Letter to the Editor

A phylogenetic study of Elizabethkingia anophelis bloodstream isolates obtained from inpatients at a single medical center

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To the Editor—Elizabethkingia anophelis is a rapidly emerging nosocomial pathogen reported to cause bacteremia in immune-compromised elderly people and neonates.1,2 The unknown pathogenesis and unclear resistance mechanism of E. anophelis and their phenotypic similarity to E. meningoseptica mislead and complicate the infection management of this pathogen, resulting in treatment failure. Inherent resistance to multiple classes of drugs and absence of an antibiotic sensitivity profile standard for this bacterium makes empirical treatment nearly impossible. Elizabethkingia anophelis bacteremia has recently been considered clinically significant, leading to high morbidity and mortality that has been mistakenly attributed to E. meningoseptica because of their phenotypic similarity.3,4 Molecular epidemiological analyses of recent Elizabethkingia bacteremia infections and outbreaks have been conducted in United States, Singapore, China, and Korea. These outbreaks were predominated by E. anophelis.3,5 This finding warrants the implementation of molecular typing for an accurate diagnosis to guide appropriate antibiotic regimen instead of relying solely on conventional phenotypic identification with a compact automated VITEK-2 system, which uses a factory default database and lacks timely amendments.3,5

In the first report of an outbreak in a tertiary healthcare center of Eastern India, the clinical and molecular epidemiology of 9 bacteremia episodes during 2 months of surveillance from August to September 2017 were identified as E. meningoseptica by the VITEK-2 system. These findings were genetically validated by species-specific markers, such as lipid-A disaccharide synthase gene for E. anophelis and sodium-proton antiporter for E. meningoseptica,5 and 16s rRNA gene sequencing. An antibiotic susceptibility study was conducted using the VITEK-2 compact automated system (BioMerieux) with the GN-AST-N280 card. Sensitivity was interpreted according to Clinical Laboratory Standards Institute (CLSI) guidelines (2013).3,7 The clonal relatedness among 9 isolates was investigated using repetitive-element polymerase chain reaction (rep-PCR) and (GTG)5 PCR according to the method described by Adiguzel et al.8

Nonrepeated Elizabethkingia spp (EA1-9) were isolated from 9 inpatients, and we analyzed the demographic data, clinical characteristics, and outcomes for these cases (Table S1 online). In these 9 cases, Elizabethkingia bacteremia prevailed mostly among elderly people (n=8; median age 52 years), but 1 patient was a 2-year-old child. The male: female ratio among these patients was 7:2 (Table S1 online). Overall, 5 Elizabethkingia isolates were obtained from blood; the rest were obtained from tracheal aspiration (n = 3) and cerebrospinal fluid (n = 1). All of these patients were reported as having hospital-acquired, clinically significant bacteremia, with a high mortality rate (33.3%). Of these 9 patients, 3 died within 1 month of their hospital stay despite treatment with antibiotics (eg, quinolones, penicillin, cephalosporins, carbapenems, etc, either alone or in combination) due to several associated complications: pneumonia, lower respiratory infection, meningitis, acute kidney injury, and metabolic encephalopathy, etc. These isolates showed resistance to different groups of antibiotics with varying percentages ranging from ~80% to 100% (Table S2 online). However, the highest susceptibility was found against tigecycline and piperacillin-tazobactam, which corroborates the previous reports3,4,9 except isolate EA1, which matched a single study from China.6 The resistance profile against levofloxacin was analogous to isolates identified in Korea and Wisconsin.3,4 However, the alteration of the antibiotic resistance profile depends generally on different types of stress on different sources of Elizabethkingia isolates.6

All 9 isolates were identified as E. meningoseptica by the VITEK-2 compact automated system. Because the identification of Elizabethkingia spp has been reported to be misleading using the VITEK 2 and MALDI-TOF MS systems,2 these samples were subjected to genotypic validation. However, upgrading the VITEK-2 system with better antibiotic sensitivity profiles, updating the CLSI guidelines, and expanding the database for MALDI-TOF mass spectra of E. anophelis will improve their proper identification. All 9 Elizabethkingia spp showed amplification of lipid-A disaccharide synthase gene, a species-specific primer of E. anophelis, and were further confirmed to be E. anophelis by 16s rRNA gene sequence analysis (GenBank accession no: MH121154-MH121158, MN038050-MN0380053). Rep PCR- and (GTG)5 PCR–based phylogenetic analysis of 9 isolates revealed a close clustering of EA1 and EA2 with EA4; EA6 with EA8; and EA5 with EA7. These findings explain the considerable clonal similarity among E. anophelis isolates belonging to the same in-patient departments (Fig. 1).

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This study is the first molecular epidemiological report on a bacteremia outbreak of India with prevalence of *E. anophelis* bacteria establishing *E. meningoseptica* to be the more remote cause of bacteremia infection. However, future prospective studies with population-based data over longer surveillance periods should be performed to determine the prevalence and incidence of *E. anophelis* bacteremia. A repeated molecular epidemiological study should be employed for accurate diagnosis and appropriate treatment regimen.

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**Supplementary material.** To view supplementary material for this article, please visit https://doi.org/10.1017/ice.2019.213

**References**


