Section: Medicine

Original Research Article

EVALUATION OF CARDIAC TROPONIN I LEVELS IN SEPSIS AND ITS CORRELATION WITH SOFA SCORE AND INFLAMMATORY MARKERS (HS-CRP AND TNF-A): A CROSS-SECTIONAL OBSERVATIONAL STUDY

Piyush Prabha Singh[1], M. Narang[2], Mukul Singh[2]

[1]*Senior Resident, Department of Medicine, Vardhman Mahavir Medical College and Safdarjung Hospital, Delhi, India*
[2]*Consultant and Professor, Department of Medicine, Vardhman Mahavir Medical College and Safdarjung Hospital, Delhi, India*

Received *: 07/05/2026*
Received in revised form *: 18/06/2026*
Accepted *: 02/07/2026*

Corresponding Author:
Dr. Piyush Prabha Singh,
Senior Resident, Department of Medicine, Vardhman Mahavir Medical College and Safdarjung Hospital, Delhi, India.
Email: piyushprabhasingh@yahoo.com

DOI: 10.70034/ijmedph.2026.3.140

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 863-868

ABSTRACT

Background: Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection. Myocardial injury is frequently observed in sepsis and may occur even without acute coronary syndrome. Cardiac Troponin-I is a sensitive marker of myocardial injury and may reflect disease severity. This study evaluated Troponin-I levels in sepsis and correlated them with SOFA score and inflammatory markers. The aim is to study the correlation between cardiac Troponin-I levels with SOFA score and inflammatory markers, hs-CRP and TNF-α, in patients with sepsis.

Materials and Methods: This observational cross-sectional study was conducted in the Department of General Medicine, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, over 18 months. A total of 38 adult patients diagnosed with sepsis according to Sepsis-3 guidelines were included. Patients with acute coronary syndrome, severe renal/hepatic dysfunction, pulmonary embolism, and malignancy were excluded. Troponin-I, hs-CRP, TNF-α, complete blood count, liver and renal function tests, arterial blood gas analysis, ECG, and SOFA score were assessed within 24 hours of admission. Statistical analysis was performed using SPSS version 17.0. Spearman correlation was used for non-normally distributed variables, and $p<0.05$ was considered statistically significant.

Results: The mean age was 45.08 ± 14.26 years, and 71.1% were males. The mean SOFA score was 7.76 ± 4.85, hs-CRP was 10.26 ± 3.78 mg/L, TNF-α was 343.58 ± 175.58 pg/mL, and Troponin-I was 0.56 ± 0.52 ng/mL. Troponin-I showed a significant positive correlation with SOFA score (rho=0.55, $p<0.001$) and hs-CRP (rho=0.51, $p<0.001$). Troponin-I was significantly higher among patients requiring inotropes compared with those not requiring inotropes (0.97 ± 0.54 vs 0.43 ± 0.45 ng/mL; p=0.006). Significant negative correlations were observed between Troponin-I and MAP (rho=-0.48, p=0.002), PaO_2/FiO_2 ratio (rho=-0.48, p=0.002), and GCS (rho=-0.38, p=0.017). TNF-α showed a weak positive but statistically non-significant correlation with Troponin-I (rho=0.19, p=0.257).

Conclusion: Cardiac Troponin-I levels were significantly correlated with SOFA score and hs-CRP levels in sepsis, suggesting that Troponin-I may serve as a useful biomarker of myocardial injury and disease severity. Its association with hypotension, respiratory dysfunction, altered sensorium, and inotropic requirement highlights its potential role in early risk stratification of septic patients.

Keywords: Sepsis, Troponin-I, SOFA score, hs-CRP, TNF-α, myocardial injury.

