

Rapidly fatal bacteremic pneumonia caused by *Klebsiella pneumoniae* with K1 hypermucoviscosity phenotype in a previously healthy young man receiving levofloxacin treatment

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Fatal bacteremic *Klebsiella pneumoniae* pneumonia is commonly encountered in alcoholic and diabetic patients. This report describes a previously healthy young man with rapidly fatal bacteremic pneumonia caused by *K. pneumoniae* serotype K1, complicated by septic shock and multiple organ dysfunction.

Key words: Bacteremia; *Klebsiella pneumoniae*; Pneumonia

Introduction

Rapidly fatal bacteremic *Klebsiella pneumoniae* pneumonia (BKPP) is commonly encountered in alcoholic and diabetic patients [1-3], but has not been reported in previously healthy young adults. This report is of a previously healthy young adult with atypical presentation of BKPP, septic shock, multiple organ failure, and rapidly fatal outcome despite aggressive treatment, including recombinant human activated protein C (rhAPC; drotrecogin alfa).

Case Report

A 24-year-old well-nourished previously healthy man presented to the emergency department in Taoyuan General Hospital, Taoyuan, Taiwan, in 2008 with a common cold for the previous 5 days and epigastralgia, vomiting, and diarrhea. He had no relevant medical or drug use history. Physical examination revealed epigastric local tenderness and hyperactive

bowel sounds. He was admitted to the hospital and treated for acute gastroenteritis with intravenous fluids. Hypotension and fever were noted 3 and 8 h after admission, respectively. Parenteral cefuroxime 1.5 g was initiated after diagnostic tests for septicemia had been performed. Chest radiograph revealed infiltrates over the right lower and middle lung fields (Fig. 1A). His white blood cell count was 5640/ μ L with 92% segment and creatinine was 1.0 mg/dL. Dyspnea and tachypnea developed 12 h after admission. He was admitted to the intensive care unit for treatment of septic shock and acute respiratory failure due to pneumonia. He had a low Acute Physiology And Chronic Health Evaluation (APACHE) II score of 14. Early goal-directed therapy of parenteral penicillin G 3 MU every 4 h, levofloxacin 750 mg daily, and oral oseltamivir 75 mg every 12 h were given.

Follow-up chest radiograph revealed diffuse infiltrates over bilateral lung fields (Fig. 1B). The patient developed multiorgan dysfunction with hepatic dysfunction (total bilirubin, 3.2 mg/dL [reference range, 0.3-1.2 mg/dL]; aspartate aminotransferase, 122 U/L [reference range, 20-48 U/L]; alanine aminotransferase, 176 U/L [reference range, 10-40 U/L]), lactic acidosis (lactate, 9.0 mEq/L [reference range,

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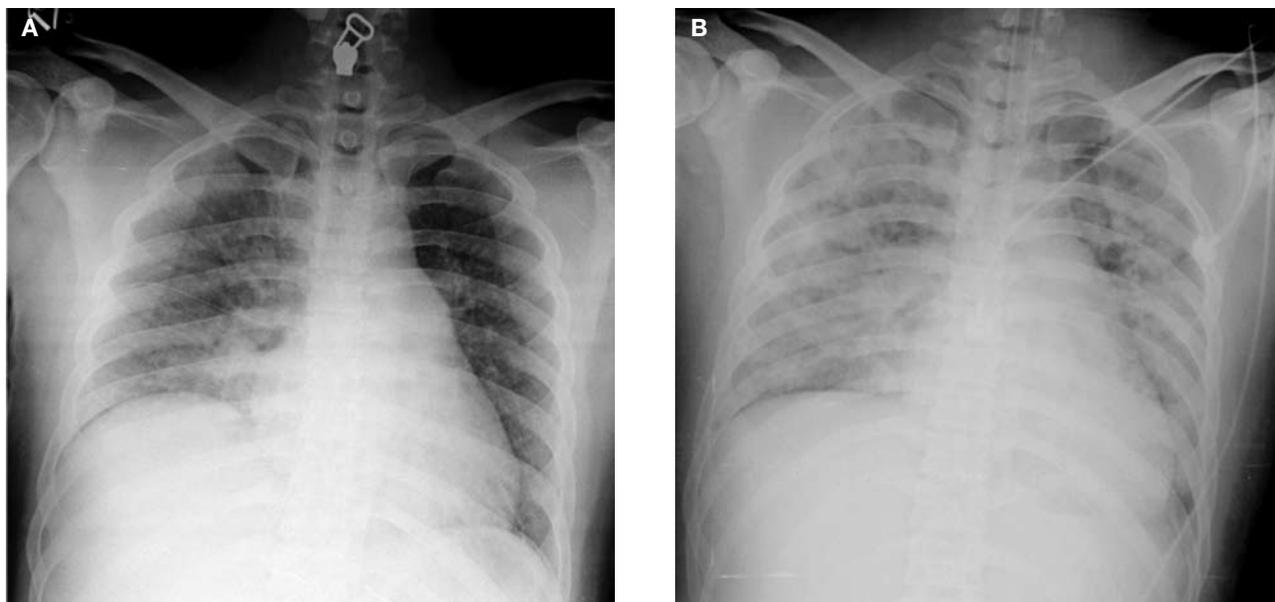


