Neonatal Meningitis with Septicemia by *Elizabethkingia meningoseptica*: A Case Report

Neetha S. Murthy, Sowmya G. Shivappa, A. Tejashree, Krishna MVS Karthik, R. Deepashree

1 Department of Microbiology, JSS Medical College, Mysuru, Karnataka, India

**Address for correspondence** Sowmya G. Shivappa, MBBS, MD, Department of Microbiology, JSS Medical College, JSS AHER, Sri Shivarathreshwaranagar, Mysuru, 570015, Karnataka, India (e-mail: sowmyashivappa@gmail.com).

**Abstract**

*Elizabethkingia* is ubiquitary aerobic bacillus abundantly found in the community as well as hospital environments. *Elizabethkingia meningoseptica* is an emerging nosocomial pathogen with an elemental ability to acclimate and survive in diversified environmental circumstances. Prompt diagnosis and an early therapeutic intervention are preponderant in the management of these infections. We report a case of meningitis with sepsis caused by *E. meningoseptica* in a 1-day-old outborn neonate. The child was stabilized with anticonvulsants and, based on laboratory findings, the neonate was started on ciprofloxacin in addition to symptomatic management. The child responded well to the treatment and was discharged on day 7 after treatment initiation. Perceptive treatment protocols backed with accurate laboratory evidence remain instrumental to avert unpropitious outcomes while combatting rare multidrug-resistant opportunistic infections.

**Keywords**

► septicemia
► *Elizabethkingia meningoseptica*
► meningitis
► multidrug resistant
► neonate

**Introduction**

*Elizabethkingia meningoseptica* is an emerging cause of life-threatening nosocomial infections among neonates. This opportunistic pathogen was primarily placed under genus *Flavobacterium* (1959) and later moved to genus *Chryseobacterium* (1994). Eventually, in the year 2005 based on 16s rRNA phylogenetic studies, this aerobic bacillus was classified in the genus *Elizabethkingia* (an eponym from its discoverer Elizabeth O. King).[^2] *E. meningoseptica* is a gram-negative bacillus that is nonmotile, nonfermentative bacteria capable of splitting tryptophan to produce indole and is oxidase positive. Whole genome sequence analysis has established the intrinsic ability of this microbe to form biofilms and serve as a potential stockpile of novel-β-lactamase genes.[^3][^4]

Literature documents sparse case reports of *E. meningoseptica* from India and around the world. Few cases of wound infections, keratitis, nosocomial sepsis, nosocomial pneumonia, endocarditis, and meningitis in immunocompromised adults and preterm neonates have been reported globally.[^5][^6] Several culpable sources of this opportunistic pathogen such as contaminated water supply, equipment tubing, infant formulas, and saline solutions have been documented apart from the environmental niches.[^7] Yet another remonstrance with respect to this bacillus is its habitual multidrug resistance with no established Clinical and Laboratory Standards Institute drug breakpoints.[^8] Given the above circumstances, empirical therapy and treatment standardization with respect to *E. meningoseptica* is a farfetched dream. We report a case of neonatal meningitis with septicemia caused by *E. meningoseptica* in an outborn term neonate.

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[^2]: Elizabeth O. King
[^3]: 16s rRNA phylogenetic studies
[^4]: aerobic bacillus
[^5]: septicemia
[^6]: *Elizabethkingia meningoseptica*
[^7]: meningitis
[^8]: multidrug resistant
[^9]: neonate


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Case Report

We present a case of 1-day-old term outborn infant with *E. meningoseptica* attributed meningitis and septicemia presenting with convulsions. A single live, full-term, male baby delivered by normal vaginal delivery at a peripheral district hospital presented with birth asphyxia. The APGAR (appearance, pulse, grimace, activity, and respiration) score at birth unknown, the baby presented to our tertiary care center with two to three episodes of convulsions, sepsis screen positive, and respiratory distress. The neonate was stabilized with a loading dose of phenobarbitone 20 mg/kg followed by a maintenance dose of 10 mg/kg. Injection calcium was given 2 mL/kg and the child was shifted to the neonatal intensive care for further management.

At admission, the child was moderately active with a heart rate of 146/minute and respiratory rate of 93/minute. Weight of the neonate was 2.88 kg. The ultrasound brain yielded a normal study with no intracranial bleed, brain parenchymal lesions, or ventricular enlargement. The cerebral hemispheres, basal ganglia, corpus callosum, and posterior fossa structures appeared normal. Magnetic resonance imaging brain showed mild hypoxic ischemic encephalopathy.

