

Assessment Of The Degree Of Endothelial Dysfunction In Patients With Chronic Obstructive Pulmonary Disease Complicated By Chronic Heart Failure

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ABSTRACT: When chronic obstructive pulmonary disease (COPD) and chronic heart failure (CHF) coexist, not only pulmonary hypertension and right ventricular failure, but also left ventricular failure, can occur. An rise in sodium uretic peptide (NT-proBNP) levels may accompany this.

Key words: COPD, CHF, sodium uretic peptide, echocardiography, diastolic dysfunction, right ventricle.

1. INTRODUCTION

COPD is the fourth biggest cause of death in the United States. COPD is expected to become the third biggest cause of mortality worldwide by 2030, according to WHO projections. Every year, over 3 million people die from COPD. The main causes of COPD are smoking and obesity, which are also major risk factors for heart disease and hypertension. These disorders' pathogenetic pathways have been extensively researched. If a patient's lungs have a disease, it suggests the cardiovascular system has a pathology as well. Chronic lung disorders, different arrhythmias, chronic heart failure, and the emergence of pulmonary fibrosis can all be associated with cardiovascular pathology.

The main cause of death in patients with COPD is not as a result of respiratory failure, but as a result of changes in the cardiovascular system. Cardiovascular diseases occur in 50% of patients with chronic obstructive pulmonary disease, and their presence increases the risk of chronic heart failure by 2-3 times [3].

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In most cases, patients with COPD develop pulmonary hypertension and right ventricular insufficiency (RV), followed by left ventricular insufficiency, even in the absence of cardiovascular pathology manifested in the clinic of the disease [8], this may occur as a result of an increase in the amount of uretic sodium peptide (NT).-proBNP). NT-proBNP is primarily considered a non-invasive marker of left ventricular heart failure. However, its concentration may increase with a violation of the function of the right ventricle and increased pressure of the pulmonary artery jamming (DLA). [5]. Studies have shown a relationship between NT-proBNP and indicators characterizing the ventilation function of the lungs (FEV1-the volume of forced exhalation per second, VEL-the vital capacity of the lungs, OEL-the total volume of the lungs). PaO2 (partial reduction of oxygen in arterial blood) [7], which is a promising marker for monitoring the course and development of NT-proBNP COPD.

The aim of the study was to determine the diagnostic value of NT-proBNP in patients with COPD complicated by chronic heart failure associated with obesity.

2. MATERIALS AND METHODS

The study was carried out in the department of pulmonology of the Samarkand regional multidisciplinary medical center. The most common combinations of cardiovascular disease and COPD are ischemic heart disease, arterial hypertension, heart failure, peripheral vascular disease and arrhythmias. 60 patients with chronic obstructive pulmonary disease (COPD) complicated by chronic heart failure (CHF) and obesity were examined, the control group consisted of 30 patients with COPD with a normal body mass index against the background of CHF.

The study involved patients with COPD belonging to the GOLD stages III and IV [6]. Exclusion criteria included clinically significant joint disease, previous breast surgery, type 2 diabetes mellitus, and a history of acute vascular disease.

All patients received inhaled glucocorticosteroids and drugs such as a b2-adrenergic agonist (budesonide / formoterol) and a long-acting M-anticholinergic drug (tiotropium bromide), which were administered according to treatment standards for the diagnosis and treatment of COPD [6].

In patients with chronic heart failure, standard therapy was used with ACE inhibitors (enalapril, perindopril, lisinopril), antiplatelet agents (thrombopols), anticoagulants (clexane), metabolites (thiotriozolin, corvitin), K-channel inhibitors (including ivabradine), nitrates. Patients were included in the study only with their consent.

Patients were divided into two groups: First - a group of patients with chronic obstructive pulmonary disease complicated by obesity and chronic heart failure (n = 60), Second - a control group with COPD against a background of 30 CHF with a normal body mass index.

The severity of heart failure was determined based on the recommendations of the New York Heart Association. The severity of patients with COPD complicated by CHF was determined using the GOLD scale.

All patients in the study underwent 12 standard ECG images. The presence of ischemia, rhythm and conduction disturbances, and focal changes were assessed. The study of FVD (function of external respiration) was carried out using a spirograph, three respiratory cycles in a row and computer processing of the results.

