Polish Annals of Medicine

Journal homepage: https://www.paom.pl

Case report

Spontaneus pneumomediastinum and subcutaneus emphysema in nonventilated COVID-19 patient

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Article info

Article history
Received: March 24, 2022
Accepted: August 11, 2022
Available online: September 9, 2022

Keywords
Subcutaneous emphysema
Noninvasive ventilation
Pneumomediastinum

Doi
https://doi.org/10.29089/paom/152734

Abstract

Introduction: The most common complications of COVID-19 infection are: pneumonia, acute respiratory distress syndrome, pulmonary fibrosis, pulmonary embolism, pneumothorax and pneumomediastinum.

Aim: We would like to highlight the rarity of pneumomediastinum and subcutaneous emphysema in nonventilated COVID-19 patient.

Case study: A 50-year-old man was addmitted to the COVID-19 Department with SARS-CoV-2 pneumonia. The patient wasn't vaccinated against COVID- 19. Upon admission the general condition was quite good with mild dyspnea.

Results and discussion: Upon admission a CT scan was performed in which there were bilateral infiltrates consistent with COVID-19 infection, covering approximately 50% of the lungs. On the 5th day of hospitalization the general condition deteriorated and a drop in saturation was observed. A follow-up CT scan revealed progression of lung inflammatory changes that spanned approximately 60%–70% of lung parenchyma; there was pneumomediastinum and subcutaneous emphysema in the neck, left subclavian, and axillary area. High flow nasal oxygen therapy (60 L/min) was administered. On the 13th day the general condition of the patient further deteriorated and blood saturation continued to drop which prompted the decision to escalate treatment. Initially, noninvasive ventilation was used, however, shortly after the patient was intubated. Immediately after intubation the patient went into cardiac arrest that ultimately led to his death.

Conclusions: The development of spontaneus pneumomediastinum in the patient can be mainly attributed to the intense cough and rapidly developing acute respiratory distress syndrome in the course of SARS-CoV-2 infection despite aggressive treatment with steroids, tolicizumab, and antibiotics for staphylococcal pneumonia.

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1. INTRODUCTION

Subcutaneous emphysema (SE) and spontaneus pneumomediastinum (SPM) refer to the presence of air in the subcutaneous tissue and mediastinum. SPM results from a sudden rise in intra-alveolar pressure, resulting in the rupture of alveoli and subsequent dissection of air along the bronchovascular sheath into the mediastinum (Macklin effect).[1,2] The most common cause of pneumothorax is trauma to the neck, chest, abdominal cavity, and mechanical ventilation. Rarely it may accompany diseases such as bronchial asthma, pulmonary tuberculosis, lung abscess, acute respiratory distress syndrome (ARDS), or simply cough.

2. AIM

We would like to highlight the rarity of SE and SPM in a nonventilated COVID-19 patient.

