Flavobacterium indologenes Bacteremia: Clinical and Microbiological Characteristics

Po-Ren Hsueh, Tzuen-Ren Hsiue, Jiunn-Jong Wu, Lee-Jene Teng, Shen-Wu Ho, Wei-Chuan Hsieh, and Kwen-Tay Luh

To our knowledge, Flavobacterium indologenes has never been reported as a cause of bacteremia in humans. F. indologenes bacteremia was diagnosed in 12 patients at a tertiary referral center in southern Taiwan between 1 January 1992 and 31 December 1994. Six of these patients had ventilator-associated pneumonia, two had primary bacteremia, and one patient each had pyonephrosis, peritonitis, biliary tract infection, and surgical wound infection. Five patients (42%) had malignancies, and three (25%) had multiple burns. Polymicrobial bacteremia was diagnosed in eight patients (67%). Two (17%) of the patients in this study died; both had polymicrobial bacteremia. Antimicrobial susceptibility testing of the blood isolates from the 12 patients showed that >90% of the isolates were susceptible to piperacillin, cefoperazone, ceftazidime, and minocycline. The chromatograms of esterified fatty acids for the isolates were identical. F. indologenes should be considered an etiologic agent of bloodstream infection, especially in hospitalized patients with severe underlying diseases.

Patients and Methods

Bacterial isolates. From 1 January 1992 to 31 December 1994, we reviewed the records on positive blood cultures from the microbiological laboratories at the National Cheng Kung University Hospital (Tainan, Taiwan) for isolates of F. indologenes. Blood specimens were processed by the Bactec 660 nonradiometric blood culture system (Becton Dickinson, Sparks, MD) in BACTEC 6A and 7B media. The Flavobacterium species were identified by conventional methods, as previously described [1, 10]. The identification of F. indologenes was confirmed by means of the API 20 NE system (bioMérieux, Marcy-l’Etoile, France) and the Vitek GNI system (bioMérieux Vital, Hazelwood, MO). F. indologenes and F. gleum were further differentiated with use of the eight biochemical tests described by Yabuuchi et al. [9].

Analysis of cellular fatty acids. For analysis of fatty acids, isolates were incubated on trypticase soy agar (BBL Microbiology Systems, Cockeysville, MD) at 35°C for 24 hours in ambient air. Harvest and lysis of cells, saponification, methylation of fatty acids, and extraction of fatty acid methyl esters were performed according to the manufacturer’s instructions [11]. Fatty acid methyl esters were analyzed by gas-liquid chromatography (GLC) with use of the Microbial Identification System (MIS; Microbial Identification System (MIS; Microbial ID Inc., Newark, DE) and a Hewlett-Packard 5890A (Hewlett-Packard, Palo Alto, CA) equipped with a fused-silica capillary column (length, 25 m; internal diameter, 0.2 mm) and flame ionization detector. The conditions for GLC were those recommended in the operational manual of the MIS [11]. Cellular fatty acids were identified by comparing retention times with those of calibration standards (Hewlett-Packard, Avondale Division, Avondale, PA).
Antimicrobial susceptibility testing. MICs of 20 antimicrobial agents for the 12 blood isolates of *F. indologenes* were determined by use of the agar dilution method described by the National Committee for Clinical Laboratory Standards (NCCLS) [12]. The following antimicrobial agents were obtained as standard reference powders of known potency: cephalothin, clindamycin, erythromycin, gentamicin, netilmicin, amikacin, trimethoprim-sulfamethoxazole (TMP-SMZ), and vancomycin (Sigma Chemical, St. Louis); piperacillin and minocycline (Lederle Laboratories, Pearl River, NY); cefotaxime (Hoechst AG, Frankfurt, Germany); ceftriaxone (Roche Laboratories, Nutley, NJ); ceftazidime (Glaxo Operations, Greenford, England); cefoperazone (Pfizer, New York, NY); moxalactam (Shionogi Pharmaceutical, Osaka, Japan); aztreonam (Bristol-Myers Squibb Laboratories, New York); imipenem (Merck Sharp & Dome, West Point, PA); ofloxacin, (Daiichi Siiyaku, Tokyo); ciprofloxacin (Bayer AG, Leverkusen, Germany); and teicoplanin (Marion Merrill Dow, Kansas City, MO).

