Bilateral tension pneumothorax with late-stage acute respiratory distress syndrome without mechanical ventilation: A case study

Hyun-Joo Oh[1], Jong Hoo Lee[2]

[1]School of Medicine, Jeju National University, Jeju, [2]Department of Internal Medicine, Jeju National University Hospital, School of Medicine, Jeju National University, Jeju, Korea

(Received May 24, 2013; Revised May 31, 2013; Accepted June 7, 2013)

Abstract

Pneumothorax associated with ventilator induced lung injury is a frequent and fatal complication for patients with acute respiratory distress syndrome (ARDS) receiving mechanical ventilation. There have been few cases of spontaneous bilateral pneumothorax in patients with late-stage ARDS who had not received mechanical ventilation. We describe the detailed course of our case and a brief literature review on pneumothoraces in patients with late-stage ARDS who did not receive mechanical ventilation. Our case suggests that even in patients with ARDS not receiving ventilatory support, monitoring for the presence of pneumothorax seems to be needed. (J Med Life Sci 2013;10(1):58-61)

Key Words : *Respiratory Distress Syndrome, Adult; Pneumothorax; Respiration, Artificial*

Introduction

Acute respiratory distress syndrome (ARDS) is the clinical result of a group of variable processes such as physical or chemical injury, and develop extensive activation of innate inflammatory response[1]. Most patients with ARDS require mechanical ventilation[1]. However, in practive, few are capable of maintaining arterial oxygenation without mechanical ventilation and do not require intubation.

Pneumothorax is a feared complication of mechanical ventilation and is associated with increased morbidity and mortality[2]. In general, pneumothoraces are known to develop more frequently in patients with ARDS or interstitial lung diseases[2]. A prospective, multicenter cohort study showed that 6.5% of patients with ARDS experienced barotrauma such as pneumothorax in spite of limited tidal volume and airway pressure[1]. The prognosis of ARDS markedly worsens if barotrauma such as pneumothorax occurs[1].

To the best of our knowledge, the incidence of pneumothorax in non-mechanically ventilated patients with ARDS is unknown. Furthermore, case reports regarding bilateral pneumothorax developing in non-ventilated ARDS patients are scarce. We report a case of bilateral tension pneumothorax in a patient with late-stage ARDS who had not been mechanically ventilated.

Corresponding Author: Jong Hoo Lee, M.D.
Department of Internal Medicine, Jeju National University Hospital, School of Medicine, Jeju National University, Aran 13 gil 15, Jeju-si, Jeju Special Self-Governing Province, Korea 690-767
E-mail: lovlet@paran.com

Case Report

Our patient was a 69-year-old man who had been hospitalized at a local hospital with fever for 4 days. He had a self-report health status of healthy and did not have a history of smoking. His chest radiography showed pulmonary consolidation in both lower lung fields and community acquired pneumonia was diagnosed by the attending physician. Despite treatment with intravenous moxifloxacin (400 mg/day), the patient remained highly febrile and chest radiography revealed more prominent bilateral pulmonary infiltrates.

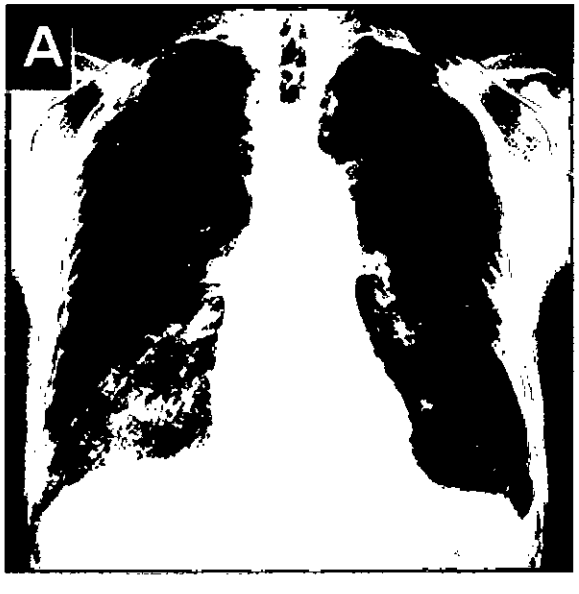
Eight days later, the patient was transferred to our hospital for further evaluation and treatment. On exam, he appeared acutely ill. His vital signs were as follows: blood pressure 139/79 mmHg; heart rate 82 beats/min; respiratory rate 24/min; and body temperature 38.1℃. Auscultation revealed crackles in both lower lung fields. Systemic review of drug history, occupation, travel and home environment did not aid in revealing the reason for the progression of the pulmonary infiltrates. And clinical signs suggested with autoimmune diseases such as morning stiffness, hair loss, joint pain and swelling, raynaud' s phenomenon, dry eye and mouth, and malar rash did not showed.

