DOI: 10.7860/JCDR/2026/82833.23460

Original Article

Microbiology Section

Clinico-epidemiological Profile, Risk Factors, and Outcomes of *Elizabethkingia* Infections: A Retrospective Observational Study

NIDHI YADUVANSHI[1], CHINMOY SAHU[2], KANCHAN KUMARI[3], SANGRAM SINGH PATEL[4]

ABSTRACT

Introduction: *Elizabethkingia* spp. is an emerging nosocomial pathogen that primarily causes bloodstream infections, pneumonia, and sepsis, particularly in critically-ill individuals. This organism is typically resistant to commonly used antibiotics for Gram-negative infections and is associated with significant morbidity, mortality, and prolonged hospital stays.

Aim: To evaluate the clinicoepidemiological profile, risk factors, species identification, antimicrobial susceptibility pattern, and outcome of infections caused by *Elizabethkingia* species.

Materials and Methods: This was a retrospective laboratory-based observational study conducted in the Department of Microbiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS), Lucknow, Uttar Pradesh, India, over a one-year period from June 2024 to June 2025. All clinical samples {blood, Cerebrospinal Fluid (CSF), Endotracheal (ET) aspirate, Bronchoalveolar Lavage (BAL) and urine} showing pure growth of *Elizabethkingia* species were included. A total of 48 non duplicate *Elizabethkingia* isolates obtained from individual patients were included. Identification was initially performed using the VITEK-2 automated system and subsequently confirmed by Matrix-Assisted Laser Desorption/Ionisation Time-of-Flight Mass Spectrometry (MALDI-TOF MS). Antimicrobial susceptibility testing was carried out by–Kirby-Bauer disk diffusion method. Demographic and clinical data (age, sex, diagnosis, co-morbidities, risk factors, and outcome) were retrieved from patient records. Statistical analysis was done using Statistical Package for the Social Sciences (SPSS) version 14.0; results were expressed as n (%), and Chi-square test was applied, considering p-value<0.05 significant.

Results: A total of 48 patients were positive for *Elizabethkingia* spp., comprising 39 adults (81.3%) and nine neonates (18.7%). Respiratory infections predominated among adults, 64.1% (25/39), whereas meningitis and sepsis were the main presentations in neonates, 88.9% (8/9), including five cases of meningitis (55.6%) and three cases of meningitis with sepsis (33.3%). Both *E. meningoseptica* and *E. anophelis* isolates showed 100% susceptibility to minocycline and piperacillin–tazobactam. Overall, favourable outcomes were observed in 83.3% of patients, with mortality in 16.7%, primarily due to meningitis and sepsis.

Conclusion: The present study highlights the emergence of *E. meningoseptica* and *E. anophelis* as significant multidrug-resistant pathogens causing severe infections in both adult and neonatal patients. The findings emphasise the importance of rapid and accurate species identification using MALDI-TOF MS to guide appropriate therapy. Early diagnosis and targeted treatment were associated with improved patient outcomes. Strengthening infection control practices and continuous antimicrobial surveillance are vital to prevent hospital transmission and ensure optimal management of these multidrug-resistant pathogens.

Keywords: Antibacterial agents, Bactearemia, Intensive care units, Mass spectrometry, Sepsis

INTRODUCTION

The *Elizabethkingia* genus is widespread and commonly found in environmental sources such as soil and water [1]. The bacterium is aerobic, Gram-negative bacilli, non fermenting, non motile, and both catalase and oxidase positive within the family Flavobacteriaceae [2]. To date, seven species of the genus *Elizabethkingia* have been validly described and recognised. These include *E. meningoseptica*, *E. miricola*, *E. anophelis*, *E. bruuniana*, *E. ursingii*, *E. occulta*, and *E. argenteiflava* [3]. Amongst species, *E. meningoseptica* and *E. anophelis* are most frequently implicated in healthcare associated infections (HAIs). They are associated with a range of serious conditions, including pneumonia, bloodstream infections, sepsis, urinary tract infections, surgical site infections, and meningitis [4,5].

Infections from this bacterium typically occur in hospital settings, especially among ICU patients on ventilators or those with medical devices such as central lines or indwelling urinary catheters [6,7]. Additional risk factors for *Elizabethkingia* infection include prolonged antibiotic therapy, extended hospital stays, long-term immunosuppressive treatment, and pre-existing co-morbidities that heighten the risk of infection and contribute to increased morbidity and mortality [8,9].

Elizabethkingia infections are particularly difficult to manage due to their resistance to many antimicrobial classes commonly used for Gram-negative bacteria, including β-lactams (penicillins, cephalosporins, and carbapenems), fluoroquinolones, aminoglycosides, macrolides, and even colistin. This leaves clinicians with very limited effective therapies. However, these pathogens often show susceptibility to minocycline, and in many isolates, to piperacillin-tazobactam and trimethoprim-sulfamethoxazole, providing some viable treatment options [10,11]. They are inherently multidrug resistant due to their ability to produce both Extended-Spectrum β-Lactamases (ESBLs) and Metallo-β-Lactamases (MBLs). The primary genes involved are bla_CME (encoding ESBL enzyme) and two chromosomal MBL genes, BlaB and bla_GOB [12,13].

Distinguishing *E. meningoseptica* from *E. anophelis* has been challenging when using routine conventional methods or automated systems, such as VITEK 2. As a result, many clinical isolates that were initially reported as *E. meningoseptica* were later confirmed to be *E. anophelis*. Accurate species-level identification requires MALDI-TOF MS with an extended or updated reference database for reliable differentiation [14]. Although *Elizabethkingia* species have emerged as important opportunistic pathogens in critically-ill and immunocompromised patients. Current literature remains limited,

largely comprising isolated case reports or small case series, with scarce data from India [4,15,16].

