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Predictive Value of High-Sensitivity Troponin I for Left Ventricular Ejection Fraction in Patients with Non-ST-Elevation Myocardial Infarction

ABSTRACT

Background: According to the World Health Organization's 2021 statistics, cardiovascular diseases (CVDs), particularly coronary artery disease (CAD), remain among the leading causes of global morbidity and mortality, affecting both high-income and low-income countries like Vietnam. **Objective:** Acute myocardial infarction (AMI) remains a major cause of mortality and cardiovascular complications, with a poor prognosis in patients with left ventricular systolic dysfunction (LVSD). High-sensitivity cardiac troponin I (hs-cTnI) is a specific biomarker of myocardial injury linked to infarct size and LVSD. However, its role in predicting left ventricular ejection fraction (LVEF) in non-ST-elevation myocardial infarction (NSTEMI) is underexplored. This study investigates the correlation between hs-cTnI and LVSD in NSTEMI patients. **Methods:** A descriptive, cross-sectional study was conducted on 117 patients with first-time NSTEMI treated at Cho Ray Hospital from February 2024 to April 2024. Admission hs-cTnI levels were measured and correlated with LVEF, assessed via echocardiography. The predictive value and optimal cut-off points of hs-cTnI for LVSD (LVEF < 50% and ≤ 40%) were determined using receiver operating characteristic (ROC) curve analysis. **Results:** Hs-cTnI levels showed a significant inverse correlation with LVEF ($r = -0.569$, $p < 0.001$). Patients with moderate-to-severe LVSD (LVEF ≤ 40%) had the highest median hs-cTnI levels (25,000 pg/mL, $p < 0.001$). The area under the ROC curve (AUC) for predicting LVEF < 50% was 0.78, with a cut-off of 12,344 pg/mL (sensitivity 68.5%, specificity 82.5%). For LVEF ≤ 40%, the AUC was 0.82, with a cut-off of 20,979 pg/mL (sensitivity 73.3%, specificity 88.5%, accuracy 84.6%). These findings underscore hs-cTnI's utility in identifying LVSD. **Conclusion:** Hs-cTnI is inversely correlated with LVEF and serves as a reliable biomarker for predicting LVSD in NSTEMI patients, facilitating risk stratification and early management decisions.

Keywords: Non-ST-elevation myocardial infarction, left ventricular systolic dysfunction, high-sensitivity troponin I, coronary angiography.

1. BACKGROUND

According to the World Health Organization's 2021 statistics, cardiovascular diseases (CVDs), particularly coronary artery disease (CAD), remain among the leading causes of global morbidity and mortality, affecting both high-income and low-income countries like Vietnam (1). This burden continues to escalate despite significant advancements in diagnosis and treatment. Between 1991 and 2021, the global prevalence of ischemic heart disease increased by 81%, with Southeast Asia experiencing a rise of up to 221% (2). Acute myocardial infarction (AMI) is a primary cause of cardiovascular-related deaths, with non-ST-segment elevation myocardial infarction (NSTEMI) accounting for 50–70% of all AMI cases (3). NSTEMI is a severe clinical condition with a high risk of progressing to heart failure and death if not promptly diagnosed and treated.

Left ventricular ejection fraction (LVEF) is a crucial factor in assessing

cardiac function and prognosis in AMI. Patients with left ventricular systolic dysfunction (LVSD) have a 2- to 3-fold higher risk of death and hospitalization due to heart failure compared to those without LVSD. According to the 2025 recommendations of the American Heart Association and the American College of Cardiology, left ventricular function should be routinely evaluated in patients with acute coronary syndrome before discharge, as LVEF plays a vital role in guiding treatment and risk stratification. Diagnosing left ventricular dysfunction is significant because it can lead to decisions to initiate or optimize guideline-directed therapies for patients with heart failure or reduced left ventricular function (4–7). Additionally, assessing LVEF helps identify patients with reduced ejection fraction, allowing consideration of implantable cardioverter-defibrillator implantation for primary prevention of sudden cardiac death in the future. However, in clinical practice, evaluating LVEF via transthoracic echocardiography using the biplane Simpson method often faces limitations due to equipment and personnel requirements. This leads to many patients not receiving timely echocardiography during hospitalization, despite all these patients having indications for echocardiographic assessment of left ventricular systolic function with a class I recommendation and level of evidence A (8) Studies have reported that the proportion of patients with ST-segment elevation myocardial infarction (STEMI) and NSTEMI not undergoing LVEF assessment during hospitalization ranges from 9% to 50% (8). Even in developed countries like the UK, Ireland, and Wales, the echocardiography rate in STEMI patients in some hospitals remains low, accounting for only about 50% of patients (9).

