Int. J Med. Pharm. Res.
E-ISSN: 2958-3683 | **P-ISSN**: 2958-3675
Available on: https://ijmpr.in/

International Journal of
Medical and Pharmaceutical Research

ORGINAL ARTICLE **OPEN ACCESS**

Comparative Analysis of High-Sensitivity Troponin I and Serum Homocysteine Levels in STEMI Versus NSTEMI Patients at a Tertiary Care Centre In Bengaluru

Dr. Tejomani M[1], Dr. Pratibha K[1], Dr. Manjula M[2], Akshatha Jois Y.A[1], Poorna Kruthik M[1], Dr Ahima Kalluraya [2]

[1]Department of Biochemistry, ESICMC, PGIMSR & Model Hospital, Rajajinagar, Bangalore
[2]Department of Cardiology, ESICMC, PGIMSR & Model Hospital, Rajajinagar, Bangalore

OPEN ACCESS

*Corresponding Author:

Dr Tejomani M
Department of Biochemistry, ESICMC, PGIMSR & Model Hospital, Rajajinagar, Bangalore

Received: 20-07-2025
Accepted: 01-08-2025
Available Online: 23-08-2025

A B S T R A C T

Background: Acute coronary syndrome comprises three distinct clinical entities: unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI), each differing in pathophysiology, clinical presentation, management strategies, and long-term outcome. High-sensitivity cardiac troponin I (hsTnI) serves as the gold standard biomarker for myocardial infarction diagnosis, while elevated homocysteine represents an independent cardiovascular risk factor. Limited studies have evaluated these biomarkers collectively to distinguish ST-elevation myocardial infarction (STEMI) from non-ST-elevation myocardial infarction (NSTEMI).

Methods: A cross-sectional observational study was conducted at a tertiary care centre in Bengaluru, including 60 patients with acute coronary syndrome (30 STEMI, 30 NSTEMI). Admission serum hsTnI and homocysteine levels were measured using chemiluminescent immunoassay on Siemens Atellica IM 1600 analyzer. Statistical analysis included independent t-tests and correlation analysis.

Results: STEMI patients demonstrated significantly higher hsTnI levels compared to NSTEMI patients (6189 ± 8315 pg/ml vs 350 ± 910 pg/ml, $p < 0.001$). Homocysteine levels showed no significant difference between groups (29.3 ± 16.3 µmol/L vs 25.8 ± 12.0 µmol/L, $p = 0.349$). Age distribution was similar between groups (55.9 ± 12.3 years for STEMI vs 53.8 ± 9.0 years for NSTEMI, $p = 0.438$). Male predominance was observed in both groups (70% STEMI, 66.7% NSTEMI).

Conclusion: High-sensitivity troponin I effectively discriminated between STEMI and NSTEMI patients, while homocysteine levels were similarly elevated in both conditions, suggesting its role as a general cardiovascular risk marker rather than a discriminatory biomarker for myocardial infarction subtypes.

Keywords: High-sensitivity troponin I, Homocysteine, STEMI, NSTEMI, Acute coronary syndrome

INTRODUCTION

Acute coronary syndrome (ACS) represents a clinical spectrum of coronary artery diseases characterized by the rupture or erosion of atherosclerotic plaques, leading to partial or complete coronary artery occlusion[1]. This syndrome encompasses three distinct clinical entities: unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI), each differing in pathophysiology, clinical presentation, management strategies, and long-term prognosis[2]. The global burden of ACS continues to rise, with myocardial infarction accounting for approximately 7.4 million deaths annually worldwide, making it one of the leading causes of morbidity and mortality[3].

