MAK HILL Publications Research Journal of Medical Sciences

Research Article
doi: 10.36478/makrjms.2025.4.143.147

OPEN ACCESS

Key Words
Cardiac injury, sepsis, troponin, echocardiography, blood culture

Corresponding Author
C. Senthil,
Department of General Medicine, ACS Medical College and Hospital, Chennai, Tamil Nadu, India

Author Designation
[1]Postgraduate
[2]Professor and Head
[3]Assistant Professor

Received: 21st May 2025
Accepted: 10th June 2025
Published: 01st July 2025

Citation: N. Tharana Shamim, C. Senthil and Valarmathi, 2025. Assessment of Cardiac Injury in Sepsis and Its Relation to in-Hospital Mortality: A Clinical Study. Res. J. Med. Sci., 19: 143-147, doi: 10.36478/ makrjms.2025.4.143.147

Assessment of Cardiac Injury in Sepsis and Its Relation to in-Hospital Mortality: A Clinical Study

[1]N. Tharana Shamim, [2]C. Senthil and [3]Valarmathi
[1-3]Department of General Medicine, ACS Medical College and Hospital, Chennai, Tamil Nadu, India

Abstract
Sepsis is a major cause of morbidity and mortality in hospitalized patients, often involving multiple organ dysfunctions, including the heart. Myocardial injury, identified by elevated troponin levels, is increasingly recognized in sepsis, but its prognostic significance remains underutilized in routine practice. This study evaluates the incidence of cardiac injury in sepsis and its association with clinical outcomes, particularly in-hospital mortality. To assess the incidence of myocardial injury in sepsis patients using serum troponin I levels and evaluate its association with echocardiographic findings, blood culture results, ICU stay duration and in-hospital mortality. This was a hospital-based observational study conducted in the Department of Medicine, ACS Medical College and Hospital, Chennai. A total of 50 adult patients with sepsis, diagnosed as per Sepsis-3 criteria, were enrolled. Serum troponin I levels were measured within 24 hours of admission. Echocardiography was performed within 48 hours. Data on demographics, laboratory tests, ECG, blood culture, ICU stay and mortality were collected. Statistical analysis was done using SPSS software. Myocardial injury (troponin I >0.04 ng/mL) was observed in 44% of patients. In-hospital mortality was significantly higher among those with myocardial injury (45.5%) compared to those without (17.8%). Left ventricular dysfunction (EF <45%) was noted in 36% of patients with elevated troponin, versus 7% in the normal troponin group. Mean ICU stay was longer in patients with myocardial injury (7.8±2.4 days) compared to those without (4.6±1.8 days). Blood culture positivity was also more frequent in the myocardial injury group (64% vs 50%). Myocardial injury is a frequent and clinically relevant complication in sepsis, associated with increased mortality, prolonged ICU stay and cardiac dysfunction. Early detection using troponin I and echocardiography may aid in risk stratification and improve outcomes. Routine cardiac evaluation should be considered in sepsis management protocols.

INTRODUCTION

Sepsis is a life-threatening organ dysfunction resulting from a dysregulated host response to infection and remains a major cause of morbidity and mortality in critically ill patients. One of the lesser emphasized but clinically significant complications of sepsis are myocardial injury, which contributes to worsening prognosis. Myocardial dysfunction in sepsis is characterized by reversible biventricular dilatation, reduced ejection fraction, and elevated cardiac biomarkers such as troponins, even in the absence of coronary artery disease[1,2]. The pathophysiology involves inflammatory cytokine release, endothelial dysfunction, mitochondrial injury and impaired myocardial oxygen utilization[3]. Despite these insights, the routine evaluation of cardiac involvement in septic patients is not standardized in clinical practice, particularly in low-and middle-income countries. Previous studies have reported a significant association between elevated cardiac troponin levels and increased mortality in sepsis[4,5]. However, the interpretation of troponin elevation remains controversial due to its potential release from non-ischemic sources during critical illness. Echocardiographic evaluation has also been used to assess cardiac dysfunction, but findings have varied due to methodological differences and patient heterogeneity[6]. While several studies have explored sepsis-related cardiac injury, many are limited by small sample sizes, focus on specific subgroups, or lack detailed correlation with mortality outcomes in different healthcare settings. There remains a gap in systematically assessing the incidence and clinical outcomes of cardiac injury in sepsis in general hospital populations, especially in resource-limited environments. Most available data are from high-income countries, leaving uncertainty about the burden and prognostic implications in different clinical contexts. This study aims to assess the incidence of cardiac injury among patients with sepsis and to evaluate its association with in-hospital mortality using cardiac biomarkers and clinical parameters. The study also seeks to contribute region-specific data to inform early identification and management strategies for sepsis-related myocardial dysfunction.

