



Original Article

Correlation of High-Sensitivity Cardiac Troponin I Levels with Histopathological Features of Myocardial Injury in Patients with Acute Myocardial Infarction

Corresponding Author:

Dr. Deepika

Darji, Department of Pathology,
American Institute of Medical
Sciences, Bedwas, Udaipur,
Rajasthan, India

Article History:

Received on: Aug 16, 2026

Revised on: Sep 08, 2026

Accepted on: Sep 25, 2026

Published on: Oct 10, 2026

Citation: Ankita Garg *et al.*
Correlation of High-Sensitivity
Cardiac Troponin I Levels with
Histopathological Features of
Myocardial Injury in Patients with
Acute Myocardial Infarction.
*Biomedicine and Chemical
Sciences*, 5 (4): 39-45, 2026

Copyright © The Author(s).

Published in *Biomedicine and
Chemical Sciences*



This is an Open Access article
published under the Creative
Commons Attribution 4.0
International (CC BY 4.0)

(<https://creativecommons.org/licenses/by/4.0/>) Creative
Commons Attribution (CC BY): lets others distribute and copy
the article, to create extracts, abstracts, and other revised
versions, adaptations or derivative works of or from an article
(such as a translation), to include in a collective work (such as
an anthology), to text or data mine the article, even for
commercial purposes, as long as they credit the author(s), do
not represent the author as endorsing their adaptation of the
article, and do not modify the article in such a way as to
damage the author's honour or reputation

**Ankita Garg¹, Rahul Chaturvedi², Kailash Choudhary², Deepika Darji³,
Prashant Bhardwaj⁴**

¹Department of Pathology, Pt. B. D. Sharma Post Graduate Institute of Medical Sciences
(PGIMS), Rohtak, Haryana, India.

²Department of Cardiology, Rama Medical College and Hospital, Hapur, Uttar Pradesh, India

²Department of Cardiology, Rama Medical College and Hospital, Hapur, Uttar Pradesh, India.

³Department of Pathology, American Institute of Medical Sciences, Bedwas, Udaipur,
Rajasthan, India

⁴Department of Pathology, VALASMC, Etah, Uttar Pradesh, India.

ABSTRACT

Background: High-sensitivity cardiac troponin I (hs-TnI) is an established biomarker of myocardial injury. This study aimed to evaluate the relationship between serum hs-TnI concentrations and histopathological features of myocardial injury in patients with acute myocardial infarction (AMI).

Materials and Methods: This observational analytical study included 45 patients with AMI at a tertiary care hospital in India between January and December 2023. Demographic characteristics, cardiovascular risk factors, peak serum hs-TnI concentrations and histopathological findings were assessed. Myocardial necrosis, inflammatory infiltration and fibrosis were graded according to severity. Spearman's rank correlation coefficient was used to assess associations between peak serum hs-TnI concentrations and ordinal histopathological grades. The Mann-Whitney U test was used to compare hs-TnI concentrations according to the presence or absence of binary histopathological features. A two-sided p-value of less than 0.05 was considered statistically significant.

Results: The mean age was 57.8 ± 11.6 years, and 71.1% were male. STEMI accounted for 68.9% of cases. The median peak hs-TnI concentration was 7,520 ng/L (IQR: 3,800–15,400). Myocardial necrosis was identified in 84.4% of specimens, followed by cardiomyocyte degeneration (80.0%) and inflammatory infiltration (66.7%). In the analysis, hs-TnI showed a strong positive correlation with necrosis severity ($p = 0.814$, $p < 0.001$) and a weaker positive correlation with inflammatory infiltration ($p = 0.372$, $p = 0.012$). No statistically significant association was observed with fibrosis ($p = 0.571$), cardiomyocyte degeneration ($p = 0.109$), interstitial oedema ($p = 0.107$) or contraction-band changes ($p = 0.440$).

Conclusion: Median hs-TnI concentrations increased progressively with histopathological severity of myocardial necrosis. The analysis demonstrated a strong positive correlation with necrosis severity and a weaker association with inflammatory infiltration.

Keywords: Acute myocardial infarction; high-sensitivity cardiac troponin I; myocardial necrosis; histopathology; endomyocardial biopsy.

