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Myocardial Dystrophy In Chronic Obstructive Pulmonary Disease: Mechanisms Of Development, Early Markers, And Treatment Approaches

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Abstract
This article examines the relationship between chronic obstructive pulmonary disease (COPD) and myocardial dystrophy, focusing on pathophysiological mechanisms, diagnostic approaches, and therapeutic strategies. A total of 120 COPD patients were evaluated for myocardial dysfunction using echocardiography, cardiac biomarkers (NT-proBNP, troponin I, CK-MB), and inflammatory markers (IL-6, TNF-α, CRP). The results demonstrated that myocardial dystrophy is present in 68.3% of COPD patients, with severity correlating with disease stage. Hypoxia-driven oxidative stress and systemic inflammation were identified as the primary mechanisms. Combined bronchodilator therapy supplemented with cardioprotective agents (trimetazidine 35 mg twice daily) significantly improved both pulmonary and cardiac functional parameters.

Keywords: chronic obstructive pulmonary disease, myocardial dystrophy, cor pulmonale, hypoxia, oxidative stress, NT-proBNP, trimetazidine, cardioprotection.

Chronic obstructive pulmonary disease (COPD) is among the leading causes of morbidity and mortality worldwide, affecting approximately 384 million people globally and accounting for over 3 million deaths annually. According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD), COPD is projected to become the third leading cause of death by 2030. In Uzbekistan, COPD prevalence among adults over 40 years of age reaches 14.3%, with the majority of cases diagnosed at moderate to severe stages [1].

Cardiovascular complications represent the most common cause of death in COPD patients, responsible for approximately 30% of all COPD-related mortality. Among these, myocardial dystrophy — a non-inflammatory, metabolic injury to the myocardium driven by chronic hypoxia, systemic inflammation, oxidative stress, and electrolyte imbalance — is increasingly recognized as a significant comorbidity. Despite its clinical relevance, myocardial dystrophy in the context of COPD remains underdiagnosed, partly due to symptom overlap between pulmonary and cardiac dysfunction and a lack of standardized diagnostic criteria [2, 3].

In COPD, sustained arterial hypoxemia triggers pulmonary vasoconstriction, leading to pulmonary hypertension and progressive right ventricular overload (cor pulmonale). Simultaneously, systemic inflammation — mediated by IL-6, TNF-α, and CRP — promotes myocardial fibrosis and impairs contractile protein function. Oxidative stress further disrupts mitochondrial energy metabolism, reducing myocardial ATP synthesis and culminating in cardiomyocyte dysfunction. These mechanisms together form a cardiopulmonary vicious cycle that accelerates both organ systems' deterioration [4].

Aim of the study: To assess the prevalence and severity of myocardial dystrophy in COPD patients across disease stages, to identify early diagnostic biomarkers, and to evaluate the efficacy of cardioprotective therapy added to standard bronchodilator treatment.

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Material and methods: The study enrolled 120 patients with confirmed COPD (GOLD stages II–IV) treated at the Republican Specialized Scientific-Practical Medical Center of Pulmonology. The control group consisted of 25 age- and sex-matched healthy individuals. Mean age was 61.4±6.8 years; 78 patients were male and 42 female. All participants underwent spirometry (FEV_1, FVC, FEV_1/FVC), 12-lead ECG, echocardiography (left and right ventricular ejection fraction, TAPSE, diastolic function), and measurement of serum NT-proBNP, troponin I, CK-MB, IL-6, TNF-α, and CRP. Arterial blood gases (PaO_2, $PaCO_2$, SaO_2) and the 6-minute walk test were also performed.

Patients were randomized into two treatment groups after baseline assessment: Group 1 (n=60) — standard COPD therapy (long-acting bronchodilators + inhaled corticosteroids); Group 2 (n=60) — standard therapy + trimetazidine 35 mg twice daily for 6 months. Outcomes were assessed at 1, 3, and 6 months. Statistical analysis was performed using SPSS 26.0; $p<0.05$ was considered significant.

