Vol. 5 No. 1 (2026)

Pakistan Journal of Medical & Cardiological Review

https://pakjmcr.com/index.php/1/about

Online ISSN 3007-2387

Print ISSN 3007-2379

Ullah et al., 2026

RELATIONSHIP BETWEEN TROPONIN LEVELS AND CLINICAL OUTCOMES AMONG CRITICALLY ILL PATIENTS WITH SUSPECTED CARDIOVASCULAR INJURY

Inam Ullah
Lecturer AHS Surgical Technology Iqra National University Swat Campus. Email: inamullahkhan@inu.edu.pk
Dr. Masooma Talib
Professor (Biochemistry) at Bahria University College of Medicine, Islamabad. Email: masooma.bilal@gmail.com
Paghunda Nobat*
MS AHS Cardiology Technology Superior University Lahore. Cath Lab Technologist at Akbar Medical Complex Mardan. Echo Technologist at Aziz Biotech Medical Lab & Research Center Islamabad. Corresponding Author Email: paghundadurrani@gmail.com
Ali Ghulam
Lecturer Institute of Health & Development Studies. Email: alighulamjani512@gmail.com
Asghar Hussain
Lecturer Surgical Technology at Fatma College of Professional Sciences Kabal Swat. Email: hussainasghar698@gmail.com
Atta Ur Rahman
Demonstrator AHS Surgical Technology Iqra National University Swat Campus. Email: attaurrahman@inu.edu.pk

Author Details

Received on 08 Feb, 2026
Accepted on 01 March, 2026
Published on 04 March, 2026

Corresponding E-mails & Authors*:
Paghunda Nobat*
paghundadurrani@gmail.com

Abstract

Background: Cardiac troponin is a sensitive biomarker of myocardial injury and is frequently elevated in critically ill patients, even without acute coronary syndrome. Elevated concentrations have been linked to greater disease severity, prolonged intensive care unit (ICU) stay, organ dysfunction, and higher mortality. To assess the relationship between serum troponin levels and clinical outcomes among critically ill patients with suspected cardiovascular injury.

Methods: Previously published observational studies of critically ill adults admitted to ICUs were reviewed. Patients were grouped by normal or elevated troponin, and

reported outcomes (ICU mortality, hospital mortality, ICU stay, mechanical ventilation, organ dysfunction) were examined. **Results:** Elevated troponin-I was found in 81% of patients in one large medical-surgical ICU study, and hospital mortality rose from 23.4% in the lowest troponin category to 57.4% in the highest. Another study found myocardial injury in 32% of patients, with mortality of 51% versus 16% in those without injury. Elevated troponin was also associated with longer ICU stay, mechanical ventilation, vasopressor requirement, and multiple-organ dysfunction. In adjusted analyses, elevated troponin was independently associated with hospital mortality (pooled adjusted OR 2.5, 95% CI 1.9–3.4, in one meta-analysis). **Conclusion:** Elevated troponin is associated with poorer outcomes in critically ill patients and serves as a marker of myocardial injury and overall illness severity. Because elevation also occurs in sepsis, renal failure, and other critical illness without myocardial infarction, interpretation must take the full clinical picture into account.

Keywords: Cardiac troponin; myocardial injury; intensive care units; critical illness; mortality; prognosis

1. Introduction

Cardiac troponin is the preferred biomarker for detecting myocardial injury because of its high sensitivity and cardiac specificity. In the ICU, however, troponin is often raised in patients with no acute coronary syndrome. Sepsis, hypoxaemia, hypotension, catecholamine exposure, renal dysfunction, and critical illness itself can all injure myocardium or reduce troponin clearance.

Previous studies have reported that elevated troponin in critically ill patients is associated with greater illness severity, longer ICU stay, organ dysfunction, and higher mortality (1–4). Whether troponin should be treated as a prognostic marker or as a signal of treatable cardiac pathology remains debated. This article therefore examines how troponin levels relate to clinical outcomes in critically ill patients with suspected cardiovascular injury.

