Research Article

Prognostic Significance and Early Kinetic Changes of BNP, H-FABP, and Oxidative Stress Biomarkers (MDA and SOD) in Acute Myocardial Infarction

Roshan Kumar Sah[1], Dr. Zunjarrao G. Badade[2], Dr. Shilpa Kadam[3], Dr. Dattatray Bhusare[4], Dr. Kshitij Z. Badade[5], Dr. Kanchan Sonone[6], Dr. Sandip Lambe[7], Dr. Santosh Gawali[8]

[1]Ph.D Scholar, Department of Biochemistry, MGM Medical College, Kamothe, Navi Mumbai. Tutor, Department of Biochemistry, SMBT IMSRC Nashik
[2]Professor, Department of Biochemistry, MGM Medical College, Kamothe, Navi Mumbai
[3] Professor, Department of Cardiology, MGM Medical College, Kamothe, Navi Mumbai
[4] Professor & HOD, Department of Emergency Medicine, MGM Medical College, Kamothe, Navi Mumbai
[5]Associate Professor, Department of Orthopedic, MGM Medical College, Kamothe, Navi Mumbai
[6]Professor and HOD, Department of Biochemistry, SMBT IMSRC, Nashik
[7]Professor and Deputy Dean, Department of Biochemistry, SMBT IMSRC, Nashik
[8]Professor and HOD, Department of Biochemistry, MGM Medical College, Kamothe, Navi Mumbai
Corresponding Author- Dr. Zunjarrao G. Badade- 9322880252- badadezg@gmail.com, (Professor, Department of Biochemistry, MGM Medical College, Kamothe, Navi Mumbai)
Email ID: badadezg@gmail.com, roshankumarsah766@gmail.com

Abstract

Background: Acute myocardial infarction (AMI) is associated with myocardial ischemia, ventricular dysfunction, and excessive oxidative stress leading to significant cardiovascular morbidity and mortality. Early assessment of biomarkers reflecting hemodynamic stress, myocardial injury, and oxidative damage may improve prognostic evaluation and clinical management in AMI. **Aim:** To evaluate the early kinetic changes and prognostic significance of B-type Natriuretic Peptide (BNP), 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 enrolled after obtaining written informed consent. Blood samples were collected at admission (0 hour) and at 12 hours post-admission. BNP and H-FABP were 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 t-test, and $p < 0.05$ was considered statistically significant. **Results:** Significant increases were observed in serum BNP and H-FABP levels at 12 hours post-admission compared to admission values ($p < 0.0001$). 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:** BNP and H-FABP exhibit significant early kinetic changes and provide valuable prognostic information in acute myocardial infarction. H-FABP serves as an important ultra-early biomarker of myocardial injury, while BNP reflects ventricular stress and remodeling risk. Dynamic alterations in MDA and SOD indicate oxidative injury and endogenous antioxidant defense recovery. A combined assessment of neurohormonal and oxidative stress biomarkers may enhance prognostic evaluation and risk stratification in patients with acute myocardial infarction.

Keywords: Acute Myocardial Infarction; BNP; H-FABP; Malondialdehyde; Superoxide Dismutase; Oxidative Stress; Cardiac Biomarkers; Prognostic Biomarkers; Ventricular Stress; Lipid Peroxidation.

1. Introduction

Acute myocardial infarction (AMI) represents a pan-systemic physiological crisis triggered by the abrupt cessation of coronary blood flow, leading to extensive myocardial ischemia, cellular 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). 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 the long-term risk of heart failure (3).

Furthermore, to close the ultra-early diagnostic blind spot before other markers peak, Heart-type Fatty Acid Binding Protein (H-FABP) has emerged as a crucial adjunct, leaking rapidly into the circulation within hours of ischemic injury and offering unparalleled sensitivity (4).

Simultaneously, ischemia-reperfusion injury triggers an immense oxidative storm that outpaces the heart's defenses. The massive, immediate production of reactive oxygen species causes highly toxic lipid peroxidation, which is accurately quantified by tracking Malondialdehyde (MDA) levels (5).

Evaluating the capacity of endogenous antioxidant defenses, such as Superoxide Dismutase (SOD), to rebound and neutralize this damage is therefore essential for understanding tissue recovery and overall clinical prognosis (6). This study comprehensively evaluates the 12-hour dynamic kinetics and prognostic value of BNP, H-FABP, MDA, and SOD.

