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ORIGINAL RESEARCH

Construction of a Nomogram Prediction Model for Individualized Prediction of the Risk of Pulmonary Fungal Infection in Lung Cancer

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Objective: To construct a nomogram model for individualized prediction of pulmonary fungal infection risk in lung cancer patients.
Methods: A total of 483 lung cancer patients hospitalized between August 2021 and August 2024 were retrospectively analyzed and randomly divided into a modeling group (n=338) and validation group (n=145). Patients in the modeling group were categorized based on the presence or absence of pulmonary fungal infection. Clinical data were analyzed using logistic regression, and a nomogram was developed using R software. Model performance was assessed using ROC curves, the Hosmer-Lemeshow (H-L) test, and Decision Curve Analysis (DCA).
Results: Pulmonary fungal infections occurred in 99 out of 483 patients (20.50%). In the modeling group, the infection rate was 21.30%. Multivariate logistic regression identified age, smoking history, diabetes, glucocorticoid use, type of antimicrobial agents, invasive procedures, and length of hospitalization as independent risk factors ($P<0.05$). The Area Under the Curve (AUC) was 0.933 in the modeling group and 0.954 in the validation group. H-L tests indicated good model calibration ($P>0.05$). DCA demonstrated high clinical utility when the predicted probability ranged from 0.08 to 0.93.
Conclusion: The nomogram based on key clinical factors effectively predicts the risk of pulmonary fungal infection in lung cancer patients and is a promising tool for assisting in early identification and intervention.
Keywords: lung cancer, pulmonary fungal infection, influencing factors, nomogram

Introduction

With the continuous advancement of diagnostic and therapeutic technologies, the mortality rate of lung cancer has decreased; however, it remains the leading cause of cancer-related deaths globally.[1,2] After undergoing surgery, radiotherapy, and chemotherapy, lung cancer patients often experience damage to normal cells, suppression of bone marrow function, and decreased immunity, making them more susceptible to pulmonary fungal infections. These infections not only impact the efficacy of surgical treatment but also lead to poor prognosis and prolonged hospital stays for patients.[3] Pulmonary fungal infections are respiratory diseases caused by various fungi and predominantly affect immunocompromised individuals. The incidence of such infections is increasing. Without timely intervention, these infections can progress to sepsis, thereby significantly impacting patient outcomes.[4,5] Studies have shown that the mortality rate among cancer patients with fungal infections is relatively high. Lung cancer patients often have chronic pulmonary diseases, and the sensitivity of early diagnosis based on clinical symptoms and imaging is low, posing challenges for timely identification. This increases the risk of pulmonary fungal infections in these patients.[6] Therefore, identifying risk factors for fungal infections is crucial for implementing early preventive and therapeutic strategies, thereby improving patient outcomes. A nomogram is a tool that provides a visual representation of predictive models to quantify the risk of clinical events, thereby helping clinicians predict the likelihood of an event and formulate corresponding preventive measures.[7] Despite this utility, there are currently few reports on the application of nomograms for predicting pulmonary fungal

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infections among lung cancer patients. Consequently, this study aimed to construct and validate a nomogram-based predictive model for individualized prediction of the risk of pulmonary fungal infections in lung cancer patients.

Materials and Methods

General Information

This study retrospectively selected 483 lung cancer patients admitted to our hospital from August 2021 to August 2024. The cases were randomly divided into a modeling group (338 cases) and a validation group (145 cases) at a 7:3 ratio. The modeling group was further categorized into an infection group and a non-infection group based on whether pulmonary fungal infection occurred. The case collection flowchart is shown in Figure 1. Inclusion criteria: (1) Meeting the diagnostic criteria for lung cancer,[8] confirmed by pathological results; (2) Meeting the diagnostic criteria for fungal infections[9] without infection before admission; (3) Age ≥18 years; (4) Complete clinical data available. Exclusion criteria: (1) Survival time <6 months; (2) Severe organ failure; (3) presence of other active infectious diseases; (4) Bacterial infections; (5) Immunodeficiency; (6) Long-term use of immunosuppressive drugs; (7) Cognitive impairment. The study protocol was approved by the Institutional Ethics Committee of our hospital.

Determination of Pulmonary Fungal Infection

The diagnosis of pulmonary fungal infection[9] was based on the following criteria: (1) fever (body temperature exceeding 38°C); (2) Worsening clinical symptoms (eg, cough, sputum production), with gradually increasing airway secretions; (3) Presence of pulmonary moist rales; (4) White blood cell count $\geq 10\times10^{9}$/L, with an increased proportion of neutrophils; (5) Radiological evidence of pulmonary inflammation evident on imaging; (6) Positive fungal culture in sputum specimens. Diagnosis of pulmonary fungal infection required fulfillment of criteria (1)-(4) plus at least one of criteria (5) or (6).