2. Methods

2.1 Design

Narrative review of published observational studies, organised according to the IMRaD structure. No new patient data were collected, and no patient-level data were pooled.

2.2 Eligibility

Studies were considered if they enrolled critically ill adults (18 years or older) admitted to an ICU, measured cardiac troponin, and reported at least one clinical outcome.

2.3 Exposure and outcomes

Patients were categorised as having normal or elevated troponin, or by ascending troponin category where studies reported this. Outcomes of interest were ICU mortality, hospital mortality, ICU length of stay, mechanical ventilation, vasopressor use, and organ dysfunction.

2.4 Data handling

Percentages were extracted as reported by the source studies. No statistical pooling or meta-analysis was performed, and the figures below are descriptive displays of the reported values only. Measures of association (odds ratios with 95% confidence intervals and P values) are reported exactly as published by the source studies and were not recalculated.

3. Results

3.1 Prevalence of troponin elevation

In a large medical-surgical ICU cohort, elevated troponin-I was present in 81% of patients (Figure 1) (3). In a separate cohort, myocardial injury was present in 32% of critically ill patients (Figure 2) (2).

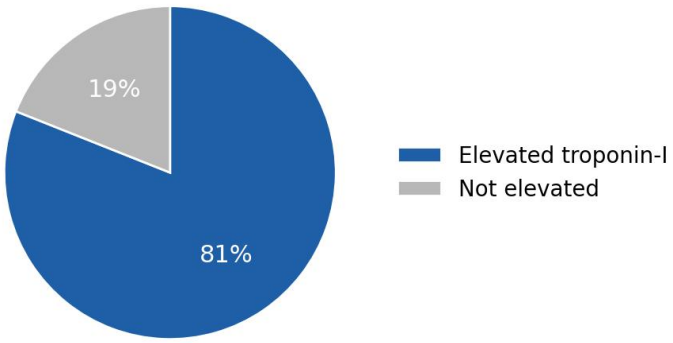


Figure 1. Prevalence of elevated troponin-I in a medical-surgical ICU cohort.

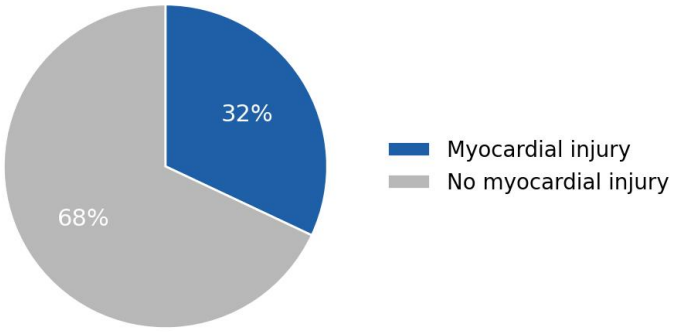


Figure 2. Prevalence of myocardial injury in a second ICU cohort.

3.2 Mortality by troponin level

Hospital mortality increased progressively with troponin concentration, from 23.4% in the lowest category to 57.4% in the highest (Figure 3) (3). In the second cohort, mortality was 51% in patients with myocardial injury and 16% in those without (Figure 4) (2).

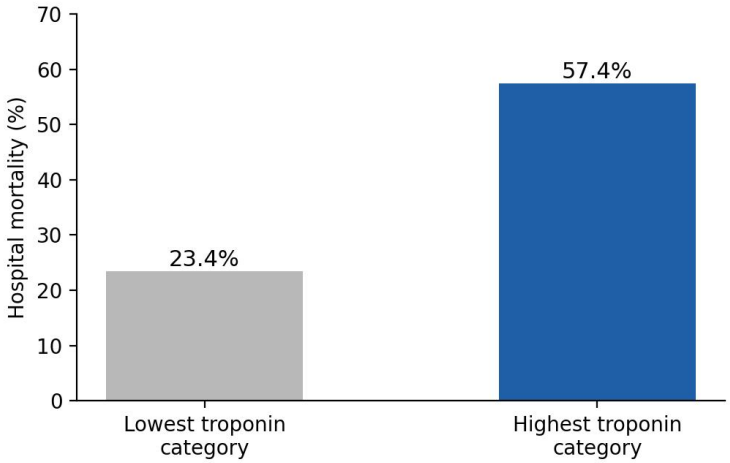


Figure 3. Hospital mortality in the lowest and highest troponin categories.

