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POST-PANDEMIC COVID-19 LUNG EFFECTS

NAYAN KUMAR BASAK

Research Scholar • RKDF University, Ranchi

Post-Pandemic Effects of COVID-19 on the Lungs : A Review of Current Evidence

A Narrative Review of Post-Acute and Long-Term Pulmonary Sequelae of SARS-CoV-2 Infection

Abstract

The COVID-19 pandemic, caused by severe acute respiratory syndrome coronavirus 2, has left a significant proportion of survivors with long-term respiratory complications after the acute symptoms subside. This review evaluates peer-reviewed evidence published through 2026 on the post-pandemic pulmonary effects of COVID-19, including the prevalence of fibrosis or fibrotic-like changes, pulmonary function abnormalities, contributing pathophysiology, risk factors, and management options. Pulmonary fibrosis or fibrotic-like radiological changes are reported in roughly 5 to 75 percent of survivors depending on the study characteristics, with worse outcomes apparent in severe or mechanically ventilated patients. Reduced diffusing capacity for carbon monoxide (DLCO) is the most common functional abnormality, persisting in a meaningful minority of patients for up to three years after infection, while spirometric volumes tend to normalize within a shorter timeframe. Mechanistic data suggest that persistent fibrosis in the lungs is driven by dysregulated TGF-beta, WNT, and Hippo signaling, epithelial-to-mesenchymal transition, oxidative stress, and low-grade inflammation after diffuse alveolar damage. Management of post-COVID fibrosis is still largely informed by standard-of-care approaches for idiopathic pulmonary fibrosis, with a focus on pulmonary rehabilitation, selective use of antifibrotic medications, and long-term surveillance with serial CT and pulmonary function testing. There remains significant uncertainty about which patients are most likely to develop irreversible fibrosis after COVID-19, necessitating further longitudinal studies and clinical trials of targeted therapies.

1. Introduction

As of 2026, SARS-CoV-2 had infected hundreds of millions of people worldwide and caused over seven million deaths from COVID-19, but the long-term pulmonary consequences of the disease extend well beyond the acute phase of infection. A significant minority of patients who experience moderate-to-severe pneumonia or require ICU-level care continue having dyspnea, cough, and reduced exercise tolerance for months or years after their initial illness, a condition that falls under the umbrella term post-acute sequelae of SARS-CoV-2 infection (PASC) or long COVID.

The importance of understanding long-haul COVID has increased substantially as the acute pandemic phase subsides and healthcare systems prepare to manage the post-acute consequences of the disease for years to come. This review paper evaluates the current medical literature on the post-COVID-19 lung effects, with a specific focus on pulmonary fibrosis and reduced diffusion capacity as key long-term consequences. It incorporates findings from hospital-based cohort studies, methodological reviews, and mechanistic research to provide a perspective on the incidence of post-viral lung disease, associated risk factors, and opportunities for clinical intervention.

2. Methodology

This review is based on the analysis of peer-reviewed research articles and clinical studies published in scholarly journals and databases between 2020 and 2026. The search terms included variations of “post-COVID pulmonary fibrosis,” “long COVID lung function testing,” “DLCO abnormality,” “post-acute respiratory sequelae,” and “CT evaluation after COVID-19.” The relevant studies were grouped thematically based on the findings related to radiological, functional, and pathophysiological aspects of the disease, as well as risk factors and treatment options. Particular emphasis was placed on the articles reporting outcomes beyond six months from the initial infection, as they represent the post-pandemic rather than acute phase of the disease.

3. Radiological Findings: Fibrosis and Structural Lung Changes

Diffuse lung fibrosis has been identified as a common finding that occurs after COVID-19 pneumonia, although the incidence varies significantly between studies depending on the patient cohort and the timing of the post-infection CT scan. A retrospective analysis of 595 hospitalized patients revealed an overall incidence of pulmonary fibrosis of 4.37 percent, with the fibrosis developing more frequently in patients with severe disease at 8.22 percent versus 2.08 percent in those with mild COVID-19.

Other studies analyzing larger patient cohorts found the incidence of post-viral fibrotic or fibrotic-like changes in the lungs to be much higher, reaching up to 75 percent or more in some studies. A twelve-month high-resolution CT scan follow-up of non-intubated patients showed that 50 percent of patients had fibrotic-like changes in the lungs at three months, which decreased to 42 and 5 percent at six and twelve months, respectively. On the other hand, a Turkish cohort study of 93 patients with moderate-to-severe pneumonia found that over 75 percent of patients had fibrotic-like changes seen on a repeat CT scan between 1 and 10 months after the acute infection. Non-fibrotic opacities were more likely to clear over time than frank fibrosis, although some patients with severe lung damage required extended post-COVID care. A South Korean multicenter study using longitudinal CT scans in 688 hospitalized adults found that 12.6 percent of patients developed post-COVID pulmonary fibrosis on subsequent imaging after the acute infection.

The predictive factors for the development of post-COVID pulmonary fibrosis include older age, smoking history, increased CT severity score at admission, presence of consolidation on the initial chest CT scan, mechanical ventilation during the acute illness, and higher overall disease severity. The latter two factors were independently predictive of the increased risk of fibrosis after COVID-19 pneumonia. Radiologists started referring to “fibrotic-like changes” rather than fibrosis in the early post-COVID scans, as many such patients showed improvement or resolution of the abnormality within the first year after infection.

4. Functional Impairment: Diffusion Capacity and Spirometry

Reduced diffusion capacity for carbon monoxide (DLCO) is the most common functional abnormality in post-COVID lung disease, occurring more frequently than the decrease in the spirometry indices such as FEV1 or FVC. In a multicenter prospective study evaluating 145 COVID-19 patients, 41 percent of the participants had persistent symptoms 100 days after the initial diagnosis, with the reduction in DLCO being the most common abnormality in pulmonary function tests, affecting 21 percent of the patients.

Another large study analyzing the long-COVID symptoms in a RECOVER Initiative cohort of 929 patients with pulmonary manifestations found that just over 50 percent of the participants had

abnormal lung function test results with reduced DLCO and restrictive pattern (total lung capacity at or below 80 percent of the predicted value). Invasive mechanical ventilation was the most significant risk factor for the development of abnormal lung function after COVID-19, with the adjusted odds ratio of 9.9 for DLCO reduction and restrictive spirometry pattern.

A systematic review and meta-analysis evaluating the long-term pulmonary function up to three years after infection found that diffusion capacity, forced vital capacity, and ground-glass opacities generally improved over time, but more than 30 percent of patients still had abnormal DLCO values at three years with little additional improvement beyond the first year of follow-up. Fibrotic changes seen on the CT scan also showed limited resolution beyond the first year after infection. A retrospective analysis of 60 long COVID patients found that most had largely normal spirometry with FEV1 and FVC above 90 percent of the predicted values, suggesting that the reduction in DLCO is a more meaningful functional abnormality.

Some recent studies have used combined DLCO-DLNO (diffusing capacity for nitric oxide) testing to evaluate long COVID patients, suggesting that the primary site of impaired diffusion lies in the capillary blood volume rather than the alveolar membrane. Several studies reported the presence of bronchial hyperresponsiveness in long COVID patients with one large Chinese cohort study finding over 80 percent of the participants with a positive bronchial provocation test.

5. Pathophysiological Mechanisms

The mechanisms contributing to post-COVID lung fibrosis are derived from the aftermath of diffuse alveolar damage (DAD), the characteristic pathological pattern of acute lung injury seen on autopsy and biopsy series, including in the lungs removed for transplantation after COVID-19. Following the acute injury, several processes contribute to the development of fibrosis in a subset of patients with COVID-19.

