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Neonatal Meningitis Due to *Elizabethkingia anophelis* Complicated by Ventriculitis and Tetraventricular Hydrocephalus: A Case Report

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Abstract

Neonatal meningitis is a severe infection associated with high mortality and a substantial risk of neurological sequelae. *Elizabethkingia anophelis* is an emerging opportunistic pathogen responsible for severe invasive infections, particularly in neonates and other vulnerable patients. Its management represents a major challenge because of its intrinsic multidrug resistance, the absence of standardized treatment recommendations, and the high risk of neurological complications. We report the first documented case at the Neonatal Intensive Care Unit of Mohammed VI University Hospital in Agadir of *E. anophelis* neonatal meningitis complicated by ventriculitis and tetraventricular hydrocephalus in a term neonate aged 15 days. The diagnosis was confirmed by isolation of *E. anophelis* from cerebrospinal fluid (CSF). Despite initial empirical antibiotic therapy, the clinical course was complicated by neurological deterioration with the development of hydrocephalus requiring external ventricular drainage. Following antimicrobial susceptibility testing, therapy was adapted to a combination of ciprofloxacin and vancomycin. The clinical course is currently favorable under treatment, although persistent ventriculitis requires ongoing specialized management. This case highlights the virulence of *E. anophelis*, the diagnostic and therapeutic challenges associated with its multidrug-resistant profile, and the importance of early microbiological identification, prompt adjustment of antimicrobial therapy, and multidisciplinary management to improve neurological outcomes. Written informed consent for publication of the clinical details and radiological images was obtained from the patient's parent(s).

Subject Areas

Pediatrics

Keywords

Elizabethkingia anophelis, Neonatal Meningitis, Ventriculitis, Hydrocephalus, Neonate, Neonatal Infection

1. Introduction

Neonatal meningitis is a severe infectious disease associated with substantial mortality and long-term neurological morbidity. Late-onset neonatal infections are particularly challenging because clinical manifestations may be nonspecific, while the rapid progression of invasive bacterial infections requires prompt empirical antimicrobial therapy [1].

Elizabethkingia species are rare Gram-negative, non-fermenting bacilli that have emerged as important opportunistic pathogens, particularly among neonates, premature infants, immunocompromised patients, and critically ill individuals [2] [3]. Among the clinically relevant species, *Elizabethkingia anophelis* has gained increasing recognition as a cause of severe bloodstream and central nervous system infections [4].

E. anophelis was first described as a distinct species in 2011. Subsequent advances in bacterial identification, including 16S rRNA gene sequencing, MALDI-TOF mass spectrometry, and whole-genome sequencing, demonstrated that a number of isolates previously identified as *E. meningoseptica* were actually *E. anophelis* [5]. This taxonomic clarification has substantially improved our understanding of the epidemiology, antimicrobial susceptibility, and clinical significance of the species.

Therapeutic management remains particularly challenging because of intrinsic resistance to several antimicrobial classes. *Elizabethkingia* species possess multiple β-lactamases and may display variable susceptibility to fluoroquinolones, trimethoprim-sulfamethoxazole, minocycline, rifampicin, and other agents, making antimicrobial susceptibility testing essential for treatment selection [6] [7]. In addition, environmental persistence and biofilm formation may facilitate healthcare-associated transmission and contribute to outbreaks [7].

Neurological complications, including ventriculitis, hydrocephalus, brain abscesses, and other intracranial complications, have been described in neonatal *E. anophelis* infections and may significantly worsen the neurological prognosis [8]. However, because of the rarity of these infections, no standardized international treatment recommendations currently exist.

We report a case of neonatal meningitis caused by *E. anophelis* in a term neonate, complicated by ventriculitis and tetraventricular hydrocephalus, illustrating the diagnostic, therapeutic, and neurosurgical challenges associated with this

emerging pathogen.

2. Case Report

We report the case of a male neonate admitted to the Neonatal Intensive Care Unit (NICU) of Mohammed VI University Hospital in Agadir at 15 days of life because of a fever of 39˚C associated with irritability, without feeding difficulties, seizures, vomiting, diarrhea, or respiratory distress.

