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Clinical Utility of Procalcitonin for Stratifying Severity in Sepsis Secondary to Pneumonia

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ABSTRACT

Introduction: Procalcitonin (PCT) is a biomarker that reflects the systemic inflammatory response to bacterial infections and has been widely studied in the context of sepsis. In patients with pneumonia, early identification of the severity of sepsis is essential for appropriate clinical management and prognostic evaluation. This study aimed to assess the clinical utility of serum procalcitonin levels in stratifying sepsis severity in patients with pneumonia.

Methods: This analytical cross-sectional study was conducted between February 2013 and March 2014 in the Emergency Department and Internal Medicine Wards of Dr. Zainoel Abidin General Hospital in Banda Aceh, Indonesia. Thirty patients diagnosed with pneumonia were enrolled in this study using quota sampling method. Serum procalcitonin levels were measured and categorized according to the severity of sepsis. Statistical analysis was performed using one-way analysis of variance (ANOVA), followed by the least significant difference (LSD) post-hoc test using the SPSS software. Statistical significance was set at $P < 0.05$.

Results: Serum procalcitonin levels progressively increased with increasing severity of sepsis. The lowest PCT level was observed in patients with pneumonia without sepsis (0.091 ng/mL), followed by those with sepsis (0.686 ng/mL), severe sepsis (3.593 ng/mL), and septic shock (21.703 ng/mL). Significant differences in PCT levels were found across the severity groups ($P < 0.05$), indicating a strong relationship between elevated procalcitonin levels and worsening clinical severity.

Conclusion: Serum procalcitonin levels correlate with the severity of sepsis in patients with pneumonia and may serve as a useful biomarker for sepsis severity stratification and clinical risk assessment.

Keywords

Procalcitonin, Sepsis Severity, Pneumonia, Biomarkers, Systemic Infection, Septic Shock.

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INTRODUCTION

Respiratory diseases are a major cause of morbidity and mortality worldwide. Among these conditions, pneumonia is one of the most prevalent and clinically significant acute lower respiratory tract infections, accounting for approximately 15–20% of respiratory disease cases worldwide. Pneumonia is defined as inflammation of the lung parenchyma and distal airways, including the terminal bronchioles, and is commonly caused by bacterial infections. Epidemiological data from the United States indicate that bacterial pneumonia accounts for more than half of the reported pneumonia cases, with a prevalence of 56.4% between 1979 and 2000, underscoring its persistent burden on public health [1,2].

Pneumonia is recognized as one of the leading infectious causes of sepsis in various healthcare settings. A national prospective multicenter study conducted in Germany demonstrated that community-acquired pneumonia was the most frequent cause of sepsis, accounting for 527 (39.1%) of 1,348 cases in that study.

Hospital-acquired pneumonia accounted for 186 cases (13.8%), ventilator-associated pneumonia for 443 cases (32.9%), and pneumonia of unknown origin for 192 cases (14.2%) [3]. Sepsis itself remains a major cause of mortality, with an estimated 400,000–500,000 deaths annually in the United States. Long-term surveillance data from 750 hospitals over a 22-year period revealed approximately 10 million sepsis cases, with incidence rates increasing from 82.7 per 100,000 population in 1979 to 240.4 per 100,000 population in 2000, highlighting its growing clinical and public health impact [4]. Given the high mortality associated with sepsis in hospitalized patients, an accurate and timely assessment of disease severity is essential for guiding clinical decision-making and prognostication. This clinical demand has prompted the development of novel biomarkers that complement the conventional clinical evaluations and laboratory tests. Procalcitonin (PCT) has emerged as a promising biomarker for both early detection and severity assessment of sepsis, and its clinical utility has been extensively evaluated in diverse patient populations [5-7]. Previous studies have demonstrated a stepwise increase in serum procalcitonin concentrations in parallel with increasing sepsis severity. Meisner reported that PCT concentrations typically range from 0.05–0.5 ng/mL in non-septic infections, increase to 0.5–2 ng/mL in sepsis, rise further to 2–10 ng/mL in severe sepsis, and exceed 10 ng/mL in septic shock, supporting its role in severity stratification [8].