The blood investigations showed a raised total cell count (128,900) and reduced platelet count (67,000). C-reactive protein was raised with a value of 27.91. Creatinine was found to be 1.32 mg/dL. Liver function parameters were within normal limits. Arterial blood gas analysis at admission showed metabolic acidosis with compensatory respiratory alkalosis. Cerebrospinal fluid (CSF) analysis at admission showed predominant neutrophils with normal protein and sugar levels.

Blood sample (4 mL) from the neonate was inoculated into a culture bottle and incubated in BacT/ALERT Microbial Colorimetric Detection System. Sample flagged positive in 23 hours. Flagged sample was subjected to direct gram staining and subsequently plated on MacConkey along with 5% sheep blood agar. Gram smear showed gram-negative bacilli and culture yielded aerobic, pale pink (Fig. 1) oxidase positive colonies on MacConkey agar and nonhemolytic colonies on blood agar. Culture isolate smear showed gram-negative bacillus in concordance with the direct smear findings (Fig. 2). VITEK-2 (bio-Mérieux) identification yielded *E. meningoseptica* with 99% probable confidence of identification. CSF sample from the child was subjected to automated culture (BacT/ALERT System). CSF culture also yielded *E. meningoseptica* by the VITEK 2 system with a 99% identification confidence. Both the blood and CSF isolate showed resistance to β-lactams, carbapenems, and aminoglycosides. Second- and third-generation quinolones were effective with an in vitro minimal inhibitory concentration of 0.5 μg/mL. The child was started on parenteral ciprofloxacin along with symptomatic management. In vivo response to therapy was clinically admirable and the child was discharged on day 7 post treatment initiation with absolutely no residual morbidity.

Discussion

Genus *Elizabethkingia* houses a notorious bunch of rare opportunistic pathogens responsible for multidrug resistant lethal infections. The mortality rate associated with this rare pathogen is around 23%. Essentially the genus is composed of saprophytic bacteria capable of survival in chlorinated water, hospital equipment, and pediatric nurseries. Interestingly although deemed to be an opportunistic pathogen commonly infecting the immunocompromised subjects, *Elizabethkingia* species are not part of the normal human microbial flora. The bacteria belonging to this genus are known to possess virulence attributes such as proteases, catalases, acetyltransferases, peroxidases, heat shock proteins, capsular polysaccharide, and lipooligosaccharides. The adherence ability of *E. meningoseptica* NCTC 10016 onto abiotic surfaces such as intravascular devices via formation of biofilms has been well established. Multiple possible pathways of exposure and mechanisms of
pathogenesis have been contemplated. However, exact mechanism of pathogenesis remains obscure.11

Considering the fact that bacterial species belonging to the genus Elizabethkingia are usually multidrug resistant, laboratory-aided identification is of prime importance. On the laboratory front, strain-dependent variabilities2 in culture growth make manual identification unreliable. Commercial automated microbial identification systems provide accurate and quick genus level identification of Elizabethkingia species. However, Elizabethkingia reference databases on commercial microbial identification systems find partial concordance with 16S rRNA gene sequencing-based specific species identification.13,14 Advances in automation and microbiological diagnostics bring with it the advantage of early diagnosis and better detection rates of Elizabethkingia species, thereby averting inappropriate antimicrobial therapy and outbreak prevention.

Members of the genus Elizabethkingia are by and large resistant to antimicrobials such as tetracycline, chloramphenicol, extended-spectrum β-lactams, and aminoglycosides. Biologically plausible chromosome and plasmid mediated β-lactam resistance due to Ambler class D extended-spectrum serine-β-lactamase coding blabME genes and carbapenem resistance due to blabR (subclass B1) and blacGob (subclass B3) genes have been documented.15 Fluoroquinolones exhibit uniform volume distribution and better penetration of the blood brain barrier by virtue of their lipophilic nature.15,16 The child in our case report also demonstrated clinical improvement with 7 days of parenteral ciprofloxacin therapy and was discharged on day 8 of admission with no documented untoward incidents.

**Conclusion**

*E. meningoseptica* is a difficult to diagnose saprophytic non-fermenter with a handful of cases being reported from pediatric nurseries and critical care units in the recent times. This agrandize with respect to notification of *E. meningoseptica* infections is attributable to the availability of rapid, accurate, commercial identification systems for laboratory diagnosis. In conclusion, given the independent attributable mortality/morbidity and multidrug resistance profile of Elizabethkingia, development of robust standard infection control practices to combat this emerging pathogen is inevitable. Armed with automated identification systems, the microbiology laboratory can combat the formidable rapid identification challenge paving way toward early appropriate treatment initiation, resulting in favorable patient outcome as documented in our case report.

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**Conflict of Interest**

None declared.

**References**