Case Report

Spontaneous bilateral pneumothorax in a previously healthy COVID-19 patient: A case report

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ABSTRACT

Coronavirus disease 2019 (COVID-19) is commonly linked with mild cough, fever, and shortness of breath symptoms. However, there have been reports of pneumothorax, which particularly occurred at least 1 week following symptom onset in elderly COVID-19 patients. Spontaneous pneumothorax (SP) is an uncommon but possibly fatal complication of COVID-19 pneumonia and is rarely reported in non-intubated patients. We report a case of a healthy, non-smoker 35-year-old young woman who presented with a 7-day cough, fever, and sudden shortness of breath. She was diagnosed with severe COVID-19 pneumonia, experienced a right SP, and developed a second pneumothorax on the contralateral side. She improved gradually following chest tube insertion in the right lung and conservative management for the left pneumothorax.

Key words: Bilateral spontaneous pneumothorax, Chest tube insertion, Conservative management, Severe pneumonia COVID-19

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has spread globally, causing a pandemic [1]. Until May 2023, the World Health Organization (WHO) reported 766,895,075 confirmed COVID-19 cases worldwide, with 6,935,889 confirmed deaths [2]. The respiratory manifestation varies from mild upper respiratory symptoms to severe pneumonia or, even worse, acute respiratory distress syndrome (ARDS). It is also related to less common but potentially fatal pulmonary problems such as pneumothorax, necessitating a distinct management approach [3]. COVID-19-induced pneumothorax is rare, although numerous cases have been reported [1]. It has been documented in a small number of cases, with a 1% incidence in hospitalized patients and a 2% incidence in those requiring critical care. Most COVID-19-related pneumothorax patients have no typical risk factors or underlying pulmonary disease. Pneumothorax in COVID-19 patients can indicate a poor prognosis [3].

Here, we report an interesting case of COVID-19 pneumonia that developed a spontaneous pneumothorax (SP) bilaterally in a healthy 35-year-old woman without any prior history of lung disease. The patient was successfully treated with combined invasive and conservative management. This case emphasizes the early diagnosis and therapy of this severe and infrequent COVID-19 pneumonia complication such as pneumothorax to prevent life-threatening consequences.

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CASE REPORT

Our patient was a 35-year-old woman who presented with sudden onset of progressive shortness of breath after a coughing episode. She was a non-smoker, had no prior medical history, her height was 160 cm, her weight was 55 kg, and her body mass index was 21.5. The patient had a fever, dry cough, and nausea 1 week before admission, and she has not taken any medication.

On presentation, the patient was tachypneic, 27 breaths/min, and tachycardic, pulse rate of 134 beats/min. Her blood pressure was 137/81 mm Hg, and oxygen saturation was 57% on room air and increased to 88% on 15 L by a non-rebreathe mask (NRM) plus a 5 L nasal cannula of oxygen administration. Physical examination showed decreased vesicular breath sounds and hyperresonance to percussion on the right hemithorax and bilateral crackles.

An electrocardiogram showed sinus tachycardia without ischemic changes. Laboratory testing demonstrated abnormality in increased white blood cell count (13.000/uL), neutrophilia (84.5%), lymphocytopenia (8.0%), increased lactate dehydrogenase (LDH) (1439 U/L), mild hyponatremia (134 meq/L), and blood gas analysis showed pH 7,468, PCO2 29,8, PO2 58, HCO3 21,7, BE -2, SpO2 92%, PaO2/FiO2 ratio 58 on 15 L NRM plus a 5 L nasal cannula of oxygen supplementation.

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Chest X-ray demonstrated right pneumothorax and bilateral infiltrates consistent with the diagnosis of COVID-19 pneumonia (Fig. 1a). Real-time reverse transcription–polymerase chain reaction (RT-PCR) swab nasopharyngeal revealed positive for SARS-CoV-2. *Mycobacterium tuberculosis* was undetected from the PCR GeneXpert examination.

