**Int. J Med. Pharm. Res.**
**E-ISSN**: 2958-3683 | **P-ISSN**: 2958-3675
**Available on**: https://ijmpr.in/

International Journal of
**Medical and Pharmaceutical Research**

**ORGINAL ARTICLE** **OPEN ACCESS**

# To Study the Role of Procalcitonin in Acute Pancreatitis Severity Prediction in Patients Attending a Tertiary Care Centre

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**OPEN ACCESS**

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*Received: 25-06-2025*
*Accepted: 27-07-2025*
*Available Online: 17-08-2025*



## A B S T R A C T

**Background:** Acute pancreatitis (AP) is a highly variable inflammatory disorder with clinical presentations ranging from mild, self-limiting illness to severe disease associated with systemic inflammatory response syndrome (SIRS), infected pancreatic necrosis, and multi-organ failure. Early stratification of severity is crucial for timely intervention.

**Aim and Objective:** To evaluate the diagnostic and prognostic significance of serum procalcitonin (PCT) levels in predicting the severity of acute pancreatitis in patients admitted to a tertiary care centre**.**

**Materials and Methods:** A case-control study was conducted on 100 patients, including 50 diagnosed cases of acute pancreatitis and 50 healthy controls. Serum procalcitonin levels were measured within 24 hours of hospital admission. The severity of AP was assessed using the Revised Atlanta Classification. Microbiological samples were collected for culture and sensitivity testing. Associations between PCT levels, severity markers, and microbial isolates were analyzed using statistical tools.

**Results:** In the present study among 50 AP cases, 34% had isolates of *Acinetobacter baumannii*, followed by *E. coli* (24%), *Klebsiella pneumoniae* (22%), and *Pseudomonas aeruginosa* (20%). Coinfection with *A. baumannii* and *P. aeruginosa* was found in 16% of patients. PCT levels were significantly elevated in patients with infected necrosis, systemic complications, and those requiring ICU admission. PCT >0.5 ng/mL was significantly associated with severe AP and multiorgan failure ($p<0.05$).

**Conclusion:** Serum procalcitonin is a reliable biomarker for early prediction of AP severity and infection-related complications. It may supplement clinical scores for better risk stratification and management.

**KEYWORDS:** Procalcitonin, Acute Pancreatitis, Serum Procalcitonin, Pct, Severity Markers

## INTRODUCTION

Acute pancreatitis (AP) is a potentially life-threatening inflammatory condition of the pancreas that presents with a wide clinical spectrum ranging from a mild, self-limiting illness to a severe form associated with systemic inflammatory response syndrome (SIRS), multi-organ dysfunction, and significant mortality. The annual incidence of AP varies globally, ranging from 13 to 45 cases per 100,000 population, and appears to be rising in many countries due to factors like gallstones, alcohol abuse, hypertriglyceridemia, and increasing obesity prevalence [1,2].

The clinical severity of AP is unpredictable during the early course of the disease. Around 15–20% of patients develop severe AP (SAP), which is associated with high morbidity and mortality [3]. Early identification of patients likely to progress to SAP is therefore crucial to initiate timely aggressive interventions, optimize resource allocation, and improve outcomes [4].

Over the years, a variety of clinical scoring systems (e.g., Ranson's score, APACHE II, BISAP), imaging modalities (e.g., contrast-enhanced CT), and biomarkers have been explored to assess the severity and predict complications in AP [5,6]. Among biochemical markers, serum C-reactive protein (CRP), interleukins, and trypsinogen activation peptide have been studied extensively. However, these have limitations, particularly with regard to timing, specificity, and accessibility [7].

Procalcitonin (PCT), a 116-amino acid precursor of the hormone calcitonin, is synthesized in response to systemic inflammation, particularly bacterial infections. In non-infectious inflammation, PCT levels remain low. This unique behavior has led to investigations into its role in various inflammatory and septic conditions, including AP [8].

Under normal physiological conditions, PCT is produced in the C-cells of the thyroid gland. However, in systemic inflammation, its expression is upregulated in multiple tissues, particularly in response to bacterial endotoxins and pro-inflammatory cytokines like IL-1β, IL-6, and TNF-α [9]. PCT rises earlier than CRP and may provide a real-time estimate of systemic inflammatory burden. It peaks within 6–24 hours of insult and has a half-life of about 24 hours, making it a dynamic and responsive biomarker [10].

