Research Article

Association Between Oxidative Stress Biomarkers And Cardiac Injury Markers In Patients With Acute Myocardial Infarction: A Prospective Observational Study

Roshan Kumar Sah[1], Dr. Zunjarrao G. Badade[2*], Dr. Shilpa Kadam[3], Dr. Dattatray Bhusare[4]

[1]Ph.D Scholar, Department of Biochemistry, MGM Medical College, Kamothe, Navi Mumbai.
[2*]Professor, Department of Biochemistry, MGM Medical College, Kamothe, Navi Mumbai. badadezg@gmail.com
[3] Professor, Department of Cardiology, MGM Medical College, Kamothe, Navi Mumbai,
[4] Professor & HOD, Department of Emergency Medicine and Critical Care, MGM Medical College, Kamothe, Navi Mumbai,
Corresponding Author- Dr. Zunjarrao G. Badade, Professor, Department of Biochemistry, MGM Medical College, Kamothe, Navi Mumba, Email ID: badadezg@gmail.com

Abstract

Background: Acute myocardial infarction (AMI) is a major cause of morbidity and mortality worldwide. In addition to myocardial injury, oxidative stress plays an important role in the development and progression of AMI. Cardiac biomarkers such as Troponin-T and CK-MB are widely used for diagnosing myocardial injury, whereas malondialdehyde (MDA) and superoxide dismutase (SOD) reflect oxidative stress and antioxidant defense. However, the relationship between cardiac injury biomarkers and oxidative stress markers during the early phase of AMI remains unclear. **Objective:** To evaluate the relationship between oxidative stress biomarkers (MDA and SOD) and cardiac injury markers (Troponin-T and CK-MB) at the time of admission (At 0 hour) and At 12 hours after admission in patients with acute myocardial infarction.
Materials and Methods: This prospective observational study included 110 patients with confirmed acute myocardial infarction admitted to the Departments of Cardiology and Emergency Medicine at MGM Medical College and Hospital, Navi Mumbai, between August 2024 and April 2026. Blood samples were collected at the time of admission (At 0 hour) and again at 12 hours after admission. Troponin-T was measured by electrochemiluminescence immunoassay, CK-MB by the enzymatic kinetic method, MDA by the Kei Satoh method, and SOD activity by the Marklund and Marklund method. Changes in biomarker levels over time were analyzed using the paired Student's *t*-test, while Pearson's correlation analysis was performed to determine the relationship between oxidative stress and cardiac biomarkers. A p-value of <0.05 was considered statistically significant.
Results: Troponin-T and CK-MB levels increased significantly from at the time of admission to 12 hours after admission ($p < 0.0001$), indicating ongoing myocardial injury. In contrast, MDA levels decreased significantly, while SOD activity increased significantly over the same period ($p < 0.0001$), suggesting improvement in oxidative stress following treatment. Pearson correlation analysis showed no significant relationship between oxidative stress markers and cardiac biomarkers at either at the time of admission or at 12 hours after admission.
Conclusion: Acute myocardial infarction is associated with significant changes in both cardiac injury biomarkers and oxidative stress biomarkers during the first 12 hours after admission. However, the absence of significant correlations between these biomarkers suggests that oxidative stress and myocardial injury represent independent pathophysiological processes during the early phase of AMI. Assessing both cardiac and oxidative stress biomarkers may therefore provide complementary information for understanding disease progression and patient management.

Keywords: Acute myocardial infarction; Troponin-T; CK-MB; Oxidative stress; Malondialdehyde; Superoxide dismutase; Cardiac biomarkers; Prospective observational study.

1. Introduction

Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality worldwide despite significant advances in its diagnosis and management (1). Early diagnosis and timely risk assessment are essential for improving clinical outcomes and reducing complications in patients with AMI. Cardiac biomarkers have become indispensable tools for confirming myocardial injury and assessing disease severity during the acute phase of infarction (2).