Results. Myocardial dystrophy was identified in 82 of 120 COPD patients (68.3%). Its prevalence increased proportionally with GOLD stage: 41.2% in GOLD II, 72.5% in GOLD III, and 94.1% in GOLD IV. Echocardiography revealed right ventricular dilatation in 61.7% of patients and reduced TAPSE (<17 mm) in 54.2%, indicating significant right ventricular dysfunction. Left ventricular diastolic dysfunction (E/A ratio <1) was present in 47.5% of the total cohort.

Figure 1. Prevalence of myocardial dystrophy and right ventricular dysfunction by COPD GOLD stage.

Serum NT-proBNP was significantly elevated in patients with myocardial dystrophy compared to those without (487.3±62.1 pg/ml vs 142.6±28.4 pg/ml; $p<0.001$), and correlated inversely with FEV_1 ($r=-0.74$, $p<0.001$) and SaO_2 ($r=-0.81$, $p<0.001$). Troponin I was elevated above the 99th percentile in 23.3% of patients, predominantly those at GOLD III–IV stages. IL-6 and TNF-α levels were markedly higher in the myocardial dystrophy group (IL-6: 18.7±3.2 vs 7.4±1.9 pg/ml; TNF-α: 24.1±4.6 vs 10.2±2.3 pg/ml; both $p<0.001$), confirming the central role of systemic inflammation in myocardial injury.

Figure 2. NT-proBNP and inflammatory marker dynamics in COPD patients with and without myocardial dystrophy.

At 6-month follow-up, Group 2 (trimetazidine + standard therapy) demonstrated significantly better outcomes compared to Group 1. Left ventricular ejection fraction improved from 48.2±3.1% to 54.7±2.8% in Group 2 versus from 47.9±3.4% to 49.6±3.2% in Group 1 ($p<0.001$). NT-proBNP decreased by 38.4% in Group 2 versus 14.7% in Group 1 ($p<0.001$). The 6-minute walk distance increased by 87±12 m in Group 2 compared to 34±9 m in Group 1 ($p<0.001$). IL-6 and TNF-α levels normalized significantly faster in the trimetazidine group ($p<0.05$) [5].

The high prevalence of myocardial dystrophy in COPD patients observed in this study is consistent with data from international registries, yet the degree of correlation between GOLD stage and cardiac dysfunction severity underscores the need for routine cardiac screening in all COPD patients. NT-proBNP emerged as the most sensitive early biomarker, with its inverse correlation to both FEV_1 and oxygen saturation providing a practical tool for risk stratification without requiring advanced imaging. Trimetazidine, a partial fatty acid oxidation inhibitor that shifts myocardial energy metabolism toward more oxygen-efficient glucose utilization, proved effective in reversing myocardial dysfunction in the comorbid setting, supporting its inclusion in treatment protocols for COPD patients with documented myocardial dystrophy [6, 7].

Conclusion:

- Myocardial dystrophy is a frequent and clinically significant comorbidity in COPD, detected in 68.3% of patients, with prevalence rising to 94.1% at GOLD stage IV.
- Chronic hypoxia, systemic inflammation (IL-6, TNF-α), and oxidative stress are the principal pathophysiological drivers of myocardial injury in COPD.

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- NT-proBNP is the most reliable early biomarker for myocardial dysfunction in COPD; it correlates inversely with FEV_1 ($r=-0.74$) and SaO_2 ($r=-0.81$) and should be incorporated into routine COPD assessment.
- Addition of trimetazidine (35 mg twice daily) to standard bronchodilator therapy significantly improved left ventricular ejection fraction (+6.5%), reduced NT-proBNP by 38.4%, and increased 6-minute walk distance by 87 m ($p<0.001$).
- Routine echocardiographic screening and cardiac biomarker assessment are recommended for all COPD patients at GOLD stage II and above to enable timely detection and treatment of myocardial dystrophy.

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