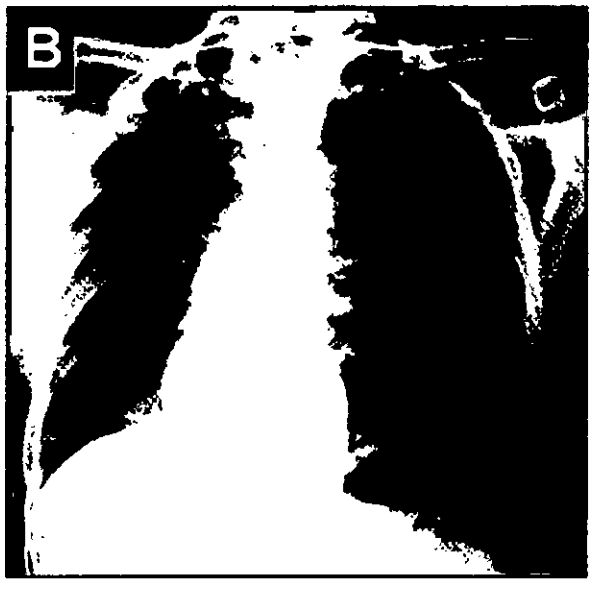
Complete blood count showed a white blood cell count of

11,900/μl, a hemoglobin of 9.0 g/dl and a platelet count of 465,000/μl. Arterial blood gas analysis (ABGA) at FiO_2 0.5 demonstrated severe hypoxia with an oxygen partial pressure (PaO_2)of 71.0 mmHg, a carbon dioxide partial pressure ($PaCO_2$)of 32.4 mmHg, a pH of 7.480, and an oxygen saturation (SaO_2)of 93.3%. The PaO_2/FiO_2 ratio was 142. C-reactive protein and procalcitonin were 16.53 mg/dl and 0.27 ng/ml, respectively. Renal and hepatic function tests were within normal limits. Autoimmune disease-related markers including ANA and anti-CCP were negative, except for a mild elevation of rheumatoid factor (52.2IU/ml).

Chest radiography revealed symmetrical and homogenous hazy opacities bilaterally (Figure 1A). Chest CT showed bilateral dense consolidation at the dependent regions with sparing of the non-dependent areas (Figure 2A and 2B). Cultures of sputum and blood did not reveal any causative pathogens. However, urine antigen for Streptococcus pneumoniae was positive. Finally, we concluded that his ARDS was triggered by an S. pneumoniae infection that was not sensitive to the respiratory fluoroquinolone. Moxifloxacin was discontinued and a 3rd-generation cephalosporin (ceftriaxone, 2.0 g/day) was initiated. At the same time, intravenous methylprednisolone (1.0 mg/kg) was started for treatment of early ARDS. Following administration of ceftriaxone and systemic glucocorticoids, clinical parameters, including a decrease in oxygen demand, gradually stabilized or improved.

