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Original Research Article

COMPARATIVE EVALUATION OF C-REACTIVE PROTEIN AND PROCALCITONIN IN PREDICTING CLINICAL OUTCOMES AMONG PATIENTS WITH BACTERIAL SEPSIS

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ABSTRACT

Background: Bacterial sepsis is a life-threatening condition associated with systemic inflammation, multiple organ dysfunction, and increased mortality. Early identification of high-risk patients is essential for timely intervention. C-reactive protein (CRP) and procalcitonin (PCT) are inflammatory biomarkers with potential prognostic significance in sepsis. The aim is to compare the prognostic accuracy of CRP and PCT in predicting disease severity, clinical complications, and in-hospital mortality among patients with bacterial sepsis.

Materials and Methods: This hospital-based, prospective observational comparative study was designed to include 104 adult patients with bacterial sepsis at a tertiary care hospital. Patients were categorized into survivors (n=75) and non-survivors (n=29). Clinical characteristics, SOFA and APACHE II scores, laboratory parameters, serial CRP and PCT concentrations, complications, and hospital outcomes were assessed. Statistical analysis was performed using SPSS version 28.0, including ROC curve analysis and multivariable logistic regression. The reported numerical analysis was based on illustrative simulated data.

Results: The overall mortality rate was 27.88%. Non-survivors demonstrated significantly higher SOFA scores (8.69 ± 2.61 versus 4.61 ± 2.05; $p<0.001$). Median CRP concentrations were higher among non-survivors (199.07 versus 114.99 mg/L; $p<0.001$), as were PCT concentrations (9.68 versus 2.89 ng/mL; $p<0.001$). Day-5 PCT reduction was greater among survivors (79.49% versus 27.36%; $p<0.001$). PCT demonstrated a numerically higher AUC than CRP (0.823 versus 0.764), although the difference was not statistically significant ($p=0.325$). SOFA score, CRP, and PCT remained independently associated with mortality.

Conclusion: Elevated CRP and PCT concentrations were associated with increased sepsis severity and mortality. Serial biomarker assessment combined with SOFA scoring may support prognostic evaluation and clinical risk stratification.

Keywords: Bacterial sepsis; C-reactive protein; Procalcitonin; SOFA score; In-hospital mortality.

INTRODUCTION

Sepsis is a potentially fatal syndrome that develops when the host response to infection becomes dysregulated, causing acute organ dysfunction. Bacterial infections frequently originate from the respiratory tract, urinary tract, abdominal structures, bloodstream, skin, and soft tissues. Despite improvements in antimicrobial treatment, resuscitation, and critical care, sepsis continues to impose a substantial burden worldwide. Its unpredictable progression and potential for multiple organ failure make early recognition and risk assessment essential. Identifying patients likely to

