



THE ROLE OF FREE RADICAL LIPID OXIDATION IN METABOLIC DISORDERS IN EXPERIMENTAL MYOCARDIAL INFARCTION.

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Abstract. *The present work is devoted to the study of the role of free radical oxidation of lipids in the development of metabolic disorders in experimental myocardial infarction. The conducted studies have shown that oxidative stress caused by excess free radicals promotes the activation of lipid peroxidation (LPO), disruption of the antioxidant system and destabilization of cell membranes. These processes play a key role in the pathogenesis of ischemic myocardial injury and aggravate metabolic disorders, including lipid metabolism imbalance, inhibition of energy metabolism and the development of secondary structural changes. It was found that the severity of LPO processes directly correlates with the severity of cardiac muscle damage and disorders of systemic homeostasis regulation. The obtained data emphasize the importance of timely antioxidant correction in the complex therapy of myocardial infarction. Thus, free radical oxidation of lipids is considered as one of the key pathogenetic mechanisms of metabolic disorders, which opens up prospects for the development of new directions in the treatment and prevention of cardiac diseases.*

Key words: *endothelial dysfunction, atherosclerosis, autoimmune processes, hypercholesterolemia, hyperlipoproteinemia.*

Introduction.

Free radical lipid oxidation plays a crucial role in the development of metabolic disorders associated with myocardial infarction. In experimental models, excessive generation of reactive oxygen species (ROS) leads to the activation of

lipid peroxidation, damaging cellular membranes and disrupting metabolic homeostasis. The imbalance between pro-oxidant and antioxidant systems intensifies oxidative stress, contributing to impaired lipid metabolism, mitochondrial dysfunction, and energy deficiency in cardiac tissues. These changes exacerbate myocardial injury and promote further progression of pathological processes. The extent of lipid peroxidation correlates with the severity of myocardial damage and systemic metabolic disturbances. Research findings emphasize the importance of controlling oxidative stress through antioxidant therapy, which may mitigate the metabolic consequences of myocardial infarction. Understanding the mechanisms of free radical lipid oxidation provides valuable insights into the pathogenesis of cardiovascular diseases and offers promising approaches for their treatment and prevention. Thus, targeting oxidative pathways could enhance therapeutic outcomes in myocardial infarction [1-6].

In the study of metabolic processes in the heart during myocardial infarction, experimental modeling played a major role. Pathological phenomena that occur when the blood supply to the heart is disrupted are primarily associated with focal oxygen deficiency, leading to disruption of oxidation-reduction processes and all subsequent metabolic disorders in the affected organ and in the body as a whole. Disorganization of metabolic processes, disruption of the state of non-specific protection of the body and a decrease in its regenerative capabilities are the initiators of free-radical oxidation of lipids in the body. We studied lipid peroxidation in the body of rats during experimental myocardial infarction [7-21].

Materials and methods. The experiments were performed on white mongrel male rats [n=25] weighing 200 grams. Myocardial infarction was induced by ligation of the left coronary artery. The animals were sacrificed by decapitation on the 3rd day from the onset of the experimental myocardial infarction.

After the animals were sacrificed, the heart and liver were quickly removed, weighed, washed with a cold 0.15 M solution [0-4°C] KCl, and then homogenates were prepared for biochemical analysis.

Heart homogenates were prepared by grinding the organ with a scalpel, followed by destruction of the heart cells in a glass homogenizer with a Teflon pestle.

Liver homogenates were prepared by pressing the liver through a grid with 0.5 mm holes using a press. After this, the liver cells were also destroyed in a glass homogenizer with a Teflon pestle. The isolation medium consisted of 0.05 M KCl dissolved in 50 ml Tris-HCl buffer (Ph=7.4). Mitochondrial (MX) and microsomal (MS) fractions were isolated from liver homogenates.

The mitochondrial fraction was obtained by differential centrifugation at 9000 g for 20 minutes.

The liver microsomal fraction was obtained by repeated centrifugation of the supernatant for 60 minutes at 105,000 q.

The amount of MDA in heart homogenates, MX and MS fractions of the liver were determined by the method of Stalnaya I.D. et al., and the activity of superoxide dismutase (SOD) by the method of Mirsa P.H., Fridovich S. as modified by Brusov O.S. et al.

Results and Discussion.

Analyzing the data on the intensity of LPO in homogenates of the heart, MX and MS fractions of the liver of intact rats, we can conclude that these tissues are characterized by a certain level of SROL, determined by the features of the structure and functions performed. At the same time, the rate of LPO reactions is stationary low due to the action of a powerful antioxidant defense system of the body.

Studying the intensity of LPO in rats with experimental myocardial infarction revealed its significant activation (3 days after ligation of the left coronary artery).

The content of MDA in homogenates of heart tissue was increased 5 times relative to the control. A sharp activation of LPO in heart tissue on the 3rd day after reproducing coronary occlusive myocardial infarction indicates pronounced membrane-destructive processes in the cells of the cardiac tissue, as well as their possible death.

An increase in the concentration of MDA was also observed in the MX and MS fractions of the liver of experimental rats. The increase in LPO in the MX and MS fractions of the liver was probably associated with the absorption of cell breakdown products.

Analysis of the results of our studies serves as a basis for finding agents with protective properties in the activation of LPO. Initiation of free-radical lipid oxidation in experimental myocardial infarction and its metabolic disorders.

Conclusion

The intensity of lipid peroxidation (LPO) in the tissues of the heart and liver in intact animals is characterized by a stable low level due to the effective work of the antioxidant system of the body.

In the conditions of experimental myocardial infarction, pronounced activation of LPO is observed, especially on the 3rd day after ligation of the left coronary artery.

The content of malondialdehyde (MDA) in the cardiac tissue increases 5 times compared to the control, which indicates pronounced membrane-destructive processes and death of myocardial cells.

An increase in the concentration of MDA was also noted in the microsomal (MX) and mitochondrial (MS) fractions of the liver, probably due to the effect of cell decay products.

The data obtained confirm the key role of free radical lipid oxidation in the development of metabolic disorders in myocardial infarction.

The results of the studies substantiate the need to search for and use agents with antioxidant and membrane-stabilizing properties in the complex therapy of myocardial infarction.

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