

Multidrug-Resistant *Elizabethkingia anophelis* Bacteremia in Northern Taiwan: Focusing on Prognostic Factors and Antimicrobial Susceptibility to Minocycline and Rifampin

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Purpose: *Elizabethkingia anophelis* is an emerging multidrug-resistant pathogen associated with high mortality, particularly in healthcare-associated bacteremia. Treatment is complicated by frequent species misidentification and limited availability of effective antibiotics. This study aimed to investigate the clinical characteristics, predictors of early and late mortality, and antimicrobial resistance profiles, including associated resistance genes.

Patients and Methods: A retrospective cohort study was conducted from 2018 to 2022 at a center in northern Taiwan, involving patients with *E. anophelis* bacteremia. Demographic and clinical data, including comorbidities and laboratory parameters, were collected. Clinical severity was assessed using the Pitt bacteremia score. Bacterial isolates were identified using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry and species-specific polymerase chain reaction. Antimicrobial susceptibility was determined using broth microdilution, and resistance genes were detected by PCR.

Results: The 14-day and 28-day mortality rates after admission were 35% and 40%, respectively. The 14-day mortality rate was associated with high Pitt bacteremia scores, chronic kidney disease, anemia, and hyperbilirubinemia. Anemia and high Pitt bacteremia scores were consistently associated with 28-day mortality. Most isolates were phenotypically resistant to β -lactams, fluoroquinolones, and trimethoprim-sulfamethoxazole, while susceptibility to minocycline (1.6%) and rifampin (9.5%) was preserved. The detected resistance genes included multiple determinants (*bla_B*, *bla_{GOB}*, *bla_{CME}*, and *dfrE*), with a notable absence of *arr-1*.

Conclusion: *E. anophelis* bacteremia is associated with higher mortality and multidrug resistance. Prognosis is significantly influenced by host factors and specific laboratory findings. Given the high resistance of these bacteria to traditional antibiotics, minocycline and rifampin may serve as key treatment options when susceptibility is confirmed. Further studies are needed to validate their clinical efficacy, dosing, and combination strategies.

Keywords: Bacteremia mortality, antibiotic resistance genes, Pitt bacteremia score, minocycline susceptibility, rifampin efficacy, broth microdilution

Introduction

Elizabethkingia is an emerging non-fermenting Gram-negative bacillus frequently reported as a cause of hospital-acquired infections, especially among immunocompromised and critically ill patients.¹ Beyond clinical settings, it persists in water systems and soil, serving as environmental reservoirs for multidrug-resistant strains.² Its intrinsic resistance to multiple antibiotics and frequent misidentification, especially between *E. meningoseptica* and *E. anophelis*, complicates both diagnosis and infection control.^{1–3}

Of the six currently recognized species, *E. anophelis* has emerged as the leading cause of nosocomial infections in many geographic regions. In Taiwan, Lee et al reported that a fluoroquinolone-resistant clonal lineage of *E. anophelis* emerged in 2013, exhibiting distinct genomic features compared to other global strains.⁴ However, species distribution appears to vary regionally. *E. anophelis* dominates Taiwan and Singapore, whereas other species such as *E. miricola* and *E. meningoseptica* are more prevalent in Malaysia and Australia.^{2,5}

Treatment of *Elizabethkingia* infections is challenging owing to widespread resistance to β -lactams, carbapenems, and aminoglycosides.^{1,2,6} Genomic analyses have shown that metallo- β -lactamase genes (*bla_B*, *bla_{GOB}*, *bla_{CME}*) and efflux pumps are widely distributed, providing potential explanations for its multidrug-resistant phenotype.^{2,4,7} Although agents such as minocycline, rifampin, and trimethoprim-sulfamethoxazole (TMP-SMX) have historically been considered effective,⁷ studies from Singapore and Korea report greater variability in fluoroquinolone resistance, reflecting geographic and strain-specific differences in resistance gene profiles.^{3,8} This high level of resistance increases the risk of inappropriate initial antimicrobial therapy, identified as one of the key factors associated with poor clinical outcomes. A recent meta-analysis established that inappropriate antimicrobial treatment, intensive care unit (ICU) admission, immunosuppression, and respiratory failure are significant risk factors for mortality in *Elizabethkingia* infections.⁸ These observations underscore the importance of region-specific surveillance and genome-informed therapeutic strategies for effective management.

Prior to the 2019 update of the VITEK® MS database, *Elizabethkingia* species were frequently misidentified as *E. meningoseptica* in a tertiary medical center in Northern Taiwan. Following the update, infection control surveillance revealed a sharp increase in *E. anophelis* cases and a corresponding decline in *E. meningoseptica* cases, exposing a long-standing misidentification issue. Similar findings were reported in Singapore, where species-specific polymerase chain reactions (PCR) identified *E. anophelis* as the predominant species in blood isolates, challenging earlier assumptions and underscoring the limitations of matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) and the importance of molecular confirmation in routine diagnostics.³

Based on internal observations from 2009 to 2013, *E. meningoseptica* isolates at our center were fully susceptible to minocycline (100%) but exhibited high resistance to levofloxacin (16.7% susceptible), piperacillin-tazobactam (28.4% susceptible), and TMP-SMX (0% susceptible). These findings, along with recommendations from the Sanford Guide,⁹ informed our empirical treatment choices, which were primarily limited to minocycline, levofloxacin, and piperacillin-tazobactam. Nevertheless, despite adherence to these protocols, the clinical outcomes remained poor, and infections were difficult to treat. The sudden and unexpected rise in cases of *E. anophelis* (reported by updating the VITEK® MS database in 2019), as well as persistent treatment failures, warranted further investigation. To address this, we conducted a retrospective cohort study combining clinical outcome analysis, antimicrobial susceptibility testing, and resistance gene profiling in *E. anophelis* bacteremia. This study aimed to identify mortality predictors and explore the relationship between genotype and phenotype, thus providing insights into optimal management strategies for this emerging multi-drug-resistant pathogen.