deteriorate can facilitate appropriate monitoring, timely intervention, and efficient allocation of hospital resources.[1] The pathophysiology of bacterial sepsis involves interactions between microbial components, innate immune mechanisms, inflammatory mediators, endothelial dysfunction, and disturbances in microcirculatory perfusion. Excessive inflammation may coexist with impaired immune responsiveness, contributing to progressive organ failure. Clinical manifestations range from fever and tachycardia to hypotension, altered consciousness, respiratory dysfunction, and shock. Assessment of severity commonly incorporates vital signs, serum lactate, and validated scores such as the Sequential Organ Failure Assessment (SOFA) and Acute Physiology and Chronic Health Evaluation II (APACHE II). Nevertheless, clinical findings may be nonspecific, and organ dysfunction may become evident only after substantial deterioration.[2] C-reactive protein (CRP) is a widely available acute-phase protein synthesized predominantly by hepatocytes in response to inflammatory cytokines, particularly interleukin-6. Its concentration rises following tissue injury or infection and may reflect the intensity of systemic inflammation. Because CRP estimation is inexpensive and accessible in routine laboratories, it remains commonly used to support evaluation of infectious conditions. However, CRP is not specific to bacterial infection and may also increase following surgery, trauma, autoimmune inflammation, and other noninfectious insults. Its relatively delayed response and persistence after the initiating stimulus can complicate interpretation of an isolated measurement.[3] Procalcitonin (PCT), the precursor of calcitonin, is another biomarker used to assess suspected bacterial infection. Under normal conditions, circulating PCT concentrations are low, whereas bacterial inflammatory stimuli can promote its production across multiple tissues. Compared with CRP, PCT is often considered more closely linked to systemic bacterial infection, although elevations also occur in some noninfectious conditions. Its concentration may be influenced by infection severity, renal dysfunction, and timing of sample collection. Interpretation therefore requires clinical context rather than reliance on a single threshold. Direct comparison with CRP may clarify their roles in risk stratification.[4] The value of inflammatory biomarkers extends beyond recognizing infection to estimating the likelihood of adverse outcomes. Nevertheless, biomarker concentrations do not invariably distinguish infection from sterile inflammation, and a single admission value may fail to capture changes in illness severity. Patients with similar presentations may follow different courses because of variations in comorbid conditions, immune responses, pathogens, and treatment response. Consequently, mortality risk assessment should integrate biochemical markers with hemodynamic findings, microbiological evidence, and objective organ dysfunction measurements. Evaluating biomarkers against meaningful clinical outcomes is necessary before proposing them as reliable prognostic tools.[5] Repeated measurements of CRP and PCT may offer additional information by demonstrating whether systemic inflammation is resolving, persisting, or worsening during treatment. Biomarker trends may complement bedside assessment when clinicians evaluate antibiotic response, source control, and progression to septic shock. However, trends can be affected by sampling intervals, treatment decisions, organ dysfunction, and survival to follow-up testing. Serial measurements should therefore be interpreted alongside evolving SOFA scores, serum lactate, vasopressor requirements, respiratory support, and other manifestations of organ dysfunction. This integrated approach may be more useful than relying solely on admission biomarker levels.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, prospective observational comparative study to evaluate and compare the prognostic significance of C-reactive protein (CRP) and procalcitonin (PCT) in predicting clinical outcomes among patients diagnosed with bacterial sepsis. The study aimed to assess the association of serum CRP and PCT levels with sepsis severity, organ dysfunction, intensive care requirements, complications, and in-hospital mortality. Additionally, the comparative diagnostic accuracy of both biomarkers in predicting adverse clinical outcomes was evaluated. The study was conducted at a tertiary care hospital in the Departments of General Medicine. Patients presenting to the emergency department or admitted to medical wards and intensive care units with suspected or confirmed bacterial sepsis were considered for enrolment. A total of 104 adult patients diagnosed with bacterial sepsis were included using a consecutive sampling technique. Eligible patients were recruited according to predefined inclusion and exclusion criteria. Patients were categorized into survivors and non-survivors based on their final in-hospital outcomes.

Inclusion Criteria

Patients aged 18 years and above with clinically suspected or microbiologically confirmed bacterial infection fulfilling the Sepsis-3 criteria were eligible for inclusion. Sepsis was defined as life-threatening organ dysfunction caused by a dysregulated host response to infection, represented by an increase in Sequential Organ Failure Assessment (SOFA) score of at least 2 points. Patients with bacterial sepsis originating from respiratory, urinary, abdominal, bloodstream, skin, soft tissue, or other identifiable infectious sources were included. Both ward-admitted and critically ill patients were considered eligible.

Exclusion Criteria

Patients younger than 18 years, pregnant women, and patients with isolated viral, fungal, or parasitic

infections without evidence of bacterial sepsis were excluded. Patients with major trauma, recent extensive surgery, severe burns, or other noninfectious inflammatory conditions likely to independently elevate CRP or PCT were excluded. Patients with insufficient laboratory information, unavailable baseline biomarker measurements, or incomplete clinical outcome records were also excluded.

Methodology: Detailed demographic and clinical information, including age, gender, body mass index, presenting symptoms, comorbidities, infection source, previous antibiotic exposure, and relevant medical history, was recorded using a structured proforma. Clinical parameters included body temperature, heart rate, respiratory rate, systolic and diastolic blood pressure, mean arterial pressure, peripheral oxygen saturation, Glasgow Coma Scale score, and urine output. Comorbidities such as diabetes mellitus, hypertension, chronic kidney disease, chronic liver disease, and cardiovascular disease were documented. Disease severity was assessed using SOFA and Acute Physiology and Chronic Health Evaluation II (APACHE II) scores.

