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Pulmonary hypertension in COPD: Is it always the consequence of end-stage disease?

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Pulmonary hypertension (PH) defined as mean resting pulmonary artery pressure (mPAP) equal or higher than 25 mm Hg (measured directly), is found in 50% of the patients with end-stage COPD. In most cases mild PH is observed. Nevertheless in 2-7% of patients, severe PH (defined as mPAP>35 mm Hg or CI<2.5 l/min) develops. What's most interesting, severe PH in COPD is not always combined with end-stage disease. The differential diagnosis on such occasion should exclude the influence of other comorbidities on PH (left heart disease, venous thromboembolism and sleep disordered breathing). Latest publications indicate that clinical phenotype of severe PH in COPD is characterized by profound hypoxemia, hypocapnia and very low diffusion capacity of the lung for carbon dioxide (DLCO), despite mild or moderate airway obstruction. It is still not known, if such phenotype is combined with certain genetic rearrangements. According to latest PH guidelines, optimal COPD treatment combined with long term oxygen therapy in the patients with PaO2 below 60 mm Hg is indicated in PH-COPD. Nevertheless in the patients with severe PH, the referral to an expert center is advised. Latest results of clinical trials with PH-specific drugs are disappointing. Despite the improvement in pulmonary hemodynamics, no significant changes in exercise capacity or quality of life of the patients are reported. Future research should be directed towards the identification of those PH-COPD patients, in whom maximal exercise capacity is limited by low cardiac output, and not by the exhausted ventilatory reserve.

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COPD and its association with gene polymorphism and cigarette smoke

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Chronic Obstructive Pulmonary Disease (COPD) is considered to be one of the most common preventable diseases. Large number of population studies has cited the alpha-1 antitrypsin gene polymorphism is one of the primary genetic reasons for its pathogenesis. Among the environmental factors, along with C-F compounds, cigarette smoke is considered to be the main factor for the disease progression. But the question is how the cigarette smoke is related with gene polymorphism? The question is indeed a complex issue. We are trying to understand the issue, in terms of the cigarette smoke compounds, how they are regulating the antioxidant enzymes. We are getting some interesting results, how the cigarette smoke extracts affecting the glutathione S-transferase enzyme machinery. We are searching for the mechanism concerning the inflammatory effect of the extracts. From the genetic point of view, the gene expression studies of the determinants of cellular GSH levels are also under progress. We have performed the experiments on gene polymorphism studies of COPD patients and have shown, the GSTM1 gene polymorphism is the main factor for the disease among the coal miners. The studies relating the understanding of the whole cigarette smoke metabolizing pathway is going on and will be presented and discussed.

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