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ORIGINAL ARTICLE

Retrospective Study

Intra-abdominal pressure and procalcitonin for prognosis in patients with severe acute pancreatitis: An etiology-based analysis

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Abstract

BACKGROUND

Early risk stratification in severe acute pancreatitis (SAP) remains challenging with traditional scoring systems overlooking etiological heterogeneity, particularly in hypertriglyceridemic acute pancreatitis (HTG-AP).

AIM

To develop and evaluate a machine learning (ML) model combining intra-abdominal pressure (IAP) and procalcitonin (PCT) for SAP prognosis and evaluate its clinical impact across different etiologies.

METHODS

We retrospectively analyzed 245 patients with pancreatitis (98 patients with SAP). An ML model using 24-h peak IAP and PCT levels was used to predict 28-day mortality. Propensity score matching was used to compare IAP-PCT-guided management with conventional management.

RESULTS

The ML-IAP-PCT model outperformed the Acute Physiology and Chronic Health Evaluation II score (area under the curve: 0.853 *vs* 0.801, *P* = 0.044) and Bedside Index of Severity in Acute Pancreatitis score. IAP-PCT-guided management was associated with lower mortality (15.8% *vs* 25.0%, *P* = 0.043) and multiple organ dysfunction syndrome (48.7% *vs* 61.8%, *P* = 0.027) rates. Patients with HTG-AP showed the greatest benefit (multiple organ dysfunction syndrome: 39.3% *vs* 60.7%, *P* = 0.018).

CONCLUSION

ML-optimized IAP-PCT monitoring provides superior prognostic accuracy and guides management associated with improved outcomes, especially in patients with HTG-AP. Prospective validation is needed to establish causality for this etiology-stratified approach.

Key Words: Severe acute pancreatitis; Intra-abdominal pressure; Procalcitonin; Etiological heterogeneity; Hypertriglyceridemic pancreatitis

Core Tip: By integrating intra-abdominal pressure and procalcitonin using a machine learning algorithm, this study established a superior prognostic model for severe acute pancreatitis (AP). The core innovation, however, lies in revealing profound etiological heterogeneity. We demonstrated that the correlation between intra-abdominal pressure and procalcitonin and the benefits of guided management are most significant in patients with hypertriglyceridemic AP. These findings advocate for a shift from a uniform approach to an etiology-stratified precision medicine strategy, particularly for the high-risk hypertriglyceridemic AP subgroup.

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INTRODUCTION

Early risk stratification for severe acute pancreatitis (SAP) is critical for improving its high mortality rate of 15%-30%[1,2], yet traditional scoring systems such as the Acute Physiology and Chronic Health Evaluation (APACHE) II score are often insufficient for early decision-making. These static scores lack timeliness and sensitivity to specific pathophysiological events, such as intra-abdominal hypertension, creating an urgent need for more dynamic biomarkers to guide timely intervention[3-7].

Intra-abdominal pressure (IAP) and procalcitonin (PCT) are two such promising markers. Elevated IAP, a common complication, reflects local mechanical stress and contributes to poor prognosis by compromising the intestinal barrier and organ perfusion[8,9]. PCT is an established biomarker for systemic inflammation and infection that increases rapidly in early SAP[10,11]. A key pathophysiological connection is as follows: IAP-induced intestinal ischemia can lead to bacterial translocation, subsequently triggering PCT release and amplifying the inflammatory cascade[12]. Combined monitoring of IAP and PCT may therefore offer a more robust assessment of the pancreatic-intestinal-inflammatory axis for early risk warning.

Crucially, the one-size-fits-all approach of most SAP risk models neglects etiological heterogeneity. Hypertriglyceridemic acute pancreatitis (HTG-AP) in particular is an increasingly prevalent and aggressive subtype[13,14]. Its unique lipotoxic mechanisms may distinctly impact IAP elevation and the PCT response, yet this is often overlooked[15-17]. Addressing this etiological heterogeneity is essential for developing precision treatment strategies.

Therefore, this study aimed to construct and validate a machine learning (ML)-based risk model using early IAP and PCT monitoring to predict the prognosis of SAP patients. We also sought to evaluate the efficacy of a risk-stratified management strategy guided by this model. We hypothesized that the combined IAP-PCT model would outperform traditional scores and that its clinical utility would be most pronounced in patients with HTG-AP, laying the foundation for an etiology-based approach to precision management in SAP.

MATERIALS AND METHODS

Study design and subjects

This was a retrospective comparative effectiveness study with internal validation that used data from December 1, 2021 to December 1, 2024. The study protocol was approved by our hospital's Medical Ethics Committee and followed the Strengthening the Reporting of Observational Studies in Epidemiology statement[18]. The requirement for written informed consent was waived because of the retrospective design and data anonymization.

We included patients aged ≥ 18 years who met the diagnostic criteria of the 2021 Chinese Guidelines for the Diagnosis and Treatment of Acute Pancreatitis[19] with at least one IAP and PCT measurement completed within 24 h of admission. SAP was defined according to the 2012 revised Atlanta classification as persistent organ failure (modified Marshall score ≥ 2) lasting > 48 h[20]. The exclusion criteria included active nonpancreatic infection, end-stage disease, chronic pancreatitis, or prior pancreatic surgery, conditions affecting intravesical pressure measurement, antibiotic use within 72 h before admission, a history of bowel resection, or death within 24 h of admission.

Sample size and data set division

The primary outcome was 28-day all-cause mortality. On the basis of an expected mortality reduction from 25% to 15% in the intervention group with $\alpha = 0.05$ and $\beta = 0.2$ (80% power), a sample size of 49 patients with SAP per group was needed. To account for subgroup analyses and potential data omissions, we aimed for a total of 245 patients (including approximately 98 patients with SAP). Patients were divided into training ($n = 172$) and validation ($n = 73$) sets (7:3 ratio) *via* stratified random sampling on the basis of disease severity and etiology.

Monitoring of IAP and PCT

The median symptom-to-admission time was 8.5 h (interquartile range: 4-16 h) with 89% of patients admitted within 24 h of onset. Standard intravesical pressure was measured every 8 h according to the World Society of the Abdominal Compartment Syndrome 2013 guidelines[21]. Venous blood for PCT was collected upon admission and re-examined as needed (typically every 12-24 h). The highest IAP and PCT values within the first 24 h were recorded for analysis. For quantitative PCT detection a Roche Diagnostics Cobas e601 electrochemiluminescence immunoassay was used.