From pathophysiological hypotheses, greater myocardial cell damage or larger infarct size leads to more severe regional wall motion abnormalities, thereby reducing left ventricular contractility. High-sensitivity cardiac troponin (hs-cTn) is a biomarker of myocardial cell injury and plays a central role in diagnosing myocardial infarction (10). Clinical experimental studies have shown that cardiac troponin I (cTnI) is closely associated with the extent of myocardial injury and infarct size (11–14) suggesting that cTnI may help predict LVEF in patients with acute myocardial infarction. In patients with STEMI, several studies have demonstrated an inverse correlation between cTnI and LVEF (15, 16). However, data on this correlation in patients with NSTEMI are limited, and no studies in Vietnam have documented this relationship. Therefore, we conducted a study on high-sensitivity cardiac troponin I (hs-cTnI) levels in first-time NSTEMI patients to evaluate the correlation between hs-cTnI and LVEF.

2. OBJECTIVE

This aim to help identify which patients should be prioritized for left ventricular function assessment, thereby supporting prognosis and optimizing treatment, especially in resource-limited countries like Vietnam.

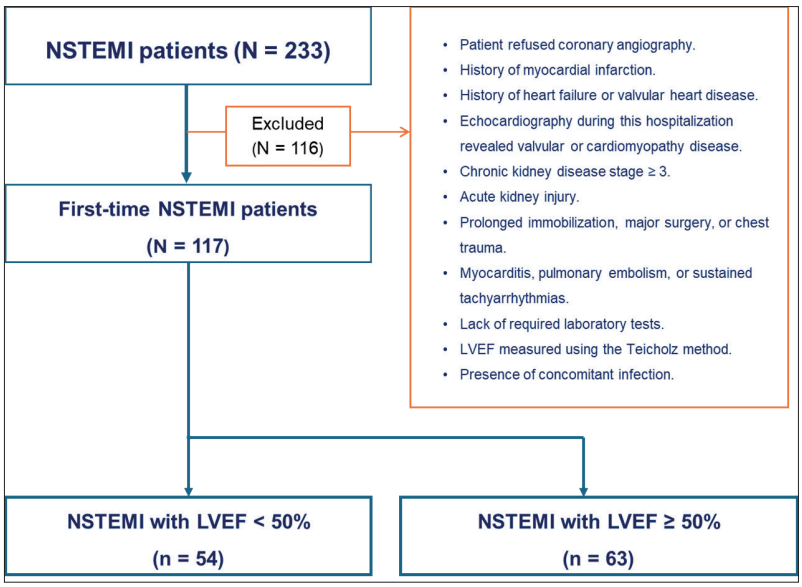

Figure 1. Flowchart of the study population selection. Abbreviation: NSTEMI, Non-ST-Elevation Myocardial Infarction; LVEF, Left Ventricular Ejection Fraction

3. MATERIAL AND METHODS

Subjects and study design

This was a cross-sectional descriptive study conducted at the Interventional Cardiology Department of Cho Ray Hospital from January 2024 to June 2024. All patients included in the study were aged 18 years or older. The Interventional Cardiology Department at Cho Ray Hospital is one of the largest coronary intervention centers in Southern Vietnam and serves as a tertiary referral hospital and a leading unit in interventional cardiology for the region. The study population consisted of patients diagnosed with first-time NSTEMI and treated at the hospital.