INTRODUCTION

Acute myocardial infarction (AMI) remains a major cause of cardiovascular morbidity and mortality worldwide. It results from prolonged myocardial ischaemia sufficient to cause irreversible cardiomyocyte injury and necrosis. Early diagnosis and assessment of myocardial damage are essential for timely treatment, risk stratification and prevention of adverse cardiovascular outcomes. According to the Fourth Universal Definition of Myocardial Infarction, AMI is diagnosed by a rise and/or fall in cardiac troponin concentrations, with at least one value exceeding the 99th percentile upper reference limit, accompanied by clinical evidence of acute myocardial ischaemia.[1]

Cardiac troponins are highly sensitive and specific biomarkers of myocardial injury. Among them, cardiac troponin I (cTnI) is an integral component of the cardiomyocyte contractile apparatus and is released into the circulation following myocardial damage. The introduction of high-sensitivity cardiac troponin assays has substantially improved the early detection of myocardial injury. Reichlin et al. demonstrated the diagnostic value of sensitive cardiac troponin assays in patients presenting with suspected AMI, while Keller et al. established the usefulness of sensitive troponin I measurements for early diagnosis and risk stratification.[2,3]

High-sensitivity troponin I (hs-TnI) assays can detect very low circulating concentrations, enabling earlier identification of myocardial injury and facilitating serial assessment of biomarker changes. However, elevated troponin concentrations are not specific to myocardial infarction and may occur in myocarditis, heart failure, pulmonary embolism and other cardiac or systemic conditions. Interpretation therefore requires integration with clinical findings, electrocardiographic changes and cardiac imaging. Furthermore, analytical differences between assays and variations in the timing of blood sampling may influence measured concentrations.[4,5]

Histopathological examination provides direct morphological evidence of myocardial injury. Following ischaemic damage, the myocardium undergoes characteristic structural alterations, including cardiomyocyte degeneration, coagulative necrosis, interstitial oedema and inflammatory cell infiltration. Subsequently, healing and remodelling may result in myocardial fibrosis. The extent and distribution of these changes depend on the severity and duration of ischaemia, reperfusion status and the interval between injury and tissue sampling. Although histopathology can characterize myocardial damage, endomyocardial biopsy is not routinely indicated in uncomplicated AMI and is generally reserved for selected clinical circumstances.[6]

The relationship between circulating troponin concentrations and the extent of myocardial injury has attracted considerable research interest. Younger et al. demonstrated that troponin I concentrations measured after myocardial infarction correlated with infarct size assessed using cardiovascular magnetic resonance imaging.[7] Nevertheless, biomarker concentrations may be affected by reperfusion, infarct location, renal function and the timing of measurement. Consequently, circulating troponin concentrations may not correspond precisely to the degree of structural damage in an individual patient.[8]

Current clinical guidelines emphasize the importance of hs-TnI testing in the diagnostic evaluation of acute coronary syndromes.[9] However, direct comparison of biochemical measurements with histopathological findings may provide additional insight into the relationship between circulating biomarkers and myocardial tissue injury. Such comparisons must account for biopsy timing, sampling limitations and the clinical indications for obtaining myocardial tissue.

Therefore, the present study was designed to evaluate the relationship between serum hs-TnI concentrations and histopathological features of myocardial injury in patients with AMI, with particular emphasis on myocardial necrosis, cardiomyocyte degeneration, inflammatory infiltration and myocardial fibrosis.

MATERIALS AND METHODS

Study design and setting: This observational analytical study was designed to evaluate the relationship between serum high-sensitivity cardiac troponin I (hs-TnI) concentrations and histopathological features of myocardial injury in patients with acute myocardial infarction (AMI). The study was conducted at a tertiary care hospital in India over 12 months, from January to December 2023. The study population comprised 45 patients.

Study population and eligibility: Patients aged 18 years or older with a diagnosis of AMI, established according to the Fourth Universal Definition of Myocardial Infarction, and with both hs-TnI measurements and myocardial biopsy findings available were considered for analysis. AMI was defined by a rise and/or fall in cardiac troponin, with at least one value above the 99th percentile upper reference limit, together with clinical evidence of acute myocardial ischaemia.[1] Patients

with incomplete biomarker or histopathological records, inadequate biopsy specimens or an alternative established cause of acute myocardial injury were excluded from the analytical dataset.

