Case Report

Central Line-Associated Bloodstream Infection Due to Elizabethkingia anophelis: Case Report and Literature Review on Pediatric Infections

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Central Line-Associated Bloodstream Infection due to *Elizabethkingia anophelis*: Case Report and Literature Review on Pediatric Infections

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Abstract: *Elizabethkingia anophelis* is an opportunistic pathogen causing lifethreatening infections in humans, particularly in immunocompromised patients, neonates and elderly. Infections caused by this species are exceptionally challenging to treat due to its multiresistance to antimicrobials. We report a case of central line-associated bloodstream infection by *E. anophelis* in a 2.5-year-old girl with acute lymphoblastic leukemia successfully treated with a combination of piperacillin/tazobactam and amikacin. The literature was also reviewed on paediatric infections caused by *E. anophelis*. Accurate identification with MALDI-TOF, or using molecular techniques, is crucial because of varying susceptibilities among species. Early, accurate diagnosis and prompt effective treatment optimize outcomes.

Keywords: *Elizabethkingia anophelis*; pediatric infections; neonate; identification; antimicrobial susceptibility

1. Introduction

Elizabethkingia species are aerobic, glucose-nonfermenting, catalase-positive, oxidase-positive, and indole-positive Gram-negative bacilli widely distributed in natural environments such as soil, water, and plants, as well as in healthcare settings [1]. The genus *Elizabethkingia* comprises 6 species, namely, *E. meningoseptica*, *E. anophelis*, *E. miricola*, *E. bruuniana*, *E. ursingii*, and *E. occulta* [1]. *E. anophelis* is an opportunistic pathogen most commonly affecting infants or critically ill adults with underlying comorbidities [1,2]. It is particularly known to cause neonatal sepsis and meningitis especially in premature newborns and sometimes is involved in outbreaks of life threatening infections, with mortality rates ranging from 24% to 60% [1-5].

Herein, we describe a case of central line-associated bloodstream infection (CLABSI) due to *E. anophelis* in a 2.5-year-old girl with acute lymphoblastic leukemia and review the literature on pediatric cases caused by *E. anophelis.*

2. Case Description

A 2.5-year-old girl was diagnosed with acute lymphoblastic leukemia of B lineage (B-ALL). The full blood count (FBC) at diagnosis was WBC: 2,300/mm^3, Hb: 6.9g/dL and PLT: 16,000/ mm^3. The myelogram showed full infiltration by lymphoblasts and the immonophenotyping revealed common pre-B ALL (EGIL classification). A central venous catheter (CVC) Hickman type was inserted and

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then commenced on intensive chemotherapy according to ALL IC-BFM 2009 Protocol. Due to prognostic factors and treatment response she was classified to receive treatment of intermediate risk group.

In a febrile neutropenia episode three months post starting intensive chemotherapy, *Streptococcus mitis* was isolated from blood cultures taken from CVC. Based on the results of the susceptibility testing the patient was given teicoplanin as a loading dose at 10mg/kg every12 hours intravenously for three doses, followed by the maintainance dose of 10mg/kg once daily, along with teicoplanin lock therapy. The CVC was kept in place. She was started again on chemotherapy according to her protocol.

Seven months after diagnosis and a month before ending intensive protocol when she was receiving cytarabine 70mg/m^2/d and thioguanine 60mg/kg/m^2 she became febrile. Blood, urine, stool, and pharyngeal cultures were taken and she was started on empirical treatment with intravenous piperacillin/tazobactam at a dosage of 300mg/kg every 6 hours. Full blood count was WBC: 200/ mm^3 with absolute neutrophil count (ANC): 0/µl, Hb: 8,6g/dl, PLT: 38000/µl, and CRP: 4.5mg/dl (normal value <0.5mg/dl). Chemotherapy was stopped during the episode. Blood specimens taken from the CVC the first day of the febrile episode were inoculated into BacT/Alert PF bottles and incubated in a BacT/Alert 3D blood culture system (BioMérieux, Marcy L' Etoile, France). After 17.2 hours the blood cultures turned positive for a Gram-negative microorganism, *Elizabethkingia anophelis* as identified by Matrix-assisted laser desorption ionization-time of flight (MALDI-TOF) mass spectrometry (VITEK MS system, BioMérieux; version 3.2). The blood cultures continued to be positive for *E. anophelis*, and the patient remained febrile for 3days.

