

Pneumothorax with Pneumomediastinum and Extensive Subcutaneous Emphysema Related to Covid-19 Infection

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Abstract

Spontaneous pneumothorax, which is not related to positive pressure ventilation, are currently seen in patients with COVID-19 pneumonia. It has been suggested that the possible pathophysiological mechanism is due to a diffuse alveolar injury leading to alveolar rupture and air leakage. We present a case of COVID-19 pneumonia, which was complicated by pneumothorax, pneumomediastinum and extensive emphysema on admission. The patient had no identifiable risk factors. He is a non-smoker without lung comorbidities or reported coughing. The patient underwent medical treatment for his COVID-19 infection with drainage but did not require invasive or non-invasive ventilation. He could be discharged home in stable condition. A review of the available literature revealed 15 other cases of spontaneous pneumomediastinum associated with COVID-19 pneumonia.

Keywords: Covid-19 Infection; Corona Infection; Covid-19 Related Pneumonia; Covid-19 Related Pneumothorax; Covid-19 Related Subcutaneous Emphysema; Covid-19 Related Pneumomediastinum

Introduction

By the end of 2019, the first notified cases of pneumonia with unknown aetiology were reported in Wuhan, China [1,2]. The clinical picture reminded of an acute respiratory distress syndrome (ARDS), which was identified as being a novel coronavirus [3]. It resulted in a worldwide spread of the virus and was declared a worldwide pandemic on 11 March 2020. To date, more than 127 Mio cases of infections have been noted with 2.8 Mio fatalities [4]. Over time, physicians have learned that the virus is of intrusive nature and does not only affect lungs but the entire organ systems creating various complications.

This case study presents a rare case of SARS-CoV-2 with pneumonia that was complicated by spontaneous pneumothorax, pneumomediastinum and extensive subcutaneous emphysema without the use of any pressure ventilation. Treatment could be managed conservatively although the patient was at a high risk due to his underlying complications.

Case Report

A 62-year-old male patient with a 6-day history of fever (maximum body temperature was about 39°C) and a rapidly progressing dyspnea since the day before A&E presentation was admitted to the hospital. On admission, he presented with extensive subcutaneous emphysema that involved the entire body from the head down to the thighs. Oxygen saturation under 2 l oxygen was 89%. He was tested positive for Covid 19 infection by corona virus nucleic acid testing by RT-PCR.

Initial X-rays revealed a pneumothorax on the left with additional bilateral opacities in the upper and middle lobes. CT images showed multiple bilateral ground glass opacities, which were consistent with COVID-19 changes.

His complete blood count revealed leukocytes $7.20 \times 10^9 /L$ ($3.5 - 9.5 \times 10^9/L$), thrombopenia $118 \times 10^9/L$ ($125 \text{ to } 350 \times 10^9/L$), lymphopenia $0.61 \times 10^9/L$ ($1.1 \text{ to } 3.2 \times 10^9/L$) and neutrophils $5.55 \times 10^9/L$ ($1.8 \text{ to } 6.3 \times 10^9/L$). Serum procalcitonin was 0.6 ng/mL ($< 0.05 \text{ ng/mL}$), LDH 598 U/l ($< 250 \text{ U/l}$) with C-reactive protein 112 mg/L ($< 3 \text{ mg/L}$). Blood gas analysis showed pH 7.46 ($7.35 \text{ to } 7.45$), oxygen partial pressure of 104 mmHg ($83 \text{ to } 108 \text{ mmHg}$) and carbon dioxide 45 mmHg ($33 \text{ to } 46 \text{ mmHg}$).



Figure 1: Extensive subcutaneous emphysema.