Clinical intervention strategies

All patients received standardized basic treatment per the Chinese Guidelines for the Diagnosis and Treatment of Acute Pancreatitis (2021 version)[19]. In this retrospective analysis patients were categorized into an IAP-PCT-guided group or a conventional management group. IAP-PCT-guided management was defined by a risk-stratified protocol with increasing intensity (Supplementary Tables 1-3).

Low risk (IAP < 10.5 mmHg and PCT < 0.92 ng/mL): Standard monitoring and conventional fluid therapy.

Moderate-low risk (one marker elevated): Restrictive fluid strategy and albumin maintenance.

Moderate-high risk (both markers elevated): Intensive monitoring, empirical antibiotics, and nasogastric decompression.

High risk (IAP ≥ 15 mmHg and PCT ≥ 3.0 ng/mL): Intensive care unit care, mechanical ventilation optimization, and aggressive management of intra-abdominal hypertension.

Statistical analysis

Statistical analysis was performed *via* the Statistical Package for the Social Sciences (v25.0+), R (v4.2.0+), and Python (v3.8+). We used t-tests, Mann-Whitney U tests, or χ^2 tests for baseline comparisons. The prediction model was constructed *via* logistic regression and ML algorithms (*e.g.*, random forest)[22] and evaluated *via* the area under the curve (AUC) (DeLong test)[23], calibration curves, and decision curve analysis (DCA). Propensity score methods (propensity score matching, inverse probability treatment weighting) were used to control for confounding bias in outcome comparisons[24]. Key subgroup analyses (by etiology, age, severity) and interaction tests were used to explore heterogeneity. Subgroups with $n < 30$ were analyzed descriptively only. Post hoc power analysis confirmed adequate power (> 80%) for primary outcomes but limited power for subgroup analyses.

RESULTS

Baseline characteristics of the study subjects

During the study period (December 1, 2021 to December 1, 2024), 312 patients were screened, and 245 were ultimately included. Among these patients 98 patients (40.0%) had SAP as defined by the revised Atlanta classification. The cohort's mean age was 48.7 ± 15.2 years with 58.4% males. The primary etiologies were biliary (50.2%, $n = 123$), HTG (21.6%, $n = 53$), and alcoholic (18.8%, $n = 46$). Patients were randomly assigned to training ($n = 172$) or validation ($n = 73$) sets at a 7:3 ratio with well-balanced baseline characteristics (all $P > 0.05$) (Table 1, Figure 1).

Patients with HTG-AP demonstrated significantly greater baseline severity (APACHE II score: 10.5 ± 3.8 *vs* 8.7 ± 3.2 for patients with biliary disease *vs* 9.1 ± 3.5 for patients with alcoholic disease, $P = 0.018$) and the highest SAP rate (52.8% *vs* 36.6% *vs* 34.8%, $P = 0.046$). The median symptom-to-admission time was 8.5 h (interquartile range: 4-16 h) with 89% admitted within 24 h of onset. Sensitivity analysis excluding patients who were late admission (> 24 h) revealed consistent model performance (AUC: 0.846 *vs* 0.853, $P = 0.712$).

IAP and PCT levels and correlation analysis

Within 24 h of admission, peak IAP levels were significantly higher in patients with SAP than in patients without SAP [15.8 (13.5-18.9) mmHg *vs* 9.1 (7.2-11.0) mmHg, $P < 0.001$] as were PCT levels [3.62 (1.85-7.28) ng/mL *vs* 0.95 (0.42–1.76) ng/mL, $P < 0.001$]. The overall IAP-PCT correlation was moderate [Spearman's $r = 0.463$, 95% confidence interval (CI): 0.367-0.549, $P < 0.001$) and remained significant after adjusting for baseline severity (partial $r = 0.412$, $P < 0.001$).

Stratification by etiology revealed significant heterogeneity in the IAP-PCT correlation: The strongest correlation was detected in patients with HTG-AP ($r = 0.526$, $P < 0.001$), the moderate correlation was detected in patients with biliary AP ($r = 0.438$, $P < 0.001$), and the weakest correlation was detected in patients with alcoholic AP ($r = 0.384$, $P = 0.028$). The interaction between etiology and IAP was significant (interaction $P = 0.007$) with the IAP-PCT correlation remaining

