

CHARACTER OF IHD COURSE IN WOMEN OF CLIMACTERIC AGE

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Annotation

Menopause is one of the factors that impair the function of the endothelium of the vascular wall. In patients with coronary artery disease in this age group, with ultrasound examination of the brachial artery, there is a violation of endothelium-dependent relaxation, which is manifested by a decrease in the increase in the diameter of the brachial artery in response to reactive hyperemia or paradoxical vasoconstriction. Changes in endothelial reactivity can be considered an early manifestation of atherosclerosis.

Key words: ischemic heart disease, menopause, atherosclerosis, hypoestrogenism.

Introduction

According to research data, most of the metabolic risk factors for coronary artery disease are common for men and women. However, women are more likely to have dyslipidemia and disorders of carbohydrate metabolism, and an increased level of total cholesterol (TC) increases the risk of CVD to a greater extent than men. By the age of 55, more than half of women have a total cholesterol level higher than 6.2 mmol / l. In the fairer sex, a decrease in HDL cholesterol and an increase in triglycerides (TG) are of greater prognostic value [1]. The relationship between HDL cholesterol levels and coronary artery disease is more pronounced in women than in men. An increase in HDL cholesterol by 1 mg / dL is accompanied by a 2% decrease in the risk of coronary heart disease in men and by 3% in women. TG levels rise by about 10% after ovarian failure in women. Perhaps this is due to altered TG clearance, which is reduced in elderly women [2]. According to the Framingham study, despite a lower TG level compared with men, the regression coefficient between this indicator and the risk of developing coronary heart disease was 5 times higher in women than in men. The level of LDL cholesterol as a predictor of CHD is less important in young women, but in the older age group it is comparable to the level of LDL cholesterol in men and is a significant risk factor for cardiovascular diseases, in particular myocardial infarction [3]. A number of studies have shown that in women, the transition from favorable to unfavorable lipid metabolism indicators is associated not with age, but with the extinction of ovarian function. In experimental studies,



convincingly shows the cardioprotective effect of estrogens, realized through specific estrogen receptors (ER), in particular ERa and ERp (CJ Gruber et al.), contained in large amounts in cardiomyocytes (CMs), fibroblasts and coronary vessels (S. Needling et al.). Thus, menopause can be considered a unique "female" risk factor for diseases of the cardiovascular system, which explains the later (10-15 years) onset of CHD in women compared to men.

Hypoestrogenemia plays the role of a trigger factor in the development of a complex of disorders included in the concept of "menopausal metabolic syndrome", which includes an increase in total cholesterol and LDL cholesterol, lipoprotein (a) (LP (a)), triglycerides, a decrease in the concentration of HDL cholesterol, a violation of carbohydrate metabolism, vascular function, increased levels of fibrinogen and platelet aggregation. Since the source for the biosynthesis of sex hormones is cholesterol, which consists mainly of low density lipoproteins, it gradually develops "lack of demand" as a hormonal substrate with a significant increase in serum concentration. This contributes to damage to the vascular endothelium. At the same time, with a decrease in the concentration of estrogen, the activity of liver lipase decreases, which leads to a decrease in the level of HDL cholesterol. The above changes in the lipid spectrum develop on average 2-3 years before the onset of menopause [4] and progress in postmenopausal. In postmenopausal women, among the subfractions of LDL, a subtype of the smallest, most dense particles, especially atherogenic, was identified, probably due to their high sensitivity to oxidation or a great tendency to penetrate into the walls of blood vessels due to their small size. The increased content of these particles is most often observed in persons with abdominal obesity and insulin resistance, which is typical for women with climacteric syndrome [5]. In prospective population studies, it has been shown that individuals with a smaller LDL particle size have a higher risk of developing myocardial infarction and other manifestations of coronary artery disease. The number of small dense subfractions of LDL cholesterol increases in parallel with an increase in the level of apoprotein (a), which is also considered an independent risk factor for cardiovascular and cerebrovascular diseases. With a drug-induced decrease in the amount of small dense LDL-C, there is a significant decrease in angiographic manifestations of coronary atherosclerosis. A decrease in all LDL subfractions (small and large particles) was noted under the influence of atorvastatin at a dose of 80 mg / day [6], and when atorvastatin was combined with omega-3 fatty acids, the number of small dense particles significantly decreased in parallel with a decrease in postprandial hypertriglyceridemia, and the concentration of peroxide products also decreased. oxidation. According to the literature, fibrates also contribute to a decrease in the level



of small dense LDL particles along with an increase in the catabolism of triglyceriderich lipoproteins, which indicates the need for regular intake of lipid-lowering drugs by patients with coronary artery disease in menopause, taking into account metabolic disorders.

Many factors have been identified that play an important role in the stimulation of oxidative stress in postmenopausal women. First of all, these are angiotensin-2 and endothelin, which increase the synthesis of superoxide. Accordingly, a decrease in the bioavailability of NO leads to vasoconstriction. Modified LDL easily form aggregates that are absorbed by phagocytes. Oxidized LDL penetrates under the endothelium, accumulates in the intima of the vascular wall, forming the lipid core of the plaque, the size and composition of which depends on its stability. It is recognized that small, soft plaques containing many lipids and having a thin fibrous capsule are more susceptible to rupture. Critical for rupture is the lipid content in the atheroma, more than 40% of its volume. Oxidized LDL also affects the antithrombogenic activity of the endothelial surface, decreasing the level of tissue plasminogen activator and increasing the level of plasminogen activator inhibitor-1. Modified oxidized LDL, which is one of the important components of oxidative stress, is involved in the activation of the inflammatory process in atherosclerotic plaques [7].

Thus, despite the fact that the development of CVD in women is delayed by about 10 years compared to men, the course of the disease and its prognosis are often more unfavorable than in men. When assessing the risk of developing coronary pathology, it is necessary to take into account the peculiarities of the pathophysiology of the female body in the climacteric period. The approaches to the treatment of coronary artery disease in men and women should be differentiated.

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