Journal of Clinical Respiratory: Open Access

Impact of Coronavirus on Respiratory System

Hamza Khan*

Department of Rhinology, University of Mohaghegh Ardabili, Ardebil, Iran

Commentary

Received: 01-Aug-2022, Manuscript No. JCROA-22-66481; **Editor assigned:** 03-Aug-2022, Pre QC No. JCROA-22-66481 (PQ); **Reviewed:** 17-Aug-22, **QC No**. JCROA-22-66481;
Revised: 24-Aug-2022, **Manuscript No.** JCROA-22-66481 (R); **Published:** 31-Aug-2022, DOI: 10.4172/jclinresp.4.S1.003

***For Correspondence:**
Hamza Khan, Department of Rhinology, University of Mohaghegh Ardabili, Ardebil, Iran

E-mail: halahmed@gmail.com

ABOUT THE STUDY

The COVID-19 pandemic has culminated in an extraordinary increase in viral pneumonia patients admitted to hospitals. Patients with comorbidities, older men, black and Asian minority ethnicities, and those with comorbidities are the most adversely affected. COVID-19 is also linked to an increased risk of thromboembolism and hypercoagulability. The vast majority of patients brought to hospitals suffer respiratory failure, and while the majority may be treated on regular wards, a significant number require intensive care. Long-term symptoms of COVID-19 pneumonia are just beginning to emerge, but data from earlier coronavirus outbreaks like SARS and MERS show that some patients will develop long-term respiratory difficulties. Interstitial lung disease and pulmonary vascular disease are predicted to be the most important respiratory consequences, based on the pattern of thoracic imaging abnormalities and rising clinical experience. There is a need for a consistent pathway for the respiratory follow-up of COVID-19 patients that balances the delivery of high-quality clinical care with limited National Health Service resources. The structure for the respiratory follow-up of patients with clinicoradiological confirmation of COVID-19 pneumonia was suggested in this commentary paper.

COVID-19 enters the human body through the respiratory tract and produces systemic damage over time. The virus causes multiorgan failure and dysfunction. In 81 percent of cases, the condition is mild, while the remaining cases are severe. Respiratory failure, septic shock, and multiorgan dysfunction are reported in 15% of positive cases, with

half of these resulting in death. The lungs are the most affected organ, and the cardiovascular system follows closely behind. The kidneys and intestines are two other organs that are experiencing considerable malfunction. In this group of patients, life-saving interventions like breathing support are especially important. The researchers concentrated on the pulmonary and cardiovascular impacts of COVID-19 infection, as well as its development and therapy. Exudative and proliferative diffuse alveolar disease with capillary congestion, desquamation of pneumocytes, hyaline membrane development, interstitial edoema, hyperplasia of pneumocytes, platelet–fibrin thrombi, and interstitial infiltration by macrophages were found in part of the COVID-19 cases. Pneumocytes had viral particles in their cytoplasm. The lungs have been found to be filled with clear liquid jelly substance in recent autopsies. COVID-19's lungs have high levels of inflammatory cytokines, which cause hyaluronan-synthase-2 to develop in lung alveolar epithelial cells and fibroblasts. Although the jelly's composition has yet to be confirmed, it is most likely hyaluronan, which can absorb water up to 1000 times its molecular weight.

The creation of these pro-inflammatory cytokines and chemokines is triggered by aggressive viral replication, which causes enormous epithelial and endothelial cell death and vascular leakage. The renin–angiotensin system failure, which enhances inflammation and vascular permeability, is hypothesized to produce acute lung injury when pulmonary ACE2 function is lost. COVID-19 triggers an aggressive host inflammatory response, which is responsible for the severe cytokine storm, by causing rapid viral multiplication, cell damage, ACE2 receptor down regulation, and antibody-dependent enhancement. A powerful autoimmune onslaught is triggered by the cytokine storm, which leads to ARDS and multiple organ failure. Case fatality is caused by uncontrolled pulmonary inflammation.

Cardiac arrhythmia cases have been discovered in the COVID-19 condition, making it difficult to distinguish between myocarditis and myocardial infarction. COVID-19 individuals with acute myocarditis may experience transitory ST elevation that resolves without treatment. In a patient with acute myocarditis, an endomyocardial biopsy may reveal myocardial inflammation and viral particles within the interstitial cells of the myocardium. An electron microscope image revealed a cytopathic inflammatory cell with viral particles with conspicuous viral crown spikes. Five endomyocardial biopsies from patients with suspected myocarditis or unexplained heart failure were confirmed as SARS-CoV-2 infections by employing reverse real-time transcriptase-polymerase chain reaction to detect SARS-CoV-2 genomes. Some of the articles included echocardiogram findings from COVID-19 patients who were evaluated within 24 hours of admission, with a 42% by 2021 of patients having a normal echocardiogram at baseline. Reduced Right Ventricular (RV) function (dilatation and dysfunction) was found in 40% of patients, as was reduced Left Ventricular (LV) diastolic function in 16%, and LV systolic dysfunction in 15%. As a result, LV systolic failure was less common. Increased troponin levels in the early stages of RV dysfunction may predict a worse clinical outcome. In the absence of clinical suspicion, a regular echocardiographic testing was not advised. About 20% of patients with clinical worsening, however, echocardiographic follow-up is required.

The outbreak of COVID-19 has wreaked havoc on the healthcare system and the budget. The virus has a strong infectivity and can paralyze people who acquire severe sickness. The virus causes multiorgan damage, with the most severe effects occurring in the airway epithelium, small intestinal epithelium, and vascular endothelium, all of which have significant ACE2 expression. Lack of oxygen patients with symptoms are treated with oxygen supplementation at first, but those with severe symptoms require mechanical ventilation. There are two phenotypes of COVID-19 infection, and the breathing approach should be determined by the phenotype. When the disease

invades the cardiovascular system, it creates significant hemodynamic abnormalities. To protect health-care personnel and prevent nosocomial transmission, strict personal protective standards are required.