The *in vitro* susceptibility testing performed by E-test revealed that *E. anophelis* was susceptible to piperacillin/tazobctam, amikacin, minocycline, doxycycline, ciprofloxacin, levofloxacin, trimethoprim-sulfamethoxazole and tigecycline, intermediate to vancomycin, and resistant to piperacillin, ceftriaxone, ceftazidime, imipenem, meropenem, ceftazidime/avibactam, meropenem/ vaborbactam, imipenem/relebactam, eravacycline, plazomicin and tetracycline (Table 1).

Table 1. MICs of isolated *Elizabethkingia anophelis* as determined by E-test.

Antimicrobial agents	MIC Breakpoints (µg/ml)			MIC (µg/ml)	Interpretation*
Piperacillin	≤16	32-64	≥128	≥256	R
Piperacillin/tazobactam	≤16/4	32/4-64/4	≥128/4	12	S
Ceftazidime	≤8	16	≥32	≥256	R
Ceftriaxone	≤8	16-32	≥64	64	R
Cefepime	≤8	16	≥32	16	I
Imipenem	≤4	8	≥16	≥32	R
Meropenem	≤4	8	≥16	≥32	R
Ceftazidime/avibactam	≤8/4	-	≥16/4	12	R
Imipenem/relebactam	≤1/4	2/4	≥4/4	≥32	R
Meropenem/vaborbactam	≤4/8	8/8	≥16/8	≥64	R
Gentamicin	≤4	8	≥16	6	I
Amikacin	≤16	32	≥64	12	S
Plazomicin	≤2	4	≥8	64	R
Tetracycline	≤4	8	≥16	48	R
Doxycycline	≤4	8	≥16	3	S
Minocycline	≤4	8	≥16	0.75	S
Eravacycline	≤0.5	-	>0.5	0.75	R
Tigecycline	≤2	4	≥8	0.75	S

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Ciprofloxacin	≤1	2	≥4	0.25	S
Levofloxacin	≤2	4	≥8	0.25	S
TMP/SXT	≤2/38	-	≥4/76	0.19	S
Vancomycin	≤4	8-16	≥32	12	I
Rifampicin	≤1	2	≥4	0.5	S

S, susceptible; I, intermediate; R, resistant; MIC, minimum inhibitory concentration; TMP/SMX, trimethoprim-sulfamethaxole. *CLSI breakpoints for "other non-Enterobacterales" were applied per CLSI document M100-Ed32 guidelines. The breakpoints used for ceftazidime/avibactam, imipenem/relebactam, meropenem/vaborbactam and plazomicin were those reported for Enterobacterales. The breakpoints used for vancomycin and rifampicin were those reported for *Staphylococcus* spp. For tigecycline the FDA-recommended MIC breakpoints were applied.

Based on the profile of the antibiogram, amikacin 20mg/kg every 24 hours was added. Four days after the start of the episode the patient became afebrile and blood cultures became negative. She continued on antibiotics for a total of 10 days. The CVC was not removed. She is now on maintenance treatment with chemotherapeutical agents per os.

The source of the infection remained undetermined because the microorganism was not isolated from any of the environmental samples (water supplies, surfaces and medical equipment).

