Identification, Genotypic Relation, and Clinical Features of Colistin-Resistant Isolates of Acinetobacter Genomic Species 13BJ/14TU from Bloodstreams of Patients in a University Hospital

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Colistin resistance remains rare among clinical isolates of Acinetobacter species. We noted the emergence of colistin-resistant bloodstream isolates of the Acinetobacter genomic species (GS) 13BJ/14TU from patients at a university hospital between 2003 and 2011. We report here, for the first time, the microbiological and molecular characteristics of these isolates, with clinical features of Acinetobacter GS 13BJ/14TU bacteremia. All 11 available patient isolates were correctly identified as Acinetobacter GS 13BJ/14TU using partial rpoB gene sequencing but were misidentified using the phenotypic methods Vitek 2 (mostly as Acinetobacter baumannii), MicroScan (mostly as A. baumannii/Acinetobacter haemolyticus), and the API 20 NE system (all as A. haemolyticus). Most isolates were susceptible to commonly used antibiotics, including carbapenems, but all were resistant to colistin, for which it is unknown whether the resistance is acquired or intrinsic. However, the fact that none of the patients had a history of colistin therapy strongly suggests that Acinetobacter GS 13BJ/14TU is innately resistant to colistin. The phylogenetic tree of multilocus sequence typing (MLST) showed that all 11 isolates formed a separate cluster from other Acinetobacter species and yielded five sequence types. However, pulsed-field gel electrophoresis (PFGE) revealed 11 distinct patterns, suggesting that the bacteremia had occurred sporadically. Four patients showed persistent bacteremia (6 to 17 days), and all 11 patients had excellent outcomes with cleared bacteremia, suggesting that patients with Acinetobacter GS 13BJ/14TU-associated bacteremia show a favorable outcome. These results emphasize the importance of precise species identification, especially regarding colistin resistance in Acinetobacter species. In addition, MLST offers another approach to the identification of Acinetobacter GS 13BJ/14TU, whereas PFGE is useful for genotyping for this species.

Acinetobacter species have emerged as important causative pathogens of nosocomial infections, including ventilator-associated pneumonia, bacteremia, and urinary tract infections (1, 2). Among these infections, the most common species isolated from clinical specimens is Acinetobacter baumannii, and a number of other Acinetobacter species are also frequently observed to be clinically relevant (1). To date, more than 32 Acinetobacter species have been reported, including at least 17 named species; the remaining species are generally referred to as genomic species (GS) pending further characterization (2–6). For species other than A. baumannii, the majority of Acinetobacter clinical isolates are isolated from blood and are associated with severe bacteremia or septicemia (1, 7), highlighting the increasing clinical importance of non-baumannii Acinetobacter species in bloodstream infections.

As the prevalence of multidrug-resistant (MDR) Acinetobacter infections has increased, the polymyxin antibiotic colistin has emerged as a first-line therapy for the treatment of MDR Acinetobacter infections (2, 8, 9). While colistin-resistant Acinetobacter strains remain rare among clinical isolates of Acinetobacter species, colistin resistance has been documented in Acinetobacter GS 13BJ/14TU, an uncommon Acinetobacter species that rarely causes disease in humans (1, 8). Acinetobacter GS 13BJ and Acinetobacter GS 14TU were independently described in 1989 by two groups based on DNA-DNA hybridization (10, 32); however, these two isolates were later found to represent a single species (11). In our previous study, Acinetobacter GS 13BJ/14TU accounted for 4.7% of all Acinetobacter isolates from blood cultures at Chonnam National University Hospital (a 1,000-bed tertiary care hospital in Gwangju, South Korea) over a 4-year period (12). However, despite the frequency of these infections, little is known regarding the microbiological, genotypic, and clinical characteristics of Acinetobacter GS 13BJ/14TU bloodstream isolates. In this study, we examined the results of the identification and antimicrobial susceptibilities for all 11 available bloodstream isolates of Acinetobacter GS 13BJ/14TU. Additionally, we evaluated the genetic relationships of these isolates using both multilocus sequence typing (MLST) and pulsed-field gel electrophoresis (PFGE) and the clinical characteristics of the patients with Acinetobacter GS 13BJ/14TU bacteremia.

