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SYMPTOMATOLOGY, COMPLICATIONS AND MODERN LABORATORY DIAGNOSTIC METHODS OF DIAGNOSIS OF ATYPICAL FORMS OF MYOCARDIAL INFARCTION

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Abstract:
Myocardial infarction (MI) is the term used for the event of a heart attack, which is damage to the heart muscle due to reduced blood flow and lack of oxygen to the heart due to the formation of plaque on the inner walls of the arteries. Myocardial infarction symptoms include chest pain from the left arm to the neck, shortness of breath, sweating, nausea, vomiting, abnormal heartbeat, anxiety, fatigue, weakness, stress, depression, and more. factors include. Myocardial infarction is a limited necrosis of the heart muscle and occurs as a result of an acute imbalance between its blood supply and demand. This article talks about the clinical picture of myocardial infarction, types of myocardial infarction, clinical periods, early and late complications, measures aimed at prevention and treatment.

Keywords: Myocardial infarction, prodromal (period before infarction), scarring (period after myocardial infarction), early and late complications, arrhythmic and asthmatic types.

Introduction

Myocardial infarction is a pathology that leads to ischemic necrosis of the heart muscle as a result of acute violation of coronary circulation. This is most often caused by the formation of blood vessels or, in 90% of cases, by atherosclerosis or other factors, a strong occlusion of an arterial blood vessel. Clinically, the disease manifests as aching, pinching, or pinching pain behind the sternum and can spread to the oral cavity, neckline, scapula, and left arm. [1]

Etiology and pathogenesis. On the basis of myocardial infarction lies atherosclerosis of the crown arteries (in most cases), spasm and blood flow to the atherosclerotic plaques. Its main cause is atherosclerosis in 95% of cases. And in the remaining 5%, The Crown lies

vascular embolism (infectious endocarditis, intra-ventricular thrombi) and congenital and acquired other types of lesions (systemic red volchanka, sclerodermia, rheumatism and coronaritis in rheumatoid arthritis), and in this myocardial infarction is considered as its complication, and not a separate clinical form of YUIK.

In most cases, a sharp cessation or delimitation of blood circulation in the veins of the crown occurs at the expense of thrombosis, which is formed in the area of the "complicated" atherosclerotic plaques in them. It activates tissue thromboplasty and blood platelet and plasmatic clotting factors with collagen. First the wall is formed a "white" thrombus of the acquired thrombus. At one time, a number of biologically active substances (endothelin, serotonin, thrombin, antithrombin A2) are released in this area, which have a strong vasoconstrictor effect. In addition, small platelet aggregates cause small crown vascular embolism and further worsen coronary circulation.

If the patient does not observe the natural activation of the fibrinolytic system or the spontaneous dissolution (lysis) of the thrombus as a result of thrombolytic treatment, he gradually enlarges and completely closes the vein opening, and develops a Q – toothed (transmural) MI. For some reason, when the crown vein does not close completely or suddenly the thrombus dissolves on its own (even in cases where the large crown veins are completely closed and the collaterals are well developed), subendocardial or intramural (without a Q tooth) MI forms.

In some cases (75%), the process of thrombus formation, which closes the crown vein butinlay, takes from 2 days to 2-3 weeks. This period corresponds from a clinical point of view to the course of non-stable stenocardia (pre-infarction syndrome). In very rare cases (¼of patients), the process of forming a thrombus that completely blocks the vein is carried out at lightning speed. In such patients, the period of receiving a heart attack (prodromal) is not observed.