The organism exhibits intrinsic resistance to multiple antibiotic classes, making timely identification and appropriate therapy challenging. Given the scarcity of regional data and the increasing clinical relevance of *Elizabethkingia* infections in hospital settings.

This study aimed to evaluate the clinicoepidemiological profile and outcome of infections caused by *Elizabethkingia* species.

The primary objective of this study was to analyse the antimicrobial susceptibility patterns of *Elizabethkingia* isolates and to assess clinical outcomes, including morbidity and mortality, among affected patients. The secondary objectives were to identify associated risk factors and underlying co-morbidities in patients with *Elizabethkingia* infections. Also, to evaluate species-level distribution and resistance trends of *Elizabethkingia* isolates.

MATERIALS AND METHODS

This was a retrospective laboratory-based observational study conducted in the Department of Microbiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India, over a one-year period from June 2024 to June 2025, during which data were collected, and data analysis was performed from July 2025 to September 2025. The study was approved by the Institute Ethics Committee (IEC No. 2021-48-EMP-EXP, dated November 29, 2021).

As this was a retrospective record-based study, the requirement for informed consent was waived. A total of 48 clinical isolates were included. The sample size was not calculated statistically; rather, all consecutive, non duplicate *Elizabethkingia* isolates obtained during the study period were included, making it a time-bound study.

Inclusion criteria: All clinical specimens yielding pure growth of *Elizabethkingia* species were included. Patients of all age groups were eligible. Patients were categorised according to clinical conditions such as Ventilator-Associated Pneumonia (VAP), defined as pneumonia developing more than 48 hours after ET intubation or within 48 hours after extubation [17], and Central Line-Associated Bloodstream Infection (CLABSI), defined as a laboratory-confirmed bloodstream infection with *Elizabethkingia* isolated from both central and peripheral blood cultures in a patient with a central line for ≥48 hours, with no other identifiable source [18].

Exclusion criteria: Clinical specimens yielding organisms other than *Elizabethkingia* species were excluded from the study.

Study Procedure

The data on demographic characteristics, risk factors, associated co-morbidities/clinical conditions, pathogen isolated, antimicrobial susceptibility profile, prior hospitalisation, antibiotic prescription and outcome of the patients were collected from Hospital Information System (HIS) and laboratory record registers. Also, data were extracted from case sheets of admitted patients, including duration of hospital stay, length of Mechanical Ventilation (MV), length of pre-Intensive Care Unit (ICU) admission, length of ICU admission and diagnostic parameters, specifically Total Leukocyte Count (TLC) and Procalcitonin (PCT) assay. A normal reference range for TLC was considered as 4,000-11,000 cells/mm^3, and for PCT, levels of <0.5 ng/mL were taken as within normal limits. Values above these cut-offs were interpreted as elevated, suggestive of systemic infection or inflammation [19,20].

Samples processing: All clinical specimens received in the microbiology laboratory during the study period were processed according to standard protocols. These included ET aspirates, BAL fluids, blood, CSF, and urine samples that yielded growth of *Elizabethkingia* species. For respiratory samples, ET aspirate and BAL fluid samples were first subjected to Gram staining. Each specimen was then cultured on Sheep blood agar, MacConkey agar, and chocolate agar plates. These plates were incubated at 37°C for 24-72 hours as per standard laboratory protocol. For semiquantitative culture, a calibrated loop was used to inoculate the samples. If bacterial growth extended beyond the tertiary streak on the plate, it was interpreted as ≥10^5 colony-forming units per millilitre (cfu/mL). A colony count of ≥10^5 cfu/mL for ET aspirate, and ≥10^4 cfu/mL for BAL fluid, was considered significant, as per established quantitative culture criteria [21]. The criteria for BAL fluid have also been described previously [22]. Also, counts below these levels were considered as colonisation or contamination.

The CSF samples were first subjected to direct microscopic examination by using wet mount and Gram staining, and cultured on Sheep blood, MacConkey, chocolate agar and then inoculated in Brain Heart Infusion (BHI) broth for subculture to look for fastidious organisms. CSF samples were incubated for 72 hours before reporting the samples as sterile or showing positive growth. For blood culture, 8-10 mL of whole blood from adult patients was inoculated directly into paired BD BACTEC Aerobic and Anaerobic bottles, while in paediatric cases, 2-3 mL of blood was placed into a paediatric Peds Plus bottle. The inoculated bottles were transported to the bacteriology laboratory and loaded into the BD BACTEC automated continuous monitoring system (Bact/Alert, bioMérieux, Marcy l'Étoile, France). Once a bottle "flagged" positive, a Gram stain was performed directly from the broth, and in parallel, the broth was streaked onto both Sheep blood agar and MacConkey agar plates. These plates were incubated at 35-37°C for 24-48 hours. If a bottle did not flag within five days of incubation, it was considered culture negative and reported as such. All the culture media used were obtained from HIMEDIA Laboratories (Mumbai, India).

