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Case Report

Hydrocephalus Due to Neonatal Septicemia, Meningitis and Ventriculitis Caused by Multi-Resistant Elizabethkingia Anophelis: A Not So Uncommon Complication

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Abstract

Elizabethkingia anophelis is a rare pathogen causing an increasing incidence of neonatal meningitis including neurological sequelae–and mortality. The path towards diagnosis and adequate treatment may be challenging due to its rarity and multi-resistant antimicrobial susceptibility patterns. We describe the therapeutic challenges of a case of neonatal septicemia, meningitis and ventriculitis with accompanying hydrocephalus. In addition, we show that hydrocephalus in *E. anophelis* meningitis/ventriculitis may be a common complication requiring interventions such as ventriculoperitoneal drainage. Therefore, repeated brain imaging during disease course is advised. Awareness of the multi-resistant features of this pathogen may lead to earlier adequate treatment, while awaiting antimicrobial susceptibility patterns.

Keywords: *Neonates, Elizabethkingia, Meningitis, (Post-Infectious) Hydrocephalus*

Introduction

Infections with *Elizabethkingia* spp. are rare yet feared for their multi-resistant antimicrobial susceptibility patterns and potency for nosocomial outbreaks [1]. In children, *Elizabethkingia* spp. infections may cause a variety of diseases, including bacteremia/septicemia and pneumonia, but mostly neonatal meningitis, with a mortality rate of approximately 30% [1]. In this report, we describe the therapeutical challenges encountered in a premature neonate with septicemia, meningitis and ventriculitis with progressive hydrocephalus due to multi-resistant *E. anophelis*.

In 1959, Elizabeth King described *Flavobacterium meningosepticum* as a previously unclassified bacterium associated with meningitis in children [2]. The gram-negative aerobic, non-motile rods were reclassified as *Chryseobacterium meningosepticum* in 1994, and in 2005 to *Elizabethkingia meningoseptica* [3]. However, recently it was identified that many infections attributed to specific *E. meningoseptica,* should have been identified as *E. anophelis*, and that the latter may be the main human pathogen of the *Elizabethkingia* genus [4,5]. The first description of an infection with *E. anophelis* dates from 2011, where a case of neonatal meningitis was described in the Central African Republic [6]. Up to date, only a few hundred cases of pediatric *Elizabethkingia* spp. infections have been reported, the majority being in neonates [1].

Case Presentation

A female neonate was born at 27 1/7 weeks of gestation via caesarean section because of imminent labour and breech position. Her birthweight was 875 grams (p42) and Apgar scores were 9 and 10 after 1 and 5 minutes after birth. Empiric antibiotics were stopped after 48 hours when blood culture remained negative. Initial respiratory support was sufficient with CPAP, FiO2 0.21, and was switched to flow after three days. At day 13, she became less active and showed episodes with desaturation and bradycardia. She had no intravenous access at that time.

Respiratory support was intensified and sepsis workup showed CRP of 44 mg/dL. Urine and blood culture were taken prior to start of empirical antibiotics for late-onset sepsis. Fortyeight hours after start of antibiotics, there was no clinical improvement, and a gram-negative rod was found in the blood culture. Antibiotics were switched to meropenem and lumbar puncture showed a gram-negative rod. CRP elevated to 152 mg/dL but started to decrease after 3 days of meropenem, meanwhile clinical irritability continued. The gram-negative rod was determined by MALDI-TOF (Bruker) as *Elizabethkingia anophelis.* A combination of vancomycin, ciprofloxacin and piperacillin/tazobactam was initiated based on previous case reports.

Minimal inhibitory concentration (MIC) in μg/ml as measured with MIC test strips (MTS ™, Liofilchem®) was for amoxicillin/clavulanic acid >256, for piperacillin/tazobactam 64, for ceftazidime >256, for ceftriaxone >32, for meropenem >32, and for gentamicin 64 μg/ml. There are no specific breakpoints for antibiotic susceptibility for *Elizabethkingia* spp. The isolate was carbapenemase positive and sequencing showed *blaGOB-4, 4lab-11, blaCME-1* as metallo-beta-lactamases in *Elizabethkingia* spp. [7]. Possible antibiotic activity was measured for vancomycin, MIC 8 μg/ml, for rifampicin 0.38 μg/ml, for ciprofloxacin 0.5 μg/ml and for ceftazidime/avibactam 6 μg/ml.

