Journal of Inflammation Research

Dovepress
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ORIGINAL RESEARCH

Factors Associated with ICU Admission and In-Hospital Mortality in Patients with Acute Pancreatitis: A Single-Center Retrospective Study

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Background: Early identification of patients with acute pancreatitis (AP) who may require intensive care unit (ICU) admission is critical for optimizing resource allocation and improving clinical outcomes. This study aimed to identify factors associated with ICU admission and to explore markers associated with in-hospital mortality in patients with AP using routinely available clinical and laboratory data.

Methods: This retrospective cohort study included 931 patients with AP admitted to a tertiary care center between June 2020 and June 2024. Demographic, clinical, and laboratory parameters obtained within 24 hours of admission were analyzed. Multivariable logistic regression with forward stepwise selection and least absolute shrinkage and selection operator (LASSO) regression were employed to identify factors associated with ICU admission. Among ICU-admitted patients, LASSO-Cox regression was performed as an exploratory analysis to identify markers associated with in-hospital mortality. Model performance was assessed using receiver operating characteristic (ROC) curve analysis and internal validation with bootstrap resampling.

Results: Of the 931 patients, 133 (14.3%) required ICU admission. Hypertriglyceridemic pancreatitis (OR 2.88, 95% CI 1.59–5.21), elevated D-dimer (OR 1.13, 95% CI 1.07–1.18), blood urea nitrogen (BUN) (OR 1.36, 95% CI 1.25–1.48), and red blood cell distribution width (RDW) (OR 1.14, 95% CI 1.09–1.19) were independently associated with ICU admission. A nomogram incorporating these factors demonstrated good discrimination (AUC 0.89, 95% CI 0.86–0.93) and calibration (Hosmer–Lemeshow P = 0.517). Among ICU patients (n = 133, 20 deaths), LASSO-Cox regression identified elevated C-reactive protein-to-albumin ratio (CAR) as a possible predictor of in-hospital mortality (HR 2.12, 95% CI 1.76–3.27), with an AUC of 0.78 (95% CI 0.69–0.88). Kaplan-Meier analysis revealed significantly lower survival in ICU-admitted patients (log-rank P < 0.001).

Conclusion: Routinely available inflammatory and biochemical markers might be associated with ICU admission and in-hospital mortality in patients with acute pancreatitis. These findings may assist early clinical risk stratification and complement existing clinical assessments. However, prospective multicenter studies with larger cohorts are warranted to validate these findings.

Keywords: acute pancreatitis, ICU admission, risk factors, nomogram, inflammatory markers

Introduction

Acute pancreatitis (AP) is an inflammatory disorder triggered by premature activation and infiltration of pancreatic enzymes, with a global incidence of approximately 34 cases per 100,000 person-years.[1–3] The clinical course of AP is highly heterogeneous, ranging from mild, self-limiting episodes to severe disease accompanied by organ failure.[4] According to the Revised Atlanta Classification (2012),[4] AP is categorized into interstitial edematous pancreatitis and

Journal of Inflammation Research downloaded from https://www.dovepress.com/ For personal use only.

https://doi.org/10.2147/JIR.S591892
Received: 9 January 2026
Accepted: 26 March 2026
Published: 3 April 2026

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necrotizing pancreatitis, with severity classified as mild, moderately severe, or severe based on the presence and duration of organ failure and the presence of local or systemic complications.

Early identification of patients who may require intensive care unit (ICU) admission is critical for several reasons. First, timely transfer to an appropriate monitoring setting and proactive initiation of organ support can improve clinical outcomes.[5] Second, ICU beds represent a scarce and expensive resource; their rational allocation necessitates objective tools to distinguish patients who will benefit from intensive care from those who can be safely managed in a general ward. Third, early risk stratification enables individualized treatment planning and may help prevent healthcare system overload.

Multiple scoring systems have been developed to assess severity in AP, including the Ranson criteria,[6] Bedside Index for Severity in Acute Pancreatitis (BISAP),[7] Acute Physiology and Chronic Health Evaluation II (APACHE II),[8] Harmless Acute Pancreatitis Score (HAPS), Japanese Severity Score (JSS),[9] Modified Computed Tomography Severity Index (MCTSI),[10] and the Balthazar grading system.[11] Despite their widespread use, these tools have notable limitations. Their complexity—often requiring multiple clinical and laboratory parameters—poses challenges for rapid bedside application. Many variables require 48 hours to complete (eg, Ranson) or necessitate repeated measurements, limiting their utility for early decision-making. Furthermore, subjective interpretation may introduce variability in scoring accuracy.[12–15] These limitations underscore the need for simpler, more objective tools for early risk assessment, particularly for predicting the need for ICU admission.

Given these limitations, there is growing interest in alternative biomarkers that can be assessed rapidly and reproducibly in the early phase of AP. Various inflammatory and metabolic markers—such as blood urea nitrogen (BUN), creatinine, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), C-reactive protein (CRP), procalcitonin (PCT), and D-dimer—have been investigated as prognostic indicators in AP. Hong et al[16] developed a logistic regression model using admission high-density lipoprotein cholesterol together with 24-hour BUN and creatinine to stratify patients with SAP. Zhu et al[17] reported that the combination of NLR, PCT, and MCTSI provided strong predictive value for infected pancreatic necrosis. Kaplan et al[18] demonstrated that the combination of PLR and NLR had predictive efficacy comparable to established scoring systems.

More recently, novel markers have been explored specifically for their ability to predict ICU admission. Xu et al[19] demonstrated that the lymphocyte-to-C-reactive protein ratio (LCR) at admission is a simple and reliable predictor of disease progression and a useful screening tool for ICU admission in adult patients with AP. Similarly, Kolber et al[20] found that serum urokinase-type plasminogen activator receptor (uPAR) measured within 24 hours of AP onset predicted the need for intensive care, with diagnostic accuracy comparable to conventional markers such as CRP, PCT, and D-dimer.

Despite these advances, data specifically focusing on the clinical characteristics and outcomes of AP patients requiring ICU admission remain limited. Most existing studies have focused on predicting SAP as defined by the Revised Atlanta Criteria, rather than the real-world clinical decision of ICU admission. Furthermore, few studies have systematically examined whether a combination of routinely available admission parameters can reliably identify patients at risk of ICU admission and subsequent in-hospital mortality.

Therefore, the present study aimed to: (1) identify factors associated with ICU admission in a cohort of patients with AP using routinely available clinical and laboratory data obtained within 24 hours of hospital presentation; (2) explore factors associated with in-hospital mortality among patients admitted to the ICU; and (3) evaluate survival differences between ICU-admitted and non-ICU patients using Kaplan-Meier analysis. By focusing on early, readily available parameters, we sought to develop a practical tool to assist clinicians in early risk stratification and decision-making regarding ICU admission.

Materials and Methods

Study Design and Patient Selection

This retrospective cohort study included patients diagnosed with acute pancreatitis (AP) at a tertiary care center in Xi'an between June 2020 and June 2024. Demographic, clinical, and laboratory data were collected from electronic medical records. Complete blood count (CBC) was performed using semiconductor laser flow cytometry and sheath flow DC

impedance methods with Sysmex reagents on an XN9000 analyzer. CRP was measured using nephelometry with Siemens high-sensitivity CRP reagents on a BNII automated protein analyzer. PCT was measured by electrochemiluminescence using a Roche Cobas 8000 analyzer with original reagents. Biochemical parameters were analyzed using a Hitachi LABOSPECT 008 analyzer with original reagents. D-dimer was measured by immunoturbidimetry using a Sysmex CS-5100 analyzer with original reagents.

