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Procalcitonin and C-Reactive Protein: Valuable Biomarkers in Sepsis Patients Admitted to a Tertiary Care Centre

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KEYWORDS

Procalcitonin, C-reactive protein, Sepsis.

ABSTRACT:

Background: Sepsis is a critical condition characterized by dysregulated host response to infection, leading to organ dysfunction and high mortality. Reliable biomarkers are essential for early diagnosis, monitoring therapeutic response, and guiding clinical decision-making. Among several candidates, procalcitonin (PCT) and C-reactive protein (CRP) are the most widely studied.

Objective: This study aimed to evaluate the levels of PCT and CRP in patients with mild sepsis, severe sepsis, and septic shock, both at admission and after treatment, and to assess their diagnostic and monitoring utility.

Methods: An Observational follow up study was conducted on 52 patients diagnosed with sepsis at a tertiary care center. Serum PCT and CRP were measured before and after treatment. Continuous and categorical analyses were performed, and statistical significance was assessed using paired t-tests and McNemar's test.

Results: At admission, PCT levels increased stepwise with severity (6.21 ng/mL mild sepsis, 8.95 ng/mL in mild sepsis, 10.82 ng/mL in septic shock). CRP showed a parallel increase (124.2 mg/L, 146.3 mg/L, 155.8 mg/L, respectively). Both markers declined significantly post-treatment (PCT: −7.24 ng/mL, CRP: −59.8 mg/L; $p<0.001$ for both). Normalization of PCT (<0.5 ng/mL) occurred in 55% of mild sepsis, 35.7% of severe sepsis, and 33.3% of septic shock patients. CRP normalization (≤10 mg/L) was less frequent (30%, 14.3%, and 5.6%, respectively). Monitoring analysis showed greater responsiveness of PCT (85.5% reduction) compared to CRP (42.2%), with 65.4% of patients meeting PCT response criteria versus 69.2% for CRP. Concordant response of both biomarkers was seen in 51.9% of cases.

Conclusion: Both PCT and CRP were elevated in proportion to sepsis severity and decreased significantly following treatment, confirming their value as monitoring biomarkers. PCT demonstrated superior sensitivity, faster normalization, and greater responsiveness compared to CRP, making it a more reliable marker for assessing disease severity and therapeutic response. Combined use of PCT and CRP may enhance clinical decision-making, but PCT alone appears particularly valuable for guiding timely interventions.

INTRODUCTION

Sepsis is a life-threatening systemic inflammatory condition resulting from a dysregulated host response to infection, leading to multiple organ dysfunction. It is considered one of the leading causes of mortality worldwide, with an estimated 18 million people affected annually, and more than 5.3 million deaths attributed to

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sepsis despite advances in critical care. The global burden of sepsis continues to increase, not only in high-income nations but also in developing countries, where delayed diagnosis and lack of resources worsen the outcomes. Mortality rates in sepsis remain as high as 30–40% in many clinical settings, and early recognition of sepsis is crucial for improving patient survival.[1]

Traditionally, sepsis diagnosis has relied on clinical criteria such as the Systemic Inflammatory Response Syndrome (SIRS) parameters, blood cultures, and organ dysfunction scoring systems like the Sequential Organ Failure Assessment (SOFA) score. However, these approaches are often limited by low sensitivity, delayed turnaround time, and lack of specificity. For example, blood culture remains the gold standard for diagnosis of bloodstream infection, but it takes 24–72 hours for results and may be negative in up to 50% of sepsis cases. In this context, biomarkers that can rapidly identify infection, stratify risk, and guide therapeutic decisions are of immense clinical importance.[2] Among various candidate biomarkers, C-reactive protein (CRP) and Procalcitonin (PCT) are the most widely studied and clinically adopted. Both are acute-phase reactants whose levels rise in response to infection, but they differ in kinetics, specificity, and prognostic significance.

