

A Clinical Data–Based Nomogram Prediction Model for ARDS in Patients With Acute Pancreatitis

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Background: Acute pancreatitis (AP) is a common gastrointestinal emergency that may rapidly progress to severe disease. Acute respiratory distress syndrome (ARDS) is one of the most fatal complications of AP, yet reliable early prediction tools remain limited. Early identification of high-risk patients may improve clinical outcomes.

Purpose: To develop and validate a nomogram prediction model for AP complicated by ARDS based on clinical data.

Methods: A total of 280 AP patients admitted to our hospital between February 2022 and March 2024 were retrospectively enrolled as the training set, and 129 patients admitted between April 2024 and June 2025 served as the validation set. Patients were divided into ARDS and non-ARDS groups according to whether ARDS occurred within 14 days of admission. Clinical and laboratory data were collected and analyzed.

Results: In the training set, 74 patients (26.43%) developed ARDS. Multivariate analysis identified age, history of alcohol consumption, lactate (Lac), red cell distribution width (RDW), fasting blood glucose (FBG), and procalcitonin (PCT) as independent risk factors, while albumin (ALB) was a protective factor. These variables were incorporated into the nomogram. The area under the ROC curve (AUC) was 0.899 in internal validation and 0.927 in external validation. Hosmer–Lemeshow tests demonstrated good calibration in both cohorts ($P > 0.05$). Decision curve analysis indicated favorable clinical net benefit across a wide range of threshold probabilities.

Conclusion: Age, alcohol consumption history, Lac, RDW, ALB, FBG, and PCT are key predictors of ARDS in patients with AP. The proposed nomogram demonstrates good discrimination, calibration, and clinical utility, and may assist clinicians in early risk stratification.

Keywords: acute pancreatitis, acute respiratory distress syndrome, nomogram, influence factors

Introduction

Acute pancreatitis (AP) is an acute inflammatory disease caused by various mechanisms following injury to the pancreas. It is also a relatively common critical illness of the digestive system. Approximately 10–20% of patients with AP develop complicated multiple organ dysfunction syndrome, with a mortality rate of 10%–15%, which can be as high as 36–50% in severe cases.¹ In recent years, the incidence of AP has been increasing annually. Although the overall mortality rate of AP is not high, its progression to severe AP significantly increases the mortality rate.^{2,3} Multiple organ failure is also a major cause of death, among which acute respiratory distress syndrome (ARDS) is a more common complication of pulmonary failure. It has a sudden onset and rapid progression, with an in-hospital mortality rate of approximately 35%–45%. Respiratory failure is the most common organ dysfunction in both the early and late stages of acute respiratory diseases and is associated with a high mortality rate. During the first week of acute pancreatitis (AP), up to 60% of deaths are considered to be caused by acute lung injury related to pancreatitis and ARDS.⁴ Despite continuous improvements in current treatment methods, the therapeutic effect for AP complicated by ARDS is still not ideal, with a mortality rate as high as 44.5%, and there is currently a lack of early warning for such patients, preventing early intervention.^{5,6} Therefore, clinically identifying the influencing factors related to AP complicated by ARDS and intervening in a timely manner can

effectively improve patient prognosis. The Nomogram model is a clinical tool for assessing patient prognosis. It can transform data into a graph, is simple to operate, and predicts the risk value of adverse prognostic events by incorporating multiple influencing factors identified through Logistic regression analysis, thereby assisting physicians in formulating corresponding strategies.^{7,8} Previous studies have explored early prediction of acute respiratory distress syndrome (ARDS) in patients with acute pancreatitis (AP). Models based on multiple machine learning algorithms identified partial pressure of oxygen, C-reactive protein (CRP), procalcitonin (PCT), lactate, Ca^{2+} , neutrophil-to-lymphocyte ratio, white blood cell count, and amylase as optimal predictive features, with the Bayesian classifier showing superior predictive performance in the test set.⁸ In addition, a predictive nomogram for ARDS in patients with AP complicated by acute kidney injury has been developed, in which intra-abdominal pressure (IAP), shock, CRP, and lactate dehydrogenase (LDH) were identified as independent predictors of ARDS by multivariate analysis.⁷ Currently, there are few reports on nomograms for AP complicated by ARDS. Therefore, this study aims to explore the construction of a nomogram prediction model for AP complicated by ARDS based on the collection of patients' general clinical characteristics and laboratory indicators, to assist clinicians in identifying high-risk patients and optimizing treatment strategies.

Aim of the Study

The aim of this study was to identify clinical factors associated with the development of acute respiratory distress syndrome (ARDS) in patients with acute pancreatitis (AP) and to construct a nomogram prediction model based on these variables. Furthermore, the study sought to evaluate the performance of the model through internal and external validation to determine its reliability, accuracy, and clinical utility.

Materials and Methods

General Data

A retrospective collection of 280 patients with AP admitted to our hospital from February 2022 to March 2024 was conducted (as the training set). The sample size was calculated using PASS 15 software with a two-sided test ($\alpha = 0.05$, power = 90%, $d = 0.50$), which indicated a required total sample size of 255 patients; considering a 10% dropout rate, at least 280 patients were ultimately included. Additionally, 129 AP patients admitted to our hospital from April 2024 to June 2025 were collected (as the validation set). The sample size was calculated using PASS 15 software with a two-sided test ($\alpha = 0.05$, power = 70%, $d = 0.50$), which indicated a required total sample size of 118 patients; considering a 10% dropout rate, at least 129 patients were ultimately included. According to whether the patients in the training and validation sets developed ARDS within 14 days of admission, they were divided into an ARDS group and a non-ARDS group. The flowchart of case collection is shown in [Figure 1](#). Inclusion criteria: (1) Met the diagnostic criteria for AP;⁹ (2) Met the diagnostic criteria for ARDS;^{10,11} (3) Age > 18 years; (4) Admitted within 24 hours of onset; (5) Had complete clinical data. Exclusion criteria: (1) Received relevant treatment before enrollment; (2) Had pre-existing diseases affecting respiration (chronic lung disease); (3) Had malignant tumors; (4) Had psychiatric disorders; (5) Had hepatic or renal dysfunction; (6) Had immune dysfunction; (7) Were in a state of pregnancy or lactation. This study was approved by the ethics committee of our hospital.

