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Surgical Neurology International
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SNI: Infection

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Case Report

Nosocomial *Elizabethkingia meningoseptica* meningitis and bacteremia in a post transsphenoidal hypophysectomy complicated with sagittal sinus thrombosis: A case report

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Received : 07 June 2022
Accepted : 27 July 2022
Published : 19 August 2022

DOI
10.25259/SNI_526_2022

Quick Response Code:

ABSTRACT

Background: *Elizabethkingia meningoseptica* meningitis is rare and challenging to manage infection. As this infection is always associated with superimposed multidrug-resistant organisms, a combination and prolonged antibiotic treatment are necessary to ensure the complete eradication of infections.

Case Description: We report successful antibiotic therapies in a patient with *E. meningoseptica* bacteremia and meningitis complicated with superimposed extreme-drug-resistant *Acinetobacter baumannii* infection in a patient post transsphenoidal hypophysectomy complicated with central venous thrombosis.

Conclusion: Antibiotic combination therapy with prolonged duration in those with *E. meningoseptica* with concomitant multi-resistant organisms is needed. Diagnosing associated prothrombotic risk with the infection and prompt treatment would also be essential.

Keywords: *Elizabethkingia meningoseptica*, Intensive care, Multidrug resistance, Neurosurgery, Sinus thrombosis

INTRODUCTION

Elizabethkingia spp. can be divided into six subtypes, and *Elizabethkingia meningoseptica* is one of them. Previously, *Elizabethkingia* species was subdivided under the *Chryseobacterium* genus. It is a Gram-negative bacillus with a characteristic of oxidase positive, nonmotile, nonfermentative, and nonsugar fermenting.[5] It is commonly found in the environment, especially in fresh water, salt water, and soil. It has never been a human flora; however, it can colonize the human body causing detrimental effects to humans. With its unique ability to produce biofilm, this bacterium is very difficult to treat as they are often resistant to our common antibiotics and consistently associated with mortality in critically ill patients. Biofilm formation also increases the ability of the bacteria to persist on various surfaces and result in meningitis through contaminated medical fluids or devices and hematogenous spread, but the exact pathogenesis is still being studied.[10] We report a case of successful treatment of simultaneous nosocomial *Elizabethkingia* meningitis and septicemia in post transsphenoidal hypophysectomy (TSH) for a pituitary macroadenoma patient.

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CASE REPORT

A 65-year-old gentleman with underlying allergic rhinitis, eczema, dyslipidemia, and benign prostatic hyperplasia initially presented with insidious onset of the left eye blurring of vision over the past 3 years which progressively worsened. On examination, the patient had left visual field quadrantanopia with no papilloedema and no other neurological deficit. MRI brain reported a pituitary macroadenoma size 2.0 cm × 1.8 cm × 2.0 with compression to the left optic chiasma. The patient's neuroendocrine workup showed low cortisol, low growth hormone, and low testosterone. Other neuroendocrine hormones were normal. He was electively admitted for TSH and excision of the tumor. Intraoperatively, the dura was not breached, and the procedure went uneventful. He was extubated postoperatively and transferred to the ward for observation. He developed polyuria on the 1st day postoperatively and was treated for cranial diabetes insipidus with desmopressin. On day 7, he was febrile, tachypneic, tachycardic, and had minimal cough with no neurological symptoms. There was no cerebrospinal fluid (CSF) rhinorrhea on examination. Air entry in his lung was equal with no adventitious sound. The chest X-ray revealed minimal perihilar haziness. White blood cell (WBC) count was 9000 cells/cm^3, and C-reactive protein was 200 mg/L, and he was started on IV Tazosin 4.5 g 6 hourly empirically for hospital acquired pneumonia. Subsequently, the patient complained of headache and neck pain on the same day. After counseling, he was indecisive about a lumbar puncture to diagnose meningitis. On day 9, the patient's Glasgow Coma Scale dropped from E4V5M6 to E3V3M5. The pupils were still 3/3 reactive but with neck stiffness with positive Kernig's and Brudzenski signs. Blood sepsis parameter showed increased WBC to 14,000 cells/cm^3 and CRP >200 mg/L. Blood culture preliminary report taken on day 7 of illness came back as *E. meningoseptica*, Carbapenam Resistance Enterobacteriaceae (CRE). On MacConkey agar, a nonlactose fermenter colony was seen, and the Gram stain showed the Gram-negative bacilli and nonmotile, and oxidative positive was identified by Vitek GN ID card. Antibiotic susceptibility testing using minimum inhibitory concentration and not disk diffusion following Clinical and Laboratory Standard Institute, CLSI M100. The patient was intubated for septic shock with meningitis and transferred to the neurocritical care unit for intensive care management. He was started on IV Meropenem 2 g TDS and IV Ciprofloxacin 400 mg TDS. On day 9, lumbar puncture and the CSF results were reported as clear and yellowish, total WBC increased 10 cells/mm^3 with predominant polymorphs cell, RBC was nil glucose 2.2 mmol/L, and protein 0.57 g/L with positive culture *E. meningoseptica* CRE. CSF lactate dehydrogenase was not performed. The patient was diagnosed with bacteremia and meningitis due to positive clinical signs and multiple isolations of *E. meningoseptica* from the blood and CSF cultures and biochemistry profiles. Subsequently, the patient did not improve neurologically post antibiotics despite improvement of the septic parameters and was subjected to a contrast-enhanced CT brain. On assessment of the CT scan, the patient developed short-segment superior sagittal sinus thrombosis. The patient was started on SC clexane 80 mg BD. On day 13, the tracheal aspirate culture and sensitivity grew *Acinetobacter baumannii* Extreme Drug-Resistant (XDR). IV Polymyxin B 750000 U BD was added for this patient to cover the CRE and XDR. The patient completed IV Polymyxin for 14 days and IV Meropenem for 14 days. IV Ciprofloxacin was completed for 14 days and followed by 14 days of T. Levofloxacin 750 mg BD. The patient's condition markedly improved, and he was extubated well on day 17. The course of culture and sensitivity and antibiotics therapy is simplified in Figure 1. On repeated CTV Brain, the venous sinus thrombosis was resolving. The patient's septic parameter normalized to WBC 7000 cells/cm^3, and CRP was negative. The patient was seen in a follow-up clinic after hospital discharge will full neurological recovery.