MATERIALS AND METHODS

Bacterial isolates and identification. In this study, we analyzed all 11 available Acinetobacter GS 13BJ/14TU bloodstream isolates, recovered from 11 patients at Chonnam National University Hospital (a 1,000-bed tertiary care hospital in Gwangju, South Korea) between January 2003 and December 2011; two duplicate isolates were obtained from two patients (patients 8 and 11) during this period. Laboratory strains Acinetobacter GS 13BJ/14TU ATCC 17905 and A. baumannii ATCC 19606 were used as references for comparison.

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controls. All bloodstream isolates were identified by partial rpoB gene analysis using a 450-bp sequence (zone 2) in the rpoB gene region (13). Primers Ac1055F (5'-GTGATAARCTGCGCGTCGT-3') and Ac1598R (5'-CGBGCTTGCTATYTTGCTT-3') were used to amplify the rpoB gene zone 2 sequence. All loci were sequenced in both the forward and reverse directions with the same primers as used for the amplification. Sequences were assembled and compared with reference sequences using the BLAST tool at the NCBI database (http://www.ncbi.nlm.nih.gov/blast). Phylogenetic analysis was performed using Molecular Evolutionary Genetics Analysis version 5 (MEGA5) (14) comparing the 450-bp rpoB gene sequences (zone 2) obtained in this study to Acinetobacter reference sequences obtained from GenBank. A dendrogram was constructed using the neighbor-joining method and bootstrap analysis with 1,000 replicates (15, 16). Phenotypic identification of bloodstream isolates was performed using the Vitek 2 XL (Gram-negative identification [GN-ID] card; bioMérieux, France), MicroScan WalkAway 96 plus (Neg Breakthrough Combo Panel; Siemens Healthcare Diagnostics), and API 20 NE systems (bioMérieux, France) according to the manufacturers' instructions. API 20 NE identification results were interpreted using the apiweb identification software with a 7-digit numerical profile (code number). Microbiological features were investigated by streaking of isolates on blood agar plates (bioMérieux, France) and by reading the reaction of the API 20 NE strip.

Antimicrobial susceptibility testing. Antimicrobial susceptibility testing of bloodstream isolates was performed using the disk diffusion method (Becton, Dickinson and Company) on Mueller-Hinton agar for ampicillin-sulbactam, piperacillin-tazobactam, ticarcillin-clavulanic acid, cefazidime, cefepime, ceftiraxone, imipenem, meropenem, gentamicin, tobramycin, amikacin, tetracycline, and ciprofloxacin; colistin susceptibility was determined using the agar dilution method according to the Clinical Laboratory and Standards Institute guidelines (17–19).

Multilocus sequence typing. MLST was performed for all Acinetobacter GS 13BJ/14TU bloodstream isolates and control strains (Acinetobacter GS 13BJ/14TU ATCC 17905 and A. baumannii ATCC 19606) using the Institut Pasteur MLST scheme (http://www.pasteur.fr/mlst). The MLST scheme uses internal fragments of the following seven housekeeping genes: cnpA60 (60-kDa chaperonin), fusA (elongation factor EF-G), gltA (citrate synthase), pyrG (CTP synthase), recA (homologous recombination factor), rplB (50S ribosomal protein L2), and rpoB (RNA polymerase subunit B). The rpoB region used for MLST is distinct from the partial rpoB gene region (zone 2) used for identification. A phylogenetic tree was inferred from 2,976-bp concatenated sequences of seven housekeeping genes for the 11 bloodstream isolates and two control strains tested in this study, along with reference Acinetobacter species sequences obtained from the MLST database and previous reports (20, 21). Phylogenetic relationships were determined using MEGAS (14), using a 1,000-replicate bootstrap analysis; a phylogenetic tree was constructed using the neighbor-joining method (15).

Pulsed-field gel electrophoresis. Restriction endonuclease analysis of Apal-digested genomic DNA was performed using PFGE, as described previously (22). The CHEF DNA size standard (Bio-Rad Laboratories) was used as a molecular weight standard. PFGE patterns were analyzed visually, and the epidemiological relatedness inferred from the number of band differences in the PFGE pattern, as described previously (23). Acinetobacter GS 13BJ/14TU ATCC 17905 and A. baumannii ATCC 19606 isolates were used as control strains.

