Elizabethkingia meningosepticum: An Emerging Cause of Septicemia in Critically Ill Patients

Sir,

We report an uncommon case of nosocomial septicemia due to *Elizabethkingia meningoseptica* in a 60-year-old female with hepatic encephalopathy. The patient was admitted in the emergency ward with a history of hematemesis and melena. She was a known case of cryptogenic liver cirrhosis with portal hypertension. She was a known diabetic on regular follow-up since 2007. On admission, the patient was unconscious but responsive to painful stimuli. The pulse rate was 120/min and the blood pressure was 120/70 mmHg. The breath sounds were normal and the abdomen was soft. The patient was stabilized and endoscopic ligation of variceal bleed was performed. Further to the procedure, she was admitted in the Intensive Care Unit.

The laboratory investigations at the time of admission revealed a total leucocyte count of 11,860/dl, with a polymorph count of 71%, platelets 51 × 10^3/µl, bilirubin (total) 13.5 mg/dl, bilirubin (direct) 3.5 mg/dl, total protein 5.5 g/dl (albumin/globulin = 0.6), aspartate aminotransferase 436 U/L, alanine transaminase 541 U/L, gamma glutamyl transferase 41 U/L, alkaline phosphatase 151 U/L, urea 99 mg/dl, creatinine 0.80 mg/dl, Na 157 mmol/L and procalcitonin 0.36 ng/ml.

On the second day of admission, the patient developed hepatic encephalopathy. Her condition gradually deteriorated and she had several episodes of hematemesis. The blood cultures sent at the time of admission were negative till 5 days of aerobic incubation. Subsequent blood cultures (three separate samples in duplicate) grew Gram negative bacilli after 48–72 h of incubation. The patient was started on Teicoplanin, Meropenem and Fluconazole along with supportive measures. She died before the identification and sensitivity of the isolates were reported. The identification and sensitivity was performed in the Vitek 2 automated system (Biomerieux, France) using the cards GN1 and AST-N090, respectively. The isolates were identified as *Elizabethkingia meningoseptica* and were sensitive to Ciprofloxacin (MIC 1 µg/ml), Tigecycline (MIC 2 µg/ml) and Trimethoprim/Sulfamethoxazole (MIC 40 µg/ml), but resistant to Ampicillin/Sulbactam, Piperacillin/Tazobactum, Ceftriaxone, Cefipime, Cefaperazone/Sulbactum, Imipenem, Meropenem, Amikacin, Gentamicin, Tobramycin and Colistin.

Elizabethkingia meningoseptica, formerly known as *Flavobacterium meningosepticum*, was first reported by King in 1959 at CDC Atlanta.[1] It was reclassified in the genus Cryseobacterium and, later on, placed in the new genus *Elizabethkingia* named after the original discoverer.[1] It is a Gram negative, obligate aerobic, non-spore forming, non-fermentative and non-motile rod that is catalase, oxidase and urease positive. It is widely distributed in nature. In the literature, most of the reported cases of *Elizabethkingia meningosepticum* infection are hospital acquired and usually occur in immunodeficient patients. In our case, the infection was presumed to be acquired from the hospital environment as the cultures were negative at the time of admission.

It has been reported as a causative agent of meningitis in premature and newborn infants.[2] In adults, it has been isolated from cases of pneumonia, endocarditis and meningitis, usually in association with some underlying severe illness.[3] On a Pubmed literature search, no case of *Elizabethkingia meningosepticum* septicemia in patients with hepatic encephalopathy has been reported from India. There were few reports of isolation of this organism from cases of renal failure, endocarditis and meningitis.

The organism is inherently resistant to many antimicrobial agents commonly used to treat infections caused by Gram negative bacteria (aminoglycosides, beta-lactam antibiotics, tetracyclines and chloramphenicol), but are often susceptible to agents generally used to treat infections caused by Gram positive bacteria (rifampicin, clindamycin, erythromycin, trimethoprim-sulfamethoxazole, quinolones and vancomycin).These resistance phenotypes could be explained by the presence of beta-lactamases, including extended-spectrum beta-lactamases and metallo-beta-lactamases.

The choice of an effective drug for the empirical treatment of nosocomial *Elizabethkingia meningosepticum* infections is difficult. Studies conducted by some authors have shown that the combination of vancomycin and rifampicin appears to be the most appropriate therapy based on clinical outcomes. On the other hand, there are few reports that show that vancomycin has a poor activity against *Elizabethkingia meningosepticum*.[4,5] The rifampicin or fluoroquinolones alone are not commonly used in a country like India where tuberculosis is endemic. The clinical effectiveness of antibiotics could not be ascertained in our case as the patient died before the institution of appropriate antibiotics. Infection with this pathogen is potentially fatal unless diagnosed and treated early. The incidence of *Elizabethkingia meningosepticum* may be underreported as correct identification is difficult unless an automated system is used. All oxidase- and urease-positive non-lactose fermenters from immunocompromised or critically ill patients should be subjected to Quinolones/trimethoprim-sulfamethoxazole combinations until laboratory identification of the isolate is confirmed.[5] Thus, awareness about this organism in clinical samples along with correct identification and sensitivity testing is required to reduce the morbidity and mortality associated with such infections.

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