Identification of isolates: The isolates were initially identified using gram staining, colony morphology characteristics, and standard biochemical tests such as oxidase, catalase, motility, urease, nitrate reduction, indole, citrate utilisation, mannitol fermentation, esculin hydrolysis, gelatin hydrolysis, and Triple Sugar Iron (TSI) agar reaction. Non fermenting, Gram-negative bacilli that were catalase and oxidase positive, non motile, showing an alkaline/alkaline (K/K) reaction on TSI agar, negative for mannitol and urease, weakly positive for indole, unable to utilise citrate as a sole carbon source, positive for esculin and gelatin hydrolysis, and negative for nitrate reduction were provisionally identified as *Elizabethkingia* species. Further identification was performed using the VITEK 2 system, and the results were confirmed through MALDI-TOF MS.

Antibiotic Susceptibility Tests (AST): Routine antibiotic susceptibility tests were performed by Kirby-Bauer disk method following Clinical and Laboratory Standards Institute (CLSI)-recommended procedures (CLSI M100, 34th edition, 2024) [23], using commercially available antibiotic discs (HiMedia, Mumbai, India). Minimum inhibitory concentrations (MIC) values generated by the VITEK-2 system and zone diameters obtained by disc diffusion were interpreted cautiously with reference to CLSI recommendations for other non Enterobacteriaceae, as organism-specific interpretive criteria for *Elizabethkingia* species are not available. Multidrug resistant organism (MDR) was recognised in an isolate when it was not susceptible to one antibiotic agent from at least three or more classes of antibiotics. Isolates not found susceptible to at least one agent in two or fewer antimicrobial categories were deemed extremely drug-resistant [24].

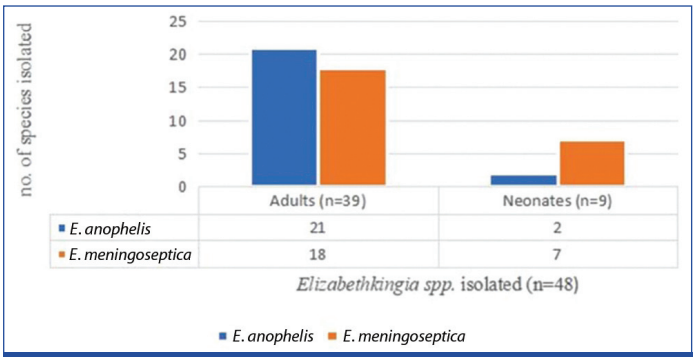
STATISTICAL ANALYSIS

Statistical analyses were performed using SPSS software for Windows, version 14.0 (SPSS Inc., Chicago, IL, USA). Categorical variables were summarised as numbers and percentages {n (%)}. Comparisons between categorical variables were performed using

the Chi-square test or Fisher's exact test, as appropriate. Continuous variables were compared using the Mann-Whitney U test. A p-value ≤ 0.05 was considered statistically significant.

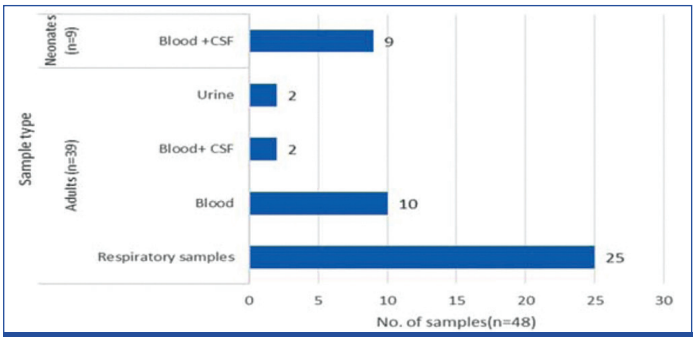
RESULTS

In this study, a total of 48 culture-positive *Elizabethkingia* isolates were included. Among the study population, 39 (81.25%) were adults and 9 (18.75%) were neonates [Table/Fig-1].



[Table/Fig-1]: Shows the distribution of *Elizabethkingia* spp. isolated in the study population (n=48).
The X-axis shows *Elizabethkingia* spp. isolated (*E.anophelis* and *E. meningoseptica*). The y-axis shows the number of species isolated in adults and neonates

In the overall study population (n=48), the majority of isolates were obtained from respiratory specimens (25/48, 52.1%), followed by blood samples (10/48, 20.8%), combined blood and CSF samples (2/48, 4.2%), and urine samples (2/48, 4.2%). In neonates, all isolates were obtained from combined blood and CSF samples (9/48, 18.8%), as shown in [Table/Fig-2].



[Table/Fig-2]: Shows distribution of samples among study group (n=48).
The X-axis shows number of samples. The y-axis indicates the type of samples

The mean age among adults was 55.5±9.9 years, while that among neonates was 9.6±2.1 days. A male predominance was observed in both groups 27 (69.2%) among adults and 5 (55.6%) among neonates - which was not statistically significant (p-value=0.46) [Table/Fig-3].

Of the total 48 isolates, MALDI-TOF MS identified 25 (52.1%) as *Elizabethkingia meningoseptica* and 23 (47.9%) as *Elizabethkingia anophelis*. All 25 *E. meningoseptica* isolates were correctly identified by the VITEK-2 system. However, among the 23 *E. anophelis* isolates, 20 were misidentified as *E. meningoseptica* and 3 as *Chryseobacterium indologenes* by VITEK-2, underscoring the limitations of conventional automated systems in accurate species-level identification.

The laboratory parameters and outcome of all patients are presented in [Table/Fig-4].