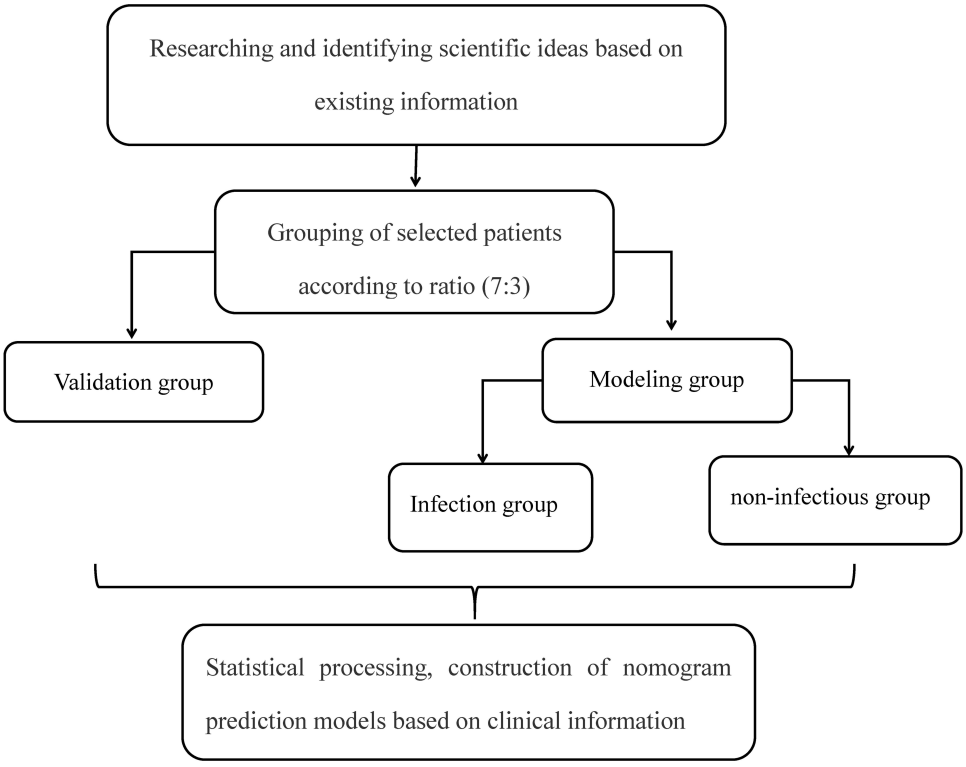

Figure 1 Case flow collection diagram.

Data Collection and Variables

(1) Comparison of clinical data between the modeling group and validation group. Data were collected from the electronic medical record system, including age, sex, body mass index (BMI), smoking history, drinking history, hypertension, diabetes, respiratory diseases, tumor stage, pathological type, anemia, use of glucocorticoids, types of antibiotics used, invasive procedures, hospitalization duration, hypoproteinemia, and treatment methods. (2) Comparison of clinical data between the infection group and non-infection group. (3) Analysis of factors influencing pulmonary fungal infection in lung cancer patients. (4) Construction of a nomogram model for predicting pulmonary fungal infection in lung cancer patients. (5) Analysis of the nomogram models for the modeling and validation groups. (6) Construction of the DCA (Decision Curve Analysis) curve for the nomogram model.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Categorical data were analyzed using the chi-square (χ^2) test or Fisher's exact test, as appropriate, and expressed as frequencies (n) and percentages (%). Continuous data conforming to a normal distribution were analyzed using independent samples *t*-tests and expressed as mean ± standard deviation ($\bar{x} \pm s$). Multivariate logistic regression analysis was used to identify factors influencing pulmonary fungal infections in lung cancer patients. The nomogram model was constructed using R software (R software version 3.6.3 and the rms package). The discriminative ability of the nomogram was evaluated using Receiver Operating Characteristic (ROC) curve analysis, and its area under the curve (AUC) was calculated. Calibration was assessed using the Hosmer-Lemeshow (H-L) goodness-of-fit test and calibration plots. The clinical utility of the nomogram was evaluated using Decision Curve Analysis (DCA). A P-value <0.05 was considered statistically significant.

Results

Comparison of Clinical Data Between the Modeling and Validation Groups

There were no significant differences in clinical data such as age and sex between the modeling group and the validation group (P>0.05). Detailed comparisons are presented in Table 1.

