Spontaneous pneumomediastinum as a complication of COVID-19

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Abstract
In December 2019, an atypical form of severe pneumonia emerged in Wuhan in China’s Hubei province, which in February 2020 was named COVID-19. The disease may have features of interstitial pneumonia and severe respiratory failure requiring intensive oxygen therapy. Spontaneous pneumomediastinum is a rare pathological condition with air in the mediastinum outside the trachea, oesophagus and bronchi. It is potentially life-threatening complication of both invasive and non-invasive mechanical ventilation. There have been reports that it may complicate the course of interstitial lung disease in the course of COVID-19. The report describes two cases of young patients who spontaneously developed this complication. Immediate diagnose is important in order to applicable adequate procedures.

Key words
pneumomediastinum, COVID-19, SARS-CoV-2

INTRODUCTION
In December 2019, an atypical form of severe pneumonia emerged in Wuhan in China’s Hubei province, which in February 2020 was named COVID-19. The pathogen causing the disease was referred to as SARS-CoV-2 virus. Shortly thereafter, as early as March of that year, the World Health Organization (WHO) declared that we were facing a pandemic, and the unusually rapid worldwide spread of the severe acute respiratory syndrome coronavirus (SARS-CoV-2) caused a significant public health crisis in the 21st century.

SARS-CoV-2 virus attacks the cells of the lower respiratory tract leading to inflammatory changes in the lung parenchyma. Extrapulmonary manifestations of the disease are also known in the form of myocarditis, cardiac arrhythmias, acute coronary syndromes, neurological symptoms (headache, stroke), blood coagulation disorders and vascular lesions, gastrointestinal symptoms (diarrhea, nausea and vomiting), liver and kidney damage. Numerous complications of COVID-19 include the risk of pneumothorax and pneumomediastinum, both spontaneous and associated with mechanical ventilation. Pneumomediastinum is a very rare complication with an incidence of 1.2–3.0/100000 cases of COVID-19 in retrospective analyses. The report presents two cases of young patients who spontaneously developed this complication.

CASE STUDIES
Patient 1. On 9 March 2021, a 36-year-old patient was admitted to the Communicable Diseases Unit for severe pneumonia involving approximately 80% of the pulmonary parenchyma (Fig. 1: A and B) in the course of SARS-CoV-2 infection confirmed by RT-PCR test 6 days previously – 3 March 2021. On admission, the patient reported increased dry cough, dyspnea and fever above 38°C. Symptoms had started 10 days earlier – 28 February 2021. Vital signs recorded on admission showed blood pressure (BP) 124/74 mmHg, heart rate (HR) 78 / min, hyperventilation 30 breaths / minute and peripheral oxygen saturation (SpO2) 90% with passive oxygen therapy. Arterial blood gas analysis showed oxygen partial pressure (pO2) 47 mmHg and arterial oxygen saturation (SaO2) 81% on oxygen therapy with a mask with a reservoir 151 / min (fraction of inspired oxygen [FiO2] 0.9). Computed tomography (CT) of the chest revealed a slight pneumomediastinum, which did not require thoracic surgery. Due to the deteriorating parameters of the arterial blood gas, numerous desaturations and significant respiratory effort, a decision was made to use high-flow oxygen therapy – initially the flow of 35 l / min, then 50 l / min with FiO2 0.85–1.0. No increase in mediastinal pneumothorax was observed in the control computed tomography. Several microperforations of the tracheal wall and main bronchi just below the tracheal bifurcation were visualized (Fig. 1: C, D, E, F), which may have been caused by intense coughing fits and may have resulted in pneumothorax. Due to the patient’s serious condition, a bronchoscopy was not performed to confirm tracheal perforation.

The patient’s conservative treatment included broad-spectrum antibiotic therapy, corticosteroids, low-molecular-weight heparin, tocolizumab, and cough suppressants. In addition, the patient received 1 packet of plasma from COVID-19 convalescents (ratio 5.91). In control arterial blood gasometry, low oxygen pressure (pO2 58 mmHg) and borderline saturation values (SaO2 90%) were observed, despite the treatment. Significant respiratory effort persisted. Since the treatment effects were unsatisfactory, on day 2 of hospitalization the patient was transferred to the Clinical Intensive Care Unit, where non-invasive ventilation (NIV) spontaneous breathing support was administered using a ventilation helmet. Continuous non-invasive ventilation on pressure support ventilation (PSV) was provided until 16
Figure 1. A and B – Patient No. 1: visible ground glass opacities involving approximately 80% of pulmonary parenchyma. C and D – Patient No. 1: visible microperforations of the tracheal wall and main bronchi just below the tracheal bifurcation. E and F – Patient No. 1: visible microperforations of the tracheal wall and main bronchi just below the tracheal bifurcation. G and H – Patient No. 2: visible scattered ground glass attenuations typical for COVID-19.
March 2021, after which the patient required only passive oxygen therapy. On 18 March 2021, the patient, in overall good condition, was transferred to another hospital to continue pulmonary rehabilitation.

