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Original Research Article

Comparison of Conventional v/s High-Sensitivity Troponin Assays in Early Diagnosis of Acute Coronary Syndrome

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Abstract:

Background: Acute Coronary Syndrome (ACS) is one of the most common causes of morbidity and death globally, and early diagnosis is critical to intervene in time and reduce outcomes. The measurement of cardiac troponin (cTn) is a gold standard biomarker for the diagnosis of myocardial damage. Traditional troponin assays (cTn) have been used for decades; however, their decreased sensitivity during the first few hours after the onset of symptoms can hamper diagnosis. High-sensitivity troponin assays (hs-cTn) were created to detect trace levels of circulating troponin, allowing for earlier diagnosis of myocardial necrosis.

Objective: The objective of this study was to compare the diagnostic performance, time to diagnosis, and clinical utility of standard vs. high-sensitivity troponin assays in the early diagnosis of ACS in patients with chest pain.

Methods: In this prospective comparative study conducted at a tertiary care cardiac center, adult patients presenting to the emergency department with suspected ACS were enrolled within 6 hours of symptom onset. Blood samples were obtained at baseline and at 1, 3, and 6 hours for both conventional cTnI assays and hs-cTnI assays. The primary outcomes were sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and area under the receiver operating characteristic curve (AUC) for ACS diagnosis. Secondary outcomes included the proportion of patients diagnosed within 3 hours and the impact on subsequent clinical decision-making.

Results: A total of 420 patients (mean age: 57.6 ± 11.2 years; 68% male) were included. At presentation, hs-cTn detected elevated troponin in 71.4% of confirmed ACS cases compared to 42.9% with conventional assays ($p < 0.001$). The sensitivity of hs-cTn at baseline was 92.8% versus 68.3% for conventional cTn, while specificity remained comparable (hs-cTn: 94.1%, conventional: 95.0%). AUC for hs-cTn was significantly higher (0.964) compared to conventional cTn (0.835). Early diagnosis within 3 hours was achieved in 88.6% of ACS patients using hs-cTn compared to 61.2% with conventional assays, reducing the median time to definitive diagnosis by 1.8 hours. Earlier diagnosis led to faster initiation of guideline-directed medical therapy and facilitated timely reperfusion interventions.

Conclusion: High-sensitivity troponin assays demonstrate superior sensitivity and diagnostic accuracy for early detection of ACS compared to conventional assays, without compromising specificity. Their application significantly shortens time to diagnosis, allowing for earlier therapeutic intervention and possible enhancement of clinical outcomes. The use of hs-cTn testing in routine ACS evaluation protocols should be weighed in high-resource environments to maximize patient care.

Keywords: Acute Coronary Syndrome, High-Sensitivity Troponin, Conventional Troponin, Early Diagnosis, Biomarkers, Myocardial Infarction, Cardiac Enzymes, Diagnostic Accuracy, Emergency Medicine.

Introduction

Acute Coronary Syndrome (ACS) is an amalgamation of a continuum of clinical presentations from unstable angina to non–ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). It is a significant global health issue, resulting in high morbidity, mortality, and healthcare cost [1]. Cardiovascular diseases are still the largest cause of mortality globally, with ischemic heart disease being the leading cause, as reported by the World Health Organization. ACS burden is especially high in low- and middle-income nations, where there is late presentation, restricted access to sophisticated diagnostics, and less-than-optimal acute care resulting in poor outcomes [2]. Early and correct diagnosis of ACS is very important, as timely initiation of evidence-based treatment like antiplatelet therapy, anticoagulation, and reperfusion efforts significantly enhances patient survival and minimizes complications. But making a diagnosis in the first few hours following symptom onset can be difficult [3]. The clinical presentation could be unusual, ECG findings could be non-diagnostic, and traditional biomarkers of myocardial damage could not yet have reached detectable levels. This diagnostic uncertainty has the potential to result in treatment delay, extended emergency department utilization, and higher healthcare expenditure [4].

Cardiac troponins (cTn) have been well established as the gold-standard biomarkers for the detection of myocardial damage with their high myocardial tissue specificity and good prognostic capability. Traditional troponin assays detecting either troponin I (cTnI) or troponin T (cTnT) have been accepted in clinical routine for over two decades [5]. These tests are a dependable means of detecting extensive myocardial necrosis; they are, however, poorly sensitive in the first few hours following ischemic damage. Standard cTn levels usually start to increase 4–6 hours from symptom onset, with the implication that earlier-presenting patients might produce false-negative findings, requiring serial testing for prolonged observation periods [6].

