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ROLE OF SERUM PROCALCITONIN IN ANTIBIOTIC UTILIZATION AND EARLY PROGNOSTICATION IN ACUTE PANCREATITIS: A RETROSPECTIVE COHORT STUDY

KEY WORDS: Acute Pancreatitis; Procalcitonin; Severity Prediction; Organ Failure

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ABSTRACT

Background: Early identification of severe acute pancreatitis and rational antibiotic use remain major clinical challenges. Procalcitonin (PCT) has emerged as a potential biomarker for severity stratification and infection-guided therapy. **Methods:** This retrospective cohort study included 60 adult patients admitted with acute pancreatitis between November 2024 and November 2025. Admission serum PCT levels (within 48 hours) were categorized and analyzed in relation to Atlanta severity classification, organ failure, ICU and hospital stay, in-hospital mortality, and antibiotic utilization. Receiver operating characteristic (ROC) analysis was performed to determine prognostic accuracy. **Results:** Median admission PCT increased significantly across Atlanta severity categories ($p < 0.001$). ROC analysis demonstrated excellent performance for predicting severe acute pancreatitis (AUC 0.98; 95% CI 0.94–1.00). A threshold of ≥2.0 ng/mL showed 100% sensitivity and 98% specificity for severe disease. Organ failure occurred predominantly in patients with PCT ≥2.0 ng/mL, and all non-survivors had elevated PCT levels. ICU and hospital stay increased progressively with higher PCT categories. Antibiotics were administered to 71.7% of patients and were significantly associated with higher admission PCT and WBC levels; however, 18.6% received antibiotics despite low PCT (<0.5 ng/mL). **Conclusions:** Admission serum procalcitonin is a reliable early prognostic marker in acute pancreatitis, demonstrating strong association with severity, organ failure, mortality, and resource utilization. While antibiotic prescribing partially aligned with biomarker elevation, variability highlights the need for standardized PCT-guided stewardship protocols. Prospective validation is warranted.

INTRODUCTION

Acute pancreatitis represents one of the most frequently encountered emergencies in gastrointestinal practice, with reported incidence rates worldwide varying between 5 and 80 cases per 100,000 population.[1] Although many patients experience a mild disease course, acute pancreatitis continues to be associated with substantial morbidity and mortality. Severe forms of the disease develop in approximately 15–20% of patients and are associated with mortality rates ranging from 15% to 30%, even with advances in contemporary management strategies.[1,2]

The inappropriate use of antibiotics in acute pancreatitis continues to pose a significant challenge in routine clinical care. International consensus guidelines issued by the International Association of Pancreatology and the American Pancreatic Association advise against the routine administration of prophylactic antibiotics in patients with acute pancreatitis, including those with pancreatic necrosis.[3]

Nevertheless, adherence to guideline-based recommendations in routine clinical practice remains inconsistent, primarily due to challenges in accurately differentiating sterile inflammatory processes from bacterial infection. This uncertainty frequently results in empirical antibiotic use, contributing to excessive antimicrobial exposure, increased healthcare expenditure, the emergence of antimicrobial resistance, and a higher incidence of drug-related adverse events.[3,4]

Conventional inflammatory markers, including C-reactive protein, RDW, LDH have limited specificity for differentiating sterile pancreatic inflammation from bacterial infection, particularly early in acute pancreatitis. This limitation contributes to uncertainty in antibiotic decision-making, highlighting the need for an objective biomarker for early infection detection and severity assessment.[5]

Procalcitonin is a peptide composed of 116 amino acids and serves as the precursor of calcitonin. It has gained recognition as an inflammatory biomarker with greater specificity for bacterial infection compared with conventional markers.[6,7] Under normal physiological conditions, circulating procalcitonin concentrations are minimal, typically measuring below 0.01 µg/L. In the presence of bacterial infection, serum levels increase rapidly within 4-12 hours and may rise to markedly elevated values in cases of severe sepsis exceeding 100 µg/L.[7]

Emerging evidence indicates that procalcitonin is useful for early assessment of disease severity, risk stratification, timely detection of complications, optimization of antimicrobial duration, and prediction of mortality in acute pancreatitis.[8]

In this context, the present study was undertaken to evaluate the role of serum procalcitonin as an early prognostic marker in acute pancreatitis and to assess its utility in guiding antibiotic therapy. By comparing clinical outcomes, severity indices, and antibiotic usage patterns, this retrospective cohort study aims to provide insight into the potential role of procalcitonin in improving risk stratification and optimizing antibiotic stewardship in patients with acute pancreatitis.