Material and Methods

Patient's Group Selection and Case Setting

All *Elizabethkingia* spp. isolates were obtained from blood cultures of patients with bacteremia treated at a tertiary medical center in Northern Taiwan between January 1, 2018, and December 31, 2022. Bacterial identification was performed using MALDI-TOF MS. Initially, 87 strains were identified. There were no age restrictions; all patients with confirmed *E. anophelis* bacteremia during the study period were included, ranging from pediatric to older populations. Patients with recurrent bacteremia, defined as a positive blood culture result from the same organism within 3 months of the initial positive blood culture, and those with two sets of positive blood cultures (considered a single event), or two sets of blood cultures within 3 days, were excluded from the analysis. All included cases were monomicrobial in terms of blood culture, with *E. anophelis* as the sole bloodstream isolate. The presence of other organisms in non-blood specimens (eg, respiratory or wound cultures) was not considered an exclusion criterion. Based on these criteria, the final analysis included 63 patients and their isolates.

Clinical data were retrieved from the hospital electronic medical records and included demographics, comorbidities, disease severity scores, laboratory results, and antibiotic therapies. The primary outcome was mortality at days 14 and 28. The Charlson Comorbidity Index (CCI)⁹ was calculated according to the patient's condition at admission, whereas the Pitt Bacteremia Score (PBS)¹⁰ was determined based on clinical and laboratory data taken during the 48 h prior to the first positive blood culture. Acute kidney injury (AKI) and moderate-to-advanced chronic kidney disease (CKD) were diagnosed according to KDIGO Clinical Practice Guidelines.^{11,12}

Treatment antibiotics were defined as those administered for >4 days within 7 days of the first positive blood culture. Exposure antibiotics were defined as those administered for >4 days before the first positive blood culture.

Susceptibility Testing and Identification of Bacterial Isolates

All isolated bacteria were preserved at -80°C in a bacterial bank, then subcultured in thioglycolate broth (CMP® PTM, Taiwan) and incubated at 37°C in a 5% CO_2 atmosphere for 24 h. The cultures were subsequently transferred to blood agar plates (tryptic soy agar with 5% sheep blood; CMP® PPM, Taiwan) and incubated for an additional 24 h under identical conditions.

PCR

Bacterial DNA was extracted using the TANBead Bacteria DNA Auto Plate Kit (Taiwan). The *anoR*, *meng*, and *ureG* genes were amplified using specific PCR primers³ to detect the target genes. Each 50 μL reaction mixture contained 5 μL of DNA template, 50 pmol of each primer, 1.5 mM MgCl_2 , 0.1 mg/mL bovine serum albumin, and 1.25 U of Taq DNA polymerase. PCR amplification was performed under the following conditions: an initial denaturation at 95°C for 5 min, 35 cycles of denaturation at 95°C for 1 min, annealing at 57°C for 1 min, and extension at 72°C for 1 min, followed by a final extension at 72°C for 10 min. The PCR products were concentrated to a final volume of 25 μL by incubation at 75°C for 45 min and subsequently separated by electrophoresis on 2% agarose gels for 5 h at 2.5 V/cm. In addition, resistance genes, including *PEDO-1*, *PEDO-3*, *CPS-1*, *ESP-1*, *SPM-1*, *catB3*, *rpsJ*, *tetB(48)*, *LRA-12*, *TLA-1*, *TLA-3*, *TEM-113*, *bla_B*, *bla_{GOB}*, and *bla_{CME}*, were detected by PCR. [Supplementary Figure 1](#) shows the agarose gel electrophoresis result for *blaB*-positive isolates. The corresponding primer sequences are listed in [Supplementary Table 1](#), and detailed experimental protocols are provided in the [Supplementary Methods](#).

MALDI-TOF MS

A small portion of a freshly grown colony was inoculated onto a 48-well target plate and immediately overlaid with 1 μL α -cyano-4-hydroxycinnamic acid matrix solution (bioMérieux, France). The plate was then dried and transferred to the VITEK® MS system (bioMérieux, France), a MALDI-TOF MS-based platform for bacterial identification.¹³ The resulting spectral fingerprints were automatically compared to the VITEK MS database version 2.0 and reanalyzed with the SARAMIS™ Knowledge Base (research-use-only, version 4.14). As previously demonstrated in a multicenter study investigating the accuracy of MALDI-TOF MS for *Elizabethkingia* species identification,¹⁴ the identification results correlated 100% with PCR-based species confirmation. [Supplementary Figure 2](#) provides the MALDI-TOF MS spectrum of *Elizabethkingia anophelis*.

Minimum Inhibitory Concentration (MIC) via Broth Microdilution

Owing to the high antibiotic resistance of *Elizabethkingia* spp., antibiotics were selected based on previous reports of susceptibility or possible clinical efficacy.¹⁵ MICs of 16 selected antimicrobials were determined using the reference broth microdilution method according to the Clinical and Laboratory Standards Institute (CLSI)-recommended guidelines.¹⁶

All antibiotics were purchased from Sigma-Aldrich (USA). MIC breakpoints for other species were adopted according to CLSI or United States Food and Drug Administration (US FDA) breakpoints,¹⁷ as CLSI has no established MIC breakpoint for *Elizabethkingia* spp. Amikacin, gentamicin, ciprofloxacin, levofloxacin, imipenem, cefepime, cefoperazone, ceftazidime, piperacillin-tazobactam, TMP-SMX, minocycline, and doxycycline were obtained from other non-Enterobacteriaceae species in the CLSI. Vancomycin and rifampin were obtained from *Enterococcus* spp.

from the CLSI. Tigecycline, which currently has no CLSI breakpoint in non-Enterobacteriaceae and *Enterococcus spp.*, was adopted from the US FDA criteria ([Supplementary Table 2](#)). Detailed protocols are provided in the [Supplementary Methods](#).

