Liver abscess caused by *Brevundimonas vesicularis* in an immunocompetent patient

Seu Hee Yoo,¹ Min Ja Kim,¹ Kyoung Ho Roh,² Si Hyun Kim,¹ Dae Won Park,¹ Jang Wook Sohn¹ and Young Kyung Yoon¹

¹Division of Infectious Diseases, Department of Internal Medicine, Korea University College of Medicine, Seoul, Republic of Korea
²Department of Laboratory Medicine, Korea University College of Medicine, Seoul, Republic of Korea

Invasive infections caused by *Brevundimonas vesicularis* are very rare in humans. We experienced an unusual case of liver abscess due to *B. vesicularis* in an immunocompetent young male. The patient was successfully treated by liver abscess drainage and with antimicrobial therapy of ceftriaxone followed by ampicillin/sulbactam. The organism found in the aspiration culture of the abscess material was initially reported, by using a VITEK 2 system, as *Sphingomonas paucimobilis*. However, later, *B. vesicularis* was confirmed as the true pathogen through 16S rRNA gene sequencing. To our knowledge, this is the first case of liver abscess caused by *B. vesicularis*.

**Introduction**

In 1994, *Brevundimonas vesicularis*, formerly grouped with *Pseudomonas*, was reclassified as a *Brevundimonas* species based on DNA–rRNA hybridization studies, 16S rRNA cataloguing and 16S rRNA gene sequencing (Segers et al., 1994). This organism has been isolated from various environmental sources, but there are rare reports in the literature of infections caused by *B. vesicularis* in both immunocompromised and immunocompetent patients.

Universally, *Brevundimonas* species have been considered to be opportunistic pathogens in immunocompromised hosts. However, the role that they play in human clinical specimens from patients without significant underlying diseases has not been determined. Furthermore, there has been no establishment of antibiotic therapeutic options, because of insufficient clinical data about the efficacy of antimicrobial agents and the wide variation of *in vitro* susceptibilities to antibiotics. Here, we describe what we believe to be the first case of a patient presenting with liver abscess caused by *B. vesicularis* who was successfully treated with ceftriaxone, followed by ampicillin/sulbactam and by drainage of the liver abscess.

**Case report**

A 30-year-old man was admitted with abdominal pain and fever. He had no previous disease and a non-contributory past medical history. On admission, his body temperature was 38.8 °C. Abdominal examination revealed tenderness of the right upper quadrant area. Abdominal computed tomography (CT) showed an ill-defined, low-attenuating lesion which was 2.2 cm in size on the subcapsular portion of the liver segment 6, suggesting a liver abscess (Fig. 1a). Initial laboratory results showed a white blood cell count of 16 800 μl⁻¹, with 86.1 % neutrophils. The level of aspartate aminotransferase was 27 IU l⁻¹, alanine aminotransferase 29 IU l⁻¹, total bilirubin 1.13 IU l⁻¹ and C-reactive protein 72.54 mg l⁻¹. The anti-amoebic antibody serology test (ELISA) was negative. Under the clinical diagnosis of liver abscess, intravenous ceftriaxone (2 g, once a day) was given and percutaneous drainage of the abscess was performed. The drained pus showed Gram-negative bacilli and was negative for malignant cells on cytology analysis. On the third day of hospitalization, the patient became afebrile and was maintained with antibiotic therapy and abscess drainage. On the sixth day of hospitalization, follow-up laboratory tests showed a white blood cell count of 8100 μl⁻¹, with 68.1 % neutrophils and C-reactive protein 31.451 mg l⁻¹. On the seventh day of hospitalization, the percutaneous catheter for abscess drainage was removed after confirming the shrinkage of the abscess cavity by a tubogram. The causative organism from the pus culture was identified as *Sphingomonas paucimobilis*, by using the VITEK 2 system (bioMérieux), with a probability of 93.58 %. The antibiogram of the isolate showed susceptibility to amikacin (MIC ≤2 μg ml⁻¹), ampicillin/sulbactam (MIC ≤2 μg ml⁻¹) and imipenem (MIC ≤0.25 μg ml⁻¹), but resistance to aztreonam (MIC ≥64 μg ml⁻¹), ceftazidime (MIC ≥64 μg ml⁻¹), cefepime (MIC ≥64 μg ml⁻¹) and ciprofloxacin (MIC ≥4 μg ml⁻¹). A VITEK 2 AST-132 kit
Liver abscess caused by *Brevundimonas vesicularis*

Fig. 1. Contrast-enhanced CT scan images. (a) An abdominal CT scan showing an ill-defined, low attenuating lesion, 2.2 cm in size, on the subcapsular portion of liver segment 6, suggesting a liver abscess (arrow). (b) Follow-up abdominal CT scan showing that the abscess lesion had decreased to 0.8 cm in size (arrow).

