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Case Report

# Elizabethkingia Meningoseptica Bacteremia with Meningitis in an Immunosuppressed Host

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## Abstract:

*Elizabethkingia Meningoseptica,* previously known as *Chryseobacterium Meningosepticum and Flavobacterium Meningosepticum,* is a rare multi drug resistant bacteria found in soil and water that can cause nosocomial infections. It is an opportunistic human pathogen mostly associated with neonatal meningitis. However, it is infrequently found to cause bacteremia in immunocompromised adults. Several outbreaks have been reported worldwide in India, Taiwan, United Kingdom, and United States. Outbreak from *Elizabethkingia Anophelis* has been reported in Wisconsin between 2015-2016, however most cases reported for *Elizabethkingia Meningoseptica* in the United States have been reported prior to 1996. We report a case of *Elizabethkingia Meningoseptica* with septic shock secondary to bacteremia and meningitis in an immunocompromised patient.

**Keywords:** *Elizabethkingia Meningoseptica;* bacteremia; meningitis

## Introduction:

*Elizabethkingia Meningoseptica* is a non-fermenting gram-negative organism commonly detected in soil and water but traditionally, rarely reported to be the causative agent of infections in humans,[3]. However, it has recently emerged as a cause of life-threatening infections especially in immunocompromised hosts. The genus is usually multi-drug resistant, exhibiting sensitivity to Minocycline but resistance to most beta lactams, beta lactams / beta lactam inhibitors, carbapenems and aminoglycosides,[4]. Most of the cases have been traced down to rubber stoppers used for milk bottles in neonatal meningitis, respiratory equipment, sink drains and water, [6, 7].

We present a unique case of community acquired *Elizabethkingia Meningoseptica* presenting with septic shock due to bacteremia and subclinical meningitis without alarm symptoms.

## Case Description

A female patient in early 70s with a past medical history of marginal zone B cell lymphoma in remission (treated with 6 cycles of bendamustine and rituximab 1.5 years ago), autoimmune hemolytic anemia treated with splenectomy and hypogammaglobulinemia on monthly intravenous immunoglobulin (IVIG) therapy, disseminated zoster virus infection 3 months prior to presentation, postherpetic neuralgia, chronic diarrhea - secondary to IVIG therapy, presented to the emergency department (ED) with generalized weakness of 1 week duration. The patient was doing

relatively well until she started to feel progressively weak to an extent where she did not have enough strength to ambulate and therefore called Emergency Medical Services (EMS). She was noted to be hypotensive with a systolic blood pressure of 60 by EMS. Denied any fever, chills, headaches, nausea, vomiting, photophobia, nuchal rigidity, abdominal pain, dysuria, frequency, cough, or shortness of breath.

On presentation in the ED, her blood pressure was 63/33 mmHg, heart rate was 129 beats per minute with temperature of 101.1oF. Initial laboratory testing was significant for leukocyte count, 21.27 K/uL with 93.9% neutrophilia; blood urea nitrogen, 63 mg/dL; creatinine, 2.9 mg/dL (increased from a baseline of 0.7 mg/dL); lactic acid, 8.0 mmol/L. Sepsis protocol was initiated, and she was administered 2 grams of aztreonam, 1 grams of vancomycin, 2-liter bolus of lactated ringers and continuous norepinephrine infusion. She was admitted to the Intensive Care Unit (ICU) with a diagnosis of septic shock.

Her two initial sets of blood cultures sent from the ED grew *Elizabethkingia Meningoseptica*. Urine culture was positive for Klebsiella. She was initially treated with Meropenem, which was switched to Meropenem/Vaborbactam 2 grams Intravenously (IV) every 12 hours 1 day later when the sensitivity report showed resistance to meropenem. The final sensitivity report resulted 3 days later that showed sensitivity to ciprofloxacin (S <=0.25), Piperacillin/Tazobactam (S <= 8) and TMP/SMX (S 1/19).

A transthoracic echocardiogram performed on day 2 of hospitalization showed no valvular vegetations with an ejection fraction of 15%, severely decreased left ventricular systolic function, grade II diastolic dysfunction, mild mitral regurgitation, mild tricuspid regurgitation, mild pulmonic regurgitation, mild aortic stenosis, and multiple left ventricular wall motion abnormalities. Transesophageal echocardiogram performed on day 8 of admission revealed EF of 35-40% with no evidence of valvular vegetations. Global systolic LV function was moderately to severely decreased. A cardiac catheterization was scheduled as outpatient. A whole-body nuclear scan was performed on Day 9 as blood cultures remained persistently positive that showed focal uptake in Left proximal greater trochanter region. Follow up Magnetic Resonance Imaging showed trochanteric bursitis without osteomyelitis, however clinically trochanter bursa was unremarkable. Patient's blood lactate and hemodynamic status gradually improved, and she was tapered off pressors on Day 9.