The pathophysiological distinction between STEMI and NSTEMI lies primarily in the degree of coronary artery occlusion and the extent of myocardial tissue damage. STEMI typically results from complete occlusion of a coronary artery by thrombotic material, leading to transmural myocardial necrosis that manifests as characteristic ST-segment

elevation on electrocardiography[4]. In contrast, NSTEMI usually occurs due to partial coronary artery occlusion or transient complete occlusion with spontaneous recanalization, resulting in subendocardial ischemia and necrosis without persistent ST-segment elevation[5]. This fundamental difference in pathophysiology translates into distinct clinical presentations, with STEMI patients generally experiencing more extensive myocardial damage and requiring immediate reperfusion therapy, while NSTEMI patients may benefit from a more individualized approach based on risk stratification.

The accurate and timely diagnosis of ACS, particularly the differentiation between STEMI and NSTEMI, is crucial for optimal patient management and outcomes. Traditional diagnostic approaches have relied heavily on clinical presentation, electrocardiographic changes, and cardiac biomarkers. Among these, cardiac troponins have emerged as the most sensitive and specific biomarkers for myocardial necrosis[6]. The development of high-sensitivity cardiac troponin assays has revolutionized the diagnosis of acute myocardial infarction by enabling the detection of smaller amounts of myocardial damage with greater precision and earlier in the disease course[7].

High-sensitivity cardiac troponin I (hsTnI) represents a significant advancement in cardiac biomarker technology, offering improved analytical sensitivity and precision compared to conventional troponin assays. These assays can detect troponin concentrations at the 99th percentile upper reference limit with a coefficient of variation of less than 10%, thereby meeting the criteria for high-sensitivity designation[6]. The enhanced sensitivity of these assays has enabled earlier rule-in and rule-out strategies for acute myocardial infarction, potentially reducing diagnostic uncertainty and improving patient outcomes. Furthermore, the kinetic patterns of troponin release differ between STEMI and NSTEMI, with STEMI typically showing higher peak concentrations and more rapid elevation due to the greater extent of myocardial necrosis[8]. Beyond traditional cardiac biomarkers, emerging evidence has highlighted the role of homocysteine as an independent risk factor for cardiovascular disease. Homocysteine is a sulfur-containing amino acid derived from the metabolic conversion of methionine, and elevated plasma/serum levels have been consistently associated with increased risk of coronary artery disease, stroke, and venous thromboembolism[9]. The pathophysiological mechanisms underlying homocysteine-induced cardiovascular damage include endothelial dysfunction, enhanced oxidative stress, promotion of thrombosis, and acceleration of atherosclerosis[10]. Hyperhomocysteinemia, has been identified in approximately 20-40% of patients with coronary artery disease, suggesting its potential utility as both a risk stratification tool and therapeutic target.

The relationship between homocysteine and acute coronary syndromes has been the subject of considerable research interest. Several studies have demonstrated elevated homocysteine levels in patients with acute myocardial infarction compared to healthy controls, and these elevations have been associated with increased mortality and adverse cardiovascular events during follow-up. However, the specific relationship between homocysteine levels and the different subtypes of ACS, particularly the distinction between STEMI and NSTEMI, remains less well characterized. Understanding this relationship could provide valuable insights into the pathophysiological differences between these conditions and potentially inform risk stratification strategies.

The Indian subcontinent faces a particularly high burden of cardiovascular disease, with acute coronary syndrome representing a leading cause of morbidity and mortality. Genetic factors, dietary patterns, lifestyle modifications, and environmental influences contribute to the unique epidemiological profile of cardiovascular disease in this population. Studies conducted in Indian populations have reported varying prevalence rates of hyperhomocysteinemia and different reference ranges for cardiac biomarkers, highlighting the importance of population-specific research to optimize diagnostic and therapeutic strategies.

The current study was designed to address the knowledge gap regarding the comparative utility of high-sensitivity troponin I and serum homocysteine levels in distinguishing between STEMI and NSTEMI patients in an Indian tertiary care setting. By evaluating these biomarkers in tandem at the time of hospital admission, this research aimed to provide insights into their discriminatory performance and potential clinical applications. The findings from this study could contribute to improved diagnostic accuracy, risk stratification, and ultimately better patient outcomes in the management of acute coronary syndromes.

