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DEVELOPMENT AND VALIDATION OF A PREDICTION MODEL FOR IN-HOSPITAL MORTALITY IN PATIENTS WITH SEVERE PNEUMONIA: A RETROSPECTIVE COHORT STUDY

RAZVOJ I VALIDACIJA MODELA ZA PREDVIĐANJE BOLNIČKE SMRTNOSTI KOD PACIJENATA SA TEŠKOM PNEUMONIJOM: RETROSPEKTIVNA KOHORTNA STUDIJA

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Summary

Background: Severe pneumonia is a major cause of mortality worldwide, and accurate early risk assessment is clinically important. This study aimed to develop and validate a mortality prediction model based on routinely available clinical variables.
Methods: In this retrospective cohort study, we enrolled 300 patients with severe pneumonia: 200 in the training cohort and 100 in the validation cohort. Univariate analysis and least absolute shrinkage and selection operator (LASSO) regression were used to identify nine key predictors for model development. Model performance was evaluated using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA) in both cohorts. Multivariable logistic regression was further performed, and a nomogram was constructed for mortality prediction.
Results: Baseline characteristics were comparable between the training and validation cohorts. The prediction model demonstrated good discrimination in the training cohort,

Kratak sadržaj

Uvod: Teška pneumonija predstavlja jedan od značajnih uzroka smrtnosti širom sveta, zbog čega je pravovremena i precizna procena rizika od smrtnog ishoda od velikog kliničkog značaja. Cilj ove studije je bio razvoj i validacija modela za predviđanje smrtnog ishoda na osnovu rutinski dostupnih kliničkih parametara.
Metode: U ovu retrospektivnu kohortnu studiju je uključeno 300 pacijenata sa teškom pneumonijom, od kojih je 200 činilo kohortu za razvoj modela, a 100 kohortu za validaciju. Za identifikaciju devet ključnih prediktora korišćene su univarijantna analiza i regresija metodom najmanjeg apsolutnog skupljanja i selekcije (LASSO). Performanse modela su procenjene pomoću ROC krivih, kalibracionih krivih i analize krive odlučivanja (DCA) u obe kohorte. Dodatno je sprovedena multivarijantna logistička regresija, a za predviđanje smrtnog ishoda konstruisan je nomogram.
Rezultati: Početne karakteristike pacijenata bile su uporedive između kohorte za razvoj modela i kohorte

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List of abbreviations: ALT, alanine aminotransferase; ALB, albumin; AUC, area under the curve; BASO, basophils; CI, confidence interval; Cr, creatinine; DBP, diastolic blood pressure; DCA, decision curve analysis; EO, eosinophils; FIB, fibrinogen; GLB, globulin; HCO_3^-, bicarbonate; HGB, haemoglobin; ICU, intensive care unit; IL-6, interleukin-6; IQR, interquartile range; LASSO, least absolute shrinkage and selection operator; LYM, lymphocytes; MONO, monocytes; NEU, neutrophils; OR, odds ratio; $PaCO_2$, partial pressure of arterial carbon dioxide; PaO_2, partial pressure of arterial oxygen; PCT, procalcitonin; PLT, platelet count; PSI, Pneumonia Severity Index; PTA, prothrombin activity; ROC, receiver operating characteristic; SBP, systolic blood pressure; SD, standard deviation; TB, total bilirubin; WBC, white blood cell count.

with an area under the ROC curve (AUC) of 0.855 (95% CI: 0.801–0.908), and acceptable discrimination in the validation cohort, with an AUC of 0.750 (95% CI: 0.640–0.861). Calibration and decision curve analyses indicated favourable clinical utility. Multivariable logistic regression identified age, coronary heart disease, pulmonary hypertension, and mechanical ventilation as independent predictors of in-hospital mortality in severe pneumonia.
Conclusion: The proposed nomogram may serve as a preliminary tool for estimating in-hospital mortality risk in patients with severe pneumonia. External validation in independent populations is required before clinical application.

Keywords: severe pneumonia, in-hospital mortality, prediction model

za validaciju. Model je pokazao dobru diskriminativnu sposobnost u kohorti za razvoj, sa površinom ispod ROC krive (AUC) od 0,855 (95% CI: 0,801–0,908), dok je u kohorti za validaciju utvrđena zadovoljavajuća diskriminativna sposobnost, sa AUC vrednošću od 0,750 (95% CI: 0,640–0,861). Rezultati kalibracione analize i analize krive odlučivanja su ukazali na dobru kliničku primenljivost modela. Multivarijantnom logističkom regresijom je utvrđeno da su starost, koronarna bolest srca, plućna hipertenzija i mehanička ventilacija nezavisni prediktori bolničkog smrtnog ishoda kod pacijenata sa teškom pneumonijom.
Zaključak: Predloženi nomogram može da posluži kao preliminarni alat za procenu rizika od smrtnog ishoda tokom hospitalizacije kod pacijenata sa teškom pneumonijom. Pre njegove primene u kliničkoj praksi neophodna je eksterna validacija na nezavisnim populacijama.

Ključne reči: teška pneumonija, bolnička smrtnost, model za predviđanje

Introduction

Severe pneumonia remains a major contributor to hospital mortality worldwide, with the greatest burden observed in older adults and patients with substantial comorbidity (1, 2). Although antimicrobial therapy, respiratory support, and critical care have advanced considerably, outcomes in this population remain unsatisfactory. Reported in-hospital mortality rates still range from 20% to 50% across studies (3–5). Accurate early risk estimation is therefore important for identifying patients who may benefit from closer monitoring, timely escalation of care, and individualized treatment strategies (6).