He was born after an uneventful, well-monitored pregnancy to a 28-year-old mother (G3P2, with one spontaneous miscarriage), with no history of pregnancy-related complications. The maternal infectious history was unremarkable, with no premature rupture of membranes, peripartum fever, or documented urinary or genital infection. Delivery was performed by elective cesarean section at 38 weeks of gestation because of a previous cesarean scar. Adaptation to extrauterine life was satisfactory, with Apgar scores of 8 and 10 at 1 and 5 minutes, respectively. Birth weight was 3200 g, length was 51 cm, and head circumference was 35 cm. The neonatal examination was unremarkable, and the infant was discharged from the maternity ward at 72 hours of life.

At 15 days of life, the infant developed a fever of 39˚C associated with persistent irritability, prompting evaluation in the pediatric emergency department followed by admission to the NICU.

On admission, the neonate weighed 3300 g. He was conscious and responsive but irritable. Primitive reflexes, including the sucking reflex, were present, with preserved axial and peripheral muscle tone. Neurological examination revealed a bulging anterior fontanelle. Head circumference was 36 cm.

Vital signs showed a temperature of 39.2˚C, heart rate of 210 beats/min, respiratory rate of 63 breaths/min, peripheral oxygen saturation of 100% on room air, and mean arterial pressure of 52 mmHg. Cardiovascular examination revealed no mottling, purpura, or cold extremities. The respiratory examination was normal, with no signs of respiratory distress or abnormal auscultatory findings. The abdomen was soft and non-distended, without hepatosplenomegaly, and bowel function was preserved. The remainder of the clinical examination was unremarkable.

Given the suspicion of neonatal bacterial infection, empirical antibiotic therapy with meningitis-dose cefotaxime and gentamicin was initiated after microbiological samples had been obtained.

Laboratory investigations showed a hemoglobin level of 11.3 g/dL, white blood cell count of 16,890/mm^3 (neutrophils: 9900/mm^3; lymphocytes: 4780/mm^3), thrombocytosis with a platelet count of 814,000/mm^3, and a C-reactive protein level of 157 mg/L. Blood cultures remained sterile.

Cerebrospinal fluid (CSF) analysis revealed a xanthochromic fluid containing 306 leukocytes/μL (55% neutrophils), 45 erythrocytes/μL, marked hyperproteinorrachia with a protein level of 2.60 g/L, and severe hypoglycorrhachia with a glucose level of 0.05 g/L, compared with a concomitant blood glucose level of 0.75 g/L. Direct examination after Gram staining revealed Gram-negative bacilli. CSF

culture yielded *Elizabethkingia anophelis*, which was susceptible to ciprofloxacin and vancomycin according to antimicrobial susceptibility testing.

An early transfontanellar cranial ultrasound revealed tetraventricular hydrocephalus.

Based on the microbiological findings, antibiotic therapy was switched to a combination of ciprofloxacin (10 mg/kg every 12 hours) and vancomycin (10 mg/kg every 6 hours): intravenous administration for both drugs.

Despite targeted antibiotic therapy, the clinical course was complicated three days after admission by a rapid increase in head circumference from 36 to 38 cm. Brain computed tomography (CT) demonstrated non-obstructive tetraventricular hydrocephalus (Evans index: 0.37), (**Figure 1**) with no identifiable obstructive lesion and no clear evidence of transependymal CSF flow.

Given this deterioration, an external ventricular drain (EVD) was inserted.

At 25 days of life, while the patient was still receiving targeted antibiotic therapy, the clinical course was complicated by status epilepticus, requiring a loading dose of phenobarbital.

Brain magnetic resonance imaging (MRI) demonstrated ventriculitis with intraventricular purulent material (**Figures 2-4**) associated with communicating tetraventricular hydrocephalus and a patent cerebral aqueduct, without evidence of transependymal CSF flow. Leptomeningeal enhancement of the basal cisterns was also observed, consistent with active meningitis.

