

MARKED PROCALCITONIN ELEVATION IN CULTURE-NEGATIVE HOSPITALIZED PATIENTS: CLINICAL SPECTRUM AND DIAGNOSTIC IMPLICATIONS FROM A RETROSPECTIVE OBSERVATIONAL STUDY

Clinical Microbiology

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ABSTRACT

Background Procalcitonin (PCT) is widely used as a biomarker of bacterial sepsis and to guide antimicrobial therapy. However, elevated PCT levels have increasingly been reported in non-bacterial inflammatory and critical illness states, which may lead to diagnostic confusion and unnecessary antibiotic use. We evaluated the magnitude and clinical correlates of elevated PCT in culture-negative hospitalized patients.

Objectives To identify the non-bacterial conditions associated with markedly elevated PCT and characterize the extent of PCT elevation in the absence of microbiological evidence of bacterial infection. **Methods** In this retrospective observational study at a tertiary care center in Northeast India, we reviewed hospitalized patients with elevated serum PCT, including demographic details, clinical diagnoses, microbiological culture results, and laboratory parameters. Patients with culture reports documented as negative, or contaminated were classified as culture-negative. PCT levels were analyzed descriptively and associated non-bacterial conditions were identified from medical records. **Results** The study was conducted for a period of 6 months from July 2025 to December 2025, 140 hospitalized patients with elevated serum procalcitonin (PCT), 96 (68.6%) were culture-negative, and quantitative PCT values were available for 93 patients. Serum PCT concentrations were markedly elevated despite the absence of microbiological evidence of bacterial infection, with a median value of 45.65 ng/mL (interquartile range [IQR] 21.3-90.8 ng/mL) and a mean value of 53.05 ng/mL. Marked PCT elevation (>10 ng/mL) was observed in 88 of 96 culture-negative patients (91.7%). Elevated PCT levels were associated with a broad spectrum of non-bacterial conditions, most commonly chronic kidney disease and dialysis-dependent states, cardiac disorders and shock, postoperative and trauma-related conditions, neurological insults, severe viral infections, malignancy, pancreatitis, autoimmune disorders, and drug-related reactions. Serum PCT concentrations showed substantial overlap between culture-negative and culture-positive patients, with median values of 51.05 ng/mL and 62.70 ng/mL, respectively, and no statistically significant difference between groups (Mann-Whitney U = 1859.0, $p = 0.280$). These findings demonstrate that markedly elevated PCT levels may occur in diverse non-bacterial inflammatory and critical illness states. **Conclusion** Marked PCT elevation occurs across a wide range of non-bacterial conditions, particularly in critically ill patients with severe systemic inflammation, organ failure, or tissue ischemia. PCT should not be used in isolation to diagnose bacterial infection, its interpretation requires integration with clinical assessment and microbiological findings to avoid unnecessary antibiotic exposure.

KEYWORDS

Antimicrobial Stewardship; Biomarkers; Chronic Kidney Disease; Critical Illness; Culture-negative Sepsis; Non-bacterial Inflammation; Procalcitonin; Systemic Inflammatory Response Syndrome

INTRODUCTION

Procalcitonin (PCT), the prohormone of calcitonin, is widely used as a biomarker for the diagnosis of bacterial infection and to guide antimicrobial therapy, particularly in patients with suspected sepsis [1,2]. Circulating PCT is negligible under physiological conditions; however, during systemic bacterial infection it is produced extrathyroidally by multiple tissues in response to pro-inflammatory cytokines and bacterial endotoxin [3]. Its relatively rapid rise and correlation with disease severity have made PCT a prominent tool for the early detection of sepsis and for antibiotic stewardship in emergency and critical care settings [4].

Despite this widespread use, accumulating evidence indicates that elevated PCT is not specific to bacterial infection. Marked elevations have been reported after major surgery and trauma, and in cardiogenic shock, acute coronary syndromes, chronic kidney disease, malignancy, severe viral infection, autoimmune disease, and other systemic inflammatory states [5,6]. In critically ill patients, these non-infectious triggers can produce a systemic inflammatory response syndrome (SIRS) with cytokine release patterns resembling those of bacterial sepsis, resulting in substantial PCT elevation even without microbiologically proven infection [2,5].