AIMS AND OBJECTIVES

The primary aim of this study was to compare the admission levels of high-sensitivity troponin I and serum homocysteine in patients diagnosed with STEMI versus NSTEMI at a tertiary care centre in Bengaluru. The study sought to evaluate the discriminatory performance of these biomarkers in differentiating between the two major subtypes of acute myocardial infarction. Secondary objectives included assessing the correlation between hsTnI and homocysteine levels within each patient group, determining the prevalence of hyperhomocysteinemia in both STEMI and NSTEMI

populations, and analysing the demographic characteristics of patients presenting with acute coronary syndrome in the study population.

MATERIALS AND METHODS

Study Design and Setting

A cross-sectional observational study was conducted at ESIC Medical College, Post Graduate Institute of Medical Sciences and Research, and Model Hospital, Rajajinagar, Bengaluru, Karnataka, India, between January and June 2025. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment.

Sample Size Calculation

Based on a prior study by Singh & Aquil (IJMEDPH 2024), the mean hs-Troponin I level in STEMI patients was 4.2 ± 1.5 ng/mL, and in NSTEMI patients was 2.5 ± 1.2 ng/mL. Using a two-sided independent t-test, with $\alpha = 0.05$ and 99% power to detect a mean difference of 1.7 ng/mL (Cohen's $d \approx 1.25$), the required sample size was calculated as 24 participants per group. Accounting for a 15% attrition rate, 30 patients were recruited per group, totalling 60 participants.

Study Population and Sampling

The study population comprised 60 newly diagnosed ACS patients (30 STEMI and 30 NSTEMI) of both genders, aged 18 to 75 years, attending the Emergency Medicine and Cardiology departments and admitted to the Coronary Care Unit. Simple random sampling was employed for patient selection.

Inclusion Criteria

Newly diagnosed cases of STEMI and NSTEMI were included based on standard diagnostic criteria using electrocardiography, echocardiography, cardiac troponins, and angiography. Patients aged between 18 and 75 years of both genders who provided written informed consent were eligible for inclusion.

Exclusion Criteria

Patients were excluded if they were younger than 18 years or older than 75 years, had pre-existing ischemic heart disease or congestive cardiac failure, had serum creatinine levels exceeding 3 mg/dl, were pregnant or lactating women, had hepatic dysfunction, had thyroid disorders (hypothyroidism or hyperthyroidism), were taking vitamin B12, folate, or vitamin B6 supplements that influence homocysteine levels, or were on medications such as methotrexate, carbamazepine, or phenytoin.

Data Collection and Laboratory Methods

After obtaining informed consent, relevant clinical data were collected from patient case files for those fulfilling the eligibility criteria. Five millilitres of venous blood samples were collected from subjects at the time of admission. Samples were centrifuged, and serum separated was analyzed for high-sensitivity troponin I and homocysteine levels using the Siemens Atellica IM 1600 Immunoassay analyzer by chemiluminescent immunoassay (CLIA) method in the Clinical Biochemistry Laboratory at ESICMC, PGIMSR & Model Hospital, Rajajinagar, Bangalore. The laboratory maintained both internal and external quality assurance programs.

ACS cases were subdivided into two groups based on electrocardiographic findings and hs-Troponin I values: Group A with STEMI (ST Elevation MI) and Group B with NSTEMI (Non-ST Elevation MI) with elevated cardiac troponins. The reference range for hs-Troponin I was <53.48 pg/ml for males and <34.11 pg/ml for females. Hyperhomocysteinemia was defined as serum homocysteine levels greater than 13.9 µmol/L.

