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Evaluation of High-Sensitivity Cardiac Troponin I in Patients with Acute Myocardial Infarction

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Abstract

Background: Acute myocardial infarction (AMI) is a major global cause of morbidity and death, and prompt treatment depends on a quick diagnosis using high-sensitivity cardiac troponin I (hs-cTnI).

Objective: This study's objective was to evaluate hs-cTnI's diagnostic value in individuals with suspected AMI.

Methodology: The study was a prospective observational study carried out from March 2024 to February 2025 in the Department of Cardiology, Saidu Teaching Hospital, Swat, and the Department of Pharmacy, Abasyn University Islamabad Campus. All eligible patients with suspected AMI were recruited using consecutive sampling and the levels of hs-cTnI were measured at baseline and 3–6 hours; the final clinical diagnosis was the gold standard for diagnostic accuracy. Data were analysed with the SPSS software version 25.

Results

Of the 326 patients, 152 (46.63%) had no AMI and 174 (53.37%) had an AMI diagnosis. The most prevalent symptom (88.65%) was chest discomfort. The most common risk factors were diabetes mellitus (45.40%) and hypertension (53.99%). For hs-cTnI, the corresponding

sensitivity, specificity, positive predictive value, negative predictive value, and overall accuracy were 91.95%, 81.58%, 85.11%, 89.86%, and 87.12%. Excellent diagnostic performance was shown by the area under the ROC curve, which was 0.91 (95% CI: 0.88–0.94). AMI patients had considerably higher mean hs-cTnI levels (12.84 ± 4.56 ng/L) than non-AMI patients (2.36 ± 1.18 ng/L, $p < 0.001$).

Conclusion

Hs-cTnI is a strong discriminator and a reliable early biomarker for the diagnosis of AMI.

Introduction

Acute myocardial infarction (AMI) is a significant burden on the healthcare system, particularly in low- and middle-income countries, and remains one of the leading causes of morbidity and death globally [1]. Early and precise diagnostic techniques are necessary since the prevalence of cardiovascular risk factors, such as diabetes, hypertension, and smoking, is rising in Pakistan [2]. For prompt treatment and improved clinical outcomes, an early and precise diagnosis is essential. The clinical presentation, electrocardiogram (ECG) results, and biochemical indicators of myocardial damage have all been used to diagnose AMI. The most precise indicators of heart tissue damage among them are cardiac troponins [3, 4].

High-sensitivity cardiac troponin I (hs-cTnI) assays have transformed the approach to the diagnosis of AMI during the past several years [5]. These assays can detect very small amounts of troponin in the blood, which means that they can detect myocardial injury earlier than traditional assays [6]. This increased sensitivity allows an earlier diagnosis of AMI even when the symptoms are not typical or the ECG results are not diagnostic. Moreover, assays of hs-cTnI are used for better risk stratification and prognosis, which helps in clinical decision making and patient treatment [7].

The higher sensitivity of the hs-cTnI assays, however, also poses a number of difficulties, especially the ability to distinguish between acute and chronic elevation of troponin levels [8,9]. Increased levels of hs-cTnI can be seen in many non-ischemic states, including heart failure, renal dysfunction, and systemic infections, which may cause confusion in diagnosis. Thus, one should be mindful of the interpretation of hs-cTnI levels in relation to clinical context and serial levels to avoid misdiagnosis [10].

Furthermore, differences in the sensitivity/specificity of the assays, population differences and the presence of comorbidities require the assessment of hs-cTnI in specific clinical scenarios [11]. A diagnosis of AMI is made when the level of cardiac troponin rises and/or falls, with at least one level exceeding the 99th percentile upper reference level and other clinical or ECG changes support the diagnosis [12]. It is crucial to understand the diagnostic value and clinical implications of hs-cTnI in areas with low resources and high cardiovascular risk factor burden. This highlights the importance of research evaluating the use of hs-cTnI to improve the diagnosis and management of AMI.

Research Objective

To determine the diagnostic accuracy of use of hs-cTnI in patients presenting with suspected AMI, with AMI diagnosis based on standard diagnostic criteria.

