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Original Article

Coagulation and inflammatory markers independently predict in-hospital mortality in aspiration pneumonia patients undergoing bronchoalveolar lavage

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Abstract: Objectives: To evaluate the prognostic value of serum coagulation and inflammatory markers for in-hospital mortality in patients with aspiration pneumonia (AP) undergoing bronchoalveolar lavage, and to develop a predictive model. Methods: This retrospective study included 220 AP patients admitted to XianJu People's Hospital between January 2022 and October 2024. Data on demographics, coagulation parameters, inflammatory markers, and in-hospital outcomes were collected. Multivariate logistic regression was used to identify independent predictors of mortality, and a nomogram was constructed based on significant variables. Results: Among the 220 patients, 42 (19.1%) died during hospitalization. Multivariate logistic regression identified age (OR = 1.057, P = 0.006), fibrinogen (FIB; OR = 1.456, P = 0.002), D-dimer (OR = 2.414, P < 0.001), leukocyte count (OR = 1.128, P = 0.027), and procalcitonin (PCT; OR = 9.240, P < 0.001) as independent predictors of in-hospital mortality. The nomogram model incorporating these variables demonstrated good discriminative ability with an area under the curve of 0.835. Calibration plots and decision curve analysis further confirmed the model's accuracy and clinical utility. Conclusion: Age, FIB, D-dimer, leukocyte count, and PCT are independent predictors of in-hospital mortality in AP patients undergoing bronchoalveolar lavage. The nomogram based on these markers shows strong predictive performance and may facilitate individualized risk assessment and clinical decision-making.

Keywords: Aspiration pneumonia, coagulation function, inflammation, D-dimer, procalcitonin, predictive model

Introduction

Aspiration pneumonia (AP) is a pulmonary infection caused by the inhalation of gastric contents, oropharyngeal secretions, or foreign material into the lower respiratory tract. It is particularly common in elderly individuals and in patients with dysphagia, impaired consciousness, or neurodegenerative disorders [1, 2]. A retrospective cohort study based on medical insurance claims in China reported that the 6-month mortality rate for AP patients exceeds 35%, with 1-year mortality reaching up to 42% [3]. Despite the widespread use of antibiotics and respiratory support, many AP patients continue to have poor clinical outcomes. This is often attributed to severe pulmonary infection, coagulation dysfunction, multiple organ failure, or complications such as acute respiratory distress syndrome, leading to refractory hypoxemia, systemic inflammation, and ultimately death [1, 4].

Bronchoalveolar lavage (BAL) is widely used in the management of AP, initially as a diagnostic tool for microbiological sampling. In recent years, its role has expanded to include therapeutic applications, especially in severe cases, where it is employed to remove aspirated material, improve airway clearance, reduce pulmonary inflammation, and potentially enhance oxygenation and clinical outcomes [1, 5, 6]. However, despite these potential benefits, some patients experience disease progression or mortality during or after BAL treatment [5]. Given the high in-hospital mortality - particularly in severe cases - identifying reliable prognostic factors in AP patients undergoing BAL is

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essential. Previous studies have identified several factors associated with mortality in AP, including advanced age, comorbidities, infection severity, and airway management strategies [7, 8]. However, prognostic indicators specific to patients receiving BAL remain poorly understood.

Coagulation dysfunction and inflammation are known to contribute significantly to disease progression in pneumonia and other respiratory conditions, by exacerbating tissue damage, impairing lung function, and worsening clinical outcomes [1, 9-11]. In infectious diseases, the coagulation system plays a dual role - participating in thrombus formation and amplifying the inflammatory response - thereby promoting microvascular injury, tissue hypoxia, and multiorgan failure [10, 12]. Abnormalities in coagulation parameters, including prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen (FIB), and D-dimer, have been closely linked to the severity of pneumonia [10]. Additionally, inflammatory markers such as leukocyte count, neutrophil count, lymphocyte count, C-reactive protein (CRP), and procalcitonin (PCT) are frequently elevated in AP and reflect the magnitude of systemic inflammation. These markers not only aid in diagnosis but also serve as important prognostic indicators in community-acquired pneumonia (CAP) [1, 13, 14]. However, there is a lack of comprehensive evaluation regarding the impact of coagulation and inflammatory markers on in-hospital mortality specifically in AP patients undergoing BAL, and their predictive value remains uncertain.

