Case Report

COVID19- Spontaneous Pneumomediastinum On Noninvasive Ventilation in Interstitial Lung Disease Patient - Rare Case

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Abstract

Pneumomediastinum is a rare condition usually seen in patients with underlying pulmonary pathology (COPD, asthma), infections, or mechanical intubation due to barotrauma. The development of Pneumomediastinum in COVID-19 infection is considered a possible indicator of worsening disease and a high degree of suspicion of development of pneumothorax and expansion of pneumomediastinum should be considered in case of any sudden hemodynamic worsening.

Keywords: *COVID19, Spontaneous Pneumomediastinum, Non-invasive Ventilation, Interstitial Lung Disease*

Case history

A 67 years old male known case of liver cirrhosis, renal carcinoma, early ILD presented to the ED complaining of cough, difficulty breathing and fever for 3 days. No cough, runny nose, chest pain, palpitations, abdominal pain. The systemic review was otherwise negative

On examination in ER vitals were as follows: Blood pressure 135/75, pulse 89, temperature 37.1 °C (98.8 °F), temperature source Tympanic, resp. rate 20, weight 64 kg, SpO2 98 %. Physical Exam was normal except for minimal crept in basal area and normal vesicular breath sounds.

Labs in ER revealed: FBC, LFT, RFT, within normal limits, PCT was 0.09, in view of the patient was stable clinically with no Shortness of breath, chest pain, or palpitation (no GIT symptoms), Septic markers not much high, hence the patient was sent For home isolation as per family request. After 2 days patient came back to ER with increasing shortness of breath and desaturation hence, he was started on oxygen with a face mask and gradually his oxygen requirements went high and started on 15L/min O2 via a non-rebreather mask (NRBM), later on, Patient was started on NIV, due to tachypnea and desaturation on 15L/min O2 via NRBM and shifted to high dependency and was started on steroids, enoxaparin, antibiotics and antivirals and symptomatic treatment. Routine labs were within the normal range however, Ferritin, CRP, dimer were high and COVD RT PCR was positive.

ABG showed metabolic alkalosis and hypoxemia on 15 LPM oxygen and NIV, during hospital admission his Spo2 worsened and he was continued on NIV and NRBM. In view of desaturation HRCT was done which revealed early changes of ILD (bilateral scattered ill-defined areas of ground glass opacities more evident at the upper lobes & Thickened interlobular septae forming interlacing subpleural reticular showing and honeycombing suggestive of underlying interstitial lung disease Mild bilateral pleural reactions are seen. Retro-caval and pretracheal lymph nodes about 12 mm in diameter are detected. No pericardial effusion detected. Ectatic ascending aorta and arch with atheromatous calcifications of the latter. Scanned bones show no definite interval change), the patient was continued on the same management, 2 days after he became more hypoxic and elevated septic markers and dimers, hence Urgent multi-slice CT pulmonary angiography (CTPA) was done to rule out pulmonary embolism. In comparison to previous HRCT dated 3/11/2020, CTPA showed no obvious filling defects are seen in central pulmonary and segmental pulmonary arteries to the visualized extent to suggest pulmonary embolism with Interval appearance of a moderate amount of free air lucency seen surrounding the heat & great vessels denoting pneumomediastinum with small right-sided pneumothorax w*ith underlying changes of ILD.*

Gradually patient became more hypoxic and the crash call was announced due to respiratory distress and desaturation and considering that patient was In severe respiratory distress and Hypoxic despite maximum NIV setting intubation and mechanical ventilation was done after well informing the family and the patient was shifted to ICU and continued on standard management for COVID and symptomatic and supportive treatment including empiric antibiotics. Over few days patient deteriorated further and expired due to respiratory failure and cardiac events.

