CASE REPORT

SARS-COV2 Pneumonia Revealed by Extensive Subcutaneous Emphysema

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Spontaneous subcutaneous emphysema (SCE), pneumomediastinum, and pneumothorax in nonventilated COVID-19 patients are being increasingly recognized as complications of COVID-19 pneumonia. We report a case of a young man with no predisposing risk factors for pneumothorax who was diagnosed with a severe acute respiratory syndrome-COV2 pneumonitis revealed by extensive SCE with fatal outcome. Computed tomography of the thorax, abdomen, and pelvis revealed a diffuse lung injury, a pneumothorax, and pneumomediastinum. These complications of COVID-19 pneumonia may be associated with poor prognosis.

Keywords: COVID-19, Pneumomediastinum, Pneumothorax, Subcutaneous emphysema

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Introduction

Novel coronavirus named severe acute respiratory syndrome (SARS-COV2) was identified as the causal agent of COVID-19 disease. The virus has been involved in a significant number of deaths across the world. Acute respiratory distress syndrome (ARDS) secondary to COVID-19 pneumonia is one of the main causes of morbidity and mortality along with multi-organ failure,[1] acute renal failure, cardiomyopathy,[2,3] and microthrombosis.[4]

Pneumothorax, pneumomediastinum, and/or subcutaneous emphysema (SCE) have recently been reported as serious complications in patients with COVID-19 disease associated with poor prognosis. These complications were unrelated to positive pressure ventilation,[5–7] occurring at admission or during hospitalization.[8–11]

In this article, we report the case of severe COVID-19 pneumonia revealed by spontaneous pneumomediastinum (SPM), pneumothorax, and massive SCE with a fatal outcome.

Case Report

A 49-year-old patient with a past medical history of treated hepatitis C presented to the emergency department with a 4-day history of persistent dry cough, fever, and worsening shortness of breath. On examination, the patient was tachypneic with a respiratory rate of 35 breaths/min, oxygen saturation of 50% on room air improved to 85% on 15 L/min of oxygen via a nonrebreather mask, he was apyrexial, heart rate was 95 beats/min, blood pressure 165/67 mmHg, and capillary blood glucose was 500 mg/dl. He had a dysphonia and a significant SCE with palpable subcutaneous crepitations over the head, neck, thorax, and abdominal wall.

Computed tomography (CT) of the thorax showed multifocal bilateral ground-glass opacities (typical pattern of SARS-CoV-2 pulmonary infection) affecting 80% of the pulmonary parenchyma, small right-sided pneumothorax, and pneumomediastinum extending to the retroperitoneum and around the diaphragm. He also had a massive SCE extending from the infratemporal fossa to the anterior abdominal wall and left palpebral emphysema, but there was no pulmonary embolism (Fig. 1). Reverse-transcriptase polymerase chain reaction (RT-PCR) was positive for SARS-COV-2.

The patient was started on hydroxychloroquine, azithromycin, zinc sulfate, and enoxaparin. He was given 60 mg intravenous methylprednisolone and hyperglycemia was managed with variable rate intravenous insulin infusion. The pneumothorax was not drained given its small size. Despite initiating high-flow oxygen therapy, oxygen requirements continued to increase with no clinical improvement. Arterial blood gas on 100% oxygen showed a pH of 7.14, PCO_2 of 31 mmHg, PO_2 of 56 mmHg, and HCO_3-of 10.4 mmol/L. Two hours following his admission to the emergency department, the patient was continuing to deteriorate, he was intubated and transferred to the intensive care unit.

Respiratory status continued to worsen and he was intubated 2 h after admission without clinical improvement and without SCE getting worsening. Sadly, the patient died 10 h following his admission to the intensive care unit from hypoxic acute respiratory failure.