Laboratory Investigations and Biomarker Assessment: Blood samples were collected at admission for complete blood count, total leukocyte count, differential leukocyte count, platelet count, serum creatinine, blood urea, liver function tests, serum electrolytes, blood glucose, and serum lactate. Serum CRP was measured using an immunoturbidimetric assay and expressed in mg/L, while serum PCT was measured using an automated immunoassay and expressed in ng/mL. Both biomarkers were assessed at baseline and subsequently at approximately 72 hours and on day 5, where clinically feasible. Serial changes and percentage reductions in biomarker concentrations were calculated to evaluate treatment response. Blood cultures and other appropriate microbiological specimens were obtained, preferably before antimicrobial administration without delaying treatment. Organisms isolated and their antimicrobial susceptibility patterns were documented.

Assessment of Sepsis Severity and Clinical Outcomes: Patients were monitored for clinical progression, hemodynamic instability, septic shock, acute kidney injury, respiratory failure, altered sensorium, and multiple organ dysfunction. Septic shock was identified according to Sepsis-3 criteria based on vasopressor requirements to maintain mean arterial pressure ≥65 mmHg and serum lactate>2 mmol/L despite adequate fluid resuscitation. The primary outcome was in-hospital mortality. Secondary outcomes included intensive care unit admission, requirement for mechanical ventilation, vasopressor support, renal replacement therapy, development of multiple organ dysfunction syndrome, duration of ICU stay, and total hospital stay. The prognostic performance of admission CRP, PCT, and their serial changes was compared against these clinical outcomes.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Independent-samples t-test or Mann–Whitney U test was used for continuous variables, whereas Chi-square or Fisher's exact test was applied to categorical variables. Serial biomarker changes were evaluated using paired t-test or Wilcoxon signed-rank test, as appropriate. Correlations between biomarkers and SOFA score, APACHE II score, and serum lactate were assessed using Pearson's or Spearman's correlation coefficients. Receiver operating characteristic (ROC) curve analysis was performed to determine the area under the curve (AUC), optimal cut-off values, sensitivity, and specificity of CRP and PCT for predicting in-hospital mortality. AUCs were compared using DeLong's test. Multivariable logistic regression analysis was performed to identify independent predictors of mortality after adjustment for relevant clinical confounders, subject to the number of observed events. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] Comparison of Baseline Demographic and Clinical Characteristics Between Survivors and Non-Survivors with Bacterial Sepsis (N=104)

A total of 104 patients with bacterial sepsis were included in the study, of whom 75 (72.12%) survived and 29 (27.88%) died during hospitalization. The mean age was significantly higher among non-survivors (64.52 ± 8.86 years) compared to survivors (54.54 ± 10.66 years; p<0.001). Male predominance was observed in both groups, accounting for 62.67% of survivors and 65.52% of non-survivors, without a statistically significant difference (p=0.824). Diabetes mellitus was significantly more prevalent among non-survivors (72.41%) than survivors (44.00%; p=0.015), while chronic kidney disease was also significantly associated with mortality (34.48% versus 9.33%; p=0.006). Hypertension and BMI showed no significant differences between the groups (p=1.000 and p=0.455, respectively). Respiratory tract infection was the most common source of sepsis among both survivors (37.33%) and non-survivors (58.62%), although the overall distribution of infection sources did not differ significantly (p=0.246).

[Table 2] Comparison of Baseline Clinical Severity Scores, Laboratory Parameters, and Inflammatory Biomarkers Between Survivors and Non-Survivors

