Iatrogenic Pneumothorax and Pneumomediastinum in a Patient with COVID-19

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ABSTRACT

Co-occurrence of pneumothorax and pneumomediastinum is rare in COVID-19 patients. Positive airway pressure therapy used to improve oxygenation may sometimes worsen clinical outcomes in some patients with severe COVID-19 pneumonia. In this case report, we describe an individual who was diagnosed with COVID-19 and developed bilateral pneumothorax and pneumomediastinum after initiating non-invasive positive airway pressure therapy.

KEYWORDS: COVID-19, SARS-CoV-2, pneumothorax, pneumomediastinum, BiPaP

CASE REPORT

A 77-year-old male with a past medical history of coronary artery disease, paroxysmal atrial fibrillation on lifelong anticoagulation, cerebrovascular disease with minimal residual deficits, and recent hospitalization for knee replacement presented to the hospital for evaluation on account of shortness of breath. He also reported dysgeusia and subjective fever. He denied having chest pain, nausea, vomiting, odynophagia, dysphagia, palpitations or any recent chest trauma. He denied any history of tobacco use. His home medications were aspirin, atorvastatin, pantoprazole, apixaban and tramadol.

Physical exam on admission revealed bilaterally diminished breath sounds in addition to being tachycardic, tachypneic and hypoxic. He had elevated inflammatory markers including an LDH of 582IU/L (100-220 IU/L), ferritin of 2721ng/ml (22-322 ng/ml), and CRP of 206mg/L (0-10 mg/L). D dimer was also elevated at 1,332ng/ml (0-300 ng/ml). Respiratory viral panel came back positive for SARS-CoV-2. X-ray imaging revealed multifocal airspace opacities suggestive of COVID-19 pneumonia (**Figure 1**). CT scan of the chest with contrast revealed diffuse bilateral groundglass airspace disease. There was no evidence of pulmonary embolism. He was placed on 10 L /min of oxygen through high flow nasal cannula. He received Dexamethasone, Remdesivir and was enrolled in a Monoclonal Antibody study. In view of concern for presumed bacterial infection, he was also started on ceftriaxone and azithromycin. He was also given bronchodilators, incentive spirometer

Figure 1. X-Ray imaging of the chest showing multifocal airspace opacities.

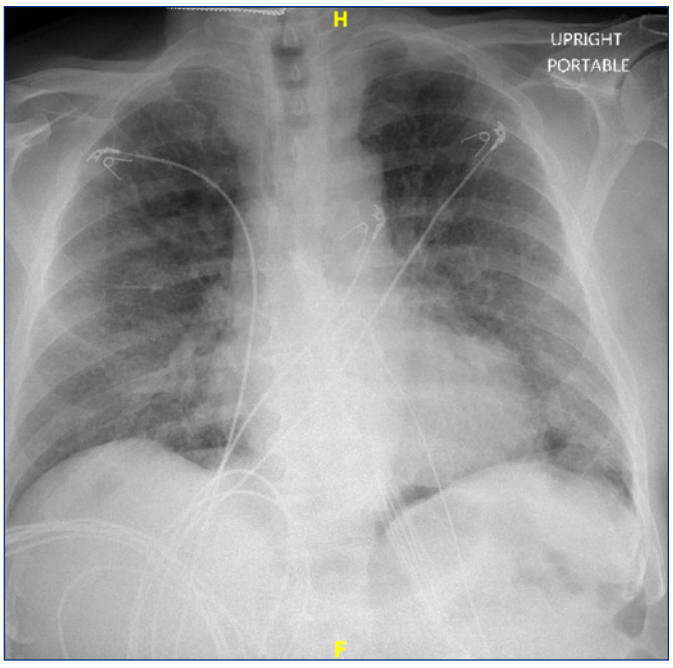
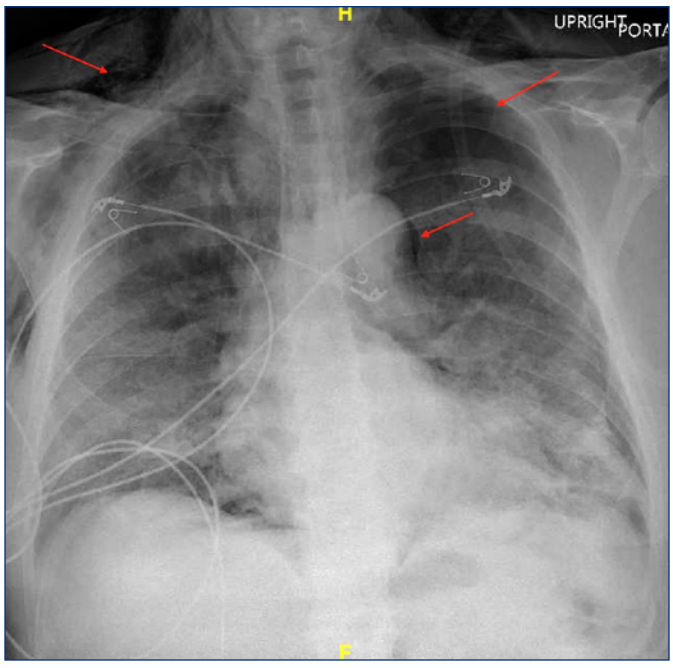