Several clinical tools, including CURB-65 and the Pneumonia Severity Index (PSI), are widely used to assess risk in community-acquired pneumonia (7, 8). However, their performance may be suboptimal in patients with severe pneumonia. This is particularly relevant for patients requiring mechanical ventilation or intensive care unit (ICU) management, in whom disease progression is rapid and clinical heterogeneity is substantial (9, 10). Moreover, models that depend on complex or subjective variables may be difficult to apply promptly in emergency and critical care settings (11). However, these conventional scoring systems were mainly developed for general pneumonia risk stratification. They may not fully incorporate patient-specific comorbidities, laboratory abnormalities, and treatment-related indicators relevant to mortality prediction in severe pneumonia. Therefore, models integrating routinely available clinical variables may provide complementary information for individualized risk assessment.

Data-driven prediction models have increasingly been used to improve prognostic assessment. The least absolute shrinkage and selection operator (LASSO) method helps select predictors from multiple candidate variables while reducing overfitting (12–14). Multivariable logistic regression can then provide an interpretable framework for estimating outcome risk. As a nomogram, such a model may further support individualized risk communication and bedside decision-making (15). However, existing prognostic models for pneumonia often differ in target populations, variable selection, and validation strategy, limiting their clinical generalizability (16).

In this single-centre retrospective cohort study, we integrated LASSO regression and multivariable logistic regression to identify clinically relevant predictors of in-hospital mortality in patients with severe pneumonia. We then developed and validated a practical nomogram-based prediction model to support early risk stratification and individualized management.

Materials and Methods

Study design and participants

This retrospective study included 300 patients with severe pneumonia admitted to Peking University Shenzhen Hospital between December 2020 and December 2025. The sample size was determined by the number of eligible patients available during the predefined study period rather than by an a priori sample size calculation. All eligible patients were included and randomly divided into a training cohort (n=200) and an internal validation cohort (n=100) at a 2:1 ratio. This study did not include an external validation cohort.

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All diagnoses were based on the Chinese Guidelines for the Diagnosis and Treatment of Adult Community-Acquired Pneumonia (2016 Edition) and established criteria for severe pneumonia (17). Community-acquired pneumonia was defined as pulmonary parenchymal infection occurring outside the hospital or within 48 h of admission, diagnosed according to clinical symptoms, laboratory findings, and chest imaging.

Severe pneumonia was defined using major and minor criteria (18). Major criteria included invasive mechanical ventilation or septic shock requiring vasopressors. Minor criteria included respiratory rate ≥30 breaths/min, PaO_2/FiO_2 ≤250 mmHg, multilobar infiltrates, altered mental status, azotemia, leukopenia, thrombocytopenia, hypothermia, or hypotension requiring aggressive fluid resuscitation. Patients meeting at least one major criterion or multiple minor criteria were classified as having severe pneumonia. For patients with mechanical ventilation, ICU admission, shock, severe hypoxemia, or multiple organ dysfunction, clinicians adjudicated the diagnosis according to predefined clinical, imaging, and laboratory criteria.

The primary endpoint was in-hospital mortality. The Ethics Committee of Peking University Shenzhen Hospital approved the study. The Ethics Committee waived the requirement for informed consent because this retrospective study used anonymized data.

Inclusion and exclusion criteria

Patients were eligible if they were aged 18 years or older, met the diagnostic criteria for severe pneumonia, had the key variables required for the present analysis available, and had a clearly documented in-hospital outcome. Patients were excluded if they had malignancy, severe hepatic, renal, or hematologic disease, immunodeficiency or long-term immunosuppressive therapy, missing key variables, or repeated admissions. For repeated admissions, only the first hospitalization was retained.

Data collection and variable definitions

Clinical data were extracted from the electronic medical record system. Variables included demographic characteristics, vital signs, laboratory tests, comorbidities, complications, treatment-related factors, and in-hospital death. Demographic variables included age and sex. Vital signs and laboratory variables included systolic blood pressure, diastolic blood pressure, white blood cell count, neutrophil count, lymphocyte count, monocyte count, eosinophil count, basophil count, haemoglobin, platelet count, alanine aminotransferase, albumin, creatinine, fibrinogen, prothrombin activity, procalcitonin, and arterial blood gas parameters, including pH, partial pressure of carbon dioxide, partial pressure of oxygen, and bicarbonate.

Comorbidities and complications included hypertension, coronary heart disease, diabetes, pulmonary hypertension, and sepsis. Treatment-related variables included mechanical ventilation and intensive care unit admission. The outcome was defined as in-hospital death. Categorical variables were coded as binary variables, and continuous variables were retained in their original form. Missing data were handled using complete-case analysis. Patients with missing values for key variables required for model development were excluded, and no multiple imputation was performed.

Statistical analysis

Statistical analyses were performed using R software. All tests were two-sided, and $P<0.05$ was considered statistically significant.

Baseline characteristics were summarized according to data type and distribution. Continuous variables were presented as mean ± standard deviation or median (interquartile range) and compared using the independent-samples t-test or Mann–Whitney U test, as appropriate. Categorical variables were presented as frequencies and percentages and compared using the χ^2 test or Fisher's exact test.

Candidate predictors were selected in the training cohort. Univariate analysis and least absolute shrinkage and selection operator (LASSO) regression were performed separately in the training cohort. Variables selected by both methods were retained to improve model parsimony and robustness.

Model performance was assessed in terms of discrimination, calibration, and clinical utility. Discrimination was evaluated using receiver operating characteristic (ROC) curves and the area under the curve (AUC). Calibration was assessed using calibration curves. Clinical utility was examined using decision curve analysis (DCA).

The variables retained after the selection procedure were subsequently entered into a standard multivariable logistic regression model. LASSO was used primarily for variable selection and shrinkage, while the subsequent multivariable logistic regression provided unpenalized coefficient estimates, odds ratios (ORs), and 95% confidence intervals (CIs), facilitating clinical interpretation of associations between the selected predictors and in-hospital mortality. The fitted multivariable logistic

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regression model was then used to construct the nomogram for individualized risk estimation.