Objectives

To evaluate the association between admission serum procalcitonin levels and disease severity, organ failure, and in-hospital mortality, and to identify optimal prognostic cut-off values in acute pancreatitis using retrospective analysis.

To evaluate patterns of antibiotic use in relation to serum procalcitonin levels and to compare these patterns with conventional clinician-driven antibiotic practices.

MATERIALS AND METHODS

Study Design and Setting

This was a retrospective cohort study conducted in the Department of General Surgery at RL Jalappa Hospital and Research Centre, Tamaka, Kolar, Karnataka, India. Medical records of patients admitted with acute pancreatitis between November 2024 and November 2025 were reviewed.

Study Population

The sample size was determined by the availability of complete medical records within the specified timeframe .Of 200 screened case records 60 files with complete information are included according to calculated sample size. All eligible patients admitted during the study period who fulfilled the predefined inclusion and exclusion criteria as mentioned below were analyzed.

Inclusion Criteria

Patients aged ≥18 years admitted with a diagnosis of acute pancreatitis according to Atlanta Classification 2013 criteria, requiring ≥2 of the following: characteristic abdominal pain (epigastric, radiating to back), serum amylase and/or lipase ≥3 times upper limit of normal, or characteristic imaging findings on contrast-enhanced CT;

Exclusion Criteria

Patients younger than 18 years were excluded. Additional exclusion criteria included absence of serum procalcitonin measurement within 48 hours of admission and incomplete medical records, chronically elevated serum lipase levels in patients with chronic pancreatitis, and elevated lipase levels attributable to other causes such as pancreatic malignancy, pancreatic trauma, post-endoscopic retrograde cholangio-pancreatography for choledocholithiasis, or cirrhosis in the absence of clinical features of acute pancreatitis.[9]

Data Collection and Variable Categorization

Demographic, laboratory, radiological, and outcome data were retrospectively obtained from case records. Collected variables included baseline characteristics, serial inflammatory and pancreatic biomarkers, contrast-enhanced CT findings, established severity scores, antibiotic utilization patterns, major complications, length of stay, and in-hospital mortality.

Several continuous variables demonstrated a non-normal distribution and were categorized for analytical purposes. Serum procalcitonin levels measured within 48 hours of admission were retrospectively stratified into four categories based on published thresholds: 0.10–0.49 ng/mL (minimal inflammatory response), 0.50–1.99 ng/mL (moderate inflammatory response), 2.00–9.99 ng/mL (severe systemic inflammatory response), and ≥10.00 ng/mL (high likelihood of severe sepsis or septic shock).[9] No predefined biomarker-based treatment protocol was applied, and all clinical decisions were made by the treating physicians at the time of care.

Ethical Approval

The study was approved by the Institutional Ethics Committee of RL Jalappa Hospital and Research Centre. Given the retrospective nature of the study and use of case records, the requirement for informed consent was waived.

Statistical Analysis:

Statistical analysis was performed using SPSS software (version 22.0; IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation or median with interquartile range, as appropriate, while categorical variables were summarized as frequencies and percentages. Comparisons between groups were conducted using the independent samples t-test or Mann–Whitney U test for continuous variables and the chi-square test or Fisher's exact test for categorical variables. Receiver operating characteristic curve analysis was performed to evaluate the prognostic performance of serum procalcitonin and to identify optimal cut-off values for predicting adverse outcomes. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

A total of 60 patients with acute pancreatitis were included in the study. The mean age of the study population was 41.6 ± 11.8 years, with a median age of 42 years (interquartile range, 33–50 years).

There was a marked male predominance, with 48 patients (80.0%) being male and 12 patients (20.0%) female.

According to the Revised Atlanta Classification, 30 patients (50.0%) had mild acute pancreatitis, 22 patients (36.7%) had moderately severe disease, and 8 patients (13.3%) had severe acute pancreatitis.

With respect to etiology, alcohol was the most common cause of acute pancreatitis, accounting for 40 cases (66.7%). Gallstone disease was the second most frequent etiology, observed in 11 patients (18.3%). Less common causes included trauma in 4 patients (6.7%), hypertriglyceridemia in 2 patients (3.3%), and other etiologies in 3 patients (5.0%) depicted in table-1.

