

Impact of the COVID-19 pandemic in lung disease patients

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Editorial

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic has generated several questions about how patients with chronic obstructive pulmonary disease (COPD) should be managed and whether therapeutic adjustments are necessary. Given the closeness of symptoms, it has prompted concerns about detecting and distinguishing coronavirus illness (COVID-19) from COPD. Basic infection control measures, such as social distance, hand washing, and wearing a mask or face covering, should be followed by COPD patients. Patients should have all of their vaccines up to date, especially the annual influenza vaccine. Inhaled corticosteroids, long-acting bronchodilators, roflumilast, or chronic macrolides should be taken as needed for stable COPD therapy, notwithstanding the lack of evidence.

Patients with COPD do not appear to be at an elevated risk of SARS-CoV-2 infection, but this could be due to preventative tactics. They have a higher risk of COVID-19 hospitalization, as well as a higher risk of developing severe disease and death. COPD has also been linked to an increased risk of serious illness or death in some studies, but not all. Many factors, including past poor adherence to medicine, problems with self-management, limited availability to care during the pandemic, and a diminished pulmonary reserve, have been hypothesised to explain for the increased risk of poor outcomes.

During the COVID-19 pandemic, diagnostic and therapeutic bronchoscopy may be required in some COPD patients. Patients should wait until they get a negative PCR test before having an elective bronchoscopy. In emergency situations where the presence of COVID-19 infection is unknown, all cases should be treated as if they are positive. If a disposable bronchoscope is available, it should be utilised, and personnel should wear personal protection equipment. The dramatic shifts in world health have raised various clinical and scientific problems and concerns for the ILD community at large, especially in the context of so much ambiguity about the status of the COVID-19 pandemic. This involves a significant and disruptive impact on the conduct of ILD clinical studies, with both short- and long-term implications. While patient screening and recruitment for ongoing clinical trials has been put on hold indefinitely, the safety of patients who have been enrolled and are participating in trials is being ensured through modified protocols that include home visits by nurses and/or telemedicine to monitor clinical status.

It was most likely a successful lockdown that resulted in a relatively clean environment and the usage of masks outside on a daily basis. As a result, there

were fewer asthma outpatient visits and hospitalizations due to acute severe asthma. The same tendency was seen in COPD and ILDs. While the COVID-19 period had no effect on the outcome of COPD and ILD exacerbations, it did have a negative impact on asthmatics. Surprisingly, pulmonologists continued to use the same amount of inhaled steroids in asthma patients during the pandemic, contrary to popular belief.

Lung structural damage is caused by chronic airway inflammation and mucus clogging. PCD is a genetically diverse condition defined by ciliary dysfunction, which results in reduced mucociliary activity and infection of the upper and lower respiratory tract. PCD morbidity is linked to pulmonary damage caused by chronic suppurative airway infections, deterioration of lung function, and bronchiectasis, all of which start in childhood. ILD is a heterogeneous group of diffuse parenchymal lung disease that affects a small number of people. Children with chronic lung disorders already have disease anxiety; however, during a pandemic, it can intensify due to increased parent worry, as seen by greater frequency of discussing the coronavirus at home, higher protection pressure due to COVID-19, and parental psychological issues. Rather than hyaline membranes, the histologic pattern was acute fibrinous and organising pneumonia (AFOP), which was marked by extensive intra-alveolar fibrin deposition, referred to as fibrin. Knowing the proper pathophysiology is critical for creating the foundation for appropriate treatment, both in terms of mechanical ventilation and future follow-up and possible antifibrotic medication management in progressive morphologies [1-5].

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