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Comorbid patient with chronic obstructive pulmonary disease with ischemic heart disease and arterial hypertension

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Abstract. A comorbid patient with the chronic obstructive pulmonary disease (COPD) in combination with cardiovascular diseases (CHD) has a poor prognosis due to the early progression of the pulmonary hypertension (PH). The study surveyed an opportunity of the PH detection using an active PH verification strategy in outpatients with COPD and stable angina pectoris. Goal. A comorbid patient with the chronic obstructive pulmonary disease (COPD) in combination with cardiovascular diseases (CHD) has a poor prognosis due to the early progression of the pulmonary hypertension (PH). The study surveyed an opportunity of the PH detection using an active PH verification strategy in outpatients with COPD and stable angina pectoris.

Keywords: chronic obstructive pulmonary disease, angina pectoris, arterial hypertension, comorbidity, pulmonary hypertension, echo.

Arterial hypertension (AH) remains one of the most pressing health problems due to its high prevalence and socioeconomic significance. A distinctive feature of hypertension is the high frequency of comorbidity. Patients with hypertension usually have one or more comorbidities, the most common of which include chronic obstructive pulmonary disease (COPD), cerebrovascular disease, diabetes mellitus, hypothyroidism, and others. Comorbidity leads to a mutual influence on the course of diseases, the nature and severity of complications, often complicates diagnosis, and determines the features of the choice of antihypertensive drugs. Arterial hypertension and chronic obstructive pulmonary disease Currently, one of the frequent comorbid conditions in the clinic of internal diseases is hypertension and COPD, associated with high rates of disability and death. The increase in the number of patients with a combination of AH and broncho-obstructive diseases is due to both an increase in the incidence of AH and CW. Introduction. A comorbid patient with chronic obstructive pulmonary disease (COPD) in combination with coronary heart disease (CHD) has a poor prognosis due to the early development of pulmonary hypertension (PH). The study investigated the possibility of detecting PH using an active verification strategy in outpatients with COPD and stable angina. A comorbid patient with chronic obstructive pulmonary disease (COPD) in combination with coronary heart disease (CHD) has a poor prognosis due to the early development of pulmonary hypertension (PH). The study investigated the possibility of detecting PH using an active verification strategy in outpatients with COPD and stable angina. A comorbid patient with chronic obstructive pulmonary disease (COPD) in combination with coronary heart disease (CHD) has a poor prognosis due to the early development of pulmonary

hypertension (PH). The study investigated the possibility of detecting PH using an active verification strategy in outpatients with COPD and stable angina.

An increase in the area of the right atrium according to echocardiography, along with other indicators of morphological and functional changes in the right parts of the heart, may be an additional diagnostic criterion for PH in comorbid patients with COPD and IHD.

Difficulties in the treatment of patients with hypertension and COPD are due to the fact that some antihypertensive drugs (beta-blockers, angiotensin-converting enzyme inhibitors (ACE inhibitors)) and bronchodilators can have an undesirable effect, aggravating the course of comorbid pathology.

According to a number of scientific studies, the leading cardiovascular pathology in patients with chronic obstructive pulmonary disease (COPD) is coronary heart disease (CHD) and its most common form - angina pectoris. The prevalence of this comorbid pathology is 20-22%, while in both sexes in the general population; angina pectoris is detected in approximately 9% of cases. A patient with COPD and IHD is prognostically unfavorable, since this combination of diseases is considered from the standpoint of the existence of close pathophysiological relationships between them, leading to mutual aggravation. In patients with COPD with coronary artery disease with little experience of disease, signs of functional changes in the right and left parts of the heart are already determined, which leads to a deterioration in the clinical course of COPD, aggravation of ventilation disorders, increased remodeling of the right and left ventricles, and the development of pulmonary hypertension (PH) largely determines the poor prognosis of comorbid syndrome, so the timely detection of PH and its treatment is one of the most difficult, but extremely important tasks facing a cardiologist, internist, and general practitioner. PH is diagnosed when the mean pulmonary artery pressure (Mean PAP) is equal to or greater than 25 mm Hg. Art.

