ISSN: 2833-9312

Case report

World Journal of Clinical & Medical Images

Pneumomediastinum In Covid-19 Infection with Fatal Outcome

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Submitted: 26 Oct 2022; **Accepted:** 31 Oct 2022; **Published:** 12 Nov 2022

Citation: *M. Benabdellah, Y. Motiaa, M. A. El Yaacoubi, S. Labib , H. Sbai. Pneumomediastinum In Covid-19 Infection with Fatal Outcome, World J Clin Med Img, 2022, 1(2), 103-105*

Abstract

We report the case of a 51-year-old female patient admitted to the intensive care unit for a severe form of SARS COV 2 pneumonia. The diagnosis based on suggestive clinical symptoms and confirmed by a non-injected CT revealed pneumomediastinum, associated to lung injuries extended to 75% of pulmonary parenchyma. After 5 days, the patient was initially put on oxygen therapy by non-rebreather mask and then on high flow nasal cannula, followed by intermittent noninvasive ventilation NIV. The patient died of respiratory failure 4 days later. We will discuss the mechanisms and risk factors for the occurrence of spontaneous pneumomediastinum during Covid-19 infection and the appropriate means of oxygen therapy.

Keywords: COVID-19, pneumomediastinum, outcome, inflammation.

Introduction

Coronavirus infection is caused by a new virus, SARS-CoV 2 (Several Acute Respiratory Syndrome Coronavirus 2), which appeared in China in December 2019 and is the cause of the current pandemic [1]. The first cases were reported in Wuhan, a Chinese city in Hubei province. Although the suggestive symptoms of coronavirus infection are similar to those of other viral infections, many associated symptoms are not described or explained [2], including spontaneous pneumomediastinum PMS.

Pneumomediastinum or mediastinal emphysema, defined as the presence of air in the mediastinum, can be incidental: traumatic or spontaneous. In patients admitted to an intensive care unit, iatrogenic PM may occur during mechanical ventilation: invasive or non-invasive or during intubation that has caused tracheal trauma. [1]

During the 2002-2003 SARS outbreak, spontaneous pneumomediastinum PMS was described in several patients. However, it has been rarely reported in Covid-19 [2].

We share an observation of PMS in a patient admitted for SARS CoV2 pneumonia to the intensive care unit during the second wave, with fatal outcome.

Case report

A 51 years old women, with no past medical history, unvaccinated for SARS-COV2, admitted to the intensive care unit, on July 2021, for hypoxemic viral pneumopathy due to Sars Cov 2 was diagnosed with a 5 days dry cough history, vomiting, headache, anosmia and fever. As the patient's shortness of breath worsened, a thoracic CT scan was performed, which showed a multifocal bilateral ground-glass opacities (typical pattern of SARS-CoV-2 pulmonary infection) affecting 75 % of the pulmonary parenchyma, with a medium-sized pneumomediastinum (figure 1). The Reverse transcriptase–polymer chain reaction (RT-PCR) was positive for SARS-COV-2.

