FEATURES OF TREATMENT OF CHF IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Annotation: *Currently, based on most studies, statistical reports obtained during screening tests using spirometry show that the level of chronic obstructive pulmonary disease (COPD) significantly exceeds the actual prevalence of mild, rather than severe and moderate severity. In 474 patients over 40 years old who came to the clinic with other complaints, spirometry revealed COPD in 10.3% of people. COPD is often associated with CHF in clinical practice. Both diseases are characterized by high morbidity and mortality. Such patients require a comprehensive approach to objectively detect both diseases at an early stage. Additional research is needed to provide new information on the pathogenesis and management of patients with COPD and CHF, which will improve life expectancy as well as allow these patients to live longer.*

Keywords: *chronic obstructive pulmonary disease, chronic heart failure, treatment, inhaled corticosteroids, beta-blockers, β2-agonists. COPD is now considered a preventable and treatable disease.*

Chronic obstructive pulmonary disease is a preventable and treatable disease that has a significant extrapulmonary manifestation that can determine the severity of the disease in patients. It is characterized by persistent airflow limitation. The disease is usually progressive and is associated with the pathogenic effects of particles or gases that cause chronic inflammation in the lungs. Epidemiological studies have shown that the gradual decline in lung function is as strong as the main cardiovascular risk factors for cardiovascular death. According to the results of the study of external respiratory activity, 5887 smokers aged 35-60 years had moderate bronchial obstruction. In all of them, a 10% decrease in forced expiratory volume in the first second (FEV1) and a 10% decrease in forced expiratory volume increased overall mortality by 14%, cardiovascular mortality by 28%, and the risk of coronary heart disease by 20%. The risk of developing CHD in patients with CHD is 2-3 times higher than in people without CHD. Chronic heart failure (CHF) is a pathophysiological syndrome in which, as a result of a particular disease of the cardiovascular system or under the influence of other etiological causes, there is a violation of the ability of the heart to relax or contract, which is accompanied by an imbalance of neurohumoral systems (renin-angiotensin-aldosterone system (RAAS), sympatho-adrenal system, natriuretic peptide system, kininkallikrein system). Narrowing of blood vessels and fluid retention, which leads to further dysfunction of the heart and other organs, as well as to

the fact that the supply of blood and oxygen to the organs and tissues of the body does not correspond to their metabolic needs.

The use of modern diagnostic and therapeutic methods, a clear algorithm for the diagnosis and treatment of patients with CHF over the past 30 years has allowed to increase the survival rate of patients and reduce the frequency of hospitalization of patients with heart failure. Approaches to the treatment of CHF in patients with CHF should be based on clinical recommendations for chronic heart failure, since it has not been proven that CHF should be treated differently in the presence of CHF. Although β-blockers improve symptoms and quality of life in patients with chronic heart failure, they are often not prescribed in patients with CHF due to reduced bronchodilator effect of β2-agonists and increased risk of bronchospasm. According to a Cochrane meta-analysis, in trials of patients with OSA and SJU, the use of selective β1-adrenoblockers has been shown to be safe in only 35% of patients. According to NICE and the European Society of Cardiology (ESC) recommendations, cardioselective β-adrenoblockers such as metoprolol, bisoprolol, and nebivolol are not contraindicated in patients with OSA. The study shows that treatment with bisoprolol and carvedilol in patients with moderate to severe bronchial obstruction and COPD has a positive effect on lung function. The potential risks of prescribing selective β1-adrenoblockers for the treatment of heart failure outweigh the risks in treating patients with COPD, even in severe obstruction. It is recommended to prescribe drugs gradually, in small doses. A slight deterioration in lung function should not be an indication for stopping selective β-adrenoblockers. Doctors are reluctant to continue β-adrenoblockers during the exacerbation of COPD, when the patient has difficulty breathing. Inhaled glucocorticosteroids Under the influence of etiological factors of COPD, the function of the respiratory tract is impaired, and the amount of air remaining in the lungs is preserved - the so-called “trap” for the respiratory system, which largely determines the clinical manifestations of the disease. The drugs used to treat patients with COPD must effectively suppress dynamic hyperinflation, and this requirement is met by modern long-acting inhaled bronchodilators (anticholinergics and β2-agonists). Inhaled glucocorticosteroids have a place in the treatment of COPD patients as part of combination therapy, and are effective in the treatment of bronchial hyperreactivity, high levels of nitric oxide in exhaled air, and eosinophilic sputum (>3%). The choice of drugs for the treatment of COPD should be accompanied by reliable data on the safety of respiratory drugs associated with cardiovascular risk in chronic heart failure.

