MORPHOLOGICAL CHANGES OF THE LUNGS IN EXPERIMENTAL ATHEROSCLEROSIS

Karimov Akmal Raxmonovich

Abu Ali Ibn Sino nomidagi Buxoro davlat tibbiyot instituti, A.Navoiy ko'chasi, Buxoro, O'zbekiston.

Annotation. It has been established that patients with Chronic Obstructive Pulmonary Disease die more often from cardiovascular causes than from Chronic Obstructive Pulmonary Disease itself, and coronary heart disease (CHD) is the leading cause of death. The morphological substrate of CHD is atherosclerotic plaque (AP), and numerous studies have been published assessing and comparing the degree of atherosclerotic lesions in the coronary arteries in patients with CHD and COPD using multislice computed tomography of the heart [6–8] and invasive coronary angiography (CAG).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.

Relevance. The problem of atherosclerosis is recognized as one of the most pressing issues today. Atherosclerosis and its complications continue to be the leading cause of morbidity and mortality in Western countries and Russia. Mortality from cardiovascular pathologies worldwide is twice that of cancer and 10 times higher than that of accidents [1]. Cardiovascular disease (CVD) risk factors play a crucial role in atherogenesis. Numerous studies have established a

link between virtually all known atherosclerosis risk factors and endothelial dysfunction. One of the main risk factors for atherosclerosis is lipid metabolism disorders. Dyslipidemia, characterized by decreased alpha-lipoprotein (HDL) and elevated beta-lipoprotein (LDL) and pre-beta-lipoprotein (VLDL), contributes to the development of atherosclerosis. Moreover, modified LDL, most often subjected to peroxidation (PO), and oxidized (oxy-LDL), possess atherogenic properties [5].

They promote increased synthesis of caveolin-1, which leads to decreased nitric oxide (NO) production by the endothelium [2,5]. Oxidized lipoproteins are active irritants for monocytes, which penetrate the subendothelial space, transforming into macrophages and then, as modified LDL accumulates within them, into foam cells [5,10]. Activated macrophages and foam cells release biologically active substances—growth factors, proinflammatory cytokines, and cell adhesion molecules—that promote platelet aggregation, vasoconstriction, and leukocyte adhesion, thereby promoting inflammation in the arterial wall and the progression of atherosclerosis. Oxy-LDL also induces vascular smooth muscle cell proliferation [1,5]. HDL, on the other hand, reverses cholesterol transport from the vascular wall and macrophages to the liver.

Arterial hypertension (HTN) is the second major risk factor for atherosclerosis. It has been shown that drug-based blood pressure control in hypertensive patients reduces the risk of stroke by 40%, myocardial infarction by 8%, and overall mortality from heart disease by 10% [1,2,10]. It has been proven that in isolated hypertension in men aged 47.5±8.4 years, lipid spectrum parameters shift towards an increase in TC, TG, LDL, a decrease in HDL, and an increase in the atherogenic coefficient [2]. Hypertension contributes to an increase in endothelial permeability and the accumulation of lipoproteins in the intima [5]. It has been proven that the cause of activation of protein and lipid peroxidation in rats with spontaneous hypertension is an increase in the production of oxygen radicals and the inefficiency of endogenous systems for their inactivation. It is also known that the development of spontaneous hypertension in rats is accompanied

by systemic inflammatory response syndrome: its initial stage is the activation (priming) of polymorphonuclear leukocytes, increased production and secretion of reactive oxygen species (O2 and H2O2), and intensification of PO of proteins and fatty acids. The reaction of O2 with NO forms peroxynitrite (ONOO-) and deprives NO of its biological effect as a relaxation factor. A decrease in NO leads to an increase in blood pressure, creating a vicious cycle [2].

From a modern perspective, endothelial dysfunction (ED) is considered a key link in the pathogenesis of atherosclerosis. It is an imbalance between the main functions of the endothelium: vasodilation and vasoconstriction, inhibition and promotion of proliferation, antithrombotic and prothrombotic functions, antioxidant and prooxidant [1,6]. NO is an important regulator in the cardiovascular system, a messenger with auto- and paracrine effects [2,7]. In the body, NO synthesis is catalyzed by the NO synthase family. NO synthases use L-arginine as a substrate and NADPH diaphorase as a cofactor. NADPH diaphorase is involved in the transport of electrons to the prosthetic group of the enzyme. The detection of NADPH diaphorase is based on the formation of diformazan in the presence of endogenous βNADPH and tetrazolium salts.

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