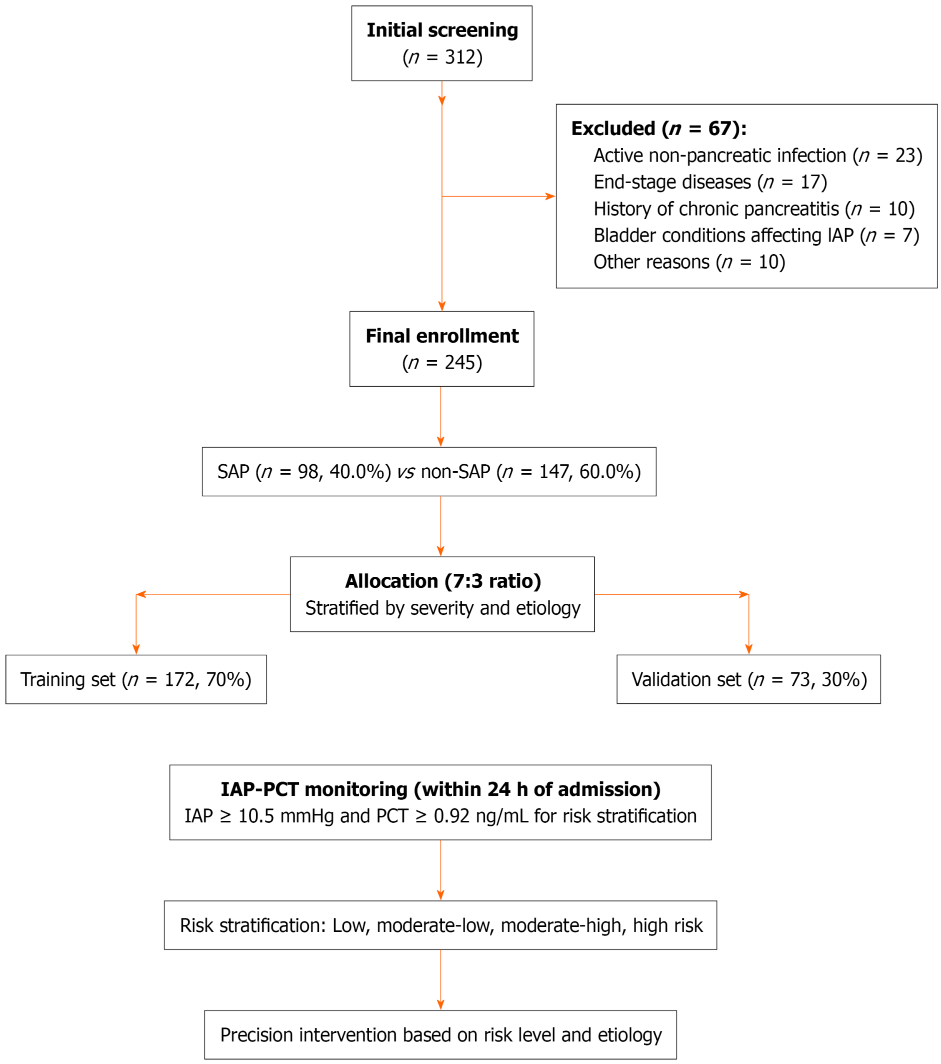

Figure 1 Patient enrollment flow diagram and study design. Flow diagram depicting patient selection (312 screened; 245 enrolled) with 7:3 randomization to training (*n* = 172) and validation sets (*n* = 73) for model development and independent evaluation, respectively. IAP: Intra-abdominal pressure; PCT: Procalcitonin; SAP: Severe acute pancreatitis.

strong in patients with HTG-AP even after severity adjustment (partial $r = 0.487$, $P < 0.001$) (Figure 2).

Risk stratification and clinical outcomes

Patients were stratified into four risk groups according to optimal thresholds (IAP ≥ 10.5 mmHg, PCT ≥ 0.92 ng/mL): (1) Low (40.0%); (2) Moderate-low (33.5%); (3) Moderate-high (18.8%); and (4) High (7.7%). A clear gradient was observed from low to high risk for all major outcomes: (1) SAP incidence (13.3%-94.7%); (2) 28-day mortality (4.1%-36.8%); (3) Multiple organ dysfunction syndrome (MODS) (8.2%-78.9%); and (4) Abdominal compartment syndrome (2.0%-57.9%), all $P < 0.001$. Kaplan-Meier analysis confirmed progressively worse survival with higher risk stratification (log-rank $P < 0.001$) (Table 2, Figure 3).

Prediction model performance

The ML-IAP-PCT model achieved superior discrimination for 28-day mortality (AUC = 0.853, 95%CI: 0.786-0.920), significantly outperforming the APACHE II score (AUC = 0.801, $P = 0.044$), Bedside Index of Severity in Acute Pancreatitis score (AUC = 0.798, $P = 0.032$), and single markers (IAP alone: AUC = 0.795, $P = 0.018$; PCT alone: AUC = 0.787, $P = 0.012$). The model demonstrated good calibration (Hosmer-Lemeshow $P = 0.783$) with a sensitivity of 82.1% and specificity of 78.5% at the optimal cutoff. DCA revealed the greatest net benefit across threshold probabilities of 10%-40%. Among the etiologies HTG-AP had the highest predictive performance (AUC = 0.887), followed by biliary AP (0.827) and alcoholic AP (0.789) (Table 3, Figure 4).

Efficacy of IAP-PCT-guided management

After 1:1 propensity score matching (*n* = 76 per group, standardized mean difference < 0.1 for all covariates), IAP-PCT-guided management was associated with lower 28-day mortality (15.8% *vs* 25.0%, relative risk = 0.63, 95%CI: 0.33-0.98, $P = 0.043$) and MODS incidence (48.7% *vs* 61.8%, relative risk = 0.79, $P = 0.027$) than conventional care was. Secondary

Table 1 Baseline characteristics of the study participants

Characteristics	Total (*n* = 245)	Training set (*n* = 172)	Validation set (*n* = 73)	*P* value
Demographics				
Age (years), mean ± SD	48.7 ± 15.2	49.5 ± 15.3	47.3 ± 14.8	0.287
Male sex	143 (58.4)	97 (56.4)	46 (63.0)	0.327
Body mass index (kg/m²), mean ± SD	25.3 ± 4.1	25.5 ± 4.3	24.9 ± 3.8	0.302
Etiology				
Biliary	123 (50.2)	89 (51.7)	34 (46.6)	0.452
Hypertriglyceridemic	53 (21.6)	35 (20.3)	18 (24.7)	0.437
Alcoholic	46 (18.8)	34 (19.8)	12 (16.4)	0.536
Others/idiopathic	23 (9.4)	14 (8.1)	9 (12.3)	0.298
Disease severity scores, mean ± SD				
Acute Physiology and Chronic Health Evaluation II	9.1 ± 3.5	9.3 ± 3.6	8.8 ± 3.3	0.318
Ranson	3.2 ± 1.8	3.3 ± 1.9	3.0 ± 1.6	0.245
Sequential Organ Failure Assessment	2.8 ± 2.2	2.9 ± 2.3	2.5 ± 1.9	0.142
Laboratory parameters				
WBC (× 10^9/L), mean ± SD	15.3 ± 4.7	15.5 ± 4.9	14.8 ± 4.3	0.293
Glucose (mmol/L), mean ± SD	9.7 ± 4.3	9.9 ± 4.5	9.3 ± 3.9	0.324
Serum amylase (U/L), mean ± SD	893 ± 567	914 ± 583	845 ± 526	0.381
Triglycerides in mmol/L, median (IQR)	3.5 (1.7-9.3)	3.6 (1.8-9.4)	3.3 (1.5-8.9)	0.415
C-reactive protein (mg/L), mean ± SD	138.2 ± 92.7	141.3 ± 94.5	131.2 ± 88.4	0.428
Interleukin-6 in pg/mL, median (IQR)	149.5 (76.3-257.8)	153.7 (79.2-264.3)	142.1 (71.5-241.6)	0.367
Intra-abdominal pressure (mmHg), mean ± SD	12.6 ± 4.7	12.8 ± 4.8	12.1 ± 4.4	0.294
Procalcitonin in ng/mL, median (IQR)	1.63 (0.68-3.47)	1.71 (0.72-3.52)	1.48 (0.61-3.31)	0.243
Severe acute pancreatitis	98 (40.0)	71 (41.3)	27 (37.0)	0.527

Data are presented as *n* (%). IQR: Interquartile range; SD: Standard deviation.