(bioMérieux) was used and the criteria of the Clinical and Laboratory Standards Institute (CLSI) (CLSI, 2011) were followed. Meanwhile, antimicrobial susceptibility testing, with MICs determined by E tests, showed susceptibility to amoxicillin/clavulanate (1 μg ml⁻¹), ceftriaxone (8 μg ml⁻¹) and cefotaxime (2 μg ml⁻¹), and resistance to ceftazidime (128 μg ml⁻¹), imipenem (32 μg ml⁻¹) and ciprofloxacin (32 μg ml⁻¹). After completion of intravenous ceftriaxone treatment for 7 days, the patient was discharged with oral ampicillin/sublactam (750 mg, three times a day). Since a case of liver abscess caused by *S. paucimobilis* is very rare, we carried out 16S rRNA gene sequencing of the isolate, according to the CLSI recommendations (CLSI, 2008). Comparative sequence analysis revealed 100 and 87.0 % identity with the respective sequences corresponding to the 16S rRNA genes of *B. vesicularis* (ATCC 11426T) and *S. paucimobilis* (ATCC 31461), respectively. In addition, the commercial biochemical test API 20NE (bioMérieux) was used to determine the biochemical characteristics, which resulted in the identification of the isolate as *B. vesicularis* with 96.5 % probability and *S. paucimobilis* with 3.4 % probability. The isolate was found to have enzymic activity with positivity for oxidase, β-glucosidase and β-galactosidase, and the ability to weakly oxidize glucose and to hydrolyse aesculin. Twenty-one days after discharge, a follow-up abdominal CT scan showed a decrease in the size of the abscess cavity from 2.2 to 0.8 cm (Fig. 1b). The patient received a total of 5 weeks of therapy with ampicillin/sublactam before the termination of treatment. On the 6-week follow-up visit after the diagnosis, the patient was found to be free of disease.

Discussion

*B. vesicularis* is a Gram-negative, non-fermenting rod that has been very rarely implicated in human infections (Sofer et al., 2007). There have been several case reports of human infections caused by *B. vesicularis*, such as necrotizing cellulitis, septicemia, septic arthritis, subacute endocarditis, meningitis, peritonitis, etc. (Planes et al., 1992; Gilad et al., 2000; Oberhelman et al., 1994; Vanholder et al., 1992; Calegari et al., 1996; Chi et al., 2004; Papaefstathiou et al., 2005; Choi et al., 2006; Yang et al., 2006; Kwak et al., 2009; Vahid, 2006; Mondello et al., 2006; Pelletier et al., 2010; Bhawadkar & Sharma, 2011; Chandra et al., 2010). However, this is the first case of liver abscess caused by *B. vesicularis*, to our knowledge. Furthermore, although *B. vesicularis* infection has been classified as opportunistic and is rare in immunocompetent patients, our case indicates the possibility of *B. vesicularis* infection in a healthy young male. The source of the infection remains unknown in our case, since the patient had no predisposing risk factors. The clinical significance of this pathogen, therefore, should not be confined solely to opportunistic infections in immunocompromised hosts.

In our case, the pathogen from the pus culture was misidentified as *S. paucimobilis* by the VITEK 2 system, but subsequently was confirmed as *B. vesicularis* by sequencing the 16S rRNA gene. *S. paucimobilis* is a yellow-pigmented, non-fermenting, Gram-negative bacillus that has a single polar flagellum with slow motility (Von, 1995; Yabuuchi et al., 1990). In a study undertaken by Zbinden et al. (2007) to compare the VITEK 2 system with 16S rRNA gene sequencing for the identification of Gram-negative, non-fermentative rods, the VITEK 2 system identified 59 % (n=53) of the isolates to the species level and 10 % (n=9) to the genus level; 31 % (n=28) of the isolates were misidentified. Our findings support that any non-fermentative, Gram-negative rods identified by the VITEK 2 system, other than *Achromobacter xylosoxidans*, *Acinetobacter* species, the *Burkholderia cepacia* complex, *Pseudomonas aeruginosa* and *Stenotrophomonas maltophilia*, should be subjected to 16S rRNA gene sequencing for accurate identification.
Standards for the treatment of B. vesicularis infection have not been well established to date. According to a study from the Centers for Disease Control and Prevention, B. vesicularis is highly susceptible to all aminoglycosides (98%), piperacillin (100%), carbenicillin (94%) and cefotaxime (94%), with moderate susceptibility to cefamandole (50%) and cefoxitin (75%), and lower susceptibility to penicillin (12%) and ampicillin (22%) (Clark et al., 1984). However, a wide variation of susceptibility to antimicrobial agents has been described in previous reports (Planes et al., 1992). The Centers for Disease Control and Prevention, have not been well established to date. According to a study from the Centers for Disease Control and Prevention, B. vesicularis is highly susceptible to all aminoglycosides (98%), piperacillin (100%), carbenicillin (94%) and cefotaxime (94%), with moderate susceptibility to cefamandole (50%) and cefoxitin (75%), and lower susceptibility to penicillin (12%) and ampicillin (22%) (Clark et al., 1984). However, a wide variation of susceptibility to antimicrobial agents has been described in previous reports (Planes et al., 1992). To our knowledge, this is the first case of liver abscess due to B. vesicularis infection. Although B. vesicularis infection has been classified as opportunistic, especially in immunocompromised patients, this case highlights the possibility of B. vesicularis infection in an immunocompetent human with no predisposing risk factor. In addition, B. vesicularis isolates might be misidentified as S. paucimobilis by an automated biochemical system.

References