The study protocol was approved by the Ethics Committee of the First Affiliated Hospital of Xi'an Jiaotong University (Approval No. XJTU1AF2025LSYY-325). The requirement for informed consent was waived due to the retrospective nature of the study.

Reporting Guidelines

This study was conducted and reported in accordance with the RECORD (REporting of studies Conducted using Observational Routinely-collected Data) guidelines.

Definitions

Diagnosis and Classification of AP

AP was diagnosed according to Guidelines for diagnosis and treatment of acute pancreatitis in China (2021).[21] Mild acute pancreatitis (MAP) was defined as the absence of organ failure and local or systemic complications. Moderately severe acute pancreatitis (MSAP) was characterized by transient organ failure (<48 hours), local complications, or exacerbation of comorbidities. Severe acute pancreatitis (SAP) was defined as persistent organ failure (>48 hours) involving the respiratory, cardiovascular, or renal systems.

ICU Admission Criteria

ICU admission decisions were made by the attending physician and intensive care team based on clinical judgment, considering factors such as persistent organ failure, hemodynamic instability, or severe respiratory dysfunction.

Nomogram

A nomogram is a multivariable prediction tool that quantifies the contribution of each predictor and presents the results graphically, enabling clinicians to make rapid decisions based on individual patient data. Each variable in the nomogram corresponds to a point value, and the total score reflects the predicted probability of the outcome.

ICU Interventions

ICU care for patients with acute pancreatitis typically includes continuous cardiorespiratory monitoring, a 1:1 or 1:2 nurse-to-patient ratio, 24/7 in-house intensivist coverage, and access to organ support modalities such as invasive mechanical ventilation, vasopressor therapy, and continuous renal replacement therapy when indicated. General ward care, by contrast, involves intermittent vital sign monitoring, standard medical therapy, and transfer to ICU if deterioration occurs.

Follow-Up

All patients were followed until hospital discharge or death; no post-discharge follow-up was conducted.

Inclusion and Exclusion Criteria

Patients were eligible for inclusion if they met the following criteria:

(1) Diagnosis of AP according to the 2021 Chinese Guidelines for the Diagnosis and Treatment of Acute Pancreatitis;[21]

(2) Age ≥ 18 years;

(3) Completion of abdominal and chest imaging and key laboratory examinations within 24 hours of admission.

Exclusion criteria were as follows:

(1) Acute exacerbation of chronic pancreatitis;

(2) Pregnancy or lactation;

(3) Severe psychiatric disorders;

(4) Malignant tumors;
(5) Incomplete clinical data.

All included patients underwent routine laboratory testing within 24 hours of admission, including complete blood count, liver and renal function tests, electrolyte panels, and blood and urine amylase measurements. Abdominal ultrasonography and contrast-enhanced abdominal CT were also performed as clinically indicated.

A total of 1158 patients were initially screened. After exclusions for malignancy (n = 79), acute exacerbation of chronic pancreatitis (n = 10), age < 18 years (n = 20), and incomplete clinical data (n = 118), 931 patients were included in the final analysis.

Clinical Data Collection

The following data were extracted from electronic medical records:

(1) Baseline characteristics, including age, sex, etiology of AP, and medical history;
(2) Laboratory parameters obtained within 24 hours of admission, including triglycerides (TG), D-dimer, white blood cell count (WBC), BUN, creatinine (CREA), aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin (TB), calcium (Ca), gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), albumin (ALB), glucose (GLU), C-reactive protein (CRP), PCT, platelet count (PLT), neutrophil count (NEU), monocyte count (MO), lymphocyte count (LY), and red cell distribution width (RDW);
(3) Derived inflammatory indices, including CRP-to-albumin ratio (CAR), platelet-to-albumin ratio (PAR), NLR, PLR, systemic immune-inflammation index (SII), and systemic inflammatory response index (SIRI);
(4) In-hospital mortality.

Data Quality Control

Independent Data Review

To ensure data consistency and reliability, all patient data were independently reviewed by two researchers during data collection and analysis. Each patient's electronic medical record (EMR) was screened against the pre-defined inclusion and exclusion criteria, and data were verified by both reviewers. Any discrepancies in key data points were resolved through further discussion to ensure consistency.

Accuracy Verification

Two experienced clinical researchers independently reviewed key variables in the EMR (eg, laboratory results, admission records, and ICU admission status) to ensure data completeness and accuracy. If any inconsistencies or missing data were identified, the original records were re-checked, or clarification was sought from clinical staff.

Statistical Analysis

Statistical analyses were performed using SPSS (version 26.0), R (version 4.1.2), and MedCalc software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range (IQR), as appropriate, based on their distribution. Comparisons between groups were conducted using the independent samples *t*-test or Mann–Whitney *U*-test. Categorical variables were presented as frequencies and percentages and analyzed using the chi-square test. A two-sided P value < 0.05 was considered statistically significant.

To identify factors independently associated with ICU admission, forward stepwise logistic regression (FSR) and least absolute shrinkage and selection operator (LASSO) regression were employed to screen potential prognostic factors. For the exploratory analysis of in-hospital mortality among ICU-admitted patients, LASSO-Cox regression was used.

The discriminatory ability of individual markers and the combined model was evaluated using receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) with 95% confidence intervals (CI) was reported. A nomogram was constructed based on the multivariable logistic regression results to visualize the relative contribution of each predictor to ICU admission. Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. Internal validation was performed using bootstrap resampling (1000 iterations) to evaluate model stability

and optimism. Survival differences between ICU-admitted and non-ICU patients were compared using Kaplan–Meier curves with the Log rank test.

Results

Demographic Characteristics of the Patients

A total of 1158 patients with acute pancreatitis (AP) were initially screened. After applying the inclusion and exclusion criteria, 931 patients were included in the final analysis. Of these, 798 patients were managed without ICU admission, while 133 patients required ICU admission (Figure 1).

Baseline demographic and clinical characteristics are summarized in Table 1. No statistically significant differences were observed between the ICU and non-ICU groups with respect to age, sex, smoking history, alcohol consumption, or prior history of pancreatitis, diabetes, hypertension, or fatty liver disease (all $P > 0.05$). In contrast, patients admitted to the ICU had a higher proportion of hypertriglyceridemic pancreatitis, higher in-hospital mortality, and longer hospital stays compared with non-ICU patients (all $P < 0.05$).

Clinical Characteristics and Laboratory Findings

Patients admitted to the ICU showed significantly higher levels of TG, D-dimer, WBC, BUN, CREA, AST, TB, GLU, CRP, PCT, NEU, RDW, CAR, NLR, PLR, SII, and SIRI compared with non-ICU patients (all $P < 0.05$). Conversely, LY counts were significantly lower in the ICU group ($P < 0.05$). No significant differences were observed between groups in ALT, Ca, GGT, ALP, ALB, PLT, MO, or PAR (all $P > 0.05$) (Table 1).