In the context of AP, elevated PCT levels may reflect either severe sterile inflammation or early infection of pancreatic necrosis. Studies have shown a correlation between elevated PCT levels and severity of AP as defined by Atlanta classification [11]. PCT levels >0.5 ng/mL have been associated with increased risk of infected pancreatic necrosis, multiorgan dysfunction, and higher mortality [12].

Risk stratification at admission is essential to decide the level of monitoring and intensity of treatment required. While scoring systems like Ranson's or APACHE-II are helpful, they have limitations due to complexity, delayed applicability, or requirement for multiple parameters over 48 hours [13]. In contrast, biomarkers like PCT offer the advantage of early, single-time-point assessment and can be repeated during the hospital course.

The revised Atlanta classification defines AP severity into mild (no organ failure), moderately severe (transient organ failure or local complications), and severe (persistent organ failure) [14]. Identifying severe and moderately severe cases early helps guide ICU admission, antibiotic stewardship, and aggressive supportive care.

Despite promising findings, PCT has not yet been universally adopted as a standard marker for AP severity. Variability in cut-off values, timing of sample collection, and small sample sizes in studies have limited its widespread use. Additionally, PCT can be elevated in other conditions such as trauma, surgery, and burns, potentially confounding its specificity [15].

Nevertheless, PCT remains one of the most promising single biomarkers, with potential use in combination with clinical scores to enhance prognostic accuracy in AP [16]. Furthermore, serial measurement of PCT may provide dynamic insight into disease progression and therapeutic response . Therefore the present study was undertaken to study the role of procalcitonin in acute pancreatitis severity prediction in patients attending a tertiary care centre

## MATERIALS AND METHODS

A prospective, hospital-based, case-control study was conducted over a period of 12 months in a tertiary care center in the Department of Microbiology. A total of 100 individuals were enrolled, including 50 confirmed cases of acute pancreatitis and 50 age- and gender-matched healthy controls.

**Inclusion criteria for cases:**

1. Patients ≥18 years old.
2. Diagnosed with acute pancreatitis as per the Revised Atlanta Criteria.
3. Presented within 72 hours of symptom onset.

**Exclusion criteria:**

1. Chronic pancreatitis.
2. Recent major surgery or trauma.
3. Known malignancy, burns, or other systemic infections.

**Data Collection:** Detailed history, clinical findings, and laboratory investigations were recorded. Serum PCT levels were measured using a standardized chemiluminescence assay within 24 hours of admission. The severity of AP was categorized as mild, moderately severe, or severe.

**Microbiology:** Specimens (TT tube, blood, tracheal aspirate, CVP line) were collected aseptically and processed using standard culture techniques to identify bacterial isolates. Antibiotic susceptibility was tested.

Microbiology: Specimens (tracheostomy tube [TT tube], blood, tracheal aspirate [TA], and central venous pressure [CVP] line) were collected aseptically from patients and immediately transported to the microbiology laboratory for processing. Samples were inoculated on appropriate culture media, including blood agar, MacConkey agar, and chocolate agar, and incubated under aerobic conditions at 35–37°C for 18–24 hours. Bacterial growth was identified based on colony morphology, Gram staining, and biochemical tests. For precise species identification, isolates were analyzed using Matrix-Assisted Laser Desorption Ionization–Time of Flight Mass Spectrometry (MALDI-TOF MS) (Bruker Daltonics, Germany) according to the manufacturer's protocol. Antimicrobial susceptibility testing (AST) was performed using the Kirby–Bauer disk diffusion method as per Clinical and Laboratory Standards Institute (CLSI) guidelines. Quality control strains (Escherichia coli ATCC 25922, Pseudomonas aeruginosa ATCC 27853, and Staphylococcus aureus ATCC 25923) were used for validation of procedures [17].