The reporting of this prediction model was reviewed with reference to the TRIPOD statement.

Results

Baseline characteristics

Baseline demographic characteristics, vital signs, laboratory findings, and comorbidities were generally comparable between the training and validation cohorts (*Table I*). Systolic blood pressure differed significantly between the two cohorts (128.5 vs 120.5 mmHg, P=0.026). In-hospital mortality was numerically higher in the training cohort than in the validation cohort (32.0% vs 22.0%). However, the difference was not statistically significant (P=0.071).

Within the training cohort, we stratified patients into non-survivor (n=64) and survivor (n=136) groups (*Table II*). Non-survivors were significantly older than survivors (P<0.001), suggesting that advanced age was associated with increased mortality risk in severe pneumonia. The proportion of male patients was slightly higher among non-survivors, although the

Table I Comparison of baseline characteristics between the training and validation cohorts.

	Training cohort	Validation cohort	P value
N	200	100	
Age (years)	73.00 (58.00–84.00)	72.00 (57.00–80.00)	0.288
SBP (mmHg)	128.50 (112.00–143.00)	120.50 (112.00–134.00)	0.026
DBP (mmHg)	72.00 (63.00–81.25)	72.50 (63.00–80.00)	0.735
WBC ($\times10^9$/L)	9.50 (6.28–12.59)	9.17 (6.32–13.60)	0.509
NEU ($\times10^9$/L)	7.91 (4.81–11.12)	7.85 (4.55–11.29)	0.908
LYM ($\times10^9$/L)	0.76 (0.46–1.13)	0.78 (0.53–1.32)	0.253
MONO ($\times10^9$/L)	0.47 (0.29–0.76)	0.50 (0.27–0.80)	0.33
EO ($\times10^9$/L)	0.02 (0.00–0.08)	0.03 (0.00–0.11)	0.129
BASO ($\times10^9$/L)	0.01 (0.00–0.03)	0.01 (0.00–0.03)	0.959
HGB (g/L)	109.50 (93.75–123.25)	105.00 (91.75–125.00)	0.742
PLT ($\times10^9$/L)	213.50 (148.25–289.00)	236.00 (158.75–302.25)	0.317
ALT (U/L)	26.50 (16.00–44.00)	22.50 (14.00–39.50)	0.331
ALB (g/L)	29.90 (27.60–33.10)	30.60 (26.98–35.85)	0.437
GLB (g/L)	32.20 (28.67–36.23)	34.20 (27.48–37.50)	0.305
TB (μmol/L)	13.05 (9.95–19.02)	14.35 (9.70–20.95)	0.241
CR (μmol/L)	74.00 (57.00–114.50)	71.00 (54.00–98.50)	0.41
FIB (g/L)	5.04 (3.81–6.73)	5.24 (4.31–6.25)	0.612
PTA (%)	78.00 (69.00–86.50)	82.00 (69.50–90.50)	0.174
IL-6 (pg/L)	58.75 (22.47–189.75)	88.92 (28.17–279.25)	0.139
PCT (ng/ml)	0.42 (0.13–1.68)	0.52 (0.10–1.90)	0.828
PH (arterial)	7.43 (7.39–7.47)	7.43 (7.38–7.47)	0.98
$PaCO_2$ (mmHg)	36.00 (32.00–42.00)	35.00 (32.00–39.00)	0.324
PaO_2 (mmHg)	77.20 (65.00–91.10)	79.00 (67.12–111.25)	0.117
HCO_3^- (mmol/L)	25.10 (22.90–27.80)	24.55 (21.70–26.78)	0.124

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Mechanical Ventilation Duration (hours)	37.50 (0.00–220.75)	33.00 (0.00–175.75)	0.701
Length of ICU Stay (Days)	4.00 (0.00–14.00)	3.00 (0.00–11.00)	0.372
Length of Hospital Stay (Days)	14.00 (9.00–22.25)	11.50 (8.00–19.00)	0.092
Sex			0.791
Female	61 (30.50%)	32 (32.00%)	
Male	139 (69.50%)	68 (68.00%)	
Hypertension			0.179
No	118 (59.00%)	67 (67.00%)	
Yes	82 (41.00%)	33 (33.00%)	
Coronary Heart Disease			0.434
No	180 (90.00%)	87 (87.00%)	
Yes	20 (10.00%)	13 (13.00%)	
Diabetes			0.732
No	171 (85.50%)	84 (84.00%)	
Yes	29 (14.50%)	16 (16.00%)	
Pulmonary Hypertension			0.648
No	143 (71.50%)	74 (74.00%)	
Yes	57 (28.50%)	26 (26.00%)	
Sepsis			0.593
No	142 (71.00%)	68 (68.00%)	
Yes	58 (29.00%)	32 (32.00%)	
Death			0.071
No	136 (68.00%)	78 (78.00%)	
Yes	64 (32.00%)	22 (22.00%)	
Mechanical Ventilation			0.934
No	81 (40.50%)	41 (41.00%)	
Yes	119 (59.50%)	59 (59.00%)	
ICU			0.504
No	76 (38.00%)	42 (42.00%)	
Yes	124 (62.00%)	58 (58.00%)	

Note: Continuous variables are presented as median (interquartile range), and categorical variables are presented as number (%). Continuous variables were compared using the independent-sample t-test or Mann–Whitney U test, as appropriate, whereas categorical variables were compared using the χ^2 test or Fisher's exact test. A two-sided $P<0.05$ was considered statistically significant.