Clinical characteristics. Demographic and clinical data for the 11 patients whose blood isolates were identified as Acinetobacter GS 13BJ/14TU were obtained from electronic medical records. Data were collected regarding clinical variables, including comorbidities, number of blood culture-positive days, stays in the intensive care unit (ICU), previous antibiotic usage, invasive procedures at the time of bacteremia onset, and outcomes. Neutropenia was defined as a neutrophil count <1,500/mm³ at the onset of infection (24). Previous use of antimicrobial agents or steroids was defined as administration within 30 days prior to onset of bacteremia (25). Recent operation was defined as an operation occurring within 30 days prior to the onset of bacteremia. Mortality was defined as Acinetobacter-related death in the absence of another definitive cause of death (26).

RESULTS

Molecular identification. A BLAST search using the 450-bp partial rpoB gene sequences from all 11 isolates included in this study identified Acinetobacter GS 13BJ/14TU as the closest match, which had 97 to 100% identity with the reference strain Acinetobacter GS 13BJ/14TU CIP 64.2 (GenBank accession number DQ207478.1) (Table 1).

A phylogenetic tree was generated using the 450-bp partial rpoB gene sequences to identify relationships among the 11 Acinetobacter GS 13BJ/14TU bloodstream isolates (isolates 1 to 11), along with the Acinetobacter species reference strains available in GenBank (Fig. 1). In this analysis, all 11 bloodstream isolates were well differentiated from other Acinetobacter species, clearly clustering with Acinetobacter GS 13BJ/14TU ATCC 17905 (CIP 64.2). The partial rpoB gene sequences of isolates 1, 3, 7, 9, and 10 were identical, as were those of isolates 5, 8, and 11.

Phenotypic identification and antimicrobial susceptibilities. All 11 Acinetobacter GS 13BJ/14TU bloodstream isolates were characterized using three commercial methods: Vitek 2, MicroScan, and the API 20 NE system (Table 1). Vitek 2 misidentified 10 of 11 isolates as A. baumannii, with the remaining isolate identified as Acinetobacter lwaffii. Similarly, MicroScan misidentified 9 of 11 isolates as A. baumannii/A. haemolyticus, with the remaining two isolates identified as Providencia stuartii and A. lwaffii. The API 20 NE system identified all isolates as A. haemolyticus with high predictive values. The code numbers obtained by the API 20 NE system were 0010053 for nine isolates and 0010051 for two isolates. Using the API 20 NE system, 100% of isolates (11/11) showed gelatin hydrolysis, and assimilation of capric acid, malate, and citrate; 82% (9/11) showed assimilation of phenylacetic acid. All 11 bloodstream isolates exhibited beta hemolysis on blood agar plates.

Antimicrobial susceptibility testing revealed that all (100%) bloodstream isolates were susceptible to the following 11 antimicrobial agents: ampicillin-sulbactam, piperacillin-tazobactam, ticarcillin-clavulanic acid, cefazidime, cefepime, ceftiraxone, imipenem, meropenem, gentamicin, tobramycin, and amikacin. Ten of 11 isolates (91%) were also susceptible to tetracycline and ciprofloxacin. However, despite this widespread susceptibility to common antimicrobial agents, all 11 isolates were resistant to colistin as determined by the agar dilution method (Table 1).

