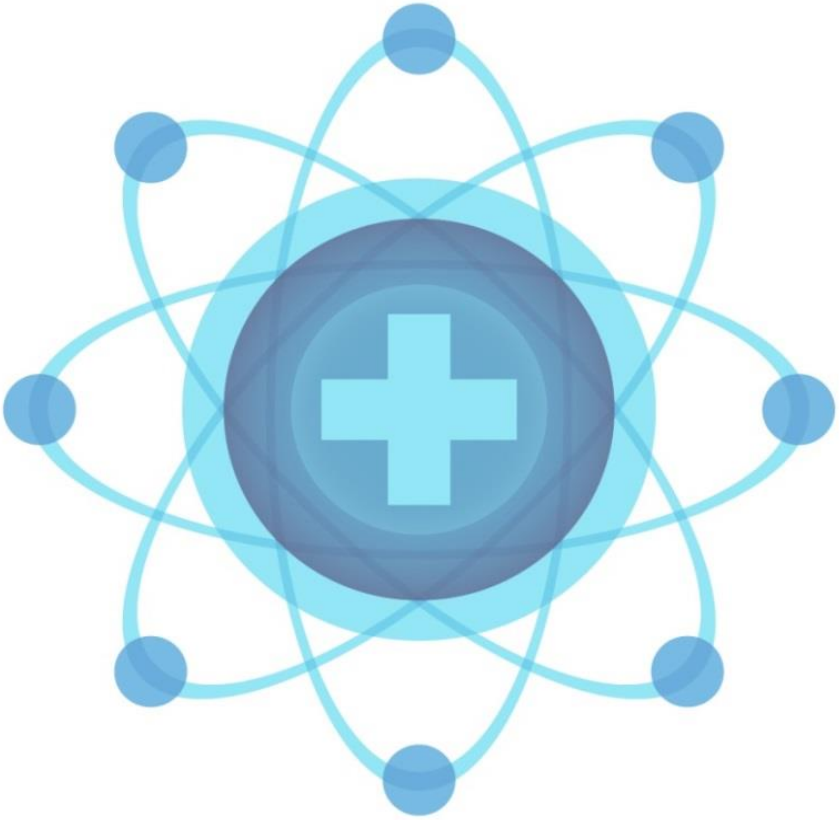

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EVALUATION OF CARDIOSPIRATORY INDICATORS ON THE BASIS OF COMPLEX TREATMENT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH PULMONARY HYPERTENSION

Kholov G.A., Djuraeva N.O.
Bukhara regional multi-profile clinical center, Bukhara state medical institute, Bukhara, Uzbekistan

Abstract. Chronic obstructive pulmonary disease (COPD) is one of the most common diseases among the population, and its pathogenesis is influenced by inflammation, endothelial dysfunction, oxidative process, as well as changes in the state of hypoxia of the lungs and other organs. Improving the diagnosis and treatment of this disease is one of the urgent and complex tasks of modern pulmonology. This is also confirmed by the increasing number of patients suffering from COPD all over the world.

According to the results of a number of studies, the prevalence of COPD among the population over 40 years of age is 11.8% in men and 8.5% in women, and the average is 10.1%. It is one of the leading causes of physician visits, emergency department visits, and hospitalizations.

Keywords: pulmonary hypertension, chronic obstructive pulmonary disease, hypoxia, hypercapnia.

Actuality. According to the available data, about 20% of patients with COPD and treated in the hospital were diagnosed with OG [2,5,9,12]. In COPD with a severe clinical sign, OG was recorded in more than 50% of cases. OG is considered a multifactorial pathophysiological condition in which vasoconstriction, obstructive remodeling, inflammatory and biochemical changes and other processes are involved [1,3,4,11]. Among them, pathological activity of potassium channels in smooth muscle cells and endothelial dysfunction are of particular importance, considering that vasoconstriction occurs early in the formation of OG. Intestinal peptide, a substance with vasodilating and antifibrotic effects, decreases in the blood serum of patients with OG [8,10]. Endothelial dysfunction leads to chronic depletion of vasodilators such as nitric oxide and protocyclin and concomitant overproduction of vasoconstrictors such as thromboxane A2 and endothelin-1 [6,7,9]. Most of these noted changes increase vascular tone and cause remodeling in them.

The purpose of the study. Coordination of treatment measures by identifying changes in cardiac hemodynamics, external respiratory activity, gas content of blood in patients with chronic obstructive pulmonary disease and pulmonary hypertension.

