Acute Infective Endocarditis Caused by *Delftia acidovorans*, a Rare Pathogen Complicating Intravenous Drug Use

Saima Mahmood, Kent E. Taylor, Timothy L. Overman, and Malkanthie I. McCormick

Department of Internal Medicine, Division of Infectious Diseases and Division of Cardiology, and Department of Pathology and Laboratory Medicine, University of Kentucky, and Pathology and Laboratory Medicine Service, Veterans Affairs Medical Center, Lexington, Kentucky, USA

Gram-negative bacilli causing infective endocarditis (IE) is rare, even in intravenous drug users. This case report underscores several clinically important aspects of *Delftia acidovorans* IE: the organism’s ability to cause rapid destruction of normal native valves and to cause embolic occlusion of large arteries and its resistance to all aminoglycosides.

CASE REPORT

In November 2010, a 30-year-old male, with a history of intravenous drug use (IDU), hepatitis C, and posttraumatic stress disorder, presented to the Lexington Veterans Affairs Medical Center (VAMC) with a 2- to 3-week history of malaise and right knee pain. The patient reported having a fever for 2 days prior to presentation, with a temperature of 103°F (noted at home). He was in his usual state of health until a month prior to presentation, when he developed right knee pain, which was first observed when he jogged for exercise. The pain progressively worsened, and he stopped jogging 2 weeks prior to presentation. One week prior to presentation, the patient went to the VAMC emergency room and was diagnosed with a viral illness; flu PCR was negative, and he was instructed to follow up with his primary care physician. No anti-infective medication was given. On the day of hospitalization, he returned to the VAMC emergency department with increased pain in his right leg, fever, and a painful red lesion on the ring finger of his left hand. Upon physical examination, his temperature was 104°F and he was found to have a new grade 3/6 diastolic murmur (not previously noted in his clinic records), diminished pedal pulse and posterior tibial pulse in his right leg, without knee effusion, and a tender erythematous nodule on the 4th digit of his left hand.

The patient’s laboratory studies were unremarkable. His human immunodeficiency virus test and urine drug screening were both negative. Blood cultures were drawn, and vancomycin and piperacillin-tazobactam (2 days) were started empirically. The echocardiogram demonstrated a severe aortic insufficiency with aortic valve vegetation of 1.0 by 1.4 cm. An arteriogram of his right leg showed occlusions in the right posterior tibial artery and right peroneal artery.

The patient’s social history was only remarkable for past IDU. He stated that he was in a drug rehabilitation facility for 6 weeks prior to the onset of symptoms but confessed to occasional relapses in his IDU behavior. The patient admitted using the water from the bathroom and kitchen faucets to prepare his drugs for injection. He denied smoking and drinking alcohol regularly.

The patient became afebrile on antibiotics in 48 h. He was evaluated by cardiothoracic surgery and, on the third hospital day, was transferred to the adjacent University of Kentucky Hospital (UKH) where he underwent successful aortic valve replacement.

Two sets of blood cultures at VAMC and a culture of tissue from the patient’s aortic valve at UKH grew a Gram-negative bacillus. The Gram-negative bacillus was identified as *Delftia acidovorans*, resistant to all aminoglycosides. His antibiotic was changed to ceftriaxone, and the bacteremia cleared a few days following surgery. His hospital stay was complicated with a left upper extremity arterial occlusion of his brachial artery. He required debridement of the left brachial artery with replacement at its bifurcation with a reversed saphenous vein graft by vascular surgery. He was continued on ceftriaxone and was discharged to long-term acute care to continue his antibiotic course for 6 weeks from the first day of negative blood culture.

Since his discharge, the patient has had two other admissions for *Candida albicans* and *Candida glabrata* fungemia and *Streptococcus sanguis* bacteremia related to IDU that have required prolonged treatment with anti-infective agents but not surgery. The patient has had no further admissions for the past year. His last clinical follow-up with vascular surgery was in February 2012, and he was doing satisfactorily.

**Microbiology data.** Upon admission to the VAMC, two blood cultures were ordered. Blood was inoculated into BacT/Alert (bio-Mérieux, Durham, NC) standard aerobic and anaerobic blood culture bottles and placed in a BacT/Alert 3D instrument (bio-Mérieux, Durham, NC). The next day, three of the four bottles were flagged positive by BacT/Alert. The time of detection ranged from 1.11 days (26 h 38 min) for two bottles to 1.35 days (32 h 24 min) for the third bottle. The fourth bottle was negative after 5 days of incubation with BacT/Alert.

Gram stains of the blood cultures revealed Gram-negative bacilli which grew on sheep blood, chocolate, and MacConkey agars, forming small colonies after 24 h of incubation at 35°C in 5% CO₂. The organism was oxidase positive.

Organism identification was determined using a no. 44 MicroScan Gram-negative combo breakpoint panel and a MicroScan W/A 40 instrument (Siemens Healthcare Diagnostics, West Sacramento, CA). The organism was identified as *D. acidovorans*.
D. acidovorans (MicroScan biotype, 0026356). The probability of the identification was 99.17%.

This organism is a nonfermenter and nonoxidizer of glucose. For glucose nonfermenters, the MicroScan identification combines biochemical data and single concentration antimicrobial resistance to generate a biotype. The organism was resistant to 4 \( \mu \)g/ml of colistin, 4 \( \mu \)g/ml of kanamycin, 4 \( \mu \)g/ml of penicillin, and 4 \( \mu \)g/ml of tobramycin. The organism had the following positive biochemical tests: acetylamide utilization, malonate utilization, nitrate reduction, oxidase, and tartrate utilization.

Antimicrobial susceptibility was determined using the same no. 44 MicroScan Gram-negative combo breakpoint panel and a MicroScan W/A 40 instrument. The isolate was resistant to aminoglycosides, cefazolin, and cefepime and was susceptible to cefazidime, carbenapemem, fluoroquinolones, pipercillin-tazobactam, tetracycline, and trimethoprim-sulfamethoxazole. The isolate was susceptible to ceftizoxime (MIC \( \leq 8 \mu \)g/ml) using the pre-2010 CLSI breakpoints; using the 2010 breakpoints, the ceftizoxime susceptibility could not be determined, as 8 \( \mu \)g/ml is the lowest dilution on this panel type. (Automated systems vary in their ability to adapt to the 2010 CLSI cephalosporin breakpoint changes.)

The Gram-negative bacillus from valve tissue was also identified as *D. acidovorans* by the Phoenix system (BD Diagnostic Systems, Sparks, MD) at UKH. The Phoenix antibiogram was similar to that obtained at VAMC. The only differences between VAMC and UKH susceptibility patterns were cefepime and pipercillin-tazobactam. The Phoenix system reported them as indeterminate. UKH performed an Etest (bioMérieux, Durham, NC) with these two antimicrobial agents. Etest results matched the VAMC MicroScan susceptibility pattern of cefepime resistant and pipercillin-tazobactam susceptible.

The International Collaboration on Endocarditis-Prospective Cohort Study identified the microbial etiology in 2,781 patients from 58 sites in 25 countries with definite endocarditis as defined by Duke criteria (9). Gram-negative bacteria accounted for only 6% of pathogens; HACEK bacteria (6) and non-HACEK bacteria accounted for 2% and 4%, respectively. This rarity of Gram-negative bacilli as causative organisms is equally true in IDU with endocarditis. We have seen an increase in IDU-related endocarditis at UKH in the past 10 years, yet Gram-negative bacterial endocarditis accounts for 2% and 4%, respectively. This rarity of Gram-negative bacteria is phenotypically similar to *Comamonas*.