On the fifteenth day after hospitalization, the patient abruptly presented with difficulty breathing and chest pain while on conventional nasal cannula with FiO2 at 0.4. Blood pressure fell to 74/47 mmHg. Pulse and respiration rate were 130 beats/min and 32/min, respectively. On physical examination, decreased breath sounds were noted on the left side of the chest. Chest radiography revealed a large hyperlucency in the left lung field (Figure 1B). ABGA on FiO2 0.5 showed respiratory acidosis with severe hypoxemia: pH of 7.31, PaCO2 of 53.3 mmHg, PaO2of 60.7 mmHg and SaO2of 87.7%. With the diagnosis of left-sided tension pneumothorax, a 24-French chest tube was placed and connected to a water seal device. On the following day, the patient complained of right-sided chest pain and aggravated dyspnea. A right-sided pneumothorax was observed on chest radiography (Figure 1C). A chest CT revealed newly developed subpleural cysts with resolving consolidation, as well as bilateral pneumothorax (Figure 2C and 2D). Because of the prolonged air leak, negative pressure was increased to −25 cm H_2O and the chest tube remained for two weeks. Air leakage from the chest tube finally stopped and fully expanded lungs were observed on chest radiography (Figure 1D). At the 2-month follow-up,t he patient was doing well, without any recurrence of pneumothorax.





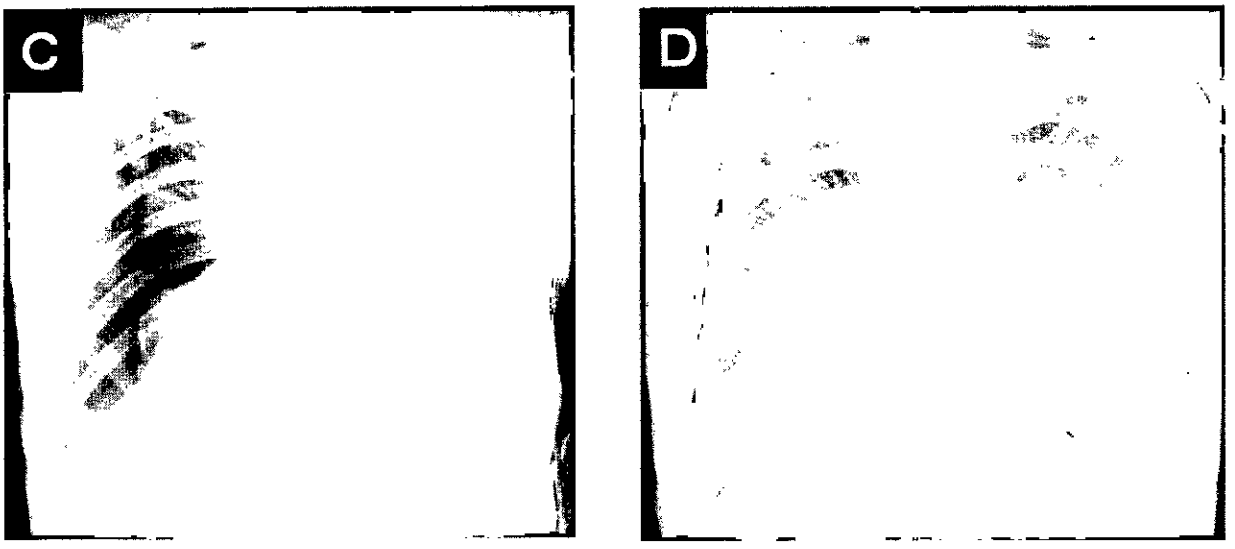


Figure 1. Chest radiography shows bilateral pulmonary infiltrates on admission
(A), newly developed large radiolucency on both lungs at an interval of one day (B and C), and full expansion of the both lungs after removal of the chest tube (D), respectively.