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. Brain natriuretic peptide (BNP) and heart-type fatty acid-binding protein (H-FABP) were estimated using the ELISA method. Malondialdehyde (MDA) levels were estimated by the Kei Satoh method, and superoxide dismutase (SOD) activity was determined by the Marklund and Marklund 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 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 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
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 Kinetics and Prognostic Value of B-type Natriuretic Peptide (BNP) Our study results documented a robust admission surge of BNP reflecting escalating hemodynamic stress, rising significantly at 12 hours ($p < 0.0001$) compared to at 0 hour. 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 (7).

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 in patients with ST-segment elevation myocardial infarction (8). Our study results were supported by Manola et al. who confirmed that admission natriuretic peptide levels reliably forecast future cardiac remodeling and act as strong predictors of heart failure (9).

Our study results were supported by Lee et al. who found that short-term follow-up BNP levels after hospital discharge serve as a powerful predictive biomarker for all-cause mortality and major adverse cardiovascular events (10). Our study results were supported by Aleesha et al. who showed that peak BNP levels accurately estimate extensive tissue dysfunction and mechanical complications following acute myocardial infarction (11).

4.2 Kinetics of Heart-type Fatty Acid-Binding Protein (H-FABP) Our findings revealed a highly significant ultra-early release of H-FABP at 12 hours ($p < 0.0001$) compared to at 0 hour. Our study results were supported by Pavel et al. who established that H-FABP shows supreme sensitivity specifically in the ultra-early 0-4 hour window, proving significantly superior to standard markers for immediate clinical triaging in suspected infarction (12). Our study results were supported by Güneş et al. who found that H-FABP begins to elevate rapidly post-damage, peaking sharply and offering critical insights into coronary artery disease progression (13). Our study results were supported by Qaddoori et al. who demonstrated that novel cardiac biomarkers like H-FABP dramatically improve acute detection limits within the first 1-4 hours of chest pain, facilitating earlier intervention (14).

Our study results were supported by Venu et al. whose systematic review confirmed that circulating levels of H-FABP strongly reflect early cellular leakage and are intimately associated with future cardiovascular events (15). Our study results were supported by Prakash S. who showed that H-FABP acts as an excellent early cardiac biomarker that elevates considerably during the critical initial window of acute myocardial infarction diagnosis (16).

4.3 Malondialdehyde (MDA) and Oxidative Stress Peak Kinetics Our study results 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 ($p < 0.0001$). 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 (17).

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 (18). Our study results were supported by Ceylan and Demir who demonstrated that oxidative stress-related lipid peroxidation, measured by MDA, strongly reflects the anatomical severity of acute coronary events utilizing SYNTAX and ACEF risk scores (19).

Our study results were supported by Gaber et al. who proved that lipid peroxidation measured as MDA increases significantly during the acute inflammatory phase of premature ST-segment elevation myocardial infarction (20). Our study results were supported by Roumeliotis et al. who highlighted that extreme lipid peroxidation and oxidative stress play key roles in the pathogenesis and evolution of severe cardiovascular complications (21). Our study results were supported by Sun et al. who identified that the active generation of toxic aldehydes and oxidative stress-related genes act as major pathological drivers and biomarkers in acute ischemia (22).

4.4 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 ($p < 0.0001$). 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 (23).

Our study results were supported by Duan et al. who concluded that timely drug intervention targeting antioxidant and anti-inflammatory pathways effectively lowers oxidative pressure, subsequently restoring cardiac pump function and significantly improving patient prognosis post-infarction (24).

5. Conclusion

This study comprehensively details the early kinetic shifts and prognostic value of specific neurohormonal, structural, 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. Concurrently, BNP tracking provides an irreplaceable real-time 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.

