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Baykulov Azim Kenjayevich*

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Research Article

Oxidative Stress and Lipid Peroxidation in Experimental Myocardial Infarction: Biochemical Evaluation in Cardiac and Hepatic Tissue

Baykulov Azim Kenjayevich

Department of Pharmaceutical and Toxicological Chemistry, Samarkand State Medical University, Uzbekistan, Samarkand.

***Corresponding Author:** Baykulov Azim Kenjayevich, Department of Pharmaceutical and Toxicological Chemistry, Samarkand State Medical University, Uzbekistan, Samarkand.

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Abstract

Introduction: Myocardial infarction (MI) remains one of the most common and life-threatening cardiovascular diseases worldwide. Experimental models of MI are essential for understanding its pathophysiology and evaluating therapeutic interventions. Objective: The aim of this study was to evaluate lipid peroxidation and antioxidant defense during experimental MI in rats. Methods: MI was induced in adult male rats by ligation of the left coronary artery. On the third day after MI, cardiac and liver tissues were collected. Heart and liver homogenates, as well as mitochondrial (MF) and microsomal (MSF) fractions of the liver, were analyzed for malondialdehyde (MDA) concentration and superoxide dismutase (SOD) activity.

Results: Compared with the control group, MDA concentration increased fivefold in cardiac homogenates and 6.1-fold in liver MF, while MSF showed the most pronounced increase (28.2-fold). SOD activity was significantly reduced: by 31.5% in MF and by 73.1% in MF. Conclusion: Experimental MI induces pronounced oxidative stress, characterized by excessive lipid peroxidation and suppression of antioxidant defenses. This model represents a valuable tool for investigating cardioprotective strategies and antioxidant therapy.

Keywords: myocardial infarction; oxidative stress; lipid peroxidation; malondialdehyde; superoxide dismutase; experimental model

Introduction

Cardiovascular diseases remain the leading cause of death worldwide, accounting for over 17 million deaths annually and representing a substantial global health and economic burden. Among them, myocardial infarction (MI) remains one of the most acute and life-threatening conditions, characterized by irreversible ischemic necrosis of cardiac tissue, which often leads to heart failure, severe disability, or death. Despite significant progress in interventional cardiology and pharmacotherapy, the morbidity and mortality associated with MI remain unacceptably high. This underscores the urgent need for a deeper understanding of the molecular and biochemical processes that drive myocardial damage, which could lead to the development of novel cardioprotective strategies [1-6].

Recent advances in experimental modeling provide researchers with robust and reproducible tools to mimic MI in laboratory animals, enabling the investigation of pathophysiological Given the close interplay between oxidative damage and antioxidant defense systems, evaluating the balance between pro-oxidant and antioxidant mechanisms is essential for identifying critical windows of therapeutic intervention [16-18].

Therefore, the aim of this study was to investigate lipid peroxidation levels and antioxidant system activity in both cardiac and hepatic tissues during the early phase of experimental MI, with the goal of identifying biochemical targets for future cardioprotective therapies.

Materials and Methods

Twenty-five adult male white outbred rats (200 ± 10 g) were housed under standard laboratory conditions (22 ± 2°C, 12 h light/dark cycle) with free access to food and water. All experimental procedures complied with institutional and international guidelines for the care and use of laboratory animals (EU Directive 2010/63/EU). MI was induced by permanent ligation of the left coronary artery under general anesthesia (ketamine/xylazine). The animals were observed for 72 hours, after which they were euthanized by decapitation.

Heart and liver tissues were rapidly excised, washed in ice-cold 0.15M KCl, blotted, and weighed. Heart homogenates were prepared in a Teflon-glass homogenizer. Liver homogenates were pressed through a 0.5 mm mesh and homogenized in 0.05M KCl (pH=7.4). Mitochondrial fractions were obtained by differential centrifugation (9000g×20min), and microsomal fractions by ultracentrifugation (105,000g×60min).

MDA concentration was determined by the TBARS method according to Stalnaya et al., and SOD activity by the method of Misra and Fridovich (modified by Brusov et al.). Data are expressed as mean ± SEM. Statistical comparisons were made using Student's t-test, with $p < 0.05$ considered significant.

Results

Baseline measurements in control rats revealed relatively low MDA levels in cardiac tissue, consistent with a normal redox balance and minimal oxidative stress under physiological conditions. Significant, but stable, levels of lipid peroxidation products were observed in both mitochondrial (MF) and microsomal (MSF) liver fractions, reflecting basal membrane turnover and ROS generation typical for metabolic activity in healthy animals.

