OPEN ACCESS Freely available online

Internal Medicine: Open Access

ISSN: 2165-8048

Commentary

Chronic Obstructive Pulmonary Disease and Lung Disease-Related Pulmonary Hypertension

Pierre Oliver*

Department of Acute Medicine, University Hospital Basel, Basel, Switzerland

DESCRIPTION

Mild-to-moderate pulmonary hypertension is a common consequence of Chronic Obstructive Pulmonary Disease (COPD), it is linked to an increased risk of exacerbation and a lower chance of survival. During exercise, sleep, and exacerbation, pulmonary hypertension normally worsens. A small percentage of COPD patients may have "out-of-proportion" pulmonary hypertension, defined as a mean pulmonary artery pressure greater than 35-40 mmHg (normal is less than 20 mmHg) and relatively preserved lung function (low to normal arterial carbon dioxide tension) that cannot explain prominent dyspnoea and fatigue. It is estimated that the prevalence of out-of-proportion pulmonary hypertension in COPD is quite close to the prevalence of idiopathic pulmonary arterial hypertension.

Cor pulmonale is a common complication of pulmonary hypertension induced by respiratory diseases. It is defined as right ventricular hypertrophy and dilatation. More research is needed to determine the role of cor pulmonale in impaired exercise capacity in COPD. Improved imaging techniques and biomarkers, such as B-type natriuretic peptide, as well as exercise testing regimens with gas exchange measures, should be included in this research. The effects of pulmonary arterial hypertension medicines should be studied in patients with chronic obstructive lung disease who have severe pulmonary hypertension. Meanwhile, supplementary oxygen and a number of therapies targeted at relieving airway congestion continue to be used to manage cor pulmonale in Chronic Obstructive Pulmonary Disease(COPD).

In the early stages of COPD, structural and functional alterations in the pulmonary circulation are visible. Endothelial dysfunction and alterations in the expression of endothelium-derived mediators that carry out vascular tone and cell development have been discovered in the pulmonary arteries of individuals with moderate illness. Some of these alterations occur in smokers with normal lung function as well. As a result, it has been proposed that the first event in the natural history of pulmonary hypertension in COPD could be a lesion of pulmonary endothelium caused by cigarette smoke components. In chronic obstructive pulmonary disease, the only treatment that decreases the progression of pulmonary hypertension is long-term oxygen administration. Nonetheless, with this treatment, pulmonary artery pressure rarely returns to normal levels, and pulmonary vascular anatomical abnormalities remain unaffected. Vasodilators are not indicated due to their low clinical efficacy and impairment of pulmonary gas exchange. Recognizing the significance of endothelial dysfunction in the physiopathology of pulmonary hypertension in chronic obstructive pulmonary disease offers up new possibilities for treatment. Exercise has been used to detect familial pulmonary hypertension risk. Exercise in COPD may cause significant increases in Ppa, particularly if there is pre-existing pulmonary hypertension during rest.

Long-term resting pulmonary hypertension is more common in patients with exercise-induced pulmonary hypertension. Ppa rises more than predicted by the PVR equation in exercising COPD patients, indicating pulmonary vasoconstriction. This unexpectedly substantial increase could be attributed to increased hypoxic pulmonary vasoconstriction caused by lower mixed venous partial pressure of oxygen, increased sympathetic nervous system tone, and decreased arterial pH caused by aggravated hypercapnia, lactic acidosis, or both. Changes in intrathoracic pressures could potentially be a factor. An increase in ventilation caused by exercise may aggravate dynamic hyperinflation and so raise alveolar pressure at expiration. In the context of blocked airways, increased ventilation is associated with significantly negative inspiratory pleural pressures. Negative pleural pressures are associated with lower ventricular pressures relative to alveolar pressure, and hence with an increase in right ventricular afterload.

Treatment for Pulmonary Hypertension (PH) does not always improve patients' outcomes. Before beginning a PH treatment for patients with chronic lung disease, it must be determined whether improving pulmonary hemodynamics has an impact on patient outcome, because most of these patients' symptoms are caused by ventilation limitations rather than CO. The primary goal of treatment is to manage the underlying lung disease, as there is presently no medicine proven to effectively treat PH associated with chronic lung disease. Patients with severe PH should be sent to experienced PAH centers and participation in clinical trials should be considered.

Correspondence to: Pierre Oliver, Department of Acute Medicine, University Hospital Basel, Basel, Switzerland, E-mail: pierre.oliver@po.ch

Received: 28-Jul-2023, Manuscript No. IME-23-26741; **Editor assigned:** 02-Aug-2023, PreQC No IME-23-26741 (PQ); **Reviewed:** 16-Aug-2023, QC No. IME-23-26741; **Revised:** 23-Aug-2023, Manuscript No. IME-23-26741 (R); **Published:** 30-Aug-2023, DOI: 10.35248/2165-8048.23.13.421

Citation: Oliver P (2023) Chronic Obstructive Pulmonary Disease and Lung Disease-Related Pulmonary Hypertension. Intern Med. 13:421.