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PREVALENCE OF ACUTE MYOCARDIAL INFARCTION AMONG YOUNG PEOPLE AND PROGNOSIS OF ITS COMPLICATIONS

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✓ ***Resume***

Myocardial infarction (MI) occurs mainly in the middle-aged and elderly population, but in recent years the disease has increasingly developed in people under 45 years of age, which seems to be associated with modern lifestyle changes. In the group of increased risk of early development of MI are primarily young men, smokers, people with a hereditary predisposition to the early development of cardiovascular pathology and those with problems with employment. At a young age, the most typical is the development of acute coronary syndrome with ST-segment elevation on the electrocardiogram, which occurred in patients without a previous history of angina. Sebaceous cyst rupture as a cause of coronary thrombosis is more common in young people than erosion. Traditionally, in the pathogenesis of atherothrombosis, the key point is considered to be the rupture of the tyre of the atherosclerotic plaque, which is more typical for atheroma with a large lipid nucleus, a thin cap and high inflammation activity. Such a plaque is traditionally called "unstable". Tire erosion is more common in plaques that have opposite characteristics and is more common in women and the elderly. Non-atherogenic causes of the development of MI at a young age can also be observed, for example, vasospasm, embolism in the coronary arteries in patients with endocarditis, etc. It has been established that young people have the lowest risk of developing it.

Keywords: myocardial infarction, acute coronary syndrome, young age, etiology, atherothrombosis, tygrombus rupture, erosion, unstable plaque, prognosis, chronic heart failure

РАСПРОСТРАНЕННОСТЬ ОСТРОВОГО ИНФАРКТА МИОКАРДА СРЕДИ МОЛОДЫХ ЛЮДЕЙ И ПРОГНОЗ ЕГО ОСЛОЖНЕНИЙ

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✓ ***Резюме***

Инфаркт миокарда (ИМ) встречается преимущественно у населения среднего и пожилого возраста, однако в последние годы заболевание все чаще развивается у лиц до 45 лет, что, по-видимому, связано с современными изменениями образа жизни. В группу повышенного риска раннего развития ИМ входят преимущественно молодые мужчины, курильщики, лица с наследственной предрасположенностью к раннему развитию сердечно-сосудистой патологии и лица, имеющие проблемы с трудоустройством.

В группу повышенного риска раннего развития ИМ входят преимущественно молодые мужчины, курильщики, лица с наследственной предрасположенностью к раннему развитию сердечно-сосудистой патологии и лица, имеющие проблемы с трудоустройством. В молодом возрасте наиболее типичным является развитие острого коронарного синдрома с подъемом сегмента ST на электрокардиограмме, возникающего у пациентов, не имевших в анамнезе стенокардии. Разрыв сальной кисты как причина коронарного тромбоза чаще встречается у молодых людей, чем эрозия. Традиционно в патогенезе атеротромбоза ключевым моментом считают разрыв покрышки атеросклеротической бляшки, что более характерно

для атеромы с крупным липидным ядром, тонкой шляпкой и высокой активностью воспаления. Такую бляшку традиционно называют «нестабильной». Эрозия шин чаще встречается в бляшках, имеющих противоположные характеристики, и чаще встречается у женщин и пожилых людей. Могут наблюдаться и неатерогенные причины развития ИМ в молодом возрасте, например спазм сосудов, эмболия коронарных артерий у больных эндокардитом и др. Установлено, что наименьший риск развития ИМ имеют молодые люди.

Ключевые слова: инфаркт миокарда, острый коронарный синдром, молодой возраст, этиология, атеротромбоз, разрыв тигромба, эрозии, нестабильная бляшка, прогноз, хроническая сердечная недостаточность.

Relevance

In recent years, the features of the development and course of coronary heart disease, in particular its acute forms, in various groups of patients depending on gender, age, comorbid and other characteristics have been actively studied. The data obtained in some cases lead to a revision of traditional views. For example, for a long time it was believed that myocardial infarction (MI) occurs mainly in the population of middle-aged and elderly people, but now its development in people under 45 years of age ceases to be casuistry [1;2;3].