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cell count; NEU, neutrophils; LYM, lymphocytes; MONO, monocytes; EO, eosinophils; BASO, basophils; HGB, haemoglobin; PLT, platelet count; ALT, alanine aminotransferase; ALB, albumin; GLB, globulin; TB, total bilirubin; CR, creatinine; FIB, fibrinogen; PTA, prothrombin activity; IL-6, interleukin-6; PCT, procalcitonin; $PaCO_2$, partial pressure of carbon dioxide; PaO_2, partial pressure of oxygen; HCO_3^-, bicarbonate; ICU, intensive care unit.

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Table II Baseline characteristics of survivors and nonsurvivors in the training cohort.

Death	No	Yes	P value
N	136	64	
Age (years)	67.00 (53.00–81.00)	81.00 (69.00–86.25)	<0.001
SBP (mmHg)	127.00 (112.00–142.00)	135.00 (112.00–145.25)	0.139
DBP (mmHg)	71.50 (63.75–81.25)	72.00 (60.00–81.25)	0.448
WBC ($\times 10^9$/L)	9.31 (6.29–12.59)	9.61 (6.19–12.42)	0.909
NEU ($\times 10^9$/L)	7.75 (5.03–11.13)	8.16 (4.42–10.85)	0.739
LYM ($\times 10^9$/L)	0.78 (0.50–1.10)	0.73 (0.42–1.21)	0.637
MONO ($\times 10^9$/L)	0.47 (0.30–0.75)	0.47 (0.25–0.77)	0.845
EO ($\times 10^9$/L)	0.01 (0.00–0.07)	0.02 (0.00–0.10)	0.407
BASO ($\times 10^9$/L)	0.01 (0.01–0.03)	0.01 (0.00–0.03)	0.949
HGB (g/L)	112.00 (96.75–122.00)	104.50 (89.25–128.00)	0.392
PLT (E+9/L)	218.50 (166.75–295.00)	187.50 (124.75–264.25)	0.038
ALT (U/L)	27.50 (16.75–45.25)	24.00 (14.75–36.00)	0.257
ALB (g/L)	29.80 (27.45–32.80)	30.50 (28.37–34.40)	0.111
GLB (g/L)	31.35 (28.25–35.62)	34.10 (29.73–36.80)	0.022
TB (mmol/L)	12.55 (10.15–16.60)	15.10 (9.18–21.60)	0.129
CR (mmol/L)	72.00 (54.75–106.00)	81.00 (60.75–151.75)	0.097
FIB (g/L)	5.32 (4.08–6.99)	4.63 (3.63–5.97)	0.039
PTA (%)	79.50 (71.00–88.00)	75.00 (64.50–82.50)	0.030
PCT (ng/mL)	0.43 (0.14–1.68)	0.36 (0.11–1.56)	0.669
PH (arterial)	7.44 (7.40–7.47)	7.42 (7.38–7.44)	0.020
$PaCO_2$ (mmHg)	35.30 (32.00–40.00)	36.00 (31.40–47.85)	0.265
PaO_2 (mmHg)	76.00 (64.90–87.35)	80.00 (66.25–99.00)	0.282
HCO_3^- (mmol/L)	25.10 (23.25–27.00)	25.05 (21.65–29.25)	0.959
Sex			0.617
Female	43 (31.62%)	18 (28.12%)	
Male	93 (68.38%)	46 (71.88%)	
Hypertension			0.003
No	90 (66.18%)	28 (43.75%)	
Yes	46 (33.82%)	36 (56.25%)	
Coronary Heart Disease			<0.001
No	131 (96.32%)	49 (76.56%)	
Yes	5 (3.68%)	15 (23.44%)	

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Diabetes			0.242
No	119 (87.50%)	52 (81.25%)	
Yes	17 (12.50%)	12 (18.75%)	
Pulmonary Hypertension			0.001
No	107 (78.68%)	36 (56.25%)	
Yes	29 (21.32%)	28 (43.75%)	
Sepsis			<0.001
No	108 (79.41%)	34 (53.12%)	
Yes	28 (20.59%)	30 (46.88%)	
Mechanical Ventilation			<0.001
No	69 (50.74%)	12 (18.75%)	
Yes	67 (49.26%)	52 (81.25%)	
ICU			<0.001
No	66 (48.53%)	10 (15.62%)	
Yes	70 (51.47%)	54 (84.38%)	

Note: Continuous variables are presented as median (interquartile range), and categorical variables are presented as number (%). Group comparisons were performed using the independent-sample t test or Mann–Whitney U test for continuous variables and the χ^2 test or Fisher's exact test for categorical variables. A two-sided P<0.05 was considered statistically significant.

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; WBC, white blood cell count; NEU, neutrophils; LYM, lymphocytes; MONO, monocytes; EO, eosinophils; BASO, basophils; HGB, haemoglobin; PLT, platelet count; ALT, alanine aminotransferase; ALB, albumin; GLB, globulin; TB, total bilirubin; CR, creatinine; FIB, fibrinogen; PTA, prothrombin activity; PCT, procalcitonin; $PaCO_2$, partial pressure of carbon dioxide; PaO_2, partial pressure of oxygen; HCO_3^-, bicarbonate; ICU, intensive care unit.

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difference was not significant (71.88% vs 68.38%, P=0.617). Regarding comorbidities, hypertension, coronary heart disease, pulmonary hypertension, and sepsis were more prevalent in non-survivors than survivors (all P<0.05). Laboratory analyses showed lower platelet counts and higher globulin levels in non-survivors (P=0.038 and P=0.022, respectively). Arterial blood gas analysis showed a lower pH in non-survivors than in survivors (7.42 vs 7.44, P=0.020), suggesting a potential association between acid–base imbalance and mortality risk. Non-survivors were also more likely to require mechanical ventilation (81.25% vs 49.26%, P<0.001) and ICU admission (84.38% vs 51.47%, P<0.001), reflecting greater disease severity among patients who died during hospitalization.