A chest tube insertion with continuous suction was done on the right pleural space. The patient then received oxygenation therapy through NRM 15 L/min, IV remdesivir (200 mg/day on day 1 and 100 mg/day after day 2), IV methylprednisolone, anticoagulant prophylaxis, antibiotics, and multivitamin based on local management guideline. Her clinical condition and CXR improved gradually (Fig. 1b), and the right pleural catheter was detached. However, the patient had a worsening cough and dyspnea 6 days later. She had hypoxemia with an oxygen saturation of 87% with NRM 15 L/min. Chest examination revealed decreased breath sound in the left lung. A chest X-ray identified a left-sided pneumothorax and multifocal patchy opacities (Fig. 1c), but she refused to insert chest drainage again. Her laboratory result showed increased white blood cells (23.400/uL), high LDH 704 U/L, D-dimers (11.12 mg/L), and raised inflammatory markers (ferritin 658.68 µg/L and C-reactive protein 62.4 mg/L). She was only given conservative treatment oxygenation with 15 L through an NRM, antibiotics replacement, and chest physiotherapy. However, her oxygen requirement decreased gradually, and she was moved to the general ward after RT-PCR testing of the nasopharyngeal swab became negative on day 15 after admission. Because of our hospital limitation, a thorax computed tomography (CT) scan was performed after conversion negative of RT-PCR on day 27 of hospitalization, which showed pulmonary fibrosis and ground-glass opacities complicated by left pneumothorax (Fig. 2a). The patient showed clinical and radiological improvement with minimal left pneumothorax (Fig. 1d) after 36 days of treatment and could be managed as an outpatient.

2 weeks after discharge, the patient returned to the clinic for routine follow-up and showed considerable improvement. The last CT scan of the thorax was taken 2 months after the onset and revealed complete lung expansion bilaterally, with fibrosis and bullous emphysema subpleural bilateral (Fig. 2b).

DISCUSSION

Secondary SP is an accumulation of air in the pleural cavity caused by lung air leakage due to an underlying lung condition with no trauma [3]. The common causes are males, smokers, underlying lung disease, and barotrauma from positive pressure ventilation [4]. Our patient had no prior medical history, smoking history, or positive pressure ventilation during therapy, making her unpredisposed. This patient's pneumothorax likely resulted from structural lung injury after COVID-19 pneumonia.

There is a relation between SARS-CoV-2 and SARS-CoV-1 and the Middle East respiratory disease coronavirus (MERS-CoV). It was found that approximately 1.7–4% of 2003 SARS-CoV-1 cases had pneumothorax and 50% had bilateral pneumothoraxes. Before SP, most patients were healthy, without risk factors, and had not experienced positive pressure ventilation or invasive procedures [1,4]. Free air from ruptured alveoli (produced by diffuse alveolar damage) tracked up the bronchovascular sheath toward the mediastinum [2,5]. SP developed in 7.1–16% of MERS-CoV cases, and all patients received positive pressure ventilation [5]. Meanwhile, in SARS-CoV-2, SP has been observed in 1% of hospitalized patients and 2% of those requiring critical care [3].

COVID-19 symptoms can be confused with pneumothorax. A pneumothorax usually manifests as abrupt-onset pleuritic chest discomfort with a sense of breathlessness. Our patient complained of cough, fever, and breathlessness, the most common symptoms of COVID-19. We checked the right hemithorax for decreased vesicular breath sounds and hyperresonance to percussion. She

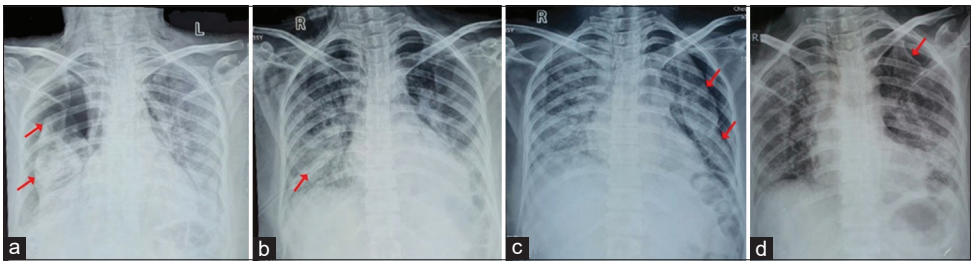

Figure 1: Chest X-ray Findings. (a). Chest X-ray (CXR) patients, when admitted to the hospital, bilateral infiltrates with right pneumothorax. (b). CXR 2 days after chest tube insertion, expanded right lung (c). 6 days hospitalized, CXR showed left-sided pneumothorax with multifocal patchy opacities. (d). CXR before patients discharged from hospital on 32nd day, improved of infiltrates but still minimal of left pneumothorax