MATERIALS AND METHODS

This hospital-based observational study was conducted in the Department of Medicine, ACS Medical College and Hospital, Velappanchavadi, Chennai, over a period of 6 months. The study included a total of 50 patients diagnosed with sepsis who were admitted to the medical wards and intensive care unit during the study period.

Study Population: Patients aged 18 years and above who fulfilled the Sepsis-3 criteria, defined as life-threatening organ dysfunction caused by a dysregulated host response to infection with a SOFA (Sequential Organ Failure Assessment) score ≥2, were included in the study.

Inclusion Criteria:

- Adult patients (≥18 years) diagnosed with sepsis.
- Willing to give informed consent or consent given by a legally authorized representative.

Exclusion Criteria:

- Patients with known structural heart disease.
- Recent history of acute coronary syndrome within the past 30 days.
- End-stage renal disease on dialysis.
- Pre-existing cardiomyopathy or chronic heart failure.

Data Collection: All participants underwent detailed clinical evaluation, including history, vital signs and systemic examination. Blood investigations were performed on admission and included:

- Complete blood count (CBC).
- Renal and liver function tests.
- Serum electrolytes.
- Blood culture.
- Serum troponin I levels (measured within 24 hours of admission).
- Electrocardiogram (ECG).
- Echocardiography (performed within 48 hours of admission).

Cardiac injury was defined based on elevated troponin I above the institutional upper reference limit (>0.04 ng/mL) in the absence of other non-septic cardiac causes. Echocardiographic assessment included measurement of ejection fraction and evaluation for regional wall motion abnormalities.

Outcome Measures: The primary outcome assessed was in-hospital mortality. Secondary outcomes included length of hospital stay and need for inotropic or ventilatory support.

Statistical Analysis: Data were entered in Microsoft Excel and analyzed using SPSS version 25. Continuous variables were expressed as mean± standard deviation (SD) and categorical variables were expressed as percentages. Chi-square test and Student's t-test were used to assess the association between cardiac injury and clinical outcomes. A p-value <0.05 was considered statistically significant.

Ethical Considerations: The study was approved by the Institutional Ethics Committee of ACS Medical College and Hospital. Informed consent was obtained from all patients or their legal guardians prior to participation.

RESULTS AND DISCUSSIONS

Table 1: Complete Blood Count Parameters (n=50)

Parameter	Mean±SD	Reference Range
Hemoglobin (g/dL)	10.4±1.8	13.0-17.0 (males)/12.0-15.0 (females)
Total Leukocyte Count (×10³/µL)	15.8±5.2	4.0–11.0
Platelet Count (×10³/µL)	165±72	150–400

The hematological parameters of patients with rhinosporidiosis revealed notable deviations from normal reference ranges. The mean hemoglobin level was 10.4±1.8 g/dL, indicating mild to moderate anemia in most cases, which may be attributed to chronic inflammation or recurrent bleeding such as epistaxis. The total leukocyte count was elevated (15.8±5.2 ×10³/µL), suggesting an active inflammatory or infectious response. Platelet counts were within the lower normal range (165±72 ×10³/µL), which, although not critically low, may reflect a systemic response to chronic infection (Table 1).

Table 2: Renal, Liver Function Tests and Electrolytes (n=50)

Parameter	Mean±SD	Reference Range
Serum Creatinine (mg/dL)	1.8±0.9	0.6–1.2
Blood Urea (mg/dL)	68±24	15–40
Total Bilirubin (mg/dL)	1.9±1.1	0.2–1.2
AST (IU/L)	78±40	5–40
ALT (IU/L)	70±36	5–45
Serum Sodium (mmol/L)	134±6	135–145
Serum Potassium (mmol/L)	4.2±0.6	3.5–5.0
Serum Chloride (mmol/L)	99±5	98–107
Serum Bicarbonate (mmol/L)	20±3	22–28

The biochemical analysis in rhinosporidiosis patients (n=50) showed evidence of renal and hepatic dysfunction along with mild electrolyte imbalances. Elevated serum creatinine (1.8±0.9 mg/dL) and blood urea (68±24 mg/dL) levels suggest varying degrees of renal impairment, potentially due to systemic infection or dehydration. Liver function tests revealed raised total bilirubin (1.9±1.1 mg/dL), AST (78±40 IU/L) and ALT (70±36 IU/L), indicating hepatic involvement or stress. Electrolyte assessment showed mild hyponatremia (134±6 mmol/L) and reduced bicarbonate (20±3 mmol/L), which may reflect dehydration, systemic inflammation, or metabolic acidosis. Serum potassium and chloride levels remained within normal limits (Table 2).

