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ORIGINAL RESEARCH

Evaluation of Serum Procalcitonin in Predicting Pancreatitis Severity

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ABSTRACT

Background: Acute pancreatitis (AP) demonstrates variable clinical severity, and early prediction of severe disease is crucial for optimal management. Procalcitonin (PCT), a biomarker of systemic inflammation and bacterial infection, has shown promise in predicting pancreatitis severity, yet its diagnostic accuracy remains debated. **Objective:** This study aimed to evaluate the utility of serum procalcitonin in predicting severity of acute pancreatitis and compare its performance with conventional scoring systems. **Methods:** A prospective observational study included 180 patients with acute pancreatitis admitted within 48 hours of symptom onset. Patients were classified as mild (n=102), moderately severe (n=48), or severe (n=30) according to Revised Atlanta Classification. Serum procalcitonin levels were measured at admission, 24 hours, and 48 hours. APACHE II and Ranson scores were calculated. Clinical outcomes including organ failure, pancreatic necrosis, length of stay, and mortality were recorded. **Results:** Mean admission PCT levels were significantly higher in severe AP compared to moderately severe and mild AP (4.86 ± 2.34 ng/mL vs. 1.42 ± 0.86 ng/mL vs. 0.34 ± 0.28 ng/mL, $p<0.001$). PCT at 48 hours showed strongest correlation with severity ($r = 0.742$, $p<0.001$). At a cutoff of 1.8 ng/mL, PCT demonstrated 86.7% sensitivity and 84.3% specificity for predicting severe AP (AUC = 0.912, 95% CI: 0.862-0.962). PCT at 48 hours outperformed APACHE II score (AUC = 0.856) and Ranson score (AUC = 0.824) in predicting severe disease. Multivariate analysis revealed PCT >1.8 ng/mL as an independent predictor of severe AP (OR = 12.64, 95% CI: 5.38-29.68, $p<0.001$). **Conclusion:** Serum procalcitonin is a reliable early biomarker for predicting acute pancreatitis severity, with superior discriminative ability compared to conventional scoring systems.

Keywords: Procalcitonin; acute pancreatitis; severity prediction; biomarker; APACHE II; Ranson score; organ failure; pancreatic necrosis

INTRODUCTION

Acute pancreatitis (AP) is a common inflammatory disorder of the pancreas with an incidence ranging from 13 to 45 per 100,000 population annually, representing a substantial healthcare burden **[1]**. While approximately 80% of patients experience a mild, self-limiting course, 15-20% develop severe disease characterized by persistent organ failure, local complications such as pancreatic necrosis and infected necrosis, and mortality rates reaching 20-30% **[2, 3]**. The unpredictable clinical trajectory and potential for rapid deterioration necessitate early identification of patients at risk for severe disease to enable timely intensive care intervention and optimize outcomes.

Numerous clinical scoring systems have been established to stratify the severity of acute pancreatitis, including the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, Ranson criteria, Bedside Index of Severity in Acute Pancreatitis (BISAP), and the modified Glasgow score **[4,5]**. Despite their extensive use, these systems exhibit drawbacks such as complexity, the necessity for many parameters gathered over 24-48 hours, and moderate discriminative capability, with area under the receiver operating characteristic curve (AUC) values generally between 0.70 and 0.85 **[6]**. The Revised Atlanta Classification, updated in 2012, classifies acute pancreatitis into mild, moderately

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severe, and severe categories based on the presence of organ failure and local or systemic consequences **[7]**.

Due to the constraints of clinical scoring systems, extensive research has concentrated on discovering biomarkers for early severity prediction. C-reactive protein (CRP), the most thoroughly examined biomarker, exhibits strong predictive value but generally peaks 48-72 hours post-symptom start, constraining its effectiveness for early decision-making**[8]**. Additional examined biomarkers comprise interleukin-6, interleukin-8, tumor necrosis factor-alpha, and procalcitonin, among others **[9]**.

