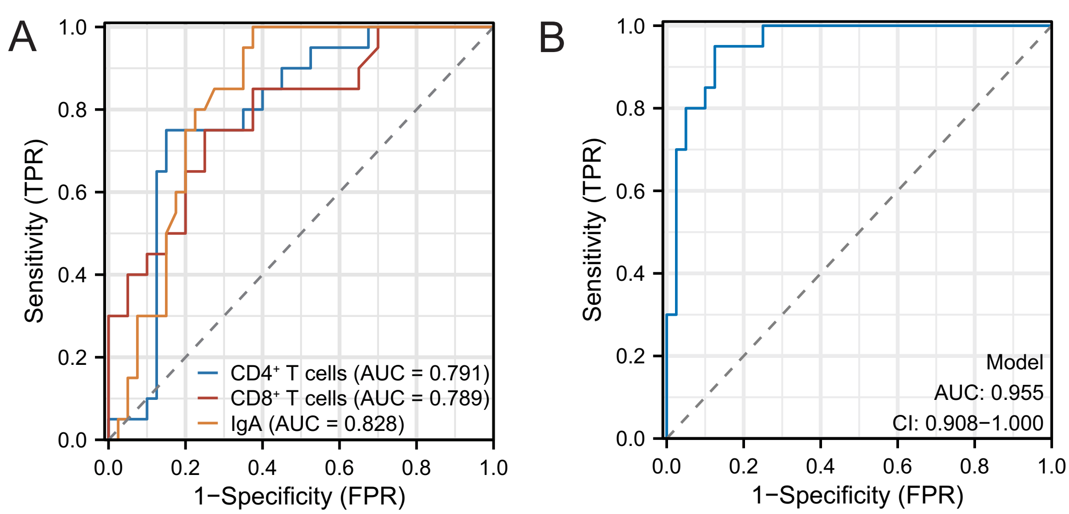

Figure 3 ROC curves of the prediction model. (**A**) ROC curves for the three variables included in the nomogram model; (**B**) ROC curve for the nomogram model.

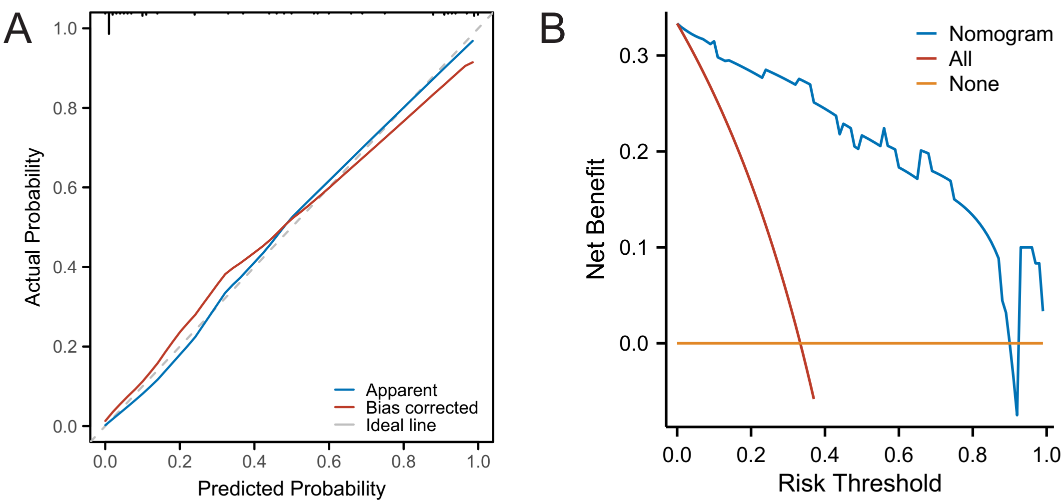

Figure 4 Model evaluation plots. (**A**) Decision curve of the prediction model; (**B**) Calibration curve of the prediction model.

Discussion

Nocardiosis is a disease caused by *Nocardia* species infection leading to tissue and organ damage, primarily affecting immunocompromised patients. Among transplant recipients, lung transplant recipients are at particularly high risk for nocardiosis.[11] Through a retrospective study of a large sample of lung transplant recipients, this study established the first prediction model for nocardiosis in lung transplant recipients, providing an early identification tool for screening high-risk patients.

