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## Bacteremia Caused by *Acinetobacter ursingii*

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***Acinetobacter ursingii* has not been reported in infectious processes apart from its recent description as a new species. A bacteremia caused by *A. ursingii* in a patient with a pulmonary adenocarcinoma confirms that this microorganism is an opportunistic human pathogen. The isolate was susceptible to imipenem, aminoglycosides, rifampin, and fluoroquinolones.**

### CASE REPORT

A 63-year-old man presenting with a pulmonary adenocarcinoma diagnosed in July 2001 was admitted to the University Hospital Hôtel Dieu, Paris, France. A fifth course of intravenous chemotherapy consisting of cisplatin (50 mg/m<sup>2</sup> of body area) and vinorelbine (30 mg/m<sup>2</sup>) was initiated through a catheter chamber, which had been implanted 12 weeks earlier. The patient had received corticosteroids for 2 months because of thoracic pain due to tumoral compression. On the day following admission (day 1), his condition deteriorated with fever (39.5°C) and chills associated with an increase of white blood cell count (15 × 10<sup>3</sup> cells per μl with 90% neutrophils), C-reactive protein (9.5 mg/dl), and fibrinogen (0.51 mg/dl). Thus, he received cefotaxime (3 g per day) and gentamicin (180 mg per day) for 2 days. Urine analysis was normal. The chest X ray and the abdominal and cardiac echography were not modified. A strain of an *Acinetobacter* sp. was isolated from five blood samples, including two obtained from the catheter chamber. The catheter chamber was removed, but its culture was sterile. On day 3, the antimicrobial therapy was changed to imipenem (2 g per day) and amikacin (900 mg per day) for 2 weeks according to the susceptibility of the strain. On day 5, rifampin (1.2 g per day) was added as the patient remained febrile. On day 7, the patient was afebrile, with normalization of white blood cell count and C-reactive protein.

Blood samples were inoculated in aerobic and anaerobic blood culture vials (BACTEC PLUS; BD Diagnostic Systems, Sparks, Md.). Aerobic vials were positive and were subcultured on nutrient agar at 37°C. After 24 h of incubation, colonies were 1 to 1.5 mm in diameter, circular, convex, smooth, and slightly opaque with entire margins. Staining of the bacteria showed gram-negative coccobacilli. Growth in brain heart infusion (BHI) broth was observed at 37°C but not at 41 and 44°C. The microorganism (isolate 954) was nonmotile, strictly aerobic, and oxidase negative. It grew on MacConkey agar (colorless colonies), was nonhemolytic on sheep blood agar, did not oxidize D-glucose, did not reduce nitrate, and was

urease and gelatinase negative. In an attempt to identify this isolate, the strips API 20 NE and API ID 32 GN (bioMérieux, Marcy l'Etoile, France) were used as recommended by the manufacturer. The repeated bacterial identifications obtained with API 20 NE and API ID 32 GN strips were *Acinetobacter junii* or *Acinetobacter johnsonii* (code no. 0000071; percentage of identification [*p*] = 63.5%; index of typicity [*T*] = 0.77) and *A. johnsonii* (code no. 00270063062; *p* = 90.5%; *T* = 0.87), respectively. These results prompted us to determine the 16S rRNA gene (16S ribosomal DNA [rDNA]) sequence of the isolate as previously described (3, 6). Briefly, the 16S rDNA was amplified by PCR with the primers Ad (5'-AGAGTTTG ATC[A,C]TGGCTCAG-3') and rJ (5'-GGTTACCTTGTTAC GACTT-3'). A total of 1,484 continuous nucleotides of 16S rDNA were determined. The complete 16S rDNA sequence of the isolate was compared to all bacterial sequences available from the GenBank database by using the Blast program (National Center for Biotechnology Information) and showed 99% similarity to that of the type strain of *Acinetobacter ursingii* (GenBank accession no. AJ275038). 16S rDNA sequences from phylogenetically related strains were obtained from the GenBank database. All 16S rDNA sequences were aligned with CLUSTAL X, and a phylogenetic tree was constructed by using DENDROGRAF, a program of the Taxotron package (Taxolab Institut Pasteur, Paris, France) (Fig. 1). Antimicrobial susceptibility of the isolate was determined by the agar diffusion method using the Epsilometer test (E test; AB BIO-DISK, Solna, Sweden) on Mueller-Hinton agar as recommended by the manufacturer. MIC results were as follows: amoxicillin, 16 μg/ml; piperacillin, 12 μg/ml; cefotaxime, 32 μg/ml; cefepime, 24 μg/ml; ceftazidime, 128 μg/ml; imipenem, 0.125 μg/ml; gentamicin, 0.25 μg/ml; amikacin, 1 μg/ml; tobramycin, 0.5 μg/ml; rifampin, 3 μg/ml; ciprofloxacin, 0.19 μg/ml. An assay for the detection of beta-lactamase by using a nitrocefin disk (BD Cefinase; BD Diagnostic Systems) was positive.