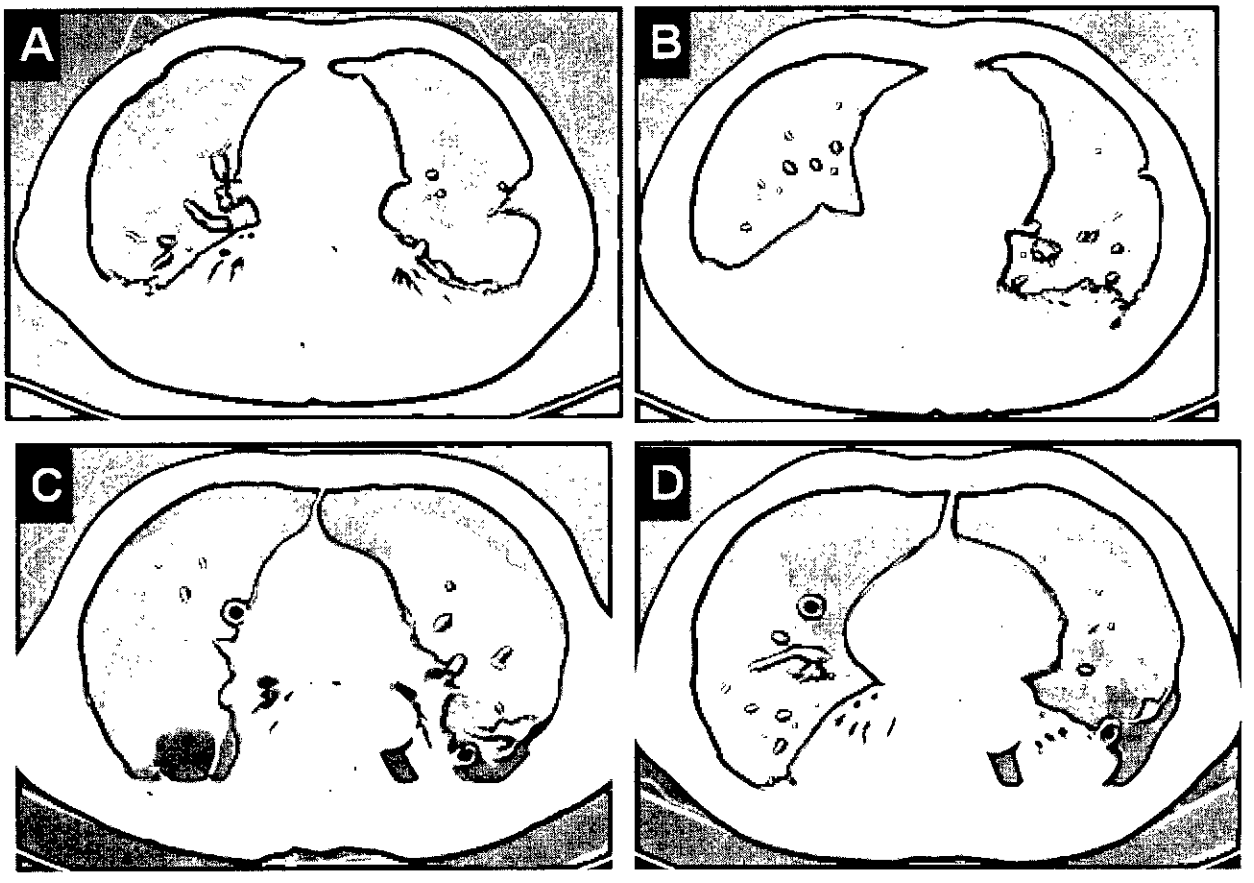


Figure 2. Initial chest tomography shows bilateral consolidation of the dependent regions of the lung with sparing of the non-dependent regions. (A and B) Chest tomography at the time of the second tension pneumothorax revealed newly developed subpleural cysts with resolving consolidations, in addition to the pneumothorax. (C and D)

Discussion

The incidence rate of pneumothorax in patients on mechanical ventilation has been reported from 4% to 15%[3]. In ventilated patients with underlying ARDS, the incidence of pneumothorax is significantly higher and is most commonly associated with ventilator-induced lung injury. One study reported that the overall incidence of pneumothorax was 14% in 139 mechanically ventilated patients, whereas a subgroup of 29 ARDS patients had a 41.3% incidence of pneumothorax[4]. Additionally, another study reported a 48.8% incidence of pneumothorax in mechanically ventilated patients[5]. However, to our knowledge, studies on spontaneous pneumothorax in patients with late stage ARDS not receiving mechanical ventilation have been not reported.