Our center's data showed that among 20 nocardiosis patients, all had pulmonary nocardiosis, with 5 patients having concurrent *Nocardia* bloodstream infections and 3 patients having infections in other organs (2 intracranial infections, 1 gallbladder infection). Our findings indicate that nocardiosis can affect multiple organs throughout the body, with the lungs being the most commonly affected organ. Pulmonary nocardiosis can also disseminate to other organs, leading to disseminated nocardiosis, consistent with previous research findings.[10] Additionally, our team found that before 2020, using traditional detection methods, the diagnosis rate of nocardiosis was 3.5%.[5] In the past five years, the diagnosis rate has significantly increased to 6.1%, likely due to the adoption of mNGS. Traditional detection methods primarily rely on bacterial culture, but *Nocardia* has a low culture positivity rate and requires extended culture time, leading to potential missed or delayed diagnoses and delayed treatment initiation or missed treatment opportunities. Although mNGS offers high sensitivity, rapid detection, and high specificity, significantly shortening the detection period, the diagnosis of pulmonary nocardiosis in lung transplant recipients largely depends on detecting *Nocardia* in BALF.[12] However, obtaining BALF is an invasive procedure with significant trauma, contraindications, and various drawbacks, which some patients cannot tolerate. This study screened and analyzed routine immune status indicators and biochemical parameters through regression analysis to construct a prediction model for nocardiosis development, using non-invasive, sensitive detection methods for early warning and assisting in precise pathogenic diagnosis, providing a signal for doctors to start treatment as early as possible.

Our study showed significant differences between nocardiosis and control groups in lymphocyte count, albumin, hemoglobin, $CD4^+$ T cells, $CD8^+$ T cells, and IgA levels. Through LASSO regression and logistic regression screening, we identified three independent risk factors ($CD4^+$ T cells, $CD8^+$ T cells, and IgA) for nocardiosis in lung transplant recipients and constructed a prediction model based on these factors. This prediction model demonstrated high predictive accuracy with an AUC of 0.955 and showed good clinical value in calibration and decision curves. By calculating nomogram scores, this model can be used to screen high-risk patients for nocardiosis post-transplantation, providing clinicians with a visualized individualized risk assessment tool.

Our study found that lymphocyte counts were significantly lower in the nocardiosis group compared to controls. This finding is consistent with previous research showing that decreased lymphocyte counts are closely associated with

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infection.[13] Sarah Soueges et al[14] found that low lymphocyte counts were associated with disseminated nocardiosis. Additionally, our study found that in 3 cases of refractory nocardiosis, lymphocyte count reduction was particularly pronounced, both in severity and duration. K Shannon et al[15] found similar results in their study of hematopoietic stem cell transplant recipients. Combined with these studies, our results further suggest that both nocardiosis occurrence and treatment efficacy are closely related to immune status, and severe lymphocyte reduction may be not only closely associated with nocardiosis development but also a major factor in poor treatment response. Furthermore, our study found that albumin and hemoglobin levels were also significantly lower in nocardiosis patients compared to controls. Albumin and hemoglobin are important indicators of malnutrition. Albumin plays crucial roles in improving inflammation, maintaining vascular endothelial integrity, and maintaining acid-base balance.[16] Our findings align with previous research showing that hypoalbuminemia is closely associated with infectious diseases and their severity.[16] Shan Li et al[16] found that over one-third of autoimmune disease patients with nocardiosis had decreased albumin levels. While the causal relationship between plasma albumin reduction and nocardiosis remains unclear, decreased plasma albumin can serve as one clinical characteristic for auxiliary diagnosis of nocardiosis. Additionally, our study found that nocardiosis patients all had varying degrees of anemia with decreased hemoglobin levels. Currently, there are no studies analyzing the correlation between nocardiosis and hemoglobin. However, previous research has shown that decreased hemoglobin levels are an independent risk factor for 90-day mortality in community-acquired pneumonia.[17] This suggests that decreased hemoglobin levels may be associated with the severity of pulmonary infection. Therefore, our findings suggest that clinicians should be alert for nocardiosis in lung transplant recipients with infectious complications when accompanied by decreased lymphocyte counts, albumin, and hemoglobin levels.

Our study further analyzed the cellular and humoral immune status of nocardiosis patients. Results showed that $CD4^+$ T cells and $CD4^+/CD8^+$ T cell ratios were significantly lower in the nocardiosis group. Tejasvi Kanagiri et al[18] reported similar findings, showing that low $CD4^+$ T cell counts were closely associated with increased risk of nocardiosis. CD4 and CD8 are core receptors on T cell surfaces and essential components of cellular immunity, playing crucial roles in T cell recognition and activation,[19] $CD4^+$ T cells are vital in protecting against pathogen infections. Therefore, our results suggest that low $CD4^+$ T cell counts and high $CD8^+$ T cell counts are closely associated with nocardiosis development in lung transplant recipients. The main pathophysiological mechanism may be: $CD4^+$ T cells secrete interferon-γ (IFN-γ) to activate macrophages, enhancing their killing ability against *Nocardia*.

