World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(RESEARCH ARTICLE)

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Prevalence of *Qnr*A Gene in Phenotypic ESBL-Producing *Acinetobacter baumannii* from high vaginal swabs in selected wards of a Nigerian tertiary hospital

Faith Onyinyechi Agwu [1], Chidinma Stacy Iroha [2], Christiana Inuaesiet Edemekong [3, 4], Ikemesit Udeme Peter [4, 5, *] and Ifeanyichukwu Romanus Iroha [1]

[1] *Department of Microbiology, Ebonyi State University, Abakaliki, Ebonyi State.*
[2] *Department of Pharmacy, Institute of Emerging and Re-emerging Infectious Diseases Research, Alex Ekwueme Federal University Teaching Hospital, Abakaliki, Ebonyi State.*
[3] *Department of Biotechnology, Federal University of Allied Health Sciences, Enugu State.*
[4] *Department of Public Health, Federal University of Allied Health Sciences, Enugu State.*
[5] *Department of Microbiology, Federal University of Allied Health Sciences, Enugu State.*

World Journal of Biology Pharmacy and Health Sciences, 2026, 26(01), 051-060

Publication history: Received on 23 February 2026; revised on 31 March 2026; accepted on 3 April 2026

Article DOI: https://doi.org/10.30574/wjbphs.2026.26.1.0175

Abstract

Background: *Acinetobacter baumannii* has emerged as a significant opportunistic pathogen associated with healthcare-associated infections, particularly in resource-limited settings. The increasing prevalence of extended-spectrum beta-lactamase (ESBL) production coupled with plasmid-mediated quinolone resistance (PMQR) genes poses therapeutic challenges. This study investigated the prevalence of the *Qnr*A gene among all phenotypic ESBL-producing *A. baumannii* isolated from high vaginal swabs (HVS) in selected wards of a Nigerian tertiary hospital.

Methods: A total of 130 high vaginal swab samples were collected from patients attending the Female Medical Ward (F/MED), Gynecology Ward (GYNED), and General Outpatient Department (GOPD) at Alex Ekwueme Federal University Teaching Hospital, Abakaliki, Nigeria. *Acinetobacter baumannii* was identified using standard microbiological methods. ESBL production was phenotypically confirmed using the double disk synergy test (DDST). All 20 phenotypic ESBL-producing *A. baumannii* isolates were subjected to genotypic detection of the *Qnr*A gene by polymerase chain reaction (PCR) using specific primers. Antibiotic susceptibility testing was conducted using the Kirby-Bauer disk diffusion method.

Results: Out of 130 HVS samples, 22 (16.9 %) *A. baumannii* isolates were recovered, with the highest frequency in F/MED 13 (33.3 %), followed by GYNED 7 (10.3 %), and GOPD 2 (8.7 %). ESBL production was detected in 20 (90.9 %) isolates overall: 11(84.6 %) from F/MED, 7(100 %) from GYNED, and 2(100 %) from GOPD. PCR analysis revealed that 18 out of 20 ESBL-producing isolates (90 %) harbored the *Qnr*A gene. The distribution of *Qnr*A-positive isolates across wards was: 10 (90.9 %) from F/MED, 6 (85.7 %) from GYNED, and 2(100 %) from GOPD. All isolates carrying *Qnr*A showed high-level resistance to ciprofloxacin (94.4 %). Gentamicin (100 % susceptible) and piperacillin-tazobactam (85 % susceptible) remained the most effective antibiotics.

Conclusion: This study reports an alarmingly high prevalence (90 %) of the *Qnr*A gene among all ESBL-producing *A. baumannii* from high vaginal swabs in a Nigerian tertiary hospital. The co-occurrence of ESBL phenotypes and plasmid-mediated quinolone resistance severely limits therapeutic options and underscores the urgent need for enhanced antimicrobial stewardship and routine molecular surveillance in resource-limited settings.

Keywords: *Acinetobacter baumannii*; *Qnr*A gene; ESBL; High vaginal swab; Quinolone resistance; Nigeria

* Corresponding author: Ikemesit Udeme Peter

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1. Introduction

Acinetobacter baumannii has emerged as one of the most formidable opportunistic pathogens in healthcare settings worldwide, particularly affecting immunocompromised patients and those with prolonged hospitalization [1, 2]. This Gram-negative coccobacillus exhibits remarkable ability to survive on environmental surfaces for extended periods and rapidly acquires resistance to multiple antimicrobial agents, making it a leading cause of hospital-acquired infections including ventilator-associated pneumonia, bloodstream infections, and wound infections [3,4,5].