Table 3: Blood Culture Findings Among Sepsis Patients (n=50)

Culture Result	No of Patients (n)	Percentage (%)
Positive Blood Culture	28	56%
Negative Blood Culture	22	44%
Among Positive Cultures (n=28):		
-Escherichia coli	9	32.1%
-Klebsiella pneumoniae	6	21.4%
-Staphylococcus aureus (MRSA + MSSA)	5	17.9%
-Pseudomonas aeruginosa	4	14.3%
-Acinetobacter baumannii	2	7.1%
-Polymicrobial Growth	2	7.1%

Among the 50 sepsis patients studied, blood culture was positive in 28 cases (56%), indicating a significant burden of bloodstream infections. Escherichia coli was the most frequently isolated organism (32.1%), followed by Klebsiella pneumoniae (21.4%) and Staphylococcus aureus (17.9%), which included both MRSA and MSSA strains. Other notable pathogens included Pseudomonas aeruginosa (14.3%) and Acinetobacter baumannii (7.1%). Polymicrobial growth was observed in 7.1% of positive cases (Table 3).

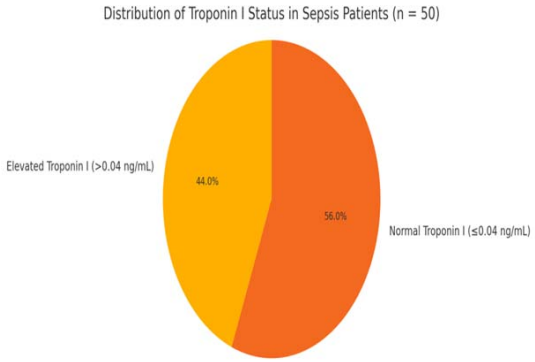

Fig. 1: Serum Troponin I Levels Among Sepsis Patients (n=50)

Among the 50 sepsis patients evaluated, elevated serum troponin I levels (>0.04 ng/mL) were observed in 22 patients (44%), indicating myocardial injury in nearly half of the cases. The remaining 28 patients (56%) had troponin I levels within the normal range (≤0.04 ng/mL). These findings suggest that sepsis is frequently associated with cardiac involvement and measuring troponin I can be a useful marker for detecting subclinical myocardial injury in such patients (Fig. 1).

Table 4: Electrocardiogram (ECG) Findings in Sepsis Patients (n=50)

ECG Abnormality	No. of Patients (n)	Percentage (%)
Normal ECG	21	42%
Sinus Tachycardia	14	28%
ST-T Segment Changes (non-specific)	6	12%
T Wave Inversions	3	6%
Atrial Fibrillation	2	4%
Ventricular Ectopics	2	4%
Prolonged QT Interval	2	4%

Electrocardiogram (ECG) findings among sepsis patients revealed abnormalities in 58% of cases, highlighting frequent cardiac involvement. Sinus tachycardia was the most common abnormality, observed in 28% of patients, likely reflecting a physiological response to infection or hypovolemia. Non-specific ST-T segment changes (12%) and T wave inversions (6%) suggested possible myocardial strain or ischemia. Less frequent but clinically significant findings included atrial fibrillation, ventricular ectopics, and prolonged QT intervals, each present in 4% of patients. A normal ECG was recorded in 42% of patients (Table 4).

Table 5: Echocardiographic Findings in Sepsis Patients (n=50)

Echocardiographic Parameter	No of Patients (n)	%
Normal LV Function (EF=55%)	26	52%
Mild Left Ventricular Dysfunction (EF 45-54%)	12	24%
Moderate to Severe LV Dysfunction (EF<45%)	7	14%
Regional Wall Motion Abnormalities (RWMA)	3	6%
Diastolic Dysfunction (Grade I/II)	9	18%
Pericardial Effusion (small, clinically insignificant)	4	8%
Right Ventricular Dysfunction	5	10%

Echocardiographic evaluation in sepsis patients revealed that 48% had some degree of cardiac dysfunction. While 52% maintained normal left ventricular (LV) function (EF ≥55%), 24% showed mild dysfunction (EF 45-54%) and 14% had moderate to severe LV dysfunction (EF <45%). Diastolic dysfunction (18%) and right ventricular dysfunction (10%) were also noted, suggesting both systolic and diastolic impairment in a subset of patients. Additional findings included regional wall motion abnormalities (6%) and small, clinically insignificant pericardial effusions (8%) (Table 5).