Parameter	Adults (n=39)	Neonates (n=9)
Age distribution		
Mean±SD (range)	55.5±9.9 years	9.6±2.1 days
Sex distribution		
Male	27 (69.2%)	5 (55.6%)
Female	12 (30.8%)	4 (44.4%)

Clinical diagnosis	LRTI (n=13) 33.33%	Meningitis (n=5) 55.56%
	VAP (n=8) 20.51%	
	Sepsis (n=7)17.95%	
	LRTI, VAP (n=4) 10.25%	Meningitis , Sepsis (n=3) 33.33%
	Sepsis, CLABSI (n=3) 7.69%	
	Sepsis, Meningitis (n=2) 5.13%	Sepsis (n=1)11.11%
	CAUTI (n=2) 5.13%	
Underlying co-morbidities	COPD (n=9) 23.07%	Prematurity (n=5) 55.56%
	DM, HTN (n=9) 23.07%	
	CLD (n=6) 15.38%	
	CKD (n=4) 10.25%	
	ALL (n=3) 7.69%	Anaemia, Jaundice, Prematurity (n=2) 22.22%
	Lung cancer (n=2) 5.12%	
	AML (n=2) 5.12%	
	Liver failure, cirrhosis (n=2) 5.12%	Low birth weight (n=2) 22.22%
	Liver cancer (n=1) 2.56%	
	Gallbladder cancer (n=1) 2.56%	
Risk factors	Pulmonary infections (n=25) 64.10%	MV/Intubation (n=3) 33.33%
	MV/ Intubation (n=12) 30.76%	Parenteral nutrition (n=3) 33.33%
	Foley's catheter (n=11) 28.20%	Central lines (n=2) 22.22%
	Chemotherapy/Immunosuppression (n=9) 23.07%	Umbilical catheters (n=1) 11.11%
	Haemodialysis (n=4) 10.25%	
	Blood transfusion (n=4) 10.25%	
	Central venous catheter (n=3) 7.69%	
	Organ transplant (n=2) 5.12%	

[Table/Fig-3]: Demographic and clinical characteristics of study group (n=48).
SD: Standard deviation; LRTI: Lower respiratory tract infection; COPD: Chronic obstructive pulmonary disease; VAP: Ventilator associated pneumonia; CLABSI: Central line associated blood stream infection; DM: Diabetes mellitus; HTN: Hypertension; MV: Mechanical Ventilation; CLD: Chronic Liver disease; CKD: Chronic kidney disease; ALL: Acute lymphoblastic leukaemia; AML: Acute myeloblastic leukaemia; UTI: Urinary tract infection

Parameter	Adults (n=39)	Neonates (n=9)	Test statistic	p-value	Odds ratio (95%CI)
Laboratory parameters					
Procalcitonin (ng/mL) Mean ±SD (range)	1.82±0.59 (1.0-2.9)	2.24±0.29 (1.8-2.6)	-	0.0046*	-
Leukocyte count (cells/ mm^3) Mean±SD (range)	15,228±894 (13,800-16,900)	16,711±342 (16,100-17,200)	-	<0.0001*	-
Other parameters					
Length of stay in hospital (days) Mean±SD (range)	24.77±3.14 (20-30)	16.71±3.42 (12-22)	-	<0.0001*	-
Length of MV (days) Mean±SD (range)	21.11±1.12 (19-23)	14.21±1.32 (12-16)	-	<0.0001*	-
Length of ICU admission (days) Mean ±SD (range)	17.31±1.43 (15-20)	11.12±1.21 (9-13)	-	<0.0001*	-
Length of Pre ICU admission (days) Mean ±SD (range)	15.45±1.32 (13-18)	9.21±1.54 (7-12)	-	<0.0001*	-
Days from admission to development of infection (days) Mean±SD (range)	11.54±2.04 (8-15)	28.33±1.41 (26-30)	-	<0.0001*	-

Outcome					
Recovered (n=40),83.3%	33 (84.6%)	7 (77.8%)	χ^2=0.14	1.000	1.57 (0.23–10.7)
Death (n=8),16.7%	6 (15.4%)	2 (22.2%)	χ^2=0.14	1.000	1.57 (0.23–10.7)

[Table/Fig-4]: Shows laboratory parameters and outcome among the study group (n=48).
The p-values for continuous variables were calculated using the Mann-Whitney U test. The p-values for categorical variables were calculated using the Chi-square test (χ^2value) or Fisher's-exact test; *p-value ≤0.05 is statistically significant; SD: Standard deviation; MV: Mechanical ventilation.

Of the 48 patients, 36 (75%) initially received empirical broad-spectrum antibiotics, most commonly carbapenems, colistin, or fluoroquinolones. Following species identification and susceptibility testing, targeted therapy (mainly minocycline or piperacillin–tazobactam) was initiated in 42 patients (87.5%). Among these, 35 (83.3%) recovered, while 7 (16.7%) died. In contrast, among patients who did not receive targeted therapy, three out of 6 (50%) recovered and 3 (50%) died. Although this difference was not statistically significant (p-value=0.09), patients who received early targeted therapy had a trend toward better survival outcomes.

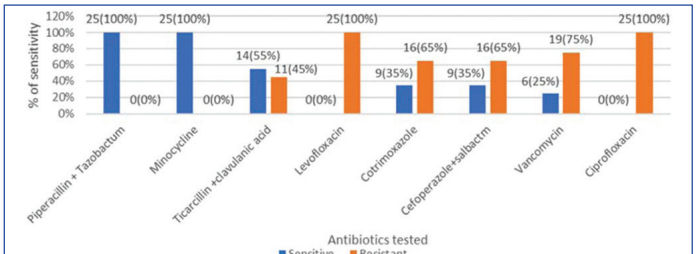
Among adults who had used antibiotics in the past month, the most common class was cephalosporins, 17 (43.6%), followed by carbapenems at 9 (23.1%), aminoglycosides 5 (12.8%), tetracyclines 3 (7.7%), and cotrimoxazole at 2 (5.1%) as depicted in [Table/Fig-5].