Among the available cardiac biomarkers, cardiac troponins, particularly Troponin-T, are considered the gold standard for the diagnosis of myocardial infarction because of their high sensitivity and specificity for myocardial injury. Creatine kinase-MB (CK-MB) also continues to play an important role in evaluating myocardial damage, especially for monitoring the progression of infarction and detecting reinfarction (3,4). Serial measurement of these biomarkers provides valuable information regarding the extent of myocardial injury and assists clinicians in making appropriate therapeutic decisions (5).

In addition to myocardial necrosis, oxidative stress plays a crucial role in the pathogenesis of AMI. During ischemia and subsequent reperfusion, excessive production of reactive oxygen species (ROS) disrupts the balance between oxidants and antioxidants, resulting in oxidative damage to cardiomyocytes (6). This imbalance contributes to lipid peroxidation, inflammation, endothelial dysfunction, and further myocardial injury, thereby influencing the progression of the disease (7).

Malondialdehyde (MDA), a stable end product of lipid peroxidation, is widely used as a biomarker of oxidative stress. Elevated MDA levels indicate increased oxidative damage to cell membranes during acute myocardial infarction (8). Conversely, superoxide dismutase (SOD) is one of the major endogenous antioxidant enzymes that protects cells against oxidative injury by converting superoxide radicals into less harmful molecules. Changes in SOD activity reflect the body's antioxidant defense response during acute myocardial injury (7,8).

Lipid abnormalities are well-established risk factors for atherosclerosis and coronary artery disease. Evaluation of the lipid profile during the early phase of AMI provides important information regarding the patient's baseline cardiovascular risk and metabolic status (9). However, while the independent clinical significance of cardiac biomarkers and oxidative stress markers has been extensively investigated, limited information is available regarding their relationship during the early hours of acute myocardial infarction (10).

Therefore, the present study was undertaken to evaluate the relationship between oxidative stress biomarkers (MDA and SOD) and cardiac injury biomarkers (Troponin-T and CK-MB) in patients with acute myocardial infarction at the time of admission and at 12 hours after admission. Understanding this relationship may provide additional insights into the pathophysiological mechanisms of AMI and contribute to a more comprehensive biomarker-based assessment of affected patients.

2. Materials and Methods

2.1 Study Design

This prospective observational study was conducted in the Department of Biochemistry in collaboration with the Departments of Cardiology and Emergency Medicine at MGM Medical College and Hospital, Navi Mumbai, from August 2024 to April 2026. The study protocol was approved by the Institutional Ethics Committee and written informed consent was obtained from all participants before enrollment.

2.2 Study Population

A total of 110 patients aged 18-80 years diagnosed with acute myocardial infarction based on clinical presentation, electrocardiographic findings, and biochemical investigations were included. Patients with recent surgery, severe trauma, pregnancy, lactation, or medical conditions known to affect oxidative stress parameters were excluded.

2.3 Sample Collection

Peripheral venous blood samples were collected at two time points: immediately after hospital admission (At 0 hours) and At 12 hours after admission. Serum was separated by centrifugation and stored under appropriate conditions until biochemical analysis.

2.4 Biochemical Analysis

Serum Troponin-T levels were measured by an electrochemiluminescence immunoassay method, while CK-MB levels were determined by the enzymatic kinetic method. Serum MDA levels were estimated by the Kei Satoh method, while SOD activity was measured by Marklund and Marklund method.

2.5 Statistical Analysis

Data were expressed as mean ± standard deviation (SD). Differences in biomarker levels between at the time of admission and at 12 hours after admission were analysed using the paired Student's *t*-test. Pearson's correlation coefficient (r) was used to evaluate the relationship between oxidative stress biomarkers (MDA and SOD) and cardiac injury markers (Troponin-T and CK-MB) at both time points. A p-value of <0.05 was considered statistically significant.