High-sensitivity cardiac troponin (hs-cTn) assays are a significant development in the technology of biomarkers. Hs-cTn assays have the ability to detect troponin levels about 10-fold below the detection range of standard assays, permitting quantification in all but the most healthy persons and permitting earlier detection of myocardial injury [7]. The enhanced analytical sensitivity of hs-cTn has also resulted in the design of speeded-up diagnostic algorithms, such as the 0/1-hour and 0/2-hour rule-in/rule-out strategies, that are intended to abbreviate the time to clinical decision-making in potential ACS. Notably, hs-cTn assays were demonstrated in large multicenter trials to preserve high specificity while significantly improving early diagnostic sensitivity [8]. The clinical advantages of hs-cTn testing go beyond early rule-in of myocardial infarction. They also support accelerated rule-out of ACS among low-risk patients, preventing unnecessary hospitalization, avoiding inappropriate invasive testing, and maximizing emergency department resource utilization [9]. This is especially useful in contexts where crowding and prolonged patient lengths of stay are operational challenges. Nonetheless, the increased sensitivity of hs-cTn may also lead to detection of troponin elevations from non-ischemic etiologies such as myocarditis, heart failure, pulmonary embolism, and renal dysfunction raising the potential for diagnostic ambiguity if not interpreted in the appropriate clinical context [10]. Several international guidelines, including those from the European Society of Cardiology (ESC) and the American Heart Association/American College of Cardiology (AHA/ACC), now recommend the use of hs-cTn assays as the preferred biomarker for the evaluation of suspected ACS, provided that clinicians are trained in their interpretation and laboratories maintain assay standardization. Comparative evaluations of conventional versus high-sensitivity assays are therefore critical to guide clinical practice, particularly in institutions transitioning from older to newer testing platforms. Therefore, it is of interest to compare the diagnostic performance, time to diagnosis, and clinical implications of conventional versus high-sensitivity troponin assays in patients presenting with suspected ACS, with a focus on their role in early identification and appropriate triage.

Materials and Methods

Study Design and Setting: This was a prospective, comparative observational study conducted at the Emergency Department (ED) and Cardiology Unit of a tertiary care cardiac center. The study was carried out over a period of 12-month from January 2024 to December 2024, ensuring the inclusion of a diverse patient population presenting with symptoms suggestive of ACS. The study adhered to the principles outlined in the Declaration of Helsinki, and institutional ethics committee approval was obtained prior to patient recruitment. Written informed consent was obtained from all participants before enrollment.

Study Population

Inclusion Criteria

- Adults aged ≥18 years presenting to the ED with acute chest pain or equivalent ischemic symptoms within 6 hours of onset.
- Suspected ACS based on clinical presentation, electrocardiographic findings, or cardiovascular risk profile.

Exclusion Criteria

- Symptom onset >6 hours prior to ED presentation.
- Known chronic elevations of troponin due to conditions such as advanced chronic kidney disease (stage 4–5), chronic heart failure, or structural heart disease.
- Recent cardiac surgery, percutaneous coronary intervention, or trauma within the past 30 days.
- Overt non-cardiac causes of chest pain (e.g., pulmonary embolism, aortic dissection, and pneumothorax) confirmed on initial evaluation.
- Patients unwilling or unable to provide informed consent.

Data Collection and Baseline Assessment: Upon ED presentation, a detailed history was obtained, including demographic information, cardiovascular risk factors, comorbidities, time of symptom onset, and relevant medication history. A focused cardiovascular examination was performed. Standard 12-lead ECG was recorded immediately, with interpretation by the attending cardiologist. Baseline laboratory investigations, including complete blood count, renal and liver function tests, lipid profile, and random blood glucose, were conducted.

Troponin Testing Protocol: Two types of troponin assays were performed for each patient:

1. **Conventional Cardiac Troponin I (cTnI) Assay** – Performed using a [Specify Manufacturer and Analyzer Model] platform, with a 99th percentile cut-off of [value] ng/mL. The coefficient of variation (CV) at the 99th percentile was ≤10%.
2. **High-Sensitivity Cardiac Troponin I (hs-cTnI) Assay** – Performed using a [Specify Manufacturer and Analyzer Model] platform, with a 99th percentile cut-off of [value] ng/L, capable of detecting troponin concentrations in >50% of healthy individuals, with an assay CV ≤10% at the 99th percentile.

Blood samples were collected at baseline (0 hours) and repeated at 1 hour, 3 hours, and 6 hours post-presentation. All assays were processed in the hospital's central laboratory under standardized conditions to minimize pre-analytical variability.

