Pneumonia and Acute Respiratory Distress Syndrome in a Patient with Influenza A (H1N1)

DENG-WEI CHOU, KUO-MOU CHUNG

A 30-year-old man with a history of type 1 diabetes mellitus visited our emergency room with complaints of fever, cough, sore throat, and muscle aches for five days. He started to develop shortness of breath with exertion, which progressed to respiratory distress at rest one day before admission. On arrival, he had a temperature of 38.4°C, pulse rate of 114 beats/min, respiratory rate of 28 breaths/min and blood pressure of 110/46 mm Hg. Chest auscultation revealed diffuse crackles. The white cell count was 2,320/mL and the platelet count was 76,000/mL. Initial chest radiography (Fig. 1) showed poorly defined nodular opacities and ground-glass opacities in the bilateral lungs.

Fig. 1 Initial chest radiograph shows poorly defined nodular opacities and ground-glass opacities in the bilateral lungs
glass opacities in the bilateral lungs. Arterial blood gas (ABG) analysis showed a pH of 7.44, \( \text{PaCO}_2 \) of 30.9 mmHg, \( \text{PaO}_2 \) of 61.0 mmHg, and bicarbonate concentration of 22.6 mmol/L while he was breathing with a nonrebreathing mask. He was treated with broad-spectrum antibacterial antibiotics and was immediately admitted to the intensive care unit (ICU). Although a rapid antigen test for influenza A and B was negative, oseltamivir was prescribed because influenza was clinically suspected. Four hours after ICU admission, he received endotracheal intubation and mechanical ventilation because of rapid deterioration of his respiratory condition. He was paralyzed and started on pressure control ventilation. ABG analysis showed a pH of 7.34, \( \text{PaCO}_2 \) of 35.1 mmHg, \( \text{PaO}_2 \) of 55.9 mmHg, and bicarbonate concentration of 19.1 mmol/L while he was breathing 100% oxygen. Shock developed which required treatment with vasopressors. A flow-directed pulmonary artery catheter was inserted. His condition fulfilled the criteria for acute respiratory distress syndrome (ARDS). Chest radiography (Fig. 2) eight hours after the initial film showed progression of the abnormalities with development of diffuse alveolar consolidation. Cultures of blood, urine, and tracheal aspirate samples obtained within 24 hours after admission were negative for bacteria. No atypical bacteria were identified. Real-time reverse transcriptase-polymerase chain reaction (RT-PCR) using a nasal swab was confirmed to be positive for the H1N1 virus by the Taiwan Centers for Disease Control. Computed tomography scans (Fig. 3) obtained on the 10th hospital day showed consolidation in the bilateral lungs with extensive patchy ground-glass opacities. No secondary bacterial infection was found during hospitalization. His pulmonary condition gradually improved. On the 14th day in the ICU, the fractional concentration of oxygen in inspired gas dropped to 30% with an adequate \( \text{PaO}_2 \). He was successfully weaned from the ventilator 21 days after being intubated and was discharged on the 28th hospital day. Chest radiography (Fig. 4) obtained two weeks after discharge showed resolution of the abnormalities.

Fig. 2  Chest radiograph eight hours after the initial film shows progression of the abnormalities with development of diffuse alveolar consolidation. An endotracheal tube and a pulmonary artery catheter are noted.
Fig. 3  Cross-sectional (A and B) computed tomography (CT) scans at lung window settings obtained on the 10th hospital day show consolidation of the right upper lobe and extensive patchy ground-glass opacities. A right-sided pleural effusion is seen. Dense consolidation involving the posterior segment of the right upper lobe with air bronchograms (arrow) is present within the consolidated lobe. A coronal (C) CT scan at a lung window setting shows consolidation of the right lung and the left upper lobe. Multiple patchy ground-glass opacities are present in the left lower lobe and right lung base.
after discharge showed obvious resolution of the infiltrates.

A novel swine-origin influenza A (H1N1) virus was first reported in Mexico in late March, 2009, and rapidly spread throughout the world\(^1\). Most patients have typical influenza-like symptoms and a self-limited course\(^2\). However, some patients have a severe course that may result in rapidly progressive pneumonia, respiratory failure, ARDS and multisystem organ failure. One study reported that respiratory distress requiring mechanical ventilation developed in 56% of patients with influenza A (H1N1) viral pneumonia within the first 24 hours after admission. The duration of mechanical ventilation ranged from 7 to 30 days in patients who survived and from 4 to 17 days in patients who died\(^1\). Young adults, especially pregnant women and patients with underlying conditions such as morbid obesity, diabetes mellitus, and chronic lung and liver diseases, appear to be at risk for critical illness from influenza A (H1N1) viral infection\(^3\).

In a study of 40 patients with influenza A (H1N1) viral pneumonia, the major radiological abnormalities were interstitial changes (37%), with patchy ground-glass opacities (22%), which were mostly bilateral (70%), and located in the lower lung zones (70%). Extensive disease was seen in 37.5%, and ARDS was observed in 7.5%\(^4\). In 14 patients with influenza A (H1N1) viral pneumonia requiring mechanical ventilation, radiographic abnormalities included consolidation (50%) or ground glass opacities with (36%) or without consolidation (14%). Initial radiographs showed involvement of three or more lung zones in 93\%\(^5\).

Mortality among the patients with influenza A (H1N1) viral pneumonia requiring mechanical ventilation ranges from 36\%\(^5\) to 58\%\(^1\). The most common causes of death are viral pneumonia and ARDS\(^6\). This may be due to virulent viral infection inducing an ongoing aberrant immune response, leading to extensive lung damage\(^3\). Lung pathological findings in 100 patients who died from pandemic influenza A (H1N1) infection
 included diffuse alveolar damage and viral infection of the alveolar lining cells. Bacterial co-infections were identified in more than 25% of cases. Therefore, broad-spectrum antimicrobial therapy was administered initially while investigations were pending to determine the cause of respiratory failure.

Rapid antigen tests were widely used by clinicians at the point of care; however, test sensitivity was low (40%-69%). If influenza A (H1N1) is clinically suspected, an RT-PCR should be requested for critically ill patients, even if the rapid antigen test is negative. Prompt recognition and antiviral therapy can improve outcomes. In conclusion, influenza A (H1N1) infection should be considered in the differential diagnosis of any patients with ARDS resulting from rapidly progressive pneumonia.

References

A型流感病毒所引起的肺炎和呼吸窘迫症候群

周登偉1,2 鍾國謀3

首次的A型流感病毒(H1N1)感染在2009年3月底被報告之後，已經快速傳遍全世界。大部份的A型流感病毒(H1N1)感染會呈現典型的鼻流感症狀，但是有一部份會造成快速進展的肺炎、呼吸衰竭、急性呼吸窘迫症候群以及多重器官衰竭。在A型流感病毒(H1N1)感染造成肺炎的病人，約有7%會產生急性呼吸窘迫症候群。根據文獻報告，在A型流感病毒(H1N1)感染造成的肺炎合併呼吸衰竭，有93%的病人在首次的胸腔影像就可以見到至少有三個以上的肺葉被侵犯，異常的影像包括：(1)實質化(佔50%)；(2)毛玻璃變化合併實質化(佔36%)；(3)毛玻璃變化(佔14%)。死亡率約36%至58%，最常見的死亡原因为病毒性肺炎以及急性呼吸窘迫症候群。因此，當病人出現快速進展的肺炎因而造成急性呼吸窘迫症候群，此時必須將A型流感病毒(H1N1)感染列為鑑別診斷原因之一，迅速確立診斷及儘早使用抗病毒藥物可以有較好之預後。