Beyond its diagnostic value in acute bacterial infections, serum procalcitonin measurement has been widely applied to differentiate systemic inflammatory response syndrome (SIRS) from sepsis and to monitor the treatment response during antimicrobial therapy [2]. Compared with other inflammatory biomarkers, such as tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), interleukin-1 (IL-1), and C-reactive protein (CRP), procalcitonin has demonstrated superior performance in reflecting disease severity and predicting clinical outcomes, reinforcing its prognostic significance in patients with sepsis [9]. Although elevated serum procalcitonin levels have consistently been observed in patients with sepsis compared to non-septic individuals, some studies have reported limitations in establishing a universally accepted threshold value for sepsis diagnosis, indicating ongoing uncertainty regarding its optimal clinical application [10]. Procalcitonin is a precursor peptide of calcitonin that is released in response to systemic inflammatory stimuli, particularly during bacterial infection. Its serum concentration has been shown to correlate closely with disease severity, and rapid declines following antibiotic administration in patients with community-acquired pneumonia suggest a dynamic relationship between inflammatory burden and procalcitonin kinetics [11]. Collectively, accumulating evidence supports the role of procalcitonin as a valuable biomarker for early sepsis detection and severity assessment, particularly in pneumonia-associated sepsis [12,13].

METHOD

This analytical cross-sectional study was conducted in the Emergency Department and Internal Medicine inpatient wards of Dr. Zainoel Abidin Regional General Hospital, Banda Aceh, Indonesia. Data were collected over a one-year period, from February 2013 to March 2014. Patients aged ⩾18 years who were diagnosed with pneumonia complicated by sepsis were eligible for inclusion in the study. Additional inclusion criteria included a hemoglobin level of at least 8 g/dL and provision of informed consent obtained either directly from the patient when conscious or from a legally authorized family member when the patient was unable to provide consent. Patients were excluded if they had conditions known to influence serum procalcitonin levels or confound inflammatory responses, including pancreatitis, thyroid carcinoma, severe trauma, lung carcinoma, and meningitis. A total of 30 patients who met the inclusion criteria were enrolled in this study.

Pneumonia was diagnosed according to the Indonesian National Guidelines for Pneumonia Diagnosis and Management. Diagnosis was primarily based on clinical history and physical examination findings. The clinical symptoms included fever, productive cough with purulent sputum (occasionally blood-streaked), dyspnea, and chest pain. Physical examination findings included asymmetric chest movement on the affected side, increased tactile fremitus on palpation, dullness on percussion over the involved lung areas, and abnormal breath sounds ranging from bronchovesicular to bronchial breathing accompanied by crackles. Clinical diagnosis without routine chest radiography was performed according to institutional practice and national clinical guidelines. Venous blood samples were collected from all enrolled patients under aseptic conditions.

Blood samples (3 mL) were collected in EDTA-containing tubes for complete blood count analysis, and an additional 3 mL was collected in plain tubes without anticoagulant for serum separation. Serum procalcitonin levels were measured using an enzyme-linked fluorescent assay (ELFA) with the BRAHMS PCT assay (VIDAS) according to the manufacturer's instructions. A reference value of <0.05 ng/mL was used. Based on serum procalcitonin concentrations, patients were classified into five categories: normal (<0.05 ng/mL), non-septic infection (0.05–0.5 ng/mL), sepsis (0.5–2.0 ng/mL), severe sepsis (2.0–10.0 ng/mL), and septic shock (>10.0 ng). Statistical analyses were performed using a 95% confidence interval, with a significance level set at $\alpha = 0.05$. Associations between clinical variables and sepsis severity were analyzed, including systolic blood pressure, heart rate, respiratory rate, body temperature, mental status, inflammatory markers, and organ function. One-way analysis of variance (ANOVA) and Spearman's correlation tests were applied as appropriate. All statistical analyses were performed using the SPSS software (IBM Corp., Armonk, NY, USA).