The state of central hemodynamics was assessed using echocardiography. The indices characterizing the systolic function of the left ventricle (LV) were calculated: end-diastolic



value (EDV), ejection fraction (EF) and myocardial mass index (MI) of LV. Assessment of the state of the right heart was carried out by measuring the end diastolic size of the RV, the thickness of the myocardium of the anterior wall of the RV in diastole. SPPA was assessed using continuous wave Doppler sonography.

Today, plasma natriuretic peptides BNP, NT-proBNP are fast and sensitive biomarkers for the diagnosis of CHF in patients with COPD. On the basis of standard methods of treatment and diagnostic methods for CHF, a threshold level of NT-proBNP was adopted to exclude CHF from 125 pg / ml, taking into account the gradual onset of symptoms.

3. RESEARCH RESULTS

There was no significant difference in age, sex and smoking history in the analyzed groups of patients (Table 1). The duration of the disease, depending on the history and year, was significantly higher in patients with COPD (group 1) complicated by chronic heart failure. In patients of group 1, FEV1 values were initially low on spirography, while in patients in group 2, these values were low (Table 1). All examined patients complained of shortness of breath, which is more common in patients with CHF and COPD complicated by obesity than in patients with chronic heart failure.

Table 1.

| Index | Group 1 (n=60) | Group 2 (n=30) |
|----------------------------------|----------------|-------------------|
| Age, years | 65,55±6,19 | 67,8±6,54 |
| Smoking experience, pack / years | 40,45±10,42 | 42,85±9,54 |
| Duration of the disease | 9,8±2,14 | 8,8±5,09 |
| Number of exacerbations per year | 4,15±0,15 | 3±1,37 |
| FEV1,% | 28,24±5,56 | 29,16±5,5 |
| FEV1 / FVC,% | <0,7 | <0,7 |

Note: p <0.05 when compared with group 2, FEV₁ - forced expiratory volume in 1 second, FVC - forced vital capacity of the lungs.

It should be noted that, according to echocardiography, LVEF, MI, and LVPD did not differ in the groups and did not exceed the norm (Table 2). In contrast to the left one, anomalies of the right chambers of the heart were more often observed in patients of the 1st group. Comparative analysis of indicators of intracardiac hemodynamics in the studied groups revealed certain structural changes in the heart. In both groups of patients, there was an



increase in the LA cavity, an increase in the thickness of the LV, LV and IVS. In addition, changes were noted in design parameters such as LVEF and VA pressure. These changes are associated with the course of CHF and COPD and are their natural manifestation.

The results of echocardiography complement the previously performed methods of laboratory and functional examinations and describe the prevalence of FC CHF in patients, as well as their severity in terms of the presence of pulmonary hypertension. Table 2 shows the main echocardiographic parameters of the examined patients of groups 1-2 at the stage of joining the study.

Table 2.

| Index | Group 1 | Group 2 | р |
|------------------|--------------|--------------|---------|
| | (n=60) | (n=30) | • |
| CRD LV, cm | 5,8 | 5,5 | < 0,05* |
| | [5,3; 6,6] | [5,1; 6,0] | |
| CSR LV, cm | 4,4 | 4,0 | < 0,05* |
| | [3,7; 5,4] | [3,7; 4,4] | |
| LVEF,% | 45,5 | 49,5 | > 0,05 |
| | [37,5; 53,0] | [42,0; 51,0] | |
| TZS LV diast, cm | 1,1 | 1,2 | > 0,05 |
| | [1,0; 1,2] | [1,1; 1,3] | |
| TMZhP LV, cm | 1,1 | 1,2 | > 0,05 |
| | [1,0; 1,2] | [1,1; 1,3] | |
| LP, cm | 4,6 | 4,6 | > 0,05 |
| | [4,2; 5,5] | [4,0; 5,0] | |
| RV, cm | 3,2 | 3,0 | > 0,05 |
| | [2,9; 3,7] | [2,9; 3,5] | |
| PP, cm | 4,5 | 4,4 | > 0,05 |
| | [4,2; 5,2] | [4,0; 4,8] | |
| Air pressure | 45,0 | 40,0 | > 0,05 |
| | [38,5; 46,0] | [36,0; 47,0] | |

Note: * - reliability of differences, significant when p<0.05

The presented data show that reliably high rates of CVD and LVEDD were observed in patients of group 1 compared with patients in group 2. This is due to the large number of patients with severe CHF, characterized by signs of recovery. Otherwise, there were no statistically significant differences in the patient samples.