Inclusion criteria included patients with a confirmed first-time NSTEMI diagnosis according to the Fourth Universal Definition of Myocardial Infarction (10, presenting with coronary artery stenosis of 50% or greater on percutaneous coronary angiography, and having left ventricular ejection fraction measured using the biplane Simpson method before undergoing coronary angiography, with echocardiography performed within 72 hours of hospital admission. Patients were excluded from the study if they were diagnosed with STEMI, had a history of myocardial infarction or heart failure, or had other cardiovascular conditions such as valvular heart disease, congenital heart disease, or cardiomyopathy (Figure 1). Additionally, patients with non-cardiac conditions that could elevate high-sensitivity cardiac troponin I (hs-cTnI) levels, including systemic infections, chronic kidney disease stage ≥ 3, acute kidney injury, prolonged immobilization, major surgery, chest trauma, myocarditis, pulmonary embolism, or sustained tachyarrhythmias, were also excluded (17).

The study was approved by the Ethics Committee for Biomedical Research at the University of Medicine and Pharmacy, Ho Chi Minh City (ID: 1323/HDDD-DHYD) in December 2023, and written informed consent was obtained from all patients

Study Variables

In this study, LVEF was measured within 72 hours after hospital admission and before coronary angiography. LVEF was assessed using the Simpson biplane method on echo-

cardiography, as recommended by the American Society of Echocardiography (ASE) in 2015. Global left ventricular systolic function was calculated as the difference between end-diastolic and end-systolic volumes measured in a single-plane, 2D echocardiographic view, divided by the end-diastolic volume. The final LVEF value was the average of measurements from two apical views: three-chamber and four-chamber. End-diastole was defined as the first frame after mitral valve closure or the point in the cardiac cycle when the left ventricular size and volume were largest. End-systole was defined as the first frame after aortic valve closure or the point when the left ventricular size and volume were smallest (18).

For patients with regular heart rhythms, M-mode echocardiography, pulsed-wave, or continuous-wave Doppler was used to assess valve opening and closing times for accurate determination of ventricular cycle phases. The echocardiographic assessment was performed using the Philips CX50 ultrasound system with an S5-1 probe at a frequency of 5 MHz. Two cardiologists, each with at least five years of experience in echocardiography and certified in cardiac ultrasound, conducted the assessments. The final LVEF value was the average of two independent measurements. If the difference between the two values was ≥ 5% and impacted LVSD classification, a third expert adjudicated the final result. The echocardiographic evaluation was conducted in a manner that avoided delaying revascularization in patients classified as very high-risk NSTE-ACS per the 2023 European Society of Cardiology guidelines (5).

Per the 2015 American Society of Echocardiography guidelines, patients were categorized into two groups: no LVSD (LVEF ≥ 50%) and LVSD present (LVEF < 50%) (18). Within the LVSD group, mild LVSD corresponded to LVEF 41–49%, whereas moderate-to-severe LVSD was defined as LVEF ≤ 40%. High-sensitivity cardiac troponin I (hs-cTnI) levels were measured at admission using the Abbott Architect i2000SR automated immunoassay analyzer, which is FDA-approved for the diagnosis of acute myocardial infarction. The ARCHITECT STAT High Sensitivity Troponin-I