Methodology

Study Design and Settings

The purpose of this prospective observational research was to evaluate the usage of hs-cTnI for individuals who may have AMI. The study was conducted at the Abasyn University Islamabad Campus' Department of Pharmacy and the Saidu Teaching Hospital's Department of Cardiology in Swat. The process of patient recruitment, clinical assessment and laboratory investigations were carried out at Saidu Teaching

Hospital, Swat and the organization of data and statistical analysis were done at Abasyn University Islamabad Campus.

Study Duration
The study period was from March 2024 to February 2025.

Study Population
Adult patients attending the emergency department at Saidu Teaching Hospital (STH) and the cardiology department with clinical signs and symptoms of AMI were included in this study.

Sample Size and Sampling Technique
The research covered 326 patients in total. The sample size was determined using the stated AMI prevalence rates and the estimated diagnostic accuracy based on a hs-cTnI at a 95% confidence level with a 5% margin of error. All eligible patients were enrolled using a non-probability successive sampling procedure until the target number of patients was attained.

Inclusion and Exclusion Criteria
The study comprised patients who were willing to sign informed consent and who were at least eighteen years old and had symptoms indicative of an AMI. To minimize confounding, patients with conditions that are known to markedly affect baseline Troponin levels, such as advanced chronic kidney disease, recent major surgery or trauma, active systemic infection, or chronic inflammatory disease were excluded.

Data Collection Procedure
Following informed consent, careful clinical histories were taken and comprehensive physical examinations conducted. At presentation, electrocardiograms were performed. Venous blood samples were drawn to measure the amount of hs-cTnI at baseline (0 hours) and repeated at 3–6 hours in line with recommended diagnostic guidelines. A structured data collection proforma was used to systematically record all the relevant demographic, clinical and laboratory information.

Laboratory Analysis
Standardized immunoassays methods were used in the clinical laboratory of Saidu Teaching Hospital, Swat for assaying the high sensitivity cardiac troponin I. Results were interpreted according to the upper reference limit determined at 99th percentile; values above the upper reference limit were defined as positive.

Statistical Analysis
Data input and analysis were done using the Statistical Package for Social Sciences (SPSS) version 25. For continuous variables, the data were shown as mean and standard deviation values; for categorical variables, frequencies and percentages were employed. The chi-square test was used to evaluate associations between categorical variables, and the area under the curve (AUC) was calculated using Receiver Operating Characteristic (ROC) curve analysis to evaluate the diagnostic performance of hs-cTnI. With the final diagnosis of AMI serving as the gold standard, diagnostic accuracy metrics including sensitivity, specificity, positive predictive value, and negative predictive value were calculated. A P value of less than 0.05 was considered statistically significant.

Ethical Considerations

The institutional review board at Saidu Teaching Hospital in Swat granted their approval to the study. Before beginning the trial, each participant provided written informed permission, and patient data confidentiality was maintained.

Results

Table 1 presents the demographic, clinical characteristics, and final diagnosis of the study population (n = 326). The majority of patients were in the age group 41–60 years (43.56%), followed by >60 years (38.65%), while only 17.79% were aged 18–40 years. Males constituted 60.74% of the study population, whereas females accounted for 39.26%. The most common presenting symptom was chest pain (88.65%), followed by shortness of breath (50.31%), sweating (42.33%), and nausea/vomiting (29.45%). Among risk factors, hypertension was most prevalent (53.99%), followed by diabetes mellitus (45.40%) and smoking (40.49%). Based on the final diagnosis, 174 patients (53.37%) were confirmed to have AMI, while 152 patients (46.63%) were categorized as non-AMI cases.

Table 1: Demographic, Clinical Characteristics and Final Diagnosis of Study Population (n = 326)

Category	Variable	Frequency (n)	Percentage (%)
Age (years)	18–40	58	17.79
	41–60	142	43.56
	>60	126	38.65
Gender	Male	198	60.74
	Female	128	39.26
Presenting Symptoms	Chest pain	289	88.65
	Shortness of breath	164	50.31
	Sweating	138	42.33
	Nausea/Vomiting	96	29.45
Risk Factors	Hypertension	176	53.99
	Diabetes Mellitus	148	45.40
	Smoking	132	40.49
Final Diagnosis	Confirmed AMI	174	53.37
	Non-AMI	152	46.63

Figure 1 demonstrates the distribution of hs-cTnI results compared with the final confirmed diagnosis of AMI. Among 188 hs-cTnI-positive patients, 160 cases were true positives (AMI present), while 28 were false positives (AMI absent). Among 138 hs-cTnI-negative patients, 124 were true negatives and 14 were false negatives. Overall, out of 326 patients, 174 were diagnosed with AMI and 152 were non-AMI cases, showing a strong correlation between hs-cTnI positivity and confirmed myocardial infarction.

