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Cardiohemodynamic Disorders and The Efficiency of Combination Therapy for COPD Associated with Pulmonary and Arterial Hypertension

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Abstract: COPD is often associated with cardiovascular diseases, including pulmonary and arterial hypertension, which contributes to a worse prognosis and the progression of cardiorespiratory impairment. Pulmonary hypertension, associated with lung disease and hypoxia, is one of the most common forms of secondary pulmonary hypertension following left heart disease.

Keywords: Chronic obstructive pulmonary disease, arterial hypertension, pulmonary hypertension, heart rate variability indicators.

Introduction: Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disease and a leading cause of death worldwide, ranking among the top causes of death according to global epidemiological studies and GOLD and WHO reports [1,3,9].

Chronic hypoxia, which causes hypoxic vasoconstriction and subsequent vascular remodeling, is considered the primary mechanism for increased pulmonary artery pressure in COPD. Systemic hypoxia in COPD can contribute to increased systemic blood pressure through impaired endothelium-dependent vasodilation and activation of neurohumoral systems (RAAS and the sympathetic nervous system). Common pathogenetic links in the combination of COPD and arterial hypertension include systemic inflammation, oxidative stress, and endothelial dysfunction, which contribute to the progression of cardiovascular disorders and increased stress on the right side of the heart in secondary PH [2,5].

Treatment of COPD complicated by pulmonary and arterial hypertension is challenging due to its multifactorial pathogenesis, including hypoxia, inflammation, oxidative stress, and endothelial dysfunction. Angiotensin-converting enzyme inhibitors can have a beneficial effect on vascular tone and pulmonary artery pressure, while drugs with antihypoxic and antioxidant effects, including L-carnitine, help improve exercise tolerance and cardiorespiratory hemodynamic parameters. Combined pathogen-directed therapy can improve the clinical condition of patients with COPD complicated by pulmonary and arterial hypertension.

METHODS

The study included 66 patients with COPD with pulmonary and arterial hypertension, treated in the therapeutic department of the Tashkent City Clinical Hospital, confirmed by clinical, laboratory and biochemical studies, chest X-ray, spirometry, ECG, echocardiography examinations. The control group included 20 practically healthy volunteers. The diagnosis and stage of COPD were determined in accordance with the GOLD recommendations (2024-2025), the presence of pulmonary hypertension - according to the ESC/ERS recommendations (2022). Hypertension was established according to the ESH/ESC criteria from 2024. The group was divided into subgroups depending on the treatment. The first subgroup (n=35) received ACE inhibitors with the basic treatment, the second subgroup (n=31) with the basic treatment, ACE inhibitors and additionally L-carnitine. The initial dose of perindopril was 1-2 mg/day taken orally once, with subsequent dose adjustments to 2-4 mg/day with subsequent monitoring every 2 weeks. L-carnitine was prescribed at 1.0 g/day – intravenous drip for the first 3 days, then 0.5 mg/day intravenous jet stream for 10 days.

RESULTS

With increasing obstruction severity in chronic obstructive bronchitis with pulmonary hypertension and hypertension, an increase in the CDR (2.98±0.30, 3.41±0.18, 3.44±0.39) and CSR (2.47±0.12, 2.59±0.31, 2.86±0.41) was observed in the groups of COPD patients with pulmonary hypertension and hypertension, although their values were at the upper limit of normal.

With an increase in RV EDV depending on the severity of obstruction (45.90±3.63 ml, 47.01±4.12 ml, 49.11±4.93 ml) and RV ESV (25.11±1.63 ml, 28.91±1.58 ml, 30.42±2.98 ml) compared to the control group (39.71±5.08 ml, 23.01±3.02 ml), a decrease in RV SV (22.01±0.4 ml, 17.34±2.13 ml, 14.01±2.46 ml) and RV MO (1.43±0.21, 1.32±0.31, 1.15±0.12) was noted.

The right ventricular ejection fraction was lower (49.01±1.72%, 43.14±2.13%, and 38.34±3.74%, depending on the degree of obstruction) compared to the control group (53.64±5.59%).

A hypertrophied and dilated right ventricle contributed to a drop in left ventricular pressure and ejection. Increased compression of the left ventricle by the right ventricle impaired left ventricular filling.

