The incidence of myocardial infarction in young people.

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Abstract

Myocardial infarction (MI) occurs mainly in the population of middle-aged and elderly people, however, in recent years, the disease is increasingly developing in people under 45 years old, which, apparently, is associated with modern changes in lifestyle. Young men, smokers, people with a hereditary predisposition to the early development of cardiovascular pathology and having problems with employment are in the group at increased risk of early development of myocardial infarction. At a young age, the development of acute coronary syndrome with ST segment elevation on the electrocardiogram is most typical, which arose in patients without a previous history of angina pectoris. Rupture of the lining of atheroma as a cause of coronary thrombosis occurs in young people more often than erosion. Traditionally, in the pathogenesis of atherothrombosis, rupture of the atherosclerotic plaque cover is considered the key moment, which is more typical for atheroma with a large lipid core, thin cover and high inflammation activity. This plaque is traditionally called "unstable". Tire erosion is more common in plaques with opposite characteristics and is more common in women and the elderly. Non-atherogenic causes of myocardial infarction at a young age can also be observed, for example, vasospasm, embolism in the coronary arteries in patients with endocarditis, etc. For the prognosis of the disease, it is important to assess the risk of complications, in particular, heart failure. It was found that young people have the lowest risk of its development.

Key words: myocardial infarction, acute coronary syndrome, young age, etiology, atherothrombosis, tire rupture, erosion, unstable plaque, prognosis, chronic heart failure

Introduction

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 signs, have been actively studied. The data obtained lead, in a number of cases, to a revision of traditional views. For example, for a long time it was believed that a heart attack myocardium (MI) occurs mainly in the population of middle-aged and elderly people, however, at present, its development in persons under 45 years old ceases to be casuistry [1].

Apparently, a large contribution to this trend is made by changes in the lifestyle of young people in many countries, including hypodynamia, an increase in the consumption of easily digestible carbohydrates, transgenic fats, which is accompanied by the development of dyslipidemia, obesity, and diabetes mellitus. In addition, young people often take extra and overtime work, they have a high overall pace of life, they are subject to chronic stress, which in some cases leads to smoking, alcohol consumption, energy drinks and overeating [2, 3]. It was found that young patients with myocardial infarction have factors that contribute to the early development and progression of coronary atherosclerosis. Thus, among young patients with MI, prevailing men are obese and / or have some form of dyslipidemia, smokers [4, 5]. As you know, atherosclerosis of the coronary artery in the stage
of destabilization of an atherosclerotic plaque and the formation of an intravascular thrombus over it is the main cause of MI and acute coronary syndrome (ACS) in general, although there are other etiopathogenetic mechanisms of its development [6, 7]. The most important risk factor for the early development and progression of atherosclerosis, along with smoking, arterial hypertension, dyslipidemia, obesity, is a 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 among relatives of patients with early onset of myocardial infarction has been traced [8].

It is being actively studied how and by what factors the hereditary predisposition is realized in practice. The influence of genes for apolipoproteins, lipoprotein receptors, and key enzymes of lipoprotein metabolism on the risk of atherosclerosis is discussed. According to a meta-analysis of 15 studies that included 3870 people, the E allele of the apolipoprotein B gene is strongly associated with the development of MI [9]. It has also been shown that the early formation of atherosclerosis and atherothrombosis is influenced by polymorphism in the genes of the matrix metalloproteinases (MMP) system, innate immunity receptors, in particular Toll-like receptors - Tolllikereceptors, endothelial NO synthase, etc. [10, 11].

Atherothrombosis of the coronary artery is the main mechanism for the development of myocardial infarction. The formation of atherothrombosis in the coronary artery most often occurs due to damage to the atheroma lining by the mechanism of tire rupture or its erosion. There are traditional views formed on the basis of the results of the late XX - early XXI century. morphological, clinical and angiographic studies, the main provisions of which will be outlined below. The development of all forms of ACS occurs much more often due to rupture and only in 25–40% of cases due to erosion of the plaque lining. Plaque rupture in a coronary artery usually occurs at the weakest point (shoulder region), where the lining is thinnest and most infiltrated by inflammatory cells.

One of the factors that determine the mechanism of damage to atherosclerotic plaques in patients with ACS of any age is the initial degree of stenosis by atheroma of the lumen of the coronary artery [12, 13]. According to H. C. Stary et al. (1995), in 81% of patients who died from a thrombosis that developed at the site of an eroded endothelium, hemodynamically significant stenoses occurred before the event, and in those who died from coronary artery thrombosis that developed at the site of a burst plaque, hemodynamically insignificant ones were recorded in 60% of cases [14].

Plaque rupture is more common in men (approximately 80% of ACS cases) than in women (60%) [15]. Tire erosion is relatively more common in elderly patients. According to R. Virmani et al. (2006), in women with ACS under 50 years of age, endothelial erosion as an etiological factor atherothrombosis can be observed in 80% of all cases [16]. Erosion of the plaque lining 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 reactions, for example, the level of circulating C-reactive protein, is closely associated with the incidence of ACS, which occurs due to rupture of the tectum of unstable atheroma, 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 quantities precisely in the area of the gap. Plaque erosion is not as closely associated as rupture with the activity of systemic and local inflammation and apoptosis [17]. The mechanisms of erosion on the surface of the plaque are less studied, but the destruction and restructuring of the extracellular
matrix are definitely important in this process. While damage to the plaque by the mechanism of tire rupture is a sudden, rapid process, erosion of the tire with the formation of a parietal thrombus can take several days. The formation of a non-occlusive thrombus over the erosion of the lining can proceed without clinical manifestations, but leads to a gradual increase in the degree of stenosis of the vessel by this atheroma.

**Disease prognosis**

Regardless of the reason, the acute discrepancy between the myocardial demand for oxygen and its delivery through the coronary arteries immediately leads to the disruption and restructuring of metabolic processes in cardiomyocytes, first of all, the key processes of energy metabolism and ion homeostasis are affected. 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 anginal attack, a significant proportion of patients die before the arrival of an ambulance team. The most vulnerable contingent of patients who die before admission to hospitals are those under 50 years of age [6, 7]. At the onset of the disease, young people of working age typically seek medical help late, 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 case of damage to the plaque lining 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 history), death of cardiomyocytes begins within 20 minutes, and after 6 hours, almost all cardiomyocytes in the area of the ischemic focus die.

**References:**

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