When examining a patient at rest according to the data of catheterization of the heart cavities. According to the existing modern classification, PH that developed as a result of lung pathology and / or hypoxemia is classified as group 3, and according to the hemodynamic classification it is characterized as precapillary. According to some authors, the prevalence of PH in isolated COPD ranges from 20 to 63%. This information is not inconsistent with research findings. where PH was detected in 35–66% of severe patients with COPD. However, the epidemiology of PH groups 2–3 (due to pathology of the left heart and lung pathology and / or hypoxia) remains an insufficiently studied problem, although it often occurs in practice. So, in 2015, A. M. Kirillov revealed in comorbid patients with COPD and IHD the maximum levels of LH, compared with isolated COPD, and a very strong correlation both with spirometric parameters and with the size of the right ventricle and confirmed the propensity of this part of the heart when overloaded, to a greater extent to dilation than to hypertrophy. As the literature search shows, with the above comorbidity, a

more complex restructuring of the heart chambers occurs, and as COPD progresses, the size of the right ventricle increases, somewhat ahead of the processes of remodeling of the pulmonary artery and the development of pulmonary hypertension, and the earliest changes in the heart recorded in patients with COPD during echocardiography (EchoCG) are a slight relative dilatation of the right ventricle (RV) and right atrium (RA). This also confirmed that coronary artery disease, of course, aggravates the course of COPD, enhances remodeling of the right heart, diastolic dysfunction of the right and left ventricles. They revealed changes in the end-diastolic size of the right ventricle and established correlations between the sizes of the right and left ventricles with the level of MAP. The 2015 ESC / ERS recommendations for the diagnosis and treatment of pulmonary hypertension contain indications of the need to use in practice not only MAP based on the calculation of the peak rate of tricuspid regurgitation, including the contrast method, but also other echocardiographic parameters that may raise or reinforce the suspicion of PH regardless of the rate of tricuspid regurgitation. Although few studies have been conducted to quantify the size of the RA, according to experts in the field of echocardiography, the area of the right atrium can become one of the evaluation criteria for the diagnosis of PH. The foregoing regarding the lack of information about the prevalence of PH in comorbid patients with non-severe COPD in combination with coronary artery disease and the possibility of its early active detection was the reason for this study. The area of the right atrium can become one of the evaluation criteria for the diagnosis of PH. The foregoing regarding the lack of information about the prevalence of PH in comorbid patients with non-severe COPD in combination with coronary artery disease and the possibility of its early active detection was the reason for this study. The area of the right atrium can become one of the evaluation criteria for the diagnosis of PH. The foregoing regarding the lack of information about the prevalence of PH in comorbid patients with non-severe COPD in combination with coronary artery disease and the possibility of its early active detection was the reason for this study.

Thus, the combination of IHD and COPD has a negative impact on the state of the vascular wall. Endothelial damage and endothelial dysfunction in comorbid pathology are more pronounced than in monopathology.

Cardiovascular diseases, including coronary artery disease, are one of the main causes of primary and repeated hospitalizations of patients with COPD, one of the main causes of death in patients with COPD. The long latent period between the onset of the disease and the clinical manifestation of atherosclerosis requires the identification of individuals with a subclinical, potentially reversible stage of cardiovascular disease. In this regard, various markers of subclinical atherosclerosis are being studied as potential predictors of cardiovascular risk. The thickness of the intima-media complex (ITM) of PA is considered as a new, easily determined parameter for verification of subclinical atherosclerosis.