Table-1 Baseline Demographic and Clinical Characteristics [n=60]

Variable	Category	n (%) / Mean ± SD / Median (IQR)
Age (years)		41.6 ± 11.8
	Median (IQR)	42 (33–50)
Sex	Male	48 (80.0%)
	Female	12 (20.0%)
Severity (Atlanta classification)	Mild	30 (50.0%)
	Moderately severe	22 (36.7%)
	Severe	8 (13.3%)
Etiology	Alcohol	40 (66.7%)
	Gall stones	11 (18.3%)
	Trauma	4 (6.7%)
	Hyper-triglyceridemia	2 (3.3%)
	Other	3 (5.0%)

Procalcitonin Values and Trends

Procalcitonin (PCT) at admission was negative or undetectable in 10 patients (16.7%), indicating no detectable systemic inflammatory response at presentation.

Among the 50 patients with detectable PCT levels, the values ranged from 0.27 ng/mL to 10.5 ng/mL. The mean PCT level was 1.88 ± 2.42 ng/mL, while the median PCT was 1.15 ng/mL (interquartile range [IQR]: 0.34–1.78 ng/mL), demonstrating a right-skewed distribution. All values are stratified according to predefined inflammatory response categories as described in table-2

Table 2 - Distribution of Procalcitonin Levels at Admission, Day-3 and Day-5

Procalcitonin category	PCT range (ng/mL)	Ad-mission (n = 60)	Day 3 (n = 47)	Day 5 (n = 46)
Negative (undetectable)	Below assay detection limit	10 (16.7%)	—	1 (2.2%)
Minimal inflammatory response	0.10–0.49	18 (30.0%)	17 (36.2%)	18 (39.1%)

Moderate inflammatory response	0.50–1.99	21 (35.0%)	21 (44.7%)	23 (50.0%)
Severe systemic inflammatory response	2.00–9.99	10 (16.7%)	6 (12.8%)	3 (6.5%)
High likelihood of severe sepsis / septic shock	≥10.00	1 (1.7%)	3 (6.4%)	1 (2.2%)
Total assessed	—	60 (100%)	47 (100%)	46 (100%)

*Missing Day 3 and Day 5 values were due to early mortality or clinical non-availability.

Serial procalcitonin (PCT) trends showed heterogeneity over time as shown in table-2 .Ten patients had negative admission PCT and were not retested, while additional missing Day 3 and Day 5 values were related to early mortality or clinical non-availability, potentially influencing longitudinal interpretation. Although only one patient had PCT ≥10 ng/mL at admission, three patients reached this threshold on Day 3, suggesting progression of systemic inflammation in a subset. By Day 5, persistently elevated PCT ≥10 ng/mL was observed in one patient, whereas declining PCT levels were predominantly seen among survivors. These findings should be interpreted cautiously, given survivorship bias inherent to serial measurements in retrospective cohorts.

Association Between Admission Procalcitonin and Atlanta Severity

Admission procalcitonin (PCT) differed significantly across Atlanta severity categories (Table). Median PCT was 0.31 ng/mL (IQR 0.29–0.36; n = 19) in mild pancreatitis, 1.45 ng/mL (IQR 1.13–1.68; n = 22) in moderately severe pancreatitis, and 6.8 ng/mL (IQR 3.5–8.2; n = 9) in severe pancreatitis. Due to non-normal distribution, a Kruskal–Wallis test was used and showed a significant difference across groups ($H = 41.35$, $p < 0.001$). Negative PCT values were included in the mild group for descriptive analysis but excluded from statistical testing. Higher admission PCT levels were associated with increasing Atlanta severity.

Table-3 Admission Procalcitonin Levels According to Atlanta Severity Classification

Atlanta severity	Patients (n)*	Median PCT (IQR), ng/mL	Mean ± SD, ng/mL
Mild	19	0.31 (0.29–0.36)	0.35 ± 0.10
Moderately severe	22	1.45 (1.13–1.68)	1.44 ± 0.44
Severe	9	6.8 (3.5–8.2)	6.20 ± 2.85
Statistical test	—	Kruskal–Wallis H = 41.35	p < 0.001

*Negative PCT values were included within the mild severity group for descriptive purposes. Statistical comparisons were performed excluding negative values due to their non-quantitative nature.