The drugs were incorporated into the agar in serial twofold concentrations that ranged from 0.03 μg/mL to 128 μg/mL. The MIC of each antibiotic was defined as the lowest concentration of the drug that inhibited visible growth of the organism. *Staphylococcus aureus* strain ATCC (American Type Culture Collection) 29213, *Escherichia coli* strain ATCC 25922, and *Pseudomonas aeruginosa* strain ATCC 27853 were used as controls in each set of tests.

Clinical characteristics. The medical records of all patients whose blood cultures were positive for *F. indologenes* were studied retrospectively. Data on underlying diseases, other associated conditions (use of an indwelling catheter or ventilator and administration of chemotherapy), the clinical syndrome, day of first in-hospital blood culture positive for *F. indologenes*, polymicrobial bacteremia, other site from which the organism was isolated, antibiotic regimens before and after blood culture results were available, complications (shock, acute renal failure, disseminated intravascular coagulation, or adult respiratory distress syndrome), and outcome were collected from clinical presentations.

Episodes of bacteremia that developed at least 72 hours after admission were regarded as nosocomial, while episodes of bacteremia identified by positive blood cultures earlier than 72 hours after admission were considered community acquired. Neutropenia was defined as a WBC count of <4 × 10^9/L. Antibiotic therapy was considered to be appropriate if the drugs chosen proved to be active against the isolate during susceptibility testing.

Results

Bacterial isolates. Twelve isolates of *F. indologenes* were identified, accounting for 0.26% of the total number of positive blood cultures over the 3-year period of this study. For all patients but one, at least two sets of blood cultures were positive for *F. indologenes*. Subculture onto sheep blood agar within 24 hours of incubation revealed smooth, circular, yellow-pigmented colonies that were 1–2 mm in diameter. All isolates were oxidase-positive gram-negative rods that did not ferment glucose. On the basis of biochemical profiles produced by the API 20NE and Vibrio GNI card, the probability that the organism was *F. indologenes* was determined to be >99%. All isolates failed to grow at 41°C, did not produce acid from xylose and L-arabinose, and were negative for esculin hydrolysis within 4 hours of incubation but positive after 24 hours of incubation. These results corresponded with the identification of the organism as *F. indologenes*.

*F. indologenes* was also isolated simultaneously from other sites in nine patients (75%) (table 1). Eight patients (67%) had polymicrobial bacteremia, and seven of these bacteremic episodes were nosocomial. The organisms most frequently cultured simultaneously with *F. indologenes* were nonfermentative gram-negative bacilli (six patients) and gram-positive cocci (three patients).

Antimicrobial susceptibility. The results of susceptibility tests with 20 antimicrobial agents are shown in table 2. All isolates were uniformly susceptible to piperacillin (MICs, 4 μg/mL), and 11 of the isolates (92%) were susceptible to cefoperazone (MICs, 16 μg/mL), ceftazidime (MICs, 8 μg/mL), and minocycline (MICs, 4 μg/mL). Susceptibility to TMP-SMZ, ofloxacin, and ciprofloxacin was variable. All but one of the isolates were resistant to vancomycin (MICs, 16 μg/mL). The isolates were consistently resistant to other β-lactam antibiotics including aztreonam and imipenem; to aminoglycosides; and to erythromycin, clindamycin, and teicoplanin.

Analysis of cellular fatty acids. The chromatogram of cellular fatty acids for one of the *F. indologenes* isolates (figure 1) was characterized by the presence of four major peaks and two minor peaks. All of the isolates had identical cellular fatty acid profiles.

Clinical characteristics of the patients. The clinical characteristics of the 12 patients with *F. indologenes* bacteremia are summarized in table 1. Male patients predominated (67%), and the mean age was 47 years (range, 1–80 years); six patients (50%) were >60 years old. Underlying diseases were present in 10 patients: five had malignancies, three had multiple burns, and two were neutropenic (chemotherapy induced) when *F. indologenes* was isolated. *F. indologenes* bacteremia was nosocomial in nine patients (75%) and developed in all of these patients after the first week of hospitalization.

