**Elizabethkingia meningoseptica**: an unusual cause for septicaemia

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**Introduction:** *Elizabethkingia meningoseptica*, a Gram-negative non-fermenting bacterium, is usually associated with neonatal meningitis and other infections, especially in immunocompromised patients. It is a potential nosocomial pathogen and is usually resistant to several commonly used antimicrobials.

**Case presentation:** We here report a rare case of septicaemia caused by *E. meningoseptica* associated with peritonitis and choledocholithiasis. The patient succumbed in spite of diagnosis and institution of appropriate antibiotics.

**Conclusion:** A prompt diagnosis of infection with *E. meningoseptica* is important so as to guide the institution of appropriate antibiotic treatment.

**Keywords:** *Elizabethkingia meningoseptica*; peritonitis; septicaemia.

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**Introduction**

*Elizabethkingia meningoseptica*, previously known as *Chryseobacterium meningosepticum* and *Flavobacterium meningosepticum*, is a ubiquitous non-fermenting, non-motile, oxidase-positive, Gram-negative bacillus (King, 1959, Kim, 2005 & Vandamme, 1994). This organism is resistant to many antimicrobial agents, including those frequently used to target Gram-negative bacterial infections. It occasionally causes meningitis, nosocomial pneumonia, sepsis and bacteraemia in immune-compromised adults and neonates (Tak et al., 2013). We here report a case of peritonitis leading to septicaemia, caused by this emerging pathogen, in an immune-competent individual.

**Case report**

A 58-year-old man was brought to the intensive care unit of our hospital in a conscious but disoriented state. He had a history of abdominal pain for 9 days followed by high-grade fever and abdominal distension for 7 days. On examination, the patient had hypotension, tachycardia, cold clammy extremities and tender abdomen in the right upper quadrant with mild ascites, while examination of other systems was unremarkable. Emergency ultrasonography revealed several stones in the gall bladder fossa and peritoneal collection with multiple internal septation, but CT scan of the head and chest X-ray were normal. His leukocyte count was significantly raised at 22 000 cm$^{-1}$ (normal 4000–10 000 cm$^{-1}$) and he had an electrolyte imbalance, while all other haematological and biochemical parameters were within normal range at the time of admission. He had no past history of any other diseases and was HIV sero-negative. He was diagnosed as having choledocholithiasis with peritonitis. The management plan was first to control the infection, if any, and then to remove the gallstones later. The patient was handled with all sterile precautions, and started empirically with parenteral imipenem–cilastatin and teicoplanin along with intravenous fluids and other supportive managements, after collection of blood from two different sites, peritoneal fluid by diagnostic paracentesis and urine for culture. Urine culture did not show any significant growth, whereas peritoneal aspirate and two sets of peripheral blood samples cultured by an automated method (Bact/ALERT 3D; Biomerieux) flagged positive within 24–30 h. Samples cultured on sheep blood agar plates (Hi-Media) showed 1–2 mm smooth, circular, grey–white, non-haemolytic colonies after overnight incubation at 37°C, whereas there was no growth on MacConkey agar plates. This was a non-motile, catalase-, indole- and oxidase-positive, mannitol-, urease-, citrate- and nitrate-reduction-negative, Gram-negative bacillus with non-fermenting reaction on TSI agar, showing no growth at 42°C. In both samples the isolates were identified as being the same and belonging to the non-fermenter group. Antimicrobial susceptibility testing by the Kirby–Bauer disc-diffusion method on Mueller–Hinton agar as per Clinical Laboratory Standards Institute guidelines (CLSI, 2010) gave the same result for these. Identification and sensitivity was further confirmed by the
Vitek-2 compact automated system (BioMérieux), using the GN and N280 cards, respectively. The organism was identified as extended spectrum β-lactamase (ESBL)-producing *E. meningoseptica*, and it was sensitive to ceftoperazone–sulbactam (MIC ≤ 8), nalidixic acid (MIC 8), ciprofloxacin (MIC 0.5) and trimethoprim–sulfamethoxazole (MIC 40); in contrast, it was resistant to tigecycline (MIC 4). Therefore, ESBL production was suspected. The antibiotics were changed to ceftoperazone–sulbactam and ciprofloxacin. To prevent the spread of this infection in the hospital, adequate control measures were taken immediately. Unfortunately, the patient died of septicaemia caused by *E. meningoseptica* within hours of starting the specific therapy.

**Discussion**

*E. meningoseptica* is most commonly associated with meningitis and a variety of infections in premature infants and newborns. Till today, *E. meningoseptica* infection has been very rare in immunocompetent hosts such as our patient. Different types of infection by this bacterium were either nosocomial or occurred in patients with known predisposing factors like malignancy, neutropenia, diabetes, steroid use, malnutrition and being on dialysis. *E. meningoseptica* has a low degree of pathogenicity and only a small percentage of colonized patients develop sepsis, while others remain asymptomatic (Steinberg, 2000). But in our case the organism was isolated from an adult without prior history of any ailments or hospitalization. Also, this bacterium had not previously been isolated in our hospital. In our patient, septicemia and peritonitis following choledocholithiasis were attributed to this organism. This bacterium proliferates in the hospital environment, growing on moist surfaces such as sinks, water tanks, ventilator tubing, saline solution used for flushing devices, etc., thus making it an emerging hospital-acquired pathogen (Jean et al., 2014). As the isolate from our patient was an ESBL producer, it was treated with ceftoperazone–sulbactam and ciprofloxacin, to which the organism was sensitive. Infection with *E. meningoseptica* is clinically important as the organism is intrinsically resistant to multiple antibiotics, such as β-lactams, aminoglycosides, tetracycline, tigecycline, colistin, chloramphenicol and carbapenems (Bloch et al., 1997). However, it is susceptible to the agents used to treat Gram-positive bacteria: rifampicin, ciprofloxacin, vancomycin and trimethoprim–sulfamethoxazole. Yet adequate treatment for this organism has not been outlined. Vancomycin alone or in combination with other agents, which include rifampicin, has in the past been successful in treatment, but its efficacy has been questioned by recent studies (Ceyhan, 2011; Mland, 2011). These resistance phenotypes can be explained by the presence of β-lactamas, including ESBLs and metallo-β-lactamas. Thus, most of the cases due to improper antibiotic use cause serious life-threatening infections, particularly in intensive care units, where there is selective antibiotic pressure owing to the higher antibiotic use and presence of critically ill patients on multiple life-support devices. As our isolate was a community-acquired multidrug-resistant pathogen, the patient succumbed to septicemia before adequate management was instituted. In view of its multidrug-resistant nature, and its ability to infect an immunocompetent individual, as in our case, and to spread in the hospital environment, its prompt diagnosis in clinical samples and sensitivity testing along with reinforcement of standard infection control measures are essential to reduce the morbidity and mortality associated with such infections.

**References**


