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Pneumothorax Aggravated Respiratory Failure in a Patient with SARS-CoV-2 Pneumonia

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Abstract

High-flow oxygen inhalation, noninvasive and invasive ventilation were used for a 68-year-old female with laboratory-confirmed SARS-CoV-2 pneumonia successively. Secondary-infection, injury of liver and kidney, respiratory acidosis and coagulation disorder were revealed by laboratory findings. Two weeks after admission, chest radiograph showed pneumothorax. She died of respiratory failure four days later.

Keywords: Pneumothorax • Respiratory failure • SARS-CoV-2 • Acute respiratory distress syndrome

Introduction

Since the beginning of 2020, severe acute respiratory syndrome coronavirus 2(SARS-CoV-2) pneumonia has resulted in thousands of death in China. The clinical and radiological features of critically ill patients with SARS-CoV-2 infection have been widely reported. However, there are few publications reporting fatal cases with respiratory failure aggravated by pneumothorax [1-3].

Case Presentation

An 68-year-old female resident of Wuhan had fever, cough, expectoration, dyspnea, diarrhea and fatigue on the first days of February 2020. As reverse transcriptase polymerase chain reaction for SARS-CoV-2 was positive, she was admitted to designated hospital. Pulse oxygen saturation (SpO_2) at admission was 97%, while blood pressure, heart rate, breathing rate and pulse were normal [4-6].

On Feb 15, her SpO_2 dropped to 85% under high flow oxygen inhalation, thus noninvasive ventilation was used. 90%-99% SpO_2 was maintained with fraction of inspired oxygen (FiO_2) 70%-100%. The main abnormalities in laboratory findings were as follows: leukocyte count 9.76×10^9 L, neutrophil 8.86×10^9 L, lymphocyte 0.50×10^9 L, albumin 26.5 g/L, Glutamic Pyruvic Transaminase (GPT) 189 U/L, Glutamic Oxaloacetic Transaminase (GOT) 172 U/L, D-dimer 17.10 μg/ml FEU, Lactate Dehydrogenase (LDH) 712 U/L, ferritin 5325.4 μg/L. Abide, methylprednisolone and moxifloxacin were used for antiviral, anti-inflammatory and anti-infection respectively [7].

Invasive ventilation had to be used for her on Feb 19, as SpO_2 dropped to 84% under noninvasive ventilation with FiO2 100%, indicating severe Acute Respiratory Distress Syndrome (ARDS). The main laboratory findings after invasive ventilation were as follows: leukocyte count $4.78\text{-}17.64 \times 10^9$/L, neutrophils $4.64\text{-}16.19 \times 10^9$/L, lymphocyte $0.41\text{-}0.44 \times 10^9$ /L, platelet count $83.0\text{-}184.0 \times 10^9$/L, hemoglobin 93.0 g/L-135.0 g/L, glucose 8.20-14.66 mmol/L, total protein 56.3 g/L-57.8 g/L, albumin 26.2 g/L-27.6 g/L, total bilirubin 19.8 μmol/L-26.7 μmol/L, direct bilirubin 14.2 mol/L-19.6 mol/L, creatinine 180 μmol/L-220 μmol/L, urea 8.9 mmol/L-10.7 mmol/L, PH 7.239-7.325, partial pressure of CO_2 ($PaCO_2$) 75.9 mmHg-99.7 mmHg, partial pressure of O_2 (PaO_2) 47.1 mmHg-96 mmHg, lactate 1.7 mmol/l-7.6 mmol/l, prothrombin time 15.6-16.5 seconds, prothrombin activity 62.0%-64.0%, fibrinogen 7.58 g/L-8.47 g/L, D-dimer 3.66 μg/ml-6.74 μg/ml FEU, fibrinogen degradation product 25.1 μg/ml-26.5 μg/ml, N terminal pro brain natriuretic peptide 1450 pg/ml, ferritin 4134.1 μg/L-5654.3 μg/L, high sensitive C-reactive protein 235.6 mg/L-285.4 mg/L, procalcitonin 0.32 ng/ml-0.54 ng/ml, IL-6 154.7 pg/ml-178.70 pg/ml [8].

On Feb 25, SpO_2 dropped to 70% with FiO_2 100%. A moblie X-ray was performed at the same day for assessing SARS-CoV-2 pneumonia. The chest radiograph revealed pneumothorax, with the left lung 30-40% compressed. Chest CT at the next day (Feb 26) confirmed the pneumothorax (see Figure 1). Positive end expiratory pressure ventilation was reduced from 14 cm H_2O to 10 cm H_2O and closed thoracic drainage was applied to her. The SpO_2 of the patients increased to 88% after drainage. Albumin was added to improve colloidal osmotic pressure and reduce pulmonary edema [9].

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Results

On Feb 28, SpO_2 dropped to 50%. The main laboratory findings at the same day were as follows: PH 7.173, $PaCO_2$ 110.5 mmHg PaO_2 36.6 mmHg , lactate 3.8 mmol/L, glucose 12.22 mmol/L, LDH 679 U/L, urea 36.90 mmol/L, creatinine 320 umol/L, estimated glomerular filtration rate 26.7 ml/min/1.73 m^2, lymphocyte 0.39×10^9/L. The next day (Feb 29) she died of respiratory failure (Figure 1) [10].