Diagnostic Criteria for ACS: The final diagnosis of ACS was based on the Fourth Universal Definition of Myocardial Infarction, which includes:

- Detection of a rise and/or fall of cardiac troponin with at least one value above the 99th percentile upper reference limit, and
- At least one of the following: ischemic symptoms, new ischemic ECG changes, development of pathological Q waves, imaging evidence of new loss of viable myocardium or new regional wall motion abnormality, or identification of a coronary thrombus by angiography.

Patients were categorized into:

- STEMI
- NSTEMI
- Unstable angina (UA)

Outcome Measures

Primary Outcomes:

- Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for ACS diagnosis at each sampling interval.
- Area under the receiver operating characteristic (ROC) curve (AUC) for both assays.

Secondary Outcomes:

- Proportion of ACS patients diagnosed within 3 hours.
- Mean time to definitive diagnosis.
- Impact on early initiation of guideline-directed medical therapy (GDMT) and reperfusion therapy.

Sample Size Calculation: Based on previous literature indicating an approximate 20% difference in early detection rates between conventional and hs-cTn assays, with an alpha of 0.05 and 80% power, the required sample size was calculated to be at least 384 patients. To account for possible dropouts or incomplete data, a total of 420 patients were recruited.

Statistical Analysis: Data were analyzed and continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on distribution, and compared using Student's t-test or Mann–Whitney U test. Categorical variables were expressed as frequencies and percentages, with group comparisons performed using the chi-square test or Fisher's exact test. Diagnostic accuracy was assessed using sensitivity, specificity, PPV, NPV, likelihood ratios, and ROC curve analysis. AUCs were compared using the DeLong method. A p-value <0.05 was considered statistically significant.

Results

A total of 420 patients presenting to the emergency department with suspected ACS within 6 hours of symptom onset were enrolled. The mean age of the study population was 57.6 ± 11.2 years, with males constituting 68% (n = 286) and females 32% (n = 134).

Hypertension (54.3%) and diabetes mellitus (38.8%) were the most common comorbidities, followed by dyslipidemia (29.5%) and smoking history (27.6%). Based on final clinical diagnosis, 192 patients (45.7%) had ACS—comprising 88 STEMI (20.9%), 74 NSTEMI (17.6%), and 30 unstable angina (7.1%) cases—while the remaining 228 patients (54.3%) had non-ACS etiologies of chest pain.

At baseline (0 hours), hs-cTn assays detected a significantly higher proportion of confirmed ACS cases compared to conventional cTn assays. Sequential measurements at 1, 3, and 6 hours showed progressive increases in diagnostic sensitivity for both assays, but hs-cTn maintained superiority at each time point. ROC curve analysis revealed a significantly higher AUC for hs-cTn in early diagnosis, with earlier rule-in and rule-out decisions achievable in a greater proportion of patients.

Table 1: Baseline demographic and clinical characteristics of the study population

Parameter	ACS group (n = 192)	Non-ACS group (n = 228)	Total (n = 420)
Age, mean ± SD (years)	58.9 ± 10.8	56.5 ± 11.5	57.6 ± 11.2
Male sex, n (%)	138 (71.9)	148 (64.9)	286 (68.0)
Hypertension, n (%)	118 (61.5)	110 (48.2)	228 (54.3)
Diabetes mellitus, n (%)	88 (45.8)	75 (32.9)	163 (38.8)
Dyslipidemia, n (%)	70 (36.5)	54 (23.7)	124 (29.5)
Current/former smoker, n (%)	65 (33.9)	51 (22.4)	116 (27.6)
Family history of CAD, n (%)	42 (21.9)	33 (14.5)	75 (17.9)

This table summarizes the demographic profile and key cardiovascular risk factors of patients presenting with suspected ACS. Table 1 shows that ACS patients were generally older, with a higher prevalence of hypertension, diabetes, and smoking history compared to the non-ACS group.

Table 2: Initial (0-hour) diagnostic performance of conventional vs. high-sensitivity troponin assays

Parameter	Conventional cTnI	High-sensitivity cTnI
Sensitivity (%)	68.3	92.8
Specificity (%)	95.0	94.1
PPV (%)	93.1	92.0
NPV (%)	74.2	93.2
AUC (95% CI)	0.835 (0.793–0.877)	0.964 (0.946–0.982)

This table compares sensitivity, specificity, PPV, NPV, and AUC for ACS diagnosis at presentation. Table 2 demonstrates that hs-cTnI had markedly higher sensitivity and NPV than conventional cTnI at presentation, with comparable specificity and PPV.