Factors Associated with ICU Admission in Patients with AP

Variables demonstrating significant differences in univariate analyses were subsequently included in a multivariable logistic regression analysis using forward stepwise selection to identify factors independently associated with ICU admission. Prior to model building, multicollinearity diagnostics were performed among the candidate variables. Substantial collinearity was observed between CRP and the CAR, with a variance inflation factor (VIF) exceeding 10. To ensure model stability, CRP was excluded from subsequent analyses. The remaining variables—including hypertriglyceridemic pancreatitis, TG, D-dimer, WBC, BUN, CREA, AST, TB, GLU, PCT, NEU, RDW, CAR, NLR, PLR, SII, SIRI, and LY—were entered into the forward stepwise selection procedure to optimize the model. Four variables

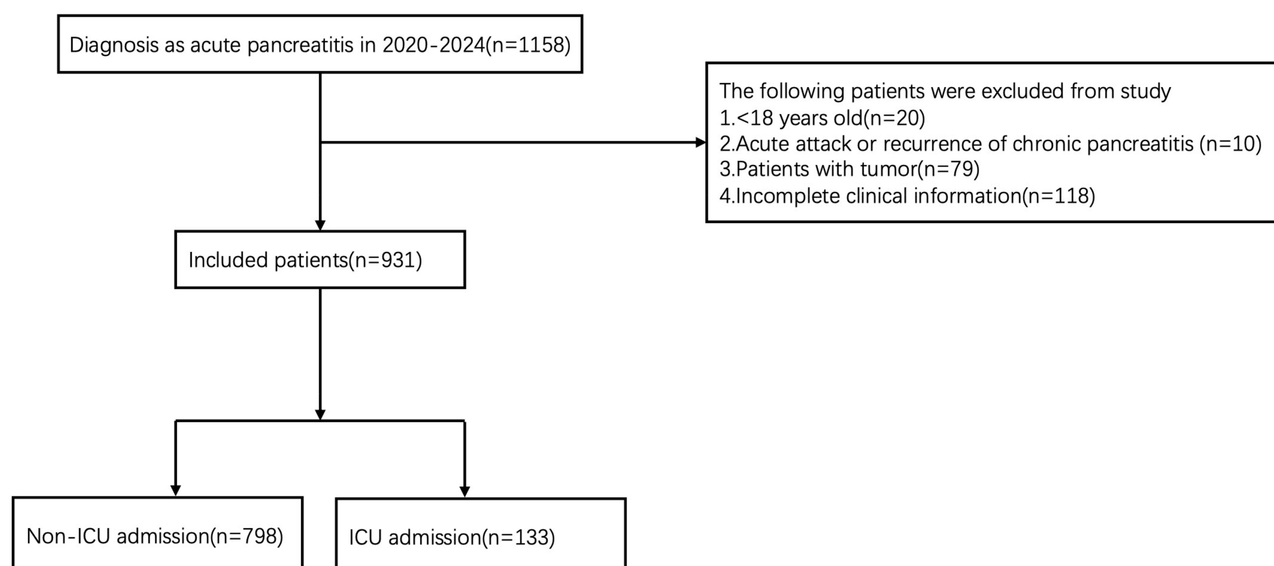

Figure 1 Flowchart of patient selection.
Abbreviation: ICU, Intensive care unit.

Table 1 Baseline Characteristics of Patients with AP

Variables	Total (n = 931)	Non-ICU Admission Group (n = 798)	ICU Admission Group (n = 133)	Statistic	*P* value
Demographics					
Age (years), M (Q_1, Q_3)	43.00 (34.00, 55.00)	43.00 (34.25, 56.00)	41.00 (34.00, 53.00)	Z=−0.70	0.482
Gender, n (%)				χ^2=0.05	0.822
Female	608 (65.31)	520 (65.16)	88 (66.17)		
Male	323 (34.69)	278 (34.84)	45 (33.83)		
Smoking, n (%)				χ^2=0.14	0.712
No	622 (66.81)	535 (67.04)	87 (65.41)		
Yes	309 (33.19)	263 (32.96)	46 (34.59)		
Alcohol, n (%)				χ^2=1.45	0.229
No	730 (78.41)	631 (79.07)	99 (74.44)		
Yes	201 (21.59)	167 (20.93)	34 (25.56)		
History of AP, n (%)				χ^2=0.21	0.645
No	771 (82.81)	659 (82.58)	112 (84.21)		
Yes	160 (17.19)	139 (17.42)	21 (15.79)		
Etiology of AP, n (%)				χ^2=18.47	<0.001
Billary	267 (28.68)	232 (29.07)	35 (26.32)		
Alcoholic	14 (1.50)	13 (1.63)	1 (0.75)		
Hypertriglyceridemic	387 (41.57)	311 (38.97)	76 (57.14)		
Others	263 (28.25)	242 (30.33)	21 (15.79)		
Status, n (%)				χ^2=115.59	<0.001
Survival	911 (97.85)	798 (100.00)	113 (84.96)		
Mortality	20 (2.15)	0 (0.00)	20 (15.04)		
Inpatient days, M (Q_1, Q_3)	9.00 (7.00, 14.00)	9.00 (7.00, 13.00)	18.00 (12.00, 29.00)	Z=−10.77	<0.001
Comorbidities					
Diabetes, n (%)				χ^2=0.14	0.709
No	752 (80.77)	643 (80.58)	109 (81.95)		
Yes	179 (19.23)	155 (19.42)	24 (18.05)		
Hypertension, n (%)				χ^2=0.05	0.818
No	735 (78.95)	629 (78.82)	106 (79.70)		
Yes	196 (21.05)	169 (21.18)	27 (20.30)		
Hypertriglyceridemia, n (%)				χ^2=12.08	<0.001
No	387 (41.57)	350 (43.86)	37 (27.82)		
Yes	544 (58.43)	448 (56.14)	96 (72.18)		
Fatty liver, n (%)				χ^2=0.17	0.683
No	651 (69.92)	560 (70.18)	91 (68.42)		
Yes	280 (30.08)	238 (29.82)	42 (31.58)		
Laboratory parameters					
TG (mmol/L) M (Q_1, Q_3)	1.95 (1.07, 3.58)	1.89 (1.03, 3.44)	2.26 (1.34, 4.04)	Z=−2.64	0.008
D-dimer (mg/L) M (Q_1, Q_3)	2.11 (0.84, 4.80)	1.62 (0.76, 4.02)	6.97 (3.25, 10.61)	Z=−11.37	<0.001
WBC (10^9/L), M (Q_1, Q_3)	11.62 (8.05, 15.38)	11.48 (7.95, 15.13)	12.50 (9.39, 18.49)	Z=−2.60	0.009
BUN (mmol/L), M (Q_1, Q_3)	4.69 (3.50, 6.04)	4.45 (3.40, 5.66)	7.18 (5.16, 11.90)	Z=−10.37	<0.001
CREA (μmol /L), M (Q_1, Q_3)	56.00 (45.00, 68.00)	55.00 (45.00, 67.00)	62.00 (44.00, 116.00)	Z=−4.11	<0.001
AST (U/L) M (Q_1, Q_3)	24.00 (19.00, 33.00)	24.00 (18.00, 31.00)	27.00 (20.00, 40.00)	Z=−3.52	<0.001
ALT (U/L) M (Q_1, Q_3)	29.00 (19.00, 46.00)	28.00 (19.00, 46.00)	29.00 (19.00, 48.00)	Z=−0.79	0.432
TB (μmol /L) M (Q_1, Q_3)	17.10 (12.30, 26.15)	16.80 (12.12, 24.78)	20.30 (13.80, 36.90)	Z=−3.50	<0.001
Ca (mmol/L) M (Q_1, Q_3)	2.14 (2.04, 2.25)	2.14 (2.04, 2.24)	2.15 (2.04, 2.28)	Z=−0.43	0.669
GGT (U/L) M (Q_1, Q_3)	64.00 (34.00, 129.00)	63.50 (33.00, 128.00)	70.00 (36.00, 135.00)	Z=−0.84	0.398
ALP (U/L) M (Q_1, Q_3)	87.00 (70.00, 114.00)	87.00 (70.00, 112.00)	92.00 (67.00, 127.00)	Z=−1.25	0.213
ALB (g/L), M (Q_1, Q_3)	35.40 (31.90, 38.95)	35.55 (32.02, 39.10)	35.10 (31.30, 38.20)	Z=−1.48	0.138
GLU (mmol/L) M (Q_1, Q_3)	6.60 (5.13, 9.50)	6.36 (5.02, 9.14)	8.65 (6.46, 10.78)	Z=−5.71	<0.001
CRP (mg/L), M (Q_1, Q_3)	35.40 (9.11, 93.30)	31.75 (7.94, 91.97)	57.90 (31.00, 100.30)	Z=−4.41	<0.001

