Short Communication

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Control of Respiratory Diseases in Patients with Amyotrophic Lateral Sclerosis During the COVID-19 Pandemic

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Abstract

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Short Communication

Persons older than 60 with hypertension, diabetes, COPD, cardiovascular, cerebrovascular, liver, kidney, and gastrointestinal diseases are more susceptible to the infection by SARS-CoV-2 and experience higher mortality when they develop COVID-19 [1-3]. COVID-19 cases with pre-existing COPD, or complicated by secondary bacterial pneumonia, are more severe and this may be due to complex immune pathogenesis[4]. Acute exacerbation of COPD has an intimate relationship with respiratory tract virus infection5. The prevalence of SARS-CoV-2 infection in COPD patients is not clear, but smokers and COPD patients are more susceptible to the infection of middle east respiratory syndrome coronavirus (MERS-CoV). MERS-CoV does the dipeptidyl peptidase IV (DPP4) such as a receptor, that had a higher expression in smokers and COPD patients than in non-smokers [5,6], showing a correlation with smokers and a coronavirus specie.

Asthma and chronic obstructive pulmonary disease (COPD) are associated with chronic inflammation of the respiratory tract. Asthma affects about 10% of adults and an even greater proportion of children. COPD affects about 10% of people over 40 years of age [7]. Amyotrophic lateral sclerosis (ALS) and asthma are an possible association. ALS is a complex pathology involving many factors and signaling pathways, such as oxidative stress, dysfunction of mitochondria, altered intracellular protein traffic, protein aggregation, and glutamate excitotoxicity related with a reduction in the expression of glutamate transporters, apoptosis, proinflammatory cytokines and deficiency of cholinergic synapses

[8-11]. Asthma is a chronic inflammatory disease with complete and/or parcial reversible airway obstruction and nonspecific airway hyper-reactivity asthma, there is a characteristic pattern of inflammation (type 2 immunity) [12] usually characterized by allergic inflammation with increased production of IgE by B cells, mast cell degranulation and eosinophil infiltration orchestrated by TH2 cells, but the same pattern of inflammation may also occur in non-allergic individuals [13].

Neurodegenerative disorders have the neuro-inflammation as characteristic of the pathologically affected tissue. There is extensive evidence that neurons and immune cells communicate, his interactions are involved in the lung inflammation of asthmatic patients [14,15]. Thereby, patients with both comorbidities (ALS and asthma and/or COPD) need a special managed according to its symptoms and severity, to decrease the clinical exacerbations, improve pulmonary function, and reduce morbidity. Although there aren't direct correlations , the similar multifactorial triggers, and the critical roles of neuronal inflammation, suggest that patients with ALS and asthma and/or CPOD must to be differential attention during the covid-19 pandemic.The major risk factor for COPD in western countries is cigarette smoking. COPD is a syndrome by progressive airflow obstruction, affecting peripheral airways, which leads to air trapping, dynamic hyperinflation and shortness of breath on exertion. In some patients, there are features of both diseases, and this has been termed asthma–COPD overlap syndrome [16,17] both are characterized by chronic inflammation of the respiratory tract, although the nature of the inflammation and its location differ18. Patients with COPD have a different pattern of inflammation (type 1 and type 3 immunity) [19,20]. This inflammation is an amplification of the mucosal inflammatory response to inhaled irritants, such as tobacco.

Approximately six million people worldwide die due to tobacco use each year [21]. The cigarette contributes to the pathogenesis and a recognized risk factor of chronic obstructive pulmonary disease (COPD), hypertension, cardiovascular disease, cancer, chronic systemic diseases with inflammatory components such as atherosclerosis, Crohn's disease, rheumatoid arthritis, psoriasis, Graves' ophthalmopathy, and noninsulin-dependent diabetes mellitus [22,23]. The cigarette smoke promotes inflammation by inducing the production of pro-inflammatory cytokines, such a TNF-α, IL-1, IL-6, IL-8 and granulocyte-macrophage colony-stimulating factor (GM-CSF), and increasing the accumulation of immune cells in the airway [24,25]. Studies in vitro provide evidence for its immunosuppressive properties [22,26,27], which is demonstrated to increased susceptibility towards respiratory tract infections , bacterial meningitis, periodontitis and poorer wound healing [22,23,28].

Recently, a histopathological study with 4 patients in pos-mortem presented a minucious lungs examination showing a bilateral diffuse alveolar damage with a comparatively mild-to-moderate lymphocytic infiltrate, composed of a mixture of CD4+ and CD8+ lymphocytes. The dominant process in all cases was consistent with diffuse alveolar damage, with a mild to moderate mononuclear response consisting of notable CD4+ aggregates around thrombosed small vessels, and significant associated hemorrhage [29].

Therefore, the previous extensive medicine and immunological knowledge provide us with the understanding that carriers of neurodegenerative diseases and his extensive association with lung dysfunction may be a severe risk factor to COVID-19 and pulmonary complications, principally, when tobacco's associated or not to COPD and/or asthma. Neuromuscular Disorders (NMD) can affect breathing function, but do not in general damage lung tissue, impaired breathing in NMD is due to weak diaphragm and breathing muscles. Thus, the strong physician's presence, nurses and caregivers in the pandemic process and in the search for measures to stop smoking are of paramount importance, because the cigarette smoke alters many signaling pathways, immune responses and increases the chance of complications in a covid-19 infection.

Conflict of Interest

No conflict of interest.

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