Table 1 Comparison of Clinical Data Between the Modeling Group and the Validation Group

factor	Modeling Group (n=338)	Validation Group (n=145)	χ^2	*P*
Age (years)			0.062	0.803
<65	179 (52.96)	75 (51.72)		
≥65	159 (47.04)	70 (48.28)		
Genders			0.005	0.944
Man	183 (54.14)	78 (53.79)		
Woman	155 (45.86)	67 (46.21)		
BMI (kg/m^2)			0.114	0.736
<24	206 (60.95)	86 (59.31)		
≥24	132 (39.05)	59 (40.69)		
Smoking history			0.204	0.651
Yes	144 (42.60)	65 (44.83)		
No	194 (57.40)	80 (55.17)		
Drinking history			0.037	0.848
Yes	82 (24.26)	34 (23.45)		
No	256 (75.74)	111 (76.55)		
Hypertension			0.167	0.682
Yes	78 (23.08)	31 (21.38)		
No	260 (76.92)	114 (78.62)		

(*Continued*)

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Table 1 (Continued).

factor	Modeling Group (n=338)	Validation Group (n=145)	χ^2	*P*
Diabetes			0.102	0.750
Yes	60 (17.75)	24 (16.55)		
No	278 (82.25)	121 (83.45)		
Respiratory diseases			0.045	0.833
Yes	134 (39.64)	56 (38.62)		
No	204 (60.36)	89 (61.38)		
Pathological staging			0.970	0.325
I~II stage	161 (47.63)	62 (42.76)		
III~IV stage	177 (52.37)	83 (57.24)		
Pathological type			0.220	0.639
Adenocarcinoma	238 (70.41)	99 (68.28)		
Squamous carcinoma	100 (29.59)	46 (31.72)		
Anemic			0.244	0.621
Yes	96 (28.40)	38 (26.21)		
No	242 (71.60)	107 (73.79)		
Use of glucocorticoids			0.175	0.676
Yes	128 (37.87)	52 (35.86)		
No	210 (62.13)	93 (64.14)		
Types of antimicrobial applications			0.046	0.830
<3	204 (60.36)	86 (59.31)		
≥3	134 (39.64)	59 (40.69)		
Intrusive operations			0.264	0.607
Yes	139 (41.12)	56 (38.62)		
No	199 (58.88)	89 (61.38)		
Length of hospitalisation			0.361	0.548
>2weeks	131 (38.76)	52 (35.86)		
≤2weeks	207 (61.24)	93 (64.14)		
Liver injury			0.064	0.800
Yes	101 (29.88)	45 (31.03)		
No	237 (70.12)	100 (68.97)		
Hypoproteinemia			0.103	0.748
Yes	117 (34.62)	48 (33.10)		
No	221 (65.38)	97 (66.90)		
Treatment method			0.134	0.714
Surgeries	75 (22.19)	30 (20.69)		
Radiotherapy	263 (77.81)	115 (79.31)		

Comparison of Clinical Data Between the Infection and Non-Infection Groups

Among the 483 included patients, 99 developed a pulmonary fungal infection, with an incidence rate of 20.50%. In the modeling group, 72 out of 338 patients experienced infection, with an incidence rate of 21.30%. Univariate analysis revealed significant differences between the two groups within the modeling cohort in terms of age, smoking history, diabetes, use of glucocorticoids, types of antibiotics used, invasive procedures, and hospitalization duration ($P<0.05$). Other clinical data showed no significant differences ($P>0.05$). See Table 2.

