PERSPECTIVE

New pharmaceutical approaches to address the cognitive impairment associated with chronic obstructive pulmonary disease

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ABSTRACT

With over 3.23 million fatalities per year, Chronic Obstructive Pulmonary Disease (COPD) is currently the third leading cause of mortality in the world and a significant global health burden. Globally, COPD costs about €82 billion annually and significantly increases morbidity and mortality. Importantly, managing COPD's comorbidities and acute exacerbations brought on by viruses and bacteria accounts for a large portion of the disease burden and health care use in this condition. Recent clinical investigations have demonstrated that cognitive dysfunction, including deficits in executive function, memory, and attention, is present in up to 60% of persons with COPD. This has an impact on crucial outcomes like quality of life, hospitalization, and survival.

INTRODUCTION

Incurable chronic obstructive pulmonary disease (COPD) is characterized by ongoing respiratory symptoms and partially reversible airflow restriction. It places a heavy strain on society and the economy because it is the third greatest cause of mortality worldwide. Globally, 174.5 million cases of COPD were documented in 2015, with a mortality rate of up to 3.2 million fatalities. The estimated €82 billion in global economic expenses associated with COPD are expected to continue to climb. Notably, due to greater levels of Cigarette Smoke (CS) and non-smoking home air pollution, such as biomass fuel used for cooking and heating, more than 90% of COPD-related mortalities take place in low and middle-income nations. In reaction to noxious particles and gases, COPD-induced airflow limitation is linked to an increase in lung inflammation, pro-inflammatory cytokine production, cell apoptosis, epithelial cell death, and oxidative stress in both the airways and lung parenchyma. People with COPD frequently experience dyspnea, weariness, a chronic cough, and sputum production as symptoms. COPD includes lung parenchyma and structural damage to both small and big airways (airways disease) (emphysema). Chronic airways disease is characterized by ongoing bronchial and bronchiole inflammation, which restricts airflow to and from the lungs and results in breathing difficulty. Emphysema causes breathing problems due to expansion of the alveoli and damage to the alveolar walls (via rigidity and degradation). Despite the fact that these pathologies permanently harm the respiratory system in COPD patients, there are preventative strategies and management options that will be discussed in this study. A significant portion of the burden of COPD is related to treating acute exacerbations of COPD (AECOPD), which are frequently brought on by bacterial and/or viral infections, with co-infections also being very common. Exacerbations can happen to patients many times a year, and for those who need to be hospitalized, the in-patient death rate may be as high as 30%. Poorer health status, more severe airflow limitation, and/or a history of exacerbations are factors that predict mortality. Key COPD symptoms such as exacerbated dyspnea, increased coughing and

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sputum production (which may be purulent in the presence of infection), increased wheezing, and fevers are all signs of an exacerbation. The rhinovirus (36%), respiratory syncytial virus (22%) and Influenza A Virus (IAV) (25%) are viral infections frequently linked to AECOPD. In addition, it has been established that certain bacterial species, such as Moraxella catarrhalis, Streptococcus pneumoniae, and Haemophilus influenzae and parainfluenzae, are frequently isolated from the lower respiratory tract and contribute to episodes of exacerbations in COPD patients. The disease is known to be associated with a number of disabling extra-pulmonary symptoms, or comorbidities, including skeletal muscle atrophy, cardiovascular disease, and metabolic syndrome, in addition to the respiratory discomfort that is the hallmark of COPD. People with COPD exhibit cognitive deficits, which have an effect on crucial outcomes like quality of life, hospitalization, and overall survival, according to recent clinical studies. Therefore, more research is required to fully understand how COPD affects the central nervous system (CNS), specifically cognitive integrity. In this review, we'll concentrate on the clinical and experimental data showing a connection between cognitive dysfunction and COPD, CS exposure, or both (CS exposure being the main risk factor for COPD). Second, with a focus on central pathways, we will discuss probable pathobiological mechanisms relating COPD and cognitive decline. In addition to being linked to traumatic experiences (such as physical and/or psychological occurrences) and substance abuse (such as using illegal substances, prescription medications, and/or alcohol), cognitive impairment has been linked to a number of neurodegenerative and psychiatric disorders. Recent clinical studies have shown that there is a sizable population of stable COPD patients who experience cognitive dysfunction. Up to 61% of these patients experience cognitive losses, including impairments in memory, executive function, and attention, compared to only 12% of the age-equivalent healthy population. It is challenging to determine from the literature the degree of cognitive decline as well as whether the patients included had stable COPD or were going through an exacerbation because the presence or absence of an exacerbation is not always defined and a variety of neuropsychological cognitive tests have been used, varying between studies. The length of stable COPD symptoms and pathology in patients has been linked to an increased prevalence of cognitive impairment. According to a study using the MMSE, those with stable COPD had statistically significant lower cognitive scores than healthy, age-matched individuals by an average of about 3 points. Furthermore, investigations have revealed a link between cognitive impairment and the intensity of respiratory symptoms in those with COPD, as it was found that those with more severe COPD had worse cognitive functioning than those with milder-to-moderate disease. The prevalence of cognitive impairment is very high among COPD patients. Very few research has been done to date to investigate the underlying causes of cognitive dysfunction with COPD. We hypothesize that the lung injury and chronic inflammatory state brought on by COPD may be a significant component in these people's altered neuropathology and apparent cognitive impairment. The "spill-over" of lung inflammation and oxidative stress into the systemic circulation could be targeted with pharmacotherapeutic and non-pharmacotherapeutic treatments to help those who are impacted. These can include anti-inflammatory/antioxidants like apocynin and ebselen as well as pulmonary rehabilitation, which could stop the BBB from being disrupted, reduce microglial activation, and improve astrocytic and neuronal function, all of which could stop cognitive deficits brought on by neuroinflammation.