Data Statistics

Statistical analyses comparing the deceased and surviving patients were performed using Pearson's chi-square test and independent sample *t*-test, with a significance threshold of $p < 0.05$. A multivariable logistic regression model was then created to identify the independent determinants of 14-day and 28-day mortality risks. Variables with clinical relevance and a p -value < 0.1 in the univariate analysis were initially included in the model. A backward stepwise selection method was applied to eliminate variables sequentially based on the Akaike Information Criterion until an optimal model was achieved. Statistical significance was defined as $p < 0.05$. Data were analyzed using SAS Enterprise Guide 8.4.

Results

Risk Factors Associated with Clinical Outcomes in Patients

Initially, 87 strains were isolated from the 68 patients. Five patients were inadvertently counted more than once owing to separate hospital admissions within a 3-month period; their duplicate episodes and corresponding five strains were excluded. Additionally, 19 strains were excluded as duplicate isolates obtained from the same patient within a 3-day interval.

The demographic and baseline clinical characteristics of the 63 patients with *Elizabethkingia anophelis* bacteremia are summarized in [Table 1](#). The mean age of the patients was 66.6 years, and 61.9% were male. The most common comorbidities were diabetes mellitus (34.9%), malignancies (30.2%), and congestive heart failure (30.2%). Other notable conditions included moderate or advanced CKD (27%) and chronic obstructive pulmonary disease (14.3%). The median PBS was 3.8, and 54% of patients had a $\text{PBS} \geq 4$. Thirty-one isolates collected in 2018 and 2019 were initially identified as *E. meningoseptica* by MALDI-TOF MS. All these isolates were later confirmed as *E. anophelis* by species-specific PCR. Upon rechecking the updated MALDI-TOF MS database, the identification results were found to be 100% consistent with PCR findings. Prior to the onset of bacteremia, the most commonly administered antibiotics were carbapenem (24 patients, 38.1%) and a third-generation cephalosporin (23 patients, 36.5%). [Table 1](#) shows the results of the univariate analysis of the 14-day and 28-day mortality. Higher PBS ($p=0.01$) and AKI ($p=0.04$) were significantly associated with an increased 14-day mortality. At 28 days, statistically significant associations were found between high PBS levels ($p=0.008$) and anemia ($p=0.042$). Minocycline, levofloxacin, and piperacillin-tazobactam treatments were not significantly associated with survival ([Table 1](#)).

Multivariate logistic regression analysis revealed that 14-day mortality was independently associated with elevated PBS (odds ratio [OR] 1.768; 95% confidence interval [CI], 1.228–2.574; $p=0.011$), moderate-to-advanced CKD (OR, 9.954; 95% CI, 1.381–71.725; $p=0.023$), anemia (OR, 55.587; 95% CI, 3.683–839.064; $p=0.004$), and hyperbilirubinemia (OR, 25.457; 95% CI, 3.034–213.616; $p=0.006$). Although treatment containing piperacillin-tazobactam appeared protective, the association was not statistically significant (OR, 0.225; 95% CI, 0.037–1.360; $p=0.067$) ([Figure 1](#)). For 28-day mortality, longer hospitalization after the onset of bacteremia was associated with lower mortality risk (OR, 0.861; 95% CI, 0.79–0.94; $p < 0.001$), while PBS (OR, 1.30; 95% CI, 0.98–1.73; $p=0.071$) and anemia (OR, 6.79; 95% CI 0.85–54.09; $p=0.071$) were not significantly correlated ([Figure 2](#)).

Of the 63 patients included in the analysis, only three were identified with primary bacteremia upon presentation to the emergency department, suggesting the possibility of community-acquired infections, while the remaining cases were presumed to be hospital-acquired based on the timing of bacteremia onset and associated clinical circumstances.

In vitro Antibiotic Susceptibility of *E. anophelis*

MICs were determined using broth microdilution, revealing that *E. anophelis* was highly resistant to several antibiotics. Antibiotic breakpoints were adopted from CLSI 2024 and the United States Food and Drug Administration Report of 2023¹⁷ ([Supplementary Table 2](#)). The MICs of all isolates are shown in [Supplementary Table 3](#). All isolates were

Table 1 Demographic Variables Associated with Elizabethkingia Anophelis Bacteremia by 14-Day and 28-Day Mortality

