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Mild chronic obstructive pulmonary disease: new insights of pathophysiology

Abstract

By giving their relatively well-preserved spirometry, some have discussed that respiratory symptoms in patients with mild COPD are unlikely to be related to lung function abnormalities. There is emerging physiological and clinical evidence of peripheral airway dysfunction, reduced physical activity levels, and diminished quality of life, and increased exercise intolerance, hospitalization, mortality in patients with mild COPD. The objective of the present review article was to summarized recent studies regarding the pathophysiology of mild COPD.

Keywords: pathophysiology, screening, diagnosis, treatment, chronic obstructive pulmonary disease

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Abbreviations: COPD, chronic obstructive pulmonary disease; FEV1, forced expiratory volume in one second; FVC, forced vital capacity; NA, not available; VA/Q, lung ventilation/perfusion ratio

Introduction

Chronic obstructive pulmonary disease (COPD) is an inflammatory abnormality of the lung that is partial reversible or irreversible airflow limitation.[1] The disease is caused by a complex interaction between genes and environment.[2] COPD is major global epidemic, affecting 5-15% of all adults in industrialized countries[3] and accounting for over 3 million deaths each year, globally.[4] Smokers constitute only approximately 50 % of the global COPD cases although cigarette smoking is the single most important risk factor.[2] Smoking cessation does not stop the underlying inflammatory process in the COPD lungs.[5] Other known risk factors are genetic abnormalities, such as alpha-1-antitrypsin deficiency, occupational dusts, chemicals and biomass smoke exposures, air pollution, respiratory infections, particularly during childhood, poor nutrition, and airway hyperresponsiveness.[6] The inflammatory process may extend beyond the pulmonary system, resulting in a state of persistent low grade systemic inflammation[7,8] which has been implicated in complications of COPD including ischemic heart disease,[9] arrhythmias,[10,11] and cachexia.[12] The current treatment is aimed at relieving patient's clinical symptoms[13] and is modified by disease severity.[1]

Definition of mild COPD: Patients with mild COPD is a group of patients with abnormal spirometry, but are otherwise asymptomatic. The physical examination is completely unremarkable. Nevertheless, patients may reveal a positive cough test (which is defined as recurrent coughing after patients take a deep breath to maximal lung capacity and coughs more than once), and have a forced expiratory time at the bedside of 9seconds or greater.[14] Mild COPD is defined by a post-bronchodilator FEV_1 that is 80% of predicted or greater in the presence of an FEV_1/FVC ratio of less than 70% and characteristic symptoms, such as exertional dyspnea in most cases.[1,15]

Pathophysiology of mild COPD: Routine spirometry does not accurately reflect the most pathophysiological heterogeneity that exists in patients with mild COPD.[16] Ofir and colleagues reported that symptomatic patients with mild disease revealed diminished exercise performance and reduced peak oxygen consumption relative to controls.[17–19] They found that dyspnea intensity was consistently elevated for any given work rate and ventilation in the patients with mild disease.[17] These differences occurred at relatively low work rates that mimic the metabolic requirements of basic daily activities (eg, stair walking).[17] More interestingly, the effects of dyspnea in mild COPD appeared to be even more pronounced in female patients.[18] Several studies revealed greater ventilator inefficiency during exercise as reflected by an increased ventilator equivalent for carbon dioxide (CO_2).[17–19] This is likely due to an increased physiological dead space because of regional increases in lung ventilation/perfusion (VA/Q). The partial pressure end-tidal CO_2 is also consistent lower at rest and during all exercise intensities in mild COPD relative to controls, indicating the presence of chronic alveolar hyperventilation and potential, and as yet unidentified alterations in the ventilator control system. Patients with mild COPD cannot appropriately increase tidal volume that is associated with a greater increase in dyspnea intensity in these patients. The respiratory system in mild disease reaches or approaches its physiological limits at lower work rates and ventilations relative to healthy controls.

Conclusion

Physiological abnormalities in mild COPD often mimic the responses characteristically observed in more advanced stages of COPD. Spirometric measures of airflow limitation do not adequately reflect the complex pathophysiology of mild COPD. When the respiratory system is experimentally stressed with exercise, several respiratory abnormalities with attendant negative sensory consequences can be uncovered in this patient group.

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Conflict of interest

The author declares no conflict of interest.

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