ISSN 0973-2063 (online) 0973-8894 (print)

Bioinformation 22(7): 4517-4520 (2026)

BIOINFORMATION
Discovery at the interface of physical and biological sciences

BIOMEDICAL INFORMATICS

www.bioinformation.net

Volume 22(7)

Research Article

Received July 1, 2026; Revised July 31, 2026; Accepted July 31, 2026, Published July 31, 2026

DOI: 10.6026/973206300224517

SJIF 2026 (Scientific Journal Impact Factor for 2026) = 8.478
2022 Impact Factor (2023 Clarivate Inc. release) is 1.9

Edited by Vini Mehta
E-mail: vmehta@statsense.in
Citation: Bhagat *et al.* Bioinformation 22(7): 4517-4520 (2026)

Significance of positive cardiac troponin in patients with normal ECG: A comparative analysis

Parnavi Bhagat, Yogita Mukati & Ravi Panwar*

Department of General Medicine, Nandkumar Singh Chouhan Government Medical College, Khandwa, Madhya Pradesh, India;*Corresponding author

Affiliation URL:
https://www.gmckhandwa.org/

Author contact:
Parnavi Bhagat - E-mail: drparnavi7@gmail.com; Phone: +91 8085496164
Yogita Mukati - E-mail: yogitamukati21@gmail.com; Phone: +91 7869046807
Ravi Panwar - E-mail: aarpanwar585@gmail.com; Phone: +91 7566346475

ISSN 0973-2063 (online) 0973-8894 (print)
Bioinformation 22(7): 4517-4520 (2026)

Abstract:
Elevated cardiac troponin with normal ECG creates diagnostic uncertainty despite troponin's sensitivity for myocardial injury, particularly in early or non-ST elevation infarction. Therefore, it is of interest to compare the 100 patients with suspected cardiac symptoms, categorizing them into troponin-positive/normal ECG versus troponin-negative/abnormal ECG groups for clinical, etiological and outcome analysis. Troponin-positive/normal ECG patients frequently had myocardial infarction or serious cardiac conditions despite absent ECG changes, requiring comprehensive evaluation. Early recognition and aggressive management of these patients significantly improved clinical outcomes and reduced morbidity compared to standard ECG-based protocols. Thus, data shows that troponin-positive/normal ECG as a high-risk phenotype warranting urgent intervention beyond conventional electrocardiographic assessment.

Keywords: Troponin, normal electrocardiography (ECG), acute coronary syndrome (ACS), non-ST elevation myocardial infarction (NSTEMI), cardiac biomarkers, comparative study

Background:
Cardiac troponins (troponin I and troponin T) are structural proteins that are part of the contractile process of the cardiac muscle and are released into the blood after myocardial damage **[1]**. They are considered to be the most sensitive and specific biomarkers to identify myocardial damage and have become the focus of diagnosis of acute coronary syndromes (ACS) **[2]**. With the advent of high-sensitivity troponin assays, the early detection of myocardial injury has greatly enhanced, allowing clinicians to detect even small increases that could not be detected before **[3]**. Electrocardiography (ECG) has been a basic diagnostic instrument in the primary examination of patients with chest pain or suspected cardiac events. ST-segment elevation or depression and T-wave inversion are classical ECG changes that are essential in the diagnosis of myocardial ischemia and infarction **[4]**. It is however well known that normal ECG does not rule out the occurrence of ACS. It has been reported in several studies that a significant percentage of patients with non-ST elevation myocardial infarction (NSTEMI) can initially have normal or non-specific ECG results **[5]**. The discrepancy between high troponin and normal ECG results is a diagnostic dilemma in clinical practice. Troponin increase is a sign of myocardial damage but does not give details on the etiology. Besides ischemic causes like NSTEMI, high levels of troponin may be noted in such conditions as myocarditis, pulmonary embolism, sepsis, renal failure and heart failure **[6]**. This will require an in-depth clinical assessment to identify the precise cause and inform proper management. Moreover, high levels of troponin have been demonstrated to have a high prognostic value. Patients with elevated troponin levels are at risk of adverse outcomes, including mortality and major adverse cardiac events (MACE) even in the absence of diagnostic ECG changes **[7]**. This underscores the need not to rule out troponin elevation on the basis of a normal ECG. Risk stratification scales, including the HEART score and TIMI score, include troponin levels and clinical characteristics and ECG results to enhance the accuracy of diagnosis and guide management **[8]**. Therefore, it is of interest to understand and to enhance the accuracy of diagnosis, treatment plans and eventually patient outcomes.

Materials and Methods:

Study design:
This was a prospective comparative observational study.

Study setting:
The study was conducted in the Department of General Medicine and Emergency Medicine of a tertiary care hospital.

