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Case Description

Case 1
A 45-year-old male with chronic kidney disease had a fever, myalgia, and mild cough for 2 days. COVID-19 was confirmed by a rapid antigen test. He had no complaints of breathlessness, chest pain, or vomiting. Following the fever, he developed worsening hypoxia and respiratory distress in the 2nd week of illness, for which he required oxygen through a nonrebreather mask. He was started on corticosteroids, heparin, and antibiotics. High-resolution computed tomography (HRCT) of the thorax showed severe COVID-19 pneumonia and normal mediastinum. He never received positive pressure ventilation, and he was transferred out of the COVID-19 care facility on day 31. On reception in the medical intensive care unit, his respiratory rate was 32 breaths/minute and oxygen saturation (SpO₂) was 96% on a nonrebreather mask at 12 L/minute of oxygen. Hamman’s crunch was absent. Normal vesicular breath sounds were heard bilaterally. Hemogram showed leucocytosis with a total count of 22,800 cells/µL—neutrophils 86% and lymphocytes 9%. His serum urea was 167 mg/dL, and serum creatinine—2.98 mg/dL. His arterial oxygen pressure/fraction of inspired oxygen ratio was 79, suggesting severe acute respiratory distress syndrome (ARDS). HRCT of the thorax (on day 31) showed pneumomediastinum extending from the thoracic inlet to the diaphragm (Fig. 1A). We managed him conservatively for isolated pneumomediastinum. Repeat HRCT of the thorax on day 36 showed near-total resolution of pneumomediastinum (Fig. 1B). His oxygen requirement was gradually tapered and he was discharged on room air after 42 days of hospitalization.

Case 2
A 34-year-old male without comorbidities presented with fever, headache, cough, and breathlessness for 5 days. Reverse transcription polymerase chain reaction assay for COVID-19 was positive. At presentation, his SpO₂ was 95% at 10 L/minute of oxygen and respiratory rate at 28 breaths/minute. Systemic examination was normal. He was started on enoxaparin, corticosteroids, remdesivir, and antibiotics. His hemoglobin was 13.3 mg/dL, and total leucocyte count at 16,040 cells/µL—neutrophils 94% and lymphocytes 4%. C-reactive protein was 5.4 mg/dL and serum ferritin was at 1226.3 ng/mL. On day 10 of symptoms patient developed sudden onset of swelling over the right side of the neck extending to the right side of the chest and right upper limb. Examination revealed palpable crepitus below the skin, suggestive of subcutaneous emphysema (SCE). He also had worsening hypoxia, tachypnea, and increasing oxygen requirements. HRCT of the thorax revealed moderate pneumomediastinum, gross SCE and minimal PTX in the right apical area (Fig. 2). Noninvasive ventilation was initiated. Since the size of the PTX was increasing, we placed an intercostal tube. Subsequently, he required intubation and mechanical ventilation. He later expired on day 22 of illness.

This report highlights two unusual complications of COVID-19 pneumonia—SPM and spontaneous PTX. Mechanisms of

Fig. 1A and B: (A) HRCT-thorax showing pneumomediastinum (black arrow). Bilateral lung parenchyma showed peripheral and central ground-glass opacities, with a CT severity score of 24/25; (B) axial cross-section of HRCT-thorax showing only two tiny air foci (two black arrows) remaining in the anterior mediastinum.
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pneumomediastinum remain unclear. The pressure gradient that normally exists between the marginal alveoli and lung interstitium, when suddenly elevated as in case of violent bouts of cough, may rupture alveoli. The leaked air initially gets collected in the perivascular spaces and causes the condition called pulmonary interstitial emphysema, which is commonly missed in chest X-ray. With further leakage, air tracks along the bronchovascular bundles into the hilum of the lung and eventually into the mediastinum, giving rise to pneumomediastinum. This phenomenon is known as the "Macklin effect." It may progress to PTX and SCE if air continues to leak down the low-pressure gradient.

The incidence of PTX among patients admitted with COVID-19 has been estimated to be around 1%. A systematic review on COVID-19-associated air leak syndromes (like PTX, pneumomediastinum, and SCE) showed that men were affected more (75.2%), with a mean age of 58 years. The most common was isolated PTX (48.5%), among whom 17.65% had a spontaneous PTX. The mean time to the occurrence was 11.6 days. The mortality rate can be as high as 40% in patients with air leak syndrome.

Spontaneous pneumomediastinum (SPM) is usually a self-limiting disease requiring only supportive treatment with analgesia, oxygen, and adequate rest. However, malignant forms may require decompression with thoracotomy. In patients with ARDS and persistent cough, dextromethorphan may help to avoid these complications by cough suppression.

To conclude, pneumomediastinum, PTX, and SCE can be spontaneous complications seen in COVID-19 patients. COVID-19 causes severe diffuse alveolar damage, leading to alveolar rupture, thereby causing air leak syndrome. It is a harbinger of worsening respiratory failure and heralds a poorer prognosis for the patients. As a potential indicator of worsening disease, COVID-19 patients with SPM needs close monitoring.

References


Fig. 2: HRCT-thorax showing moderate pneumomediastinum (black arrow), gross SCE involving the lower neck, shoulders, and chest wall (white asterisk), and minimal PTX in the right apical area (white arrow). Bilateral lung parenchyma had peripheral and central ground-glass opacities with a CT severity score of 23/25