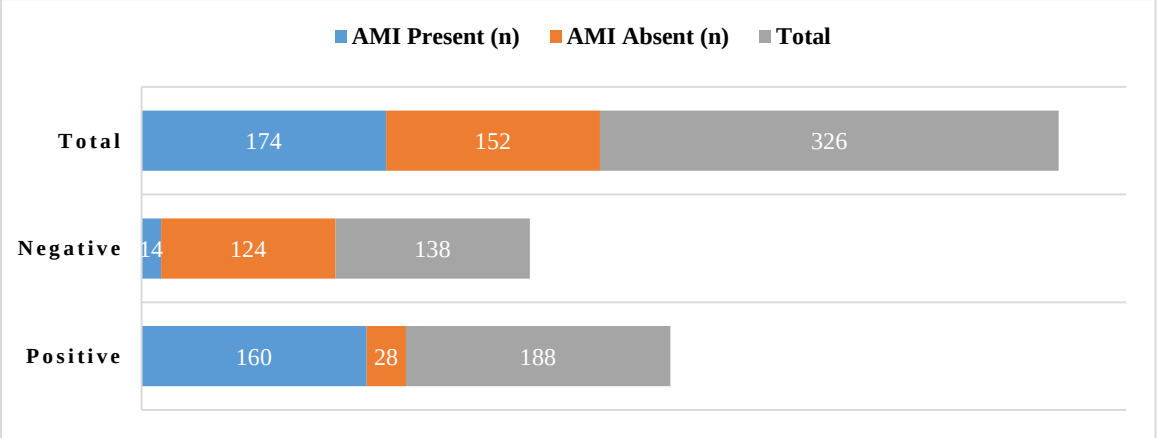

Figure 1: Hs-cTnI Results Compared with Final Diagnosis

The diagnostic accuracy of hs-cTnI for the identification of AMI is summarized in Figure 2. The specificity was 81.58%, suggesting good exclusion of non-AMI patients, while the sensitivity was 91.95%, demonstrating a strong capacity to properly detect AMI instances. Both the positive and negative predictive values were 85.11% and 89.86%, respectively. With an overall diagnosis accuracy of 87.12%, hs-cTnI performed well in the study population.