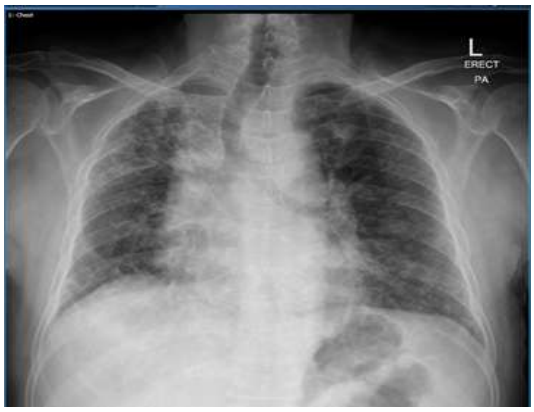


Figure 1: October 26, 2020

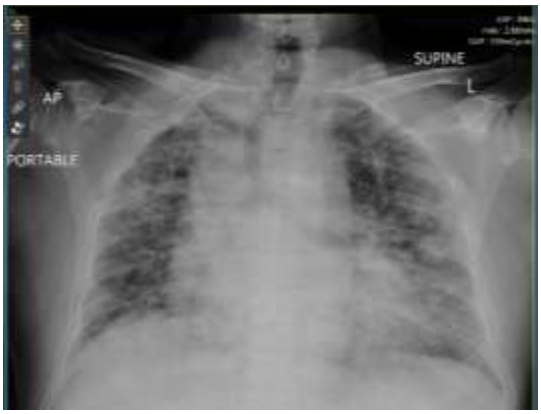
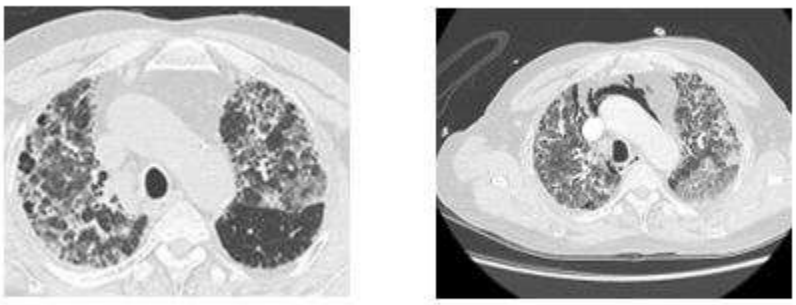
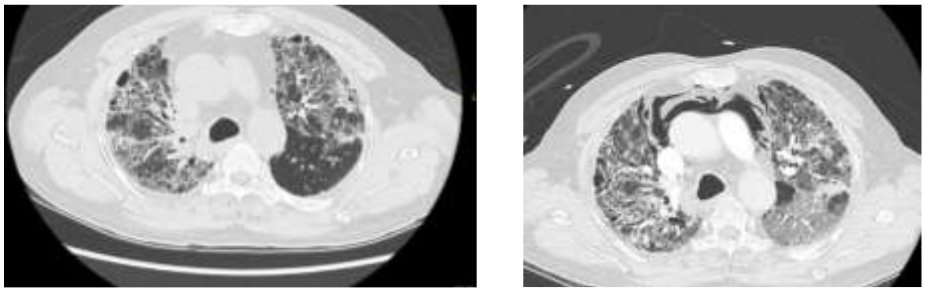


Figure 2: November 6, 2020, CXR - Interval appearance of the right lung volume loss with multifocal infiltrates in the right lung, predominantly in the upper lobe.
Patchy infiltrates are also seen in the left lung. Please recommend that correlation to exclude active infective/inflammatory disease.



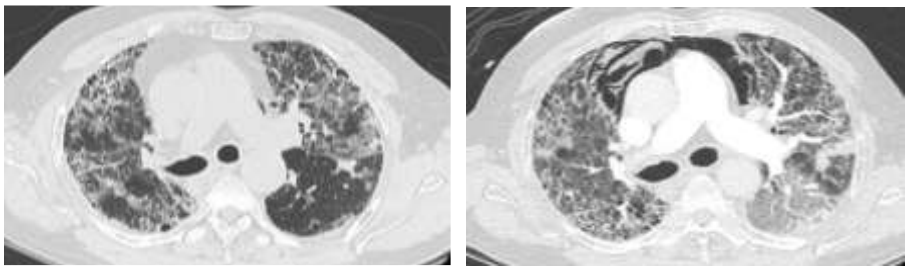
HRCT(A) CTPA(B)

Figure 3: HRCT(A) and CTPA(B) at the level of aortic arch trunk showing pneumomediastinum in CTPA