Repeat CSF analysis revealed persistent xanthochromia, with 630 leukocytes/μL (80% neutrophils) and 18 erythrocytes/μL. Gram staining again demonstrated Gram-negative bacilli. CSF culture remained positive for *Elizabethkingia anophelis*. The bacterial isolate recovered from cerebrospinal fluid was identified as Elizabethkingia anophelis using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) indicating persistent microbiological infection.

At the time of manuscript preparation, the neonate was 28 days old (**Table 1**). He remained hospitalized in the NICU, breathing spontaneously, and had been receiving targeted therapy with ciprofloxacin and vancomycin for 13 days. The external ventricular drain remained functional. The clinical course was considered stable, but active microbiological infection persists with close neurological, microbiological, and neuroradiological monitoring.

Table 1. Clinical timeline.

Time	Event/treatment/findings
Day 15 of life (admission)	Fever, irritability, bulging fontanelle; blood cultures and lumbar puncture; CSF Gram-negative bacilli, inflammatory CSF profile; cefotaxime plus gentamicin started after sampling.

Continued

Day 16 of life	Cranial ultrasound: tetraventricular hydrocephalus; CSF culture identification of E. anopheli; switch to ciprofloxacin plus vancomycin.
Day 18 of life (admission + 3 days)	Head circumference increased from 36 to 38 cm; CT: communicating tetraventricular hydrocephalus (Evans index 0.37); EVD insertion
Day 20 of life	Brain MRI: ventriculitis with purulent intraventricular material, communicating hydrocephalus, basal leptomeningeal enhancement.
Day 25 of life	Status epilepticus; phenobarbital loading dose.
Day 25 of life	Repeat CSF: 630 leukocytes/µL, 80% neutrophils, Gram-negative bacilli and persistent E. anophelis growth.
Day 28 of life	Day 13 of targeted therapy; spontaneous breathing, functioning EVD; clinically stable, but active microbiological infection persists.

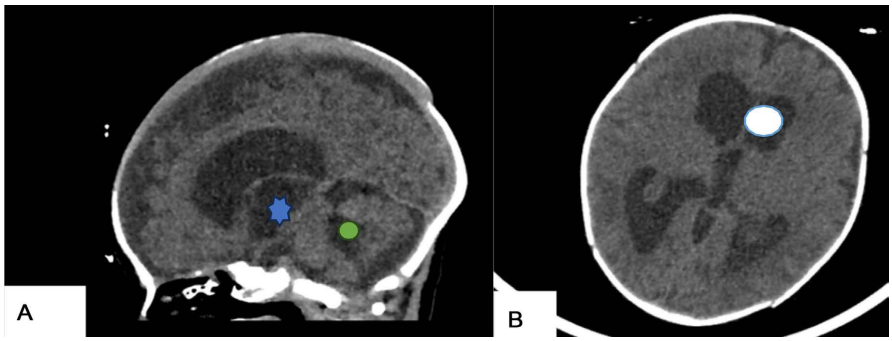


Figure 1. Non-contrast brain CT scan showing communicating, non-obstructive tetraventricular hydrocephalus without signs of active transependymal CSF resorption: (A) sagittal view; (B) axial view. (): fourth ventricle; (): third ventricle; (): lateral ventricle.