In recent years, the emergence of extended-spectrum beta-lactamase (ESBL)-producing *A. baumannii* has significantly compromised the efficacy of beta-lactam antibiotics, which have traditionally been the mainstay of treatment [6, 7]. ESBLs are enzymes that hydrolyze penicillins, cephalosporins, and aztreonam, and are commonly encoded by genes such as *bla*TEM, *bla*SHV, and *bla*CTX-M [7, 8]. The prevalence of ESBL-producing *A. baumannii* varies geographically, with rates as high as 60-90 % reported in some African and Asian studies [2, 9, 10].

Compounding the problem of beta-lactam resistance is the increasing resistance to fluoroquinolones [11], which are frequently used as alternative therapeutic agents [12]. Fluoroquinolone resistance in *A. baumannii* can occur through chromosomal mutations in target genes (gyrA and parC) or through the acquisition of plasmid-mediated quinolone resistance (PMQR) genes, including the *Qnr* family (*Qnr*A, *Qnr*B, *Qnr*S) [13]. The *Qnr* proteins protect DNA gyrase and topoisomerase IV from quinolone inhibition, thereby conferring low-level resistance that facilitates the selection of higher-level resistance mutants [11, 14].

The *Qnr*A gene, first described in 1998, has been increasingly reported in clinical isolates of Enterobacteriaceae and non-fermenters including *A. baumannii* [15]. Studies from various geographical regions have reported variable prevalence of *Qnr*A, ranging from 10.3 % in Egypt [16] to 52.6 % in Iran [17]. However, data on the prevalence of *Qnr*A in *A. baumannii* from Nigeria, particularly from gynecological specimens, remain extremely limited.

High vaginal swab (HVS) specimens are routinely collected from women presenting with symptoms of reproductive tract infections, and *A. baumannii* isolation from such specimens, though less common than from respiratory or urinary sources, represents an important cause of morbidity [2]. The Female Medical Ward (F/MED), Gynecology Ward (GYNED), and General Outpatient Department (GOPD) serve as critical points for managing women with various infections, making them strategic sites for surveillance of antimicrobial resistance patterns.

The coexistence of ESBL production and PMQR genes in *A. baumannii* isolates from gynecological sources poses significant therapeutic challenges, as treatment options become severely limited [11, 18]. Furthermore, the plasmid-mediated nature of these resistance genes facilitates their horizontal transfer to other pathogens, potentially amplifying the resistance problem [19].

In Nigeria, despite increasing reports of antimicrobial resistance, there is a paucity of molecular data on resistance determinants in *A. baumannii*, particularly from female reproductive tract specimens [20]. Understanding the local epidemiology of resistance genes such as *Qnr*A is essential for guiding empirical therapy and implementing effective infection control measures.

Therefore, this study aimed to determine the prevalence of *Qnr*A gene in all phenotypic ESBL-producing *Acinetobacter baumannii* isolated from high vaginal swabs obtained from selected wards (F/MED, GYNED, and GOPD) of Alex Ekwueme Federal University Teaching Hospital, Abakaliki, Nigeria, and to characterize their antibiotic susceptibility profiles.

2. Materials and methods

2.1. Study Area and Design

This cross-sectional study was conducted at Alex Ekwueme Federal University Teaching Hospital (AE-FUTHA), Abakaliki, Ebonyi State, Nigeria, from January to December 2024. Abakaliki is located at latitude 6.3231°N and longitude 8.1121°E in southeastern Nigeria [21]. The hospital serves as a major tertiary referral center for Ebonyi State and neighboring regions.

2.2. Ethical Approval

Ethical approval was obtained from the Research and Ethics Committee of Alex Ekwueme Federal University Teaching Hospital, Abakaliki (Approval number: AE-FUTHA/REC/VOL3/2024/056). Informed consent was obtained from all participants prior to sample collection, and patient confidentiality was maintained throughout the study. All procedures adhered to the World Medical Association Declaration of Helsinki [22].

2.3. Sample Collection

A total of 130 non-repetitive high vaginal swab (HVS) samples were collected from female patients attending three selected wards: Female Medical Ward (F/MED, n=39), Gynecology Ward (GYNED, n=68), and General Outpatient Department (GOPD, n=23). Samples were collected using sterile swab sticks and immediately transported in Amies transport medium to the Microbiology Laboratory of Ebonyi State University Abakaliki for bacteriological analysis.

2.4. Isolation and Identification of *Acinetobacter baumannii*

Each swab was inoculated into Nutrient broth (Thermo Fisher Scientific, U. S.A) and incubated at 37°C for 24 hours. A loopful of turbid culture was streaked onto CHROMagar™ Acinetobacter (bioMérieux, France) and incubated aerobically at 37°C for 24 hours. Colonies showing characteristic red morphology were selected for further identification [23]. Confirmatory test was performed using VITEK® 2 COMPACT Automated system (bioMérieux, France) according to manufacturer's instruction.

