Pseudomonas stutzeri Necrotizing Pneumonia in Pre-existing Pulmonary Tuberculosis

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Abstract

Pseudomonas stutzeri (*P. stutzeri*) is a Gram-negative, non-fermenting rod. It is a rare pathogen; therefore, its isolation is often associated with colonization or contamination. We herein describe the first reported case of necrotizing pneumonia caused by *P. stutzeri* in a non-HIV infected patient with previously undiagnosed pulmonary tuberculosis. The isolate was found to be antibiotic resistant, which led to the failure of the initial treatment. This case highlights the unique presentation of necrotizing pneumonia caused by *P. stutzeri* and the importance of emerging antimicrobial resistance in *P. stutzeri*.

Key words: *Pseudomonas stutzeri*, necrotizing pneumonia

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Introduction

Pseudomonas stutzeri (*P. stutzeri*) is a Gram-negative, non-fermenting, environmental bacterium that does not typically cause human infection. This microorganism has been reported in causing infections in patients with various underlying medical conditions (1). In previous reports, infections caused by *P. stutzeri* were usually responsive to treatments with β -lactam, and the treatment outcomes were usually good (1-4). We herein report a case of *P. stutzeri*-induced necrotizing pneumonia in a patient with previously undiagnosed pulmonary tuberculosis. The antimicrobial resistance of *P. stutzeri* led to the initial unresponsiveness to treatment.

Case Report

A 79-year-old man, who had a smoking history of 30 packs/day for years, with chronic obstructive pulmonary disease (COPD) was admitted to the emergency department due to progressive dyspnea along with a one-week history of fever. On the physical examinations, he was 168 cm tall and weighed 65 kg. His vital signs were as follows: blood pressure, 108/65 mmHg; pulse, 104 beats/min; respiration,

24 breaths/min; body temperature, 39.4°C. His level of consciousness was alert. His auscultation finding indicated coarse crackles.

The laboratory investigation data on admission (day 1) is listed in the Table. The chest X-ray demonstrated increased vascular marking over both lung fields with peribronchial thickening but without infiltration shadow (Fig. 1).

Ampicillin-sulbactam (1.5 g every 6 hours) was initially prescribed for the treatment of the acute exacerbation of COPD. After 3 days of treatment, the antibiotic therapy was changed to levofloxacin (750 mg once daily) due to persistent respiratory distress and infiltration progression in the left lower lung. The patient became afebrile, but left lower lung pneumonia was still present despite the levofloxacin treatment. The antibiotic therapy was changed to vancomycin (1 g twice daily) and meropenem (1 g every 8 h) (Fig. 2). Sputum acid-fast staining was performed on hospital day 5 as the chest X-ray showed increased infiltration in the left lower lung field (Fig. 3). The acid-fast staining revealed acid-fast bacilli in the sputum while the *Mycobacterium tuberculosis* DNA polymerase chain reaction (PCR) (COBAS® TaqMan® Mycobacterium tuberculosis (MTB) Test, Roche, Basel, Switzerland) revealed a positive result. The patient was started on anti-mycobacterial therapy with

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Figure 1. The chest X-ray on admission revealed increased vascular marking over both lung fields with peribronchial thickening but without infiltration shadow.

Figure 3. The chest X-ray (day 5), following sputum acid-fast staining, demonstrated increased infiltration in the left lower lung field.

rifampin (600 mg once daily), ethambutol (1,200 mg once daily), pyrazinamide (1,500 mg once daily) and isoniazid (300 mg once daily). The treatment was discontinued five days later due to a jaundice episode. The biochemical data following the jaundice episode (day 16) is listed in the Table. The screen for hepatitis B virus, hepatitis C virus, and human immunodeficiency virus was negative. The patient did not have purulent sputum until 18 days after admission and much purulent sputum was expectorated. Chest computed tomography (CT) revealed left lung necrotizing pneumonia with air-fluid level (Fig. 4). The sputum culture yielded *P. stutzeri*, identified by the biochemistry testing, two days later. According to Clinical and Laboratory Standards Institutes (CLSI) breakpoints, the isolate was susceptible to amikacin, imipenem, piperacillin and cefepime and resistant to gentamycin, ciprofloxacin, levofloxacin and cefazidime (disc diffusion method). The antibiotic therapy was therefore changed to imipenem-cilastatin (500 mg every 8

Figure 2. The hospital course and duration of treatments. AMPC/SBT: ampicillin-sulbactam, LVFX: levofloxacin, VCM: vancomycin, MEPM: meropenem, INH+RFP+EB+PZA: isoniazid, rifampin, ethambutol and pyrazinamide, IPM/cs: imipenem/cilastatin

Table. The Laboratory Data on Admission (day 1) and during an Episode of Jaundice (day 16)