Variables	Overall N= 63 ^a	14-Day Mortality		p ^b	28-Day Mortality		p ^b
		Survivors N=41 ^a	Non-Survivors N= 22 ^a		Survivors N=38 ^a	Non-Survivors N= 25 ^a	
Age	66.6±21.3	64.7±24.6	70.1±12.3	0.937	64.5±25.7	69.8±12.3	0.343
Charlson Comorbidity Index ^c	5.7±2.9	5.4±3.1	6.4±2.3	0.162	5.3±3.1	6.4±2.6	0.176
Pitt bacteremia score ^d	3.8±3.1	3±2.3	5.4±3.7	0.01	3±2.3	5.1±3.7	0.008
Days in hospital before bacteremia	31.8±25.9	32±27.4	31.2±22	0.724	33.1±28.3	29.8±22.0	0.625
Days in hospital after bacteremia	26±50.2	38±58.3	3.7±3.7	<0.001	39.2±61.1	6±7.8	0.009
Total hospitalization days	57.8±59.9	70±68.8	35±22.1	0.008	72.2±71.7	35.7±22.0	0.017
Male sex	39(61.9)	23(56.1)	16(72.7)	0.195	22(57.9)	17(68.0)	0.419
Comorbidity							
Moderate or advanced CKD ^e	17(27)	8(19.5)	9(40.9)	0.068	8(21.1)	9(36.0)	0.191
Diabetes mellitus	22(34.9)	14(34.1)	8(36.4)	0.86	14(36.8)	8(32.0)	0.693
Liver cirrhosis	2(3.2)	1(2.4)	1(4.5)	1	1(2.6)	1(4.0)	1
COPD	9(14.3)	3(7.3)	6(27.3)	0.075	3(7.9)	6(24.0)	0.138
Congestive heart failure	19(30.2)	10(24.4)	9(40.9)	0.173	9(23.7)	10(40.0)	0.167
Malignancy	19(30.2)	13(31.7)	6(27.3)	0.715	11(28.9)	8(32.0)	0.796
Laboratory finding							
Acute kidney injury ^f	29(46)	15(36.6)	14(63.6)	0.04	15(39.5)	14(56.0)	0.315
Anemia ^g	46(73)	27(65.9)	19(86.4)	0.08	24(63.2)	22(88.0)	0.042
Elevated GOT	55(92.1)	36(87.8)	22(100)	0.437	31(81.6)	24(96.0)	0.63
Elevated GPT	19(30.2)	11(26.8)	8(36.4)	0.432	10(26.3)	9(36.0)	0.375
Hyperbilirubinemia ^h	25(39.7)	12(29.3)	12(54.5)	0.053	13(34.2)	12(48.0)	0.298
Hypoalbuminemia ⁱ	27(42.9)	14(34.1)	12(54.5)	0.249	13(34.2)	14(56.0)	0.093
Markedly elevated CRP ^j	38(60.3)	22(53.7)	16(72.7)	0.147	20(52.6)	18(72.0)	0.127
Treatment contains							
Minocycline	24(38.1)	16(39)	8(36.4)	0.836	13(34.2)	11(44.0)	0.434
Levofloxacin	32(50.8)	22(53.7)	10(45.5)	0.535	21(55.3)	11(44.0)	0.382
Piperacillin-tazobactam	22(34.9)	17(41.5)	5(22.7)	0.137	16(41.2)	6(24.0)	0.14

Notes: a Mean (± standard deviation) or number (%). b Independent t test; Pearson's chi-square test. Differences with P < 0.05 were considered statistically significant and are shown in bold. c Patient's initial medical record upon admission. d All criteria are graded within 48 h before or on the day of first positive blood culture. The highest score was recorded at that time. e CKD was defined according to the 2024 KDIGO Clinical Practice Guideline 12, categories G3b–G5. f Acute kidney injury was defined according to the KDIGO 2012 Clinical Practice Guideline 13. g Anemia was defined as a hemoglobin level <11 g/dL. h Hyperbilirubinemia was defined as total bilirubin >1.2 mg/dL. i Hypoalbuminemia was defined as a serum albumin <3.0 g/dL. j Markedly elevated CRP level was defined as a CRP level >5 mg/dL.

Abbreviations: CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CRP, C-reactive protein; GOT, Glutamic Oxaloacetic Transaminase; GPT, Glutamic Pyruvic Transaminase.

resistant to amikacin, gentamicin, imipenem, cefepime, ceoperazone, ceftazidime, piperacillin-tazobactam, chloramphenicol, and TMP-SMX. Only one isolate was susceptible and five exhibited intermediate susceptibility to tigecycline. Most of the isolates demonstrated sensitivity to minocycline (98.41%, 62 isolates), rifampin (90.48%, 57 isolates), and doxycycline (98.41%, 62 isolates) (Table 2).

Genetic Determinants of E. anophelis Resistance

The five genes encoding carbapenem-hydrolyzing metallo-β-lactamases (MBL) (*PEDO-1*, *PEDO-3*, *CPS-1*, *ESP-1*, *SPM-1*), the *catB3* gene, and the genes of the determinant of fluoroquinolone resistance (*rpsJ*, *tetB(48)*) were found in all isolates. Beta-lactamase genes (*LRA-12*, *TLA-1*, *TLA-3*, *TEM-113*, *bla_B*, *bla_{GOB}*, *bla_{CME}*) were identified in 58–63

