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Case Report

Spontaneous pneumothorax and pneumomediastinum as a rare complication of COVID-19 pneumonia: Report of 6 cases [☆]

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ABSTRACT

Spontaneous pneumothorax (SPT) and pneumomediastinum (SPM) have been reported as uncommon complications of coronavirus disease (COVID-19) pneumonia. The exact incidence and risk factors are still unrecognized. We report 6 nonventilated, COVID-19 pneumonia cases with SPT and SPM and their outcomes. The major risk factors for development of SPT and SPM in our patients were male gender, advance age, and pre-existing lung disease. These complications may occur in the absence of mechanical ventilation and associated with increasing morbidity (chest tube insertion, sepsis, hospital admission) and mortality. SPT and SPM should be considered as a potential predictive factor for adverse outcome and probable cause of unexplained deterioration of clinical condition in COVID-19 pneumonia.

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Introduction

From the beginning of the coronavirus disease (COVID-19) outbreak in December 2019 in Wuhan City, China, the novel COVID-19 has emerged as a global healthcare crisis [1]. COVID-19 manifests as a multisystem disease, however, the lung represents the most common affected target organ.

The typical clinical presentation of this disease consists of fever, dry cough, shortness of breath, fatigue, headache, and myalgia [1]. The characteristic CT scan findings of COVID-19 pneumonia are mainly bilateral, lower lobe, and peripheral distributed ground-glass opacities. Pneumothorax and pneumomediastinum are rare findings [2]. There are case reports of spontaneous pneumothorax (SPT) and pneumomediastinum (SPM) in patients with COVID-19 [3,4], and some suggest the

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presence of pneumothorax and pneumomediastinum may be predictive of worse prognosis [5]. Here, we reported 6 nonventilated, COVID-19 cases with SPT and SPM.

Case 1

A 90-year-old man with history of diabetes, chronic renal insufficiency, chronic obstructive pulmonary disease (COPD), and intensive care unit (ICU) admission due to COVID-19, 3 weeks before current presentation, was brought to the emergency department with fever, pleuritic chest pain and severe shortness of breath in the preceding 24 hours. His labs were significant for hyperglycemia, leukocytosis, and inflammatory (CRP, LDH) markers. He was hypoxic, with an SpO₂ of 85% with 14 L/min oxygen via nasal cannula, tachypneic and tachycardic. There was no breath sound on the left side.

The chest CT scan showed large, left side tension hydropneumothorax causing collapse of left lung with contralateral mediastinal shift, minimal right-sided pneumothorax Fig. 1A. In addition to tension pneumothorax, bilateral patchy ground-glass opacities and small subpleural blebs in the lower lobes Fig. 1B.

During previous ICU admission, he had received azithromycin, ceftriaxone, dexamethasone, and supplementary 10 L/oxygen by mask face with maximum 90% FiO₂ without mechanical ventilation. There was no history of trauma or central venous line insertion.

The tension pneumothorax was treated by insertion of a pleural tube. In spite of ICU admission and appropriate management, he deceased with clinical features of sepsis, 3 days after diagnosis of tension pneumothorax .

Case 2

A 67-year-old man with productive cough, fever, shortness of breath, and abnormal chest radiograph presented to the emergency service. He had a history of major depression, COPD, opium addiction and tobacco smoking. Diagnosis of COVID-19 pneumonia was made based on PCR. Lymphopenia, elevated bilirubin and IL-6 level were noted on initial blood work. Chest CT scan at that time showed: Bilateral high-density patchy ground glass opacities and consolidations more prominent at the bases, bullae and cystic spaces in the peripheral and subpleural distribution Fig. 2A. He was febrile and hypoxic (SpO₂ of 83% with 6 L/min oxygen). He was started on supplementary 15 L/oxygen via nasal cannula, corticosteroid, antibiotics and hydroxychloroquine. His ICU course was unremarkable and with no need mechanical ventilation. After 5 days he was discharged from ICU and transferred to the ward. Three days later, he experienced pleuritic chest pain after prolonged coughing. CT scan at this time shows new right-sided pneumothorax Fig. 2B. His pneumothorax was treated by insertion of pleural tube with appropriate resolution.

