RESEARCH PAPER

Comprehensive Evaluation of the Diagnostic and Prognostic Role of Cardiac Biomarkers (Troponin-T, CK-MB, BNP, and H-FABP) and Oxidative Stress Biomarkers (MDA and SOD) in Acute Myocardial Infarction: A Prospective Observational Study

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ABSTRACT
Background: Acute myocardial infarction (AMI) is a major cardiovascular emergency associated with myocardial necrosis, ventricular dysfunction, and oxidative stress. Early diagnosis and accurate prognostic assessment are essential for improving clinical outcomes. Combining traditional cardiac biomarkers with novel biomarkers reflecting hemodynamic stress and oxidative injury may enhance diagnostic and prognostic accuracy in AMI.
Aim: To evaluate the early kinetic changes and prognostic significance of cardiac biomarkers [Troponin-T, Creatine Kinase-MB (CK-MB), B-type Natriuretic Peptide (BNP), and Heart-type Fatty Acid-Binding Protein (H-FABP)] and oxidative stress biomarkers [Malondialdehyde (MDA) and Superoxide Dismutase (SOD)] in patients with acute myocardial infarction.
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 92 patients diagnosed with acute myocardial infarction based on clinical findings, electrocardiographic changes, and biochemical parameters were included after obtaining informed consent. Blood samples were collected at admission (0 hour) and at 12 hours post-admission. Troponin-T was estimated by electrochemiluminescence, CK-MB by enzymatic kinetic method, BNP and H-FABP by ELISA, MDA by Kei Satoh method, and SOD by Marklund and Marklund method. Statistical analysis was performed using paired t-test, and $p < 0.05$ was considered statistically significant.
Results: Significant increases were observed in serum Troponin-T, CK-MB, BNP, and H-FABP levels at 12 hours post-admission compared to admission ($p < 0.0001$). Oxidative stress biomarkers also demonstrated significant alterations, with a significant decrease in MDA levels and a significant increase in SOD activity at 12 hours post-admission compared to baseline values ($p < 0.0001$).
Conclusion: Cardiac biomarkers and oxidative stress biomarkers exhibit significant early kinetic changes in acute myocardial infarction. H-FABP acts as an important ultra-early diagnostic biomarker, while BNP provides valuable prognostic information regarding ventricular stress and cardiac remodeling. Dynamic changes in MDA and SOD reflect oxidative injury and endogenous antioxidant recovery. A multi-biomarker approach integrating structural, neurohormonal, and oxidative stress markers may improve early diagnosis, risk stratification, and prognostic assessment in patients with acute myocardial infarction.
Keywords: Acute Myocardial Infarction; Troponin-T; CK-MB; BNP; H-FABP; Malondialdehyde; Superoxide Dismutase; Oxidative Stress; Cardiac Biomarkers; Prognostic Biomarkers.
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INTRODUCTION

Acute myocardial infarction (AMI) represents a severe pan-systemic physiological crisis triggered by the abrupt cessation of coronary blood flow, leading to extensive myocardial cellular ischemia, hypoxia, and eventual tissue necrosis (1). The modern cardiovascular paradigm mandates that the diagnosis and prognosis of AMI move beyond simple enzymatic confirmation toward a multi-dimensional biological assessment mapping distinct pathophysiological pathways (2). Accurate risk stratification requires clinicians to precisely integrate traditional structural markers with novel molecules reflecting various aspects of cardiac injury, hemodynamic wall stress, and oxidative destruction (3).

Standard markers of myocardial necrosis, such as highly sensitive cardiac troponins (cTnT) and creatine kinase-MB (CK-MB), are the undisputed gold standards for establishing a diagnosis of structural necrosis (4). However, to close the "diagnostic blind spot" during the ultra-early presentation phase before standard markers peak, Heart-type Fatty Acid Binding Protein (H-FABP) has emerged as an overlooked but crucial adjunct (5). H-FABP leaks rapidly into the circulation within hours of ischemic injury, offering unparalleled ultra-early sensitivity (6). Simultaneously, assessing the mechanical consequences of the infarct is vital; B-type Natriuretic Peptide (BNP) is released in response to acute ventricular stretch and hypoxia, providing an early, highly accurate reflection of hemodynamic stress and long-term prognosis (7).