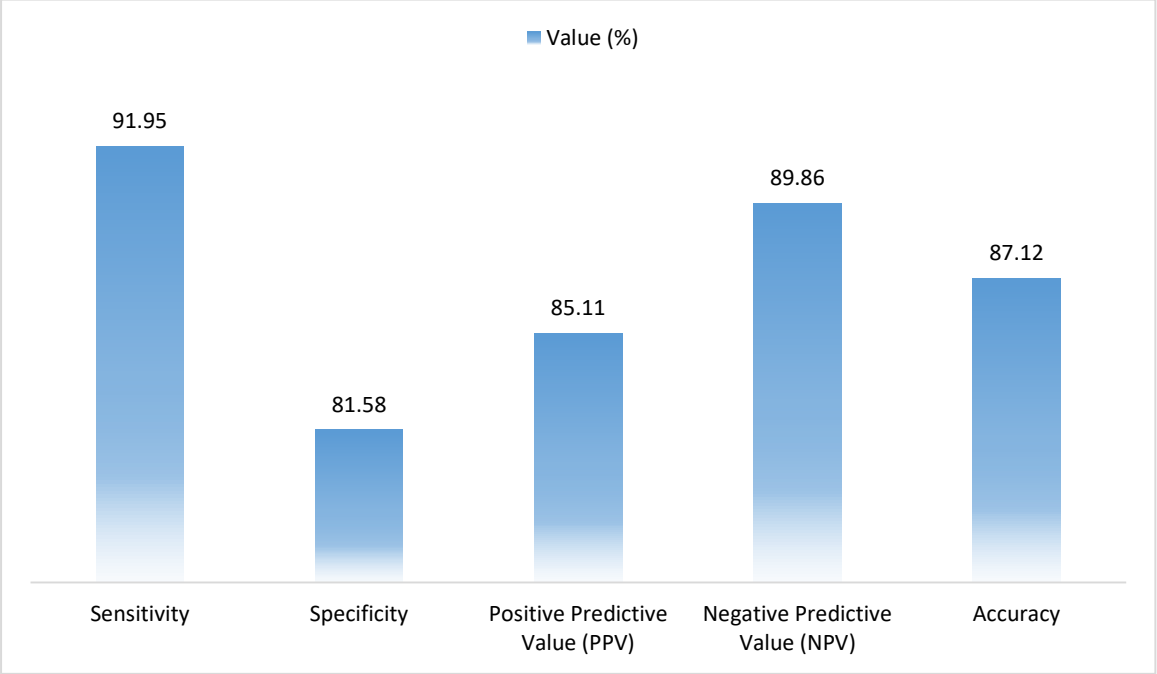

Figure 2: Diagnostic Accuracy of hs-cTnI for Detection of AMI

The Receiver Operating Characteristic (ROC) curve analysis of hs-cTnI for AMI diagnosis is shown in Table 2. Excellent diagnostic accuracy was indicated by the area under the curve (AUC) of 0.91. The results were statistically significant, with a p-value of less than 0.001, and the 95% confidence interval ranged from 0.88 to 0.94, demonstrating the great discriminative capacity of hs-cTnI in separating AMI from non-AMI patients.

Table 2: ROC Curve Analysis of hs-cTnI

Parameter	Value
Area Under Curve (AUC)	0.91
95% Confidence Interval	0.88 – 0.94
p-value	<0.001

Table 3 shows the association between hs-cTnI positivity and final diagnosis of AMI. A significantly higher proportion of AMI patients had positive hs-cTnI results (91.95%) compared to non-AMI patients (18.42%), while negative hs-cTnI results were more common in non-AMI cases (81.58%) than in AMI cases (8.05%). The association was statistically significant with a p-value of <0.001, indicating a strong relationship between hs-cTnI positivity and confirmed myocardial infarction.

Table 3: Association of hs-cTnI Positivity with Final Diagnosis

Variable	hs-cTnI Positive n (%)	hs-cTnI Negative n (%)	p-value
AMI	160 (91.95)	14 (8.05)	<0.001
Non-AMI	28 (18.42)	124 (81.58)	
Footnote: *Chi-square test applied*			

Table 4 compares the mean hs-cTnI levels between AMI and non-AMI groups. Patients with AMI had significantly higher mean hs-cTnI levels (12.84 ± 4.56 ng/L) compared to non-AMI patients (2.36 ± 1.18 ng/L). The difference between the two groups was statistically significant with a p-value of <0.001, as analyzed using an independent sample t-test, indicating markedly elevated troponin levels in AMI patients.

Table 4: Comparison of Mean hs-cTnI Levels Between AMI and Non-AMI Groups

Group	Mean hs-cTnI (ng/L)	Standard Deviation (SD)	p-value
AMI (n = 174)	12.84	4.56	<0.001
Non-AMI (n = 152)	2.36	1.18	
Footnote: *Independent sample t-test applied*			

Discussion

In the present study, in combination with clinical parameters, high sensitivity cardiac troponin I (hs-cTnI) had a high sensitivity, specificity, PPV, NPV, and accuracy values for the diagnosis of AMI of 91.95%, 81.58%, 85.11%, 89.86% and 87.12% respectively. These results show good performance as to both ruling in and ruling out AMI. A prospective multicenter study reported comparable diagnostic characteristics of hs-cTnI with a sensitivity of approximately 80–91% and a specificity of approximately 79–82%, reflecting a good balance in the diagnostic performance in an emergency setting [13].

The present study demonstrated a good discriminatory ability with an area under the ROC curve of 0.91 (95% CI: 0.88–0.94) between AMI and non-AMI cases. A similar performance was found in a large European multicenter cohort, with an AUC of 0.93 for hs-cTnI, and high levels of diagnostic performance across various cohorts and assay platforms [14]. Such close association lends greater external validity to hs-cTnI as a good early myocardial injury detection biomarker.