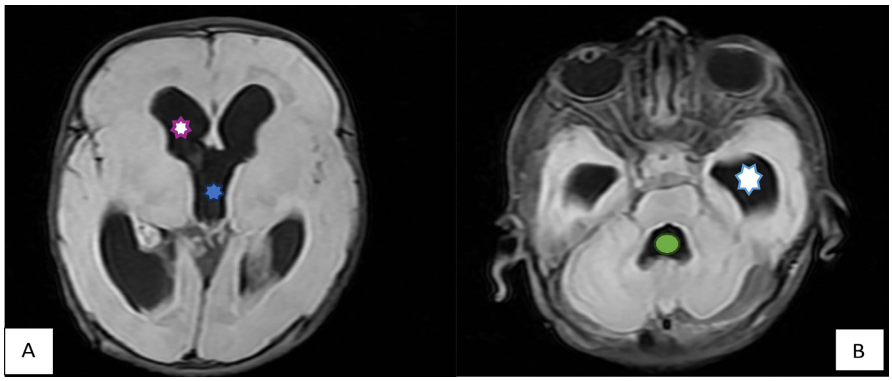


Figure 2. Brain MRI, axial FLAIR sequences, showing non-obstructive tetraventricular hydrocephalus without signs of active transependymal CSF resorption: (A) Dilatation of the lateral ventricles () and the third ventricle (); (B) Dilatation of the fourth ventricle () and the temporal horns of the lateral ventricles ().

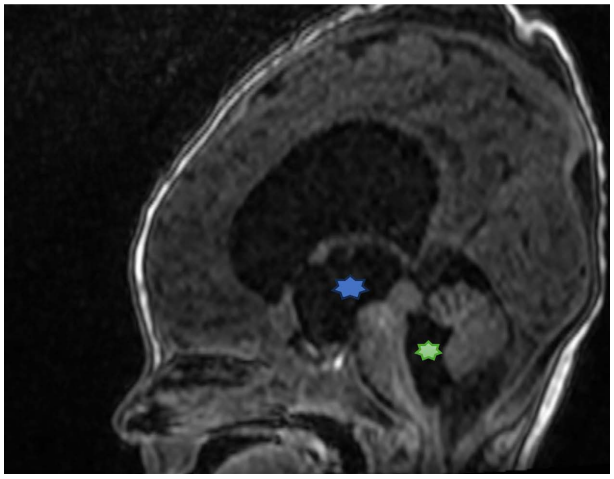


Figure 3. Brain MRI, sagittal BRAVO sequence, showing non-obstructive tetraventricular hydrocephalus without signs of active transependymal CSF resorption. (): fourth ventricle; (): third ventricle.

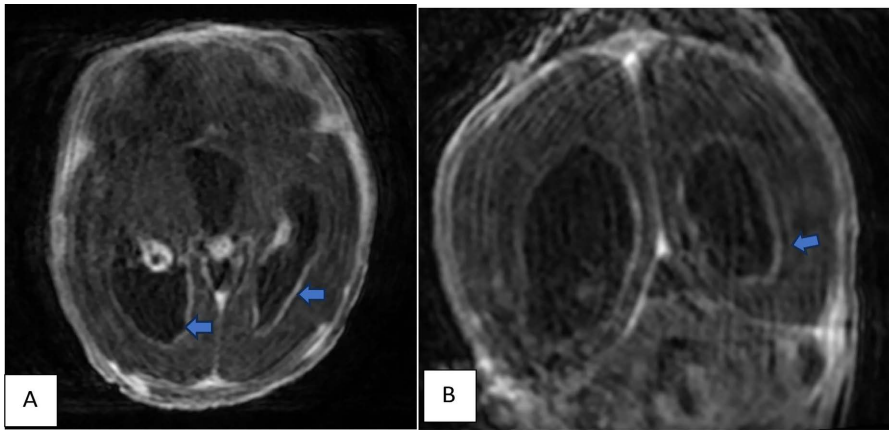


Figure 4. Brain MRI, post-gadolinium contrast-enhanced T1-weighted sequences in axial (A) and coronal (B) planes, showing tetraventricular dilatation associated with ependymal enhancement after gadolinium administration, suggestive of ventriculitis. Bilateral enhancement of the walls of the occipital horns of the lateral ventricles is also observed ().

3. Discussion

Elizabethkingia anophelis is now recognized as an emerging opportunistic pathogen responsible for severe invasive infections, particularly in neonates, premature infants, and immunocompromised patients [2]-[4]. The increasing recognition of *E. anophelis* has been facilitated by improvements in microbiological identification, particularly 16S rRNA gene sequencing, MALDI-TOF mass spectrometry, and whole-genome sequencing. These techniques have demonstrated that a substantial proportion of isolates previously identified as *Elizabethkingia meningoseptica* were actually *E. anophelis* [5]-[7]. In a study based on 16S rRNA sequencing, *E. anophelis* accounted for 59.3% of the clinical *Elizabethkingia* isolates identified, illustrating its importance as a human pathogen [7].

