ALTERNATIONS IN THE STRUCTURE OF THE BRONCHOPULMONARY AND CARDIOVASCULAR SYSTEMS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract. *The World Health Organization (WHO) predicts that chronic obstructive pulmonary disease (COPD) will rank as the leading cause of death globally by 2030. Currently, around 3 million deaths annually are attributed to this condition. The main risk factors for COPD are smoking and metabolic syndrome, especially obesity, which also contribute to tumor development and are key factors in cardiovascular disease such as coronary heart disease and hypertension.*

The progression of these disorders is driven by various pathogenetic mechanisms that result in indirect vascular damage, endothelial dysfunction, and arterial thickening. Contributing factors include hypoxemia, diabetes, smoking, oxidative stress, and elevated systemic inflammation, both at rest and during physical exertion.

Key words: *chronic obstructive pulmonary disease, ischemic heart disease, cor pulmonale, oxidative stress.*

ИЗМЕНЕНИЯ В СТРОЕНИИ БРОНХОЛЕГОЧНОЙ И КАРДИОВАСКУЛЯРНОЙ СИСТЕМ ПРИ ХРОНИЧЕСКОЙ ОБСТРУКТИВНОЙ БОЛЕЗНИ ЛЕГКИХ

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Аннотация. *По данным Всемирной организации здравоохранения (ВОЗ), к 2030 году хроническая обструктивная болезнь легких (ХОБЛ) окажется на третьем месте среди основных причин смертности в мире. Ежегодно от этого заболевания погибает около 3 миллионов человек. Главными факторами риска являются курение и метаболический синдром, включая ожирение, которые не только способствуют возникновению опухолей, но и играют важную роль в развитии сердечно-сосудистых заболеваний, таких как ишемическая болезнь сердца и гипертония. Развитие этих расстройств связано с несколькими патогенетическими процессами, которые вызывают косвенное повреждение сосудов, дисфункцию эндотелия и утолщение артерий. К таким факторам относятся гипоксемия, диабет, курение, окислительный стресс и системное воспаление, как в покое, так и при физической активности.*

Ключевые слова: *хроническая обструктивная болезнь легких, ишемическая болезнь сердца, гипертония, окислительный стресс.*

SURUNKALI OBSTRUKTIV O‘PKA KASALLIGIDA BRONXPULMONAR VA KARDIOVASKULYAR TIZIMIDAGI STRUKTURAVIY O‘ZGARISHLAR

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Annotatsiya. *Dunyo bo‘yicha JSST ma’lumotlari bo‘yicha, 2030 yilga kelib, surunkali obstruktiv o‘pka kasalligi (SOO‘K) o‘lim sabablarida uchinchi o‘rinni egallaydi. Hozirda har yili 3 millionga yaqin odam SOO‘K tufayli vafot etadi. Chekish va metabolik sindrom, ayniqsa semizlik, SOO‘K rivojlanishida yondosh kasaliklar turli yurak kasalliklari, jumladan, yurak ishemik kasalligi va gipertoniya rivojlanishining asosiy xavf omillaridan hisoblanadi. Ushbu kaslliklar rivojlanish jarayonida turli patogenetik mexanizmlar yuazaga keladi. Agar bemorda o‘pkada patologik o‘zgarishlar bo‘lsa, demak, yurakda ham muammolar mavjud bo‘lishi ehtimoli katta. Oqibatda surunkali o‘pka yurak, turli ritm buzilishlari va o‘pka tromboemboliyasining rivojlanishiga olib kelishi mumkin. SOO‘Kda renin-angiotenzin-aldosteron tizimi (RAAT) va boshqa neyrogormonlar hamda mediatorlarning (sitokinlar, endotelin, vazopressin va boshqalar) yurak rivojlanishiga sabab bo‘ladi.*

Kalit so‘zlar: *o‘pkaning surunkali obstruktiv kasalligi, yurak ishemik kasalligi, gipertoniya, o‘pkali yurak, oksidlovchi stress.*

Introduction.