Research materials and methods. 120 patients with pulmonary hypertension of various degrees of severity were taken as the object of research in the multidisciplinary medical center of Bukhara region. Follow-ups were divided into 3 functional classes (II, III, IV) according to the Eurasian clinical recommendation for the diagnosis and treatment of pulmonary hypertension (2019) and according to the treatment methods used (bosentan or sildenafil on the basis of the complex standard treatment of COPD), each of them was less than two comparison in groups was studied.

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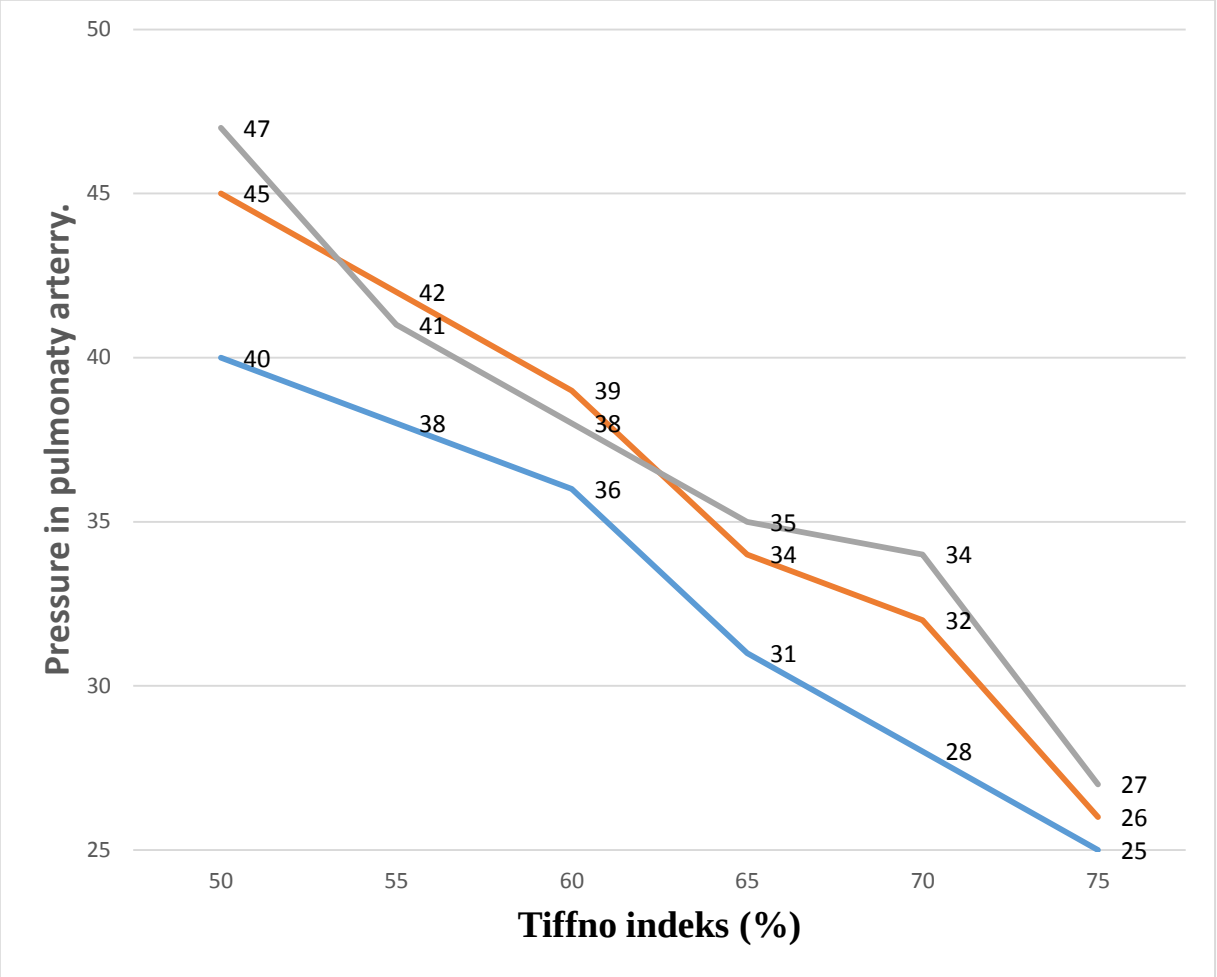
Planned examinations were carried out before and 3 months after treatment. Questionnaire and scales, static methods were used to compare the obtained data (clinical, laboratory and functional-instrumental examination results). Clinical and biochemical analyzes of blood (ALT, AST, bilirubin, etc.), blood gas content (SO2, PaO2, SaO2) and indicators of external respiratory activity, instrumental examinations - EKG, ExoKG (central hemodynamic indicators) before treatment and during 6 months of the treatment process. was conducted and their physical activity and emotional state, professional adaptation, and satisfaction with treatment were evaluated using the Seattle questionnaire. Statistical analysis was carried out based on the Pearson square method.