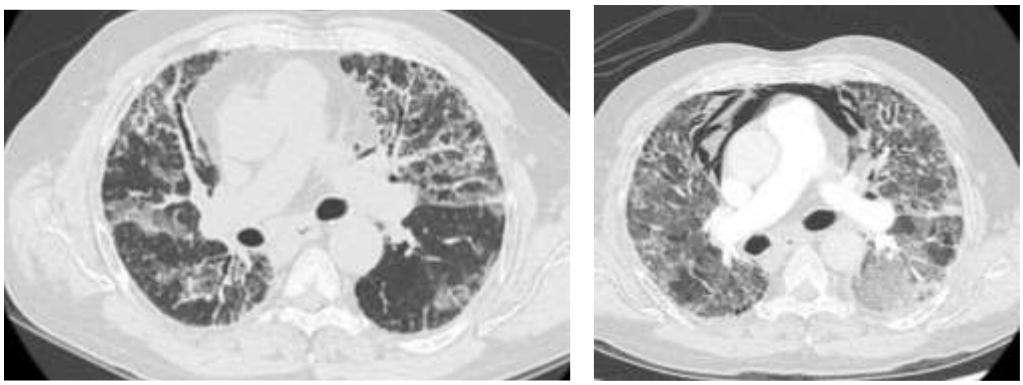
HRCT(A) CTPA(B)

Figure 4: HRCT(A) and CTPA (B) at the level of end of aortic arch showing pneumomediastinum in CTPA



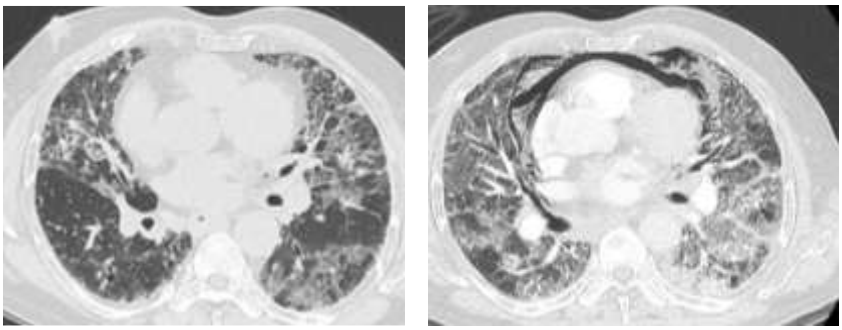
HRCT(A) CTPA(B)

Figure 5: HRCT(A) and CTPA (B) at the level of pulmonary trunk showing pneumomediastinum in CTPA



HRCT(A) CTPA(B)

Figure 6: HRCT(A) and CTPA (B) at the level of pulmonary trunk showing pneumomediastinum in CTPA



HRCT(A) CTPA(B)

Figure 7: HRCT(A) and CTPA (B) at the level of cardiac chambers showing pneumomediastinum in CTPA

Discussion

Spontaneous pneumomediastinum is a rare condition, most commonly caused by chronic respiratory conditions such as asthma, COPD, chronic lung disease, infections and mechanical ventilation. While most cases are self-limited and managed conservatively, the condition must be monitored carefully as it can lead to life-threatening cardiovascular and respiratory compromise if remain undiagnosed. **(**1**)**

In Our patient, he had a history of early ILD with severe COVID pneumonia requiring high Fio2 and NIV support, during NIV support he developed pneumomediastinum and small pneumothorax most likely due to stiff lungs and underlying parenchymal destruction by severe COVID pneumonia and with the contribution of PEEP by NIV. After the development of pneumomediastinum and small right-sided

pneumothorax, he became unstable and required mechanical ventilation. Spontaneous pneumomediastinum is uncommon in viral pneumonia. It has been reported in cases with severe acute respiratory syndrome-associated coronavirus pneumonia. **(**2**)**

Although the exact mechanism is unknown, increased alveolar pressure and diffuse alveolar injury in severe COVID-19 pneumonia are common which may make the alveoli more prone to rupturing, especially as patients often have pronounced violent cough with underlying pulmonary disease. To date, there have been few reports on spontaneous pneumomediastinum from COVID-19 in the setting of non-mechanical ventilation, although some cases have been complicated by pneumothorax. **(**3**)**
In addition, an inflammatory reaction could also contribute to spontaneous pneumothorax during lung infections. Some studies have reported that SARS might independently result in cyst formation, even in the absence of mechanical ventilation, and inflammatory exudate could play a relevant role in this. **(**4,5**)**