Therefore, this study aimed to assess the prognostic significance of serum coagulation and inflammatory markers in predicting in-hospital mortality among AP patients treated with BAL. Given the absence of a validated predictive model for this patient population, we developed a nomogram based on independently associated risk factors. This tool may assist in early risk stratification, support clinical decision-making, and ultimately improve outcomes in high-risk AP patients.

Materials and methods

Patient selection

This retrospective study analyzed the clinical records of 220 AP patients admitted to XianJu People's Hospital between January 2022 and October 2024. Patients were divided into two groups based on in-hospital mortality status, with 178 patients in the survivor group (those discharged alive from the hospital) and 42 patients in the non-survivor group (those who died during hospitalization).

Inclusion criteria were as follows: age ≥ 18 years; a primary discharge diagnosis of AP; and receipt of BAL therapy.

Exclusion criteria included: concurrent infections in other organs; history of severe organ dysfunction; long-term use of immunosuppressive agents or known immune disorders; malignancy; or end-stage disease.

The diagnostic criteria for AP included radiographic or clinical evidence of pneumonia, presence of predisposing risk factors such as altered consciousness or swallowing dysfunction, and clinical signs of aspiration-such as irritative cough, sudden dyspnea, presence of food particles in sputum, fever, or tachycardia [15].

Because standardized swallowing assessments were unavailable for all patients, those exhibiting overt signs of dysphagia or a documented history of aspiration were considered to have swallowing dysfunction [16].

This study was approved by the Ethics Committee of XianJu People's Hospital.

Data collection

Demographic and clinical data were collected, including age, sex, educational attainment, marital status, residence (urban or rural), history of alcohol use, smoking status, and comorbidities (e.g., diabetes, chronic obstructive pulmonary disease, stroke, hypertension), along with in-hospital mortality status. Coagulation parameters included PT, APTT, FIB, and D-dimer. Inflammatory markers included CRP, PCT lymphocyte count, leukocyte count, neutrophil count, and lymphocyte count.

Outcome measures

All laboratory tests were performed using standardized automated equipment. Coagulation parameters (PT, APTT, FIB, D-dimer) were measured using an automated coagulation analyzer (CX-9000, Mindray). Hematological parameters