Apparently, a great contribution to this trend is made by changes in the lifestyle of young people in many countries, including physical inactivity, increased consumption of easily digestible carbohydrates, transgenic fats, which is accompanied by the development of dyslipidemia, obesity, diabetes mellitus. In addition, young people often take extra and overtime work, they have a high general pace of life, they are subject to chronic stress, which in some cases leads to smoking, drinking alcohol, energy drinks and overeating [5]. It has been established that young patients with MI have factors that contribute to the early development and progression of atherosclerosis of the coronary arteries. Thus, among young patients with MI, men who are obese and/or have some form of dyslipidemia, smokers prevail [3].

As is known, atherosclerosis of the coronary artery at the stage of destabilization of the atherosclerotic plaque and the formation of an intravascular thrombus over it is the main cause of the development of MI and acute coronary syndrome (ACS) in general, although there are other etiopathogenetic mechanisms of its development [5]. The most important risk factor for the early development and progression of atherosclerosis, along with smoking, arterial hypertension, dyslipidemia, obesity, is genetic predisposition. Genealogical studies demonstrate not only the influence of hereditary mechanisms on the early development of atherosclerosis as such, but also on its predominant localization. For example, an increased prevalence of coronary atherosclerosis was traced among relatives of patients with early onset of MI [10].

It is actively studied how and with the help of what factors the hereditary predisposition is realized in practice. The influence of apolipoprotein genes, lipoprotein receptors and key enzymes of lipoprotein metabolism on the risk of atherosclerosis is discussed. According to a meta-analysis of 15 studies including 3870 people, the E allele of the apolipoprotein B gene has a strong association with the development of MI [15].

It has also been proved that the early formation of atherosclerosis and atherothrombosis is influenced by polymorphisms of genes of the matrix metalloproteinase system (MMP), innate immunity receptors, in particular, To11-like receptors - To11-1ikereceptors, endothelial NO synthase, etc. [14].

The formation of atherothrombosis in the coronary artery most often occurs due to damage to the sebaceous cyst cap by the mechanism of tyre rupture or its erosion. There are traditional views formed on the basis of the results of morphological, clinical and angiographic studies carried out in the late XX - early XXI centuries, the main provisions of which will be set out below.

The development of all forms of ACS is much more likely due to rupture and only in 25-40% of cases due to erosion of the plaque cover. Plaque rupture in the coronary artery usually occurs at the weakest point (the shoulder area), where the cap is thinnest and most infiltrated by inflammatory cells.

One of the factors determining the mechanism of atherosclerotic plaque damage in patients with ACS of any age is the initial degree of stenosis of the coronary artery lumen sebaceous cysts [12]. In 81% of patients who died from thrombosis that developed at the site of an eroded endothelium, hemodynamically significant stenosis was present before the event, and in those who died from coronary

artery thrombosis that developed at the site of a burst plaque, hemodynamically insignificant stenosis in the coronary arteries was recorded in 60% of cases [14].

Plaque rupture is more common in men (about 80% of ACS cases) than in women (60%) [13]. Tire erosion is relatively more common in elderly patients. In women with ACS younger than 50 years of age, endothelial erosion as an etiological factor of atherothrombosis can be observed in 80% of all cases [7]. Erosion of the plaque cover as a trigger for the formation of atherothrombosis is also relatively more common in patients with diabetes mellitus, in smokers and in the presence of hypertriglyceridemia. Blood cholesterol levels are significantly more associated with the likelihood of plaque rupture than with erosion.