Table 6: Comparison of LVEF Between Patients with and Without Elevated Troponin I

Troponin I Status	No of Patients (n)	Mean LVEF (%) ± SD	LVEF < 45% (n)	Percentage with LV Dysfunction
Elevated Troponin I (>0.04 ng/mL)	22	46.8 ± 7.2	8	36.4%
Normal Troponin I (≥0.04 ng/mL)	28	56.3 ± 5.4	2	7.1%
Total	50	—	10	20%

Comparison of left ventricular ejection fraction (LVEF) between patients with and without elevated troponin I levels revealed a clear association between myocardial injury and reduced cardiac function. Patients with elevated troponin I had a significantly lower mean LVEF (46.8±7.2%) compared to those with normal troponin levels (56.3±5.4%). Among those with elevated troponin, 36.4% had LVEF <45%, indicating moderate to severe left ventricular dysfunction, whereas only 7.1% of patients with normal troponin levels showed such dysfunction[6].

Table 7: Association Between Blood Culture Positivity and Myocardial Injury

Blood Culture Status	Patients with Elevated Troponin I (n=22)	Patients with Normal Troponin I (n=28)	Total (n=50)
Positive Blood Culture	16	12	28
Negative Blood Culture	6	16	22

(Table 7) shows, the association between blood culture positivity and myocardial injury in sepsis patients indicates a notable trend. Among the 22 patients with elevated troponin I (suggestive of myocardial injury), 16 (72.7%) had a positive blood culture, whereas only 12 (42.9%) of the 28 patients with normal troponin I showed culture positivity. In contrast, a higher proportion of patients with normal troponin had negative cultures (57.1%) compared to those with elevated troponin (27.3%).

Table 8: Outcome Comparison Between Patients With and Without Myocardial Injury

Parameter	With Myocardial Injury (n=22)	Without Myocardial Injury (n=28)
Mean Age (years)	58.2±10.4	52.6±12.1
In-hospital Mortality (%)	45.5%	17.8%
ICU Stay (days, mean±SD)	7.8±2.4	4.6±1.8
LVEF <45% (%)	36%	7%
Positive Blood Culture (%)	64%	50%

The comparison of outcomes between sepsis patients with and without myocardial injury reveals significant differences in clinical severity and prognosis. Patients with myocardial injury were older on average (58.2±10.4 years vs. 52.6±12.1 years) and had markedly higher in-hospital mortality (45.5% vs. 17.8%). They also experienced longer ICU stays (7.8±2.4 days vs. 4.6±1.8 days), indicating more severe illness and prolonged recovery. Left ventricular dysfunction (LVEF <45%) was observed in 36% of patients with myocardial injury compared to only 7% without it. Additionally, a higher proportion of patients with myocardial injury had positive blood cultures (64% vs. 50%), suggesting that systemic infection may contribute to cardiac involvement and worsen outcomes. These findings emphasize the prognostic significance of myocardial injury in septic patients (Table 8).

In this observational study conducted among 50 sepsis patients at a tertiary care hospital, we observed a high prevalence of myocardial injury (44%) as defined by elevated serum troponin I levels. The study further demonstrated a significant association between myocardial injury and adverse clinical outcomes, including increased in-hospital mortality, longer ICU stays, and reduced left ventricular function. Our findings are consistent with several earlier studies that have highlighted the burden of cardiac dysfunction in sepsis. Altmann *et al.* (2010) reported that 36% of septic patients had elevated troponin levels, which were independently associated with poor outcomes, even in the absence of underlying coronary artery disease[7]. Similarly, Brown *et al.* (2012) observed that myocardial dysfunction, especially when accompanied by diastolic impairment, significantly increased the risk of death in septic shock patients[8]. In our study, 45.5% mortality was recorded in patients with elevated troponin, compared to 17.8% in those with normal levels. This mirrors the results of Wilson[9], who found troponin elevation to be a strong predictor of mortality in sepsis, suggesting ongoing myocardial stress or injury likely driven by systemic inflammation, oxidative stress and microvascular dysfunction. The mean ICU stay was also longer in the myocardial injury group (7.8±2.4 days vs. 4.6±1.8 days), indicating a more prolonged recovery and critical care requirement. These findings align with the study by Pulido *et al.*

