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CASE REPORT

Delftia acidovorans Bacteremia Associated with Ascending Urinary Tract Infections Proved by Molecular Method

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KEY WORDS: ascending urinary tract infection; bacteriemia; Delftia acidovorans A 93-year-old male was hospitalized due to complicated urinary tract infection. *Delftia acidovorans* was isolated from patient's blood and urine culture. The blood and urine strains were identical and confirmed by pulse field gel electrophoresis. Symptoms resolved after specific antibiotic therapy with intravenous administration with imipenem 500 mg every 6 hours. *D acidovorans* is an environment microorganism and rarely caused human infections. So far as we know, this is the first report of ascending urinary tract infection associated with *D acidovorans* bacteremia in Taiwan.

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1. Introduction

Delftia acidovorans, formerly called *Comamonas acidovorans* or *Pseudomonas acidovorans*, is a Gram-negative rod with a worldwide distribution. It is an environment organism found in water and soil. It can cause human infection and only few case report were found in the literature. It could be cause endocarditis, ocular infections, urinary tract infection, catheter-related bacteriemia and empyema.^{1–6} We report a case of *D acidovorans* bacteremia associated with ascending urinary tract infection related to urinary catheter irrigation.

2. Case report

A 93-year-old man was admitted to ward due to urosepsis. He was brought to our emergency department because of fever with chillness and hematuria. He had underlying disease of benign prostate hyperplasia with obstructive uropathy and received a long-term placed urinary catheter. He also had chronic bronchitis and hypertensive cardiovascular disease with regular treatment at an outside patient department.

The patient experienced bladder urine retention by blood clot in urinary catheter one day before admission. Irrigation of urinary catheter with normal saline was performed by his layman caregiver immediately. Fever developed 1 day later and he was sent to our emergency department (ED) due to fever, chills, and hematuria. On

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physical examination, he had a temperature of 40.9°C and blood pressure 92/56 mmHg. The dry skin turgor and hypernatremia (Na 156 mEq/L) were noted. The breathing sound revealed bilateral basal rhonchi of lung and a grade II/VI systolic murmur at right side sternal border. The finding of chest radiograph revealed emphysematous change of bilateral lung fields and cardiomegaly. Bedside abdominal sonogram revealed an enlarged urinary bladder with urine retention. His urinary catheter was replaced after irrigation again at the ED. Urinary analysis yielded pyuria and bacteriuria. No blood clot or obvious obstruction developed after admission. Blood and urine samples were obtained for culture before antibiotic therapy. Because the patient had fever and chills, the blood cell count was performed and the laboratory data of blood test showed that white blood cell count was 18,600/dL, platelets were 167,000/ dL, and hemoglobin was 12.8 mg/dL. The value of C-reactive protein was 18.5 and erythrocyte sedimentation rate was 78. The arterial blood gas analysis revealed metabolic acidosis. The patient was diagnosed as urinary tract infection with urosepsis, and received empirical broad-spectrum antibacterial therapy with flomoxef 2 g intravenously every 8 hours. However, fever persisted for 24 hours and then amikacin 375 mg was given intravenously every 24 hours. He became afebrile after fluid hydration. D acidovorans was identified from blood and urine cultures 3 days later by Phoneix 100 PX0753 panel (Beckton-Dickson, Taiwan, R.O.C). The susceptibility test of D acidovorans was performed by Clinical Laboratory Standard Institute (2010) guidelines.⁷ It resisted to all aminoglycosides, quinolones and β-lactam antibiotics except cefoperazone/sulbactam, imipenem, and doripenem. But fever flared up again on the next day. Antibiotics were shifted to cefoperazone/sulbactam (1 g/ 1 g) intravenously every 6 hours.

