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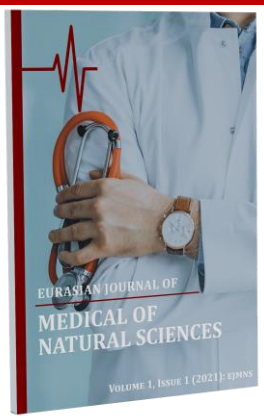

UNSTABLE PROGRESSIVE ANGINA PECTORIS

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ABSTRACT

Despite the widespread introduction of modern diagnostic technology, the appearance of a large number of medications, the problem of verification and treatment of coronary heart disease (CHD) remains very urgent. In particular, issues related to the diagnosis and treatment of the disease during its exacerbation, the development of myocardial infarction (MI) and sudden death (VS) are of particular importance.The review presents data on progressive angina pectoris - exacerbation of chronic ischemic heart disease. The risk of sudden death and myocardial infarction in such patients is higher than in those with stable angina.

IHD is a common disease caused by atherosclerosis of the coronary arteries, as a result of which the balance between oxygen delivery and myocardial demand is disturbed. Recently, the terms "unstable angina" (UA) and "acute coronary syndrome with and without ST segment elevation" (ACS-ST and ACSb-ST) have been widely used in the definition of various forms of its course. ACS includes NS and MI and was introduced for convenience as a preliminary diagnosis, allowing the doctor to determine urgent organizational and therapeutic measures at the first contact with the patient. In the future, when monitoring the patient, taking into account the results of clinical studies, a final, more specific diagnosis is made. NS is a severe period of exacerbation of coronary artery disease, threatening the development of MI or VS. In terms of clinical manifestations and prognostic value, it occupies an intermediate position between stable exertional angina and acute myocardial infarction. Previously, such definitions were used as: “pre-infarction state”, “threatening myocardial infarction”, “pre-infarction angina”, “prodromal syndrome” [5].

Thus, we can say that we have, as cardiologists say, a “matryoshka doll”: ACS is divided into MI and NS, which in turn consists of several of the above and other well-known (Chernov S.A., Chernov A.P.) clinical forms, one of which is the most common PSK. PSK is characterized by an increase in the frequency, intensity and duration of attacks of retrosternal or other pain equivalent to angina pectoris, a decrease in tolerance to habitual physical or emotional stress, and an increase in the number of nitroglycerin tablets consumed to relieve pain [2].

Etiology and pathogenesis of PSK To date, it has become obvious that the causes of the progressive course of coronary artery disease in most cases are due to changes in atherosclerosis. oral plaque (AB) (inflammation, erosion and rupture of plaques with subsequent thrombosis and microembolism of the coronary arteries) [4].

At the same time, the size of the plaques is of relative importance for the development of critical conditions. It is necessary to have the so-called “vulnerable” plaque, which features a large lipid core and a thin cap [14, 15, 16]. Factors contributing to atherosclerotic plaque damage can be divided into external and internal. The former may include arterial hypertension, increased activity of the sympathoadrenal system, vasoconstriction (spasm of the coronary arteries), high levels of LDL, triglycerides, molecules such as fibrinogen, fibronectin, von Willebrand factor, the presence of a pressure gradient before and after stenosis, which, along with periods of "extension - compression" in the places of branching and bending of the vessels leads to a weakening of the plaque structure [6,9]. Internal factors that contribute to the weakening of the plaque structure are the predominance of the lipid core, a decrease in the number of smooth muscle cells and collagen synthesis, an increase in the activity of macrophages inside the plaque and their apoptosis, inflammation inside the plaque, accompanied by infiltration of its cap by macrophages.

Angiographic data, the results of intravital angioscopy [13] showed that in most cases PSCs are accompanied by tears, surface defects, ruptures of atherosclerotic plaques with the release of extremely thrombogenic content, platelet activation, release of vasoactive substances and thrombus formation [15] . In some cases, a thrombus forms on the surface, i.e. located above the gap (crack, defect) of the atherosclerotic plaque, penetrates into the plaque, leading to a rapid increase in its size [7, 11, 13]. In other cases, intermittent arterial occlusion occurs. The thrombus, protruding into the lumen of the vessel, does not cause its complete occlusion, but reduces blood flow, which is manifested by the PSC clinic. Thrombi, both parietal and occlusive, are dynamic, so the blood flow in the corresponding vessel can repeatedly either resume or stop within a short time. Thrombosis can develop suddenly or gradually (over several days) and is a dynamic process. But blood clots can completely close the lumen of the artery for a long time, leading to the development of myocardial infarction. The clot that has not dissolved is replaced by scar tissue produced by smooth muscle cells. The results of this process can be a wide range of changes, from complete chronic occlusion of the vessel to complete or partial restoration of its patency. The latter, apparently, determines the transition of PSC to a stable state, but often with an increase in the functional class of the disease.