Characteristics	Total N = 117	LVEF ≥ 50% n = 63	LVEF < 50% n = 54	p-value
Age, years	65.5 ± 11.1	65.2 ± 1.5	65.9 ± 1.4	0.73
Gender, n (%)				
Male	73 (62.4)	40 (63.5)	33 (61.1)	0.79
Female	44 (37.6)	23 (36.5)	21 (38.9)	
BMI, Kg/m2	22.9 ± 3.2	22.7 ± 3.3	23.2 ± 2.9	0.38
Hypertension, n (%)	90 (76.9)	51 (80.9)	39 (72.2)	0.26
Diabetes mellitus, n (%)	40 (32.4)	20 (31.8)	20 (37.0)	0.55
Dyslipidemia, n (%)	57 (48.7)	27 (42.9)	30 (55.6)	0.17
Smoking, n (%)	59 (50.4)	30 (47.6)	29 (53.7)	0.51
Family history of early CAD, n (%)	6 (5.1)	2 (3.2)	4 (7.4)	0.41
Overweight/Obesity, n (%)	58 (49.6)	28 (44.4)	30 (55.6)	0.23
Ischemic stroke, n (%)	11 (9.4)	5 (7.9)	6 (11.1)	0.56
Chronic obstructive pulmonary disease, n (%)	9 (7.7)	3 (4.8)	6 (11.1)	0.30
Peripheral artery disease, n (%)	4 (3.4)	1 (1.6)	3 (5.6)	0.33
Heart rate, bpm	79.4 ± 12.3	77.2 ± 10	82 ± 14.1	0.03
Systolic blood pressure, mmHg	127.1 ± 16.3	128.7 ± 14.7	125.2 ± 17.9	0.25
Diastolic blood pressure, mmHg	76.2 ± 8.7	76.8 ± 7.6	75.5 ± 9.8	0.40
Killip, n (%)				
I	98 (83.8)	60 (95.2)	38 (70.4)	< 0.001
II	17 (14.5)	3 (4.8)	14 (25.9)	
III	2 (1.7)	0 (0)	2 (3.7)	
IV	0 (0)	0 (0)	0 (0)	
Total cholesterol, mg/dL	188 [153 – 222]	192 [152 – 227]	179.5 [158 – 216]	0.83
HDL-C, mg/dL	124.6 [89 – 151]	38 [33 – 47]	38 [32 – 43]	0.69
LDL-C, mg/dL	38 [33 – 60]	128.2 [85 – 152]	123.5 [92 – 145.6]	0.97
Triglycerides, mg/dL	172 [120 – 245]	170 [120 – 256]	174.5 [128 – 231]	0.87
hs-cTnI, pg/mL	8703.7 [2430.5 – 21134.9]	3277.8 [1684.2 – 10604.9]	15314.8 [7544.9 – 25.000]	< 0.001
Single-vessel disease, n (%)	28 (23.9)	16 (25.4)	12 (22.2)	0.74
Two-vessel disease, n (%)	35 (29.9)	20 (31.8)	15 (27.8)	
Three-vessel disease, n (%)	54 (46.2)	27 (42.9)	27 (50)	
Length of hospital stay, days	5 [4 – 7]	5 [4 – 6]	5.5 [5 – 8]	0.04

Table 1. Comparison of Clinical and Laboratory Characteristics Between NSTEMI Patients With and Without LVSD. Abbreviations: SD, Standard Deviation; LVEF, Left Ventricular Ejection Fraction; BMI, Body Mass Index; CAD, Coronary Artery Disease; COPD, Chronic Obstructive Pulmonary Disease; bpm, Beats Per Minute; IQR, Interquartile Range; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; hs-cTnI, High-Sensitivity Cardiac Troponin I.

assay is a chemiluminescent microparticle immunoassay (CMIA) used to quantify cardiac troponin I (cTnI) in human plasma (EDTA dipotassium [K2]) on the ARCHITECT i2000SR system (19).

In addition to LVEF and hs-cTnI, demographic and clinical variables were collected, including age, sex, body mass index, hypertension, diabetes mellitus, dyslipidemia, smoking status, family history of premature coronary artery disease, history of stroke, chronic obstructive pulmonary disease, peripheral artery disease, Killip classification, lipid profile, and length of hospital stay. Coronary angiography results were used to determine the severity of coronary artery disease, with significant stenosis defined as ≥ 50% luminal narrowing in an epicardial coronary artery. Patients were categorized based on the number of affected vessels: single-vessel disease, two-vessel disease, three-vessel disease, and multivessel disease (≥ 2 vessels with ≥ 50% stenosis).

Statistical Analysis

Data entry and processing were performed using Stata 17.0 (StataCorp, 2021, College Station, TX). Categorical variables were expressed as frequencies and percentages and compared using the chi-square test. Continuous variables were assessed for normality with the Shapiro–Wilk test; normally distributed data were reported as mean ± SD and compared using t-tests or ANOVA, while non-normal data were presented as median (IQR) and analyzed with the Mann–Whitney U or Kruskal–Wallis test. Linear regression evaluated the correlation between LVEF and hs-cTnI levels. Logistic regression and ROC curve analysis determined the optimal hs-cTnI cutoff for predicting LVSD using the Youden index. Statistical significance was set at $p < 0.05$ for all tests.

