Chronic obstructive lung disease effecting brain pathology and its treatment.

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Abstract

Brain pathology is a poorly understood systemic manifestation of chronic obstructive pulmonary disease (COPD). Imaging techniques using magnetic resonance (MR) diffusion tensor imaging (DTI) and resting state functional MR imaging (rfMRI) provide measures of white matter microstructure and gray functional activation, respectively. The patients with COPD would have reduced white matter integrity and that functional communication between gray matter resting-state networks would be significantly different to control subjects. In addition, we tested whether observed differences related to disease severity, cerebrovascular comorbidity and cognitive dysfunction.

Keywords: Hypoxemia, Hypercapnia, Exacerbations, Physical activity, Pulmonary disease.

Introduction

Over the past few decades, chronic obstructive lung disease (COPD) has been considered a disease of the lungs, often caused by smoking. Nowadays, COPD is regarded as a systemic disease. Both physical effects and effects on brains, including impaired psychological and cognitive functioning, have been demonstrated. Patients with COPD may have cognitive impairment, either globally or in single cognitive domains, such as information processing, attention and concentration, memory, executive functioning and self-control. Possible causes are hypoxemia, hypercapnia, exacerbations and decreased physical activity. Cognitive impairment in these patients may be related to structural brain abnormalities, such as gray-matter pathologic changes and the loss of white matter integrity which can be induced by smoking. Cognitive impairment can have a negative impact on health and daily life and may be associated with widespread consequences for disease management programs. It is important to assess cognitive functioning in patients with COPD in order to optimize patient-oriented treatment and to reduce personal discomfort, hospital admissions and mortality. COPD is recognized as a disease with many systemic components. Among them, the neuro-psychical component (anxiety/depression) , has already been recognized. Besides this, in the last decade, research has been initiated on the neuro morphological substrate to explain whether certain manifestations such as cognition alteration, the occur fence of balance disorders, etc. could also be due to the impact of COPD, or would only be manifestations related to age and/or other comorbidities [1].

Treatments for COPD-induced cognitive dysfunction

Currently there is no available therapy to slow or reverse the progression of COPD and its associated comorbidities. However, current knowledge of therapies involved in the management of COPD and/or cognitive dysfunction associated with other neurological disorders could be utilised in order to assist in the production of treatments [2, 3].

Anti-inflammatory treatments to manage COPD-induced cognitive dysfunction: Given the damaging consequences of excessive inflammation and oxidative stress in COPD, targeting these both in the pulmonary and systemic circulations may reverse and prevent the pathological effects they have on the respiratory system and comorbidities associated with COPD [4]. One such therapy that is commonly used in COPD is corticosteroids, which are known to reduce lung inflammation and suppress airway obstruction in AECOPD, thus resulting in bronchi dilation and ease of breathlessness. However, this treatment has been shown to have limited efficacy as people with COPD are generally resistant to the steroid treatment, but is recommended that inhaled corticosteroids are taken alongside long-acting-β2-agonists to improve difficulties in respiration. Interestingly, a study found that patients with COPD having high blood eosinophil less than 2% counts took inhaled corticosteroids combined with long-acting-β2-agonists overall reduced the risk of exacerbation episodes, compared to their placebo counterparts [5].

Conclusion

As previously described, patients with COPD have excessive levels of ROS in the lungs and it has been hypothesised

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that the oxidative burden in people with COPD could be reduced through antioxidant therapies. We have shown that GPX-1-deficient mice exposed to CS have increased lung inflammation when compared to room air exposed mice and wild-type CS-exposed mice. Similarly, GPX-1 deficient mice infected with IAV had significantly more lung inflammation compared to vehicle treated wild-type/GPX-deficient mice. The GPX-mimetic, ebselen (SPI-1005; a synthetic organ selenium drug), has undergone several clinical trials for the treatment for hearing loss, tinnitus and Meniere's disease.

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