Antibiotic class	n (%)
Cephalosporins	17 (43.6)
Carbapenems	9 (23.1)
Aminoglycosides	5 (12.8)
Tetracyclines	3 (7.7)
Fluoroquinolones	3 (7.7)
Cotrimoxazole	2 (5.1)

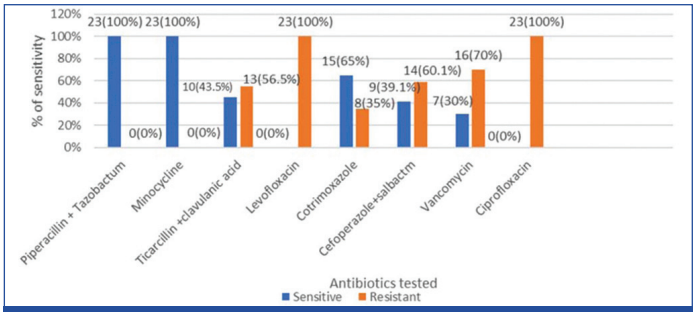
[Table/Fig-5]: Prior use of different antibiotic classes among adult patients (n=39).

The antibiotic sensitivity profile shows that both *E. meningoseptica* (n=25) and *E.anophelis* (n=23) isolates were 100% sensitive to piperacillin-tazobactam and minocycline. On the other side, they were 100% resistant to levofloxacin and ciprofloxacin. In case of *E. meningoseptica*, 75% of the isolates demonstrated resistance to vancomycin, while 65% were resistant to cefoperazone/sulbactam and cotrimoxazole. Additionally, 45% showed resistance to ticarcillin/clavulanic acid. For *E. anophelis*, 70% of the isolates were resistant to vancomycin, 60.1% to cefoperazone/sulbactam, 35% to cotrimoxazole, and 56.5% to ticarcillin/clavulanic acid as illustrated in [Table/Fig-6,7]. Using the VITEK-2 automated system, piperacillin-tazobactam demonstrated MIC values ranging from 4-16 µg/mL, with an MIC_{50} of 8 µg/mL and an MIC_{90} of 16 µg/mL, while minocycline showed MIC values of 0.25-2 µg/mL, with an MIC_{50} of 1 µg/mL and an MIC_{90} of 2 µg/mL.

In 25 adult patients, empirical therapy included piperacillin-tazobactam, meropenem, cefepime, and vancomycin. Definitive therapy in these



[Table/Fig-6]: Antibiotic sensitivity profile of *Elizabethkingia meningoseptica* isolates (n=25).
X-axis represents various antibiotics tested for *Elizabethkingia meningoseptica* isolates. Y-axis represents percentage of sensitivity (Sensitive or Resistant) among isolates tested



[Table/Fig-7]: Antibiotic sensitivity profile of *Elizabethkingia anophelis* isolates (n=23).
X-axis shows various antibiotics tested for *Elizabethkingia anophelis* isolates. Y-axis shows percentage of sensitivity (Sensitive or Resistant) among isolates tested

patients consisted of cefoperazone-sulbactam, and vancomycin. In 12 adult patients, empirical therapy involved ceftriaxone, meropenem, and vancomycin, followed by definitive therapy with cotrimoxazole. In two patients, empirical therapy consisted of ceftriaxone, piperacillin-tazobactam, and nitrofurantoin, while definitive therapy included cotrimoxazole as shown in [Table/Fig-8].

Clinical diagnosis	Empirical antibiotic	Post-AST antibiotic	Number of patients n=39 (%)
LRTI, Sepsis, CLABSI, Meningitis	Piperacillin-tazobactam, meropenem, cefepime, vancomycin	Minocycline, piperacillin-tazobactam, cefoperazone-sulbactam, vancomycin	25 (64.10%)
VAP	Ceftriaxone, meropenem, vancomycin	Minocycline, piperacillin-tazobactam, cotrimoxazole	12 (30.77%)
CAUTI	Ceftriaxone, piperacillin-tazobactam, nitrofurantoin	Minocycline, piperacillin-tazobactam, ticarcillin-clavulunate	2 (5.13%)

[Table/Fig-8]: Shows empirical antibiotic therapy and definitive therapy administered to adult patients (n=39).
LRTI: Lower respiratory tract infection; CLABSI: Catheter-associated urinary tract infection; VAP: Ventilator-associated pneumonia; CAUTI: Catheter-associated bloodstream infection.

Amongst five neonates, empirical therapy was vancomycin with cefotaxime/meropenem, later changed to cotrimoxazole. In two neonates, ampicillin + gentamicin was switched to ticarcillin–clavulanate. In another two, ampicillin + cefotaxime was changed to cefoperazone-sulbactam, and vancomycin. Piperacillin–tazobactam and minocycline was part of definitive therapy in all neonates and adult patients.

DISCUSSION

Infections caused by *Elizabethkingia* species are uncommon but clinically significant, as these emerging pathogens exhibit intrinsic resistance to many antibiotics commonly used against Gram-negative bacteria. Reports from various regions have described diverse clinical presentations, including meningitis, sepsis, Central line associated blood stream infection (CLASBI), VAP, Catheter Associated Urinary Tract Infection (CAUTI), osteomyelitis, endocarditis, and keratitis. The ability of *E. meningoseptica* to persist in chlorinated water and hospital plumbing makes it an important nosocomial pathogen, with transmission through contaminated medical devices or fluids such as ventilators, humidifiers, and intubation tubes [3,25,26].