Methods

Diagnostic Criteria for ARDS

The diagnostic criteria for ARDS were mainly determined by referencing the literature:^{10,11} (1) A known etiology, with the onset of acute respiratory symptoms within 1 week; (2) Oxygenation index < 300 mmHg with positive end-expiratory pressure > 5 mmH₂O; (3) Respiratory failure not fully explained by fluid overload, requiring echocardiography to exclude hydrostatic pulmonary edema; (4) Reduced transparency in both lungs on imaging.

Collection of Clinical Data

Clinical data were collected through the patients' electronic medical record system, mainly including age, sex, Body mass index (BMI), etiology, diabetes, hypertension, coronary heart disease, smoking history, alcohol consumption history, heart rate, systolic blood pressure, diastolic blood pressure, partial pressure of oxygen in arterial blood (PaO₂), partial pressure of

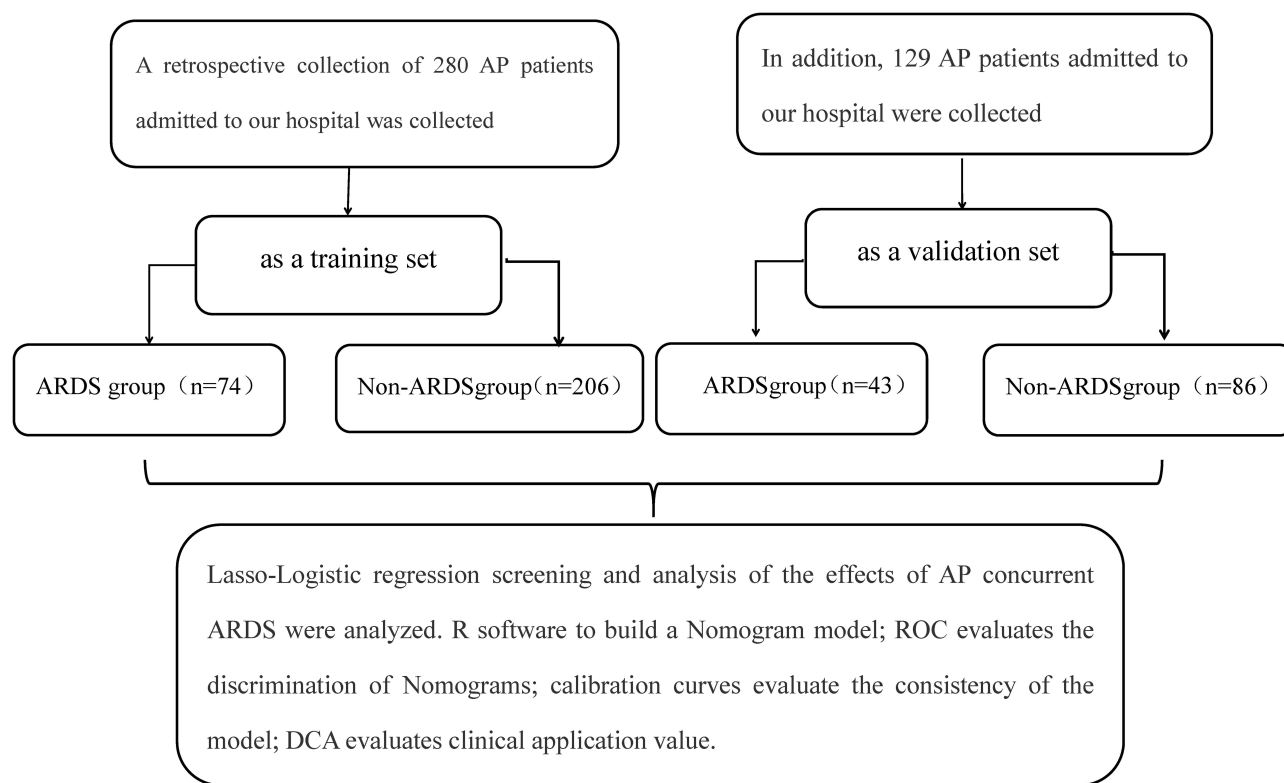


Figure 1 Case flow collection diagram.

carbon dioxide in arterial blood (PaCO₂), white blood cell count (WBC), platelet count (PLT), neutrophil percentage (NE%), hemoglobin (HGB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin (TBIL), direct bilirubin (DBIL), lactic acid (Lac), red blood cell distribution width (RDW), lactate dehydrogenase (LDH), serum creatinine (SCr), blood urea nitrogen (BUN), albumin (ALB), fasting blood glucose (FBG), procalcitonin (PCT), C-reactive protein (CRP), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen (FIB), sodium ion (Na⁺), potassium ion (K⁺), calcium ion (Ca⁺), APACHE II score, and SOFA score. Variables with more than 5% missing data were excluded from the analysis to enhance the reliability and methodological rigor of the results.