4. RESULTS

Study Population Characteristics

Over the study period, 117 patients fulfilled the inclusion criteria. The study cohort had a mean age of 65.5 ± 11.1 years and was predominantly male (62.4%). Among the 117 patients in the study sample, the proportion of those with left ventricular systolic dysfunction (LVSD) (LVEF < 50%) was 46.1%, which was lower than the proportion of patients without LVSD (LVEF ≥ 50%) at 53.9%. Within the LVSD group, 30 patients had an LVEF ≤ 40%, accounting for 25.6%, while 24 patients had an LVEF ranging from 41% to 49%, representing 20.5%. Baseline clinical and laboratory characteristics of patients with NSTEME are summarized in Table 1. Among these patients, those with LVSD demonstrated statistically significant differences in several clinical and laboratory parameters compared to those without LVSD. Notably, the mean heart rate was significantly elevated in the LVSD group relative to the non-LVSD group (82 bpm vs. 77.2 bpm, $p = 0.03$). Furthermore, the proportion of patients with a Killip classification of ≥ II was markedly higher in the LVSD group (29.6% vs. 4.8%, $p < 0.001$), suggesting a greater degree of heart failure severity (Table 1).

Regarding biochemical markers, the mean hs-cTnI level at admission was significantly higher in the LVSD group compared to the non-LVSD group (15,314.8 pg/mL vs. 3,277.8 pg/mL; $p < 0.001$), reflecting more extensive myocardial necrosis. Additionally, the mean length of hospital stay was longer in the LVSD group than in the non-LVSD group (5.5 days vs. 5 days; $p = 0.04$), suggesting a more severe disease course requiring prolonged treatment.

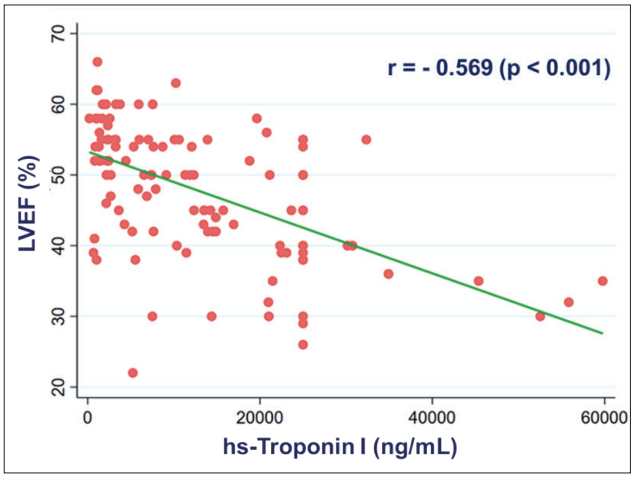

Figure 2. Correlation Between Admission hs-cTnI Levels and LVEF. Abbreviations: LVEF, Left Ventricular Ejection Fraction; hs-Troponin I, High-Sensitivity Troponin I.

Correlation Between Admission hs-cTnI Levels and LVEF

Regarding the relationship between high-sensitivity cardiac troponin I (hs-cTnI) levels at admission and LVEF, we observed a negative correlation in patients with first-time NSTEMI. Specifically, hs-cTnI levels at admission and LVEF assessed by transthoracic echocardiography using the Simpson biplane method showed an inverse correlation (Figure 2), with a Spearman correlation coefficient of $r = -0.569$ ($p < 0.001$). According to Guilford's classification (1963), this correlation is of moderate strength.20

In this study, we observed that in patients with first-time NSTEMI, lower LVEF was associated with higher hs-cTnI levels, with the highest hs-cTnI concentrations observed in patients with LVEF ≤ 40%. Based on LVEF and the severity of left ventricular systolic dysfunction (LVSD), patients were categorized into two groups: those with and without LVSD. Within the LVSD group, LVEF between 41–49% corresponded to mild LVSD, while LVEF ≤ 40% was classified as moderate-to-severe LVSD. Patients with moderate-to-severe LVSD (LVEF ≤ 40%) exhibited the highest hs-cTnI levels, with a median concentration of 25,000 pg/mL. The differences in hs-cTnI levels among the three groups - patients without LVSD, those with mild LVSD, and those with moderate-to-severe LVSD - were statistically significant ($p < 0.001$) (Table 2).