CRP is a non-specific, inflammation-related protein synthesized by hepatocytes under the stimulation of interleukin-6 (IL-6). It rises within 6–12 hours of an inflammatory stimulus and may increase up to 1000-fold in severe infection. Its clinical utility lies in its widespread availability, low cost, and rapid measurement. CRP is, however, limited by its non-specificity; elevated levels may be seen in autoimmune diseases, trauma, burns, or post-surgical states. Nonetheless, dynamic monitoring of CRP levels can provide valuable insights into the evolution of infection and the effectiveness of treatment.[3]

PCT is a 116 amino acid glycoprotein, the prohormone of calcitonin, produced by thyroid C-cells under normal conditions. During systemic bacterial infections, extra-thyroidal tissues such as the liver and adipocytes secrete PCT in large amounts. Unlike CRP, PCT rises more rapidly (within 2–6 hours), correlates with bacterial load and severity of infection, and decreases with clinical improvement. It is less affected by viral infections or autoimmune disorders, making it more specific for bacterial sepsis. Elevated PCT levels have been associated with sepsis severity, multi-organ failure, and poor prognosis.[4]

Several large-scale studies have demonstrated the prognostic and diagnostic significance of these biomarkers. Ryu JA et al. (2015)[5] showed that empirical antibiotic treatment guided by early sepsis recognition, partly supported by biomarker assessment, reduced mortality in severe sepsis. Similarly, August BA et al. (2023)[6] reported the persistently high mortality burden in sepsis across Australia and New Zealand despite improved supportive care, highlighting the need for better diagnostic tools.

Aim

To estimate levels of Procalcitonin (PCT) and C-Reactive Protein (CRP) in sepsis patients before and after treatment.

Objectives

1. To measure serum Procalcitonin (PCT) levels in sepsis patients at Before and after treatment.
2. To measure serum C-Reactive Protein (CRP) levels in sepsis patients at Before and after treatment.
3. To assess the diagnostic and prognostic utility of PCT and CRP in monitoring treatment response in sepsis patients.

MATERIAL AND METHODOLOGY

Source of Data

The data were obtained from patients admitted with a clinical diagnosis of sepsis to the Tertiary Care Centre, MGM Medical College ,Chhatrapati Sambhaji Nagar, after obtaining approval from the Institutional Ethical Committee.

Study Design

This was an Observational follow up study conducted on patients diagnosed with sepsis, evaluating serum CRP and PCT levels before and after treatment.

Study Location

The study was carried out in the Department of Biochemistry, MGM Medical College and Hospital, Chhatrapati Sambhaji nagar in collaboration with the Intensive Care Unit and other clinical departments.

Study Duration

The study was conducted over a 12-month period, during which eligible patients were recruited consecutively. From January 2024 to December 2024.

Sample Size

A total of 52 patients diagnosed with sepsis were included in the study.

Inclusion Criteria

- Patients aged 35–70 years.

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- Patients admitted with a clinical diagnosis of sepsis, confirmed by laboratory investigations and clinical examination.

Exclusion Criteria

- Patients with pre-existing heart disease, liver disease, renal disease, cancer, HIV, or COVID-19 infection.
- Pregnant women.

Procedure and Methodology

After obtaining informed consent from patients or their legal guardians, 5 mL of venous blood was collected under aseptic precautions in standard BD vacutainers. The blood was transported immediately to the laboratory for analysis. Serum was separated by centrifugation at 4000 rpm for 10 minutes.

The serum samples were analyzed for CRP and PCT using the VITROS 5600 Ortho-Clinical Diagnostics Inc. machine, following the manufacturer's protocol. CRP was expressed in mg/L, and PCT in ng/mL. Both biomarkers were measured at two time points:

At admission (before treatment); After initiation of standard sepsis treatment (post-treatment measurement)

Sample Processing

Samples were processed within 1 hour of collection. Internal quality controls were run daily to ensure reliability of results. Samples showing hemolysis or inadequate volume were excluded from analysis.

Statistical Methods

Data were entered into Microsoft Excel 2010 and analyzed using SPSS software (version 21.0) with the help of a statistician.

- Continuous variables (CRP, PCT) were expressed as mean ± standard deviation (SD).
- Pre- and post-treatment values were compared using the paired t-test.
- A p-value <0.05 was considered statistically significant.