The underlying clinical syndromes included pneumonia (six patients), primary bacteremia (two patients), and pyonephrosis, peritonitis, biliary tract infection, and surgical wound infection (one patient each). All six cases of pneumonia were ventilator associated, and each patient had received ventilatory support for at least 1 week before developing bacteremia. Of these six patients, five had polymicrobial bacteremia, and four had cultures of tracheal aspirates that were positive for *F. indologenes* as well as other nonfermentative gram-negative bacilli.
Table 1. Clinical characteristics, treatment, and outcome for 12 patients with bacteremia caused by Flavobacterium indologenes.

<table>
<thead>
<tr>
<th>Patient no./age (y)/sex</th>
<th>Underlying conditions</th>
<th>Other associated condition(s)</th>
<th>Clinical syndrome</th>
<th>Other bacteria isolated</th>
<th>Antibiotic treatment</th>
<th>Complication(s)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/60/M</td>
<td>Uremia</td>
<td>Ventilator</td>
<td>Nosocomial pneumonia</td>
<td>Staphylococcus aureus</td>
<td>Tracheal aspirate</td>
<td>Cefotaxime, netilmicin</td>
<td>Ciprofloxacin</td>
</tr>
<tr>
<td>2/80/F</td>
<td>Diabetes mellitus, COPD</td>
<td>Ventilator</td>
<td>Nosocomial pneumonia</td>
<td>None</td>
<td>Tracheal aspirate</td>
<td>Cefazolin, tobramycin</td>
<td>Ofloxacin</td>
</tr>
<tr>
<td>3/30/M</td>
<td>Burns (23% TBSA)</td>
<td>Ventilator</td>
<td>Nosocomial pneumonia</td>
<td>Stenotrophomonas maltophilia, Enterococcus faecalis</td>
<td>Tracheal aspirate</td>
<td>Amp/Sulb, gentamicin</td>
<td>Moxalactam, vancomycin</td>
</tr>
<tr>
<td>4/1/M</td>
<td>Burns (35% TBSA)</td>
<td>Ventilator</td>
<td>Nosocomial pneumonia</td>
<td>Escherichia coli, Morganella morgani, Alcaligenes species</td>
<td>None</td>
<td>Ciprofloxacin, ceftizoxime</td>
<td>Ciprofloxacin, ceftoxitin, amikacin</td>
</tr>
<tr>
<td>5/20/M</td>
<td>Burns (35% TBSA)</td>
<td>Ventilator</td>
<td>Nosocomial pneumonia</td>
<td>Stenotrophomonas maltophilia, Pseudomonas fluorescens, Pseudomonas aeruginosa, Enterobacter cloacae</td>
<td>Tracheal aspirate</td>
<td>Amp/Sulb, gentamicin</td>
<td>Cefazidine, vancomycin</td>
</tr>
<tr>
<td>6/80/M</td>
<td>Gastric adenocarcinoma</td>
<td>Ventilator</td>
<td>Nosocomial pneumonia</td>
<td>None</td>
<td>Tracheal aspirate</td>
<td>Imipenem, gentamicin</td>
<td>Imipenem, ceftizidine</td>
</tr>
<tr>
<td>7/70/M</td>
<td>Bladder carcinoma</td>
<td>PCND</td>
<td>Community-acquired pneumonia</td>
<td>None</td>
<td>Pus, urine</td>
<td>Piperacillin, amikacin</td>
<td>Cefazidine, amikacin</td>
</tr>
<tr>
<td>8/44/M</td>
<td>Diverticulitis</td>
<td>None</td>
<td>Community-acquired pneumonia</td>
<td>Group D streptococci</td>
<td>Acetic fluid</td>
<td>Cefoxinid, tobramyacin, metronidazole</td>
<td>Cefoxitin, tobramyacin, metronidazole</td>
</tr>
<tr>
<td>9/66/M</td>
<td>Cholangiocarcinoma</td>
<td>PTCD</td>
<td>Community-acquired biliary tract infection</td>
<td>None</td>
<td>Bile</td>
<td>Cefazolin, netilmicin</td>
<td>Cefetazid, gentamicin</td>
</tr>
<tr>
<td>10/60/M</td>
<td>Aortic mycotic aneurysm</td>
<td>Duodenostomy and drainage</td>
<td>Nosocomial surgical wound infection</td>
<td>None</td>
<td>Drainage fluid</td>
<td>Amp/Sulb, netilmicin, chloramphenicol</td>
<td>Cefazidime, metronidazole, amikacin</td>
</tr>
<tr>
<td>11/31/F</td>
<td>Breast carcinoma</td>
<td>Port-a-catheter, neutropenia</td>
<td>Nosocomial primary bacteremia</td>
<td>Acinetobacter baumannii</td>
<td>None</td>
<td>Piperacillin, netilmicin</td>
<td>Piperacillin, netilmicin</td>
</tr>
<tr>
<td>12/32/F</td>
<td>Acute leukemia</td>
<td>Neutropenia</td>
<td>Nosocomial primary bacteremia</td>
<td>Acinetobacter Iwafuji</td>
<td>None</td>
<td>Piperacillin, netilmicin</td>
<td>Cefazidime, vancomycin, amikacin</td>
</tr>
</tbody>
</table>

