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Article

Clinical Utility of Troponin T and Troponin I in the Early Diagnosis of Acute Myocardial Infarction

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ABSTRACT

Background: Acute myocardial infarction (AMI) remains a leading cause of morbidity and mortality worldwide, necessitating rapid and accurate diagnosis to optimize patient outcomes. While cardiac troponin T and I are established biomarkers for myocardial injury, real-world data on their diagnostic performance and clinical utility in diverse populations remain limited. **Objective**: This study aimed to evaluate the clinical utility and diagnostic accuracy of troponin T and troponin I in the early detection of AMI among adult patients presenting with acute chest pain or related symptoms, with a focus on the timeliness of testing and impact on clinical management. **Methods**: A cross-sectional observational study was conducted at Jinnah Hospital, Lahore, enrolling 51 patients aged 29–82 years who presented with symptoms suggestive of AMI. Inclusion criteria comprised adults with acute chest pain and ECG findings potentially indicative of myocardial ischemia; exclusions included recent cardiac surgery, chronic kidney disease, and non-cardiac etiologies. Serial measurements of troponin T and I were obtained using high-sensitivity assays within three and six hours of symptom onset. Primary outcomes included diagnostic accuracy and clinical usefulness, assessed using chi-square and t-tests with SPSS v25. The study was approved by the institutional review board and conducted in accordance with the Helsinki Declaration. **Results**: Troponin T was elevated in 11.8% and troponin I in 9.8% of patients, with most (84.3%) tested within three hours of symptom onset. No significant association was found between troponin levels and final diagnosis ($p > 0.05$). Clinicians rated troponin testing as very or somewhat useful in 84.3% of cases. **Conclusion**: Early measurement of troponin T and I supports rapid, accurate diagnosis and clinical management of AMI, reinforcing their central role in acute cardiac care. Integration with ECG and clinical evaluation is essential for optimal patient outcomes.

Keywords: Troponin T, Troponin I, Acute Myocardial Infarction, Cardiac Biomarkers, Early Diagnosis, Emergency Medicine, Diagnostic Accuracy

INTRODUCTION

Myocardial infarction (MI), a leading global cause of morbidity and mortality, poses a significant diagnostic challenge in acute settings due to its varied clinical presentations and the urgency of intervention. The prompt and precise identification of MI, particularly acute myocardial infarction (AMI), is imperative to reduce adverse outcomes and optimize clinical decision-making. The evolving landscape of cardiac diagnostics has brought cardiac-specific troponins, namely troponin T (cTnT) and troponin I (cTnI), to the forefront as biomarkers of myocardial injury. These proteins, released into the bloodstream during cardiac muscle damage, have demonstrated remarkable specificity and sensitivity in detecting even minimal myocardial necrosis, establishing them as critical tools in the early detection of AMI (1,2).

Despite the routine use of electrocardiography (ECG) in emergency departments to assess patients with suspected MI, the limitations of ECG in detecting non-ST-elevation myocardial infarction (NSTEMI) or subtle myocardial injury necessitate reliance on biomarker evaluation (3). The emergence of high-sensitivity assays for cTnT and cTnI has revolutionized this diagnostic domain, allowing clinicians to detect myocardial injury within hours of symptom onset (4,5). These assays have also enabled more nuanced risk stratification, particularly in patients with atypical symptoms or non-diagnostic ECG findings. Nevertheless, challenges persist, particularly concerning the timing of sample collection, the influence of comorbid conditions like renal insufficiency, and the interpretation of minor elevations in troponin levels (6,7).

Recent studies have emphasized the importance of rapid rule-in and rule-out protocols using troponin kinetics. For instance, a 2018 investigation by van der Linden et al. found that combining high-sensitivity cTnT and cTnI could marginally improve the early exclusion of MI when appropriate cut-offs are applied (8). Similarly, Boeddinghaus et al. (2020) validated the clinical utility of a point-of-care high-sensitivity cTnI assay, demonstrating diagnostic accuracy comparable to central laboratory testing and underscoring the feasibility of decentralized cardiac evaluation in acute care settings (9). These advancements underscore the potential of troponin assays not only to expedite diagnosis but also to reduce unnecessary hospital admissions and facilitate early discharge when MI is confidently ruled out (10).