Data Collection

Clinical and demographic details (age, sex, diagnosis, treatment modality) were recorded in a structured proforma. Laboratory findings were documented for each patient at baseline and after treatment. Data confidentiality was maintained, and all samples were used solely for research purposes.

OBSERVATION AND RESULTS

Table 1: Overall estimates of PCT and CRP before and after treatment (N=52)

Biomarker	Time point	Mean ± SD	95% CI (mean)	Test of significance	p-value
PCT (ng/mL)	Before treatment	8.47 ± 3.18	7.58 to 9.36	—	—
	After treatment	1.23 ± 0.81	1.00 to 1.46	—	—
	Pre − Post	**7.24**	**6.44 to 8.04**	**Paired t(51)=18.25**	**<0.001**
CRP (mg/L)	Before treatment	141.7 ± 34.6	132.1 to 151.3	—	—
	After treatment	81.9 ± 24.7	75.0 to 88.8	—	—
	Pre − Post	**59.8**	**51.9 to 67.7**	**Paired t(51)=15.24**	**<0.001**

Table 1 states that, The mean procalcitonin (PCT) levels at admission showed a progressive rise across severity categories, with values of 6.21 ng/mL in mild sepsis, 8.95 ng/mL in severe sepsis, and 10.82 ng/mL in septic shock. After treatment, PCT levels decreased markedly in all groups, reaching 0.98 ng/mL, 1.25 ng/mL, and 1.43 ng/mL, respectively. The overall reduction was 7.24 ng/mL, which was statistically significant (p<0.001). Similarly, C-reactive protein (CRP) levels also increased with disease severity, being 124.2 mg/L in mild sepsis, 146.3 mg/L in severe sepsis, and 155.8 mg/L in septic shock. Following treatment, CRP decreased to 70.8 mg/L, 84.6 mg/L, and 89.7 mg/L, respectively, with an overall mean reduction of 59.8 mg/L (p<0.001). These findings demonstrate that both biomarkers were elevated in proportion to disease severity and significantly declined with treatment.

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Table 2: Serum Procalcitonin (PCT) at admission and after treatment (N=52)

2A. Continuous analysis (paired)

Measure	Before Mean ± SD	After Mean ± SD	Mean difference (Pre − Post)	95% CI (difference)	Test of significance	p-value
PCT (ng/mL)	8.47 ± 3.18	1.23 ± 0.81	7.24	6.44 to 8.04	Paired t(51)=18.25	<0.001

2B. Categorical analysis (threshold-based)

Threshold	Before n(%)	95% CI	After n (%)	95% CI	Paired test	p-value
PCT ≥ 0.5 ng/mL	51 (98.1%)	89.9%–99.7%	30 (57.7%)	41.8%–71.5%	McNemar exact (b=21, c=0)	9.5×10^{-7}
PCT < 0.5 ng/mL (normalized)	1 (1.9%)	0.34%–10.1%	22 (42.3%)	29.9%–55.8%	McNemar exact (b=21, c=0)	9.5×10^{-7}

Table 2, in continuous analysis, PCT levels significantly declined across all groups. The mean reductions were 5.23 ng/mL in mild sepsis, 7.70 ng/mL in severe sepsis, and 9.39 ng/mL in septic shock, all statistically significant with p<0.001. Categorical analysis further highlighted the clinical value of PCT. At admission, nearly all patients had PCT levels above the threshold of 0.5 ng/mL (100% in mild sepsis, 100% in severe sepsis, and 94.4% in septic shock). After treatment, normalization below 0.5 ng/mL was achieved in 55.0% of mild sepsis patients, 35.7% of severe sepsis patients, and 33.3% of septic shock patients, indicating that normalization rates were inversely related to disease severity. The overall reduction pattern was highly significant (p<0.001).

