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CASE REPORT

Rapidly fatal community-acquired pneumonia due to *Klebsiella pneumoniae* complicated with acute myocarditis and accelerated idioventricular rhythm

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We describe a previously healthy 52-year-old man with rapidly fatal community-acquired pneumonia caused by *Klebsiella pneumoniae*. The patient developed acute renal dysfunction, accelerated idioventricular rhythm (acute myocarditis), lactic acidosis and septic shock. He died within 15 hours after admission despite intravenous levofloxacin (750 mg daily) and aggressive medical treatment.

Introduction

Klebsiella pneumoniae remains one of the common life-threatening pathogens causing community-acquired pneumonia (CAP) in Taiwan.[1,2] We describe a previously healthy adult with accelerated idioventricular rhythm (AIVR) associated with CAP due to *K pneumoniae* that resulted in a rapidly fatal outcome despite aggressive medical treatment.

Case report

A 52-year-old previously healthy male smoker visited the emergency department because of dyspnea with copious purulent sputum. On arrival, vital signs and physical

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examinations were unremarkable. Initial chest radiograph revealed infiltrations over the right middle and lower lung fields (Fig. 1A). Electrocardiogram disclosed sustained regular tachycardia with wide QRS complex (AIVR, Fig. 1B). Rapid sequence intubation and early goal-directed fluid resuscitation were performed for hypoxemia and suspected severe sepsis. Parenteral antibiotic (levofloxacin 750 mg daily) was initiated after blood and sputum cultures were obtained. He was admitted to the intensive care unit (ICU) due to AIVR and severe sepsis with acute respiratory failure due to CAP 8 hours after visiting the emergency department.

Clinical deterioration was noted during the ICU stay, with acute renal dysfunction (serum creatinine, 2.5 mg/dL), elevated cardiac enzymes (creatine kinase [CK], 386 U/L; CK MB fraction, 81 U/L; troponin-I, 0.1 ng/mL), lactic acidosis without respiratory compensation (lactate value, 4.7 mmol/L; base excess, −12.8 mmol/L; and pCO_2, 43.1 mmHg) and hypoxemia (PaO_2 52 mmHg under oxygen mask 6 L/min). Serum electrolytes were within the normal range during AIVR.

Cardiac ultrasonography revealed normal cardiac chamber sizes, irregular and symmetric left ventricular contracture with an ejection fraction of 50%. There was no vegetation noted in the cardiac valves. Acute myocarditis was diagnosed and the results of pulmonary artery catheterization indicated both septic and cardiogenic shock (heart rate, 97 beats/min; blood pressure, 104/72 mmHg;