RESULTS

The baseline demographic and clinical characteristics of the study population are summarized in Table 1

Table 1. Baseline Clinical Characteristics of the Study Population (n = 30)

Characteristic	Value
Male/Female, n (%)	17 (56.7) / 13 (43.3)
Age, years	58.3 ± 12.5
Level of consciousness, n (%)	CM 40.0; Ap 46.7; So 6.7; St 6.7
Sepsis severity, n (%)	Sepsis 40.0; Severe 26.7; Shock 33.3
SBP, mmHg	106.7 ± 24.5
Heart rate, bpm	106 (90–122)
Respiratory rate, bpm	28 (25–40)
Temperature, °C	38.0 (36.4–39.2)
Hemoglobin, g/dL	10.1 (8.5–13.5)
Leukocytes, /µL	14.1 ± 4.4 ×10³
Platelets, /µL	158 (65–660) ×10³
Creatinine, mg/dL	1.75 (0.57–7.30)
Urine output, mL/24 h	125 (50–800)
Procalcitonin, ng/mL	24.6 (0.45–192.7)

Notes: CM, compos mentis; Ap, apathetic; So, somnolent; St, stupor.

Comparisons of vital signs and clinical variables across the sepsis severity categories are presented in Table 2.

Table 2. Sepsis Severity and Clinical Variables

Variable	Sepsis	Severe	Shock	p
SBP*, mmHg	115 (100–150)	125 (100–150)	80 (60–90)	<0.001
Heart rate*, bpm	98 (90–122)	103 (98–110)	120 (110–122)	<0.001
Respiratory rate, bpm	27 (25–34)	30 (26–40)	30 (26–38)	0.059
Temperature, °C	38.0 ± 0.8	38.1 ± 0.9	37.9 ± 1.0	0.883
Mental status*, n	CM 12	Ap 8	Ap 6 / So 2 / St 2	0.001

Notes: Data are presented as *median (range)* or *mean ± SD*. *Statistical significance was set at P < 0.05.*

One-way analysis of variance demonstrated a statistically significant association between serum procalcitonin levels and the severity of sepsis ($p < 0.001$). The median procalcitonin level increased progressively with worsening sepsis severity. In contrast, the leukocyte counts did not differ significantly among the severity groups. Significant associations were observed between sepsis severity and renal function. Serum creatinine levels and urine output differed significantly across the severity categories, whereas platelet counts showed no significant association.

Table 3. Sepsis Severity and Inflammatory Markers

Variable	Sepsis	Severe	Shock	p
*PCT, ng/mL**	0.85 (0.62–1.21)	5.66 (4.24–7.95)	35.09 (20.95–70.48)	<0.001
Leukocytes, ×10³/µL	13.0 (4.2–22.1)	14.5 (12.0–23.4)	16.1 (2.0–19.0)	0.307

Notes: Values are presented as medians (ranges). Statistical significance was set at P < 0.05.

Table 4. Sepsis Severity and Organ Function Parameters

Variable	Sepsis	Severe	Shock	p
*Serum creatinine, mg/dL**	0.95 (0.57–1.70)	2.10 (1.80–2.50)	1.80 (0.80–7.30)	<0.001
Platelets, ×10³/µL	215 (106–455)	140 (106–420)	145 (65–660)	0.285
*Urine output, mL/24 h**	320 (100–800)	100 (50–280)	100 (50–200)	0.001

Notes: Values are presented as medians (ranges). Statistical significance was set at P < 0.05.

A stepwise increase in serum procalcitonin levels was observed across the sepsis severity groups, with statistically significant differences between all groups.

Table 5. Comparison of Procalcitonin Levels Among Sepsis Severity Groups

Group	n	PCT, ng/mL (median)
Sepsis	12	0.85[a]
Severe Sepsis	8	5.66[b]
Septic Shock	10	35.09[c]

Note: Different superscript letters indicate statistically significant differences (P < 0.05).