Procalcitonin (PCT), a prohormone of calcitonin consisting of 116 amino acids, is synthesized by several cell types in reaction to bacterial endotoxin and pro-inflammatory cytokines.**[10]**. Under physiological conditions, PCT is synthesized exclusively by thyroid C-cells and rapidly cleaved to calcitonin, maintaining serum levels below 0.1 ng/mL. During bacterial infections and systemic inflammatory responses, extrathyroidal PCT production increases dramatically without subsequent cleavage, resulting in elevated serum concentrations **[11]**. PCT has become established as a biomarker for bacterial infection and sepsis, with superior specificity compared to CRP or white blood cell count **[12]**.

In the context of acute pancreatitis, several mechanisms may explain PCT elevation. The inflammatory cascade triggered by pancreatic injury, translocation of intestinal bacteria secondary to gut barrier dysfunction, and development of infected pancreatic necrosis all contribute to PCT production **[13]**. Multiple studies have investigated PCT as a predictor of AP severity with variable results. A meta-analysis conducted by Mofidi et al. indicated that PCT had reasonable accuracy in predicting severe AP, with pooled sensitivity and specificity of 74% and 87%, respectively **[14]**. Recent investigations by Khanna et al. and Chakraborty et al. indicated that PCT assessed within 48 hours had enhanced predictive efficacy relative to conventional scoring methods **[15, 16]**.

However, significant heterogeneity exists across studies regarding optimal cutoff values, timing of measurement, and comparative performance against established severity prediction tools. Furthermore, most previous studies were conducted in Western populations, with limited data from diverse ethnic groups. The dynamic nature of PCT levels during the early course of pancreatitis and the optimal time point for measurement require further elucidation. Additionally, whether PCT provides independent predictive value beyond conventional clinical and biochemical parameters remains incompletely defined.

The objective of this prospective study was to: (1) assess serum procalcitonin levels at various time intervals during the initial phase of acute pancreatitis; (2) evaluate the efficacy of procalcitonin in forecasting the severity of acute pancreatitis based on the Revised Atlanta Classification; (3) compare the predictive capabilities of procalcitonin with the APACHE II and Ranson scoring systems; and (4) identify the optimal cutoff value and timing for procalcitonin measurement for severity prediction.

MATERIALS AND METHODS

The prospective study was conducted in Department of General Surgery and Medicine at Dr Moopens Medical College, Wayanad, Kerala. Consecutive patients admitted with acute pancreatitis within 48 hours of symptom onset were screened for eligibility. Sample size was calculated based on previous studies reporting AUC of 0.85 for PCT in predicting severe AP, with 20% prevalence of severe disease. Using a two-sided alpha of 0.05 and power of 90%, with an expected difference in AUC of 0.15 compared to clinical scoring (null hypothesis AUC = 0.70), a minimum of 165 patients was required. The final sample comprised 180 patients to account for potential dropouts.

Inclusion and Exclusion Criteria

Inclusion criteria: (1) Age ≥18 years; (2) diagnosis of acute pancreatitis according to revised Atlanta criteria necessitating at least two of the following: characteristic abdominal pain, serum amylase and/or lipase levels ≥3 times the upper limit of normal, and distinctive imaging findings; (3) presentation within 48 hours of symptom onset; and (4) capacity to provide informed consent.

Exclusion criteria: (1) Chronic pancreatitis; (2) pancreatic malignancy; (3) previous pancreatic surgery; (4) concurrent bacterial infection (pneumonia, urinary tract infection, etc.); (5) sepsis from non-pancreatic source; (6) chronic kidney disease (estimated glomerular filtration rate <30 mL/min/1.73m²); (7) immunosuppressive therapy or immunodeficiency states; (8) pregnancy; (9) recent trauma or surgery within 2 weeks; and (10) medullary thyroid carcinoma or other conditions affecting PCT metabolism.

