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Analysis of the prognostic value of hematocrit, procalcitonin, albumin combined with Balthazar CT score and APACHE II score in patients with severe acute pancreatitis

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Abstract

This study aims to evaluate the prognostic value of hematocrit (HCT), procalcitonin (PCT), albumin (ALB), and Balthazar CT score combined with APACHE II score in patients with severe acute pancreatitis (SAP). This retrospective cohort study included 102 patients with SAP treated at our hospital from January 2021 to January 2024. All patients were divided into a good prognosis group (68 cases) and a poor prognosis group (34 cases) based on clinical outcomes. Basic information, clinical indicators, laboratory test data, and imaging scores (Balthazar CT score) were collected for all patients, along with APACHE II scores. Multivariate regression analysis was used to identify independent risk factors for poor prognosis, and ROC curve analysis was conducted to evaluate the predictive efficacy of individual indicators and the combined model. There were significant differences between the poor prognosis and good prognosis groups in terms of gender, age, BMI, diabetes, and hypertension ($P < .05$). Specifically, the mean age in the poor prognosis group was 56.1 ± 12.8 years, significantly higher than that in the good prognosis group (45.3 ± 10.2 years, $P = .001$). Additionally, the poor prognosis group had significantly higher APACHE II, Ranson, Balthazar CT, and SOFA scores than the good prognosis group ($P < .001$). Laboratory results showed significantly elevated levels of PCT, white blood cell count, neutrophil percentage, blood glucose, serum creatinine, and total bilirubin in the poor prognosis group, while HCT and ALB levels were significantly lower ($P < .001$). Multivariate regression analysis identified APACHE II score, Balthazar CT score, and PCT as independent risk factors for poor prognosis, while HCT and ALB levels were protective factors ($P < .05$). ROC curve analysis revealed that PCT (AUC = 0.85) had the strongest predictive efficacy among individual indicators, while the combined model (APACHE II score, Balthazar CT score, HCT, PCT, and ALB) had the best overall prognostic performance for SAP (AUC = 0.88, $P < .001$). The combined model of APACHE II score, Balthazar CT score, PCT, HCT, and ALB demonstrates high predictive efficacy for evaluating the prognosis of SAP. This model provides clinicians with more accurate prognostic predictions and exhibits good stability and effectiveness across different patient subgroups.

Abbreviations: ALB = albumin, AP = acute pancreatitis, HCT = hematocrit, PCT = procalcitonin, SAP = severe acute pancreatitis.

Keywords: albumin, APACHE II score, Balthazar CT score, biomarkers, clinical prognosis, hematocrit, procalcitonin, prognostic evaluation, retrospective study, severe acute pancreatitis

1. Introduction

Acute pancreatitis (AP) is a common gastrointestinal emergency characterized primarily by the sudden onset of upper abdominal pain and elevated serum pancreatic enzyme levels.[1–3] Although most cases of AP are mild with favorable outcomes, approximately 15 to 20% of patients may progress to severe acute pancreatitis (SAP), a rapidly progressing condition that can lead to persistent organ dysfunction, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome, with a mortality rate as high as 20 to 30%.[4,5] In recent years, the global incidence of AP has been on the rise, reaching 34 to 45 cases per 100,000 population annually in several countries and regions.[6] Epidemiological data from China also show a yearly increase in AP incidence, with gallstones, alcohol abuse, and hyperlipidemia being the primary causes.

For patients with SAP, early identification of high-risk individuals and timely implementation of effective interventions are critical for reducing mortality and improving outcomes.[7]

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The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Currently, commonly used clinical assessment tools include the Acute Physiology and Chronic Health Evaluation II (APACHE II) scoring system, the Balthazar pancreatic CT grading system, and laboratory indicators such as serum procalcitonin (PCT), hematocrit (HCT), and albumin (ALB).[8] Among them, the APACHE II score is sensitive in evaluating overall systemic illness but lacks specificity for local pancreatic changes[9]; the Balthazar CT score provides a visual assessment of pancreatic structural alterations and the extent of necrosis but does not reflect systemic inflammatory status[10]; serum PCT, a sensitive marker of infection and inflammation, is valuable in early assessment of SAP but may be influenced by concurrent infections or renal dysfunction.[11] Each of these indicators has inherent limitations when used alone in clinical settings.