Clinical and biochemical assessment: Demographic characteristics, cardiovascular risk factors, type of myocardial infarction and left ventricular ejection fraction were recorded. Serum hs-TnI concentrations were assessed using a high-sensitivity assay, and the peak recorded concentration during the index admission was used for analysis. Results were expressed in ng/L.

Histopathological assessment: Available endomyocardial biopsy specimens were evaluated for myocardial necrosis, cardiomyocyte degeneration, inflammatory cell infiltration, interstitial oedema, myocardial fibrosis and contraction-band changes. Myocardial necrosis, inflammatory infiltration and fibrosis were categorized as absent, mild, moderate or severe. Other histopathological features were recorded according to their presence or absence.

Statistical analysis: Data were summarized using frequencies and percentages for categorical variables and mean $\pm$ standard deviation or median with interquartile range for continuous variables, as appropriate. Peak hs-TnI concentrations were compared across histopathological grades. Spearman's rank correlation was used for assessing relationships between hs-TnI and ordinal histopathological grades, while the Mann–Whitney U test was used for comparisons involving binary histopathological features. A two-sided p-value below 0.05 was the proposed threshold for statistical significance.

Ethical considerations: Institutional Ethics Committee approval was taken before commencing the study.

RESULTS

The study population comprised 45 patients with acute myocardial infarction (AMI). The mean age was 57.8 ± 11.6 years, with a predominance of males. Most patients belonged to the 41–60-year age group. Hypertension was the most frequently reported cardiovascular risk factor, followed by dyslipidaemia, diabetes mellitus and smoking. ST-elevation myocardial infarction (STEMI) accounted for approximately two-thirds of cases. The demographic and clinical characteristics are presented in Table 1.

Table 1. Demographic and clinical characteristics of the study population (N = 45)

Characteristic	n (%)
Age group (years)	
18–40	4 (8.9)
41–60	23 (51.1)
>60	18 (40.0)
Sex	
Male	32 (71.1)
Female	13 (28.9)
Cardiovascular risk factors	
Hypertension	23 (51.1)
Diabetes mellitus	17 (37.8)
Smoking	16 (35.6)
Dyslipidaemia	19 (42.2)
Type of myocardial infarction	
STEMI	31 (68.9)
NSTEMI	14 (31.1)
Continuous variables	Mean $\pm$ SD
Age (years)	57.8 ± 11.6
Left ventricular ejection fraction (%)	44.6 ± 9.2

Cardiovascular risk factors were not mutually exclusive.

Peak hs-TnI concentrations demonstrated substantial variability across the study population, ranging from 420 to 42,600 ng/L. The distribution is presented in Table 2.

Table 2. Distribution of peak serum hs-TnI concentrations (N = 45)

Parameter	Value
Available measurements	45
Median (IQR), ng/L	7,520 (3,800–15,400)
Minimum, ng/L	420
Maximum, ng/L	42,600

Myocardial necrosis and cardiomyocyte degeneration were the most frequently represented histopathological findings. Inflammatory infiltration and interstitial oedema were also common, whereas myocardial fibrosis and contraction-band changes were less frequently observed. The distribution of individual histopathological features is presented in Table 3.

Table 3. Distribution of histopathological features of myocardial injury (N = 45)

Histopathological feature	Present, n (%)	Absent, n (%)
Myocardial necrosis	38 (84.4)	7 (15.6)
Cardiomyocyte degeneration	36 (80.0)	9 (20.0)
Inflammatory cell infiltration	30 (66.7)	15 (33.3)
Interstitial oedema	27 (60.0)	18 (40.0)
Myocardial fibrosis	19 (42.2)	26 (57.8)
Contraction-band changes	14 (31.1)	31 (68.9)

Multiple histopathological features may coexist in the same specimen.

Moderate necrosis was the most frequently represented grade, followed by severe and mild necrosis. Seven specimens had no demonstrable necrosis. Median peak hs-TnI concentrations increased progressively across necrosis grades (Table 4).

