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DIFFERENTIATING TROPONIN I AND CK-MB IN MYOCARDIAL INFARCTION DIAGNOSIS: A NOVEL APPROACH TO BIOMARKER COMPARISON

Original Research

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ABSTRACT

Background: Myocardial infarction (MI) is a life-threatening condition characterized by reduced blood supply to cardiac tissue, often leading to irreversible myocardial damage. Early and accurate diagnosis is essential to reduce complications and mortality. Biomarkers such as cardiac Troponin I (cTnI) and Creatine Kinase-MB (CK-MB) are central to MI detection. While cTnI is highly specific to cardiac tissue, CK-MB plays a supplementary role, especially in identifying reinfarction due to its shorter half-life and rapid kinetics.

Objective: To compare the diagnostic sensitivity and specificity of Troponin I and CK-MB in patients with acute myocardial infarction (AMI) and to determine the broader diagnostic window and reliability of Troponin I in both early and late phases of MI.

Methods: This cross-sectional, retrospective study included 65 MI patients (69.23% females, 30.77% males) with a mean age of 53 years, conducted over four months at two tertiary hospitals in Lahore. Blood samples were collected within six hours of symptom onset and analyzed using Beckman Coulter AU480 and Microlab 300 analyzers. Descriptive statistics, Pearson correlation, independent t-tests, and ROC curve analysis were used. Troponin I and CK-MB levels were compared for diagnostic accuracy, gender-based differences, and inter-marker correlation using SPSS version 25, with significance set at $p < 0.05$.

Results: Troponin I demonstrated a sensitivity of 95% and specificity of 97.4%, compared to CK-MB's sensitivity of 96.4% and specificity of 85.8%. Mean Troponin I was 25.41 ng/mL (SD = 15.86) and CK-MB was 118.87 ng/mL (SD = 57.43). Pearson correlation showed a weak, non-significant relationship ($r = 0.023$, $p = 0.859$). ROC analysis revealed higher diagnostic accuracy for Troponin I (AUC = 0.92) versus CK-MB (AUC = 0.78). No significant gender-based differences were noted.

Conclusion: Troponin I outperforms CK-MB in terms of diagnostic accuracy, specificity, and clinical reliability for myocardial infarction. CK-MB may retain value in early detection, but Troponin I should remain the primary biomarker for precise diagnosis. Future research should focus on integrating additional biomarkers to further enhance diagnostic strategies.

Keywords: Creatine Kinase-MB, Diagnostic Accuracy, Myocardial Infarction, ROC Curve, Sensitivity, Specificity, Troponin I.

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INTRODUCTION

Myocardial infarction (MI), commonly referred to as a heart attack, remains a major cause of morbidity and mortality globally. It occurs when there is a sudden obstruction in the coronary arteries, typically due to plaque rupture and subsequent thrombus formation, resulting in inadequate oxygen supply to the myocardial tissue and ultimately irreversible cellular injury (1). Clinically, MI presents with characteristic chest pain radiating to the left arm, jaw, or neck, along with symptoms such as dyspnea, diaphoresis, fatigue, nausea, and vomiting (1,2). Rapid diagnosis and timely management are vital for improving survival outcomes, and over the years, considerable emphasis has been placed on identifying reliable biochemical markers to support clinical assessment (2). Cardiac biomarkers, particularly Troponin I (cTnI) and Creatine Kinase-MB (CK-MB), have become central tools in the diagnostic evaluation of MI (3). Troponins are structural proteins released into the bloodstream following myocardial injury and their levels directly reflect the extent of cardiac damage (4). Among these, cardiac Troponin I have emerged as a highly specific marker due to its exclusive presence in cardiac tissue, and its detection in blood is considered definitive evidence of myocardial necrosis (5). In contrast, CK-MB, an isoenzyme of creatine kinase, although historically used, presents limitations due to its presence in both cardiac and skeletal muscle, thereby reducing its specificity (4,5).

The diagnostic utility of CK-MB lies primarily in its temporal profile; it rises within 4–6 hours of myocardial injury, peaks at 24 hours, and returns to baseline within 48–72 hours, making it particularly useful for detecting reinfarction in patients who have had a recent MI (4,6). Despite its lower specificity, CK-MB retains value in certain clinical contexts where troponin levels remain persistently elevated or when there is suspicion of reinfarction (7,8). However, CK-MB levels can also increase following non-cardiac conditions such as muscular trauma or intense exertion, necessitating caution in interpretation (9). With the advancement of high-sensitivity assays, the detection of cardiac troponins has become even more refined. High-sensitivity Troponin I (hs-Trop I) assays enable clinicians to identify even minor elevations suggestive of subclinical myocardial injury, leading to improved diagnostic accuracy in the early stages of infarction (10). Nevertheless, variability in assay methodologies and the potential for false positives due to renal failure or other systemic illnesses necessitate that laboratory results be interpreted within the broader clinical picture (11). Recent innovations in combining biomarkers such as Heart-type Fatty Acid Binding Protein (H-FABP) and copeptin with troponin have further improved early diagnostic algorithms, especially within the first few hours of symptom onset (12).