Members of the genus *Acinetobacter* belong to the gamma subdivision of the class *Proteobacteria*. In 1986, Bouvet and Grimont described four new species of *Acinetobacter*, namely, *Acinetobacter baumannii*, *Acinetobacter haemolyticus*, *A. johnsonii*, and *A. junii* (2). In addition, these authors emended the descriptions of two other species, *Acinetobacter calcoaceticus* and *Acinetobacter lwoffii*. In 1988, *Acinetobacter radioresistens*,

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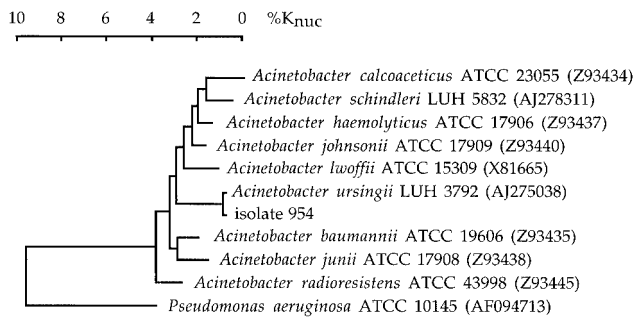


FIG. 1. Phylogenetic tree of isolate 954 of *A. ursingii* and the type strains of related species based on comparative analysis of 16S rRNA gene sequences. Sequence alignment was by the CLUSTAL method. The dendrogram was generated by using the neighbor-joining algorithm and choosing *Pseudomonas aeruginosa* as the outgroup. %K<sub>nuc</sub>, percentage of nucleotide substitution.

from the environment, was described (9). More recently, *A. ursingii* and *Acinetobacter schindleri*, isolated from human clinical specimens, have been delineated (7, 8). Thus, at present, the genus *Acinetobacter* consists of the above nine species. However, this remains insufficient to name all the members of this genus, as shown by the 21 different DNA groups (genospecies) previously reported (5). In clinical laboratories, the identification of nonfermentative gram-negative rods is usually carried out by using identification systems such as the API 20 NE and the API ID 32 GN strips (bioMérieux). *A. baumannii*, the most frequent species of *Acinetobacter* involved in nosocomial infections, is easily identified with these systems. However, the discriminative power of the tests using API strips has been shown to be insufficient for accurate identification of the other species of *Acinetobacter* (1). In the present case, the preliminary misidentification was due to the absence of *A. ursingii* in the databases of the API 20 NE and API ID 32 GN strips. Additional phenotypic tests, such as growth in BHI broth at 37 and 41°C and oxidization of glutarate and L-aspartate, may help to differentiate *A. ursingii* from *A. junii* and *A. johnsonii* (Table 1).

*Acinetobacter* species are commonly isolated from the environment and may also be isolated from humans (skin and mucous membranes) (10). During the last 2 decades, they have emerged as nosocomial pathogens. In debilitated patients, they may be responsible for severe and fatal infections involving the respiratory tract, the urinary tract, and wounds (including catheter sites). Risk factors for infection include serious underlying

TABLE 1. Phenotypic tests for differentiating between *A. ursingii*, *A. junii*, and *A. johnsonii*<sup>a</sup>

Test	Result <sup>b</sup> for:		
	<i>A. ursingii</i>	<i>A. junii</i>	<i>A. johnsonii</i>
Growth at 37°C in BHI broth	+	+	–
Growth at 41°C in BHI broth	–	+	–
Oxidization of glutarate	+	–	–
Oxidization of L-aspartate	+	–	V

<sup>a</sup> Data are from Nemeč et al. (8).

<sup>b</sup> +, positive for 90 to 100% of strains; –, positive for 0 to 10% of strains; V, positive for 11 to 89% of strains.

disease such as cancer, intravascular or intravesical catheterization, treatment with broad-spectrum antibiotics or corticosteroids, prolonged hospital stay, and stay in intensive care units. Due to their prolonged survival in the environment, *Acinetobacter* species may spread among patients and cause hospital-associated outbreaks (4, 11). In the present case, the pulmonary adenocarcinoma and the corticosteroid administration were two major risk factors for the dissemination of *A. ursingii* in blood. Although *A. ursingii* has been isolated solely from humans, its natural habitat is not known. We assume that this isolate colonized the patient's skin and that the intravascular catheterization triggered its spread to the bloodstream. The accurate identification of the species of *Acinetobacter* is important for epidemiological and therapeutic reasons. Improvement of the taxonomy and of the methods of identification must be taken into account when analyzing previous studies, which often used names or methods no longer valid. The *Acinetobacter* species involved in human infections and their antimicrobial susceptibilities remain partly undetermined. *A. ursingii* has not been reported in infectious processes apart from its recent description as a new species (8). However, this species may have the same medical importance as our isolate. Indeed, out of the 29 strains reported in the literature, 13 were isolated from the blood of patients suffering from severe underlying disease. Furthermore, *A. ursingii* strains may have the potential to spread to other patients, as demonstrated by molecular typing (7). For these reasons, clinical microbiologists must be aware of the opportunistic pathogenicity of this newly described species, which deserves further studies to determine its prevalence in humans.

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