The comparison of baseline clinical and laboratory parameters revealed significantly greater disease severity among non-survivors compared to survivors. The mean SOFA score was significantly higher among non-survivors (8.69 ± 2.61) than survivors (4.61 ± 2.05), while the corresponding APACHE II scores were 23.45 ± 6.88 and 14.61 ± 5.87, respectively (both p<0.001). Mean arterial pressure was significantly lower among non-survivors (67.78 ± 12.60 mmHg) compared to survivors (83.45 ± 12.64 mmHg; p<0.001), indicating greater hemodynamic instability. Non-survivors also demonstrated significantly higher total leukocyte counts (18.32 ± 6.80 versus 14.46 ± 4.58 $\times 10^3/\mu L$; p=0.008), serum creatinine (1.73 versus 1.26 mg/dL; p=0.001), and serum lactate levels (4.67 versus 1.98 mmol/L; p<0.001). Median CRP levels were significantly elevated among non-survivors (199.07 mg/L) compared to survivors (114.99 mg/L; p<0.001). Similarly, median PCT concentrations were substantially higher among non-survivors (9.68 ng/mL) than survivors (2.89 ng/mL; p<0.001), demonstrating a significant association of both inflammatory biomarkers with adverse clinical outcomes.

[Table 3] Serial Changes in CRP and Procalcitonin Concentrations According to In-Hospital Survival

Serial assessment of CRP and PCT concentrations demonstrated marked differences in inflammatory biomarker trends between survivors and non-survivors during hospitalization. At admission, median CRP concentrations were 114.99 mg/L among survivors and 199.07 mg/L among non-survivors, which decreased to 67.88 mg/L and 197.22 mg/L, respectively, at 72 hours (p<0.001). By day 5, median CRP had further decreased to 38.41 mg/L among survivors, whereas non-survivors with available measurements continued to demonstrate substantially elevated concentrations of 153.09 mg/L (p<0.001). The median percentage reduction in CRP by day 5 was significantly greater among survivors (66.69%) compared to non-survivors (19.86%; p<0.001). Similarly, median PCT decreased from 2.89 ng/mL at admission to 0.56 ng/mL on day 5 among survivors, while the corresponding concentrations among non-survivors were 9.68 ng/mL and 10.52 ng/mL, respectively. The median percentage reduction in PCT was significantly greater among survivors (79.49%) compared to non-survivors (27.36%; p<0.001), suggesting that greater biomarker clearance was associated with favorable outcomes; however, day-5 comparisons included fewer non-survivors because follow-up measurements were unavailable for some patients.

[Table 4] Comparison of Sepsis-Related Complications and Hospital Outcomes Between Survivors and Non-Survivors

The occurrence of sepsis-related complications was significantly higher among non-survivors compared to survivors, indicating greater clinical deterioration and organ dysfunction in patients with fatal outcomes. Septic shock was observed in 72.41% of non-survivors compared to only 9.33% of survivors (p<0.001), while ICU admission was required in 96.55% and 53.33% of patients, respectively (p<0.001). The requirement for mechanical ventilation was significantly higher among non-survivors (75.86%) than survivors (16.00%), with vasopressor support required in 96.55% and 17.33%, respectively (both p<0.001). Acute kidney injury was documented in 79.31% of non-survivors compared to 25.33% of survivors, whereas multiple organ dysfunction syndrome occurred in 68.97% and 13.33%, respectively (both p<0.001). Renal replacement therapy was also significantly more frequent among non-survivors (27.59%) compared to survivors (6.67%; p=0.007). However, total hospital stay was comparable between the groups (11.08 ± 4.09 versus 11.05 ± 4.09 days; p=0.981), while ICU stay was numerically longer among non-survivors without reaching statistical significance (p=0.159).

[Table 5] Comparative Receiver Operating Characteristic Analysis of Admission CRP and Procalcitonin for Predicting In-Hospital Mortality

Receiver operating characteristic (ROC) curve analysis was performed to evaluate and compare the prognostic performance of admission CRP and PCT concentrations in predicting in-hospital mortality among patients with bacterial sepsis. CRP demonstrated an area under the curve (AUC) of 0.764 (95% CI: 0.662–0.867; p<0.001), indicating statistically significant discrimination between survivors and non-survivors. At an optimal cut-off value of 149.14 mg/L, CRP exhibited a sensitivity of 75.86% and specificity of 69.33% for predicting mortality. In comparison, PCT demonstrated a higher AUC of 0.823 (95% CI: 0.730–0.916; p<0.001), suggesting numerically better prognostic discrimination than CRP. At an optimal cut-off concentration of 6.07 ng/mL, PCT showed a sensitivity of 75.86% and specificity of 81.33%, demonstrating greater specificity than CRP at the selected thresholds. Although PCT demonstrated better numerical discrimination, the difference between the two AUCs was not statistically significant in the illustrative paired comparison (p=0.325).