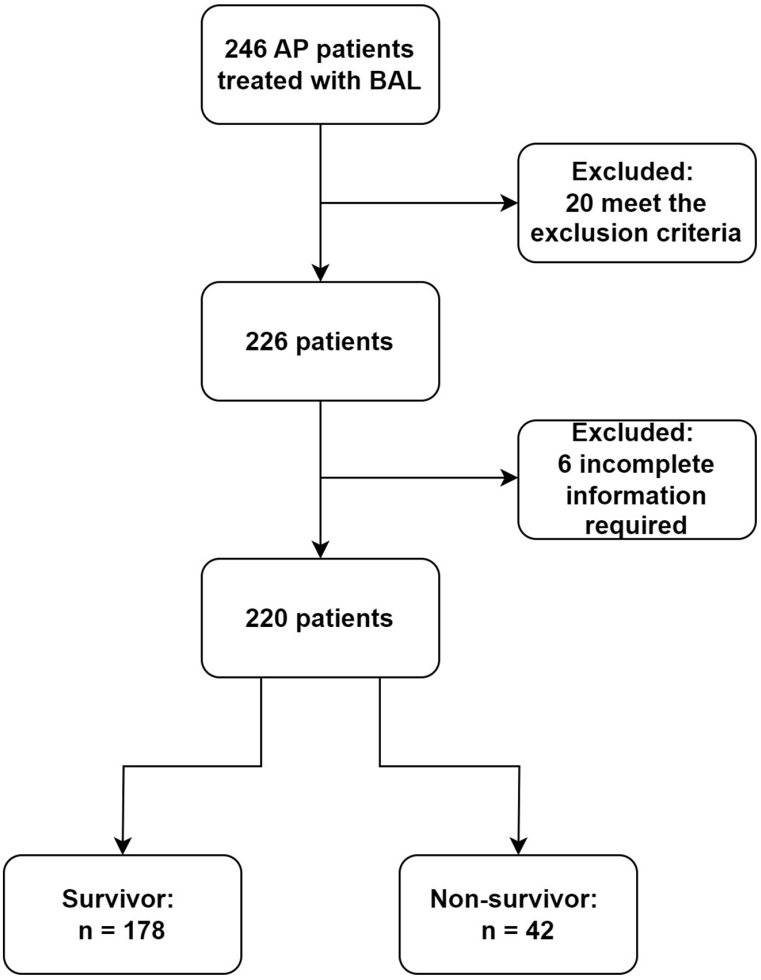

Figure 1. Flow chart for the patient selection. AP: Aspiration pneumonia; BAL: Bronchoalveolar lavage.

(leukocytes, neutrophils, lymphocytes) were analyzed using an automated hematology analyzer (BC-760 CS, Mindray). Inflammatory markers (CRP, PCT) were measured using an automated biochemical analyzer (BS-1000M, Mindray).

Statistical analysis

Continuous variables were represented as mean ± standard deviation (SD). Depending on the distribution, independent sample t-tests or nonparametric methods were used for group comparisons. Categorical variables were presented as counts and percentages, with group differences assessed using the chi-square test. Univariate and multivariate logistic regression analyses were performed to identify prognostic factors associated with in-hospital mortality in AP patients. Variables with P < 0.1 in the univariate analysis were entered into the multivariate model. A backward stepwise selection approach was employed to determine the final set of independent predictors. The predictive model was evaluated using the area under the receiver operating characteristic curve (AUC), calibration plots, and decision curve analysis (DCA) to assess its discriminative ability, calibration, and clinical utility. All statistical analyses were conducted using SPSS version 27.0, and a *P*-value < 0.05 was considered statistically significant.

Results

Comparison of baseline characteristics between survivor and non-survivor groups

As shown in **Figure 1**, a total of 220 patients were included in the final analysis after screening. **Table 1** presents the baseline characteristics of the AP patients. The median age of the non-survivor group was significantly higher than that of the survivor group (74.00 [68.00, 79.75] vs. 67.00 [60.25, 75.00], P = 0.004), suggesting that advanced age is associated with an increased risk of in-hospital mortality. There were no statistically significant differences between the groups in terms of sex, BMI, educational level, marital status, residence, smoking and alcohol history, or comorbidities such as diabetes, hypertension, and stroke (all P > 0.05).

Comparison of coagulation parameters between survivor and non-survivor groups

Table 2 summarizes the coagulation parameters, including PT, APTT, FIB, and D-dimer. Compared to survivors, non-survivors had significantly lower APTT values (31.60 [28.88, 34.77] vs. 34.15 [30.10, 37.30], P = 0.024),

Table 1. Comparison of Baseline information

Variables	Survivor (n = 178)	Non-survivor (n = 42)	$t/Z/\chi^2$	P
Age, M (Q_1, Q_3)	67.00 (60.25, 75.00)	74.00 (68.00, 79.75)	-2.887	0.004
Sex, n (%)			0.074	0.785
Male	119 (66.85)	29 (69.05)		
Female	59 (33.15)	13 (30.95)		
BMI, Mean ± SD	20.67 ± 1.82	20.36 ± 2.22	0.922	0.358
Education, n (%)			-	0.778
Primary school and below	100 (56.18)	26 (61.90)		
Junior high school	54 (30.34)	10 (23.81)		
Senior high school	18 (10.11)	4 (9.52)		
College and above	6 (3.37)	2 (4.76)		
Current marital status			0.134	0.715
Married	103 (57.87)	23 (54.76)		
Unmarried	75 (42.13)	19 (45.24)		
Place of residence			0.565	0.452
Rural	86 (48.31)	23 (54.76)		
Urban	92 (51.69)	19 (45.24)		
Smoking, n (%)			0.124	0.724
Yes	107 (60.11)	24 (57.14)		
No	71 (39.89)	18 (42.86)		
Alcohol consumption, n (%)			0.929	0.335
Yes	91 (51.12)	18 (42.86)		
No	87 (48.88)	24 (57.14)		
Diabetes, n (%)			0.032	0.858
Yes	79 (44.38)	18 (42.86)		
No	99 (55.62)	24 (57.14)		
Hypertension, n (%)			0.308	0.579
Yes	89 (50.00)	19 (45.24)		
No	89 (50.00)	23 (54.76)		
Stroke history, n (%)			1.458	0.227
Yes	32 (17.98)	11 (26.19)		
No	146 (82.02)	31 (73.81)		

BMI: Body mass index.