Therefore, piperacillin/tazobactam was switched to rifampicin. Cranial ultrasound performed at day 2, 4 and 11 after sepsis work-up, initially showed no abnormalities. From day 11, signs of ventriculitis and progressive hydrocephalus (maximum Levene index 43 mm on the left, 39 mm on the right) were identified, requiring multiple lumbar and ventricular punctures for alleviation of high intracranial pressure (Figure 1a and 1b). Three weeks after onset of illness, fever recurred, and CRP was elevated again. Cerebrospinal fluid (CSF) showed pleiocytosis and vancomycin was switched to ceftazidime/avibactam, as penetration of vancomycin into CSF may be limited. Blood and CSF cultures remained negative. Clinically our patient improved, but hydrocephalus with symptoms of high intracranial pressure remained, requiring multiple ventricular punctures a week. A ventriculoperitoneal drain (VPD) was placed followed by neurological improvement, although Levene indices changed minimally (Figure 1c and 1d).

Antimicrobial treatment was continued with rifampicin, ceftazidime/avibactam and ciprofloxacin for eight weeks in total until two days after placement of VPD. At 3.5 months of age (corrected age 2 weeks) she was discharged home. At 17 months old (corrected age 14 months), our patient showed axial hypotonia, peripheral hypertonia and neurodevelopmental delay. Brainstem Evoked Response Audiometry shows no reproducible curves on both sides.

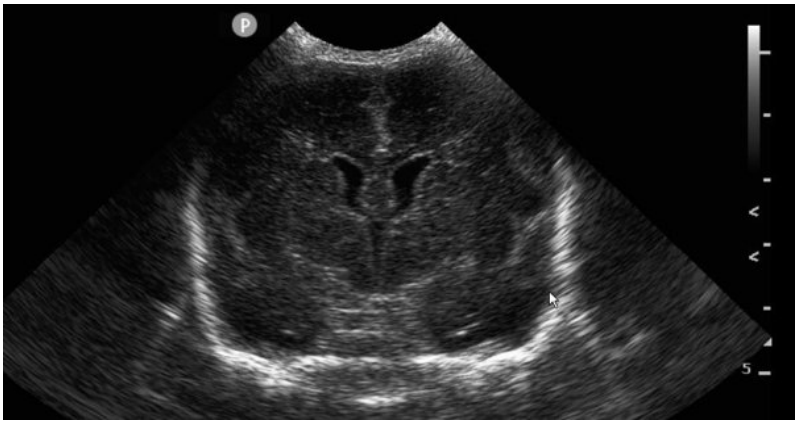

Figure 1A. Normal brain ultrasound made 4 days after the sepsis work-up.

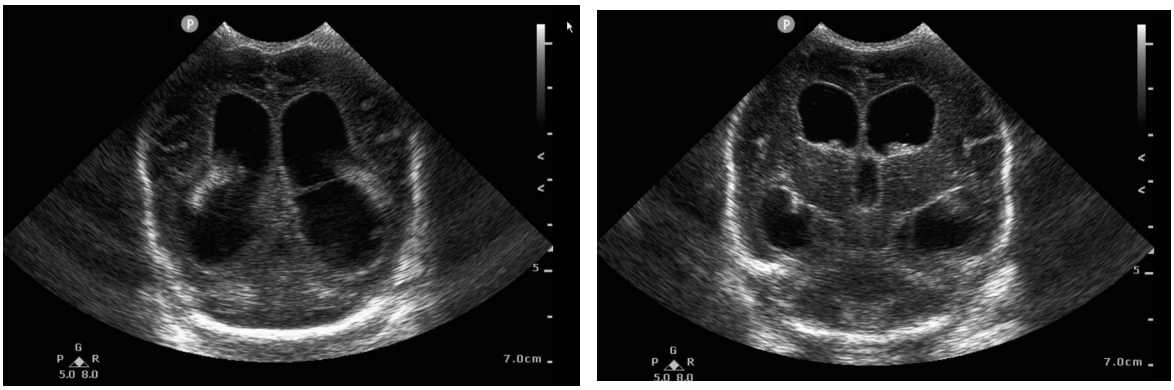

Figure 1B and 1C. Brain ultrasound made 11 days after the sepsis work-up, showing intraventricular septation, suggestive of ventriculitis. There is also dilatation of the lateral ventricle and third ventricle
(Davies on the left side 13.7 mm, on the right side 13.9 mm (both compatible with severe ventriculomegaly), Levene index on the left side 16.2 mm (>+2 SD+4 mm), on the right side 14.4 mm (> +2 SD).