MLST and PFGE analysis. Three new alleles (gltA 42, pyrG 28, and recA 46) and four new sequence types (STs) (ST198, ST199, ST200, and ST201) were identified by MLST and were added to the MLST database (see http://www.pasteur.fr/mlst). Overall, five distinct STs were obtained from a combination of seven housekeeping genes (Table 1). Of the five STs, ST198 and ST200 were shared by six and two isolates, respectively, with the remaining three STs (ST69, ST199, and ST201) found in one isolate each. The control strain, Acinetobacter GS 13BJ/14TU ATCC 17905, was classified as ST69. Within the phylogenetic tree, all 11 bloodstream isolates clustered with the reference strains Acinetobacter GS 13BJ/14TU ATCC 17905 and Acinetobacter GS 13BJ/14TU LUH 1718 (Fig. 2). This cluster was distinct from the Acinetobacter calcoaceticus-A. baumannii complex that comprised A. baumannii, A. calcoaceticus, A. nosocomialis, and A. pittii. Acinetobacter GS
<table>
<thead>
<tr>
<th>Isolate no.</th>
<th>Date of isolation (yr-mo-day)</th>
<th>Species identified by:</th>
<th>Antimicrobial susceptibility</th>
<th>MLST</th>
<th>PFGE</th>
<th>rpoB gene sequence analysis</th>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>1</td>
<td>2000-01-01</td>
<td>Acinetobacter GS 13BJ/14TU (97)</td>
<td>A. baumannii/haemolyticus (99.99)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except colistin (R) 18-22-42</td>
</tr>
<tr>
<td>2</td>
<td>2004-03-15</td>
<td>Acinetobacter GS 13BJ/14TU (100)</td>
<td>A. baumannii/haemolyticus (98.18)</td>
<td>A. haemolyticus (95.2)</td>
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<td>All S, except tetracycline (R) and colistin (R) 18-22-42</td>
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<td>2005-10-18</td>
<td>Acinetobacter GS 13BJ/14TU (97)</td>
<td>A. baumannii/haemolyticus (98.18)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except colistin (R) 21-25-24</td>
</tr>
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<td>4</td>
<td>2008-08-03</td>
<td>Acinetobacter GS 13BJ/14TU (100)</td>
<td>A. baumannii/haemolyticus (98.18)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except colistin (R) 21-25-24</td>
</tr>
<tr>
<td>5</td>
<td>2009-07-24</td>
<td>Acinetobacter GS 13BJ/14TU (99)</td>
<td>A. baumannii/haemolyticus (98.18)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except colistin (R) 21-25-24</td>
</tr>
<tr>
<td>6</td>
<td>2009-10-01</td>
<td>Acinetobacter GS 13BJ/14TU (97)</td>
<td>A. baumannii/haemolyticus (98.18)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010051</td>
<td>All S, except colistin (R) 18-22-42</td>
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<td>7</td>
<td>2009-11-03</td>
<td>Acinetobacter GS 13BJ/14TU (97)</td>
<td>A. baumannii/haemolyticus (66.99)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except colistin (R) 18-22-42</td>
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<td>8</td>
<td>2010-05-11</td>
<td>Acinetobacter GS 13BJ/14TU (99)</td>
<td>A. baumannii/haemolyticus (95.16)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except colistin (R) 18-22-42</td>
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<td>9</td>
<td>2010-07-03</td>
<td>Acinetobacter GS 13BJ/14TU (97)</td>
<td>A. lwoffii (97)</td>
<td>A. baumannii/haemolyticus (98.18)</td>
<td>A. haemolyticus (95.2)</td>
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<td>10</td>
<td>2011-07-11</td>
<td>Acinetobacter GS 13BJ/14TU (97)</td>
<td>A. baumannii/haemolyticus (98.18)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except ciprofloxacin (I) and colistin (R) 18-22-42</td>
</tr>
<tr>
<td>11</td>
<td>2011-08-09</td>
<td>Acinetobacter GS 13BJ/14TU (99)</td>
<td>A. baumannii/haemolyticus (75.88)</td>
<td>A. haemolyticus (95.2)</td>
<td>0010053</td>
<td>All S, except colistin (R) 21-25-24</td>
</tr>
</tbody>
</table>

**Note:** Numbers in parentheses are the probabilities of correct identification (as percentages).

**a** Antimicrobial susceptibility testing was performed using the disk diffusion method for ampicillin-sulbactam, piperacillin-tazobactam, ticarcillin-clavulanic acid, ceftazidime, cefepime, ceftriaxone, imipenem, meropenem, gentamicin, tobramycin, amikacin, tetracycline, and ciprofloxacin; colistin susceptibility was determined using the agar dilution method. S, susceptible; I, intermediate; R, resistant.