(*Continued*)

Table 1 (Continued).

Variables	Total (n = 931)	Non-ICU Admission Group (n = 798)	ICU Admission Group (n = 133)	Statistic	*P* value
PCT (ng/mL), M (Q_1, Q_3)	0.16 (0.06, 0.48)	0.13 (0.05, 0.37)	0.67 (0.19, 2.15)	Z=−9.94	<0.001
PLT (10^9/L), M (Q_1, Q_3)	234.00 (182.00, 296.50)	234.00 (183.00, 290.00)	252.00 (171.00, 353.00)	Z=−0.83	0.409
NEU (10^9/L), M (Q_1, Q_3)	7.12 (4.45, 10.41)	6.77 (4.30, 10.10)	9.12 (5.49, 13.37)	Z=−4.61	<0.001
MO (10^9/L), M (Q_1, Q_3)	0.45 (0.32, 0.64)	0.45 (0.32, 0.62)	0.46 (0.33, 0.71)	Z=−1.14	0.256
LY (10^9/L), M (Q_1, Q_3)	1.31 (0.93, 1.73)	1.33 (0.96, 1.73)	1.15 (0.79, 1.62)	Z=−3.29	0.001
RDW (%), M (Q_1, Q_3)	44.00 (42.00, 46.60)	43.70 (41.80, 45.80)	48.40 (45.10, 51.70)	Z=−10.49	<0.001
Inflammatory indexes					
CAR, M (Q_1, Q_3)	1.04 (0.25, 2.83)	0.91 (0.23, 2.78)	1.67 (0.84, 3.38)	Z=−4.61	<0.001
PAR, M (Q_1, Q_3)	6.60 (4.96, 8.60)	6.57 (4.96, 8.37)	6.87 (4.85, 10.27)	Z=−1.29	0.197
NLR, M (Q_1, Q_3)	5.50 (2.96, 9.44)	5.06 (2.75, 8.76)	7.85 (4.79, 13.82)	Z=−5.82	<0.001
PLR, M (Q_1, Q_3)	180.82 (128.30, 253.48)	176.51 (128.13, 243.67)	220.00 (135.90, 310.81)	Z=−2.95	0.003
SII, M (Q_1, Q_3)	1294.73 (677.80, 2258.64)	1245.57 (640.86, 2069.12)	1872.87 (927.67, 3168.46)	Z=−5.03	<0.001
SIRI, M (Q_1, Q_3)	2.50 (1.18, 5.21)	2.38 (1.10, 4.70)	4.00 (1.89, 7.75)	Z=−4.96	<0.001

Notes: Data are presented as n (%) for categorical variables and as median (M) with interquartile range (Q_1, Q_3) for continuous variables, where Q_1 and Q_3 represent the 25th and 75th percentiles, respectively. $P < 0.05$ was considered statistically significant. Z: Mann–Whitney test, χ^2: Chi-square test.

Abbreviations: TG, Triglyceride; WBC, White blood cell; BUN, Blood urea nitrogen; CREA, Serum creatinine; AST, Aspartate aminotransferase; ALT, Alanine Aminotransferase; TB, Total bilirubin; Ca, Calcium; GGT, Gamma-Glutamyl Transferase; ALP, Alkaline Phosphatase; ALB, Albumin; GLU, Glucose; CRP, C-reactive protein; PCT, Procalcitonin; PLT, Platelet; NEU, Neutrophil; MO, Monocyte; LY, Lymphocyte; RDW, Red blood cell distribution width; CAR, C-reactive protein to Albumin Ratio; PAR, Platelet to Albumin Ratio; NLR, Neutrophil to lymphocyte ratio; PLR, Platelet to lymphocyte ratio; SII, Systemic immune-inflammation index; SIRI, Systemic inflammatory response index; AP, Acute pancreatitis; ICU, Intensive care unit.

were retained as independently associated with ICU admission: hypertriglyceridemic pancreatitis (odds ratio [OR] 2.88, 95% CI 1.59–5.21), D-dimer (OR 1.13, 95% CI 1.07–1.18), BUN (OR 1.36, 95% CI 1.25–1.48), and RDW (OR 1.14, 95% CI 1.09–1.19) (Table 2).

To further validate the selection of predictors, LASSO regression was applied as a sensitivity analysis. Using 10-fold cross-validation with the minimum criteria to tune the regularization parameter (λ), four variables with non-zero coefficients were identified as potential predictors: hypertriglyceridemic pancreatitis (OR 2.21, 95% CI 2.12–5.82), D-dimer (OR 1.32, 95% CI 1.63–2.19), BUN (OR 1.91, 95% CI 2.25–4.69), and RDW (OR 1.33, 95% CI 1.58–2.41). These findings were consistent with those obtained from the forward stepwise logistic regression, confirming the robustness of the selected variables.

Nomogram for Predicting ICU Admission

A nomogram was constructed based on the multivariable logistic regression analysis to visualize the association between selected variables and ICU admission (Figure 2). Etiology of acute pancreatitis was coded as follows: 1 = biliary, 2 = alcoholic, 3 = hypertriglyceridemic, and 4 = other causes.

ROC curve analysis was performed to evaluate the discriminatory ability of individual predictors and the combined model within the study cohort. The AUC values for hypertriglyceridemic pancreatitis, D-dimer, BUN, and RDW were 0.61 (95% CI 0.56–0.66), 0.81 (95% CI 0.77–0.85), 0.78 (95% CI 0.73–0.83), and 0.78 (95% CI 0.74–0.83), respectively. The multivariable model incorporating these four predictors demonstrated good apparent discrimination, with an AUC of 0.89 (95% CI 0.86–0.93) (Figure 3).