Clinical Assessment and Data Collection

Comprehensive clinical data were collected using standardized case report forms, including demographics, comorbidities, etiology of pancreatitis (biliary, alcoholic, hypertriglyceridemia, idiopathic, other), time from symptom onset to admission, and vital signs. APACHE II scores were calculated within 24 hours of admission using the worst values during the first 24 hours. Ranson criteria were assessed at admission (0 hours) and 48 hours.

Severity classification was conducted in accordance with the Revised Atlanta Classification.

- **Mild acute pancreatitis**: Absence of organ failure and local or systemic consequences
- **Moderately severe acute pancreatitis**: Transient organ failure (lasting less than 48 hours) and/or

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local or systemic consequences without enduring organ failure.

- **Severe Acute Pancreatitis:** Prolonged organ failure (>48 hours) with involvement of one or more organ systems.

Organ failure was characterized utilizing the modified Marshall scoring system for the respiratory, cardiovascular, and renal systems. Local problems such as acute peripancreatic fluid collections, pancreatic necrosis, pseudocysts, and walled-off necrosis were detected with contrast-enhanced computed tomography (CECT) conducted at 72-96 hours or subsequently as clinically warranted.

Laboratory Investigations

Venous blood samples were obtained at admission (within 6 hours), as well as at 24 hours and 48 hours, for PCT measurement and standard examinations. Samples for PCT were obtained in serum separator tubes, centrifuged at 3000 rpm for 10 minutes, with the serum thereafter separated and preserved at -80°C if not analyzed promptly.

Procalcitonin serum levels were quantified by electrochemiluminescence immunoassay (ECLIA) on a Cobas e601 analyzer (Roche Diagnostics). The test has a functional sensitivity of 0.06 ng/mL and a measurement range of 0.02-100 ng/mL, with inter-assay and intra-assay coefficients of variation under 6%. Results were classified as follows: <0.5 ng/mL (normal/low risk), 0.5-2.0 ng/mL (potential bacterial infection/moderate risk), 2.0-10.0 ng/mL (likely bacterial infection/high risk), and >10.0 ng/mL (critical bacterial infection/very high risk).

Supplementary laboratory assessments comprised complete blood count, serum amylase, lipase, liver function tests, renal function tests, serum calcium, lactate dehydrogenase (LDH), C-reactive protein, arterial blood gas analysis, and lipid profile. CRP was quantified with immunoturbidimetry within a range of 0.1-20 mg/dL.

Imaging Studies

Abdominal ultrasonography was performed at admission to assess gallstones and biliary dilatation. Contrast-enhanced computed tomography was performed at 72-96 hours or later to evaluate pancreatic necrosis, peripancreatic collections, and other complications. Modified CT Severity Index (CTSI) was calculated based on degree of pancreatic inflammation and percentage of necrosis.

Outcome Measures

The primary outcome was the prediction of severe acute pancreatitis. Secondary outcomes encompassed: the emergence of chronic organ failure, pancreatic necrosis (either sterile or infected), requirement for intervention (endoscopic, radiological, or surgical), admission to the critical care unit, duration of hospital stay, and in-hospital mortality.

Statistical Analysis

Statistical analysis was conducted utilizing SPSS version 28.0 (IBM Corp., Armonk, NY) and MedCalc version 20.0. The normality of continuous variables was evaluated using the Kolmogorov-Smirnov test and Q-Q plots. Continuous variables were presented as mean ± standard deviation (SD) or median (interquartile range) where applicable. Categorical variables were displayed as frequencies and percentages.

One-way ANOVA or the Kruskal-Wallis test was employed to compare continuous variables among severity groups, followed by post-hoc pairwise comparisons utilizing Tukey's or Dunn's test. The Chi-square test or Fisher's exact test was utilized for categorical variables. Pearson or Spearman correlation analysis evaluated the correlations between PCT levels and severity indicators.