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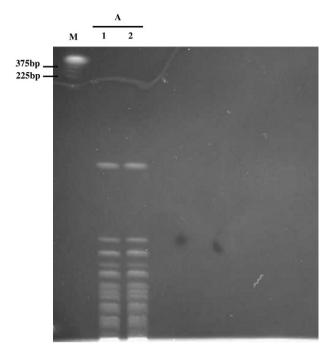


Figure 1 Genotyping of two strains of *Delftia acidovorans* from blood and urine that revealed identical by pulsed-field gel electrophoresis method.

On the eighth admission day, the antibiotic regimen was changed to imipenem 500 mg, intravenously every 6 hours due to recurrent fever. The patient became afebrile within 2 days. Cardioechography examination was arranged to rule out infective endocarditis because of bacteremia and recurrent fever, and the results, which demonstrated calcified aortic valve with moderate

 Table 1
 Characteristics of antimicrobial susceptibility profiles of Delftia acidovorans

aortic and pulmonary regurgitation. There was no vegetation found from the transthoracic cardioechography. He recovered well after 14 days complete antibiotic therapy.

3. Discussion

D acidovorans is an environment microorganism can be found in water and soil. It is an opportunistic pathogen,^{6,8} and has been isolated from blood pressure monitoring devices⁹ and surgical equipments.¹⁰ Because of the ubiquitous characteristics of *D* acidovorans, establishing its pathogenicity may be difficult. Our patient presented with bacteremia due to urinary tract ascending infection. For proving the two *D* acidovorans strains were the same pathogens isolated from the urine ascending to blood, we performed pulse field gel electrophoresis (Figure 1) molecular study and proved the two strains as the same clone. Since symptoms were resolved after the organism was eliminated using specific antibiotic treatment, this organism should be the etiologic agent of infection.^{1,4–8}

Our isolate shows a higher antimicrobial resistance than previous studies, showing that *D* acidovorans was generally resistant to all aminoglycosides, but susceptible to third- and fourthgeneration cephalosporins, quinolones, and trimethoprimsulfamethoxazole (Table 1). Our isolate was only susceptible to cefoperazone/sulbactam, imipenem, and doripenem but resistant to all aminoglycoside, quinolones, third- and fourth-generation β -lactam antibiotics. The development of emergent resistance to antimicrobial agents of this rare human pathogen is alarming and could be a threat in the future. In this case, we proved by pulse field gel electrophoresis molecular method that the bacteria entry to blood through urinary catheter. Either during urinary catheter change or irrigation, it stressed the importance of sterility of water and aseptic technique during handling these procedures.

Antibiotics\References	$\frac{\text{Horowitz et al}^1}{1}$	Castagnola et al ⁴ 2	Ravaoarinoro et al ⁸		Ojeda-vargas et al ⁵	Kawamura et al ⁶			Our isolate
			3	4	5	6	7	8	9
Amikacin	R	S	R	R	R	R	R	R	R
Cefotaxime	S		S	S	S	R			R
Gentamicin	R	R	R	R		R	R	R	R
Piperacillin/tazobactam						R			R
TMP/SMX	S	S			S	R	S	S	R
Levofloxacin						R	S	S	R
Cefoperazone/sulbactam						S	S	S	S
Aztreonam	MS		R	S	S	R	S	S	R
Ceftazidime	S	S				R	S	S	R
Imipenem		S	S	S	S	S	S	S	S
Meropenem						MS	S	S	S
Ciprofloxacin	S	S	S	S	S	R	S	S	R
Chloramphenicol	MS					R			R
Cefepime						R	S	S	R
Tigecycline						R			R
Tobramycin	R	S	R	R	R		R	R	R
Cefazolin	R						S	S	R
Cefuroxime	R								R
Cefoxitin	S								
Cefoperazone	S		R	S					R
Ceftriaxone	S	S							R
Ampicillin	R	R			R				R
Ticarcillin	R	S	R	S	S				R
Mezlocillin	S								
Piperacillin	MS	S	S	S			S	S	R
Netilmicin			R	R					R
Norfloxacin			S	S	S				R
Ofloxacin			S	S					R
Fosfomycin					R				

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