Association of CRP/Troponin Ratio with Mortality in Pneumosepsis Patients Admitted from the Emergency Department

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Abstract

Aim: This study aimed to investigate the association between the C-reactive protein (CRP)/troponin ratio at ICU admission and mortality in patients with pneumosepsis admitted from the emergency department (ED).

Methods: This retrospective, single-center cohort study included 40 adult patients admitted to the intensive care unit with pneumosepsis between September 2024 and September 2025. Demographic characteristics, comorbidities, laboratory parameters at admission (including CRP and high-sensitivity troponin), disease severity scores (APACHE II and SOFA), need for respiratory support, inotrope use, and ICU outcomes were collected from electronic medical records. Patients were stratified into survivor and non-survivor groups. Continuous variables were compared using the Mann–Whitney U test, and categorical variables using the chi-square or Fisher's exact test.

Results: ICU mortality occurred in 23 patients (57.5%), whereas 17 patients (42.5%) survived to hospital discharge. The CRP/troponin ratio was significantly lower in non-survivors compared with survivors (3.7 [IQR: 1.4–8.7] vs. 8.9 [IQR: 4.0–51.8], p = 0.006). Troponin levels were significantly higher in the non-survivor group (24.4 vs. 9.7 ng/L, p = 0.005). Non-survivors required longer durations of invasive mechanical ventilation (p < 0.001) and more frequent inotrope support (p = 0.004). APACHE II and SOFA scores were higher in non-survivors, although these differences did not reach statistical significance.

Conclusions: A lower CRP/troponin ratio at ICU admission was significantly associated with increased mortality in pneumosepsis patients admitted from the emergency department. This ratio may serve as a simple and accessible biomarker for early risk stratification in this high-risk population.

Keywords: *Sepsis; pneumonia; troponin; C-reactive protein; mortality*

1. Introduction

Pneumosepsis, defined as sepsis secondary to pneumonia, remains one of the leading causes of intensive care unit (ICU) admissions worldwide and is associated with substantial morbidity and mortality.[1-3] Sepsis, characterized by a dysregulated host response to infection resulting in life-threatening organ dysfunction, continues to account for a significant proportion of ICU deaths, with reported mortality rates ranging between 25% and 40% despite advances in supportive care and antimicrobial therapy.[4,5] Early identification of patients at high risk of death is therefore crucial to guide timely interventions, optimize resource allocation, and potentially improve outcomes.[6,7]

Among the biomarkers routinely measured in septic patients, C-reactive protein (CRP) serves as a widely used indicator of systemic inflammation and correlates with infection severity, while cardiac troponins reflect myocardial injury, which is frequently observed in sepsis and portends a worse prognosis.[8-10] Traditionally, these parameters have been evaluated independently; however, integrating them into a ratio (CRP/troponin) may provide a more comprehensive assessment of the balance between systemic inflammatory response and cardiac involvement.

Emerging evidence suggests that a low CRP/troponin ratio may indicate either inadequate systemic inflammation (immune dysfunction) or disproportionate myocardial injury, both of which are linked to adverse outcomes.[11,12] Nonetheless, studies directly evaluating the prognostic value of the CRP/troponin ratio in septic populations are limited, and data specific to pneumosepsis patients—particularly those admitted from the emergency department (ED)—are scarce.[12,13]

Therefore, the present study aimed to investigate the prognostic significance of the CRP/troponin ratio in pneumosepsis patients admitted to the ICU from the ED and to explore its association with in-hospital mortality.

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2. Materials and Methods

2.1. Study Design and Setting

This study was designed as a single-center, retrospective, observational cohort study. It was conducted in the tertiary care adult intensive care units (ICUs) of Eskişehir City Hospital, a high-volume referral center with over 120 ICU beds, and its emergency department (ED). The study period covered patients admitted between September 1, 2024, and September 1, 2025.

2.2. Patient Selection

Eligible participants were adults aged 18 years or older who were admitted to the ICU directly from the ED with clinically and radiologically confirmed pneumosepsis and had complete electronic medical records. Patients younger than 18 years, those admitted to the ICU for other indications, those transferred to another center before completion of ICU treatment, and those with missing or incomplete data were excluded from the analysis.