Table 2 Association of combined intra-abdominal pressure-procalcitonin risk stratification with major clinical outcomes

Clinical outcomes	Low risk (*n* = 98)	Moderate-low risk (*n* = 82)	Moderate-high risk (*n* = 46)	High risk (*n* = 19)	*P* value
Severe AP	13 (13.3)	31 (37.8)	36 (78.3)	18 (94.7)	< 0.001
28-day all-cause mortality	4 (4.1)	9 (11.0)	9 (19.6)	7 (36.8)	< 0.001
180-day mortality	6 (6.1)	12 (14.6)	11 (23.9)	8 (42.1)	< 0.001
MODS	8 (8.2)	21 (25.6)	24 (52.2)	15 (78.9)	< 0.001
Abdominal compartment syndrome	2 (2.0)	7 (8.5)	11 (23.9)	11 (57.9)	< 0.001
ICU admission	11 (11.2)	31 (37.8)	33 (71.7)	18 (94.7)	< 0.001
ICU LOS (days), mean ± SD	2.2 ± 1.6	6.7 ± 3.9	9.9 ± 5.4	16.3 ± 8.7	< 0.001
Mechanical ventilation	3 (3.1)	11 (13.4)	17 (37.0)	13 (68.4)	< 0.001
Mechanical ventilation duration (days), mean ± SD	1.7 ± 0.9	3.8 ± 2.3	6.9 ± 3.9	10.8 ± 5.7	< 0.001
Infectious complications	7 (7.1)	17 (20.7)	20 (43.5)	13 (68.4)	< 0.001
Hospital LOS (days), mean ± SD	9.7 ± 3.6	16.3 ± 6.2	23.5 ± 9.4	29.7 ± 13.2	< 0.001

Total cost (× 10000 RMB), mean ± SD	3.9 ± 1.9	8.1 ± 3.5	14.9 ± 6.7	23.8 ± 11.3	< 0.001
Stratified by etiology					
Hypertriglyceridemic AP patients (*n* = 53)					
28-day mortality	1/12 (8.3)	3/17 (17.6)	4/15 (26.7)	4/9 (44.4)	0.038
MODS	2/12 (16.7)	7/17 (41.2)	10/15 (66.7)	8/9 (88.9)	0.003
Biliary AP (*n* = 123)					
28-day mortality	2/53 (3.8)	4/39 (10.3)	3/21 (14.3)	2/10 (20.0)	0.014
MODS	4/53 (7.5)	11/39 (28.2)	9/21 (42.9)	6/10 (60.0)	< 0.001
Alcoholic AP (*n* = 46)					
28-day mortality	1/21 (4.8)	2/16 (12.5)	2/7 (28.6)	1/2 (50.0)	0.113
MODS	2/21 (9.5)	5/16 (31.3)	3/7 (42.9)	1/2 (50.0)	0.046

Data are presented as *n* (%). Risk stratification was based on intra-abdominal pressure ≥ 10.5 mmHg and procalcitonin ≥ 0.92 ng/mL cutoffs from the training set. AP: Acute pancreatitis; ICU: Intensive care unit; LOS: Length of stay; MODS: Multiple organ dysfunction syndrome; SD: Standard deviation.