Blood cultures remained persistently positive until day 14 of the hospitalization. Antibiotics regimen was changed in accordance with their sensitivities as shown in Table 1 and Table 2. On day 11, patient complained of diffuse headache without any fever, chills, nausea, vomiting, photophobia, or nuchal rigidity. Patient was alert and oriented to name, place, and time without any focal neurological deficits. As no focal source of infection was identified at that time, a diagnostic lumbar puncture was performed. Cerebrospinal Fluid (CSF) appearance was slightly cloudy. CSF studies were consistent with bacterial meningitis with glucose of 14 mg/dL, total nucleated cells 830 (lymphocytes 4%, neutrophils 92%, monocytes 4%), protein 90 mg/dL, Red Blood Cells 260, and lactate dehydrogenase 61. CSF culture grew Elizabethkingia Meningoseptica resistant to Aztreonam, Cefepime, Ceftriaxone and Meropenem. Additional sensitivities were run for vancomycin and Daptomycin with MIC of 16 and > 256 respectively.

Blood cultures collected from day 14 showed no growth and remained negative on the subsequently from days 15 to 19. The patient was discharged on day 23 on the final antibiotic regimen of Levofloxacin 750 mg IV every 24 hours, Minocycline 100 mg IV every 12 hours, and Rifampin 600 mg IV every 24 hours for a total duration of four weeks from the first negative blood culture. Acyclovir 400 mg orally every 12 hours was continued for antiviral prophylaxis due to patient's immunocompromised status.

| **Hospitalization Day** | | Day 1 | Day 3 | Day 5, 7, 8 | Day 6 | Day 9 | Day 11 | Day 11 | Day 13 | Day 14, 16-19 |
|---|---|---|---|---|---|---|---|---|---|---|
| **Culture Results** | | Blood – Elizabethki ngia meningose ptica | Blood – Elizabethki ngia meningose ptica | Blood – Elizabethki ngia meningose ptica | Blood – Elizabethki ngia meningose ptica | Blood – Elizabethki ngia meningose ptica | Blood – Elizabethki ngia meningose ptica | CSF – Elizabethki ngia meningose ptica | Blood – Elizabethki ngia meningose ptica | Blood – No growth |
| **Sensitivities** | **Amikacin** | R>32 | No sensitivities Run | No sensitivities Run | R>32 | R>32 | | | No sensitivities Run | |
| | **Amoxycillin/ Clavulanic Acid** | | | | | | | | | |
| | **Ampicillin** | | | | | | | | | |
| | **Ampicillin/Sulbactam** | | | | | | | | | |
| | **Aztreonam** | R>16 | | | R>16 | R>16 | | R>16 | | |
| | **Cefepime** | R>16 | | | R>16 | R>16 | | R>16 | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| **Cefiderocol** | | | | 16 | | |
| **Cefoxitin** | | | | | | |
| **Ceftriaxone** | R>32 | R>32 | R>32 | | R>32 | |
| **Chloramphenicol** | | | | 7 | 256 | |
| **Ciprofloxacin** | S 0.5 | S 0.5 | R > 2 | I 2 | | |
| **Daptomycin** | | | > 256 | | > 256 | |
| **Ertapenem** | | | | | | |
| **Gentamicin** | R > 8 | R > 8 | R > 8 | | | |
| **Imipenem** | | | | | | |
| **Levofloxacin** | S <= 0.5 | S <= 0.5 | S 2 | S 1 | | |
| **Meropenem** | R > 8 | R > 8 | R > 8 | | R > 8 | |
| **Minocycline** | | | | 1 | | |
| **Nitrofurantoin** | | | | | | |
| **Piperacillin/ tazobactam** | S 16 | I 32 | R > 64 | S <=8 | | |
| **Rifampin** | | | | 0.25 | | |
| **Tigecycline** | | | | | | |
| **Tobramycin** | R >8 | R >8 | R >8 | | | |
| **TMP/SMX** | S 1/19 | S 1/19 | R > 2/38 | S 1/19 | | |
| **Vancomycin** | | | 16 | | 16 | |

**Table 1:** Blood Culture growth and sensitivities in MIC (Minimum Inhibitory Concentration) chronologically by hospital days.