Other hypothesized etiologies for COVID patients developing pneumothorax and or pneumomediastinum could be as follow **(**6**)**:

1. In ventilated patients, high PEEP or insertion of the central line could be the risk factor for the development of pneumothorax which is not uncommon in ICU care.
2. Another possible hypothesis could be necrotizing small infarct of the lung secondary to micro and macro-vascular thrombosis and which could lead to a rupture in the pleura and the risk is high if the patient is ventilated and on high PEEP
3. Another associated infection viz TB, MRSA, bacterial pneumonia can also cause necrotizing pneumonia followed by a rupture into pleural space and consequent pneumothorax.
4. Underlying COPD or blebs or bullae which are common in the smokers and old/middle age population can also lead to rupture following the vascular compromise after COVID induces micro embolism and necrosis.

The pathologic features in the lungs of patients with COVID-19-related pneumonia greatly resemble those seen in SARS and Middle East respiratory syndrome coronavirus infection. **(**7,8**)**
Histologic examinations reported diffuse bilateral alveolar damage with cellular fibromyxoid exudates in COVID-19 and pulmonary cystic lesions. Pulmonary cystic lesions may develop in response to cellular fibromyxoid exudates, which form a valve in the bronchus. **(**9**)**

In fact, the cytokine storm syndrome, a critical clinical condition induced by a cascade of cytokine activation, characterized by overwhelming systemic inflammation, hyperferritinemia, hemodynamic instability, and multiple organ failure, is now recognized as being the main cause of the severity of SARS-

CoV-2 infection.33 It could, therefore, be hypothesized that patients with a higher inflammatory response in SARS-CoV-2 infection could be at higher risk of developing lung damage favoring Spontaneous pneumothorax.

In one recent Spanish study - The location of Spontaneous pneumothorax was in the right lung in 81% of cases, with an extension ranging from minimal to massive, and was accompanied by pneumomediastinum and subcutaneous emphysema in 16% of cases **(**10**)**
Volpi S. et al presented a case series of Pneumomediastinum, a rare complication in COVID-19 patients. They emphasize that conservative management is usually all that is required, with gradual resorption of the air from the tissues **(**11**).**

There have been 2 other COVID-19 patients with pneumomediastinum described in the literature to date, who were also treated conservatively1,2,3 However, it is important to continue to clinically monitor with serial imaging for the development of pneumothorax. In the context of the severe respiratory disease associated with COVID-19, it is highly likely that (unless there is a history of retching/vomiting) the pathogenesis of the pneumomediastinum is due to alveolar rupture secondary to barotrauma associated with mechanical ventilation, due to the high PEEP required to maintain adequate oxygenation in these severely compromised patients. Indeed, barotrauma is a recognized complication of mechanical ventilation. Tracheobronchial injury secondary to intubation can also be a cause. To try and minimize the risk of barotrauma, patients should be ventilated with the least damaging settings possible to achieve adequate oxygenation. In patients requiring escalating PEEP, efforts should be focused on identifying potentially reversible causes and strategies to reduce the PEEP should be sought, for example, pruning the patients early **(**12,13**).**

Conclusion

Pneumomediastinum is a rare condition usually seen in patients with underlying pulmonary pathology (COPD, asthma), infections, or mechanical intubation due to barotrauma. The development of Pneumomediastinum in COVID-19 infection is considered a possible indicator of worsening disease and a high degree of suspicion of the development of pneumothorax and expansion of pneumomediastinum should be considered in case of any sudden hemodynamic worsening. Patients with COVID-19 complaining of dyspnea and chest pain, and exhibiting tachycardia, tachypnea, and hypoxemia, should be assessed to rule out pneumothorax and pneumomediastinum.
Although it's not common in viral pneumonia, few cases have been described in COVID pneumonia. NIV should be used with low PEEP in patients with COVID pneumonia and preexisting underlying lung disease.

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