In the present study, MALDI-TOF MS identified *E. meningoseptica* and *E. anophelis* in nearly equal proportions. Conventional systems like VITEK-2 misidentified most *E. anophelis* isolates, a limitation also highlighted by Lin JN et al., [14]. This misidentification has important clinical implications, as minor differences in antimicrobial susceptibility between the two species can influence therapeutic choices. Such errors may delay administration of effective agents, leading to suboptimal treatment and poorer outcomes. Moreover, failure to correctly identify *E. anophelis* can obscure its true epidemiological burden and hinder infection-control measures.

Hence, reliable species-level identification using MALDI-TOF MS or molecular assays is crucial for appropriate management and surveillance [14].

Both *E. meningoseptica* and *E. anophelis* isolates in the present study showed 100% susceptibility to minocycline and piperacillin-tazobactam. These findings are in agreement with earlier reports from Asia and other regions [8,10,12]. Colistin and carbapenems, commonly used as empirical agents for multidrug-resistant Gram-negative infections, showed poor activity against *Elizabethkingia* species.

The present findings indicate that the timely initiation of targeted therapy following accurate species identification contributed to improved clinical outcomes. Patients who received appropriate therapy with minocycline or piperacillin–tazobactam after MALDI-TOF confirmation showed a higher recovery rate compared to those maintained on empirical regimens. Although the difference did not reach statistical significance, the trend suggests that early switch to targeted therapy may positively influence survival.

Respiratory infections were the predominant clinical presentation in adults, comparable to observations by Feng M et al., who found pulmonary infections and ventilator dependence strongly associated with *Elizabethkingia* in critically-ill patients [9]. Among neonates, meningitis and meningitis with sepsis predominated, in agreement with Goel S et al., and other Indian series describing *E. meningoseptica* as an emerging cause of neonatal meningitis, particularly in premature and low-birth-weight infants [27].

Common co-morbidities in adults, such as diabetes and COPD, have similarly been reported as predisposing factors in a prior study [28]. In neonates, prematurity and low birth weight increased susceptibility, reflecting compromised immunity and prolonged ICU exposure. The markedly longer duration from admission to infection in neonates compared to adults indicates that these were mostly late-onset, hospital-acquired infections. A Brazilian NICU cohort and a Swiss national study also demonstrated higher bloodstream-infection risk after prolonged hospitalisation, reinforcing the present study observation [28,29].

Both *E. meningoseptica* and *E. anophelis* in the present cohort displayed multidrug resistance, consistent with global trends [10]. All isolates were susceptible to piperacillin–tazobactam and minocycline, whereas resistance to levofloxacin and ciprofloxacin was universal. Comparable susceptibility profiles have been documented worldwide, identifying minocycline and β-lactam/β-lactamase inhibitor combinations as the most reliable therapeutic options [10]. The resistance patterns are explained by production of ESBLs (bla_CME) and MBLs (bla_BlaB, bla_GOB), as reported by Wang L et al., who found co-carriage of these genes in most clinical isolates [30].

Empirical therapy in present study patients often included cephalosporins and fluoroquinolones- agents to which *Elizabethkingia* is intrinsically resistant- emphasising the importance of early species identification and susceptibility testing for prompt therapy adjustment. Early initiation of effective drugs such as piperacillin-tazobactam or minocycline likely contributed to improved survival. The overall mortality of 16.7% in this study was lower than previously reported rates (25-50%) [4,31,32], possibly due to earlier recognition and targeted treatment following MALDI-TOF implementation. Although mortality was slightly higher in neonates (22.2%) than in adults (15.4%), the difference was not statistically significant, likely due to the small sample size.

Accurate and timely identification of *Elizabethkingia* species is essential for guiding therapy and infection control. Laboratories should update MALDI-TOF databases regularly to minimise misidentification. From a clinical standpoint, awareness of this pathogen's resistance profile will prevent inappropriate empirical use of cephalosporins or carbapenems. Strengthening antibiotic-stewardship programs and maintaining strict hand hygiene and water-supply monitoring can curb nosocomial transmission. Future studies incorporating molecular characterisation of resistance genes and multicentric surveillance are needed to better define regional epidemiology and therapeutic outcomes.

Limitation(s)

This study had several limitations. Being a single-centre, retrospective analysis, the findings may not be fully generalisable to other settings. Molecular methods for the detection of antimicrobial resistance genes were not performed, limiting insights into the underlying genetic mechanisms of resistance. Although species-level identification was confirmed using MALDI-TOF MS, limitations of the VITEK-2 automated system were evident. While all *Elizabethkingia meningoseptica* isolates were correctly identified by VITEK-2, a substantial proportion of *Elizabethkingia anophelis* isolates were misidentified, most commonly as *E. meningoseptica* and occasionally as *Chryseobacterium indologenes*. This highlights the restricted ability of conventional automated systems to accurately differentiate *Elizabethkingia* species, particularly *E. anophelis*. Despite these limitations, the study provides valuable insights into species distribution, antimicrobial resistance patterns, and clinical outcomes of *Elizabethkingia* infections in both adult and neonatal populations.