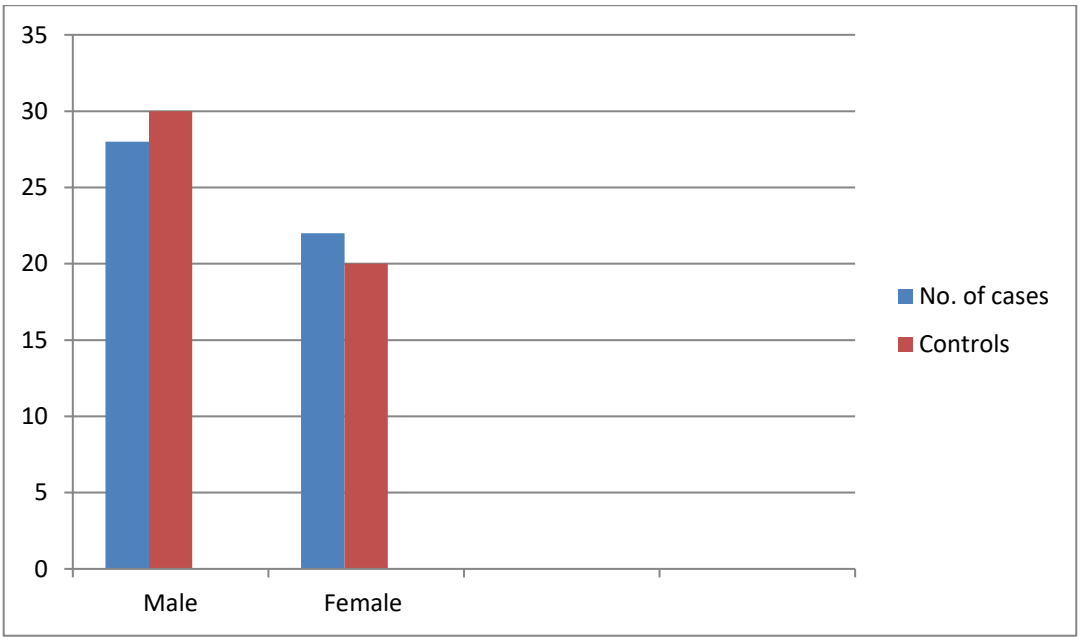
**Statistical Analysis:** Data were analyzed using SPSS software. Chi-square and t-tests were used to evaluate the association between variables. A p-value <0.05 was considered statistically significant.

**RESULTS**

In the present study among 50 AP cases, 34% had isolates of *Acinetobacter baumannii*, followed by *E. coli* (24%), *Klebsiella pneumoniae* (22%), and *Pseudomonas aeruginosa* (20%). Coinfection with *A. baumannii* and *P. aeruginosa* was found in 16% of patients. PCT levels were significantly elevated in patients with infected necrosis, systemic complications, and those requiring ICU admission. PCT >0.5 ng/mL was significantly associated with severe AP and multiorgan failure (p<0.05).

**Table No. 1: Genderwise distribution (N=100)**

| Gender | Cases (n=50) | Controls( n=50) | Total (N=100) | P value |
|---|---|---|---|---|
| Male | 28 (56%) | 30 (60%) | 58(58%) | |
| Female | 22 (44%) | 20 (40%) | 42 (42%) | 0.68 |




**Graph No. 1: Graphical Representation of Genderwise distribution (N=100)**

A total of 100 participants were included in the study, consisting of 50 cases and 50 controls. Among the cases, 28 (56%) were males and 22 (44%) were females, whereas the control group included 30 (60%) males and 20 (40%) females. The gender distribution was statistically not significant between the groups (p = 0.68).

**Table No. 2: Agewise distribution (N=100)**

| Age Group ( Years) | Cases (n=50) | Controls (n=50) | Total (N=100) |
|---|---|---|---|
| ≤30 | 5 (10%) | 7 (14%) | 12 (12%) |
| 31-40 | 9(18%) | 10 (20%) | 19 (19%) |
| 41-50 | 14 (28%) | 12(24%) | 26 (26%) |
| 51-60 | 13(26%) | 14 (28%) | 27 (27%) |
| >60 | 9 (18%) | 7 (14%) | 16 (16%) |

Age-wise distribution showed that the most common age group among both cases and controls was 51–60 years, comprising 13 (26%) of cases and 14 (28%) of controls. The next most prevalent age group was 41–50 years with 14 (28%) cases and 12 (24%) controls. Other age groups were less represented. There was no statistically significant difference in age distribution between cases and controls (p = 0.89).