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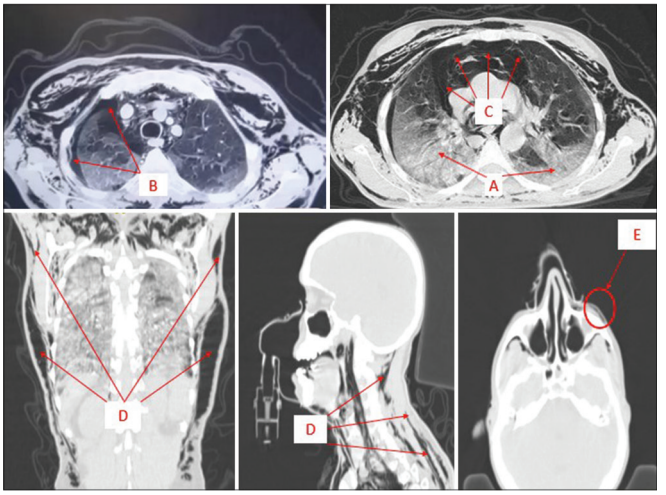


Fig. 1: Computed tomography revealing multifocal bilateral ground-glass opacities associated with pneumothorax, pneumomediastinum, and extended subcutaneous emphysema. (A) Multifocal bilateral ground-glass opacities with consolidation. (B) Right pneumothorax. (C) Pneumomediastinum. (D) Subcutaneous emphysema extending to the neck, thorax, and abdomen. (E) Subcutaneous emphysema of the left eyelid

The patient's condition deteriorated sharply, it was difficult to maintain oxygenation through mechanical ventilation and proning. Despite the staff's efforts, he eventually died of ARDS.

Discussion

Typically described as a consequence of barotrauma in patients undergoing mechanical ventilation, SPM, pneumothorax, and SCE are unusual presentations of COVID-19 pneumonia. A literature review shows other reported cases of spontaneous pneumothorax in nonventilated COVID patients, occurring during the hospital stay or at admission.

The incidence of SPM and SCE in nonventilated patients is still unknown. In 2003, 11.6% of patients who were diagnosed with SARS caused by novel COV (SARS-COV) developed SPM at 19.6+/−4.6 days from symptom onset.[12]

SARS-COV can lead to alveolar rupture by causing severe diffuse alveolar damage and interstitial emphysema. The resulting air then dissects the bronchovascular sheaths into the pulmonary hila and reaches the mediastinum. In some patients, the air may progress to SCE and pneumothoraces which are not systematically associated.[12] This pathophysiological etiology was confirmed in COVID-19 patients in pathology from postmortem studies. Carsana *et al*. found multiple aspects of lung tissue damage in postmortem studies of 38 patients who died of COVID-19 disease. They reported features of alveolar damage similar to those described with SARS and Middle East respiratory syndrome, capillary congestion, necrosis of pneumocytes, hyaline membranes, interstitial and alveolar edema, type 2 pneumocyte hyperplasia, and platelet-fibrin thrombi in pulmonary arterial vessels.[13]

SCE and SPM were diagnosed alone or associated with pneumothorax in ventilated or nonventilated patients with COVID-19 pneumonia. They were either present on admission or they were diagnosed during the hospital stay on chest X-ray or CT of the thorax in view of clinical deterioration.[5–7] The diagnostic interval of these complications varies from few days to several weeks.[8–11]

Our patient was admitted to the emergency department with a massive SCE along with a typical expression of severe COVID-19 pneumonia, rapid worsening shortness of breath, and persistent dry cough. The CT was performed to assess the extension SCE and search for typical signs of SARS-COV2 infection given the current epidemiological context and showed a right-sided pneumothorax with typical radiological signs of COVID-19. RT-PCR confirmed the diagnosis of COVID-19 pneumonia. The patient was without underlying conditions or risk factors for spontaneous pneumothorax.

Clinical deterioration due to diffuse lung injury, SCE, and SPM are complications associated with poor prognosis.[6] The patient was intubated and ventilated using ARDSnet protective lung ventilation and he was proned. There was no indication for decompression or drainage of the pneumothorax.[14] Extracorporeal membrane oxygenation as a rescue therapy for hypoxemic respiratory failure is not available in our setup.

Conclusion

SCE and SPM associated or not to pneumothorax can reveal or complicate a COVID-19 disease even in non ventilated patients. The extension of SCE and SPM reflects the severity of alveolar damage and lung injury and is associated with a poor prognosis.

Consent

Written informed consent was obtained from the patient's next of kin for publication of this case report and any accompanying images.

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