Post-COVID-19 pulmonary cavitation and tension pneumothorax in a non-ventilated patient

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Abstract

Cavitation and pneumothorax are independently associated with high morbidity and mortality in coronavirus disease-2019 (COVID-19). While spontaneous (non-traumatic) pneumothorax formation has commonly been observed among mechanically ventilated COVID-19 patients, there are few rare reports of COVID-19 associated pneumothorax without any history of barotrauma and other conventional risk factors. Here, we report a unique case of post-COVID-19 cavitation and tension pneumothorax which was further complicated by hydropneumothorax formation in a young patient who suffered severe COVID-19 pneumonia 4 weeks back. As the patient was devoid of any conventional risk factors, we believe that persistent inflammatory alveolar damage even after clinical recovery from COVID-19 played a key role in pulmonary cavitation followed by pneumothorax formation. With prompt clinical and radiological recognition of these fatal, yet treatable complications of COVID-19 pneumonia, the patient was saved and had an uneventful recovery.

Keywords: COVID-19, hydropneumothorax, lung cavitation, pneumothorax, post-covid complications

Introduction

Pneumothorax is the accumulation of air within the pleural space and can be classified into two types: traumatic and spontaneous (primary or secondary). Secondary spontaneous pneumothorax (SSP) develops in association with an already diseased lung; whereas primary spontaneous pneumothorax (PSP) happens in patients without a history of associated pulmonary pathologies. While coronavirus disease-2019 (COVID-19) can result in a plethora of pulmonary and non-pulmonary complications, pneumothorax and cavity formation are relatively uncommon, yet carry fatal outcomes. Two retrospective analyses showed the occurrence of pneumothorax in 1–2% of COVID-19 patients who almost invariably required hospitalization and management in the intensive care unit. The patients who required positive pressure ventilation (PPV) showed a relatively higher incidence of spontaneous pneumothorax, as high as 15%. Multiple cases of PSP have also been reported in non-intubated patients and/or patients with no prior lung diseases and even as an initial presentation of worsening lung function due to COVID-19 pneumonia, albeit the underlying mechanism and impact of this association remain uncertain. However, tension pneumothorax is an extremely rare but life-threatening complication of COVID-19 pneumonia and only a handful of cases have been reported to date. Herein, we report a rare case of severe post-COVID-19 pulmonary cavitation and tension PSP which was further complicated by hydropneumothorax formation in a patient without any associated lung disease or prior exposure to PPV.

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Case Report

A 28-year-old (non-vaccinated against COVID-19), non-smoker, overweight male without any other comorbidity presented to the emergency with complaints of cough and chest pain for 3 days and sudden onset shortness of breath (SOB) for 1 day. He was admitted to the same institute 6 weeks back with the diagnosis of severe COVID-19 pneumonia [COVID-19 Reporting and Data System (CORADS)-6, computed tomography severity score (CTSS)- 20/25] [Figure 1a]. He was treated as per standard national guideline, required no ventilator support, and discharged 4 weeks back with a stable hemodynamic state and SpO₂ >94% for 3 days without oxygen support. He was doing well at home for 4 weeks until he developed sudden onset of SOB while taking inhalational budesonide (prescribed on discharge).

On physical examination, he was conscious, alert, oriented, restless, and dyspneic. His SpO₂ was 56% (room air), BP 110/70 mm Hg, and pulse 158 beats/min. Breathing sound was diminished on the left side. On percussion, there was hyper-resonant note on the left side of the chest as compared to the right side. Examination of other systems did not show any abnormality clinically. About 100% oxygen supply and nebulization with bronchodilators and inhalational steroids were started. Urgent chest X-ray (CXR) was done and revealed tension pneumothorax on the left side [Figure 1b]. Water-sealed intercostal chest drainage (ICD) was performed on an emergency basis and thereafter patient’s symptoms, clinical parameters improved markedly (SpO₂ - 98% with 2 L/min O₂) with a radiological resolution of pneumothorax [Figure 1c].