Predictor selection

To identify variables for model development, we performed univariate regression and LASSO regression in the training cohort. The LASSO coefficient profiles are shown in *Figure 1A*, and the binomial deviance curve used to determine the optimal λ is shown in *Figure 1B*. A Venn diagram (*Figure 1C*) identified nine overlapping predictors selected by both methods from 12 variables in each approach. These predictors were age, hypertension, coronary heart disease, pulmonary hypertension, sepsis, fibrinogen, prothrombin activity, mechanical ventilation, and ICU admission.

Model performance

The predictive performance of the model was evaluated in both the training and validation cohorts. ROC analysis showed good discrimination in the training cohort (*Figure 2A*), with an AUC of 0.855 (95% CI: 0.801–0.908). In the validation cohort (*Figure 2B*), the model retained acceptable discrimination, with an AUC of 0.750 (95% CI: 0.640–0.861). These findings indicate good apparent discrimination in the training cohort and acceptable performance in the internal validation

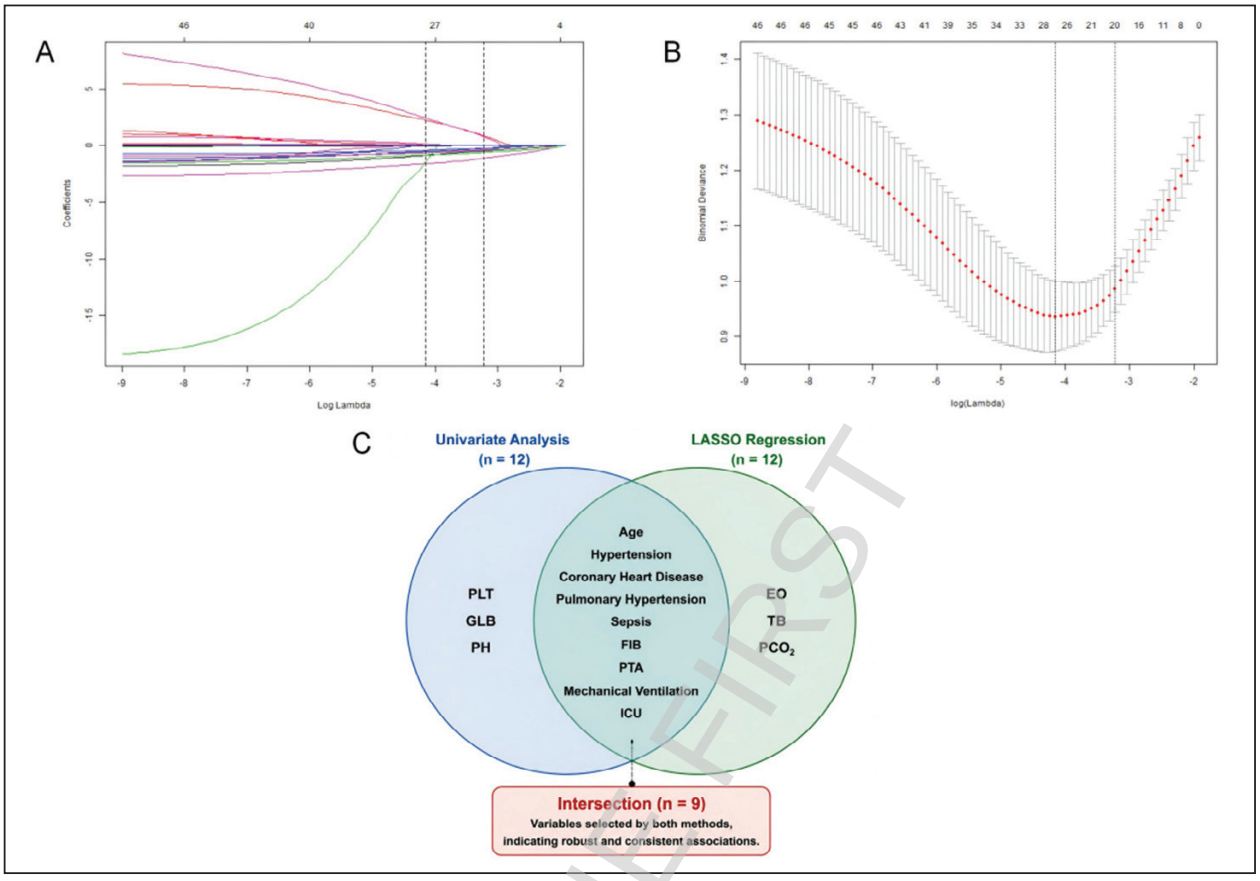


Figure 1 Variable selection using univariate analysis and lasso regression.

Note: Panel A shows the coefficient profiles generated by least absolute shrinkage and selection operator (LASSO) regression. Panel B shows the binomial deviance plot used to determine the optimal penalty parameter (λ). Panel C presents the overlap of variables identified by univariate analysis and LASSO regression.

Abbreviations: LASSO, least absolute shrinkage and selection operator; PLT, platelet count; GLB, globulin; pH, arterial pH; EO, eosinophils; TB, total bilirubin; PCO_2, partial pressure of carbon dioxide; FIB, fibrinogen; PTA, prothrombin activity; ICU, intensive care unit.