2.3. Data Collection

Laboratory parameters obtained at ICU admission included complete blood count, serum glucose, blood urea nitrogen, creatinine, C-reactive protein (CRP), procalcitonin, ferritin, B-type natriuretic peptide, D-dimer, and cardiac biomarkers. Cardiac troponin levels were measured using a high-sensitivity cardiac troponin I (hs-cTnI) assay (Abbott ARCHITECT STAT High Sensitive Troponin-I, Abbott Diagnostics, USA). The upper reference limit (99th percentile) for hs-cTnI in our laboratory was 34 ng/L for males and 16 ng/L for females, in accordance with the manufacturer's recommendations. The CRP/troponin ratio was calculated by dividing the CRP value (mg/L) by the hs-cTnI level (ng/L) obtained at ICU admission. Treatment-related data such as the need and duration of mechanical ventilation, high-flow nasal cannula (HFNC) support, mask or nasal cannula oxygen therapy, and inotrope use were also recorded. Outcomes included length of ICU stay and ICU/hospital mortality.

2.4. Statistical Analysis

Normality of distribution was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Continuous variables were non-normally distributed and are therefore presented as median and interquartile range (IQR). Comparisons between survivors and non-survivors were performed using the Mann–Whitney U test for continuous variables. Categorical variables were summarized as counts and percentages and compared using the chi-square or Fisher's exact test as appropriate. A two-sided p value <0.05 was considered statistically significant. Statistical analyses were performed using SPSS software, version 22.0 (IBM Corp, Armonk, NY, USA).

3. Results

A total of 40 patients admitted to the ICU with a diagnosis of pneumosepsis from the emergency department during the study period were included in the analysis. The median age of the study cohort was 69 years (IQR: 62–80), and 41.2% of the patients were male. The most common comorbidities were diabetes mellitus (n=13, 32.5%), hypertension (n=13, 32.5%), chronic obstructive pulmonary disease (COPD) (n=12, 30%), coronary artery disease (n=8, 20%), and chronic kidney disease (n=3, 7.5%). The median APACHE II and SOFA scores on admission were 16 (IQR: 12–20) and 29.1 (IQR: 17.1–38.9), respectively. Laboratory and clinical characteristics of the overall study population are summarized in **Table 1.**

ICU mortality occurred in 23 patients (57.5%), whereas 17 patients (42.5%) survived to hospital discharge. Comparisons between survivors and non-survivors are presented in **Table 2.** The CRP/troponin ratio was significantly lower among non-survivors compared with survivors (3.7 [IQR: 1.4–8.7] vs. 8.9 [IQR: 4.0–51.8], p=0.006). Troponin levels were also significantly higher in non-survivors (24.4 [11.8–51.2] vs. 9.7 [6.0–19], p=0.005).

Regarding ICU course, non-survivors had a significantly longer duration of invasive mechanical ventilation (p<0.001) and a greater need for inotropic support (p=0.004). In contrast, the duration of oxygen therapy via face mask and nasal cannula was longer in survivors (p=0.001 and p<0.001, respectively). Although APACHE II and SOFA scores were numerically higher in non-survivors compared with survivors (median APACHE II: 18 vs. 16; median SOFA: 29.1 vs. 23.5), these differences did not reach statistical significance (p = 0.107 and p = 0.090, respectively). This lack of statistical significance is most likely attributable to the limited sample size and reduced statistical power, suggesting a potential type II error, rather than the absence of a clinically meaningful association.

Table 1

Baseline Demographic, Clinical, and Laboratory Characteristics of the Study Population

	Value (Median [IQR] or n [%])
Age, (years)	69 (62–80)
Gender (male)	17 (41.2)
Comorbidities	
Diabetes mellitus	13 (32.5)
Hypertension	13 (32.5)
Chronic obstructive pulmonary disease	12 (30.0)
Coronary artery disease	8 (20.0)
Chronic kidney disease	3 (7.5)
APACHE II score	16 (12–20)
SOFA score	29.1 (17.1–38.9)
Laboratory parameters	
CRP, mg/L	122.9 (63.7–193.9)
Hs Troponin I, ng/L	16.7 (8.6–36.9)
CRP/Troponin ratio	5.7 (2.1–18.8)
Procalcitonin, ng/mL	0.3 (0.1–1.1)
Ferritin, ng/mL	478.5 (205.7–908.7)
WBC, $\times 10^9$/L	11.2 (7.4–15.3)
Hemoglobin, g/dL	12.7 (10.7–13.9)
Platelet count, $\times 10^3$/μL	239 (192–326)
Glucose, mg/dL	139 (116.5–265.5)
BNP, pg/mL	104.1 (47.5–324.2)
D-dimer, mg/L	1.7 (1.0–4.1)
BUN, mg/dL	23.4 (17.8–32.2)
Creatinine, mg/dL	0.8 (0.7–1.1)
ICU course	
Length of ICU stay, days	10 (5.2–18.5)
Duration of invasive MV, days	1 (0–4.7)
Duration of HFNC, days	2.5 (0–6.7)
Duration of mask O_2, days	3 (0–6)
Duration of nasal cannula O_2, days	0 (0–2)
Duration of inotrope use, days	1.5 (0–3)