High-resolution computed tomography (HRCT) scan of chest depicted predominantly peripheral ground glass opacities with consolidative lesions and subpleural fibrotic changes in both lungs, a cavitary lesion (with air-fluid level within) in left lower lobe and left-sided hydropneumothorax. His initial CTSS during first-time hospitalization was 25/25 [Figure 1d]. He was treated as per standard national guideline, considering it to be a late sequel. His rapidly deteriorating SpO₂ level compelled us to go for immediate ICD insertion as a working diagnosis of tension pneumothorax had been made by the CXR findings. HRCT, which was performed three days after ICD insertion, showed a thick-walled cavitary lesion (4 cm × 2.3 cm × 5.4 cm) with air-fluid level within in the left lower lobe and loculated hydropneumothorax in left lung with chest tube in situ of 98–100% on room air for 3 days before discharge with further planning to referral to a higher centre for consideration of lung transplantation.

Discussion

Pneumothorax can occur in any phase and at any point during the course of illness (as long as after several weeks of apparent recovery) irrespective of the severity of the COVID-19 infection. Pneumothorax occurred in our patient 4 weeks later (from the time of discharge during the initial attack of COVID-19), considering it to be a late sequel. His rapidly deteriorating SpO₂ level compelled us to go for immediate ICD insertion as a working diagnosis of tension pneumothorax had been made by the CXR findings. HRCT, which was performed three days after ICD insertion, showed a thick-walled cavitary lesion in the lower lobe of the left lung as well as left-sided hydropneumothorax. His initial CTSS during first-time hospitalization was 20/25.
admission was 20/25 suggestive of severe COVID-19, and this time CTSS was 25/25 implying further extensive progression of the disease even after discharge. The cavity may be attributed to post-COVID changes as there was no evidence of any cavity in the previous HRCT scan and the possibility of co-existent mycobacterial, fungal, and autoimmune diseases were ruled out by appropriate tests. Post-COVID cavitation may also be due to direct alveolar damage by excess cytokine release and as a result of infarct caused by microthrombi formation.[9,10] Although unlikely from the clinical point of view, the coexistent pulmonary thromboembolism could not be ruled out in our patient due to the non-availability of CT pulmonary angiography in this set-up.[9]

The presence of subpleural bulla/mini-blebs and pneumatocele has been most popularly discussed as the cause of PSP in general. Patients with a history of smoking, prolonged cough, pre-existing lung pathology, connective tissue disorder, tall stature, and male gender, in particular, are at risk.[9,10] Pneumothorax can rarely occur as a complication of acute respiratory distress syndrome (ARDS) too with the possible mechanism being alveolar rupture due to barotrauma or pressure-overload following induced ventilation.[12] Rarely spontaneous tension pneumothorax can occur in association with COVID-19 without any history of prior PPV.[9,14] Some researchers suggested that chronic cystic changes in the lung may be caused by a spectrum of severe COVID-19 itself, which later on may lead to the development of pneumothorax after getting ruptured by the greater force of cough due to greater lung damage.[9,19] Zoumot et al.[9] also noted severe morbidity and mortality in patients presented with post-COVID-19 lung cavitation irrespective of the presence of pneumothorax; they have also hypothesized that cavitary lesions can heighten the risk of pneumothorax by extending themselves to the pleura and rupture of the cavity wall. Surprisingly, our patient had no known risk factor except being male and no obvious subpleural bulla/bleb or cystic changes were evident on HRCT chest at any point during the whole course of his illness. Therefore, the development of spontaneous pneumothorax, in this case, was most likely due to structural lung damages resultant of COVID-19-induced inflammatory injury (as evidenced by persistent high serum concentration of interleukin-6) and rupture of the cavity. Development of hydropneumothorax (which was not evident in CXR, but later unfurled in HRCT) was probably the result of ICD insertion.[19]

**Conclusion**

Although ARDS appears to be the prime suspect as a cause of rapid deterioration of SpO₂ in COVID-19, recognition of uncommon, fatal but potentially curable etiology like tension pneumothorax is crucial for the management of such cases. Misdiagnosis can lead to protocol-based initiation of PPV and the outcome may be detrimental with underlying unrecognized pneumothoraces.[11] Cavitation, primary spontaneous tension pneumothorax complicated by hydropneumothorax in a post-COVID patient without any history of mechanical ventilation, and other conventional risk factors made this index case unique. Moreover, our patient was saved by prompt recognition of the condition by astute physical examination and immediate radiography and appropriate multidisciplinary management in time even in a resource-constrained setup.

**Informed consent**

Written consent has been taken for using the medical data of the patient for academic purposes while maintaining full anonymity.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

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