Damage to an atherosclerotic plaque is not a purely mechanical process. It has been shown that the activity of local and systemic inflammatory responses, such as circulating C-reactive protein levels, is closely associated with the incidence of ACS due to tygrombus rupture of unstable sebaceous cysts, but does not correlate with the likelihood of endothelial erosion. As a result of morphological studies, activated macrophages, mast cells and tissue factor are found in large numbers in the area of rupture. Plaque erosion is not as closely related as rupture to the activity of systemic and local inflammation and apoptosis [11]. The mechanisms of erosion on the surface of the plaque are less well understood, but the destruction and rearrangement of the extracellular matrix are definitely important in this process.

While plaque damage by the mechanism of tyre rupture is a sudden, rapid process, tyre erosion with the formation of a parietal thrombus can take several days. The formation of a non-occlusive thrombus over the erosion of the tyre can proceed without clinical manifestations, but leads to a gradual increase in the degree of stenosis of the vessel with this sebaceous cyst.

The term "unstable atherosclerotic plaque" has become commonly used, it is such plaques that in most cases are the cause of the development of ACS [13]. Morphologically, an unstable plaque is distinguished from a stable one by a thin, fragile cap and a large lipid nucleus, which occupies a significant volume. In addition, unstable plaque is characterized by signs of active inflammation, which further weakens the structure of the plaque, in particular due to the release of enzymes by inflammatory cells that reduce the mechanical strength of the cap. The unstable plaque is infiltrated by macrophages, mast cells, and T cells, and has an increased content of tissue factor and inflammatory mediators (e.g., cytokines).

In 2015, an article by R. Libby and G. Paster under the intriguing title "Requiem for an 'unstable' plaque" [19].

The aim of the study was to study the prevalence of acute myocardial infarction among young people and the prognosis of its complications.

Results and discussion

Based on the results of studies completed in the last few years in the United States and European countries, the authors noted modern changes in the traditional course of atherosclerosis and the mechanisms of atherosclerotic plaque damage. It turned out that according to the work of recent years, sebaceous cysts with instability characteristics can remain intact for a long time, and in the case of damage, the mechanism of tyre erosion is more common, and rupture is less common. than previously described.

Changes in the "usual" progression of atherosclerosis can be explained by several reasons. First of all, food stereotypes of the population in different countries are gradually changing with a steady increase in the consumption of easily digestible carbohydrates and transgenic fats, which is accompanied by an increase in the prevalence of diabetes mellitus [6].

In addition, for a number of reasons, long before the debut of ACS, many patients are already on therapy with drugs with a pleiotropic, "plaque-stabilizing" effect. A significant proportion of patients, especially in Europe and North America, receive various antithrombotic drugs, statins, and drugs that block the activity of the renin-angiotensin-aldosterone system for various indications. Many drugs of these groups have been described as having a variety of pleiotropic antiatherogenic effects.

Angiotensin-converting enzyme inhibitors and angiotensin receptor I blockers are able to reduce the activity of MMPs, which appears to be related to the induction effect of angiotensin II on MMP expression [8]. There is also data on the antiapoptotic and other antiatherogenic effects of drugs of these groups.

Statins not only reduce the level of low-density lipoprotein cholesterol and triglycerides, but also reduce the severity of inflammation in the sebaceous cyst, reduce the volume of the lipid nucleus, and help stabilize even a relatively thin tire [16]. Under the influence of long-term therapy with statins, the content of connective tissue elements in the sebaceous cystrum increases, it becomes more fibrous in structure, and according to some data, it calcifies more often. A common reason for prescribing statins at a young age is hereditary dyslipidemia.

The presence of a burdened family history is the most important risk factor for the development of ACS after smoking.

At a young age, the most typical development of ST-segment elevation ACS (ST ACS) on an electrocardiogram (ECG) occurred in patients without a previous history of angina pectoris. The mechanism of sebaceous cyst tyre rupture is more common than erosion. The predominant contingent of such patients are men, smokers [12]. Analysis of the population of patients with ACSpST showed that among them, young patients are more likely to smoke than the elderly. The risk of developing MI decreases after the patient quits smoking, and the beneficial effect of smoking cessation on the prognosis correlates with the duration of smoking.