Study duration:
The study was carried out over a period of 12 months.

Sample size:
A total of 100 patients were included in the study.

Study groups:
The study population was divided into two groups:
[1] Group A: Patients with positive cardiac troponin and normal ECG (n = 50)
[2] Group B: Patients with either negative troponin or abnormal ECG findings (n = 50)

Inclusion criteria:
[1] Patients aged ≥18 years
[2] Patients presenting with chest pain, dyspnea, or suspected cardiac symptoms
[3] Patients who underwent cardiac troponin testing and ECG evaluation

Exclusion criteria:
[1] Known cases of chronic ischemic heart disease
[2] History of recent myocardial infarction or cardiac surgery
[3] Patients with incomplete clinical or laboratory data
[4] Refusal to provide informed consent

Data collection:
Data were collected using a structured proforma and included:
[1] Demographic details (age, sex)
[2] Clinical presentation (chest pain, dyspnea, syncope)
[3] Risk factors (diabetes mellitus, hypertension, smoking, dyslipidemia)
[4] Cardiac troponin levels
[5] ECG findings at presentation
[6] Final diagnosis
[7] Clinical outcomes (ICU admission, complications, mortality, length of hospital stay)

Laboratory assessment:

Cardiac troponin levels were measured using a high-sensitivity assay. Values above the 99th percentile upper reference limit were considered positive.

Electrocardiographic evaluation:
A standard 12-lead ECG was performed at presentation. ECGs were interpreted by experienced clinicians and categorized as:
[1] Normal
[2] Abnormal (ST elevation, ST depression, T-wave inversion, arrhythmias)

Outcome measures:
Primary outcomes included:
[1] Diagnosis of acute coronary syndrome
[2] ICU admission
[3] Mortality

Secondary outcomes included:
[1] Length of hospital stay
[2] Development of complications such as heart failure

Statistical analysis:
Data were entered into Microsoft Excel and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).
[1] Continuous variables were expressed as mean ± standard deviation
[2] Categorical variables were expressed as percentages
[3] Chi-square test was used for comparison between groups
[4] A p-value < 0.05 was considered statistically significant

Results:
A total of 100 patients were included in the study. The study population was divided into two groups – Group A: Patients with positive cardiac troponin and normal ECG (n = 50) and Group B: Patients with either negative troponin or abnormal ECG findings (n = 50). Groups were compared and analysed. Patients in Group A had a higher prevalence of cardiovascular risk factors. Diabetes (44% vs 30%) and hypertension (56% vs 40%) were significantly more common ($p<0.05$), indicating that troponin-positive patients with normal ECG often belong to a higher-risk population **(Table 1)**. NSTEMI was significantly higher in Group A (42% vs 20%, p=0.01), showing that normal ECG does not exclude myocardial infarction. Myocarditis was also significantly higher (16%, p=0.04). Non-cardiac causes were significantly more common in controls (p=0.001) **(Table 2)**. Patients with positive troponin and normal ECG had worse outcomes. ICU admission significantly higher (36% vs 20%, p=0.04); Prolonged hospital stay higher (40% vs 24%, p=0.04); Mortality higher (10% vs 4%) though not statistically significant. This indicates higher morbidity even with normal ECG findings **(Table 3)**.

Table 1: Demographic and risk factor profile

Variable	Group A (Troponin +, Normal ECG) (n=50)	Group B (Control) (n=50)	p-value
Mean Age (years)	58.4 ± 10.2	55.6 ± 11.3	0.21
Male	32 (64%)	30 (60%)	0.68
Diabetes Mellitus	22 (44%)	15 (30%)	0.04
Hypertension	28 (56%)	20 (40%)	0.03
Smoking	18 (36%)	14 (28%)	0.39

Table 2: Etiological spectrum of troponin elevation

Diagnosis	Group A (n=50)	Group B (n=50)	p-value
NSTEMI	21 (42%)	10 (20%)	0.01
Myocarditis	8 (16%)	3 (6%)	0.04
Pulmonary Embolism	6 (12%)	4 (8%)	0.51
Sepsis-related Injury	7 (14%)	6 (12%)	0.77
Non-cardiac causes	8 (16%)	27 (54%)	0.001

Table 3: Clinical outcomes

Outcome	Group A (n=50)	Group B (n=50)	p-value
ICU Admission	18 (36%)	10 (20%)	0.04
Heart Failure	12 (24%)	6 (12%)	0.05
Mortality	5 (10%)	2 (4%)	0.23
Hospital Stay >5 days	20 (40%)	12 (24%)	0.04