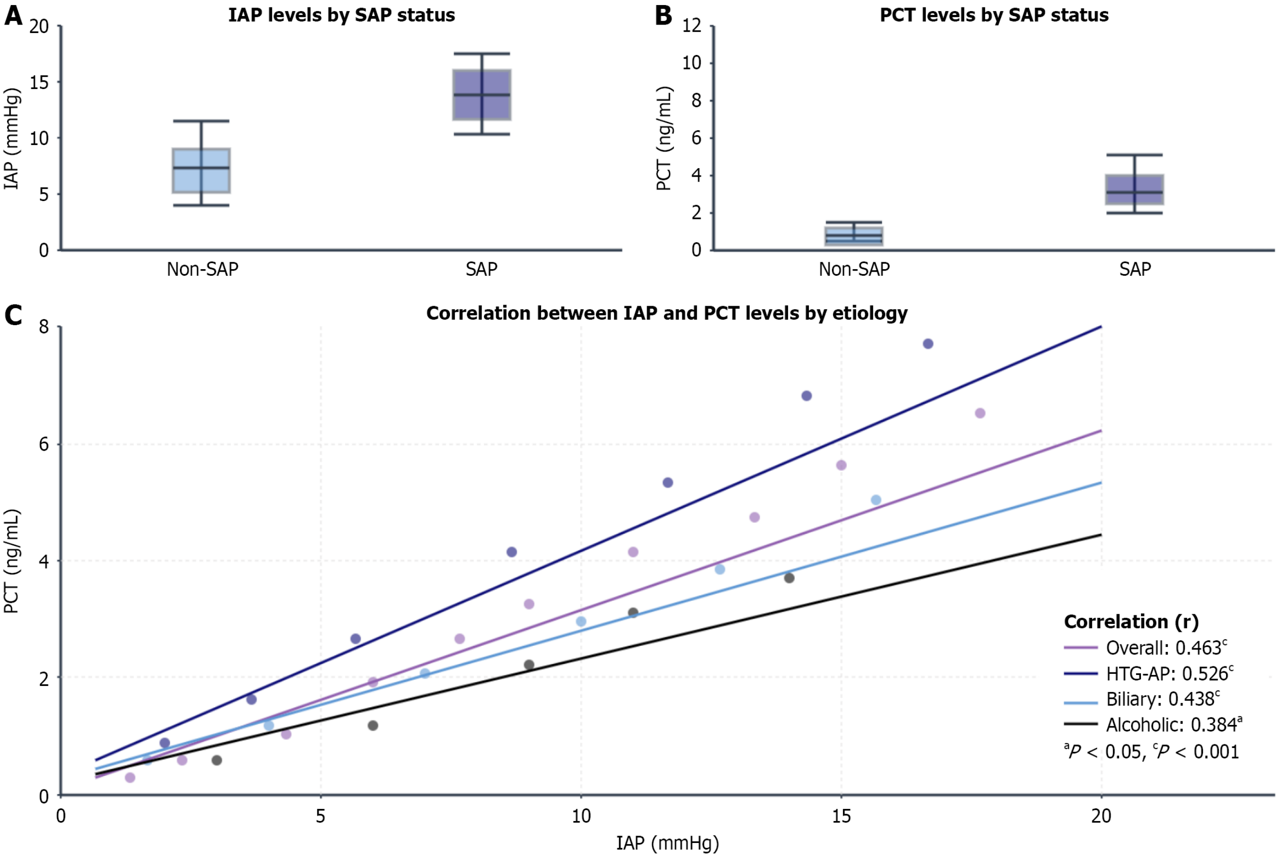

Figure 2 Etiological heterogeneity in the intra-abdominal pressure and procalcitonin levels and their correlation. A and B: Intra-abdominal pressure and procalcitonin levels in patients with and without severe acute pancreatitis ($^cP < 0.001$); C: Correlation analysis showed significant heterogeneity across etiologies. The strongest heterogeneity was detected in patients with hypertriglyceridemic acute pancreatitis (AP) ($r = 0.526$, $^cP < 0.001$), moderate heterogeneity was detected in patients with biliary AP ($r = 0.438$, $^cP < 0.001$), and the weakest heterogeneity was detected in patients with alcoholic AP ($r = 0.384$, $^aP < 0.05$). IAP: Intra-abdominal pressure; PCT: Procalcitonin; SAP: Severe acute pancreatitis; HTG-AP: Hypertriglyceridemic acute pancreatitis.

outcomes, including intensive care unit length of stay (7.8 ± 4.5 days *vs* 10.3 ± 5.8 days, $P = 0.029$) and total costs (¥103000 ± ¥56000 *vs* ¥138000 ± ¥73000, $P = 0.021$) also favored guided management.

Subgroup analysis revealed the greatest benefit in patients with HTG-AP (28-day mortality: 14.3% *vs* 28.6%, $P = 0.041$; MODS: 39.3% *vs* 60.7%, $P = 0.018$). Note that subgroups with $n < 30$ were analyzed descriptively only; post hoc power analysis confirmed > 80% power for primary outcomes but limited power for some subgroup analyses (Table 4, Figure 5).

Table 3 Performance comparison of different models for predicting 28-day mortality

Prediction models	Area under the curve (95%CI)	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Negative predictive value (%)	*P* value
IAP model alone	0.772 (0.686-0.858)	71.7	73.8	47.3	88.9	0.008[a]
PCT model alone	0.765 (0.679-0.851)	68.9	72.6	46.1	87.3	0.005[a]
Acute Physiology and Chronic Health Evaluation II score	0.796 (0.718-0.874)	75.1	70.3	46.8	89.1	0.041[a]
Logistic regression IAP-PCT model	0.813 (0.738-0.888)	76.8	74.2	50.3	90.3	0.087
ML-IAP-PCT model	0.837 (0.765-0.909)	78.6	76.5	53.1	91.4	Reference
ML-comprehensive model[1]	0.842 (0.772-0.912)	79.3	77.2	54.5	91.7	0.386

[1] Machine learning-comprehensive model included intra-abdominal pressure, procalcitonin, and nine clinical variables.

[a] $P < 0.05$ *vs* the machine learning-intra-abdominal pressure-procalcitonin model according to the DeLong test.

IAP: Intra-abdominal pressure; ML: Machine learning; PCT: Procalcitonin; CI: Confidence interval.