Statistical Analysis

Data were processed using SPSS 25.0. Count data were analyzed using the χ^2 test and are expressed as n (%). Measurement data were analyzed using the *t*-test and are expressed as (mean \pm SD). Lasso-Logistic regression was used to screen and analyze the influencing factors for AP complicated by ARDS. R software (R version 4.5.1 and the *rms* package) was used to construct the Nomogram model. The receiver operating characteristic (ROC) curve was used to evaluate the discrimination of the Nomogram; the calibration curve was used to assess the consistency of the model and Decision Curve Analysis (DCA) was used to evaluate the clinical application value. A P-value < 0.05 was considered to indicate a statistically significant difference.

Results

Comparison of Clinical Data Between ARDS and Non-ARDS Groups in the Training Set

In the training set of 280 patients, 74 developed ARDS (26.43%). There were significant differences between the ARDS group and the non-ARDS group in terms of age, history of alcohol consumption, Lac, RDW, ALB, FBG, PCT, APACHE II score, and SOFA score (P<0.05). There were no significant differences in other data (P>0.05). See [Table 1](#).

Table 1 Comparison of Clinical Data Between ARDS and Non ARDS Groups in the Modeling Set

Factor	ARDS Group (n=74)	Non ARDS Group (n=206)	t/ χ^2	P
Age			18.175	<0.001
≥60 years old	47 (63.51)	72 (34.95)		
<60 years old	27 (36.49)	134 (65.05)		
Gender			0.018	0.893
Male	41 (55.41)	116 (56.31)		
Female	33 (44.59)	90 (43.69)		
BMI			0.153	0.696
≥24 kg/m ²	35 (47.30)	92 (44.66)		
<24 kg/m ²	39 (52.70)	114 (55.34)		
Pathogen			3.966	0.265
Biligenic	32 (43.24)	96 (46.60)		
Hyperlipidemia	20 (27.03)	60 (29.13)		
Alcoholic	14 (18.92)	41 (19.90)		
Other	8 (10.81)	9 (4.37)		
Diabetes	16 (21.62)	42 (20.39)	0.050	0.822
Hypertension	25 (33.78)	52 (25.24)	1.992	0.158
Coronary heart disease	4 (5.41)	10 (4.85)	0.035	0.852
Smoking history	24 (32.43)	64 (31.07)	0.047	0.828
History of drinking	46 (62.16)	82 (39.81)	10.965	0.001
Heart rate (times/min)	93.26±20.34	88.61±18.67	1.794	0.074
Systolic blood pressure (mmHg)	131.25±23.24	129.63±22.53	0.526	0.599
Diastolic blood pressure (mmHg)	80.21±13.26	78.62±11.05	1.005	0.316
PaO ₂ (mmHg)	78.65±10.54	80.26±11.45	1.059	0.291
PaCO ₂ (mmHg)	35.16±6.52	34.68±6.75	0.529	0.597
WBC (×10 ⁹ /L)	18.62±3.24	18.96±3.52	0.727	0.468
PLT (×10 ⁹ /L)	231.25±46.32	229.34±47.62	0.298	0.766
NE%	83.04±3.04	83.11±3.12	0.167	0.868
HGB (g/L)	134.29±21.24	137.21±22.09	0.648	0.518
ALT (U/L)	33.26±3.21	33.42±3.19	0.369	0.712
AST (U/L)	34.21±7.65	32.86±7.16	1.731	0.630
ALP (U/L)	114.26±21.25	112.26±20.64	1.366	0.173
TBIL (mmol/L)	30.82±4.32	30.49±4.21	0.574	0.566
DBIL (mmol/L)	4.82±1.01	4.79±0.96	0.227	0.820
Lac (mmol/L)	1.64±0.31	1.14±0.30	12.190	<0.001
RDW (%)	14.02±2.34	12.79±2.24	4.004	<0.001
LDH (U/L)	206.24±26.24	201.25±27.63	1.350	0.178
SCr (μmol/L)	71.65±8.62	69.59±8.16	1.835	0.068
BUN (mmol/L)	6.01±1.05	5.81±0.98	1.477	0.141
ALB (g/L)	36.28±5.32	42.26±6.01	7.560	<0.001
FBG (mmol/L)	8.53±1.06	6.32±1.09	15.068	<0.001
PCT (ng/mL)	1.05±0.21	0.32±0.07	43.698	<0.001
CRP (mg/L)	69.16±9.68	67.89±7.36	1.166	0.244
TG (mmol/L)	1.62±0.31	1.64±0.33	0.454	0.650
LDL-C (mmol/L)	1.38±0.33	1.43±0.35	1.070	0.286
HDL-C (mmol/L)	2.46±0.42	2.41±0.38	0.020	0.888
PT (s)	12.64±1.98	12.31±1.89	0.944	0.346
APTT (s)	29.86±3.24	30.11±3.18	0.577	0.564
FIB (mg/dL)	391.68±16.24	390.62±15.78	0.492	0.623
Na ⁺ (mmol/L)	143.26±32.14	142.21±30.98	0.248	0.805

(Continued)

Table 1 (Continued).