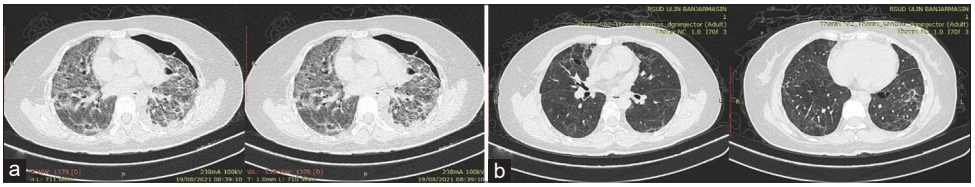

Figure 2: Chest CT-scan findings. (a). Chest CT scan on day 27 of hospitalization (13 days after discharge from isolation room; 37 days from onset) showed pulmonary fibrosis and ground-glass opacities complicated by left pneumothorax. (b). Chest CT scans 36 days after discharge (63 days from onset) showed complete lung expansion bilateral, with fibrosis and bullous emphysema subpleural bilateral

developed severe symptoms and was admitted for respiratory distress. COVID-19 infections most often cause posterior and peripheral ground-glass opacities. Uncommon imaging findings include pleural effusion, pneumothorax, lymphadenopathy, pericardial effusion, and cavitation [4]. In this case, infiltrates were seen in imaging dominantly on both sides of the lung with pneumothorax.

Although the exact cause of SP in COVID-19 is obscure, it could be related to some mechanisms. In patients with COVID-19 pneumonia, SP is a complication frequently associated with severe cytokine storm cases, an overactive immune response to SARS-CoV-2 infection [1,6]. A severe COVID-19 infection caused widespread alveolar destruction, making the alveoli vulnerable to rupture and causing pneumothorax. Without non-invasive or invasive breathing support, pneumothorax may develop. Our patient had a significant cough, which could trigger an alveolar rupture. It is consistent with Rafiee *et al*., who found that a severe cough and underlying COPD were risk factors [7,8]. Prolonged inflammation, ischemia, and fibrosis in COVID-19 patients with ARDS may predispose them to alveolar injury and produce pleural air leakage [2,8]. Our patient had no known history of pulmonary disease but had lung parenchymal abnormality with 75% involvement, indicating that SARS-CoV-2-induced alveolar destruction produced the pneumothorax. COVID lung inflammation can cause pneumatocele and SP. Mechanical ventilation contributes to pneumatocele; however, cases have been observed without it [1,6]. Another possible pathophysiologic mechanism for SP is the formation of subpleural organized microinfarctions due to peripheral thrombosis. These microinfarctions could cause pleural leakage and, eventually, pneumothorax [9].

In general, there is no difference in the management of pneumothorax in COVID-19, specifically the insertion of a thoracic drainage system allowing appropriate lung re-expansion in 48–72 h. However, the progression of the underlying lung parenchymal disease determines the patient's prognosis. A literature review shows 18 case reports on SP in COVID-19 patients. Ten patients required chest tube insertion, whereas eight were treated conservatively [1]. In our case, chest tube insertion was performed only in the right lung, while the left lung pneumothorax was managed conservatively.

Even after the viral infection has resolved, the patients may have several sequelae, one of which is post-COVID-19 fibrosis, as seen in this patient. According to a study by Nabahati *et al*. on post-COVID-19 pulmonary fibrosis, over half of 90 patients who recovered from moderate or severe COVID-19 exhibit anomalies in the form of pulmonary fibrosis [10].

Our case had limitations; due to hospital resources, a chest CT scan cannot be conducted when a patient has a positive RT-PCR; hence we lacked detailed data on pulmonary infiltration and pleural involvement in the early-stage illness of COVID-19 pneumonia.

CONCLUSION

This case illustrates that bilateral pneumothorax in COVID-19 can develop in non-intubated and previously healthy women. Any patient with suspected or confirmed COVID-19 developing new acute symptoms or consistent clinical worsening with pneumothorax should be evaluated for SP. Early diagnosis and treatment are critical and can prevent life-threatening consequences.

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