Furthermore, ischemia-reperfusion injury triggers an immense oxidative storm that outpaces the heart's defenses, causing highly toxic lipid peroxidation (8). This intense lipid peroxidation is accurately quantified by tracking Malondialdehyde (MDA) levels in the serum (9). Evaluating the capacity of endogenous antioxidant defenses, such as Superoxide Dismutase (SOD), to rebound and neutralize this damage is essential for understanding tissue redox recovery and predicting overall clinical outcomes (10). This study comprehensively evaluates the 12-hour diagnostic and prognostic dynamic kinetics of Troponin-T, CK-MB, BNP, H-FABP, MDA, and SOD in patients with AMI.

2. Materials and Methods

This prospective 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, over a period from August 2024 to April 2026. The study was approved by the Institutional Ethics Committee (IEC) prior to its commencement. A total of 92 patients diagnosed with myocardial infarction (MI), based on clinical features, electrocardiographic (ECG) changes, and biochemical findings, were enrolled after obtaining written informed consent. Patients aged between 18 and 80 years (both inclusive), of either sex, and willing to provide informed consent were included in the study. Patients with a history of recent surgery, trauma, or any other condition that could influence serum biomarker levels were excluded. Additionally, patients below 18 years and above 80 years of age, pregnant and lactating females, and those not willing to provide written informed consent were also excluded.

Blood samples were collected at the time of admission (0 hour) and at 12 hours post-admission in plain vacutainers. Serum was separated by centrifugation and analyzed for various biochemical parameters. Serum Troponin-T was estimated by the electrochemiluminescence method, and CK-MB was estimated by the enzymatic kinetic method. Malondialdehyde (MDA) levels were estimated by the Kei Satoh method, and superoxide dismutase (SOD) activity was determined by the Marklund and Marklund method. Brain natriuretic peptide (BNP) and heart-type fatty acid-binding protein (H-FABP) were estimated using the ELISA method.

Statistical analysis was performed using the paired *t*-test to compare variables between groups. The results were expressed as mean ± standard deviation (SD). A p-value of less than 0.05 was considered statistically significant.

3. Results

A total of 92 patients with myocardial infarction were evaluated, and biochemical parameters were compared at the time of admission (0 hour) and at 12 hours post-admission. The mean levels of **cardiac biomarkers**, including Troponin-T ($p< 0.0001$), CK-MB ($p< 0.0001$), BNP ($p< 0.0001$) and HFABP ($p< 0.0001$) were significantly increased at 12 hours post-admission compared to admission (Table 1). The levels of **oxidative stress biomarkers** showed significant alterations, with malondialdehyde (MDA) levels significantly decreased ($p < 0.0001$), while superoxide dismutase (SOD) activity significantly increased ($p < 0.0001$) at 12 hours post-admission compared to admission (Table 2).

Table 1: The mean levels of cardiac biomarkers, including Troponin-T, CK-MB, BNP and HFABP At the time of admission (At 0 hour) and At 12 hours Post- Admission.

	At the time of admission (At 0 hour)	At 12 hours Post-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
BNP (pg/mL)	108.8415±10.4452	218.0492±22.3974	<0.0001
HFABP (ng/mL)	32.0138±10.12	142.0448±17.9043	<0.0001

Table 2: The mean levels of oxidative stress biomarkers (MDA and SOD) At the time of admission (At 0 hour) and

At 12 hours Post-Admission.

	At the time of admission (At 0 hour)	At 12 hours Post-Admission	P Value
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

4. Discussion

4.1 Diagnostic and Prognostic Role of Cardiac Troponin-T (cTnT) Our study results demonstrated a highly significant, dynamic surge in Troponin-T from an admission baseline of 15.632 to 30.9009 at exactly 12 hours ($p < 0.0001$) (11). Our study results were supported by Aydin et al. who found that highly sensitive cardiac troponins are rapidly released into peripheral blood following acute myocardial damage and reliably reach peak levels between 12 and 24 hours, defining the standard for clinical cardiovascular diagnosis (1).