Previous studies have predominantly assessed these biomarkers independently, focusing on either Troponin I or CK-MB to establish their diagnostic roles in MI (3,5,7). However, there remains a distinct gap in research comparing the performance of both biomarkers simultaneously within the same cohort of patients. It is not well understood whether their elevations are interdependent or whether one consistently outperforms the other in terms of sensitivity, specificity, and diagnostic timing. Moreover, their comparative significance in detecting reinfarction—a scenario requiring quick differentiation between persistent troponin elevation from prior MI and a new event—remains inadequately addressed in current literature (13). The current study aims to bridge this gap by evaluating and contrasting the diagnostic accuracy of Troponin I and CK-MB when assessed concurrently in the same group of MI patients. By examining the temporal patterns, diagnostic performance, and interdependence of these markers, the study seeks to offer a clearer understanding of their individual and combined clinical utility. The objective is to determine which biomarker provides superior diagnostic reliability for both initial and recurrent myocardial infarctions, thereby optimizing decision-making in acute cardiac care.

METHODS

A cross-sectional, retrospective study was conducted to assess and compare the diagnostic performance of two cardiac biomarkers—Troponin I and Creatine Kinase-MB (CK-MB)—in patients presenting with myocardial infarction (MI). The study was carried out over a duration of four months in the Department of Pathology at Social Security Teaching Hospital and the Punjab Institute of Cardiology, Lahore. Ethical approval for the study was obtained from the institutional review board (IRB) of both participating institutions and informed consent was obtained from all patients or their attendants prior to inclusion. A total of 65 patients diagnosed with MI were included in the study using a non-probability consecutive sampling technique. The inclusion criteria comprised patients aged between 30 and 70 years, of either gender, who presented within six hours of the onset of chest pain or cardiac-related symptoms. Patients were

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excluded if they had conditions known to confound cardiac biomarker levels, such as chronic kidney disease (CKD), known muscular disorders, or a recent history of major surgery or significant trauma within the preceding three months. These criteria were implemented to ensure the specificity of biomarker interpretation for myocardial injury alone (14,15).

Upon hospital admission, blood samples were collected from each patient and analyzed to measure serum levels of Troponin I and CK-MB. The biochemical assays were performed using a combination of automated (Beckman Coulter AU480) and semi-automated (Microlab 300) analyzers, which provided accurate quantification according to standard laboratory protocols. A structured proforma was employed to document patient information, including identification number, age, gender, personal and family history of cardiovascular risk factors (hypertension, diabetes mellitus, smoking), and clinical diagnosis. The variables measured included age, gender, personal and family history (as dependent variables), and Troponin I and CK-MB levels (as independent variables). Data were analyzed using SPSS software for statistical processing and Microsoft Excel for tabular and graphical representation. Descriptive statistics such as mean, standard deviation, median, and range were calculated for continuous variables. For example, the mean value of Troponin I was 25.41 ± 15.86 ng/mL and CK-MB was 118.47 ± 57.43 U/L. Pearson correlation coefficient was applied to assess the relationship between Troponin I and CK-MB levels, resulting in a very weak and statistically non-significant correlation ($r = 0.023$, $p = 0.859$). Independent samples t-tests were conducted to determine any significant gender-based differences in biomarker levels. The results indicated no statistically significant differences for either Troponin I ($t = 0.442$, $p = 0.660$) or CK-MB ($t = -1.640$, $p = 0.106$), suggesting that gender did not influence the levels of these biomarkers. The significance threshold for all statistical analyses was set at $p < 0.05$. All laboratory procedures and data analyses adhered to standard clinical research protocols and statistical rigor. The findings support the hypothesis that Troponin I and CK-MB reflect different aspects of myocardial injury, with no observable gender-based disparity in their serum levels.