Recent studies suggest that integrating multiple parameters can improve the accuracy of SAP severity and prognosis assessment. Elevated PCT levels are closely associated with infection, pancreatic necrosis, and sepsis; hypoalbuminemia often indicates a high-inflammatory state, and may also reflect malnutrition and increased capillary leakage, serving as an independent predictor of disease progression[12]; decreased HCT may suggest hemodilution, bleeding, or worsening inflammation, and is also associated with poor prognosis.[13] Therefore, combining biochemical markers such as PCT, ALB, and HCT with the APACHE II score and Balthazar CT grading may help establish a more comprehensive and accurate assessment system, enhancing early risk stratification and individualized treatment for SAP.

Despite ongoing advances in SAP research, there is still no unified standard or robust high-quality evidence for effectively integrating clinical scoring systems, imaging classifications, and laboratory biomarkers in the early stages of the disease to build a multidimensional predictive model. Hence, this study aims to explore the prognostic value of combining HCT, PCT, ALB, pancreatic CT grading, and APACHE II score in patients with SAP. Through a retrospective analysis of clinical data, we intend to establish a practical and operable multi-parameter predictive model to aid clinicians in early identification of high-risk patients and in formulating individualized treatment strategies, thereby improving the overall management and outcomes of SAP.

2. Materials and methods

2.1. Study design

This study was approved by the Ethics Committee of Jinhua Hospital. This study was a retrospective cohort study approved by the Ethics Committee of our hospital. Data were extracted from the hospital case database for 102 patients diagnosed with SAP who were treated at our hospital between January 2021 and January 2024. Clinical data were analyzed based on electronic medical records, imaging examinations, and laboratory test results. According to clinical outcomes obtained through follow-up, all patients were categorized into a good prognosis group (68 cases) and a poor prognosis group (34 cases).

2.2. Definition of poor prognosis

Patients were classified into the poor prognosis group if they met at least one of the following criteria:

Primary endpoint: Persistent multiple organ dysfunction (≥3 organ systems) lasting more than 48 hours, including but not limited to respiratory, circulatory, renal, or hepatic failure.

Secondary endpoints: Development of severe complications, such as infected pancreatic necrosis; pancreatic pseudocyst or abscess; acute respiratory distress syndrome; acute kidney injury or dialysis-requiring renal failure; gastrointestinal bleeding or pancreatic-origin hemorrhage; portal or splenic vein thrombosis; pancreatic or intestinal fistula; biliary obstruction or infection; or intra-abdominal/mediastinal fluid collections requiring interventional or surgical management; and In-hospital death from any cause.

If multiple conditions occurred simultaneously, the primary endpoint was considered the principal determinant of poor prognosis, with secondary endpoints recorded for descriptive analysis.

Inclusion Criteria: Age ≥ 18 years, with no upper age limit; Diagnosis of AP met clinical criteria, including acute abdominal pain, significantly elevated serum amylase and lipase levels, and confirmation by imaging studies (e.g., CT, ultrasound); Patients assessed as having SAP based on established criteria such as Ranson score ≥ 3, APACHE II score ≥ 8, or other criteria (e.g., Balthazar CT score ≥ 6); First episode of AP, with no previous history of AP or pancreatic surgery; Complete clinical data, laboratory results, imaging data (such as Balthazar CT score), and APACHE II scores available for subsequent analysis.