Internal Validation

Internal validation was performed using bootstrap resampling with 1000 iterations. The calibration curve demonstrated good agreement between observed and predicted probabilities within the derivation cohort (Figure 4). The Hosmer–Lemeshow goodness-of-fit test yielded a *P* value of 0.517, indicating no evidence of poor calibration.

 https://doi.org/10.2147/JIR.S591892

Table 2 Multivariable Analysis of Factors Associated with ICU Admission in AP

Predictive factor	β	S.E.	Wald	*P* value	OR (95% CI)
Intercept	−11.12	1.09	−10.20	<0.001	0.00 (0.00 ~ 0.00)
Etiology of AP					
Hypertriglyceridemic	1.06	0.30	3.49	<0.001	2.88 (1.59 ~ 5.21)
D-dimer (mg/L)	0.12	0.03	4.54	<0.001	1.13 (1.07 ~ 1.18)
BUN (mmol/L)	0.31	0.04	7.35	<0.001	1.36 (1.25~ 1.48)
RDW (%)	0.13	0.02	5.95	<0.001	1.14 (1.09 ~ 1.19)

Notes: β, Regression coefficient; S.E., Standard error. $P < 0.05$ was considered statistically significant.
Abbreviations: AP, Acute pancreatitis; ICU, Intensive care unit; BUN, Blood urea nitrogen; RDW, Red blood cell distribution width; OR, Odds ratio; CI, Confidence interval.

Exploratory Prognostic Analysis of In-Hospital Mortality Among ICU Patients

Among the 133 ICU-admitted patients, 20 in-hospital deaths were recorded. Given the limited number of events (n = 20) and the associated risk of overfitting, LASSO-Cox regression was performed as a sensitivity analysis. Using 10-fold cross-validation with the lambda.min criterion, the optimal penalty parameter (λ) was selected. At the optimal λ value (lambda.1se), the LASSO model retained a single predictor with a non-zero coefficient: elevated CAR (HR 2.12, 95% CI 1.76–3.27). By applying a stricter penalty to reduce overfitting, LASSO regression identified CAR as a possible predictor of in-hospital mortality in this critically ill population.

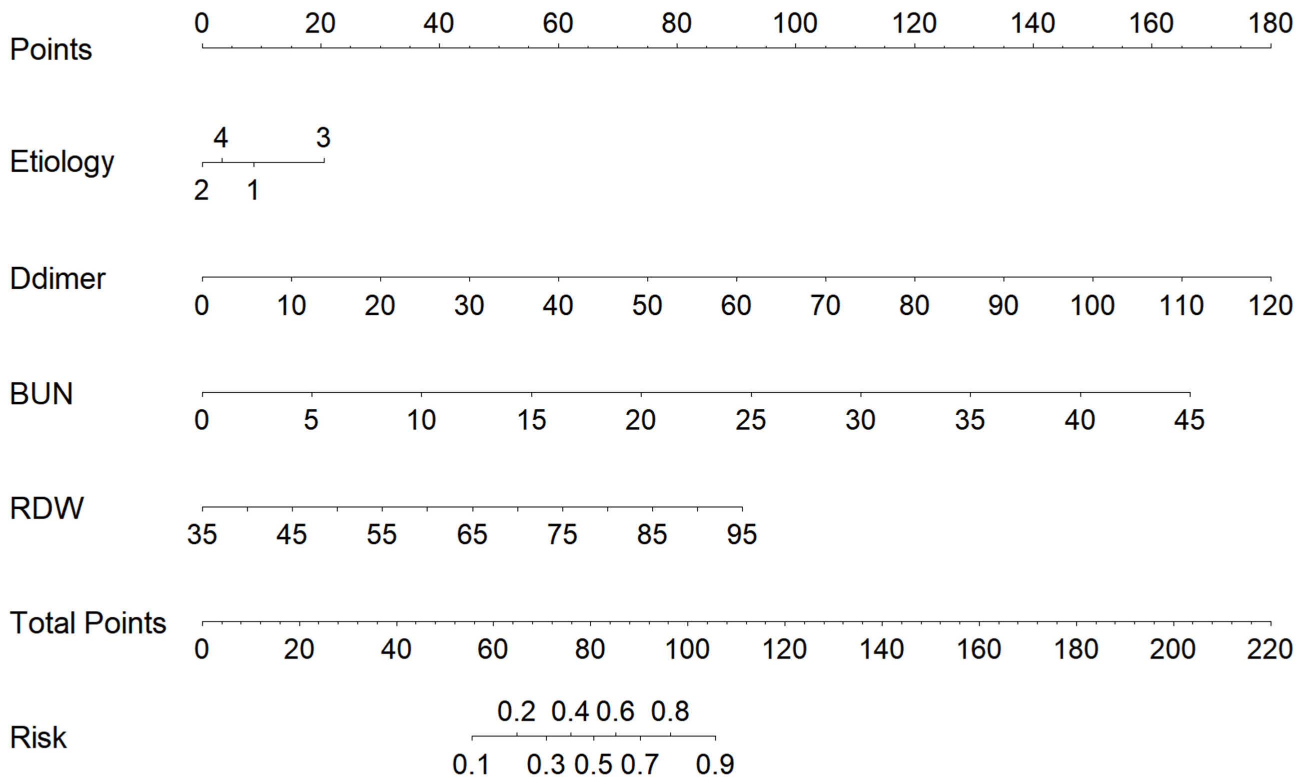

Figure 2 Nomogram for predicting ICU admission in patients with AP. The nomogram was constructed based on a multivariable logistic regression model and incorporates four predictors: hypertriglyceridemic pancreatitis, D-dimer, BUN, and RDW. Each predictor is assigned a point value on the "Points" scale, and the total points accumulated across all predictors correspond to the predicted probability of ICU admission on the "Risk" scale. This nomogram allows individualized risk estimation based on admission characteristics. Etiology coding: 1, biliary; 2, alcoholic; 3, hypertriglyceridemic; 4, other.
Abbreviations: AP, Acute pancreatitis; BUN, Blood urea nitrogen; RDW, Red blood cell distribution width; ICU, Intensive care unit.