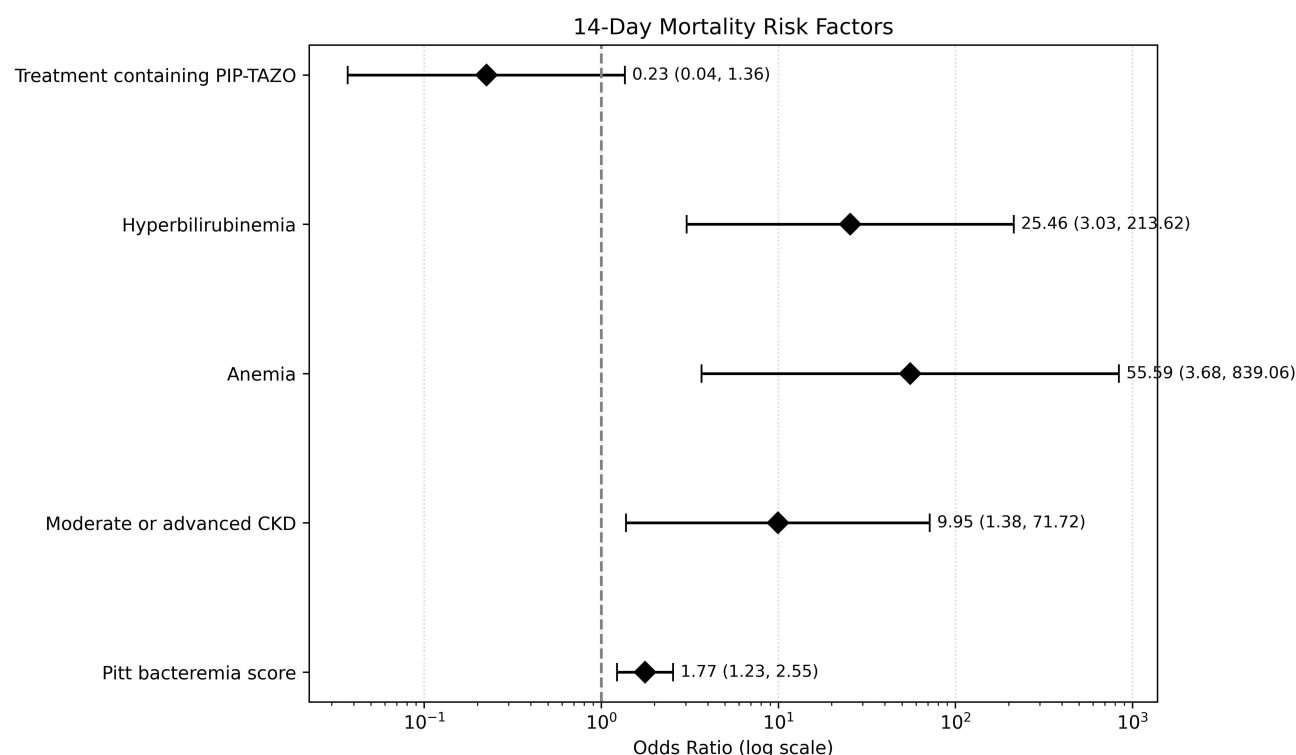


Figure 1 Multivariate logistic regression analysis of 14-day mortality. Significant predictors of 14-day mortality included a high Pitt bacteremia score (OR, 1.77; 95% CI, 1.23–2.55), moderate-to-advanced chronic kidney disease (CKD) (OR, 9.95; 95% CI, 1.38–71.73), anemia (hemoglobin <11 g/dL) (OR, 55.59; 95% CI, 3.68–839.06), and hyperbilirubinemia (total bilirubin >1.2 mg/dL) (OR, 25.46; 95% CI, 3.03–213.62). Treatment containing piperacillin–tazobactam (PIP-TAZO) was associated with reduced 14-day mortality (OR, 0.23; 95% CI, 0.04–1.36).

Abbreviations: OR, odds ratio; CI, confidence interval; CKD, chronic kidney disease; PIP-TAZO, piperacillin–tazobactam.

isolates. All isolates harbored *dfrE*, which is a known determinant of diaminopyrimidine resistance. None of the isolates were found to harbor the *arr-1* gene, an antibiotic inactivation enzyme commonly found in other resistant bacteria.

Discussion

E. anophelis bacteremia is a severe infection characterized by high mortality and extensive antimicrobial resistance. In this study, the observed 14- and 28-day mortality rates were 35% and 40%, respectively, notably higher than those reported in previous studies, possibly reflecting host vulnerability, inclusive of elevated PBS, anemia, kidney, and liver function impairments. However, although the strains here were resistant to even more antibiotics than those described in previous reports, minocycline and rifampin demonstrated very good in vitro activity (over 90% susceptibility rates). Although oral minocycline demonstrated no clear clinical benefit and rifampin was not administered, genomic analysis confirmed the phenotypic susceptibility profiles, as all resistance genes were associated with in vitro resistance to antibiotics, except for minocycline, which remained highly active despite the presence of *tetB(48)*, and rifampin, whose resistance gene *arr-1* was absent in all isolates. These results emphasize both the therapeutic challenges and potential of minocycline and rifampin, which require further clinical evaluation.

Elizabethkingia spp. infections have been correlated with high mortality rates worldwide,^{5,18} with *E. anophelis* infections specifically linked to increased hospital mortality.^{19,20} In this study, independent factors associated with 14-day mortality in *E. anophelis* bacteremia were elevated PBS, moderate-to-advanced CKD, anemia, and hyperbilirubinemia. For 28-day mortality, extended hospitalization following bacteremia was an independent protective factor, whereas PBS and anemia showed non-significant trends. These results were consistent with those of previous studies. Sarathi et al identified diabetes, pneumonia, COVID-19 co-infection, and prior antibiotic exposure as mortality-associated factors in bloodstream infections.²¹ Seong et al similarly found that elevated SAPS II scores, high CRP/albumin ratios, and rifampin resistance predicted poor outcomes, reflecting the impact of host status and systemic inflammation.⁶ A meta-

