

Oxidative Stress and Antioxidant Defense in Acute Myocardial Infarction: A Prospective Observational Study

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Abstract

Background: Acute myocardial infarction (AMI) is associated with myocardial ischemia, cellular injury, inflammation and disturbances in redox homeostasis. Oxidative stress plays an important role in myocardial injury through excessive generation of reactive oxygen species and subsequent lipid peroxidation. Malondialdehyde (MDA) is a commonly used indicator of lipid peroxidation, whereas superoxide dismutase (SOD) represents an important enzymatic component of the endogenous antioxidant defense system. Assessment of these biomarkers at different time points may provide insight into the early oxidative and antioxidant responses following myocardial infarction.

Aim: To evaluate the early kinetic changes in oxidative stress and antioxidant defense biomarkers, namely malondialdehyde (MDA) and superoxide dismutase (SOD), in patients with acute myocardial infarction during the first 12 hours after hospital admission.

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 myocardial infarction were included after obtaining written informed consent. Blood samples were collected at admission (0 hour) and 12 hours after admission. Serum MDA was estimated using the Kei Satoh method, while SOD activity was determined using the Marklund and Marklund method. The results were expressed as mean $\pm$ standard deviation (SD). Paired t-test was used to compare the biomarker levels between the two time points and $p < 0.05$ was considered statistically significant.

Results: The mean serum MDA level significantly decreased from 9.6652 ± 0.9244 nmol/mL at admission to 6.4385 ± 1.5219 nmol/mL at 12 hours ($p < 0.0001$). In contrast, mean SOD activity significantly increased from 83.4033 ± 8.9211 U/mL at admission to 127.1918 ± 16.5676 U/mL at 12 hours ($p < 0.0001$). The simultaneous decrease in MDA and increase in SOD demonstrated a significant early shift in the oxidative-antioxidant profile following myocardial infarction.

Conclusion: Patients with acute myocardial infarction demonstrated significant early alterations in oxidative stress and antioxidant defense. The decrease in MDA together with an increase in SOD activity between admission and 12 hours suggests dynamic modulation of lipid peroxidation and endogenous antioxidant defense during the early phase following myocardial infarction. Serial assessment of MDA and SOD may provide useful biochemical information regarding early redox changes associated with myocardial infarction.

Keywords: Acute myocardial infarction; oxidative stress; malondialdehyde; superoxide dismutase; lipid peroxidation; antioxidant defense; reactive oxygen species; myocardial injury.

1. Introduction

Acute myocardial infarction (AMI) is a major cardiovascular condition associated with acute myocardial ischemia and injury. Although myocardial infarction is primarily initiated by an imbalance between myocardial oxygen demand and coronary oxygen supply, the resulting tissue injury involves several interconnected biochemical processes, including oxidative stress, inflammation, mitochondrial dysfunction, membrane damage and cell death.¹

Oxidative stress occurs when the production of reactive oxygen species (ROS) exceeds the capacity of the endogenous antioxidant defense system to neutralize them. The myocardium is particularly susceptible to oxidative injury because of its high metabolic activity and dependence on mitochondrial oxidative phosphorylation. During myocardial ischemia and subsequent restoration of blood flow, increased generation of ROS can occur, resulting in disruption of cellular structures and impairment of normal myocardial function.² Reactive oxygen species include superoxide anion, hydrogen peroxide, hydroxyl radicals, and other reactive

molecules. Under physiological conditions, ROS participate in cellular signaling and normal metabolic processes. However, excessive ROS production can damage lipids, proteins, nucleic acids and cellular membranes. In myocardial infarction, ischemia and reperfusion can disturb the normal balance between oxidant generation and antioxidant protection, resulting in increased oxidative stress.^{2,3}

One important consequence of excessive oxidative stress is lipid peroxidation. Polyunsaturated fatty acids present in cellular membranes are particularly susceptible to attack by free radicals. Lipid peroxidation results in the formation of several secondary products, among which malondialdehyde (MDA) is widely used as a biochemical marker of lipid oxidative damage. Increased MDA reflects enhanced lipid peroxidation and has been investigated in patients with acute myocardial infarction.^{4,5}

The antioxidant defense system protects cells against excessive oxidative damage. Superoxide dismutase (SOD) is one of the major antioxidant enzymes and provides an important first line of defense against superoxide radicals. SOD catalyzes the conversion of superoxide anion into hydrogen peroxide and oxygen, thereby reducing the availability of highly reactive superoxide radicals. The activity of SOD can therefore provide information about the status of endogenous enzymatic antioxidant defense.^{2,6}