3. Results

3.1 Changes in Cardiac and Oxidative Stress Biomarkers

Serial analysis demonstrated significant changes in both cardiac injury markers and oxidative stress biomarkers during the first 12 hours following admission. Serum Troponin-T increased significantly from 15.63 ± 2.54 ng/L at the time of admission to 30.90 ± 4.74 ng/L at 12 hours after admission ($p < 0.0001$). Similarly, CK-MB levels increased significantly from 33.33 ± 3.10 IU/L at the time of admission to 57.88 ± 5.07 IU/L at 12 hours after admission ($p < 0.0001$), indicating ongoing myocardial injury.

Among the oxidative stress biomarkers, serum MDA levels decreased significantly from 9.67 ± 0.92 nmol/mL at the time of admission to 6.44 ± 1.52 nmol/mL at 12 hours after admission ($p < 0.0001$). In contrast, SOD activity increased significantly from 83.40 ± 8.92 U/mL at the time of admission to 127.19 ± 16.57 U/mL at 12 hours after admission ($p < 0.0001$), suggesting improvement in antioxidant defence during the early phase after admission. (Table 1)

Table 1: The mean levels of cardiac biomarkers, including Troponin-T and CK-MB and oxidative stress biomarkers (MDA and SOD) At the time of admission (At 0 hour) and At 12 hours after Admission.

	At the time of admission (At 0 hour)	At 12 hours after Admission	P Value
Troponin-T (ng/L)	15.6327±2.5403	30.9009±4.7383	<0.0001
CK-MB (IU/L)	33.3279±3.1009	57.8753±5.0737	<0.0001
MDA (nmol/ml)	9.6652±0.9244	6.4385±1.5219	<0.0001
SOD (U/ml)	83.4033±8.9211	127.1918±16.5676	<0.0001

3.2 Correlation Between Oxidative Stress and Cardiac Injury Biomarkers

Pearson's correlation analysis was performed to determine the relationship between oxidative stress biomarkers (MDA and SOD) and cardiac injury markers (Troponin-T and CK-MB) at the time of admission and at 12 hours after admission.

Correlation between oxidative stress biomarkers and cardiac injury markers at the time of admission (At 0 hour)

At admission, MDA showed weak positive correlations with both Troponin-T (r = 0.061) and CK-MB (r = 0.043). SOD demonstrated a weak positive correlation with Troponin-T (r = 0.109) and a negligible negative correlation with CK-MB (r = –0.026). None of these correlations were statistically significant ($p > 0.05$). (Table 2)

Table 2. Correlation between oxidative stress biomarkers and cardiac injury markers at the time of admission (At 0 hour)

Pearson's correlation coefficient (r)	Troponin-T	CK-MB
MDA	0.061	0.043
SOD	0.109	–0.026

Correlation between oxidative stress biomarkers and cardiac injury markers at 12 hours after admission

At 12 hours after admission, MDA demonstrated weak negative correlations with both Troponin-T (r = –0.097) and CK-MB (r = –0.108). SOD showed a weak positive correlation with Troponin-T (r = 0.100) and a weak negative correlation with CK-MB (r = –0.099). Similar to the findings at admission, none of the observed correlations reached statistical significance ($p > 0.05$). (Table 3)

Table 3. Correlation between oxidative stress biomarkers and cardiac injury markers at 12 hours after admission

Pearson's correlation coefficient (r)	Troponin-T	CK-MB
MDA	–0.097	–0.108
SOD	0.100	–0.099

4. Discussion

The present prospective observational study evaluated the early changes in cardiac injury biomarkers and oxidative stress biomarkers in patients with acute myocardial infarction (AMI). Our findings demonstrated a significant increase in serum Troponin-T and CK-MB levels during the first 12 hours after admission, indicating the progressive release of myocardial proteins following acute ischemic injury. These observations support the established role of cardiac biomarkers in the diagnosis and early assessment of patients with AMI.