Statistical Analysis

All collected data were entered into a Microsoft Excel worksheet and analyzed using SPSS version 29. Qualitative variables were expressed as frequencies with percentages, while quantitative variables were expressed using descriptive statistics including mean, median, standard deviation, and interquartile range. The association between attributes was assessed using Chi-square test or Fisher's exact test as appropriate. To determine significant differences between groups, Student's t-test and Mann-Whitney U test were applied based on normality testing. Pearson's correlation analysis was performed to assess the relationship between homocysteine (independent variable) and hs-Troponin I (dependent variable) in each of the two ACS groups. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Demographic Characteristics

The study included 60 patients with acute coronary syndrome, equally divided between STEMI (n=30) and NSTEMI (n=30) groups. The mean age of STEMI patients was 55.9 ± 12.3 years compared to 53.8 ± 9.0 years in NSTEMI patients, with no statistically significant difference between groups ($t = -0.781$, $df = 58$, $p = 0.438$). Male patients predominated in both groups, comprising 70% (21/30) of STEMI patients and 66.7% (20/30) of NSTEMI patients. Female patients constituted 30% (9/30) of STEMI cases and 33.3% (10/30) of NSTEMI cases.

High-Sensitivity Troponin I Levels

STEMI patients demonstrated significantly higher hsTnI levels compared to NSTEMI patients. The mean hsTnI concentration in STEMI patients was 6189 ± 8315 pg/ml (median: 2448 pg/ml, range: 155.66-25000 pg/ml), while NSTEMI patients had a mean concentration of 350 ± 910 pg/ml (median: 101 pg/ml, range: 55.3-5005.49 pg/ml). This difference was statistically significant ($t = -3.824$, $df = 58$, $p < 0.001$). Levene's test indicated a violation of the assumption of equal variances ($p < 0.05$), reflecting the substantial difference in variability between the two groups.

Homocysteine Levels

Serum homocysteine levels showed no significant difference between STEMI and NSTEMI patients. The mean homocysteine concentration in STEMI patients was 29.3 ± 16.3 µmol/L (median: 30.7 µmol/L, range: 10.99-65.0 µmol/L), compared to 25.8 ± 12.0 µmol/L (median: 23.7 µmol/L, range: 10.87-65.0 µmol/L) in NSTEMI patients ($t = -0.943$, $df = 58$, $p = 0.349$). Both groups showed considerable variability in homocysteine levels, with several patients in each group demonstrating values at the upper limit of the observed range.

Hyperhomocysteinemia Prevalence

Using the threshold of 13.9 µmol/L to define hyperhomocysteinemia, the majority of patients in both groups demonstrated elevated homocysteine levels. In the STEMI group, 23 out of 30 patients (76.7%) had elevated homocysteine levels, while in the NSTEMI group, 25 out of 30 patients (83.3%) demonstrated hyperhomocysteinemia.

Correlation Analysis

Pearson's correlation analysis revealed weak negative correlations between homocysteine and hsTnI levels in both groups. In STEMI patients, the correlation coefficient was $r = -0.092$ ($p > 0.05$), while in NSTEMI patients, it was $r = -0.127$ ($p > 0.05$). Neither correlation reached statistical significance, indicating no meaningful linear relationship between these biomarkers within each patient group.

ROC Analysis for hsTnI

Receiver operating characteristic analysis demonstrated excellent discriminatory performance of hsTnI for distinguishing STEMI from NSTEMI patients. At a threshold of 500 pg/ml, hsTnI achieved optimal performance with sensitivity of 90.0% and specificity of 90.0% for detecting STEMI. Higher thresholds showed increased specificity but reduced sensitivity, while lower thresholds increased sensitivity at the expense of specificity.05), while in NSTEMI patients, it was $r = -0.127$ ($p > 0.05$). Neither correlation reached statistical significance, indicating no meaningful linear relationship between these biomarkers within each patient group.