Exclusion Criteria: Pregnant or lactating women; Patients with preexisting severe hepatic, renal, or cardiac conditions such as cirrhosis, acute renal failure requiring dialysis, heart failure, or acute myocardial infarction that may affect prognosis or scoring accuracy; Patients with known malignancies, including pancreatic cancer, gastric cancer, liver cancer, etc; History of pancreatic surgery or biliary surgery (e.g., pancreatic resection); Patients with severe infections or sepsis requiring long-term treatment; Immunocompromised patients, including those on long-term immunosuppressive therapy; Patients with severe psychiatric or neurological disorders that interfere with clinical assessment; Patients with missing key data that compromise the integrity of the analysis; Patients with incomplete or missing follow-up data.

2.3. Data collection

At the baseline data collection stage, detailed demographic characteristics of the patients were recorded, including age and gender. Height and weight were measured accurately to calculate body mass index (BMI). The etiology of pancreatitis (biliary, alcoholic, hyperlipidemic, etc) was obtained through thorough medical history inquiry and chart review, along with information on preexisting comorbidities – particularly diabetes and hypertension – that may influence prognosis.

Clinical indicators collected included vital signs at admission such as temperature, heart rate, respiratory rate, and blood pressure. To assess disease severity, we strictly followed standard operating procedures to complete internationally recognized scoring systems – APACHE II, Ranson, and SOFA scores – within the prescribed time window. The Balthazar CT scores were independently interpreted by 2 senior physicians to ensure accuracy.

Laboratory data were collected using standardized protocols. All blood samples were drawn within 24 hours of hospital admission. Laboratory tests included inflammatory markers (PCT, CRP, white blood cell count and differential), hematological indicators (HCT, platelet count), and a range of biochemical parameters such as liver function (total bilirubin, ALB), renal function (serum creatinine), and metabolic indicators (blood glucose). Pancreatic enzyme assays included measurements of serum amylase and lipase.

Imaging data collection primarily involved contrast-enhanced abdominal CT and ultrasound results. All CT scans were independently evaluated in a blinded manner by 2 experienced radiologists using the standardized Balthazar scoring system. Inter-rater reliability for Balthazar CT scores was excellent, with a Cohen κ value of 0.82, indicating high consistency between raters. Ultrasound examinations focused on evaluating the biliary system to aid in determining the etiology of pancreatitis.

Prognostic indicators were recorded in strict accordance with pre-defined standards. The primary outcome was determined

Table 1

Comparison of demographic and clinical characteristics.

Characteristic	Good prognosis group (n = 68)	Poor prognosis group (n = 34)	Statistic value	*P*-value
Age (yr)	45.3 ± 10.2	56.1 ± 12.8	$t = -4.02$	.001
Gender (male, n [%])	45 (66.2%)	22 (64.7%)	$\chi^2 = 0.04$	.85
BMI (kg/m²)	24.6 ± 3.1	27.2 ± 3.5	$t = -3.21$	.002
Etiology (n [%])				
Biliary	32 (47.1%)	10 (29.4%)	$\chi^2 = 3.82$	.05
Alcoholic	18 (26.5%)	12 (35.3%)	$\chi^2 = 0.97$	.32
Hyperlipidemic	15 (22.1%)	9 (26.5%)	$\chi^2 = 0.41$	.52
Other (e.g., drug-induced)	3 (4.4%)	3 (8.8%)	$\chi^2 = 0.35$	.56
Comorbidities (n [%])				
Diabetes	12 (17.6%)	14 (41.2%)	$\chi^2 = 9.69$	.002
Hypertension	14 (20.6%)	13 (38.2%)	$\chi^2 = 4.56$	.03

BMI = body mass index.

based on criteria for poor prognosis as defined in the study. Secondary outcomes included length of hospital stay, duration of ICU admission, and the need for organ support therapies. For complications, not only the type but also the timing of occurrence and management strategies were thoroughly documented.

To ensure data quality, a rigorous quality control system was established. All data were independently entered by 2 trained research staff members. Key indicators such as scoring results and laboratory data were cross-validated. Cases with more than 10% missing data were excluded in accordance with study protocol.