Parameter analyzed	Result(day 1)	Result(day 16)
Blood cells count		
White blood cell	14,900 / μ L	17,900 / μ L
Hemoglobin	16 g/dL	12.6 g/dL
Platelet	92×10^3 / μ L	89×10^3 / μ L
Segment	63 %	90.8 %
Lymphocyte	16 %	4.4 %
Monocyte	3 %	4.6 %
Band form	17 %	0 %
Biochemistry		
Blood urea nitrogen	21 mg/dL	23 mg/dL
Creatinine	1.59 mg/dL	0.84 mg/dL
Sodium	134 mmol/L	145 mmol/L
Potassium	4.6 mmol/L	3.17 mmol/L
AST	28 IU/L	32 IU/L
ALT	11 IU/L	16 IU/L
Total Bilirubin	1.29 mg/dL	4.3 mg/dL
Direct Bilirubin		1.85 mg/dL
C-reactive protein	15.45 mg/dL	7.69 mg/dL
Albumin	2.2 g/dL	1.8 g/dL
Cortisol	>60 μ g/dL	
HBs-Ag		(-)
HCV-Ab		(-)
HIV-Ab		(-)
Arterial blood Gas (under room air condition)		
pH	7.35	
PaO ₂	86.9 mmHg	
PaCO ₂	55.5 mmHg	
HCO ₃	30.2 mEq/L	

h). Because of persistent respiratory distress, an endotracheal tube was inserted and the patient was transferred to the intensive care unit (ICU). Repeated sputum cultures from the endotracheal aspirates also grew *P. stutzeri* four days later. No other bacterium was ever isolated. The patient's clinical condition improved after one more week of treatment with imipenem-cilastatin. The chest X-ray also showed decreased

Figure 4. Left lung pneumonia progressed to necrotizing pneumonia with air-fluid level (arrow) (day 18).

infiltration of the left lower lung. The endotracheal tube was removed, and the patient was transferred to the regular ward. Anti-mycobacterial medication was gradually resumed. The patient had respiratory distress and desaturation 12 days after extubation due to sputum impaction. The patient and his family refused further intubation. He developed respiratory failure and expired 32 days after admission.

Discussion

P. stutzeri is an aerobic, non-fermenting, Gram-negative rod with polar monotrichous flagella. *P. stutzeri* is a ubiquitous saprophyte, normally found in soil, water and hospital environments, that rarely causes human infection. However, patients with various underlying medical conditions had been reported to have infections caused by *P. stutzeri* (1). Pneumonia and hemodialysis-related blood stream infection were the most frequently reported infections (1). Empyema, septic arthritis, osteomyelitis and soft tissue infection has also been reported (1, 2). Patients may also get infected through trauma or medical procedures (3). We herein reported a case of *P. stutzeri*-induced necrotizing pneumonia in a patient with previously undiagnosed pulmonary tuberculosis.

In this case, necrotizing pneumonia progressing from community acquired pneumonia is the most probably etiology. The quantity of expectorated purulent sputum increased due to the abscess cavity communicating with the bronchioles, while the antibiotic treatment became more effective. Repeated sputum cultures before and after the endotracheal intubation all yielded *P. stutzeri* only, thus it was considered as the causative pathogen.

An underlying *Mycobacterium* infection may cause lung parenchyma destruction and promote necrosis of the lung caused by *P. stutzeri*. In previous reports, patients with *P. stutzeri* infection may have had underlying diseases, such as liver cirrhosis, COPD, malignancy, uremia and HIV infection (1, 2, 4, 5). Patients with these underlying diseases, such as our patient, were also at risk for *Mycobacterium* in-

fection. Previous cases of underlying *Mycobacterium* infection superimposed by *P. stutzeri* infection have only been reported in HIV-infected patients (4, 5). Our case indicated that this infection in non-HIV infected patients is possible, and *P. stutzeri* should be considered as a true pathogen in patients at risk.

In previous reports, *P. stutzeri* isolates were susceptible to various antibiotics, such as piperacillin, third-generation cephalosporins, imipenem, fluoroquinolones and aminoglycosides, and the response to treatment was usually good (1-5). Lee et al. reported that a few *P. stutzeri* isolates with metallo- β -lactamase were resistant to third-generation cephalosporins and some fluoroquinolones (6). Nicolosi et al. also reported a *P. stutzeri* isolate causing pneumonia that was resistant to fluoroquinolones, piperacillin and imipenem (7). The *P. stutzeri* isolate from our patient showed resistance to third-generation cephalosporin and fluoroquinolones. This resistance also initially contributed to deterioration of pneumonia. Metallo- β -lactamase was not checked in this isolate, therefore, we do not know if the resistance is due to the inactivation of β -lactam. The emerging antimicrobial resistance of *P. stutzeri* could potentially lead to antibiotic treatment failure. Therefore, determine the antimicrobial profile of the isolate and changing the treatment accordingly is necessary.

In conclusion, our case highlights a unique presentation of necrotizing pneumonia caused by *P. stutzeri* that rarely causes human infection. *P. stutzeri* should therefore be considered a true pathogen in patients with underlying medical conditions, such as liver cirrhosis, COPD, uremia, malignancy and HIV infection. The emerging antimicrobial resistance of *P. stutzeri* could lead to treatment failure. The antibiotic sensitivity of the organism therefore should be checked for those patients who do not respond to initial treatment.

The authors state that they have no Conflict of Interest (COI).

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