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Spontaneous Pneumomediastinum in COVID-19 Unrelated to Mechanical Ventilation: A Case Report

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Abstract

BACKGROUND: Pneumomediastinum is a rare disease associated with barotrauma and uncommonly occurs in viral pneumonia. Although the underlying mechanism of the incidence of pneumomediastinum in COVID-19 patients is not fully understood, barotrauma is the most probable cause.

CASE REPORT: We reported a case of a 27-year-old woman with the chief complaint that was shortness of breath and diagnosed with COVID-19 based on reverse transcription polymerase chain reaction examination. On the 6th day after being admitted to the hospital, suddenly, the intensity of dyspnea was increased with the decrease of oxygen saturation. Computerized tomography of the chest confirmed pneumomediastinum and pneumonia COVID-19. There was no improvement of symptoms after oxygen and steroid administration. Emergency thoracotomy was not performed; yet, and the patient has died.

CONCLUSIONS: Although pneumomediastinum is benign disease and self-limited disease, the presents of pneumomediastinum may relate to worse outcomes in COVID-19 infections.

Introduction

The first case of COVID-19 in Indonesia was reported on March 2, 2020 with a clinical appearance that was moderate symptoms, including fever, cough, and shortness of breath. Then, it spreads all over the countries until it reaches 143,445,675 cases per April 22, 2020, with a mortality rate was 3,051,736 cases [1]. The symptoms were varied from mild disease, including fever, cough, anosmia, ageusia, rhinorrhea, and nasal obstruction until breathing failure [2]. The progression of disease was affected by the immunological state of the patient, comorbid, and the laboratory results [3].

COVID-19 involved pulmonary and extrapulmonary manifestations. Chest computerized tomography (CT) scan was a substantial diagnostic tool for assessing the pulmonary manifestation, particularly ground-glass opacities, and consolidation. Further, a chest CT scan can describe the mediastinal involvement in COVID-19 manifestation [4], [5]. Pneumomediastinum is an uncommon manifestation of COVID-19 that can be diagnosed from the chest CT Scan [2], [6]. In the SARS-COV-1 pandemic in 2002, a retrospective study revealed that there were 13 from 123 cases of COVID-19 infections diagnosed with pneumomediastinum. From those cases, 5% of cases were needed a mechanical ventilator, and four patients were reported dead [7].

The mechanism of pneumomediastinum in COVID-19 still becomes controversial due to many aspects that can be related to the presence of air in mediastinum space [8]. The accumulation of air in the mediastinal space cause pressure to the few vital organs in the mediastinum, including great vessels, trachea, and heart. Few cases reported spontaneous resolution, but few studies revealed the need for invasive intervention and related to the poor prognosis [6]. Here, we present a case of a 27-year-old pregnant woman with a diagnosis of long COVID-19 and complicated with pneumomediastinum.

Case Report

A 27-year-old-pregnant woman with 35 weeks of gravida was admitted to the hospital due to a chief

complaint of fever for 1 day. She has come from the high-risk zone of COVID-19 for 2 weeks. There was no history of comorbidities, including hypertension, diabetes mellitus, cardiovascular disease, kidney disease, and malignancy. On admission, she was stable without any complaints besides moderate grade of fever with 38.3°C and oxygen saturation was 98% room air. Reverse transcription polymerase chain reaction test revealed positive results, and the patient was charged in the isolation room. Three days on admission, there was a sudden progression of disease with a high fever reached 39.5°C. Emergency sectio-cesarean (SC) procedure was performed due to fetal distress, and the patient's condition was stable after the SC procedure. However, on the 9th day on day admission, the patient was desaturated, with average oxygen saturation was 80–85%. There was no good clinical progression on the 9th–25th of the day of symptoms. The patient remained short of breath with oxygen saturation 86–90% in using a 15 l/min non-rebreathing mask. On the 25th day of symptoms, the patient was voluntarily transferred into our hospital.

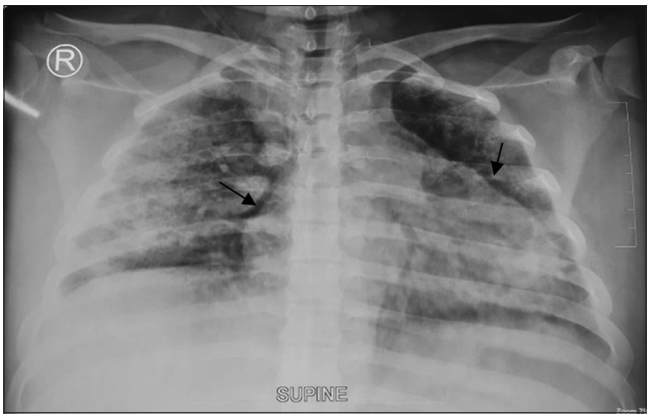

Figure 1: Chest X-ray revealed massive fibrotic and infiltrate in both of lung zone