In women who develop ACS at a young age, along with smoking, the use of oral contraceptives is an important risk factor.

In both men and women at a young age, the risk of developing ACS is strongly associated with socioeconomic factors, in particular job loss or unemployment [10]. It is possible that one of the reasons for the increase in the incidence of MI among young people is the economic crisis observed in many countries, as a result of which the unemployment rate has increased significantly.

In addition to factors associated with atherothrombosis, spasm, coronary artery obstruction due to hemorrhage in atherosclerotic plaque, young people may have other causes of acute mismatch of myocardial oxygen demand and delivery. Thus, anemia and hypotension contribute to a decrease in oxygen delivery, and tachycardia, increased blood pressure, hyperthermia and other factors increase the need for it. The causes of MI can be vasculitis involving the arteries of the heart, trauma or hematological disease. In a population of young patients, a factor can cause the development of MI either on its own, or in combination with other factors, or in combination with atherosclerosis of the coronary arteries.

Regardless of the cause, an acute discrepancy between the myocardial demand for oxygen and its delivery through the coronary arteries immediately leads to a violation and restructuring of metabolic processes in cardiomyocytes, the key processes of energy metabolism and ion homeostasis are the first to suffer. The result is a change in the transmembrane potential, which creates conditions for the development of arrhythmias, including life-threatening ones. Up to 50% of deaths in patients with ACS occur in the first 1.5-2 hours from the onset of an angina attack, a significant part of patients dies before the arrival of the ambulance team. The most vulnerable group of patients who die before admission to hospitals are people under 50 years of age [5].

At the onset of the disease for young, able-bodied people, late medical care is typical, which can have a significant adverse effect on the course, outcome and development of complications in any form of ACS. The heart is an organ that is extremely sensitive to ischemia. In the case of damage to the plaque cover by the mechanism of rupture and total occlusion of a large artery in the absence of effective collateral blood flow (which is also typical for young patients without a previous coronary anamnesis), cardiomyocyte death begins after 20 minutes, and after 6 hours, almost all cardiomyocytes in the area of the ischemic focus die.

Due to the fact that the development of ST-segment elevation MI on ECG is more typical for young people, the relevant issue is to determine the prognosis of the disease and, in particular, the likelihood of developing pathological remodeling of the left ventricular myocardium. An important aspect of the problem is the expected adherence to treatment, including taking drugs aimed at preventing the development of chronic heart failure.

The most reliable information on the issue of adherence to treatment and determining the short- and long-term prognosis is provided by independent registries, the interest in which has only been growing in the Russian Federation in recent years [14]. It has been established that if a young patient with ACS receives medical care in a timely manner in full, the risk of developing chronic heart failure is significantly lower compared to older patients [16].

In order to predict the course and outcome of the disease, various clinical diagnostic scales are currently used: TIMI ST elevation (Thrombolysis In Myocardial Infarction), CADILLAC (Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications), PAMI (Primary Angioplasty in Myocardial Infarction), etc., but the search for new risk factors for the development of cardiovascular complications continues [16].

Conclusion

Despite the fact that MI in young people is still a fairly rare event, in recent years there has been a steady increase in the frequency of its occurrence. In the group of increased risk of early development of MI are primarily young men, smokers, people with a hereditary predisposition to the early development of cardiovascular pathology and those with problems with employment.

The causes of ACS are diverse and represent various variants of an acute discrepancy between the myocardial oxygen demand and its delivery through the coronary arteries. In young patients with ACS, blood flow in the coronary artery may be impaired due to atherothrombosis, spasm, arterial obstruction due to hemorrhage into atherosclerotic plaque, and many other non-atherogenic causes.

Despite the presence of universal patterns, the process of development of myocardial ischemia is always individual, the features of its course are determined by many factors. With the timely provision of medical care in full, the prognosis in young patients with ACS is much better than in older patients. A better understanding of the causes and mechanisms of ACS development in young patients is a serious medical and social task.

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