Receiver operating characteristic (ROC) analysis demonstrated excellent diagnostic performance of admission serum procalcitonin for predicting severe acute pancreatitis (Figure 1). The area under the ROC curve (AUC) was 0.98 (95% confidence interval [CI]: 0.94–1.00), which was significantly greater than the reference line of no discrimination (AUC = 0.50; $p < 0.001$).

At an admission procalcitonin cut-off value of ≥0.5 ng/mL, the sensitivity for predicting severe acute pancreatitis was 100%, with a specificity of 52.9%, indicating excellent screening ability. Increasing the threshold to ≥2.0 ng/mL improved specificity to 98.0% while maintaining 100% sensitivity, suggesting strong discriminative power for confirming severe disease.

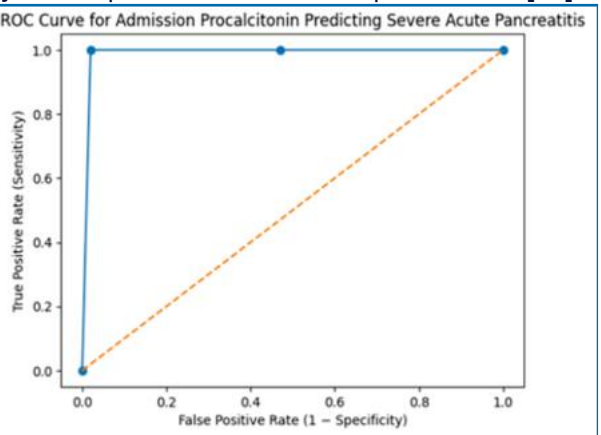

Figure 1- ROC Analysis of Admission Procalcitonin in Predicting Severe Acute Pancreatitis

≥0.5 ng/mL: excellent for screening / ruling out severe disease

≥2.0 ng/mL: excellent for confirming severe disease

Association Between PCT and Antibiotic Utilization

Antibiotic utilization was defined as initiation of any systemic antibacterial agent during hospital admission for acute pancreatitis. Accordingly Antibiotics were administered to 43 of 60 patients (71.7%). Patients receiving antibiotics had significantly higher admission WBC counts and procalcitonin (PCT) levels compared with those who did not (Table 4).

Table 4. Admission Procalcitonin and WBC According to Antibiotic Use

Variable	Antibiotics used (n = 43)	No antibiotics (n = 17)	p-value*
WBC count ($\times 10^9$/L), median (IQR)	15.4 (13.7–19.6)	9.6 (8.9–10.9)	< 0.001
Admission PCT (ng/mL), median (IQR)†	1.40 (0.35–3.50)	0.30 (0.28–0.36)	< 0.001

†Negative PCT values were included as 0 ng/mL for descriptive comparison.

*p-values calculated using Mann–Whitney U test due to non-normal distribution.

Admission procalcitonin (PCT) showed a strong positive correlation with admission WBC counts (Spearman = 0.93, $p < 0.001$; n = 50). Higher PCT values were consistently associated with higher leukocyte counts, indicating concordance between biochemical and hematological markers of systemic inflammation at presentation.

Among antibiotic recipients, PCT-guided initiation (PCT ≥0.5 ng/mL) and leucocytosis were the most frequently documented indications, followed by sepsis or septic shock and clinical suspicion of infection. Ceftriaxone with metronidazole was the most commonly prescribed regimen, whereas broader-spectrum antibiotics, including piperacillin –tazobactam and meropenem, were predominantly used in patients with sepsis, septic shock, or higher inflammatory marker levels. However, 8 patients (18.6%) received antibiotics despite low admission PCT values (<0.5 ng/mL), including 4 patients (9.3%) with undetectable PCT. Additionally, 5 patients (11.6%) had low admission WBC counts, and the same 5 patients (11.6%) had both low or negative PCT and low WBC at presentation (Table 5), indicating antibiotic initiation in the absence of biochemical evidence of infection.

Table 5. Antibiotic Use Despite Low Admission Inflammatory Markers

Parameter among antibiotic recipients (n = 43)	n (%)
Low PCT (<0.5 ng/mL)	8 (18.6%)
Negative (undetectable) PCT	4 (9.3%)

Low WBC (<11 ×10^9/L)	5 (11.6%)
Low WBC + low/negative PCT	5 (11.6%)

Association Between PCT and Clinical Outcomes.

