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E-Mail :
editor.ijasem@gmail.com
editor@ijasem.org

www.ijasem.org

Pulmonary Fibrosis Present in a Patient With a History of COVID-19: A Case Report

A.L.T. KALYANI, S.V.S.S SWETHA, P. KANCHANA

ABSTRACT--A COVID-19 pneumonia patient with a CORADS - Score of 5 (CORAD-5) who was treated with conventional procedures, oxygen support, and nasal inhalational ventilation (NIV) was unable to wean off of oxygen support. A CT pulmonary angiography was done due to concerns about pulmonary thromboembolism. Extensive pulmonary fibrosis was detected by CTPA after just 16 days.

Keywords: Noninvasive ventilation (NIV), pulmonary fibrosis (PF), pulmonary embolism (PE), and COVID-19 pneumonia

INTRODUCTION

In December of this year, news surfaced that a coronavirus never previously seen in humans was spreading across the population of Wuhan, China[1]. The severe acute respiratory syndrome coronavirus 2 (SARS-COV-2) or new coronavirus 2019(COVID-19) is an enveloped RNA virus of the family beta-coronaviridae[2]. COVID-19 has been linked to a high death rate[3] in patients with severe pneumonia and acute respiratory distress syndrome (ARDS). COVID-19, which also originates in bats like the more well-known severe acute respiratory syndrome coronavirus(SARS-COV) and middle East respiratory syndrome coronavirus(MERS-COV), has recently captured the attention of the medical community[2].

The World Health Organization declared COVID-19 a pandemic on March 11, 2020. Patients may first arrive with fever and/or respiratory symptoms; nevertheless, chest computed tomography (CT) imaging will reveal pulmonary abnormalities of

varying degrees in all patients. We reported the case of an older guy who, although having a normal chest computed tomography scan, presented with malaise, fever, and cough; yet, real-time polymerase chain reaction (RT-PCR) from a nasopharyngeal swab revealed that he was COVID-19 negative. A CORADS score of 5 (CORAD-5) on computed tomography (CT) imaging at presentation, which subsequently worsened into post-inflammatory pulmonary fibrosis.

Case Study II

A 59-year-old nonsmoking man with a history of hypertension and diabetes arrived to the outpatient clinic on August 5th, 2020 complaining of shortness of breath and other symptoms dating back to July 31st.

Heart rate was 93 beats per minute, oxygen saturation (SpO₂) was 70 percent on room air, and blood pressure was 110 over 61. A high-level care facility accepted him without delay.

Department of BIOTECHNOLOGY¹, PHARMACOLOGY^{2,3}

NRI College Of Pharmacy,

Pothavarappadu Village, AgiripalliMandal, Krishna Dist, Andhra Pradesh Pin Code:521212

To keep his oxygen saturation (SpO₂) at 92%, he needed to breathe in large amounts of oxygen (10 lpm) with a non rebreathing mask. He, too, was made to lie on his stomach. On August 5th, 2020, a chest X-ray was taken and revealed extensive homogeneous opacities, most prominently in the lower lung lobes (figure 1).

The gold standard, a covid-19 - Real time polymerase chain reaction test, was negative on a nasopharyngeal swab. On August 5, 2020, a CT scan of the patient's chest revealed a CORADS score of 5 (figure 2). He first received an empirical treatment schedule consisting of antibiotics, antivirals, and a brief course of steroids. On August 5th, 2020, however, the patient's health worsened. He was first

patient's inability to wean off oxygen assistance and many bouts of nocturnal de-saturation over the first few days of hospitalization.

prompting a recommendation. No pulmonary embolism was found, however the study did highlight

placed on nasal intubation and ventilation (NIV) in AVAPS mode, with IPAP set to a range of 16 to 14 cm of H₂O and EPAP set to 5 cm of H₂O. High flow oxygen at 15 lpm was used to keep the patient's oxygen saturation (SpO₂) at 98% after NIV was discontinued on day six of hospitalization. His oxygen saturation (SpO₂) was about 94% when he was placed on oxygen assistance and weaned off NIV. The patient's oxygen needs decreased over time after admission, but they still required 3 lpm as a minimum to be alive.