3. CASE STUDY

A 50-year-old, physically active male was admitted to the COVID-19 Department due to worsening of shortness of breath/dyspnoea, cough, weakness and fever up to 39.8°C for about 8 days. On the 7 days before hospitalization the patient tested positive for SARS-CoV-2 in a RT-PCR test. The patient wasn't vaccinated against COVID-19. He had no comorbidities or drug intake. He denied drug allergies, tobacco, or alcohol intake. Upon admission the general condition of the patient was quite good and the patient was experiencing mild dyspnea. Arterial saturation without oxygen therapy was 90%. When administering oxygen supplementation of 8 L/min via a face mask the saturation increased to 94%, blood pressure 139/93 mm Hg, heart rate 108 bpm. Arterial blood gas analysis revealed elevated CRP 156.7 mg/L (normal ranges 0–5 mg/L), D-dimer 1.24 μl/mL (<0.5 μl/mL), AST 194 U/L (0–50 U/L), and ALT 46 U/L (0–50 U/L), hyponatremia 131 mmol/L (136–145 mmol/L), slightly elevated interleukin 6 level 58 pg/mL (<6 pg/mL), lymphopenia 11.60% (1%–3%), thrombocytopenia 87000/μl (150–400/ μl), eosinopenia 0% (1%–6%). The arterial blood gas panel displayed pH 7.5 (7.35–7.45), $pC0_2$ 25.4 mm Hg (35–48 mm Hg), $p0_2$ 63.5 mm Hg (75–100 mm Hg), oxygen saturation 93.9% (94%–100%), HCO_3 19.5 mmol/L (21–26 mmol/L); mono CT scan showed ground-glass opacity with 50% distribution bilaterally (Figure 1A). Therapy as recommended by the Polish Society of Epidemiology and Physicians of Infectious Diseases was initiated. The patient was given steroids, intermediate dose of low molecular weight heparin, empirical and then targeted antibiotic therapy, intravenous fluids, and antipyretic drugs. The patient required face mask oxygenation therapy (6–15 L/min). A nasopharyngeal culture detected *Staphylococcus aureus*. Echocardiography indicated no pathologies, ejection fraction of approximately 55%, and low probability of pulmonary hypertension (PAH). After initial improvement of the patient's status, we observed gradual deterioration of the patient's general condition (resting saturation decreased to 80%–85%, CRP increased to 178 mg/L, in arterial blood gas analysis $p0_2$ 55.8 mm Hg, saturation 88%). Follow up examinations revealed significantly elevated interleukin 6 levels (97.83 pg/mL) and further progression of lungs inflammatory changes to approximately 60%–70% of the lungs. Moreover, pneumomediastinum and SE were identified in the neck, left subclavian, and axillary area. There were no signs of accompanying pneumothorax in the CT scan (Figures 1B and 2B). High flow nasal oxygenation was started with an oxygen flow of about 60 L/min (95% O_2), prone position was used, and tolicizumab was administered. During the next few days we observed an improvement which allowed us to reduce the FiO_2 to 65%–70%. On 13th day of hospitalization the patient developed a severe cough and ultimately desaturated to 75%–78%. Despite increasing the oxygen flow during high flow nasal oxygen therapy, we observed no significant improvement in the saturation level and general condition of the patient. Noninvasive ventilation (NIV) with continuous positive airway pressure (CPAP) was used. Because of pneumomediastinum and SE low positive and expiratory pressure (PEEP) was utilized (8 cm H_2O) with a FiO_2 value of 100%. The patient was stable for next few hours with a saturation of 93%–95%, blood pressure 151/94 mm Hg, heart rate 88 bpm. Eventually, due to respiratory

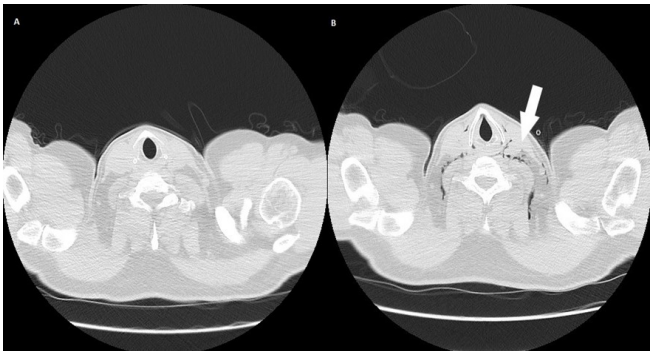

Figure 1. (A) ground-glass opacity with 50% distribution bilaterally (CT on admission); (B) ground-glass opacity with 60%–70%, distribution bilaterally, pneumomediastinum (arrows).

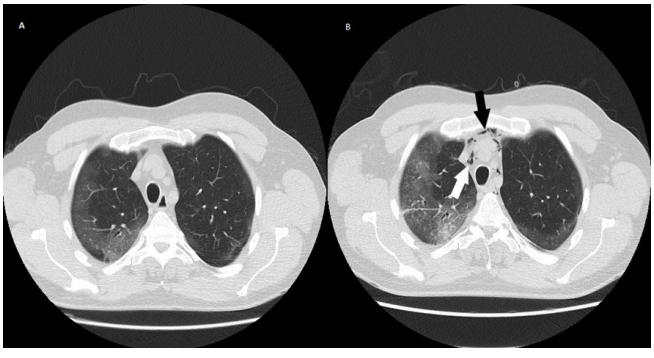

Figure 2. (A) CT scan of the neck region (on admission); (B) subcutaneous emphysema in the neck (arrow).

exhaustion the patient was intubated and mechanically ventilated. Shortly after intubation the patient went into cardiac arrest and cardiopulmonary resuscitation was conducted for nearly an hour during which 7 mg of adrenaline was administered. Despite the resuscitation the patient died.