Table 2 Comparison of Clinical Data Between Infection and Non-Infection Groups

Factor	Infection Group (n=72)	Non-Infection Group (n=266)	χ^2	*P*
Age (years)			16.217	<0.001
<65	23 (31.94)	156 (58.65)		
≥65	49 (68.06)	110 (41.35)		
Genders			0.074	0.786
Man	40 (55.56)	143 (53.76)		
Woman	32 (44.44)	123 (46.24)		
BMI (kg/m^2)			0.263	0.608
<24	42 (58.33)	164 (61.65)		
≥24	30 (41.67)	102 (38.35)		
Smoking history			16.951	<0.001
Yes	46 (63.98)	98 (36.84)		
No	26 (36.11)	168 (63.16)		
Drinking history			0.616	0.433
Yes	20 (27.78)	62 (23.31)		
No	52 (72.22)	204 (76.68)		
Hypertension			0.191	0.662
Yes	18 (25.00)	60 (22.56)		
No	54 (75.00)	206 (77.44)		
Diabetes			21.122	<0.001
Yes	26 (36.11)	34 (12.78)		
No	46 (63.89)	232 (87.22)		
Respiratory diseases			0.881	0.348
Yes	32 (44.44)	102 (38.35)		
No	40 (55.56)	164 (61.65)		
Pathological staging			0.119	0.730
I~II stage	33 (45.83)	128 (48.12)		
III~IV stage	39 (54.17)	138 (51.88)		
Pathological type			0.144	0.705
Adenocarcinoma	52 (72.22)	186 (69.92)		
Squamous carcinoma	20 (27.78)	80 (30.08)		
Anemic			0.026	0.871
Yes	21 (29.17)	75 (28.20)		
No	51 (70.83)	191 (71.80)		
Use of glucocorticoids			26.324	<0.001
Yes	46 (63.89)	82 (30.83)		
No	26 (36.11)	184 (69.17)		
Types of antimicrobial applications			17.619	<0.001
<3	28 (38.89)	176 (66.17)		
≥3	44 (61.11)	90 (33.83)		
Intrusive operations			13.070	<0.001
Yes	43 (59.72)	96 (36.09)		
No	29 (40.28)	170 (63.91)		
Length of hospitalisation			21.728	<0.001
>2 weeks	45 (62.50)	86 (32.33)		
≤ 2weeks	27 (37.50)	180 (67.67)		
Liver injury			1.694	0.193
Yes	26 (36.11)	75 (28.20)		
No	46 (63.89)	191 (71.80)		

(*Continued*)

Table 2 (Continued).

Factor	Infection Group (n=72)	Non-Infection Group (n=266)	χ^2	*P*
Hypoproteinemia			0.738	0.390
Yes	28 (38.89)	89 (33.46)		
No	44 (61.11)	177 (66.54)		
Treatment method			0.935	0.334
Surgeries	19 (26.39)	56 (21.05)		
Radiotherapy	53 (73.61)	210 (78.95)		

Analysis of Factors Influencing Pulmonary Fungal Infection in Lung Cancer Patients

Whether pulmonary fungal infection occurred in lung cancer patients was taken as the dependent variable (yes=1, no=0). Factors demonstrating significant differences in Table 2 were included as independent variables, with the variable assignment shown in Table 3. Multivariate Logistic regression analysis(Stepwise forward selection method with entry criterion α=0.05 and removal criterion α=0.10) revealed that age(OR:12.094,95% CI:4.927~29.684), smoking history (OR:6.765,95% CI:2.761~16.574), diabetes(OR:4.710,95% CI:1.964~11.293), use of glucocorticoids(OR:5.569,95% CI:2.448~12.669), types of antibiotics used(OR:2.814,95% CI:1.168~6.779), invasive procedures(OR:3.706,95% CI:1.374~9.999), and hospitalization duration(OR:3.805,95% CI:1.409~10.273) were risk factors for pulmonary fungal infection in lung cancer patients (P<0.05). See Table 4.

Table 3 Assignment Methods of Argument Variables

variable	Assignment Method
Age	<65 years old=0, ≥65 years old=1
Smoking history	Yes=1, no=0
Diabetes	Yes=1, no=0
Use of glucocorticoids	Yes=1, no=0
Types of antimicrobial applications	≥3=1, <3=0
Intrusive operations	Yes=1, no=0
Length of hospitalisation	≤2 weeks=0, >2 weeks==1

Table 4 Analysis of Factors Affecting Pulmonary Fungal Infection of Patients with Lung Cancer

variable	β Value	SE Variable	Wald χ^2 Variable	*P* Variable	OR Variable	95% CI
Age	2.493	0.458	29.604	<0.001	12.094	4.927~29.684
Smoking history	1.912	0.457	17.487	<0.001	6.765	2.761~16.574
Diabetes	1.550	0.515	12.059	0.001	4.710	1.964~11.293
Use of glucocorticoids	1.717	0.446	16.766	<0.001	5.569	2.448~12.669
Types of antimicrobial applications	1.035	0.419	5.320	0.021	2.814	1.168~6.779
Intrusive operations	1.310	0.506	6.692	0.010	3.706	1.374~9.999
Length of hospitalisation	1.336	0.507	6.954	0.008	3.805	1.409~10.273
Constant	−5.256	0.563	87.037	<0.001	0.005	–

Construction of the Nomogram Model for Pulmonary Fungal Infection in Lung Cancer Patients

