

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

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Annotation. Currently, among lung pathologies in the world, chronic obstructive pulmonary disease is the fourth leading cause of death worldwide, claiming 3.5 million lives in 2021, which is approximately 5% of all deaths worldwide. 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.

Atherosclerosis is a chronic immunomodulatory, multifocal, and immunoinflammatory 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 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.

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