Research results. Impairment of external breathing is important in COPD, and it is one of the factors leading to increased pulmonary hypertension. Taking this into account, we studied the correlation between the Tiffno index, which is considered one of the main indicators of external respiratory activity, and heart hemodynamics and size indicators, as well as blood gas content in patients with COPD pulmonary hypertension. There is a correlation between Tiffno index and OASB and pulmonary artery internal pressure in patients with COPD II severity level, respectively r = -0.3; P< 0.05 and r = -0.4; P< 0.005 negative correlation relationship was noted. This determined indicator confirms that a decrease in the Tiffno index leads to an increase in the pressure in the pulmonary artery in parallel. Also, a decrease in this indicator leads to an increase in the diameters of the right ventricle and pulmonary artery (r = -0.3; P< 0.05 and r = -0.4; P< 0.007, respectively). The Tiffno index was negatively correlated with blood gas content, in particular, pCO2 (r = -0.5; P< 0.001) and positively (r = 0.66; P< 0.001) with pO2. Naturally, a decrease in the Tiffno index led to an increase in pCO2 and a decrease in pO2 in the blood.

There is III severity of the disease, that is, in the second group of patients, between Tiffno's index and O'ASB and the internal pressure of the pulmonary artery, respectively, r = -0.39; P< 0.01 and r = -0.6; A negative correlation of P< 0.001 was noted.Also, a decrease in this indicator leads to an increase in the diameters of the right lobe, right ventricle and pulmonary artery (r = -0.46; P< 0.003, r = -0.48; P< 0.002 and r = -0.38; P < 0.001). The Tiffno index was negatively correlated with blood gas content, in particular, pCO2 (r = -0.6; P< 0.001) and positively (r = 0.77; P< 0.001) with pO2. Naturally, a decrease in the Tiffno index led to an increase in pCO2 and a decrease in pO2 in the blood. In addition to the above, end-diastolic and systolic size (r = -0.38; P< 0.01 and r = -0.55; P< 0.001, respectively) and end-diastolic and systolic volume (r = -0, respectively) with the Tiffno index .45; P< 0.003 and r = -0.6; P< 0.001) also revealed inverse reliable correlations. These figures confirm that as the severity of COPD is accompanied by pulmonary hypertension, remodeling of the right and left parts of the heart is added to the process, and negative changes in their size and volume are observed.

In the third group of patients under our observation, there was a negative (r = -0.39; P< 0.01) correlation between the indicators that were found to be correlated

with the Tiffno index in the second group above, as well as the size of its left.



The identified changes confirm the existence of an organic relationship between Tiffno's index, which is one of the main indicators of external respiratory activity, and pulmonary hypertension and heart hemodynamics and sizes in our patients. When the chronic obstructive pulmonary disease is accompanied by pulmonary hypertension (different functional classes) of various severity levels, the Tiffno index decreases, causing the studied indicators to shift to the negative side.

It is known that one of the changes observed in the cardiovascular system of COPD is the dysfunction of the right ventricle, right ventricle, and pulmonary hypertension in a number of cases. In the existing literature, very little attention has been paid to the changes in the left ventricle when COPD is accompanied by pulmonary hypertension. In addition, in this group of patients, we did not find information on the comparative study of the effect of endothelin receptor antagonist - usenta 125 (bosentan) and sildenafil, a typical representative of the 5-type phosphodiesterase group, with eplerenone on cardiac hemodynamics. Taking this into account, as mentioned in the second chapter, 120 patients diagnosed with COPD and pulmonary hypertension were divided into groups based on their severity and recommended complex treatments, and intracardiac hemodynamics were evaluated. The first group consisted of 40 patients with COPD II functional class (severity) pulmonary hypertension, who were divided into two subgroups on the basis of

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standard treatment. The first subgroup consisted of 20 patients who received bosentan and eplerenone on the basis of standard treatment, and the second group of 20 patients who received eplerenone together with sildenafil (table-1).

Table 1. Peripheral blood pressure and echocardiographic parameters before and after treatment in chronic obstructive pulmonary disease with functional class II pulmonary hypertension.