The significant rise in serum Troponin-T observed in the present study is consistent with the well-established diagnostic and prognostic value of cardiac troponins in acute myocardial infarction. Kocatürk et al. (2022) highlighted that a multi-biomarker approach improves prognostic assessment in patients with AMI and emphasized the importance of cardiac biomarkers in predicting clinical outcomes (11). Similarly, Toprak et al. (2024) evaluated a multi-biomarker panel in patients with suspected myocardial infarction and reported that integrating selected biomarkers with established clinical models improved prediction of major adverse cardiovascular events, reinforcing the value of biomarker-based risk stratification. These findings support our observation that serial measurement of cardiac biomarkers provides important information regarding the progression of myocardial injury during the early phase of infarction (12).

Likewise, Munir et al. emphasized the importance of biochemical cardiac markers in the early diagnosis and management of acute coronary syndrome and reported that serial assessment of these biomarkers improves diagnostic accuracy and facilitates timely clinical intervention (13). Shinde et al. (2023) also described the clinical utility of cardiac biomarkers and biosensor-based technologies for the rapid diagnosis of acute myocardial infarction, highlighting the importance of Troponin-T as a sensitive indicator of myocardial injury (14). These reports are in agreement with our findings, where Troponin-T levels increased significantly from admission to 12 hours, reflecting ongoing myocardial damage.

The present study also demonstrated a significant increase in CK-MB levels during the first 12 hours after admission. Although high-sensitivity cardiac troponins have become the preferred biomarkers for diagnosing AMI, CK-MB continues to provide valuable clinical information regarding infarct evolution. Rizvi et al. (2025) reviewed the physiological and clinical significance of cardiac biomarkers and confirmed that both Troponin-T and CK-MB remain important indicators of myocardial injury and prognosis (15). Kopec et al. (2017) further demonstrated that combining high-sensitivity cardiac troponin with additional biomarkers improves prediction of adverse cardiovascular outcomes, supporting the concept of a multimarker strategy for risk assessment (16). Horiuchi et al. (2020) also reported that biomarkers significantly improve the diagnosis and prognostic evaluation of myocardial infarction, particularly in patients with complex clinical presentations (17).

Our findings regarding CK-MB are consistent with previous studies. Al-Gani reported that CK-MB levels increase significantly following acute myocardial infarction and are closely associated with myocardial tissue damage (18). Similarly, Al-Hadi and Fox (2009) described CK-MB as a valuable biomarker for the early diagnosis and clinical management of acute coronary syndrome, particularly when interpreted together with cardiac troponins (19). Malhotra and Ahmed (2022) also demonstrated a positive association between CK-MB and myocardial infarction and highlighted its usefulness in assessing myocardial injury (20).

Further evidence supporting our findings comes from Sravanthi et al. (2024), who reported that combined evaluation of cardiac biomarkers improves the diagnostic assessment of patients with acute coronary syndrome (22). Likewise, Shah and Haridas (2007) observed a characteristic serial rise in cardiac marker enzymes during the first week after acute myocardial infarction, confirming the predictable release kinetics of CK-MB after myocardial injury (23). Tilea et al. (2021) reviewed the evolution of blood biomarkers in myocardial infarction and concluded that conventional biomarkers such as CK-MB continue to complement modern cardiac troponin assays in clinical practice (24). Similarly, Stătescu et al. (2022) emphasized that combining classical and contemporary cardiac biomarkers provides more comprehensive prognostic information than reliance on a single biomarker alone (25).

Overall, the significant increase in Troponin-T and CK-MB observed in the present study is in agreement with previously published evidence demonstrating that these biomarkers remain fundamental for the diagnosis, monitoring, and prognostic assessment of patients with acute myocardial infarction. Their serial evaluation during the early hours after admission reflects the progression of myocardial injury and supports their continued use as essential components of multimarker strategies for risk stratification in clinical practice.

Oxidative stress is a key mechanism involved in the pathogenesis of acute myocardial infarction (AMI). Excessive production of reactive oxygen species (ROS) during myocardial ischemia and subsequent reperfusion contributes to lipid peroxidation, endothelial dysfunction, inflammatory responses, and further myocardial injury. In the present study, serum malondialdehyde (MDA) levels were significantly elevated at admission and decreased markedly after 12 hours, while superoxide dismutase (SOD) activity increased significantly during the same

period. These findings indicate an initial increase in oxidative stress followed by partial restoration of antioxidant defense during the early phase of AMI.

