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Case report

COVID-19 pneumonia complicated by bilateral pneumothorax: A case report

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ABSTRACT

Background: Pneumothorax is a rare but life-threatening complication associated with pneumonia caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).
Case presentation: Informed consent was obtained from the patient himself.A 50-year-old man presented with a 9-day history of fever, cough, and dyspnoea. He was diagnosed with coronavirus disease 2019 (COVID-19) pneumonia and was admitted to the Medical Hospital, Tokyo Medical and Dental University. Chest CT showed diffuse patchy ground-glass opacities (GGOs). His state of oxygenation deteriorated, and mechanical ventilation was initiated on day 4 after admission (12th day from onset). He improved gradually and was weaned from ventilation on day 15. Sudden onset of bilateral pneumothorax occurred on day 21 with severe respiratory failure, and chest CT revealed pneumatocele formation on both lower lobes.
Conclusions: Pneumothorax is a notable complication in cases of severe COVID-19 pneumonia, especially in those who require positive-pressure ventilation.

1. Introduction

In early 2020, infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) became a pandemic. The outbreak also occurred in Japan, especially in urban areas with high population densities [1]. According to a report analysing the clinical characteristics of 1099 patients in China, 15.7% exhibited severe pneumonia, and 2.3% needed invasive mechanical ventilation [2].

Pneumothorax is reported as a rare but life-threatening complication of coronavirus disease 2019 (COVID-19) pneumonia [3–7]. We consider severe COVID-19 pneumonia complicated with bilateral pneumothorax and pneumatocele formation to be a cause of pneumothorax.

1.1. Case presentation

The patient was a 50-year-old man with a history of mandibular carcinoma (at age 45) and a former smoker of 2 packs of cigarettes daily for 10 years, though he quit smoking 15 years previously. He experienced fever of 38.5 °C and dry cough and visited a physician. A rapid influenza test was negative, and he received antifebrile agents. However, his fever continued, and he had worsening dyspnoea within a few days. Polymerase chain reaction (PCR) for SARS-CoV-2 from nasal swabs

Abbreviations: COVID-19, coronavirus disease 2019; CRP, *C*-reactive protein; GGOs, ground-grass opacites; PCR, polymerase chain reaction; PEEP, positive end-expiratory pressure; P/F, PaO2/fraction of inspired oxygen; SARS, severe acute respiratory syndrome; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

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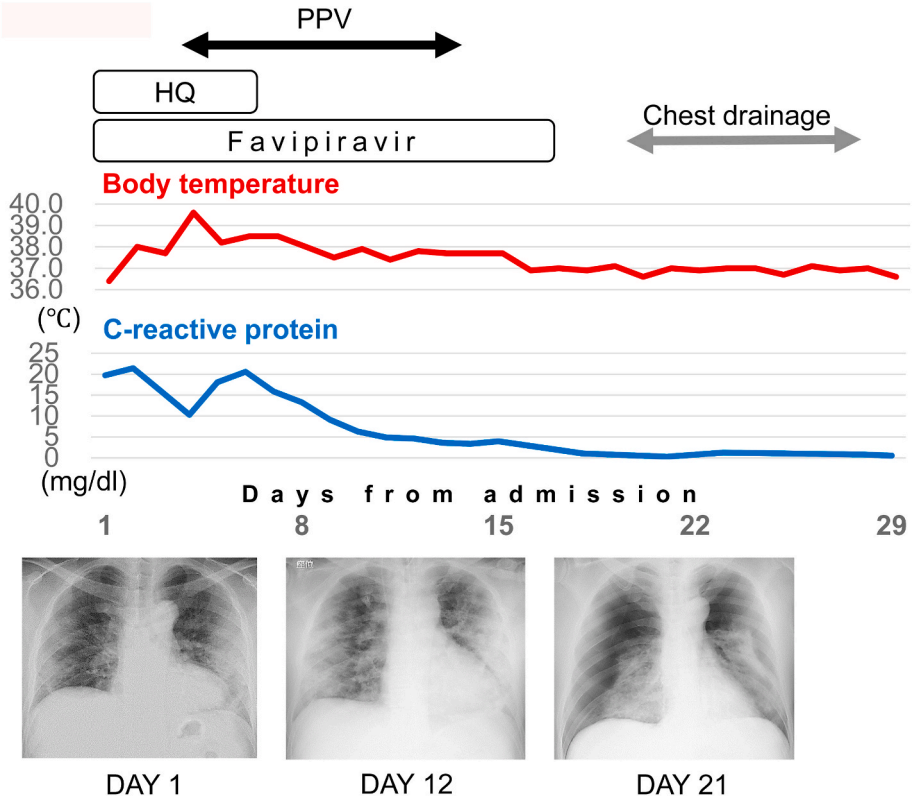