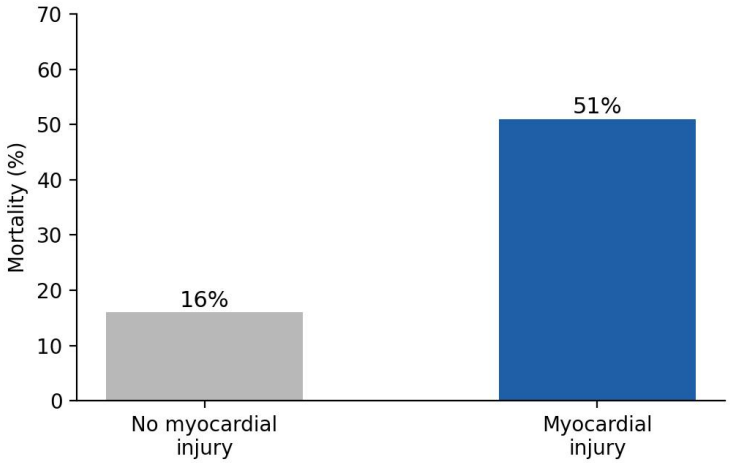


Figure 4. Mortality with and without myocardial injury.

3.3 Association between troponin and clinical outcomes

Association between troponin and outcome was examined in the source studies using adjusted analyses (Table 1, Figure 6). In a meta-analysis of 23 studies, elevated troponin was independently associated with death (pooled adjusted OR 2.5, 95% CI 1.9–3.4, P < 0.001) (1). In a prospective ICU cohort, myocardial injury remained independently associated with hospital mortality after adjustment for SAPS II score and mechanical ventilation (adjusted OR 2.09, 95% CI 1.06–4.11, P = 0.01) (2).

The association showed a dose-response pattern. In the medical-surgical ICU cohort, hospital mortality rose steadily across four troponin-I categories: 23.4% (Group I), 33.2% (Group II), 49.6% (Group III) and 57.4% (Group IV) (Figure 5). Relative to Group I, adjusted odds of hospital death were 1.08 (95% CI 0.84–1.38) in Group II, 1.64 (1.24–2.17) in Group III and 1.80 (1.30–2.49) in Group IV, so the association was not significant in the lowest elevated category and became significant at higher levels (3). Higher high-sensitivity troponin T groups were likewise associated with higher in-hospital mortality (P < 0.001) and longer ICU and hospital stay (P < 0.01) (7).

Findings were not uniform. In one small prospective cohort, an elevated troponin alone was not independently predictive of ICU or hospital mortality, whereas myocardial infarction was (hospital mortality 50.0% vs 22.0%, P = 0.010) (6).

Table 1. *Reported measures of association between troponin and mortality in the source studies.*

Study	Comparison	Outcome	Association (as published)
Meta-analysis, 23 studies (1)	Elevated vs normal troponin	Mortality	Pooled adjusted OR 2.5 (95% CI 1.9–3.4), P < 0.001
ICU cohort, n = 217 (2)	cTnI > 0.1 ng/mL vs lower	Hospital mortality	51% vs 16%, P < 0.001; adjusted OR 2.09 (1.06–4.11), P = 0.01
Medical-surgical ICU (3)	Group II vs Group I	Hospital mortality	Adjusted OR 1.08 (0.84–1.38)
Medical-surgical ICU (3)	Group III vs Group I	Hospital mortality	Adjusted OR 1.64 (1.24–2.17)
Medical-surgical ICU (3)	Group IV vs Group I	Hospital mortality	Adjusted OR 1.80 (1.30–2.49)
Medical ICU, hs-troponin T (7)	Higher vs lower hsTnT groups	Mortality; length of stay	Higher mortality (P < 0.001); longer stay (P < 0.01)
Prospective ICU cohort (6)	Elevated troponin	Hospital mortality	Not independently predictive; myocardial infarction 50.0% vs 22.0%, P = 0.010