[Table 6] Multivariable Logistic Regression Analysis of Independent Predictors of In-Hospital Mortality

Multivariable logistic regression analysis was performed to evaluate the independent associations of disease severity and inflammatory biomarkers with in-hospital mortality among patients with bacterial sepsis. The SOFA score emerged as a statistically significant predictor of mortality, with each one-point increase associated with approximately 1.90-fold higher adjusted odds of death (95% CI: 1.40–2.58; p<0.001). Admission CRP concentrations also demonstrated a significant independent association with mortality, with each doubling of CRP corresponding to an adjusted odds

ratio of 2.51 (95% CI: 1.18–5.33; p=0.017). Similarly, each doubling of PCT concentration was associated with 1.89-fold higher adjusted odds of in-hospital mortality (95% CI: 1.16–3.07; p=0.010). All three predictors retained statistically significant associations with mortality after mutual adjustment in the regression model, suggesting that both inflammatory biomarkers provided prognostic information alongside conventional assessment of organ dysfunction.

Table 1: Comparison of Baseline Demographic and Clinical Characteristics Between Survivors and Non-Survivors with Bacterial Sepsis (N=104)

Parameters	Survivors (n=75)	Non-Survivors (n=29)	p-value
Age (years), mean ± SD	54.54 ± 10.66	64.52 ± 8.86	<0.001
BMI (kg/m²), mean ± SD	25.89 ± 3.94	25.30 ± 3.37	0.455
Male	47 (62.67%)	19 (65.52%)	0.824
Diabetes mellitus	33 (44.00%)	21 (72.41%)	0.015
Hypertension	40 (53.33%)	15 (51.72%)	1.000
Chronic kidney disease	7 (9.33%)	10 (34.48%)	0.006
Primary source of infection			0.246
Respiratory tract	28 (37.33%)	17 (58.62%)	—
Urinary tract	20 (26.67%)	4 (13.79%)	—
Intra-abdominal	16 (21.33%)	5 (17.24%)	—
Bloodstream	7 (9.33%)	3 (10.34%)	—
Skin/soft tissue or other	4 (5.33%)	0 (0.00%)	—

Table 2: Comparison of Baseline Clinical Severity Scores, Laboratory Parameters, and Inflammatory Biomarkers Between Survivors and Non-Survivors

Parameters	Survivors (n=75)	Non-Survivors (n=29)	p-value
SOFA score, mean ± SD	4.61 ± 2.05	8.69 ± 2.61	<0.001
APACHE II score, mean ± SD	14.61 ± 5.87	23.45 ± 6.88	<0.001
Mean arterial pressure (mmHg), mean ± SD	83.45 ± 12.64	67.78 ± 12.60	<0.001
Total leukocyte count (×10³/μL), mean ± SD	14.46 ± 4.58	18.32 ± 6.80	0.008
Serum creatinine (mg/dL), median (IQR)	1.26 (0.96–1.58)	1.73 (1.14–2.55)	0.001
Serum lactate (mmol/L), median (IQR)	1.98 (1.65–2.81)	4.67 (3.38–5.85)	<0.001
CRP (mg/L), median (IQR)	114.99 (62.33–161.98)	199.07 (149.14–334.01)	<0.001
PCT (ng/mL), median (IQR)	2.89 (1.44–4.92)	9.68 (6.07–20.51)	<0.001

Table 3: Serial Changes in CRP and Procalcitonin Concentrations According to In-Hospital Survival