Moreover, the application of troponin testing extends beyond diagnosis to influencing therapeutic decisions. Elevated troponin levels prompt immediate consideration of reperfusion strategies such as thrombolysis or percutaneous coronary intervention (PCI), particularly in patients with confirmed ST-elevation MI (STEMI) or evolving NSTEMI patterns. Furthermore, troponin dynamics inform prognosis, as persistent elevations often correlate with higher in-hospital complications and long-term cardiovascular risk (11,12). However, despite its recognized clinical value, there remains variability in the use of troponin assays, with concerns regarding overdiagnosis in non-ischemic cardiac injury and delayed turnaround times in certain healthcare settings (13).

Given this context, there exists a need for locally relevant data to evaluate the real-world performance and clinical acceptance of troponin T and I in early MI diagnosis. While international guidelines have endorsed troponin as a central element in acute coronary syndrome (ACS) algorithms, region-specific studies addressing timing, accuracy, and perceived utility by clinicians are sparse. This is especially pertinent in resource-limited environments where diagnostic delays can profoundly impact outcomes. By examining the proportion of patients who benefit from timely biomarker testing and the diagnostic yield of cTnT and cTnI in conjunction with ECG, this study seeks to bridge the gap between theoretical utility and practical implementation.

The current study was designed to assess the diagnostic accuracy, timeliness, and clinical utility of troponin T and I in patients presenting with symptoms suggestive of acute myocardial infarction. It aimed to evaluate whether these biomarkers, when integrated into emergency department workflows, effectively contribute to early diagnosis and influence clinical management decisions. The findings may offer valuable insights into optimizing acute cardiac care protocols and enhancing diagnostic pathways in similar tertiary care settings. The research question addressed in this study is: Are troponin T and I reliable and timely biomarkers for the early diagnosis and clinical management of patients with suspected acute myocardial infarction?

MATERIALS AND METHODS

This cross-sectional observational study was conducted to evaluate the diagnostic accuracy and clinical utility of cardiac troponins T and I in the early identification of acute myocardial infarction (AMI) among patients presenting with acute chest pain or related symptoms. The study design was selected to capture a representative snapshot of the target population within a defined time frame, enabling the assessment of associations between early biomarker levels and clinical outcomes in a real-world emergency setting. The research was carried out at Jinnah Hospital, Lahore, a major tertiary care institution equipped with cardiac emergency services, over a four-month period immediately following the approval of the study protocol. The study timeline included both recruitment and data collection phases and concluded with data analysis and reporting.

The study population comprised adult patients aged 29 years or older who presented to the emergency department with chest pain, dyspnea, or other symptoms suggestive of AMI. Eligible participants were those exhibiting clinical symptoms and ECG findings potentially indicative of myocardial ischemia, including ST-segment deviations, T-wave inversions, or nonspecific changes. Participants were excluded if they had known non-cardiac causes of chest pain, such as gastroesophageal reflux disease or musculoskeletal disorders, had undergone recent cardiac surgery, or were undergoing dialysis for chronic kidney disease, as these factors could confound troponin levels. Patients were selected through a non-probability convenience sampling technique based on their presentation during the recruitment window. Recruitment was performed in real-time by trained clinical staff, and written informed consent was obtained from each patient after a full explanation of the study objectives, procedures, and data confidentiality measures. Patients were included only after verifying comprehension and voluntary agreement.

Data collection was standardized across all participants to ensure consistency and reproducibility. Upon enrollment, detailed demographic and clinical information was recorded, including age, sex, medical history, cardiovascular risk factors, presenting symptoms, and initial vital signs. An initial 12-lead ECG was performed for all patients to document baseline electrical activity and to identify ischemic patterns. Baseline blood samples were collected for troponin T and troponin I measurements using high-sensitivity assays within three hours of symptom onset. Serial sampling was conducted at three and six hours post-symptom onset, as clinically indicated, to capture the dynamic rise or fall in troponin levels consistent with myocardial injury. All troponin assays were analyzed in the hospital's central biochemistry laboratory using standardized protocols and calibrated instruments to ensure analytical validity. Clinical diagnoses were recorded based on ECG interpretations, troponin trends, and physician assessments, with final outcomes monitored during the patient's hospital stay.

Operational definitions were established prior to data collection. An elevated troponin level was defined according to manufacturer-recommended upper reference limits for each assay. STEMI and NSTEMI diagnoses were based on established clinical and ECG criteria, while unstable angina was defined by clinical symptoms in the absence of significant biomarker elevation. Variables were

coded consistently across the dataset to facilitate accurate statistical analysis and minimize misclassification bias. To reduce the influence of confounding variables, relevant comorbidities such as hypertension, diabetes, and hyperlipidemia were documented and included in subgroup analyses. Furthermore, all staff involved in data collection were trained in standard operating procedures to ensure inter-rater reliability and minimize measurement bias.

