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RELATIONSHIP BETWEEN HEART-TYPE FATTY ACID-BINDING PROTEIN AND OXIDATIVE STRESS BIOMARKERS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT
Background: Acute myocardial infarction (AMI) is one of the leading causes of morbidity and mortality worldwide. Early diagnosis is essential for timely intervention and improved clinical outcomes. Heart-type Fatty Acid-Binding Protein (H-FABP) is an early cardiac biomarker released rapidly after myocardial injury, while oxidative stress during AMI leads to increased lipid peroxidation, reflected by Malondialdehyde (MDA) and activation of antioxidant defense mechanisms, represented by Superoxide Dismutase (SOD). However, the relationship between H-FABP and oxidative stress markers remains incompletely understood. Objective: To evaluate the changes in H-FABP, MDA and SOD during the first 12 hours after hospital admission in patients with acute myocardial infarction and to determine the correlation between H-FABP and oxidative stress markers. Materials and Methods: 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. A total of 110 patients diagnosed with acute myocardial infarction based on clinical findings, electrocardiographic changes and biochemical parameters were enrolled after obtaining written informed consent. Blood samples were collected at admission (0 hour) and at 12 hours post-admission. H-FABP was estimated using the ELISA method. MDA levels were estimated by the Kei Satoh method, while SOD activity was determined by the Marklund and Marklund method. Statistical analysis was performed using paired Student's t-test and Pearson's correlation coefficient. $p < 0.05$ was considered statistically significant. Results: Serum H-FABP levels increased significantly at 12 hours of admission compared with 0 hour (at the time of admission) ($p < 0.0001$). In contrast, MDA levels decreased significantly, whereas SOD activity increased significantly at 12 hours of admission compared with 0 hour (at the time of admission) ($p < 0.0001$). Correlation analysis demonstrated no significant association between serum H-FABP and either MDA or SOD At 0 hour (at the time of admission) or At 12 hours of admission (all $p > 0.05$). Conclusion: Patients with acute myocardial infarction demonstrated significant temporal changes in H-FABP and oxidative stress markers. However, H-FABP showed no significant correlation with MDA or SOD, suggesting that myocardial injury and oxidative stress represent independent biological processes. A multi-biomarker approach may improve early diagnosis and risk stratification.

KEYWORDS: Acute myocardial infarction, H-FABP, Malondialdehyde, Superoxide Dismutase, Oxidative stress, Cardiac biomarkers.

1. INTRODUCTION

Acute myocardial infarction (AMI) remains one of the leading causes of mortality and morbidity worldwide and represents a major cardiovascular health challenge (1). It occurs due to sudden occlusion of a coronary artery, resulting in myocardial ischemia and irreversible cardiac muscle injury if timely reperfusion is not achieved (2). Early diagnosis and prompt treatment are essential for reducing infarct size and improving patient survival (3). Conventional cardiac biomarkers require several hours to become detectable after myocardial injury, creating a diagnostic gap during the early phase of AMI (4).

Heart-type Fatty Acid-Binding Protein (H-FABP) is a small cytoplasmic protein with a molecular weight of approximately 15 kDa that is rapidly released into the circulation following myocardial cell injury (5). Because of its rapid release kinetics, H-FABP has emerged as a valuable biomarker for the early diagnosis and prognostic assessment of acute myocardial infarction (6).

Myocardial ischemia is also associated with excessive production of reactive oxygen species (ROS), leading to oxidative stress and cellular damage (7). Lipid peroxidation caused by ROS produces malondialdehyde (MDA), which is widely used as a marker of oxidative damage (8). In response to oxidative stress, antioxidant enzymes such as superoxide dismutase (SOD) are activated to neutralize free radicals and protect myocardial cells from further injury (9).

Although H-FABP is an established marker of myocardial injury and MDA and SOD are recognized indicators of oxidative stress, their relationship during the acute phase of myocardial infarction has not been fully explored (10). Understanding this relationship may improve early diagnosis, prognostic assessment and risk stratification of patients with AMI (11). Therefore, the present study aimed to evaluate the changes in serum H-FABP, MDA and SOD during the first 12 hours after admission and to determine the correlation between H-FABP and oxidative stress markers (12).

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 H-FABP concentrations were measured using the Enzyme-Linked Immunosorbent Assay (ELISA) 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). Paired Student's t-test was used to compare biomarker levels between at the time of admission (At 0 hour) and At 12 hours of Admission. Pearson's correlation coefficient (r) was used to assess the relationship between H-FABP and oxidative stress markers. A p-value <0.05 was considered statistically significant.