The development of acute necrosis in the heart muscle can be caused by three other conditions:

1) The Crown is a pronounced spasm of the veins;

2) poor development of the collateral vascular system;

3) a sharp increase in myocardial oxygen demand as a result of physical and emotional strain, a sharp increase in AB, and other causes. These three factors lead to an increase in necrosis foci and an increase in hajmi. In contrast the crown, which slowly occurs when the collateral circulation develops well, may also not develop MI in some cases as a result of complete occlusion of the vessel. [5]

Clinical picture

Common symptoms observed in this disease include:

- Extremely strong, long-lasting "wedge-shaped" pain behind the suddenly appearing collar, a feeling of fear of death;
- Sudden cardiac rhythm and conduction disorders, acute left ventricular failure, collapse or shock;

- Unusual for the patient, pain in the epigastral area or in the arms, neck, teeth, lower jaw areas;
- Sudden acute deterioration of the patient's condition, heart failure, drop in blood pressure.

Clinical periods:

1. Prodromal (pre - infarction) - lasts from several hours to a month (on average 2-18 days), and sometimes may not be. During this period, clinical signs of some type of non-stable stenocardia (first appearing, exacerbating, spontaneous and other) are observed. In most cases, the ST segment and T tooth change in the ECG. At the same time, it would be wrong to say that all cases accompanied by signs of non-stable stenocardia during this period end with Mi. Because only 15-20% of them form MI.
2. The acute period is the period from the onset of the first clinical and/or asbobium (ECG) symptoms of myocardial acute ischemia to the formation of a necrosis furnace (from 30 minutes to 2 hours). During this period, it is possible to reverse morphological changes in the myocardium and prevent the formation of a foci of necrosis by timely applying thrombolytic drugs that allow the crown to restore blood flow in the vessels. The first clinical sign of MI is associated with the termination of the process of thrombotic occlusion of the coronary artery or its sudden occurrence (in the absence of signs of the prodromal period in the patient).
3. Acute period. The acute period corresponds to the period of development of the resorption - necrotic syndrome, which is associated with the response reaction of the whole organism in relation to the formation of a necrosis furnace and the absorption (resorption) of necrosis-affected tissue into the blood. During this period, a complex process, accompanied by a violation of the activity of the cardiovascular system, begins to remodel the left ventricle. This period lasts 7-10 days when it goes without complications.
4. Acute subterranean period. In the acute sub - MI, tissue that undergoes necrosis begins to be replaced slowly by connective tissue. The process of remodeling the left ventricle continues. The duration of this period depends on the size of the foci of necrosis, the state of the surrounding tissue that is not included in this process, the degree of development of collaterals, the accompanying disease and the complication of MI. In most cases, it lasts 4-6 weeks. The patient's condition gradually improves. Pain symptom and life-threatening rhythm disturbances (paroxysmal ventricular tachycardia and fibrillation) are not observed when the disease is uncomplicated. Other heart rhythm disturbances (extrasystoles, sinus tachycardia, etc.), high levels of Av and SA blockade symptoms disappear. Symptoms of heart failure that occur during the acute period are reduced.
5. Scarring (period after a heart attack). At the beginning of the scarring period, the amount of collagen in the scar area increases, and its hardening is completed. To balance hemodynamics at a time, a series of compensatory mechanisms (hyperfunctions and hypertrophy of the undamaged myocardium according to The Starling mechanism, a slight dilation of the left ventricle, an increase in the volume of heart firing, etc.) are triggered.

Patients with myocardial infarction may experience early and late complications at different periods of the disease. Complications can be early and late complications:

Early complications: heart rhythm and conduction disorders, cardiogenic shock, acute heart failure, thromboembolism, thromboendocarditis, acute and chronic heart aneurysm, internal and external rupture of the heart.

Late complications: complications that occur early in the myocardial infarction mentioned above can also occur in its late stages. In combination with this, Dressler syndrome and SYY in patients are observed only in the late period of myocardial infarction. Dressler's syndrome-develops in 2-8 weeks of illness and is accompanied by pleurisy, pneumonitis, pericarditis. [3]

Atypical clinical types of myocardial infarction:

Asthmatic– manifested by clinical signs of cardiac asthma or pulmonary edema. Patients complain of a feeling of lack of air, that the cough is first dry, then sputum with a pink foam will separate. They are in an orthopnoe position, and crepitation and small bubble wet wheezes are heard over the lungs. This type of MI is more commonly seen in the elderly, patients with QD, and Re-MI.