DISCUSSION

Elizabethkingia species are oxidase-positive, nonmotile, nonfermentative, and nonglucose fermenting Gram-negative aerobic bacilli, and *E. meningoseptica* is one of the medically important species.[5] It is a common saprophyte found in the soil environment, saltwater, and fresh water and even survives in chlorine-treated water supplies. In hospitals, sink basins and taps are potential reservoirs, and transmission to patients occurs through the contaminated fluid of medical devices including respirators, intubation tubes, humidifiers, incubators, and even antiseptic and saline solutions.[7] It can spread by wet and dry materials and surfaces, including the hands of hospital staff.[2]

Elizabethkingia spp. is an emerging nosocomial pathogen. It is known that *E. meningoseptica* colonized the human oropharynx, respiratory secretions, aerosol tubes, endotracheal tubes, and the respiratory tract in ventilated adult patients.[1] Those at risk include patients undergoing medical and surgical interventions with usages of an indwelling central venous catheter and other invasive medical devices, patients showing immunosuppression, prolonged hospital stay, and exposure to multiple broad-spectrum antibiotics.[4] Most of the reported cases involved neonatal meningitis.[4] Tak *et al.* described a case of *E. meningoseptica* meningitis in a patient with post traumatic brain injury.[9]

A possible explanation for the high mortality rate may be its potential to form biofilms. The biofilms produced by biofilm-forming pathogens, like *Elizabethkingia* spp., might decrease the immune response and increase resistance against antibiotics, resulting in a high mortality rate. In addition, prolonged antibiotic treatment regimens are necessary to