The importance of TIM PA as an independent predictor of cardiovascular events has been demonstrated. In our work in patients with COPD (both with the presence, and the absence of coronary artery disease) showed signs of VA remodeling in the form of VA wall thickening and damage to the vascular wall. At the same time, in patients with COPD and CAD, these manifestations are somewhat less pronounced than in patients with COPD without CAD. This may be due to the fact that after myocardial infarction, patients receive more active and regular therapy (angiotensin-converting enzyme inhibitors, statins, etc.). In our work, no relationship was found between IMT PA and the risk of cardiovascular events, while in the study by MH Urban et al. (2017) found a two-fold increase in the 10-year risk of cardiovascular events in patients with COPD and elevated IMT PA. Perhaps this is due to different risk assessment methods (SCORE and Framingham scale).

At the same time, the relationships we have identified between the development of atherosclerosis in COPD patients and impaired flow-dependent vasodilation are supported by data from other authors. Early diagnosis of any disease is a determining factor for timely effective treatment with a favorable prognosis and prevention of complications. The endothelium is an active metabolic organ that becomes the first target organ in cardiovascular diseases; endothelial dysfunction is one of the key links in pathogenesis and precedes vascular damage, regardless of their organ localization. Evaluation of vasoregulatory endothelial function has now been convincingly shown to correlate with other invasive methods, and this procedure is presented for non-invasive and rapid peripheral evaluation of arterial function. Flow-dependent vasodilation is an independent predictor of cardiovascular morbidity and mortality and correlates well with invasive assessment of coronary artery endothelial function and CAD severity. Violations of the vasoregulatory activity of the endothelium in patients with COPD have previously been demonstrated in some studies. In our work, as in a number of others, it was found that endothelial dysfunction in patients with COPD in combination with CAD is more pronounced than in patients with COPD without CAD or in patients with CAD without COPD. Comparison of the results of the study of endothelial function in patients of the two groups presents certain methodological difficulties, since in some patients the blood flow velocity significantly increased during the test with RG, but there was no significant change in the diameter of the vessel, while in other patients, the blood flow velocity changed less with a significant increase in the diameter of the artery. Considering these changes, we calculated the sensitivity of PA to a change in the stimulus - shear stress on the endothelium in patients with COPD with and without CAD.

In patients with COPD, both with CAD and without CAD, a decrease in the sensitivity of PA to shear stress during the test with RG was found, which indicates a pronounced violation of the vasoregulatory function of the vascular wall in this category of patients. Our results confirm the data of other authors on a decrease in the

sensitivity of PA to shear stress on the endothelium due to increased blood flow in comorbid patients with a combination of coronary artery disease and COPD. the sensitivity of PA to a change in the stimulus - shear stress on the endothelium in patients with COPD with and without CAD was calculated. In patients with COPD, both with CAD and without CAD, a decrease in the sensitivity of PA to shear stress during the test with RG was found, which indicates a pronounced violation of the vasoregulatory function of the vascular wall in this category of patients. Our results confirm the data of other authors on a decrease in the sensitivity of PA to shear stress on the endothelium due to increased blood flow in comorbid patients with a combination of coronary artery disease and COPD. the sensitivity of PA to a change in the stimulus - shear stress on the endothelium in patients with COPD with and without CAD was calculated. In patients with COPD, both with CAD and without CAD, a decrease in the sensitivity of PA to shear stress during the test with RG was found, which indicates a pronounced violation of the vasoregulatory function of the vascular wall in this category of patients. Our results confirm the data of other authors on a decrease in the sensitivity of PA to shear stress on the endothelium due to increased blood flow in comorbid patients with a combination of coronary artery disease and COPD. which indicates a pronounced violation of the vasoregulatory function of the vascular wall in this category of patients. Our results confirm the data of other authors on a decrease in the sensitivity of PA to shear stress on the endothelium due to increased blood flow in comorbid patients with a combination of coronary artery disease and COPD. which indicates a pronounced violation of the vasoregulatory function of the vascular wall in this category of patients. Our results confirm the data of other authors on a decrease in the sensitivity of PA to shear stress on the endothelium due to increased blood flow in comorbid patients with a combination of coronary artery disease and COPD. which indicates a pronounced violation of the vasoregulatory function of the vascular wall in this category of patients. Our results confirm the data of other authors on a decrease in the sensitivity of PA to shear stress on the endothelium due to increased blood flow in comorbid patients with a combination of coronary artery disease and COPD.