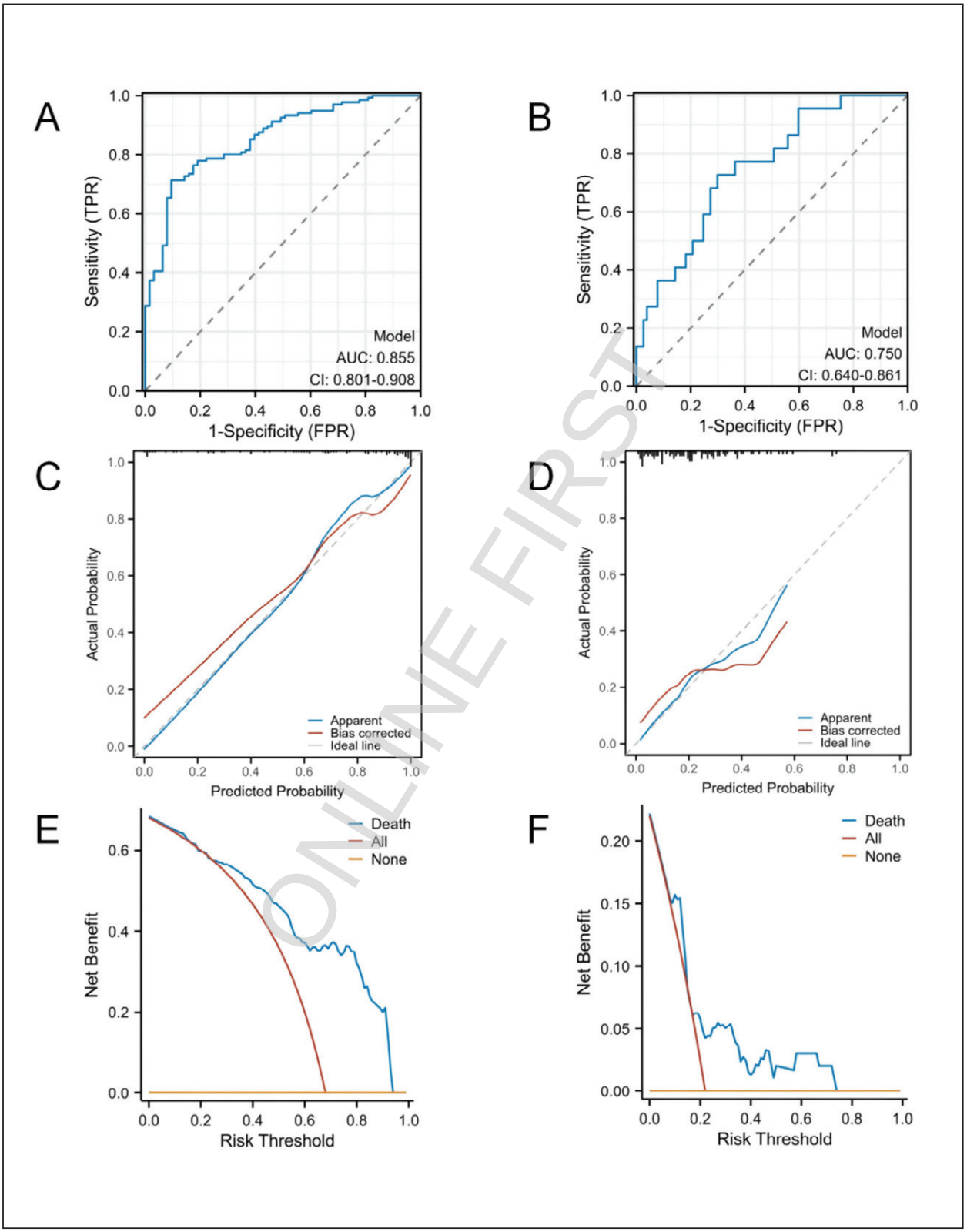
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cohort for distinguishing non-survivors from survivors.

Figures 2C and *2D* show calibration curves for the training and internal validation cohorts. The apparent and bootstrap bias-corrected prediction curves were generally close to the ideal 45° reference line in both cohorts. However, the internal validation cohort showed some deviation. Decision curve analysis (*Figures 2E* and *2F*) showed that the model provided a greater net benefit than the treat-all and treat-none strategies over threshold probabilities of approximately 20%–90% in the training cohort and approximately 20%–70% in the internal validation cohort. These results indicate that the model may support clinical decision-making by identifying patients with severe pneumonia who are at increased risk of in-hospital death.

Multivariable logistic regression and nomogram construction

The nine predictors retained for model development were age, hypertension, coronary heart disease, pulmonary hypertension, sepsis, fibrinogen, prothrombin activity, mechanical ventilation, and ICU admission (*Table III*). Among them, age, coronary heart disease, pulmonary hypertension, and mechanical ventilation were statistically significant. At the same time, the remaining predictors were retained in the prediction model according to the predefined modelling strategy. The nomogram was constructed based on the fitted multivariable logistic regression model (*Figure 3*). Age was significantly associated with in-hospital mortality; each 1-year increase in age was associated with 4% higher odds of death (OR=1.04, 95% CI: 1.02–1.07, P<0.001). Coronary heart disease (OR=3.91, 95% CI: 1.61–9.96, P=0.003),



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Figure 2 Model performance in the training and validation cohorts.

Note: ROC curves for the training (A) and validation (B) cohorts. Calibration curves in the training (C) and validation (D) cohorts; blue line = apparent prediction, red line = bias-corrected prediction, dashed line = ideal calibration. Decision curve analysis in training (E) and validation (F) cohorts; blue line = net benefit of the prediction model.

Table III Multivariable logistic regression results for the final prediction model.