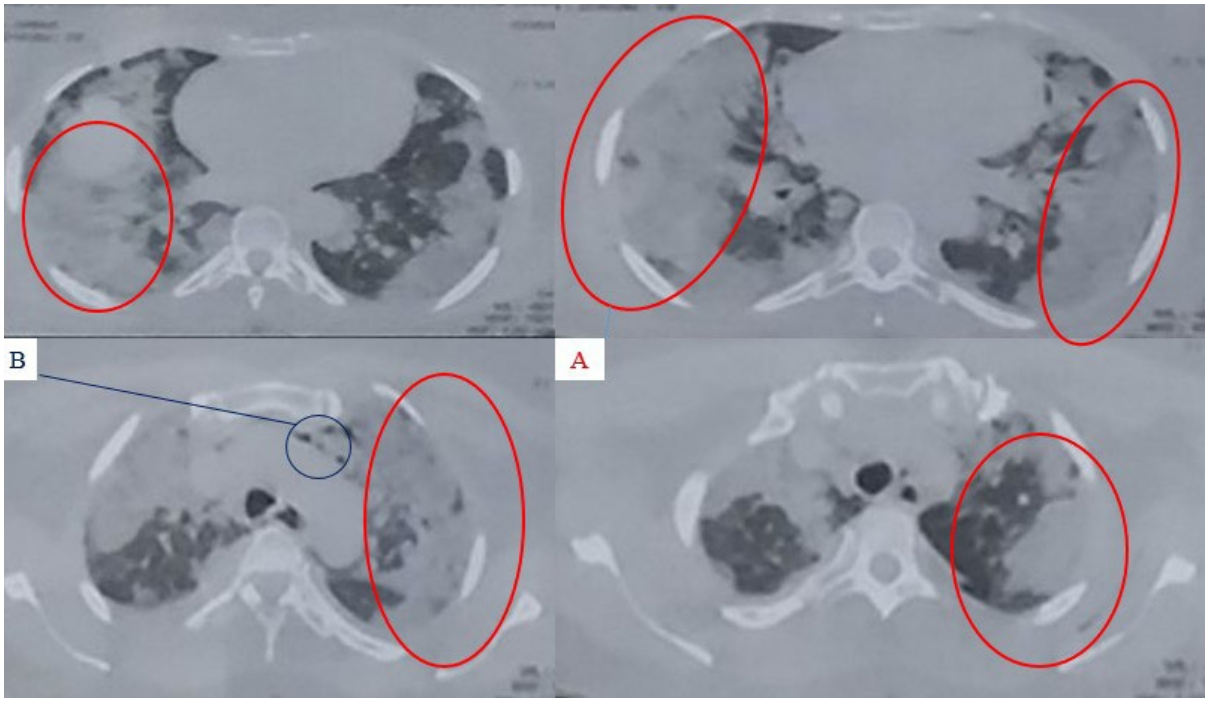

Figure 1 : chest coputed tomography

- A : multifocal bilateral ground-glass opacities
- B : pneumomediastinum

On examination, the patient was conscious; she was tachypneic with a respiratory rate of 30 breaths per minute, oxygen saturation of 64% on room air improved to 86% on 15 l/min of oxygen, via a non-rebreather mask, and an improvement to 91% on lateral decubitus; she was apyrexial, heart rate was 105 beats per minute and blood pressure 130/90 mmHg, capillary blood glucose was 102mg/dl. There was no subcutaneous emphysema and no dysphonia. Arterial blood gas on 100% oxygen showed a respiratory alkalosis with a pH of 7.53, PCO2 of 33 mmHg, PO2 of 71.8 mmHg, and HCO3 - of 27.5 mmol/L, base excess of 5.

The patient was started on methylprednisolone 80 mg intravenously per day, vitamin C, vitamin D and Zinc. Therapeutic anticoagulation was started using enoxaparin 1 mg/kg twice daily, LMWH 0.8/12h, rapid insulin depending on the dextro. Biological investigation on admission showed leukocytosis at 9700/mm3, lymphopenia at 1106/mm3, an elevated C-reactive protein level of 430 mg/l, lactate dehydrogenase was 983 UI/l, ferritin level was 300 ng/ml, D-dimer was 1980 µg/L and fibrinogen at 8g/l.

The remainder of her blood test results were within normal range, including renal function, hepatic function tests, and platelet count.

Twenty-four hours after her admission, the patient's respiratory status worsened with polypnea and desaturation despite 15 l/min of oxygen via a non- rebreather mask, imposing the use of high flow nasal cannula (HFNC, FIO2: 100%, flow: 70 l/min). The patient was placed on prone position, with intravenous infusion of 800 mg Tocilizumab for cytokine storm syndrome.

On the day 4 of her admission, the respiratory status continued to deteriorate (respiratory rate 40 breaths per minute, saturation at 80% under HFNC), indicating the use of non invasive ventilation (NIV) (PEEP: 6 cmH2O; pressure support PS: 15 cmH2O; pressure-assisted ventilation mode, via a full-face mask) without clinical improvement and without any subcutaneous emphysema. Arterial blood gas showed a respiratory acidosis with severe hypoxemia after two sessions of NIV (Ph : 7.31, PCo2= 51.8 mmHg, PO2 to 221 mmHg, HCO3- 25.6 mmol/L).

The patient was intubated for clinical worsening despite NIV and was ventilated on assisted control volume mode. It was difficult to maintain oxygenation through mechanical ventilation, proning and use of neuromuscular blockade agent infusion. She eventually died of ARDS.

Discussion

Pneumomediastinum associated with SARS-COV2 pneumonia was reported before and it is associated with alveolar damage, secondary to a hyperinflammation state. Lymphatic infiltration and alveolar exudation are generated by viral particles invasion of the alveolar wall and interlobular septum [3].

The physiopathology of pneumomediastinum was described by Macklin in 1939: it involves an alveolar rupture followed by the bronchovascular walls' dissection by the released air. This air, following the bronchial walls, spreads into the mediastinum. Two mechanisms create this pressure gradient: the increase in intra-alveolar pressure or the decrease in pressure in the peri-alveolar interstitial space. The first mechanism is seen in the intentional Valsalva maneuver or similar conditions such as coughing, sneezing, nausea and vomiting. The second mechanism is during extreme respiratory effort, diabetic ketosis and rapid reduction of atmospheric pressure [3, 4], in addition SARS-COV may cause diffuse alveolar damage and interstitial emphysema, this was confirmed for SARS and Middle East Re-

spiratory Syndrome (MERS) virus and in post mortem studies for SARS-COV2 [5]. In addition, the use of corticosteroids for several days, even at a low dose, can contribute to the phenomenon of weakened interstitial tissue.

The diagnosis is based on chest X-rays and especially chest Computed tomography, which allow to evaluate the severity and extension of lung injuries, to research bacterial superinfection and to diagnose other complications, especially pneumothorax, which can modify the therapeutic approach. No specific therapy seems to have proven its effectiveness, the evolution is towards recovery and the treatment is symptomatic.

The initial Chinese and American studies described no cases of PMS, while an Iranian study found a proportion of 16.6% in patients with COVID 19 with no previous pulmonary history and no use of mechanical ventilation. Given these results, further research was carried out and 7 cases of PMS were found. In their studies, they found that the PMS may or may not be associated with pneumothorax; it may or may not be associated with subcutaneous emphysema. Patients had no previous respiratory history [6-11].

Our patient had an isolated pneumomediastinum without pneumothorax, associated with a severe form of SARS-COV2 pneumonia. Chest CT showed lung injuries affecting 75% of pulmonary parenchyma, in addition to cytokine storm syndrome reflecting hyperinflammation reaction induced by immunity activation. This CSS was diagnosed upon clinical deterioration and biological parameters (CRP, LDH, Ferritin and d-dimeres) for which tocilizumab was administered, without clinical status improvement. The acute respiratory failure was managed by using HFNC, NIV and intubation while monitoring clinical parameters (saturation, subcutaneous emphysema), airway pressure (plateau pressure) and arterial blood gas. ECMO was not available in our hospital which could be discussed especially after using neuromuscular blockade agent and prone position.

Conclusion

Pneumomediastinum associated with SARS-COV2 pneumonia is linked to alveolar damage secondary to hyperinflammation status. This complication may be associated with this disease's severity. More studies are needed to analyze this association.

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