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EXPERIENCE OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN PATIENTS WITH ISCHEMIC DISEASE

Summary. At present, much attention is paid to the study of various soluble forms of adhesive molecules belonging to a new class of immunoregulatory microparticles in various pathological processes. It should be noted that information about the participation of these molecular forms in the pathogenesis of COPD and IHD is sporadic and contradictory. In addition, with the combination of COPD and IHD, exertional angina pectoris II, III functional classes (FC), there are no complex clinical studies devoted to the relationship of myocardial remodeling processes with biomarkers of the inflammatory process, including molecules of cell adhesion and adhesion of vascular walls, as well as pro-inflammatory cytokines and lipid profile ...

Key words: cardiovascular morbidity, left ventricular hypertrophy (LVH), glomerular filtration rate (GFR), blood pressure (BP).

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ОПЫТ ХРОНИЧЕСКОЙ ОБСТРУКТИВНОЙ БОЛЕЗНИ ЛЕГКИХ У БОЛЬНЫХ ИШЕМИЧЕСКОЙ БОЛЕЗНЬЮ

Резюме. В настоящее время большое внимание отводится изучению при разнообразных патологических процессах различных растворимых форм адгезивных молекул, относящихся к новому классу иммунорегуляторных микрочастиц. Необходимо отметить, что сведения об участии в патогенезе ХОБЛ и ИБС этих молекулярных форм единичны и разноречивы. Кроме того, при сочетании ХОБЛ и ИБС, стенокардии напряжения II, III функциональных классов (ФК) отсутствуют комплексные клинические исследования, посвященные взаимосвязям процессов ремоделирования миокарда с биомаркерами воспалительного процесса, включая молекулы клеточной адгезии и адгезии сосудистых стенок, а также провоспалительных цитокинов и липидного профиля.

Ключевые слова: сердечно-сосудистой заболеваемости, гипертрофии левого желудочка (ГЛЖ), клубочковой фильтрации (СКФ), артериального давления (АД).

Relevance. Chronic obstructive pulmonary disease is one of the main causes of morbidity and mortality and ranks 4th in the general population. Cardiovascular disease (CVD) in patients with COPD is detected in approximately 50%. The increased risk of cardiovascular complications in patients with COPD, according to some researchers, is due to the presence of systemic inflammation in these patients. It is known that markers of systemic inflammation in both COPD and atherosclerosis are an increase in the concentrations of inflammatory mediators, including C-reactive protein (CRP), tumor necrosis factor alpha (TNF-a), interleukin-4, interleukin-6. At present, much attention is paid to the study of various soluble forms of adhesive molecules belonging to a new class of immunoregulatory microparticles in various pathological processes. It should be

noted that information on the participation of these molecular forms in the pathogenesis of COPD and IHD is sporadic and contradictory. In addition, with the combination of COPD and IHD, exertional angina pectoris II, III functional classes (FC), there are no complex clinical studies devoted to the relationship of myocardial remodeling processes with biomarkers of the inflammatory process, including molecules of cell adhesion and adhesion of vascular walls, as well as pro-inflammatory cytokines and lipid profile.

Objective: to optimize the diagnosis of myocardial remodeling in patients with chronic obstructive pulmonary disease, combined with ischemic heart disease, exertional angina II, III functional classes, taking into account structural and functional features.

Materials and research methods. Inclusion criteria: patients with COPD II-IV spirometric stages, patients with coronary artery disease, angina pectoris II, III functional classes, patients with a combination of COPD II-IV spirometric stages and coronary artery disease, angina pectoris II, III functional classes. Criteria for exclusion: decompensated chronic cor pulmonale, persistent rhythm disturbances (atrial fibrillation, atrial flutter), pneumonia, unstable angina pectoris, acute and postponed myocardial infarction, CHF stages II-III.

Research results. Depending on the presence of concomitant coronary artery disease, the following groups of patients were identified: 1st - with COPD without concomitant coronary artery disease (n = 56); The second - with COPD + ischemic heart disease (n-31), and 5 people from this group had angina pectoris II FC and 26 people had angina pectoris III FC. Group 3 consisted of patients with coronary artery disease (n = 36), mainly with exertional angina SFC.

The control group consisted of clinically healthy individuals who did not have violations of spirometric parameters and ECG, comparable to the examined patients by sex and age (30 people, average age 52 ± 1.2 years, of which 19 were men, 8 were women). IHD was diagnosed by a specialist cardiologist based on the results of Holter ECG monitoring performed during the current or previous hospitalizations; in 37% - previously performed coronary angiography. Patients in

the COPD + IHD and IHD groups were prescribed cardiotropic therapy (ACE-i, Ca-channel blockers, antiplatelet agents, nitrates, statins), selective beta-blockers (in the IHD group). Patients from the COPD + IHD and COPD groups were admitted to the hospital with an exacerbation of respiratory disease and received standard therapy for the relief of exacerbation of COPD: nebulized short-acting and / or prolonged-acting bronchodilators (m-anticholinergics, (32 -agonists); nebulized forms of inhaled glucocorticosteroids) ; mu-colitis, as well as systemic glucocorticosteroids (sGKS) in a short course (5-10 days). In case of signs of an infectious exacerbation of COPD, patients were additionally prescribed antibiotics empirically. All patients were examined twice: in the period of exacerbation of COPD, in the first 1-3 days after admission to the department (I period) and in the phase of induction of COPD remission, after 8-12 days, before discharge (I period). Criteria for induction of remission: a decrease in the severity of dyspnea (NPV20 / min; dyspnea index on a scale <2), Ba02> 92%, a decrease in the severity of brochoobstructive syndrome (physically, an increase in FEV1 / PSV by 100 ml or more), heart rate [390 / min (clinically , ECG).

Output. We used general clinical and special research methods. The diagnosis of COPD was carried out according to the clinical guidelines [GOLD, 2012], and the severity of the course was established according to the results of spirography, taking into account the FEV value] and a decrease in the FEV1 / FVC index less than 70%. The degree of respiratory failure (DN) was determined according to pulse oximetry data. The severity of dyspnea was assessed using the Medical Research Counsel (mMRC) scale. ITI was calculated using the formula: ITI (packs / years) = number of cigarettes smoked per day x smoking experience (years) / 20. ITI> 10 is a significant risk factor for the development of chronic obstructive pulmonary disease. On an automatic ("Eton-01") pneumotachometer, the function of external respiration (FVD) was studied. Doppler echocardiography (Doppler Echo-KG) was performed on the APOGEE-SX apparatus using a transducer with a frequency of 3.5 MHz.

Evaluated end-systolic (ESR) and end-diastolic (EDD) dimensions, the thickness of the posterior wall of the left ventricle (LV) in systole (TZSLVS) and diastole (TZSLZhd), the thickness of the interventricular septum in systole (TMZHPS) and diastole) (TM the anterior wall of the heart (TPJ). When the ratio E / A <1.0, the hypertrophic type was determined, with E / A> 2 - the pseudo-normal type of diastolic dysfunction. Determined the ejection fraction (FI). The value of the MPA is> 30 mm Hg. was considered a marker of pulmonary hypertension.

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