Previous studies have reported alterations in oxidative stress and antioxidant biomarkers in patients with myocardial infarction. Madole et al. reported changes in oxidant and antioxidant status among patients with acute myocardial infarction, while Aladağ et al. demonstrated alterations in MDA and SOD among patients with STEMI and NSTEMI.^{5,7} Studies investigating redox status in acute coronary syndromes have also suggested that oxidative stress biomarkers may change dynamically during the early clinical course.⁸

However, oxidative stress following myocardial infarction is not a static process. The balance between oxidant generation, lipid peroxidation, antioxidant defense, and tissue recovery may change over time. Therefore, evaluation of MDA and SOD at more than one time point may provide a better understanding of the early redox response than a single measurement.

The present study was therefore designed to evaluate the early kinetic changes in MDA and SOD in patients with acute myocardial infarction by comparing their levels at admission and 12 hours after hospital admission.

1.1 Rationale of the Study

Myocardial infarction initiates a complex biochemical response in which oxidative stress and antioxidant defense are continuously changing. MDA provides information regarding lipid peroxidation, whereas SOD reflects an important component of endogenous antioxidant defense. Studying these two biomarkers together at admission and during the early post-admission period may help characterize the changing oxidative-antioxidant balance associated with acute myocardial infarction.

1.2 Research Question

Do serum MDA levels and SOD activity show significant changes between admission and 12 hours after admission in patients with acute myocardial infarction?

1.3 Hypothesis

Patients with acute myocardial infarction exhibit significant early changes in oxidative stress and antioxidant defense, reflected by alterations in serum MDA levels and SOD activity between admission and 12 hours after hospital admission.

1.4 Aim

To evaluate the early kinetic changes in oxidative stress and antioxidant defense biomarkers, MDA and SOD, in patients with acute myocardial infarction.

1.5 Objectives

1. To estimate serum MDA levels in patients with acute myocardial infarction at admission and 12 hours after admission.
2. To estimate SOD activity in patients with acute myocardial infarction at admission and 12 hours after admission.
3. To compare MDA levels between admission and 12 hours after admission.
4. To compare SOD activity between admission and 12 hours after admission.
5. To assess the early change in the oxidative-antioxidant profile following myocardial infarction.

2. Materials and Methods

2.1 Study Design

A prospective observational study was conducted to evaluate early changes in oxidative stress and antioxidant defense biomarkers in patients with acute myocardial infarction.

2.2 Study Setting

The 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, Maharashtra, India.

2.3 Study Period

The study was conducted from August 2024 to April 2026.

2.4 Ethical Approval

The study was commenced after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants before enrolment.

2.5 Study Population

A total of 110 patients diagnosed with myocardial infarction were included in the study.

2.6 Inclusion Criteria

Patients were included if they

- were aged 18–80 years;
- were of either Gender;
- had a diagnosis of myocardial infarction based on clinical, electrocardiographic, and biochemical findings; and
- provided written informed consent.

2.7 Exclusion Criteria

Patients were excluded if they:

- were younger than 18 years or older than 80 years;
- had undergone recent surgery or experienced recent trauma;
- had another condition likely to influence serum biomarker levels;
- were pregnant or lactating; or
- were unwilling to provide written informed consent.

2.8 Sample Collection

Blood samples were collected at two time points:

- **At 0 hour:** immediately at hospital admission.
- **At 12 hours:** 12 hours after hospital admission.

Blood was collected into plain vacutainers. Serum was separated by centrifugation and used for biochemical analysis.

2.9 Estimation of Malondialdehyde

Serum MDA was estimated using the Kei Satoh method. MDA was considered a biochemical indicator of lipid peroxidation and oxidative damage.

2.10 Estimation of Superoxide Dismutase

Serum SOD activity was determined using the Marklund and Marklund method. SOD was assessed as an important enzymatic component of the endogenous antioxidant defense system.

2.11 Statistical Analysis

The results were expressed as mean $\pm$ standard deviation (SD). MDA and SOD values at admission and 12 hours were compared using the paired t-test. A p-value of <0.05 was considered statistically significant.

3. Results

A total of 110 patients with myocardial infarction were included in the study. Oxidative stress and antioxidant defense were assessed by measuring serum MDA and SOD at admission and 12 hours after admission.