INTRODUCTION

Sepsis is a complex clinical syndrome characterized by life-threatening organ dysfunction resulting from a dysregulated host immune response to infection. Despite substantial advances in antimicrobial therapy, intensive care support, and organ support strategies, sepsis continues to be a major cause of morbidity and mortality worldwide. According to the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3), sepsis is defined as infection associated with an acute increase in Sequential Organ Failure Assessment (SOFA) score of two points or more, reflecting significant organ dysfunction. Early identification of disease severity remains essential for timely intervention and improved clinical outcomes.[1,2]

Cardiovascular dysfunction represents one of the earliest and most important manifestations of severe sepsis. Septic myocardial dysfunction is characterized by reversible impairment of ventricular contractility occurring in the absence of primary ischemic heart disease. The underlying mechanisms are multifactorial and include excessive inflammatory cytokine release, oxidative stress, mitochondrial dysfunction, endothelial injury, impaired calcium homeostasis, nitric oxide overproduction, and microvascular abnormalities. These mechanisms collectively contribute to myocardial cell injury, ventricular dilatation, and reduced ejection fraction, ultimately increasing mortality risk.[3,4]

Cardiac troponin I (cTnI) is a highly specific biomarker of myocardial injury and has become the gold standard for detecting myocardial cell damage. Although elevated cTnI levels are classically associated with acute coronary syndromes, numerous studies have demonstrated significant elevations among patients with sepsis even in the absence of obstructive coronary artery disease. Elevated troponin concentrations in sepsis have consistently been associated with increased disease severity, prolonged intensive care stay, higher incidence of multiorgan dysfunction, and increased mortality. Consequently, cTnI has emerged as a potentially valuable prognostic biomarker in septic patients.[5,6]

The Sequential Organ Failure Assessment (SOFA) score is one of the most widely accepted scoring systems for assessing the severity of organ dysfunction in critically ill patients. The score incorporates respiratory, cardiovascular, hepatic, coagulation, neurological, and renal parameters to objectively quantify organ failure. Increasing SOFA scores have demonstrated strong associations with adverse clinical outcomes, making it an essential component of modern sepsis assessment. Correlating cardiac biomarkers with SOFA score may provide valuable insights into the relationship between myocardial injury and systemic organ dysfunction.[2,7]

Inflammation plays a central role in the pathophysiology of sepsis. High-sensitivity C-reactive protein (hs-CRP), an acute-phase reactant synthesized by hepatocytes in response to interleukin-6 stimulation, reflects the magnitude of systemic inflammation and has been widely used as a marker of disease activity. Similarly, tumor necrosis factor-alpha (TNF-α) is a pivotal pro-inflammatory cytokine that initiates the inflammatory cascade responsible for endothelial dysfunction, myocardial depression, apoptosis, and multiorgan failure during sepsis. Persistent elevation of these inflammatory mediators has been associated with worse clinical outcomes and increased mortality.[4,8]

Although previous investigations have independently evaluated cardiac troponins, inflammatory biomarkers, and SOFA score in septic patients, relatively few studies have comprehensively examined their interrelationship. Establishing correlations among cTnI, hs-CRP, TNF-α, and SOFA score may improve understanding of septic myocardial injury and facilitate early prognostic assessment. Therefore, the present study was undertaken to evaluate cardiac troponin I levels in patients with sepsis and determine their correlation with organ dysfunction severity and inflammatory biomarkers.

MATERIALS AND METHODS

Study Design: The present study was a hospital-based observational cross-sectional study designed to evaluate cardiac troponin I levels in patients diagnosed with sepsis and determine their correlation with SOFA score and inflammatory biomarkers (hs-CRP and TNF-α).

Study Setting: The study was conducted in the Department of General Medicine, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, a tertiary care teaching hospital catering to a large number of critically ill medical patients.

Study Duration: The study was carried out over a period of 18 months following approval from the Institutional Ethics Committee.

Study Population: The study population comprised adult patients admitted with sepsis fulfilling Sepsis-3 diagnostic criteria.

Sample Size: Sample size calculation was performed according to the method described by Hulley et al. Considering an expected correlation coefficient (r = 0.48) reported by Zhang et al., a minimum sample size of 32 patients was obtained. Allowing for approximately 20% attrition or incomplete data, the final sample size was fixed at 38 patients.

Inclusion Criteria

Patients fulfilling all of the following criteria were included:

- Age ≥18 years.
- Both males and females.