	OR	CI Low	CI High	P value
Age	1.04	1.02	1.07	<0.001
Hypertension				
No	Reference			
Yes	1.80	0.97	3.36	0.063
Coronary Heart Disease				
No	Reference			
Yes	3.91	1.61	9.96	0.003
Pulmonary Hypertension				
No	Reference			
Yes	2.30	1.22	4.33	0.010
Sepsis				
No	Reference			
Yes	1.88	0.98	3.61	0.055
FIB	0.93	0.79	1.09	0.381
PTA	0.99	0.97	1.01	0.206
Mechanical Ventilation				
No	Reference			
Yes	2.41	1.08	5.52	0.034
ICU				
No	Reference			
Yes	1.79	0.82	4.02	0.150

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Note: Multivariable logistic regression analysis was performed to identify independent predictors of in-hospital mortality in patients with severe pneumonia. Odds ratios (ORs), 95% confidence intervals (CIs), and corresponding P values are presented. Variables with categorical coding used the "No" group as the reference category. A two-sided $P<0.05$ was considered statistically significant.

Abbreviations: OR, odds ratio; CI, confidence interval; FIB, fibrinogen; PTA, prothrombin activity; ICU, intensive care unit.

pulmonary hypertension (OR=2.30, 95% CI: 1.22–4.33, P=0.010), and mechanical ventilation (OR=2.41, 95% CI: 1.08–5.52, P=0.034) were also independently associated with increased mortality risk. Hypertension (OR=1.80, 95% CI: 0.97–3.36, P=0.063) and sepsis (OR=1.88, 95% CI: 0.98–3.61, P=0.055) showed positive but nonsignificant associations. Fibrinogen and prothrombin activity were not statistically significant in the multivariable model (P>0.05), suggesting limited independent predictive contribution after adjustment. However, as markers of coagulation and systemic inflammation, they may still have clinical relevance and warrant further evaluation.

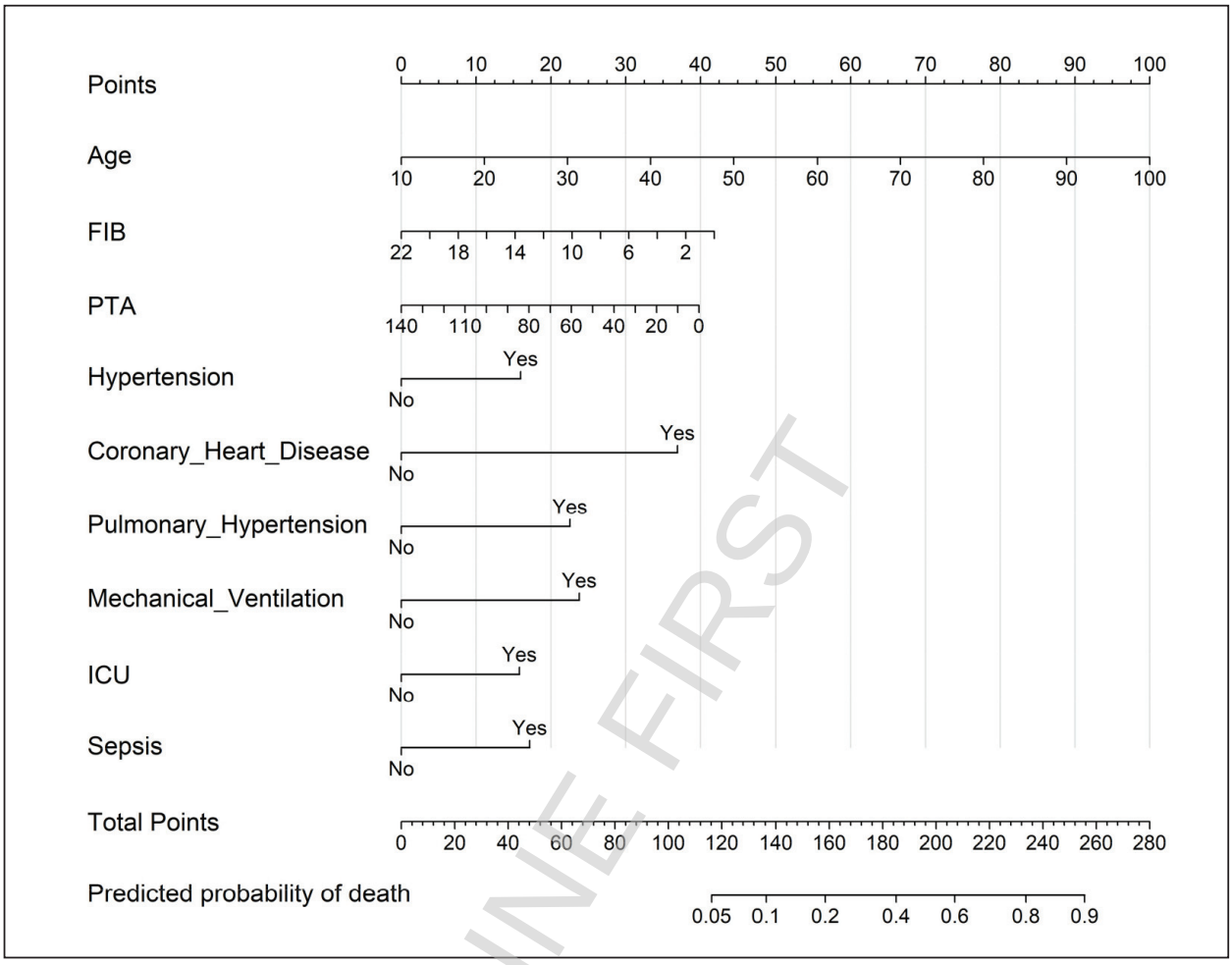


Figure 3 Nomogram for predicting mortality risk in patients with severe pneumonia based on the multivariable logistic regression model.

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Discussion

In this single-centre retrospective cohort study, we developed a mortality prediction model for patients with severe pneumonia and visualized individualized risk using a nomogram. Multivariable analysis identified age, coronary heart disease, pulmonary hypertension, and mechanical ventilation as independent predictors of in-hospital mortality. Hypertension and sepsis did not reach statistical significance in the multivariable model. Still, they showed consistent signals in univariate analysis and LASSO selection.