Case 3

A 66-year-old man with hypertriglyceridemia, hypertension, and bronchial asthma presented to the emergency department with sudden onset pleuritic chest pain and dyspnea. His blood work demonstrated leukocytosis, lymphopenia,

hypocalcemia, elevated triglyceride and inflammatory markers (CRP, LDH). Chest radiograph finding was suggestive for left-sided pneumothorax and complementary chest CT scan showed large left-sided and very small right-sided pneumothorax Fig. 3A. Evidence of ground-glass opacities and consolidations bilaterally due to COVID-19 were seen Fig. 3B. A pleural catheter was placed on the left side. Follow-up chest images revealed good resolution of right-sided pneumothorax.

About 1 month earlier, he had history of COVID-19 pneumonia based on nasopharyngeal swap and ICU admission. During ICU time, he was hypoxic and received 12/L oxygen by nasal cannula with maximum 90% FiO₂ and no need for mechanical ventilation.

Case 4

A 60-year-old man with a diagnosis of COVID-19 pneumonia and ICU admission about 2 months earlier, presented to the emergency service with 3 days new chest pain, increased severity of dyspnea.

He had no risk factor. At the emergency, his vital signs were stable, with oxygen saturation of 95% in room air. There was asymmetry of breath sounds on auscultation with decreasing breath sounds on the left side. With clinical suspicion to pneumothorax or lung collapse, he was admitted and sent for CT scan. CT scan of chest demonstrated left-sided pneumothorax and heterogeneous ground-glass opacities and linear scarring compatible with absorption stage of COVID-19 pneumonia Fig. 4B. Drainage pleural catheter was inserted to control pneumothorax. There was a significant clinical improvement. In the following chest images, absorption of the pneumothorax was observed.

During the previous ICU admission, he had received corticosteroid, antibiotics, and supplementary oxygen by mask face. His ICU staying was smooth and transferred to the ward after 4 days. CT scan at that time showed bilateral patchy high-density ground-glass opacities and consolidative lesions Fig. 4A that are typical for progressive stage of COVID-19 in the peripheral and peribronchovascular distribution.

Case 5

A 30-year-old man with 1-week history of progressive leg swelling and pain, shortness of breath and cough was referred to CT pulmonary angiography. CT pulmonary angiography demonstrated multiple filling defects in the segmental and subsegmental arteries of right lower lobe, extensive bibasilar parenchymal consolidations Fig. 5B and as an incidental finding linear air streaks and bubbles in the mediastinal soft tissue (pneumomediastinum) Fig. 5A. The patient never smoked, no trauma or surgery and had no hematologic or thrombophilia abnormality. Anticoagulant therapy was started and in the follow-up images, no progression of pneumomediastinum.

One month before this presentation, he had severe COVID-19 pneumonia based on current epidemiologic criteria (physical findings, CT scan of chest, and blood oxygen saturation) and 7 days hospital admission including 1 day ICU staying. During hospitalization time, he received dexamethasone, antibiotics, supplementary oxygen by mask face with maximum

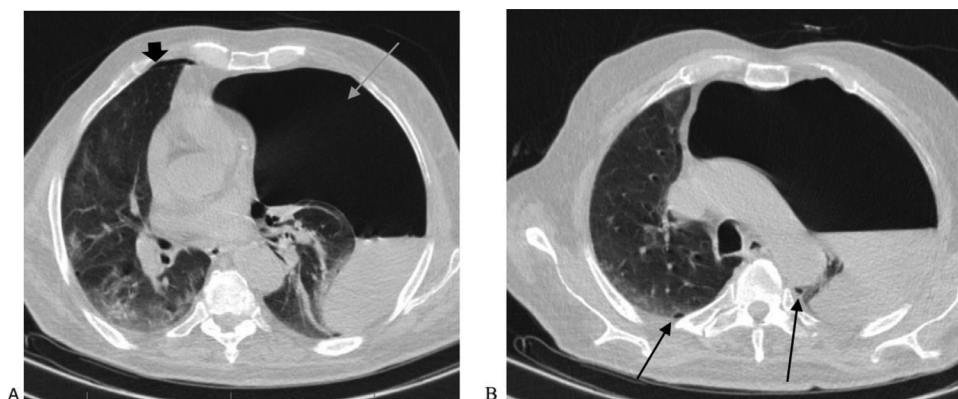


Fig. 1 – Patient 1 90-year-old man who presented with fever, severe dyspnea, and hypoxemia. (A) Axial section of CT of the chest shows large left side tension hydropneumothorax causing collapse of left lung and contralateral mediastinal shift (long arrow), minimal air (short arrow) in right pleural space. There are patchy ground-glass opacities in the right base. (B) Tiny subpleural blebs (long arrow) in the posterior part of upper lobes.

