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Clinical picture

CLINICAL PICTURE

Post-COVID-19 pneumonia pulmonary fibrosis

Case

A 48-year-old male patient with no prior co-morbidities presented to the emergency department with complaints of dry cough, fever and throat pain for the preceding 5 days followed by shortness of breath for the past 2 days. He had no significant travel or contact history. His vitals were stable except for mild hypoxemia on room air (SpO_2 of 92%). On laboratory investigation he had lymphopenia, elevated Lactate dehydrogenase (LDH), raised C-reactive protein and elevated D-dimer levels. Initial chest radiograph revealed peripheral multiple inhomogeneous opacities in bilateral lung fields. His reverse transcriptase-polymerase chain reaction nasal and oropharyngeal swab for COVID-19 came out be positive and he was managed conservatively with supplemental oxygen, low molecular weight heparin, dexamethasone and antipyretics. He was symptomatically improved but continued to have hypoxemia even after 3 weeks of treatment, so a high resolution computed tomography of the chest was performed and it showed architectural distortion, interlobar septal thickening and traction bronchiectasis features (shown in Figure 1A and B) suggestive of fibrotic lung disease. He was discharged on home oxygen therapy and planned to enroll him in antifibrotic therapy trail during the subsequent follow-up.

Discussion

Clinical manifestations of Corona virus disease-2019 (COVID-19) have ranged from asymptomatic/mild symptoms to severe illness and mortality.[1] Most of the mild and moderate cases are recovered completely but a small proportion of severe cases with acute respiratory distress syndrome continued to remain hypoxemic despite adequate treatment. Chest imaging of this subset of patients revealed fibrotic changes in the form of traction bronchiectasis, architectural distortion and septal thickening similar to the changes seen in other fibrotic lung diseases.[2] The pathogenesis of post-infective pulmonary fibrosis includes dysregulated release of matrix metalloproteinases during the inflammatory phase of adult respiratory distress syndrome (ARDS) causing epithelial and endothelial injury with unchecked fibroproliferation. There is also a vascular dysfunction which is a key component of the switch from ARDS to fibrosis, with vascular endothelial growth factor and cytokines such as interleukin-6 and tumor necrosis factor alpha being implicated.[3] Although the role of presently available antifibrotic drugs (pirfenidone and nintedanib) for fibrotic lung diseases beyond idiopathic pulmonary fibrosis have been evaluated by some authors[4] their role in post-COVID-19 pneumonia pulmonary fibrosis need further research in the present pandemic.

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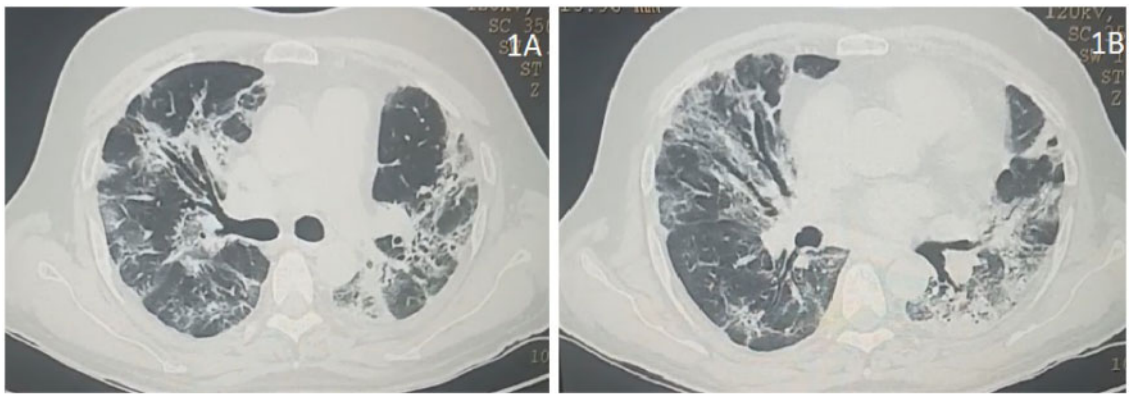

Figure 1. (**A** and **B**) Traction bronchiectasis, architectural distortion and interlobar septal thickening suggestive of pulmonary fibrosis.

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