| Date | Antibiotic Regimen |
|---|---|
| Day 1 | Aztreonam 2g IVPB stat, Vancomycin 1g IVPB stat |
| Day 2 | cefepime 1g q12h – got 1 dose; Meropenem 1g q12h started |
| Day 3 | meropenem discontinued; changed to Meropenem/vaborbactam 2g q12h |
| Day 4 | Meropenem/vaborbactam 2g q12h |
| Day 5 | Meropenem/vaborbactam 2g q12h |
| Day 6 | Ciprofloxacin 400mg IV q12h; piperacillin/tazobactam 3.375g IV q6h |
| Day 7 | Ciprofloxacin 400mg IV q12h; piperacillin/tazobactam 3.375g IV q6h |
| Day 8 | Ciprofloxacin 400mg IV q12h; piperacillin/tazobactam 3.375g IV q6h |
| Day 9 | Ciprofloxacin 400mg IV q12h; piperacillin/tazobactam 3.375g IV q6h |
| Day 10 | Ciprofloxacin 400mg IV q12h; piperacillin/tazobactam 3.375g IV q6h |
| Day 11 | Ciprofloxacin 400mg IV q12h; piperacillin/tazobactam 3.375g IV q6h, TMP/SMX 120mg IV q6h |
| Day 12 | Ciprofloxacin 400mg IV q12h; piperacillin/tazobactam 3.375g IV q6h, TMP/SMX 120mg IV q6h |
| Day 13 | Ciprofloxacin 400mg IV q12h discontinued; TMP/SMX 120mg IV q6h discontinued; Started Levofloxacin 750mg IV q24h; Started Minocycline 200mg STAT then 100mg q12h; Started Rifampin 600mg IV q24h |
| Day 14 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; TMP/SMX 260mg IV q8h |
| Day 15 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; TMP/SMX 260mg IV q8h |
| Day 16 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; TMP/SMX 260mg IV q8h |
| Day 17 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; TMP/SMX 260mg IV q8h |
| Day 18 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; TMP/SMX 260mg IV q8h |

| | |
|---|---|
| Day 19 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; TMP/SMX 260mg IV q8h |
| Day 20 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; |
| Day 21 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; |
| Day 22 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; |
| Day 23 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; |
| Day 24 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; |
| Day 25 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; |
| Day 26 | Levofloxacin 750mg IV q24h; Minocycline 100mg q12h; Rifampin 600mg IV q24h; |

**Table 2:** Antibiotic Regimen – Day of hospitalization and antibiotics regimen.

## Discussion

*Elizabethkingia Meningoseptica* is a ubiquitous, non-fermenting, non-motile, oxidase-positive, gram-negative bacillus. It was first discovered in 1959 by Elizabeth O. King as an environmental pathogen associated with neonatal meningitis outbreaks in premature infants. In the past decade, the annual incidence of *E. Meningoseptica* infection has increased from 0.01 per 1000 admissions to 0.04 per 1000 admissions,[1]. It has been identified as an emerging pathogen in hospital settings and has been isolated in hospital water supplies, medical devices, and disinfectants. A 22-month outbreak affecting 30 patients was recently reported in 2016 a London, UK critical care unit. Water was identified as the source of infection,[6].

In immunocompromised adults, *E. Meningoseptica* can cause meningitis, sepsis, and pneumonia. *Elizabethkingia* infections are associated with a mortality rate of 30% due to the multidrug resistance associated with this organism and lack of effective therapeutic regimens,[2]. A recent antibiotic exposure is usually identified and patients with hematologic malignancies and organ transplant are at particularly high risk due to their immunocompromised state,[5]. Rate of mortality was as high as 53% in patients with malignancies and diabetes mellitus.

Our patient had persistently positive blood cultures for *Elizabethkingia Meningoseptica* without any clinical symptoms of meningitis on her initial presentation. A thorough workup was unrevealing of the infection source and therefore a diagnostic lumbar puncture was performed in this patient with mild headache which revealed meningitis. It is unclear whether patient had *Elizabethkingia Meningoseptica* meningitis with bacteremia at initial presentation without manifesting the full expected symptoms of meningitis. We would recommend based on this case that the threshold of performing a diagnostic lumbar puncture in a patient with *E. Meningoseptica* bacteremia can be extremely low especially if primary focus of the infection cannot be identified

## Conclusion

*Elizabethkingia Meningoseptica* is an emerging pathogen in healthcare setting which can cause severe and life-threatening infection in immunocompromised hosts. There should be a high index of suspicion *Elizabethkingia* M*eningoseptica* among immunocompromised patients presenting with septic shock. It is a multidrug-resistant organism and treatment should be guided based on the antibiotic sensitivity pattern. Blood cultures can remain positive for up to 2 weeks and should be followed up until negative. Most of the isolates of Elizabethkingia Meningoseptica are sensitive to Levofloxacin, Ciprofloxacin, Tigecycline, Piperacillin-tazobactam and Trimethoprim- sulfamethoxazole. Antibiotic resistance can emerge during therapy. Multidrug regimen should be used and tailored to the sensitivity pattern based on repeated blood cultures. All patients presenting with Elizabethkingia Meningoseptica bacteremia should undergo meningitis workup including a lumbar puncture early on during the treatment course to tailor the antibiotics regimen and prevent multi-drug resistance. Further studies are required to establish the full spectrum of disease and natural history of this rare pathogen, and to determine optimal empiric antibiotic regimen.

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