The nomogram model was constructed as follows: $P=e^x/(1+e^x)$, x=12.094×Age+ 6.765×Smoking History +4.710×Diabetes+5.569×Use of Glucocorticoids+2.814×Types of Antibiotics Used+3.706×Invasive Procedures +3.805×Hospitalization Duration. The relative contribution of these factors to infection risk, from greatest to least, were age, smoking history, use of glucocorticoids, diabetes, types of antibiotics used, invasive procedures, and hospitalization duration. To illustrate the nomogram's utility, consider a hypothetical patient: under 65 years old (0 points), with a smoking history (78.5 points), diabetes (62.0 points), no use of glucocorticoids (0 points), application of three or more types of antibiotics (42.5 points), invasive procedures (36.5 points), and hospitalization longer than two weeks (33.5 points), the total score would be 257.5 points. By projecting a vertical line from this total score on the nomogram scale, a predicted probability of approximately 73% for developing pulmonary fungal infection is obtained. The nomogram is depicted in Figure 2.

Nomogram Model in the Modeling Group

The AUC of the nomogram model in the modeling group was 0.933, with a 95% CI of 0.892–0.974. The Hosmer-Lemeshow test result was χ^2=7.120, P=0.705, indicating good calibration. These results are illustrated in Figure 3.

Nomogram Model in the Validation Group

The AUC of the nomogram model in the validation group was 0.954, with a 95% CI of 0.923–0.994. The Hosmer-Lemeshow test result was χ^2=6.576, P=0.637, further supporting good model calibration. These findings are presented in Figure 4.

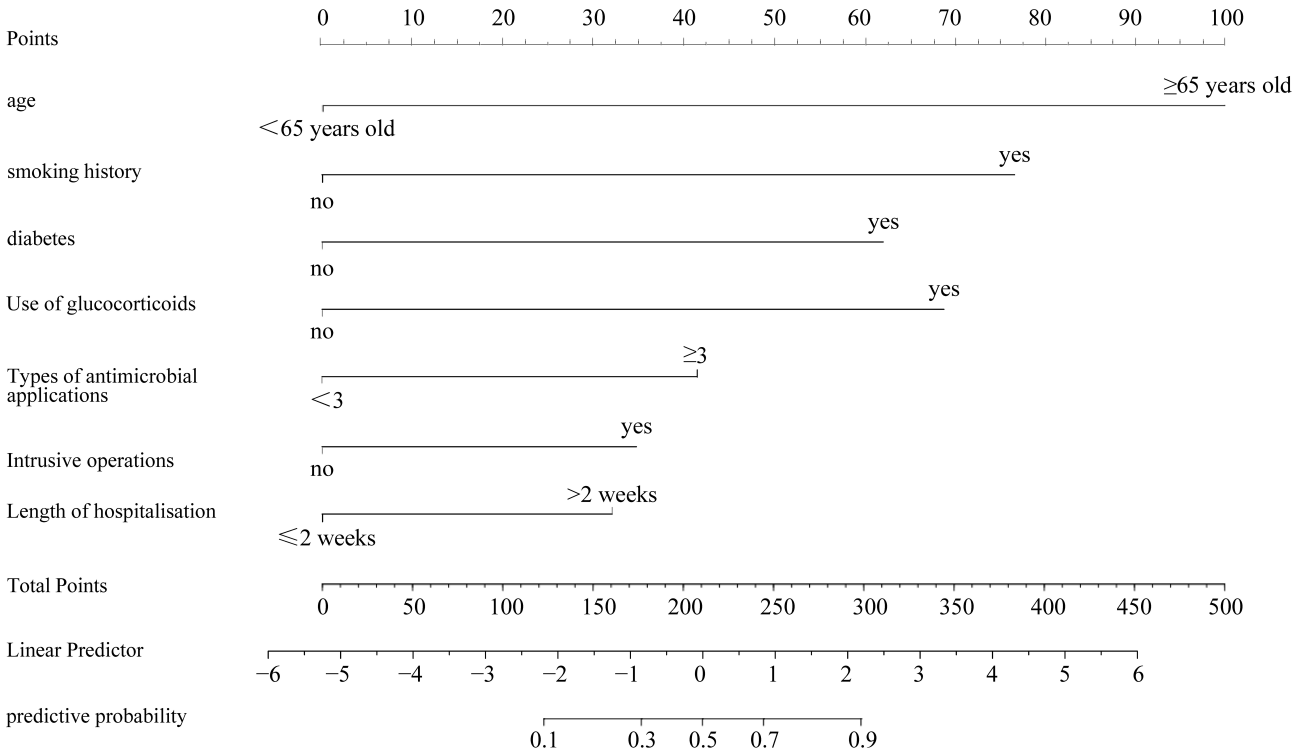

Figure 2 Construction of a nomogram model of pulmonary fungal infection of patients with lung cancer.