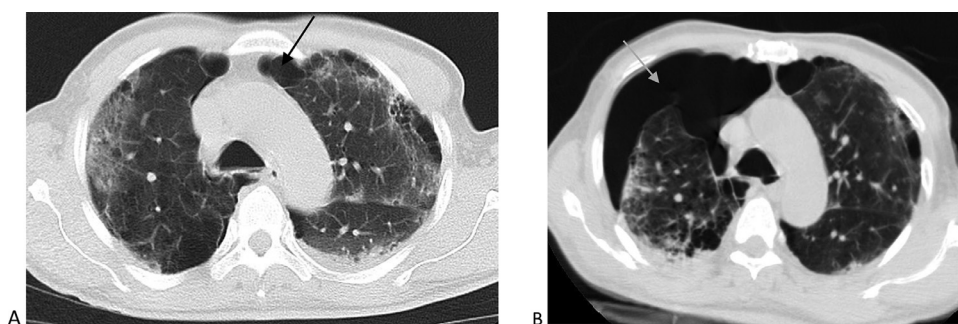


Fig. 2 – Patient 2 67-year-old man with sudden onset pleuritic chest pain and history of COPD. (A) Axial section in lung window shows multifocal patchy ground glass opacity, reticulations. Subpleural cysts in the upper lobes (arrow). (B) Right-sided pneumothorax (arrow) 3 days later.

90% FiO₂ and no need for intubation and mechanical ventilation.

Case 6

A 53-year-old male with no significant history presented to the emergency department with shortness of breath, chest pain, and cough. He was febrile and hypoxic on presentation with SpO₂ of 88% requiring 5/L oxygen via nasal cannula. Lymphopenia, elevated serum troponin level, and inflammatory (CRP, LDH) markers were noted on initial blood work. PCR for COVID-19 was positive. CXR revealed bilateral and extensive air-space opacity. He was admitted and started on hydroxychloroquine, azithromycin, ceftriaxone, and dexamethasone. He received oxygen by nasal cannula with maximum 90% FiO₂ and no need to be mechanically ventilated.

However, the patient clinically became deteriorated with hemodynamic instability, diminished renal function and was transferred to the ICU. About 14 days after presentation, we noticed worsening respiratory function and significant decreasing breath sound on the right side. CT scan of chest revealed: Right-sided pneumothorax and extensive bilateral ground-glass opacities Fig. 6. A decision was made to place a

pleural catheter under CT guidance. Unfortunately, on the following days, the patient's clinical status continued to worsen with hypotension and features of septic shock syndrome, then he expired after comfort measures were taken (Figs. 1-6).

Discussion

The clinical course of the COVID-19 is unpredictable and varies from asymptomatic or subclinical symptoms to severe disease [6] with development of acute respiratory distress syndrome and organ failure. Due to high sensitivity and rapid access, chest CT plays an important role in diagnosis and management of COVID-19 infection [2] and has been recognized as the most sensitive imaging modality to detect small amounts of pneumomediastinum and pneumothorax [7]. The most common manifestations of COVID-19 pneumonia in chest CT scan are multifocal ground-glass opacities with or without consolidative areas, predominantly in peripheral, lower-lobes, and posterior anatomic distribution [8]. These imaging findings mostly correlate with histopathologic manifestations of acute lung injury caused by SARS-CoV-2. Histologic findings of lung injury in COVID-19 are heterogeneous and have a broad

