Journal of Clinical Respiratory Open Access

Extended Abstract
Vol. 1, Iss. 1
2019

COPD 2016: Pulmonary hypertension in COPD: Is it always the consequence of end-stage disease?_Monika Szturmowicz_National Tuberculosis and Lung Diseases Research Institute, Poland

Monika Szturmowicz

Pulmonary hypertension (PH) defined as an average resting pulmonary arterial pressure (mPAP) equal to or greater than 25 mm Hg (measured directly), is found in 50% of patients with end-stage COPD. In most cases, a slight pH is observed. However, in 2 to 7% of patients, a severe pH (defined as mPAP> 35 mm Hg or CI <2.5 l / min) develops. Most interestingly, the severe PH in COPD is not always associated with a terminal illness. The differential diagnosis on this occasion should exclude the influence of other comorbidities on HP (left heart disease, venous thromboembolism and sleep-disordered breathing). The latest publications indicate that the clinical phenotype of severe PH in COPD is characterized by deep hypoxemia, hypocapnia and a very low diffusion capacity of the lung for carbon dioxide (DLCO), despite a slight or moderate obstruction of the respiratory tract. It is still unclear whether such a phenotype is associated with certain genetic rearrangements. According to the latest PH guidelines, optimal treatment for COPD combined with long-term oxygen therapy in patients with PaO2 less than 60 mm Hg is indicated in the PH-COPD. However, in patients with severe PH, it is advisable to consult an expert center. The latest results from clinical trials with specific PH drugs are disappointing. Despite the improvement in pulmonary hemodynamics, no significant change in patients' exercise capacity or quality of life is reported. Future research should be directed to the identification of patients with PH-COPD, in whom the maximum exercise capacity is limited by low cardiac output and not by the exhausted ventilatory reserve.

Lung disease is one of the most common causes of pulmonary hypertension (PH). The development of HP influences the course of lung disease, worsening clinical symptoms and prognosis. According to the most recent publications, HP during pulmonary diseases develops both as a result of a "parenchymal" and vascular pathology, in patients with a genetic predisposition. Prolonged infection (especially viral) can be an additional promotional factor. Right cardiac catheterization (CHR), which is an invasive procedure, is the only objective method of diagnosing PH. According to the latest recommendations, the algorithm for managing PH and coexisting interstitial lung disease is based on the RHC and the results of pulmonary function tests. The majority of patients develop mild PH during advanced lung disease. The best treatment for the underlying lung disease combined with long-term oxygen therapy is recommended in this group. In case of severe PH (average pulmonary arterial pressure at rest (mPAP) ≥ 35 mm Hg), the alternative cause of PH should be sought. The use of PAH-specific drugs should be limited to patients with severe PH participating in clinical trials.

The established model of PH during DPLD was that linked to vascular remodeling due to interstitial pathology and/or hypoxia. However, the presence of vascular pathology in areas of minor interstitial lung disease and the occurrence of PH at the early stage of the disease in patients without significant hypoxemia indicate a more complex pathogenesis. Recent studies suggest the role of alveolar epithelial cells, fibroblasts and vascular cells in the development of PH. In IPF, the process is initiated by fibrocytes carrying CD34 or CD45 markers, derived from the bone marrow or from the population of pulmonary steam cells, responsible for the induction of collagen production and the recruitment of inflammatory mediators. profibrogenic. The process appears to be regulated by a wide variety of angiogenesis promoters and inhibitors as well as by growth factors.

monika.szturmowicz@gmail.com

This abstract is partly presented at 3rd International Conference on Chronic Obstructive Pulmonary Disease on July 11-12, 2016 held at Brisbane, Australia