Our case has several noteworthy features. To our knowledge, this is the first

case of neonatal meningitis caused by *E. anophelis* reported at our institution. Unlike many previously reported cases, our patient was a term neonate with no identified maternal infectious risk factors, born by elective cesarean section, who developed symptoms on day 15 of life. This chronology is more suggestive of a late-onset infection acquired after birth rather than a documented vertical transmission. However, vertical transmission has also been demonstrated in neonatal *E. anophelis* infections, emphasizing the complexity of its epidemiology [2] [3].

The natural reservoir and transmission pathways of *E. anophelis* remain incompletely understood. The species was initially isolated from the midgut of the mosquito *Anopheles gambiae*, but subsequent evidence has highlighted the importance of environmental and healthcare-associated reservoirs. *Elizabethkingia* species can persist in water and healthcare environments and may colonize sinks, water systems, humidifiers, ventilators, and other medical devices. Their ability to form biofilms may contribute to environmental persistence and facilitate transmission [9].

In 2024, Brandsema *et al.* reported a neonatal case of *E. anophelis* meningitis in the Netherlands in which the clinical isolate was clonally related to isolates recovered from the water reservoir of an automated infant milk dispenser located at the patient's home [10]. This observation demonstrates that contaminated water reservoirs may represent a potential source of community-acquired neonatal infection. Although no environmental investigation was performed in our unit, the absence of maternal risk factors and the late onset of symptoms make an environmental acquisition plausible, although a healthcare-associated source cannot be confirmed.

As in many reported cases, the initial clinical manifestations in our patient were nonspecific, consisting of fever and irritability. This presentation illustrates the diagnostic challenge of neonatal meningitis, in which early clinical manifestations may be subtle. In our patient, however, the presence of a bulging anterior fontanelle on admission rapidly raised suspicion of central nervous system involvement and prompted early lumbar puncture. CSF analysis showed the typical features of severe bacterial meningitis, including marked pleocytosis with neutrophil predominance, profound hypoglycorrhachia, marked hyperproteinorrachia, and a positive culture for *E. anophelis*.

The neurological course observed in our patient further illustrates the severe neurological complications that may accompany *Elizabethkingia* meningitis. Despite targeted antimicrobial therapy, the patient rapidly developed tetraventricular hydrocephalus requiring external ventricular drainage, followed by MRI-confirmed ventriculitis with intraventricular purulent material. Hydrocephalus and ventriculitis have been described in neonatal *E. anophelis* meningitis, including a particularly severe case reported by Honavar *et al.*, in which the infection was complicated by ventriculitis and hydrocephalus requiring neurosurgical management [11]. Other neurological complications, including brain abscesses, have also been reported in neonatal infections [12].

The persistence of a positive CSF culture despite several days of targeted antimicrobial therapy represents another important feature of our case. Persistent microbiological positivity may reflect a high bacterial burden, involvement of the ventricular system, biofilm formation, or inadequate antimicrobial penetration into infected compartments [12]. In the recently published case by Wu *et al.*, neonatal *E. anophelis* meningitis was complicated by a brain abscess and required individualized antimicrobial management based on susceptibility testing [12]. These observations support the importance of repeated CSF sampling in patients with ventriculitis or persistent clinical abnormalities to document microbiological clearance and guide treatment duration.

The therapeutic management of *E. anophelis* infections remains particularly challenging because of the organism's intrinsic multidrug-resistant phenotype. *Elizabethkingia* species possess chromosomally encoded β-lactamases, including the metallo-β-lactamases BlaB and GOB and the class A β-lactamase CME, which contribute to resistance to most β-lactam antibiotics, including carbapenems [13]. The genomic characteristics of *E. anophelis* also include multiple determinants associated with antimicrobial resistance and virulence. This intrinsic resistance profile likely explains the lack of efficacy of the initial empirical regimen of cefotaxime and gentamicin in our patient.