**Table No. 3: Type of Isolates**

| Type of Isolates | No. of Isolates | Percentage | P value |
|---|---|---|---|
| *A. baumanni* | 17 | (34%) | 0.89 |
| *E.coli* | 12 | (24%) | |
| *Klebsiella pneumoniae* | 11 | (22%) | |
| *Pseudomonas. aerogenosa* | 10 | (20%) | |

Among the isolates obtained from the cases, Acinetobacter baumannii was the most common organism identified, found in 17 (34%) isolates, followed by E. coli in 12 (24%), Klebsiella pneumoniae in 11 (22%), and Pseudomonas aeruginosa in 10 (20%). Coinfection with A. baumannii and P. aeruginosa was seen in 8 (16%) of the cases.

**Table No. 4: Number of cases with co infection**

| Type of Isolates | No. of Isolates Coinfection | Percentage |
|---|---|---|
| *Acinetobacter baumannii* and *Pseudomonasaerogenosa* | 8 | (16%) |

**Table No. 5: Type of Isolates**

| Type of Specimen | No. of Isolates | Percentage |
|---|---|---|
| TT Tube | 15 | (30%) |
| Blood | 14 | (28%) |
| CVP | 10 | (20%) |
| TA | 11 | (55%) |

Regarding specimen type, the highest number of isolates were from TT (Tracheostomy Tube) samples (15; 30%), followed by blood (14; 28%), TA (Tracheal Aspirate) (11; 22%), and CVP (Central Venous Pressure) lines (10; 20%).

**Table No. 6: Risk Factors/Comorbidity**

| Risk Factors/Comorbidity | Cases (n=50) | Controls (n=50) | Total (N=100) |
|---|---|---|---|
| Diabetes Mellitus | 20 (40%) | 15 (30%) | 35 (35%) |
| Hypertension | 16(32%) | 12 (24%) | 28 (28%) |
| Chronic Liver Disease | 5 (10%) | 3(6%) | 8 (8%) |
| Chronic Kidney Disease | 6(12%) | 2(4%) | 8 (8%) |
| COPD | 3 (6%) | 4 (8%) | 7 (7%) |
| Co Comorbidity | 10 (20%) | 20 (40%) | 30 (30%) |
| | | | |

In terms of comorbid conditions, diabetes mellitus was present in 20 (40%) of cases and 15 (30%) of controls, a difference that was not statistically significant (p = 0.29). Hypertension was observed in 16 (32%) of cases and 12 (24%) of controls (p = 0.38). Other risk factors included chronic liver disease (10% vs 6%, p = 0.45), chronic kidney disease

(12% vs 4%, p = 0.14), and COPD (6% vs 8%, p = 0.69). Interestingly, co-comorbidity (presence of multiple risk factors) was higher among controls (40%) than cases (20%), which was statistically significant (p = 0.03).

In the present study, among 50 AP cases, 34% had isolates of *Acinetobacter baumannii*, followed by *E. coli* (24%), *Klebsiella pneumoniae* (22%), and *Pseudomonas aeruginosa* (20%). Coinfection with *A. baumannii* and *P. aeruginosa* was found in 16% of patients.
Serum procalcitonin (PCT) levels in AP cases ranged from 0.12 ng/mL to 9.8 ng/mL, with a mean value of 2.45 ± 1.18 ng/mL, significantly higher than in controls (0.05 ± 0.02 ng/mL, p<0.001). In severe AP, mean PCT levels were 3.72 ± 1.05 ng/mL, compared to 1.26 ± 0.64 ng/mL in mild to moderately severe AP. PCT >0.5 ng/mL was present in 82% of severe AP patients and was significantly associated with infected necrosis, systemic complications, multiorgan failure, and ICU admission (p<0.05).