(2012), where sepsis-related myocardial injury was linked to prolonged hospitalization and poor functional outcomes[10]. Echocardiography in our study revealed that 36% of patients with elevated troponin had EF <45%, compared to only 7% in those without myocardial injury. This is supported by Vieillard-Kim[11], who reported a high frequency of left ventricular systolic dysfunction among patients with septic shock. The presence of diastolic dysfunction and pericardial effusion in a subset of our patients further reflects the spectrum of cardiac involvement during sepsis. Our study also explored the relationship between blood culture positivity and myocardial injury. A higher proportion of culture-positive patients had elevated troponin (57.1%), suggesting that bacteremia and higher pathogen burden may contribute to myocardial stress. While few studies have directly compared blood culture status with cardiac biomarkers, this association warrants further investigation. Despite these valuable insights, our study has certain limitations. The sample size was relatively small and advanced cardiac imaging such as tissue Doppler or speckle-tracking echocardiography was not performed, which could provide more detailed assessment of subclinical dysfunction. In addition, the study was single-centre and observational in nature.

CONCLUSION

This study highlights that myocardial injury is a common and clinically significant complication of sepsis, affecting nearly half of the patients. Elevated troponin I levels were associated with increased mortality, longer ICU stay, and higher prevalence of cardiac dysfunction on echocardiography. These findings support the need for routine cardiac evaluation in septic patients, including early measurement of troponins and focused echocardiography, especially in critically ill individuals. Early identification of cardiac involvement could guide clinical decisions regarding monitoring, supportive care and prognostication. Larger multicentre studies are recommended to validate these findings and determine whether targeted cardioprotective therapies can improve outcomes in patients with sepsis-induced myocardial injury.

REFERENCES

1. Vieillard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F. Hemodynamic instability in sepsis: bedside assessment by Doppler echocardiography. Am J Respir Crit Care Med. 2003;168 (11):1270-1276. doi:10.1164/ rccm.200306-816CC
2. Landesberg G, Gilon D, Meroz Y, *et al.* Diastolic dysfunction and mortality in severe sepsis and septic shock. Eur Heart J. 2012;33(7):895-903. doi:10.1093/eurheartj/ehr351
3. Hollenberg SM, Singer M. Pathophysiology of sepsis-induced cardiomyopathy. Nat Rev Cardiol. 2021;18(6):424-434. doi:10.1038/ s41569-020-00492-2
4. Smith A, John M, Trout R, Davis E, Moningi S. Elevated cardiac troponins in sepsis: what do they signify?. W V Med J. 2009;105(4):29-32.
5. Zochios V, Valchanov K. Raised cardiac troponin in intensive care patients with sepsis, in the absence of angiographically documented coronary artery disease: A systematic review. J Intensive Care Soc. 2015 Feb;16(1):52-57. doi: 10.1177/17511 43714555303. Epub 2014 Dec 9. PMID: 28979375; PMCID: PMC5593290.
6. Kakihana Y, Ito T, Nakahara M, Yamaguchi K, Yasuda T. Sepsis-induced myocardial dysfunction: pathophysiology and management. J Intensive Care. 2016;4:22. Published 2016 Mar 23. doi:10.1186/s40560-016-0148-1
7. Altmann DR, Korte W, Maeder MT, Fehr T, Haager P, Rickli H, Kleger GR, Rodriguez R, Ammann P. Elevated cardiac troponin I in sepsis and septic shock: no evidence for thrombus associated myocardial necrosis. PLoS One. 2010 Feb 3;5(2):e9017. doi: 10.1371/journal.pone.0009017. PMID: 20140242; PMCID: PMC2815772.
8. Brown SM, Pittman JE, Hirshberg EL, Jones JP, Lanspa MJ, Kuttler KG, Litwin SE, Grissom CK. Diastolic dysfunction and mortality in early severe sepsis and septic shock: a prospective, observational echocardiography study. Critical ultrasound journal. 2012 Dec;4:1-9.
9. Wilson MF, Baig MK, Ashraf H. Quality of life in octagenarians after coronary artery bypass grafting. The American journal of cardiology. 2005 Mar 15;95(6):761-4.
10. Pulido JN, Afessa B, Masaki M, et al. Clinical spectrum, frequency, and significance of myocardial dysfunction in severe sepsis and septic shock. Mayo Clin Proc. 2012;87(7):620-628. doi:10.1016/j.mayocp.2012.01.018
11. Kim S, Lee JD, Kim BK, Kim YH, Kim JH. Association between Left Ventricular Systolic Dysfunction and Mortality in Patients with Septic Shock. Journal of Korean medical science. 2020 Feb 3;35(4):e24-.