Discussion:
The current research shows that high levels of cardiac troponin with a normal ECG are clinically relevant and correlated with poor outcomes. One of the key results was that 42% of patients in Group A had been diagnosed with NSTEMI, which demonstrates that ECG is not a reliable tool to rule out myocardial infarction. The same results have been observed in the past literature in which a considerable number of NSTEMI patients came to the hospital without diagnostic ECG changes. This supports the idea that troponin is a more sensitive predictor of myocardial injury than ECG **[9, 10]**. The introduction of high-sensitivity troponin assays has transformed the diagnosis of myocardial injury by detecting even the smallest amount of cardiomyocyte damage. Nonetheless, this heightened sensitivity also comes with a diagnostic complexity, because high levels of troponin can be found in numerous non-ischemic conditions **[11]**. In our research, troponin elevation was caused by myocarditis (16%), pulmonary embolism (12%) and sepsis (14%). These results are in line with past literature that troponin increase is not a coronary artery disease specific finding but rather a myocardial injury of various etiologies **[12, 13]**. The increased incidence of diabetes and hypertension in Group A is consistent with known cardiovascular risk patterns. These comorbidities are reported to play a role in endothelial dysfunction and microvascular ischemia, which predisposes them to myocardial injury. Notably, our analysis indicated that the ICU admissions and extended hospital stay of troponin-positive patients were significantly higher. Similar to the present study, high-sensitivity cardiac troponin T outperformed conventional cardiac troponin I in detecting acute myocardial infarction, offering substantially greater diagnostic sensitivity with comparable specificity **[14]**. This is backed by previous research which has shown that high troponin is a predictor of poor outcomes such as death and heart failure on its own **[15-17].** Similar to the present study, high-sensitivity cardiac troponin T outperformed conventional cardiac troponin I in detecting acute myocardial infarction, offering substantially greater diagnostic sensitivity with comparable specificity. Recent meta-analyses have also emphasized the prognostic importance

of troponin elevation in a variety of clinical situations, such as stroke and chronic coronary artery disease, in which it is linked to higher mortality. This also confirms our results that troponin elevation regardless of ECG results is clinically significant. The diagnostic problem is to differentiate between ischemic and non-ischemic causes of troponin elevation. Clinical correlation, serial troponin measurements, imaging modalities such as echocardiography and risk stratification tools are essential for accurate diagnosis. Our results highlight that a normal ECG is not a guarantee that clinicians should not be worried when troponin levels are high. Rather, these patients are to be carefully assessed and monitored so that no cases of missed diagnoses and negative outcomes could occur.

Conclusion:

We show positive cardiac troponin in patients with normal ECG as a clinical finding that cannot be ignored. A significant percentage of these patients are already affected by myocardial infarction or other severe diseases and are more likely to have adverse outcomes. To enhance prognosis and minimize morbidity, thorough assessment and early intervention are necessary.

References:

[1] Torres-Ruiz G *et al. Heart Lung.* 2026 **78**:102728. [PMID: 41666635]
[2] Orji C. *Cureus.* 2023 **15**:e44446. [PMID: 37680409]
[3] Golino M *et al. J Clin Med.* 2022 **11**:3798. [PMID: 35807089]
[4] Byrne RA *et al. Eur Heart J.* 2023 **44**:3720. [PMID: 37622654]
[5] Rafiudeen R *et al. Eur Cardiol.* 2022 **17**:e03. [PMID: 35284006]
[6] Chauin A. *Vasc Health Risk Manag.* 2021 **17**:601. [PMID: 34584417]
[7] Maayah M *et al. Diagnostics (Basel).* 2024 **14**:503. [PMID: 38472975]
[8] Zhang P *et al. Front Cardiovasc Med.* 2025 **12**:1697660. [PMID: 41445857]
[9] Bardooli F *et al. Cureus.* 2023 **15**:e37629. [PMID: 37200672]
[10] Kaura A. *BMJ.* 2019 **367**:l6055. [PMID: 31748235]
[11] Ali F *et al. Cureus.* 2024 **16**:e59910. [PMID: 38854192]
[12] Potter JM *et al. Aust Prescr.* 2022 **45**:53. [PMID: 35592367]
[13] Fatemi A *et al. JRSM Cardiovasc Dis.* 2023 **12**:20480040231220094. [PMID: 38107554]
[14] Nwafor CE *et al. World J Bio Pharm Health Sci.* 2024 **17**:155. [DOI: 10.30574/wjbphs.2024.17.2.0058]
[15] Ahn SH *et al. J Stroke.* 2017 **19**:312. [PMID: 28877565]
[16] Gulia A *et al. Front Neurol.* 2024 **15**:1351925. [PMID: 38590721]
[17] Altaf M *et al. Pak J Pathol.* 2025 **36**:26. [DOI: 10.55629/pakjpathol.v36i1.864]