PATHOLOGICAL PHYSIOLOGY OF CORONARY HEART DISEASE

Rakhimova Gulnoz Shamsiyevna
Bukhara State Medical Institute
https://orcid.org/0009-0003-5023-0656
raximova.gulnoz@bsmi.uz

Annotation. Ischemic heart disease is a disease that develops as a result of absolute insufficiency of the coronary circulation and manifests itself by myocardial damage of varying degrees. Insufficiency of the coronary circulation is a pathological condition characterized by the inability of the coronary vessels to provide blood supply to the heart in accordance with its energy needs. In other words, there is a discrepancy between the energy needs of the heart and the delivery of oxygen and nutrients by the coronary vessels. Insufficiency of the coronary circulation can be relative and absolute.

Keywords: ischemia, coronary arteries, heart failure, hypoxia, myocard.

Introduction: Relative coronary insufficiency occurs in the case of a primary increase in the energy needs of the heart (an increase in the load on the heart during physical work, arterial hypertension). At the same time, the intensity of the coronary blood flow may increase, but this turns out to be insufficient to meet the increased needs of the heart. Absolute coronary insufficiency occurs in the case of a primary violation of the coronary circulation, as a result of which the delivery of oxygen and nutrients to the myocardium decreases both at rest and with an increase in the energy needs of the heart.

Causes of myocardial ischemia:

I. The obturation mechanism is a decrease in the lumen of the coronary arteries. Its causes may be:

a) stenosing atherosclerosis (is the cause of myocardial ischemia in 90% of cases);

b) thrombosis of the coronary arteries (most often a consequence of atherosclerosis);

c) embolism of the coronary arteries;

d) inflammatory processes in the wall of the heart vessels - coronariitis (with rheumatism, syphilis).

For the development of clinical signs of myocardial ischemia, the value of "critical stenosis" is important. This is a minimal decrease in the lumen of the vessels, in which ischemia occurs. In humans, this indicator is 75%.

II. Angiospastic mechanism - spasm of coronary vessels. Its causes may be: a) excitation of α-adrenoreceptors against the background of blockade of β-

adrenoreceptors; b) vasopressin; c) angiotensin II; d) thromboxane A1; e) hypocapnia; e) endothelin is a biologically active substance of endothelial origin. A known clinical form of coronary angiospasm is Prinzmetal angina.

III. Compression mechanism - compression of the coronary vessels. It may occur with tachycardia (the total duration of the period of compression of the coronary vessels during the systole of the heart increases). Sometimes it is caused by scars and tumors.

Pathogenetic factors in coronary insufficiency:

1. Hypoxia.

2. Acidosis. It develops as a result of the accumulation of acidic metabolic products due to impaired blood outflow and activation of glycolysis.

3. An increase in the extracellular concentration of potassium ions in the ischemic area. It is noted from the very beginning of disorders of the coronary circulation (after a few minutes), when there is still no damage to cardiomyocytes. During this period, for reasons still unknown, there is a passive release of potassium ions from cells (the permeability of K^+ channels may increase), and its extracellular concentration increases to 8 mmol/ l or more. After a few hours, the "outflow" of K^+ ions from the cells may be due to a malfunction of the Na-K pumps (ATP deficiency) and an increase in the permeability of damaged membranes.

Consequences of coronary circulatory insufficiency:

1. Violation of myocardial contractility with the development of heart failure.

2. The appearance of abnormal electrical activity – electrical instability of the heart, the development of arrhythmias.

3. Damage to cardiomyocytes caused by ischemia.

4. Reperfusion syndrome.

The main clinical forms of ischemic heart disease:

1) angina pectoris - attacks of short-term (up to 20 minutes) acute coronary insufficiency, which are accompanied by pain syndrome, a sense of fear and related vegetative reactions. There are tension angina, rest angina and Prinzmetal angina (coronary artery spasm);

2) preinfarction condition (unstable angina, intermediate coronary syndrome, or acute focal myocardial dystrophy) - develops with a duration of myocardial ischemia from 20 to 40 minutes;

3) myocardial infarction - necrosis of the heart muscle caused by disorders of the coronary circulation. Occurs with reversible (transient) ischemia lasting over 40-60 minutes, or with irreversible disorders of coronary blood flow;

4) cardiosclerosis - sclerotic changes of the heart muscle.

They can be diffuse (atherosclerotic cardiosclerosis) and focal (postinfarction cardiosclerosis).

The main causes of myocardial infarction:

1. Atherosclerosis of the coronary arteries. Its development is accompanied by a violation of the supply of oxygen to the myocardium.

2. Increased load on the heart (physical strain, hypertension). At the same time, the heart's need for oxygen increases.

3. Stress. The role of catecholamines in the development of myocardial infarction:

A. Violation of coronary circulation. This is due to the fact that catecholamines:

a) contribute to the development of atherosclerosis;

b) cause contractural spasm of the smooth muscles of the coronary arteries (catecholamine damage to the cells of the vascular wall);

c) activate thrombosis and blood clotting.

B. Increase the oxygen demand of the heart. The energy needs of the heart increase as a result of the realization of the positive ino- and chronotropic effects of catecholamines and an increase in total peripheral resistance, which increases the load on the heart.

C. Non-coronary catecholamine damage to cardiomyocytes. They arise as a result of the fact that large doses of catecholamines activate lipid and calcium mechanisms of cell damage.

Clinical syndromes of myocardial infarction:

1. Pain syndrome.

2. Acute heart failure. It develops when large areas of the myocardium are affected. It may manifest itself as a syndrome of cardiac asthma and pulmonary edema or cardiogenic shock.

3. Arrhythmic syndrome. It is possible to develop all types of arrhythmias. The most dangerous is the appearance of ventricular fibrillation.

4. Resorption-necrotic syndrome.

Non-coronary necrosis of the heart:

1. Hypoxic myocardial necrosis. It develops with a vascular, hemic form of hypoxia. At the same time, against the background of a general lack of oxygen in the body, which in itself leads to an increase in the load on the circulatory system, necrotic damage to the muscle fibers of the heart develops.

2. Electrolyte-steroid cardiopathy with necrosis. According to Selye's observations, when rats are injected with a significant amount of sodium salts in combination with some anions (sulfate-, phosphate-), lesions of a degenerative-necrotic type appear in the heart, often accompanied by hyalinosis of the vessels of other organs. The introduction of even small doses of norepinephrine, calciferol derivatives, hypoxia, muscle tension or significant restriction of mobility lead to the

development of extensive myocardial necrosis. At the same time, potassium and magnesium salts have a protective effect.

3. Immune damage to the heart. They are possible when a heterogeneous serum containing antibodies against heart proteins (cardiocytotoxins) is injected into the body. In the body, in certain situations, antibodies and sensitized lymphocytes may occur, directed against the tissues of one's own heart and having a damaging effect on them.

4. Neurogenic lesions of the heart. Dystrophic changes and myocardial necrosis can also be caused by acute or chronic irritation of the cervical-thoracic node of the sympathetic trunk, vagus nerve, hypothalamus, brainstem or other parts of the brain. The introduction of large doses of adrenaline or norepinephrine into the bloodstream also leads to heart damage. The mechanism of neurogenic damage is based on the discrepancy between the levels of function, metabolism and blood supply.

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