Abbreviations: APACHE II = Acute Physiology and Chronic Health Evaluation II; BNP = B-type natriuretic peptide; BUN = blood urea nitrogen; CRP = C-reactive protein; HFNC = high-flow nasal cannula; ICU = intensive care unit; IQR = interquartile range; MV = mechanical ventilation; SOFA = Sequential Organ Failure Assessment; WBC = white blood cell.

Table 2

Comparison of Clinical, Laboratory, and Outcome Parameters Between Survivors and Non-Survivors

	Survivors (n = 17)	Non-Survivors (n = 23)	p value
Age, years	66 (62–80)	69 (62–80)	0.645
Male sex, n (%)	7 (41.2)	13 (56.5)	0.337
APACHE II score	16 (12–20)	18 (16–22)	0.107
SOFA score	23.5 (14.7–35.6)	29.1 (23.5–42.4)	0.090
Laboratory parameters			
CRP, mg/L	139.5 (65.6–275.5)	107.5 (63.6–179.3)	0.182
Hs Troponin I, ng/L	9.7 (6.0–19)	24.4 (11.8–51.2)	0.005
CRP/Troponin ratio	8.9 (4.0–51.8)	3.7 (1.4–8.7)	0.006
Procalcitonin, ng/mL	0.2 (0.1–0.7)	0.4 (0.1–2.1)	0.143
Ferritin, ng/mL	379 (199–617)	631 (318–1492)	0.173
WBC, $\times 10^9$/L	10.5 (7.1–15.0)	13.0 (7.3–16.3)	0.645
Hemoglobin, g/dL	12.0 (10.1–15.1)	12.9 (11.3–14.5)	0.277
Platelet count, $\times 10^3/\mu L$	238 (190–283)	240 (196–334)	0.859
Glucose, mg/dL	139 (116.5–265.5)	158 (118–271)	0.705
BNP, pg/mL	76.7 (25.3–224.4)	174.8 (60.4–948.5)	0.075
BUN, mg/dL	22.8 (17.5–30.6)	26.3 (19.2–32.6)	0.374
Creatinine, mg/dL	0.8 (0.7–1.0)	0.9 (0.7–1.2)	0.464
ICU course			
Length of ICU stay, days	15 (5.5–21.5)	10 (5–16)	0.254
Duration of invasive MV, days	0 (0–0)	3 (1–7)	<0.001
Duration of HFNC, days	4 (0–9)	2 (0–5)	0.277
Duration of mask O_2, days	6 (3–10)	0 (0–5)	0.001
Duration of nasal cannula O_2, days	2 (1–3)	0 (0–0)	<0.001
Duration of inotrope use, days	0 (0–2)	2 (1–5)	0.004

Abbreviations: APACHE II = Acute Physiology and Chronic Health Evaluation II; BNP = B-type natriuretic peptide; BUN = blood urea nitrogen; CRP = C-reactive protein; HFNC = high-flow nasal cannula; ICU = intensive care unit; IQR = interquartile range; MV = mechanical ventilation; SOFA = Sequential Organ Failure Assessment; WBC = white blood cell.

4. Discussion

In this retrospective cohort of 40 patients with pneumosepsis admitted from the Emergency Department (ED) to the ICU, our findings indicate that the CRP/troponin ratio at admission is significantly associated with ICU mortality. Non-survivors had a markedly lower median CRP/troponin ratio than survivors (3.7 vs. 8.9, p=0.006), suggesting that when myocardial injury (as reflected by troponin) is proportionally greater than systemic inflammatory response (as reflected by CRP), outcomes are worse. To our knowledge, there is no previous large-scale study specifically examining the prognostic value of the CRP/troponin ratio in pneumosepsis, although associations between troponin, CRP, and outcomes in sepsis have been described.[12,14]

Previous studies have extensively evaluated troponin elevation in sepsis as an independent prognostic factor.[15-17] For instance, Zheng et al. found that elevated cardiac troponin levels were strong predictors of both hospital and long-term mortality in septic patients.[16] Similarly, Frencken et al. demonstrated that myocardial injury is associated with increased case fatality in sepsis and that high troponin levels were linked to both short-term and long-term adverse outcomes.[17] Our findings align with these observations, as troponin levels were significantly higher among non-survivors (24.4 vs. 9.7, p=0.005). Moreover, Ostermann et al. demonstrated that troponin release is linked not only to myocardial strain but also to biomarkers of systemic inflammation, including CRP, suggesting a complex interaction between cardiac injury and inflammatory response.[14] This supports the concept that integrating troponin with CRP into a ratio might offer a more comprehensive picture of disease severity.

