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A Short Commentary on Pulmonary Fibrosis and COVID-19 Pandemic

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Commentary

A severe acute respiratory illness caused by severe acute respiratory syndrome coronavirus 2 was reported in Wuhan, China, in December 2019. (SARS-CoV-2). Over 3 million individuals had been proven sick by the end of April 2020, with over 1 million in the United States alone, and over 215 000 fatalities. COVID-19 causes a wide range of symptoms, from moderate upper respiratory tract symptoms to severe acute respiratory distress syndrome. Idiopathic pulmonary fibrosis (IPF) and severe COVID-19 share a number of risk factors, including growing age, male sex, and comorbidities including hypertension and diabetes. Antifibrotic therapy's function in patients with IPF who develop SARS-CoV-2 infection, as well as the scientific justification for its continuation or discontinuation, remains unclear. A number of approved and prospective antifibrotic drugs have also been tested in models of acute lung damage and viral pneumonia. Previous coronavirus infections, such as severe acute respiratory syndrome and Middle East respiratory syndrome, as well as new findings from the COVID-19 pandemic, show that SARS-CoV-2 infection may have significant fibrotic effects.

Antifibrotic treatments that are now available or in development may be useful in avoiding severe COVID-19 in people with IPF, treating severe COVID-19 in persons without IPF, and preventing fibrosis following SARS-CoV-2 infection. Despite the fact that pulmonary fibrosis can develop without a definite initiating factor or a clinically obvious first acute inflammatory phase, it is more often linked with serious lung damage. Respiratory infections, persistent granulomatous illnesses, medicines, and connective tissue abnormalities are all possible causes. Pulmonary fibrosis is linked to irreversible lung failure and persistent pulmonary architectural deformity. Pulmonary fibrosis is fundamental to the pathophysiology of severe acute respiratory distress syndrome (SARS) and MERS, according to available clinical, radiographic, and postmortem data, and recent evidence shows that pulmonary fibrosis might potentially worsen infection with SARS-CoV-2. The aim of the commentary is to look at the existing knowledge on the pathogenesis of COVID-19 infection lung damage. We assess the evidence for possible risk factors for the development of pulmonary fibrosis in patients with the illness and recommend risk-reduction methods. We conclude that advanced age and COVID-19 infection are predictors of pulmonary fibrosis, based on the current research. Because there is no proven effective targeted therapy for pulmonary fibrosis, risk reduction strategies should focus on reducing the disease's severity and protecting the lungs from additional damage. COVID-19 mostly harms the lungs, but it may also harm other organs and systems such as the heart, immune system, and so on. Despite the fact that the aetiology of COVID-19 has been thoroughly explained, there is currently no particular treatment for the condition, with most therapies consisting of supportive care. Stem cell therapy has showed promise in preclinical trials as a possible treatment for refractory and uncontrollable lung diseases.

The pathogenic development and possible processes underpinning stem cell treatment in COVID-19, as well as registered COVID-19 clinical studies, in this study. So far, preclinical investigations and clinical trials have shown that mesenchymal stem cells (MSCs) or MSC-like derivatives are the most promising stem cell treatments for COVID-19 treatment. By secreting a variety of factors, MSCs have been shown to alleviate cytokine release syndrome (CRS) and preserve alveolar epithelial cells, indicating safety and potential effectiveness in COVID-19 patients with acute respiratory distress syndrome (ARDS). However, because stem cell quality consistency and homogeneity cannot be measured or guaranteed at this time, additional research is needed. Since the first reports of coronavirus disease 2019 (COVID-19) in late 2019, infections caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have spread fast, culminating in a worldwide pandemic that has killed millions. The high number of infected persons first necessitated the coordination of global healthcare resources to offer supportive treatment for the critically ill. While clinical trials for safe and efficient antiviral medicines are underway, and vaccine research efforts are speeding up, the long-term consequences of SARS-CoV-2 infection are becoming more well-known and worrying.

Although SARS-CoV-2 enters the body mostly through the upper and lower respiratory tracts, leading in COVID-19 pneumonia as the most frequent symptom, acuity is also a common symptom. In addition, accumulating evidence suggests that SARS-CoV-2 infection affects the central nervous system (CNS) and peripheral nervous system (PNS), causing direct or indirect neuron damage and long-term neurological consequences. An update on the processes behind the long-term consequences of SARS-CoV-2 infection in the three primary areas of lung injury, brain injury, and neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and multiple sclerosis, as well as highlights the need of patient surveillance after an acute SARS-CoV-2 infection to offer a justification for the prevention, diagnosis, and therapy of these long-term consequences Several crossing mechanisms between coronavirus infection and fibrotic pathways are reviewed in order to identify variables and processes that may be targeted to enhance patient outcomes. The objective is to enhance detection of possible contributory risk factors for fibrotic illness by presenting reports of post-infection sequelae from past coronavirus epidemics.

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