- Diagnosed with sepsis according to Sepsis-3 guidelines.
- Provided written informed consent.

Exclusion Criteria

Patients were excluded if they had:

- Acute coronary syndrome based on clinical findings and ECG.
- Chronic kidney disease or severe renal dysfunction.
- Severe hepatic dysfunction.
- Acute pulmonary embolism.
- Known malignancy.
- Refusal to provide informed consent.

Sampling Technique: Consecutive eligible patients admitted during the study period were enrolled until the required sample size was achieved.

Study Procedure: Following enrollment, each participant underwent comprehensive clinical evaluation.

Detailed demographic characteristics including age, sex, presenting symptoms, duration of illness, medical history, comorbid conditions, medication history, and physical examination findings were recorded in a structured case record form.

Written informed consent was obtained before participation.

Laboratory Investigations: Within 24 hours of admission, venous blood samples were collected under strict aseptic precautions.

The following investigations were performed:

- Complete Blood Count (CBC
- Hemoglobin
- Total Leukocyte Count
- Differential Leukocyte Count
- Platelet Count
- Liver Function Tests
- Serum Bilirubin
- Kidney Function Tests
- Serum Creatinine
- Serum Cardiac Troponin I
- High-sensitivity C-Reactive Protein (hs-CRP
- Tumor Necrosis Factor-alpha (TNF-α)
- Arterial Blood Gas Analysis
- Electrocardiography

Estimation of Cardiac Troponin I

Serum cardiac troponin I was measured using the electrochemiluminescence immunoassay (ECLIA) method on the Cobas e411 analyzer.

Estimation of hs-CRP

High-sensitivity CRP levels were estimated using the Biocell Medicare BN II analyzer according to manufacturer recommendations.

Estimation of TNF-α

Serum TNF-α concentrations were measured using the ADVIA Centaur CP Immunoassay System employing chemiluminescence technology.

SOFA Score Assessment

SOFA score was calculated within 24 hours of admission using standard criteria based on six organ systems:

- Respiratory
- Cardiovascular
- Hepatic
- Coagulation
- Neurological
- Renal

FiO_2 was calculated using:

$FiO_2 = 20 + (4 \times \text{Oxygen Flow Rate in L/min})$

Outcome Measures

Primary Outcome

- Correlation between serum cardiac troponin I levels and SOFA score.

Secondary Outcomes

- Correlation between cTnI and hs-CRP.
- Correlation between cTnI and TNF-α.
- Association of cTnI with severity of sepsis.

Data Collection: Clinical findings and laboratory results were entered into a predesigned proforma and subsequently transferred to Microsoft Excel before statistical analysis.

Statistical Analysis: Data were analyzed using SPSS version 17.0.

Continuous variables were expressed as mean ± standard deviation or median (interquartile range) depending on distribution.

Categorical variables were summarized as frequencies and percentages.

Comparisons between groups were performed using:

- Student's independent t-test
- Mann–Whitney U test
- Chi-square test
- Fisher's exact test

Correlation between continuous variables was assessed using:

- Pearson correlation coefficient for normally distributed variables.
- Spearman rank correlation coefficient for non-parametric variables.

A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol received approval from the Institutional Ethics Committee of Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi before initiation.

Written informed consent was obtained from every participant. Confidentiality of patient information was maintained throughout the study. All procedures adhered to the ethical principles outlined in the Declaration of Helsinki.

RESULTS

A total of 38 adult patients with sepsis were included in the study. The mean age was 45.08 ± 14.26 years, with maximum participants in the 31–40-year age group (28.9%). Males constituted 71.1%, while females constituted 28.9%.