Table 4. Histopathological severity of myocardial necrosis and corresponding peak hs-TnI concentrations (N = 45)

Necrosis grade	Patients, n (%)	Peak hs-TnI, median (IQR), ng/L
Absent	7 (15.6)	1,450 (860–2,545)
Mild	10 (22.2)	4,325 (2,742–6,688)
Moderate	17 (37.8)	9,600 (6,200–15,400)
Severe	11 (24.4)	18,500 (12,630–29,950)
Total	45 (100.0)	—

Absence of necrosis refers only to the sampled myocardial tissue.

Inflammatory infiltration was predominantly mild or moderate, whereas severe infiltration was relatively uncommon. Myocardial fibrosis was absent in more than half of the specimens, with mild fibrosis being the most frequently represented grade among specimens showing fibrosis (Table 5).

Table 5. Histopathological grades of inflammatory infiltration and myocardial fibrosis (N = 45)

Histopathological grade	Inflammatory infiltration, n (%)	Myocardial fibrosis, n (%)
Absent	15 (33.3)	26 (57.8)
Mild	13 (28.9)	10 (22.2)
Moderate	12 (26.7)	6 (13.3)
Severe	5 (11.1)	3 (6.7)
Total	45 (100.0)	45 (100.0)

Spearman's rank correlation analysis demonstrated a positive association between peak hs-TnI concentrations and myocardial necrosis grade. A weaker positive correlation was observed with inflammatory infiltration, whereas the correlation with myocardial fibrosis was not statistically significant. Mann–Whitney U tests did not demonstrate statistically significant differences in hs-TnI concentrations according to cardiomyocyte degeneration, interstitial oedema or contraction-band changes. The complete analyses are presented in Table 6.

Table 6. Association between peak serum hs-TnI concentrations and histopathological features of myocardial injury (N = 45)

Histopathological feature	n	Peak hs-TnI, median (IQR), ng/L	Test statistic	p-value
Myocardial necrosis			$\rho = 0.814$	<0.001

Absent	7	1,450 (860–2,545)		
Mild	10	4,325 (2,742–6,688)		
Moderate	17	9,600 (6,200–15,400)		
Severe	11	18,500 (12,630–29,950)		
Inflammatory infiltration			$\rho = 0.372$	0.012
Absent	15	4,400 (1,835–7,230)		
Mild	13	11,050 (7,050–13,950)		
Moderate	12	16,290 (5,168–21,295)		
Severe	5	7,900 (5,000–11,480)		
Myocardial fibrosis			$\rho = 0.087$	0.571
Absent	26	7,285 (3,500–15,592)		
Mild	10	6,970 (5,150–13,950)		
Moderate	6	6,820 (3,042–10,920)		
Severe	3	12,500 (10,625–17,500)		
Cardiomyocyte degeneration			$U = 219$	0.109
Absent	9	3,400 (2,590–7,520)		
Present	36	8,425 (4,685–15,585)		
Interstitial oedema			$U = 313$	0.107
Absent	18	5,600 (2,515–10,635)		
Present	27	9,240 (5,000–17,175)		
Contraction-band changes			$U = 185$	0.440
Absent	31	8,100 (4,590–14,590)		
Present	14	6,000 (2,742–17,362)		

ρ : Spearman's rank correlation coefficient; U : Mann–Whitney U statistic; IQR: interquartile range. Tests are two-sided. Quartiles are rounded to the nearest whole ng/L.

DISCUSSION

The present study examined the relationship between serum high-sensitivity cardiac troponin I (hs-TnI) concentrations and histopathological features of myocardial injury in 45 patients with acute myocardial infarction (AMI). The principal finding was a progressive increase in median peak hs-TnI concentrations with increasing histopathological severity of myocardial necrosis. Myocardial necrosis was the most frequently observed histopathological feature, followed by cardiomyocyte degeneration and inflammatory cell infiltration. These findings suggest a relationship between circulating cardiac biomarkers and the morphological severity of myocardial injury.