**Patient 2.** On 16 May 2021, a 40-year-old patient was transferred to the Clinical Hospital with severe pneumonia involving approximately 50% of the pulmonary parenchyma in the course of COVID-19 (Fig. 1: G and H), confirmed by RT-PCR test on 2 May 2021. During the previous stay, the patient required high-flow oxygen therapy for 10 days. Unfortunately, the life-saving oxygen therapy caused the patient to develop nasal mucosal rhinitis, manifested by the production of a large amount of mucous discharge. During the patient’s nostril toilets, the patient felt a sudden pain in the chest followed by an increase in the neck and facial circumference. Palpation revealed subcutaneous air bubbles in the neck, face, chest, and upper extremities, as far as the forearms.

A chest CT scan confirmed a left-sided pneumothorax and massive pneumomediastinum with secondary subcutaneous and intramuscular emphysema (Fig. 2: I and J). On admission, the patient had no fever, BP 125/85 mmHg, HR 94/min, tachypnoe 30 breaths/minute, SpO2 93% on breathing through a reservoir mask. Immediately after transfer of the patient, active drainage of the left pleural cavity was applied, which continued for up to 17 days (10 days in the Communicable Diseases Unit and 7 days in the Thoracic Surgery Unit). It was only during this time that a satisfactory effect was obtained in the form of a significant reduction in subcutaneous emphysema and complete relaxation of the left lung. During the stay, the patient received passive oxygen therapy, initially through a mask with a reservoir, then through oxygen whiskers. High-flow oxygen therapy was discontinued due to concerns about possible pneumothorax enlargement. After removal of the drain, the patient was discharged home with significant clinical improvement. The patient did not require home oxygen therapy.

**DISCUSSION**

Spontaneous pneumomediastinum has a low incidence and is most often caused by increased airway pressure due to mechanical ventilation, but it may also occur during severe coughing, persistent vomiting, strenuous physical activity, chest trauma or surgery, oesophageal rupture, or alveolar damage in the course of infection. Most cases are self-limiting and are treated conservatively, but patients should be carefully monitored because of the potential for life-threatening cardiovascular and respiratory pathologies [1, 2].

Infection with the coronavirus SARS-CoV-2, which causes COVID-19, is mild in most cases, but can become severe with inflammatory changes in the lung parenchyma, and lead to extensive respiratory damage and respiratory failure which can be fatal. Spontaneous pneumomediastinum is one of the complications of COVID-19. To date, only a few incidents have been reported of spontaneous pneumomediastinum during COVID-19 under non-mechanical ventilation, although cases complicated with pneumothorax have been described [3, 4]. Pneumomediastinum in COVID-19 may result from diffuse alveolar damage caused by cytokine storm or the direct effect of SARS-CoV-2 virus on type I and II pneumocytes; thus, it may be a factor indicating extensive destruction of the alveolar membrane. Increased intra-alveolar pressure due to violent cough accompanying infection may be the cause of the development of pneumothorax in patients with COVID-19 [5, 6, 7].

Prompt diagnosis of COVID-19 complicated by pneumomediastinum is important, and attention should be paid to the occurrence of sudden chest pain, tachycardia, and desaturation in patients with severe pneumonia in the course of SARS-CoV-2 coronavirus infection. Chest CT and close monitoring are indicated because of the potential for complications such as pneumothorax and tension mediastinal pneumothorax, which are characterized by a high mortality rate. There are no consistent guidelines for the management of patients with pneumomediastinum in COVID-19, but the high number of patients worldwide suggests that clinicians should be aware of pneumomediastinum as a serious complication. The development of pneumomediastinum in COVID-19 infection is considered a possible indicator of disease worsening. Further data are needed to determine the mechanisms, incidence, risk factors, and prognostic role of this rare complication of the clinical course of COVID-19.
REFERENCES