4. RESULTS AND DISCUSSION

The most common complications of COVID-19 infection are pulmonary complications such as: pneumonia, ARDS, pulmonary fibrosis, pulmonary embolism, PAH, pneumothorax, pneumomediastinum.[3] The most common cause of pneumothorax is trauma to the neck, chest, abdominal cavity, and mechanical ventilation. Rarely it may accompany diseases such as bronchial asthma, pulmonary tuberculosis, lung abscess, ARDS, or simply cough. Spontaneous pneumomediastinum most commonly arises in the setting of increased intra-alveolar pressure, which causes rupture of alveoli and subsequent dissection of air along the bronchovascular sheath into the mediastinum. In the general population the average frequency of SE and SPM is 1.2 to 3 per 100 thousand people.[4,5] In literature, it is a common to come across descriptions referring to ventilated patients with SPM, however, SPM unrelated to mechanical ventilation is a newly described complication of COVID-19 with only a few cases described so far. Vazzana et al. from Italy described very similar case in 80-year old man with pneumomediastinum and SE after NIV COVID-19 patient.[6] Analyzing the previous SARS outburst in 2003. Peiris at al. demonstrated that 12% of patients with SARS developed SPM unrelated to intubation and mechanical ventilation.[7] Very similar results were obtained in a small study by Chu et al. SPM developed in 11.6% of the SARS cohort at 19.6 ± 4.6 days from the onset of symptoms; a high peak LDH level was associated with its development.[5] Moreover, development of SP correlated to a significantly high intubation and mortality rate.[8] SPM is uncommon in viral pneumonia, although, it has been reported in cases with SARS-associated coronavirus pneumonia.[9,10] SPM can also lead to other complications such as extensive pneumothorax (PNX) and extensive SE which was observed in the described patient. Both of these clinical conditions are affiliated with a poor prognosis and the low chance of survival. Additionally, SPM can cause a rare complication such as staphylococcal pneumonia and fungal pneumonia.[11,12] In our case the nasopharyngeal culture tested positive for *S. aureus* during admission, which could have influenced the final outcome, even though initial empiric and then directed antibiotic therapy was administered. In literature there is little evidence pointing to staphylococal pneumonia as the causitive source of SPM. The cases that illustrate this correlation are outdated and refer to immunoincompetent patients.[13] In the presented case the immune status of the patient is affected due to the viral infection itself as well as the use of tolicizumab and steroids. Aggressive steroid therapy has also been speculated to play a significant role in the pathogenesis of spontaneous pneumothorax in SARS patients,[12] however, this theory does not have significant evidence and steroids remain useful in restricting the rapid and damaging inflammatory response seen in viral pneumonia, especially in the course of SARS-CoV-2 infection.[14,15] No evidence was found in regards to tolicizumab influencing the risk of SPM in patients with viral pneumonia, particularly in coraonvirus pneumonitis.

Based on the above differentiated conditions, the main reasons accounting for the development of PMS in the presented patient is the intense cough and rapidly developing ARDS in the course SARS-CoV-2 pneumonitis despite aggressive treatment with steroids, tolicizumab and antibiotics for staphylococcal pneumonia. No other sources of mediastinal air could be identified in the course of the diagnostic procedures. While most cases of spontaneous pneumomediastinum are self-limited and could be managed conservatively, the condition can lead to life threatening circulatory and respiratory pathology as it happened in the described case.[16]

5. CONCLUSIONS

(1) There are many pulmonary complications of COVID-19 patients, but the pneumomediastinum and SE are not very often in nonventilated patients.
(2) Both of these conditions can be accounted by the SARS-CoV-2 inflammation, damage in the alveolar epithelium, ARDS and persistent exertive cough.
(3) Considering the growing number of patients with COVID-19 pneumonia and its complications, an attentive description of such cases and their treatment is necessary to deal with the very high mortality rate, which seems to be dominant in the available cases described.

Conflict of interest
No conflict of interest.

Funding
This manuscript is not funded by any organization.

Acknowledgments
We would like to thank the COVID-19 Department in University Clinical Hospital in Olsztyn for contribution in getting patient's data and images.

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