Case Report

First case of post-endoscopic retrograde cholangiopancreatography bacteraemia caused by Acinetobacter ursingii in a patient with choledocholithiasis and cholangitis

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We describe what we believe to be the first case of biliary sepsis caused by Acinetobacter ursingii. The patient was a healthy woman with no comorbidities who presented with choledocholithiasis and cholangitis. The performance of an endoscopic cholangiopancreatography was the trigger for A. ursingii bacteraemia. This report highlights the inadequacies of conventional phenotypic tests usually available in clinical microbiology laboratories for the identification of Acinetobacter species.

Case report

A 47-year-old woman was admitted to hospital with fever and ictericia. Twelve days earlier, she had undergone a cholecystectomy due to cholelithiasis. Seven days after surgery, an endoscopic retrograde cholangiopancreatography (ERCP) was performed because of cholestatic jaundice with suspicion of choledocholithiasis. The ERCP confirmed choledocholithiasis and cholangitis with abundant purulent exudate within the choledochus. An endoscopic sphincterotomy was performed. No samples were taken for culture and no microbiological studies were done at that time. Two days after the ERCP was performed, the patient was discharged from hospital, but 24 h later she had to be admitted again because of high fever. Three blood cultures were taken and therapy with cefotaxime was started.

Blood samples were inoculated in aerobic and anaerobic blood culture vials (Bactec Plus; BD Diagnostics Systems). Two aerobic vials were positive and were subcultured on chocolate and 5% horse blood agar plates (bioMérieux). After 24 h incubation, colonies were 1–1.5 mm in diameter, circular and convex. No haemolysis was produced on 5% horse blood agar. The isolate was a strictly aerobic Gram-negative coccobacillus, with positive catalase and negative oxidase reactions. It did not grow on MacConkey agar (bioMérieux).

Phenotypic identification was made using the NegCombi 36 panel MicroScan (Dade Behring) and the API 20 NE strip (bioMérieux) in accordance with the manufacturers’ instructions. The strain was identified as Acinetobacter lwoffii. With API 20 NE, the percentage of identification obtained was 86.9% (code no. 0040000). According to the MicroScan system database, the only identification obtained was A. lwoffii (biotype 0000040).

Antimicrobial susceptibility of the isolate was determined using the NegCombi 36 panel (MicroScan). The strain was resistant to cephalotin and cephalozin (MIC >16 µg ml⁻¹), and susceptible to ampicillin (MIC ≤8 µg ml⁻¹), amoxicillin-clavulanate (MIC ≤8 µg ml⁻¹), cefuroxime (MIC ≤4 µg ml⁻¹), cefotaxime (MIC ≤8 µg ml⁻¹), cefotaxime (MIC =4 µg ml⁻¹), ceftazidime (MIC ≤1 µg ml⁻¹), cefepime (MIC =4 µg ml⁻¹), imipenem (MIC ≤2 µg ml⁻¹), aztreonam (MIC ≤1 µg ml⁻¹), ciprofloxacin (MIC =1 µg ml⁻¹) and aminoglycosides (MIC ≤4 µg ml⁻¹).

For genotypic identification, the isolate was sent to the Microbiology National Center (Instituto de Salud Carlos III, Madrid, Spain) to determine the 16S rRNA gene sequence. A total of 1477 bp of 16S rRNA was determined using a method previously described (Drancourt et al., 2000). The sequence obtained was compared to bacterial sequences available from the GenBank database using the BLAST program (National Center for Biotechnology Information). The strain was identified as Acinetobacter ursingii and showed 99% similarity to the type strain of A. ursingii.

Abbreviation: ERCP, endoscopic retrograde cholangiopancreatography.
The patient had a favourable clinical course. She received therapy with intravenous cefotaxime for 1 week and oral cefuroxime for another week.

Discussion
To the best of our knowledge, this is the first description of biliary sepsis caused by *A. ursingii* that occurred in an immunocompetent patient with choledocholithiasis and cholangitis.

The taxonomy of the *Acinetobacter* genus has undergone extensive revision since 1986 (Juni, 2005); the genus currently includes 32 genomic species, 17 of which have been validated. Since its recent description in 2001 as a novel species (Nemec *et al.*, 2001), *A. ursingii* has been isolated in a few human cases (Loubinoux *et al.*, 2003; Romero-Gómez *et al.*, 2006; Dortet *et al.*, 2006). Apart from the strains reported in the taxonomic description of the species, only five cases of bacteraemia caused by *A. ursingii* have been reported, and all of them occurred in patients with severe underlying comorbidities, most of them immunocompromised patients (Loubinoux *et al.*, 2003; Romero-Gómez *et al.*, 2006; Dortet *et al.*, 2006). Most of the strains were isolated from blood or other sterile body site cultures in seriously ill hospitalized patients (Nemec *et al.*, 2001; Loubinoux *et al.*, 2003; Romero-Gómez *et al.*, 2006; Dortet *et al.*, 2006). To our knowledge, only one previous strain of *A. ursingii* has been recovered from a patient considered to have cholangitis (Dortet *et al.*, 2006). However, in that patient, *A. ursingii* was isolated in a single blood culture and only a ‘possible’ causality relationship could be established. In the present case report, the bacterium was isolated from two different blood cultures and cholangitis was evident by ERCP, thus confirming that this species can cause bacteraemic biliary tract infection. The case is also unique because, unlike in previous reports, our patient was a healthy woman with no comorbidities who presented with cholelithiasis, choledocholithiasis and cholangitis. The performance of ERCP was, in this patient, the most likely trigger for *A. ursingii* bacteraemia.

This report highlights the inadequacies of conventional phenotypic tests usually available in clinical microbiology laboratories for the identification of *Acinetobacter* species. At present, no rapid and accurate commercial phenotypic identification exists for new *Acinetobacter* species (Dortet *et al.*, 2006). Genotypic methods are needed for definitive identification confirmation since accurate identification of *Acinetobacter* species may have epidemiological and therapeutic implications.

References