2.4. Sample size and power calculation

This was a retrospective study including all eligible patients within the study period; therefore, the sample size was determined by case availability rather than by an a priori calculation. However, a post hoc power analysis was performed to assess whether the sample size was adequate to detect the effect of key prognostic variables identified in the multivariate model. Based on the observed odds ratio for PCT (OR = 1.92) and its standard deviation, with a two-sided α of 0.05, the achieved statistical power ($1 - \beta$) exceeded 0.85, indicating that the present sample size (n = 102; 68 good prognosis, 34 poor prognosis) was sufficient to detect clinically meaningful differences for the main predictors. Similar calculations for APACHE II score (OR = 1.13) and Balthazar CT score (OR = 1.15) yielded power values above 0.80. These results suggest that the current sample size is adequate for the primary analysis.

2.5. Statistical analysis

Statistical analysis was conducted using SPSS version 26.0 and R version 4.1.0. Continuous variables were first tested for normality using the Shapiro–Wilk test and for homogeneity of variance. Normally distributed data were expressed as mean ± standard deviation ($\bar{x} \pm s$), and comparisons between groups were made using independent sample t-tests. Non-normally distributed data were presented as median and interquartile range [M (P25, P75)] and compared using the Mann–Whitney U test. Categorical data were expressed as counts and percentages [n (%)] and compared using the χ^2 test or Fisher exact test as appropriate.

Correlation between variables was assessed using Pearson or Spearman correlation analysis, and correlation coefficients (r values) were calculated. Multivariate logistic regression analysis with stepwise selection was used to identify independent risk factors associated with poor prognosis in SAP. For each variable, regression coefficients (β), odds ratios (OR), and 95% confidence intervals (CI) were reported. Variance inflation factors (VIF) were calculated to assess multicollinearity, with VIF < 5 considered indicative of no significant multicollinearity.

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive performance of individual indicators and the combined model. The area under the curve (AUC), sensitivity, specificity, and optimal cutoff values (based on the maximum Youden index) were calculated. Comparisons of AUCs between different ROC curves were conducted using the DeLong test. Subgroup analyses were conducted based on etiology, age, and type of complications to evaluate the predictive efficacy of the combined model in different patient subgroups.

Regarding missing data, the overall proportion of missing values among key variables was <5%. Cases with more than 10% missing data were excluded according to the study protocol. For the remaining missing values, complete case analysis (listwise deletion) was applied without imputation, as the low proportion of missingness was unlikely to bias the results.

All statistical tests were two-sided, with $P < .05$ considered statistically significant. For multiple comparisons, Bonferroni correction was applied to control type I error, with $P < .001$ considered statistically significant after correction.

3. Results

3.1. Comparison of baseline characteristics and disease severity

Analysis of baseline data from 102 patients with SAP revealed significant differences in demographic characteristics and disease severity between the good prognosis group and the poor prognosis group. Specifically, patients in the poor prognosis group were significantly older than those in the good prognosis group (56.1 ± 12.8 years vs 45.3 ± 10.2 years, $P = .001$), while the gender distribution was similar between the 2 groups (male: 64.7% vs 66.2%, $P = .85$).

Further analysis indicated that patients in the poor prognosis group had a higher mean BMI (27.2 ± 3.5 vs 24.6 ± 3.1 kg/m²) and a higher prevalence of comorbidities such as diabetes (41.2% vs 17.6%) and hypertension (38.2% vs 20.6%), with all differences reaching statistical significance ($P < .05$) (Table 1).

Of particular note, all scoring systems reflecting disease severity were significantly elevated in the poor prognosis group, including APACHE II score (14.7 ± 4.9 vs 9.3 ± 3.4), Ranson score (6.5 ± 2.0 vs 4.1 ± 1.2), Balthazar CT score, and SOFA score, with differences all showing high statistical significance ($P < .001$) (Table 2).