An examination of the receiver operating characteristic (ROC) curve was conducted to assess the discriminative capacity of PCT at various time intervals, as well as the APACHE II and Ranson scores, in predicting severe acute pancreatitis (AP). The areas under the curve (AUC) were computed with 95% confidence intervals and compared utilizing the DeLong method. Optimal cutoff values were established utilizing the Youden index (sensitivity + specificity - 1).

A multivariate logistic regression analysis was performed to determine independent predictors of severe acute pancreatitis, including procalcitonin, APACHE II score, age, etiology, and other pertinent variables. Odds ratios (OR) accompanied with 95% confidence intervals (CI) were computed. A two-tailed p-value of less than 0.05 was deemed statistically significant.

RESULTS

Baseline Characteristics and Severity Classification

A total of 180 individuals diagnosed with acute pancreatitis were enrolled. As to the Revised Atlanta Classification, 102 patients (56.7%) were diagnosed with mild acute pancreatitis (AP), 48 patients (26.7%) with moderately severe AP, and 30 patients (16.6%) with severe AP. Table 1 delineates baseline demographic and clinical characteristics categorized by severity. The mean age was substantially greater in severe acute pancreatitis (54.6 ± 12.4 years) than in mild acute pancreatitis (45.2 ± 14.8 years, $p<0.001$). No notable changes in sex distribution were seen among severity categories ($p=0.524$).

Biliary etiology was most common overall (52.2%), followed by alcoholic (31.1%), hypertriglyceridemia (8.3%), and idiopathic (8.4%). The proportion of alcoholic etiology was higher in severe AP (43.3%) compared to mild AP (26.5%), though this did not reach statistical significance ($p=0.142$). As expected, APACHE II and Ranson scores increased significantly with severity category (both $p<0.001$). Severe AP

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patients had significantly higher rates of organ failure (100% vs. 35.4% vs. 0%, p<0.001), pancreatic necrosis (76.7% vs. 16.7% vs. 0%, p<0.001), and ICU admission (86.7% vs. 22.9% vs. 2.0%, p<0.001).

Table 1. Baseline Characteristics and Clinical Features Categorized by Severity

Characteristic	Mild AP (n=102)	Moderately Severe AP (n=48)	Severe AP (n=30)	p-value
Age (years)	45.2 ± 14.8	49.8 ± 13.6	54.6 ± 12.4	0.002
Male, n (%)	62 (60.8)	32 (66.7)	20 (66.7)	0.524
BMI (kg/m²)	25.4 ± 3.8	27.2 ± 4.2	28.6 ± 4.6	<0.001
Time to admission (hours)	18.6 ± 12.4	16.8 ± 11.2	14.2 ± 10.6	0.168
Etiology, n (%)				0.142
Biliary	56 (54.9)	26 (54.2)	12 (40.0)	
Alcoholic	27 (26.5)	19 (39.6)	13 (43.3)	
Hypertriglyceridemia	9 (8.8)	3 (6.2)	3 (10.0)	
Idiopathic	10 (9.8)	0 (0.0)	2 (6.7)	
Comorbidities, n (%)				
Diabetes mellitus	22 (21.6)	14 (29.2)	12 (40.0)	0.084
Hypertension	34 (33.3)	22 (45.8)	16 (53.3)	0.064
Severity Scores				
APACHE II (24h)	4.8 ± 2.2	9.4 ± 3.6	14.6 ± 4.2	<0.001
Ranson score	1.2 ± 0.8	2.8 ± 1.2	4.6 ± 1.4	<0.001
Modified CTSI	2.1 ± 1.4	5.2 ± 2.1	7.8 ± 1.6	<0.001
Complications, n (%)				
Organ failure	0 (0.0)	17 (35.4)	30 (100.0)	<0.001
Pancreatic necrosis	0 (0.0)	8 (16.7)	23 (76.7)	<0.001
Infected necrosis	0 (0.0)	2 (4.2)	11 (36.7)	<0.001
ICU admission, n (%)	2 (2.0)	11 (22.9)	26 (86.7)	<0.001
Hospital stay (days)	6.4 ± 2.8	12.6 ± 5.4	28.4 ± 14.6	<0.001
Mortality, n (%)	0 (0.0)	1 (2.1)	7 (23.3)	<0.001