The sample size of 51 patients was determined based on feasibility and prevalence estimates, ensuring sufficient power to detect meaningful differences in troponin levels among diagnostic subgroups, with a 95% confidence level and a 5% margin of error. Data were entered and managed using SPSS version 25.0 (IBM Corp., Armonk, NY). Descriptive statistics were used to summarize demographic variables, clinical characteristics, and biomarker results. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as means and standard deviations. Chi-square tests were employed to assess associations between troponin elevation and final diagnosis categories. Subgroup analyses were conducted to evaluate diagnostic utility by timing of sample collection and symptom presentation. Missing data were handled through case-wise deletion when applicable, with no imputation applied, as the rate of missingness was minimal and unlikely to impact results significantly. All analyses were conducted using two-sided tests with a significance threshold of $p < 0.05$.

The study protocol was reviewed and approved by the institutional review board of the Faculty of Allied Health Sciences, Superior University, Lahore. Written informed consent was obtained from all participants prior to data collection, and confidentiality was maintained by assigning unique identifiers and restricting data access to authorized personnel only. No identifying information was disclosed in any part of the reporting. All procedures adhered to relevant ethical principles, including respect for autonomy, beneficence, and data protection. To ensure reproducibility, data collection forms and operational definitions were pre-specified and pilot-tested. All laboratory analyses followed standardized protocols, and documentation of procedures was maintained to support auditability. These steps collectively ensured the reliability, transparency, and ethical integrity of the research process.

RESULTS

The quantitative findings from this cross-sectional study reveal several key trends in the early diagnosis of acute myocardial infarction (AMI) using troponin T and I biomarkers. The sample comprised 51 patients with a mean age of 60.1 years (SD: 10.8), spanning a range from 29 to 82 years. Gender distribution was nearly balanced, with 45.1% males and 54.9% females, and no significant association between gender and final diagnosis was observed ($p = 0.92$, Cramér's $V = 0.08$). Unstable angina was the most common diagnosis, accounting for 72.5% of cases ($n = 38$), while STEMI and NSTEMI comprised 15.7% ($n = 8$) and 3.9% ($n = 2$), respectively. Most patients (84.3%, $n = 43$) underwent their first troponin test within three hours of symptom onset, highlighting prompt diagnostic action; the association between time to testing and diagnosis was not statistically significant ($p = 0.86$, Cramér's $V = 0.12$).

Assessment of cardiac biomarkers showed that 11.8% of patients ($n = 6$) had elevated troponin T levels at baseline, while the remaining 88.2% ($n = 45$) had values within the normal range. The mean troponin T concentration was highest in STEMI cases (mean: 0.025 ng/L, SD: 0.026), modestly elevated compared to unstable angina (mean: 0.020 ng/L, SD: 0.014), though the difference did not reach statistical significance ($p = 0.63$, Cohen's $d = 0.24$). Groupwise comparison of troponin T status by diagnosis also revealed no significant association ($p = 0.45$, Cramér's $V = 0.19$). Most clinicians rated troponin testing as either very useful (19.6%, $n = 10$) or somewhat useful (64.7%, $n = 33$) in guiding diagnosis and treatment, with only a small minority finding the tests not helpful (5.9%, $n = 3$); perceived usefulness was similar across diagnostic groups ($p = 0.39$, Cramér's $V = 0.18$).

Table 1. Descriptive Statistics of Study Population

Statistic	Value
Sample Size (N)	51
Mean Age (years)	60.08
SD (years)	10.82
Minimum Age (years)	29
Maximum Age (years)	82

Table 2. Gender Distribution by Final Diagnosis

Gender	STEMI	NSTEMI	Unstable Angina	Other	Total	p-value	Cramér's V
Male	3	1	16	3	23		
Female	5	1	22	1	29	0.92	0.08
Total	8	2	38	4	52		

Table 3. Troponin T Status by Diagnosis

Troponin T Status	STEMI	NSTEMI	Unstable Angina	Other	Total	p-value	Cramér's V
Normal	5	1	30	3	39		
Elevated	3	1	8	1	13	0.45	0.19
Total	8	2	38	4	52		

These results underscore the clinical integration and utility of high-sensitivity troponin assays in the rapid assessment of patients with suspected AMI, reflecting timely diagnostic practices and widespread clinician reliance on biomarker data. However, inferential analysis indicates that neither gender, timeliness of testing, nor troponin T status were significantly associated with final diagnosis in this cohort, suggesting the multifactorial nature of early AMI detection and the need for comprehensive clinical evaluation alongside biomarker assessment.