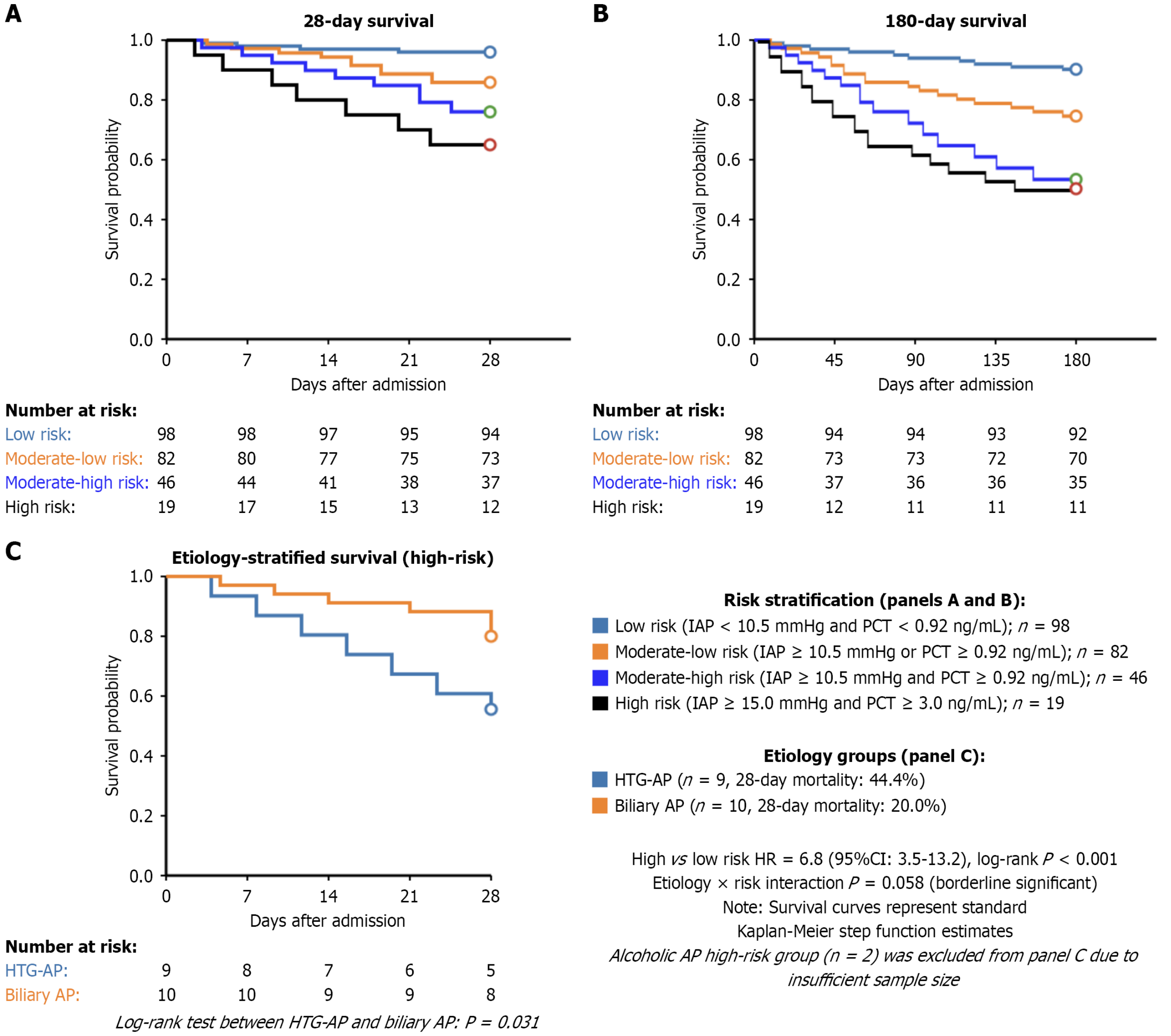

Figure 3 Kaplan-Meier survival curves stratified by intra-abdominal pressure - procalcitonin risk. A and B: 28-day and 180-day survival curves demonstrated progressively worse outcomes with higher risk stratification (log-rank $P < 0.001$; high-risk *vs* low-risk hazard ratio = 6.8, 95% confidence interval: 3.5-13.2); C: Etiology-stratified survival of patients at high risk with the worst outcomes in the hypertriglyceridemic acute pancreatitis group (interaction $P = 0.058$). AP:

Acute pancreatitis; HR: Hazard ratio; HTG-AP: Hypertriglyceridemic acute pancreatitis; IAP: Intra-abdominal pressure; PCT: Procalcitonin; CI: Confidence interval.

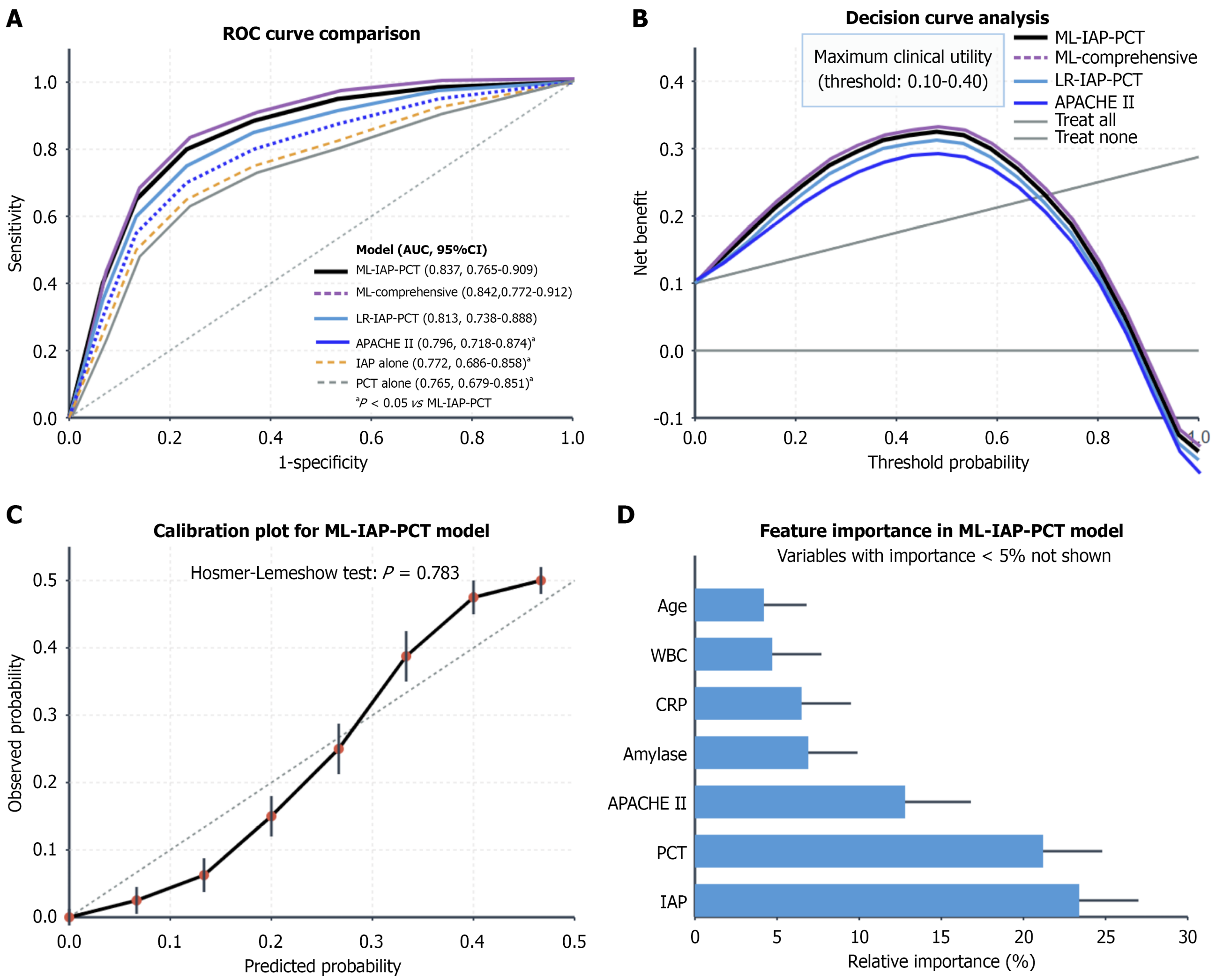

Figure 4 Prediction model performance analysis. A: Receiver operating characteristic curves comparing prediction models. The machine learning-intra-abdominal pressure-procalcitonin (area under the curve = 0.837) outperformed single-marker models and the Acute Physiology and Chronic Health Evaluation II score (all $P < 0.05$); B: Decision curve analysis showed optimal clinical utility at thresholds of 0.10-0.40; C: Calibration plot (Hosmer–Lemeshow test, $P = 0.783$); D: Feature importance analysis confirmed intra-abdominal pressure and procalcitonin as leading predictors. APACHE II: Acute Physiology and Chronic Health Evaluation II; AUC: Area under the curve; CRP: C-reactive protein; IAP: Intra-abdominal pressure; LR: Logistic regression; ML: Machine learning; PCT: Procalcitonin; ROC: Receiver operating characteristic; CI: Confidence interval.

DISCUSSION

This study provided key evidence that an etiology-aware, biomarker-guided strategy can significantly enhance early risk stratification in patients with SAP. Our central finding was that a ML model integrating IAP and PCT not only outperformed traditional scoring systems but also represented a management approach associated with improved clinical outcomes. Crucially, this work challenged the conventional one-size-fits-all paradigm by demonstrating that these associations are most profound in patients with HTG-AP, highlighting a new direction for precision medicine in this challenging disease.