This lack of specificity poses a clinically important challenge. Over-reliance on PCT values without adequate clinical correlation or microbiological confirmation may lead to misdiagnosis of bacterial sepsis and to unnecessary initiation or prolongation of broad-spectrum antibiotic therapy, with attendant risk of antimicrobial resistance [7,8]. This concern is particularly relevant in patients with chronic kidney disease, multiorgan dysfunction, postoperative stress, or malignancy, in whom baseline inflammation and impaired clearance may further confound interpretation.

Data on the magnitude and clinical contexts of PCT elevation in culture-negative hospitalized patients remain limited, particularly from low- and middle-income countries. Characterizing the spectrum

of non-bacterial conditions associated with elevated PCT is important for improving diagnostic accuracy and antimicrobial stewardship. We therefore analyzed PCT levels in culture-negative hospitalized patients at a tertiary care center and identified the non-bacterial clinical conditions associated with marked PCT elevation.

Materials and Methods Study Design and Setting

This retrospective observational study was conducted at a tertiary care teaching hospital in Northeast India. We analyzed hospitalized patients in whom serum PCT testing was performed as part of routine clinical evaluation during the study period.

Study Population

The study was conducted for a period of 6 months from July 2025 to December 2025 and total of 140 hospitalized patients with elevated serum procalcitonin levels were included in the study. Patients of all age groups and both sexes were eligible. Clinical records, laboratory data, and microbiological reports were reviewed.

Patients with elevated serum procalcitonin with corresponding microbiological culture results, including blood, urine, respiratory, or other clinically indicated specimens, were available for review. Patients whose culture reports were documented as negative growth, no growth, or contamination were classified as culture-negative and included in the final analysis.

Patients with confirmed bacterial growth on culture were excluded from the primary analysis of non-bacterial causes of PCT elevation.

Data Collection

Data were retrospectively retrieved from hospital laboratory records and medical case files. Information collected included demographic characteristics such as age and sex, clinical diagnoses and relevant comorbidities, admission specialty, microbiological culture findings, laboratory parameters including serum procalcitonin, C-reactive

protein, total leukocyte count, thyroid-stimulating hormone, and lactate dehydrogenase where available, together with documented clinical outcomes including survival status. Clinical diagnoses were subsequently reviewed to identify underlying non-bacterial conditions associated with systemic inflammation.

Procalcitonin Measurement

Serum PCT was measured using a standardized automated immunoassay routinely employed in the hospital laboratory and reported in nanograms per milliliter (ng/mL). Values exceeding the upper measurable limit were recorded as the maximum reported value.

Microbiological Evaluation

Microbiological cultures were performed according to standard hospital protocols using specimens appropriate to the clinical indication. Results reported as negative growth, nil growth, or contamination were considered culture-negative for the purposes of this study.

Definitions

Variable	Definition
Culture-negative patient	Absence of microbiological evidence of bacterial infection based on available culture reports
Markedly elevated procalcitonin	Serum PCT >10 ng/mL
Non-bacterial cause	Clinical condition associated with systemic inflammation without confirmed bacterial infection

RESULTS

Study Population

A total of 140 hospitalized patients with elevated serum procalcitonin (PCT) levels were screened during the study period. Based on microbiological findings, 96 patients (68.6%) were classified as culture-negative, with culture reports documented as negative growth, no growth, or contamination. Quantitative PCT values were available for 93 of these patients, who were included in the detailed analysis.

The study cohort comprised patients from multiple clinical specialties, including internal medicine, nephrology, cardiology, critical care, surgery, and pediatrics, reflecting the diverse clinical settings in which elevated PCT levels were encountered despite the absence of microbiologically confirmed bacterial infection.

Figure 1. Flow Diagram of patient selection

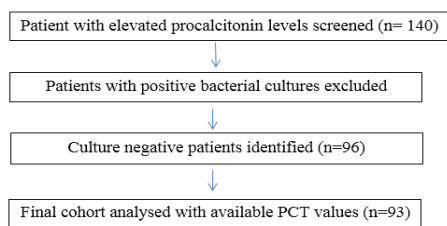


Figure 1. Flow diagram of patient selection and classification, including screening, exclusion of culture-positive cases, and final inclusion of culture-negative patients for analysis.