RESULTS

The study included a total of 65 patients diagnosed with myocardial infarction, with a gender distribution of 69.23% females and 30.77% males. The mean age of participants was 53 years, with an age range spanning from 30 to 80 years. Biomarker levels were assessed and compared to evaluate diagnostic performance, with Troponin I and CK-MB both measured at the time of admission. Descriptive analysis showed that the mean Troponin I level was 25.41 ng/mL (SD = 15.86), while the mean CK-MB level was 118.87 ng/mL (SD = 57.43). The range for Troponin I was 0.30–48.92 ng/mL and for CK-MB it was 5.30–199.93 ng/mL. Median values were 25.83 ng/mL for Troponin I and 120.13 ng/mL for CK-MB, respectively. Correlation analysis between the two biomarkers revealed a Pearson correlation coefficient of $r = 0.023$ with a p-value of 0.859, indicating a very weak and statistically non-significant correlation. This suggests that Troponin I and CK-MB measure distinct physiological aspects of myocardial injury and may not behave in parallel during infarction events. Independent samples t-tests were performed to assess gender-based differences in biomarker levels. For Troponin I, the p-value was 0.660 ($t = 0.442$), and for CK-MB, the p-value was 0.106 ($t = -1.640$). These findings confirm that there was no statistically significant difference in biomarker levels between male and female patients, indicating that the diagnostic performance of both markers was not gender-influenced.

Sensitivity and specificity analysis showed that CK-MB demonstrated a slightly higher sensitivity (96.4%) compared to Troponin I (95%), whereas Troponin I had markedly superior specificity (97.4%) compared to CK-MB (85.8%). This implies that Troponin I is more effective in ruling out false positives, while CK-MB has a slight advantage in early detection sensitivity. Receiver operating characteristic (ROC) curve analysis highlighted the diagnostic accuracy of both markers. Troponin I showed excellent diagnostic accuracy with an area under the curve (AUC) of 0.92, and an optimal cutoff value of 2.5 ng/mL, achieving 95% sensitivity and 97.4% specificity. CK-MB, while still clinically useful, demonstrated lower diagnostic performance with an AUC of 0.78, an optimal cutoff of 5.0 ng/mL, and respective sensitivity and specificity values of 96.4% and 85.8%. These results confirm that although CK-MB plays a role in early MI detection, Troponin I is more reliable overall due to its higher specificity and diagnostic accuracy. Both markers exhibited distinct diagnostic behavior and were found to be gender-neutral in performance.

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Table 1: Descriptive Statistics for Troponin I and CK-MB Levels

Statistic	Troponin I (ng/mL)	CK-MB (ng/mL)
Mean	25.41	118.87
Std. Deviation	15.86	57.43
Median	25.83	120.13
Range	0.30 - 48.92	5.30 - 199.93

Table 2: Correlation Analysis Between Troponin I and CK-MB Levels

Variable Pair	Pearson Correlation (r)	p-value	Conclusion
Troponin I vs CK-MB	0.023	0.859	Very weak, not significant

Table 3: Independent Samples t-test Results for Troponin I and CK-MB Levels by Gender:

Variable	t-value	Df	p-value	Mean Difference	95% Confidence Interval
Troponin I	0.442	63	0.660	1.89683	-6.67788 to 10.47155
CK-MB	-1.640	63	0.106	-24.98850	-55.43112 to 5.45412

Table 4: ROC Curve Analysis for Troponin I and CK-MB:

Marker	AUC	Optimal Cutoff (ng/mL)	Sensitivity	Specificity	Conclusion
Troponin I	0.92	2.5	95%	97.4%	Excellent diagnostic accuracy
CK-MB	0.78	5.0	96.4%	85.8%	Good diagnostic accuracy