**b** MLST, multilocus sequence typing.

**c** Allele profiles for *cpn60-fusA-gltA-pyrG-recA-rplB-rpoB*.

**d** Three new alleles and four new STs identified in this study were added to the Institut Pasteur MLST database.

**e** PFGE, pulsed-field gel electrophoresis.

**f** ST, sequence type.
15BJ LUH 1729, previously described as ST66, was also distinct from the cluster of *Acinetobacter* GS 13BJ/14TU. The representative PFGE patterns for each of the 11 bloodstream isolates are shown in Fig. 3. PFGE of *ApaI*-digested genomic DNA revealed 11 distinct PFGE patterns (Table 1). In the two patients (patients 8 and 11) from whom serial bloodstream isolates were collected, all strains identified from each patient exhibited identical MLST and PFGE patterns.

**Clinical characteristics.** The clinical characteristics of the 11 patients with *Acinetobacter* GS 13BJ/14TU bacteremia are summarized in Table 2. Most patients had severe comorbidities, such as leukemia, infective endocarditis, and acute myocardial infarct...
tion, or underwent invasive procedures, such as tracheal intubation, central venous catheterization, coronary artery intervention, and peritoneal dialysis. No previous use of colistin was identified in any of the 11 patients. Ten patients were deemed to have nosocomial infections, while the remaining patient was an outpatient undergoing regular peritoneal dialysis. While all patients with Acinetobacter GS 13BJ/14TU bacteremia had excellent outcomes with cleared bacteremia, four patients (patients 1, 5, 8, and 11) were repeatedly culture positive (two to seven times), with continuous, persistent bacteremia (6 to 17 days). These four patients with continuous bacteremia were implicated in necrotizing pneumonia, infective endocarditis, primary bacteremia, and peritonitis.

Nucleotide sequence accession numbers. The zone 2 rpoB sequences of isolates 1, 5, and 6 have been deposited in GenBank under accession numbers KC138716, KC138718, and KC138717, respectively.
species identification, as reported previously (2, 29–31). This study describes the first description of the Acinetobacter GS 13BJ/14TU isolate. In addition, this isolate is intrinsically resistant to colistin. In this study, all 11 bloodstream isolates of Acinetobacter GS 13BJ/14TU were susceptible to a wide range of clinically relevant antibiotics, but all exhibited significant in vitro resistance to colistin. As none of the isolates included in this study were recovered from patients with a history of colistin therapy, these data strongly suggest that Acinetobacter GS 13BJ/14TU is intrinsically resistant to colistin. In addition, this report is noteworthy from a clinical standpoint in that it represents the first description of Acinetobacter GS 13BJ/14TU as an opportunistic pathogen capable of causing health-care-associated bacterial infections with favorable outcomes. These data highlight the importance of proper identification of Acinetobacter at the species level, and the need for physicians to be aware of the antimicrobial susceptibility profiles of different Acinetobacter species, particularly those causing bloodstream infections.

Although all 11 bloodstream isolates were misidentified by the phenotypic identification methods using the Vitek 2, MicroScan, and API 20 NE systems, all were correctly identified as Acinetobacter GS 13BJ/14TU by rpoB gene (zone 2) sequencing, confirming the suitability of this region for species identification, as reported previously (13, 27, 28). Misidentification by commercial systems may have arisen due to limitations in existing databases and the lack of nonspecialized substrates for Acinetobacter GS 13BJ/14TU species identification, as reported previously (2, 29–31).

All of our isolates produced beta hemolysis on blood agar plates, a phenotype not observed in A. calcoaceticus-A. baumannii complex species (2, 10, 32). Further characterization of these isolates using the API 20 NE system revealed gelatin hydrolysis and assimilation of capric acid, malate, and citrate, similar to A. haemolyticus (33). These phenotypic characteristics likely explain why the API 20 NE system misidentified these isolates as A. haemolyti-
cus with high predictive values. However, further characterization revealed assimilation of phenylacetic acid in 9 of 11 isolates (82%), a finding that is inconsistent with identification of A. haemolyticus because <1% of all A. haemolyticus isolates exhibit such a phenotype, information which is present in the manufacturer’s database and a previous report (33). A previous report also showed 100% susceptibility (10/10 isolates) of A. haemolyticus to colistin (8), in contrast to the results described here. Taken together, our study identified three characteristic features that can be used to diagnose Acinetobacter GS 13BJ/14TU infections in clinical microbiology laboratories. These features include (i) evidence of beta hemolysis on blood agar plates, (ii) colistin resistance, and (iii) positive identification as A. haemolyticus by the API 20 NE system, especially when combined with positive assimilation of phenylacetic acid. Molecular methods remain necessary for definitive species identification; however, these criteria may allow early detection of Acinetobacter GS 13BJ/14TU infections.