Fig. 1. Clinical course of the patient. Changes of body temperature and levels of *C*-reactive protein from the admission (the 9th day from onset) to the discharge. HQ: Hydroxychloroquine; PPV: positive-pressure ventilation.

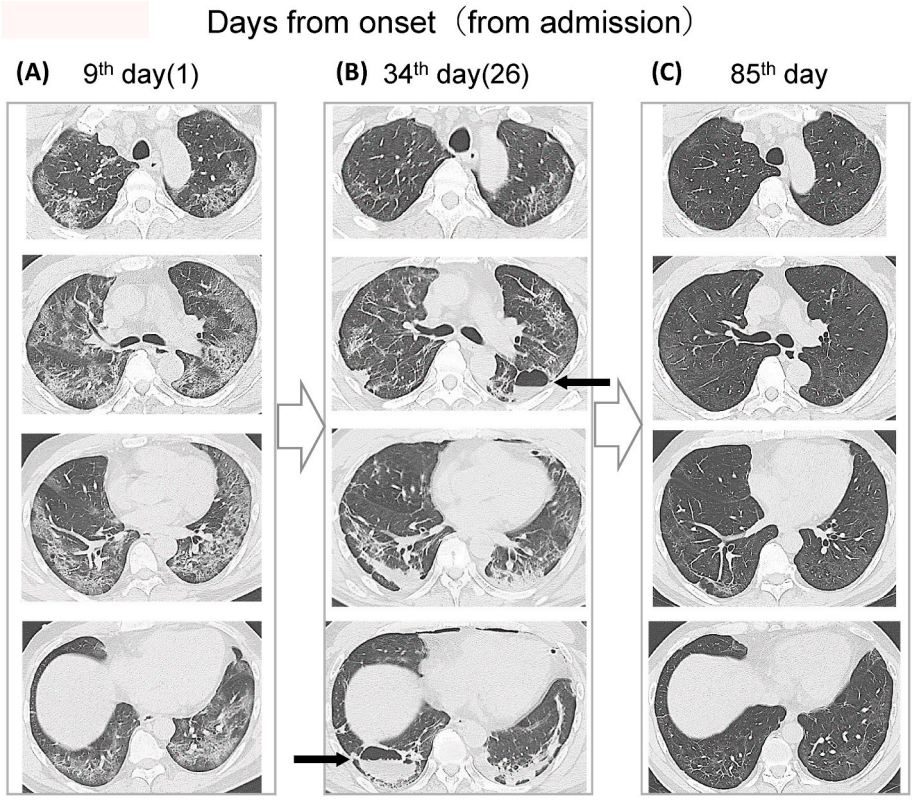

Fig. 2. Findings of Chest CT scan. Chest CT performed on admission (A: on the 9th day from onset), after drainage of pneumothorax (B: on the 34th day), and 1 month after discharge (C: on the 85th day). Pneumatocele formations were found on the both lower lobe (B: indicated as the black arrow).

was performed on the 6th day from onset, and he was diagnosed with coronavirus disease 2019 (COVID-19) on the 9th day. His dyspnoea continued to worsen during this period, and he was admitted to our hospital on the same day. On physical examination, his body temperature was 36.4 °C, and saturation of peripheral oxygen (SpO_2) was 87% for room air. Laboratory tests showed a total white blood cell count of $7.4 \times 10^3/\mu l$, with neutrophil and lymphocyte proportions of 84.9% and 8.4%, respectively. The *C*-reactive protein (CRP) level was elevated to 19.74 mg/dl. A chest CT scan showed bilateral patchy ground-glass opacities (GGOs) distributed predominantly in the subpleural regions. Favipiravir (3600 mg/day on day 1 and 1600 mg/day after day 2) and hydroxychloroquine (6 mg/kg/day) were started. GGOs had progressed, and his hypoxia deteriorated, requiring 5 L/min of supplemental oxygen on day 4 of admission (12th day from onset). He was moved to the ICU, and mechanical ventilation was initiated and continued for 11 days. The mean positive end-expiratory pressure (PEEP) and minute volume were 9.8 mmH_2O and 8.94 L/min respectively. The PaO_2/fraction of inspired oxygen (P/F) ratio decreased, and GGOs on chest X-ray persisted until day 11 after admission. However, these parameters improved gradually, and he was weaned off ventilation on day 13.