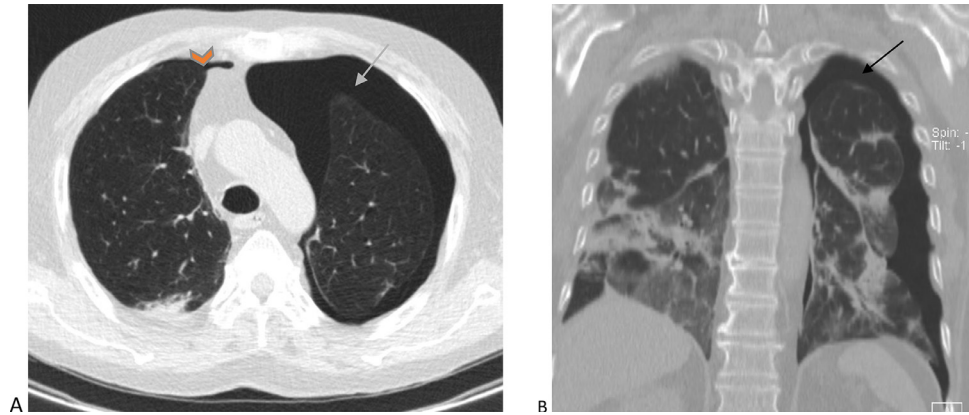


Fig. 3 – Patient 3 66-year-old man who presented with acute onset pleuritic chest pain. (A) Axial section of CT of the chest reveals large left (arrow) and minimal right sided (arrow head) pneumothorax. (B) Coronal MPR reconstruction shows pneumothorax (arrow), extensive areas of GGOs, consolidation seen commonly bilaterally, more predominantly in the posterior part of mid and lower third of lung.

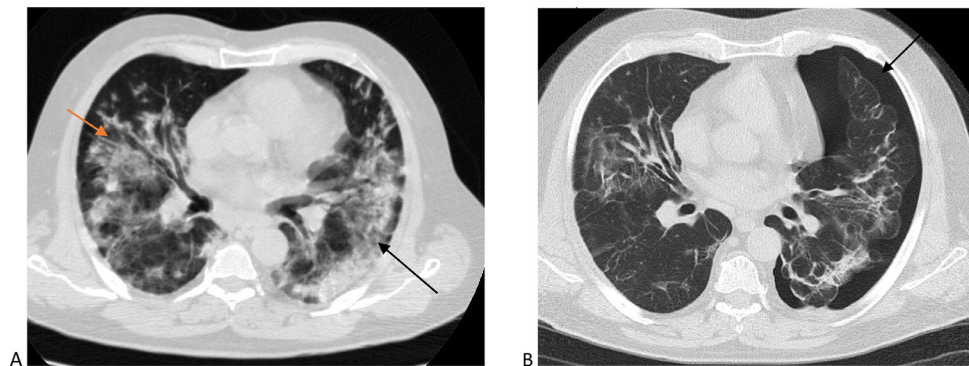


Fig. 4 – Patient 4 60-year-old man with 3 days new chest pain, increased severity of dyspnea. (A) Baseline CT scan of the chest reveals extensive GGOs (arrows), consolidations bilaterally, more predominant in the peripheral and peribronchovascular distribution on April 1. (B) Left-sided pneumothorax (arrow), heterogeneous ground-glass opacities and linear scarring compatible with absorption stage, 50 days later.

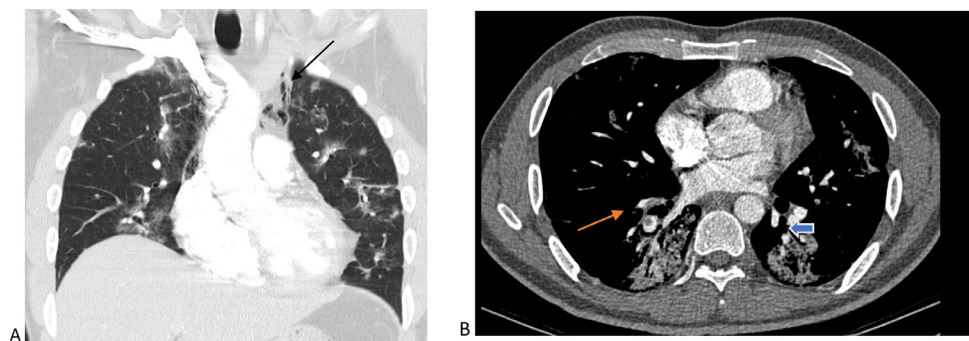


Fig. 5 – Patient 5 30-year-old man with 1-week history of progressive leg swelling and pain, shortness of breath and cough. (A) Coronal MPR reconstruction in parenchymal window shows linear air streaks and bubbles in the mediastinal soft tissue (pneumomediastinum). (B) Axial slice in mediastinal window demonstrates multiple filling defects in the segmental and subsegmental arteries right lower lobe (arrow), bilateral consolidations (short arrow) in the lower lobes.