Factor	ARDS Group (n=74)	Non ARDS Group (n=206)	t/χ^2	P
K ⁺ (mmol/L)	3.96±0.84	4.12±0.96	1.269	0.205
Ca ⁺ (mmol/L)	1.72±0.24	1.78±0.31	1.510	0.132
APACHE II score (points)	16.54±2.52	8.69±1.62	30.515	<0.001
SOFA score (points)	8.34±2.14	3.62±0.98	25.195	<0.001

Lasso-Logistic Regression Analysis of Influencing Factors for AP Complicated by ARDS

Using the development of ARDS in AP patients in the training set as the dependent variable (Yes=1, No=0), and the factors with significant differences from Table 1—age (≥ 60 years=1, < 60 years=0), history of alcohol consumption (Yes=1, No=0), Lac, RDW, ALB, FBG, PCT, APACHE II score, and SOFA score—as independent variables, a Lasso analysis was performed using R software. The results showed that when the penalty coefficient λ was 0.02342007, the model demonstrated excellent performance, and seven predictive factors were selected: age, history of alcohol consumption, Lac, RDW, ALB, FBG, and PCT, as shown in Figure 2. A logistic analysis (forward stepwise method) of the independent variables selected by Lasso regression showed that age, history of alcohol consumption, Lac, RDW, FBG, and PCT were risk factors for AP complicated by ARDS ($P < 0.05$), while ALB was a protective factor ($P < 0.05$). See Table 2.

Construction of the Nomogram Model for AP Complicated by ARDS

A nomogram model was constructed based on the factors selected from the Lasso-Logistic regression analysis, with $P = \frac{e^x}{1+e^x}$, where $x = -19.475 + 1.296 \times \text{age} + 1.389 \times \text{history of alcohol consumption} + 0.700 \times \text{Lac} + 0.523 \times \text{RDW} - 1.038 \times \text{ALB} + 1.296 \times \text{FBG} + 0.896 \times \text{PCT}$. In this model, the most important influencing factor was PCT. The risk of ARDS complicating AP associated with each factor was represented as a score, and the total score was obtained by summing the scores of all factors. The predicted probability of ARDS occurrence in individual patients was then determined according to the corresponding total score. The higher the total score, the greater the probability of developing ARDS. For example, when the total score is between 150 and 160, the probability of occurrence is 71% to 83%. See Figure 3.

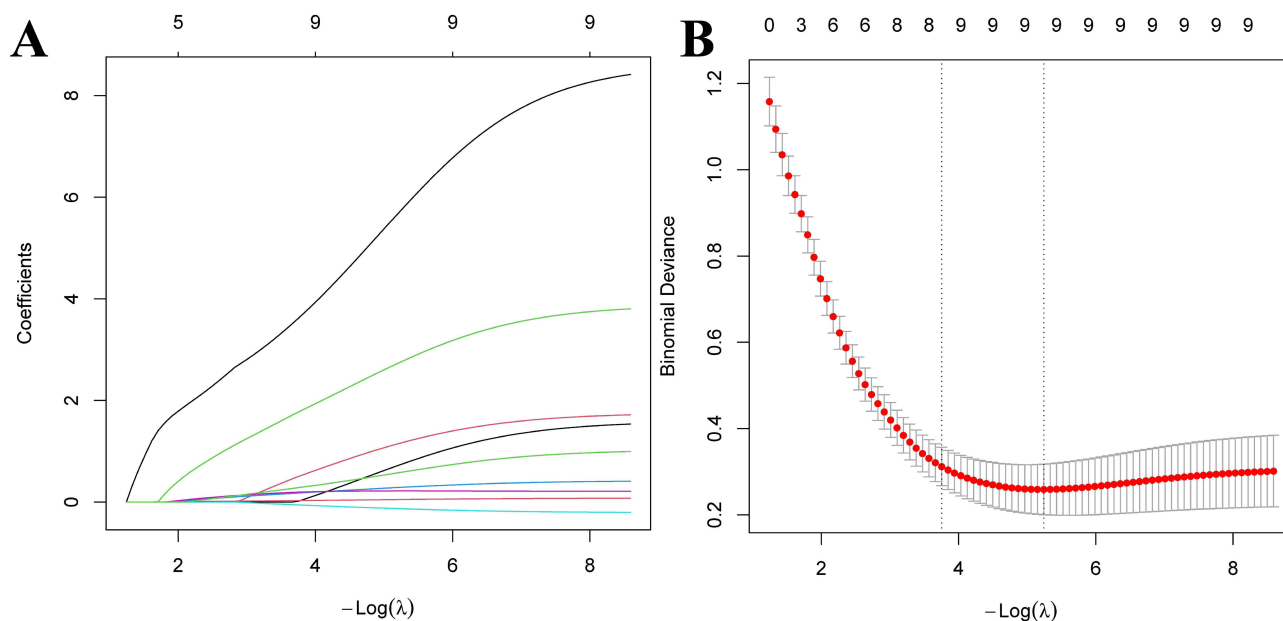


Figure 2 Lasso-logistic regression analysis of risk factors for ARDS in acute pancreatitis. (A) Relationship diagram of Lasso regression coefficient; (B) Lasso regression 10-fold cross-validation results.

Table 2 Lasso-Logistic Regression Analysis of the Influencing Factors of AP Concurrent ARDS

Variable	B value	SE value	Wald χ^2 value	P value	OR value	95% CI
Age	1.296	0.302	18.411	<0.001	3.654	2.022~6.604
History of drinking	1.389	0.267	27.075	<0.001	4.012	2.377~6.771
Lac	0.700	0.333	4.420	0.036	2.014	1.049~3.868
RDW	0.523	0.211	6.143	0.013	1.687	1.116~2.551
ALB	-1.038	0.310	11.222	0.001	0.354	0.193~0.650
FBG	1.296	0.286	20.529	<0.001	3.654	2.086~6.401
PCT	0.896	0.355	6.377	0.012	2.451	1.222~4.915
Constant	-19.475	3.445	31.949	<0.001	0.004	-