However, *Nocardia* can suppress IFN-γ production, thereby inhibiting macrophage clearance of *Nocardia*.[20] Thus, when $CD4^+$ T cell counts are reduced, decreased IFN-γ secretion leads to insufficient macrophage activation, preventing *Nocardia* clearance and resulting in nocardiosis. Few studies have focused on the relationship between $CD8^+$ T cells and nocardiosis. However, HIV infection studies have shown that elevated $CD8^+$ T cell counts are associated with cytomegalovirus co-infection when $CD4^+$ T cell counts are low.[21] Yamagata et al[22] and Shrestha et al[23] indicated that $CD8^+$ T cells increase significantly in immunosuppressed patients, representing an active immune response to infection. Additionally, our assessment of humoral immunity in lung transplant recipients found that low IgA levels were associated with nocardiosis. While many previous studies have focused on the correlation between low IgG and increased infection rates in lung transplant recipients, Sudish C Murthy et al[24] found that pre-transplant low IgA was associated with infection in lung transplant recipients, but few studies have explored the relationship between post-transplant IgA levels and infection.[25,26] Similar research found that IgA deficiency led to increased mortality in liver transplant recipients.[27] Our study suggests that decreased IgA levels may also be a major risk factor for nocardiosis in lung transplant recipients. The potential mechanism may be: IgA is the most abundant immunoglobulin in the human body, primarily present in mucosal areas, protecting against infection by neutralizing toxins and preventing microorganisms from crossing mucosal epithelial barriers.[28] Healthy adults produce approximately 3 grams of IgA daily.[29] Low IgA levels may weaken mucosal immune defense, making it easier for *Nocardia* to colonize and cause infection.

Our study's univariate and multivariate logistic regression analyses showed that decreased $CD4^+$ T cells and IgA levels, and elevated $CD8^+$ T cells are independent risk factors for post-lung transplant nocardiosis, and abnormal changes in these three indicators suggest the potential development of nocardiosis. The prediction model constructed based on multivariate analysis demonstrated good predictive performance in validation, with an AUC of 0.955, indicating high accuracy in both sensitivity and specificity. These three indicators are derived from routine laboratory tests, making them easily accessible and cost-effective. By monitoring these indicators, clinicians can identify high-risk lung transplant recipients for infection, take timely intervention measures, develop individualized treatment plans, and improve patient outcomes.

In clinical practice, this model can be incorporated into routine follow-up assessments for lung transplant recipients by integrating laboratory indicators—$CD4^+$ T cells, $CD8^+$ T cells, and IgA—into electronic medical record systems. The risk score calculated via the nomogram enables clinicians to quickly assess nocardiosis risk using regularly obtained blood test results. Based on the ROC analysis, a cutoff probability of 0.64 offers a balance between sensitivity and specificity, and could serve as an actionable threshold. Patients exceeding this threshold may benefit from early imaging evaluations, microbiological monitoring, or even empirical antibiotic treatment when clinical suspicion is high. This implementation strategy may help prevent delays in diagnosis and improve outcomes through timely intervention.

This study has certain limitations. Due to the relatively low incidence of post-lung transplant nocardiosis, the number of cases was limited, and the prediction model was not split into training and validation sets. Besides, we provide the complete scoring rules in Figure 3 to enable verification by external cohorts. Therefore, we look forward to future multi-center studies with larger sample sizes. Additionally, some nocardiosis patients in this study had concurrent infections with other pathogens, which might affect the accuracy of the study results.

Summary

Through a retrospective study of a large sample of lung transplant recipients, this study analyzed laboratory indicators in nocardiosis patients and constructed a nomogram model based on LASSO regression and logistic regression analyses. The model shows that decreased $CD4^+$ T cells and IgA levels, and elevated $CD8^+$ T cells can predict the occurrence of post-lung transplant nocardiosis. Additionally, our results suggest that clinicians should be alert for nocardiosis in lung transplant recipients with infectious complications when accompanied by decreased lymphocyte counts, albumin, and hemoglobin levels. This study may provide reference value for early diagnosis and preemptive treatment of nocardiosis in clinical lung transplant recipients.

Conclusion

$CD4^+$ T cells, $CD8^+$ T cells, and IgA are independent risk factors for nocardiosis in lung transplant recipients. The prediction model based on these three indicators can effectively differentiate between patients with and without nocardiosis, facilitating early identification of nocardiosis in clinical practice.

Data Sharing Statement

The datasets generated and/or analyzed during the current study are not publicly available but are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The study followed the Declaration of Helsinki. Given that this study is retrospective, the Ethics Committee of the First Affiliated Hospital of Guangzhou Medical University approved the study and consented to waive the informed consent statement (Ethics number: ES-2024-K144-01).

Consent for Publication

All authors consent to the publication of this study.

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The authors are grateful to the patients involved in this study.

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 report no conflicts of interest in this work.

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