CONCLUSION(S)

This study highlights the clinicoepidemiological profile and antimicrobial susceptibility pattern of *Elizabethkingia* infections in a tertiary-care hospital. The organism showed multidrug resistance, with minocycline, piperacillin + tazobactam remaining the most active agents. Early and accurate species-level identification, guided by MALDI-TOF, is crucial for targeted therapy and improved outcomes. Continuous surveillance and rational antibiotic use are essential to limit resistance and optimise patient management.

Acknowledgement

The authors would like to thank the Institutional Ethical Committee, SGPGIMS (IEC, SGPGIMS) for granting permission to conduct this study.

Authors' contribution: NY: Protocol development, Conception and design; NY, CS: Methodology used to collect isolates; KK: Data collection; CS, SSP: Data analysis, Data curation; CS: Supervision; NY, CS: Writing original draft, Interpretation of Data; CS, SSP; Writing review and editing. All authors read and approved the final manuscript for submission.

Availability of data and materials: The data presented in this study are available on request from the corresponding author.

REFERENCES

[1] Singh NS, Singh A, Gupta P, Agarwal A. *Elizabethkingia anophelis* infections: A case series from a tertiary care hospital in Uttar Pradesh. Cureus. 2022;14(11):e32057. Doi: 10.7759/cureus.32057. PMID: 36600834; PMCID: PMC9802642.

[2] Geremia N, Marino A, De Vito A, Giovagnorio F, Stracquadanio S, Colpani A, et al. Rare or unusual non-fermenting Gram-negative bacteria: Therapeutic approach and antibiotic treatment options. Antibiotics (Basel). 2025;14(3):306. Doi: 10.3390/antibiotics14030306. PMID: 40149115; PMCID: PMC11939765.

[3] Zajmi A, Teo J, Yeo CC. Epidemiology and characteristics of *Elizabethkingia* spp. infections in Southeast Asia. Microorganisms. 2022;10(5):882. Doi: 10.3390/microorganisms10050882. PMID: 35630327; PMCID: PMC9144721.

[4] Singh S, Sahu C, Singh Patel S, Singh S, Ghoshal U. Clinical profile, susceptibility patterns, speciation and follow-up of infections by *Elizabethkingia* species: Study on a rare nosocomial pathogen from an intensive care unit of North India. New Microbes New Infect. 2020;38:100798. Doi: 10.1016/j.nmni.2020.100798. PMID: 33294193; PMCID: PMC7689178.

[5] Wang B, Cheng R, Feng Y, Guo Y, Kan Q, Qian A, et al. *Elizabethkingia anophelis*: An important emerging cause of neonatal sepsis and meningitis in China. Pediatr Infect Dis J. 2022;41(5):e228-e232. Doi: 10.1097/INF.0000000000003464. PMID: 35067644.