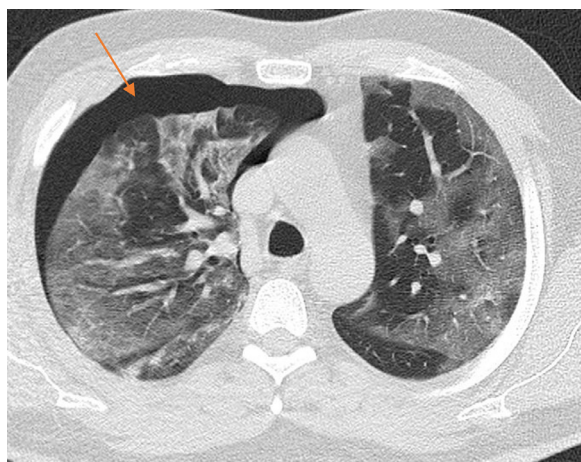


Fig. 6 – Patient 6 53-year-old male with shortness of breath, chest pain and cough. Axial section in parenchymal window reveals right sided pneumothorax (arrow) with extensive ground-glass opacities, enlarged small vessels, and fine reticulation (crazy paving) bilaterally.

spectrum ranging from diffuse alveolar damage with hyaline membrane formation to organizing pneumonia (OP), fibrosis [9,10], and microvascular thrombosis [11].

Some patients may have atypical or extrapulmonary manifestations as pleural effusion, mediastinal lymphadenopathy, pneumomediastinum, and pneumothorax during course of the disease. These findings may have potential prognostic value [12,13].

Although pneumothorax appears as a frequent and potentially life-threatening complication in acute respiratory distress syndrome, especially in those who had mechanical ventilation support [14], it has only been reported in 1% of COVID-19 patients [15].

McGuinness et al. [16] reported approximately 15% incidence of barotrauma among the COVID-19 patients requiring invasive mechanical ventilation. Yet to date, the incidence of SPT and SPM in the patients with no history of mechanical ventilation is unknown.

Etiology of pneumothorax in patients with severe COVID-19 pneumonia is not well recognized and many factors may precipitate the occurrence, such as the barotrauma during mechanical ventilation [16], complication of central line catheter insertion [18], and underlying pulmonary pathology (such as pre-existing emphysema, bulla, and cyst). The cause of pneumomediastinum may also be related to the abrupt increase in intrathoracic pressure associated with Valsalva maneuver (cough, vomiting, vigorous activity, shouting or inhalation of an illicit drugs) and subsequently rupture of the alveoli, followed by air dissection through the bronchovascular bundles into the mediastinum (Macklin's effect) [19].

Here, we describe 6 cases that developed SPT and SPM in the course of COVID-19 pneumonia.

In our case reports, the patients were male above the age of 29 years. They did not have mechanical ventilation and received supplementary oxygen only by facemask or nasal cannula, 3 patients had pre-existing lung disease (COPD). Mortality rate was 30%. The diagnosis of SPT or SPM was

made more than 1 week after the onset of symptoms (8-50 days). In the most of patients, the lung findings at the time of pneumothorax and pneumomediastinum diagnosis were compatible with the absorption stage of disease [17].

In conclusion, the SPT and SPM in our patients could be attributable to the sudden increase in intrathoracic pressure during forceful coughing and subsequently rupture of damaged alveolar wall or small lung cysts which had developed in the fibrotic stage of disease [20]. The risk factors in our patients were male gender, advance age and pre-existing lung disease especially COPD. This complication might be associated with poor outcome in the COVID-19 pneumonia [21]. The teaching points are that possibility of pneumothorax and pneumomediastinum is still existing in nonventilated patients and should be considered as a cause of clinical deterioration in hospitalized and nonhospitalized COVID-19 patients [22].

Consent statement

The authors of this article “Spontaneous Pneumothorax and Pneumomediastinum as A Rare Complication of COVID-19 Pneumonia” are confirming that informed consent for publication of this case reports was obtained from the patients.

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