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# Pulmonary Fibrosis in COVID-19 Survivors

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## ABSTRACT

Pulmonary fibrosis can arise without a distinct initiating factor, and it is more often linked with significant lung damage without a clinically evident first acute inflammatory phase. 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.

**Keywords:** SARS-CoV-2; Pulmonary fibrosis; Respiratory syndrome

## INTRODUCTION

Fibrosis may be thought of as a result of a wound-healing mechanism that is linked to the severity of the triggering event. COVID-19 lung damage has been linked to a variety of causes, including viral and immune-mediated processes. Aside from these, other variables may predispose people to serious lung damage, increasing their chance of death or pulmonary fibrosis in survivors [1].

Discuss the pathological processes involved in fibrosis formation and investigate the pathophysiology of lung damage in COVID-19 infection of pulmonary fibrosis after previous and current human coronavirus outbreaks in this study [2]. In addition, we examine the evidence for risk factors for lung fibrosis progression after COVID-19 infection, as well as potential risk reduction methods.

Other medications with antiviral and immunomodulatory properties being measured comprise ivermectin and nitazoxanide. Though these medications have shown in vitro movement against SARS-CoV-2, supplementary studies are required to regulate their efficacy in the treatment of COVID-19. Because cytokine storm caused by hyperinflammation is a key role in lung damage in SARS-CoV-2 infection, immunosuppressive drugs have been widely used in the treatment of COVID-19 infection [3]. The IL-1 receptor blocker anakinra, which has shown increased survival benefits in cytokine storm related to sepsis, and the IL-6 receptor blocker are two immunosuppressive medicines now under investigation. Tocilizumab is presently being tested in COVID-19 patients in a multicenter clinical study. Despite their widespread usage, corticosteroids have little evidence of therapeutic benefit. Clinical investigations have indicated that the use of convalescent plasma has some potential. Furthermore, mesenchymal stem cells, which have the multipotent ability to repair injured alveolar epithelium, produce anti-inflammatory factors and suppress fibroproliferation, have been proposed as a therapy for ARDS, the primary cause of death in COVID-19 illness [4]. Clinical studies for the utilisation of mesenchymal stem cells are presently underway.

## CONCLUSION

The SARS-CoV-2 virus has continued to spread throughout the world, affecting millions of people. Pulmonary fibrosis has been identified as a possible sequela in survivors of earlier human coronavirus epidemics. The process of fibrogenesis is centred on virus-induced lung damage, immune response, and efforts at repair. Advanced age, disease severity, length of ICU hospitalisation and mechanical ventilation, smoking, and persistent drinking are all thought to be predictors of pulmonary fibrosis. Because there is no proven effective targeted therapy for pulmonary fibrosis, risk reduction strategies should focus on reducing the severity of the condition and protecting the lungs from additional damage.

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