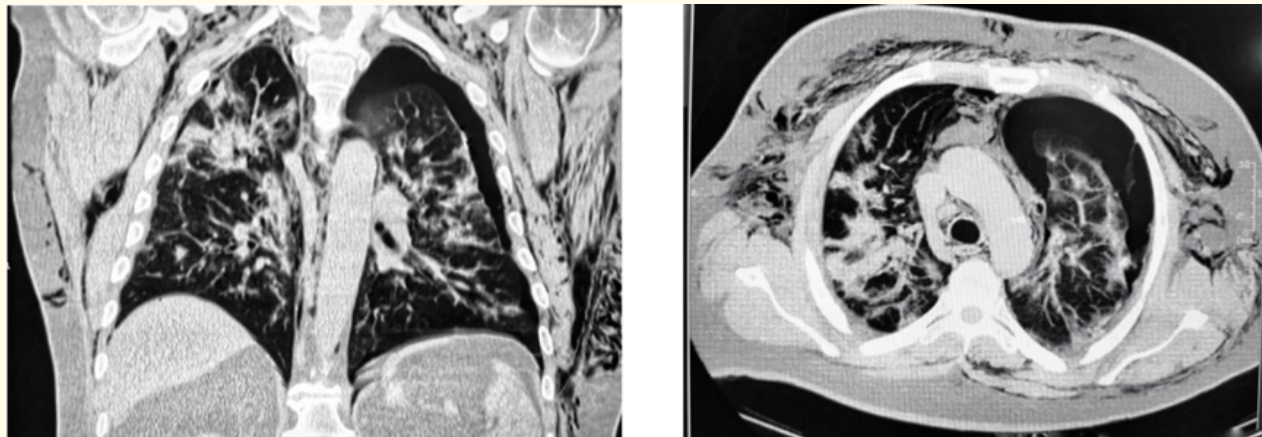


Figure 2: CT chest: Pneumothorax of the left lung with pneumomediastinum and extensive subcutaneous emphysema.

The patient received a chest drain and was put on 20 mmHg suction, 2l oxygen, lopinavir/ritonavir as well as antibiotics (initially cefoperazone-sulbactam followed by imipenem- cilastatin). Besides, he was also treated with steroid therapy (methylprednisolone 40 mg daily iv).

With high-flow nasal cannula and oxygen, intravenous steroid therapy over three days (methylprednisolone 20 mg/d) followed by oral Prednisone tablets for further four days (10 mg/d) for suspected cytokine storm. X-ray follow-ups could demonstrate full expansion of the collapsed lung. Clinical symptoms gradually improved over a period of 6 days. Pneumothorax and subcutaneous emphysema were absorbed completely. Ten days after admission, the patient had no more evidence of Corona virus in 2 consecutive RT-PCR tests.

Discussion

Spontaneous rupture of a subpleural bulla is the most common cause of primary spontaneous pneumothorax [5]. Contributing risk factors for the development of spontaneous pneumothorax have been identified by Wang [6] as tobacco smoking, male sex, age, low body mass index, thin stature, strenuous exercise, prolonged cough, and lung diseases, i.e. chronic obstructive pulmonary disease (COLD).

Spontaneous pneumomediastinum and pneumothorax, which are not related to positive pressure ventilation, have been reported as a rare and unusual complication in patients with severe COVID-19 pneumonia. It must be assumed that the pathophysiological mechanism results from a diffuse alveolar injury leading to alveolar rupture and air leak [7]. Subcutaneous emphysema usually occurs when air leaks into tissues under the skin, which is common after pneumothorax. Dyspnea is non-specific and a common symptom for severe COVID-19 pneumonia, pneumothorax and pneumomediastinum. This may not occur in the acute phase of infection but also as late sequelae in the course of recovery weeks after infection [8].

During the pandemic, typical features of COVID-19 have been identified. CT scans in the early phase can reveal bilateral multilobar and reticular ground-glass opacification that are localised in the periphery or the posterior aspects of the lungs with middle/lower predominance [9]. About one percent of COVID-19 patients develop a pneumothorax [10]. And finally, spontaneous pneumomediastinum, usually a rare condition, refers to alveolar rupture due to an increase in intrathoracic pressure, followed by air dissection through the bronchovascular sheath into the mediastinum.

Our case had spontaneous pneumothorax, additional pneumomediastinum and subcutaneous emphysema at the same time, which contributes to a rare clinical complication as a result of Covid-19 pneumonia.

Conclusion

This case is a reminder that acute clinical deterioration with oxygen desaturation in a COVID-19 patient with pneumonia can indicate pneumothorax or pneumomediastinum. The best management of such cases is yet still unknown. In this case, we treated with steroid therapy because we suspected a possible cytokine storm. The patient recovered well and without further surgical interventions. It is of note that the significance of the timing and the efficacy of steroid therapy in COVID-19 patients with severe pneumonia also remains unknown.

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