Two MLST schemes for Acinetobacter species identification are currently available, those of Bartual et al. (6) and the Institut Pasteur. Of these two, only the Institut Pasteur MLST scheme provided a successful performance of MLST testing for all isolates of Acinetobacter GS 13BJ/14TU, suggesting that the scheme of the Institut Pasteur may be a more suitable MLST method for this species. All bloodstream Acinetobacter GS 13BJ/14TU isolates are well clustered with the ST 65 of Acinetobacter GS 13BJ/14TU LUH 1718 (SEIP 5.84) and the ST 69 of Acinetobacter GS 13BJ/14TU LUH 1717 (ATCC 17905) reference strains of previous reports (20, 21), suggesting that MLST is suitable for data transfer and comparison and thus providing a tool for long-term or global epidemiological studies (6, 34). Furthermore, all MLST concatenated sequences for Acinetobacter GS 13BJ/14TU were easily differentiated from other Acinetobacter species (Fig. 2). These findings suggest that STs 65, 69, 198, 199, 200, and 201 may be indicative of Acinetobacter GS 13BJ/14TU strains, and the MLST can be used as genetic identification tool for this species.

This study represents the first report of the genotypic relationships among Acinetobacter GS 13BJ/14TU clinical isolates using...
<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (yr)/gender</th>
<th>Department/ward</th>
<th>Hospital days from admission to first bacteremia</th>
<th>Underlying diseases</th>
<th>No. of blood cultures (days)</th>
<th>ICU stay</th>
<th>Clinical status at positive culture</th>
<th>Previous use of colistin/other antibiotics</th>
<th>Previous use of steroid/neutropenia</th>
<th>Recent operation</th>
<th>Probable primary site of infection</th>
<th>Invasive procedures at bacteremia onset</th>
<th>Antibiotic therapy</th>
<th>Outcome of bacteremia/mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>76/F</td>
<td>CS/SICU</td>
<td>46</td>
<td>Leukemia</td>
<td>7 (0, 1, 2, 9, 11, 14, 17)</td>
<td>No</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Necrotizing pneumonia</td>
<td>Tracheal intubation, CVC</td>
<td>Y</td>
<td>Cleared/alive</td>
</tr>
<tr>
<td>2</td>
<td>76/M</td>
<td>ID/ERID</td>
<td>58</td>
<td>Duodenal ulcer bleeding and perforation, coronary artery disease</td>
<td>2 (0, 4, 7)</td>
<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Primary bacteremia</td>
<td>CVC, TPN, GI endoscopy, NG tube, IVI, Foley catheter</td>
<td>Y/N</td>
<td>Cleared/alive</td>
</tr>
<tr>
<td>3</td>
<td>71/M</td>
<td>CV/8761</td>
<td>78</td>
<td>Renal impairment, gout</td>
<td>1 (0, 2, 3, 4, 7, 8)</td>
<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Pneumonia</td>
<td>Coronary angiography/intervention, TPN</td>
<td>Y</td>
<td>Cleared/alive</td>
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<tr>
<td>4</td>
<td>80/M</td>
<td>CV/8766</td>
<td>32</td>
<td>Liver cirrhosis, gastric ulcer</td>
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<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Pneumonia</td>
<td>CVC, hemodialysis</td>
<td>Y</td>
<td>Cleared/alive</td>
</tr>
<tr>
<td>5</td>
<td>68/M</td>
<td>CV/MICU</td>
<td>78</td>
<td>Mitral regurgitation, heart failure</td>
<td>2 (0, 6)</td>
<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Pneumonia</td>
<td>Coronary angiography/TPN, TPN</td>
<td>Y</td>
<td>Cleared/alive</td>
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<td>6</td>
<td>85/F</td>
<td>HPBS/B866</td>
<td>6</td>
<td>Dermatomyositis, DM</td>
<td>4 (0, 4, 7, 8)</td>
<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Pneumonia</td>
<td>TPN, arterial catheter, TPN, NG tube, IVI, Foley catheter</td>
<td>Y</td>
<td>Cleared/alive</td>
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<tr>
<td>7</td>
<td>75/M</td>
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<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
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<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
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<td>83/F</td>
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<td>12</td>
<td>Choledocholithiasis</td>
<td>4 (0, 4, 7, 8)</td>
<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Pneumonia</td>
<td>CVC, TPN, TPN, Coronary angiography</td>
<td>Y/N</td>
<td>Cleared/alive</td>
</tr>
<tr>
<td>10</td>
<td>48/M</td>
<td>CV/MICU</td>
<td>11</td>
<td>Cerebral vascular accident</td>
<td>1 (0, 1, 2, 9, 11, 14, 17)</td>
<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Pneumonia</td>
<td>TPN, TPN, None</td>
<td>Y</td>
<td>Cleared/alive</td>
</tr>
<tr>
<td>11</td>
<td>42/M</td>
<td>DM</td>
<td>11</td>
<td>End-stage renal disease, DM</td>
<td>1 (0, 1, 2, 9, 11, 14, 17)</td>
<td>N/N</td>
<td>Growth</td>
<td>N/Y</td>
<td>N/N</td>
<td>N</td>
<td>Pneumonia</td>
<td>TPN, TPN, None</td>
<td>Y</td>
<td>Cleared/alive</td>
</tr>
</tbody>
</table>