A 92% oxygen saturation (SpO₂). On the 16th day of admission, a CT pulmonary angiography was performed due to concerns about a possible pulmonary thromboembolism as a result of the

the fast emergence of severe lung fibrosis. The patient was administered anti fibrotic treatment with pirfenidone and released home on day 20 requiring 3 lpm of oxygen to maintain oxygen saturation (SpO₂) of 92%.



Figure1 Chest X-ray showing Right Lower zonedensehomogenousopacification

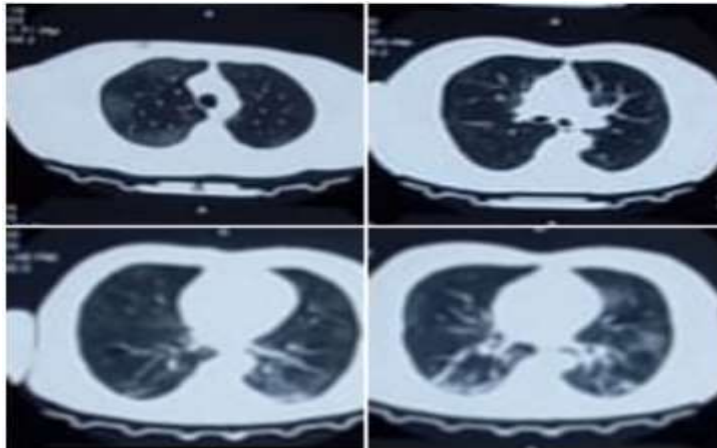
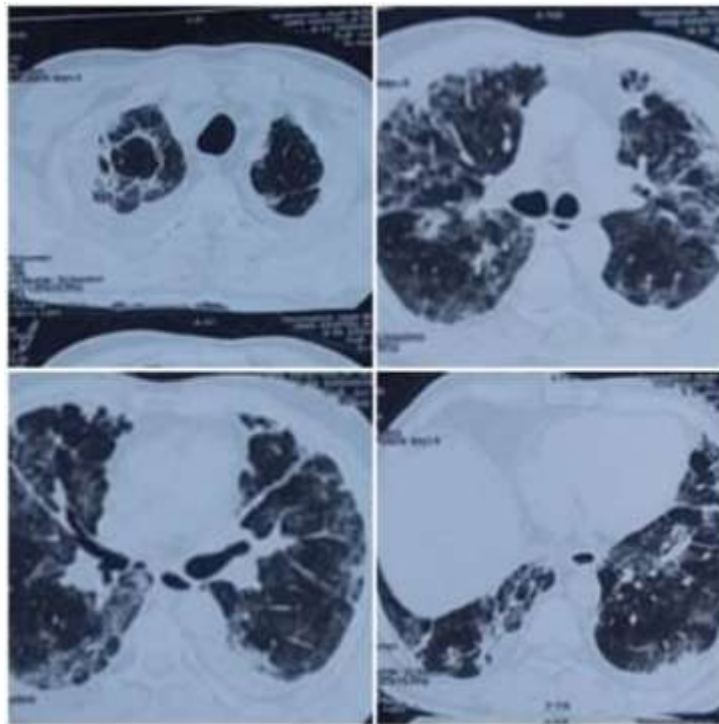


Figure2 The first CT scan 6 days after symptom onset showed Bilateral ground glass opacities predominantly involving lower lobes



Figure

3CTPAscanperformed16dayslaterprogressedintoExtensivePulmonaryfibrosisinvolvingallobesofbothlungs