The use of ratios for risk stratification in sepsis has precedent. For example, the CRP/albumin ratio has been validated as a prognostic biomarker in large cohorts and incorporated into predictive nomograms.[18] Similar to CRP/albumin, the CRP/troponin ratio may capture the balance between systemic inflammation and end-organ damage, potentially outperforming either marker alone in prognostic accuracy. Our data provide preliminary evidence that such a ratio has clinical relevance and merits further investigation in larger, prospective .[18]

Moreover, our study shows that non-survivors also had significantly higher troponin levels (24.4 vs. 9.7, p=0.005), greater dependency on invasive mechanical ventilation (p<0.001), and more frequent inotropic support (p=0.004). These findings are consistent with the pathophysiology of severe sepsis, where cardiac dysfunction and respiratory failure impose additive risks. Yang et al. identified troponin-I elevation as an independent predictor of short-term mortality in septic shock patients presenting to the ED, particularly when combined with clinical scoring systems.[11]

In terms of comorbidities, diabetes mellitus, hypertension, COPD, coronary artery disease, and chronic kidney disease were common in our population, reflecting the typical multimorbidity observed in ICU pneumosepsis patients. These comorbidities did not differ significantly between survivors and non-survivors, suggesting that acute biomarkers and physiological derangements may outweigh baseline comorbidity burden in short-term prognostic discrimination. This observation is consistent with prior reports in which troponin elevation was more strongly associated with mortality than underlying chronic disease burden.[19]

While scores such as APACHE II and SOFA are established tools for mortality prediction in critical illness and sepsis, in our data both were numerically higher among non-survivors but did not reach statistical significance. This lack of statistical significance is most likely attributable to the limited sample size and the resulting risk of type II error rather than the absence of a true clinical association, as the observed trends were consistent with established prognostic expectations in sepsis. Studies such as those by Zheng et al. and Lörstad et al. have found that troponin measured at admission, particularly high-sensitivity troponin, is associated with 30-day to 1-year mortality in sepsis, even when controlling for established clinical risk factors.[16,20]

From a clinical standpoint, the CRP/troponin ratio could be adopted as a rapid, cost-effective tool for initial risk stratification in pneumosepsis patients presenting to the ED. Although our study was not designed to establish a definitive cut-off value for the CRP/troponin ratio, exploratory evaluation suggested that lower ratios were consistently associated with poor outcomes. However,

due to the limited sample size, any proposed threshold would lack sufficient statistical robustness and therefore requires validation in larger, multicenter prospective studies before clinical implementation.

This study has several limitations. First, its retrospective and single-center design may limit the generalizability of the findings. Second, the relatively small sample size (n = 40) restricts statistical power and increases the risk of type II error. Importantly, this limited sample size precluded the performance of multivariate regression analysis; therefore, the independent predictive value of the CRP/troponin ratio could not be verified after adjustment for potential confounders. Additionally, only admission biomarker values were analyzed, and serial measurements over time were not available. Finally, causality cannot be inferred due to the observational nature of the study.

5. Conclusion

Our findings indicate that the CRP/troponin ratio measured at ICU admission is significantly associated with ICU mortality among patients with pneumosepsis admitted from the Emergency Department. Troponin elevation, prolonged mechanical ventilation, and increased inotrope requirement were also significant markers of poor prognosis. These results suggest that the CRP/troponin ratio may serve as a simple, inexpensive, and readily available tool for early risk stratification in this high-risk population. However, due to the retrospective single-center design and limited sample size of this study, these findings should be interpreted with caution and require confirmation in larger, multicenter prospective cohorts before routine clinical implementation.

Statement of ethics

The study protocol was reviewed and approved by the Institutional Ethics Committee of City Hospital (Approval No: ESH/BAEK 2025/225, Date: 11/09/2025). As this was a retrospective chart review, the requirement for informed consent was waived. The study was conducted in accordance with the Declaration of Helsinki and local regulatory requirements.

genAI

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Conflict of interest statement

The authors declare that they have no conflict of interest.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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