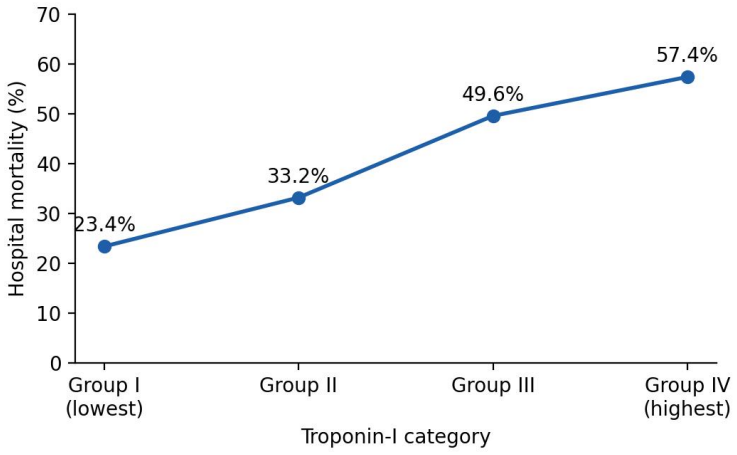


Figure 5. Hospital mortality across four troponin-I categories (Group I lowest, Group IV highest).

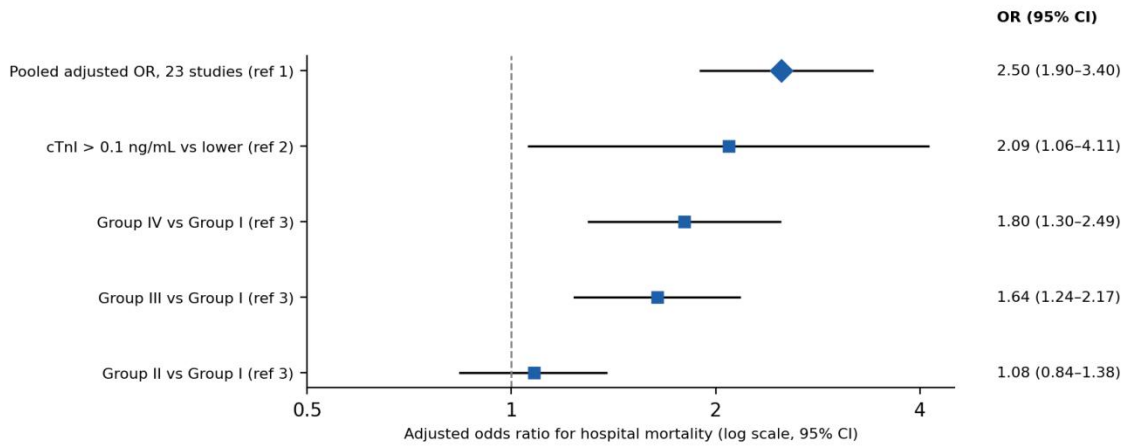


Figure 6. Adjusted odds ratios (95% CI) for mortality in the source studies. Each estimate is from a different study or comparison and is not pooled here.

3.4 Other outcomes

Elevated troponin was also associated with longer ICU stay, mechanical ventilation, vasopressor requirement, and multiple-organ dysfunction (1,4,7). These associations are summarised in Table 2, because the available reported values do not allow a numerical graph.