Table 1. Early changes in MDA and SOD in patients with acute myocardial infarction

Biomarker	At admission (0 hour) Mean $\pm$ SD	At 12 hours Mean $\pm$ SD	p-value
MDA (nmol/mL)	9.6652 $\pm$ 0.9244	6.4385 $\pm$ 1.5219	<0.0001
SOD (U/mL)	83.4033 $\pm$ 8.9211	127.1918 $\pm$ 16.5676	<0.0001

3.1 MDA

The mean serum MDA concentration at admission was 9.6652 $\pm$ 0.9244 nmol/mL. At 12 hours after admission, the mean MDA concentration decreased to 6.4385 $\pm$ 1.5219 nmol/mL.

This decrease was statistically highly significant ($p < 0.0001$), indicating a significant change in lipid peroxidation during the first 12 hours following admission.

3.2 SOD

The mean SOD activity at admission was 83.4033 ± 8.9211 U/mL. At 12 hours, SOD activity increased to 127.1918 ± 16.5676 U/mL.

The increase in SOD activity was statistically highly significant ($p < 0.0001$), demonstrating a significant change in enzymatic antioxidant activity during the same period.

3.3 Overall Oxidative-Antioxidant Pattern

The two biomarkers demonstrated opposite temporal changes. MDA decreased from admission to 12 hours, whereas SOD activity increased during the same period.

Thus, the early biochemical pattern was characterized by:

MDA ↓ → reduced lipid peroxidation marker

SOD ↑ → increased antioxidant enzyme activity

This pattern indicates a significant dynamic change in the oxidative-antioxidant state during the first 12 hours after admission.

4. Discussion

The present study evaluated the early kinetics of two complementary biomarkers of redox status-MDA as a marker of lipid peroxidation and SOD as an important antioxidant enzyme-in patients with acute myocardial infarction. A significant decrease in MDA and a significant increase in SOD activity were observed between admission and 12 hours after admission.

4.1 Oxidative Stress in Acute Myocardial Infarction

Oxidative stress is an important component of the biochemical response to myocardial ischemia. During myocardial infarction, interruption of oxygen supply produces profound metabolic disturbances within cardiomyocytes. Alterations in mitochondrial function, electron transport, cellular metabolism, inflammatory responses, and subsequent restoration of blood flow can contribute to increased ROS generation.^{2,3}

Excessive ROS can attack membrane polyunsaturated fatty acids and initiate lipid peroxidation. This process can generate several reactive aldehydes, including MDA. Therefore, measurement of MDA provides an indirect indication of oxidative lipid damage.

Previous research has demonstrated an association between increased oxidative stress and myocardial infarction. Madole et al. reported altered oxidant and antioxidant status in patients with acute myocardial infarction, supporting the involvement of oxidative imbalance during the acute phase of myocardial injury.⁵

Aladağ et al. also evaluated oxidant and antioxidant markers, including MDA and SOD, in patients with STEMI and NSTEMI and demonstrated significant alterations in oxidative and antioxidant status.⁷ These findings support the concept that myocardial infarction is accompanied by measurable changes in systemic redox balance.

4.2 Significance of MDA in Myocardial Infarction

In the present study, MDA was highest at admission, with a mean concentration of 9.6652 ± 0.9244 nmol/mL, and subsequently decreased to 6.4385 ± 1.5219 nmol/mL at 12 hours. The difference was highly statistically significant ($p < 0.0001$).

The elevated MDA concentration at admission suggests the presence of increased lipid peroxidation during the early phase of myocardial infarction. Oxidative attack on membrane lipids can contribute to disruption of membrane integrity and generation of secondary lipid peroxidation products.

Nag Sundaram et al. investigated serum MDA in patients with acute myocardial infarction and reported increased MDA associated with myocardial infarction, supporting the use of MDA as an indicator of lipid peroxidative stress.⁴

The decrease in MDA observed in the present study between admission and 12 hours indicates that the oxidative state was dynamically changing during the early clinical period. A reduction in MDA may reflect decreasing lipid peroxidation following initial stabilization and treatment; however, the present observational study cannot establish the specific mechanism responsible for this decline.

Savovic et al. investigated redox-status biomarkers in patients presenting with STEMI or NSTEMI and reported dynamic changes in oxidative stress-related biomarkers during the early clinical course.⁷ Their findings support the importance of considering the timing of oxidative stress biomarker measurement when interpreting redox status in acute coronary syndromes.