To determine the features of the course of CHF in the treatment of patients with COPD complicated by chronic heart failure, at the initial stage of the study, a correlation analysis was carried out on all patient samples.

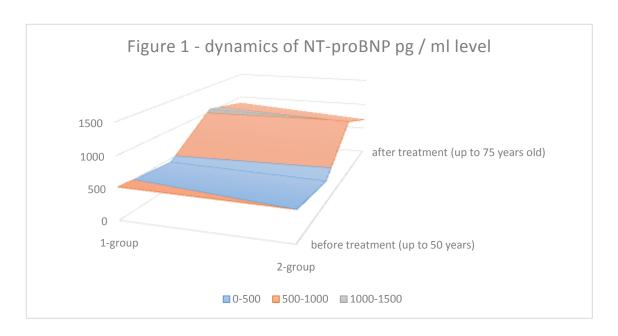
When examining the timing of admission of patients in groups 1 and 2 with chronic obstructive pulmonary disease complicated by chronic heart failure, the following changes in NT-proBNP pg / ml were observed.

NT-proBNP was 523.5 ± 89.5 pg / ml in group 1 patients with an average cardiac EF of 40–49% upon admission to the hospital. NT-proBNP decreased to 435.5 ± 92.5 pg / ml after treatment. During hospitalization, the mean NT-proBNP value of 1050 ± 92.8 pg / ml in



patients with EF of the group less than 40% of the heart rate under the age of 75 received treatment after NT-proBNP treatment $859.5 \pm 94.5 \text{ pg} / \text{ml}$.

NT-proBNP was 499.5 \pm 91.5 pg / ml in group 2 patients with an EF of 45% of heart rate under the age of 50 at admission, NT-proBNP - 376.5 \pm 92.5 pg / ml after treatment. On admission, the mean NT-proBNP was 993 \pm 93.6 pg / ml in group 1 of patients with ejection fraction less than 45% of cardiac ejection fraction under the age of 75 years after treatment with NT-proBNP 678.5 \pm 93.6 pg / ml.



The results obtained once again confirm the participation of the right ventricle in the increase in the production of uretic sodium peptide. NT-proBNP is produced primarily in the ventricular myocardium in response to increases in end-diastolic pressure and volume. To a lesser extent, it is synthesized in the atrial myocardium. A number of authors associate an increase in NT-proBNP concentration in patients with COPD with a hemodynamic response to the development of pulmonary hypertension as a result of pulmonary vasospasm [4]. Alternative reasons, promotes an increase in the concentration of NT-proBNP in COPD,

Alternative reasons, promotes an increase in the concentration of NT-proBNP in COPD, mechanical compression from the outside of the LV wall due to hyperinflation and increased intrathoracic pressure is considered [1]. The content of NT-proBNP is significantly increased, which may be associated with more transient hemodynamic disorders, including a temporary increase in systolic pressure in the pulmonary artery, the addition or exacerbation of right ventricular failure [2].

4. DISCUSSION

Many researchers are interested in respiratory pathology in individuals with COPD who also have chronic heart failure, because the sodium-uretic peptide can be used to measure the muscle activity of the left ventricle and so detect chronic heart failure early.

According to the data presented, patients in both groups had a similar tolerance to physical activity at the time of enrollment in the study. Patients with severe CHF outperformed both

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groups, according to the findings. Although the data showed that the average distance was longer in group 2, there were no significant statistical differences between the groups..

Comparative analysis of indicators of intracardiac hemodynamics in the studied groups revealed certain structural changes in the heart. In both groups of patients, there was an increase in the LA cavity, an increase in the thickness of the LV, LV and IVS. In addition, changes were noted in design parameters such as LVEF and VA pressure. These changes are associated with the course of CHF and COPD and are their natural manifestation.

5. CONCLUSION

There is an increase in sodium-uretic peptide concentration with chronic heart failure in COPD patients, which can be used as a possible dynamic biomarker of increased pulmonary hypertension as well as the development of chronic gait failure.

Long-term treatment of patients with chronic obstructive pulmonary disease complicated by chronic heart failure not only lowers pulmonary artery systolic pressure and improves the condition of the right heart chambers, but also lowers sodium uretic peptide concentration, increasing therapy effectiveness. For monitoring, I prefer NT-proBNP.

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