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MODERN ASPECTS OF THE PATHOGENESIS OF COMORBIDITY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract:

This review article presents modern aspects of the pathogenetic mechanisms of the formation of bronchial obstruction and pulmonary emphysema in chronic obstructive pulmonary disease occurring against the background of comorbid diseases. The author summarizes the latest research on the interaction of cardio-respiratory pathologies and prevention of complications of these conditions.

Keywords: Chronic Obstructive Pulmonary Disease, Comorbid Pathology, Prognosis, Respiratory System

Comorbidity is one of the most important problems of clinical practice, which fully applies to COPD, the combination of which with some diseases significantly worsens its prognosis. Thus, according to various studies, from 20 to 50% of deaths in COPD patients are associated with "vascular" comorbidity, primarily with coronary heart disease and arterial hypertension [5]. These patients are at high risk of sudden cardiac death, the main causes of which are fatal cardiac arrhythmias and myocardial infarction (MI). The probability of its development in patients with COPD is 40% higher than in the general population, and the 5-year survival rate decreases by 34% compared to patients with MI without concomitant COPD [6]

The comorbid background of patients with COPD is burdened by no less than the somatic status of "vascular" patients, while it is obvious that COPD, in turn, aggravates the clinical course of the absolute majority of diseases known today according to a number of clinical and laboratory indicators [3, 4]. At the same time, patterns between changes in the clinic, course, and the outcomes of COPD and a number of diseases have not been studied enough. However, today it has become obvious that due to the presence of systemic effects of COPD, a number of diseases (arterial hypertension, coronary heart disease, diabetes mellitus, osteoporosis, malignant neoplasms, etc.) may be a consequence of the natural course of this disease [5, 6]. The pathogenetic mechanisms of the development of concomitant pathology in patients with COPD are tissue hypoxia, oxidative stress, endothelial dysfunction, activation of neurohumoral systems, violation of water-electrolyte metabolism, as well as chronic systemic inflammation [7, 8].

Since COPD is a disease of the second half of life, the occurrence of osteoporosis as a concomitant pathology is not surprising. An increased risk of osteoporosis in COPD is associated with age, physical inactivity, low body mass index (BMI), smoking, hypogonadism, malnutrition and the use of corticosteroids [6]. According to WHO data, the prevalence of osteoporosis among COPD patients ranges from 24 to 69% [4].

COPD is a disease with a comorbid component. COPD polymorbidity includes cardiovascular pathology, diabetes mellitus (DM), depression, lung cancer, osteoporosis, etc. [1].

It is known that the risk of fatal cardiovascular complications in patients with COPD is 2-3 times higher than in patients without comorbid pathology [8].

Of particular importance is the comorbidity of arterial hypertension (AH) with COPD, since these diseases have many common risk factors. Cardiovascular risk increases due to hypoxia, chronic inflammation in the bronchi, damage to the vascular endothelium, activation of free radical oxidation. Modern approaches to the management of AH patients are not limited only to achieving the target blood pressure level. The main goal of hypertension treatment is to reduce the overall cardiovascular risk, as well as the correction of all identified modifiable risk factors and to ensure the protection of target organ damage [1, 5, 7, 8].

Bacterial colonization of the lower respiratory tract in COPD outside of exacerbation affects the nature and frequency of exacerbations of COPD [8]. Possible mechanisms of bacterial action on the course of stable COPD include stimulation of mucus hypersecretion, suppression of mucociliary activity, direct damage to the epithelium, increased epithelial expression of proinflammatory cytokines IL-6, IL-8 and TNF-▪ , which can trigger the inflammatory process [11].

Oxidants also mediate other pathological processes: DNA degradation, decreased surfactant activity, increased permeability of the epithelium and

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endothelium. Thus, in COPD, under the influence of cigarette smoke and other airborne pollutants, free radical oxidation is activated and the equilibrium in the "oxidants-antioxidants" system is disturbed and, as a result, pathological processes in the lungs are stimulated. In addition, cigarette smoke and oxidative stress contribute to the aging process, and therefore COPD is interpreted as accelerated lung aging.

Smoking stimulates the development of oxidative stress, which is a pronounced imbalance of oxidant-antioxidant systems. As a result, a pathogenic concentration of active oxygen metabolites occurs in the lung tissue [10].

In patients with a combination of COPD and DM, there is an increase in the frequency of exacerbations of COPD, an increase in the duration of hospitalization, and higher mortality [11, 12]. Currently, the influence of factors such as systemic inflammation, oxidative stress, hypoxemia, hyperglycemia, low physical activity, smoking, on the development of both diseases has been proven [7, 13, 14].

Domestic and foreign epidemiological studies indicate that patients with COPD are characterized by a wide range of comorbidity, which includes both somatic and mental disorders: arterial hypertension (in 35-70% of COPD cases), coronary artery disease, atherosclerosis of coronary vessels with thrombotic complications (from 20 to 50-60%), bronchial asthma (up to 21%), obesity (on average in 40% of patients) or, conversely, a decrease in body mass index (14%), oncological diseases (most often – lung cancer (up to 30%), osteoporosis (up to 70% of COPD cases), diabetes mellitus (31%), maladaptive and mental disorders [7-10]. The definition of COPD in the Global Strategy for Prevention, Diagnosis and Treatment (Global Strategy for Prevention, Diagnosis and Management of COPD, GOLD, 2020) highlights the impact of concomitant pathology on morbidity and mortality. The focus is on diseases of the cardiovascular system, osteoporosis, lung cancer, gastroesophageal reflux disease, bronchiectatic disease, obstructive sleep apnea syndrome, metabolic syndrome (MS) and diabetes mellitus, anxiety and depressive disorders. In addition, the influence of multiple comorbidity on the severity of the clinical course of COPD, the frequency of exacerbations, the quality of life of patients is discussed, the urgency of finding safe and effective ways to treat complicated forms, especially in the elderly, is emphasized [10].

CONCLUSION.

Thus, among the concomitant pathology of COPD patients, much attention is paid to cardiovascular pathology, osteoporosis, diabetes mellitus, obesity, lung cancer. The gastroenterological component of the comorbid background is gastroesophageal reflux disease (GERD), which is associated in COPD patients with a high risk of exacerbations and poor health status. The so-called gastrocomorbidity of COPD patients, manifested along with GERD and peptic ulcer disease, does not accidentally attract the attention of researchers. Gastroenterological manifestations, depression, and, in fact, COPD progressing, mutually burden each other.

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