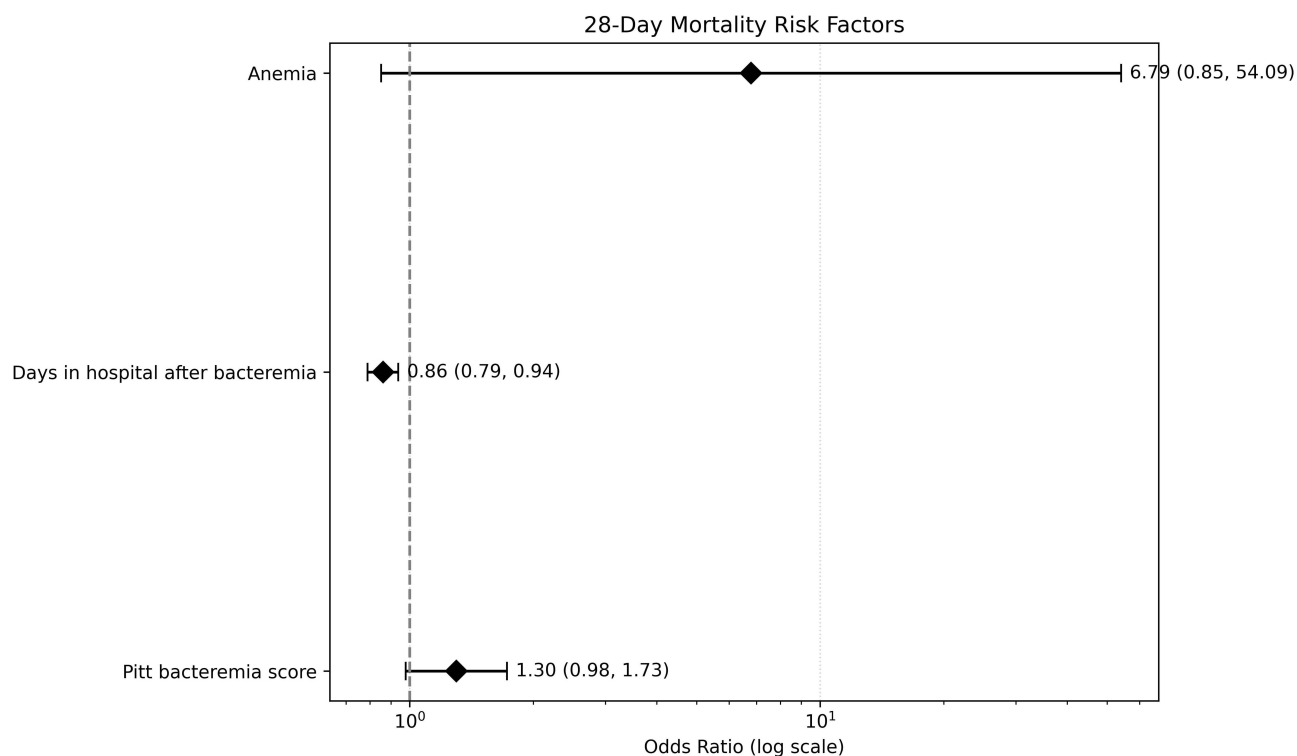


Figure 2 Multivariate logistic regression analysis of 28-day mortality. Pitt bacteremia score (OR, 1.30; 95% CI, 0.98–1.72) and anemia (hemoglobin <11 g/dL) (OR, 6.79; 95% CI, 0.90–51.18) showed borderline associations with mortality. Longer duration of hospitalization after bacteremia onset was inversely correlated with mortality (OR, 0.86; 95% CI, 0.79–0.93).

Abbreviations: OR, odds ratio; CI, confidence interval.

Table 2 In Vitro Antibiotic Susceptibility of *E. Anophelis*

	MIC(s) (mg/L)		Susceptibility (%)		
	MIC50	MIC90	S	I	R
Amikacin	>64	>64	0%	0%	100%
Gentamicin	>16	>16	0%	0%	100%
Ciprofloxacin	>4	>4	0%	6.35%	93.65%
Levofloxacin	4	>8	0%	12.70%	87.30%
Imipenem	>8	>8	0%	0%	100%
Cefepime	>16	>16	0%	0%	100%
Cefoperazone	>64	>64	0%	0%	100%
Ceftazidime	>32	>32	0%	0%	100%
Piperacillin-Tazobactam	>128	>128	0%	0%	100%
Trimethoprim-Sulfamethoxazole	4/76	4/76	0%	0%	100%
Minocycline	<2	<2	98.41%	1.59%	0%
Doxycycline	<2	4	98.41%	1.59%	0%
Tigecycline	2	8	1.59%	7.94%	90.47%
Rifampin	0.25	1	90.48%	9.52%	0%
Vancomycin	32	64	0%	12.70%	87.30%
Chloramphenicol	>64	>64	0%	0%	100%

Abbreviations: MIC50, MIC at which 50% of the isolates were inhibited; MIC90, MIC at which 90% of the isolates were inhibited; S, susceptible; I, intermediate; R, resistant.

analysis by Huang et al confirmed ICU admission, ventilator use, liver disease, and inappropriate antibiotics as major contributors to mortality.⁸ Although hepatic disease was not significant in this cohort, moderate-to-advanced CKD and anemia consistently appeared as strong risk factors.

In addition, Lee et al demonstrated that a dominant *E. anophelis* lineage in Taiwan, harboring high-level ciprofloxacin resistance, was associated with increased 14-day mortality, suggesting that bacterial genomic features may also influence outcomes.⁴ Zajmi et al emphasized diagnostic delay and immunosuppression, particularly in neonates, as contributors to poor prognosis.⁵ Notably, the inverse association observed between longer hospitalization following bacteremia and 28-day mortality in this study likely reflects survivor bias, as early mortality inherently shortens hospital stay, while patients who survive the initial phase may benefit from extended supportive care. These findings suggest that underlying diseases, such as renal dysfunction and anemia, and the adequacy of inpatient management significantly influence survival. In this study, treatment with minocycline, levofloxacin, or piperacillin-tazobactam showed no statistically significant differences in the 14-day and 28-day mortality rates.

Overall, while risk factors may vary, host condition and early disease severity consistently influence outcomes in *Elizabethkingia anophelis* bacteremia. Although antibiotic therapy may be a modifiable factor, our limited sample size may have hindered the detection of significant associations. However, further studies are required to elucidate the therapeutic role of specific antimicrobial agents.

In this study, *E. anophelis* isolates exhibited high susceptibility to minocycline (98.41%), doxycycline (98.41%), and rifampin (90.48%), whereas all isolates were resistant to TMP-SMX, ciprofloxacin, levofloxacin, and most β -lactams. Tigecycline showed poor activity, with only one isolate (1.59%) being susceptible and five exhibiting intermediate susceptibility. These findings are in line with those reported by Singh et al (India)¹⁸ and Chiu et al (Taiwan),²² who both observed 100% susceptibility to minocycline. Chiu et al²² and Lin et al²³ also reported similarly high susceptibility to rifampin (95.2%). These similarities may reflect conserved mechanisms of susceptibility to these antibiotics across regions or the limited clinical use of these agents, which reduces selective pressure. However, the complete resistance to TMP-SMX observed in this cohort contrasts sharply with Guerpillon et al during a French outbreak, where 95% of isolates were susceptible to TMP-SMX and 90% to ciprofloxacin.¹⁹ This discrepancy may be attributed to regional differences in antimicrobial usage and the clonal spread of resistant strains, as supported by Lee et al's study showing 100% ciprofloxacin resistance in a dominant local lineage.⁴ Levofloxacin susceptibility, which is reported to vary widely across studies (12–100%),²⁴ was not observed in this cohort. Regarding tigecycline, the susceptibility rate (1.59%) aligned with the lower end of the global range (0–52.2%) described in Huang's comprehensive review.²⁴ These regional differences may reflect clonal variation, antimicrobial usage patterns, and testing methodology.