Arrhythmic– accompanied by disorders of the acute rhythm and permeability of the heart of the soul. In this case, the typical pain syndrome is not clearly manifested. Patients in most cases suddenly complain that the heart beats more often, incorrectly, pauses, lacks air, dizziness. In this type of MI, swinging arrhythmias, ventricular or supraventricular tachycardia paroxysms, extrasystoles (more polytope), Giss tutami limbs and atroventricular blockades are observed;

Abdominal–it is observed in MI, developed in the lower basal area of the heart, accompanied by pain in the epigastral area, nausea, recording that does not cause relief, flatulence, paresis of the gastrointestinal system;

In addition to cyerebrovascular – coronary blood vessels, cranial vascular thrombosis (spasm) manifests itself in patients with clinical signs of obmork or stroke (dizziness, nausea, recording, syncopated conditions, hemiparesis, paralysis, mental disorders). This type of MI is most often observed in elderly patients who have developed severe atherosclerosis in the blood vessels of the brain;

Asymptomatic or low symptomatic eels - most often detected in patients with QD and when symptoms of SQAYe appear. Patients cannot tell exactly when MIni spent, accidentally detected when an ECG was dropped. [5]

Laboratory-asbobium inspections. Diagnosis of myocardial infarction using laboratory testing methods is based on: tissue necrosis with nospecific indications and signs of inflammation in the myocardium; hyperfermentemia; increased levels of myoglobin and troponins in the blood. As MI develops, the following main laboratory is reflected in the signs:

Rise of body harorat (from subfebrile indicators to 38.5-390S);
Leukocytosis, in most cases up to 12-15 x109 / l;
Aneosinophilia;
The leukocyte formula is shifted slightly (towards Rod nuclei) to the left;
ECHT increase per minute.

The listed laboratory changes are associated with the extent to which the pathological process in the heart has developed, and it is also possible that there are no such changes in a minor injury to the myocardium. These nospecific indications should be analyzed in comparison with clinical and ECG signs. [6]

Treatment

All patients with myocardial infarction are admitted in special intensive care units of the hospital. Treatment measures should be aimed at completely eliminating pain syndromes, preventing heart rhythm and conduction disorders, and limiting the focus of necrosis. In this case, patients are prescribed Thrombolytics (streptokinase, streptodecase), anticoagulants (heparin, fracsiparin), antiagregants (aspirin, cardiomagnil, stazex), nitrates (nitrosorbit, nitrong, monosan, oligard), β-blockers (atenolol, egilok, nebilet), antiarrhythmic (cordarone, β-blockers, allapinin) in individual doses based on the patient condition. When recommending them, it is important not to ignore the indications and contraindications.

All patients with suspected development of myocardial infarction without a Q tooth, i.e. acute coronary syndrome and persistent displacement of the RS-T interval, should be immediately admitted to cardioreanimation units and the following treatment measures should be carried out:

Pain relief (pain relief;
I Antithrombotic and antiaggregant treatment;
II.Thrombolytic treatment;
III.Oxygenotherapy
IV.Use of anti-ischemic drugs;
V.Prescribe angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonists. [4]

Prevention. Elimination of risk factors leading to ischemic heart disease: abstinence from smoking, normalization of body weight, consumption of dietary foods low in animal fats, monand treatment of AG and diabetes mellitus moderate uric acid and hypercholisterinemia in the blood. [2]

Conclusion

Myocardial infarction is when the coronary arteries of the heart become blocked by the thrombus or become compressed, resulting in necrosis of the heart muscle floor (local death) as a result of impaired blood supply to the muscle floor of the heart. It is diagnosed

by people between the ages of 45 and 60. Myocardial infarction is more common in most people with atherosclerosis, hypertension and diabetes mellitus.

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