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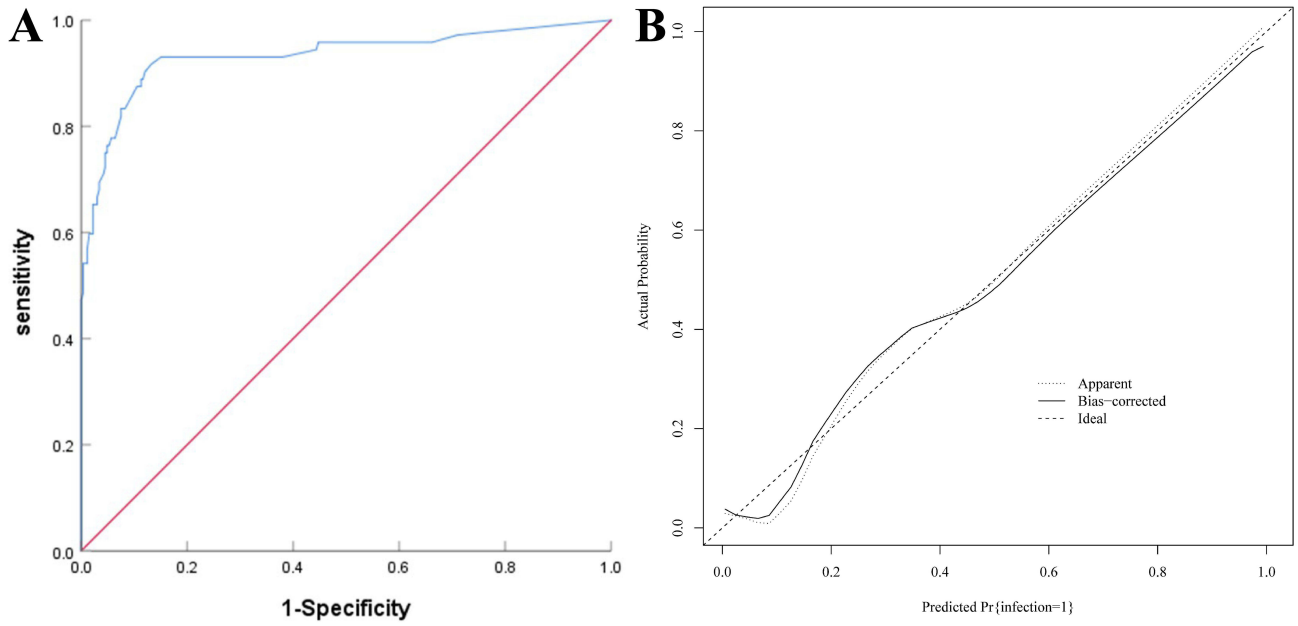

Figure 3 Nomogram Model in the Modeling Group (**A**) ROC curve of modeling group; (**B**) calibration curve of modeling group.