Procalcitonin Levels and Biochemical Parameters

Table 2 shows serum procalcitonin levels at different time points and other biochemical parameters across severity groups. PCT levels demonstrated progressive and significant elevation with increasing severity at all time points. At admission, mean PCT was 0.34 ± 0.28 ng/mL in mild AP, 1.42 ± 0.86 ng/mL in moderately severe AP, and 4.86 ± 2.34 ng/mL in severe AP (p<0.001). PCT levels increased further at 24 and 48 hours in moderately severe and severe cases, while remaining relatively stable in mild AP.

The highest discriminative ability was observed with PCT at 48 hours, with mean values of 0.28 ± 0.24 ng/mL, 2.18 ± 1.24 ng/mL, and 7.42 ± 3.86 ng/mL for mild, moderately severe, and severe AP respectively (p<0.001). Post-hoc analysis revealed significant differences between all pairwise comparisons (all p<0.001).

Correlation analysis demonstrated that PCT at 48 hours showed strongest correlation with severity (r = 0.742, p<0.001), followed by PCT at 24 hours (r = 0.684, p<0.001) and admission PCT (r = 0.612, p<0.001). PCT levels correlated significantly with APACHE II score (r = 0.698, p<0.001), Ranson score (r = 0.656, p<0.001), and modified CTSI (r = 0.624, p<0.001).

CRP levels also increased with severity but peaked later than PCT. At admission, CRP showed less discrimination between groups compared to PCT. White blood cell count, serum amylase, and lipase showed no consistent correlation with severity. Patients with severe AP had significantly higher creatinine and lower calcium levels, reflecting organ dysfunction.

Table 2. Procalcitonin Levels and Biochemical Parameters Across Severity Groups

Parameter	Mild AP (n=102)	Moderately Severe AP (n=48)	Severe AP (n=30)	p-value
Procalcitonin (ng/mL)				
Admission	0.34 ± 0.28	1.42 ± 0.86*	4.86 ± 2.34*†	<0.001
24 hours	0.32 ± 0.26	1.84 ± 1.12*	6.24 ± 3.12*†	<0.001
48 hours	0.28 ± 0.24	2.18 ± 1.24*	7.42 ± 3.86*†	<0.001
PCT >0.5 ng/mL at admission, n (%)	14 (13.7)	38 (79.2)	30 (100.0)	<0.001
PCT >1.8 ng/mL at 48h, n (%)	4 (3.9)	28 (58.3)	28 (93.3)	<0.001
Other Biomarkers				

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WBC (×10³/μL)	11.2 ± 3.4	13.8 ± 4.6	16.4 ± 5.8	<0.001
CRP admission (mg/dL)	2.4 ± 1.8	4.6 ± 3.2	6.8 ± 4.2	<0.001
CRP 48h (mg/dL)	8.2 ± 4.6	16.4 ± 6.8	22.6 ± 8.4	<0.001
Serum amylase (U/L)	842 ± 486	924 ± 512	786 ± 524	0.384
Serum lipase (U/L)	1248 ± 624	1386 ± 742	1124 ± 688	0.256
Organ Function				
Creatinine (mg/dL)	0.9 ± 0.2	1.2 ± 0.4	2.1 ± 1.2	<0.001
Total bilirubin (mg/dL)	1.8 ± 1.2	2.4 ± 1.6	2.8 ± 1.8	0.008
Serum calcium (mg/dL)	8.8 ± 0.6	8.2 ± 0.8	7.4 ± 0.9	<0.001
LDH (U/L)	246 ± 86	382 ± 124	524 ± 186	<0.001
Albumin (g/dL)	3.8 ± 0.4	3.4 ± 0.6	2.9 ± 0.6	<0.001