3. Discussion

E. anophelis was initially isolated from the midgut of the Anopheles gambiae mosquito in 2011 [6]. The first reported clinical case of *E. anophelis* infection was meningitis in an 8-day-old girl in the Central African Republic. *E. anophelis* was identified by 16S-rRNA sequencing [7]. Since this initial report sporadic cases of serious systemic infections in infants and adults and several outbreaks of *E. anophelis* have been reported in Asia, and the USA. The largest outbreak was registered in the Midwestern United States, resulting in 20 deaths among 65 infected patients [3]. To date, in Europe only 2 adult cases and one outbreak of *E. anophelis* have been described [4,8,9]. In many previous studies it has been revealed that the incidence of *E. anophelis* infections was highly underestimated due to misidentification as *E. meningoseptica* based on phenotypes and prior MALDI-TOF systems not including *E. anophelis* in their diagnostic databases [1,5]. MALDI-TOF with updated databases and molecular methods such as 16S rRNA sequencing and whole-genome sequencing (WGS) are reliable and accurate in species identification. Our isolate was identified by MALDI-TOF MS (v. 3.2) containing in its database three species of the genus *Elizabethkingia*, namely *E. meningoseptica*, *E. anophelis* and *E. miricola*.

In a Medline/Pubmed searching the keywords ''*Elizabethkingia anophelis* pediatric infections''we found only 21 previously reported cases [5,7,10-19]. Our case is the first pediatric *E. anophelis* infection described in Europe. Table 2 summarizes the patients' characteristics (gender, age, country of origin), the clinical manifestation, underlying medical conditions, the type of specimen cultured, antibiotic treatment and outcome.

The majority of cases (59.1%) involved newborns mostly premature. A slight female predominance was observed (1.2:1). Although *E. anophelis* is ubiquitous in nature with global distribution, most cases (81.8%) have been reported in Asian countries. Meningitis was the most common presentation in newborns. Other clinical manifestations included bloodstream and respiratory infections. The present case was a CLABSI. It has been demonstrated that *E. anophelis* has the ability to form biofilm that facilitate their establishment in CVCs, complicating treatment [20]. The source of the infection and the route of transmission remain unclear for all cases, except for one of vertical transmission from the mother who had chorioamnionitis to her neonate [11]. The majority of children had their immune system weakened by prematurity, by intensive medical interventions, or by other comorbities. The case fatality rate of the infected children was 33.3%, with deaths being most common among infected neonates. Five children among survivors of *E. anophelis* meningitis developed neurologic sequelae such as hyrocephalus and hearing loss [10,12,15,19].

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Table 2. Characteristics of paediatric patients with Elizabethkingia. anophelis infections.

Ref.	Country of origin	Age	Sex	Diagnosis	Underlying conditions	Specimen type	Antibiotic treatment	Outcome
7	Central African Republic	8 d	F*	Meningitis	Asphyxia at birth	CSF	Gentamicin, ampicillin	Death
10	China	22 d	M	Meningitis	Prematurity	Blood, CSF	Vancomycin, piperacillin/tazobactam	Survival (hydrocephalus)
10	China	18 d	F	Meningitis	None	CSF	Vancomycin, piperacillin/tazobactam	Survival (hydrocephalus)
11	Hong Kong	21 d	M	Meningitis	None	Blood, CSF	Vancomycin, piperacillin, rifampicin	Survival (without neurologic sequelae)
11	Hong Kong	1 d	F	Meningitis	Prematurity	Blood, CSF	Vancomycin, piperacillin/tazobactam, rifampicin	Survival (without neurologic sequelae)
5	Hong Kong	1 mo	F	Catheter-related bacteremia	Prematurity, RDS, PDA	Blood	Vancomycin, cefoperazone/sulbactam	Death
5	Hong Kong	8 d	F	Meningitis	Imperforated anus, rectovaginal fistula	Blood, CSF	Vancomycin, rifampicin	Survival
12	Cambodia	1 d	M	Sepsis	Prematurity	Blood	Imipenem	Survival
12	Cambodia	51 d	F	VAP	Ventricular septal defect	Respiratory secretion	Ciprofloxacin	Death
12	Cambodia	1 d	M	Sepsis	Prematurity	Blood	Ampicillin, gentamicin	Death
12	Cambodia	15 wk	F	Meningitis	Failure to thrive	Blood	Ceftriaxone	Unknown
12	Cambodia	8 mo	M	VAP	Duodenal atresia	Respiratory secretion	Meropenem	Death
12	Cambodia	7 d	F	Meningitis	Prematurity	Blood	Ciprofloxacin, vancomycin	Survival (hydrocephalus)
12	Thailand	1 d	F	Sepsis	Prematurity	Blood	Ampicillin, gentamicin	Death
13	India	2 y	F	Bronchopneumonia	NR	Blood	Pipercillin/tazobactam, levofloxacin, colistin, ceftriaxone/sulbactam, imipenem	Survival
14	India	11 d	M	Meningitis, sepsis	Prematurity	Blood, CSF	Pipercillin/tazobactam, vancomycin, ciprofloxacin	Survival (without neurologic sequelae)