Table 2. *Direction of association between elevated troponin and outcomes in critically ill patients.*

Outcome	Association with elevated troponin
Hospital / ICU mortality	Higher, with a graded relationship
ICU length of stay	Longer
Mechanical ventilation	More frequent
Vasopressor requirement	More frequent
Multiple-organ dysfunction	More frequent

4. Discussion

Across the cited studies, higher troponin was consistently associated with worse outcomes. The graded rise in hospital mortality, more than doubling from the lowest to the highest category, suggests a dose-response pattern, and the large gap in mortality between patients with and without myocardial injury points the same way.

A meta-analysis of 23 observational studies (4492 critically ill patients) found that elevated troponin was associated with increased risk of death (adjusted OR 2.5, 95% CI 1.9–3.4) and longer ICU stay (1). Not all studies agree, however: in one prospective ICU cohort an elevated troponin alone was not independently predictive of mortality, whereas myocardial infarction was (6).

Troponin elevation in the ICU is probably not only a sign of coronary occlusion. It more likely reflects the combined burden of myocardial injury, systemic inflammation, haemodynamic stress, and reduced renal clearance, which is why it tracks overall illness severity (5,8). Troponin should therefore be interpreted with clinical findings, ECG, and echocardiography rather than in isolation.

Limitations

This is a narrative review of observational studies, so associations are not causal. Study populations, assay generations, and cut-offs differ, and most reported mortality figures are unadjusted for illness severity or confounders such as sepsis and renal failure. Only values stated in the source abstracts are displayed, and no pooled estimates are given.

5. Conclusion

Elevated troponin in critically ill patients with suspected cardiovascular injury is associated with higher mortality, longer ICU stay, and greater organ dysfunction. It is a useful marker of myocardial injury and illness severity, but because it is raised in many non-coronary conditions, it must be interpreted within the whole clinical context.

Declarations

Ethical approval: Not applicable. This review used only published literature and involved no human participants or patient data.

Informed consent: Not applicable.

Conflict of interest: The authors declare no conflict of interest.

Funding: None

Data availability: All data are from the cited published studies.

References

1. Lim W, et al. Elevated cardiac troponin measurements in critically ill patients. Arch Intern Med. 2006;166(22):2446-54. PMID: 17159009.

2. Quenot JP, Le Teuff G, Quantin C, Doise JM, Abrahamowicz M, Masson D, et al. Myocardial injury in critically ill patients: relation to increased cardiac troponin I and hospital mortality. Chest. 2005;128(4):2758-64. doi:10.1378/chest.128.4.2758. PMID: 16236952.

3.Association between troponin-I levels and outcome in critically ill patients admitted to non-cardiac intensive care unit with high prevalence of cardiovascular risk factors. BMC Anesthesiol. 2018. PMID: 29788912.

4. Moderate elevations of serum troponin I in a surgical intensive care unit population (retrospective review of 869 patients). Crit Care Med. 2003. PMID: 14605530.

5. Ammann P, Maggiorini M, Bertel O, Haenseler E, Joller-Jemelka HI, Oechslin E, et al. Troponin as a risk factor for mortality in critically ill patients without acute coronary syndromes. J Am Coll Cardiol. 2003;41(11):2004-9.

6. Lim W, Qushmaq I, Cook DJ, Crowther MA, Heels-Ansdell D, Devereaux PJ. Elevated troponin and myocardial infarction in the intensive care unit: a prospective study. Crit Care. 2005;9(6):R636-44.

7. Baron JM, Lewandrowski EL, Januzzi JL, Bajwa EK, Thompson BT, Lewandrowski KB. Measurement of high-sensitivity troponin T in noncardiac medical intensive care unit patients: correlation to mortality and length of stay. Am J Clin Pathol. 2014;141(4):488-93. PMID: 24619748.

8. Markou N, Gregorakos L, Myrianthefs P. Increased blood troponin levels in ICU patients. Curr Opin Crit Care. 2011;17(5):454-63. PMID: 21716108.