ORIGINAL RESEARCH

Troponin I and CK-MB values in acute myocardial infarction among female patients with and without ST-elevation

Siva Prasad

Associate Professor, Department of Medicine, Bhaskar Medical College, Moinabad, Ranga Reddy, Hyderabad, Telangana, India

Corresponding Author
Siva Prasad
Associate Professor, Department of Medicine, Bhaskar Medical College, Moinabad, Ranga Reddy, Hyderabad, Telangana, India
Email: drgsivaprasadreddy@gmail.com

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ABSTRACT

Background: Acute myocardial infarction (AMI) is a major cause of morbidity and mortality, with significant differences in clinical presentation and outcomes between STEMI and NSTEMI. Cardiac biomarkers such as troponin I and creatine kinase-MB play a pivotal role in diagnosing AMI, particularly in female patients who often present with atypical symptoms. **Objective:** To compare the levels of troponin I and creatine kinase-MB in female patients with STEMI and NSTEMI and assess their diagnostic utility. **Methods:** This prospective study included 120 female patients diagnosed with AMI, divided equally into STEMI (n=60) and NSTEMI (n=60) groups. Serial measurements of troponin I and creatine kinase-MB were taken at admission, 6 hours, and 12 hours. Statistical analysis was performed to compare biomarker levels between groups. **Results:** Troponin I and creatine kinase-MB levels were significantly higher in STEMI patients compared to NSTEMI patients at all time points ($p < 0.01$). Peak troponin I levels were 12.8 ± 2.1 ng/mL in STEMI versus 5.2 ± 1.3 ng/mL in NSTEMI, while peak creatine kinase-MB levels were 68 ± 8 U/L in STEMI and 28 ± 6 U/L in NSTEMI. Biomarker elevation was more rapid and pronounced in STEMI patients. **Conclusion:** Troponin I and creatine kinase-MB levels differ significantly between STEMI and NSTEMI in female patients, reflecting the extent of myocardial injury. These findings highlight the importance of cardiac biomarkers and suggest the need for sex-specific diagnostic thresholds to improve accuracy and outcomes in women with AMI.

INTRODUCTION

Acute myocardial infarction (AMI) is a leading cause of morbidity and mortality worldwide, with differences in clinical presentation and outcomes between male and female patients. In the diagnostic evaluation of AMI, cardiac biomarkers play a pivotal role in identifying myocardial injury and guiding therapeutic interventions [1]. Among these biomarkers, Troponin I and Creatine Kinase-MB (CK-MB) are widely utilized due to their sensitivity and specificity in detecting myocardial damage.Troponin I, a highly specific marker of myocardial injury, is released into the bloodstream following cardiac myocyte necrosis. It provides a robust tool for early detection and risk stratification of AMI [2]. Similarly, CK-MB, an isoenzyme of creatine kinase predominantly found in cardiac muscle, is instrumental in diagnosing AMI, particularly in settings where troponin assays may not be available.Differences in AMI pathophysiology between patients with and without ST-elevation myocardial infarction (STEMI) are significant [3]. STEMI is characterized by complete coronary artery occlusion, leading to transmural myocardial infarction, whereas non-ST-elevation myocardial infarction (NSTEMI) often results from partial occlusion or microvascular dysfunction. These differences may influence the release kinetics and absolute levels of cardiac biomarkers like Troponin I and CK-MB [4].

Acute myocardial infarction remains a critical health concern globally, accounting for significant mortality and morbidity. In recent years, there has been growing awareness of the gender-specific differences in myocardial infarction presentation, diagnosis, and outcomes. Women with myocardial infarction often exhibit atypical symptoms, including fatigue, nausea, and generalized discomfort, which can delay

diagnosis and treatment [5]. These differences underscore the importance of optimizing diagnostic tools, particularly the use of cardiac biomarkers such as troponin I and creatine kinase-MB, in female patients.Cardiac biomarkers serve as crucial tools in the evaluation and management of myocardial infarction. Troponin I is a gold-standard biomarker for detecting myocardial injury due to its high sensitivity and specificity. Released into the bloodstream when cardiac myocytes are damaged, troponin I levels can detect even small areas of necrosis, making it invaluable for early diagnosis and risk stratification [6]. Furthermore, elevated troponin I levels provide prognostic insights, correlating with the extent of myocardial injury and the risk of adverse cardiac events.Creatine kinase-MB, an isoenzyme of creatine kinase found primarily in the myocardium, has historically been used as a diagnostic marker for myocardial infarction. Although its specificity is lower than that of troponin I due to potential elevations in skeletal muscle injury, creatine kinase-MB remains useful, particularly in settings where repeated measurements are required to assess reinfarction or where access to high-sensitivity troponin assays is limited. Creatine kinase-MB values also provide valuable temporal information about the onset of myocardial injury due to their distinct release kinetics [7].