Procalcitonin Levels in Culture-Negative Patients

Marked elevations in serum PCT were observed among culture-negative patients. Among the 93 patients with quantitative results, the median PCT concentration was 45.65 ng/mL (interquartile range [IQR], 21.3-90.8 ng/mL), while the mean concentration was 53.05 ng/mL. PCT values ranged from 7.6 ng/mL to ≥ 100 ng/mL, the upper reporting limit of the assay. Median PCT levels across major non-bacterial clinical categories are shown in Table 1.

Overall, 88 of 96 culture-negative patients (91.7%) demonstrated markedly elevated PCT concentrations (>10 ng/mL), indicating that values typically associated with severe bacterial infection were frequently observed despite the absence of microbiological evidence of infection.

Figure 3. shows the wide distribution and substantial overlap of PCT levels across non-bacterial clinical categories, including chronic kidney disease, cardiac, postoperative, and systemic inflammatory states.

Comparison of culture-negative and culture-positive groups demonstrated substantial overlap in PCT concentrations. The median PCT concentration was 51.05 ng/mL among culture-negative patients and 62.70 ng/mL among culture-positive patients. This difference was not statistically significant (Mann-Whitney U = 1859.0, $p = 0.280$), suggesting limited discriminatory ability of isolated PCT values for distinguishing bacterial from non-bacterial causes of systemic inflammation.

Clinical Conditions Associated with Elevated Procalcitonin

Review of clinical records identified a broad spectrum of non-bacterial conditions associated with elevated PCT levels. Chronic kidney disease (CKD), including dialysis-dependent states and acute-on-chronic kidney injury, represented the most common category and demonstrated the highest median PCT concentration among major diagnostic groups (63.6 ng/mL).

Elevated PCT concentrations were also frequently observed in patients with cardiovascular disorders, including acute coronary syndrome, cardiogenic shock, acute left ventricular failure, and post-cardiac surgery states. Similarly, substantial elevations were identified in postoperative patients and those with major trauma, indicating that tissue injury and physiological stress can produce marked increases in circulating PCT.

Neurological disorders, including ischemic stroke, intracerebral hemorrhage, hypoxic-ischemic encephalopathy, and osmotic demyelination syndrome, were associated with elevated PCT concentrations despite negative microbiological cultures. Additional non-bacterial conditions included severe viral respiratory infections, malignancy and chemotherapy-related complications, acute pancreatitis, hepatobiliary disease, autoimmune and hyperinflammatory disorders, and severe drug-related hypersensitivity reactions.

Marked elevations were observed across all clinical categories, with several patients demonstrating PCT concentrations ≥ 100 ng/mL. Particularly notable were patients with anaphylaxis and severe drug reactions, where PCT levels reached values commonly encountered in septic shock.

Associated Laboratory Parameters

Elevated PCT concentrations were frequently accompanied by increased inflammatory markers, including C-reactive protein and total leukocyte count. However, discordance between biomarkers was also observed. Several patients demonstrated markedly elevated PCT concentrations despite normal or only mildly elevated leukocyte counts and the absence of microbiological confirmation of bacterial infection. These findings further emphasize the limited specificity of PCT when interpreted in isolation.

Clinical Outcomes

Many culture-negative patients with markedly elevated PCT levels presented with severe systemic illness characterized by systemic inflammatory response syndrome (SIRS), shock, or multiorgan dysfunction. Mortality was documented across several diagnostic categories; however, because outcome data were analyzed descriptively, no association between PCT concentration and clinical outcome was assessed.

Table 1. Non-bacterial Clinical Categories Associated with Elevated Procalcitonin

Non-bacterial category	Patients, n	Median PCT (ng/mL)	Typical PCT range (ng/mL)*	Representative conditions
Chronic kidney disease / dialysis	18	63.6	20 to ≥ 100	CKD stage 4–5, acute-on-chronic kidney injury, maintenance hemodialysis
Cardiac / shock states	8	32.5	15–80	Acute coronary syndrome, cardiogenic shock, acute LV failure, post-CABG
Post-surgical / trauma	10	26.6	15 to ≥ 100	Major surgery, postoperative states, fractures

Neurological insults	4	38.0	30–60	Ischemic stroke, intracerebral hemorrhage, encephalopathy
Pancreatitis / hepatobiliary disorders	3	32.3	20–60	Acute pancreatitis, cholangitis, hepatic failure
Autoimmune / hyperinflammatory states	1	45.7	~45	SLE, secondary hemophagocytic syndrome
Anaphylaxis / drug-related reactions	1	100.0	≥100	Reaction following parenteral iron

* Ranges are derived from observed minimum and maximum values within each category. Patient counts shown reflect the categories with complete quantitative summaries; totals do not sum to the full culture-negative cohort because the severe viral infection and malignancy/chemotherapy categories are reported narratively above but were not included with summary statistics in the original dataset — author to confirm and add these rows if available.