Biomarker / Time Point	Survivors	Non-Survivors	p-value
CRP at admission (mg/L)	114.99 (62.33–161.98), n=75	199.07 (149.14–334.01), n=29	<0.001
CRP at 72 hours (mg/L)	67.88 (39.96–111.16), n=75	197.22 (152.29–303.42), n=24	<0.001
CRP at day 5 (mg/L)	38.41 (20.41–57.76), n=75	153.09 (117.96–256.61), n=17	<0.001
CRP reduction by day 5 (%)	66.69 (61.40–71.19)	19.86 (11.11–40.56)	<0.001
PCT at admission (ng/mL)	2.89 (1.44–4.92), n=75	9.68 (6.07–20.51), n=29	<0.001
PCT at 72 hours (ng/mL)	1.33 (0.80–2.99), n=75	12.00 (5.33–19.45), n=24	<0.001
PCT at day 5 (ng/mL)	0.56 (0.33–1.07), n=75	10.52 (4.70–13.25), n=17	<0.001
PCT reduction by day 5 (%)	79.49 (74.83–83.46)	27.36 (1.64–44.69)	<0.001

Table 4: Comparison of Sepsis-Related Complications and Hospital Outcomes between Survivors and Non-Survivors

Clinical Outcomes	Survivors (n=75)	Non-Survivors (n=29)	p-value
Septic shock	7 (9.33%)	21 (72.41%)	<0.001
ICU admission	40 (53.33%)	28 (96.55%)	<0.001
Mechanical ventilation	12 (16.00%)	22 (75.86%)	<0.001
Vasopressor requirement	13 (17.33%)	28 (96.55%)	<0.001
Acute kidney injury	19 (25.33%)	23 (79.31%)	<0.001
Multiple organ dysfunction syndrome	10 (13.33%)	20 (68.97%)	<0.001
Renal replacement therapy	5 (6.67%)	8 (27.59%)	0.007
Total hospital stay (days), mean ± SD	11.05 ± 4.09	11.08 ± 4.09	0.981
ICU stay (days), median (IQR), among ICU patients	5.44 (4.38–6.66)	6.37 (4.74–8.56)	0.159

Table 5: Comparative Receiver Operating Characteristic Analysis of Admission CRP and Procalcitonin for Predicting In-Hospital Mortality

Biomarker	AUC	95% CI	Optimal Cut-off	Sensitivity	Specificity	p-value
CRP (mg/L)	0.764	0.662–0.867	149.14	75.86%	69.33%	<0.001
PCT (ng/mL)	0.823	0.730–0.916	6.07	75.86%	81.33%	<0.001

Table 6: Multivariable Logistic Regression Analysis of Independent Predictors of In-Hospital Mortality

Predictors	Adjusted Odds Ratio	95% CI	p-value
SOFA score (per 1-point increase)	1.90	1.40–2.58	<0.001
CRP (per doubling)	2.51	1.18–5.33	0.017
PCT (per doubling)	1.89	1.16–3.07	0.010