Table 2. Comparison of coagulation parameters

Variables	Survivor (n = 178)	Non-survivor (n = 42)	Z	P
PT, M (Q_1, Q_3)	12.90 (10.83, 14.97)	11.35 (10.20, 13.40)	-1.664	0.096
APTT, M (Q_1, Q_3)	34.15 (30.10, 37.30)	31.60 (28.88, 34.77)	-2.252	0.024
FIB, M (Q_1, Q_3)	4.00 (2.50, 5.40)	5.55 (3.73, 6.40)	-3.964	< .001
D-dimer, M (Q_1, Q_3)	0.95 (0.64, 1.17)	1.38 (1.10, 1.93)	-4.818	< .001

PT: Prothrombin time; APTT: Activated partial thromboplastin time; FIB: Fibrinogen.

indicating a potential trend toward hypercoagulability. Additionally, FIB and D-dimer levels were markedly elevated in the non-survivor group (FIB: 5.55 [3.73, 6.40] vs. 4.00 [2.50, 5.40], P < 0.001; D-dimer: 1.38 [1.10, 1.93] vs. 0.95 [0.64, 1.17], P < 0.001), suggesting excessive coagulation activity may contribute to poor outcomes. PT did not differ significantly between the groups (P = 0.096).

Table 3. Comparison of inflammation indicators

Variables	Survivor (n = 178)	Non-survivor (n = 42)	t/Z	P
Neutrophils, Mean ± SD	8.89 ± 2.97	9.79 ± 4.66	-1.193	0.239
Leucocytes, Mean ± SD	11.25 ± 3.79	12.55 ± 5.00	-1.585	0.119
Lymphocytes, M (Q_1, Q_3)	1.01 (0.82, 1.23)	0.90 (0.77, 1.12)	-1.584	0.113
CRP, M (Q_1, Q_3)	70.00 (44.25, 119.00)	98.50 (84.25, 110.75)	-2.341	0.019
PCT, M (Q_1, Q_3)	0.88 (0.67, 1.05)	1.13 (0.87, 1.42)	-4.405	< .001

CRP: C-reactive protein; PCT: Procalcitonin.

Comparison of inflammatory markers between survivor and non-survivor groups

As shown in **Table 3**, inflammatory markers such as CRP and PCT were significantly higher in the non-survivor group (CRP: 98.50 [84.25, 110.75] vs. 70.00 [44.25, 119.00], P = 0.019; PCT: 1.13 [0.87, 1.42] vs. 0.88 [0.67, 1.05], P < 0.001), reflecting a more severe systemic inflammatory response. Although differences in neutrophil count (P = 0.239), leukocyte count (P = 0.119), and lymphocyte count (P = 0.113) were not statistically significant, these markers tended to be elevated in the non-survivor group, potentially indicating greater infection severity or immune dysregulation.

Univariate logistic regression analysis of in-hospital mortality

To identify potential risk factors for in-hospital mortality, univariate logistic regression analysis was performed (**Table 4**). Age (OR = 1.039, 95% CI: 1.006-1.072, P = 0.018), FIB (OR = 1.446, 95% CI: 1.195-1.751, P < 0.001), D-dimer (OR = 2.500, 95% CI: 1.548-4.037, P < 0.001), and PCT (OR = 15.297, 95% CI: 4.396-53.228, P < 0.001) were significantly associated with mortality risk. Among them, PCT showed the highest OR, suggesting it may be a strong predictor of poor prognosis. Although leukocyte count (OR = 1.085, 95% CI: 0.995-1.182, P = 0.063) and CRP (OR = 1.007, 95% CI: 1.000-1.015, P = 0.058) did not reach statistical significance, they showed a potential trend toward association with mortality.