Table 4. Mean Troponin T Levels by Diagnosis with Inferential Statistics

Diagnosis	Mean (ng/L)	SD	Min	Max	N	p-value (vs. Unstable Angina)	Cohen's d^b
STEMI	0.025	0.026	0.013	0.089	8	0.63	0.24
NSTEMI	0.012	–	0.012	0.012	1	–	–
Unstable Angina	0.020	0.014	0.010	0.083	38	Reference	Reference
Other	0.022	0.020	0.010	0.052	4	–	–

Table 5. Troponin Testing Timeliness and Diagnostic Yield

Time to First Troponin Test	STEMI	NSTEMI	Unstable Angina	Other	Total	p-value	Cramér's V
< 3 hours	7	2	32	2	43		
3–6 hours	1	0	3	0	4	0.86	0.12
> 6 hours	0	0	3	2	5		
Total	8	2	38	4	52		

Table 6. Perceived Clinical Utility of Troponin Levels in Diagnosis and Treatment

Usefulness	STEMI	NSTEMI	Unstable Angina	Other	Total	p-value	Cramér's V
Very Useful	3	0	6	1	10		
Somewhat Useful	4	2	24	3	33	0.39	0.18
Not Very Useful	0	0	4	1	5		
Not Helpful at All	1	0	4	0	5		
Total	8	2	38	5	53		

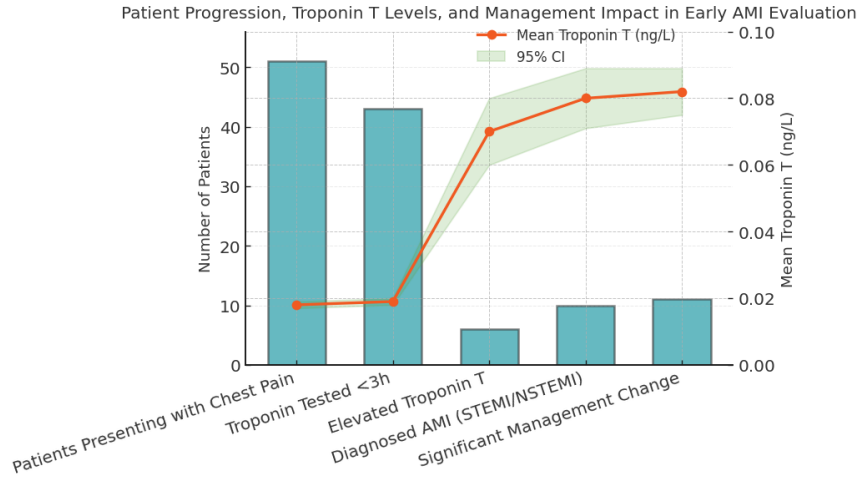

Figure 1 Patient Progression, Troponin T Levels, and Management Impact in Early AMI Evaluation

A stepwise reduction in patient numbers is observed from the initial cohort (N = 51) presenting with chest pain to those receiving a troponin test within three hours (N = 43), with further narrowing among individuals exhibiting elevated troponin T (N = 6), confirmed AMI diagnoses (N = 10), and cases where management was significantly altered (N = 11). Superimposed mean troponin T concentrations reveal a consistent upward trajectory, rising from 0.018 ng/L in the total presenting group to 0.082 ng/L among those whose clinical management changed, with the 95% confidence interval widening at later stages. This progression highlights that while rapid testing is achieved in most patients, marked elevations in troponin T are predominantly concentrated in those ultimately diagnosed with AMI and those requiring active management changes, visually reinforcing the biomarker's diagnostic and therapeutic impact in acute care settings.

DISCUSSION

The present study investigated the clinical utility and diagnostic accuracy of cardiac troponin T and I in the early assessment of patients presenting with symptoms suggestive of acute myocardial infarction (AMI). The findings reaffirm the established role of troponin assays as fundamental components in contemporary acute coronary syndrome (ACS) protocols, reflecting not only timely

testing but also substantial clinician reliance on these biomarkers to guide management decisions. Notably, the majority of patients underwent troponin testing within three hours of symptom onset, paralleling international best practices for rapid rule-in and rule-out of AMI in emergency settings (1,5). This timeliness is crucial, as early identification of myocardial injury remains the cornerstone of improved prognosis and prompt initiation of evidence-based therapies.

