

A Case of Severe Pneumonia and Respiratory Failure Due to Swine-Origin Influenza A (H1N1) Virus at Pichit Hospital, North Thailand

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ABSTRACT

Pneumonia with respiratory failure is a severe complication of swine-origin influenza A (H1N1) virus infection. An increased mortality rate has been noted in those who require mechanical ventilation on hospitalization. We present the clinical feature, laboratory investigations, radiological findings, and treatment outcome of a 81-year-old man with poorly controlled diabetes who was hospitalized at Pichit Hospital. (*J Infect Dis Antimicrob Agents* 2009;26:109-12.)

INTRODUCTION

In late March 2009, an outbreak of a respiratory illness caused by swine-origin influenza A (H1N1) virus (S-OIV) was identified in Mexico. As the virus continued to spread rapidly, the World Health Organization (WHO) raised the pandemic level to level 6.⁴ Mexico has reported the greatest number of cases with severe clinical presentation and high mortality rate.¹

The spectrum of clinical illness of S-OIV infection in Thailand has not been fully determined. Most cases in a series of severe pneumonia and respiratory failure in Mexico had occurred in previously healthy patients. We report a case of 81-year-old diabetic Thai man with severe pneumonia and respiratory failure developed on the first day of hospitalization who eventually survived, and was

discharge from our hospital. The real clinical predictors of prognosis are still unknown.

CASE REPORT

An 81-year-old Thai man with poorly controlled diabetes was hospitalized at Pichit Hospital, North Thailand on July 2, 2009. The presenting symptom was dyspnea, 1 day prior to admission. He had returned from Chonburi 5 days previously, and developed symptoms of productive cough, high fever, and dyspnea.

On admission, the patient found to have a temperature of 39°C, respiratory rate of 40/minute, pulse rate of 110/minute, blood pressure of 110/60 mmHg, and agitation. His oxygen saturation was 80-85 percent (on mask with bag with oxygen at 10 litres per minute). He had crepitations and wheezing of

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both lungs. There was no hepatosplenomegaly, and there were no palpable lymph nodes. There were no localizing neurological or meningeal irritation signs.

Laboratory investigations

Complete blood count revealed a hematocrit of 42 percent, a white cell blood count of 18,400/mm³ (88% neutrophils, 9% lymphocytes, 2% band forms, and 1% monocytes), and a platelet count of 280,000/mm³. Blood urea nitrogen and creatinine were 15 and 1.4 mg/dL, respectively. A sputum Gram's stain revealed only a few white blood cells and no bacteria. Liver function test and electrolytes were normal. Serum blood sugar was 495 mg/dL, but serum ketone

was negative. An electrocardiogram showed sinus tachycardia, with poor R waves progression from leads V₁-V₄. Chest X-ray revealed an interstitial infiltration, more on the right upper and left lower lobes (Figure 1). A cold agglutinin test for *Mycoplasma* was negative.

On the first day of admission, the patient was intubated and on ventilator due to pneumonia with acute respiratory failure. A nasopharyngeal swab was sent to test for influenza A, using polymerase chain reaction (PCR). The patient was hospitalized at an intensive care unit (ICU) on the first day. The antibiotics chosen on the first day were ceftazidime and azithromycin. Blood sugar was well controlled



Figure 1. Chest X-ray showed an interstitial infiltration of the right lung and left lower lung fields.

(130-200 mg/dL) by multiple subcutaneous short-acting insulin injections. On the second day of hospitalization, a report of PCR of nasopharyngeal swab showed a positive result for S-0IV. Oseltamivir (75 mg twice daily) was then started on the second day of hospitalization. Chest X-ray revealed an increased infiltration of the right and left lower lungs (Figure 2). However, the patient's condition was worsened, and he developed hypotension requiring inotropic treatment. On the third day of hospitalization, the fever had decreased. On the fourth day of hospitalization, the volume control mode of respirator was changed to continuous positive airway pressure mode, with inspiratory pressure support of 12-16 cm H₂O. On

the sixth day of hospitalization, the extubation could be performed, and the patient was able to breathe spontaneously on an oxygen mask with bag at 10 litres per minute with a respiratory rate of 22-24/minute and an oxygen saturation of 95-100 percent. On the eighth day of hospitalization, he was able to breathe in room air, with an oxygen saturation of 90-92 percent, and eventually discharged from the hospital.

DISCUSSION

The patient had probably had an incubation period of four days before he developed a high-grade fever, pneumonia, and respiratory failure. His complete blood count had a high white blood count,



Figure 2. Chest X-ray showed an increased interstitial infiltration of the right lung and left lower lung fields.

similar to that caused by bacterial infections. This observation is consistent with the data of severe S-0IV pneumonia reported from Mexico.¹ However, no evidence of bacterial infection was identified, either by sputum examination or sputum culture. He developed circulatory failure on the second day of hospitalization, requiring inotropic drugs to maintain his blood pressure. He also had an elevated blood creatinine level (1.6 mg/dL), probably due to acute tubular necrosis as a result of a short period of circulatory failure, which recovered to 0.8 mg/dL on the sixth day of hospitalization (data not shown).

The patient's chest X-ray showed an interstitial infiltration of the right lung and left lower lungs. This is a common finding in most severe cases of S-0IV infection reported from Mexico.¹ Other chest X-ray findings may include patchy alveolar infiltration, which may be caused by acute severe lung injury or acute respiratory distress syndrome (ARDS).

Our patient also had wheezing of both lungs upon physical examination, wheezing was observed in 11 percent of cases in the Mexican series.¹ No corticosteroid was prescribed initially because of a concern about uncontrolled bacterial infection. However, the patient eventually made a full recovery with both antibacterial and antiviral treatment and supportive care.

The case series of the first 18 patients hospitalized in Mexico City with S-0IV infection

documented the clinical findings of severe illness or death.¹ The pathology reports revealed necrosis of the bronchial wall, neutrophilic infiltrates, and diffuse damage with prominent hyaline membranes and prominent fibroblast proliferation. The mortality among patients requiring mechanical ventilation was 58 percent, and lung damage was most likely due to the direct effect of infection with S-0IV. The possible mechanisms of lung damage include a direct injury to the respiratory epithelium² with probably an indirect effect creating cytokine storm.

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