The findings demonstrate, a strong association was observed between the hs-cTnI positivity and the final diagnosis, with 91.95% of AMI patients being hs-cTnI positive, compared to only 18.42% of non-AMI patients. This is similar to what was observed in a systematic diagnostic evaluation in which sensitivity of the hs-cTn assays was between 0.71 and 0.91 and the specificity varied with patient population and comorbid conditions (e.g., renal dysfunction) [15]. The results emphasize the need to use hs-cTnI results in conjunction with clinical context to prevent false-positive misclassification.

The mean hs-cTnI level in AMI patients in this study was 12.84 ± 4.56 ng/L, significantly higher than 2.36 ± 1.18 ng/L in non-AMI patients ($p < 0.001$). This is a biochemical gradient that would indicate significant myocardial damage in those who

were confirmed with infarction. This quantitative difference has also been reported in earlier studies, with significantly higher levels of hs-cTnI in AMI patients than in non-ischemic chest pain patients, further supporting the use of hs-cTnI as a marker of myocardial necrosis and burden of injury [16].

The ROC analysis in this study also showed an excellent overall diagnostic discrimination with an AUC of 0.91 (95% CI: 0.88–0.94, $p < 0.001$). Similar studies have reported AUC values between 0.87 and 0.94 for hs-cTnI, which vary with assay type and when the samples were taken, especially in early presenters with chest pain [14]. This implies that serial measurement strategies, such as the one applied in the present study (time 0 and 3–6 hours), improve the accuracy of the diagnosis in real clinical settings.

The overall results of the diagnostic indices in this study are comparable with those reported internationally: hs-cTnI is highly sensitive and has moderate to high specificity, and can be used for rapid shortlist triage of suspected AMI patients. These results in large multicenter populations have been consistent and confirm that hs-cTnI is a valuable biomarker with excellent rule-out properties and clinically relevant predictive value in ED settings [13,17].

Strength and Limitations

The strengths of this study are the prospective observational design, the relatively large number of 326 patients and the use of serial hs-cTnI measurements at 0 and 3–6 hours, which enhances the reliability of the measurement. Moreover, the use of a clear gold standard (UDMIM) and detailed statistical analysis with the evaluation of ROC curve and the diagnostic accuracy parameters further support the results. There are some caveats, however. The study was conducted at one tertiary care centre and the results might not be generalizable to other populations. In addition, non-probability consecutive sampling may result in selection bias. Confounding was minimised by excluding patients with chronic renal failure and systemic infections, but the study findings may not be applicable to a more diverse population in which these co-morbidities are more common.

Conclusion

Finally, the present study demonstrated that the diagnostic sensitivity, specificity and the ROC curve analysis of a hs-cTnI assay are excellent in the early diagnosis of AMI. The marked difference in hs-cTnI levels of AMI and non-AMI patients further supports the clinical use of this marker to differentiate AMI from other chest pains. The results highlight the potential of hs-cTnI as a useful and reliable assay that can help identify and manage patients with suspected AMI, in addition to clinical judgement and ECG interpretation.

References

Nascimento BR, Brant LC, Marino BC, Passaglia LG, Ribeiro AL. Implementing myocardial infarction systems of care in low/middle-income countries. Heart. 2019 Jan 1;105(1):20-6. https://doi.org/10.1136/heartjnl-2018-313398

Nazir A, Nazir A, Afzaal U, Aman S, Sadiq SU, Akah OZ, Jamal MS, Hassan SZ. Advancements in biomarkers for early detection and risk stratification of cardiovascular diseases—a literature review. Health Science Reports. 2025 May;8(5):e70878. https://doi.org/10.1002/hsr2.70878

Fathil MF, Arshad MM, Gopinath SC, Hashim U, Adzhri R, Ayub RM, Ruslinda AR, Nuzaihan M, Azman AH, Zaki M, Tang TH. Diagnostics on acute myocardial infarction: Cardiac troponin biomarkers. Biosensors and Bioelectronics. 2015 Aug 15;70:209-20. https://doi.org/10.1016/j.bios.2015.03.037

Aydin S, Ugur K, Aydin S, Sahin İ, Yardim M. Biomarkers in acute myocardial infarction: current perspectives. Vascular health and risk management. 2019 Jan 17:1-0. https://doi.org/10.2147/VHRM.S166157

