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Elizabethkingia anophelis

The First Italian Report of Neonatal Meningitis and Sepsis Associated With Breast-Pump–Expressed Milk

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Abstract: *Elizabethkingia anophelis* (*E. anophelis*) is an emerging opportunistic pathogen and environmental gram-negative rod. It has been implicated in neonatal meningitis, sepsis, and nosocomial outbreaks, having also been isolated from contaminated medical equipment. Infections by *E. anophelis* are associated with morbidity and mortality, primarily due to its intrinsic multidrug resistance. The determinants of transmission, pathogenicity, antibiotic resistance, and global epidemiology remain poorly understood, and no established therapeutic guidelines are currently available. Newborn positive blood culture, CSF, and breast milk samples were inoculated on Blood Agar plates and incubated at 37 °C, 5% CO_2 for 24 hours. The isolates were identified by mass spectrometry. The minimal inhibitory concentrations (MICs) were obtained by gradient strip method following manufacturer's instructions. We report here the first documented case in Italy of neonatal meningitis and sepsis caused by *E. anophelis* associated with maternal expressed breast milk. The combination therapy of vancomycin, cotrimoxazole, and levofloxacin for pediatric *E. anophelis* infection has yielded success.

Key Words: *Elizabethkingia anophelis*, neonatal sepsis, neonatal meningitis

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Currently, the Elizabethkingia genus includes 7 distinct species.[1] These are considered environmental bacteria, ubiquitously distributed in different natural reservoirs, including soil, freshwater sources, and insect or amphibian guts.[2] Among them, *E. meningoseptica* and *E. anophelis* are the most recognized pathogenic species associated with human infections. *E. anophelis* was first isolated from the midgut of mosquito species, *Anopheles gambiae* and *Anopheles stephensi*.[3] Elizabethkingia is an obligate aerobe, non-motile, glucose nonfermenting, non-spore forming, catalase-oxidase-urease positive, weakly indole-positive, and nitrate-negative, pale yellow-pigmented gram-negative bacillus; it can be cultured on blood and chocolate agar at 37 °C.[4] *E. anophelis* is not part of the normal human microbiota, but it has been recovered from nosocomial environments, including hospital surfaces and tap water.[5] Over recent years, it has emerged as a nosocomial opportunistic pathogen causing meningitis and sepsis, especially in premature newborns and immunocompromised patients. Furthermore, *E. anophelis* poses risks for community and health care-associated outbreaks.[6–9]

The treatment of *E. anophelis* infection is very challenging due to its intrinsic resistance to several classes of antibiotics, including beta-lactams (carbapenems included), aminoglycosides, fluoroquinolones, tetracyclines, and chloramphenicol.[10] High mortality rates are reported, ranging from 18% to 70%.[11]

However, the true incidence of *E. anophelis* infection is likely underestimated, due to the lack of reliable laboratory tests. Many cases of infection initially attributed to *E. meningoseptica* have been later reclassified as *E. anophelis* by molecular analysis, and increasing evidence suggests that this species is the major human pathogen within the Elizabethkingia genus.[9,12]

To the best of our knowledge, we report the first documented case in Italy of *E. anophelis* sepsis and meningitis in a previously healthy, full-term neonate.

CASE REPORT

A 12-day-old male neonate was admitted to a level I neonatal care unit with a documented fever exceeding 38 °C, which had onset a few hours before hospital admission. He was born at term at another hospital through spontaneous vaginal delivery, following an uncomplicated pregnancy and delivery, to a healthy mother. His birth weight was 3350 g, and his Apgar score was 10/10.

Maternal TORCH serology and GBS screening were negative. No perinatal complications occurred, and the newborn was discharged on day 3 of life on exclusive

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breastfeeding with expressed breast milk collected through breast pump or manual expression. On day 9 of life, his mother developed a fever and was diagnosed with mastitis; oral antibiotic amoxicillin therapy was started.

At admission, the neonate was irritable, with poor feeding and fever (39 °C). Examination showed a bulging anterior fontanel, generalized hypertonia, abnormal reflexes, tachycardia (HR 190 bpm), and a cardiac murmur, while blood pressure and respiratory parameters were normal. Initial laboratory investigations revealed mild leukocytosis (WBC: 17,000/mm^3, neutrophils 57.3%), normal hemoglobin (Hb: 18.7 g/dL) and platelets (PLT: 383×10^9/L) count; inflammatory markers were mildly elevated with C-reactive protein (CRP) at 28.3 mg/L (reference cutoff: 8 mg/L). Chest and abdominal radiographs were unremarkable.