**Note:**
- CS, cardiothoracic surgery; SICU, surgical intensive care unit; GI, gastrointestinal; ER, emergency room; ID, infectious disease; CV, cardiovascular; RHD, rheumatic disease; MICU, medical intensive care unit; HPBS, hepatopancreaticobiliary surgery; NR, neurology; KD, kidney disease; DM, diabetes mellitus; COPD, chronic obstructive pulmonary disease; CAPD, continuous ambulatory peritoneal dialysis; CVC, central venous catheterization; TPN, total parenteral nutrition; NG, nasogastric; IVI, intravascular intervention; Y, yes; N, no.
- Days are numbered relative to the time of first positive blood culture.
both MLST and PFGE analysis. In the current study, MLST differentiated 11 isolates into 5 STs, whereas PFGE revealed 11 distinct patterns, suggesting that PFGE is a more discriminatory method than MLST for this species. Despite the apparent divergence among PFGE patterns of isolates from different patients, each of the duplicate isolates from patients 8 and 11 exhibited identical MLST and PFGE patterns, indicating a high degree of reproducibility for these two methods. In the present study, none of the bloodstream isolates were highly related spatiotemporally, and PFGE showed 11 distinct patterns, suggesting that each bacteremia occurred sporadically. However, as PFGE patterns are likely to reflect rapidly evolving genetic markers, isolates with identical STs but different PFGE patterns may have been derived from a recent common ancestor (35). These data suggest that the PFGE method may be useful for epidemiological typing of Acinetobacter GS 13B]/14TU isolates, while MLST is more effective for species identification.

Although Acinetobacter GS 13B]/14TU has been implicated in cases of septic shock, endocarditis, and pyrexia (1), little is known about the clinical characteristics of Acinetobacter GS 13B]/14TU bacteremia. Our data suggest that Acinetobacter GS 13B]/14TU can cause health care-associated bacteremia in patients with severe underlying diseases or those who had undergone invasive medical procedures. The isolates from seven patients were recovered from blood just once, and the isolate from one patient disappeared without antibiotic therapy. This finding suggests that this species may cause transient bacteremia. However, four patients who had severe underlying diseases such as leukemia, mitral re-gurgitation, uncontrolled diabetes mellitus, or end-stage renal disease showed persistent bacteremia (6 to 17 days), demonstrating this species may be implicated in necrotizing pneumonia, infective endocarditis, primary bacteremia, or sepsis. However, despite the persistent bacteremia in some patients, all Acinetobacter GS 13B]/14TU bacteremias were eventually cleared, with positive outcomes in all 11 patients. These observations are consistent with previous reports of significant differences in the clinical features, including mortality, among Acinetobacter species, with A. baumannii associated with significantly worse outcomes compared to other Acinetobacter species (36–41). This work therefore highlights the importance of correct GS identification, which may facilitate prediction of the outcome of Acinetobacter bacteremia.

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REFERENCES