3. RESULTS

3.1 Changes in H-FABP and Oxidative Stress Biomarkers from at the time of Admission (At 0 hour) to at 12 Hours after admission

A total of 110 patients with acute myocardial infarction were included in the study. Significant changes were observed in serum H-FABP and oxidative stress biomarkers during the first 12 hours after hospital admission. Serum H-FABP levels increased significantly from 32.01 ± 10.12 ng/mL at the time of admission (0 hour) to 142.04 ± 17.90 ng/mL at 12 hours after admission ($p < 0.0001$), indicating progressive myocardial injury during the early phase of acute myocardial infarction.

Significant changes were also observed in oxidative stress biomarkers. Serum MDA levels decreased significantly from 9.66 ± 0.92 nmol/mL at the time of admission (0 hour) to 6.43 ± 1.52 nmol/mL at 12 hours after admission ($p < 0.0001$), suggesting a reduction in oxidative stress following treatment. In contrast, Serum SOD activity increased significantly from 83.40 ± 8.92 U/mL at the time of admission (0 hour) to 127.19 ± 16.56 U/mL at 12 hours after admission ($p < 0.0001$), indicating an improvement in the body's antioxidant defense system during the observation period (Table 1).

Table 1: The mean levels of H-FABP and Oxidative Stress Biomarkers 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
HFABP (ng/mL)	32.0138±10.12	142.0448±17.9043	<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 H-FABP and Oxidative Stress Biomarkers

Correlation analysis was performed to evaluate the relationship between serum H-FABP and oxidative stress biomarkers (MDA and SOD) at the time of admission and At 12 hours after hospital admission.

Correlation Between H-FABP and Oxidative Stress Biomarkers at the time of Admission (At 0 Hour)

At the time of admission (At 0 hour), H-FABP showed a weak positive correlation with MDA ($r = 0.054$) and SOD ($r = 0.176$); however, neither correlation was statistically significant ($p > 0.05$) (Table 2).

Table 2. Correlation Between H-FABP and Oxidative Stress Biomarkers at the time of Admission (At 0 Hour)

Oxidative Stress Biomarker	Pearson's Correlation with HFABP (r)	P value
MDA (nmol/mL)	0.054	>0.05
SOD (U/mL)	0.176	>0.05

Correlation Between H-FABP and Oxidative Stress Biomarkers at 12 Hours after admission.
At 12 hours of admission, H-FABP showed a weak negative correlation with MDA ($r = -0.131$) and a weak positive correlation with SOD ($r = 0.102$). These correlations were also not statistically significant ($p > 0.05$) (Table 3).

Table 3. Correlation Between H-FABP and Oxidative Stress Biomarkers at 12 Hours after admission.

Oxidative Stress Biomarker	Pearson's Correlation with HFABP (r)	P value
MDA (nmol/mL)	−0.131	>0.05
SOD (U/mL)	0.102	>0.05

Overall, although significant temporal changes were observed in H-FABP, MDA and SOD levels during the first 12 hours, no significant correlation was found between H-FABP and the oxidative stress biomarkers at either time point, suggesting that myocardial injury and oxidative stress may represent independent biological processes in patients with acute myocardial infarction.

4. DISCUSSION

The present study evaluated the relationship between Heart-type Fatty Acid-Binding Protein (H-FABP) and the oxidative stress markers Malondialdehyde (MDA) and Superoxide Dismutase (SOD) in patients with acute myocardial infarction (AMI). The results demonstrated significant changes in all three biomarkers during the first 12 hours after admission. However, despite these marked biochemical changes, no statistically significant correlation was observed between H-FABP and oxidative stress markers, suggesting that myocardial injury and oxidative stress represent different pathophysiological processes.

In the present study, serum H-FABP levels increased significantly during the first 12 hours after admission, confirming its role as an early biomarker of myocardial injury. H-FABP is a low-molecular-weight cytoplasmic protein that is rapidly released into the circulation following myocardial ischemia and has been recognized as one of the earliest indicators of myocardial damage (13). Previous studies have demonstrated that H-FABP provides higher diagnostic sensitivity during the early hours of acute myocardial infarction than conventional cardiac biomarkers and is useful for early diagnosis and risk assessment (14). Güneş et al. further reported significant alterations in H-FABP concentrations in patients with cardiovascular diseases, supporting its diagnostic utility (15). Venu et al. demonstrated that elevated H-FABP levels are associated with adverse cardiovascular events and have important prognostic significance (16). Similarly, Prakash reported that H-FABP is a reliable biomarker for the early diagnosis of acute myocardial infarction (17).