NOTE: Amp/Sub = ampicillin/sulbactam; ARDS = adult respiratory distress syndrome; ARF = acute renal failure; COPD = chronic obstructive pulmonary disease; DIC = disseminated intravascular coagulation; PCND = percutaneous nephrostomy drainage; PTCD = percutaneous transhepatic cholangiography drainage; TBSA = total body surface area.

Before F. indologenes was isolated, all but three (patients 7, 11, and 12; table 1) of the 12 patients were treated with different combinations of two or more antibiotics that later proved inactive in vitro against the isolates. One patient (patient 10) had an aortic mycotic aneurysm due to Salmonella choleraeuts, which was complicated by duodenal perforation. This patient underwent graft interposition and duodenostomy with insertion of an indwelling drainage catheter. F. indologenes was isolated concurrently from multiple samples of the drained fluid and blood on the 14th hospital day, at which time the patient had clinical evidence of sepsis.

After blood cultures were found to be positive for F. indologenes, all but three (patients 3, 8, and 9) of the 12 patients were treated with appropriate antibiotics. The conditions of 10 patients improved clinically within 5 days of the initiation of treatment. Two (17%) of the patients, both of whom had pneumonia and presented with refractory shock, developed adult respiratory distress syndrome and/or acute renal failure; they died on the fifth and sixth day, respectively, after the bacteremic episodes.

Discussion

Flavobacterium species CDC group IIb as well as F. meningosepticum, which belong to the first natural group of flavobacteria designated by Holmes et al. [8], are saccharolytic and
Table 2. In vitro antimicrobial susceptibilities of the 12 blood isolates of *F. indologenes*.

<table>
<thead>
<tr>
<th>Antimicrobial agent</th>
<th>MIC range (µg/mL)</th>
<th>MIC&lt;sub&gt;90&lt;/sub&gt;</th>
<th>MIC&lt;sub&gt;50&lt;/sub&gt;</th>
<th>Susceptibility breakpoint (µg/mL)</th>
<th>No. (%) of isolates susceptible</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piperacillin</td>
<td>1–8</td>
<td>2</td>
<td>4</td>
<td>≤16</td>
<td>12 (100)</td>
</tr>
<tr>
<td>Cephalothin</td>
<td>32–&gt;12</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>≤8</td>
<td>0</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>16–&gt;128</td>
<td>32</td>
<td>64</td>
<td>≤8</td>
<td>0</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>16–&gt;128</td>
<td>32</td>
<td>64</td>
<td>≤8</td>
<td>0</td>
</tr>
<tr>
<td>Ceftazidime</td>
<td>2–&gt;128</td>
<td>8</td>
<td>8</td>
<td>≤8</td>
<td>11 (92)</td>
</tr>
<tr>
<td>Cefoperazone</td>
<td>4–&gt;128</td>
<td>8</td>
<td>16</td>
<td>≤16</td>
<td>11 (92)</td>
</tr>
<tr>
<td>Moxalactam</td>
<td>32–&gt;128</td>
<td>64</td>
<td>&gt;128</td>
<td>≤8</td>
<td>0</td>
</tr>
<tr>
<td>Aztreonam</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>≤8</td>
<td>0</td>
</tr>
<tr>
<td>Imipenem</td>
<td>32–&gt;128</td>
<td>64</td>
<td>64</td>
<td>≤4</td>
<td>0</td>
</tr>
<tr>
<td>Oftoxacin</td>
<td>2–64</td>
<td>2</td>
<td>32</td>
<td>≤2</td>
<td>6 (50)</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>0.5–128</td>
<td>1</td>
<td>32</td>
<td>≤1</td>
<td>8 (67)</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>≤0.5</td>
<td>0</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>64–&gt;128</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>≤0.5</td>
<td>0</td>
</tr>
<tr>
<td>Minocycline</td>
<td>2–16</td>
<td>4</td>
<td>4</td>
<td>≤4</td>
<td>11 (92)</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>8–&gt;128</td>
<td>64</td>
<td>&gt;128</td>
<td>≤4</td>
<td>0</td>
</tr>
<tr>
<td>Netilmicin</td>
<td>64–&gt;128</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>≤8</td>
<td>0</td>
</tr>
<tr>
<td>Amikacin</td>
<td>32–&gt;128</td>
<td>64</td>
<td>&gt;128</td>
<td>≤16</td>
<td>0</td>
</tr>
<tr>
<td>TMP-SMZ</td>
<td>0.5–16</td>
<td>2</td>
<td>16</td>
<td>≤2</td>
<td>6 (50)</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>2–128</td>
<td>16</td>
<td>16</td>
<td>≤4</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Teicoplanin</td>
<td>32–64</td>
<td>32</td>
<td>64</td>
<td>≤8</td>
<td>0</td>
</tr>
</tbody>
</table>