Notably, comparisons of susceptibility rates across studies should be interpreted with caution owing to the absence of standardized MIC breakpoints specific to *E. anophelis*. Most referenced studies adopted surrogate interpretive criteria based on the CLSI or European Committee on Antimicrobial Susceptibility Testing guidelines for other organisms such as *Enterobacteriaceae*, *Pseudomonas aeruginosa*, or *Stenotrophomonas maltophilia*. These methodological differences, often inconsistently stated, may partially account for the variations in the reported susceptibility rates. A summary of the breakpoint references used in these studies is presented in Table 3 to promote transparency.

Taken together, our findings support the continued use of minocycline and rifampin as primary agents against *E. anophelis* but raise concerns about the reliability of TMP-SMX and fluoroquinolones in empirical treatment. The observed resistance to tigecycline also suggests its limited therapeutic utility. In Taiwan, rifampin is a restricted antimicrobial primarily reserved for tuberculosis treatment, and its use is limited owing to concerns about hepatotoxicity and significant drug–drug interactions. This limited use may have contributed to the preserved susceptibility of *E. anophelis* to rifampin, as observed in our study. These findings underscore the importance of antimicrobial stewardship and the potential of rifampin as a therapeutic option when supported by susceptibility data. Overall, these findings reinforce the need for localized susceptibility surveillance to inform treatment decisions and guide infection control efforts.

Genetic analysis supported the antibiotic susceptibility patterns, revealing that *E. anophelis* harbors multiple resistance genes, including β -lactam resistance genes (*bla_{CME}*, *bla_B*, *bla_{GOB}*) and multidrug resistance efflux pumps.^{1,25} Consistent with intrinsic resistance to cephalosporins and carbapenems, all isolates possessed five carbapenem-

Table 3 Reference Breakpoint Standards Used in Comparative Susceptibility Studies of *Elizabethkingia Anophelis*

Authors/ Year	No. of Isolates	Country/Study Design	MIC Reference Standard	Reference
Lee et al/2022	288 <i>E. anophelis</i>	Taiwan/Single center	Colistin: CLSI M100-S30, <i>Pseudomonas aeruginosa</i> Other: CLSI M100-S30, other non-Enterobacterales	[4]
Lin et al/2023	167 <i>E. anophelis</i>	Taiwan/Multicenter	Vancomycin, rifampin: CLSI M100-S32, <i>Enterococcus faecalis</i>	[23]
Huang/2024	Total 960 <i>E. anophelis</i> from 12 studies	China, Taiwan, Korea, Singapore, USA/ Literature review	CLSI M100, Enterobacteriaceae EUCAST, nonspecies	[24]
Singh et al/ 2020	27 patients (9 <i>E. anophelis</i>)	India/Single center	CLSI M100-S27 other non-Enterobacterales	[18]
Guerpillon et al/2022	20 <i>E. anophelis</i>	France/Single center	SFM/EUCAST 2020, non-species-related PK-PD breakpoints	[19]
Chiu et al/ 2021	84 <i>E. anophelis</i>	Taiwan/Single center	Tigecycline: US FDA 2019, Enterobacteriaceae Rifampin and vancomycin: CLSI M100-S30, <i>Enterococcus</i> species Other: CLSI M100-S30, other non-Enterobacteriaceae	[22]

Abbreviations: CLSI, Clinical and Laboratory Standards Institute; EUCAST, European Committee on Antimicrobial Susceptibility Testing; SFM, Société Française de Microbiologie; FDA, US Food and Drug Administration; MIC, minimum inhibitory concentration; PK-PD, pharmacokinetic–Pharmacodynamic; S30/S32, CLSI M100 30th/32nd edition.

hydrolyzing metallo- β -lactamase genes (*PEDO-1*, *PEDO-3*, *CPS-1*, *ESP-1*, *SPM-1*) and the *catB3* gene, commonly found in carbapenem-resistant *Klebsiella pneumoniae*.²⁶ Additionally, beta-lactamase genes (*LRA-12*, *TLA-1*, *TLA-3*, *TEM-113*, *bla_B*, *bla_{GOB}*, *bla_{CME}*) were present in 58–63 isolates. Various *catB* genes associated with potential multidrug resistance^{15,27} were detected in all isolates. Strong β -lactam resistance has been demonstrated and is consistent with previous studies.^{1,5,7}

Interestingly, although several resistance phenotypes aligned with the corresponding resistance genes, inconsistencies were evident, suggesting that the presence of genes alone may not fully predict antimicrobial susceptibility in *E. anophelis*. Despite all isolates possessing *tetB*(48), a gene previously associated with minocycline resistance, as well as *tetX* and *abeS*, which are more commonly linked to tigecycline resistance in *Acinetobacter baumannii*,²⁸ minocycline remained highly effective against *E. anophelis*, with 98.4% of isolates susceptible. This finding suggests that the presence of *tetB* does not necessarily predict phenotypic resistance to minocycline in *E. anophelis*.²⁹ On the contrary, the *arr-1* gene, which is typically linked to rifampin resistance in *Mycobacterium* spp.,³⁰ was absent in all isolates, which may explain the high susceptibility to rifampin (90.5%) observed in our study. Given the potential significance of *arr-1*, further functional studies are warranted to clarify its contribution to rifampin resistance. For TMP-SMX, 100% resistance was observed phenotypically, which correlated well with the universal detection of the *dfrE* gene, a determinant associated with TMP resistance in *Enterococcus faecalis*.³¹ Fluoroquinolone resistance genes such as *rpsJ* and *tetB*(48)¹⁵ were also detected in all isolates, although phenotypic resistance to fluoroquinolones varied across strains.