When addressing the development of ChHF in patients with COPD, it is important to consider the presence of chronic heart defects. This condition is regarded as one of the pressing issues in contemporary medicine. In both instances, the SRAA system is triggered [Обрезан, А. Г., & Куликов, Н. В. (2017). Нейрогуморальный дисбаланс при хронической сердечной недостаточности: классические и современные позиции. *Российский кардиологический журнал*, (9 (149)).] It is crucial to note that SRAA activation takes place not only in the

circulatory system but also at the tissue level, particularly in the lungs. [Селезнева, Е., & Жданова, Т. (2015). Роль нейрогуморальной активации в развитии АГ при хронической обструктивной болезни легких. *Врач*, (5-8).]. According to several authors, COPD 30 is associated with the development of pulmonary hypertension in 50% of cases. Two-thirds of patients with copd die within 15 months to 5 years after the onset of circulatory failure decompensation. Among the causes of mortality in individuals under 40 years of age, it ranks third, following arterial hypertension and chronic coronary insufficiency. A direct contributor to the development of arterial hypertension is HIC and ChHF. Chronic obstructive pulmonary disease (COPD) is currently one of the most prevalent chronic inflammatory diseases of the lower respiratory tract and represents a significant global medical and social issue. [8,9,14]. Chronic obstructive bronchitis ranks 5th among all diseases in terms of prevalence and 3rd in terms of mortality. According to the WHO, diabetes mellitus is currently the 3rd leading cause of death globally, with approximately 2.8 million deaths annually, accounting for 4.8% of all deaths. [1,7]. The development of cardiovascular pathology in copd is influenced not only by the onset of various forms of pathology but also by the presence of cardiorespiratory rigidity. A direct contributor to the formation of arterial hypertension in this context is copd, HIC, and ChHF. These disorders arise from a combination of pathogenetic mechanisms that result in indirect vascular damage, endothelial dysfunction, and arterial stiffness. Increased levels of angiotensin II in the lungs may also contribute to reduced alveolar gas exchange, lung inflammation, and pulmonary vasoconstriction. [Авдеев, С. Н. (2020). Современные подходы к диагностике и терапии легочной гипертензии у пациентов с хронической обструктивной болезнью легких. *Пульмонология*, (1), 90-101.]. These factors include hypoxemia, diabetes, smoking, oxidative stress, and low levels of systemic inflammation during both rest and exercise. Several studies have indicated that in chronic obstructive bronchitis, factors such as the patient's age, smoking, cholesterol levels, and systolic blood pressure (SBP) contribute to the development of cardiovascular disease. Inflammatory cytokines such as α-TNF and interleukins (IL), particularly IL-6 and IL-8, play a significant role in hemostasis disorders. The main cytokine responsible for activating coagulation processes is IL-6, while α-TNF also influences coagulation, but IL-6 has an indirect effect. IL-8 is also involved in the regulation of blood clotting by influencing the secretion of procoagulant and anticoagulant factors, as well as plasminogen inhibitors. [Keer R., Stirling D., Ludlam C.A. Interleukin6 and haemostasis // Br. J. Haematol. 2001. Vol. 115, No. 1. P. 3-12; Levi M, Ten Cate H. Disseminated intravascular coagulation // N Engl J Med. 1999 Aug 19;341(8):586-92.].

Materials and Methods: The research was carried out in the pulmonology department of the Samarkand Regional Multidisciplinary Medical Center. In line with the study's objective, patient histories were reviewed, focusing on the patients' age and disease history. The examination was conducted in two comparative groups. According to the results of the study, the first main group consisted of 60 patients with stage II-III SOOC, including 14 women (23.3%) and 46 men (76.7%). The second control group included 30 healthy individuals, with 15 men (50%) and 15 women (50%). The first (primary) group included 32 patients under the age of 50, consisting of 10 women (31.25%) and 22 men (68.75%). Additionally, the group included 28 patients aged 50 and older, with 4 women (14.3%) and 24 men (85.7%). The average age of patients in group 1 under 50 years of age was 36.8 ± 0.7 years, ranging from 21 to 50 years. In contrast, the average age of patients over 50 years of age was 61.0 ± 0.6 years, with ages ranging from 51 to 76 years.

