



Elizabethkingia Meningoseptica: A Rare Pathogen Causing Community Acquired Septicemia in a Neonate.

KEYWORDS

Elizabethkingia meningoseptica , Flavobacterium meningosepticum, neonatal septicemia

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ABSTRACT We report a rare case of community acquired septicemia in neonate caused by *Elizabethkingia meningoseptica* (*E. meningoseptica*). A 26 days old premature male neonate weighing 1.9 kg presented with the history of high grade fever and refusal for feeds for 4 days. The patient was admitted and the blood sample and cerebrospinal fluid (CSF) was sent for culture. Patient was put up on ceftriaxone and amikacin. There was no response to the treatment. Sepsis screen was positive and blood culture flashed positive and the organism identified as *E. meningoseptica* based on various morphological features and biochemical tests. CSF was sterile. Antibiotic susceptibility testing was done. Combination therapy with piperacillin-tazobactam along with vancomycin was started and continued for 14 days. Patient responded and the subsequent blood culture was sterile.

Introduction:

E. meningoseptica has undergone several taxonomic modifications since first described by Elizabeth O king in 1959 as *Flavobacterium meningosepticum* (Fraser SL & Jorgensen JH,1997) (Kim , Kim , Lim , Park & Lee , 2005). It is a pigment (yellow no diffusible) producing, non motile, catalase, oxidase and indole positive, glucose non-fermenter gram- negative bacilli (Versalovic, Carroll, Funke, Jorgensen, Landry & Warnock , 2011).). It is known to cause meningitis, septicemia, pneumonia, endocarditis, and sinusitis but specifically it causes neonatal meningitis and septicemia, in premature low birth weight infants (Hoque, Graham , Kaufmann & Tabaqchali , 2001).

E. meningoseptica is usually resistant to most of the antibiotics specially used against Gram negative bacteria but are often susceptible to agents generally used to treat infections caused by Gram-positive bacteria (Hung, Lin, Lin, Lin & Shi ZY 2008). This often leads to inappropriate choice of antibiotics for initial empirical therapy and results in treatment failures (Hung et al , 2008).

Case Report:

A 26 days old preterm male neonate weighing 1.9 kg was admitted to the neonatal intensive care unit (NICU) of our institute with the history of high grade fever and refusal for feeds for 4 days. On examination the baby was lethargic, inactive and pale. The body temperature was 37.8 C, pulse rate 135 beats /min and he was maintaining a PO2 of > 90% at room air, which started dropping over 2 hr. At the same time he had tachypnea. Blood sample was sent for indirect early markers of neonatal infection. Total leukocyte count was decreased (2800/mm³) and there was absolute neutropenia (300/ mm³). C-reactive protein (CRP) was raised (15.0mg/dl) platelet count was 2, 30,000/mm³. Chest radiograph was normal. Cerebrospinal fluid analysis was done. Both blood and CSF were sent for microbial culture on day of admission. On second day, blood culture showed microbial growth and was subcultured. The colonies on nutrient agar were smooth, circular 1-2 mm in diameter with entire edges, regular margins and produced

yellow non-diffusible pigment. On blood agar the colonies were 1-2 mm smooth, circular, grayish-white non hemolytic having a characteristic smell. There was no growth on Mac Conkey agar. Gram stain of these colonies showed gram-negative, slender, slightly curved, non-sporing rods. This bacteria was a non-motile, catalase, oxidase and indole positive, non fermenting and did not hydrolyse urea. Citrate was not utilized and the organism hydrolysed esculin and gelatin. It did not reduce nitrate. Based on the growth pattern, biochemical reactions, antibiogram and identification by Vitek 2 system the isolate from blood culture was identified as *E. meningoseptica*. CSF cytology and biochemistry were found to be normal. There was no evidence of bacteria on smear examination and the CSF culture was sterile.

Antibiotic susceptibility was performed by Kirby Bauer disc diffusion method. Bacteria was resistant to ampicillin, ticarcillin, ceftriaxone, cefepime, tetracycline, chloramphenicol, imipenem, amikacin, gentamicin and colistin. The isolate was susceptible to ciprofloxacin, piperacillin-tazobactam, rifampicin, vancomycin, tigecycline, cotrimoxazole.

On admission ceftriaxone and amikacin was started empirically. There was no clinical response. After the report of antibiotic susceptibility both drugs were stopped and piperacillin-tazobactam along with vancomycin added and continued for 14 days. Over the three days baby showed significant clinical improvement. There was a decrease in the repeat CRP 4.2 mg/dL and the absolute neutrophil count also improved 1320/mm³. Subsequent repeat blood culture was done on day seven which was sterile. Baby was shifted out of NICU after seven days and discharged on day twenty one in good general condition.

Discussion:

E. meningoseptica is a rare bacteria causing septicemia in infants. It is ubiquitous in environment and found as a saprophyte in water and soil⁶. It also has been found on hospital environmental surfaces (Ceyhan, Yildirim, Tekeli, Yurdakok, Us & Altun ,2008). Prematurity is a primary risk

factor for *E. meningosepticum* infection in infants (du Moulin GC, 1979). Bloch et al (1997) reported meningitis as the predominant infection in neonates, followed by sepsis and pneumonia .

The point arising from this case worth highlighting is that this is a case of community acquired infection contrary to literature where most cases described are nosocomial (Ceyhan et al,2008). Other Indian studies have reported this rare pathogen causing neonatal and adult infection (Sarma, Kumar, Jha, Baveja & Sharma, 2011). To our knowledge this is a rare case of community acquired septicemia caused by this organism. Ubiquitous nature of this organism and prematurity with low birth weight as a risk factor explains this community acquired infection in our case.

Secondly, rarity of this organism makes the choice of antibiotic very difficult for the microbiologists as well as the clinicians. Even Clinical and Laboratory standards Institute (CLSI) breakpoints for this organism are still not established, making a therapeutic challenge (da Silva and Pereira , 2013). So once microbiologist suspects this infection, clinician should immediately be informed so that appropriate antibiotic changes may be instituted to the earliest.

Conclusion:

Microbiologist and clinicians both need to be aware of this rare organism as a potential pathogen in premature neonate who present with features of sepsis.

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