Age was an important independent predictor of mortality. This association may reflect age-related immune dysfunction, reduced phagocytic and lymphocyte activity, impaired inflammatory regulation, and limited organ reserve. These changes may increase susceptibility to multiple organ dysfunction and acute respiratory distress syndrome under severe infection and hypoxic stress (19–21). Chronic low-grade inflammation in older adults may also amplify cytokine responses, aggravate lung injury and systemic inflammation, and thereby increase mortality risk (22, 23).

Coronary heart disease was associated with substantially higher mortality, suggesting that limited cardiovascular reserve may worsen the course of severe pneumonia. During severe infection, increased myocardial oxygen demand, reduced cardiac compliance, and hemodynamic instability may promote systemic hypoperfusion and accelerate multiple organ failure (24–26). Pulmonary hypertension was also an important risk factor, possibly reflecting increased right ventricular afterload, elevated pulmonary vascular resistance, impaired oxygenation, and reduced systemic tissue perfusion (27–29). Mechanical ventilation was independently associated with in-hospital mortality. However, interpret this association cautiously

because mechanical ventilation primarily marks severe respiratory failure and may therefore partly reflect underlying disease severity rather than an independent causal effect on mortality (30–32). Moreover, because the present retrospective analysis did not explicitly model the temporal relationship between initiation of mechanical ventilation and subsequent clinical deterioration, causal direction cannot be established. Mechanical ventilation may also overlap with other indicators of disease severity, potentially introducing collinearity or residual confounding. Therefore, its role in the present model should be interpreted as a prognostic predictor rather than as a causal risk factor for mortality.

Although hypertension and sepsis were not statistically significant in the final model, their selection by univariate analysis and LASSO suggests potential clinical relevance in specific subgroups. Hypertension may contribute to organ ischemia and hypoxic stress through chronic vascular dysfunction, left ventricular hypertrophy, and impaired microcirculation (33, 34). Sepsis may promote organ failure through systemic inflammation, immune dysregulation, endothelial injury, and microvascular dysfunction (35–37). Coagulation markers, including fibrinogen and prothrombin activity, were not independently significant but may still reflect hypercoagulability, inflammation–coagulation crosstalk, and microcirculatory disturbance (38, 39). The lower platelet count observed among nonsurvivors may also indicate platelet consumption and coagulation abnormalities related to severe inflammation, potentially worsening microvascular injury and organ dysfunction (40).

This study has several limitations. First, its single-centre retrospective design and modest sample size may introduce selection bias and limit generalizability. In particular, 64 in-hospital deaths occurred in the training cohort, and the multivariable model included nine predictors, corresponding to an EPV of approximately 7.1. This was below the conventional rule-of-thumb threshold of 10 events per predictor and may have increased the risk of overfitting and unstable coefficient estimates. Consistent with this concern, the AUC decreased from 0.855 in the training cohort to 0.750 in the internal validation cohort, suggesting some degree of model optimism. Therefore, the predictive performance of the model should be interpreted cautiously and requires further validation in larger independent cohorts. Second, some laboratory and imaging variables were missing, and complete-case analysis may have affected model stability. Third, unmeasured confounding, including differences in antibiotic treatment and ICU resource utilization, could not be fully accounted for and may have influenced model performance and generalizability. Finally, this study was validated only internally using a randomly split validation cohort from the same centre. No external validation or temporal validation was performed. Future multicentre external validation and prospective validation studies are required before clinical implementation.

Overall, the proposed model demonstrated acceptable discrimination, calibration, and potential clinical utility for predicting in-hospital mortality in severe pneumonia. By integrating routinely available clinical variables into a nomogram, the model may support early risk stratification and individualized management. Although established scores such as CURB-65, PSI, and qSOFA are widely used for clinical risk assessment, direct comparisons with these scores were not performed in the present study. Therefore, the incremental predictive value of our model over existing scoring systems remains to be determined in future external validation studies.

Conclusion

We developed and validated a nomogram-based prediction model for in-hospital mortality in patients with severe pneumonia using routine clinical variables. The model showed good discrimination, calibration, and clinical applicability in both the training and validation cohorts. The proposed nomogram may serve as a preliminary tool for estimating in-hospital mortality risk in patients with severe pneumonia. External validation in independent populations is required before clinical application.

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Authors' contribution

Haodong Guo contributed to the conceptualisation, methodology, formal analysis, data curation and visualisation, and wrote the original draft of the manuscript. Changguo Li contributed to the methodology, formal analysis and validation, and

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participated in reviewing and editing the manuscript. Liusheng Wu contributed to project administration and manuscript review and editing. Xiaoqiang Li contributed to the conceptualisation, supervision and project administration, and participated in reviewing and editing the manuscript. Liusheng Wu and Xiaoqiang Li contributed equally to this work and share corresponding authorship.

Human ethics and consent to participate declarations

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Research Ethics Committee of Peking University Shenzhen Hospital (Approval No. Š2026Ć153). Because this was a retrospective study involving the analysis of existing clinical data, the requirement for informed consent was waived by the ethics committee. All patient information was anonymized and handled confidentially throughout the study.

Availability of data and material

The datasets generated and/or analysed during the current study are not publicly available because they contain confidential clinical information and are subject to institutional ethical restrictions. Requests for access to the datasets may be considered by the corresponding author upon reasonable request and approval from the institutional ethics committee.

Consent for publication

All authors have read and approved the final version of the manuscript and agree to its submission. All authors meet the ICMJE criteria for authorship and agree to be accountable for all aspects of the work.

Conflict of interest statement

All the authors declare that they have no conflict of interest in this work.

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