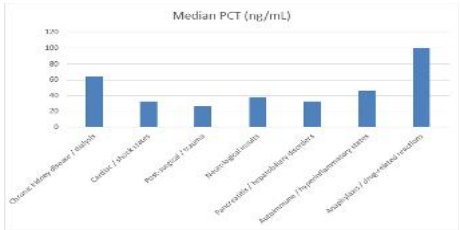


Figure 2. Median serum procalcitonin levels across major non-bacterial clinical categories in culture-negative hospitalized patients. Chronic kidney disease and dialysis-dependent states showed the highest median procalcitonin levels. Marked elevations were observed across all categories, including cardiac, postoperative, neurological, and systemic inflammatory conditions.

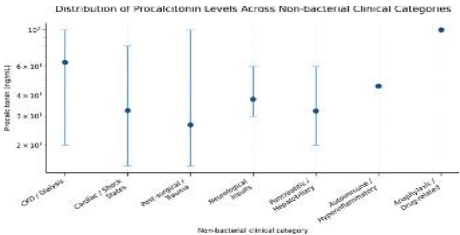


Figure 3. Distribution of serum procalcitonin (PCT) levels across non-bacterial clinical conditions.

The figure shows median PCT levels and ranges for patients with non-bacterial causes of elevated PCT, including chronic kidney disease (CKD)/dialysis, cardiac or shock states, post-surgical or trauma-related conditions, neurological insults, pancreatitis/hepatobiliary disorders, autoimmune or hyperinflammatory states, and anaphylaxis/drug-related reactions. Elevated PCT levels were observed across multiple non-infectious conditions, with the highest median levels seen in CKD/dialysis and anaphylaxis/drug-related reactions. The y-axis is displayed on a logarithmic scale to accommodate the wide range of PCT values.

Statistical Analysis

Data were entered into a spreadsheet and analyzed using descriptive and inferential statistics. Continuous variables were summarized as mean, median, standard deviation (SD), and interquartile range (IQR), as appropriate, while categorical variables were expressed as frequencies and percentages. Because serum procalcitonin (PCT) values demonstrated a non-normal distribution with marked skewness and several observations at the upper assay reporting limit, comparisons between culture-negative and culture-positive patients were performed using the Mann-Whitney U test. The median PCT concentration was 51.05 ng/mL among culture-negative patients and 62.70 ng/mL among culture-positive patients. No statistically significant difference was observed between the two groups ($U = 1859.0, p = 0.280$). All statistical tests were two-tailed, and a p -value of <0.05 was considered statistically significant.

DISCUSSION

In this retrospective observational study, markedly elevated PCT was frequently present in hospitalized patients without microbiological evidence of bacterial infection. A substantial proportion of culture-negative patients had PCT values well above thresholds traditionally considered highly suggestive of bacterial sepsis, including levels exceeding 100 ng/mL. These findings underscore the limited specificity of PCT in critically ill patients and highlight the need for cautious interpretation in complex clinical settings.

PCT is induced by pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor- α , which are released not only during bacterial infection but also in severe non-infectious inflammatory states [3,6]. In the present study, elevated PCT was commonly associated with conditions characterized by systemic inflammation, tissue ischemia, or multiorgan dysfunction. Cardiac conditions — including acute coronary syndromes, cardiogenic shock, and acute left ventricular failure — were frequently encountered; these are associated with global hypoperfusion and ischemia-reperfusion injury, which can trigger cytokine release and extrathyroidal PCT synthesis independent of infection [9,10].