Figure 2. X-Ray imaging of the chest showing new bilateral left greater than right pneumothoraces, pneumomediastinum with subcutaneous emphysema extending to the soft tissue of the neck.



and encouraged to prone as per the institutional protocol.

On day 2, his oxygen requirements continued to worsen and he was placed on 50 L/min of 100% FiO2 through Neptune device. His CRP and d dimer increased to 211.97mg/L and 5,536/ml. He was started on a therapeutic dose of enoxaparin in place of apixaban. Given concern for concurrent pulmonary edema, he received 3 doses of furosemide. He was started on Bilevel positive airway pressure ventilation (BiPap) with inspiratory IPAP of 10cm H20 and expiratory EPAP of 8cm H20 at night-time. On day 3, he started complaining of acute, severe, bandlike chest pain along with worsening shortness of breath. Physical exam was significant for crepitus in his neck. He was noted to be saturating 88% on Bipap. X-ray imaging revealed new bilateral left greater than right pneumothoraces, pneumomediastinum with subcutaneous emphysema extending to the soft tissue of the neck (**Figure 2**). D dimer was noted to be 10, 840ng/ml. Bipap was discontinued and patient was placed back on 60 L/min of 100% FiO2 via high flow nasal cannula (HFNC). Surgical service was consulted, and he had thorocostomy and bilateral chest tube placement.

In view of persistent hypoxia despite maximum non-invasive ventilatory therapy, goals of care conversation were held with the patient and his family. The patient did not want further escalation of care and was transitioned to inpatient hospice in line with his goals of care. He died the following day.

DISCUSSION

Pneumothorax, defined as the presence of air in the pleural space, is a potentially lethal condition, and is associated with increased morbidity and mortality. Pneumothorax can be categorized as primary, secondary, spontaneous, traumatic or iatrogenic in etiology. Development of pneumothorax is most closely associated with pre-existing lung conditions and presence of bullae or pneumatoceles. The risk factors for this include smoking, male gender and prolonged coughing. Spontaneous pneumothorax and pneumomediastinum have been described previously in COVID-19 patients.[1-5]

Positive airway pressure therapy has been previously associated with spontaneous pneumothorax.[6] The exact incidence is unknown but has been reported in patients with COPD, cystic fibrosis, neuromuscular diseases, Pneumocystis jiroveci pneumonia, SARS and MERS infections.[7, 8] There was one previous report of a COVID-19 patient with new mediastinal emphysema, bulla, and pneumothorax after being started on HFNC. The authors speculated that the mediastinal emphysema and pneumothorax were likely related to COVID-19 pneumonia than HFNC itself.[9] Our patient did not have any pre-existing lung conditions, history of tobacco use or presence of bullae or pneumatoceles on lung CT that would have facilitated occurrence of pneumothorax or pneumomediastinum.

Postulated mechanisms of pneumothorax and pneumomediastinum in COVID-19 include inflammation leading to pneumatocele formation and rupture and also subpleural necrosis leading to bronchopleural fistulas. Increased alveolar pressure and diffuse alveolar injury along with low compliance also make the alveoli more prone to rupture, causing free air to track along pulmonary vessels and the interstitium to hilum. Corticosteroids may also play a role in spontaneous pneumothorax by delaying healing and perpetuating air leakage.[8]

Co-occurrence of pneumothorax and pneumomediastinum is rare in COVID-19 patients. Non-invasive positive airway pressure therapy used to improve oxygenation may worsen clinical outcomes in some patients with severe COVID-19 pneumonia similar to ventilator-induced lung injury. Prompt recognition of early signs and symptoms of pneumothorax and pneumomediastinum in COVID-19 patients is necessary to minimize morbidity and mortality. The use of minimum amounts of positive airway pressure ventilation to achieve acceptable tissue oxygenation may reduce the incidence of this complication.

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