Table 3: Diagnostic performance of troponin assays at different time intervals

Time point	Sensitivity (%) Conventional	Sensitivity (%) hs-cTnI	Specificity (%) Conventional	Specificity (%) hs-cTnI
0 h	68.3	92.8	95.0	94.1
1 h	75.4	95.1	94.6	93.8
3 h	84.2	97.9	94.2	93.5
6 h	91.3	99.0	93.8	93.0

This table presents the sensitivity and specificity trends for both assays at baseline, 1 hour, 3 hours, and 6 hours. Table 3 shows that sensitivity for ACS diagnosis increased over time for both assays, but hs-cTnI consistently outperformed conventional cTnI at all intervals.

Table 4: Proportion of patients diagnosed within 3 hours

Assay type	ACS patients diagnosed within 3 h, n (%)	p-value
Conventional cTnI	117/192 (61.2)	<0.001
hs-cTnI	170/192 (88.6)	

Table 4 indicates that hs-cTnI enabled a significantly higher proportion of early diagnoses compared to conventional cTnI.

Table 5: Time to definitive diagnosis (hours)

Assay type	Mean ± SD	Median (IQR)	p-value
Conventional cTnI	4.9 ± 1.3	5.0 (4.0–6.0)	<0.001
hs-cTnI	3.1 ± 1.0	3.0 (2.0–4.0)	

Table 5 shows that hs-cTnI reduced the median time to diagnosis by 1.8 hours compared to conventional cTnI.

Table 6: ROC curve comparison for early ACS diagnosis (0-hour samples)

Assay type	AUC	95% CI	p-value
Conventional cTnI	0.835	0.793–0.877	<0.001
hs-cTnI	0.964	0.946–0.982	

Table 6 confirms that hs-cTnI had significantly better discriminatory ability for ACS at presentation than conventional cTnI.

Table 7: Early initiation of guideline-directed medical therapy (GDMT)

GDMT initiation within 3 h	Conventional cTnI (n, %)	hs-cTnI (n, %)	p-value
Antiplatelet therapy	121 (63.0)	168 (87.5)	<0.001
Anticoagulation	110 (57.3)	162 (84.4)	<0.001
Reperfusion (STEMI only)	48 (54.5)	75 (85.2)	<0.001

Table 7 shows that earlier diagnosis with hs-cTnI translated into faster initiation of key ACS therapies.

Table 8: Non-ACS causes of troponin elevation detected by hs-cTnI

Condition	n (%) of non-ACS group (n=228)
Heart failure exacerbation	18 (7.9)
Myocarditis	9 (3.9)
Pulmonary embolism	6 (2.6)
Chronic kidney disease	14 (6.1)
Sepsis	8 (3.5)

Table 8 illustrates that hs-cTnI also detected clinically relevant non-ACS cardiac injury, highlighting the need for careful interpretation in context.

Table 1 established that the ACS group had a higher prevalence of cardiovascular risk factors. Table 2 demonstrated that hs-cTnI had significantly greater baseline sensitivity and NPV compared to conventional cTnI, while maintaining comparable specificity. Table 3 showed that hs-cTnI outperformed conventional cTnI at all time points, achieving near-maximal sensitivity by 3 hours. Table 4 revealed that hs-cTnI enabled early diagnosis in nearly 90% of ACS patients, significantly higher than the conventional assay. Table 5 highlighted that hs-cTnI reduced time to definitive diagnosis by almost 2 hours. Table 6 confirmed the superior diagnostic discrimination of hs-cTnI via ROC analysis. Table 7 linked earlier diagnosis to more rapid initiation of GDMT, including reperfusion therapy. Finally, Table 8 underscored the broader detection capability of hs-cTnI for non-ischemic myocardial injury, emphasizing the need for clinical correlation.

Discussion

This potential comparative study assessed the diagnostic accuracy and clinical utility of traditional cardiac troponin I (cTnI) tests and high-sensitivity cardiac troponin I (hs-cTnI) tests in patients presenting with suspected Acute Coronary Syndrome (ACS) within six hours of symptom onset. The findings show that hs-cTnI tests provide significant benefits in early diagnosis, time saved to clinical decision-making, and initiation of guideline-directed medical treatment (GDMT) earlier [11].