Chronic kidney disease and dialysis-dependent states constituted another major group in this cohort. Reduced renal clearance of PCT, persistent low-grade inflammation, and dialysis-related immune activation are recognized contributors to elevated baseline PCT in patients with advanced kidney disease [11,12]. These findings reinforce the need for caution when interpreting PCT values in this population, particularly without corroborative clinical or microbiological evidence of infection.

Marked PCT elevation was also observed after major surgery and trauma. Surgical stress and extensive tissue injury can induce a SIRS resembling sepsis, producing transient but substantial rises in PCT [5]. Similarly, acute neurological insults — including ischemic and hemorrhagic stroke, hypoxic-ischemic encephalopathy, and osmotic demyelination syndrome — were associated with high PCT values; neuroinflammation and disruption of central regulatory mechanisms may contribute to this phenomenon [13,14].

Elevated PCT was also identified in severe viral infection [15], acute pancreatitis, hepatobiliary disorders [16,17], malignancy, autoimmune disease [18,19], and hyperinflammatory conditions [20]. These findings are consistent with the concept that PCT reflects the intensity of systemic inflammation rather than serving as a pathogen-specific marker. Notably, extremely high PCT values were observed in anaphylaxis and drug-related reactions, highlighting a diagnostic pitfall in which non-infectious shock states may mimic septic shock both clinically and biochemically [21,22].

These findings have important clinical implications. PCT is increasingly used to guide the initiation and discontinuation of antibiotic therapy; over-reliance on elevated PCT values, particularly in critically ill patients with complex comorbidities, may result in unnecessary or prolonged antibiotic use, contributing to antimicrobial resistance, increased healthcare costs, and patient harm [7,8]. Our results support the growing consensus that PCT should be interpreted as an adjunctive marker rather than a standalone diagnostic test, with its utility maximized when integrated with clinical assessment, imaging, and microbiological data [4,5].

Strengths and Limitations

This study has several limitations. First, its retrospective design limits the ability to establish causal relationships between underlying clinical conditions and PCT elevation. Second, culture-negative status does not completely exclude infection, particularly in patients who may have received antimicrobial therapy before specimen collection. Third, serial PCT measurements were unavailable for many patients, preventing evaluation of biomarker kinetics over time. Finally, the study was conducted at a single tertiary-care center, which may limit the generalizability of the findings to other healthcare settings.

CONCLUSION

This study demonstrates that markedly elevated serum procalcitonin (PCT) levels frequently occur in hospitalized patients without microbiological evidence of bacterial infection. High PCT concentrations were observed across a broad spectrum of non-bacterial conditions, including chronic kidney disease, cardiac and shock states, major surgery and trauma, acute neurological disorders,

severe viral infections, malignancy, autoimmune diseases, and drug-related hypersensitivity reactions. Importantly, PCT levels showed substantial overlap between culture-negative and culture-positive patients, highlighting the limited specificity of PCT as a standalone marker for bacterial infection.

These findings underscore that elevated PCT concentrations may reflect the intensity of systemic inflammation, tissue injury, organ dysfunction, and critical illness rather than the presence of bacterial infection alone. Consequently, reliance on PCT values in isolation may contribute to diagnostic uncertainty and inappropriate antimicrobial use.

PCT should therefore be interpreted as an adjunctive biomarker within the broader clinical context, incorporating patient history, physical examination, imaging findings, and microbiological investigations. Greater awareness of non-infectious causes of PCT elevation may improve diagnostic accuracy, support antimicrobial stewardship efforts, and reduce unnecessary antibiotic exposure. Future prospective studies are needed to further define condition-specific PCT thresholds and evaluate the role of serial PCT monitoring in distinguishing infectious from non-infectious inflammatory states.

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Ethical Approval

This was a retrospective observational study that utilized anonymized data obtained from routine clinical records. No intervention was performed, and no identifiable patient information was collected. So, the Ethics committee approval was waived by the Institutional Ethics Committee.

Consent to Participate

The requirement for informed consent was waived due to the retrospective nature of the study and the use of anonymized patient data collected during routine clinical care. No identifiable patient information was included in the analysis.

Declaration

The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki (2013 revision). As this was a retrospective observational study utilizing anonymized routinely collected clinical data without any intervention or direct patient contact, the study adhered to internationally accepted ethical principles for medical research involving human participants.

Competing interests

The authors declare no competing interests.

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Conflicts of Interest: None.

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