Multivariate logistic regression analysis of in-hospital mortality

Multivariate logistic regression was conducted on variables with P < 0.1 in the univariate analysis (**Table 5**). The analysis identified age (OR = 1.057, 95% CI: 1.016-1.099, P = 0.006), FIB (OR = 1.456, 95% CI: 1.150-1.844, P = 0.002), D-dimer (OR = 2.414, 95% CI: 1.458-3.997, P < 0.001), leukocyte count (OR = 1.128, 95% CI: 1.014-1.256, P = 0.027), and PCT (OR = 9.240, 95% CI: 2.486-34.351, P < 0.001) as independent predictors of in-hospital mortality. Among these, PCT and D-dimer exhibited the highest odds ratios, underscoring their potential clinical importance in risk stratification.

Construction of the nomogram prediction model

Based on the multivariate regression results, a nomogram was constructed (**Figure 2**) to predict in-hospital mortality in AP patients. The model incorporates five key variables-age, FIB, D-dimer, leukocyte count, and PCT - allowing individualized risk assessment and informing treatment decisions.

Validation and performance evaluation of the nomogram

To evaluate model performance, ROC analysis, calibration curve analysis, and DCA were conducted (**Figure 3**). As shown in **Figure 3A**, the nomogram yielded an AUC of 0.835, indicating excellent discriminative ability. The calibration curve (**Figure 3B**) demonstrated strong agreement between predicted and observed outcomes, indicating good model calibration. DCA (**Figure 3C**) further confirmed the model's clinical utility across a range of threshold probabilities, supporting its application in real-world clinical settings.

Discussion

This study retrospectively analyzed data from 220 AP patients who underwent BAL to evaluate the prognostic significance of serum coagulation and inflammatory markers in predicting in-hospital mortality. The findings identified

Table 4. Univariate logistic regression

Variables	β	S.E	Z	P	OR (95% CI)
Age, M (Q_1, Q_3)	0.038	0.016	2.361	0.018	1.039 (1.006-1.072)
Sex, n (%)					
Male					1.000 (Reference)
Female	-0.101	0.370	-0.272	0.785	0.904 (0.438-1.866)
BMI	-0.085	0.092	-0.922	0.356	0.918 (0.766-1.100)
Education, n (%)					
Primary school and below					1.000
Junior high school	-0.339	0.409	-0.830	0.406	0.712 (0.320-1.587)
Senior high school	-0.157	0.595	-0.264	0.792	0.855 (0.266-2.743)
College and above	0.248	0.846	0.294	0.769	1.282 (0.244-6.726)
Current marital status					
Married					1.000
Unmarried	-0.126	0.345	-0.365	0.715	0.881 (0.448, 1.734)
Place of residence					
Rural					1.000
Urban	-0.258	0.344	-0.750	0.453	0.772 (0.393, 1.517)
Smoking, n (%)					
Yes					1.000
No	0.122	0.347	0.353	0.724	1.130 (0.572-2.233)
Alcohol consumption, n (%)					
Yes					1.000
No	0.333	0.346	0.961	0.336	1.395 (0.708-2.748)
Diabetes, n (%)					
Yes					1.000
No	0.062	0.346	0.179	0.858	1.064 (0.540-2.098)
Hypertension, n (%)					
Yes					1.000
No	0.191	0.344	0.555	0.579	1.211 (0.616-2.377)
Stroke history, n (%)					
Yes					1.000
No	-0.482	0.402	-1.200	0.230	0.618 (0.281-1.357)
PT	-0.103	0.063	-1.633	0.102	0.903 (0.798-1.021)
APTT	-0.048	0.031	-1.535	0.125	0.953 (0.897-1.013)
FIB	0.369	0.097	3.787	< .001	1.446 (1.195-1.751)
D-dimer	0.916	0.245	3.747	< .001	2.500 (1.548-4.037)
Neutrophils	0.081	0.052	1.551	0.121	1.084 (0.979-1.200)
Leucocytes	0.082	0.044	1.856	0.063	1.085 (0.995-1.182)
Lymphocytes	-0.082	0.166	-0.496	0.620	0.921 (0.666-1.274)
CRP	0.007	0.004	1.897	0.058	1.007 (1.000-1.015)
PCT	2.728	0.636	4.287	< .001	15.297 (4.396-53.228)