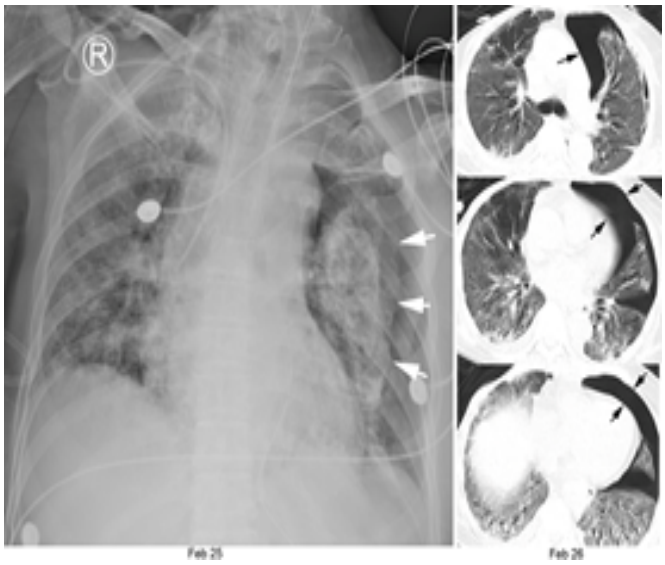

Figure 1. A female resident of Wuhan with laboratory-confirmed SARS-CoV-2 infection had fever, cough, expectoration, dyspnea, diarrhea and fatigue on the first days. With the aggravation of acute respiratory distress syndrome (ARDS), high flow oxygen inhalation, noninvasive and invasive ventilation were used for her successively.

This old female patient without underlying diseases developed ARDS and respiratory failure. The interval from onset of symptoms to laboratory-confirmation of SARS-CoV-2 infection was 10 days, to admission to hospital 11days, to noninvasive ventilation 15 days, to invasive ventilation 19 days, to pneumothorax 25 days and to death 29 days [11,12]. She was believed to have died of respiratory failure, which was aggraveted by the pneumothorax. Although closed thoracic drainage was used for treating pneumothorax, respiratory acidosis deteriorated untill death [13,14].

Discussion

Spontaneous Pneumothorax (SPX) and Spontaneous Pneumomediastinum (SPM) is explained as the appearance of free air within thepleural space and mediastinum respectively. SPX is as outcome of rupture of bulla in lung tissue, create air leak into the pleural space. SPM results from alveolar rupture secondary to an acute increase in intrathoracic pressure subsequently major to dissection of air along the bronchovascular sheath on the way to the mediastinum. Respiratory mani festations of COVD-19 infection involve pneumonia, acute lung injury and acute respiratory distress syndrome. The development of SPX and SPM secondary to COVID-19 infection is barely reported and its presence serves as a possible indicator of worsening respiratory disease. The underlying pathophysiology of SPX and SPM as a result of infection is not apparantly understood. This proposed mechanism of near to a prior explained lung disease that has a similar presentation like pneumocystis pneumonia. It is thought that SARS-CoV-2 causes ischemic parenchymal damage with subsequent inflammation coming in the formation of pulmonary blebs/cyst which ultimately results in obstruction in the small airways. Spontaneous rupture of these cystic lesions leading to SPX is uncommon, however the severe respiratory symptoms seen in COVID-19 may increase the risk. The tachypnoea seen in patients with COVID-19 infection results in acute lung injury. The physiology is similar to that seen in Patient with Self-Inflicted Lung Injury (P-SILI) where casual hyperventilation causes injury to the lungs. Strong inspiratory efforts increase tissue stress, elevates pulmonary transvascular pressures with subsequent fluid leak. SARS-CoV-2 accesses cells *via* angiotensin-converting enzyme-2, most prominent in type II alveolar cells; which leads to dysregulation of surfactant production and function. The strong inspiratory efforts as seen in P-SILI, decreased surfactant production which decreases compliance, the formation of blebs/cyst and large changes in transpulmonary pressure from COVID-19 infection results in rupture with ensuing SPX and SPM. Both patients with worsening respiratory status were placed on high flow nasal canula (HFNC). The risk of SPX and SPM may be increased with the adding of HFNC. The increased positive pressure and high flow rates in already compromised alveoli likely contributed to spontaneous rupture. We assume that patient with COVID-19 may have an increased risk of SPX and SPM. An acute deterioration with rapid oxygen desaturation in a COVID-19 patient could indicate further progressive lung injury. Clinicians should be mindful of the possible development of SPX and SPM in these critically ill patients with a low threshold for CT chest imaging to delineate the extent of pulmonary damage caused by SARS-CoV-2.

Conclusion

From the results obtained by all validation the clinical and radiological features of critically ill patients with SARS-CoV-2 infection have been widely reported. However, there are few publications reporting fatal cases with respiratory failure aggravated by pneumothorax.

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