*Editorial*

# Pulmonary Fibrosis in COVID-19 Survivors- an editorial

**Prof. Dr. HN Sarker**

An outbreak of the novel coronavirus nCoV-19 (SARS-CoV-2), responsible for the coronavirus disease-19 (COVID-19), was first detected in Hubei province, China, on December 31, 2019. It has rapidly spread globally with approximately 37,354,063 confirmed cases and 1,075,584 deaths till 10th October, 2020 [1]. World Health Organization (WHO) declared COVID- 19 pandemic on 11th March 2020.

SARS-CoV-2 primarily affects the respiratory system like severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS). The disease has a diverse course; patients may range from asymptomatic to critical disease with respiratory failure, complicated by acute respiratory distress syndrome (ARDS).

Pulmonary fibrosis is a recognized sequelae of ARDS. Pulmonary fibrosis were common following SARS and MERS and current evidence suggests pulmonary fibrosis could complicate infection by SARS-CoV-2 [2]. Pulmonary fibrosis can also occur from other causes such as connective tissue disorders, drugs, chronic granulomatous diseases, and respiratory infections [3].

Pulmonary fibrosis leads to chronic breathing difficulties, long-term disability and affects patients' quality of life.

There are no specific mechanisms that lead to this phenomenon in COVID-19, but some information arises from previous severe acute respiratory syndrome (SARS) or Middle East respiratory syndrome (MERS) epidemics.

An initial phase of lung injury is followed by acute inflammation as well as an attempt at repair. This process can result in the restoration of normal pulmonary architecture, or it may lead to pulmonary fibrosis with architectural distortion and irreversible lung dysfunction. The repair process involves regeneration by native stem cells and connective tissue deposition to replace areas of defect [4]. Alveolar macrophages play a central role in this process by phagocytizing alveolar debris and the production of cytokines and growth factors involved in the repair [5].

This repair by fibroproliferation leads to a potential increase in the risk of pulmonary fibrosis occurring as a sequela of COVID-19 [6]. Fibrotic changes have been found on chest CT scans in COVID-19 patients. Fibrotic changes have been seen in 33.9% patients in advanced-phase disease in one study. The predictors of pulmonary fibrosis in COVID-19 infection are advanced age, illness severity, length of ICU stay and

1. Professor and Head of department, Medicine, SSKMC, Gopalgonj

mechanical ventilation, smoking and chronic alcoholism [7, 8, 9, 10, 11, 12].

There is no proven effective targeted therapy against pulmonary fibrosis; although two disease-modifying drugs, nintedanib and pirfenidone, have shown promise in clinical trials in slowing down the decline in pulmonary function and have been approved for the treatment of idiopathic pulmonary fibrosis (IPF) [13, 14]. Risk reduction measures should be directed at limiting the severity of the disease and protecting the lungs from other incidental injuries.

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