In the emergency room, the patient still feels shortness of breath with oxygen saturation was 92%, with oxygen administration was 5 l in the nasal cannula. She also experienced a cough with thick whitish sputum without bloody sputum. Chest pain was experienced with the characteristics, which was triggered on breathing with VAS 4. There were no additional symptoms of COVID-19 experienced by the patient. On the presentation, his respiratory rate was 26/min, heart rate 104/min, blood pressure 134/78 mmHg, and axial temperature was 36.8°C. Chest examination revealed marked shortness of breath with coarse crackles in both hemithorax and the use of inspiratory accessory muscles. Heart examination demonstrated normal heart sound with no additional sound, including murmur, gallop, bruits, and rubs. Chest X-ray revealed massive fibrotic and infiltrate in both lung zone (Figure 1). Laboratory findings showed an increase in leucocyte count (17.2 × 10^3/μl) and d-dimer (1100.8 μ/L). Chest CT scan showed a massive crazy-paving pattern with free air in

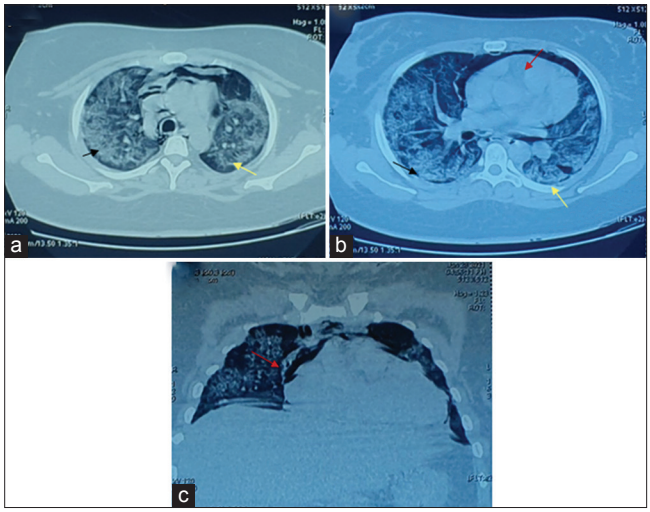

Figure 2: From axial chest CT showing peripheral crazy paving pattern multifocal in right upper, middle, and lower lung lobe (Figure AB black arrow) and left upper, lower lung lobe (Figure AB yellow arrow), small amounts of gas appear as curvilinear lucencies outlining major aortic branches and superior vena cava (Figure 2a white arrow), and large pneumopericardium (Figure 2b red arrow). Coronal lung window showing very extensive pneumomediastinum with no pneumothorax (Figure 2c red arrow)

mediastinum space around trachea, heart, and right/left bronchus (Figure 2a-c). The patient was diagnosed with long COVID-19 with pneumomediastinum and lung fibrosis. Oxygen, broad-spectrum antibiotics, bronchodilator and steroid inhalation, and multivitamins were administered to control symptoms. Chest physiotherapy was performed to rehabilitate the lung function. Close monitoring of blood pressure, oxygen saturation, heart rate, and respiratory rate were carried out in the wardroom. Clinical conditions progressively worsened, and the patient died on the 34th day of symptoms with respiratory failure.

Discussion

SARS-COV-2 is a coronavirus that invades all organs with the expression of the Angiotensin-Converting Enzyme 2 (ACE-2) receptor [9]. Lung has the highest expression of ACE-2 receptor, so the respiratory symptoms, including shortness of breath, cough, chest pain, and fever, were the main complaints of COVID-19 [10]. The symptoms of COVID-19 varied from mild disease to severe respiratory failure that needs a mechanical ventilator. Host response to virus particles played an important role in developing a hyperinflammatory state in COVID-19 infections [11]. Pregnancy is one of the predisposition factors related to developing severe COVID-19 manifestation due to the physiological and immunological change in the pregnancy period, including the change of pulmonary size, alteration in both innate and adaptive immunity, and hypercoagulable state [12].

In this presented case, we presented a pregnant woman with fever as the initial presentation and progressed into respiratory distress in 1 week. Emergency SC was performed due to fetal distress, although at that time, the patient did not experience desaturation yet. The patient begins experiencing shortness of breath after 9 days of symptoms. In the clinical course of COVID-19, the pulmonary phase, including progressed respiratory symptoms, begins after the 8th day when the inflammatory phase with cytokine storm occurs. Hence, the patient might present mild disease in the early presentation and progress into shortness of breath after the 8th day until reach ARDS after 12th days in certain populations [13], [14].