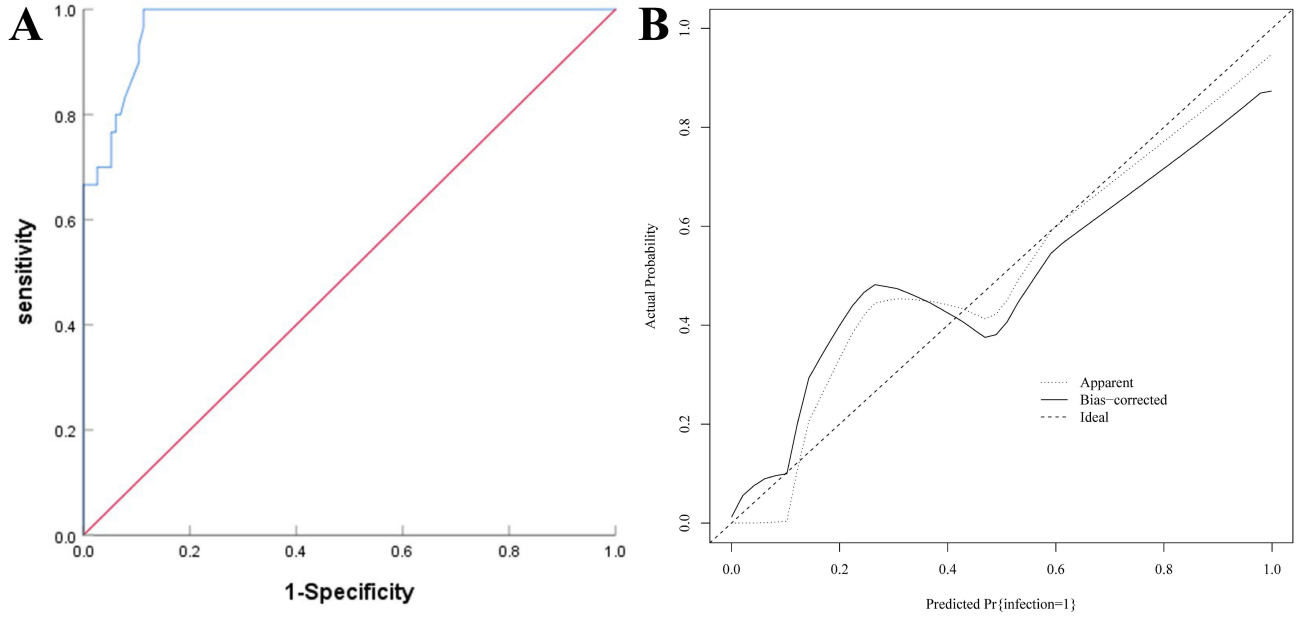

Figure 4 Nomogram Model in the Validation Group (**A**) ROC curve of validation group; (**B**) Calibration curve of validation group.

Decision Curve Analysis Curve of the Nomogram Model

The DCA curve demonstrated that the nomogram model had high clinical utility when the predicted probability ranged from 0.08 to 0.93. The DCA curve is shown in Figure 5.

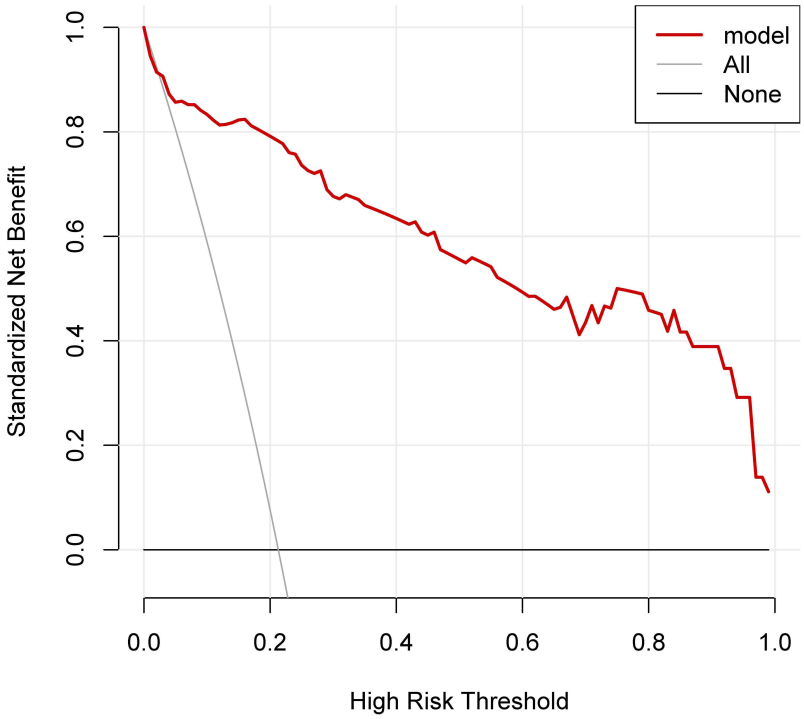

Figure 5 DCA curve for the nomogram.

Discussion

Most lung cancer patients are susceptible to developing pulmonary infections during the progression of the disease. This is primarily due to a reduction in neutrophil phagocytic function, fibrosis of the pulmonary mucosal epithelium, and weakened immunity following treatment. When fungal infections occur, they can accelerate the progression of the patient's condition.[10] Early diagnosis of fungal infections is challenging because patients with impaired immune function fail to mount an adequate inflammatory response, resulting in a lack of typical symptoms of infection.[11] Additionally, with worsening air pollution, the incidence of lung cancer has been gradually increasing. The treatment of lung cancer damages normal cells, and combined with the severity of the disease, the presence of respiratory secretions that cannot be expelled in a timely manner, and a reduced ability to clear foreign substances, the risk of infection is significantly heightened.[12,13] This study found that among the 483 patients, 99 developed a pulmonary fungal infection, resulting in an infection rate of 20.50%. In the modeling group, 72 out of 338 patients developed infections, with an infection rate of 21.30%. This observed incidence was higher than that reported in previous studies (12%),[14] a discrepancy that may be attributed to the potentially older age and more severe underlying conditions of the patients included in this study. Regional differences may also contribute to variations in incidence. Therefore, developing a risk prediction model is of great clinical importance for effective prevention.

