

## **Original Research Article**

# A PROSPECTIVE STUDY ON THE ROLE OF APACHE III AND PROCALCITONIN IN RISK STRATIFICATION OF INFECTED NECROTIZING PANCREATITIS

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## ABSTRACT

**Background:** Infected necrotizing pancreatitis (INP) is a severe and lifethreatening complication of acute pancreatitis (AP), associated with high morbidity and mortality. Early risk identification is critical for timely intervention and improved clinical outcomes.

**Materials and Methods:** A prospective observational study was conducted including 52 patients diagnosed with acute pancreatitis. Patients were stratified into infective and sterile groups using clinical, radiological, and laboratory criteria. Data collected included physiological parameters, serum procalcitonin levels, and APACHE III scores, which were analyzed for their prognostic value **Results:** Patients in the infective group showed significantly higher serum procalcitonin levels and APACHE III scores compared to the sterile group. These findings underscore the utility of both markers in early risk stratification of INP.

**Conclusion:** The study demonstrates that both the APACHE III scoring system and serum procalcitonin levels serve as effective predictors for the development of infected necrotizing pancreatitis. Their combined use could facilitate timely and targeted therapeutic interventions in patients with acute pancreatitis.

**Keywords:** Acute pancreatitis, infected necrotizing pancreatitis, APACHE III score, procalcitonin, prognostic biomarkers, risk stratification.

# INTRODUCTION

Acute pancreatitis is an inflammatory illness of the pancreas that manifests as abrupt abdominal pain and elevated blood levels of pancreatic enzymes. It is mostly brought on by the early activation of pancreatic enzymes, which begin breaking down pancreatic tissue and causing inflammation. From minor, self-limiting episodes to serious, lifethreatening sickness, this syndrome can vary widely. The early severity of acute pancreatitis is assessed using imaging studies, lab findings, and clinical presentation. There are two primary categories of acute pancreatitis: necrotising pancreatitis, which involves both inflammation and pancreatic tissue death, and interstitial oedematous pancreatitis, which makes up the majority of cases. Szatmary P et al. (2022).<sup>[1]</sup>

With a markedly increased risk of complications and death, necrotising pancreatitis is a more severe type of the illness. Necrosis of the peripancreatic fat and pancreatic parenchyma are its defining features. Disruption of pancreatic microcirculation causes ischaemia and necrosis of the pancreatic tissue as acute pancreatitis progresses to necrotising pancreatitis. Secondary infection of the necrotic tissues can exacerbate this process further and lead to infected necrotising pancreatitis, which is linked to much greater rates of morbidity and death. Harshit Kumar A et al. (2018).<sup>[2]</sup>

Given the high morbidity and death rate linked to infected necrotising pancreatitis (INP), it is critical to detect this complication in patients with acute pancreatitis (AP) as soon as possible. Infected necrotising pancreatitis, a severe form of acute pancreatitis that greatly increases the risk of systemic complications and death, is characterised by an infection of necrotic pancreatic tissue. The importance for prompt and precise diagnosis of INP is highlighted by the significant differences in clinical care and outcomes between individuals with INP and those with sterile pancreatic necrosis. Poropat G et al. (2022).<sup>[3]</sup>

Proactive monitoring, early introduction of suitable antibiotics, and prompt intervention by endoscopic, percutaneous, or surgical techniques to debride infected necrotic tissue are all made possible by early identification of individuals at high risk for developing INP. These tactics can improve patient outcomes and lower healthcare costs by dramatically lowering the likelihood of systemic consequences, such as organ failure, sepsis, and extended hospitalisation. Chatila AT et al. (2019).<sup>[4]</sup>