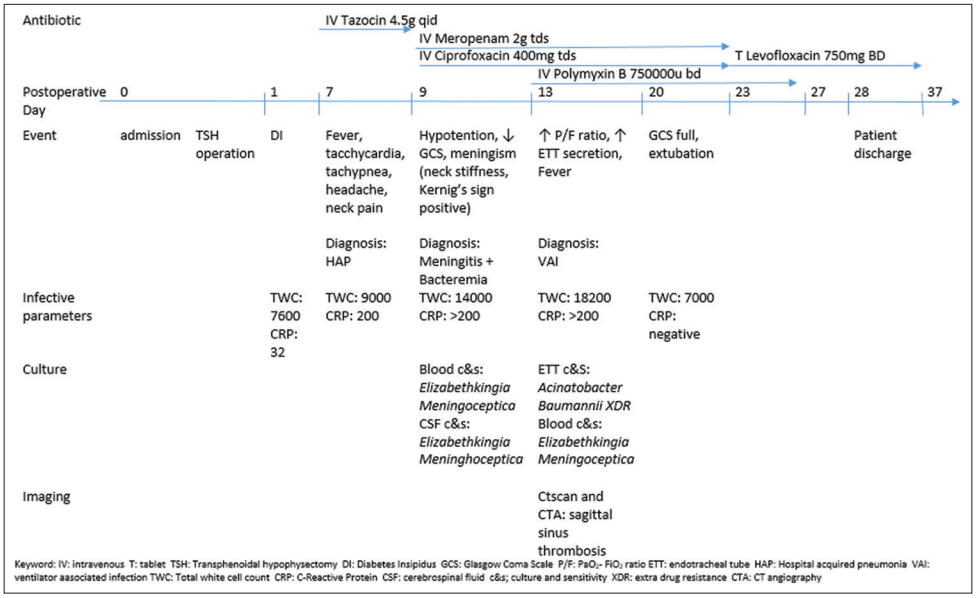

Figure 1: Clinical course of symptoms, culture, and antibiotic therapy for the patient. IV: Intravenous, T: tablet, TSH: transphenoidal hypophysectomy, DI: diabetes insipidus, GCS: Glasgow Coma Scale, P/F: PaO_2-FiO_2 ratio, ETT: endotracheal tube, HAP: hospital-acquired pneumonia, VAI: ventilator-associated infection, TWC: total white cell count, CRP: C-reactive protein, CSF: cerebrospinal fluid, c&s: culture and sensitivity, XDR: extra drug resistance, CTA: CT angiography.

eradicate the pathogen successfully. Moreover, it is known that *Elizabethkingia* spp. has virulence factors, including intracellular invasion and chromosomal and plasmid-mediated resistance to many antimicrobial drugs.[6] For example, *E. meningoseptica* possesses two types of β-lactamases (intrinsic class A extended-spectrum serine-β-lactamases and inherent class B Metallo-β-lactamases), which makes it resistant to a broad range of antimicrobials that are routinely used for empiric treatment of infections caused by Gram-negative bacteria.[3] Therefore, *E. meningoseptica* is generally resistant to carbapenems, extended-spectrum cephalosporins, aminoglycosides, aztreonam, and colistin. There is no antimicrobial regimen of choice for empiric treatment of *Elizabethkingia* spp. infections, as antimicrobial susceptibilities have been inconsistent across reports.[8] Interestingly, *E. meningoseptica* is often susceptible to agents generally used to treat infections caused by Gram-positive bacteria, such as rifampicin, clindamycin, erythromycin, trimethoprim-sulfamethoxazole, quinolones, and vancomycin.

Our patient's recognizable risk factors for *E. meningoseptica* are underlying hypocortisolism and prolonged intensive care and hospital stay. Furthermore, the superimposed multi-resistant organism with *Elizabethkingia* is common, making it more complicated for antibiotic management. Judicious use of antibiotic combination therapy with the frequent assessment of the patient clinical condition is necessary. The antibiotic of choice must have good blood barrier penetration in patients with meningitis. In our patient, since the patient also contracted XDR infection, IV Polymyxin B was also started in an antibiotic combination. Prolonged antibiotic therapy is also necessary due to the formation of biofilm. That is why we continued with oral Levofloxacin after the completion of IV ciprofloxacin. Although the mortality rate with *Elizabethkingia* is reported to be more than 50%, frequent assessment and initiation of correct antibiotic regimes may improve the patient's outcome. Moreover, since one of the virulence factor genes in *Elizabethkingia* contains phospholipase C, we postulate that the incidence of central venous thrombosis in this patient might also be contributed by the prothrombotic property of this infection.[10]

CONCLUSION

Nosocomial *E. meningoseptica* is rare but difficult to manage and often requires antibiotic combination therapy with prolonged duration, especially in those who have superimposed multi-resistant organisms. Incidence of central venous thrombosis might also be contributed by the infection necessitating prompt therapy of antithrombotic agents and optimization of intravascular volume.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Balakrishnan NK, Ghani AR, Hassan MH, Mukmin LA, Hassan WM. Nosocomial *Elizabethkingia meningoseptica* meningitis and bacteremia in a post transsphenoidal hypophysectomy complicated with sagittal sinus thrombosis: A case report. Surg Neurol Int 2022;13:370.