Conclusions. The problem of hypertension, coronary artery disease and comorbidity with its versatility attracts the attention of both scientists and clinicians, is the most frequently discussed topic on the forums of various levels, remains relevant for further scientific research, since many questions remain insufficient studied. The presence of comorbidity requires an individual approach to the patient, complex diagnostics and treatment, taking into account all available pathologies.

REFERENCES.

1. Global Strategy for diagnosis, management and prevention of COPD. Scientific information and recommendations for COPD programs. Updated 2013. http://www.goldcopd.org/uploads/users/files/GOLD_Report_2013Feb13.pdf
2. Engstrom G, Lind P, Hedblad B, et al. Lung function and cardiovascular risk. *circulation*. 2002;106(20):2555-60.

3. Sin DD, Wu L, Man SF. The relationship between reduced lung function and cardiovascular mortality: a population-based study and a systematic review of the literature. *Chest*. 2005;127(6):1952-9. <https://doi:10.1378/chest.127.6.1952>

4. Sin DD, Anthonisen NR, Soriano JB, Agusti AG. Mortality in COPD: role of comorbidities. *Eur Respir J*. 2006;28(6):1245-57. <https://doi:10.1183/09031936.00133805>

5. Macnee W, Maclay J, McAllister D. Cardiovascular injury and repair in chronic obstructive pulmonary disease. *Proc Am Thorac Soc*. 2008;5(8):824-33. <https://doi:10.1513/pats.200807-071TH>

6. Decramer M, Janssens W. Chronic obstructive pulmonary disease and comorbidities. *Lancet Respir Med*. 2013;1(1):73-83. [https://doi:10.1016/S2213-2600\(12\)70060-7](https://doi:10.1016/S2213-2600(12)70060-7)

7. Pływaczewski R, Maciejewski J, Bednarek M, Zieliński J, et al. Causes of deaths in COPD patients in primary care setting - a 6-year followup. *Pneumonol Alergol Pol*. 2015;83(3):193-202. <https://doi:10.5603/PiAP.2015.0031>

8. Curkendall SM, DeLuise C, Jones JK, et al. Cardiovascular disease in patients with chronic obstructive pulmonary disease, Saskatchewan Canada: cardiovascular disease in COPD patients. *Ann Epidemiol*. 2006;16(1):63-70. <https://doi:10.1016/j.annepidem.2005.04.008>

9. Sabit R, Thomas P, Shale DJ, et al. The effects of hypoxia on markers of coagulation and systemic inflammation in patients with COPD. *Chest*. 2010;138(1):47-51. <https://doi:10.1378/chest.09-2764>

10. MacNee W. Pulmonary and systemic oxidant/antioxidant imbalance in chronic obstructive pulmonary disease. *Proc Am Thorac Soc*. 2005;2(1):50-60. <https://doi:10.1513/11>. Chazova I.E., Chuchalin A.G., Zykov K.A., Ratova L.G. Diagnosis and treatment of patients with arterial hypertension and chronic obstructive pulmonary disease (recommendations of the Russian Medical Society for Arterial Hypertension and the Russian Respiratory Society). // Systemic hypertension. - 2013. - V.10, No. 1. - P.5-34. 12. Grigorieva N.Yu. Comorbid patient with hypertension and COPD. // Attending doctor. - 2016. - No. 7. - P.24-27. 13. Mortensen EM, Copeland LA, Pugh MJ, Restrepo MI, de Molina RM, Nakashima B. et al. Impact of statins and ACE inhibitors on mortality after COPD exacerbations. // *Respiratory Res*. - 2009. - V.10. – P.45. doi: