Title: **Post COVID-19 pulmonary fibrosis**

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An 85-year-old woman with a history of hypertension presented to our hospital with worsening hypoxia. She had been diagnosed with COVID-19 more than a month prior, then requiring mechanical ventilation for acute respiratory distress syndrome secondary to COVID-19. She received Remdesivir, convalescent plasma, and dexamethasone and was eventually weaned to high flow nasal cannula oxygen (HFNC) of 40L/min. She was subsequently discharged to a long-term acute care facility. The patient had no prior history of any lung disease and was independent before her hospitalization.

On admission to our hospital, she required HFNC of 60 L/min oxygen and 80% of fractional inspired oxygen. Repeat COVID-19 testing was negative. She was afebrile and tachypneic with a respiratory rate of 30 breaths/min. She had no crackles or wheezes on lung auscultation. A chest computed tomography (CT) showed diffuse interstitial fibrotic changes with honey combing and traction bronchiectasis but no consolidation (Figure 1). Initial course of broad-spectrum antibiotics failed to decrease her oxygen requirements; she was started on prednisone 40 mg daily with a taper by 10 mg every two weeks. After three weeks, she was finally weaned to just four liters of oxygen via nasal cannula. There was slight improvement in alveolar opacities in her follow-up CT done after three weeks.

We made a diagnosis of post-COVID-19 pulmonary fibrosis – an emerging new diagnosis in the COVID era that has been described in case reports[3,4,5]. Schwensen *et. al*. described an 80-year-old woman who died from respiratory failure secondary to extensive fibrosis and traction bronchiectasis seen on CT chest about five weeks into her illness[4]. Similarly, Scelfo *et. al*. described three middle-aged patients with extensive fibrosis on CT chest more than a month into their diagnosis of COVID-19[3]. A study involving autopsies on eight patients who died from COVID-19 showed fibrotic lung parenchymal remodeling with increased fibroblasts, micro-honeycombing, and airspace destruction[2]. This lung fibrosis may be due to several different reasons. First, viral replication within cells may activate a profibrotic pathway via migration of macrophages, monocytes and neutrophils, which secrete pro-inflammatory cytokines. The result is destruction of normal lung parenchyma eventually leading to fibrosis. Also, reactive oxygen species, barotrauma caused by mechanical ventilation, and micro-clot formation in pulmonary vasculature all may contribute to the development of lung fibrosis[3,7].

Current data on treatment is limited. Some authors have postulated that antifibrotic agents such as Pirfenidone and Nintedanib may be useful. Others have suggested a possible role for steroid therapy. There are currently no long-term studies and no treatment guidelines to mitigate lung fibrosis associated with COVID-19[6]. The sole curative treatment currently is lung transplantation which can only be performed on a select group of patients in specialized centers[1]. Further studies are needed to determine a role for steroids in the acute setting, since our patient clearly benefited from it.

Learning point for clinicians

In patients who have post COVID inflammatory fibrosis, steroids may be helpful in decreasing oxygen requirements. Long term studies are needed to assess to develop treatment guidelines for this emerging diagnosis.

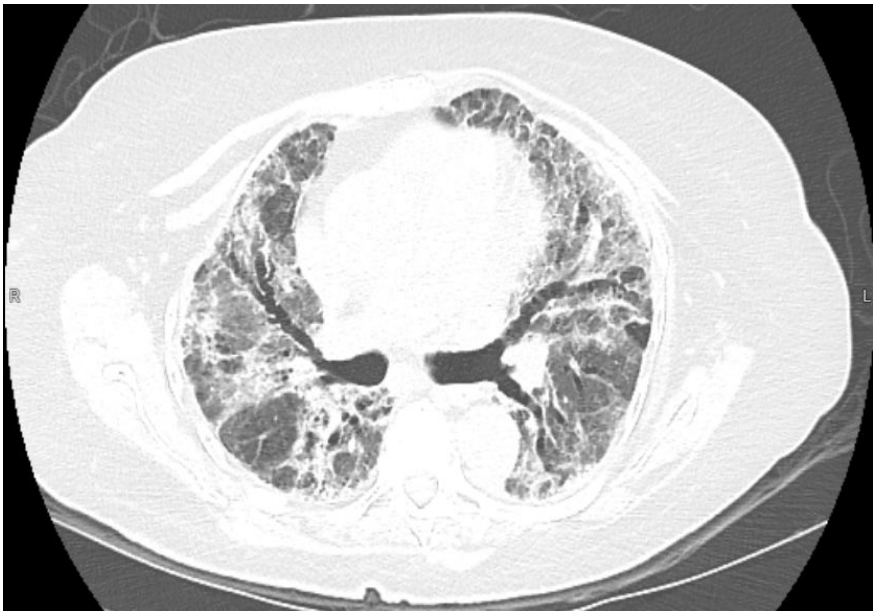

Figure 1: Extensive bronchiectasis, honeycombing and alveolar opacities on CT chest done on admission.

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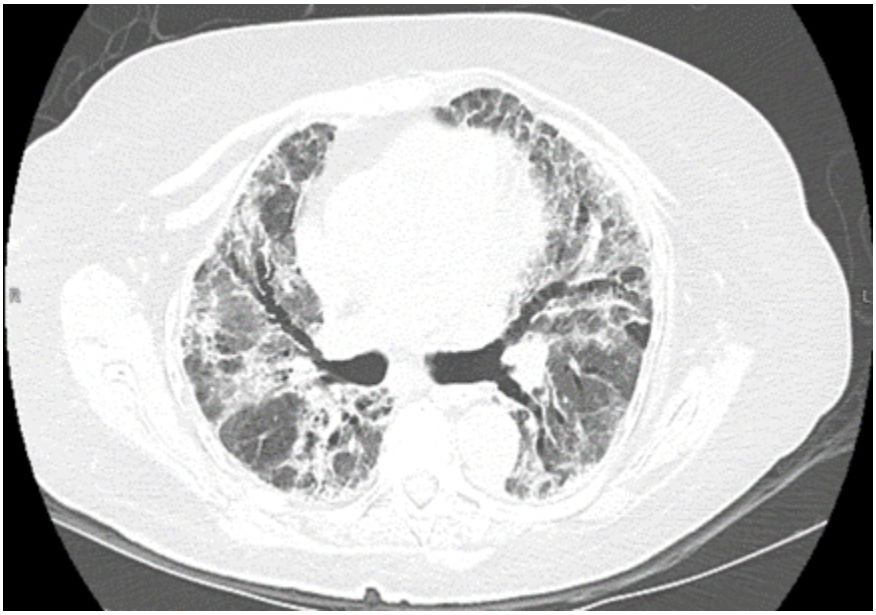

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