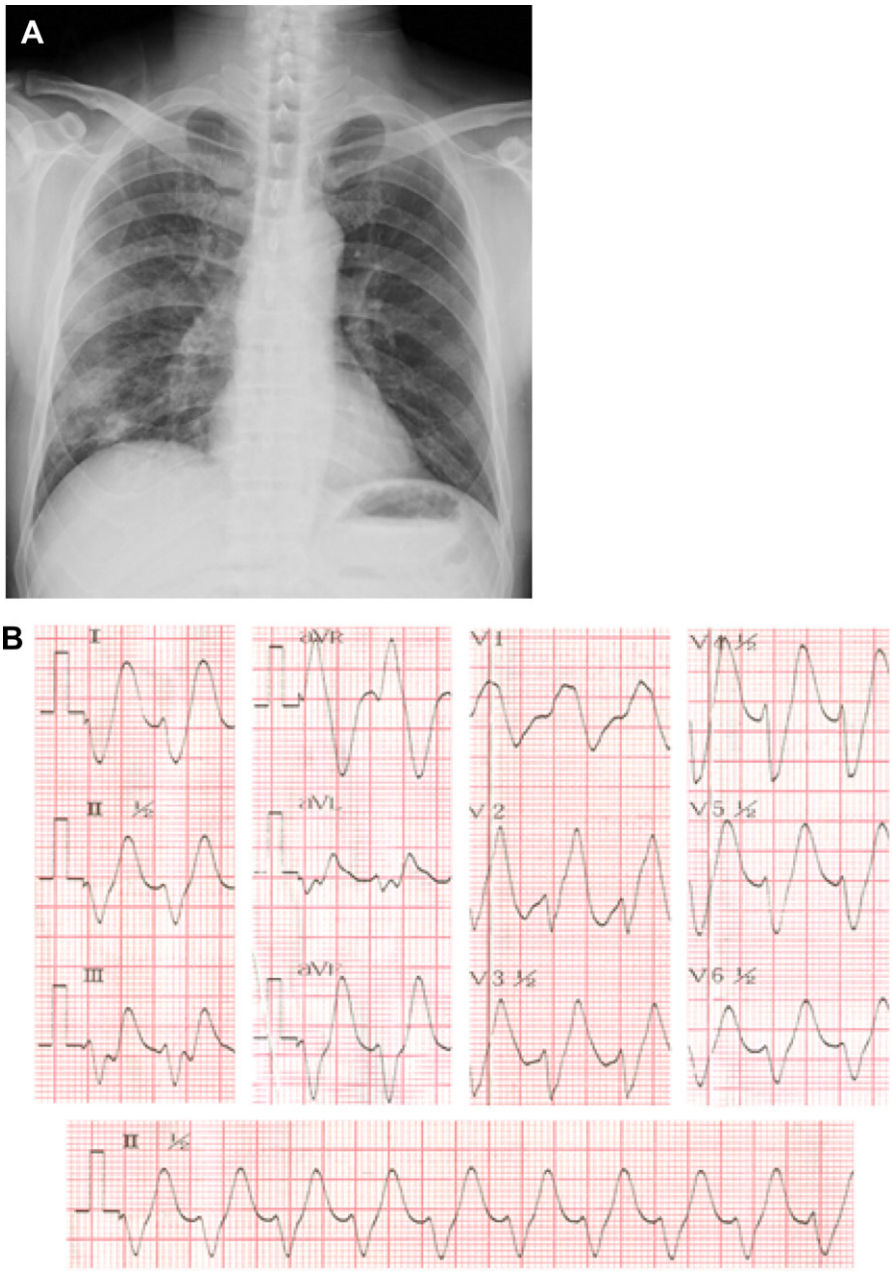

Figure 1. Chest X-ray revealed right upper and middle lung field infiltrations (A) and electrocardiogram showed an accelerated idioventricular rhythm (B) after the onset of airway symptoms.

pulmonary artery wedge pressure, 28 mmHg; cardiac index, 3.69 L/min/m^2; systemic venous resistance index, 1,453 dyn/s/cm^{-5}/m^2) under multiple vasopressors. Cardiac arrest with mixed acidosis ensued 6 hours after admission to the ICU.

Two sets of purulent sputum cultures yielded confluent growth of mucoid *K pneumoniae* that was susceptible to cefazolin, cefuroxime, ampicillin/sulbactam and levofloxacin but resistant to ampicillin by the standard disk diffusion method. Blood cultures were sterile. Serological tests for anti-*Mycoplasma pneumoniae* IgM (Savyon Diagnostics Ltd., Ashdod, Israel), *Legionella* urinary antigen test for serogroup 1 (Binax Inc., Portland, ME, USA), and BinaxNOW® *S pneumoniae* urinary antigen test (Binax Inc.) were all negative. The cause of death was regarded as septic and cardiogenic shock with multiple organ failure and AIVR due to CAP caused by *K pneumoniae*.

Discussion

The appearance of AIVR was considered to be a terminal event and prompted the diagnosis of acute myocarditis.[3] Primary coronary angiography and biopsy of the myocardium was not performed due to the rapidly fatal outcome. Acute myocarditis in association with *K pneumoniae* has been reported but did not include a pathology study or a positive blood culture finding.[4]

In Taiwan, CAP due to mucoid strains of *K. pneumoniae* was more common in younger patients without serious underlying disease (presence of end-stage liver or renal failure, metastatic malignancy, neutropenia, or age $>$70).[5] It is likely that the complicated acute myocarditis and rapidly fatal outcome of this case was related to the virulence factors and pathogenesis of *K pneumoniae*. Myocarditis due to viruses or *M. pneumoniae* still could not be excluded because there were no more clinical samples for further study due to rapidly fatal outcome of this patient. Although an organism growing from a respiratory specimen (not from blood or pleural effusion samples) is not definitive enough to make a firm etiologic diagnosis of CAP in this patient, the confluent growth of a single pathogen from two sputum specimens from a patient with CAP might strongly suggest that *K pneumoniae* is the causative organism of pneumonia.

In summary, the appearance of AIVR with CAP and severe sepsis should prompt the first-line physicians to consider the possibility of acute myocarditis, a condition that requires aggressive treatment and awareness of a rapidly fatal outcome.

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