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Case Report

Post-COVID Pulmonary Fibrosis - A Case Report

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Abstract

Introduction: Pandemic of COVID-19 is induced by serious acute respiratory syndrome coronavirus 2 (SARSCoV-2), a devastating on going global pandemic. Its disease brutality ranges from asymptomatic, mild to severe disease, which can be intricated by promoting hypoxemic respiratory failure and Acute Respiratory Distress Syndrome (ARDS). Pulmonary fibrosis is condition that occurs due to scarring of lung tissue.

Case Presentation: A 57 years old male patient presented with fever, head ache, malaise, shortness of breath and cough productive of blood tingled sputum of 0, 1 week duration. 5 days back he was seen at private clinic after he presented with the above complaints. He was given IV antibiotic at private clinic for 5 days but no improvement. Later, he developed shortness of breath and start to desaturate then referred to this Hospital for better investigation and management.

Conclusion: Due to serious inflammatory response/cytokine storm (post-COVID fibrotic ARDS) COVID-19 pneumonia with diffuse bilateral infiltrates can rapidly advances into pulmonary fibrosis which made of our patient oxygen dependent with important rebate in quality of patient life.

Keywords: *COVID-19; Pulmonary Fibrosis; Acute Respiratory Distress Syndrome (ARDS)*

Introduction

Pandemic of COVID-19 is induced by serious acute respiratory syndrome coronavirus 2 (SARSCoV-2), a calamitous on-going universal pandemic. Its disease brutality ranges from asymptomatic, mild to severe disease, which can be intricated by promoting hypoxemic respiratory failure and Acute Respiratory Distress Syndrome (ARDS). Due to the scarring of lung tissue pulmonary fibrosis condition occurs. Most usual cause being idiopathic existing over a long duration of time, due to activation of fibroblasts also it can occur for several days after serious bacterial pneumonias. Our case shows that Covid-19 infection can acutely cause pulmonary fibrosis [1].

COVID-19 disease is induced by a novel coronavirus, known as SARS-CoV-2. The Universal pandemic began in the country China in Wuhan, in December 2019 and it has since then spread whole world [2].

The clinical demonstrations simulated viral pneumonia which escorted by serious symptoms of dysgeusia, including persistent fever, cough, dyspnoea and fatigue, respiratory illnesses. It has been reported as universal pandemic by World Health Organization and named as COVID-19. COVID-19 has symptoms more severe than the Severe Acute Respiratory Syndrome (SARS) or the Middle East Respiratory Syndrome (MERS) [3].

Coronavirus disease 2019 (COVID-19) has now led to more than a million deaths across the world. These deaths are usually due to serious pulmonary involvement. In fatal cases, features of diffuse alveolar damage are a frequent finding [4].

COVID-19 and its pathogenesis: The SARS-COV 2 belongs to the family coronaviridae of the order nidovirales. The approximate length of virus genome is 29.9 kb and 80 to 120 nm diameter. Corona virus has increased transmissibility and great affinity to enter into the

host cells and it exploits a highly glycosylated heteromeric protein. It has a Receptor Binding Domain (RBD) in its spike can sticks with the ACE2 receptor of the host cell. It is negotiated by the Transmembrane Protease Serine 2 (TMPRSS2) cathepsin. ACE2 receptors are broadly expressed in the endothelial cells of tracheal, bronchial cells, alveolar, monocytes, and macrophages of the immune system. The virus reproduces its genome, mobilizes and releases huge number of viral particles [3].

During the inflammatory phase of adult respiratory distress syndrome (ARDS) the pathogenesis of post-infective pulmonary fibrosis incorporates dysregulated release of matrix metalloproteinases which is leads to epithelial and endothelial injury with unchecked fibroproliferation. Most patients with acute respiratory distress syndrome (ARDS) or severe pneumonia were found to have pulmonary fibrosis during follow-up after the cure of COVID-19. Clinical, radiographic, and autopsy reports of pulmonary fibrosis were commonplace following SARS and MERS, and current indication recommends pulmonary fibrosis could convolute infection by SARS-CoV-2. Pulmonary fibrosis is also a known sequela of serious and/or persistent damage to the lung from other causes such as chronic granulomatous diseases, respiratory infections, medications and connective tissue disorders [5].

