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Opinion Article

Early Detection of Pulmonary Hypertension in Chronic Obstructive Pulmonary Disease (COPD)

James Marc*

Department of Medicine, University of Alabama at Birmingham, Birmingham, USA

DESCRIPTION

A frequent complication of Chronic Obstructive Pulmonary Disease (COPD) is mild-to-moderate pulmonary hypertension, this complication is linked to higher exacerbation risks and lower survival rates. Exercise, sleep, and pulmonary hypertension exacerbations typically make the condition worse. The main factor contributing to a rise in pulmonary artery pressure in people with COPD is pulmonary vascular remodeling, which is assumed to be the outcome of the interactions between severe emphysema's capillary loss, inflammation, and hypoxia.

A small percentage of COPD patients may exhibit "out-of-proportion" pulmonary hypertension, which is indicated by a mean pulmonary artery pressure >35–40 mmHg (normal is not more than 20 mmHg) and a relatively preserved lung function (with low to normal arterial carbon dioxide tension), which cannot be explained by the presence of prominent dyspnea and fatigue. According to estimates, the prevalence of idiopathic pulmonary arterial hypertension and out-of-proportion pulmonary hypertension are quite similar in people with COPD.

Patients with Chronic Obstructive Pulmonary Disorders (COPD) typically have underdiagnosed and under-evaluated Pulmonary Hypertension (PH). It is crucial to have a unified approach for diagnosing and treating PH with the goal of improving the patient's quality of life and prognosis in terms of therapeutic considerations because PH is associated with a high rate of mortality from disease-related complications such cor pulmonale. Early therapy initiation and improved prognostic implications can result from early detection of this comorbidity in COPD patients. This study's objectives were to estimate the prevalence of PH in COPD and evaluate any statistically significant relationships between PH severity and COPD severity.

The 2003 WHO classification of PH places pulmonary hypertension in COPD under group 3, or PH associated with respiratory system problems and/or hypoxemia. In contrast to the criteria of primary pulmonary hypertension (mPAP >25 mm Hg), PH associated with lung disease is defined as resting mean PAP (mPAP) greater than 20 mm Hg. The prevalence of COPD is rising globally as a result of rising smoking rates and a decline in infectious disease-related mortality. The prevalence of COPD is further influenced in some areas by the widespread use of biomass fuels like wood, grass, or other organic materials.

In countries with poor access to healthcare, COPD death rates may be greater than in countries with better access to healthcare. Globally, COPD was the third highest cause of death in 2019 with 3.23 million fatalities. When genetically predisposed individuals are exposed through inhalation, their airways and alveoli might become inflamed, which can result in illness.

An increase in protease activity and a decrease in antiprotease activity are assumed to be the mediators of the process. The normal process of tissue repair involves the breakdown of elastin and connective tissue by lung proteases like neutrophil elastase, matrix metalloproteinases, and cathepsins.

Antiproteases such alpha-1 antitrypsin, airway epithelium-derived secretory leukoproteinase inhibitor, elafin, and matrix metalloproteinase tissue inhibitor typically operate as a check on their action. As part of the inflammatory process in COPD patients, active neutrophils and other inflammatory cells release proteases. When protease activity surpasses antiprotease activity, tissue damage and mucus hyper secretion occur.

Patients who continue to smoke and those who have a higher lifetime tobacco exposure typically experience rapid symptom progression. Morning headache is a symptom of more severe disease and denotes nocturnal hypoxemia or hypercapnia. Wheezing, a protracted expiratory phase of breathing, lung hyperinflation shown by diminished heart and lung sounds, and enlarged anteroposterior thorax diameter (barrel chest) are all indications of COPD. Advanced emphysema patients undergo weight loss and muscle wasting, which has been linked to inactivity, hypoxia, or the release of systemic inflammatory mediators such TNF-alpha.

The goal of treating chronic stable COPD is to enhance lung and physical function while preventing exacerbations. Short-acting beta-adrenergic medications should be used primarily to relieve symptoms quickly, while inhaled corticosteroids, long-acting beta-

Correspondence to: James Marc, Department of Medicine, University of Alabama at Birmingham, Birmingham, USA, E-mail: james232@marc.uab.edu

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adrenergic medications, and long-acting anticholinergic medications should be used to reduce exacerbations. Pulmonary hypertension has no known treatment option. To minimize symptoms, increase life expectancy, and stop the problem from growing worse, there are medicines available. Any health issues that might be causing the pulmonary hypertension may also be treated.