[6] Raj N, Ojha V, Rautela A, Singh V, Das A, Sen M, et al. One-year experience of an emerging nosocomial pathogen *Elizabethkingia meningoseptica* at a tertiary care hospital in Northern India: A case series. Int J Crit Illn Inj Sci. 2024;14(2):112-14. Doi: 10.4103/ijciis.ijciis_73_23. PMID: 39005978; PMCID: PMC11245138.
[7] Thigpen S, Walblay K, Adil H, Zelencik S, Zelinski C, Nelson K, et al. *Elizabethkingia* spp. outbreak in a ventilator-capable skilled nursing facility, Chicago 2023. Open Forum Infect Dis. 2023;10(Suppl 2):ofad500.1288. Doi: 10.1093/ofid/ofad500.1288. PMCID: PMC10676833.
[8] Huang C, Kuo S, Lin L. Mortality risk and antibiotic therapy for patients with infections caused by *Elizabethkingia* species: A meta-analysis. Medicina (Kaunas). 2024;60(9):1529. Doi: 10.3390/medicina60091529. PMID: 39336571; PMCID: PMC11433677.
[9] Feng M, Huang M, Fan Y, Liu G, Zhou S, Zhou J. Clinical characteristics and risk factors for infection and death in critically ill patients with pulmonary infection with *Elizabethkingia* spp. Infect Drug Resist. 2024;17:2673-83. Doi: 10.2147/IDR.S460640. PMID: 38953097; PMCID: PMC11216603.
[10] Huang C. Antimicrobial susceptibility patterns and antimicrobial therapy of infections caused by *Elizabethkingia* species. Medicina (Kaunas). 2024;60(12):1990. Doi: 10.3390/medicina60121990.
[11] Hu S, Lv Y, Xu H, Zheng B, Xiao Y. Biofilm formation and antibiotic sensitivity in *Elizabethkingia anophelis*. Front Cell Infect Microbiol. 2022;12:953780. Doi: 10.3389/fcimb.2022.953780.
[12] Wu C, Xiong L, Liao Q, Zhang W, Xiao Y, Xie Y. Clinical manifestations, antimicrobial resistance and genomic feature analysis of multidrug-resistant *Elizabethkingia* strains. Ann Clin Microbiol Antimicrob. 2024;23(1):32. Doi: 10.1186/s12941-024-00691-6. PMID: 38600542; PMCID: PMC11007976.
[13] González LJ, Vila AJ. Carbapenem resistance in *Elizabethkingia meningoseptica* is mediated by metallo-β-lactamase BlaB. Antimicrob Agents Chemother. 2012;56(4):1686-92. Doi: 10.1128/AAC.05835-11. PMID: 22290979; PMCID: PMC3318372.
[14] Lin JN, Lai CH, Yang CH, Huang YH, Lin HF, Lin HH. Comparison of four automated microbiology systems with 16S rRNA gene sequencing for identification of *Chryseobacterium* and *Elizabethkingia* species. Sci Rep. 2017;7(1):13824. Doi: 10.1038/s41598-017-14244-9. PMID: 29062009; PMCID: PMC5653830.
[15] Govindaraju G, Rajaiah B, Ramakrishnan S. Elizabethkingia meningoseptica infection in neonates: A threat and clinical challenge. Infect Dis Trop Med. 2021;7:e756. Doi:10.32113/idtm_20219_756.
[16] Ghafur A, Vidyalakshmi PR, Priyadarshini K, Easow JM, Raj R, Raja T. Elizabethkingia meningoseptica bacteremia in immunocompromised hosts: The first case series from India. South Asian Journal of Cancer. 2013;2(04):211-15.
[17] Kalil AC, Metersky ML, Klompas M, Muscedere J, Sweeney DA, Palmer, et al. American Thoracic Society: Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med. 2005;171:388-416.
[18] Marino PL. The ICU Book. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2013. Available from: https://surgery.lwwhealthlibrary.com/book.aspx?bookid=942&sectionid=0.
[19] Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801-10.
[20] Schuetz P, Birkhahn R, Sherwin R, Jones AE, Singer AJ, Kline JA. Procalcitonin to diagnose infection and guide antibiotics in patients with leukocytosis: Prospective study. Int J Infect Dis. 2008;12(3):319-24.
[21] Koneman EW, Allen SD, Janda WM, Schreckenberger PC, Winn WC Jr. Koneman's Color Atlas and Textbook of Diagnostic Microbiology. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2017.
[22] Forbes BA, Sahm DF, Weissfeld AS. Bailey & Scott's Diagnostic Microbiology. 14th ed. St. Louis: Elsevier; 2017.
[23] Clinical and Laboratory Standards Institute (CLSI). Performance standards for antimicrobial disc susceptibility tests. 34th ed. CLSI standard M100. Wayne (PA): CLSI; 2024.
[24] Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: An international expert proposal for interim standard definitions for acquired resistance. *Clin Microbiol Infect.* 2012;18:268-81. Doi: 10.1111/j.1469-0691.2011.03570.x.
[25] Yahşi A, Bayhan Gİ, Erat T, Güney AY, Özen S, Konca K, et al. *Chryseobacterium/Elizabethkingia* species infections in children. Asian Pacific Journal of Tropical Medicine. 2023;16(6):268-75.
[26] Balm MN, Salmon S, Jureen R, Teo C, Mahdi R, Seetoh T, et al. Bad design, bad practices, bad bugs: Frustrations in controlling an outbreak of *Elizabethkingia meningoseptica* in intensive care units. Journal of Hospital Infection. 2013;85(2):134-40.
[27] Goel S, Jhajra SD, Nangia S, Kumar A, Nanda D. Neonatal meningitis secondary to *Elizabethkingia meningoseptica* infection. J Glob Infect Dis. 2023;15(1):23-27. Doi: 10.4103/jgid.jgid_111_22.
[28] Freitas FTM, Araujo AFOL, Melo MIS, Romero GAS. Late-onset sepsis and mortality among neonates in a Brazilian intensive care unit: A cohort study and survival analysis. Epidemiol Infect. 2019;147:e208. Doi: 10.1017/S095026881900092X. PMID: 31364533; PMCID: PMC6624867.
[29] Giannoni E, Agyeman PKA, Stocker M, Posfay-Barbe KM, Heininger U, Spycher BD, et al.; Swiss Pediatric Sepsis Study. Neonatal sepsis of early onset, and hospital-acquired and community-acquired late onset: A prospective population-based cohort study. J Pediatr. 2018;201:106-114.e4. Doi: 10.1016/j.jpeds.2018.05.048. PMID: 30054165.
[30] Wang L, Zhang X, Li D, Hu F, Wang M, Guo Q, et al. Molecular characteristics and antimicrobial susceptibility profiles of *Elizabethkingia* clinical isolates in Shanghai, China. Infect Drug Resist. 2020;13:247-56. Doi: 10.2147/IDR.S240963. PMID: 32099417; PMCID: PMC6996224.
[31] Jean SS, Lee WS, Chen FL, Ou TY, Hsueh PR. *Elizabethkingia meningoseptica*: An important emerging pathogen causing healthcare-associated infections. J Hosp Infect. 2014;86(4):244-49. Doi: 10.1016/j.jhin.2014.01.009. PMID: 24680187.
[32] Lau SK, Chow WN, Foo CH, Curreem SO, Lo GC, Teng JL, et al. *Elizabethkingia anophelis* bacteremia is associated with clinically significant infections and high mortality. Sci Rep. 2016;6:26045. Doi: 10.1038/srep26045. PMID: 27185741; PMCID: PMC4868968.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Microbiology, Uttar Pradesh University of Medical Sciences, Saifai, Etawah, Uttar Pradesh, India.
2. Additional Professor, Department of Microbiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India.
3. Senior Resident, Department of Microbiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India.
4. Associate Professor, Department of Microbiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Nidhi Yaduvanshi,
Flat No. 605, S Block, Multistorey Building, UPUMS,
Saifai Campus, Etawah, Uttar Pradesh, India.
E-mail: yaduvanshinidhi89@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Sep 03, 2025
- Manual Googling: Jan 10, 2026
- iThenticate Software: Jan 14, 2026 (1%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? No
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Aug 19, 2025**
Date of Peer Review: **Oct 08, 2025**
Date of Acceptance: **Jan 17, 2026**
Date of Publishing: **Jun 01, 2026**