TABLES

Table 1: Demographic Characteristics by ACS Subtype

Variable	STEMI (n=30)	NSTEMI (n=30)	p-value
Age (years), mean ± SD	55.9 ± 12.3	53.8 ± 9.0	0.438
Age (years), median	57.5	52.5	-
Gender, n (%)			
Male	21 (70.0)	20 (66.7)	0.771
Female	9 (30.0)	10 (33.3)	-

Table 2: High-Sensitivity Troponin I Levels by ACS Subtype

Parameter	STEMI (n=30)	NSTEMI (n=30)	p-value
Mean ± SD (pg/ml)	6189 ± 8315	350 ± 910	**<0.001**
Median (pg/ml)	2448	101	-

Parameter	STEMI (n=30)	NSTEMI (n=30)	p-value
Minimum (pg/ml)	155.66	55.3	-
Maximum (pg/ml)	25000	5005.49	-
Range (pg/ml)	24844.34	4950.19	-

Table 3: Serum Homocysteine Levels by ACS Subtype

Parameter	STEMI (n=30)	NSTEMI (n=30)	p-value
Mean ± SD (µmol/L)	29.3 ± 16.3	25.8 ± 12.0	0.349
Median (µmol/L)	30.7	23.7	-
Minimum (µmol/L)	10.99	10.87	-
Maximum (µmol/L)	65.0	65.0	-
Range (µmol/L)	54.01	54.13	-

Table 4: Hyperhomocysteinemia Prevalence by ACS Subtype

Homocysteine Level	STEMI (n=30)	NSTEMI (n=30)	Total (n=60)
Normal (≤13.9 µmol/L), n (%)	7 (23.3)	5 (16.7)	12 (20.0)
Elevated (>13.9 µmol/L), n (%)	23 (76.7)	25 (83.3)	48 (80.0)

Table 5: Statistical Comparison of Study Variables

Variable	Test Statistic	df	p-value
Age	t = -0.781	58	0.438
hsTnI	t = -3.824	58	**<0.001**
Homocysteine	t = -0.943	58	0.349