References

1. Aydin S, Ugur K, Aydin S, Sahin I, Yardim M. Biomarkers in acute myocardial infarction: current perspectives. Vasc Health Risk Manag. 2019;15:1-10.
2. Statescu C, Anghel L, Tudurachi BS, et al. From Classic to Modern Prognostic Biomarkers in Patients with Acute Myocardial Infarction. Int J Mol Sci. 2022;23:9168.
3. Suzuki S, Yoshimura M, et al. Plasma Level of B-Type Natriuretic Peptide as a Prognostic Marker After Acute Myocardial Infarction. Circulation. 2004.
4. Goel H, Melot J, Krinock MD, et al. Heart-type fatty acid-binding protein: an overlooked cardiac biomarker. Ann Med. 2020;52(8):444-461.
5. Madole MB, Bachewar NP, Aiyar CM. Study of oxidants and antioxidants in patients of acute myocardial infarction. Adv Biomed Res. 2015;4:241.
6. Aladağ N, Asoğlu R, Ozdemir M, et al. Oxidants and antioxidants in myocardial infarction (MI). J Med Biochem. 2021;40(3):286-294.
7. Bassan F, Bassan R, Esporcatte R, Santos B, Tura B. Very Long-Term Prognostic Role of Admission BNP in Non-ST Segment Elevation Acute Coronary Syndrome. Arq Bras Cardiol. 2016.
8. Mega JL, Morrow DA, de Lemos JA, et al. B-Type Natriuretic Peptide at Presentation and Prognosis in Patients With ST-Segment Elevation Myocardial Infarction. J Am Coll Cardiol. 2004;44:335-339.
9. Manola S, Pavlovic N, Radeljic V, et al. B-type Natriuretic Peptide as Predictor of Heart Failure in Patients with Acute ST Elevation Myocardial Infarction. Croat Med J. 2009.
10. Lee JW, Choi E, Khanam SS, et al. Prognostic value of short-term follow-up B-type natriuretic peptide levels after hospital discharge in patients with acute myocardial infarction. Int J Cardiol. 2019;289:19-23.
11. Fazlinezhad A, et al. Plasma Brain Natriuretic Peptide (BNP) as an Indicator of Left Ventricular Function, Early Outcome and Mechanical Complications after Acute Myocardial Infarction. Clin Med Insights Cardiol. 2011;5:77-83.
12. Pavel AL, Kundnani NR, Morariu SI, et al. Importance of H-FABP in Early Diagnosis of Acute Myocardial Infarction. Int J Gen Med. 2024;17:4335-4346.
13. Güneş E, Mert N, Kaya Y, Günbatar N, Mert H. Changes in heart type fatty acid binding protein (H-FABP) and certain biochemical parameters during chronic artery diseases. J Sci Rep-A. 2023;53:147-161.
14. Qaddoori HT, Mohammed FM, Abdullah AN. Novel Cardiac Biomarkers H-FABP and GPBB for Early Detection and Prognosis of Acute Myocardial Infarction. Angiotherapy. 2024;8(3):1-12.
15. Venu AP, Rajkumar R, Roy DD, et al. Association of H-FABP with cardiovascular events: A systematic review. J Cardiovasc Thorac Res. 2024;16(2):77-87.
16. Prakash S. A Study on the Role of Heart Type Fatty Acid Binding Protein in the Diagnosis of Acute Myocardial Infarction. J Clin Diagn Res. 2016.
17. Savovic Z, Pindovic B, Nikolic M, et al. Prognostic Value of Redox Status Biomarkers in Patients Presenting with STEMI or Non-STEMI. J Pers Med. 2023;13(7):1050.
18. Nagasundaram M, Singh YR, Singh TSD, et al. Study of serum malondialdehyde level in patients with acute myocardial infarction. Int J Acad Med Pharm. 2025;7(3):625-629.

19. Ceylan Y, Demir H. Associations of oxidative stress biomarkers with SYNTAX and ACEF risk scores in acute myocardial infarction. Medicine. 2025;104:38.
20. Gaber MA, Omar OH, Meki AM, et al. The role of inflammation and oxidative stress markers in the occurrence and severity of coronary artery disease. Biomed Res Ther. 2024;11(11):6960-6970.
21. Roumeliotis S, Veljkovic A, Georgianos PI, et al. Association between Biomarkers of Oxidative Stress and Inflammation with Cardiac Necrosis and Heart Failure in Non-ST Segment Elevation Myocardial Infarction. Oxid Med Cell Longev. 2021.
22. Sun Y, Wang M, Tan X, Zhang H, Yang S. Identification of Oxidative Stress-Related Biomarkers in Acute Myocardial Infarction. Int J Gen Med. 2023.
23. Rotariu D, Babes EE, Tit DM, et al. Oxidative stress – Complex pathological issues concerning the hallmark of cardiovascular and metabolic disorders. Biomed Pharmacother. 2022;152:113238.
24. Duan D, Li H, Chai S, et al. The relationship between cardiac oxidative stress, inflammatory cytokine response, cardiac pump function, and prognosis post-myocardial infarction. Sci Rep. 2024.