Internal Validation of the Model for AP Complicated by ARDS

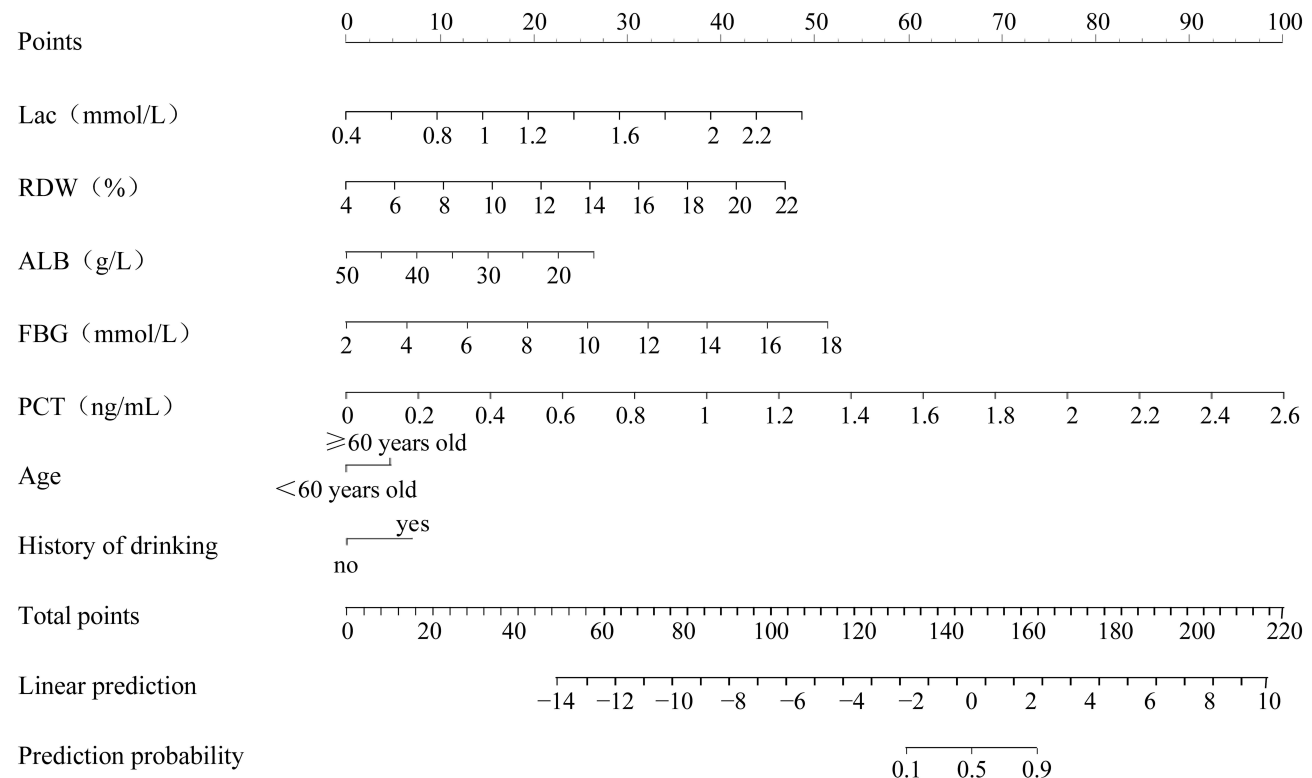
The ROC curve showed that the model predicting ARDS complicating AP had an AUC of 0.899 (95% CI = 0.857–0.942), with a sensitivity of 86.42% and a specificity of 80.35%. Internal validation using the bootstrap method with 1000 resamples yielded a C-index of 0.842. The goodness-of-fit assessment showed a Hosmer–Lemeshow test of $\chi^2 = 7.413$ ($P = 0.722$), indicating a high consistency between the predicted risk of ARDS and the observed outcomes. See [Figure 4](#).

Internal Validation Using DCA Curve

The results of the Decision Curve Analysis (DCA) showed that when the threshold probability was between 0.14 and 0.95, the net benefit for assessing AP complicated by ARDS was high, and higher than the two default lines, as shown in [Figure 5](#).

Comparison of Clinical Data Between ARDS and Non-ARDS Groups in the Validation Set

In the 129 patients of the validation set, 43 developed ARDS (33.33%). There were significant differences between the ARDS group and the non-ARDS group in terms of age, history of alcohol consumption, Lac, RDW, ALB, FBG, and PCT ($P < 0.05$). There were no significant differences in other data ($P > 0.05$). See [Table 3](#).