Case Presentation

A 57 years old male patient presented with fever, head ache, malaise, shortness of breath and cough productive of blood tingled sputum of 0, 1 week duration. 5 days back he was seen at private clinic after he presented with the above complaints'. He was given IV antibiotic at private clinic for 5 days but no improvement. Later, he developed shortness of breath and start to desaturate then referred to our Hospital for better management.

He was not hypertensive and diabetic previously but during the current illness his serum glucose and blood pressure are high.

After he come to the hospital, he was tested for COVID-19 and the report was positive.

Then he was admitted to Dilla university COVID-19 treatment centre.

Physical finings at admission COVID-19 treatment centre are:

- PE.
- GA: Acutely sick looking with respiratory distress.
- RR: 48 breaths/min, BP: 140/100 mmHg, PR: 124 beats/min, T: 38.6C and SPO_2: 85% with atmospheric air.
- Chest: Coarse crepitation over the posterior lower 2/3 the lung fields bilaterally and subcostal retraction.
- The other systems are normal.

Investigation:

- CBC, FBS, CXR and OFT
- ECG-Sinus tachycardia
- CBC
- WBC: 11.9 X 10^3

- Hct: 42.8%
- Plt: 313 X 10^6
- RBS: 103 mg/dl
- Liver function test and renal function test are within the normal range
- CXR: There is homogenous opacity over the posterior lower lung fields bilaterally.

Post contrast CT of the chest

Findings: There are bilateral patchy ill-defined ground glass opacities and consolidations with areas of dilated air ways likely suggesting traction bronchiectasis predominantly involving bilateral lower and basal, and peripheral lungs with lesser involvement of the upper lungs. There are also multiple irregular linear interstitial thickenings forming multiple reticular densities in both lungs which could represent interstitial fibrotic changes. There is no obvious sub pleural sparing. No honey combing.

The central trachea, the right and left main stem bronchus and the lobar and segmental branches are patent.

There is no pleural or pericardial fluid collection. No hilar or mediastinal LAP.

All cardiac chambers have normal size and morphology and the mediastinal great vessels (ascending aorta, aortic arch and its major branches, descending aorta, pulmonary arteries, SVC and brachiocephalic veins) have normal CT appearance.

The osseous structures appear normal. The included abdominal organs are unremarkable.

Management

He was admitted to Dilla university COVID-19 treatment centre and put on oxygen with face mask 10 l/min, ceftriaxone 1 gram IV BID for 07 days, antipyretics, low molecular weight heparin, Azithromycin 500 mg PO/daily for 3 days, dexamethasone 6 mg IV/ day for 10 days and other supportive cares. After one month of hospital stay, he was symptomatically improved but continued to have hypoxemia and discharged with appointment, 2 months after COVID-19 pneumonia he presented again with a new onset of shortness of breath and chest CT was done and showed ground glass opacity over the lower lung lobes bilateral. For which the patient was readmitted to the hospital and was on oxygen and later he developed HAP and treated for 10 days with ceftazidime and vancomycin.

Discussion

Pulmonary fibrosis leads to lung scarring. Fibrotic tissue is dead tissue, no gas exchange happens and there is a restrictive pattern on Pulmonary function tests (PFTs). There are many different conditions that can persuade pulmonary fibrosis, but it is not usual to develop post viral infections. Classic COVID-19 pneumonia induces diffuse bilateral infiltrates causing serious inflammatory response which is leading to macrophage activation, cytokine release with activation and proliferation of fibroblasts inducing tissue annihilation and scarring.

Conclusion

Covid-19 pneumonia with diffuse bilateral infiltrates can rapidly develops into pulmonary fibrosis due to serious inflammatory response/cytokine storm (post-COVID fibrotic ARDS), which made our patient oxygen dependent with important reduction in quality of life.

Ethics Approval and Consent to Participate

Not Applicable.

Consent to Publication

Informed consent was obtained from the patient to publish the case details.

Availability of Data Materials

Not Applicable.

Competing Interests

The authors declared no potential conflicts of interests with respect to the research, authorship and publication of the article.

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Authors' Contributions

BM: Conceptualization, and Validation: Project administration, Visualization, Investigation: GS; Writing-review and editing.

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