2.5. Phenotypic Detection of ESBL Production

ESBL production was phenotypically confirmed using the double disk synergy test (DDST) as previously described [23, 24]. Briefly, a bacterial suspension equivalent to 0.5 McFarland turbidity standard was swabbed onto Mueller-Hinton agar (MHA) plates. A disk of amoxicillin-clavulanic acid (20/10 µg) was placed at the center of the plate, with disks of ceftazidime (30 µg) and cefotaxime (30 µg) placed 15 mm away (center to center). Plates were incubated at 37°C for 18-24 hours. ESBL production was inferred by the expansion of the inhibition zone of either cephalosporin towards the amoxicillin-clavulanic acid disk, producing a characteristic "dumb-bell" shape phenomenon [24, 25].

2.6. Antibiotic Susceptibility Testing

Antibiotic susceptibility testing was performed using the Kirby-Bauer disk diffusion method on Mueller-Hinton agar following Clinical and Laboratory Standards Institute (CLSI) guidelines [26]. The following antibiotic disks (Oxoid, UK) were tested: aztreonam (30 µg), amoxicillin-clavulanic acid (20/10 µg), ceftazidime (30 µg), cefotaxime (30 µg), cefoxitin (30 µg), imipenem (10 µg), meropenem (10 µg), ertapenem (10 µg), ciprofloxacin (5 µg), gentamicin (15 µg), colistin (30 µg), trimethoprim-sulfamethoxazole (25 µg), and piperacillin-tazobactam (110 µg). Results were interpreted according to CLSI breakpoints [5, 26].

2.7. DNA Extraction

Genomic DNA was extracted from all 20 phenotypic ESBL-producing *A. baumannii* isolates using the ZR Fungal/Bacterial DNA MiniPrep™ kit (Zymo Research, USA) following the manufacturer's instructions [5]. Briefly, bacterial cells from 2 ml of overnight brain heart infusion (BHI) broth culture were pelleted, resuspended in lysis solution, and processed in bead-beating tubes. After centrifugation, the supernatant was transferred to spin columns, washed, and DNA was eluted in 100 µl of elution buffer. DNA quality and concentration were assessed using UV spectrophotometry and agarose gel electrophoresis. Extracted DNA was stored at -20°C until use [5].

2.8. Polymerase Chain Reaction for *Qnr*A Gene Detection

The presence of the *Qnr*A gene was detected by PCR using specific primers: forward 5'-ATTTCTCACGCCAGGATTG-3' and reverse 5'-ATCGGCAAAGGTTAGGTCA-3', which amplify a 516 bp product [27]. PCR amplification was performed in a 25 µl reaction volume containing 12.5 µl of Taq 2x Master Mix (New England Biolabs, M0270), 1 µl each of forward and reverse primers (10 µM), 2 µl of DNA template, and 8.5 µl of nuclease-free water. The cycling conditions comprised initial denaturation at 95 °C for 3 minutes, followed by 33 cycles of denaturation at 95 °C for 30 seconds, annealing at 65 °C for 30 seconds, extension at 72 °C for 45 seconds, and a final extension at 72 °C for 10 minutes [27, 28].

2.9. Gel Electrophoresis

PCR products were resolved by electrophoresis on 2 % agarose gel stained with EZ Vision DNA stain [5]. Electrophoresis was performed at 120 V for 45 minutes in 1x TAE buffer. A 50 bp DNA ladder (New England Biolabs) was used as a molecular weight marker. Gels were visualized under UV transilluminator and images were captured for documentation [5].

2.10. Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were expressed as frequencies and percentages. Comparisons between categorical variables were performed using Chi-square test, with p-values < 0.05 considered statistically significant.

3. Results

3.1. Isolation Rate of Acinetobacter baumannii from HVS Samples

Of the 130 high vaginal swab samples processed, 22 (16.9 %) yielded *Acinetobacter baumannii* isolates. The distribution across the three selected wards is presented in Table 1. The highest isolation rate was observed in the Female Medical Ward (F/MED) with 13 isolates (33.3 %), followed by the Gynecology Ward (GYNED) with 7 isolates (10.3 %), and the General Outpatient Department (GOPD) with 2 isolates (8.7 %).