**Figure 3** A Nomogram model of AP concurrent ARDS.

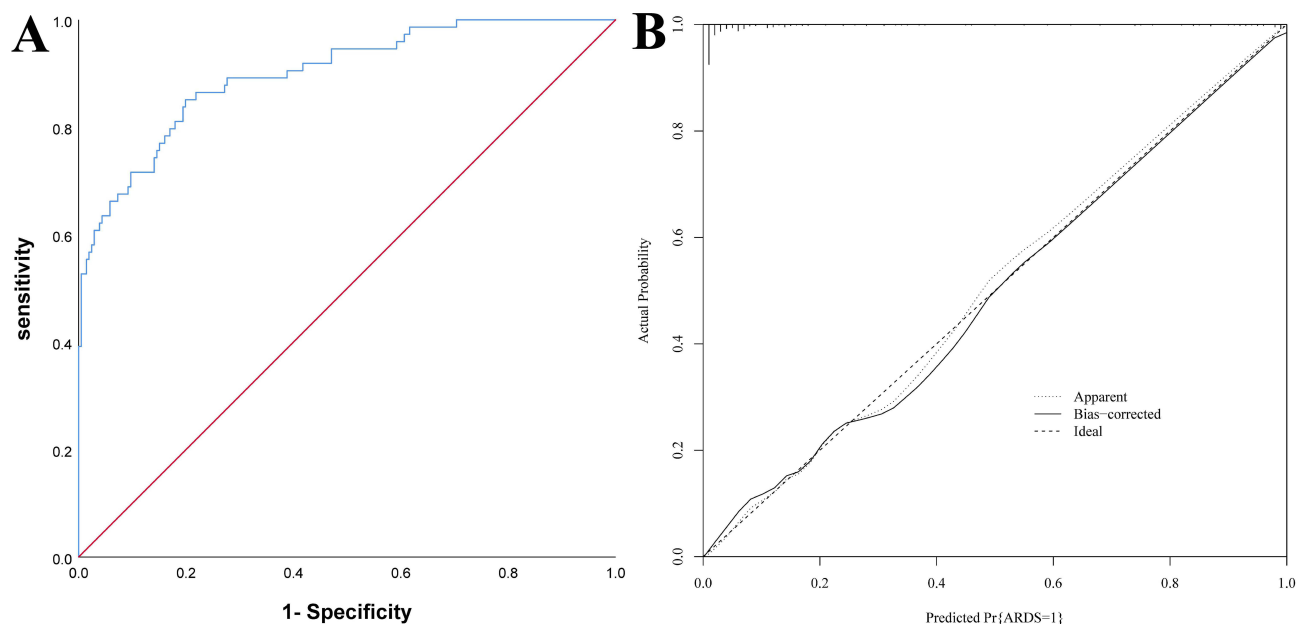


Figure 4 Internal validation of the predictive model for ARDS in acute pancreatitis. **(A)** ROC curves; **(B)** Calibration curves.

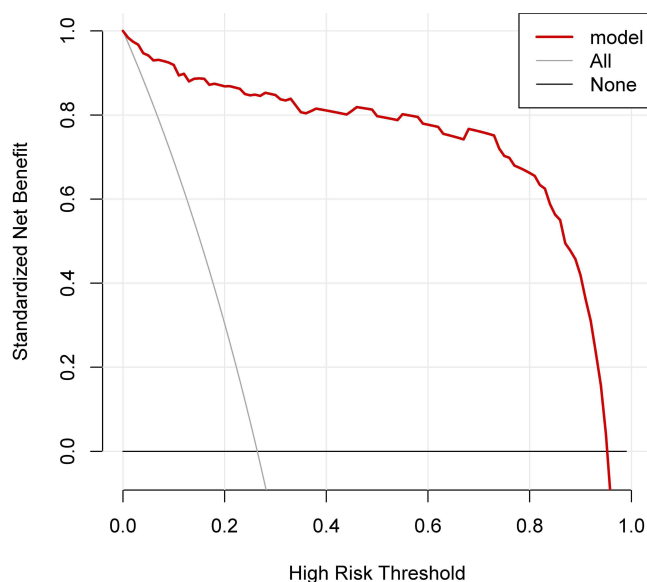


Figure 5 Internal validation of DCA curves.

Note: The X-axis represents the potential threshold probability for early recurrence, and the Y-axis represents the net benefit. The red line indicates the nomogram predicting AP complicated by ARDS. The gray line assumes intervention for all patients, and the thin black line indicates no intervention for any patient.

External Validation of the Model for AP Complicated by ARDS

The ROC curve showed that in external validation, the model predicting ARDS complicating AP had an AUC of 0.927 (95% CI = 0.878–0.976), with a sensitivity of 88.96% and a specificity of 80.35%. Internal validation using the bootstrap method with 1,000 resamples yielded a C-index of 0.824. The goodness-of-fit assessment showed a Hosmer–Lemeshow test of $\chi^2 = 7.297$ ($P = 0.716$), indicating a high consistency between the predicted risk of ARDS and the observed outcomes, as shown in Figure 6.

Table 3 Comparison of Clinical Data Between ARDS and Non ARDS Groups in Validation Set

Factor	ARDS Group (n=43)	Non ARDS Group (n=86)	t/ χ^2	P
Age			12.370	<0.001
≥60 years old	28 (65.12)	28 (32.56)		
<60 years old	15 (34.88)	58 (67.44)		
History of drinking	28 (65.12)	30 (34.88)	10.588	0.001
Lac (mmol/L)	1.57±0.34	1.11±0.24	8.888	<0.001
RDW (%)	14.36±2.25	12.67±2.15	4.144	<0.001
ALB (g/L)	34.36±5.03	41.38±5.86	6.713	<0.001
FBG (mmol/L)	8.46±1.11	6.24±1.04	11.175	<0.001
PCT (ng/mL)	1.03±0.25	0.34±0.09	22.872	<0.001

External Validation Using DCA Curve

The results of the DCA curve showed that when the threshold probability was between 0.19 and 0.94, the net benefit for assessing AP complicated by ARDS was high, and higher than the two default lines, as shown in Figure 7.

Discussion

AP is an exocrine disease of the pancreas caused by factors such as biliary tract diseases and alcohol. Clinically, patients present with symptoms like abdominal pain and can also develop multiple organ dysfunction. Among these complications, AP complicated by ARDS is a major cause of death in AP patients, and the extent of lung involvement is related to the mortality rate.¹² Currently, the pathogenesis of ARDS in patients with AP is not yet fully understood, and there is a lack of specific drugs for its treatment. Therefore, early warning and intervention for ARDS can reduce its incidence and improve patient prognosis. However, commonly used clinical assessment systems do not provide effective early warnings.^{13,14} Thus, it is necessary to identify risk factors for the early prediction of this condition in patients.