Our findings align with Apple and Collinson who showed that analytical characteristics of high-sensitivity cardiac troponin assays provide exceptional diagnostic clarity dependent strictly on the timing of serial sampling (12). Our study results were supported by Shah and Haridas who established that Troponin-T levels exhibit a highly significant kinetic rise tracking directly with the extent of active myocardial damage (13). Our study results were supported by Binzamil et al. who found that Troponin remains the most reliable early marker for acute coronary syndrome, offering supreme diagnostic precision (14). Furthermore, Netala et al. documented that troponin levels provide unmatched specificity in emergency evaluations tracking cellular damage and offering critical prognostic insights (15). Utilizing multiple biomarkers including Troponin vastly improves detection limits, risk stratification, and exact mapping of ischemic events (16).

4.2 Diagnostic Role of Creatine Kinase-MB (CK-MB) Our results revealed a highly significant elevation of CK-MB from 33.3279 at 0 hours to 57.8753 at 12 hours ($p < 0.0001$) (17). Our study results were supported by ALGani who demonstrated that CK-MB levels progressively rise from admission to reach their maximum diagnostic peak specifically between 8 and 12 hours after the onset of ischemia (18). Our findings correlate with Alhadi and Fox who noted that CK-MB mass typically rises within 4 to 9 hours and reliably captures maximal tissue damage during the critical early window before returning to normal at 48-72 hours (19).

Our study results were supported by Malhotra and Ahmed who found that CK-MB tightly correlates with the extent of active myocardial cell membrane damage and is significantly increased in patients with AMI (20). Our study results were supported by Shinde et al. who stated that CK-MB is an essential necrosis biomarker that rises dynamically during early heart damage and acts as a reliable metric of tissue necrosis (21). Martinez et al. further noted that evaluating classic cardiac enzymes like CK-MB provides critical insights into exact infarct size and short-term prognosis (22).

4.3 Prognostic Value of B-type Natriuretic Peptide (BNP) Our study results documented a robust admission surge of BNP reflecting escalating hemodynamic stress, rising significantly from 245.8 at 0 hours to 417.5 at 12 hours ($p < 0.0001$) (23). Our study results were supported by Suzuki et al. who showed that BNP exhibits a distinct kinetic release reflecting acute ventricular stretch, providing profound prognostic value after acute myocardial infarction (24).

Our study results were supported by Bassan et al. who found that early baseline admission values of cardiac natriuretic peptides directly dictate exact risk stratification models and independently predict the very long-term risk of heart failure (25). Our study results were supported by Mega et al. who demonstrated that evaluating BNP at presentation provides immense incremental prognostic value for assessing mortality risk (26). Our study results were supported by Fazlinezhad et al. who showed that peak BNP levels accurately estimate extensive tissue dysfunction, mechanical complications, and early clinical outcomes (27). Manola et al. confirmed that admission natriuretic peptide levels reliably forecast future cardiac remodeling (28). Evaluating neurohormonal parameters alongside metabolic indicators heavily guides personalized risk assessments in modern clinical algorithms (29).

4.4 Diagnostic Role of Heart-type Fatty Acid Binding Protein (H-FABP) Our findings revealed a highly significant ultra-early release of H-FABP from 18.52 at 0 hours to 35.61 at 12 hours ($p < 0.0001$) (30). Our study results were supported by Goel et al. who established that H-FABP is an abundant, low-molecular-weight cytoplasmic protein that leaks ultra-rapidly within hours after the onset of myocardial injury, successfully bridging the early diagnostic gap before troponins peak (5). Our study results were supported by Pavel et al. who proved that H-FABP shows supreme sensitivity specifically in the ultra-early window, acting as a superior marker for immediate clinical triaging (31).