Blood and urine samples were collected for culture, and empirical broad-spectrum antibiotic therapy was started with ampicillin (50 mg/kg every 8 h) and netilmicin (3 mg/kg every 12 h). After 8 hours, the clinical condition deteriorated with the onset of lethargy, vomiting, and persistent fever. Laboratory tests worsened (WBC: 8210/mm^3 with 69.4% neutrophils, Hb: 13.6 g/dL, CRP: 289.1 mg/L), therefore, a lumbar puncture was performed. Ceftazidime and acyclovir were added, and ampicillin was increased to meningeal dosing (100 mg/kg every 8 h). Cerebrospinal fluid (CSF) was cloudy with hypoglycorrhachia (2 mg/dL with concomitant blood glucose 145 mg/dL), hyperproteinorrhachia (207 mg/dl), and polymorphonuclear pleocytosis. CSF and blood samples were submitted to FilmArray meningitis/encephalitis (ME) and Blood Culture Identification 2 (BCID2) panels, respectively, which returned a negative result. CSF microscopic examination showed gram-negative bacilli. After overnight incubation, both CSF and blood culture revealed *E. anophelis.* Urine culture was negative. The isolate was resistant to all tested β-lactams (ampicillin, cefotaxime, ceftazidime, imipenem, piperacillin/tazobactam) and aminoglycosides (amikacin); it showed susceptible-increased exposure to ciprofloxacin and levofloxacin. No interpretative breakpoints were available for vancomycin, rifampicin, and cotrimoxazole. Due to the patient's clinical status, he was transferred to the referral centre for neonatal intensive care unit. Acyclovir therapy was discontinued, based on these microbiological results and literature data,[12–16] antibiotic therapy was modified to include cotrimoxazole (5 mg/kg every 8 h), vancomycin (15 mg kg every 12 h), and off-label levofloxacin (10 mg/kg every 12 h).

Significant clinical improvement followed within 48 hours, with stable defervescence and normalization of neurological findings. A repeat lumbar puncture performed one week later revealed sterile CSF. After 2 weeks of therapy, in agreement with the infectious disease specialist, vancomycin was replaced with intravenous rifampicin (7.5 mg/kg every 12 h). This therapy was continued for 3 weeks following the negative CSF result.

Initial electroencephalography showed diffuse cerebral dysfunction and left temporoparietal focality; it improved by the fourth day of targeted therapy and normalized by the time of discharge.

Likewise, in the acute phase, cranial ultrasound revealed ependymal hyperechogenicity and septa in the anterior part of the lateral ventricles, which progressively improved by the third day of effective therapy and completely recovered over the hospitalization course.

The child was discharged after 34 days of hospitalization, in good clinical condition with normal neurological examination and negative inflammatory markers. A follow-up MRI, performed 4 days later, showed no abnormalities. No neurological impairments were identified in the neuropediatric follow-up. In addition, no other relevant infectious disease occurred in the first years of life; therefore, no immunodeficiency was suspected.

To investigate the source of infection, mother's blood cultures and breast milk samples were also obtained. Blood cultures came back negative, but *E. anophelis* was isolated from the breast milk, *P. aeruginosa*, *A. baumannii complex*, *S. aureus*, *D. acidovorans,* and *S. marcescens* were also isolated. Consequently, breastfeeding was suspended and replaced with formula. Breast milk culture repeated at follow-up was negative, and breastfeeding was subsequently resumed.

Clinical isolates from blood, CSF, and breast milk samples were inoculated on blood agar plates and incubated at 37 °C with 5% CO_2 for 24 hours. Isolates were identified by matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF) mass spectrometry (MS) (VITEK MS Biomerieux) with the database version

TABLE 1. Antimicrobial Susceptibility Testing Results of *E. anophelis*; MIC in µg/mL (Measured by E-Test bioMérieux)

	Isolate 1 (Blood)		Isolate 2 (CSF)		Isolate 3 (Milk)	
Drug	**MIC**	**Interpretation**	**MIC**	**Interpretation**	**MIC**	**Interpretation**
Amikacin	> = 256	R	> = 256	R	> = 256	R
Ampicillin	> = 256	R	> = 256	R	> = 256	R
Cefepime	> 16	R	> 16	R	> 16	R
Cefotaxime	> = 32	R	> = 32	R	> = 32	R
Ceftazidime	> = 32	R	> = 32	R	> = 32	R
Ciprofloxacin	0.5	I	0.5	I	2	R
Levofloxacin	0.19	S	0.19	S	0.75	I
Imipenem	> 8	R	> 8	R	> 8	R
Meropenem	> 8	R	> 8	R	> 8	R
Piperacillin-tazobactam	> 64	R	> 64	R	> 64	R
Rifampicin	0.25	IE	0.25	IE	1	IE
Vancomycin	12	IE	12	IE	12	IE
Trimethoprim-sulfamethoxazole	0.25	IE	0.19	IE	0.25	IE

IE indicates insufficient evidence.