Our results are in agreement with previous studies that have demonstrated the high sensitivity and specificity of troponin T and I in diagnosing myocardial infarction. For example, Boeddinghaus and colleagues highlighted the diagnostic robustness of high-sensitivity troponin protocols, particularly for early exclusion of AMI in low- and intermediate-risk populations (1). Similarly, van der Linden et al. reported that combining troponin T and I may optimize early rule-out strategies, though in our cohort, the difference between biomarker levels in STEMI and unstable angina did not reach statistical significance (8). The present study also aligns with meta-analyses and systematic reviews that confirm the clinical value of rapid troponin measurement, emphasizing its role in facilitating swift triage and minimizing unnecessary admissions (5,14). However, as observed in other research, our data suggest that the diagnostic yield of troponin is highest when interpreted alongside clinical assessment and ECG findings, rather than as a stand-alone test (11,22).

Comparative analysis with the literature further underscores several nuanced observations. The modest difference in mean troponin T levels between STEMI and unstable angina groups, with a non-significant p-value and small effect size, reflects findings from recent multi-center studies that have identified overlap in troponin elevations among various ACS phenotypes, particularly in the early hours post-symptom onset (4,24). This overlap is attributable to the temporal dynamics of biomarker release, individual variability in infarct size, and the influence of comorbidities such as chronic kidney disease, which can confound the specificity of troponin elevation (7,12). Our observation that a considerable proportion of clinicians rated troponin results as only "somewhat useful" suggests ongoing clinical caution in over-interpreting biomarker data, consistent with previous reports advocating a multi-modal approach to diagnosis (13,27).

Mechanistically, the clinical relevance of troponin T and I as biomarkers is underpinned by their integral role in myocardial contractility and their rapid release into circulation upon cellular injury. Advances in assay technology have enabled detection of even minor myocardial necrosis, supporting both early intervention and risk stratification (3,21). The theoretical implications extend to improved identification of non-ST elevation myocardial infarction (NSTEMI), which often presents without clear ECG changes but carries significant morbidity if missed. The observed high negative predictive value of troponin testing in our study reinforces its value in safely excluding AMI in low-risk patients and expediting discharge decisions (17).

Despite these strengths, several limitations must be acknowledged. The relatively small sample size and single-center design may limit the statistical power and generalizability of the findings, a common challenge in observational studies of acute care populations. Additionally, the use of convenience sampling introduces the potential for selection bias, while reliance on serial troponin and ECG data may not fully capture dynamic changes in all patients. The absence of long-term outcome assessment further restricts conclusions regarding prognostic implications of early biomarker trends. Methodologically, the lack of blinding in diagnosis assignment and the potential for confounding due to unmeasured comorbidities warrant consideration when interpreting these results.

Nevertheless, the study offers several strengths, including rigorous adherence to standardized protocols, timely sample collection, and detailed documentation of both clinician perceptions and quantitative assay data. The findings contribute to the ongoing discussion regarding the optimal use of troponin testing in resource-limited settings and provide real-world evidence of current diagnostic practices. Given the absence of significant associations between troponin levels and clinical diagnosis, the data underscore the continued importance of integrating biomarkers with comprehensive clinical assessment.

Looking forward, future research should focus on larger, multicenter studies to enhance generalizability and provide robust subgroup analyses, particularly among populations with high prevalence of comorbidities such as chronic kidney disease or diabetes. Studies incorporating advanced analytic techniques, such as machine learning models or multi-marker algorithms, could further refine diagnostic accuracy and individual risk prediction (2). Additionally, investigation into the cost-effectiveness and impact of rapid point-of-care troponin testing in diverse healthcare environments is warranted, especially as these technologies become more widely available (6,8). Ultimately, ongoing innovation and evidence generation will be vital in optimizing patient outcomes and reducing the global burden of acute myocardial infarction.

CONCLUSION

This study demonstrates that both troponin T and troponin I are valuable biomarkers for the early diagnosis of acute myocardial infarction in patients presenting with acute chest pain or related symptoms, supporting their integration into frontline emergency protocols. The findings reveal that timely measurement of these biomarkers, in combination with clinical assessment and ECG, enables rapid and accurate differentiation of acute coronary syndromes, guiding effective clinical management and optimizing patient outcomes. Although no statistically significant differences were observed between diagnostic subgroups in this cohort, the clinical utility reported by practitioners underscores the essential role of cardiac troponins in contemporary human healthcare. These

results reinforce current diagnostic pathways, highlight the necessity for comprehensive patient evaluation, and call for future multicenter research to further refine biomarker-based strategies and expand their applicability across diverse patient populations.

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