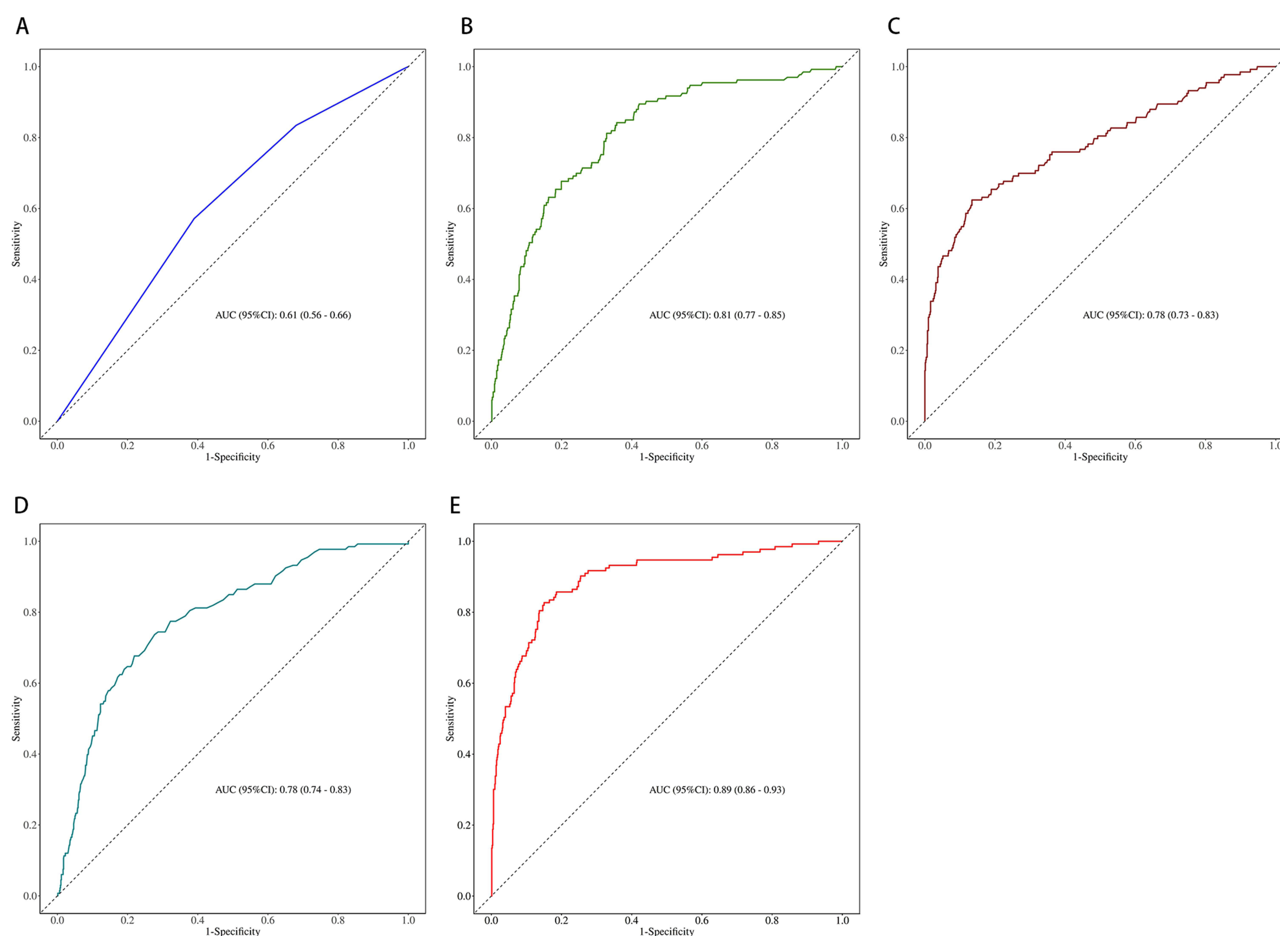

Figure 3 ROC curves showing the apparent discrimination of individual variables and the multivariable model for predicting ICU admission in AP. (**A**) Etiology of AP; (**B**) D-dimer; (**C**) BUN; (**D**) RDW; (**E**) Multivariable model for ICU admission in AP.
Abbreviations: BUN, Blood urea nitrogen; RDW, Red blood cell distribution width; ICU, Intensive care unit; AP, Acute pancreatitis; ROC, Receiver operating characteristic.

Discriminatory Performance of CAR for Mortality in ICU Patients

The ability of CAR to discriminate between survivors and non-survivors in the ICU subgroup was assessed using ROC curve analysis. CAR demonstrated moderate discriminatory ability, with an AUC of 0.78 (95% CI: 0.69–0.88) (Figure 5).

Kaplan-Meier Survival Analysis

Kaplan-Meier survival analysis revealed that patients admitted to the ICU had significantly lower survival probabilities compared to those not requiring ICU admission (log-rank $P < 0.001$) (Figure 6).

Discussion

AP is a globally prevalent disease characterized by a highly heterogeneous clinical course and widely variable outcomes.[22] Early identification of patients at risk of clinical deterioration remains a critical goal, particularly in resource-limited settings.[23] Organ dysfunction and local or systemic complications are key determinants of early risk stratification; however, their onset is often delayed.[4] Therefore, identifying reliable prognostic markers is essential for optimizing early treatment and guiding resource allocation. There is an urgent need for biomarkers that are safe, simple, rapid, cost-effective, readily available at admission, reproducible, and observer-independent.

In this study, we used real-world retrospective data to examine factors associated with ICU admission in patients with AP and to explore laboratory markers associated with in-hospital mortality among those admitted to the ICU. Importantly, these two analyses address distinct clinical questions. The ICU admission analysis focuses on early risk

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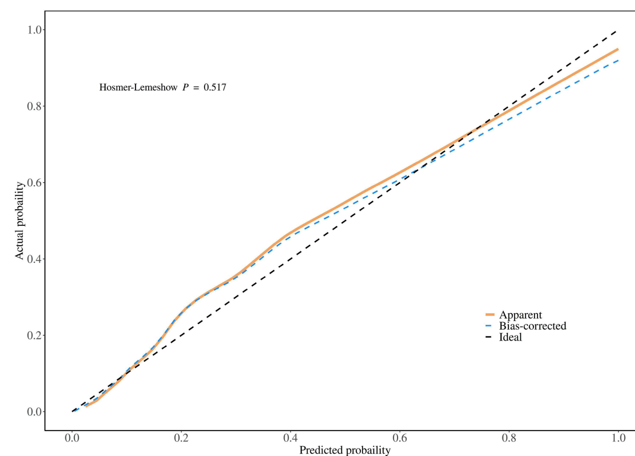

Figure 4 Calibration curve model for patients with admitted to the ICU. The x-axis represents the predicted probability calculated using the model, while the y-axis represents the observed probability. The apparent calibration curve (dotted line) indicates model performance in the original data, whereas the bias-corrected curve (solid line) represents model performance after correction for optimism using 1000 bootstrap resamples. Perfect prediction would align with the 45° (dashed) reference line.
Abbreviations: AP, Acute pancreatitis; ICU, Intensive care unit.

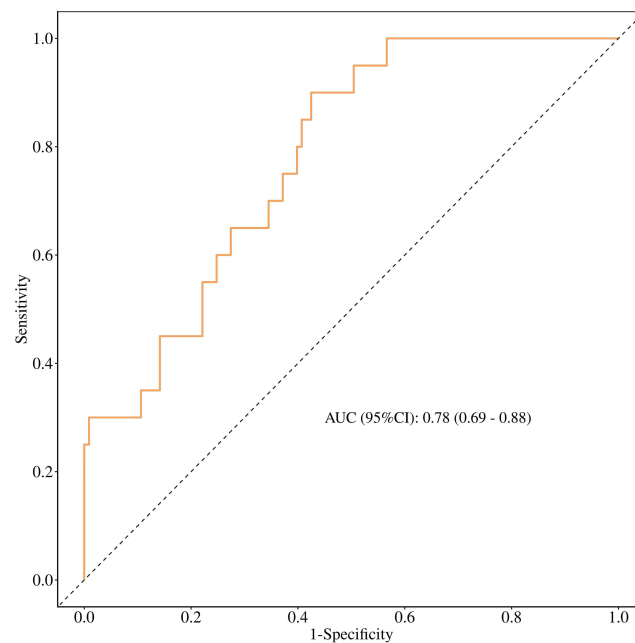

Figure 5 ROC curve of CAR for predicting in-hospital mortality in ICU patients.
Abbreviations: CAR, CRP-to-albumin ratio; ICU, Intensive care unit; ROC, Receiver operating characteristic.

stratification at initial presentation, whereas the mortality analysis is restricted to patients who have already progressed to critical illness and therefore represents an exploratory prognostic assessment within the ICU population—rather than a generalizable mortality prediction model.