Through Lasso-Logistic regression, age, history of alcohol consumption, Lac, RDW, ALB, FBG, and PCT were identified as influencing factors for AP complicated by ARDS. The reasons are analyzed as follows: (1) For older

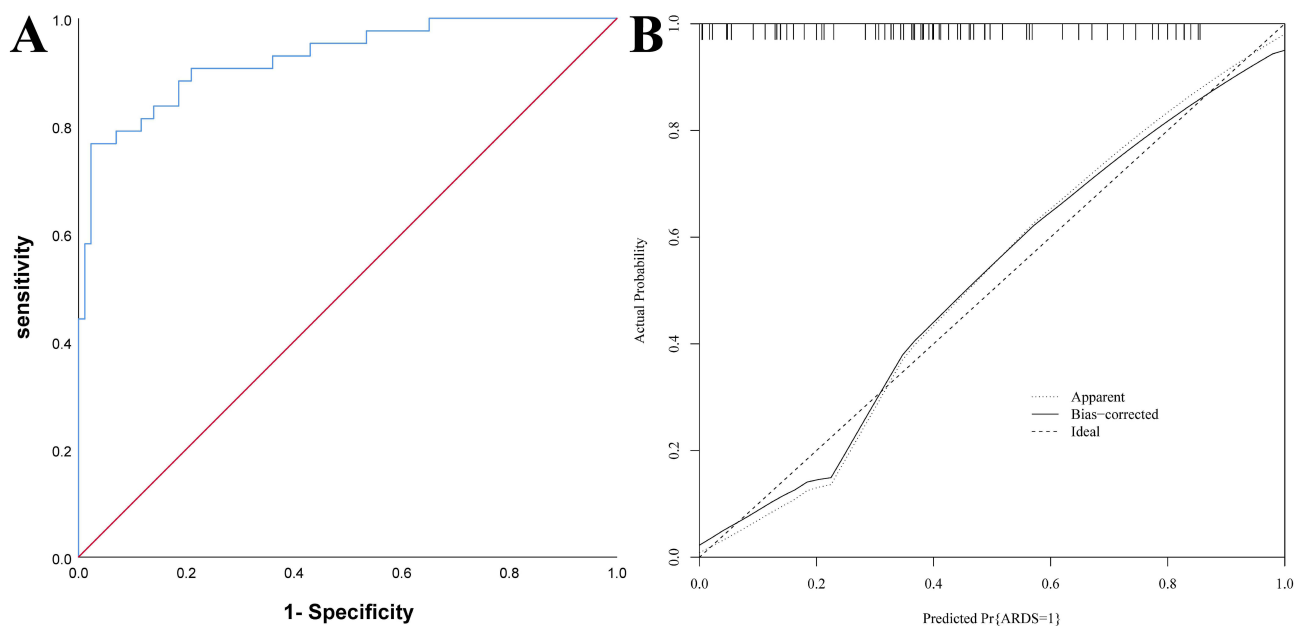


Figure 6 External validation of the predictive model for ARDS in acute pancreatitis. (A) ROC curves; (B) Calibration curves.

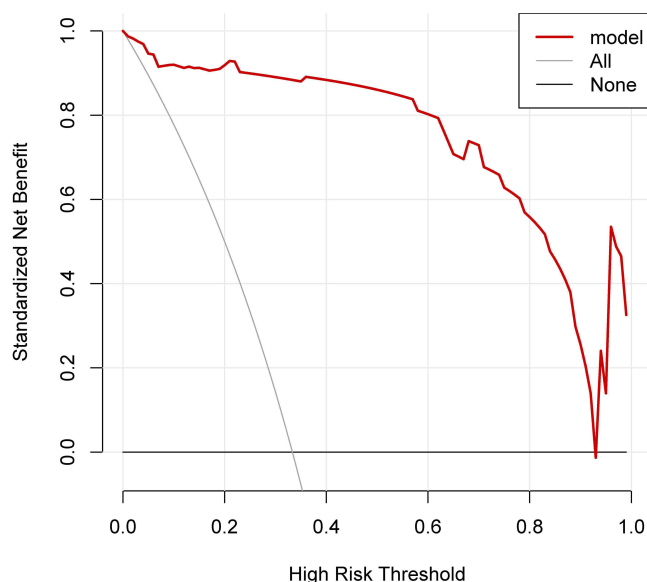


Figure 7 External validation of DCA curves.

Note: The X-axis represents the potential threshold probability for early recurrence, and the Y-axis represents the net benefit. The red line indicates the nomogram predicting AP complicated by ARDS. The gray line assumes intervention for all patients, and the thin black line indicates no intervention for any patient.

