SOME ASPECTS OF THE FORMATION OF CHRONIC KIDNEY DISEASE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract: The role of smoking, inflammation, endothelial dysfunction, hypoxia and pulmonary hypertension as factors associated with the development of renal dysfunction in chronic obstructive pulmonary disease is considered. The importance of identifying renal dysfunction in the early stages of chronic obstructive pulmonary disease is dictated by the fact that both pathological conditions are potential risk factors for cardiovascular disease and mortality.
Keywords: smoking, endothelial dysfunction, systemic inflammation, hypoxia, pulmonary hypertension.

INTRODUCTION

Conducted clinical-epidemiological and prospective studies indicate that for many years CKD occurs in the shadow of NCDs, including chronic obstructive pulmonary disease (COPD) [1]. According to literary data, kidney and lung damage often coexist. Thus, in 26% of patients with COPD, kidney function is reduced, in 60% of patients with CKD, obstructive sleep apnea syndrome is detected [2]. The development of COPD is facilitated by such factors as rapid and disorganized urbanization, globalization of unhealthy lifestyles, aging of the population, etc. As a rule, CKD and COPD have a long course and are the result of the influence of a combination of genetic, physiological, environmental and behavioral factors.

MATERIALS AND METHODS

Many studies show that patients with COPD most often have risk factors for the development of CKD [3]. On the other hand, risk factors for the development of COPD are common for the occurrence of CKD [3]. Such factors as smoking, environmental conditions, climate, infections, medications, etc. have a significant impact on the development and progression of CKD. Currently, among the factors affecting renal tissue in COPD, significant importance is given to smoking, inflammation and dysfunction of the endothelium, systemic hypoxia, pulmonary hypertension (PH).

RESULTS AND DISCUSSION

Smoking. Tobacco smoke is the most common cause of COPD. It is well known that tobacco smoking is the main modifiable risk factor for COPD. Thus, smokers have a six-fold higher risk of developing COPD; nine out of ten smokers develop COPD. According to other data, about 88% of people with COPD are smokers [4]. At the same time, smokers shorten their lives by 10-15 years. Currently, cigarette consumption is increasing every year. In the publication by K.N. Yeshchenko and co-authors, it is emphasized that the lethal dose of nicotine for a person is from 50 to 100 mg, or 2-3 drops [2]. This is the dose that enters the blood daily after smoking 20-25 cigarettes (one cigarette contains approximately 6-8 mg of nicotine, of which 3-4 mg enters the blood). When smoking, 20-25% of toxic substances enter the human body, and 50%, together with exhaled smoke, enter the air, which is breathed by others [3]. Research results show that tobacco smoke contributes to the development of oxidative stress and an imbalance of enzymes of the protease system and their inhibitors in the lung tissue, thereby contributing to a further increase in the inflammatory process in the lungs and, ultimately, to the pathomorphological changes characteristic of COPD [3].

Inflammation and endothelial dysfunction. Chronic persistent inflammation characteristic of COPD occurs with the participation of neutrophils, macrophages and T-lymphocytes. Their production of proinflammatory cytokines makes a significant contribution to the progression of

endothelial dysfunction and atherosclerosis. Vascular endothelium is a metabolically active cell lining all vessels of the body. In the work of A.A. Vizel and I.Yu. Vizel it was shown that COPD is a chronic environmentally mediated inflammatory disease of the respiratory system with damage to the airways and pulmonary parenchyma with the development of emphysema, manifested by partially reversible bronchial obstruction, characterized by progression and increasing phenomena of chronic respiratory failure [1]. The pathogenesis of COPD is based on inflammation of the bronchial tree, which is systemic in nature. It is generally accepted that at present, inflammation in COPD is not limited to the lung tissue, but also extends to the systemic blood flow, where it can participate in the development of concomitant pathologies. Now there is no doubt that systemic inflammation occurs as a result of the effect of tobacco smoke on other tissues, in particular, the vascular endothelium. In patients with COPD, the intensity of systemic inflammation increases during periods of exacerbation. Most studies conducted earlier show that COPD patients have a higher density of CD8+ T-lymphocytes of the bronchial wall, neutrophils, macrophages, eosinophils and other types of inflammatory cells [3]. Individuals with COPD have elevated levels of fibrinogen, C-reactive protein (CRP), tumor necrosis factor alpha (TNF-α), and higher leukocyte counts in peripheral blood compared with controls. From a pathophysiological point of view, TNF-α promotes the production of endothelin-1 and angiotensinogen.

Systemic hypoxia. Persistent airflow limitation detected by spirography and progressive inflammation in COPD lead to chronic (systemic) hypoxia. Accumulated data indicate the involvement of systemic hypoxia in the process of fibrosis formation in the kidneys and the progression of CKD. Under physiological conditions, 100 g of renal tissue uses approximately 400 ml of blood, which is about 20% of the cardiac output [2].

CONCLUSION

It is noted that restoration of renal function after successful transplantation contributes to the normalization of the LBP level [3]. To summarize this review, it should be noted that it is important to identify CKD in the early stages of COPD, which is dictated by the fact that both pathological conditions are risk factors for cardiovascular disease and mortality. The relationship between COPD and CKD may have deeper foundations than the commonality of risk factors and progression of both diseases. The formation and progression of CKD in individuals with COPD has a complex genesis, which requires further study of the mechanisms of its development.

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