**DISCUSSION**

In the present study, elevated serum procalcitonin (PCT) levels were strongly associated with the severity of acute pancreatitis (AP), including infected pancreatic necrosis, organ failure, and ICU admissions. Our findings are consistent with several earlier studies that have established PCT as a reliable early biomarker for stratifying the severity of AP.
A study by Rau et al. found that PCT levels >0.5 ng/mL could predict infected necrosis with 93% sensitivity and 87% specificity, suggesting a strong diagnostic value of PCT in early-stage AP complications. Similarly, Mofidi et al. demonstrated that serum PCT levels correlated significantly with APACHE II scores, CRP, and organ dysfunction [18].

Chen et al. highlighted the diagnostic value of PCT in predicting persistent organ failure in AP patients, with elevated levels correlating with prolonged ICU stays and mortality. Another prospective study by Sharma et al. showed that PCT was superior to CRP and IL-6 in assessing severity during the early course of AP [19].

Alempijevic et al. also demonstrated that high PCT levels, along with IL-6 and TNF-alpha, could serve as early predictors of severe AP and systemic complications. Hatzistilianou emphasized that PCT can aid not only in the diagnosis of sepsis but also in guiding therapeutic decisions in AP management [20].

Harbarth et al. evaluated critically ill patients with suspected sepsis and reported that PCT, along with IL-6 and IL-8, provided valuable prognostic information in distinguishing bacterial infections, which is relevant in AP patients with infected necrosis [21].

Talukdar and Vege proposed early management protocols for severe AP, emphasizing the use of reliable markers like PCT to aid timely triaging and avoid overtreatment in mild cases. A study by Assicot et al. originally demonstrated elevated PCT levels in systemic bacterial infections, laying the groundwork for its application in other inflammatory conditions including AP [22].

A study by Meisner explored the biochemical basis of PCT and confirmed that it rises earlier than CRP and has a better correlation with inflammatory response magnitude, supporting its role as a dynamic biomarker in AP. Becker et al. further elucidated the pathophysiology of PCT, linking its increase to cytokine-driven expression in extra-thyroidal tissues during systemic infections [23].

Papachristou et al. compared scoring systems and noted that combining these with biochemical markers like PCT could enhance predictive accuracy for mortality and complications in AP. Khanna et al. also suggested that while scores like Ranson, APACHE II, and BISAP have value, real-time markers such as PCT can provide immediate guidance at admission [24,25].

Yadav and Lowenfels underscored the growing incidence of AP globally, increasing the need for efficient severity prediction models like those incorporating PCT. Roberts et al. highlighted regional variations in AP etiology and incidence, reaffirming the value of universal biomarkers like PCT across different populations [1].

Vege et al. stressed early intervention in AP to reduce morbidity and mortality, advocating for tools like PCT in clinical practice to recognize severe cases early. Tenner et al., in the American College of Gastroenterology guidelines, acknowledged the role of serum markers such as PCT in guiding antibiotic use in complicated AP [26].

Rana et al. [27]demonstrated that combining PCT with imaging improved the diagnostic precision in necrotizing pancreatitis, aiding early therapeutic decisions. Chen and colleagues reiterated the role of PCT in differentiating between sterile and infected necrosis, an essential aspect of management strategies.

Collectively, these studies underscore the utility of PCT in the early prediction of AP severity, enabling better patient stratification, resource allocation, and therapeutic planning.

Infections are still one of the most common complications of cancer management. Infection in cancer patients is accompanied with increased morbidity and mortality as well as delayed treatment regimens, prolonged hospitalization, and increased financial burden of health care . Blood stream infections (BSIs) are the most life-threatening conditions among other causes of infections . This is especially dangerous in the era of antimicrobial resistance (AMR) [28].

## CONCLUSION

Procalcitonin holds significant potential as an early predictor of severity in acute pancreatitis. Its ability to rapidly reflect the systemic inflammatory burden makes it a valuable tool in early risk stratification. Integration of PCT into clinical protocols may aid in improving patient outcomes by guiding early intensive care, antibiotic use, and prognostication.

## DECLARATIONS

**Conflicts of interest:** There is no any conflict of interest associated with this study
**Consent to participate:** There is consent to participate.
**Consent for publication:** There is consent for the publication of this paper.
**Authors' contributions:** Author equally contributed the work.

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