Admission serum procalcitonin levels demonstrated a clear association with clinically relevant outcomes. Increasing procalcitonin categories were associated with progressively longer ICU and hospital stays shown in figure-2. Organ failure was observed predominantly among patients with admission procalcitonin ≥2.00 ng/mL. All non-survivors had markedly elevated procalcitonin levels (≥2.00 ng/mL), whereas no mortality occurred in patients with admission procalcitonin levels below this threshold as depicted in the table-6

Table 6. Clinical Outcomes According to Admission Procalcitonin Category (n = 60)

PCT category (ng/mL)	n	ICU stay, median (IQR), days	Hospital stay, median (IQR), days	Organ failure n (%)	Survivors n (%)	Non-survivors n (%)
Negative (<0.10)	4	1 (1–1.25)	10 (10–12)	0 (0%)	4 (100%)	0 (0%)
0.10–0.49	23	2 (1–2)	12 (8–13)	0 (0%)	23 (100%)	0 (0%)
0.50–1.99	23	4 (4–5)	15 (13–16)	1 (4.3%)	23 (100%)	0 (0%)
2.00–9.99	9	7 (3–8)	17 (14–28)	8 (88.9%)	5 (55.6%)	4 (44.4%)
≥10.00	1	2 (2–2)	2 (2–2)	1 (100%)	0 (0%)	1 (100%)

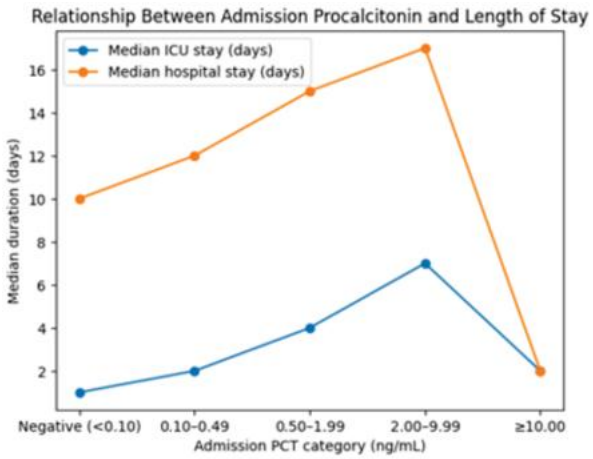

Figure-2 Relationship Between PCT and ICU / Hospital Stay [n=60]

DISCUSSION

In this study, admission serum procalcitonin demonstrated strong prognostic value in acute pancreatitis. PCT levels increased significantly across Atlanta severity categories and were closely associated with organ failure, ICU stay, hospital stay, and mortality. All non-survivors had admission PCT ≥2.0 ng/mL, and this threshold showed excellent diagnostic performance for predicting severe disease (AUC 0.98;95% CI 0.94–1.00; sensitivity 100%, specificity 98%). Additionally, higher PCT levels correlated strongly with leukocytosis and antibiotic utilization, supporting its role as an early marker of systemic inflammatory response. These findings suggest that a single admission PCT measurement may provide robust early risk stratification.

Compared with Liang et al. [10] their cohort included an older population (59.7 ± 8.9 years) with a relatively balanced sex distribution, whereas our patients were younger (41.6 ± 11.8 years) with marked male predominance (80%). Alcohol was the leading etiology in our cohort, in contrast to their more heterogeneous biliary-dominant population, indicating demographic and etiological differences that may influence disease profile.

Consistent with Rau et al. [11] we observed a strong relationship between elevated PCT and adverse outcomes. While Rau et al. required sustained PCT ≥3.5–3.8 ng/mL over two consecutive days to predict infected necrosis with MODS, our findings demonstrate that a single admission measurement can provide excellent prognostic discrimination. Differences in optimal cut-offs likely reflect variation in study endpoints and design; however, both studies confirm that higher PCT levels are strongly associated with severe complications.

Alberti et al. [12] emphasized the predictive role of PCT for infectious complications and need for urgent ERCP. In contrast, our study highlights its robust performance in predicting overall severity, organ failure, and mortality. Together, these findings reinforce admission PCT as a clinically valuable early biomarker in acute pancreatitis.

Yang et al [13] reported a SAP mortality of 16.8%, with deaths confined to the severe pancreatitis group. Similarly, in our cohort, mortality occurred exclusively among patients with elevated admission PCT levels. No deaths were observed in patients with PCT <2.0 ng/mL. In contrast, mortality was 44.4% in the 2.00–9.99 ng/mL group and 100% in those with PCT ≥10 ng/mL. These findings indicate clear stratification of mortality risk according to admission PCT levels.