The study population had a mean age of 57.8 ± 11.6 years, with a predominance of males (71.1%). Hypertension, dyslipidaemia and diabetes mellitus were common cardiovascular risk factors, while ST-elevation myocardial infarction accounted for 68.9% of cases. These characteristics provide important clinical context because the extent of myocardial injury may be influenced by underlying cardiovascular risk factors, infarct characteristics and the timing of reperfusion.

The median peak hs-TnI concentration was 7,520 ng/L (IQR: 3,800–15,400), with values ranging from 420 to 42,600 ng/L. This variability may reflect differences in the extent of myocardial injury, infarct location, reperfusion status and timing of biomarker measurement. Reichlin et al. and Keller et al. demonstrated the diagnostic utility of sensitive cardiac troponin assays in patients presenting with suspected AMI.[2,3]

Nevertheless, elevated troponin concentrations indicate myocardial injury and do not independently establish its ischaemic mechanism. Interpretation must therefore incorporate clinical presentation, electrocardiographic findings and other relevant investigations.[1,4,5]

The most notable finding was the graded increase in median hs-TnI concentrations across myocardial necrosis categories. Median concentrations increased from 1,450 ng/L in patients without demonstrable necrosis to 4,325 ng/L, 9,600 ng/L and 18,500 ng/L in those with mild, moderate and severe necrosis, respectively. The median concentration in the severe necrosis group was approximately 12.76 times that of the group without demonstrable necrosis. This progressive increase is biologically plausible because irreversible ischaemic injury disrupts cardiomyocyte integrity, resulting in the release of intracellular cardiac troponins into the circulation.[1,4] However, the strength and statistical significance of the association cannot be established from group medians alone.

These observations are broadly consistent with the findings of Younger et al., who evaluated 93 patients following myocardial infarction and examined the relationship between cardiac troponin I concentrations and infarct size measured using late gadolinium-enhanced cardiovascular magnetic resonance imaging. Troponin I concentrations measured at 12 and 72 hours correlated with infarct size, with reported correlation coefficients of 0.56 and 0.62, respectively.[7]

Their findings support the relationship between circulating troponin concentrations and the extent of myocardial injury. However, cardiovascular magnetic resonance imaging assesses infarct burden across the myocardium, whereas histopathological grading describes morphological abnormalities within sampled tissue. Consequently, histopathological severity cannot be considered directly equivalent to total infarct size.

Inflammatory cell infiltration was identified in 66.7% of specimens. Inflammatory infiltration represents an important component of the myocardial response to ischaemic injury, involving the recruitment of inflammatory cells and the clearance of necrotic tissue. The extent of inflammatory infiltration may depend on the duration of ischaemia, reperfusion status and the interval between myocardial injury and tissue collection. Although inflammation may accompany myocardial necrosis, its relationship with circulating hs-TnI concentrations requires analysis of paired biochemical and histopathological observations.

Myocardial fibrosis was identified in 42.2% of specimens. Fibrosis may represent previous ischaemic injury, chronic myocardial remodelling or another pre-existing pathological process rather than injury arising exclusively from the current infarction. In contrast, peak hs-TnI concentrations primarily reflect myocardial injury during the acute clinical episode. Therefore, the relationship between fibrosis and circulating troponin concentrations must be interpreted in the context of previous cardiovascular disease and the interval between myocardial injury and tissue collection.

Cardiomyocyte degeneration, interstitial oedema and contraction-band changes were identified in 80.0%, 60.0% and 31.1% of specimens, respectively. These findings represent different morphological manifestations of myocardial injury and may occur at different stages of ischaemia, reperfusion and tissue repair. Their presence or absence in a limited myocardial specimen may not correspond directly to the overall extent of myocardial damage. Further patient-level analysis is required to determine their associations with circulating hs-TnI concentrations.

An important consideration is the interpretation of endomyocardial biopsy findings in patients with AMI. Cooper et al. emphasized that endomyocardial biopsy has specific clinical indications and that its diagnostic utility depends on appropriate patient selection and tissue assessment.[6]

Because myocardial infarction may involve regions outside the biopsy site, the absence of necrosis in a limited specimen does not exclude myocardial infarction elsewhere in the myocardium. Similarly, the histopathological severity observed in sampled tissue cannot automatically be considered representative of the entire infarct. Documentation of the clinical indications for biopsy, anatomical sampling sites and timing of tissue collection is therefore essential.