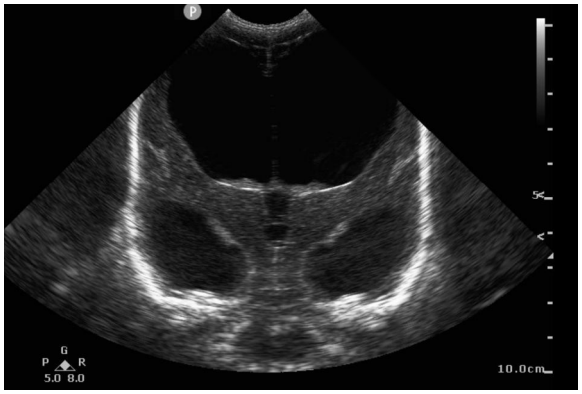

Figure 1D. Brain ultrasound at 3 months of age showing further progression of severe hydroocephalus.

Discussion

We present a case of neonatal septicemia, meningitis and ventriculitis with severe and progressive hydrocephalus due to infection with multi-resistant *E. anophelis.* In children, *Elizabethkingia* spp. infections may cause a variety of disease, including bacteremia/septicemia and pneumonia, but mostly neonatal meningitis, with mortality rates of approximately 20-30% [1,8]. The first report of an infection with specific *E. anophelis* dates from 2011, causing neonatal meningitis [6]. However, recent publications show that several infections that were previously attributed to *E. meningoseptica* may have been misidentified and should be attributed to *E. anophelis* [5].

While it has been suggested that hydrocephalus in *E. anophelis* may be a rare complication [9], it may be more common than previously acknowledged. According to literature, hydrocephalus was found in 24% due to *E. anophelis* infections [8], 30% in unspecified *Elizabethkingia* spp. infections [1], and 60% in *E. meningoseptica* infections, respectively [10]. Additionally, a literature review by Tan et al showed the occurrence of hydrocephalus in surviving patients with *Elizabethkingia* spp. infections ranging between 20-50% reported in studies on NICU/pediatric ward outbreaks [11]. The high incidence found in case reports may be due to publication bias. However, compared to the risk of hydrocephalus after a general bacterial meningitis, an estimated 7% [11], hydrocephalus after a *Elizabethkingia* spp. meningitis may occur more frequently. Due to the need for intervention, physicians should recognize the possibility for potential (evolving) hydrocephalus in *E. anophelis* meningitis and consider performing routine cerebral imaging during the disease course.

Our patient showed neurodevelopmental delay at 17 months old. Neurodevelopmental outcome after *E. anophelis* meningitis is rarely reported in literature. Of four reported patients, all had hydrocephalus requiring a VPD and neurodevelopment was noted normal in two (50%) [9, 13, 14]. Developmental delay may also be (partially) attributed to prematurity, as in our patient. Long-term neurodevelopmental outcomes of *E. anophelis* meningitis have not been published.

E. anophelis appears to be commonly resistant to multiple drug classes including aminoglycosides, third generation cephalosporines and carbapenems [15], posing a serious challenge in timely and adequate treatment for favourable outcomes. Interestingly, (partial) susceptibility to vancomycin has been reported in Elizabethkingia spp., a feature not commonly attributed to gram-negatives [15]. In our patient, indeed (limited) in vitro susceptibility was shown to vancomycin.

This *E. anophelis* infection occurred isolated on our Neonatal Intensive Care Unit (NICU). Literature reports potency for nosocomial outbreaks of *Elizabethkingia* spp., however, community-acquired *E. anophelis* infections have also been reported [15, 16, 17]. Our patient was admitted to the NICU since birth and had no respiratory support, nor intravenous access at time of onset, only a nasogastric tube. We have not been able to identify the source of this isolated infection.

Conclusion

Elizabethkingia spp. infections have a high morbidity and mortality rate and should worry all medical staff upon encounter. Hydrocephalus in *E. anophelis* meningitis and ventriculitis may be a common complication, ultimately requiring intervention such as ventriculoperitoneal drainage. Repeated brain imaging is therefore advised during the disease course. Awareness of the multi-resistant features of this pathogen may lead to faster adequate treatment, while awaiting antimicrobial susceptibility patterns.

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Statement of Ethics

This research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki.

Consent to Publish Statement

Written informed consent was obtained from the parents of the patient for publication of the details of their medical case and accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

LT, AR, GE performed clinical care for the patient and contributed to writing the case report. BdJ performed microbial and antimicrobial susceptibility analyses of the specimens and contributed to the writing of the case report.

Data Availability Statement

Data can be requested via the corresponding author.

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