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FEATURES OF THE DEVELOPMENT OF MORPHOLOGICAL CHANGES IN THE LUNGS IN EXPERIMENTAL ATHEROSCLEROSIS UNDER THE INFLUENCE OF VARIOUS RISK FACTORS

Karimov Akmal Raxmonovich

Bukhara State Medical Institute named after Abu Ali Ibn Sino, A. Navoiy Street, Bukhara, Uzbekistan.

Annotation. *The study of changes in lung tissue in atherosclerosis is undoubtedly one of the urgent problems of modern morphology. Atherosclerosis is a systemic process, therefore, if damage is detected in one part of the body, the likelihood of atherosclerosis developing in other areas, including the lungs, is very high. In atherosclerosis, as a systemic disease, changes in the lungs can manifest as narrowing of the pulmonary arteries due to the formation of atherosclerotic plaques, which leads to impaired function and the development of symptoms such as shortness of breath.*

Keywords: *atherosclerosis, lung, oxidative stress, morphology, morphometry.*

ОСОБЕННОСТИ РАЗВИТИЯ МОРФОЛОГИЧЕСКИХ ИЗМЕНЕНИЙ В ЛЕГКИХ ПРИ ЭКСПЕРИМЕНТАЛЬНОМ АТЕРОСКЛЕРОЗЕ ПОД ВЛИЯНИЕМ РАЗЛИЧНЫХ ФАКТОРОВ РИСКА

Каримов Акмал Рахмонович

Бухарский государственный медицинский институт имени Абу Али ибн Сино, ул. А. Навои, Бухара, Узбекистан.

Аннотация. *Изучение изменений в лёгочной ткани при атеросклерозе, несомненно, является одной из актуальных проблем современной морфологии. Атеросклероз – системный процесс, поэтому при поражении одного органа вероятность развития атеросклероза в других областях, включая лёгкие, весьма высока. При атеросклерозе, как системном заболевании, изменения в*

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лёгких могут проявляться сужением лёгочных артерий за счёт образования атеросклеротических бляшек, что приводит к нарушению их функции и развитию такого симптома, как одышка.

***Ключевые слова**: атеросклероз, легкие, оксидативный стресс, морфология, морфометрия.*

Relevance Atherosclerosis is a chronic pathological process of medium- and large-caliber arteries and is a major cause of morbidity and mortality worldwide. Known risk factors such as hypertension, hypercholesterolemia, and smoking create a systemic environment that promotes endothelial dysfunction, lipoprotein oxidation, free oxygen radical formation, and leukocyte migration. Lipid streaks are formed as a result of the accumulation of oxidized lipoproteins in the vessel wall. Macrophages migrate along the endothelium and phagocytose lipid-rich proteins. High intracellular cholesterol levels in macrophages lead to cell death.

Chronic hypoxia develops in atherosclerosis. Oxidative stress is another important link in the pathogenetic relationship between chronic obstructive pulmonary disease and cardiovascular disease. Oxidative stress has been shown to play a significant role in the pathogenesis of atherosclerosis, particularly in its negative impact on endothelial function. Reactive oxygen species damage cell membranes and interact with endogenous vasoactive mediators of endothelial cells, which leads to endothelial dysfunction. Reactive oxygen species also accelerate lipid peroxidation, leading to the formation of oxidized lipoproteins - a key factor in the development of vascular wall damage, which, together with impaired vascular tone, creates conditions conducive to the development of an inflammatory response, the subintimal space of which promotes leukocyte migration.

Chronic obstructive pulmonary disease is the fourth leading cause of death worldwide, after cardiovascular diseases, malignant tumors and injuries. According to WHO, the global prevalence of chronic obstructive pulmonary disease in people over 40 years of age is on average 10.1% (11.8% in men and 8.5% in women). Chronic obstructive pulmonary disease is a chronic inflammatory disease of the respiratory system, characterized by progressive bronchial obstruction and

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progressive chronic respiratory failure, affecting mainly the peripheral airways and lung parenchyma (with the development of emphysema), and is associated with environmental factors. It develops in susceptible individuals and is characterized by increased cough, sputum production, and shortness of breath. It has a stable progressive course, ultimately leading to chronic respiratory failure and pulmonary heart disease.