LVEF Groups (%)	hs-cTnI, pg/mL	LVEF, %	p-value
Group I (n = 63)			
LVEF ≥ 50%	3277.8 [1684.2 – 10604.9]	55 [52 – 58]	p < 0.001*
Group II (n = 54)			
LVEF 41 – 49% (n = 24)	13482.6 [5524.1 – 14880.9]	45 [42.5 – 45]	
LVEF ≤ 40% (n = 30)	25000 [14394.7 – 30158.1]	37 [30 – 39]	

Table 2. Admission hs-cT. I Levels in Different Patient Groups (N = 117. *Kruskal-Wallis test was used. Abbreviations: LVEF, Left Ventricular Ejection Fraction; IQR, Interquartile Range; hs-cTnI, High-Sensitivity Cardiac Troponin I.

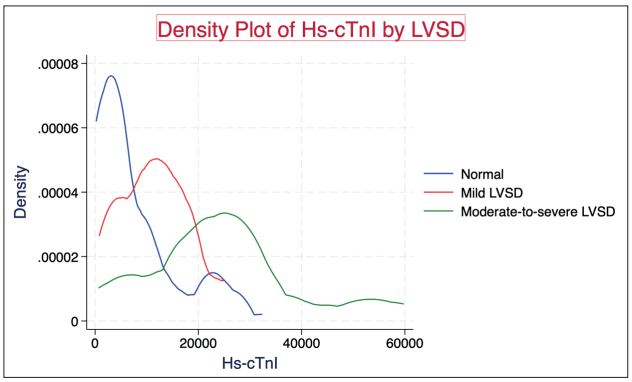

Figure 3. Distribution of hs-troponin I levels at admission according to LVSD classification. Abbreviations: LVSD, Left Ventricular Systolic Dysfunction; hs-cTnI, High-Sensitivity Cardiac Troponin I.

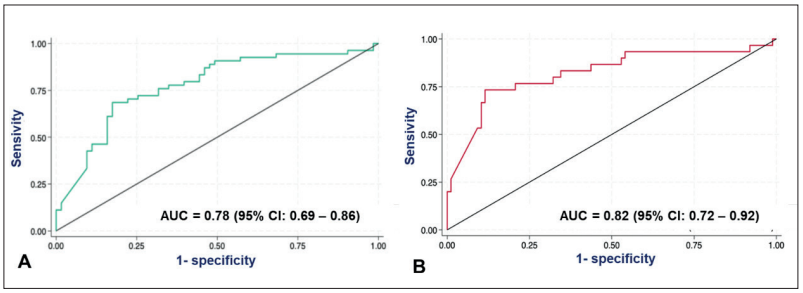

Figure 4. ROC curves for hs-cTnI levels in predicting LVEF < 50% (4A) and LVEF ≤ 40% (4B). Abbreviations: LVEF, Left Ventricular Ejection Fraction; ROC, Receiver Operating Characteristic; CI, Confidence Interval.

Although patients with moderate-to-severe left ventricular systolic dysfunction (LVSD) typically demonstrated elevated high-sensitivity cardiac troponin I (hs-cTnI) levels, the density plot revealed a significant overlap in hs-cTnI values across LVSD categories. This overlap resulted from substantial variability around the mean, as some patients without LVSD exhibited unusually high hs-cTnI concentrations, while certain individuals with severe LVSD showed comparatively lower values. Figure 3 clearly depicted this pattern, indicating that, although hs-cTnI levels correlated with the severity of LVSD, they did not fully differentiate between LVSD subgroups.