№	Indicators	Chronic obstructive pulmonary disease complicated by pulmonary hypertension II functional class n =40				
		Standard treatment + bosentan + eplerenone n=20		Standard treatment + sildenafil + eplerenone n=20		Confidence level (P) of the difference between the two groups after treatment
		Before treatment	After treatment	Before treatment	After treatment	
1	Systolic arterial blood pressure (mm. sym. above)	130±3,2	118,6±1,89***	132±3,4	120,25±1,79***	P>0,05
2	Diastolic arterial blood pressure (mm. sim. above)	82,75±1,57	75,72±1,22***	79,6±2,4	78,7±1,05	P<0,05
3	Mean arterial blood pressure (mm. sym. above)	98,5±1,7	92,55±0,86***	97,07±2,7	92,55±0,86	P>0,05
4	Left piece (mm)	35.2 ±1.2	33.9 ±1.2	35.5±1.04	34.3±1.04	P>0,05
5	Right piece (mm)	36.45±0.5	33.45±0.5***	36.05±0.36	35.1±0.37	P<0,001
6	Right ventricle (mm)	28.6±0.57	26.6±0.56*	29.25±0.56	27.4±0.54	P>0,05
7	End systolic volume (ml)	37.75±1.5	35.85±1.5	38.3±1.85	36.4±1.85	P>0,05
8	End diastolic volume (ml)	94.9±4.99	92.9±4.99	95.5±5.14	93.6±5.17	P>0,05

9	**End systolic size (cm)**	2.9±0.064	2.6±0.064**	2.83±0.08	2.75±0.09	P<0,05
10	**End diastolic size (cm)**	4.6± 0.09	4.3± 0.09*	4.58±0.10	4.39±0.10	P<0,05
11	**Pulmonary artery diameter (mm)**	22.35±0.20	20.65±0.19***	22.15±0.31	21.65±0.19**	P<0,001
12	**Pulmonary artery systolic pressure (mm.sim.ust)**	36.7±0.56	27.7±0.56***	36.25±0.65	30.9±0.68***	P<0,001
13	**Left ventricular ejection fraction (%)**	61.1±1.16	64.4±1.16	62.1±1.16	64.2±1.16	P>0,05

Systolic arterial blood pressure decreased reliably after treatment in both subgroups, respectively (130±3.2 mmHg and 118.6±1.89 mmHg. R<0.001 and 132±3.4 mmHg. wire height and 120.25±1.79 mm wire height).

Diastolic and mean blood pressure values decreased reliably after treatments only in the group receiving bosentan on the basis of complex treatment (R<0.001, table-1). This suggests that bosentan has a greater hypotensive effect than sildenafil.

In the conducted echocardiographic examinations, it was found that the size of the left ventricle was 35.2±1.2 mm in the first subgroup and 33.9±1.2 mm after the procedures. In the second subgroup, these numbers were 35.5±1.0 mm and 34.3±1.0 mm, respectively, and no reliable changes were observed in either subgroup after treatment (R>0.05). After the treatments, the indicators in the first subgroup decreased by 3.6%, and in the second subgroup by 3.4%, the difference between them (0.2%) was unreliable (R>0.05).

Right lobe size was 36.45±0.5 mm to 33.45±0.5 mm before and after treatments in the first subgroup, respectively. decreased to , the difference between them was equal to 8.2% and a reliable (R<0.001) change was observed. In the second subgroup, these numbers were equal to 36.05±0.36 and 35.1±0.37 mm, and the values before treatment and after it differed by 2.5% (R>0.05). Post-treatment scores were 5.7% higher in the first subgroup than in the second, and the differences were reliable (R<0.001).

Right ventricular size in the first subgroup was 28.6±0.57 and 26.6±2.5 mm before and after treatments, respectively. was equal to and reliably decreased by 7% (R<0.05).In the second subgroup, these numbers were 29.25±0.56 and 27.4±0.5 mm, decreased by 3.5%, and the differences were not reliable (R>0.05). The difference between the two subgroups was 3.49%, and although the former did not reliably differ from the latter, the scores were significantly higher.

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This confirms that bosentan, when used together with eplerenone, has a positive effect not only on the right ventricle, but also on the hemodynamic parameters of the right ventricle.

The end-systolic volume in the first subgroup was 37.75±1.5 and 35.85±1.5 ml before and after treatment, respectively, and the differences were 5% (R>0.05). In the second subgroup, the indicators were equal to 38.3±1.85 and 36.4±1.8 ml, respectively, and the difference before and after the treatments was 4.96% (R>0.05). The difference between the two subgroups after treatment was 0.04%, and it was confirmed that the indicators did not differ from each other reliably (R>0.05).