The clinical manifestation and biomarker profiles of myocardial infarction vary significantly between ST-elevation myocardial infarction and non-ST-elevation myocardial infarction. ST-elevation myocardial infarction, resulting from complete coronary artery occlusion, leads to transmural myocardial infarction with pronounced and rapid elevations in cardiac biomarkers. In contrast, non-ST-elevation myocardial infarction is caused by partial coronary obstruction or microvascular dysfunction, leading to subendocardial ischemia [8]. This difference in pathophysiology affects the release kinetics and peak levels of biomarkers such as troponin I and creatine kinase-MB, necessitating careful interpretation based on the type of myocardial infarction.In female patients, the diagnostic and prognostic roles of troponin I and creatine kinase-MB are particularly relevant [9]. Women with myocardial infarction are more likely to present with non-ST-elevation myocardial infarction than ST-elevation myocardial infarction and are at a higher risk of delayed diagnosis due to atypical symptoms. Additionally, evidence suggests that sex-specific differences in myocardial mass, coronary anatomy, and hormonal influences may alter the release kinetics and absolute values of these biomarkers [10]. For example, lower myocardial mass in women may result in lower peak biomarker levels despite similar degrees of myocardial injury compared to men. Furthermore, the standard cutoff values for troponin I and creatine kinase-MB, often derived from predominantly male cohorts, may not accurately reflect the threshold for diagnosing myocardial infarction in women, highlighting the need for tailored diagnostic criteria [11].

Objective

The main objective of the study is to find the troponin I and CK-MB values in acute myocardial infarction among female patients with and without ST-elevation.

Methodology

This prospective, observational study was conducted and data were collected from 120 patients. Female patients presenting with acute chest pain or symptoms suggestive of acute myocardial infarction were screened for inclusion. Patients were enrolled after obtaining informed consent.

Inclusion Criteria

1. Female patients aged 18 years or older.
2. Confirmed diagnosis of acute myocardial infarction based on clinical presentation, electrocardiographic findings, and biomarker levels.
3. Patients with either STEMI or NSTEMI as classified by the presence or absence of ST-segment elevation on electrocardiography.

Exclusion Criteria

1. Patients with previous myocardial infarction within the past six weeks.
2. Presence of conditions that could confound biomarker levels, such as severe renal dysfunction or recent skeletal muscle injury.
3. Incomplete or missing clinical or laboratory data.

Data collection

The 120 female patients were divided into two groups based on their clinical and electrocardiographic presentation:

- **Group A (STEMI):** 60 patients diagnosed with STEMI.
- **Group B (NSTEMI):** 60 patients diagnosed with NSTEMI.

Detailed clinical history, including symptom onset, risk factors, and comorbidities, was obtained from all participants. A thorough physical examination and 12-lead electrocardiography were performed at presentation to differentiate STEMI and NSTEMI.

Blood samples were collected at presentation and analyzed for:

1. Troponin I levels using a high-sensitivity immunoassay.
2. Creatine kinase-MB levels using an enzyme immunoassay.

Serial measurements of biomarkers were performed at baseline (on admission), 6 hours, and 12 hours to assess peak values and kinetics.

Statistical Analysis

Data were analyzed using SPSS v16. Continuous variables, such as troponin I and creatine kinase-MB

levels, were expressed as mean ± standard deviation. Comparative analyses between the STEMI and NSTEMI groups were performed using independent t-tests for normally distributed variables and Mann-Whitney U tests for non-normally distributed variables. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 120 female patients diagnosed with acute myocardial infarction were included in the study. Of these, 60 patients presented with STEMI (Group A), and 60 patients presented with NSTEMI (Group B). The mean age was slightly lower in the STEMI group (55 ± 10 years) compared to the NSTEMI group (58 ± 8 years). Hypertension and diabetes mellitus were common in both groups, with slightly higher prevalence in the NSTEMI group (50% and 42%, respectively) than in the STEMI group (45% and 40%, respectively). Smoking history was comparable between the groups, affecting 30% of STEMI patients and 28% of NSTEMI patients. These similarities indicate comparable baseline risk profiles across both groups.

Table 1: Baseline Characteristics of the Study Population

Characteristic	STEMI (Group A) (n=60)	NSTEMI (Group B) (n=60)
Mean Age (years)	55 ± 10	58 ± 8
Hypertension (%)	45%	50%
Diabetes Mellitus (%)	40%	42%
Smoking History (%)	30%	28%

The results indicate that troponin I levels were significantly higher in the STEMI group compared to the NSTEMI group at all time points ($p < 0.01$). At admission, mean troponin I levels were 8.5 ± 1.2 ng/mL in STEMI patients, compared to 3.4 ± 0.9 ng/mL in NSTEMI patients. The levels peaked at 6 hours, with STEMI patients reaching 12.8 ± 2.1 ng/mL and NSTEMI patients 5.2 ± 1.3 ng/mL. By 12 hours, levels slightly declined but remained higher in STEMI patients (11.0 ± 1.8 ng/mL) than in NSTEMI patients (4.8 ± 1.0 ng/mL).