For patients with LVEF < 50%, the area under the ROC curve (AUC) was 0.78, which indicated good predictive ability of hs-cTnI levels. The optimal hs-cTnI cutoff was determined to be 12,344 pg/mL, with a Youden index of 0.512, resulting in a sensitivity of 68.5% and specificity of 82.5% (PLR = 3.9; NLR = 0.4). In contrast, for patients with LVEF ≤ 40%, the AUC rose to 0.82, with an optimal hs-cTnI cutoff of 20,979 pg/mL, a Youden index of 0.618, sensitivity of 73.3%, and specificity of 88.5% (PLR = 6.4; NLR = 0.3) (Figure 4). These findings suggested that hs-cTnI served as a valuable biomarker for predicting left ventricular systolic dysfunction, with higher hs-cTnI levels corresponding to greater impairment in LVEF (Table 3). This supported the utility of hs-cTnI as an early diagnostic and risk stratification tool in patients with NSTEMI.

Parameters	LVEF < 50%	LVEF ≤ 40%
hs-cTnI cut-off value (pg/mL)	12.344	20.979
ROC AUC	0.778	0.821
Youden index	0.512	0.618
Sensitivity (%)	68.5%	73.3%
Specificity (%)	82.5%	88.5%
Positive Likelihood Ratio	3.9	6.4
Negative Likelihood Ratio	0.4	0.3

Table 3. Predictive Performance of hs-cTnI Cut-off Values for LVSD Severity. Abbreviations: LVSD, Left Ventricular Systolic Dysfunction; LVEF, Left Ventricular Ejection Fraction; hs-cTnI, High-Sensitivity Cardiac Troponin I; ROC AUC, Receiver Operating Characteristic Area Under the Curve.

5. DISCUSSION

Non-ST-elevation myocardial infarction remains one of the leading causes of mortality and cardiovascular complications worldwide. High-sensitivity cardiac troponin I (hs-cTnI) is a highly specific biomarker for myocardial injury, and previous studies have demonstrated that elevated hs-cTnI levels are closely associated with infarct size and left ventricular systolic dysfunction (LVSD) (4, 21, 22).

Our study found a negative correlation between admission hs-cTnI levels and LVEF in first-time NSTEMI patients, with a Spearman correlation coefficient of r = -0.569 (p < 0.001). According to Guilford's classification (1963), this correlation is considered moderate (20). This result is consistent with several international studies. Hossain et al. conducted a study on 132 acute coronary syndrome patients, including 57 with NSTEMI, and reported a correlation coefficient of r = -0.516 (p < 0.001) (23). Similarly, Wasyanto found a correlation coefficient of r = -0.53 (p = 0.009) in a study of 23 myocardial infarction (MI) patients, including 8 with NSTEMI (24). Khan MH, in a study involving 130 NSTEMI patients, reported r = -0.539 (p = 0.001) (25). However, our correlation coefficient is higher than that reported by Islam, who found r = -0.411 (p < 0.001) in 160 NSTEMI patients (26). Overall, these findings consistently indicate a negative correlation between hs-cTnI levels and LVEF, with a moderate correlation strength according to Guilford's classification.

Furthermore, our study observed that as LVEF decreased, hs-cTnI levels increased, particularly in patients with LVEF ≤ 40%. This finding aligns with the study by Khan MH, which reported the highest cTnI levels in patients with LVEF ≤ 35% and the lowest levels in those with LVEF ≥ 55% (p < 0.001) (25). This suggests that hs-cTnI may serve as a valuable tool for early prediction of LVSD. Higher hs-cTnI levels correspond to lower LVEF and vice versa, providing clinicians with a practical approach to assessing left ventricular function and aiding in patient management, monitoring, and prognosis.

With an hs-cTnI cutoff of ≥ 12,344 pg/mL, our study demonstrated the ability to predict LVEF < 50% with a sensitivity of 68.5% and specificity of 82.5%. This threshold is higher than those reported by Hossain and Khan MH. Hossain used a cTnI cutoff of ≥4,500 pg/mL to predict LVEF < 50%, achieving a sensitivity of 65% and specificity of 54.1%.23 Khan MH determined a cTnI cutoff of ≥ 6,600 pg/mL for predicting LVEF < 55%, with 100% sensitivity and 92.4% specificity (.25). These discrepancies may be due to differences in study design. Hossain's study used conventional cTnI assays

rather than high-sensitivity assays and had a smaller sample size, with patient groups categorized based on a predefined cutoff of 4,500 pg/mL rather than an optimized sensitivity-specificity threshold (23). Similarly, Khan MH used conventional cTnI assays instead of hs-cTnI and defined LVSD as LVEF < 55%, whereas we defined LVSD as LVEF < 50% (25).

