CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND CHRONIC HEART FAILURE: CURRENT VIEWS ON PATHOGENESIS, CLINICAL FEATURES, DIAGNOSIS AND TREATMENT

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Abstract

Chronic obstructive pulmonary disease (COPD) and chronic heart failure (CHF) are among the most common chronic diseases worldwide and frequently coexist in clinical practice. Shared risk factors, including smoking, aging, systemic inflammation, oxidative stress, and endothelial dysfunction, contribute to the development and progression of both conditions. The coexistence of COPD and CHF significantly worsens prognosis, increases hospitalization rates, reduces exercise tolerance, and decreases quality of life. Diagnostic difficulties arise due to overlapping clinical manifestations, particularly dyspnea and exercise intolerance.

Keywords: chronic obstructive pulmonary disease, COPD, chronic heart failure, comorbidity, systemic inflammation, cardiovascular diseases, treatment.

Introduction. Chronic obstructive pulmonary disease (COPD) is one of the leading causes of morbidity and mortality worldwide [1]. According to the latest GOLD recommendations, COPD is associated with significant healthcare burden and frequently coexists with cardiovascular diseases, particularly chronic heart failure (CHF) [1].

Chronic heart failure is a complex clinical syndrome resulting from structural or functional abnormalities of the heart that impair ventricular filling

or ejection of blood [2]. Both COPD and CHF are characterized by high mortality, frequent hospital admissions, and progressive deterioration of functional status [3]. Their coexistence creates substantial diagnostic and therapeutic challenges.

COPD and CHF frequently coexist because they share common risk factors, including advanced age, smoking, obesity, hypertension, diabetes mellitus, and systemic inflammation. The prevalence of heart failure among patients with COPD ranges from approximately 20% to 30%, while some studies report even higher rates depending on the population examined [3].

Recent global analyses indicate that the burden of COPD-HF comorbidity continues to increase and is expected to rise further by 2050 due to population aging and the growing prevalence of chronic noncommunicable diseases [6].

Common Pathophysiological Mechanisms. Systemic inflammation is considered one of the principal mechanisms linking COPD and CHF [2]. Chronic pulmonary inflammation in COPD extends beyond the respiratory system and contributes to vascular dysfunction, myocardial remodeling, and cardiac impairment. Elevated levels of inflammatory mediators such as interleukin-6, tumor necrosis factor-α, and C-reactive protein have been observed in both diseases [2,4].

Oxidative stress plays an important role in the progression of both COPD and CHF. Cigarette smoke exposure, chronic hypoxia, and inflammatory processes increase the production of reactive oxygen species, resulting in endothelial dysfunction and tissue damage. This process contributes to both airflow limitation and cardiac remodeling [4].

Hypoxia and Pulmonary Hypertension. Persistent hypoxemia in COPD promotes pulmonary vasoconstriction and pulmonary hypertension. Increased

pulmonary vascular resistance places an additional burden on the right ventricle, potentially leading to right-sided heart failure[5,7].

Clinical features. The coexistence of COPD and CHF is characterized by overlapping symptoms that often complicate diagnosis.

The most common manifestations include: progressive dyspnea, exercise intolerance, chronic fatigue, reduced physical activity, peripheral edema, chronic cough, sleep disturbances [3]. Dyspnea represents the predominant symptom in both diseases and often makes differentiation difficult during routine clinical assessment [7]. Patients with combined COPD and CHF generally experience more severe symptoms, increased frequency of exacerbations, and poorer health-related quality of life compared with patients suffering from either condition alone [3,5].

Diagnostic Challenges. Accurate diagnosis remains difficult because COPD and CHF share many clinical features.

Spirometry remains the gold standard for COPD diagnosis and demonstrates persistent airflow limitation with a post-bronchodilator FEV1/FVC ratio below [1]

Echocardiography is essential for evaluating cardiac structure and function and for differentiating heart failure with reduced ejection fraction (HFrEF) from heart failure with preserved ejection fraction (HFpEF) [7].

Biomarkers. Measurement of B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) is useful for identifying heart failure in patients presenting with respiratory symptoms [7]. Elevated levels strongly suggest cardiac involvement.

Imaging Studies. Chest radiography and computed tomography help identify emphysema, pulmonary congestion, and other structural abnormalities [5,9].

Combined interpretation of pulmonary and cardiac findings improves diagnostic accuracy.

Modern Treatment Approaches. COPD Management

Current GOLD recommendations emphasize individualized therapy based on symptoms and exacerbation risk. Long-acting bronchodilators remain the cornerstone of treatment. Smoking cessation, pulmonary rehabilitation, and vaccination are essential components of comprehensive management [2].

Heart Failure Management. Patients with CHF should receive guideline-directed medical therapy, including: angiotensin-converting enzyme inhibitors, angiotensin receptor-neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, sodium-glucose cotransporter-2 (SGLT2) inhibitors. Cardioselective β1-blockers have demonstrated safety and survival benefits in patients with concomitant COPD and heart failure [7,8,10].

Conclusion. COPD and chronic heart failure frequently coexist and share numerous pathogenetic mechanisms, including systemic inflammation, oxidative stress, endothelial dysfunction, and chronic hypoxia. Their combination significantly worsens clinical outcomes, increases healthcare utilization, and reduces quality of life. Early recognition, comprehensive diagnostic evaluation, and multidisciplinary treatment strategies are essential for improving prognosis and reducing disease burden.

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