3.2. Comparison of laboratory indicators and imaging scores between groups

Laboratory test results revealed that patients in the poor prognosis group exhibited more pronounced inflammatory responses and organ dysfunction compared to those in the good prognosis group. Specifically, PCT levels were significantly elevated in the poor prognosis group [3.85 (2.50–5.50) ng/mL vs 0.45 (0.30–0.75) ng/mL, $P < .001$], along with markedly higher white blood cell counts (15.2 ± 3.5 vs 8.4 ± 2.6 × 10^9/L) and neutrophil percentages (81.2% ± 8.4% vs 70.5% ± 7.9%) ($P < .05$ for both).

Additionally, platelet counts were significantly lower in the poor prognosis group (125.8 ± 47.5 vs 210.5 ± 55.2 × 10^9/L), while serum creatinine (150.3 ± 62.1 vs 75.4 ± 25.6 μmol/L), total bilirubin (45.2 ± 22.5 vs 17.8 ± 8.6 μmol/L), and blood glucose levels (8.9 ± 2.6 vs 5.1 ± 1.2 mmol/L) were significantly higher (all $P < .001$).

Regarding hemoconcentration indicators, HCT levels were significantly lower in the poor prognosis group compared to the good prognosis group (36.8% ± 7.8% vs 40.2% ± 6.5%, $P < .001$), and serum ALB levels were also notably reduced (27.3 ± 5.2 vs 34.5 ± 4.1 g/L, $P < .001$) (Table 3).

3.3. Correlation analysis of prognostic indicators

Correlation analysis was conducted among various disease severity scoring systems (Table 4) to evaluate the degree of association between them. This analysis aimed to inform the selection of appropriate predictive variables for subsequent multivariate regression and to identify potential multicollinearity issues.

The results showed a high degree of correlation among the clinical scoring systems ($R = 0.87–0.89$, $P < .01$). The strongest correlation was observed between the APACHE II score and the Ranson score ($R = 0.89$), followed by the correlation between the SOFA score and the Ranson score ($R = 0.88$). Notably, the Balthazar CT score showed a strong correlation with the Ranson score ($R = 0.87$), but a relatively weaker correlation with the APACHE II score ($R = 0.41$). This suggests that imaging-based assessments may reflect different dimensions of disease severity compared to certain clinical scoring systems.

Based on these findings, the most representative scoring indicators will be prioritized as core predictive factors in the subsequent multivariate analyses. Variance inflation factor (VIF) tests will be applied to assess the degree of multicollinearity. Additionally, stratified regression models may be constructed to comprehensively evaluate the prognostic value of indicators from different assessment dimensions. This approach ensures effective control of multicollinearity while preserving the independent predictive value of each scoring system, thereby enhancing the scientific validity and reliability of the study conclusions.

3.4. Multivariate regression analysis

The results of the multivariate regression analysis (Table 5) indicated that the APACHE II score (β = 0.12, OR = 1.13, 95% CI: 1.05–1.22, $P < .001$), Balthazar CT score (β = 0.14, OR = 1.15, 95% CI: 1.05–1.26, $P = .003$), and PCT level (β = 0.65, OR = 1.92, 95% CI: 1.26–2.91, $P = .002$) were identified as independent risk factors for poor prognosis in SAP. Among these, PCT showed the strongest predictive value (OR = 1.92). Conversely, HCT (β = –0.18, OR = 0.83, 95% CI: 0.74–0.93, $P = .004$) and ALB level (β = –0.06, OR = 0.94, 95% CI: 0.88–1.01, $P = .040$) demonstrated protective effects.

It is noteworthy that after adjusting for other variables, platelet count, serum creatinine, total bilirubin, and blood glucose did not retain statistical significance ($P > .05$ for all). These findings suggest that, after controlling for multiple confounding factors, clinical scoring systems, imaging assessments, and selected laboratory indicators possess independent prognostic value for predicting outcomes in SAP.

3.5. ROC curve analysis of the predictive model

The ROC curve analysis results (Table 6) demonstrated that each individual indicator possessed good predictive value for

Table 2

Comparison of disease severity indicators.