Our findings also showed significant changes in oxidative stress markers during the acute phase of myocardial infarction. Elevated MDA levels at admission indicated increased lipid peroxidation resulting from excessive production of reactive oxygen species during myocardial ischemia (18). Aladağ et al. demonstrated significant disturbances in oxidant and antioxidant balance in patients with myocardial infarction (19). Savović et al. reported that oxidative stress biomarkers have important prognostic value in patients with STEMI and NSTEMI (20). Nagasundaram et al. observed significantly elevated serum MDA levels in patients with acute myocardial infarction (21). Ceylan and Demir further demonstrated that oxidative stress biomarkers are associated with the severity of coronary artery disease (22). Gaber et al. reported that oxidative stress and inflammation play important roles in the occurrence and progression of coronary artery disease (23). Roumeliotis et al. also demonstrated that biomarkers of oxidative stress are significantly associated with myocardial necrosis and heart failure in patients with myocardial infarction (24).

In contrast, SOD activity increased significantly during the 12-hour observation period, indicating activation of endogenous antioxidant defense mechanisms. Superoxide dismutase plays a vital role in protecting myocardial tissue against oxidative damage by scavenging superoxide radicals and reducing oxidative stress (25). Rotariu et al. described oxidative stress as a major pathological mechanism in cardiovascular disorders and emphasized the protective role of antioxidant enzymes (26). Duan et al. reported that restoration of antioxidant defense contributes to improved myocardial recovery and better cardiac function after myocardial infarction (27). Stătescu et al. also highlighted the prognostic importance of antioxidant biomarkers in patients with acute myocardial infarction (28).

Although significant temporal changes were observed in H-FABP, MDA and SOD, correlation analysis revealed no statistically significant association between H-FABP and either oxidative stress marker at the time of admission or at 12 hours of admission. The weak correlation coefficients indicate that the degree of myocardial injury measured by H-FABP does not directly predict the severity of oxidative stress or antioxidant response (29). Malhotra and Ahmed similarly reported that correlations among different cardiac biomarkers may be weak because they reflect different biological mechanisms (30). Shrivastava et al. demonstrated that biochemical and inflammatory markers change independently during the course of acute myocardial infarction (31). Kocatürk et al. further emphasized that combining multiple biomarkers provides better prognostic information than relying on a single marker (32).

The findings of the present study support the concept of a multi-biomarker approach for the evaluation of patients with acute myocardial infarction. Toparch et al. demonstrated that a multi-biomarker panel improves prognostic assessment in

patients with suspected myocardial infarction (33). Munir et al. highlighted the importance of combining biochemical cardiac markers for accurate diagnosis of acute coronary syndrome (34). Shinde et al. emphasized the diagnostic utility of multiple cardiac biomarkers and biosensors in myocardial infarction (35). Rizvi et al. reviewed the physiological and clinical significance of various cardiac biomarkers in cardiovascular diseases (36). Kopec et al. reported that combining cardiac biomarkers significantly improves prediction of myocardial infarction and clinical outcomes (37). Horiuchi et al. also demonstrated that multiple biomarkers improve diagnostic accuracy and prognostic assessment in myocardial infarction (38). Therefore, simultaneous assessment of cardiac injury biomarkers and oxidative stress markers may provide a more comprehensive understanding of disease progression and facilitate better management of patients with acute myocardial infarction.

5. CONCLUSION

The present study demonstrated significant temporal changes in serum Heart-type Fatty Acid-Binding Protein (H-FABP), Malondialdehyde (MDA) and Superoxide Dismutase (SOD) during the first 12 hours after hospital admission in patients with acute myocardial infarction. H-FABP levels increased significantly, indicating ongoing myocardial injury, whereas MDA levels decreased and SOD activity increased, reflecting dynamic changes in oxidative stress and antioxidant defense following treatment. Despite these significant biochemical alterations, no statistically significant correlation was observed between H-FABP and either MDA or SOD, suggesting that myocardial injury and oxidative stress represent distinct yet complementary pathophysiological processes during acute myocardial infarction. These findings support the potential value of a multi-biomarker approach incorporating markers of myocardial injury and oxidative stress to provide a more comprehensive assessment of patients with acute myocardial infarction and may improve early diagnosis, risk stratification, and clinical decision-making

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