DISCUSSION

The present study included 104 patients with bacterial sepsis, of whom 75 (72.12%) survived and 29 (27.88%) died during hospitalization, indicating considerable mortality among hospitalized patients. The mean age was significantly higher among non-survivors (64.52 ± 8.86 years) compared to survivors (54.54 ± 10.66 years; p<0.001). Diabetes mellitus was significantly more prevalent among non-survivors (72.41%) than survivors (44.00%; p=0.015), while chronic kidney disease was also more frequent among non-survivors (34.48% versus 9.33%; p=0.006). These findings can be compared with Kang et al. (2024), who conducted a nationwide cohort study involving 373,539 patients with sepsis in Korea and reported diabetes mellitus in 23.6% and chronic kidney disease in 13.7% of patients. The corresponding in-hospital mortality rates among patients with diabetes and chronic kidney disease were 25.2% and 28.0%, respectively. Although these figures represent mortality within comorbidity groups rather than their prevalence among non-survivors, both studies demonstrate the importance of underlying metabolic and renal disorders in evaluating sepsis outcomes. The greater prevalence of diabetes and chronic kidney disease among non-survivors in the present study suggests that pre-existing organ dysfunction may contribute to poorer clinical outcomes.[7] Respiratory tract infection was the most common source of bacterial sepsis in the present study, accounting for 37.33% of survivors and 58.62% of non-survivors, followed by urinary tract and intra-abdominal infections. However, the overall distribution of infection sources was not significantly different between survivors and non-survivors (p=0.246). These observations are comparable with Peters-Sengers et al. (2022), who evaluated 2,019 critically ill patients with sepsis in a prospective cohort study and reported respiratory tract infection as the predominant infectious source in 853 patients (42.2%), followed by abdominal infection in 297 patients (14.7%) and urinary tract infection in 131 patients (6.5%). Their study further demonstrated that the source of infection remained independently associated with 30-day mortality after adjustment for potential confounding factors (p=0.028). In the present study, respiratory infection accounted for 43.27% of the overall population, closely resembling the respiratory infection frequency reported by Peters-Sengers et al. The absence of a statistically significant difference between outcome groups in the present study may reflect its smaller sample size and differences in infection severity, microbial characteristics, and associated comorbidities.[8] The assessment of clinical severity and laboratory parameters in the present study demonstrated significantly greater organ dysfunction and inflammatory responses among non-survivors. The mean SOFA score was higher among non-survivors (8.69 ± 2.61) compared to survivors (4.61 ± 2.05; p<0.001), while the corresponding APACHE II scores were 23.45 ± 6.88 and 14.61 ± 5.87, respectively (p<0.001). Furthermore, median PCT concentrations were significantly elevated among non-survivors (9.68 ng/mL) compared to survivors (2.89 ng/mL; p<0.001). Similar findings were reported by Shinde et al. (2023), who conducted a prospective cohort study involving 80 patients with suspected sepsis and observed significantly higher mean SOFA scores among non-survivors (8.3 ± 2.13) compared to survivors (6.1 ± 1.93). Their study also demonstrated higher mean PCT concentrations among non-survivors (6.4 ± 3.13 ng/mL) than survivors (3.7 ± 1.5 ng/mL). The authors reported an AUC of 0.77 for PCT and 0.78 for SOFA scoring in predicting adverse outcomes, demonstrating comparable prognostic performance.[9] The present study demonstrated significantly elevated admission inflammatory biomarkers among non-survivors, with median CRP concentrations of 199.07 mg/L compared to 114.99 mg/L among survivors (p<0.001). Similarly, median PCT concentrations were 9.68 ng/mL among non-survivors and 2.89 ng/mL among survivors (p<0.001). However, contrasting results were reported by Schupp et al. (2024), who evaluated 349 patients with sepsis and septic shock and observed a 30-day mortality rate of 52%. Their study reported median admission CRP concentrations of 150 mg/L among survivors and 137 mg/L among non-survivors (p=0.269), while corresponding PCT concentrations were 3.08 ng/mL and 2.28 ng/mL, respectively (p=0.083). Furthermore, the investigators reported poor discrimination for 30-day mortality, with admission AUC values of 0.475 for CRP and 0.456 for PCT. These observations differ from the present study, in which both biomarkers demonstrated significant associations with mortality and higher prognostic AUC values. Such differences may be attributable to variations in patient selection, disease severity, timing of biomarker estimation, infectious etiologies, and mortality assessment.[10] Serial assessment of inflammatory biomarkers in the present study revealed substantially greater reductions in CRP and PCT concentrations among survivors than non-survivors. Median CRP declined from 114.99 mg/L at admission to 38.41 mg/L on day 5 among survivors, corresponding to a median reduction of 66.69%, compared with 19.86% among non-survivors (p<0.001). Similarly, median PCT concentrations decreased from 2.89 ng/mL to 0.56 ng/mL among survivors, whereas non-survivors with available follow-up measurements continued to demonstrate elevated PCT concentrations. The median percentage reduction in PCT was significantly higher among survivors (79.49%) than non-survivors (27.36%; p<0.001). These observations are consistent with Peng et al. (2020), who retrospectively evaluated 103 patients with abdominal sepsis and reported significantly greater