Table 1: Baseline demographic and laboratory profile of study participants

Parameter	Mean ± SD / n (%)	Median (IQR)	Range
Age (years)	45.08 ± 14.26	43.00 (35.25–57.00)	19–69
Male	27 (71.1%)	—	—
Female	11 (28.9%)	—	—
Hemoglobin (g/dL)	10.33 ± 2.77	9.50 (8.50–12.57)	4.80–16.30
TLC (/mm³)	16292.11 ± 6271.44	15800 (12725–19375)	3200–30000
Platelets (/mm³)	153871.05 ± 109775.67	125000 (81500–203000)	11000–450000
Total bilirubin (mg/dL)	2.60 ± 2.53	1.65 (0.83–2.60)	0.40–10.40
Serum creatinine (mg/dL)	4.72 ± 4.70	3.05 (0.95–6.58)	0.30–14.90

The study population was predominantly male and middle-aged. Laboratory findings showed leukocytosis, anemia, thrombocytopenia tendency, hyperbilirubinemia, and raised creatinine, indicating systemic inflammation and multiorgan involvement.

Table 2: Clinical severity, inflammatory markers and Troponin-I levels

Parameter	Mean ± SD / n (%)	Median (IQR)	Range
MAP (mmHg)	81.71 ± 17.95	82.50 (68.50–94.00)	50–115
Inotrope requirement	9 (23.7%)	—	—
No inotrope	29 (76.3%)	—	—
PaO_2/FiO_2	392.39 ± 162.98	350 (268.75–477.50)	150–780
GCS	11.66 ± 3.94	13 (9.25–15.00)	3–15
SOFA score	7.76 ± 4.85	6 (4–10.75)	2–22
hs-CRP (mg/L)	10.26 ± 3.78	10 (7.25–14.00)	2–15
TNF-α (pg/mL)	343.58 ± 175.58	351 (205.25–437.25)	90–728
Troponin-I (ng/mL)	0.56 ± 0.52	0.46 (0.05–0.90)	0.01–2.10

The mean SOFA score was 7.76 ± 4.85, suggesting moderate organ dysfunction. Troponin-I levels were elevated in many patients, with a mean value of 0.56 ± 0.52 ng/mL, supporting the presence of myocardial injury in sepsis.

Table 3: Association and correlation of Troponin-I with clinical and inflammatory parameters

Parameter	Correlation / Mean Troponin-I	p value	Interpretation
Age	rho = -0.11	0.529	Not significant
Gender	Male: 0.61 ± 0.54; Female: 0.43 ± 0.47	0.333	Not significant
TLC	rho = -0.28	0.094	Not significant
Platelet count	rho = -0.29	0.074	Not significant
Serum creatinine	rho = 0.16	0.326	Not significant
MAP	rho = -0.48	0.002	Significant negative correlation
Inotrope use	Yes: 0.97 ± 0.54; No: 0.43 ± 0.45	0.006	Significant
PaO_2/FiO_2	rho = -0.48	0.002	Significant negative correlation
GCS	rho = -0.38	0.017	Significant negative correlation
SOFA score	rho = 0.55	<0.001	Significant positive correlation
hs-CRP	rho = 0.51	<0.001	Significant positive correlation
TNF-α	rho = 0.19	0.257	Not significant

Troponin-I showed a statistically significant positive correlation with SOFA score and hs-CRP, indicating that myocardial injury increased with worsening organ dysfunction and systemic inflammation. Troponin-I showed significant negative correlation with MAP, PaO_2/FiO_2, and GCS, suggesting higher myocardial injury among patients with hypotension, respiratory compromise, and impaired neurological status. TNF-α showed a weak positive but statistically non-significant correlation with Troponin-I.

Overall, the study demonstrated that cardiac Troponin-I levels were significantly associated with important markers of sepsis severity. Patients requiring inotropic support had higher Troponin-I levels compared with those not requiring inotropes, indicating greater myocardial stress in hemodynamically unstable patients. The moderate positive correlation between Troponin-I and SOFA score suggests that Troponin-I may reflect global organ dysfunction rather than isolated cardiac involvement. Similarly, the significant correlation with hs-CRP supports the role of systemic inflammation in sepsis-associated myocardial injury.

DISCUSSION

Sepsis is a life-threatening clinical syndrome characterized by infection-associated organ dysfunction caused by a dysregulated host response. The Sepsis-3 definition emphasizes organ dysfunction assessed by the SOFA score, and this approach has improved the clinical recognition of patients at higher risk of adverse outcomes. (2) In the present study, the mean SOFA score was 7.76 ± 4.85, reflecting moderate severity of illness among enrolled patients.