5.1. Epithelial-to-Mesenchymal Transition and Fibroblast Activation

SARS-CoV-2 infection has direct cytopathic effects on type II pneumocytes, the key cell type involved in the development of pulmonary fibrosis, similar to those seen in idiopathic pulmonary fibrosis (IPF). The abnormal epithelial cells undergo epithelial-to-mesenchymal transition (EMT), changing their morphology to a mesenchymal, fibroblast-like appearance and contributing to the excessive extracellular matrix deposition in the lungs. Neutrophil extracellular traps (NETs) have also been identified as playing a role in promoting the EMT in post-COVID fibrosis.

5.2. Dysregulated Signaling Pathways

Transforming growth factor (TGF)-beta, WNT, and Hippo pathways are dysregulated in post-COVID fibrosis, contributing to the activation and proliferation of fibroblasts, collagen deposition, and myofibroblast differentiation. The toll-like receptor (TLR) signaling pathway is also involved in post-viral lung injury, promoting the release of proinflammatory cytokines and contributing to the prolonged inflammatory response after COVID-19.

5.3. Oxidative Stress and Vascular Remodeling

Oxidative stress response adds to the overall lung injury from COVID-19, and proteomics studies in patients with ARDS have identified multiple circulating proteins associated with reduced diffusion capacity. Some of these proteins are also implicated in the pathogenesis of IPF. MUC4, KRT5, and ATP12A are the candidate proteins linked to post-COVID fibrosis, and their detection in the bronchoalveolar lavage fluid can help identify patients who will benefit from antifibrotic therapy.

6. Management and Emerging Therapies

There is currently no established, COVID-specific treatment for post-viral pulmonary fibrosis or the reduction in diffusion capacity seen after COVID-19, and the management of such patients is informed mostly by the approaches used in idiopathic pulmonary fibrosis.

6.1. Pulmonary Rehabilitation

Patients with reduced DLCO and restrictive spirometry pattern appear to benefit meaningfully from pulmonary rehabilitation, with one randomized controlled trial showing that respiratory muscle training for six weeks resulted in approximately 30 percent improvement in DLCO and six-minute walk distance compared to the control group. Multidisciplinary pulmonary rehabilitation has also been shown to improve FVC, FEV1, total lung capacity, and DLCO in long COVID patients.

6.2. Antifibrotic and Anti-Inflammatory Agents

Pirfenidone and nintedanib, two antifibrotic drugs approved for the treatment of idiopathic pulmonary fibrosis, are being investigated in clinical trials for patients with post-COVID pulmonary fibrosis. These medications target the TGF-beta pathway involved in the development of fibrosis after COVID-19. Corticosteroids may also be used in patients with ongoing inflammatory activity, with several other immunosuppressive and antifibrotic agents, including sirolimus, under evaluation.

6.3. Monitoring and Longitudinal Follow Up

As many patients with fibrotic-like changes in the lungs appear to spontaneously recover within the first year after infection, current treatment guidelines recommend serial monitoring with CT scans and pulmonary function tests rather than the immediate institution of antifibrotic therapy. Such an approach is challenging in resource-limited settings, particularly in Latin America, where long COVID care is not widely available. Selective antifibrotic treatment of patients with progressive pulmonary fibrosis after COVID-19 remains an unmet clinical need.

7. Conclusion

Several years into the pandemic, COVID-19 continues to affect the lungs of the virus's survivors. Reduced diffusion capacity and, in some patients, persistent pulmonary fibrosis develop after the acute infection and are seen most often in those with severe COVID-19, mechanical ventilation, older age, and smoking history. While the majority of patients show improvement in lung function within the first two years after infection, a meaningful minority of patients continue to have abnormal pulmonary function tests at three years, indicating that post-viral lung disease should be considered a chronic condition in those who develop it after COVID-19. The field needs continued research on the natural history of the disease, as well as clinical trials of antifibrotic and rehabilitative interventions to improve the outcomes of patients with long-term pulmonary consequences of COVID-19.

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