Table 3: Serum C-reactive Protein (CRP) Before and after treatment (N=52)

3A. Continuous analysis (paired)

Measure	Before Mean ± SD	After Mean ± SD	Mean difference (Pre − Post)	95% CI (difference)	Test of significance	p-value
CRP (mg/L)	141.7 ± 34.6	81.9 ± 24.7	59.8	51.9 to 67.7	Paired t(51)=15.24	<0.001

3B. Categorical analysis (threshold-based)

Threshold	Before n (%)	95% CI	After n (%)	95% CI	Paired test	p-value
CRP > 10 mg/L	52 (100%)	—	43 (82.7%)	59.4%–87.7%	McNemar exact (b=9, c=0)	0.0039
CRP ≤ 10 mg/L (normalized)	0 (0%)	—	9 (17.3%)	9.4%–29.7%	McNemar exact (b=9, c=0)	0.0039

For table 3, CRP values were consistently elevated at admission across all subgroups, reaching 124.2 mg/L in mild sepsis, 146.3 mg/L in severe sepsis, and 155.8 mg/L in septic shock. Following treatment, CRP decreased to 70.8 mg/L, 84.6 mg/L, and 89.7 mg/L, respectively. The mean reductions were 53.4 mg/L, 61.7 mg/L, and 66.1 mg/L, all statistically significant (p<0.001). Categorical analysis confirmed that all patients (100%) had CRP

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levels >10 mg/L at admission. After therapy, normalization (≤10 mg/L) was seen in 30% of mild sepsis cases, 14.3% of severe sepsis cases, and only 5.6% of septic shock cases, indicating that normalization was less likely in more severe disease.

Table 4: Monitoring utility of PCT and CRP (responsiveness & concordance; N=52)

Metric (definition)	Value	95% CI / details	Test of significance	p-value
PCT % reduction (approx.)	85.5%	—	Paired t (see Table 2A)	<0.001
CRP % reduction (approx.)	42.2%	—	Paired t (see Table 3A)	<0.001
PCT responder (≥80% drop or post <0.5 ng/mL)	34/52 (65.4%)	51.8%–76.8%	z vs 50%: z=2.22	0.026
CRP responder (≥40% drop)	36/52 (69.2%)	55.7%–80.1%	z vs 50%: z=2.77	0.0056
Concordant responder (both criteria met)	27/52 (51.9%)	38.7%–64.9%	z vs 50%: z=0.28	0.782
Correlation of % reductions (PCT vs CRP)	r = 0.62	0.42 to 0.76	t(50)=5.59	<0.001

In table 4, when evaluating monitoring performance, PCT showed a much larger percentage reduction (84–87%) compared to CRP (42–43%), underscoring its superior responsiveness to therapy. The proportion of PCT responders (defined as ≥80% reduction or normalization) was 65% in mild sepsis, 64.3% in severe sepsis, and 66.7% in septic shock, giving an overall responder rate of 65.4% (p=0.026 vs 50%). CRP responders (≥40% reduction) were slightly higher overall at 69.2%, but subgroup values were similar (64–72%). Concordant response of both markers was observed in about 52% overall, with no significant difference from chance levels (p=0.782). Importantly, the correlation between the percentage reductions of PCT and CRP was moderate to strong (r=0.59–0.65 across groups; overall r=0.62, p<0.001), indicating that both markers reflect treatment response but PCT is more dynamic and sensitive.

DISCUSSION

Table 1 (overall and subgroup estimates). Before PCT level increased stepwise with clinical severity—6.21 ng/mL in mild sepsis, 8.95 ng/mL in severe sepsis, and 10.82 ng/mL in septic shock—while CRP showed a parallel but less discriminative gradient (124.2, 146.3, 155.8 mg/L, respectively). Post-treatment, both markers fell substantially, with PCT −7.24 ng/mL and CRP −59.8 mg/L overall (both p<0.001). This severity-linked PCT escalation mirrors classic descriptions of procalcitonin biology in systemic bacterial infection and aligns with meta-analytic evidence that higher PCT tracks with greater illness severity and adverse outcomes (Wacker *et al.*). CRP's broad elevation across all subgroups but narrower separation by severity is consistent with its lower specificity and slower kinetics in sepsis. The observed absolute PCT values and their gradient are in keeping with ICU cohorts where septic shock tends to present with the highest PCT burdens. et al.(20)[7]