Table 6. One-Way ANOVA of Procalcitonin Levels Among Sepsis Severity Groups

Group	n	95% CI (min–max), ng/mL	p (ANOVA)
Sepsis	12	0.62–1.21	
Severe Sepsis	8	4.24–7.95	<0.001
Septic Shock	10	20.95–70.48	

Note: One-way ANOVA, p < 0.001.

Spearman's correlation analysis demonstrated strong correlations between sepsis severity and serum procalcitonin levels, creatinine, urine output, systolic blood pressure, heart rate, respiratory rate, and mental status. Leukocyte and platelet counts and body temperature were not significantly correlated with the severity of sepsis.

Table 7. Spearman Correlation with Sepsis Severity

Variable	p	r
PCT*	<0.001	0.939
Leukocytes	0.127	0.285
Creatinine*	0.003	0.520
Platelets	0.117	−0.292
Urine Output*	<0.001	−0.646
Sbp*	<0.001	−0.698
Heart Rate*	<0.001	0.697
Respiratory Rate*	0.033	0.390
Temperature	0.762	−0.058
Mental Status*	<0.001	0.915

Notes: Spearman's test; $p < 0.05$ Indicates Significance.

DISCUSSION

This study evaluated the role of serum procalcitonin levels in assessing sepsis severity in patients with pneumonia. The results indicated that key clinical parameters, including the level of consciousness, systolic blood pressure, heart rate, respiratory rate, and body temperature, as well as inflammatory markers such as leukocyte count and procalcitonin levels, showed mean values that exceeded the normal ranges. These findings are consistent with the established diagnostic criteria for sepsis. According to the consensus definitions proposed by the American College of Chest Physicians (ACCP) and the Society of Critical Care Medicine (SCCM), abnormalities in vital signs and inflammatory markers are fundamental components for identifying and classifying sepsis, supporting the clinical relevance of the variables evaluated in this study [14-17].

In this study, increased serum procalcitonin levels were accompanied by progressively higher degrees of sepsis. This pattern is consistent with the findings reported by Brunkhorst et al. in a multicenter study conducted in Germany involving 185 patients across the spectrum of systemic inflammatory response syndrome (SIRS), sepsis, severe sepsis, and septic shock. That study reported mean procalcitonin levels of 0.53 ± 2.89 ng/mL in sepsis, 6.91 ± 3.87 ng/mL in severe sepsis, and 12.89 ± 4.39 ng/mL in septic shock, demonstrating a significant stepwise increase in disease severity [18,19]. Similar results were reported by Harbarth et al. in a study conducted in Geneva involving 78 patients, which showed progressively higher median procalcitonin concentrations across severity categories, including 3.5 ng/mL in sepsis, 6.2 ng/mL in severe sepsis, and 21.3 ng/mL in septic shock [20]. These consistent findings across different populations further support procalcitonin as a reliable biomarker of sepsis severity. Distinct procalcitonin threshold ranges corresponding to increasing sepsis severity were identified in this study. Serum PCT levels ranged from 0.62–1.21 ng/mL in sepsis, increased to 4.24–7.95 ng/mL in severe sepsis, and reached markedly higher values of 20.95–70.48 ng/mL in patients with septic shock. These findings are in agreement with the data reported by Meisner, who analyzed seven studies involving 145 patients and demonstrated a progressive increase in procalcitonin concentrations across all stages of sepsis, with the highest levels observed in patients with septic shock. Meisner reported PCT concentrations of 0.05–0.5 ng/mL in non-septic infections, 0.5–2.0 ng/mL in sepsis, 2.0–10.0 ng/mL in severe sepsis, and >10 ng/mL in septic shock [8]. Comparable trends were also described by Dorizzi et al. in an Italian cohort of 103 patients and by Bourboulis et al. in a large Greek cohort of 1,156 patients, both of whom demonstrated increasing procalcitonin levels with disease progression and organ dysfunction [21]. Collectively, these findings reinforce the concept that procalcitonin reflects the presence and severity of sepsis in a dose-dependent manner.