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15	India	12 d	M	Meningitis, sepsis	Prematurity	Blood, CSF	Cefoperazone/sulbactam, vancomycin, TMP/SMX, rifampicin, ciprofloxacin	Survival (hydrocephalus)
16	India	7 mo	M	Bacteremia	NR	Blood	Vancomycin, piperacillin/tazobactam	Survival
17	Turkey	11y	M	Bacteremia	congenital tracheomalacia, cerebral palsy, SARS-CoV-2 past infection	Blood	Colistin, ciprofloxacin	Death
18	New York	17 mo	F	Sepsis, pneumonia	None	Blood	Ampicillin, ceftriaxone, amoxicillin/clavulanate	Survival
19	South Carolina	11 d	M	Meningitis, bacteremia	Prematurity	Blood, CSF	Vancomycin, rifampicin, ciprofloxacin, TMP/SMX	Survival (hearing loss, hydrocephalus)
Present case	Greece	2.5 y	F	CLABSI	ALL	Blood	pipercillin/tazobactam, amikacin	Survival

*F, female; M, male; d, days; wk, weeks; mo, months; y, years; VAP, ventilator-associated pneumonia; RDS, respiratory distress syndrome; PDA, patent ductus arteriosus; NR, not reported; CLABSI, central-line associated bloodstream infection; ALL, acute lymphoblastic leukemia.

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E. anophelis has been known to be resistant to multiple antimicrobial agents, including most β-lactams, β-lactam/β-lactamase inhibitors, carbapenems, and polymyxins [1]. The majority of the reported cases were treated with vancomycin combined with other antibiotics, such as rifampicin, ciprofloxacin, trimethoprim-sulfamethoxazole or piperacillin/tazobactam. Our isolate was intermediate to vancomycin, and resistant to β-lactams, carbapenems, and the novel β-lactam/β-lactamase inhibitors such as ceftazidime/avibactam, imipenem/relebactam, and meropenem/vaborbactam because inhibitors have low activity against the metallo-β-lactamases produced by *E. anophelis*. In our case piperacillin/tazobactam was initially given as empiric therapy and was continued with the addition of amikacin after susceptibility data became available. Notably, four of the nine reported cases of *E. anophelis* meningitis with favorable outcome were treated with combinations including piperacillin/tazobactam [10,11,14]. Comparable results suggestive of susceptibility to piperacillin/tazobactam have been reported in studies of Han et al., Jian et al. and Perrin et al. [3,21,22]. However, further evaluation of in *vivo* data and continuous surveillance of antimicrobial resistance are required to make optimal therapeutic decisions. It has been shown that inappropriate empirical antimicrobial therapy is an independent risk factor for increased mortality in patients infected with *E. anophelis*.[1] Accurate identification is essential for selecting the appropriate antimicrobial therapy because of the varying susceptibility profiles among species.

4. Conclusion

The increasing number of cases of *E. anophelis* infections which is a result of availability of new, accurate identification methods, highlight the clinical significance of this opportunistic pathogen in pathogenesis of human infections. Early diagnosis, timely and accurate species identification and prompt effective treatment optimize outcomes.

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