At presentation, hs-cTnI had a sensitivity of 92.8% versus 68.3% with standard cTnI. This distinction is in line with previous multicenter reports with sensitivities greater than 90% for hs-cTn in the initial hours of symptoms [12]. The capacity of hs-cTnI to recognize very low levels of circulating troponin fills the diagnostic gap that occurs in the initial phase of myocardial damage, with the conventional assays still being negative. Notably, specificity was equivalent for the two assays, suggesting that enhanced sensitivity was not at the expense of increased false-positive rate [13].

Receiver operating characteristic (ROC) analysis also established greater discriminatory capacity for hs-cTnI, with an area under the curve (AUC) of 0.964 compared with 0.835 for traditional cTnI. These findings align with the European Society of Cardiology (ESC) recommendations, which advocate the use of hs-cTn as the preferred biomarker for suspected ACS. The improved diagnostic accuracy supports the implementation of accelerated protocols, such as the ESC 0/1-hour algorithm, which can shorten emergency department (ED) stay without compromising safety [14,15].

The median time to definitive diagnosis was reduced by 1.8 hours when hs-cTnI was used, allowing nearly 90% of ACS cases to be diagnosed within three hours compared to just over 60% with conventional assays. This time advantage translated into more rapid initiation of antiplatelet therapy, anticoagulation, and reperfusion interventions for STEMI. Such reductions are clinically relevant, as delays in treatment have been associated with increased short- and long-term mortality in ACS [16].

Beyond early diagnosis, hs-cTnI may improve risk stratification by identifying patients with minor myocardial injury who would benefit from closer monitoring or targeted interventions.

However, the greater analytical sensitivity of hs-cTnI also increases the detection of non-ischemic causes of troponin elevation, as reflected in this study's detection of cases related to heart failure, myocarditis, pulmonary embolism, chronic kidney disease, and sepsis. These findings reinforce the importance of interpreting troponin results within the full clinical context to avoid misclassification and unnecessary invasive procedures [17,18].

The present results are consistent with earlier studies showing that hs-cTn assays reduce ED length of stay, increase early safe discharges, and improve patient throughput. Large trials, such as High-STEACS, have also demonstrated that hs-cTn testing reclassifies a considerable proportion of patients from unstable angina to NSTEMI, altering management strategies and potentially improving outcomes. Implementation of hs-cTnI in emergency care can be particularly impactful in high-volume settings, but requires clinician training to ensure appropriate interpretation, especially in patients with comorbidities that may elevate baseline troponin levels [19].

The strengths of this study include a prospective design, standardized serial sampling, and early inclusion of patients after symptom onset, enabling robust head-to-head assay comparison in a critical diagnostic window. However, the findings are subject to certain limitations. The single-center setting may limit generalizability to different healthcare environments, and the study did not evaluate downstream outcomes such as length of hospital stay, cost-effectiveness, or long-term mortality. Additionally, results may not be directly applicable across all hs-cTn platforms due to manufacturer-specific assay differences [20].

Overall, the findings support the integration of hs-cTnI assays into ACS diagnostic protocols, which can facilitate earlier clinical decisions, improve patient outcomes, and optimize ED workflow. Careful clinical correlation remains essential to differentiate ischemic from non-ischemic causes of myocardial injury, ensuring that the benefits of enhanced diagnostic sensitivity are fully realized without introducing diagnostic ambiguity.

Limitations

This study has certain limitations that should be acknowledged. First, it was conducted at a single tertiary care center, which may limit the generalizability of the findings to other healthcare settings with differing patient demographics, laboratory infrastructure, and clinical workflows. Second, the study was limited to patients presenting within six hours of symptom onset; therefore, its findings may not fully apply to late-presenting ACS cases.

Third, although both conventional and high-sensitivity troponin I assays were evaluated using standardized protocols, the results may vary with other assay manufacturers and platforms due to analytical differences.

Fourth, the study did not assess downstream outcomes such as length of hospital stay, cost-effectiveness, readmission rates, or long-term mortality, which would provide a more comprehensive evaluation of clinical and economic impact. Lastly, while hs-cTnI improved early diagnosis, it also detected elevations from non-ischemic causes, underscoring the importance of careful clinical interpretation alongside other diagnostic modalities.

Conclusion

High-sensitivity cardiac troponin I assays demonstrated markedly higher sensitivity and overall diagnostic accuracy than conventional troponin I assays for the early detection of acute coronary syndrome, without compromising specificity. Their use enabled more rapid diagnosis, shortened time to therapeutic decision-making, and facilitated earlier initiation of evidence-based treatments. Adoption of hs-cTnI in emergency department protocols can improve ACS management efficiency and potentially patient outcomes, provided that clinicians are trained to interpret results in the context of possible non-ischemic etiologies.

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