Fig. 1. Chest radiograph showing rapid progression of lung lesions from (A) an infiltrate over the right lower lobe at admission to (B) diffuse infiltrates over the bilateral lungs 18 h after admission.

0.7–2.3 mEq/L]; base excess, -8.4 mmol/L), hypoxemia (arterial oxygen partial pressure, 54 mm Hg [reference range, 80–100 mm Hg]; fractional inspired oxygen, 1.0 [reference range, 0.21]), disseminated intravascular coagulopathy (prothrombin time, 14.7 sec [reference range, 10–13 sec]; fibrin degradation products, 745 μ g/L [reference range, <10 μ g/mL]), and low protein C activity (29.2% [reference range, 76–208%]). Despite treatment with mechanical ventilation, vasopressors, and rhAPC 24 μ g/kg/h, he died of profound shock 41 h after admission.

Two sets of blood culture yielded *K. pneumoniae*, which was susceptible to cefazolin, cefmetazole, ceftriaxone, and levofloxacin but resistant to ampicillin by the standard disk diffusion method. The *K. pneumoniae* isolate recovered from this patient exhibited the K1 hypermucoviscosity phenotype, as well as *rmpA*, *kfu*, *ybtU*, *iroN*, *virB1*, and *iuc* genotypes [4,5]. Urine antigen for *Legionella* serogroup I, immunoglobulin M antibody for *Mycoplasma pneumoniae*, and enzyme-linked immunosorbent assay test for human immunodeficiency virus were negative. Polymerase chain reaction results for severe acute respiratory syndrome-associated coronavirus, influenza viruses, and hantavirus were also negative.

Discussion

The clinical course of the common cold followed by acute gastroenteritis 5 days later suggested atypical

infection in this patient. Failure of the early identification of septic symptoms could result in delay of initial resuscitation, development of organ dysfunction, and mortality. Treatment with rhAPC also failed due to delayed administration because of concern about the patient's low APACHE II score [6]. Such treatment appears to be beneficial for patients with 2 or more dysfunctional organs and low protein C activity [7]. Multiple organ dysfunction and low protein C activity could be a better indication for such treatment in patients with severe sepsis who have a low APACHE II score. Another possible explanation for the rapidly fatal outcome of this patient is the virulence of *K. pneumoniae*. Capsular serotype K1 and iron uptake gene cluster (e.g., *iuc*) have been reported as major virulence determinants for liver abscess [4,5]. The pathogenic role of this phenotype/genotype in BKPP needs further investigation.

In conclusion, delayed recognition of early septic symptoms, delay in initiating rhAPC treatment in a patient with a low APACHE II score, and the virulence of *K. pneumoniae* serotype K1 might all contribute to a rapidly fatal outcome in previously healthy patients with bacteremic pneumonia caused by *K. pneumoniae*.

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