Spasm of the coronary vessels [3, 4, 9], neurohumoral, and metabolic factors also play an important role in the pathogenesis of PSC. Arterial spasm, as well as the problem of AB instability, is inextricably linked with endothelial dysfunction (DE), which is understood as an imbalance between mediators that normally ensure the optimal course of all endothelium-dependent processes. Risk factors for coronary heart disease, such as arterial hypertension, diabetes mellitus, an increase in the amount of LDL, smoking increase the activity of lipid peroxidation, which leads to the accumulation of oxygen superoxide anions. As a result of this process, a whole cascade of reactions is triggered: inactivation of nitric oxide (NO), formation of the peroxynitrite radical, oxidation of LDL, and an increase in the formation of adhesive

molecules of vascular cells. Each of these reactions affects the processes of atherogenesis, up to the rupture of the AB (Steinberg D.A., 1989). Along with this, endothelial NO deficiency leads to the predominance of vasoconstrictor reactions. In addition, the function of the endothelium (NO-synthase) is closely related to the oxygen transport function of the blood (OTFC). For example, the inhibition of NO synthesis causes a decrease in tissue pO2, which in turn determines NO metabolism (formation of nitrate from nitrosohemoglobin). And the oxygen-binding properties of blood affect the activity of the L-arginine-NO system, which, in turn, affects the functional properties of hemoglobin, its affinity for oxygen (Zinchuk V.V., Borisyuk M.V., 2000). However, to date, with progressive angina pectoris, this interaction and the mutual influence of the function of the endothelium and CTFC have not been studied.

Glucose and free fatty acids (FFA) are the "fuel" for the heart. When a sufficient amount of oxygen is supplied, FFAs are the supplier of 60-80% of ATP. But to form the same amount of ATP, FFAs require 10% more oxygen than glucose. Therefore, under conditions of ischemia, aerobic oxidation of FFA and glucose decreases, and anaerobic glycolysis becomes the main source of ATP. When the blood flow is restored, about 95% of ATP is again formed due to the oxidation of FFA [6]. As a result of oxidation, hydroperoxides (diene conjugates) are formed, which are then metabolized into secondary ones - malondialdehyde (MDA) and tertiary products of lipid peroxidation (LPO) - Schiff bases. LPO processes occur in all cells, but the most powerful generator of free radicals are leukocytes and platelets, as well as hepatocytes [4]. Thus, hypoxia enhances lipolysis with excessive mobilization of fatty acids, which, in turn, activates the free radical oxidation of the latter. The pronounced predominance of FFA oxidation over glucose, as well as their increased content in the ischemic zone, are one of the main factors of reperfusion injury and the development of myocardial dysfunction, dangerous cardiovascular complications, including heart rhythm disturbances [7, 8].

Diagnosis of PSK is primarily based on the data of the anamnesis. Patients most often indicate the day (date) of the increase in the frequency, intensity and duration of pain. The nature of pain sensations, their irradiation may change. Pain appears in response to less exertion or first appears at rest. The effect of nitroglycerin decreases, the need for it increases. New symptoms for the patient join, such as shortness of breath, palpitations, nausea.

Among laboratory data, a special place in the diagnosis of PSC is occupied by the determination of troponins T and I in peripheral blood, which are markers of damage cardiac muscle damage in patients with unstable angina [1, 17]. Most often, they are determined in the blood of those patients in whom the last attack at rest developed within the next 48 hours or in patients with changes in the final part of the ventricular complex, transient changes in the ST segment on the ECG [7,3]. For the most accurate diagnosis of myocardial damage, it is recommended to determine the level of troponins T and I upon admission to the hospital, after 6-12 hours and after each intense attack of retrosternal pain (A.L. Syrkin, A.V. Dobrovolsky, 2001).

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