BMI: Body mass index; PT: Prothrombin time; APTT: Activated partial thromboplastin time; FIB: Fibrinogen; CRP: C-reactive protein; PCT: Procalcitonin.

age, FIB, D-dimer, leukocyte count, and PCT as independent predictors of in-hospital death, with PCT and D-dimer demonstrating the strongest associations. A nomogram model incorporating these variables showed good predictive performance, offering a practical tool for individualized risk assessment in AP patients. These results support the early identification of

Table 5. Multivariate logistic regression

Variables	β	S.E	Z	P	OR (95% CI)
Age	0.055	0.020	2.748	0.006	1.057 (1.016-1.099)
FIB	0.376	0.120	3.123	0.002	1.456 (1.150-1.844)
D-dimer	0.881	0.257	3.425	< .001	2.414 (1.458-3.997)
Leucocytes	0.121	0.055	2.210	0.027	1.128 (1.014-1.256)
PCT	2.224	0.670	3.319	< .001	9.240 (2.486-34.351)

FIB: Fibrinogen; PCT: Procalcitonin.

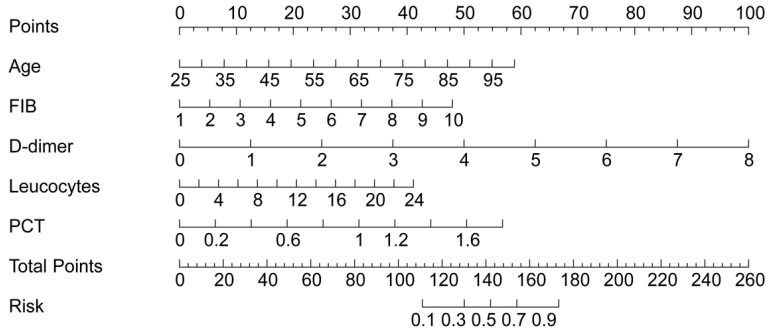

Figure 2. Nomogram prediction model. FIB: Fibrinogen; PCT: Procalcitonin.

high-risk individuals and provide a scientific basis for optimizing clinical management strategies.

Elderly individuals are particularly vulnerable to aspiration due to age-related immune decline, swallowing dysfunction, and a greater burden of comorbidities, contributing to increased infection-related mortality [2, 17]. Additionally, aging is associated with impaired antibacterial defense and dysregulated inflammatory responses, which can result in more severe pulmonary inflammation and systemic damage [1]. Previous studies have reported that older patients receiving percutaneous endoscopic gastrostomy are at higher risk for AP, with a greater prevalence in males [18]. In this study, the in-hospital mortality rate among AP patients undergoing BAL was approximately 19%, and non-survivors were significantly older than survivors-consistent with earlier research [5, 19]. Furthermore, age was confirmed as an independent predictor of mortality, aligning with findings from studies on CAP and hospital-acquired pneumonia [20]. These results highlight the need for preventive strategies in high-risk elderly populations, including swallowing assessments, nutritional support, aspiration prevention, and early antimicrobial therapy to reduce mortality.

Coagulation dysfunction is a well-documented feature in various forms of pneumonia. Studies have shown that both Mycoplasma pneumoniae and COVID-19 pneumonia can induce hypercoagulable states [9, 21, 22]. In severe pneumonia, coagulopathy is closely linked to disease progression, with elevated D-dimer levels serving as a predictor of adverse outcomes, including mortality [10]. Moreover, the combined assessment of D-dimer, FIB, and inflammatory markers has been shown to aid in predicting mortality, ICU admission, and venous thromboembolism in COVID-19 pneumonia patients [9, 23].

In this study, FIB and D-dimer levels were significantly elevated in non-survivors and identified as independent predictors of in-hospital mortality. Compared to other pneumonia types, coagulation abnormalities in AP may be more pronounced, potentially due to unique pathophysiological mechanisms. Aspiration-induced microvascular injury may trigger the coagulation cascade, resulting in pulmonary microthrombi, impaired circulation, and exacerbated infection [24]. Furthermore, AP may induce a more severe systemic inflammatory response syndrome (SIRS), aggravating coagulation system dysfunction, which could explain the strong correlation between elevated D-dimer and FIB levels and mortality risk observed in this study [25]. Therefore, dynamic monitoring of D-dimer and FIB levels may assist in identifying high-risk patients and guiding anticoagulation therapy.