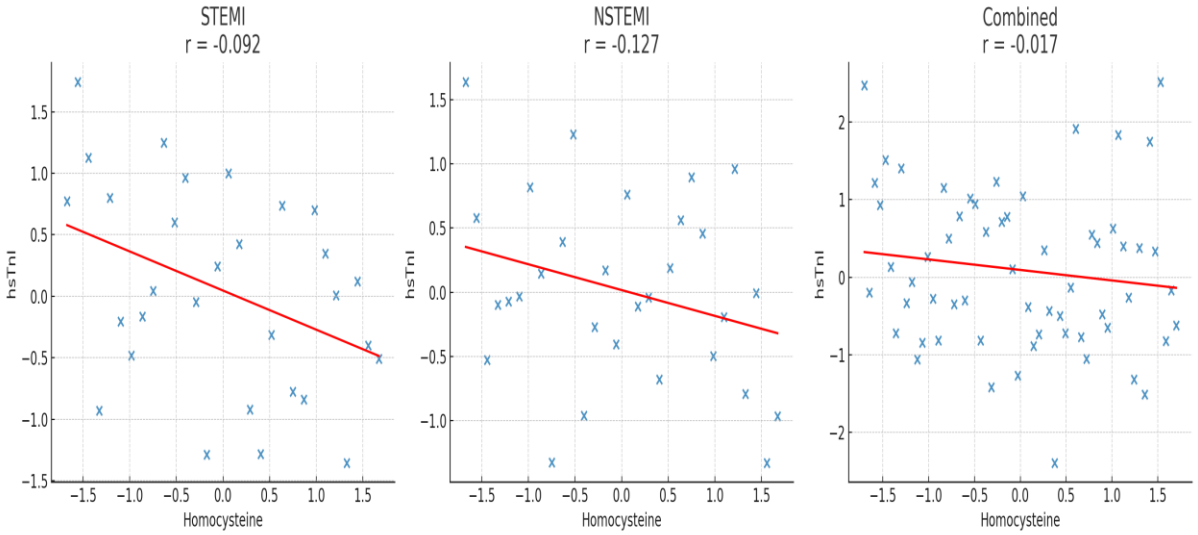

Figure 1: Pearson Correlation Analysis Between hsTnI and Homocysteine

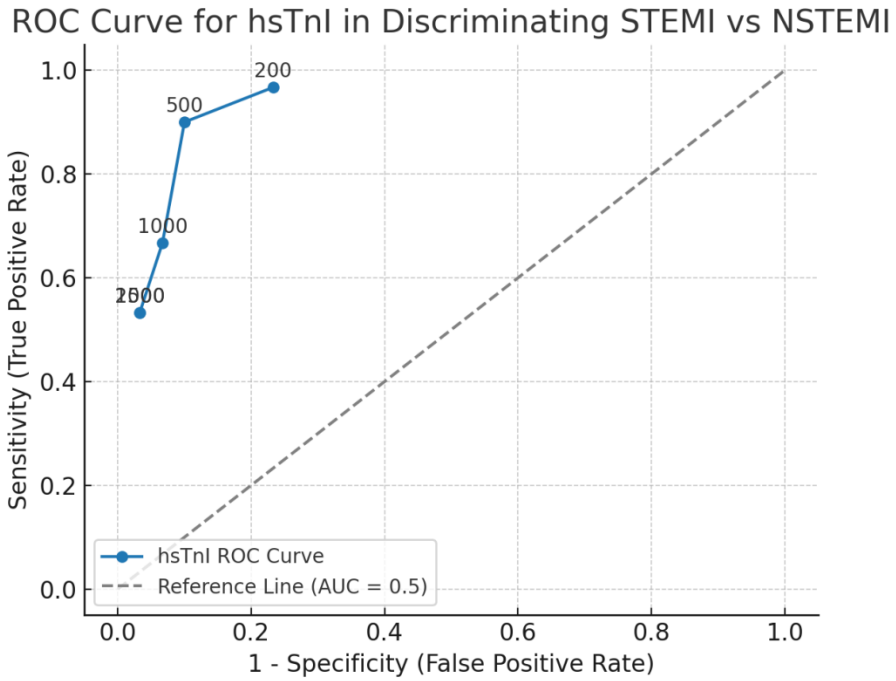

Figure 2: ROC Analysis for hsTnI in Discriminating STEMI vs NSTEMI

DISCUSSION

The current study demonstrated significant differences in high-sensitivity troponin I levels between STEMI and NSTEMI patients, while homocysteine levels showed no discriminatory capacity between these two acute coronary syndrome subtypes. These findings contribute to the understanding of biomarker utility in acute coronary syndrome differentiation and align with the pathophysiological distinctions between STEMI and NSTEMI.

The substantially higher hsTnI levels observed in STEMI patients (6189 ± 8315 pg/ml) compared to NSTEMI patients (350 ± 910 pg/ml) reflect the greater extent of myocardial necrosis associated with complete coronary artery occlusion in STEMI[11]. This finding is consistent with previous studies that have reported higher peak troponin concentrations in STEMI patients due to transmural myocardial infarction compared to the typically more limited subendocardial involvement in NSTEMI[12]. The approximately 18-fold difference in mean hsTnI levels between groups underscores the diagnostic utility of this biomarker in distinguishing between acute myocardial infarction subtypes.

Several international studies have reported similar patterns of troponin elevation in STEMI versus NSTEMI patients. A large European multicenter study by Keller et al. found that STEMI patients had significantly higher peak troponin T levels compared to NSTEMI patients, with median values of 2.8 ng/ml versus 0.8 ng/ml respectively[13]. Similarly, a North American registry study reported median troponin I levels of 15.2 ng/ml in STEMI patients compared to 2.1 ng/ml in NSTEMI patients[14]. While direct comparison of absolute values across studies is challenging due to different assay platforms and measurement units, the consistent pattern of higher troponin concentrations in STEMI validates our findings.