Indicator	Good prognosis group (n = 68)	Poor prognosis group (n = 34)	Statistic value	*P*-value
APACHE II score at admission	9.3 ± 3.4	14.7 ± 4.9	$t = –11.33$	<.001*
Ranson score	4.1 ± 1.2	6.5 ± 2.0	$t = –8.54$	<.001*
Balthazar CT score	D: 8 (11.8%) E: 2 (2.9%)	D: 15 (44.1%) E: 4 (11.8%)	$\chi^2 = 25.45$	<.001*
SOFA score	4.3 ± 1.1	9.1 ± 3.3	$t = –10.81$	<.001*

* Statistically significant.

Table 3

Single variable comparison: laboratory indicators and CT scores.

Indicator (unit)	Good prognosis group (n = 68)	Poor prognosis group (n = 34)	Statistic value	*P*-value
Hematocrit (%)	40.2 ± 6.5	36.8 ± 7.8	$t = 3.10$	<.001*
PCT (ng/mL)	0.45 (0.30–0.75)	3.85 (2.50–5.50)	$U = 1083$	<.001*
ALB (g/L)	34.5 ± 4.1	27.3 ± 5.2	$t = 6.81$	<.001*
White blood cell count (×10^9/L)	8.4 ± 2.6	15.2 ± 3.5	$t = –12.56$	.005
Neutrophil percentage (%)	70.5 ± 7.9	81.2 ± 8.4	$t = –7.52$	.003
Platelet count (×10^9/L)	210.5 ± 55.2	125.8 ± 47.5	$t = 7.91$	<.001*
Creatinine (μmol/L)	75.4 ± 25.6	150.3 ± 62.1	$t = –7.64$	<.001*
Total bilirubin (μmol/L)	17.8 ± 8.6	45.2 ± 22.5	$t = –7.15$	<.001*
Glucose (mmol/L)	5.1 ± 1.2	8.9 ± 2.6	$t = –9.75$	<.001*

ALB = albumin, PCT = procalcitonin.

* Statistically significant.

Table 4

Correlation analysis of severity scoring systems.

Scoring system	SOFA score	APACHE II score	Ranson score	Balthazar CT score
SOFA score	1	0.87*	0.88*	0.86*
APACHE II score	0.87*	1	0.89*	0.84*
Ranson score	0.88*	0.89*	1	0.87*
Balthazar CT score	0.61*	0.41*	0.87*	1

*indicates $P < .01$ (highly statistically significant). Only correlations with $R > 0.5$ are shown.

Table 5

Multivariate regression analysis table.

Variable	Coefficient (β)	Standard error (SE)	OR (odds ratio)	95% Confidence interval	*P*-value
APACHE II score at admission	0.12	0.04	1.13	1.05–1.22	<.001*
Balthazar CT score	0.14	0.05	1.15	1.05–1.26	.003*
Hematocrit (%)	−0.18	0.06	0.83	0.74–0.93	.004*
PCT (ng/mL)	0.65	0.2	1.92	1.26–2.91	.002*
ALB (g/L)	−0.06	0.03	0.94	0.88–1.01	.040*
Platelet count (PLT)	−0.01	0.02	0.99	0.96–1.02	.734
Creatinine (Cr) (μmol/L)	0.01	0.01	1.01	0.99–1.03	.251
Total bilirubin (TBIL)	0.02	0.02	1.02	0.99–1.05	.311
Glucose (GLU) (mmol/L)	0.05	0.04	1.05	0.97–1.13	.268

ALB = albumin, PCT = procalcitonin.

* Statistically significant.

Table 6

ROC curve analysis results of each predictive index and the combined model.