Consistent with our findings, APTT, is often reduced in hypercoagulable states. However, a recent study of elderly patients with severe

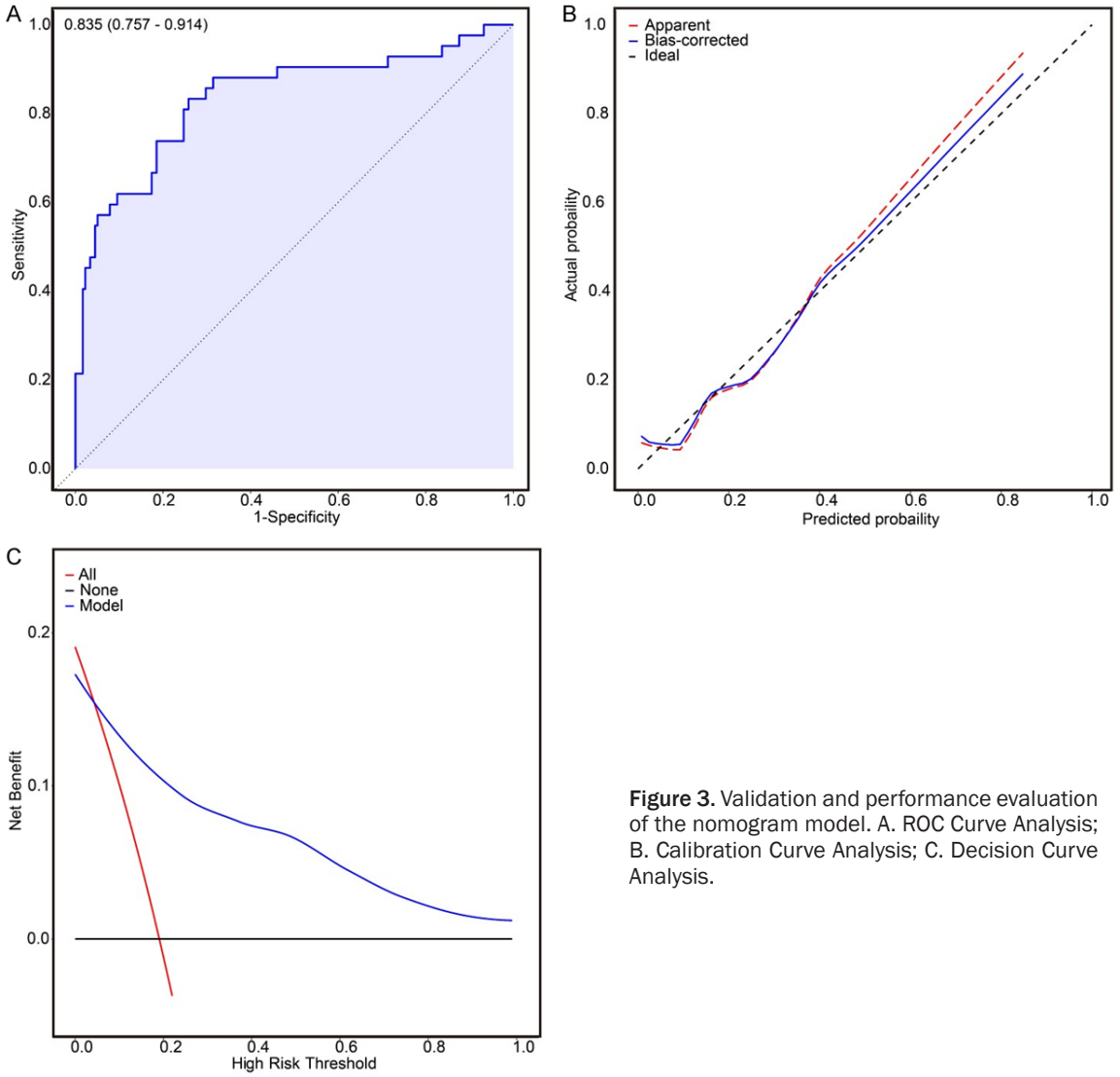

Figure 3. Validation and performance evaluation of the nomogram model. A. ROC Curve Analysis; B. Calibration Curve Analysis; C. Decision Curve Analysis.

pneumonia found that those who died within 28 days of admission had longer APTT values at baseline compared to survivors [10, 21]. This may reflect coagulation factor depletion in the setting of severe disease, especially when complicated by disseminated intravascular coagulation or consumptive coagulopathy, ultimately leading to prolonged APTT despite underlying hypercoagulability [9].