Recently, the Berlin definition for ARDS was introduced[6] and according to this definition, our patient met the clinical criteria for moderate ARDS. Althoughwe suspect that the direct cause was pneumococcal pneumonia, we could not perform a bronchoalveolar lavage due to the patient's high oxygen demand. However and fortunately, the patient did not need mechanical ventilator therapy and kept sufficient oxygenation by venture mask or HFNC.

In the early or exudative stage of ARDS, the inflammatory factors released due to lung injury damage the integrity of the alveolar-capillary barrier. Consequently, an influx of protein-rich fluid fills the alveolar space. Also, cytokines and other proinflammatory compounds aggravate the injury, and coagulation abnormalities occur. As a result, hypoxemia, inactivated surfactant, intrapulmonary shunting, and impaired alveolar ventilation is observed. Many patients during this phase require ventilatory support[7]. After the acute stage of ARDS, patients have rapid resolution of the disease or progression to fibrotic lung injury, which is correlated with an increased risk of death[7]. The late or fibrous stage begins to appear at the second week after the initial insult. During this process, a chest CT will show the architectural distortions of the interstitial and bronchovascular markings and a dramatic increase of subpleural cysts or bullae[8].

One retrospective ARDS study demonstrated that the incidence of pneumothorax in late-stage ARDS (87%) was significantly higher than intermediate-stage (46%) and early-stage ARDS (30%)[5]. Compared to early-stage ARDS, the late-stage showed lower respiratory compliance, increased dead space, higher levels of $PaCO_2$, lower venous admixture, and lower positive end-expiratory pressure requirement. In addition, the number of bullae per lung was significantly higher in late-stage ARDS than intermediate- and early-stage ARDS[5].

In spite of our patient not having high positive airway pressure or tidal volume, follow-up chest CT at the time of the second tension pneumothorax demonstrated newly developed subpleural cysts, which was not observed in the initial CT scan. His pneumothorax occurred about 3 weeks after he developed symptoms of cough and dyspnea, which is consistent with the late stage of ARDS. These cysts likely developed by progressive parenchymal fibrosis occurring during late-stage ARDS. We suggest that this was the underlying pathophysiology of the bilateral pneumothorax, as our patient did not receive mechanical ventilation.

In summary, we report a case of bilateral tension pneumothorax in a patient with late-stage ARDS who did not receive mechanical ventilation. This suggests that even in patients with late-stage ARDS without ventilatory support, careful monitoring for the presence of pneumothorax may be needed.

References

1. Weg JG, Anzueto A, Balk RA, Wiedemann HP, Pattishall EN, Schork MA and et al. The relation of pneumothorax and other air leaks to mortality in the acute respiratory distress syndrome. N Engl J Med 1998;338:341-6.
2. Anzueto A, Frutos-Vivar F, Esteban A, Alia I, Brochard L, Stewart T and et al. Incidence, risk factors and outcome of barotrauma in mechanically ventilated patients. Intensive Care Med 2004;30:612-9.
3. Petersen GW, Baier H. Incidence of pulmonary barotrauma in a medical ICU. Crit Care Med 1983;11:67-9.
4. Gammon RB, Shin MS, Buchalter SE. Pulmonary barotrauma in mechanical ventilation. Patterns and risk factors. Chest 1992;102:568-72.
5. Gattinoni L, Bombino M, Pelosi P, Lissoni A, Pesenti A, Fumagalli R and et al. Lung structure and function in different stages of severe adult respiratory distress syndrome. JAMA 1994;271:1772-9.
6. Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Caldwell E, Fan E and et al. Acute respiratory distress syndrome: the Berlin Definition. JAMA 2012;307:2526-33.
7. Ware LB, Matthay MA. The acute respiratory distress syndrome. N Engl J Med 2000;342:1334-49.
8. Gattinoni L, Caironi P, Pelosi P, Goodman LR. What has computed tomography taught us about the acute respiratory distress syndrome? Am J Respir Crit Care Med 2001;164:1701-11.