A study of LV cardiac hemodynamic parameters in patients with COPD, PH, and hypertension revealed that pulmonary circulation volume overload resulted in an increase in LA dimensions (3.63±0.41 cm, 3.72±0.31 cm, 4.01±0.15 cm) as bronchial obstruction increased compared to the control group (3.59±0.17 cm). An increase in the posterior wall of the left ventricle (0.93±0.08 cm, 0.96±0.11 cm, 1.21±0.09 cm) and interventricular septum (1.10±0.05 cm, 1.16±0.09 cm, 1.15±0.06 cm) indicates more pronounced cardiac muscle hypertrophy with an increase in pulmonary hypertension pressure and bronchial obstruction, and in the presence of hypertension. Volume overload of the left heart was accompanied by an increase in the EDD (4.78±0.48 cm, 5.11±0.73 ml, 5.32±0.41 cm) and ESR (3.51±0.16 ml, 3.37±0.37 ml, 3.81±0.32 cm) among COPD patients with PH and hypertension compared to the control group (5.01±0.64 cm and 3.58±0.16 cm, respectively), but the parameters did not exceed generally accepted norms.

The end-diastolic volume (EDV) increased (121.4±10.12 ml, 128.4±10.98 ml, and 139.1±9.21 ml) and was accompanied by an increase in ESV (59.01±5.91 ml, 62.01±7.81 ml, and 66.01±0.72 ml) compared with the control group (126.9±9.24 ml and 57.61±5.37 ml). Compensatory increases in both LVSV and LVMI, as well as cardiac index, were observed.

These data indicate that myocardial hypertrophy and compensatory cardiac activation develop in response to increased volume and pressure load, leading to more pronounced LV myocardial hypertrophy.

Treatment of patients with COPD, pulmonary hypertension, and hypertension in the study groups resulted in a significant reduction in pulmonary artery pressure (PAP) with perindopril. With perindopril and perindopril in combination with L-carnitine, MAP significantly decreased by 1.27 and 1.3 times, respectively, compared to baseline.

Analysis of data from patients with COPD, pulmonary hypertension, and hypertension treated with ACE inhibitors and L-carnitine showed a reduction in systolic and diastolic blood pressure. Target blood pressure levels (140/90 mmHg) were achieved in 23 (82.14%) patients. Systolic blood pressure decreased by 1.2 (131.9±2.1 mmHg) and 1.3 times (125.12±2.1 mmHg), diastolic pressure by 1.14 (81.4±1.5 mmHg) and 1.16 times (80.29±1.8 mmHg), respectively, in both groups. FVD indicators also improved in the compared groups, with FEV1 increasing by 6% (66.93%) and 11% (71.01%) in both groups. The indices of the functional state of the myocardium of patients with COPD, pulmonary hypertension and hypertension during pharmacotherapy with perindopril revealed a decrease in the size of the RV EDV by 5.6% and 8.5% (44.3±0.13 cm and 43.01±0.10 cm), a decrease in the value of the RV ESV by 14.3% and 19.4% in both groups (24.77±0.31 cm and 23.31±0.7 cm) with an increase in the RV SV by

13.61% and 22.83% (19.7±0.51 ml and 21.3±0.7 ml, respectively, in both groups) indicating an improvement in the function of the right ventricle.

Changes in the hemodynamic conditions of the right ventricle resulted in increases in parameters such as cardiac output (1.69±0.2 ml and 1.76±0.4 l/min, respectively, in both groups), and ejection fraction increased by 4% (47.01±0.21%) and 6% (49.5±0.31%), respectively. Significant changes were observed during pharmacotherapy with perindopril and L-carnitine.

A decrease in pulmonary hypertension was significantly more common in the 2nd group of COPD patients with pulmonary hypertension and hypertension, accompanied by a decrease in RV EDD (3.18±0.31) and RV ESR (3.03±0.4), indicating signs of reduced right ventricular overload (Figure 3). Perindopril and L-carnitine therapy also improved the size of the right atrium and right ventricle. Improvement in cardiac hemodynamics was significantly more frequently observed in the second group of patients receiving perindopril and L-carnitine, compared to patients receiving only perindopril.

Along with improved pulmonary hemodynamics, perindopril and L-carnitine therapy in COPD patients with pulmonary hypertension also altered systemic hemodynamic parameters. Treatment resulted in increases in end-diastolic volume (EDV), stroke volume (SV), left ventricular volume (LVM), and left ventricular ejection fraction (LVEF) among COPD patients with pulmonary hypertension.

CONCLUSION

The results of the analysis showed that among patients with COPD with pulmonary hypertension and hypertension, treatment with perindopril in combination with L-carnitine demonstrated clinical efficacy, as demonstrated by a significant reduction in vascular hypertension in the pulmonary and systemic circulations. Perindopril can be considered a first-line treatment for pulmonary and arterial hypertension in COPD patients with pulmonary and arterial hypertension, as well as for the correction of cardiac hemodynamic abnormalities.

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