

Coronavirus disease 2019 with spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema, France

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Abstract

To our knowledge, Complications such as pneumomediastinum and/or pneumothorax during the course of COVID-19 remain rare and their mechanism is poorly described. We present a case of COVID-19 pneumonia associated with spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema in an immunocompetent patient with no past history of smoking or chronic obstructive pulmonary disease (COPD). The only risk factor of this patient was prolonged cough. We hypothesize the mechanism underlying the pneumomediastinum is the aggressive disease pathophysiology in COVID-19 with an increased risk of alveolar damage.

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Introduction

On December 2019, an outbreak of pneumonia began in the city of Wuhan (China). The causal agent was identified as a novel coronavirus, named later the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Clinical descriptions of the coronavirus disease 2019 (COVID-19) outbreak reveal that most patients may be asymptomatic or have mild or moderate symptoms. Chest CT findings have been recommended as major evidence for clinical diagnosis of COVID-19 [1]. We report a case of COVID-19 pneumonia associated with spontaneous pneumothorax, pneumomediastinum and subcutaneous emphysema.

Case presentation

A 62-year-old man from France was admitted to the Infectious Diseases Department (Nord Franche-Comte Hospital, France) on 28 March 2020 with a 7-day history of influenza-like illness, anosmia and dyspnoea. On admission, he presented fever (39.5°C), productive cough without haemoptysis and general deterioration. Physical examination revealed bilateral crackling sounds on pulmonary auscultation. He did not have a history of smoking or chronic obstructive pulmonary disease.

On complete blood count, only the lymphocyte count (660 cells/ μ L) was decreased (normal range 1500–4000 cells/ μ L). Routine laboratory findings showed elevated C-reactive protein (139 mg/L, normal range 0–5 mg/L), serum ferritin (2100 μ g/L, normal range 22–322 μ g/L), fibrinogen (8.8 g/L, normal range 1.7–4.2 g/L) and D-dimer (25 963 mg/L, normal range <500 mg/L).

The diagnosis of COVID-19 was retained; based on microbiological data (positive RT-PCR on nasopharyngeal swab) and imaging (chest CT revealed multiple ground-glass opacities (Fig. 1a) with bilateral parenchymal consolidation, associated with pulmonary embolism).

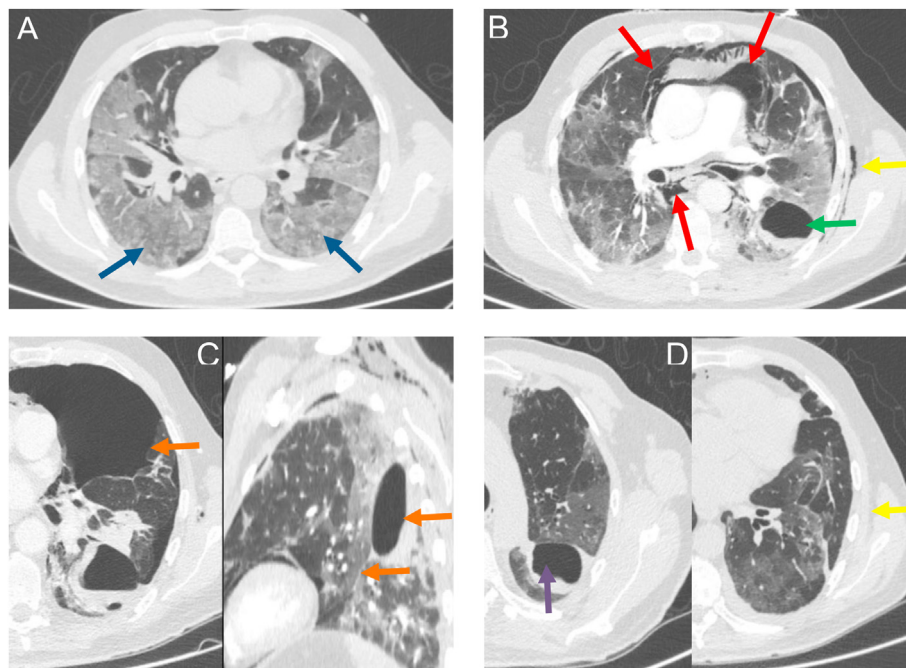


FIG. 1. (a) Chest CT showing bilateral ground-glass opacities (blue arrow) on day 1. (b) Chest CT showing persistent bilateral ground-glass opacities, important pneumomediastinum (red arrow), subcutaneous emphysema (yellow arrow) and a thin-walled lung cavity with an air–fluid level (green arrow), day 25. (c) Chest CT showing a left spontaneous intra-scissural pneumothorax (brown arrow), day 27. (d) Chest CT showing resolution of pneumomediastinum and subcutaneous emphysema (yellow arrow) and partial resolution of left pneumothorax (purple arrow), day 37.