He was moved to the general ward on day 15, and PCR testing of nasal swabs became negative twice on day 19 after admission. GGOs on chest X-ray also improved (Fig. 1). However, on day 21 after admission, intense dyspnoea suddenly appeared after he coughed. Bilateral pneumothorax was detected by chest X-ray (Fig. 1), and chest drainage on both sides of the chest cavity was immediately implemented. Both lungs rapidly fully expanded after this intervention. A chest CT scan was performed to evaluate the underlying causes of pneumothorax, and pneumatocele formations were found on the bilateral lower lobes (Fig. 2). The chest drains were removed 8 days after insertion, and the patient was discharged on day 29. On the 85th day after onset, chest CT demonstrated that most of the pneumatocele had disappeared, but GGOs in the peripheral region and a linear shadow in the right lower lobe remained.

2. Discussion

As several cases have been reported, pneumothorax is a rare but life-threatening complication of COVID-19 pneumonia [8,9]. We report that severe COVID-19 pneumonia complicated by bilateral pneumothorax occurred after mechanical ventilation for 11 days. The patient had no underlying disease leading to pneumothorax on chest CT at admission, and it occurred when the patient had improved sufficiently to wean him from ventilator support. Additionally, and of note, pneumatocele formation was identified as the cause of pneumothorax by a chest CT scan performed immediately after chest drainage.

Pneumothorax has been reported as a specific and potentially life-threatening complication in mechanically ventilated patients with severe acute respiratory syndrome (SARS), which was an epidemic in China from 2002 to 2003 [10,11]. Kao et al. [12] reported that mechanically ventilated SARS patients with higher respiratory rates, lower P/F ratios, and higher $PaCO_2$ during hospitalization had greater risks of developing pneumothorax; in contrast, there were no significant differences in the pressures and volumes of positive-pressure ventilation between patients with complicated and uncomplicated disease (mean PEEP and minute volume between complicated and uncomplicated group were 8.2 and 7.9 mmH_2O, 11.38 and 10.40 L/min respectively).

Alan et al. [13] reported 6 cases of SARS complicated by pneumothorax, and 3 of 6 cases were bilateral. Chest CT performed after chest drainage revealed honeycombing or a bronchiectasis-like appearance with diffuse inflammatory change. The authors hypothesized that the cause of pneumothorax was rupture of these dilated cystic airspaces arising from diffuse alveolar damage. They also speculated that a sustained period of lung inflammation was an important pathogenic factor in the development of pneumothorax because the onset was relatively late (the mean time from admission to development was 24.3 days). Clinical manifestations between SARS and COVID-19 are similar, with widespread GGOs on chest CT [14], higher inflammation observed in severe or critical cases of COVID-19 pneumonia than in mild and moderate cases [2], and a longer duration required for recovery [15].

The pathogenesis of pneumatoceles is considered localized pulmonary over-inflation caused by check-valve type obstruction of the bronchus [16]. Although the relationship between positive-pressure ventilation and pneumatocele formation is uncertain, positive-pressure ventilation may cause distention of these spaces, leading to pneumothorax [17]. In the present case, positive pressure ventilation may have induced distention of pneumatoceles, leading to pneumothorax with a background of sustained diffuse lung inflammation caused by SARS-CoV-2 infection.

3. Conclusion

Long-term positive-pressure ventilation can be a risk factor for the development of pneumothorax in patients with severe COVID-19 pneumonia.

Declarations

Ethics approval and consent to participate:
The Patient provided consent for publication.

4. Consent for publication

Written informed consent was obtained from the patient described in this report.

5. Availability of data and material

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Author contributions

T Shirai wrote the manuscript. TM, KA, MK, TG, T Shigematsu, JT, SA, RY, HS, TO, KH, KO, and JA collected the clinical data. TO, YO, and YM supported the intellectual advice and revised the manuscript. All authors approved the final version of the manuscript.

Declaration of competing interest

The authors have declared no conflicts of interest.

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