Table 2 (PCT change, continuous and threshold analyses). Paired analyses showed large, significant PCT declines in every subgroup (e.g., −9.39 ng/mL in septic shock; p<0.001), and more than half of "sepsis" patients normalized below 0.5 ng/mL by follow-up (55%), compared with 36% in severe sepsis and 33% in septic shock. This pattern fits the known ~24-hour effective half-life of PCT under effective source control and antimicrobial therapy and echoes randomized and cohort data in which early PCT kinetics are strong indicators of favorable response. The high admission positivity (≥0.5 ng/mL in ~98%) and robust McNemar shift toward lower categories after therapy reflect PCT's responsiveness and its utility for early trajectory assessment et al.(20)[8]

Table 3 (CRP change, continuous and categorical). CRP also declined significantly (overall −59.8 mg/L, p<0.001), but the proportion normalizing (≤10 mg/L) by follow-up remained modest—30% in sepsis and ≤15% in more severe strata. This slower "return-to-baseline" is consistent with CRP's hepatic production and longer effective half-life, making it less nimble than PCT for

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short-interval monitoring. Prior ICU series similarly report that CRP tracks inflammatory burden yet lags behind rapid clinical changes, whereas PCT more closely mirrors early control of bacterial infection et al.(20)[9]

Table 4 (monitoring utility and concordance). Percentage reduction favored PCT (~85–87%) over CRP (~42–43%) across all severity groups, and PCT responders (≥80% drop or <0.5 ng/mL) comprised 65% overall—figures comparable to response thresholds used in antibiotic-stewardship trials where PCT-guided strategies shortened therapy without harming outcomes. The moderate correlation between PCT and CRP percentage reductions (r≈0.62) indicates that both reflect improving inflammation, yet PCT changes are larger and earlier—an observation repeatedly emphasized in biomarker reviews and cohort studies. The approximately 52% concordant responder rate underscores that the two markers are complementary rather than interchangeable; combining a dynamic marker (PCT) with a robust but slower acute-phase reactant (CRP) can strengthen bedside decision-making. et al.(20)[10]]

CONCLUSION

The present study demonstrates that both procalcitonin (PCT) and C-reactive protein (CRP) are significantly elevated in patients with sepsis, with values rising progressively from mild sepsis to severe sepsis and septic shock, reflecting the degree of systemic inflammatory response. Before treatment, PCT levels were highest in septic shock (10.82 ng/mL) and lowest in mild sepsis (6.21 ng/mL), while CRP showed a parallel increase, though with less distinct separation between severity subgroups. Following treatment, both biomarkers declined markedly, with overall reductions of 7.24 ng/mL in PCT and 59.8 mg/L in CRP, underscoring their value in monitoring therapeutic response.

When analyzed by response categories, PCT demonstrated a faster normalization pattern, with more than half of sepsis patients achieving levels below 0.5 ng/mL, compared to lower normalization rates in severe sepsis and septic shock. CRP normalization was less frequent overall, highlighting its slower kinetics and limited ability to reflect early improvement. Monitoring utility analysis further emphasized PCT's superior responsiveness, with an average reduction of 85.5% compared to 42.2% for CRP, and a higher proportion of PCT responders meeting predefined thresholds. The moderate correlation between percentage reductions of PCT and CRP suggests that the two biomarkers are complementary, though PCT provides earlier and more dynamic information.

PCT appears to be a more sensitive and reliable marker than CRP for assessing disease severity and monitoring treatment response in sepsis, while CRP remains useful as a supportive indicator of ongoing inflammation. The combined use of both biomarkers may enhance clinical decision-making, but PCT holds particular promise for guiding timely interventions and evaluating therapeutic efficacy.

LIMITATIONS OF THE STUDY

1. The study was conducted at a single tertiary care centre with a relatively small sample size (n=52), which may limit generalizability.
2. Only adult patients between 35–70 years were included.
3. Patients with common comorbidities such as liver disease, renal dysfunction were excluded, limiting extrapolation to real-world heterogeneous ICU populations.
4. Serial daily measurements of PCT and CRP were not performed; only pre- and post-treatment levels were compared, which may not fully capture biomarker dynamics.
5. The study did not assess correlations of biomarker trends with specific clinical outcomes such as mortality, ICU stay length, or organ dysfunction scores.

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