NOTE. TMP-SMZ = trimethoprim-sulfamethoxazole.

indole positive [1]. *Flavobacterium* species CDC group IIb shares a number of phenotypic characteristics with strains of *F. meningosepticum*; however, unlike *F. meningosepticum*, this group is characterized by the production of a distinct orange-yellow pigment within 24 hours of culture on a blood agar plate, hydrolysis of starch, and lack of acidification of d-arabinose and galactose [1, 10]. The name *F. indologenes* was formerly proposed for all of the heterogeneous strains of *Flavobacterium* species CDC group IIb, and the name *F. gleum* was proposed for a genetically homogenous group within it [1, 7, 8]. Until 1990, *Flavobacterium* species CDC group IIb was divided into two species, *F. indologenes* and *F. gleum*, on the basis of biochemical reactions and DNA homology studies [9, 13]. To date, however, these two species are poorly characterized in most microbiological laboratories, partly because of the general lack of commercial identification systems available for full differentiation. In this study, all the isolates were identified as *F. indologenes* by means of conventional tests as well as by two commercial identification systems. Identification of these isolates was further confirmed with use of the biochemical tests described by Yabuuchi et al. [9]. The fact that these isolates had identical cellular fatty acid profiles indicates that they were identical at the species level [14].

*Flavobacteria* are ubiquitous in nature and are found in soil, plants, foodstuffs, and water sources despite adequate chlorination [1, 3]. In the hospital, indwelling vascular catheters, vials, sink traps, feeding tubes, other fluid-associated equipment, and even disinfectants may become reservoirs for *flavobacteria* [1–3, 5]. Strains of *Flavobacterium* species CDC group IIb are found more commonly in clinical specimens than are all other flavobacteria combined, but most of the isolates are recovered with other bacteria and have been judged to be of questionable pathogenicity [1–6]. Few cases of definite human infections due to *Flavobacterium* species CDC group IIb have been reported in the English-language literature since the mid-1970s [3–6]. *F. indologenes* has been isolated from a patient with ventilator-associated pneumonia only once before; in that case, the organism was recovered only from a tracheal aspirate [4].

Bacteremia caused by *Flavobacterium* species CDC group IIb is rare; to our knowledge, only 15 cases have been previously described, 14 of which were associated with a nosocomial outbreak [6] and one of which was associated with bilateral breast cancer [3]. Contaminated vascular catheters were thought to be the portals of entry for the organisms in these patients.