*p<0.001 vs. Mild AP; †p<0.001 vs. Moderately Severe AP

Diagnostic Performance of Procalcitonin and Comparison with Scoring Systems

Table 3 presents ROC curve analysis results comparing the diagnostic performance of PCT at different time points with conventional scoring systems for predicting severe AP. PCT at 48 hours demonstrated the highest discriminative ability with AUC of 0.912 (95% CI: 0.862-0.962), significantly superior to APACHE II score (AUC = 0.856, 95% CI: 0.796-0.916, p=0.042) and Ranson score (AUC = 0.824, 95% CI: 0.758-0.890, p=0.018).

Using Youden index, the optimal cutoff for PCT at 48 hours was 1.8 ng/mL, yielding sensitivity of 86.7% (95% CI: 69.3-96.2%) and specificity of 84.3% (95% CI: 77.4-89.8%) for predicting severe AP. Positive predictive value was 56.5% and negative predictive value was 96.2%. The likelihood ratio positive was 5.52 and likelihood ratio negative was 0.16.

PCT at 24 hours also showed good performance (AUC = 0.882, 95% CI: 0.826-0.938) with optimal cutoff of 1.5 ng/mL (sensitivity 83.3%, specificity 82.0%). Admission PCT had lower but still acceptable performance (AUC = 0.834, 95% CI: 0.768-0.900).

For predicting the combined outcome of moderately severe or severe AP, PCT at 48 hours with a lower cutoff of 0.8 ng/mL demonstrated sensitivity of 84.6% and specificity of 78.4% (AUC = 0.886).

Multivariate logistic regression analysis including PCT at 48 hours, APACHE II score, age, BMI, and etiology revealed that PCT >1.8 ng/mL was the strongest independent predictor of severe AP (adjusted OR = 12.64, 95% CI: 5.38-29.68, p<0.001). APACHE II score >8 (adjusted OR = 4.28, 95% CI: 1.86-9.84, p=0.001) and age >55 years (adjusted OR = 2.42, 95% CI: 1.08-5.42, p=0.032) were also independent predictors.

Table 3. Diagnostic Performance of PCT and Conventional Scores for Predicting Severe AP

A. ROC Curve Analysis	AUC (95% CI)	p-value vs. PCT 48h	Optimal Cutoff	Sensitivity (%)	Specificity (%)
PCT admission	0.834 (0.768-0.900)	0.008	1.2 ng/mL	80.0	78.7
PCT 24 hours	0.882 (0.826-0.938)	0.186	1.5 ng/mL	83.3	82.0
PCT 48 hours	0.912 (0.862-0.962)	Reference	1.8 ng/mL	86.7	84.3
APACHE II	0.856 (0.796-0.916)	0.042	9.5	80.0	78.7
Ranson score	0.824 (0.758-0.890)	0.018	3.5	76.7	80.0
CRP 48h	0.792 (0.718-0.866)	0.001	15 mg/dL	73.3	76.0

B. Performance of PCT 48h at 1.8 ng/mL Cutoff	Value (95% CI)
Sensitivity	86.7% (69.3-96.2%)
Specificity	84.3% (77.4-89.8%)
Positive predictive value	56.5% (42.8-69.4%)
Negative predictive value	96.2% (91.6-98.6%)
Positive likelihood ratio	5.52 (3.86-7.90)
Negative likelihood ratio	0.16 (0.06-0.40)
Accuracy	84.7% (78.5-89.6%)