IVD_1.0.10. The minimal inhibitory concentrations (MICs) were obtained by the gradient strip method (E-test BioMèrieux, France) following the manufacturer's instructions. MICs were interpreted according to EUCAST Clinical Breakpoint Tables v. 9.0, using PK-PD (non-species related) breakpoints. Table 1 shows the antimicrobial susceptibility pattern of 3 clinical isolates. For vancomycin, rifampicin, and cotrimoxazole, breakpoints were not available, so we did not interpret susceptible or resistant.

DISCUSSION

Elizabethkingia anophelis is a rare cause of neonatal meningitis, mostly affecting preterm infants. Accurate species identification within the *Elizabethkingia* genus can be challenging. Earlier MALDI-TOF MS databases have been associated with misidentification among *Elizabethkingia* species, particularly between *E. anophelis* and *E. meningoseptica*. However, updated databases have significantly improved diagnostic accuracy. In our case, molecular confirmation by whole-genome sequencing or 16S rRNA gene sequencing was not performed, representing a limitation of the study, as molecular methods remain the gold standard for definitive species identification.

To our knowledge, this is the first case in Italy of *E. anophelis* sepsis and meningitis in a healthy term neonate, without evidence of nosocomial origin.

Neonatal meningitis by *E. anophelis* has been already described, with molecular evidence suggesting vertical transmission from mothers with chorioamnionitis.[17] Conversely, vertical transmission in our case appears unlikely, given the latency between delivery and the onset of symptoms (12 d). Other cases in the literature, mainly from Southeast Asia, have described *E. anophelis* contamination of hospital taps and aerators with horizontal transmission by health care workers during handwashing with contaminated water in intensive care units.[5,18] This highlights its significance as a nosocomial infection. However, in most reported cases, the source of the infection could not be identified, although nosocomial transmission was usually presumed.[19]

Moreover, many neonates in hospital settings are particularly vulnerable to opportunistic infections because of weakened immune systems due to intensive medical treatment, prematurity, chronic conditions, and infections.[20] However, in our case, the baby was healthy and did not require intensive care at birth. Moreover, the 9-day interval between hospital discharge after birth and infective symptoms onset does not support nosocomial transmission.

Recent literature reports an increasing number of neonatal meningitis caused by *E. anophelis* also in full-term, otherwise healthy neonates. Two such cases have been documented in China[12] and one more healthy 17-month-old infant in New York.[21] To the best of our knowledge, only one similar case has already been reported in Europe to date: the affected neonate was formula-fed, and *E. anophelis* was identified as a contaminant in the automatic formula dispenser used for home milk preparation.[18]

In our case, the only plausible source of infection was expressed breast milk, since *E. anophelis* was isolated from both mother's expressed milk and infant's cultures. All other bacteria isolated from breast milk were considered environmental contaminants. The question remains as to how the milk could have become contaminated by *E. anophelis*. Given the organism's known ability to persist in water systems[22], contamination may have occurred through improperly cleaned breast pump or handwashing with contaminated water. Some limitations in our report included the absence of a genetic comparison between clinical isolates and the lack of environmental water sampling. However, the mother's breast pump and bottle cleaning procedures were investigated and found to be apparently appropriate. Recent studies have highlighted how human milk microbiota composition can be influenced by various factors, including milk expression. Specifically, indirect breastfeeding and breast pump use have been consistently associated with reduced microbial diversity and a shift toward environmental bacteria.[23] These findings suggest that direct breastfeeding supports maternal-infant microbial exchange, whereas indirect breastfeeding may promote the colonization by environmental bacteria, which can become true pathogens for newborns.

E. anophelis typically present a multidrug-resistant profile, with limited available therapeutic options. Interestingly, as previously mentioned, several pediatric patients have been effectively treated with vancomycin, either alone or in combination with other antibiotics such as cotrimoxazole and levofloxacin. Vancomycin is primarily active against gram-positive bacteria; the reason for its antimicrobial effects against *E. anophelis* remains unclear.[14] Interpretation of antimicrobial susceptibility for *Elizabethkingia* species is challenging because standardized clinical breakpoints are lacking for several agents, including vancomycin, rifampicin, and trimethoprim-sulfamethoxazole. Therefore, MIC values for these antibiotics were reported descriptively without categorical interpretation.

Therapeutic decisions were guided by a review of available literature, consultation with infectious disease specialists, and the patient's clinical response. Previous reports have documented successful treatment of pediatric Elizabethkingia infections using vancomycin in combination with trimethoprim-sulfamethoxazole and fluoroquinolones, despite the organism's gram-negative classification. In our case, rapid clinical improvement after initiation of targeted combination therapy supported the chosen regimen.

Recent studies employing microbial genomics have aimed to identify novel potential drug targets for the development of new antibiotics against *E. anophelis*.[24]

This case highlights the critical importance of precise and accurate sampling and culture processing techniques in identifying rare pathogens, such as *E. anophelis*. Further research is essential to elucidate the pathogen's mechanisms of virulence, modes of transmission, and to establish evidence-based therapeutic strategies.

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