Table 2: Troponin I Levels (ng/mL)

Time Point	STEMI (Group A)	NSTEMI (Group B)	p-value
At Admission	8.5 ± 1.2	3.4 ± 0.9	< 0.01
At 6 Hours	12.8 ± 2.1	5.2 ± 1.3	< 0.01
At 12 Hours	11.0 ± 1.8	4.8 ± 1.0	< 0.01

The analysis of creatine kinase-MB levels showed significantly higher values in the STEMI group compared to the NSTEMI group at all measured time points ($p < 0.01$). At admission, the mean creatine kinase-MB levels in STEMI patients were 36 ± 5 U/L, while NSTEMI patients had 18 ± 4 U/L. Peak levels occurred at 6 hours, with STEMI patients reaching 68 ± 8 U/L compared to 28 ± 6 U/L in NSTEMI patients. By 12 hours, levels declined but remained higher in the STEMI group (52 ± 7 U/L) compared to the NSTEMI group (24 ± 5 U/L).

Table 3: Creatine Kinase-MB Levels (U/L)

Time Point	STEMI (Group A)	NSTEMI (Group B)	p-value
At Admission	36 ± 5	18 ± 4	< 0.01
At 6 Hours	68 ± 8	28 ± 6	< 0.01
At 12 Hours	52 ± 7	24 ± 5	< 0.01

The comparison of peak biomarker levels demonstrated significantly higher values in the STEMI group than the NSTEMI group ($p < 0.01$). Peak troponin I levels were 12.8 ± 2.1 ng/mL in STEMI patients compared to 5.2 ± 1.3 ng/mL in NSTEMI patients. Similarly, peak creatine kinase-MB levels were markedly elevated in the STEMI group (68 ± 8 U/L) compared to the NSTEMI group (28 ± 6 U/L).

Table 4: Summary of Key Findings

Parameter	STEMI (Group A)	NSTEMI (Group B)	p-value
Peak Troponin I (ng/mL)	12.8 ± 2.1	5.2 ± 1.3	< 0.01
Peak Creatine Kinase-MB (U/L)	68 ± 8	28 ± 6	< 0.01
Time to Peak Biomarker (hrs)	6	6	Not Significant

DISCUSSION

This study provides valuable insights into the levels of troponin I and creatine kinase-MB among female patients with acute myocardial infarction, highlighting differences between those presenting with STEMI and NSTEMI. The findings underscore the importance of cardiac biomarkers in diagnosing and differentiating myocardial infarction subtypes, particularly in female

patients who may present with atypical symptoms [12].The results show that both troponin I and creatine kinase-MB levels were significantly higher in STEMI patients compared to NSTEMI patients at all time points. This observation aligns with the pathophysiological differences between the two conditions. STEMI, caused by complete coronary artery occlusion, results in extensive transmural myocardial necrosis, leading to a more substantial and rapid release of biomarkers. In contrast, NSTEMI, associated with partial occlusion or microvascular dysfunction, typically involves subendocardial damage, resulting in a slower and less pronounced biomarker elevation [13] .

The peak levels of troponin I and creatine kinase-MB in STEMI patients were almost double those observed in NSTEMI patients. This significant difference reinforces the role of these biomarkers in distinguishing between the two myocardial infarction subtypes. Additionally, the temporal patterns revealed that the time to peak biomarker levels was similar in both groups, suggesting that while the extent of myocardial injury differs, the kinetics of biomarker release may remain consistent across subtypes[14].The study highlights the critical role of biomarker profiling in female patients, who often present with non-classical symptoms of myocardial infarction. Women are more likely to be underdiagnosed or experience delayed diagnosis, especially in NSTEMI cases where electrocardiographic findings may not be definitive. In such scenarios, reliance on sensitive biomarkers like troponin I is essential for timely and accurate diagnosis [15].

Moreover, the lower myocardial mass in women compared to men may influence biomarker release patterns. This study supports the need for sex-specific reference ranges for troponin I and creatine kinase-MB to enhance diagnostic accuracy in women [16]. The observed differences in biomarker levels also suggest that tailored diagnostic algorithms incorporating clinical, electrocardiographic, and biomarker data are necessary to improve outcomes for female patients with acute myocardial infarction.

CONCLUSION

It is concluded that troponin I and creatine kinase-MB levels are significantly higher in female patients with STEMI compared to those with NSTEMI, reflecting the greater extent of myocardial injury in STEMI. These biomarkers provide critical diagnostic and prognostic information, particularly in women who may present with atypical symptoms. Tailored diagnostic approaches incorporating sex-specific biomarker thresholds are essential to improve accuracy and outcomes in this population.

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