This study identified seven independent risk factors influencing pulmonary fungal infections in lung cancer patients through multivariate analysis: age, smoking history, diabetes, use of glucocorticoids, types of antibiotics used, invasive procedures, and hospitalization duration. The reasons for their influence are analyzed as follows: (1) Elderly patients are more prone to infections because the continuous decline of tissues and organs in older individuals weakens their immunity. Furthermore, the presence of multiple comorbidities often reduces the host's ability to resist fungi and other pathogenic microorganisms. The decreased elasticity of alveoli and diminished protective function of the respiratory mucosal barrier further increase the risk of fungal infection.[15,16] Therefore, healthcare providers should enhance treatment and care for elderly patients to effectively prevent infections. (2) Smoking accelerates oxidative stress reactions in the body, activating proto-oncogenes and causing cellular malignancy. Long-term smoking introduces harmful substances such as nicotine, tar, and polycyclic aromatic hydrocarbons, which damage natural killer cells and gradually

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reduce the number of pulmonary cilia. This destruction of the respiratory barrier decreases alveolar macrophage function, thereby increasing the likelihood of fungal infections.[17] (3) High blood glucose levels increase plasma osmolality, which impairs cellular phagocytic and bactericidal functions. The antioxidant defense mechanism of the lungs is also compromised, making infections more likely. Moreover, such patients often experience metabolic imbalances and poor nutritional status, which elevate the risk of fungal infections.[18] Clinically, it is crucial to actively treat comorbidities and provide nutritional support to enhance the body's resistance. (4) Excessive use of glucocorticoids inhibits the production of reactive oxygen intermediates, reduces the phagocytic ability of alveolar macrophages toward fungal spores, and suppresses the elimination of fungal germ spores, thereby increasing the risk of infection.[19] Clinically, it is important to restrict the use of glucocorticoids. (5) Overuse of antibiotics disrupts the gut microbiota, breaking the balance of the original flora and affecting intestinal protein metabolism and vitamin B synthesis. This disrupts the body's functional recovery and increases the risk of pulmonary infections.[20] It is therefore essential to use antibiotics rationally in clinical practice to avoid excessive use that could weaken the body's functions. (6) Invasive procedures, such as catheterization or tracheal intubation, increase infection risk due to the potential for incomplete disinfection during the procedure and local mucosal damage. Such procedures compromise the skin and mucosal barriers, facilitating pathogen invasion.[21] (7) Prolonged hospitalization increases the time patients spend bedridden, can weaken their cough reflex, and may prevent smooth clearance of respiratory secretions. Additionally, extended hospital stays increase exposure to nosocomial pathogens and potentially poorly ventilated wards, all of which elevate the risk of fungal infections.[22]

This study constructed a nomogram model, which yielded AUC values of 0.933 and 0.954 for the modeling and validation groups, respectively. The H-L test indicated good model fit, demonstrating strong predictive discrimination. The DCA curve showed that when the probability ranged from 0.08 to 0.93, the nomogram model had high clinical utility. This model can assist clinicians in prevention strategies by identifying high-risk populations with poor prognosis, thereby reducing the risk of infection.

Furthermore, in this study, data on systemic anti-cancer therapies such as targeted agents, immunotherapy, or anti-angiogenic drugs were not included. Therefore, potential associations between these treatments and pulmonary fungal infections were beyond the scope of our analysis. Additionally, endpoints such as treatment response, long-term prognosis, or overall survival were not assessed. These issues are of high clinical relevance and merit further investigation in future prospective studies with broader datasets.

In conclusion, age, smoking history, diabetes, use of glucocorticoids, types of antibiotics used, invasive procedures, and hospitalization duration are influencing factors for pulmonary fungal infections in lung cancer patients. The nomogram constructed based on these factors effectively predicts the risk of pulmonary fungal infections in this patient population. This study has several limitations. As a retrospective study, there is a potential for selection bias and limitations in data completeness, such as precise dosage information. The sample size is relatively small. Additionally, it is a single-center study. Future research will involve expanding the sample size and conducting prospective multicenter validation to confirm these findings and enhance the generalizability of the model.

Research Involving Human Participants

This retrospective study involving human participants was in accordance with the ethical standards of the Institutional Review Board (or Ethics Committee) of Ganzhou People's Hospital (Approval No. GZSRMYY2024090021) and with the 1964 helsinki Declaration and its later amendments or comparable ethical standards. Written informed consent was obtained from all participants included in this study.

Data Sharing Statement

All relevant data generated or analyzed during this study are included within this published article.

Consent for Publication

All authors have read and approved the final manuscript and give their consent for publication.

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Disclosure

The authors declare that they have no conflicts of interest.

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