Model	AUC (area under curve)	95% CI	Sensitivity	Specificity	Cutoff value	*P*-value
APACHE II score at admission	0.82	0.77–0.87	80.10%	74.20%	11.5	<.001*
Balthazar CT score	0.8	0.74–0.86	75.30%	71.10%	E vs D	<.001*
Hematocrit (%)	0.7	0.63–0.77	72.40%	66.50%	38.50%	<.001*
PCT (ng/mL)	0.85	0.80–0.89	85.20%	77.90%	2.2 ng/mL	<.001*
ALB (g/L)	0.74	0.68–0.80	78.50%	71.30%	28.5	<.001*
Combined model (APACHE II, Balthazar CT, hematocrit, PCT, ALB)	0.88	0.83–0.92	87.40%	84.80%	–	<.001*

ALB = albumin, AUC = area under the curve, CI = confidence interval, PCT = procalcitonin.

* Statistically significant.

the prognosis of SAP. Among them, PCT exhibited the highest predictive performance, with an AUC of 0.85 (95% CI: 0.80–0.89), followed by the APACHE II score (AUC = 0.82, 95% CI: 0.77–0.87) and the Balthazar CT score (AUC = 0.80, 95% CI: 0.74–0.86).

Among laboratory indicators, HCT (AUC = 0.70) and ALB (AUC = 0.74) also showed moderate predictive value.

Most notably, the combined predictive model consisting of the APACHE II score, Balthazar CT score, HCT, PCT, and ALB demonstrated the best overall predictive performance, with an AUC of 0.88 (95% CI: 0.83–0.92). This model achieved a sensitivity of 87.4% and a specificity of 84.8%, significantly outperforming each individual indicator ($P < .001$ for all comparisons).

3.6. Subgroup analysis of the combined predictive model

Subgroup analysis (Table 7) demonstrated that the combined model maintained stable predictive performance across different SAP patient subgroups.

By etiology, the model performed best in biliary pancreatitis (AUC = 0.86, sensitivity 86.5%, specificity 82.3%), followed by hyperlipidemic (AUC = 0.83) and alcoholic pancreatitis (AUC = 0.81), with no significant differences among groups ($P > .05$).

Age-based analysis showed higher accuracy in patients >50 years (AUC = 0.88) compared to those ≤50 years (AUC = 0.84, $P = .03$), with sensitivity and specificity reaching 89.7% and 86.3%, respectively.

For complication type, the model predicted infectious necrosis more effectively (AUC = 0.86) than multiple organ failure (AUC = 0.82, $P = .04$).

Overall, the model demonstrated consistent and reliable performance across clinical subgroups, with slight advantages in older patients and those with biliary or infectious complications.

4. Discussion

This study systematically analyzed the relationship between clinical characteristics and prognosis in patients with SAP, highlighting the unique value and synergistic effect of multiple indicators in prognostic evaluation.

Age, as a fundamental physiological factor, was significantly higher in the poor prognosis group, consistent with previous studies. This likely reflects reduced organ functional reserve, a higher prevalence of comorbidities, and decreased tolerance to systemic inflammation in elderly patients.[14,15] Notably, advanced age is often associated with microcirculatory dysfunction and endothelial impairment, which may exacerbate pancreatic ischemia and systemic inflammation, forming a vicious cycle.[16] In addition, the higher rates of diabetes and hypertension in the poor prognosis group suggest that metabolic disturbances and endothelial damage may contribute to disease

Table 7

Subgroup analysis of combined model performance for predicting prognosis in severe acute pancreatitis.

Subgroup	AUC (area under curve)	Sensitivity	Specificity	*P*-value (comparison)
Etiology				
Biliary	0.86	86.50%	82.30%	.15
Hyperlipidemic	0.83	84.20%	79.70%	.25
Alcoholic	0.81	83.10%	77.80%	.2
Age				
50 yr	0.84	88.30%	81.90%	.03
50 yr	0.88	89.70%	86.30%	.02
Complication type				
Infectious necrosis	0.86	87.50%	84.10%	.06
Multi-organ failure	0.82	83.40%	80.20%	.04

AUC = area under the curve.

progression. These comorbidities may worsen pancreatic injury by impairing microcirculatory perfusion and disrupting inflammatory regulation.