Recent evidence supports the role of procalcitonin (PCT) in antibiotic stewardship. Siriwardena [14] highlights that PCT >1.0 ng/mL may justify antibiotic initiation in acute pancreatitis and that structured PCT-guided algorithms, such as in the PROCAP trial, reduce unnecessary antibiotic exposure. Similarly,Kim [15] (2022) summarizes randomized trial data demonstrating that PCT-guided strategies safely reduce antibiotic duration in sepsis and lower respiratory tract infections. In our retrospective cohort, antibiotic use was significantly associated with higher admission PCT and WBC levels, indicating biomarker-informed decision-making. However, 18.6% of treated patients had PCT <0.5 ng/mL, reflecting deviation from strict protocol-based thresholds. Unlike controlled trial settings, our findings represent real-world clinician-directed prescribing and underscore the need for standardized PCT-guided stewardship protocols in acute pancreatitis.

Badia et al[16] ,state that antibiotics in acute pancreatitis should be reserved for suspected or confirmed infected pancreatic necrosis and should ensure broad Gram-negative and Gram-positive coverage with adequate pancreatic penetration, such as carbapenems or piperacillin–tazobactam, while avoiding routine prophylaxis and unnecessary antifungal use. In our cohort, broader-spectrum agents, including piperacillin–tazobactam and meropenem, were mainly administered to patients with sepsis or elevated inflammatory markers, consistent with these recommendations. Ceftriaxone combined with metronidazole was the most frequently used regimen, providing appropriate Gram-negative and anaerobic coverage. These patterns indicate general concordance with guideline-based antimicrobial selection. However, 18.6% of patients received antibiotics despite low admission PCT levels, suggesting variability in initiation criteria. Therefore, although antibiotic choice largely reflected recommended coverage strategies, prescribing thresholds were not consistently biomarker-guided.

Dias et al [17] , demonstrated a strong correlation between admission PCT and total hospital stay (r = 0.892), ICU requirement (r = 0.643), and severe acute pancreatitis (r = 0.837), our findings show a similar graded relationship using categorical analysis. In our cohort, ICU stay increased from a median of 1 day in PCT <0.10 ng/mL to 7 days in 2.00–9.99 ng/mL, while hospital stay rose from 10 to 17 days across the

same strata. Organ failure was absent below 0.50 ng/mL but occurred in 88.9% of patients with PCT 2.00–9.99 ng/mL and 100% with ≥10 ng/mL

This study comprehensively evaluated admission procalcitonin in relation to Atlanta severity, organ failure, ICU and hospital stay, mortality, and antibiotic utilization within a single cohort. The use of ROC analysis with confidence intervals strengthens the validity of diagnostic performance estimates. Additionally, categorical stratification of procalcitonin enabled clinically interpretable risk gradients across outcome measures.

Limitations

The retrospective single-center design limits external validity and introduces potential selection bias. The modest sample size, particularly within higher procalcitonin strata, may have contributed to the high observed AUC and warrants external validation. Incomplete serial measurements introduce potential survivorship bias. Furthermore, antibiotic use was clinician-directed without a standardized procalcitonin-guided protocol, precluding assessment of causal stewardship impact.

Clinical Implications

Admission procalcitonin appears to be a practical early risk stratification tool in acute pancreatitis. A threshold of ≥2.0 ng/mL demonstrated strong discrimination for severe disease and mortality, supporting early escalation of care, while lower values may assist in identifying low-risk patients and promoting more judicious antibiotic use. Prospective validation of standardized procalcitonin-guided algorithms is warranted.

CONCLUSION

This retrospective study demonstrates that admission serum procalcitonin is a reliable early marker of disease severity in acute pancreatitis. Elevated levels were independently associated with organ failure, longer ICU and hospital stay, and in-hospital mortality, showing clear risk stratification across predefined categories. A cut-off of ≥2.0 ng/mL identified patients at high risk for adverse outcomes with strong diagnostic performance. Although antibiotic initiation correlated with higher procalcitonin values, variability in prescribing practices highlights the need for structured biomarker-guided protocols. Larger prospective studies are required to validate these findings and define standardized clinical algorithms.

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