Our observation of elevated MDA levels at admission is consistent with previous reports. Savović et al. (2023) demonstrated that oxidative stress biomarkers have important prognostic value in patients with ST-elevation and non-ST-elevation myocardial infarction and reported that increased oxidative stress is associated with disease severity and clinical outcomes (26). Similarly, Aladağ et al. (2021) reported significantly higher oxidant levels and reduced antioxidant capacity in patients with myocardial infarction compared with healthy controls, emphasizing the important role of oxidative stress in the pathophysiology of AMI (27). These findings are in agreement with our study, where elevated MDA levels at admission reflected increased lipid peroxidation during acute myocardial ischemia.

The significant reduction in MDA levels after 12 hours observed in our study may be attributed to early therapeutic intervention and gradual stabilization of oxidative status following admission**.** Helan et al. (2022**)** investigated the kinetics of oxidative stress biomarkers during acute systemic illness and demonstrated that oxidative stress markers change dynamically during the early phase of disease, reflecting alterations in redox homeostasis over time (28). Although their study was conducted in septic shock, the dynamic behaviour of oxidative stress biomarkers supports the concept that oxidative stress is not static but changes during the course of acute illness. Likewise, Ceylan and Demir (2025) reported that oxidative stress biomarkers, including lipid peroxidation products, were associated with the severity of coronary artery disease and acute myocardial infarction, further supporting the clinical significance of oxidative stress assessment in cardiovascular diseases (29).

In contrast to MDA, SOD activity increased significantly during the first 12 hours after admission. SOD is one of the most important endogenous antioxidant enzymes responsible for scavenging superoxide radicals generated during oxidative stress. The increase in SOD activity observed in the present study may represent activation of the body's antioxidant defence system in response to excessive free radical production during myocardial infarction. Sun et al. (2023) identified several oxidative stress-related biomarkers involved in the pathogenesis of acute myocardial infarction and highlighted the importance of antioxidant pathways in limiting myocardial injury and improving cellular recovery (30). Our findings support these observations and suggest that increased SOD activity reflects an adaptive protective response during the early phase of AMI.

Overall, the findings of the present study demonstrate that although both cardiac injury biomarkers and oxidative stress biomarkers undergo significant changes during the early phase of acute myocardial infarction, they do not exhibit a significant linear correlation. These results suggest that myocardial injury and oxidative stress represent complementary but independent pathophysiological processes. Consequently, incorporating both cardiac biomarkers and oxidative stress biomarkers into a multimarker assessment strategy may improve the understanding of disease progression and enhance early risk stratification in patients with acute myocardial infarction.

5. Conclusion

This prospective observational study demonstrated that acute myocardial infarction is associated with significant early changes in both cardiac injury biomarkers and oxidative stress biomarkers. During the first 12 hours after admission, serum Troponin-T and CK-MB levels increased significantly, indicating ongoing myocardial injury, whereas malondialdehyde (MDA) levels decreased and superoxide dismutase (SOD) activity increased significantly, reflecting dynamic alterations in oxidative stress and antioxidant defence.

Despite these significant temporal changes, no statistically significant correlation was observed between oxidative stress biomarkers (MDA and SOD) and cardiac injury markers (Troponin-T and CK-MB) at either at the time of admission or at 12 hours after admission. These findings suggest that myocardial injury and systemic oxidative stress are parallel but biologically independent processes during the early phase of acute myocardial infarction.

The present study highlights the importance of evaluating cardiac injury and oxidative stress biomarkers as complementary indicators rather than relying on a single biomarker pathway. Integrating these biomarkers may provide a more comprehensive understanding of the complex pathophysiological mechanisms underlying acute myocardial infarction and contribute to improved early clinical assessment and risk stratification.

6. References

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