Table 1.

Clinical features of patients in the main group

Parameter		**Middle age**	**Minimal age**	**Maximal age**
Age		48,9	21	76
		Total Number of Patients		%
Gender Distribution	Male	46		76,7 %
	Famale	14		23,3 %
Body Mass Index (BMI)		27,8 (25,2; 29,7)		
COPD Functional Class	Stage I	12		20,%
	Stage II	29		48,3 %
	Stage III	19		31,6 %
Duration of smoking		32 [28; 36]		
Emphysema		22		37,27 %
Pulmonary hypertension (EchoCG)	Stage I	13		34,54
	Stage II	4		8,18
	Stage III	1		2,72

It looks like you're referring to a table that provides detailed information about the distribution of patients in the main group. Here's a sample of how the table might be structured based on the details you've given [10, 11,13]. Based on the additional details you've provided, here's how you could structure the table, this table now includes the details about the smoking history, the distribution of patients by gender, and the functional class of SOOC. Feel free to adjust the BMI values if needed or add any more specific details.

Result and Discussion. To assess cardiac activity and determine hemodynamic status in all patients at various stages of the study, echocardiography was employed. A comparative analysis of cardiac activity and hemodynamic parameters between the main and control groups revealed certain changes in cardiac function. In both patient groups, enlargement of the left ventricle and left ventricular spaces, as well as thickening of the posterior wall of the left ventricle and interventricular septum, were observed. Additionally, changes in blood pressure in the left ventricle and pulmonary artery of Sue were noted. These changes were associated with the effects of violence against ChHF. The results of EXO-CG complement previously conducted laboratory and functional examination methods, providing a distribution of patients according to the functional class of chronic heart failure, as well as the severity associated with the presence of pulmonary hypertension. Table 2 presents the main echocardiographic parameters of the patient group.

Table 2.

Comparative Analysis of Hemodynamic Parameters

Parameter	Main group (60)	P
Left Ventricular End-Diastolic Volume (LVEDV)	5,7 [5,4-5,9]	< 0,05
Left Ventricular End-Systolic Volume (LVESV)	4,3 [3,6-5,25]	< 0,05
Ejection Fraction (EF)	45,25 [38,5-52,0]	< 0,05
During diastole PWLV	1,1 [1,0-1,2]	< 0,05
LA	4,5 [4,0-5,0]	< 0,05
RV	3,1 [2,9-3,3]	< 0,05
RA	4,4 [4,1-4,7]	< 0,05
Pulmonary Artery Pressure (PAP)	40,9 [39,6-46,1]	< 0,05

The data indicate that the left ventricular heart rate was significantly higher in patients in group A. These results may be attributed to the fact that when chronic obstructive pulmonary disease (COPD) is complicated by chronic heart failure, the two conditions exacerbate each other, leading to a worsening of the overall clinical status. After prolonged treatment, all patients underwent the same tests during a re-examination. Throughout the long-term therapy, patients were prescribed inhaled corticosteroids (fluticasone), two long-acting beta-agonists, antiplatelet agents, and beta-blockers, as part of the standard treatment regimen for Suqua Sue. The results of the 6-minute walk test and Clinical case assessment dynamics are presented in Table 3.

Table 3.