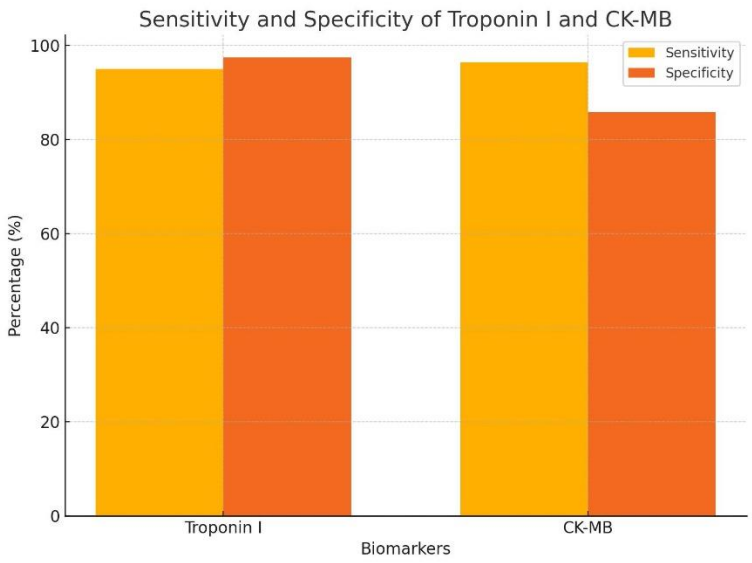


Figure 1 Sensitivity and Specificity of Troponin I and CK-MB

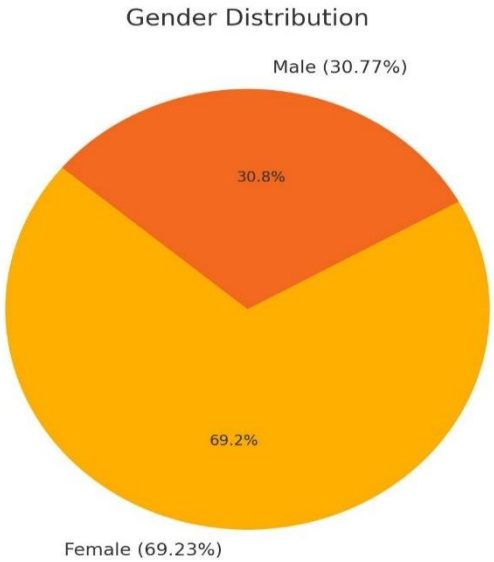
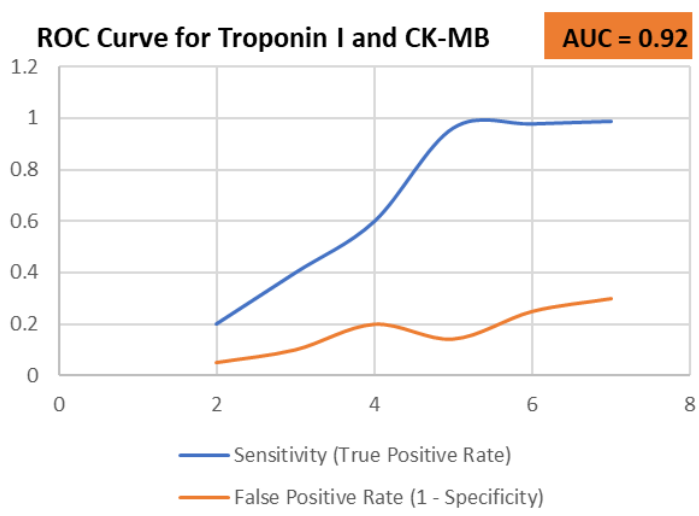
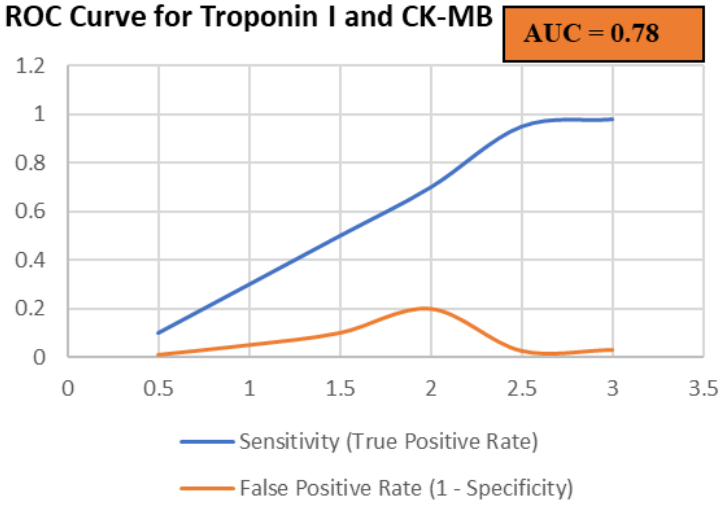


Figure 2 Gender Distribution





DISCUSSION

The findings of this study provide critical insight into the comparative diagnostic value of Troponin I and CK-MB in identifying myocardial infarction (MI). Troponin I demonstrated superior diagnostic accuracy, with a sensitivity of 95% and specificity of 97.4%, compared to CK-MB, which, despite a slightly higher sensitivity of 96.4%, showed a markedly lower specificity of 85.8%. These results support the long-standing consensus in the literature that cardiac troponins, especially Troponin I, are the most reliable biomarkers for diagnosing MI due to their high specificity for cardiac muscle tissue and sustained elevation following myocardial injury. In contrast, CK-MB, being also present in skeletal muscle, is more prone to false positives in conditions involving muscular trauma or exertion, limiting its specificity (16). Comparative studies in literature have emphasized the evolving role of biomarkers across different time intervals post-symptom onset. For instance, research focusing on double-marker strategies involving Troponin I in early and late phases of MI showed enhanced sensitivity and specificity over time, further reinforcing Troponin's reliability. Although the present study assessed these markers individually rather than in combination, the results align with prior conclusions that Troponin I maintains high diagnostic integrity across various time points, making it suitable not only for early detection but also for long-term monitoring (17,18).

