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Research Article

Combined Pulmonary Fibrosis and Emphysema: The Long-Term Dramatic Sequelae and another Tsunami that will Follow the Earthquake in the Sars-Cov-2 Era

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Abstract

The SARS-CoV-2 pandemic has already infected in excess of 98 million people worldwide and resulted in 2,1 million deaths. While the majority of those infected will not have long-term pulmonary sequelae, 5%-10% will develop severe COVID-19 pneumonia and Acute Respiratory Distress Syndrome (ARDS). The natural history of these severely affected patients is unclear at present, but using our knowledge of closely related coronavirus outbreaks like Severe Acute Respiratory Distress Syndrome (SARS) and Middle East Respiratory Syndrome (MERS), we would hypothesize that the majority will stabilize or improve over time although some patients will progress to advanced lung fibrosis or post-COVID interstitial Pulmonary Combined Disease (PC-ILD). Unlike the SARS and MERS outbreaks which affected only a few thousands, the sheer scale of the present pandemic suggests that physicians are likely to encounter large numbers of patients (potentially hundreds of thousands) with PC-ILD. We report a case of a patient presenting with persistent respiratory failure after recovery from COVID-19 infection with imaging showing evidence of new onset combined pulmonary fibrosis and emphysema. Nonetheless, given the huge numbers of individuals affected by COVID-19, even rare complications will have major health effects at the population level. It is important that plans are made now to rapidly identify whether the development of pulmonary fibrosis occurs in the survivor population.

Keywords: Combined pulmonary fibrosis; Sars-Cov-2

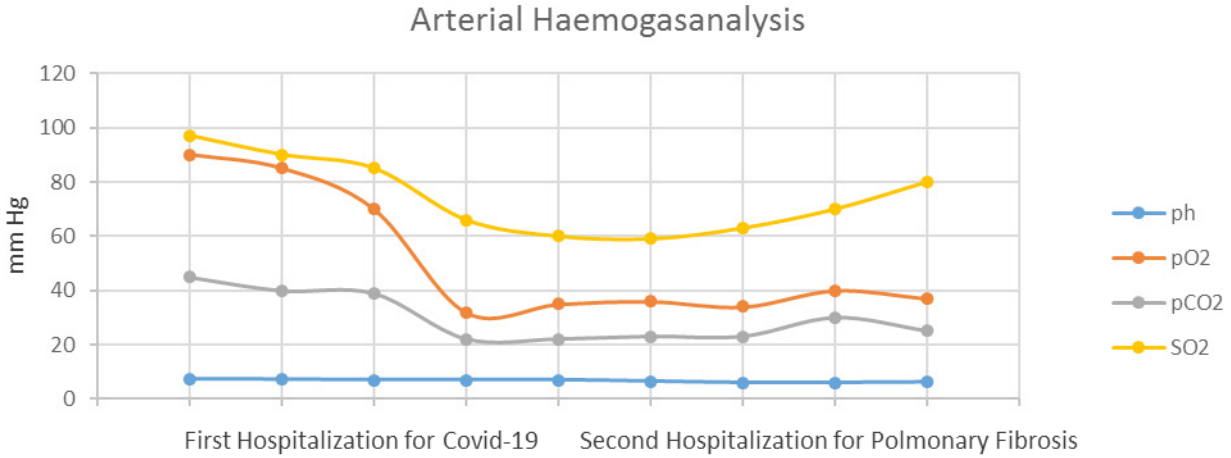
Background

The SARS-CoV-2 pandemic has already infected in excess of 98 million people worldwide and resulted in 2,1 million deaths. While the majority of those infected will not have long-term pulmonary sequelae, 5%–10% will develop severe COVID-19 pneumonia and Acute Respiratory Distress Syndrome (ARDS). The natural history of these severely affected patients is unclear at present, but using our knowledge of closely related coronavirus outbreaks like Severe Acute Respiratory Distress Syndrome (SARS) and Middle East Respiratory Syndrome (MERS), we would hypothesize that the majority will stabilize or improve over time although some patients will progress to advanced lung fibrosis or post-COVID interstitial Pulmonary Combined Disease (PC-ILD) [1]. Unlike the SARS and MERS outbreaks which affected only a few thousands, the sheer scale of the present pandemic suggests that physicians are likely to encounter large numbers of patients (potentially hundreds of thousands) with PC-ILD [2]. We report a case of a patient presenting with persistent respiratory failure after recovery from COVID-19 infection with imaging showing evidence of new onset combined pulmonary fibrosis and emphysema.