patients, the decline in bodily functions is accompanied by physiological changes in their lung tissue structure, which can reduce lung compliance and respiratory function, thereby increasing the probability of pulmonary diseases. Furthermore, older patients have relatively lower immune function and reduced defense capabilities of the respiratory mucosa, making them more susceptible to developing ARDS.^{15,16} Therefore, in clinical practice for older patients, it is necessary to closely monitor their vital signs and provide mechanical ventilation as needed to ameliorate hypoxic injury. (2) Alcohol consumption reduces alveolar epithelial glutathione. In AP patients, an inflammatory response is present, and when the body is under stress, it can increase oxygen free radicals, leading to damage to pulmonary epithelial cells. In patients with a long-term history of alcohol consumption, vascular endothelial selectin is significantly increased. Vascular endothelial selectin can enhance the adhesion of leukocytes to pulmonary vascular endothelial cells, increasing alveolar-capillary permeability and predisposing them to ARDS.^{17,18} Therefore, during clinical treatment, patients should be advised of the dangers of alcohol consumption to reduce its frequency. (3) Lac can reflect tissue hypoxia and insufficient perfusion. An elevated level can be used to assess the occurrence of ARDS. Because AP complicated by ARDS is essentially an organ function injury, ARDS reduces the body's ventilation and oxygenation functions. When tissue oxygenation is insufficient, Lac production increases. Changes in capillary permeability and hemoconcentration prevent effective tissue perfusion, thereby increasing the risk of ARDS.¹⁹ (4) RDW reflects the heterogeneity of red blood cells. In the early stages of AP, damage to pancreatic acinar cells activates inflammatory cells, leading to the massive release of inflammatory factors. The inflammatory response can damage pulmonary capillaries and alveolar epithelial cells through various pathways. It also inhibits red blood cell maturation, causing larger reticulocytes to enter the bloodstream, which increases RDW. An increased RDW can also enhance the oxidative stress response, raising the risk of ARDS.^{20,21} (5) ALB is an important indicator related to ARDS. When the body's endothelial barrier is damaged, the permeability of the alveolar-capillary barrier increases. Inflammatory cells and other components infiltrate the extracellular matrix, causing alveolar edema and reducing alveolar clearance rate. This exacerbates lung injury and accelerates the leakage of ALB into the alveoli, leading to decreased ALB levels and an increased risk of ARDS.²² (6) Hyperglycemia can cause vascular endothelial oxidative stress injury and cell apoptosis, worsening the patient's condition, promoting the development of ARDS, and increasing patient mortality.²³ Therefore, it is necessary to closely monitor patients' blood glucose fluctuations in clinical practice and adjust treatment in a timely manner. (7) Following an infection, PCT is released into the bloodstream in large quantities, causing its level to rise significantly, which reflects the severity of the body's inflammatory response. Previous studies have found that PCT levels are related to the severity of pancreatic lesions and can reflect pancreatic exudation in

the early stages. PCT is also highly expressed in ARDS, and early PCT levels are associated with long-term organ dysfunction, indicating that an elevated PCT level increases the risk of ARDS.²⁴

In this study, a nomogram model was constructed based on Lasso-Logistic regression. The nomogram is feasible to implement across different healthcare systems, allowing adjustments in the acquisition of indicators, risk calculation tools, and depth of decision-making interventions according to available resources, thereby providing a unified and standardized risk prediction tool and promoting homogeneity in clinical care. The results showed that the AUCs for the training and validation sets were 0.899 and 0.927, respectively. The H-L test found that the predicted probability of AP complicated by ARDS was in close agreement with the actual probability. Furthermore, the DCA curves for the training and validation sets showed high net benefits for assessing AP complicated by ARDS when the threshold probabilities were within the ranges of 0.14–0.95 and 0.19–0.94, respectively. This indicates that the model has high clinical value and can assist healthcare professionals in timely intervention, by identifying high-risk populations based on relevant factors, closely monitoring them, and providing prompt interventions. Within a specific range of threshold probabilities, the clinical net benefit is high, enabling precise interventions for high-risk groups, avoiding overtreatment, and optimizing resource allocation. In resource-limited settings, these thresholds can be fully applied to clinical decision-making by following the principle of unchanged thresholds with adapted actions: immediate referral for high-risk patients, close monitoring and elective referral for moderate-risk patients, and routine care for low-risk patients, thus ensuring effective treatment while minimizing missed diagnoses and overtreatment.

Conclusions

In conclusion, age, history of alcohol consumption, Lac, RDW, ALB, FBG, and PCT are influencing factors for AP complicated by ARDS. The nomogram prediction model established based on these risk factors demonstrates good consistency, discrimination, and a high net clinical benefit. This study has certain limitations. As a single-center, retrospective study with a relatively small sample size, the results may be subject to bias. In the future, we plan to expand the sample size and conduct prospective, multi-center studies for further validation. This will allow the nomogram model to become a readily accessible clinical decision-making tool for physicians, seamlessly integrating into the diagnostic and treatment workflow without additional learning costs. It will fully realize its core value of precise risk stratification and individualized intervention guidance, thereby promoting homogeneity in the quality of clinical care.

Abbreviations

AP, Acute pancreatitis; ARDS, Acute respiratory distress syndrome; RDW, Red cell distribution width; FBG, Fasting blood glucose; PCT, Procalcitonin; ALB, Albumin; BMI, Body mass index; PaO₂, Partial pressure of oxygen in arterial blood; PaCO₂, Partial pressure of carbon dioxide in arterial blood; WBC, White blood cell count; PLT, Platelet count; NE%, Neutrophil percentage; HGB, Hemoglobin; ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; ALP, Alkaline phosphatase; TBIL, Total bilirubin; DBIL, Direct bilirubin; Lac, Lactic acid; LDH, Lactate dehydrogenase; SCr, Serum creatinine; BUN, Blood urea nitrogen; CRP, C-reactive protein; TG, Triglycerides; LDL-C, Low-density lipoprotein cholesterol; HDL-C, High-density lipoprotein cholesterol; PT, Prothrombin time; APTT, Activated partial thromboplastin time; FIB, Fibrinogen; Na⁺, Sodium ion; K⁺, Potassium ion; Ca⁺, Calcium ion; ROC, Receiver operating characteristic; DCA, Decision curve analysis.

Data Sharing Statement

The data used to support the findings of this study are included within the article.

Ethics Approval and Consent to Participate

This study was reviewed and approved by the Ethics Committee of Ganzhou People's Hospital (Ganzhou, China). The ethical approval reference number is [Approval No. PJB2025-317-01]. All procedures involving human participants were conducted in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. Written informed consent to participate in this study was obtained from all participants.

Consent for Publication

All authors give consent for publication.

Funding

There is no funding to report.

Disclosure

The authors declared no conflicts of interest in this work.

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