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POST COVID PULMONARY FIBROSIS

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ABSTRACT:

The risk factors for development of fibrotic-like radiographic abnormalities after severe COVID-19 are incompletely described and the extent to which CT findings correlate with symptoms and physical function after hospitalisation remains unclear. At 4 months after hospitalisation, fibrotic-like patterns were more common in those who underwent mechanical ventilation (72%) than in those who did not (20%).

Keywords: COVID-19, Corona virus, pneumonia, pulmonary fibrosis

INTRODUCTION:

After the COVID-19 outbreak, increasing number of patients worldwide who have survived COVID-19 continue to battle the symptoms of the illness, long after they have been clinically tested negative for the disease. As we battle through this pandemic, the challenging part is to manage COVID-19 sequelae which may vary from fatigue and body aches to lung fibrosis. This review addresses underlying mechanism, risk factors, course of disease and treatment option for post covid pulmonary fibrosis.

After the COVID-19 outbreak, increasing number of patients worldwide who have survived COVID-19 continue to battle the symptoms of the illness, long after they have been clinically tested negative for the disease. They are called as long – haulers. As we battle through this pandemic, the challenging part is how to manage this COVID-19 Sequelae which may vary from mild in terms of fatigue and body aches to severe forms requiring long term oxygen therapy and lung transplantation due to lung fibrosis, significant cardiac abnormalities and stroke leading to significant impairment in Quality of health. Various studies have reported that around 70–80% of patients who recovered from COVID-19 presents with persistence of at least 1 or more symptoms, even after being declared COVID-free[1]

Mechanism of post COVID pulmonary fibrosis

Various mechanisms of lung injury in COVID-19 have been described, with both viral and immune-mediated mechanisms being implicated. Pulmonary fibrosis can be either subsequent to chronic inflammation or an idiopathic, genetically influenced and age related fibroproliferative process. Pulmonary fibrosis is a known sequela to ARDS. However, persistent radiological abnormalities after ARDS are of little clinical significance and have dwindled with protective lung ventilation.[2]

It has been found that 40% of patients with COVID-19 develop ARDS, and 20% of ARDS cases are severe. The prevalence of post-COVID-19 fibrosis will become apparent with time, but early analysis from patients with COVID-19 on hospital discharge suggests that more than a third of recovered patients develop fibrotic abnormalities. The pathological feature of ARDS is diffuse alveolar damage (DAD) which is characterized by an initial acute inflammatory exudative phase with hyaline membranes, followed by an organizing phase and fibrotic phase.[7] Previous studies highlight that duration of disease is an important determinant for lung fibrosis post ARDS. This study showed that, 4% of patients with a disease duration of less than 1 week, 24% of patients with a disease duration of between weeks 1 and 3, and 61% of patients with a disease duration of greater than 3 weeks, developed fibrosis.[3]

Treatment of post COVID 19 pulmonary fibrosis

Currently, no fully proven options are available for the treatment of post inflammatory COVID 19 pulmonary fibrosis. Various treatment strategies are under evaluation. It has been proposed that prolonged use of anti-viral, anti-inflammatory and anti-fibrotic drugs diminish the probability of development of lung fibrosis. However, it is yet to be ascertained whether early and prolonged use of antiviral agents may prevent remodeling of lung or which of the available antiviral is more effective. Though risk-benefit ratio should be assessed prior to use, prolonged low dose corticosteroid may prevent remodeling of lung in survivors. Anti-fibrotic drugs, such as pirfenidone and nintedanib, have anti-inflammatory effects as well and thus they may be used even in the acute phase of COVID-19 pneumonia. Pirfenidone exerts

anti-fibrotic, anti-oxidative and anti-inflammatory effects. Pirfenidone could attenuate ARDS induced lung injury as it reduces LPS-induced acute lung injury and subsequent fibrosis by suppressing NLRP3 inflammasome activation. There are few concerns regarding antifibrotic in acute phase. Many covid 19 patients have hepatic dysfunction in the form of raised transaminases and both antifibrotics pirfenidone and Nintedanib cause hepatotoxicity. Nintedanib is associated with increased risk of bleeding as most of the covid 19 patients are on anticoagulant.[4]

CONCLUSION:

Considering huge numbers of individuals affected by COVID-19, even rare complications like post covid pulmonary fibrosis will have major health effects at the population level. Elderly patient who require ICU care and mechanical ventilation are the highest risk to develop lung fibrosis.

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