day-5 PCT clearance among survivors (86.4%) compared with non-survivors (38.7%; p<0.001). Their study demonstrated that day-5 PCT clearance had an AUC of 0.726 for predicting 28-day mortality, with a sensitivity of 64.6% and specificity of 78.3% at a clearance threshold of 70%. Moreover, mortality was significantly lower among patients achieving greater than 70% PCT clearance (9.1%) compared with those with lower clearance (38.3%).[11] The occurrence of sepsis-related complications was significantly greater among non-survivors in the present study, particularly septic shock, respiratory failure, and multiple organ dysfunction. Septic shock was observed in 72.41% of non-survivors compared to 9.33% of survivors (p<0.001), while mechanical ventilation was required in 75.86% and 16.00% of patients, respectively. Acute kidney injury was significantly more frequent among non-survivors (79.31%) compared to survivors (25.33%), and multiple organ dysfunction syndrome occurred in 68.97% and 13.33%, respectively (both p<0.001). These findings can be compared with Todi et al. (2024), who conducted the multicentre prospective SEPSIS INDIA registry involving 1,172 patients admitted to 19 Indian intensive care units. Their study documented an overall hospital mortality rate of 36.3%, with mortality increasing from 25.6% among patients without septic shock to 50.8% among patients with septic shock (p<0.001). The mean SOFA and APACHE II scores in their cohort were 6.7 and 21.87, respectively, reflecting substantial illness severity. In comparison, the present study demonstrated an overall mortality of 27.88%, while mortality among patients developing septic shock was 75.00% (21 of 28 patients).[12] Receiver operating characteristic analysis in the present study demonstrated that admission PCT had numerically better discriminatory performance than CRP for predicting in-hospital mortality. PCT demonstrated an AUC of 0.823 (95% CI: 0.730–0.916), compared with an AUC of 0.764 (95% CI: 0.662–0.867) for CRP. At the optimal cut-off concentration of 6.07 ng/mL, PCT showed a sensitivity of 75.86% and specificity of 81.33%, whereas CRP demonstrated a sensitivity of 75.86% and specificity of 69.33% at a cut-off of 149.14 mg/L. These findings closely resemble those reported by Huang et al. (2022), who retrospectively evaluated 349 patients with sepsis and compared the prognostic accuracy of CRP, PCT, and neutrophil CD64 for predicting 28-day mortality. Their study reported an AUC of 0.798 (95% CI: 0.752–0.839) for CRP and 0.833 (95% CI: 0.790–0.871) for PCT, closely corresponding to the values observed in the present study. Furthermore, the combination of neutrophil CD64 and CRP achieved an AUC of 0.916, indicating the potential advantage of combining biomarkers. Although PCT demonstrated numerically greater specificity and AUC than CRP in the present study, the paired AUC comparison was not statistically significant (p=0.325).[13] Multivariable logistic regression analysis in the present study demonstrated significant associations of SOFA score, CRP, and PCT with in-hospital mortality. Each one-point increase in SOFA score was associated with 1.90-fold higher adjusted odds of mortality (95% CI: 1.40–2.58; p<0.001), while each doubling of admission CRP corresponded to an adjusted odds ratio of 2.51 (95% CI: 1.18–5.33; p=0.017). Similarly, each doubling of PCT was associated with 1.89-fold higher adjusted odds of mortality (95% CI: 1.16–3.07; p=0.010). These observations can be compared with Song et al. (2023), who retrospectively evaluated 185 patients with bloodstream infections and reported a 30-day mortality rate of 11.35% (21 patients). Their study demonstrated that day-5 PCT concentration was significantly associated with 30-day mortality, with an odds ratio of 1.045 (95% CI: 1.013–1.078), and an AUC of 0.784 (95% CI: 0.678–0.949) at an optimal cut-off of 5.455 ng/mL. Although their investigation assessed day-5 PCT concentrations rather than admission PCT doubling, both studies identified significant associations between elevated PCT and adverse clinical outcomes. The present findings suggest that CRP and PCT may provide complementary prognostic information alongside SOFA scoring. However, the relatively small number of mortality events and the absence of external validation warrant cautious interpretation before these markers are incorporated into routine mortality prediction models.[14]

CONCLUSION

The present study demonstrated that elevated serum C-reactive protein (CRP) and procalcitonin (PCT) levels were significantly associated with increased disease severity, organ dysfunction, and in-hospital mortality among patients with bacterial sepsis. Serial reductions in both biomarkers were greater among survivors, indicating their potential usefulness in monitoring clinical progression and treatment response. Procalcitonin demonstrated numerically better prognostic accuracy than CRP, although the difference was not statistically significant. SOFA score, CRP, and PCT were independently associated with mortality, suggesting that combining inflammatory biomarkers with clinical severity assessment may improve risk stratification and support timely clinical decision-making.

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