Cardiac troponin I is an almost exclusive protein of cardiomyocytes, released into the bloodstream only upon myocardial injury. Myocardial infarction damages cardiomyocytes, leading to troponin I release into circulation, and the magnitude of hs-cTnI elevation reflects the extent of myocardial injury. High-sensitivity cardiac troponin I assays can detect troponin levels 10–100 times lower than conventional assays, enabling early diagnosis or exclusion of MI within 1–3 hours (17). Due to its high sensitivity and accuracy, hs-cTnI is superior in detecting myocardial injury compared to conventional troponin assays (27). As myocardial damage extends, left ventricular contractility diminishes, reducing LVEF. This explains the inverse relationship between hs-cTnI levels and LVEF: patients with greater myocardial injury exhibit higher hs-cTnI levels and lower LVEF. However, in clinical practice, overlap in hs-cTnI values among patient groups is observed due to factors such as variability in test timing, early interventions, and comorbidities influencing troponin release. The substantial variation (high standard deviation) around mean hs-cTnI values reduces clear differentiation between LVSD groups, necessitating integration of hs-cTnI with other clinical assessments, such as echocardiography and ECG, for comprehensive cardiac evaluation. This partly explains the differences in hs-cTnI cutoff values for predicting LVSD in our study compared to others.

Our findings provide evidence that hs-cTnI may serve as a useful biomarker for predicting LVSD in NSTEMI patients. With ROC AUC values of 0.78 for LVEF < 50% and 0.82 for LVEF ≤ 40%, the hs-cTnI cutoff values (12,344 pg/mL and 20,979 pg/mL) demonstrate good discriminative ability between patients with normal and impaired cardiac function. To date, no studies have established an hs-cTnI cutoff for predicting LVEF ≤ 40% for direct comparison. However, among 30 patients with LVEF ≤ 40%, the majority (22 patients, 73.3%) had admission hs-cTnI levels ≥ 20,000 pg/mL. This suggests that hs-cTnI could be a valuable biomarker in assessing LVSD severity, particularly in clinical settings where timely echocardiography may be limited due to resource constraints. Utilizing hs-cTnI for risk stratification may help clinicians identify high-risk patients requiring closer monitoring and targeted interventions, thereby optimizing treatment strategies and improving patient outcomes.

A notable strength of our study is the use of hs-cTnI, a highly sensitive assay allowing early detection of myocardial injury, to evaluate the extent of cardiac dysfunction. The study sample consisted of 117 first-time NSTEMI patients, eliminating confounding factors from recurrent events or prior interventions and thereby enhancing result reliability. The determination of optimal hs-cTnI cutoff values based on the Youden index provides valuable information for risk stratification and treatment planning, particularly in healthcare settings with limited echocardiographic resources.

However, our study has some limitations. The sample size was relatively small, with only 25.6% of patients having LVEF ≤ 40%, potentially limiting the generalizability of findings. The cross-sectional design does not establish causality between hs-cTnI levels and cardiac dysfunction but rather identifies a statistical association. The overlap in hs-cTnI values among LVEF groups indicates that despite a clear correlation, hs-cTnI alone cannot replace echocardiographic assessment and should be integrated with other clinical parameters. Additionally, determining cutoff values based on the Youden index may be influenced by sample characteristics and data distribution, necessitating further studies with larger cohorts and advanced statistical methods to enhance predictive accuracy.

6. CONCLUSION

Hs-cTnI levels demonstrated a moderate inverse correlation with LVEF and proved to be a valuable biomarker for predicting LVSD. The identification of optimal hs-cTnI cut-off values enabled early detection of LVSD prior to echocardiography, thereby supporting effective risk stratification and informing timely interventions in patients with NSTEMI.

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- **Declaration of patient consent:** The authors certify that they have obtained all appropriate patient consent forms.
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