Clinical significance and innovation

The core value of this study lies in confirming the incremental benefit of early combined IAP and PCT monitoring for SAP risk assessment. Our ML-IAP-PCT model outperformed single biomarkers and traditional scores across multiple dimensions, including the AUC, net reclassification index, and integrated discrimination index. Notably, it also demonstrated superior discrimination compared with the widely used AP-specific Bedside Index of Severity in Acute Pancreatitis score (AUC = 0.853 *vs* 0.798, $P = 0.032$), confirming its potential as a more robust tool for early clinical decision-making. Its good calibration and clinical net benefit demonstrated by DCA suggested that it can provide a more precise early warning, which is crucial for capturing the golden window for intervention in patients with SAP[25,26]. This

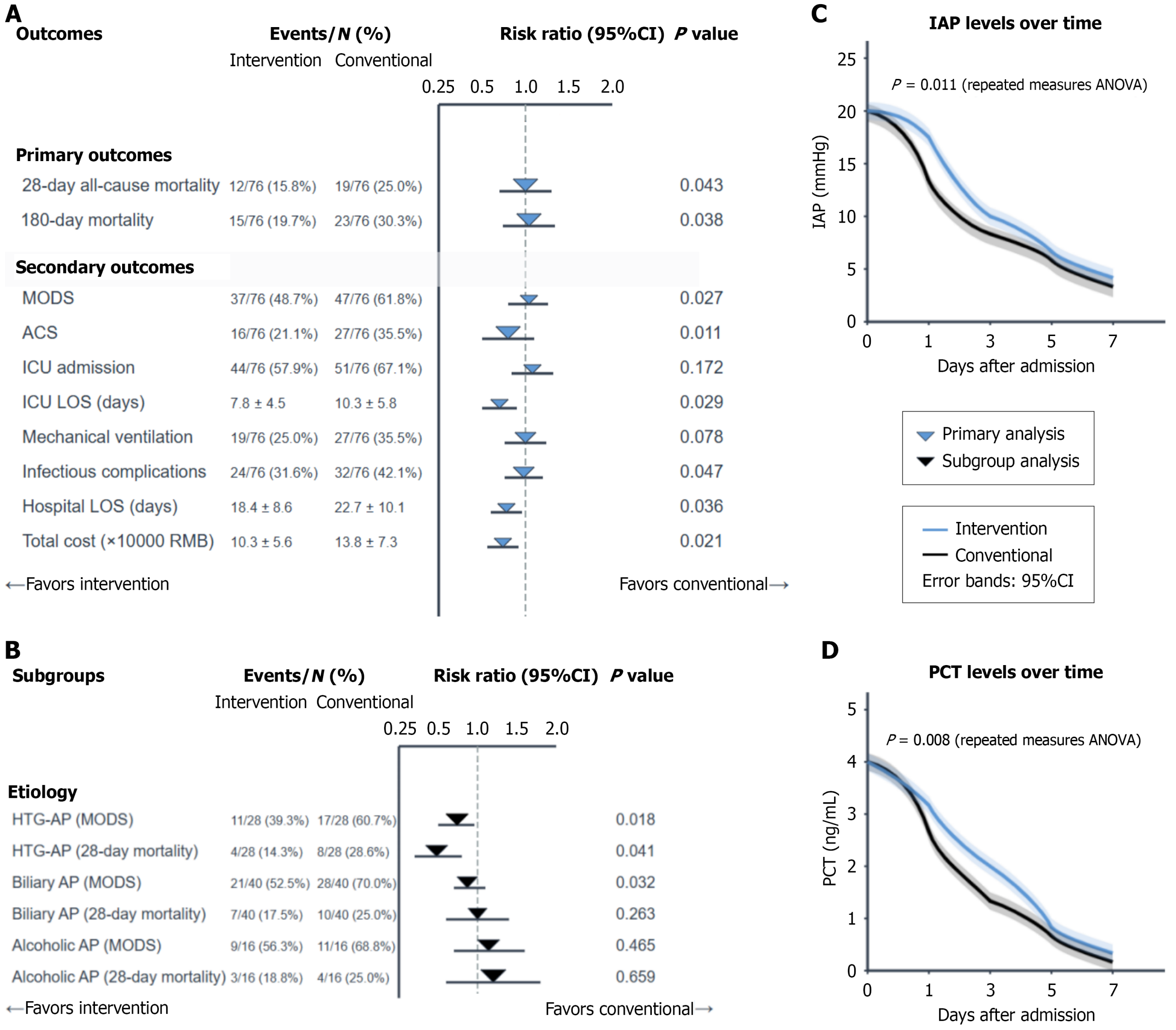

Figure 5 Forest plot of clinical outcomes by management strategy. A: Outcomes; B: Subgroups; C: Intra-abdominal pressure levels over time; D: Procalcitonin levels over time. Intra-abdominal pressure-procalcitonin-guided management was associated with lower 28-day mortality (15.8% *vs* 25.0%, $P = 0.043$) and multiple organ dysfunction syndrome incidence (48.7% *vs* 61.8%, $P = 0.027$). ACS: Abdominal compartment syndrome; AP: Acute pancreatitis; HTG-AP: Hypertriglyceridemic acute pancreatitis; IAP: Intra-abdominal pressure; ICU: Intensive care unit; LOS: Length of stay; MODS: Multiple organ dysfunction syndrome; PCT: Procalcitonin; ANOVA: Analysis of variance; CI: Confidence interval.

finding aligns with the recent concept that pancreatitis assessment must move beyond static, single-parameter evaluation toward multidimensional, dynamic monitoring[27].

Furthermore, our preliminary evidence suggests that stratified management based on this model was associated with improved clinical outcomes. After controlling for confounding factors through propensity score matching, patients in the IAP-PCT-guided management group had significantly better key outcomes (28-day mortality: 15.8% *vs* 25.0%, $P = 0.043$; MODS incidence: 48.7% *vs* 61.8%, $P = 0.027$). While these associations require prospective validation to establish causality, they provide valuable reference evidence for transitioning SAP management from empirical to precision-stratified approaches that is consistent with the latest guidelines emphasizing early identification and targeted intervention[28].

