were identified from both specimens. The *D. acidovorans* late was initially not identifiable by MicroScan WalkAway (Sie-

mensch, Munich, Germany). Additional testing was done with Vitek and API 20 NE version 6.0 (bioMérieux, Marcy l’Etoile, France). The Vitek reported *D. acidovorans* with 53% probability, and the API 20 NE version 6.0 reported *D. acidovorans* as a significant taxon with an unacceptable profile. Finally, it was further characterized with long-chain-fatty-acid analysis by gas chromatography using the MIDI 62 system (Microbial Identification Systems, Newark, DE). The susceptibilities of the second *D. acidovorans* isolate by MicroScan were now reported as resistant to cefotaxime, ceftriaxone, cefepime, aztreonam, and piperacillin-tazobactam but susceptible to ceftazidime, amoxicillin-clavulanate, ticarcillin-clavulanate, and trimethoprim-sulfamethoxazole. Treatment was changed to ceftazidime and given along with vancomycin. Blood cultures became negative after 3 days of the antibiotics. The patient remained afebrile. The antibiotics were continued to complete a 21-day course. Her catheter became malpositioned and re-

quired replacement at the end of the treatment. Blood cultures remained negative for 18 days after removal of the catheter.

Because of the changes in the antimicrobial susceptibility profiles, the second isolate was screened for genes encoding CTX-M extended-spectrum β-lactamases using multiplex PCR with previously identified primers and PCR conditions (20). No CTX-M enzymes were detected. *In vitro* susceptibilities of the second isolate were retested by MicroScan, Etest, and broth macrodilution, which revealed inconsistencies of the cephalosporin susceptibilities (Table 1). Evaluation for the presence of β-lactamases with penicillin, ceftazolin, and ceftria-

xone hydrolysis measurement by spectrophotometric assay was performed (13). Maximal hydrolysis rates using penicillin, ceftazolin, and ceftriaxone substrate were 0.05, 0.04, and 0 μmol/mg of protein/min, respectively.

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*D. acidovorans* was formerly called *Comamonas acidovorans* or *Pseudomonas acidovorans*. Prior to 1987, a variety of now independent genera, including *Burkholderia*, *Stenotrophomonas*, *Ralstonia*, *Comamonas*, *Delfia*, *Acidovorax*, and others,
were all identified within the broad genus *Pseudomonas*. As a collective, they are all generally aerobic, oxidase-positive, Gram-negative bacilli that grow on MacConkey’s agar. This broad genus was rearranged based on 16S rRNA homology studies. *D. acidovorans* belongs to the family *Comamonadaceae* based on 16S rRNA gene sequence analysis. *Comamonas acidovorans* was found phylogenetically distant from the type species of *Comamonas*, *Comamonas terrigena*, and was removed from the genus *Comamonas* and renamed *Delftia acidovorans* (6, 19).

Microscopically, *D. acidovorans* is characterized by straight to slightly curved Gram-negative bacilli, which occur single or in pairs. Strains are motile by means of polar or bipolar tufts of one to six flagella, with the distinctive feature of having a long wavelength (3.0 μm between the tops of adjacent waves). The organism is strictly aerobic; growth occurs at 30°C to 37°C, and 8% of the strains grow at 42°C. It grows well on media containing organic acids, amino acids, peptone, and carbohydrates but not glucose; 29% of the strains grow in the presence of sucrose, and maltose, and no production of hydrogen sulfide in Kligler iron agar, but 57% of the strains are positive with lead acetate paper. The organism is also negative for beta-galactosidase, and 10-μg colistin disks and 300-U polymyxin B disks, while the related species *Comamonas testosteroni* and *C. terrigena* are susceptible (9). The following tests are suggested for identification of nonfermenters: alkaline phosphatase, benzyl-arginine arylamidase, pyrrolidonyl arylamidase, and 10-μg colistin disks (9).

This organism is a common water and soil saprophyte and has wide geographic distribution (6). *D. acidovorans* appears to be an organism of limited virulence that has usually been considered nonpathogenic when isolated from clinical specimens. However, it has been reported to be a pathogen under certain circumstances. Weinstein et al. described an outbreak of *D. acidovorans* bacteremia associated with contaminated pressure-monitoring devices (18). Unfortunately, clinical and epidemiologic information was not provided in association with this outbreak. Horowitz et al. reported a case of *D. acidovorans* endocarditis in an intravenous drug user with alcoholic liver disease (7). The illness occurred soon after the patient was treated for 4 weeks with ampicillin for viridans streptococcal endocarditis. Several cases of ocular infections have been reported, including keratitis, blepharitis, and conjunctivitis in immunocompromised corneas, contact lens wearers, or otherwise healthy patients (1, 11, 12, 17).