The mean age of patients was 45.08 ± 14.26 years, with a male predominance of 71.1%. Similar demographic trends have been observed in several

sepsis studies where adult males constitute a major proportion of hospitalized septic patients, possibly due to differences in exposure risk, comorbidity burden, and healthcare-seeking behavior.[4,5]

The present study demonstrated a mean Troponin-I level of 0.56 ± 0.52 ng/mL. Troponin elevation in sepsis has been widely reported even in the absence of acute coronary syndrome. The mechanisms include inflammatory cytokine-mediated myocardial depression, microvascular dysfunction, increased membrane permeability, mitochondrial injury, oxygen supply–demand mismatch, and catecholamine-related myocardial stress.[6] Therefore, elevated Troponin-I in sepsis should not always be interpreted as primary coronary thrombosis; rather, it may indicate sepsis-induced myocardial injury.

A major finding of the present study was the significant positive correlation between Troponin-I and SOFA score (rho = 0.55, p <0.001). This indicates that myocardial injury increased with worsening organ dysfunction. Similar observations have been reported by Røsjø et al., who found that circulating cardiac troponin levels were associated with disease severity and survival in severe sepsis and septic shock.[7] Kim et al. also reported that troponin testing may help identify sepsis-induced myocardial dysfunction and adverse outcomes.[8]

Troponin-I was significantly higher among patients requiring inotropes compared with those not requiring inotropes (0.97 ± 0.54 vs 0.43 ± 0.45 ng/mL, p = 0.006). This finding supports the relationship between circulatory failure and myocardial injury. Patients receiving noradrenaline or other vasoactive drugs generally represent a more severe hemodynamic phenotype of sepsis. Septic shock is associated with myocardial depression, vasoplegia, endothelial dysfunction, and impaired tissue perfusion, all of which may contribute to troponin release.[9,10]

Troponin-I showed a significant negative correlation with MAP (rho = -0.48, p = 0.002), indicating that lower blood pressure was associated with higher myocardial injury. This can be explained by reduced coronary perfusion pressure, microcirculatory impairment, and increased myocardial oxygen demand during septic shock. Similarly, Troponin-I had a significant negative correlation with PaO_2/FiO_2 ratio (rho = -0.48, p = 0.002), suggesting that respiratory dysfunction and hypoxemia may contribute to myocardial stress in sepsis.

Another important observation was the significant positive correlation between Troponin-I and hs-CRP (rho = 0.51, p <0.001). CRP is an acute-phase reactant that reflects systemic inflammatory activity. In sepsis, inflammatory activation contributes to endothelial damage, capillary leak, oxidative stress, and myocardial cell injury. The observed correlation supports the hypothesis that septic myocardial injury is closely linked with inflammatory burden.[11,12]

In contrast, TNF-α showed only a weak and statistically non-significant correlation with Troponin-I (rho = 0.19, p = 0.257). Although TNF-α is a key cytokine in septic inflammation and myocardial depression, its circulating level may fluctuate rapidly depending on timing of sampling, infection source, immune response, and prior treatment. This may explain the absence of significant correlation in the present study. Previous experimental and clinical studies have shown that cytokine-mediated myocardial depression is complex and cannot be explained by a single mediator alone.[13,14]

The present study also found that Troponin-I was significantly associated with lower GCS (rho = -0.38, p = 0.017). This may reflect the relationship between myocardial injury and overall severity of illness, including sepsis-associated encephalopathy, tissue hypoperfusion, metabolic derangement, and multiorgan dysfunction.

Overall, the findings suggest that Troponin-I is not merely a cardiac marker in sepsis but may function as an integrated marker of disease severity, inflammatory burden, hemodynamic instability, respiratory dysfunction, and organ failure. Its correlation with SOFA score and hs-CRP supports its potential role in early risk stratification of septic patients. However, the cross-sectional design and small sample size limit causal inference. Serial Troponin-I measurements, echocardiographic correlation, and mortality follow-up would further strengthen future research.