I. DISCUSSION

Patients with Middle East Respiratory Syndrome (MERS) often had post-inflammatory pulmonary fibrosis after recovering from viral pneumonia; this condition was associated with a progressive decline in pulmonary function. SARS-COV-2, like SARS-COV, uses the angiotensin-converting enzyme 2 (ACE 2), a receptor present in many human organs including the lung, kidney, heart, and intestines, to infect human cells. In the Chinese research by Guan et al.[4], 173 of the 1099 patients with confirmed cases of COVID-19 had advanced illness. The average age was 52 years old, 57.8 percent were male, and 23.7 percent had been diagnosed with hypertension, 16.2 percent with diabetes, and 5.8 percent with coronary artery disease. The median age of the 67 patients who were hospitalized to critical care, needed mechanical ventilation, or died was 64 years old; 45 (67%) were male; and 39 (58%) had a co morbidity, the most frequent of which was hypertension, impacting 24 patients (36%) overall. In most cases, pulmonary fibrosis develops after the lungs have been subjected to extreme stress over an extended period of time. Lung fibrosis is caused by an abnormally high amount of extracellular matrix deposition in response to the persistent injury. Lung epithelium

Injured cells emit molecules called damage-associated molecular patterns (DAMPs) once a noxious stimulus is applied to them. This, along with microbe-associated pathogen-associated molecular patterns (PAMPs), are detected by alveolar macrophages, triggering a cascade of transduction events and the production of cytokines such as interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-). Chest x-rays will reveal ground glass opacity, consolidation, and septal thickening if air space exudates, alveolar collapse, and interstitial edema are present. Fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), transforming growth factor beta (TGF-beta), and interleukin-1 (IL-1)(11) all have a role in attracting fibroblasts to the site of alveolar damage. Collagen, fibronectin, and the ECM skeleton were all generated by fibroblasts. Myofibroblasts secrete IL-1, IL-6, IL-8, and monocyte chemoattractant protein-1 (MCP-1) during the inflammatory response, in addition to engaging in disordered production of extracellular matrix (ECM)(12). Early stage CT scans (stages 0-4) often show an evolving overline. Opaque ground glass, Crazy paving with fewer lobes involved, Stages 5-8 of a Progression Heightened Crazy Paving and Prolonged Groundglass Opacities at the

Pinnacle (10–13) Stage

Gradual advancement [2] was seen throughout the Consolidation and Absorption Stage (14 days).

There is mounting evidence that some people infected with covid-19 may develop a condition called cytokine storm syndrome, which is characterized by a severe inflammatory response brought on by the virus[3]. Patients with covid-19 at hospital discharge imply a significant incidence of fibrotic lung function problems, and further studies will confirm this finding. Fifty-one patients, or 47%, had problems with gas transfer, while twenty-seven patients, or 25%, had decreased total lung capacity[4]. Twenty-one (33.9%) of 62 patients in a study by Zhou et al. had fibrotic alterations on chest CT scans; these were more common in the late stage of the illness (emerging 8-14 days after the beginning of symptoms) than in the early stage (emerging 7 days after the onset of symptoms)[5]. Considering an anti-fibrotic intervention during the first week of ARDS onset is recommended for optimal effectiveness[6]. The range of pulmonary fibrotic illness shown in COVID-19, from fibrosis associated with organizing pneumonia to severe acute lung damage in which there is progression to extensive fibrotic change[7], provides support for the use of anti fibrotic treatment. VicorePharma has applied to conduct a phase 2 study of C21 (an agonist of AT2R) in IPF, and the application was approved by COVID-19[8] on March 30th, 2020. Additional biological justification for the use of pirfenidone in COVID-19[9] is provided by the fact that it decreases blood and lung IL-6 concentrations in mouse models of pulmonary fibrosis.

Post-COVID-19 fibrosis is anticipated to be a significant concern given the global scope of the COVID-19 pandemic and the large number of patients needing invasive ventilation. The interstitial lung disease community as a whole has to work together to learn more about COVID-19's long-term effects and come up with effective solutions to this new threat.

II. CONCLUSION

As a result of its global dissemination, SARS-COV-2 has infected millions of people. The development of pulmonary fibrosis in survivors is a known risk after human coronavirus epidemics. Fibrogenesis revolves on the virus-induced lung damage, immunological response, and repair processes. With no tried and true method of targeting

treatment for pulmonary fibrosis should focus on reducing the severity of the disease and preserving the lungs against accidental injury.

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