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CHOLESTEROL AS A KEY RISK FACTOR FOR ATHEROSCLEROSIS

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Introduction. Atherosclerosis causes cardiovascular diseases, which are one of the leading causes of death worldwide. Elevated cholesterol levels, specifically low-density lipoproteins, are a major factor in the development of this pathological condition. Research into the role of cholesterol is key to the diagnosis, prevention, and treatment of atherosclerosis.

The aim of the study is to analyze the role of cholesterol in the development of atherosclerosis, to investigate the mechanism of atherosclerotic plaque formation and to prevent their occurrence.

Research methods: descriptive, classificatory, comparative.

The main material. Atherosclerosis is a chronic disease of the arteries that's caused by a collection of lipids on the inside of the blood vessels and inflammation, which messes with blood flow and narrows the vessels [1].

Current research identifies not only previously known risk factors for atherosclerosis, such as hypercholesterolemia, hypertension, genetic predisposition, obesity, sugar diabetes, and smoking, but also certain inflammatory conditions, such as gout, rheumatoid arthritis, and HIV.

Cholesterol in human blood plasma is divided into exogenous cholesterol, which enters the body with food, and endogenous cholesterol, which is synthesized independently. At the same time, the amount of exogenous cholesterol does not exceed 30%, which indicates that dietary adjustments can only reduce blood plasma cholesterol levels by 10% [1].

Recent scientific research shows that most cardiovascular diseases are caused not by cholesterol itself, contrary to what was believed until recently, but by substances formed during its oxidation - compounds that form during long-term or improper storage of foods high in cholesterol. Under the influence of oxygen, heat, or light, cholesterol undergoes chemical transformations that produce oxysterols, which are products of its aging. Currently, more than eighty such compounds are known, even a minimal content of some of which can cause damage to the walls of blood vessels,

inflammatory processes, disturbances in cellular metabolism, and potentially lead to atherosclerosis. Whereas pure cholesterol, before oxidation, is necessary for the normal functioning of the human body, as it is an integral component of every cell, plays an important role in the synthesis process and as a starting material for male and female sex hormones, bile acids, and vitamin D [2].

It is important to note that cholesterol does not exist in a free state in the human body, but binds with proteins to form complex compounds called lipoproteins. These are responsible for transporting cholesterol from the liver to peripheral tissues. The most important types of lipoproteins are very low, low, and high density lipoproteins. An excess of low-density lipoproteins contributes to the formation of atherosclerotic plaques on the walls of blood vessels, while high-density lipoproteins return excess cholesterol to the liver for further utilization. Therefore, low-density lipoproteins are considered the most atherogenic, and their level determines the possibility of developing atherosclerosis and the effectiveness of lipid-lowering therapy [3].

Normally, the human body regulates cholesterol levels within the range of up to 5 mmol/L. An increase to 7 mmol/L, i.e., only two units, doubles the risk of mortality from ischemic heart disease [2].

Thus, atherosclerosis can be caused by many factors, which are divided into modifiable and non-modifiable. Modifiable risk factors for atherosclerosis, those that a person can directly influence by changing their lifestyle, include hyperlipidemia, hypertension, smoking, diabetes mellitus, a sedentary lifestyle, obesity, chronic stress, and alcohol abuse [4]. However, the following are considered to be unmodifiable risk factors for atherosclerosis: age, since after 45 years in men and 55 years in women, the risk of atherosclerotic plaques increases significantly; gender - men usually have atherosclerosis more often, but after menopause, the risk increases in women as well; heredity also plays a role, for example, familial hypercholesterolemia [2].

Conclusions. Prevention of atherosclerosis primarily involves a healthy and balanced diet. To prevent the formation of atherosclerotic plaques, it is necessary to limit the consumption of foods containing saturated fats and trans fats, engage in regular physical activity, give up alcohol and smoking (as tobacco smoke damages the walls of blood vessels and accelerates atherogenesis), and control cholesterol levels (especially for people in high-risk groups).

The results of the study prove that cholesterol, namely its oxidation products, leads to the development of atherosclerosis, but it is not the only factor that accelerates atherogenesis.

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