Case Report

Isolation of Multidrug Resistant Elizabethkingia Meningoseptica from an Immune-Compromised Patient

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ABSTRACT

Elizabethkingia meningoseptica is an emerging pathogen known to cause meningitis, pneumonia, endocarditis, bacteremia, wound & soft tissue infection, abdominal, respiratory and ocular infections, dialysis associated peritonitis and prosthesis associated septic arthritis; especially in immunodeficient hosts. We are reporting an uncommon case of nosocomial septicemia due to Elizabethkingia meningoseptica in a 56 year old male admitted with hypoglycemic encephalopathy. Patient had squamous cell carcinoma and was receiving chemotherapy. Using biochemical tests and VITEK 2 automated (Biomerieux) Identification and Antimicrobial Sensitivity Test (AST) system, the isolate was identified as Elizabethkingia meningoseptica and was found sensitive to Trimethoprim-Sulfamethoxazole only. His condition improved gradually after starting Trimethoprim-Sulfamethoxazole and repeat blood culture was sterile.

To our knowledge from literature review, this is the first reported case of multidrug resistant Elizabethkingia meningoseptica septicemia from North India.

Keywords: Elizabethkingia meningosepticum, septicemia, multidrug resistant.

INTRODUCTION

Elizabethkingia meningoseptica, formerly known as Flavobacterium meningosepticum, was first reported by King in 1959 at Centre for Disease Control (CDC) Atlanta. It was reclassified in the genus Cryseobacterium and, later on, placed in the new genus Elizabethkingia, named after the original discoverer.¹ It is widely distributed in nature. Most of the reported cases of Elizabethkingia meningoseptica infections are hospital acquired which usually occur in immunodeficient patients.

CASE REPORT

A 56 year old male was admitted with hypoglycemic encephalopathy. The patient was unconsciousness at the time of admission. Examination revealed respiratory distress, bilateral Crepitations in lungs and altered sensorium. He was treated for squamous cell carcinoma of lower limb since six months ago and had a history of recurrent seizures. He was a chronic smoker.

Laboratory investigation at the time of admission revealed Total Leucocyte Count (TLC) of 19,730/mm³. Biochemistry showed serum calcium of 6.67 mg/dl, serum phosphorus of 5 mg/dl and blood sugar of 21 mg/dl. Blood, Urine and Cerebrospinal fluid were sent for culture. Intra venous glucose infusion, Injection Ceftriaxone, Meropenem, Phenytoin and Mannitol were
started. Cerbro Spinal Fluid (CSF) microscopic examination was normal (WBC <5 cells/mm$^3$) Urine, blood & CSF cultures were negative. His condition gradually improved with normalization of blood sugars.

On 5$^{th}$ day of hospital stay, his condition deteriorated again. He developed high grade fever with worsening of respiratory distress and required ventilatory support. TLC raised with 90% polymorphs. Samples for Blood, tracheal swab and urine culture were taken again and antibiotics were changed to Teicoplanin and Aztreonam. The repeat blood culture was positive after 48 hrs of incubation. The colonies on blood agar were 1-2 mm smooth, circular, grayish-white non-hemolytic. There was no growth on MacConkey agar. Gram staining of the colony showed gram negative bacilli which were non motile, catalase positive, oxidase positive, urease and citrate negative and no change in Triple Sugar Iron TSI agar. The identification and sensitivity was performed on the Vitek 2 automated system (Biomerieux, France) using GN1 and AST-N090, cards respectively. The isolate was identified as Elizabethkingia meningoseptica sensitive to only Trimethoprim-Sulfamethoxazole (MIC 40 $\mu$g/ml), intermediate sensitive to Tigecycline (MIC 4 $\mu$g/ml) and resistant to all other antibiotics including Ampicillin-sulbactam, Piperacillin-Tazobactum, ceftriaxone, cefpime, ceferazone - sulbactum, Imipenem, Meropenem, Amikacin, Gentamicin, Tobramycin Ciprofloxacin and colistin.

The choice of an effective drug for the empirical treatment of nosocomial Elizabethkingia meningosepticum infections is difficult. Prolonged combination of rifampin with vancomycin, Trimethoprim-Sulfamethoxazole, minocycline or fluoroquinolones may have better clinical outcome. On the other hand, there are few reports that show that vancomycin has a poor activity against Elizabethkingia meningosepticum. The rifampicin or fluoroquinolones alone are not commonly used in a country like India where tuberculosis is endemic. The incidence of Elizabethkingia meningosepticum may be underreported as correct identification is difficult unless an automated system is used. All oxidase positive and urease-negative non-lactose fermenters from

**DISCUSSION**

Elizabethkingia meningosepticum has been reported as a causative agent of meningitis in premature and newborn infants. In adults, it has been isolated from patients with pneumonia, endocarditis and meningitis, usually in association with some underlying severe illness. Most of the reported cases of Elizabethkingia meningosepticum infection are hospital acquired and usually occurs in immune-deficient patients. In our case also the infection was presumed to be acquired from the hospital environment as the blood culture was negative at the time of admission. The patient was immunosuppressed and was on immnosuppressive therapy from six months.

Intrinsic multiresistance of Elizabethkingia to polymyxins and tigecycline is known owing to production of both ESBL and chromosomal MBL, but they are often susceptible to agents generally used to treat infections caused by Gram positive bacteria (rifampicin, clindamycin, erythromycin, trimethoprim-sulfamethoxazole, quinolones and vancomycin). Our isolate was sensitive only to Trimethoprim-Sulfamethoxazole, and had only intermediate sensitivity to Tigecycline (MIC 4 $\mu$g/ml). It was resistant to all other antibiotics including Ampicillin-sulbactam, Piperacillin-Tazobactum, ceftriaxone, cefpime, ceferazone - sulbactum, Imipenem, Meropenem, Amikacin, Gentamicin, Tobramycin Ciprofloxacin and colistin.
immunocompromised or critically ill patients should be subjected to Quinolones & Trimethoprim - sulfamethoxazole combinations until laboratory identification and sensitivity of the isolate is confirmed. [4]
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.
There is a possibility of encountering increased number of infections due to this opportunistic pathogen particularly in intensive care unit in compromised patients in future.

REFERENCES