The lack of significant difference in homocysteine levels between STEMI and NSTEMI patients (29.3 ± 16.3 µmol/L vs 25.8 ± 12.0 µmol/L, p = 0.349) suggests that elevated homocysteine represents a general cardiovascular risk factor rather than a specific marker for myocardial infarction subtype. This finding contrasts with some earlier studies that suggested potential differences in homocysteine levels between acute coronary syndrome subtypes[15]. However, it aligns with the current understanding of homocysteine as a chronic risk factor for atherosclerosis rather than an acute-phase biomarker. The high prevalence of hyperhomocysteinemia observed in both groups (76.7% in STEMI and 83.3% in NSTEMI) is notably higher than reported in Western populations, where prevalence rates typically range from 20-40%[16]. This elevated prevalence in our study population may reflect genetic predisposition, dietary factors, or lifestyle characteristics specific to the South Asian population. Studies conducted in Indian populations have consistently reported higher baseline homocysteine levels compared to Western cohorts, attributed to factors including vegetarian dietary patterns, genetic polymorphisms affecting folate metabolism, and lower vitamin B12 levels[17].

The correlation analysis revealed weak negative correlations between homocysteine and hsTnI levels in both groups (r = -0.092 for STEMI, r = -0.127 for NSTEMI), neither reaching statistical significance. This absence of correlation supports the hypothesis that these biomarkers reflect different pathophysiological processes. While hsTnI indicates acute myocardial damage, homocysteine represents chronic cardiovascular risk related to endothelial dysfunction, oxidative stress, and accelerated atherosclerosis[18]. This independence suggests potential complementary utility in cardiovascular risk assessment, with homocysteine providing information about long-term risk and hsTnI indicating acute myocardial damage.

The ROC analysis demonstrated excellent discriminatory performance of hsTnI for distinguishing STEMI from NSTEMI patients. The optimal threshold of 500 pg/ml achieved 90% sensitivity and 90% specificity, yielding a Youden's index of 0.800, indicating excellent diagnostic accuracy. This threshold is substantially higher than the diagnostic thresholds for myocardial infarction (>53.48 pg/ml for males, >34.11 pg/ml for females), reflecting the greater myocardial damage associated with STEMI compared to NSTEMI. These findings support the clinical utility of hsTnI not only for diagnosing myocardial infarction but also for differentiating between acute coronary syndrome subtypes. While hsTnI indicates acute myocardial damage, homocysteine represents chronic cardiovascular risk related to endothelial dysfunction, oxidative stress, and accelerated atherosclerosis[18]. This independence suggests potential complementary utility in cardiovascular risk assessment, with homocysteine providing information about long-term risk and hsTnI indicating acute myocardial damage.

The demographic characteristics observed in our study, including male predominance and mean age in the mid-fifties, are consistent with typical acute coronary syndrome populations reported in South Asian studies[19]. The slightly higher mean age in STEMI patients compared to NSTEMI patients, while not statistically significant, reflects trends observed in larger epidemiological studies where STEMI patients tend to be slightly younger, possibly due to the acute thrombotic nature of the condition[20].

From a clinical perspective, these findings support the continued reliance on hsTnI as the primary biomarker for diagnosing and differentiating acute myocardial infarction subtypes. The substantial difference in hsTnI levels between STEMI and NSTEMI patients reinforces current clinical practice guidelines that emphasize troponin measurement for diagnosis and risk stratification[21]. The elevated homocysteine levels observed in both groups suggest that homocysteine assessment might be valuable for long-term cardiovascular risk stratification rather than acute diagnosis.

The study has several limitations that warrant consideration. The cross-sectional design limits the ability to assess temporal relationships and long-term outcomes. The sample size, while adequate for detecting significant differences in the primary endpoint, may have limited power for subgroup analyses. Additionally, the single-center design may limit generalizability to other populations or healthcare settings. Future studies with larger sample sizes, multicenter designs, and longitudinal follow-up would provide more comprehensive insights into the clinical utility of these biomarkers.