The inflammatory response is the body's primary defense against pathogen invasion or tissue injury [1]. As an open system, the lungs are particularly susceptible to inhaled pathogens and aspirated material, making inflammation a central feature of respiratory diseases, especially pneumonia [1, 26, 27]. In pneumonia, immune activation leads to the recruitment of neutrophils, macrophages, and dendritic cells, triggering the release of cytokines, chemokines, and complement activation. These responses increase vascular permeability and disrupt the alveolar-capillary barrier, causing pulmonary edema and impaired gas exchange - resulting in the hallmark symptoms of pneumonia, including fever, cough, dyspnea, and hypoxemia [26]. Excessive cytokine production may result in a cytokine storm, contributing to severe lung injury and systemic immune dysregulation [28]. Additionally, as previously discussed, inflammation and coagulation are mutually reinforcing: inflammation promotes thrombosis, while hypercoagulability exacerbates inflammation, creating a vicious cycle that may culminate in SIRS [11, 12].

Leukocytes play a critical role in the innate immune system by phagocytosing pathogens, releasing inflammatory mediators, and regulating immune responses. They are also widely recognized as sensitive markers of inflammation [26, 29]. In pneumonia, elevated neutrophil and leukocyte counts often reflect the severity of infection and the host's immune response to invading pathogens [9]. In the present study, leukocyte count emerged as a significant predictor of in-hospital mortality among AP patients. Although leukocytes are essential for host defense, excessive activation can contribute to tissue injury. Neutrophils, in particular, are known to release reactive oxygen species and proteolytic enzymes, which can disrupt the alveolar-capillary barrier, promote pulmonary edema, and impair gas exchange-mechanisms that may underlie the observed association between elevated leukocyte levels and poor prognosis in AP patients undergoing BAL [30].

PCT and CRP are widely used inflammatory biomarkers induced by infection and are commonly employed for early diagnosis and severity assessment in a variety of infectious diseases [9, 31]. Previous studies have shown that CRP levels correlate closely with pneumonia severity and that elevated CRP is associated with increased mortality [14, 32]. PCT has also been identified as a reliable predictor of ICU admission in COVID-19 patients [9]. Consistent with these findings, our study demonstrated significantly higher PCT levels in non-survivors, and multivariate analysis confirmed PCT as an independent predictor of in-hospital mortality. These results underscore the importance of accurately assessing inflammatory status in pneumonia management and highlight the potential of individualized treatment strategies tailored to a patient's inflammatory profile.

Despite its valuable findings, this study has several limitations. First, as a retrospective single-center study, it is subject to inherent selection bias. Larger, multicenter prospective studies are required to validate these results. Second, the study assessed a limited set of coagulation and inflammatory markers; other potentially relevant biomarkers - such as cytokines and coagulation regulatory proteins - which were not included. Third, the analysis did not consider the impact of treatment variables (e.g., antibiotic regimens, supportive interventions) on patient outcomes, which may confound the interpretation of results. Future studies should include a broader range of biomarkers and integrate treatment-related factors to develop more robust and clinically applicable predictive models. Lastly, the nomogram developed in this study was only internally validated; external validation is needed to confirm its generalizability.

Conclusion

This study identified age, FIB, D-dimer, leucocyte count, and PCT as independent predictors of in-hospital mortality in AP patients undergoing BAL. Among these, PCT and D-dimer demonstrated particularly strong predictive value. The nomogram model incorporating these variables achieved good predictive performance, making it a valuable tool for individualized risk assessment. This model can facilitate early identification of high-risk patients and support the development of optimized treatment strategies in clinical practice.

Disclosure of conflict of interest

None.

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