DYNAMICS OF THE INFLUENCE OF LACTATE DEHYDROGENASE DURING EXPERIMENTAL MYOCARDIAL INFARCTION

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Annotation.*Cardiovascular diseases and atherosclerosis remain the same cause of disability and mortality in the world. Study of vascular dysfunction of the vascular endothelium and hyperlipoproteinemia in experimental atherosclerosis. The experiment was carried out on 28 rabbits with an average chinchilla weight of 2.5-3.0 kg. The effect of the drug is clear and dynamic: the first 3 months and 1 month after the first dose. The results obtained were compared with the results of the control and intact groups. Long-term use of cholesterol is accompanied by the development of hypertriglyceridemia , the effect of which depends on the duration of the experiment. An important role in the development of endothelial dysfunction in hypercholesterolemia is played by a decrease in the synthesis of endothelial nitric oxide and an increase in its active radicals, modification of low-density lipoproteins and their deposition in the vascular endothelium.*

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

Introduction. According to the World Health Organization (2020), "...cardiovascular diseases are becoming the leading cause of death in the world..." [1]. Myocardial infarction is one of the leading causes of death and disability. In developed countries, 20% of patients diagnosed with myocardial infarction die before emergency medical care is provided, and the 30-day mortality rate reaches up to 30%.

One of the characteristic signs of many diseases is a change in enzyme activity.

An increase in enzyme activity, as is known, may be a consequence of activation of the enzyme in the blood serum as a result of its structural rearrangement, changes in the conditions that control the function of this catalytic protein, or is the result of additional intake from tissues due to a process leading to impaired permeability, destruction of cellular and subcellular membranes structures [2-5].

Aim. This work is devoted to elucidating the relationship between an increase in enzyme activity and metabolic changes, as well as studying the possibility of influencing metabolic processes by introducing the enzyme from the outside.

Materials and methods. Experiments were carried out on 25 rabbits weighing 3.5-4.0kg. Myocardial infarction was induced by ligation of the left coronary artery. On day 3, experimental animals were intravenously injected with the enzyme preparation lactate dehydrogenase at a dose of 5000E per 1 kg of body weight. 1 hour after the introduction of the enzyme, the animal was killed by decapitation and the heart and liver were removed. Mitochondria from the infarction zone and liver tissue were isolated by differential centrifugation in a 0.25 m sucrose solution. The activity of lactate dehydrogenase (LDH) and aldolase was determined in mitochondria and supernatant of cardiac tissue and liver [6-10].

The content of reduced and oxidized metabolic products was studied in the deproteinized tissue extract. The enzyme activity was assessed using an SF-46 spectophotometer [11-14].

Results and discussion. From the results of our studies it follows that in intact animals, the activity of lactate dehydrogenase in the supernatant of cardiac tissue was equal to 1.923 + 0.134 μmol NAD-H/mg/min, and in mitochondria - 0.089 + 0.002 μmol NAD-H/mg/min, i.e. LDH activity in the supernatant of these animals was 21.6 times higher than in mitochondria.

In rabbits with coronary-occlusive myocardial infarction, in both fractions studied, LDH activity decreases, and in the supernatant by 1.49 times (1.288 ±

0.94 μmol NADH-/mg/min), and in mitochondria by 1.85 times (0.048±0.001 μmol/NADH/mg/ min) compared to control values .

After administration of LDH to rabbits with myocardial infarction, enzyme activity increased both in the supernatant and in the mitochondria of cardiac tissue by 1.27 (1.643±0.086 μmol/NADH/mg/min) and 1.60 times (0.077±0.003 μmol/NADH/mg/min) respectively, compared with those in control rabbits.

The accumulation of lactic acid and associated pH shifts are one of the main reasons leading to the development of arrhythmia in the acute period of myocardial infarction. An increase in LDH activity during hypoxia facilitates the use of lactic acid by ischemic myocardium, which is associated with an acceleration of the oxidation reaction of lactic acid into pyruvic acid, which then enters the Krebs cycle.

Intravenous administration of exogenous LDH causes increased glycolytic breakdown of glucose in the liver. In the cytosolic fraction, aldolase activity increases by 74%, LDH by 26%. In liver mitochondrial fractions, the function of membrane-bound LDH sharply increases, while aldolase remains virtually unchanged.

Activation of the glycolytic breakdown of glucose is accompanied by intense release of dihydroxyacetone phosphate (-39%), increased use of pyruvate (-83%), and accumulation of lactic acid in the liver (+111.7%). The content of *a* -glycerophosphate increased by 32%.

The dependence of changes in liver metabolism caused by administered LDH is confirmed by a predominant shift in the glycolytic enzyme-substrate system.

oxidase activity in the microsome fraction compared to the control , indicating an increase in oxidative detoxification processes with the participation of NADPH. Therefore, administration of exogenous LDH activates the microsomal oxidative defense mechanism under these experimental conditions [15].

Thus, the above data indicate that in the acute period of MI (3rd day of the experiment), LDH activity in the affected cardiac tissue decreases significantly . There is a violation of the relationship between oxidized and reduced intermediate metabolic products, which is accompanied by the accumulation of a certain type of metabolites with a general energy deficit [16-17].

Conclusion. LDH introduced into the body is actively involved in metabolism, initiates the activity of associated enzyme systems of cardiac tissue, ensures the transformation of hydrogen, the main source of ATP in living systems, determines the direction of metabolic flows, optimal electrical balance and energy supply of tissues.

The identified peculiarity of the effect of the enzyme introduced into the body can be used in the correction of metabolic disorders characteristic of myocardial infarction.

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