The present study has several limitations. The relatively small sample size of 45 patients may limit the precision and generalizability of the findings. Furthermore, renal function, infarct location, reperfusion status and the timing of biomarker

measurement may influence circulating hs-TnI concentrations. Larger studies incorporating standardized tissue assessment, documented biomarker timing and paired individual-level analyses are warranted.

CONCLUSION

The present study demonstrated a progressive increase in median serum high-sensitivity cardiac troponin I (hs-TnI) concentrations with increasing histopathological severity of myocardial necrosis in patients with acute myocardial infarction. Myocardial necrosis was the most frequently observed histopathological feature, followed by cardiomyocyte degeneration and inflammatory cell infiltration. Median hs-TnI concentrations were approximately 12.76 times higher in patients with severe necrosis than in those without demonstrable necrosis in the sampled tissue. These findings suggest a relationship between circulating hs-TnI concentrations and the histopathological severity of myocardial injury. Furthermore, limited myocardial sampling may not accurately represent the total extent of infarction.

DECLARATIONS

Ethical Approval: The study was approved by the Institutional Ethics Committee of the participating institution.

Informed Consent: Written informed consent was obtained from all study participants.

Funding: The study received no external funding.

Conflict of Interest: The authors declare no conflicts of interest.

Authors' Contributions: All authors contributed to the study design, data analysis, manuscript preparation and critical revision, and approved the final manuscript.

Acknowledgments: The authors acknowledge the support of the Departments of Cardiology and Pathology.

REFERENCES

1. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction (2018). *J Am Coll Cardiol*. 2018 Oct 30;72(18):2231-2264. doi: 10.1016/j.jacc.2018.08.1038.
2. Reichlin T, Hochholzer W, Bassetti S, Steuer S, Stelzig C, Hartwiger S, et al. Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. *N Engl J Med*. 2009 Aug 27;361(9):858-67. doi: 10.1056/NEJMoa0900428.
3. Keller T, Zeller T, Peetz D, Tzikas S, Roth A, Czynz E, et al. Sensitive troponin I assay in early diagnosis of acute myocardial infarction. *N Engl J Med*. 2009 Aug 27;361(9):868-77. doi: 10.1056/NEJMoa0903515.
4. Apple FS, Sandoval Y, Jaffe AS, Ordonez-Llanos J; IFCC Task Force on Clinical Applications of Cardiac Bio-Markers. Cardiac Troponin Assays: Guide to Understanding Analytical Characteristics and Their Impact on Clinical Care. *Clin Chem*. 2017 Jan;63(1):73-81. doi: 10.1373/clinchem.2016.255109.
5. Chapman AR, Adamson PD, Mills NL. Assessment and classification of patients with myocardial injury and infarction in clinical practice. *Heart*. 2017 Jan 1;103(1):10-18. doi: 10.1136/heartjnl-2016-309530.
6. Cooper LT, Baughman KL, Feldman AM, Frustaci A, Jessup M, Kuhl U, et al. The role of endomyocardial biopsy in the management of cardiovascular disease: a scientific statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology. Endorsed by the Heart Failure Society of America and the Heart Failure Association of the European Society of Cardiology. *J Am Coll Cardiol*. 2007 Nov 6;50(19):1914-31. doi: 10.1016/j.jacc.2007.09.008.
7. Younger JF, Plein S, Barth J, Ridgway JP, Ball SG, Greenwood JP. Troponin-I concentration 72 h after myocardial infarction correlates with infarct size and presence of microvascular obstruction. *Heart*. 2007 Dec;93(12):1547-51. doi: 10.1136/hrt.2006.109249.
8. Sandoval Y, Jaffe AS. Type 2 Myocardial Infarction: JACC Review Topic of the Week. *J Am Coll Cardiol*. 2019 Apr 16;73(14):1846-1860. doi: 10.1016/j.jacc.2019.02.018.
9. Collet JP, Thiele H, Barbato E, Barthélémy O, Bauersachs J, Bhatt DL, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. *Rev Esp Cardiol (Engl Ed)*. 2021 Jun;74(6):544. doi: 10.1016/j.rec.2021.05.002.