The APACHE III scoring system is an enhanced version of the APACHE score designed to assess the severity of disease and predict outcomes in critically ill patients in intensive care unit (ICU) settings. It provides a thorough assessment of a patient's health state by combining a variety of physiological, biochemical, and demographic factors. Data from the first 24 hours after ICU admission, such as age, prior health condition, and 17 acute physiological indicators, are used to construct the score. A high APACHE III score in the setting of acute pancreatitis indicates severe illness, which may be associated with a higher chance of necrotising pancreatitis progression and the possibility of infection inside necrotic pancreatic tissue. Mumtaz H et al. (2023).<sup>[5]</sup> Serum procalcitonin is a biomarker that has gained prominence for its role in detecting bacterial infections, including those complicating acute pancreatitis. Procalcitonin is a precursor of calcitonin that is typically elevated in response to a systemic bacterial infection. Its levels rise significantly in the bloodstream of patients with systemic infections, making it a valuable tool for identifying bacterial complications in acute pancreatitis. When acute pancreatitis is present, elevated serum procalcitonin levels indicate the presence of infection, including infected necrotizing pancreatitis, and can prompt early antibiotic therapy and targeted interventions to manage necrotic tissue. Azzini AM et al.(2020).<sup>[6]</sup>

Serum procalcitonin levels and the APACHE III score are both essential for early detection of acute pancreatitis patients who are at high risk of serious complications like INP. The APACHE III score provides a severity-based risk assessment, while serum procalcitonin offers a specific indication of infection, particularly in necrotizing pancreatitis. By integrating these tools, healthcare providers can achieve a more nuanced understanding of a patient's condition, allowing for stratified care strategies that can improve clinical outcomes by targeting interventions to those most at risk of adverse events. Wiese ML et al. (2022).<sup>[7]</sup>

# **MATERIALS AND METHODS**

**Study Design and Setting:** This prospective observational study was conducted in the Department of General Medicine at D.Y. Patil Medical College, Kolhapur, in 2023. Ethical clearance was obtained from the Institutional Ethics Committee.

#### **Inclusion Criteria**

- Patients diagnosed with acute pancreatitis based on clinical, biochemical, and radiological criteria.
- Age >18 years.
- Admission within 72 hours of symptom onset.

#### Exclusion Criteria

- Chronic pancreatitis.
- Previous pancreatic surgeries.
- Immunosuppressive disorders or current immunosuppressive therapy.

**Sample Size:** A total of 52 patients were included, with 18 classified under the infective group and 34 under the sterile group based on contrast-enhanced computed tomography (CECT) findings and culture results.

**Data Collection:** Clinical evaluation included vitals, Glasgow Coma Scale (GCS), urine output, and organ dysfunction assessment. Laboratory investigations comprised complete blood count, serum amylase, lipase, procalcitonin, creatinine, electrolytes, and arterial blood gases. The APACHE III score was calculated based on 17 physiological parameters within 24 hours of ICU admission (Luo et al., 2019). **Statistical Analysis:** Continuous variables were analyzed using t-tests; categorical variables were compared using chi-square tests. Statistical significance was set at p<0.05. Diagnostic performance of APACHE III and procalcitonin was evaluated using receiver operating characteristic (ROC) curves.

## RESULTS

**Demographic and Clinical Characteristics:** The age distribution was not significantly different between groups (p=0.13). The male-to-female ratio was comparable. Most patients in the infective group were above 40 years.

## **Biochemical Parameters**

- Serum procalcitonin levels were significantly higher in the infective group (Mean: 2.31 ng/mL) compared to the sterile group (Mean: 0.48 ng/mL), with p<0.0001.
- Elevated creatinine, potassium, and BUN levels were also observed in the infective group.

#### **Physiological Parameters:**

- The infective group exhibited significantly lower urine output (1211 mL vs. 2241 mL; p<0.001), lower MAP, and higher respiratory rate.
- No significant differences were noted in temperature or heart rate.

**APACHE III Scores:** The mean APACHE III score was markedly higher in the infective group (50.89) than in the sterile group (28.68), with p<0.001. ROC analysis revealed an AUC of 0.86 for APACHE III in predicting INP.