Clerico A, Zaninotto M, Aimo A, Padoan A, Passino C, Fortunato A, Galli C, Plebani M. Advancements and challenges in high-sensitivity cardiac troponin assays: Diagnostic, pathophysiological, and clinical perspectives: On behalf of the Italian Study Group on Cardiac Biomarkers. Clinical Chemistry and Laboratory Medicine (CCLM). 2025 Jun 1;63(7):1260-78. https://www.degruyterbrill.com/document/doi/10.1515/cclm-2024-1090/html

Osafo R, Olatunji SH, Nduka CL. Acute Myocardial Infarction (AMI) Diagnosis; Impact of Technology in Developing Highly Sensitive Biomarkers and Assays. Cardiol. Angiol. Int. J. 2022 Jun 13;11(4):124-3. DOI: 10.9734/CA/2022/v11i430215

Lanza O. Linking platelet reactivity, cardiac autonomic dysfunction and inflammation in patients with acute coronary syndromes: a complex puzzle to be solved with potential therapeutic implications. https://hdl.handle.net/20.500.14242/194963

Kozinski M, Krintus M, Kubica J, Sypniewska G. High-sensitivity cardiac troponin assays: from improved analytical performance to enhanced risk stratification. Critical Reviews in Clinical Laboratory Sciences. 2017 Apr 3;54(3):143-72. https://doi.org/10.1080/10408363.2017.1285268

Lazar DR, Lazar FL, Homorodean C, Cainap C, Focsan M, Cainap S, Olinic DM. High-sensitivity troponin: a review on characteristics, assessment, and clinical implications. Disease Markers. 2022;2022(1):9713326. https://doi.org/10.1155/2022/9713326

Savic L, Stankovic S, Antonijevic N, Matic D, Lasica R, Krljanac G, Asanin M. Elevated Cardiac Troponin in Non-Cardiac Conditions Unrelated to Acute Myocardial Infarction. International Journal of Molecular Sciences. 2025 Dec 1;26(23):11655. https://doi.org/10.3390/ijms262311655

Clerico A, Zaninotto M, Plebani M. Methodological evaluation and clinical interpretation of hs-cTnI and hs-cTnT variations: a reappraisal. Clinical Chemistry and Laboratory Medicine (CCLM). 2026 Feb 24;64(3):566-9. https://doi.org/10.1515/cclm-2025-1318

Sandoval Y, Apple FS, Saenger AK, Collinson PO, Wu AH, Jaffe AS. 99th percentile upper-reference limit of cardiac troponin and the diagnosis of acute myocardial infarction. Clinical chemistry. 2020 Sep 1;66(9):1167-80. https://doi.org/10.1093/clinchem/hvaa158

Nseir M, Mokhtari A, Stanisic M, Ekström U, Labaf A. Validation and correlation of high-sensitive troponin I and troponin T in the emergency department. BMC Cardiovascular Disorders. 2024 Oct 12;24(1):551. https://doi.org/10.1186/s12872-024-04230-1

Rubini Gimenez M, Twerenbold R, Reichlin T, Moehring B, Wildi K, Meller B, Haaf P, Zellweger C, Osswald S, Mueller C. Direct comparison of high-sensitivity cardiac troponin I versus T for the early diagnosis of acute myocardial infarction. European Heart Journal. 2013 Aug 1;34(suppl_1):20. https://doi.org/10.1093/eurheartj/eht307.20

Parikh RH, Seliger SL, deFilippi CR. Use and interpretation of high sensitivity cardiac troponins in patients with chronic kidney disease with and without acute myocardial infarction. Clinical biochemistry. 2015 Mar 1;48(4-5):247-53. https://doi.org/10.1016/j.clinbiochem.2015.01.004

Ren D, Huang T, Liu X, Xu G. High-sensitive cardiac troponin for the diagnosis of acute myocardial infarction in different chronic kidney disease stages. BMC cardiovascular disorders. 2021 Feb 17;21(1):100. https://doi.org/10.1186/s12872-020-01746-0

Aspromonte N, Zaninotto M, Aimo A, Fumarulo I, Plebani M, Clerico A. Measurement of cardiac-specific biomarkers in the emergency department: new insight in risk evaluation. International Journal of Molecular Sciences. 2023 Nov 6;24(21):15998. https://doi.org/10.3390/ijms242115998