In the present study, patients who had severely debilitating diseases, malignancy, or neutropenia and had undergone various invasive procedures or had previously received broad-spectrum antibiotics during a long period of hospitalization were at high risk for bloodstream infections caused by *F. indologenes*. Tracheobronchial colonization with gram-positive cocci and gram-negative rods, particularly nonfermentative gram-negative bacilli that originated from a hospital environment, is common in mechanically ventilated patients [4, 15, 16]. The incidence of nosocomial pneumonia is high, especially during the first 8–10 days, among patients who receive prolonged respiratory assistance [17]. Our findings were consistent with
those of other investigators. Furthermore, we emphasize that *F. indologenes* should be included in the list of pathogens that cause ventilator-associated pneumonia.

Although flavobacteria are not considered part of the normal bacterial flora in humans, these organisms can indeed be recovered from various anatomical sites in healthy persons [1, 3]. For three of our patients (patients 7, 8, and 9), the infections were unrelated to any invasive procedure, and the patients had had no exposure to a hospital environment in the preceding 3 months. Because of the diverse conditions associated with the community-acquired and nosocomial infections and because of the different antibiotic resistance profiles of the isolates, the epidemiological association among these patients is not evident.

The pathogenic role of *F. indologenes* in patients with polymicrobial bacteremia is difficult to determine. Two of our patients (patients 1 and 4) with polymicrobial bacteremia died despite the fact that they received antibiotics with in vitro activity against *F. indologenes*, which suggests that the deaths might be not fully attributable to flavobacterial bacteremia. On the contrary, two other patients (patients 3 and 8) who had polymicrobial bacteremia recovered despite the administration of antibiotics without in vitro activity against *F. indologenes*. However, the fact that three-fourths of the patients had *F. indologenes* recovered simultaneously from various sites in addition to blood indicates the invasive nature of this organism.

The susceptibility patterns of the isolates is partly consistent with those of *Flavobacterium* species CDC group IIb strains described by other authors [18–21]. These organisms have been reported to be uniformly resistant to extended-spectrum penicillins, first- and second-generation cephalosporins, and aztreonam. Susceptibilities to clindamycin, erythromycin, tetracycline, aminoglycosides, third-generation cephalosporins, imipenem, and quinolones have varied. In the present study, piperacillin, cefoperazone, ceftazidime, and minocycline were the agents most active against the isolates, while ciprofloxacin was active to a lesser extent. The activities of other antibiotics were disappointing. The wide range of antibiotic resistance exhibited by these isolates, particularly those recovered from hospitalized patients exposed to prolonged antibiotic therapy (and thus selection pressure), causes a significant therapeutic dilemma in clinical settings.

It is difficult to determine optimal therapeutic regimens for treating severe infections due to *F. indologenes* because of the small number of patients in this series as well as the presence of polymicrobial bacteremia. Vancomycin had previously been recommended as the drug of choice for treatment of meningitis caused by *F. meningosepticum* in infants [22]. However, the high MICs of vancomycin for the *F. indologenes* isolates determined in the present study, as well as those for isolates of *F. meningosepticum* and other flavobacterial species described in earlier studies [18, 23], indicate that this drug is not appropriate for treating flavobacterial infections.

Administration of the quinolones, especially ciprofloxacin, has resulted in clinical cures in patients with flavobacterial infections [21, 24]. One patient (patient 1) with *F. indologenes* and *S. aureus* bacteremia who was treated with intravenous ciprofloxacin (which had in vitro activity against the two isolates) died. However, another patient (patient 2), who was treated with oral ofloxacin for bacteremia due to the susceptible isolate, recovered. Clinical treatment of flavobacterial sepsis with a quinolone as the sole agent needs further evaluation. On the basis of the antimicrobial susceptibility patterns of these *F. indologenes* isolates and the limited clinical experience, we have concluded that piperacillin, cefoperazone, and ceftazidime may be included as part of the regimen for treating *F. indologenes* infections.

In summary, the findings of this study indicate that *F. indologenes* should be considered a potential pathogen in bloodstream infections in humans, especially in the setting of malignancy and with use of invasive procedures. The resistance of this organism to multiple antibiotics, including the newer beta-lactam agents and the high incidence of polymicrobial bacteremia make it difficult to determine optimal therapeutic options. The relatively low mortality among patients with *F. indologenes* infections...
bacteremia is not fully understood; further cases should be evaluated, and a study on the pathogenicity of this organism in humans should be conducted to elucidate this phenomenon.

References