Angiotensin-converting enzyme inhibitors Treatment with statins, ACE inhibitors (angiotensin-converting enzyme inhibitors), and ARBs (angiotensin II receptor blockers) can reduce morbidity and mortality in patients with COPD. According to Mancini et al., statins, ACE inhibitors, and/or ARBs reduce the risk of hospitalization in patients with COPD. Indeed, in patients for whom β-blockers are absolutely contraindicated, ACE inhibitors and ARBs may be a way to improve the quality of life of patients. Also, when choosing an antihypertensive drug, ARB II drugs should be given to patients on a general

basis. In addition, there is evidence that the effect of ARB II drugs in COPD is associated with a decrease in pulmonary artery pressure, increased oxygen saturation, and increased physical activity. However, it should be remembered that ACE inhibitors significantly impair the quality of life of patients with COPD and, in some cases, are mistakenly perceived as an exacerbation of pulmonary disease. However, cough can develop during treatment with any drug from the ARB group. Some of them are less likely to cause the side effects mentioned above than ACEIs, but the data are contradictory [6]. It remains difficult to prove the relationship between taking ACEIs and the development of cough. In addition, in some cases, cough may be due to causes unrelated to ACEIs (bronchial asthma, pneumonia, chronic bronchitis, laryngitis, upper respiratory tract infections, pneumocystis, left ventricular failure, lung cancer, mitral stenosis, pulmonary thromboembolism, smoking). Diagnostic difficulties in identifying cough due to ACEIs are also associated with its frequent occurrence at night and sometimes worsening in the horizontal position. In patients with heart failure, these phenomena are sometimes difficult to distinguish from paroxysmal nocturnal dyspnea. Currently, there is no clear answer to the question of whether ACE inhibitors should be discontinued with the development of cough. Although angiotensin II receptor blockers are a possible alternative to ACE inhibitors in this situation, the benefits of ACE inhibitors in this category of patients have not been proven.

Calcium antagonists in diseases. Currently, the prospects for the use of drugs from the group of calcium antagonists in cardiovascular diseases in combination with ACE inhibitors are highlighted. In patients with ischemic heart disease, the effect of calcium antagonists as peripheral vasodilators helps to improve hemodynamics and myocardial contractility. It should be noted that bronchial passivity does not worsen under the influence of representatives of this group of drugs. In addition, according to a number of data, when using calcium antagonists in this category of patients, vital capacity of the lungs, minute volume, and blood pressure in the pulmonary artery are improved. Thus, D. A. Yakhontov et al. showed that the addition of amlodipine to the treatment of patients with a history of AG and OSOK was accompanied by a clear positive dynamics of indicators of external respiratory function. The results obtained are associated with the improvement of hemodynamics of the small circle of blood circulation under the influence of amlodipine and its direct bronchodilator effect. The cause of the frequent occurrence of OSOK heart failure is pulmonary arterial hypertension, studies have been conducted on measuring the level of proBNP in patients with OSOK and pulmonary arterial hypertension against the background of taking amlodipine. According to the results of the study, a significant decrease in proBNP from 1297±912 to 554±5 PG/ml was observed after treatment with calcium channel blockers, which confirms the possible benefits of treatment with vasodilators in OSOC. Aldosterone receptor blockers. There is a lot of information about the importance and role of the renin-angiotensin-aldosterone system (RAAS) in the development of pulmonary arterial hypertension and chronic cor pulmonale in OSOC patients. Pulmonary arterial hypertension develops as a result of hypoxemia caused by

damage to the pulmonary-bronchial system and is mediated by changes in neurohormones. A significant increase in the activity of RAAS components leads to vasoconstriction and, therefore, to an increase in pressure in the pulmonary artery.

High levels of aldosterone contribute to the contraction of smooth muscle cells, the development of hypertrophy and myocardial fibrosis, vasoconstriction and an increase in the volume of circulating blood in the blood vessels. Thus, they trigger mechanisms that lead to the formation of chronic cor pulmonale. Therefore, aldosterone receptor antagonists, i.e. spironolactone, are recommended for the treatment of patients with chronic cor pulmonale on the background of OSA. Its use leads to the accumulation of potassium in the cellular structures of the lung tissue, preventing the entry of sodium and chloride ions from them, which contributes to the dilation of the bronchi and small vessels of the circulatory system. In addition, potassium, as an intracellular electrolyte, prevents dehydration of the lungs and prevents the development of pneumosclerosis and pulmonary emphysema.

Excess aldosterone plays a pathophysiological role in the relationship between high blood pressure and obstructive sleep apnea syndrome in OSA. In particular, according to the results of studies conducted in obese patients, a higher level of aldosterone is detected compared to normosthenics. It is assumed that the development of obesity leads to an increase in the amount of aldosterone, which causes a predisposition to AG in these people, which leads to obstructive sleep apnea syndrome in OSOC patients. Thus, in the treatment of changes in the cardiovascular system observed in OSOC, it is recommended to use aldosterone receptor blockers that prevent the development of endothelial dysfunction, which helps to normalize vascular tone and restore myocardial function.

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