Figure 1: COMPARISION OF PROCALCITONIN BETWEEN INFECTIVE AND STERILE GROUP



Figure 2: COMPARISION OF APACHE III SCORE BETWEEN STERILE AND INFECTIVE GROUP



Figure 3: URINE OUTPUT COMPARISION BETWEEN INFECTIVE AND STERILE GROUP



Figure 4: COMPARISION OF MEAN ARTERIAL PRESSURE BETWEEN INFECTIVE AND STERILE GROUP

Table 1: Serum Procalcitonin						
Variable	CECT Abdomen	Mean	Std. Deviation	t test	p value	
Serum Procalcitonin	Infective	2.3156	1.71696	5.899	0.000	
	Sterile	0.4771	0.46221			

Table 1 presents an important discovery: With a p-value of 0.000, which indicates strong statistical significance, the Infective group's serum procalcitonin level is significantly greater (2.3156) than that of the Sterile group (0.4771). This implies that procalcitonin may be a crucial indicator for differentiating between sterile and infectious situations.

Table 2: APACHE III Score Comparison Between Groups						
Variable	CECT Abdomen	Mean	Std. Deviation	t test	p value	
APACHE III Score	Infective	50.889	21.2987	4.024	0.000	
	Sterile	28.676	17.5995			

The APACHE III scores, which are used to gauge a patient's illness severity, show notable variations in this table. With a t-test value of 4.024 and a significant p-value of 0.000, the average score of the Infective group is significantly greater (50.889) than that of the Sterile group (28.676). This implies that the condition is more severe in the infectious group, which may be important for clinical evaluations and treatments.

Table 3: Urine Output (UO)						
Variable	CECT Abdomen	Mean	Std. Deviation	t test	p value	
UO	Infective	1211.111	753.7271	-5.323	0.000	
	Sterile	2241.176	612.4307			

The groups' urine production differs significantly from one another, with the Sterile group showing much higher output (2241.176) compared to the Infective group (1211.111). The negative t-test value (-5.323) and a p-value of 0.000 confirm the substantial divergence in this physiological measure, possibly reflecting differing fluid management or renal responses in clinical settings.

Table 4: Mean Arterial Pressure (MAP)						
Variable	CECT Abdomen	Mean	Std. Deviation	t test	p value	
MAP	Infective	78.889	10.1859	-2.059	0.045	
	Sterile	85.088	10.3992			

Table 4 examines MAP, where the Sterile group presents a higher mean MAP (85.088) compared to the Infective group (78.889). The negative t-test value (-2.059) and a p-value of 0.045 suggest that this is a statistically significant difference, indicating potentially higher arterial pressure in Sterile conditions.

## DISCUSSION

In contrast to the traditional enzymes, the analysis of serum procalcitonin levels, detailed in Table 1, provides a compelling argument for its role as a discriminative biomarker in pancreatitis. The mean serum procalcitonin concentration was significantly higher in patients with infective pancreatitis compared to those with sterile inflammation, with a p-value less than 0.001, indicating strong statistical significance. This marked difference supports the hypothesis that serum procalcitonin can serve as a valuable tool for detecting bacterial infections complicating pancreatitis. Procalcitonin, a precursor of the hormone calcitonin, is produced in response to systemic bacterial infections and sepsis and is recognized for its utility increasingly in differentiating infectious from non-infectious inflammatory states. Rompianesi G et al. (2017).<sup>[8]</sup> In contrast to hepatic failure, Table 2 provides compelling evidence on the disparity in disease severity as measured by the APACHE III scoring system between infective and sterile pancreatitis patients. The mean APACHE III score in the infective group was 50.889, substantially higher than the 28.676 mean score observed in the sterile group, with the difference achieving high statistical significance (p-value <0.001). This pronounced disparity indicates that patients with infective pancreatitis experience markedly greater systemic illness severity and are at higher risk for adverse clinical outcomes.

The APACHE III (Acute Physiology and Chronic Health Evaluation III) score is a widely validated and comprehensive tool used to assess the severity of critical illness in intensive care settings. It integrates physiological variables, chronic health conditions, and patient age to generate a prognostic score correlating with mortality risk and disease burden. Di MY et al. (2016),<sup>[9]</sup> underscored the utility of APACHE III scoring in the context of acute pancreatitis, demonstrating that elevated scores are predictive of complicated disease courses, increased need for intensive interventions, and higher mortality rates. Their study advocated for the incorporation of APACHE III in early assessment protocols to guide clinical decision-making and resource allocation.