In the ICU admission analysis, hypertriglyceridemic pancreatitis, elevated D-dimer, elevated BUN, and elevated RDW were independently associated with ICU admission. These variables are routinely available at hospital admission and reflect complementary pathophysiological processes, including metabolic disturbance, coagulation activation, renal dysfunction, and systemic inflammation.

Hypertriglyceridemic pancreatitis (HTG-AP) has emerged as an increasingly important etiology of AP in China, likely related to changes in lifestyle and dietary patterns.[21,24–26] Consistent with previous reports,[26] HTG-AP in our cohort was associated with a higher likelihood of ICU admission. Previous meta-analyses have demonstrated that HTG-

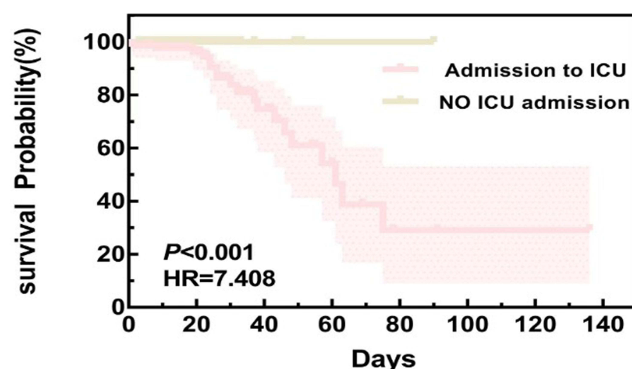

Figure 6 In-hospital survival of patients with AP stratified by ICU admission. The x-axis shows days from hospital admission to death or discharge. Tick marks indicate censoring at discharge. The ICU group had significantly lower survival.
Abbreviations: AP, Acute pancreatitis; ICU, Intensive care unit.

AP is associated with increased risks of persistent systemic inflammatory response syndrome, organ failure, and mortality.[27] The present findings further support the association between HTG-AP and more severe clinical trajectories, although etiological classification alone cannot fully capture the dynamic severity of the disease.

D-dimer reflects activation of coagulation and fibrinolytic pathways, which are increasingly recognized as key components of AP pathophysiology.[28,29] Microcirculatory dysfunction and a hypercoagulable state may contribute to organ failure in critically ill patients.[30–32] Newton et al[33] demonstrated that D-dimer levels were significantly elevated in patients requiring ICU admission compared to those managed in general wards, with a cut-off value of 933.5 ng/L predicting complicated acute pancreatitis—including the need for intensive care—supporting its utility as an early triage marker in resource-limited settings. Similarly, Wan et al[34] reported that among 3451 patients with AP, those with elevated D-dimer at admission were more likely to develop pancreatic necrosis and organ failure than those with normal levels. In a small pediatric cohort of 36 patients, Boskovic et al[35] suggested that D-dimer may serve as a simple clinical marker for predicting disease severity and local complications in children with AP. In the present study, elevated D-dimer was independently associated with ICU admission (OR 1.13, 95% CI 1.07–1.18), further supporting its role as an early, accessible marker for identifying patients at high risk of clinical deterioration.

Elevated BUN may reflect hypovolemia, renal hypoperfusion, and early organ dysfunction.[36] Faisst et al[37] demonstrated that higher BUN levels at admission were associated with prolonged ICU stay in patients with acute necrotizing pancreatitis. Similarly, Dai et al[36] reported that elevated BUN (>10.745 mmol/L) was associated with increased risks of invasive mechanical ventilation and ICU admission. Consistent with these findings, BUN was independently associated with ICU admission in our cohort (OR 1.36, 95% CI 1.25–1.48). Nevertheless, BUN is a nonspecific marker influenced by multiple clinical factors, and its prognostic value should be interpreted in conjunction with the overall clinical context.

RDW is a routinely reported hematological parameter that reflects the degree of heterogeneity in erythrocyte volume and has been shown to have clinical utility in various diseases, including AP.[38] Data on RDW as an inflammatory marker in AP remain limited. In a study of 185 patients with AP, O'Connell et al[39] reported that elevated RDW was an independent predictor of ICU or high dependency unit (HDU) admission, with an AUC of 0.63 (95% CI 0.463–0.797). At a cut-off value of 14.65%, RDW predicted ICU admission with a sensitivity of 46.7% and a specificity of 80%; at a cut-off of 15.5%, sensitivity was 33.3% and specificity was 89.4%. In the present study, RDW was independently associated with ICU admission (OR 1.14, 95% CI 1.09–1.19), and the AUC was 0.78 (95% CI 0.74–0.83). Notably, RDW is routinely included in the complete blood count report, meaning that it is immediately available to emergency and admitting clinicians at the time of patient assessment, without incurring additional costs or resource burden.

Based on the four identified predictors, we developed a cost-effective and readily available risk prediction model for early identification of patients with AP at high risk of ICU admission. The model integrates simple laboratory markers and clinical characteristics, enabling timely initiation of life-support interventions and potentially improving outcomes in

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high-risk populations. Its simplicity and low cost underscore its potential applicability across diverse healthcare settings, particularly in resource-limited environments.

To contextualize our findings, we compared the performance of our model with established scoring systems reported in the literature. In a meta-analysis by Zhu et al[40] the Ranson and BISAP scores predicted ICU admission in patients with AP with pooled AUCs of 0.92 (95% CI: 0.81–1.00) and 0.86 (95% CI: 0.67–1.00), respectively. In a prospective study of 50 patients with AP, Harshit et al[41] compared the accuracy of four scoring systems for predicting ICU admission. The modified CTSI demonstrated the highest predictive accuracy, with an AUC of 0.993 (95% CI: 0.975–1.000), followed by the Ranson score (AUC 0.910, 95% CI: 0.767–1.000), APACHE II (AUC 0.885, 95% CI: 0.783–0.987), and BISAP (AUC 0.877, 95% CI: 0.739–1.000). These findings suggest that modified CTSI and the Ranson score are reliable tools for identifying patients likely to require intensive care. Our prediction model achieved an AUC of 0.89 (95% CI: 0.86–0.93), which is comparable to the performance of these established scoring systems, supporting its potential clinical utility. However, given the retrospective, single-center design and the lack of standardized ICU admission criteria, these results should be interpreted as hypothesis-generating rather than definitive predictors of disease severity. External validation in diverse populations is warranted to confirm the generalizability of our model.