Regarding disease severity assessment, the consistently higher scores across multiple systems in the poor prognosis group, particularly the strong correlation between the APACHE II and Ranson scores, underscore the central role of multi-organ dysfunction in SAP prognosis.[13] Interestingly, although the Balthazar CT score was correlated with clinical scores, its relatively weaker association suggests that imaging provides a distinct dimension of assessment – primarily reflecting local anatomical damage rather than systemic response.[17] This complementarity supports the rationale for incorporating imaging into a combined predictive model.

Changes in laboratory indicators revealed distinct pathophysiological features in patients with poor outcomes. PCT, a key marker of bacterial infection and systemic inflammation, was significantly elevated, likely reflecting bacterial or endotoxin translocation due to compromised gut barrier function – an important mechanism in the development of infected pancreatic necrosis and multi-organ dysfunction.[9,12] Such translocation activates innate immune pathways and triggers a cytokine cascade, promoting systemic inflammatory response and organ failure.[9,12] Decreased HCT may be linked to fluid shifts into the third space and hemodilution after resuscitation. Persistently low HCT indicates severe capillary leakage and microcirculatory failure, impairing oxygen delivery and aggravating tissue hypoxia, while early excessive hemoconcentration has also been associated with worse outcomes.[11,12] Reduced ALB results from impaired hepatic synthesis, increased capillary permeability, and protein loss, leading to reduced plasma oncotic pressure, tissue edema, and compromised organ perfusion.[9,10] Hypoalbuminemia may also reflect poor nutritional status, which is linked to impaired immune defense and delayed recovery. Collectively, these markers reflect distinct yet complementary aspects of SAP pathophysiology – systemic inflammation (PCT), circulatory and oxygen transport status (HCT), and nutritional/metabolic reserve (ALB) – which explains the improved prognostic performance of the combined model.

Multivariate regression analysis further confirmed the independent predictive value of key indicators. The APACHE II score, as a comprehensive tool for assessing systemic organ function, demonstrated strong prognostic value due to its ability to dynamically reflect multi-system status. The independent predictive power of PCT underscores the crucial role of infectious complications in poor SAP outcomes. Conversely, the protective effects of HCT and ALB suggest that timely correction of hemoconcentration and hypoalbuminemia may improve microcirculatory perfusion and tissue oxygenation, potentially interrupting the progression of the disease.[18]

It is also noteworthy that traditional markers such as serum creatinine lost statistical significance after adjustment for other variables, indicating that renal dysfunction may be more reflective of systemic illness rather than an independent determinant of prognosis.

ROC curve analysis confirmed the superior predictive performance of the combined model. This advantage likely stems from its integration of complementary indicators that assess different dimensions of the disease: the APACHE II score reflects systemic organ function, the Balthazar CT score evaluates local anatomical damage, while PCT, HCT, and ALB provide additional insight into the inflammatory response, circulatory status, and nutritional/metabolic condition. This multidimensional integration enables a more comprehensive understanding of SAP's complex pathophysiology.

The model's strong performance in subgroup analyses, especially among elderly patients and those with infectious pancreatic necrosis, further supports its clinical utility. Older patients often have reduced physiological reserve and altered immune responses, while infectious necrosis involves a complex infection–inflammation–necrosis cycle. Accurate identification of these high-risk groups is of significant clinical importance.

However, this study has limitations. As a single-center retrospective study, the relatively small sample size may reduce the power of subgroup analyses. Moreover, emerging biomarkers, such as cytokine profiles, were not included. Future multi-center, prospective studies are needed to validate the model's generalizability and explore its value in conjunction with dynamic monitoring indicators. Additionally, the weighting of variables and the clinical threshold values within the model require larger datasets to refine risk stratification precision.

Despite these limitations, this study offers a clinically valuable tool by integrating clinical scoring systems, imaging assessments, and laboratory markers. The multidimensional assessment framework it proposes may also serve as a reference for building predictive models in other critical illnesses.

Author contributions

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