We identified four previous reports of vascular catheter-related *D. acidovorans* bacteremia (2, 4, 8, 10). All four patients were immunocompromised with underlying malignancies or AIDS. All responded to antimicrobial treatment, but eventually removal of the central venous catheter was required for three patients, with the exception of the case described by Ender et al. (4).

The *D. acidovorans* bacteremia in our patient was likely secondary to an intravascular catheter. Although the girl did not meet the strict criteria for catheter-related sepsis (14), the catheter was probably the origin of the bacteremia based on the clinical presentation and the exclusion of other sources.

### TABLE 1. MicroScan and Etest MICs for isolates of *D. acidovorans* from the patient

<table>
<thead>
<tr>
<th>Antibiotic(s)</th>
<th>1 (MicroScan)</th>
<th>2</th>
<th>MicroScan</th>
<th>Repeat MicroScan</th>
<th>Etest</th>
<th>Broth macrodilution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amoxicillin-sulbactam</td>
<td>16/8</td>
<td>&gt;16/8</td>
<td>16/8</td>
<td>16/8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aztreonam</td>
<td>&lt;8</td>
<td>&gt;16</td>
<td>&gt;16</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cefazolin</td>
<td>&gt;16</td>
<td>&gt;16</td>
<td>&gt;16</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ceftazidime</td>
<td>&lt;2</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>0.38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ceftaxime</td>
<td>&lt;2</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>0.38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gentamicin</td>
<td>&gt;8</td>
<td>&gt;8</td>
<td>&gt;8</td>
<td>&gt;256</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imipenem</td>
<td>&lt;4</td>
<td>&lt;4</td>
<td>&lt;4</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Levofloxacin</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>0.064</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meropenem</td>
<td>&lt;4</td>
<td>&lt;4</td>
<td>&lt;4</td>
<td>0.094</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Piperacillin-tazobactam</td>
<td>&lt;16</td>
<td>&gt;64</td>
<td>&gt;16</td>
<td>&lt;0.016</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ticarcillin-clavulanate</td>
<td>&lt;16</td>
<td>&gt;16</td>
<td>&gt;16</td>
<td>&lt;0.016</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trimeprprim-sulfamethoxazol</td>
<td>&lt;2/38</td>
<td>&lt;2/38</td>
<td>&lt;2/38</td>
<td>0.125</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The catheter was not removed immediately, and differential quantitative blood cultures were not obtained. However, the patient had the same organism isolated from the hemodialysis catheter and a peripheral blood sample. She had no other source of infection based on clinical assessment. Therefore, the infection likely represented a recurrent vascular-catheter-related bacteremia.

Information about susceptibility of this organism is limited. Case reports revealed that all were resistant to gentamicin, and some were resistant to all aminoglycosides. They are generally susceptible to broad-spectrum cephalosporins, piperacillin, aztreonam, carbenapenem, quinolones, and trimethoprim-sulfamethoxazole (1, 2, 3, 4, 7, 8, 10, 12, 17) (Table 2). *D. acidovorans* has been reported to have β-lactamase in two resistant laboratory mutant strains and inducible β-lactamase production in one resistant clinical strain (15). Kawamura et al. reported recurrent bacteremia in an 11-year-old girl with metastatic neuroblastoma during sustained neutropenia after peripheral blood transplant, where the isolate developed resistance to broad-spectrum penicillins and cephalosporins during antibiotic treatment (8). The second isolate from our case also appeared to have developed resistance to broad-spectrum cephalosporins by MicroScan after antibiotic treatment. With the previous reports of inducible β-lactamase production and a pattern of resistance after the antibiotic therapy, a PCR for genes encoding β-lactamase activity in the second isolate. Susceptibility testing by Etest and broth macrodilution demonstrated inconsistent results for cephalosporins.

In conclusion, we report a case of recurrent intravascular-catheter-related infection caused by *D. acidovorans*. Antibiotic susceptibility testing for cephalosporins using MicroScan appears to be unreliable for this organism.

We thank John Quale and David Landman for all laboratory support and for providing helpful critique on this work and thank Lee Lillian from the Public Health Laboratory, New York City Department of Health and Mental Hygiene, for performing the gas chromatography.

**REFERENCES**