Cardiovascular disease remains a leading cause of death in most developed countries. According to the WHO, cardiovascular disease causes 17.5 million deaths annually. By 2025, it is projected that more than 1.5 billion people, or one-third of the world's population over 25, will have high blood pressure—the most dangerous risk factor for atherosclerosis. Coronary heart disease (CHD) and stroke, both of which are caused by atherosclerosis, occupy a special place in the structure of cardiovascular diseases. CHD and stroke will become the leading cause of death and disability worldwide, with the number of deaths from CHD expected to reach 20 million by 2020 and 24 million by 2030. The causes of atherosclerosis are not yet fully understood, but clinical, experimental, and epidemiological studies have identified risk factors that contribute to the development and progression of cardiovascular disease.

According to WHO, health depends 50-55% on social conditions and lifestyle, 19-20% on the environment, 20-22% on genetic factors, and only 7-10% on the quality of medical care and the level of development of the healthcare system [6, 26, 29,215]. Classification of risk factors: Cardiovascular diseases: Biological (non-modifiable) factors: male gender; old age; genetic factors contributing to the development of dyslipidemia, hypertension, obesity, diabetes. Anatomical, physiological and metabolic features: hypertension; dyslipidemia; diabetes; obesity. Behavioral factors: low physical activity; dietary commitment; smoking; stress; alcohol consumption. Risk factors are individual characteristics that affect the likelihood of developing a disease in the future in a particular person. Genetic predisposition does not necessarily manifest itself throughout a person's life; controlling modifiable risk factors can prevent the onset of cardiovascular disease.

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According to the WHO, four main risk factors contribute most to the risk of sudden death: hypertension, diabetes, smoking, and dyslipidemia. Let's consider some of them. According to the WHO, 23% of deaths from coronary heart disease are due to smoking, which reduces life expectancy by an average of 20 years in smokers aged 35-69.

The risk of heart attack (including recurrent heart attack) increases with age (especially over 70 years) and with the number of cigarettes smoked per day. Sudden death among individuals who smoke more than one pack of cigarettes per day occurs five times more often than among non-smokers. Passive smoking increases the risk of coronary heart disease by 25-30%.

Damage to the intimal surface in hypertension is a significant cause of vascular wall damage. Long-term persistence of high blood pressure triggers the main mechanisms of chronic pathological (neurodegenerative) processes in organs and tissues. Normally functioning endothelium is characterized by continuous basal production of NO via endothelial NO synthase from L-arginine. This is essential for maintaining adequate basal vascular tone. Furthermore, NO has antioxidant and angioprotective properties, inhibiting endothelial-leukocyte interactions and monocyte migration, as well as platelet aggregation and adhesion. During the development of hypoxic changes associated with the transition from a physiological to a pathological state, protective mechanisms aimed at increasing the body's resistance deserve special attention.

The adaptive process during hypoxia is formed with the participation of four mechanisms of different directions:

1) The primary mechanisms are those whose mobilization leads to the stabilization of oxygen delivery to the body, which significantly compensates for its deficiency in the external environment. These include pulmonary hyperventilation, activation of cardiac activity, erythrocytosis, and an increase in the affinity of hemoglobin for oxygen.

2) The mechanisms responsible for improving oxygen transport to the brain, myocardium, and other organs are important. Their involvement in the adaptation

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process is accompanied by dilation of the arteries, arterioles of the precapillary sphincters (brain, heart, etc.), an increase in the capillary networks, and an increase in the permeability of cell membranes.

3) At later stages of adaptation to hypoxia, mechanisms are activated to increase the ability of cells and tissues to utilize oxygen from the blood with a simultaneous increase in the synthesis of ATP molecules due to an increase in the affinity of the final enzyme of the respiratory chain, cytochrome oxidase, to oxygen, and an improvement in oxidative phosphorylation caused by structural and functional changes in the mitochondria.

4) An important mechanism of adaptation to hypoxia is considered to be an increase in the intensity of anaerobic processes in the resynthesis of ATP due to the activation of glycolysis. Thus, with exogenous forms of hypoxia, despite a clear decrease in oxygen content in the environment, no significant changes in oxygen tension, carbon dioxide, or pH occur over a certain period in arterial blood and interstitial fluid. However, with a high degree of stress on adaptive mechanisms, their effectiveness decreases, which manifests as symptoms of acute or chronic oxygen deficiency. Despite a significant amount of fundamental and clinical research, the morphofunctional properties of lung tissue in atherosclerosis, its role in predicting the course of the disease and choosing optimal surgical approaches, have still not been fully understood, which requires further research.

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