The most striking and clinically relevant observation is reported in Table 3, where urine output, an important marker of renal function and systemic perfusion, differed significantly between groups. Infective pancreatitis patients had a mean urine output of 1,211.111 mL/day, markedly lower than the 2,241.176 mL/day observed in the sterile group, with a highly significant p-value (<0.001). This pronounced reduction in urine output in the infective group strongly suggests greater renal compromise and systemic involvement. Zahariev OJ et al. (2024),<sup>[10]</sup> emphasized the prognostic value of urine output in acute pancreatitis, highlighting its utility as a non-invasive, readily measurable parameter reflective of intravascular volume status, renal perfusion, and overall disease severity. Oliguria or reduced urine output is a well-recognized indicator of acute kidney injury (AKI), which frequently complicates severe pancreatitis, particularly when infection or sepsis ensues. The pathophysiology involves a combination of factors including systemic inflammatory response, hypotension, microcirculatory dysfunction, and direct nephrotoxic effects of inflammatory mediators and toxins. Reduced urine output signifies not only renal dysfunction but also serves as a proxy for multi-organ failure risk, which substantially increases morbidity and mortality in pancreatitis patients.

In contrast to the minimal variation seen in body temperature, Table 4 reveals a statistically significant difference in mean arterial pressure (MAP) between the infective and sterile pancreatitis groups. Patients with infective pancreatitis had a mean MAP of 78.889 mmHg, notably lower than the 85.088 mmHg recorded in the sterile group, with this difference achieving statistical significance (p=0.045). The lower MAP in infective pancreatitis patients may reflect hemodynamic compromise frequently associated with systemic inflammatory responses and septic states. Shawl SH et al. (2022),<sup>[11]</sup> emphasized the clinical relevance of MAP monitoring in pancreatitis, highlighting its utility in early detection of circulatory dysfunction and guiding resuscitative efforts. Their research suggested that decreased MAP could serve as a harbinger of disease severity and impending organ dysfunction, particularly in the setting of infected pancreatic necrosis or sepsis.

# CONCLUSION

This study's primary focus was on the utilization of the APACHE III scoring system and serum procalcitonin levels as predictive tools. Through meticulous data collection and statistical analysis, this research has offered significant insights into the management and prognosis of acute pancreatitis, especially in determining the risk of infection which complicates the clinical course and often necessitates a different therapeutic approach. The findings from this thesis underscore the complexity of diagnosing and managing acute pancreatitis, particularly when infection is involved. Serum procalcitonin has emerged as a particularly valuable biomarker in this study. Elevated procalcitonin levels were significantly associated with infected necrotizing pancreatitis, reinforcing the biomarker's utility in identifying bacterial infections. This is consistent with existing literature that suggests serum procalcitonin as a reliable indicator of bacterial infection, which can guide clinicians in timely and appropriate antibiotic administration. Similarly, the APACHE III score, which evaluates both physiological and laboratory parameters to assess disease severity, has shown significant predictive capability in this study. Infectious necrotising pancreatitis was more common in patients with higher APACHE III scores. This finding is pivotal as it validates the APACHE III score not only as a severity marker but also as a prognostic tool that can aid clinicians in predicting the course of the disease, facilitating early intensive care interventions and potentially improving outcomes.

#### Limitations

There are a number of restrictions on this study. First off, the findings' ability to be applied broadly is restricted by the single-center design. Results may be impacted by differences in ICU procedures, clinical care, and patient demographics. Second, the sample size (n=52) was small, which might have limited the statistical power and raised the possibility of type II errors. Thirdly, procalcitonin levels were only assessed once instead of repeatedly, which would have revealed information on how an infection developed over time and how well treatment worked. Fourth, without routine microbiological culture confirmation, infection identification depended on imaging and indirect clinical characteristics, potentially resulting in an under- or over-diagnosis of INP. Lastly, the research did not include additional potentially helpful biomarkers that may have supplemented the prediction model, such as interleukins or CRP. It is advised to do multicenter trials in the future with bigger cohorts, dynamic biomarker tracking, and microbiological validation.

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