Taken together, our findings suggest that while genotypic data can partially predict resistance phenotypes, variability in gene expression, regulatory mechanisms, and other factors may modulate antimicrobial susceptibility. Future studies integrating transcriptomic or proteomic analyses could provide deeper insights into these discrepancies and refine resistance prediction models.

Although many studies have reported the good in vitro susceptibility of *E. anophelis* to minocycline, few have explored its clinical relevance. An ICU study in northern India reported 100% susceptibility and treatment success rates of 79% for *Elizabethkingia* spp. and 83% for *E. anophelis*; however, detailed administration protocols were lacking.¹⁸ In contrast, a Korean study found no correlation between minocycline susceptibility and mortality, and no patients received the drug.⁶ Yang et al later demonstrated that spontaneous mutations could reduce minocycline efficacy in vivo, even in initially susceptible strains.³² A higher dose of minocycline was suggested in an animal study³³ to achieve better pharmacokinetics. In this study, 98.41% of isolates were susceptible; however, regimens containing oral minocycline showed no survival benefits. Intravenous administration may be more effective, particularly in critically ill patients.

Another effective agent identified in this study was rifampin, which demonstrated high susceptibility in vitro. However, none of the patients in this cohort received rifampin as part of their treatment, and their clinical experience with the drug remains limited. Chang et al (2019) demonstrated that rifampin was the most active antimicrobial against *Elizabethkingia* spp., with 94.4% of *E. anophelis* isolates susceptible, even in the presence of chromosomally encoded metallo- β -lactamases (*Bla_B* and *bla_{GOB}*).³⁴ This suggests rifampin retains efficacy despite the high-level resistance typically associated with MBL-producing strains. Similarly, Lin et al (2023) reported 95.2% susceptibility to rifampin and further supported its therapeutic potential through demonstration of synergistic activity with vancomycin in vitro and improved survival outcomes in a zebrafish infection model.²³ Yang et al (2022) found that minocycline–rifampin combination demonstrated in vitro synergy, but did not improve survival in animal models.³² These findings suggest that while minocycline remains a key agent, resistance development may compromise its efficacy. Collectively, these findings highlight the antimicrobial activity of rifampin and suggest its potential as a therapeutic option for multidrug-resistant *Elizabethkingia* infections, particularly in those with MBL-mediated resistance.

This study had some limitations that must be acknowledged. First, determining the cause of death was difficult to evaluate owing to the complexity of the clinical conditions, frequent modifications in antibiotic regimens, and multiple coinfections present in critically ill patients. Prior to this, no research has relied on the effect of switching antibiotics, despite defining the main antibiotic. The small patient cohort also restricts the analysis of specific antibiotic regimens, including combination therapies. Although the study aimed to explore the correlation between resistance patterns and clinical outcomes, further stratified analysis was constrained by the high concordance between antimicrobial susceptibility profiles and resistance gene expression among the isolates. This limited variability in resistance profiles restricted our ability to draw meaningful comparisons between survivors and non-survivors. Moreover, the retrospective, single-center design could limit the generalizability of the findings, while the relatively small sample size could reduce the statistical power and obscure clinically meaningful associations. Additional limitations include unmeasured confounding and selection bias, which may have influenced the interpretation of clinical outcomes. Unmeasured confounding factors such as differences in antimicrobial stewardship, immune status, and the presence of multidrug-resistant pathogens may have impacted the findings. Finally, due to the lack of CLSI-defined MIC breakpoints for *E. anophelis*, susceptibility interpretation was based on surrogate criteria, which may introduce variability and should be considered a methodological limitation.

Conclusion

This retrospective study revealed a higher mortality rate compared to reports from other countries. Statistically significant risk factors included high PBS, anemia, jaundice, and CKD. Although these strains are resistant to most antibiotics, consistent with their genomic resistance profiles, they exhibit notable in vitro susceptibility to minocycline and rifampin, suggesting these agents as potential therapeutic candidates. Given the limited treatment options available, minocycline and rifampin warrant further investigation. Further prospective studies are necessary to validate the association between clinical outcomes and the use of these antimicrobials, confirm their efficacy, determine optimal dosing regimens, and clarify their role in combination therapy for *E. anophelis* infections.

Abbreviations

AKI, Acute kidney injury; CCI, Charlson Comorbidity Index; CI, confidence interval; CKD, Chronic kidney disease; CLSI, Clinical and Laboratory Standards Institute; ICU, intensive care unit; MALDI-TOF MS, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry; MBL, Metallo- β -lactamases; MIC, Minimum inhibitory concentration; OR, odds ratio; PBS, Pitt Bacteremia Score; PCR, polymerase chain reaction; TMP-SMX, trimethoprim-sulfamethoxazole; US FDA, United States Food and Drug Administration.

Data Sharing Statement

The clinical and statistical datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics Approval and Informed Consent

This study was approved by the Institutional Review Board of MacKay Memorial Hospital (approval no. 20MMHIS400e) and conducted in accordance with the Declaration of Helsinki. The requirement for informed consent was waived by the Institutional Review Board due to the retrospective design of the study and the use of anonymized patient data. All patient information was fully anonymized before the analysis to ensure confidentiality.

Consent for Publication

Not applicable. This study did not include individual data in the form of images, recordings, or quotations.

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Author Contributions

All authors significantly contributed to the work reported, including the conception, study design, execution, data acquisition, analysis, interpretation, and manuscript preparation. All authors participated in drafting, revising, or critically reviewing the article, approved the final version for publication; agreed on the target journal, and accepted responsibility for all aspects of the work.

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Disclosure

The authors declare no competing interests.

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