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# CELLULAR CHANGES IN CARDIOMYOCYTES DUE TO ISCHEMIA AND NECROSIS

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**ANNOTATION:**

**In the biochemical study of the blood of patients with hypoxic myocardial ischemia, an increase in the MV fraction of troponins of creatine phosphokinase (CPK-MB) and lactate dehydrogenase (LDH) is observed.**

**Keywords: cardiomyocytes, secretory, rhythm, ischemia, necrosis, infarction.**

**INTRODUCTION:**

The transverse muscle tissue forms the myocardial layer of the heart. This type of muscle tissue, unlike skeletal muscle, is made up of cardiomyocytes, not heart muscle cells. 3 different cardiomyocytes can now be distinguished. Typical, impulsive-atypical and secretory. Reduction cardiomyocytes are cylindrical cells with a length of 50-120 microns and a width of 15-20 microns. They connect to each other to form functional fibers. In the center of cardiomyocytes there are one or two nuclei of an oval or elongated shape. Myofibrils are located around the nucleus, with a large number of mitochondria. The smooth endoplasmic reticulum and the T-system are well developed. Cardiomyocytes are covered with a sarcolemma, which is surrounded by a plasma membrane and a basement membrane, respectively. The basement membrane is not located in the area of the intervertebral discs, it only laterally surrounds the cardiomyocytes. The intercellular plates are located stepwise between the plasma membranes of two cells. In the area of intervertebral discs, cardiomyocytes fuse through desmosomes, a cleft palate (nexus), and intervertebral bridges. Actin protofibrils of myofibrils enter the intermediate plates. The structure of myofibrils resembles the tissue of the transverse skeletal muscles. Excitatory cardiomyocytes (Purkinje fibers or atypical cardiomyocytes) in the heart are larger than contractile cardiomyocytes (100 μm in length and 50 μm in width) and transmit excitation from cells that control the rhythm (pacemakers) to contracting muscle fibers. Pacemaker cells are a special type of atypical cardiomyocytes that are innervated by fibers of the autonomic nervous system. If cardiomyocytes in infants have the ability to divide, cardiomyocytes that die due to the lack of satellite cells in an adult and the elderly, as well as the loss of the ability of cardiomyocytes to divide, will not be restored and a connective tissue scar will form in their place (with myocardial infarction).

Myocardial infarction is a cardiovascular disease: infarction caused by impaired coronary circulation (arterial atherosclerosis, thrombosis, spasm). The development of myocardial infarction is caused by hypertension, diabetes, obesity, and smoking, and inactivity, physical and mental stress. In most cases, myocardial infarction occurs against the background of angina pectoris. One of the main symptoms of myocardial infarction is a decrease in the amount of oxygen and

nutrients in a specific part of the heart muscle as a result of less than one blood flow. It is known that myocardial infarction is the death of cardiomyocytes due to ischemia. The most pathognomonic sign that a really acute necrosis has occurred in the myocardium is an increase in specific markers in the blood.

Changes in ischemic necrosis, which can be detected under an electron microscope, appear much earlier. For example, 10 minutes after the onset of ischemia, mitochondria in cardiomyocytes swell (indicating that the cells are damaged by hypoxia), their matrix thickens and the integrity of the membranes is disrupted, which is a sign that the cell has undergone irreversible changes. Histochemical and biochemical changes occurring at the site are also known at an early stage. After an hour of necrosis, the amount of potassium and glycogen sharply decreases, the activity of the enzymes succinate dehydrogenase and cytochrome oxidase decreases.

Markers are usually found inside cardiomyocytes and enter the bloodstream only when their membrane is destroyed. The activity of the enzymes CPK, MVKFK, LDH, transaminases in the blood really increases with the destruction of myocardial cells.

Troponin levels also increase with very small myocardial infarctions, biochemical diagnostics (less than 1.0 g). This made it possible to recognize myocardial infarction; in about a quarter of patients, only stable angina was diagnosed with this test. Elevated levels of cardiospecific troponins T and I are associated with both the risk of death and the development of complications of other clinically significant diseases.

Rapid recovery of myocardial perfusion is accompanied by a complex of metabolic and structural changes that can be spontaneously reversed, but in some cases this causes acute heart failure.

In everyday practice, a polarizing microscope should be used to diagnose apoptosis of cardiomyocytes, since this is the most informative and sensitive method.

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