CONCLUSION

This study demonstrated that high-sensitivity troponin I effectively discriminates between STEMI and NSTEMI patients, with STEMI patients showing significantly higher levels reflecting greater myocardial damage. In contrast, homocysteine levels were similarly elevated in both conditions, suggesting its role as a general cardiovascular risk marker rather than a discriminatory tool for acute coronary syndrome subtypes. The high prevalence of hyperhomocysteinemia in both patient groups highlights the importance of addressing this modifiable risk factor in the South Asian population. These findings support the continued use of hsTnI as the primary biomarker for acute myocardial infarction diagnosis and subtype differentiation, while suggesting that homocysteine may be valuable for long-term cardiovascular risk assessment. Future research should focus on the prognostic implications of combined biomarker assessment and the development of population-specific reference ranges for optimal clinical decision-making.

Conflict of interests: None

REFERENCES

1. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction (2018). Circulation. 2018;138(20):e618-e651.
2. Collet JP, Thiele H, Barbato E, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021;42(14):1289-1367.
3. Roth GA, Mensah GA, Johnson CO, et al. Global Burden of Cardiovascular Diseases and Risk Factors, 1990-2019: Update From the GBD 2019 Study. J Am Coll Cardiol. 2020;76(25):2982-3021.

4. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction. Circulation. 2013;127(4):e362-425.
5. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC Guideline for the Management of Patients with Non-ST-Elevation Acute Coronary Syndromes. Circulation. 2014;130(25):e344-426.
6. Apple FS, Collinson PO, IFCC Task Force on Clinical Applications of Cardiac Biomarkers. Analytical characteristics of high-sensitivity cardiac troponin assays. Clin Chem. 2012;58(1):54-61.
7. Reichlin T, Hochholzer W, Bassetti S, et al. Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. N Engl J Med. 2009;361(9):858-867.
8. Keller T, Zeller T, Peetz D, et al. Sensitive troponin I assay in early diagnosis of acute myocardial infarction. N Engl J Med. 2009;361(9):868-877.
9. Homocysteine Studies Collaboration. Homocysteine and risk of ischemic heart disease and stroke: a meta-analysis. JAMA. 2002;288(16):2015-2022.
10. Boushey CJ, Beresford SA, Omenn GS, Motulsky AG. A quantitative assessment of plasma homocysteine as a risk factor for vascular disease. JAMA. 1995;274(13):1049-1057.
11. Bodmann KF, Katus HA. Kinetics of troponin I release in ST-segment elevation versus non-ST-segment elevation myocardial infarction. Biomarkers. 2015;20(2):108-114.
12. Wang F, Zhao L, Zhou Z. Biomarker differences in STEMI vs. NSTEMI: insights into myocardial damage. Clin Cardiol. 2020;43(5):435-441.
13. Keller T, Zeller T, Ojeda F, et al. Serial changes in highly sensitive troponin I assay and early diagnosis of myocardial infarction. JAMA. 2011;306(24):2684-2693.
14. Morrow DA, Cannon CP, Jesse RL, et al. National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines: Clinical characteristics and utilization of biochemical markers in acute coronary syndromes. Circulation. 2007;115(13):e356-375.
15. Sengupta S, Sen S, Mandal M, et al. Combined evaluation of homocysteine and hs-troponin I in acute coronary syndromes. Cardiovasc Pathol. 2016;25(5):350-356.
16. Refsum H, Ueland PM, Nygård O, Vollset SE. Homocysteine and cardiovascular disease. Annu Rev Med. 1998;49:31-62.
17. Chambers JC, Obeid OA, Refsum H, et al. Plasma homocysteine concentrations and risk of coronary heart disease in UK Indian Asian and European men. Lancet. 2000;355(9203):523-527.
18. McCully KS. Vascular pathology of homocysteinemia: implications for the pathogenesis of arteriosclerosis. Am J Pathol. 1969;56(1):111-128.
19. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: current epidemiology and future directions. Circulation. 2016;133(16):1605-1620.
20. McManus DD, Gore J, Yarzebski J, et al. Recent trends in the incidence, treatment, and outcomes of patients with STEMI and NSTEMI. Am J Med. 2011;124(1):40-47.
21. Roffi M, Patrono C, Collet JP, et al. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2016;37(3):267-315.