The 6-minute walk test and CCA dynamics

Main group (60)			
the 6-minute walk test			
Parameter	(n=60)	ChhF>40% (n=41)	Chhf<40% (n=19)
At the initial stage of inspections	306,0 [240,0-390,0]	378,0 [280,0-400,0]	220,0 [200,0-220,0]
After 6 month	350,0 [250,0-420,0]	400,0 [320,0-420,0]	245,0 [240,0-250,0]
P	$_{1\text{-}2}$ <0,05	$_{1\text{-}2}$ <0,05	$_{1\text{-}2}$ <0,05
CCA			
Parameter	(n=50)	ChhF >40% (n=28)	ChhF <40% (n=22)
At the initial stage of inspections	6 [6; 8]	7 [6; 8]	9 [8; 8]
After 6 month	6 [5; 7]	5 [4; 6]	7,5 [7; 8] *
P	$_{3\text{-}4}$ <0,05	$_{3\text{-}4}$ <0,05	$_{3\text{-}4}$ <0,05

After six months of treatment, positive dynamics in the 6-minute walk test were observed. The group also recorded a statistically significant ($p<0.05$) increase in the distance covered. The results are presented in **Figure 1.**

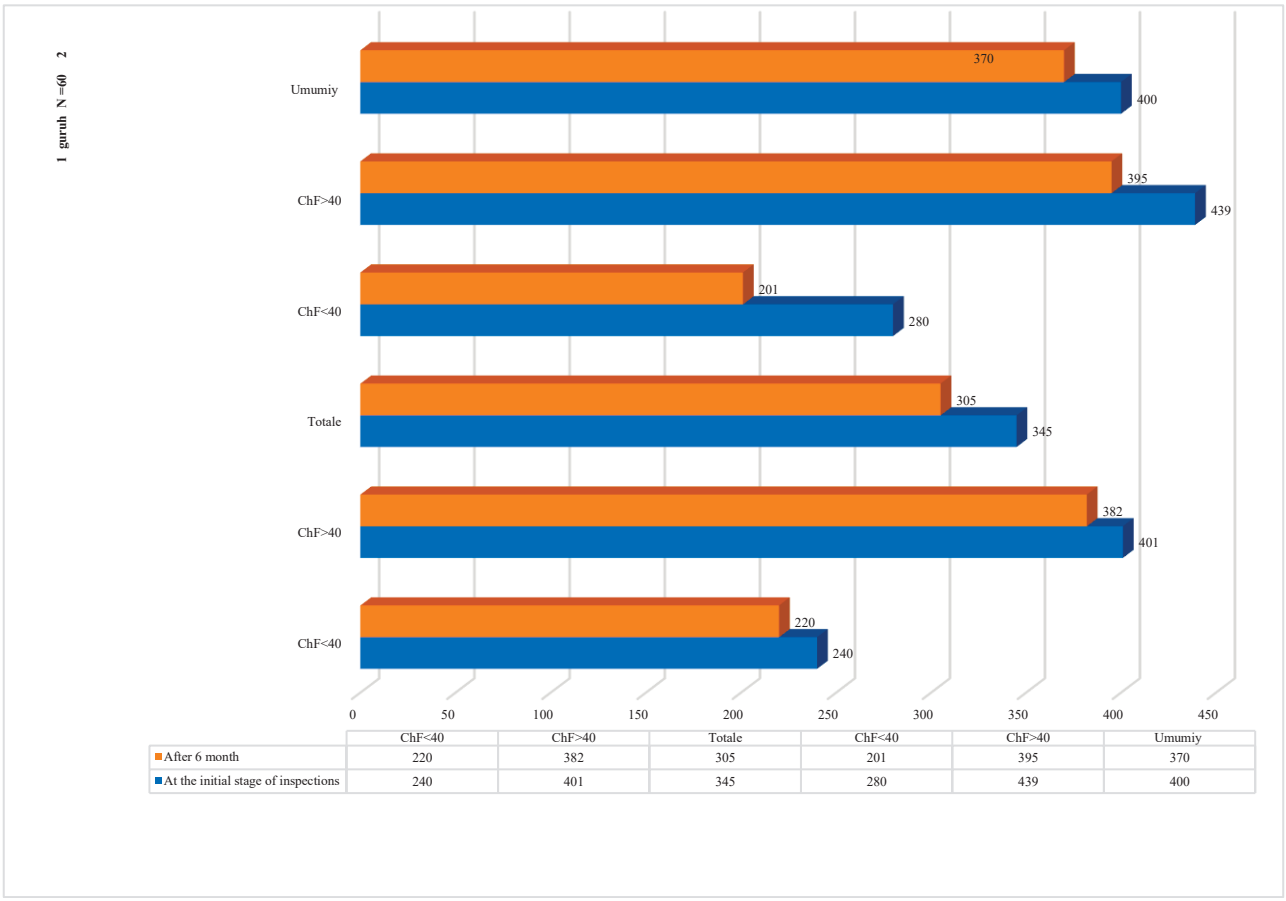


Figure 1. **The 6-minute walk test dynamics**

The CCA score showed similar results, with a noticeable improvement in the group. Specifically, there was a decrease in the number of points scored by the patients. In **Group 1,** the average CCA score at 6 months was 6, indicating a reduction in symptoms. On average, the group experienced a decrease in the number of points scored as a result of treatment. The reduction in the number of points for serious clinical manifestations did not lead to a decrease in the ChHF functional class in severe patients. However, this positive dynamic allowed for a reduction in the functional class of the disease in some patients, particularly those with mild and moderate severity. **Table 4** presents the changes in cardiohemodynamic parameters during the remission period following therapy.

Table 4.

The changes in cardiohemodynamic parameters during the remission period following therapy

Parameter	Main group (60)		P
	At the initial stage of inspections	After 6 month	
Left Ventricular End-Diastolic Volume (LVEDV)	5,7 [5,4-5,9]	5,6 [5,2-5,9]	$P_{1-2}>0,05$
Left Ventricular End-Systolic Volume (LVESV)	4,3 [3,6-5,25]	4,0 [3,8; 4,4]	$P_{1-2}>0,05$
Ejection Fraction (EF)	45,25 [38,5-52,0]	48,0 [41,5- 54,0]	$P_{1-2}>0,05$