On day 3 post-MI, MDA levels rose sharply in all studied samples, indicating a marked activation of membrane lipid peroxidation. In cardiac homogenates, MDA concentration increased more than fivefold compared with controls ($p < 0.01$), demonstrating extensive oxidative damage to cardiomyocyte membranes. In the liver, MDA levels increased 6.1-fold in MF and an impressive 28.2-fold in MSF, suggesting that microsomal membranes were particularly vulnerable to ROS-induced peroxidation during systemic response to MI

Tissue/Fraction	MDA (nmol/mg protein·min)	Ascorbate-Dependent Peroxidation (ADP)	NADH-Dependent Peroxidation (NDP)	ADP/NDP Ratio
Heart	1.65 ± 0.02	3.33 ± 0.11	6.71 ± 0.17	0.49 ± 0.03
Liver MF	8.10 ± 0.11	88.3 ± 0.73	64.5 ± 0.39	1.36 ± 0.11
Liver MSF	1.43 ± 0.06	11.2 ± 0.09	31.4 ± 0.29	0.35 ± 0.01

Table 1: MDA concentration in heart and liver fractions (control rats).

Post-MI Changes

On day 3 post-MI, MDA levels rose sharply (Table 2), indicating increased membrane lipid peroxidation.

Group	MDA (nmol/mg protein·min)	ADP	NDP	ADP/NDP Ratio
Control	1.65 ± 0.02	3.33 ± 0.11	6.71 ± 0.17	0.49 ± 0.03
MI (Day 3)	7.42 ± 0.04	20.64 ± 0.14	82.53 ± 0.02	0.25 ± 0.02

Table 2: MDA concentration in cardiac tissue (3 days post-MI).

Similar increases were observed in mitochondrial and microsomal fractions of the liver, with MSF showing the greatest change (Table 3).

Fraction	Control	MI (Day 3)	Fold Increase
MF	8.10 ± 0.11	59.9 ± 1.73	×6.1
MSF	1.43 ± 0.06	28.8 ± 1.02	×28.2

Table 3: MDA concentration in liver fractions (3 days post-MI).

In parallel with LPO activation, a significant suppression of the antioxidant defense system was observed. SOD activity declined by 31.5% in MF and by 73.1% in MSF ($p < 0.01$), indicating a profound weakening of the first-line enzymatic protection against superoxide radicals. This imbalance between excessive ROS generation and diminished antioxidant capacity reflects severe oxidative stress and may contribute to further propagation of cellular damage, not only in the myocardium but also in the liver as a key metabolic organ.

The combination of markedly elevated MDA and decreased SOD activity highlights a critical redox imbalance during the early post-infarction period, which may play a decisive role in the progression of myocardial injury and systemic complications.

Discussion

Our study demonstrates that experimental myocardial infarction (MI) in rats is accompanied by a pronounced activation of free radical lipid peroxidation (LPO) and a concomitant decrease in antioxidant enzyme activity. On day 3 post-MI, MDA levels in cardiac homogenates increased more than fivefold compared with controls, indicating severe oxidative damage to cardiomyocyte membranes. A similar pattern was observed in the liver, where MDA concentration rose 6.1-fold in the mitochondrial fraction (MF) and as much as 28.2-fold in the microsomal fraction (MSF). These findings confirm that MI induces not only local but also systemic oxidative stress, affecting organs distant from the primary ischemic site.

The observed decrease in superoxide dismutase (SOD) activity -31.5% in MF and 73.1% in MSF-suggests a significant depletion of antioxidant defenses. This is consistent with reports by other researchers showing that post-infarction oxidative stress is characterized by an imbalance between reactive oxygen species (ROS) production and detoxification capacity, leading to further propagation of cellular damage [12].

Our results align with previous studies demonstrating that excessive ROS generation during MI can trigger lipid, protein, and nucleic acid oxidation, mitochondrial dysfunction, and eventual cell death. The particularly high vulnerability of microsomal membranes to peroxidation observed in our work may be explained by their structural and biochemical properties, including the presence of oxygen-dependent monooxygenase systems and high levels of polyunsaturated fatty acids, as noted in earlier biochemical studies [18]

Taken together, these findings highlight oxidative stress as a key pathogenic factor in post-infarction myocardial remodeling and systemic metabolic disturbances. Our data, in agreement with literature reports,

suggest that therapeutic modulation of redox status—via exogenous antioxidants, SOD mimetics, or mitochondrial stabilizers—could mitigate the extent of oxidative injury and improve recovery after MI. Future research should focus on standardizing experimental models to assess the efficacy of such interventions and to clarify their translational potential for clinical use.

Conclusion

Experimental myocardial infarction in rats causes a profound increase in lipid peroxidation and suppression of antioxidant defense mechanisms. These results support the utility of this model for studying cardioprotective interventions and screening antioxidant compounds.

Abbreviations

MI– Myocardial Infarction

ROS – Reactive Oxygen Species

LPO – Lipid Peroxidation

MDA – Malondialdehyde

SOD – Superoxide Dismutase

MF – Mitochondrial Fraction

MSF – Microsomal Fraction

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Data Availability Statement

The data are available from the author upon request.

Conflicts of Interest

The author declares no conflicts of interest.

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