End-diastolic volume was 94.9±5 and 92.9±5.0 ml before and after treatment in the first subgroup, and 95.5±5.14 and 93.6±5.17 ml in the second subgroup, respectively. In this case, the difference was 2.1% in the first subgroup and 2.0% in the second subgroup, and the change after treatment was not reliably different between the two groups and was equal to 0.1% (R>0.05).

A comparative analysis showed that bosentan and sildenafil had similar effects on end-diastolic and systolic volume in subgroups with eplerenone. But in both groups, these indicators were unreliable (R>0.05).

The end-systolic size was 2.9±0.06 cm and 2.6±0.06 cm before and after the treatments in the first subgroup, respectively. The difference between them was 10.3%. In the second subgroup, these indicators were 2.83±0.08 cm and 2.75±0.09 cm, respectively, before and after treatment, and no difference was detected. In the first bosentan and eplerenone subgroup, end-systolic size was reliably reduced by 10.3% after the treatments (R<0.001). When the post-treatment differences were compared between the two subgroups, it was found that the systolic size was significantly reduced in the former (R<0.05).

The end-diastolic size in the first subgroup was 4.6±0.09 and 4.3±0.09 cm before and after treatment, respectively, and the difference between them was reliable (R<0.05). In the second subgroup, the differences after treatment were 4.58±0.1 and 4.39±0.1, respectively, and were not reliable (R>0.05). Post-treatment scores improved by 6.5% in the first subgroup and 4.1% in the second. These changes were highly reliable (R<0.05) in the first subgroup compared to the second (2.4%). This indicates a significant reduction in end-diastolic size values when bosentan is used in combination with eplerenone.Pulmonary artery diameter was 22.35±0.2 before treatment and 20.65±0.19 after treatment, respectively, in the subgroups receiving bosentan and eplerenone based on the first standard treatment of COPD. and the differences were equal to 8.8% (R<0.001). In the second group, i.e. sildenafil and eplerenone, the values before and after the treatments were 22.15±0.2 and 21.65±0.1 mm, respectively. organized. The difference between pre-treatment and post-treatment was 4.4% in this group was reliable (R<0.01). In addition, the first post-treatment group score was 4.4% higher than the second group, which confirms that bosentan has a reliable (R<0.05) higher lowering effect on pulmonary artery pressure than sildenafil.

Also, in the subgroup that received bosentan, the difference in pulmonary artery systolic pressure after treatment compared to the pretreatment value changed

by 28.4% in the positive direction (36.7±0.56 and 27.7±0.56 mm Hg, respectively) (R<0.001). In the group receiving sildenafil, it was equal to 19.6% (R<0.01) (respectively, 36.25±0.65 and 30.9±0.68 mm.sim.super.). The difference between groups after treatment was 8.8% higher in the first group than in the second group (R<0.001), which indeed indicates a greater positive effect of bosentan in reducing pulmonary artery pressure compared to sildenafil.

Left ventricular ejection fraction parameters were evaluated before and after treatments in both subgroups.Indicators in the first subgroup were 61.1±1.16% and 64.4±1.0% respectively (difference 5.4%, R>0.05) and in the second subgroup 62.1±1.1% and It was equal to 64.2±1.0% (difference 3.38%, R>0.05). The difference in positive change between groups after treatment was 2.02%, and the values were not reliable (P>0.05). In both subgroups, cardiac ejection fraction changed positively after the treatments, but they were not reliable. But bosentan has been confirmed to be more effective than sildenafil.

Conclusions.

1. In patients with chronic obstructive pulmonary disease and pulmonary hypertension, cardiac hemodynamics, blood gas content, and indicators of external respiratory activity change negatively in parallel with the severity of the disease.

2. When the chronic obstructive pulmonary disease is accompanied by pulmonary hypertension, the systolic pressure in the pulmonary artery in the II-III-IV stages of the disease in the right ventricle r = 0.4, P< 0.01; r = 0.7, P< 0.01; r = 0.52, P< 0.01 and between right lobe size r = 0.35, P< 0.02; r = 0.47; P< 0.002 positive correlation was found. Positive correlations were noted between pulmonary artery systolic pressure and diastolic and systolic volume (r = 0.55, P< 0.001 and r = 0.39, P< 0.01) in the course of the disease, not only in the right, but also in the left heart. confirms the participation of parts.

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