Case Description

Our patient 82 year-old, an ex-smoker Italian men with Diabetes mellitus, Hypercholesterolemia, Previous TIA. Twenty days after your recent hospitalization (November 17th), for Sars-Cov-2 infection he reports fever, progressive dyspnea since discharge, interfering with daily activities including walking to the bathroom and preparing food, cough, constitutional symptoms, acute respiratory failure . On his last visit (December,12th), he was hospitalized for 15 days for Sars-Cov-2 pneumonia.Laboratory diagnosis reveleaded pO2 36.2 mmHg, pCO2 22.3 mmHg, SO2 66.5%. Arterial Haemogasamalysis in FiO2 60% pH 7.45, pO2 53.2 mmHg, pCO2 35.8 mmHg, sO2 86.5%. Therapy undertaken with steroid, antibiotic, heparin (thromboembolic prophylaxis

dosage), aerosol with steroid and bronchodilator (anticholinergic muscarinic receptor agonist) and oxygen requirements were met with nasal cannula, with gradual improvement to 95% saturation on room air. He denied previous hospitalizations, family history, no occupational exposures and substance abuse. Vital Signs showed blood pressure 95/130 mmHg, Pulse 90 BPM, RR 22 Temperature 38 °C. Physical exam was pertinent for fine velcro-like inspiratory and expiratory crackles auscultated at lung bases. At chest x-ray showed patchy opacities diffusely worsened from previous visit. CTPE showed no pulmonary embolism, but diffuse bilateral patchy infiltrates with ground glass opacities and bronchiectasis, pulmonary emphysema, diffuse beehive lung appearance most prominent at bases compatible with a diffuse condition of fibrosis, no areas of consolidation of lung parenchyma. WBC was 8.64 [K/mm3], absolute lymphocyte count 1.73 K/. BNP, procalcitonin, lactate, autoimmune workup and Echocardiogram were normal. Inflammatory markers were elevated but decreased from last admission post-Covid. He was treated with methylprednisolone and oxygen to prevent further fibrosis in areas of active inflammation. After symptomatic improvement, he was discharged on home oxygen and steroid taper with outpatient follow up (Figure 1).



Note: This table show the variability of Arterial Haemaogasanalysis during the First Hospitalization for Sars-Cov-2 and the Second Hospitalization for Polmonary Fibrosis

Figure 1: Arterial Haemogasanalysis.

Discussion

Pulmonary fibrosis can develop either following chronic inflammation or as a primary, genetically influenced, and age-related fibroproliferative process, as in Idiopathic Pulmonary Fibrosis (IPF) [3]. In this moment, the long- term implications of COVID-19 and pulmonary consequences are uncertain and should not be assumed without appropriate prospective study. We highlight a patient who recovered from COVID-19 infection with mild ARDS yet has profound hypoxia on exertion with imaging showing severe fibrotic changes and areas of active inflammation. While severe ARDS alone has been shown to cause rapidly forming pulmonary fibrosis and emphysema, exact sequelae of mild COVID-19 respiratory failure is unknown. Possible mechanisms include proliferation of proinflammatory cytokines like IL-6 during the acute infection, which has been linked to development of fibrosis [4]. Nonetheless, given the huge numbers of individuals affected by COVID-19, even rare complications will have major health effects at the population level. It is important that plans are made now to rapidly identify whether the development of pulmonary fibrosis occurs in the survivor population.

Conclusion

We can hope to deliver appropriate clinical care and urgently design interventional trials to prevent a second wave of late mortality associated with this devastating pandemic.

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