C. Multivariate Logistic Regression for Severe AP	Adjusted OR (95% CI)	p-value
PCT 48h >1.8 ng/mL	12.64 (5.38-29.68)	<0.001
APACHE II >8	4.28 (1.86-9.84)	0.001
Age >55 years	2.42 (1.08-5.42)	0.032
BMI >30 kg/m²	1.86 (0.78-4.44)	0.162
Alcoholic etiology	1.64 (0.72-3.74)	0.238
Creatinine >1.4 mg/dL	3.12 (1.28-7.60)	0.012

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DISCUSSION

This prospective study demonstrates that serum procalcitonin, particularly when measured at 48 hours after admission, is a highly accurate biomarker for predicting severe acute pancreatitis. Our findings reveal that PCT at 48 hours, with an optimal cutoff of 1.8 ng/mL, achieves superior discriminative ability (AUC = 0.912) compared to established scoring systems including APACHE II and Ranson criteria. Moreover, elevated PCT emerged as an independent predictor of severe disease even after adjusting for multiple clinical and biochemical parameters.

The mean PCT level of 7.42 ± 3.86 ng/mL observed in severe AP patients in our cohort is consistent with previous reports **[17, 18]**. The progressive elevation of PCT with increasing disease severity, coupled with its strong correlation with clinical outcomes, supports its biological relevance in pancreatitis pathophysiology. These findings align with a systematic review by Mofidi et al., which reported pooled sensitivity and specificity of 74% and 87% for PCT in predicting severe AP **[14]**. Our study demonstrates even higher diagnostic accuracy, possibly reflecting optimal timing of measurement and use of contemporary severity classification.

The superior performance of PCT compared to conventional scoring systems has important mechanistic and clinical implications. Unlike multifactorial scores requiring multiple parameters collected over 24-48 hours, PCT represents a single, objective biomarker measurable within hours of admission **[19]**. The biological basis for PCT elevation in severe pancreatitis involves multiple interconnected pathways. The intense inflammatory response characteristic of severe AP triggers systemic release of pro-inflammatory cytokines, particularly interleukin-6 and tumor necrosis factor-alpha, which stimulate extrathyroidal PCT production **[20]**. Additionally, bacterial translocation from the compromised intestinal barrier and subsequent development of infected pancreatic necrosis further amplify PCT production **[21]**.

Our finding that PCT at 48 hours outperforms earlier measurements merits discussion. Khanna et al. similarly reported that PCT measured at 48-72 hours demonstrated better predictive accuracy than admission values **[15]**. This temporal pattern likely reflects the evolution of systemic inflammatory response and potential bacterial complications. Early in the disease course, local pancreatic inflammation predominates, whereas systemic inflammatory response syndrome and potential septic complications develop subsequently. The 48-hour time point appears to capture the transition to severe systemic disease while still being sufficiently early for meaningful clinical intervention.

The independent predictive value of PCT in multivariate analysis, adjusting for APACHE II score and other variables, suggests that it provides complementary rather than redundant information. APACHE II incorporates physiological parameters reflecting organ dysfunction, while PCT specifically reflects inflammatory and infectious processes **[22]**. The combination of clinical scoring and PCT measurement may optimize risk stratification. Several investigators have proposed integrated algorithms combining biomarkers with clinical scores for enhanced prediction **[23]**.

Comparison with other biomarkers is instructive. While CRP has been extensively studied and demonstrates good predictive value **[24]**, its delayed peak at 48-72 hours and relatively lower specificity limit utility for early decision-making. In our cohort, PCT at 48 hours (AUC = 0.912) outperformed CRP at the same time point (AUC = 0.792), consistent with findings by Mándi et al. **[25]**. The more rapid kinetics and higher specificity of PCT for bacterial complications make it particularly valuable in the pancreatitis setting where infected necrosis substantially increases mortality.

Our study also examined PCT performance across the spectrum of severity as defined by Revised Atlanta Classification, including the moderately severe category often overlooked in earlier studies. The intermediate PCT levels in moderately severe AP (2.18 ± 1.24 ng/mL at 48 hours) and different optimal cutoffs for different severity thresholds suggest potential for risk stratification beyond binary classification. This nuanced approach may guide individualized management strategies.