CONCLUSION

The present study concluded that cardiac Troponin-I levels were significantly correlated with SOFA score and hs-CRP levels in patients with sepsis. Higher Troponin-I levels were associated with greater organ dysfunction, lower MAP, reduced PaO_2/FiO_2 ratio, lower GCS, and need for inotropic support. TNF-α showed a weak positive but statistically non-significant correlation with Troponin-I. These findings suggest that Troponin-I may serve as a useful biomarker of sepsis severity and sepsis-associated myocardial injury when interpreted along with SOFA score and inflammatory markers.

REFERENCES

1. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M. The Third International Consensus Definitions for Sepsis and Septic Shock. JAMA. 2016;315(8):801-810. doi:10.1001/jama.2016.0287.
2. Vincent JL, Moreno R, Takala J, Willatts S, De Mendonça A, Bruining H. The SOFA score to describe organ dysfunction/failure. Intensive Care Med. 1996;22(7):707-710. doi:10.1007/BF01709751.
3. Fleischmann C, Scherag A, Adhikari NKJ, Hartog CS, Tsaganos T, Schlattmann P. Assessment of global incidence and mortality of hospital-treated sepsis. Am J Respir Crit Care Med. 2016;193(3):259-272. doi:10.1164/rccm.201504-0781OC.
4. Rudd KE, Johnson SC, Agesa KM, Shackelford KA, Tsoi D, Kievlan DR. Global, regional, and national sepsis incidence

and mortality. Lancet. 2020;395(10219):200-211. doi:10.1016/S0140-6736(19)32989-7.
5. Rudiger A, Singer M. Mechanisms of sepsis-induced cardiac dysfunction. Crit Care Med. 2007;35(6):1599-1608. doi:10.1097/01.CCM.0000266683.64081.02.
6. Landesberg G, Gilon D, Meroz Y, Georgieva M, Levin PD, Goodman S. Diastolic dysfunction and mortality in severe sepsis and septic shock. Eur Heart J. 2012;33(7):895-903. doi:10.1093/eurheartj/ehr351.
7. Røsjø H, Varpula M, Hagve TA, Karlsson S, Ruokonen E, Pettilä V. Circulating high sensitivity troponin T in severe sepsis and septic shock. Intensive Care Med. 2011;37(1):77-85. doi:10.1007/s00134-010-2051-x.
8. Kim JS, Kim M, Kim YJ, Ryoo SM, Sohn CH, Ahn S. Troponin testing for assessing sepsis-induced myocardial dysfunction. J Clin Med. 2019;8(2):239. doi:10.3390/jcm8020239.
9. ver Elst KM, Spapen HD, Nguyen DN, Garbar C, Huyghens LP, Gorus FK. Cardiac troponins I and T are biological markers of left ventricular dysfunction in septic shock. Clin Chem. 2000;46(5):650-657.
10. Ammann P, Fehr T, Minder EI, Günter C, Bertel O. Elevation of troponin I in sepsis and septic shock. Intensive Care Med. 2001;27(6):965-969. doi:10.1007/s001340100920.
11. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. N Engl J Med. 1999;340(6):448-454. doi:10.1056/NEJM199902113400607.
12. Pierrakos C, Vincent JL. Sepsis biomarkers: a review. Crit Care. 2010;14(1):R15. doi:10.1186/cc8872.
13. Kumar A, Thota V, Dee L, Olson J, Uretz E, Parrillo JE. Tumor necrosis factor alpha and interleukin 1 beta are responsible for in vitro myocardial cell depression. J Exp Med. 1996;183(3):949-958. doi:10.1084/jem.183.3.949.
14. Pathan N, Hemingway CA, Alizadeh AA, Stephens AC, Boldrick JC, Oragui EE. Role of interleukin 6 in myocardial dysfunction of meningococcal septic shock. Lancet. 2004;363(9404):203-209. doi:10.1016/S0140-6736(03)15326-3.