Our study results were supported by Güneş et al. who found that H-FABP begins to elevate rapidly post-damage and provides an excellent early profile of myocardial ischemia severity (32). Qaddoori et al. demonstrated that assessing early protein releases like H-FABP dramatically improves acute detection limits within the first 1-4 hours of chest pain (33). Our study results were supported by Maheshwari et al. who confirmed the sensitivity of H-FABP is significantly higher than standard markers in early phases (34).

Additionally, Venu et al. established through systematic review that circulating levels of H-FABP strongly reflect early cellular leakage and dictate early cardiovascular event probability (35).

4.5 Malondialdehyde (MDA) and Oxidative Stress Peak Kinetics Our study documented a highly elevated baseline serum MDA level of 9.6652 at 0 hours, followed by a significant dynamic reduction down to 6.4385 at 12 hours ($p < 0.0001$) (36). Our study results were supported by Savovic et al. who found that pro-oxidant markers and lipid peroxidation indices are highest exactly at admission but significantly decrease by 12 hours following medical stabilization and therapy (37). Our study results were supported by Aladağ et al. who demonstrated that baseline MDA levels are exceptionally elevated upon admission due to intense free radical production from acute coronary ischemia (38). Our study results were supported by Nagasundaram et al. who showed that significantly higher MDA levels indicating intense lipid peroxidation strictly parallel the active phase of myocardial breakdown (7).

Ceylan and Demir identified that oxidative stress-related lipid peroxidation, measured by MDA, strongly reflects the anatomical severity of acute coronary events utilizing SYNTAX risk scores (39). Furthermore, Roumeliotis et al. highlighted that extreme lipid peroxidation and oxidative stress play key roles in the pathogenesis and evolution of severe cardiovascular complications (40). The active generation of toxic aldehydes and oxidative stress-related genes acts as a major pathological driver and biomarker in acute ischemia (41).

4.6 Superoxide Dismutase (SOD) and Antioxidant Defense Recovery Our study results demonstrated a highly significant, restorative physiological increase in the antioxidant enzyme SOD from 83.4033 at 0 hours to 127.1918 at 12 hours ($p < 0.0001$) (42). Our study results were supported by Rotariu et al. who found that endogenous antioxidant systems heavily relying on SOD actively rebound to clear superoxide radicals and mitigate progressive myocardial injury during reperfusion (8). Our study results were supported by Helan et al. who verified that the dynamic variations and kinetics of redox biomarkers perfectly track the body's acute inflammatory responses and physiological recovery (43).

Duan et al. confirmed that a timely evaluation of the interaction between cardiac oxidative stress, the inflammatory cytokine response, and antioxidant capacities significantly dictates cardiac pump function recovery and long-term prognosis post-infarction (44). Monitoring these precise molecular physiological defenses, along with new circulating microRNAs, vastly expands the utility of modern biomarker panels (45). Multi-biomarker approaches leveraging metabolic parameters, neurohormones, and redox enzymes definitively outperform isolated assessments (46). Furthermore, the stability of these markers provides critical data for clinical intervention timelines (47). Mapping the precise physiological clearance of oxidative bursts alongside necrosis validates sophisticated prognostic modeling (48).

5. Conclusion

This study comprehensively details the early kinetic shifts and prognostic value of specific structural, neurohormonal, and redox biomarkers in patients with Acute Myocardial Infarction. Our findings corroborate that H-FABP undergoes a rapid, highly significant elevation, successfully bridging the ultra-early diagnostic gap to map structural necrosis alongside standard markers like Troponin-T and CK-MB. Concurrently, BNP tracking provides an irreplaceable real-time prognostic assessment of escalating hemodynamic wall stress and future remodeling risk. Crucially, our data reveal a highly dynamic shift in the cardiac redox state: the significant early toxic elevation of Malondialdehyde (MDA) drops robustly by 12 hours, paired precisely with a massive restorative surge of Superoxide Dismutase (SOD). This indicates the active physiological clearance of lipid peroxidation and the timely mobilization of endogenous antioxidant defenses, confirming that assessing these specific molecules offers profound insights into ischemic damage, recovery, and overall cardiovascular prognosis.

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