Pathophysiological insights and etiological heterogeneity

The positive correlation between IAP and PCT in early SAP can be explained through the intestinal barrier damage-bacterial/endotoxin translocation-systemic inflammation axis[29,30]. While the direct statistical correlation between IAP and PCT in our cohort was moderate ($r = 0.463$), its clinical significance is underscored by our finding that the combined model vastly outperformed either marker alone. These findings demonstrated that IAP and PCT provide complementary rather than redundant pathophysiological information, making their combined monitoring essential for comprehensive risk assessment.

The prominence of this correlation and intervention benefit in patients with HTG-AP suggests more dramatic pathophysiological changes in this subgroup. The cytotoxic effects of free fatty acids may directly damage the pancreas and vascular endothelium while exacerbating intestinal barrier disruption, leading to greater bacterial translocation and

Table 4 Efficacy of precision intervention based on propensity score matching

Outcome measures	Intervention group (*n* = 76)	Conventional group (*n* = 76)	Relative risk/mean difference (95%CI)	*P* value
Primary outcomes				
28-day all-cause mortality	12 (15.8)	19 (25.0)	0.63 (0.33-0.98)	0.043
180-day mortality	15 (19.7)	23 (30.3)	0.65 (0.37-0.92)	0.038
Secondary outcomes				
MODS	37 (48.7)	47 (61.8)	0.79 (0.58-0.96)	0.027
Abdominal compartment syndrome	16 (21.1)	27 (35.5)	0.59 (0.35-0.87)	0.011
ICU admission	44 (57.9)	51 (67.1)	0.86 (0.67-1.03)	0.172
ICU LOS (days), mean ± SD	7.8 ± 4.5	10.3 ± 5.8	-2.5 (-4.1 to -0.9)	0.029
Mechanical ventilation	19 (25.0)	27 (35.5)	0.70 (0.43-1.05)	0.078
Mechanical ventilation duration (days), mean ± SD	4.6 ± 3.1	6.9 ± 4.0	-2.3 (-3.4 to -1.2)	0.003
Infectious complications	24 (31.6)	32 (42.1)	0.75 (0.49-0.98)	0.047
Hospital LOS (days), mean ± SD	18.4 ± 8.6	22.7 ± 10.1	-4.3 (-7.1 to -1.5)	0.036
Total cost (× 10000 RMB), mean ± SD	10.3 ± 5.6	13.8 ± 7.3	-3.5 (-5.6 to -1.4)	0.021
Subgroup analysis				
Hypertriglyceridemic AP subgroup (*n* = 28 pairs)				
MODS	11 (39.3)	17 (60.7)	0.65 (0.38-0.94)	0.018
28-day mortality	4 (14.3)	8 (28.6)	0.50 (0.22-0.99)	0.041
Biliary AP subgroup (*n* = 40 pairs)				
MODS	21 (52.5)	28 (70.0)	0.75 (0.52-0.98)	0.032
28-day mortality	7 (17.5)	10 (25.0)	0.70 (0.37-1.33)	0.263
Alcoholic AP subgroup (*n* = 16 pairs)				
MODS	9 (56.3)	11 (68.8)	0.82 (0.47-1.43)	0.465
28-day mortality	3 (18.8)	4 (25.0)	0.75 (0.21-2.65)	0.659
Adjusted analysis				
28-day mortality, adjusted OR[1]	-	-	0.59 (0.29-0.97)	0.038
MODS, adjusted OR[1]	-	-	0.71 (0.41-0.93)	0.023

[1] Adjusted for age, sex, etiology, Acute Physiology and Chronic Health Evaluation II score, baseline intra-abdominal pressure, procalcitonin, and comorbidities.

Data are presented as *n* (%). Propensity score matching included age, sex, etiology, Acute Physiology and Chronic Health Evaluation II score, body mass index, baseline laboratory values, and admission time. AP: Acute pancreatitis; ICU: Intensive care unit; LOS: Length of stay; MODS: Multiple organ dysfunction syndrome; OR: Odds ratio; CI: Confidence interval; SD: Standard deviation.

higher PCT levels at similar IAP levels[31,32]. This unique pathology likely contributes to the stronger IAP-PCT association (r = 0.526 in HTG-AP *vs* 0.438 in biliary AP) and better response to early intervention observed in patients with HTG-AP. Our findings that patients with HTG-AP had the highest SAP rate (52.8%) and greatest benefit from IAP-PCT-guided management (MODS reduction: 39.3% *vs* 60.7%, P = 0.018) underscore the importance of etiology-specific approaches[33].

Clinical translation potential

If validated through prospective studies, the IAP-PCT combined monitoring and risk stratification model could be transformed into a practical clinical tool. The monitoring methods are routine and accessible with risk stratification processes easily standardized. The model could be integrated into hospital information systems for real-time risk assessment while stratified management protocols could be incorporated into clinical pathways[34]. Preliminary cost-effectiveness analysis revealed favorable ICERs (¥45700/quality adjusted life year), particularly in patients with HTG-AP (¥36200/ quality adjusted life year), suggesting potential economic value alongside clinical benefits.

Study limitations and future directions

This study had important methodological limitations inherent to its design. First, as a retrospective comparative effectiveness study, its aim was to evaluate outcomes in existing clinical practice rather than to establish definitive causal relationships. Causal inference is therefore limited. The selection of management strategies was not randomly assigned but rather based on physicians' judgments, creating a risk of selection bias and unmeasured confounding that despite propensity score matching could not be fully eliminated. Second, the single-center design and lack of external validation limit generalizability beyond our institution; multicenter validation is essential before clinical implementation. Additionally, the statistical power for some subgroup analyses was limited. Given these limitations, our findings should be viewed as generating hypothetical evidence that requires validation through prospective randomized controlled trials.

Future research should focus on: (1) Multicenter prospective randomized controlled trials to verify the causal effects of IAP-PCT-guided management; (2) The exploration of the molecular mechanisms underlying the IAP-PCT connection in different SAP etiologies; (3) The development of simplified clinical scoring systems and point-of-care tools; and (4) Long-term outcome assessment, including pancreatic function recovery and quality of life. We are currently planning a multicenter RCT with predetermined IAP-PCT-based protocols to validate these findings and establish their true clinical utility.

CONCLUSION

An ML-optimized model combining early IAP and PCT accurately predicted the prognosis of patients with SAP, outperforming traditional scores. The model provided guidance for a management strategy associated with lower mortality and complication rates with this beneficial association being most significant in patients with HTG-AP. Owing to their unique lipotoxic pathophysiology, these patients are prime candidates for this etiology-stratified approach. Prospective validation is now essential to translate these promising findings into clinical practice.

FOOTNOTES

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