He was given oxygen therapy, antibacterial and antiviral treatments (ceftriaxone 2 g/day for 7 days and lopinavir/ritonavir 800 mg/day for 7 days) and heparinotherapy. He also received tocilizumab (at a dosing regimen of 8 mg/kg in two intravenous infusions at 12-h intervals). Clinical outcome was favourable and he was discharged on 15 April.

On 22 April he was readmitted for exertional angina and dyspnoea after a prolonged cough. C-reactive protein was normal (0.3 mg/L) and high-sensitivity cardiac troponin I was also normal (<2.5 pg/mL, normal range <47 pg/mL). A SARS-CoV2 RT-PCR was positive on day 25. Chest CT showed multiple ground-glass opacities bilaterally with pneumomediastinum and subcutaneous emphysema (Fig. 1b). Oxygen support, analgesics and antitussives were introduced. On 24 April, chest pain persisted with dyspnoea and desaturation. Chest CT confirmed the diagnosis of left spontaneous pneumothorax (Fig. 1c) and a closed intercostal chest tube with a size 16 French was inserted. The chest tube was removed 3 days later, when imaging confirmed durable resolution of pneumomediastinum with partial resolution of the pneumothorax and reduction of parenchymal consolidation (Fig. 1d). On 5 May the patient was discharged for outpatient follow up.

Discussion

Most individuals with COVID-19 are diagnosed with pneumonia and characteristic CT imaging patterns, so radiological examinations have become vital in early diagnosis and assessment of disease course [1]. CT findings are frequently bilateral, multilobar and show peripheral ground-glass opacities with vascular enlargements. Consolidations often appear during progression as well as crazy paving and reticulation [1,2]. Lymphadenopathy, pleural effusions and complications such as mediastinal emphysema and pneumothorax are rare [2,3] and should raise concern for other disease. Chen et al. showed that only about 1% of individuals with COVID-19 have pneumothorax [4]. Pneumothorax and/or pneumomediastinum are more frequent in individuals with COVID-19 following tracheal intubation for invasive ventilation. This can be secondary to tracheobronchial injury along with the use of larger bore tracheal tubes and higher ventilation pressures [5].

The precise mechanism of spontaneous pneumothorax and/or pneumomediastinum in COVID-19 is unknown. To our knowledge, only few cases have been reported [6–10]. The particularity of our observation is that our patient had spontaneous

pneumothorax, pneumomediastinum and subcutaneous emphysema at the same time, with no past history of smoking or chronic obstructive pulmonary disease. The only risk factor of this patient was prolonged cough. Usually, spontaneous rupture of a small subpleural bulla is the cause of primary pneumothorax [11]. Spontaneous pneumomediastinum is defined as the presence or the appearance of free air in the mediastinum, typically in the absence of iatrogenic injury or external trauma. It is habitually an alveolar rupture caused by an increase in intrathoracic pressure, with a dissection of air through the bronchovascular sheath into the mediastinum. Subcutaneous emphysema then occurs when air penetrates the tissues under the skin [12].

In our case, we hypothesize that the mechanism for these simultaneous complications is the aggressive disease pathophysiology in COVID-19, which carries an increased risk of alveolar damage.

Conclusion

Acute deterioration with rapid oxygen desaturation and several parenchymal involvements in an individual with COVID-19 could indicate pneumothorax and/or pneumomediastinum and requires the performance of CT control.

Authors' contributions

SZ and TK contributed to drafting the manuscript. YBA contributed figure preparation and reviewed the final draft. CM, VG and TC also reviewed the final draft.

Conflicts of interest

The authors have stated that there are no conflict of interests in relation to this article.

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