During diastole PWLV	1,1 [1,0-1,2]	1,3 [1,1-1,3]	$P_{1-2}>0,05$
LA	4,5 [4,0-5,0]	4,5 [4,1-5,0]	$P_{1-2}>0,05$
RV	3,1 [2,9-3,3]	3,1 [2,8-3,5]	$P_{1-2}>0,05$
RA	4,4 [4,1-4,7]	4,5 [4,0-4,9]	$P_{1-2}>0,05$
Pulmonary Artery Pressure (PAP)	40,9 [39,6-46,1]	31,0 [25,0-45,0]	$P_{1-2}>0,05$

The patients showed significant improvements in certain indicators, including a decrease in pulmonary artery pressure, accompanied by a reduction in the development of irreversible changes in the "cardiopulmonary" complex. This had a positive effect on the respiratory system. Of particular interest is the assessment of the dynamics of the main echocardiographic indicators in patients from this group, divided into small subgroups with ChHF < 40% and ChHF > 40% (as shown in Table 4).

Conclusions. Thus, the results of laboratory and instrumental studies, along with the clinical dynamics after treatment, showed that patients experienced increased resistance to physical exertion, improved quality of life, and a reduction in disease relapses. As a result, an improvement in the prognosis for the major diseases was observed. It is associated with the degree of severity and their natural manifestation. The results of EXO-CG complement previously conducted laboratory and functional examination methods, providing insight into the distribution of patients according to the ChHF EF, as well as the severity level related to the presence of pulmonary hypertension. Positive dynamics were observed, including a decrease in the magnitude of the LVgap, an increase in ChHF by an average of 4%, a reduction in the LA gap, and a decrease in blood pressure in AP. The reduction of the LV cavity and the increase in Syue not only have a positive clinical effect on patients but also enhance their level of resistance to physical exertion. These improvements help reduce the manifestation of ChHF, maintain the reserve capabilities of the heart muscle, and decrease the development of the "lung-heart" syndrome. Additionally, these changes are accompanied by a reduction in pressure, further contributing to the overall health improvements.

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