Chest CT scan played an essential role in the initial assessment of COVID-19. The most frequent presentation of COVID-19 was peripheral, multifocal, and bilateral ground-glass opacities with or without consolidation. In few cases, pseudonodular condensation may present. The atypical presentation also shows lung cavitation, smooth interlobular, septal thickening, and pleural effusion [15]. In an unusual case, the accumulation of free air in pleural space and mediastinum may be seen. A multifocal crazy paving pattern was presented in this case, with a small amount of air accumulation appear as curvilinear lucencies outlining major aortic branches and superior vena cava. The crazy-paving pattern is defined by multiple, patchy ground-glass opacities with reticular, and interlobular septal thickening and intralobular lines. It is pathognomonic in COVID-19 progressive disease, which may present in the inflammatory and hyperinflammatory state [16]. This is in line with these cases, in which Chest CT was performed after the 25th day when the patient was under cytokine storm period.

Pneumomediastinum is an uncommon manifestation in COVID-19 infections. Macklin hypothesizes that alveolar rupture is the basic mechanism for pneumomediastinum incidence [17]. However, the definite mechanism that triggers the alveolar rupture was unclear. Few factors are contributing to an increase in intrapulmonary pressure including coughing, sneezing, giving birth, and vomiting. Furthermore, other factors are cigarette smoking, marijuana consumption, chronic obstructive pulmonary disease, asthma, chronic bronchitis, and diabetic ketosis [4]. After alveolar rupture, the air enters the interstitial space and gets in through the perivascular and peribronchial sheath to the mediastinum space [6]. The occurrence of pneumomediastinum in mechanical ventilators applied was more common. Pulmonary barotrauma due to high tidal volume (above 12 mL/kg body weight) and peak pressure above 35 mmHg has been discussed as the pathophysiology of pneumomediastinum in pneumonia patients [18]. Nevertheless, the same tidal pressure was also applied in other patients who did not develop pneumomediastinum. Another factor that might precipitate; the alveolar rupture was the ability of SARS-COV-2 to invade the type 1 and type 2 epithelial cells and damage the integrity of the alveolar membrane due to a high level of inflammatory cytokines. This made the alveolar cells to be susceptible to rupture when there is a slight increase of airway pressure such as coughing, sneezing, or vomiting [5]. Furthermore, pneumomediastinum incidence is more common in COVID-19 infections which in our cases, there was no history of the mechanical ventilator and the history of previous medical history related to increasing the airway pressure. Hence, the most probable mechanism for the incidence of pneumomediastinum is more common in severe to a critical case of COVID-19.

Pneumomediastinum is a benign disease. There was no need for invasive management in spontaneous pneumomediastinum. Usually, after 2 days of observation, the patient can be discharged with long-term follow-up. The recurrence of spontaneous pneumomediastinum was rare if there was no complication, including massive emphysema or oesophagal rupture [19]. Close monitoring of oxygen saturation, blood pressure, respiratory rate, and heart rate was required in case of the disease progression. Oxygen administration must be carried out to increase the spontaneous resolve pneumomediastinum by the increase the nitrogen concentration [8]. In this case, 3 l of oxygen were administrated using a nasal cannula.

Clinical outcomes in pneumomediastinum in COVID-19 varied according to the severity of acute respiratory distress (ARDS) and the complication of pneumomediastinum, including massive emphysema and sepsis. In Kangas Dick's study, 24 of 34 patients were died from breathing failure and sepsis [20]. In our case, the patient died after 9 days of admission to our hospital due to breathing failure. Juarez Llcolla *et al.* also reported more than half of their patient were died because of breathing failure resulted in the progression of COVID-19 infections in the lung parenchyma. Massive destruction in pulmonary parenchyma interferes with the diffusion process in breathing physiology [6].

In conclusion, pneumomediastinum is a benign disease with multifactorial etiology. Although it is a self-limited disease, the presents of pneumomediastinum may interrupt the circulation and breathing physiology and further relates to worse outcomes in COVID-19 infections. Further study to analyze the pathophysiology of pneumomediastinum in COVID-19 infection was needed to assess the best management.

Consent for Publication

The patient was properly informed and provided consent for her clinical information to be included in the publication of this case report and the accompanying images.

Author's Contribution

NNS is involved in treating the patient and designing the article. CS, AGI and FRA participated in editing the manuscript critically. All authors declared that they contributed to this article and that they have read and approved the final manuscript.

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