In contrast, susceptibility to fluoroquinolones, trimethoprim-sulfamethoxazole, minocycline, rifampicin, and vancomycin is variable between isolates, emphasizing the importance of antimicrobial susceptibility testing to guide definitive therapy [14]. In our case, susceptibility testing demonstrated susceptibility to ciprofloxacin and vancomycin, allowing rapid adaptation of antimicrobial therapy. Although vancomycin has been used in several reported cases of *Elizabethkingia* infection, its clinical role remains uncertain because susceptibility results may be variable and robust clinical evidence supporting its use is lacking [14].

The antimicrobial resistance profile of *Elizabethkingia* is particularly relevant when selecting empirical therapy. The organism is generally resistant to many β-lactams and aminoglycosides, although susceptibility patterns may vary substantially between species and individual isolates [15]. These characteristics reinforce the need for early species-level identification and susceptibility testing rather than relying solely on conventional empirical regimens for Gram-negative neonatal meningitis [16].

To date, no international consensus has established an optimal antimicrobial regimen or treatment duration for neonatal *E. anophelis* infections. The available evidence is largely based on case reports, case series, and retrospective studies, with treatment strategies varying according to the site and severity of infection and the antimicrobial susceptibility profile of the isolate [16]. Prolonged therapy may be required in cases complicated by ventriculitis, hydrocephalus, brain abscess, or persistent CSF positivity. In our patient, the persistence of a positive CSF culture after 13 days of targeted therapy further demonstrates the difficulty of eradicating this infection and supports an individualized treatment duration based

on the clinical, microbiological, and neuroradiological response.

Our case also highlights the importance of a multidisciplinary approach involving neonatologists, microbiologists, infectious disease specialists, and neurosurgeons. External ventricular drainage played a critical role in the management of the rapidly progressive hydrocephalus and provided access for repeated CSF monitoring. In severe neonatal infections, neurosurgical intervention may be essential for controlling intracranial pressure and managing complications such as hydrocephalus and ventriculitis.

However, the long-term neurological prognosis remains uncertain. Published pediatric and neonatal cases have reported substantial morbidity among survivors, including developmental delay, seizures, motor impairment, cognitive impairment, and other neurological sequelae [17]. Consequently, prolonged neurodevelopmental and neurological follow-up is warranted in survivors of severe neonatal *Elizabethkingia* meningitis.

Finally, this case emphasizes the importance of strengthening healthcare-associated infection prevention and environmental surveillance in neonatal intensive care settings. Identification of *E. anophelis* should prompt consideration of an environmental investigation, including assessment of water sources, sinks, medical devices, and relevant infection-control procedures, particularly when more than one case occurs or when a healthcare-associated source is suspected [18]. Early recognition of a potential environmental reservoir may allow implementation of targeted control measures and prevent secondary infections.

4. Conclusions

This case highlights that *Elizabethkingia anophelis* should be considered an emerging etiological agent of severe neonatal meningitis. Its multidrug-resistant profile, the high frequency of neurological complications, and the absence of standardized therapeutic recommendations make it a significant diagnostic and therapeutic challenge.

Early microbiological identification, prompt adjustment of antimicrobial therapy according to susceptibility testing, close neuroradiological monitoring, repeated assessment of CSF sterilization, and neurosurgical management when required are essential components of the management of complicated neonatal *E. anophelis* meningitis. Long-term neurological and neurodevelopmental follow-up is also warranted given the potential for severe neurological sequelae.

Author Contributions

Khayi, F.: Conceptualization, Methodology, Writing: Original Draft, patient management. El Hammoumi, M.: Investigation, Clinical Management. Taher, F.: Investigation, Clinical Management. Daoudi, A.: Investigation, Clinical Management. Ezzarigua, N.: Investigation (Bacteriological and microbiological investigations), Formal Analysis. Wakrim, S.: Investigation (Radiological imaging and MRI interpretation). Lmejjati, M.: Surgical Intervention/Neurosurgical Management.

Conflicts of Interest

The authors declare no conflicts of interest.

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