The biological plausibility of procalcitonin as a sepsis biomarker is further supported by experimental and clinical evidence. Meisner reported that serum procalcitonin levels may begin to rise as early as 2–3 h following inflammatory induction and can increase to several hundred nanograms per milliliter, while remaining a highly stable molecule, both in vitro and in vivo. In animal models of sepsis, procalcitonin levels increase markedly within 24 h, accompanied by parallel but less pronounced elevations in inflammatory mediators, such as interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α), highlighting the rapid and robust response of procalcitonin to systemic bacterial inflammation [22]. These findings are consistent with the immunological mechanisms underlying procalcitonin production. Procalcitonin mRNA is expressed in human peripheral blood mononuclear cells and can be stimulated by various proinflammatory cytokines as well as bacterial lipopolysaccharide (LPS). Under physiological conditions, some lymphocytes and monocytes produce basal levels of procalcitonin; however, exposure to bacterial LPS markedly increases this production. Notably, monocytes isolated from patients with septic shock exhibit elevated baseline procalcitonin levels and an exaggerated response to LPS stimulation, reflecting dysregulated immune activation during severe sepsis [23]. This study demonstrated a significant positive correlation between sepsis severity and serum procalcitonin levels in patients with pneumonia-induced sepsis. These results are consistent with the findings reported by Elkhashab et al. from the University of Alexandria Hospital, who showed significantly higher procalcitonin levels in patients with pneumonia and sepsis and concluded that procalcitonin serves as both a specific biomarker and prognostic indicator in hospitalized patients with pneumonia [24].

Additional studies by Balci et al. in Turkey and Castelli et al. in Italy further support the diagnostic and prognostic value of procalcitonin in differentiating sepsis severity and inflammatory states, with statistically significant differences observed across the severity categories [25,26]. Moreover, Jeong et al. demonstrated that procalcitonin concentrations were significantly higher in patients with sepsis than in those without, reinforcing its role as an early inflammatory marker of sepsis [27]. The high precision and accuracy of procalcitonin measurement make it a valuable tool for early sepsis detection and severity prediction, facilitating timely clinical intervention and risk stratification in patients with sepsis. [28]. This study had some limitations that should be considered. The cross-sectional design limited the assessment of temporal changes in procalcitonin levels and precluded causal inferences. Potential confounding factors influencing serum procalcitonin levels have not been comprehensively evaluated. In addition, pneumonia diagnosis was primarily based on clinical history and physical examination without routine chest radiography. However, radiographic confirmation is recommended by the Infectious Diseases Society of America/American Thoracic Society (IDSA/ATS) guidelines [29]. Several studies have supported the use of clinical diagnostic criteria alone, particularly in resource-limited settings.

CONCLUSION

Serum procalcitonin levels increase stepwise with sepsis severity in patients with pneumonia, with distinct threshold values observed between pneumonia and septic shock. The strong association between elevated PCT levels and disease severity reflects the underlying inflammatory burden and organ dysfunction. These findings support the use of procalcitonin as a reliable biomarker for severity stratification and clinical risk assessment in pneumonia-associated sepsis.

DECLARATIONS

This study was approved by the Ethics Committee of Dr. Zainoel Abidin Regional General Hospital, Banda Aceh, Indonesia. All procedures involving human participants were conducted in accordance with the principles of the Declaration of Helsinki. The relatively small sample size may limit the generalizability of our findings.

CONSENT FOR PUBLICATION

The Authors agree to the publication in the Journal of Society Medicine.

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COMPETING INTERESTS

The authors declare no conflict of interest.

AUTHORS' CONTRIBUTIONS

W.H.S. and A.P. conceived and designed this study. W. H. S. and W. performed the data collection and laboratory analyses. All authors conducted data analysis and interpretation. All authors participated in manuscript drafting and critical revision, approved the final version, and agreed to be held accountable for all aspects of the work.

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