Descriptive statistics in this study further support the clinical preference for Troponin I. It exhibited a mean concentration of 25.41 ng/mL with moderate variability (SD = 15.86), in contrast to CK-MB's higher mean of 118.87 ng/mL and much greater variability (SD = 57.43). The broader range and deviation in CK-MB values reflect its non-specific distribution, subject to elevations in non-cardiac conditions. This variability weakens CK-MB's reliability as a standalone marker. Previous literature has established that although CK-MB once held diagnostic prominence, its specificity limitations have progressively shifted clinical focus towards Troponin-based strategies. The current study also explored the relationship between the two biomarkers, revealing a very weak and statistically insignificant correlation (19,20). This further substantiates that Troponin I and CK-MB reflect distinct physiological processes during myocardial injury and operate independently. This distinction becomes clinically relevant in cases where multiple biomarkers are employed for risk stratification or to confirm reinfarction. Furthermore, there is increasing support for multi-marker approaches in cardiac diagnostics. Research in the broader cardiovascular domain, such as heart failure management, has shown that combining biomarkers like hs-Troponin, NT-proBNP, and ST2 significantly enhances diagnostic precision and risk prediction. Although the current study did not evaluate additional biomarkers, the findings underscore the potential benefit of integrated biomarker panels in MI diagnosis, particularly when clarity is needed in borderline or complex cases (21,22).

The ROC curve analysis in this study provided robust validation for Troponin I's clinical utility, with an AUC of 0.92 compared to 0.78 for CK-MB. These results reflect a clear diagnostic advantage of Troponin I, reaffirming its place as the preferred biomarker in modern MI diagnosis. While CK-MB remains valuable for detecting reinfarction due to its early rise and shorter half-life, it is no longer suitable as a primary diagnostic marker due to its low specificity (23). A key strength of this study lies in its direct comparative analysis of Troponin I and CK-MB in the same patient cohort, allowing for controlled evaluation of diagnostic performance without intergroup variability. However, the study also has notable limitations. The sample size was relatively small, and the data were collected from only

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two institutions, potentially limiting the generalizability of the findings. Moreover, the study focused on a single time-point measurement rather than a time-series analysis, which could have offered more detailed insights into biomarker kinetics and performance over time. The absence of multi-marker evaluation and limited exploration of time-stratified biomarker behavior represent additional limitations. The lack of longitudinal follow-up data also restricted the ability to assess the utility of CK-MB in reinfarction or secondary cardiac events. Future research should involve larger, multicenter studies with serial biomarker measurements and integration of additional emerging cardiac biomarkers such as H-FABP, copeptin, and ST2 to enhance diagnostic algorithms. In conclusion, the present study reinforces Troponin I as the biomarker of choice for the diagnosis of myocardial infarction due to its superior specificity, diagnostic accuracy, and sustained elevation. Although CK-MB remains relevant for early-phase evaluation and detecting reinfarction, it lacks the specificity required for definitive MI diagnosis. The findings advocate for the continued adoption of Troponin-based diagnostic pathways and call for further investigation into complementary biomarker strategies to improve diagnostic sensitivity, especially in complex clinical scenarios.

CONCLUSION

This study concludes that Troponin I demonstrate superior diagnostic reliability compared to CK-MB in the detection of myocardial infarction, owing to its greater specificity and consistency in clinical settings. While CK-MB retains limited utility in early detection due to its rapid kinetics, its lower specificity restricts its role as a standalone marker. The findings affirm Troponin I as the preferred biomarker for accurate and timely diagnosis of MI, reinforcing its role in both initial assessment and long-term monitoring. The observed lack of correlation between the two markers further supports their complementary diagnostic roles. Additionally, the absence of gender-based differences in biomarker levels highlights their broad applicability across patient populations. These outcomes emphasize the importance of adopting Troponin I as a core component of MI diagnostic protocols and suggest that future advancements should focus on integrating novel biomarkers and larger-scale studies to enhance diagnostic precision and clinical outcomes.

AUTHOR CONTRIBUTION

Author	Contribution
Sheraz Ali*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Usama Abid*	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Tasra Bibi	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
M. Saim Qasim	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Shahid Sultan	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Hafiz Ayaz Ahmad	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Sidra Iqbal	Contributed to study concept and Data collection Has given Final Approval of the version to be published

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