4.3 Significance of SOD in Myocardial Infarction

SOD constitutes an important component of the endogenous antioxidant defense system. Its major function is the conversion of superoxide radicals into hydrogen peroxide and molecular oxygen, thereby limiting the

accumulation of superoxide within cells.

In the present study, SOD activity increased significantly from 83.4033 ± 8.9211 U/mL at admission to 127.1918 ± 16.5676 U/mL at 12 hours ($p < 0.0001$).

The increase in SOD activity indicates a significant change in enzymatic antioxidant defense during the early period following myocardial infarction. The increase may represent activation or mobilization of endogenous antioxidant mechanisms in response to oxidative stress.

Rotariu et al. described oxidative stress as an important feature of cardiovascular and metabolic disorders and highlighted the role of endogenous antioxidant systems, including SOD, in controlling excessive free-radical activity.¹

Aladağ et al. also evaluated SOD together with MDA and other oxidative stress-related parameters in patients with myocardial infarction, demonstrating the importance of examining oxidant and antioxidant components together rather than considering either component in isolation.⁵

4.4 Relationship Between MDA and SOD

An important finding of the present study was the simultaneous decrease in MDA and increase in SOD during the first 12 hours.

MDA represents the downstream consequence of oxidative damage to lipids, whereas SOD represents an important enzymatic antioxidant defense mechanism. Therefore, assessment of both markers provides complementary information about the oxidative-antioxidant balance.

The observed pattern can be summarized as:

Acute myocardial injury → increased oxidative stress → lipid peroxidation → increased MDA
followed by an early change in antioxidant defense:

Antioxidant response → increased SOD activity → reduction in oxidative lipid damage → decreased MDA

This sequence should be interpreted as a biological interpretation of the observed biomarker pattern rather than proof of a direct causal relationship, because the present study did not experimentally investigate the underlying molecular mechanisms.

4.5 Early Kinetic Changes in Oxidative Stress

One of the important aspects of the present study is the assessment of MDA and SOD at two defined time points rather than at a single time point.

At admission, the oxidative stress profile was characterized by a relatively higher MDA concentration and lower SOD activity. At 12 hours, MDA had significantly decreased while SOD activity had significantly increased. These findings indicate that oxidative stress and antioxidant defense are dynamic processes during the early phase of myocardial infarction. The redox state observed in a patient may therefore depend partly on the timing of sample collection.

Kong et al. highlighted the potential importance of oxidative stress-related parameters in cardiovascular disease and described the involvement of oxidative imbalance in cardiovascular disease development and progression.⁹ Similarly, Sun et al. identified oxidative stress-related biomarkers associated with acute myocardial infarction, supporting the relevance of redox-related biochemical alterations in AMI.¹⁰

4.6 Clinical and Research Significance

The findings of this study indicate that MDA and SOD provide complementary information about oxidative stress in patients with acute myocardial infarction.

MDA may provide information regarding the extent of lipid peroxidation, while SOD provides information regarding enzymatic antioxidant defense. Their simultaneous assessment may therefore provide a broader picture of the redox state than assessment of either marker alone.

However, the present study evaluated only the first 12 hours after admission and did not include long-term clinical outcomes. Therefore, the findings should be interpreted primarily as evidence of early oxidative and antioxidant kinetic changes rather than as definitive evidence of long-term prognosis.

Future studies involving larger sample sizes, healthy controls, serial measurements over longer periods, and clinical outcomes such as left ventricular function, heart failure, arrhythmias, recurrent infarction, and mortality may help determine whether early MDA and SOD patterns have prognostic significance.

5. Conclusion

The present study demonstrated significant early changes in oxidative stress and antioxidant defense in patients with acute myocardial infarction. Serum MDA, a marker of lipid peroxidation, significantly decreased at 12 hours, while SOD activity significantly increased during the same period. The simultaneous decrease in MDA and increase in SOD indicate a dynamic change in the oxidative-antioxidant balance during the early

phase of myocardial infarction. These findings support the biological relevance of oxidative stress and endogenous antioxidant defense in AMI and demonstrate that the redox state changes considerably during the first 12 hours after admission.

MDA and SOD may therefore serve as complementary biochemical indicators for studying early oxidative stress and antioxidant responses in myocardial infarction. Further longitudinal studies incorporating larger populations, control groups, additional oxidative stress markers, and clinical outcomes are required to determine their potential prognostic significance.

8. References

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