The clinical utility of our model lies in its ability to provide an early, objective estimate of a patient's likelihood of requiring ICU admission. This estimate can inform several practical decisions: in the emergency department, it can help prioritize which patients should be considered for ICU referral; in resource-limited settings, it can guide primary care physicians in deciding which patients warrant transfer to a higher-level facility; and for hospital administrators, it can aid in anticipating ICU bed demand and allocating resources. The model does not aim to prescribe specific ICU interventions, but rather serves as an early warning tool to identify patients at heightened risk, prompting closer observation and timely clinical assessment.

In an exploratory analysis, we further investigated laboratory parameters associated with in-hospital mortality in ICU patients. Given the limited number of deaths (n = 20) and the resulting risk of overfitting, we performed LASSO-Cox regression as a sensitivity analysis. By applying a more stringent penalty to reduce overfitting, LASSO regression found that elevated CAR was a possible predictor of in-hospital mortality in this critically ill patient population (HR 2.12, 95% CI 1.76–3.27).

CAR is a novel inflammation-based biomarker that has been associated with inflammatory severity and mortality in various diseases.[42] However, evidence regarding its prognostic value in AP, particularly in Chinese populations, remains limited. Kaplan et al[43] reported that each unit increase in CAR was associated with a 1.52-fold increase in mortality risk in a cohort of 192 patients with AP. Similarly, Kiyak et al[44] found that CAR had higher sensitivity and specificity for predicting mortality in AP compared to NLR and PLR, with an AUC of 0.856. In the present study, we evaluated the discriminatory ability of CAR using ROC curve analysis in the ICU subgroup. CAR demonstrated moderate discriminatory power, with an AUC of 0.78 (95% CI: 0.69–0.88), for distinguishing between survivors and non-survivors.

To contextualize our findings, we compared the performance of CAR with established scoring systems reported in the literature. In a meta-analysis by Zhu et al[42] the pooled AUCs for predicting mortality in AP were 0.91 (95% CI: 0.88–0.93) for the Ranson score and 0.92 (95% CI: 0.90–0.94) for BISAP, with no significant difference between the two (P = 0.480). Another meta-analysis by Gao et al[45] reported that APACHE II predicted in-hospital mortality in AP with a pooled AUC of 0.83, a sensitivity of 0.95 (95% CI: 0.77–1.00), and a specificity of 0.68 (95% CI: 0.63–0.73). While the AUC of CAR in our ICU cohort (0.78) was lower than those reported for these multi-parameter scoring systems, it is important to note that CAR is a single, easily measurable marker. Its predictive performance, combined with its simplicity and low cost, suggests that CAR may serve as a useful adjunct for early risk stratification, particularly in resource-limited settings. However, given the exploratory nature of this analysis and the small number of events, these findings should be interpreted with caution and require validation in larger, multicenter cohorts.

Limitations

This study has several limitations. First, the retrospective, single-center design may introduce selection bias and residual confounding, limiting the generalizability of the findings. Second, the mortality analysis was restricted to ICU-admitted patients and included a small number of outcome events (n = 20), precluding robust predictive modeling and external

generalization; this analysis should therefore be considered exploratory. Third, the proposed models were not compared with established severity scoring systems (eg, BISAP, APACHE II, Ranson), and no conclusions regarding incremental predictive value can be drawn. Fourth, ICU admission decisions were based on clinical judgment rather than standardized criteria, which may introduce heterogeneity. Fifth, because ICU admission does not necessarily equate to severe AP, these findings should not be interpreted as direct predictors of disease severity. Finally, These findings should be viewed as hypothesis-generating, and confirmation in larger, prospective multicenter studies is essential before any clinical application can be considered, to validate our findings and confirm the generalizability of our model across different populations and settings.

Conclusion

Routinely available inflammatory and biochemical markers might be associated with ICU admission and in-hospital mortality in patients with acute pancreatitis. These findings may assist early clinical risk stratification and complement existing clinical assessments. However, prospective multicenter studies with larger cohorts are warranted to validate these findings.

Abbreviations

ALB, Albumin; ALP, Alkaline Phosphatase; ALT, Alanine Aminotransferase; AP, Acute Pancreatitis; APACHE II, Acute Physiology and Chronic Health Evaluation II; AST, Aspartate Aminotransferase; AUC, Area Under the Curve; BISAP, Bedside Index for Severity in Acute Pancreatitis; BUN, Blood Urea Nitrogen; Ca, Calcium; CAR, C-Reactive Protein-to-Albumin Ratio; CI, Confidence Interval; CREA, Creatinine; CRP, C-Reactive Protein; CT, Computed Tomography; D-dimer, A fibrin degradation product used as a marker of coagulation; GGT, Gamma-Glutamyl Transferase; GLU, Glucose; HR, Hazard Ratio; HTG-AP, Hypertriglyceridemia-induced Acute Pancreatitis; ICU, Intensive Care Unit; JSS, Japanese Severity Score; LY, Lymphocyte Count; MCTSI, Modified Computed Tomography Severity Index; MO, Monocyte Count; NLR, Neutrophil-to-Lymphocyte Ratio; NEU, Neutrophil Count; OR, Odds Ratio; PAR, Platelet-to-Albumin Ratio; PCT, Procalcitonin; PLR, Platelet-to-Lymphocyte Ratio; PLT, Platelet Count; RDW, Red Blood Cell Distribution Width; ROC, Receiver Operating Characteristic; SAP, Severe Acute Pancreatitis; SII, Systemic Immune-Inflammation Index (PLT × NEU/LY); SIRI, Systemic Inflammatory Response Index (MO × NEU/LY); SIRS, Systemic Inflammatory Response Syndrome; SPSS, Statistical Package for the Social Sciences; TB, Total Bilirubin; TG, Triglycerides; WBC, White Blood Cell Count.

Data Sharing Statement

All the data included in this study are available upon request by contacting the corresponding author.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the Ethics Committee of the First Affiliated Hospital of Xi'an Jiaotong University (Approval No. XJTU1AF2025LSYY-325). Due to the retrospective design and the use of anonymized routinely collected clinical data, the requirement for written informed consent was waived by the Ethics Committee in accordance with institutional and national guidelines.

Author Contributions

Qian Wu: Conceptualization, Methodology, Software, Formal analysis, Data curation, Visualization, Writing – original draft, Writing – review & editing.

Thomas Stuart Mughogho: Formal analysis, Software, Writing – original draft, Writing – review & editing.

Qi Zhang: Investigation, Data curation, Writing – review & editing.

Dokani Michael Ndovi: Formal analysis, Software, Writing – original draft, Writing – review & editing.

Shangwu Liu: Data curation, Formal analysis, Software, Writing – review & editing.

https://doi.org/10.2147/JIR.S591892

Xiaoqin Wang: Conceptualization, Supervision, Project administration, Methodology, Funding acquisition, Writing – review & editing, Resources.

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work. Qian Wu and Thomas Stuart Mughogho are co-first authors and have contributed equally to this work.

Funding

This research was funded by The Science and Technology Program of Xi'an (Grant Number: 24LLRHZDZX0028) and University-Industry-Research Innovation Fund of the Ministry of Education of the People's Republic of China (Grant Number: 2025MR003).

Disclosure

The authors have no relevant financial or non-financial interests to disclose.

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