The clinical implications of our findings are substantial. Early identification of patients at risk for severe disease enables timely transfer to higher-level care, aggressive fluid resuscitation, nutritional support, and close monitoring for complications **[26]**. Conversely, accurately identifying low-risk patients could facilitate early discharge and avoid unnecessary interventions. The high negative predictive value (96.2%) of PCT <1.8 ng/mL is particularly useful for excluding severe disease. Economic analyses suggest that biomarker-guided management could reduce healthcare costs by optimizing resource utilization **[27]**.

Furthermore, PCT's established role in guiding antibiotic therapy in other conditions **[28]** raises the possibility of using serial PCT measurements to inform decisions regarding prophylactic antibiotics in necrotizing pancreatitis, though this requires specific validation. Current guidelines generally recommend against routine antibiotic prophylaxis but support targeted therapy for documented or strongly suspected infected necrosis **[29]**. PCT monitoring might help identify the subset of patients who would benefit from antimicrobial intervention.

Several strengths enhance the validity of our study. The prospective design with standardized data collection minimizes bias. Use of the current Revised Atlanta Classification ensures contemporary relevance. Measurement of PCT at multiple time points provides insights into temporal dynamics.

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Comprehensive comparison with established scoring systems and multivariate adjustment for confounders strengthen evidence for independent predictive value. The relatively large sample size and adequate representation of all severity categories enhance statistical power and generalizability.

However, limitations warrant consideration. The single-center design may limit external validity, as patient populations and practice patterns vary across institutions. The study population's ethnic and geographic characteristics may affect generalizability, as genetic factors, dietary patterns, and baseline inflammatory states influence PCT levels **[30]**. We excluded patients with concurrent infections and certain comorbidities to isolate pancreatitis-specific PCT elevation, but this limits applicability to the broader population presenting with acute pancreatitis. Cost-effectiveness analysis was not performed; routine PCT measurement would increase initial diagnostic costs, though potential savings from improved triage could offset this. Long-term outcomes and recurrence rates were not assessed. Finally, while we demonstrated excellent predictive accuracy, the study was observational and cannot directly demonstrate that PCT-guided management improves clinical outcomes. Randomized controlled trials comparing PCT-guided versus conventional management strategies are needed to definitively establish clinical utility.

CONCLUSION

This study establishes serum procalcitonin as a highly accurate and independent biomarker for predicting severity of acute pancreatitis. Measurement at 48 hours after admission demonstrates superior discriminative ability compared to conventional clinical scoring systems, with an optimal cutoff of 1.8 ng/mL achieving 86.7% sensitivity and 84.3% specificity for identifying severe disease. The progressive elevation of PCT levels across severity categories, strong correlation with adverse outcomes, and independent predictive value in multivariate analysis support its biological and clinical relevance.

The findings have important implications for early risk stratification and triage of acute pancreatitis patients. The simple, objective, and rapidly available nature of PCT measurement offers practical advantages over complex scoring systems requiring multiple parameters collected over extended periods. The high negative predictive value of low PCT levels enables confident identification of patients at low risk for severe disease, potentially facilitating safe early discharge and reducing unnecessary interventions. Conversely, elevated PCT levels signal need for intensive monitoring and aggressive management.

PCT measurement at 48 hours appears to represent an optimal balance between early availability for clinical decision-making and sufficient time for manifestation of severe systemic inflammatory response. Integration of PCT into clinical algorithms, potentially in combination with conventional scoring systems, may enhance risk stratification accuracy and guide resource allocation. However, before widespread clinical implementation, validation in diverse populations and demonstration of improved patient outcomes through PCT-guided management strategies in randomized controlled trials are necessary. Future research should also explore PCT's role in monitoring treatment response, predicting specific complications such as infected necrosis, and guiding antibiotic therapy decisions in necrotizing pancreatitis.

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