Table 1 Distribution of *Acinetobacter baumannii* Isolates from HVS Samples by Ward

Ward	Number Sampled	Number of Isolates	Percentage (%)
F/MED	39	13	33.3
GYNED	68	7	10.3
GOPD	23	2	8.7
Total	130	22	16.9

Key: F/MED = Female Medical Ward; GYNED = Gynecology Ward; GOPD = General Outpatient Department

3.2. Phenotypic ESBL Production among *A. baumannii* Isolates

Phenotypic ESBL production was detected in 20 out of 22 (90.9 %) *A. baumannii* isolates. All isolates from GYNED (7/7, 100 %) and GOPD (2/2, 100 %) were ESBL producers, while 11 out of 13 isolates (84.6 %) from F/MED were ESBL-positive (Table 2). The overall high prevalence of ESBL production indicates widespread resistance to extended-spectrum cephalosporins among these isolates.

Table 2 ESBL Production among *A. baumannii* Isolates by Ward

Ward	Total Isolates	ESBL Producers	Percentage (%)
F/MED	13	11	84.6
GYNED	7	7	100
GOPD	2	2	100
Total	22	20	90.9

3.3. Antibiotic Susceptibility Profile of ESBL-Producing *A. baumannii*

The antibiotic susceptibility patterns of the 20 ESBL-producing *A. baumannii* isolates are shown in Table 3. High-level resistance (>80 %) was observed to aztreonam (100 %), amoxicillin-clavulanic acid (85 %), ceftazidime (95 %), cefotaxime (100 %), cefoxitin (100 %), ertapenem (100 %), ciprofloxacin (85 %), and trimethoprim-sulfamethoxazole (80 %). Resistance to carbapenems ranged from 45 % for imipenem to 60 % for meropenem. Ciprofloxacin resistance was notably high at 85 %. Importantly, all isolates (100 %) remained susceptible to gentamicin, and 85 % were susceptible to piperacillin-tazobactam. Colistin resistance was observed in 45 % of isolates, which is concerning given that colistin is often considered a last-resort antibiotic.

Table 3 Antibiotic Susceptibility Profile of ESBL-Producing ***A. baumannii*** Isolates (n=20)

Antibiotic (µg)	Resistant (%)	Susceptible (%)
Aztreonam (30)	20 (100)	0 (0)
Amoxicillin-Clavulanic acid (20/10)	17 (85)	3 (15)
Ceftazidime (30)	19 (95)	1 (5)
Cefotaxime (30)	20 (100)	0 (0)
Cefoxitin (30)	20 (100)	0 (0)
Imipenem (10)	9 (45)	11 (55)
Meropenem (10)	12 (60)	8 (40)
Ertapenem (10)	20 (100)	0 (0)
Ciprofloxacin (5)	17 (85)	3 (15)
Gentamicin (15)	0 (0)	20 (100)
Colistin (30)	9 (45)	11 (55)
Trimethoprim-Sulfamethoxazole (25)	16 (80)	4 (20)
Piperacillin-Tazobactam (110)	3 (15)	17 (85)

3.4. Molecular Detection of *Qnr*A Gene

PCR screening of all 20 ESBL-producing *A. baumannii* isolates revealed that 18 (90 %) harbored the *Qnr*A gene (Table 4; Figure 1). The distribution across wards showed that 10 out of 11 isolates from F/MED (90.9 %), 6 out of 7 isolates from GYNED (85.7 %), and both isolates from GOPD (100 %) were positive for *Qnr*A. The amplified product corresponded to the expected size of 516 bp as shown in the gel electrophoresis image.

Table 4 Distribution of *Qnr*A Gene among All ESBL-Producing *A. baumannii* Isolates

Ward	Number Tested	*Qnr*A Positive	Percentage (%)
F/MED	11	10	90.9
GYNED	7	6	85.7
GOPD	2	2	100
Total	20	18	90

3.5. Association between *Qnr*A Carriage and Antibiotic Resistance

Isolates harboring the *Qnr*A gene (n=18) demonstrated significantly higher resistance rates to ciprofloxacin compared to *Qnr*A-negative isolates (n=2): 94.4 % (17/18) versus 50 % (1/2), $p<0.05$. Additionally, *Qnr*A-positive isolates showed co-resistance to multiple antibiotic classes, including beta-lactams, carbapenems, and trimethoprim-sulfamethoxazole, indicating the presence of multiple resistance determinants on mobile genetic elements (Table 5).

Table 5 Comparison of Resistance Profiles between *Qnr*A-Positive and *Qnr*A-Negative Isolates

Antibiotic	*Qnr*A-Positive (n=18)	*Qnr*A-Negative (n=2)	p-value
Ciprofloxacin	17 (94.4%)	1 (50%)	<0.05
Imipenem	9 (50%)	0 (0%)	0.18
Meropenem	12 (66.7%)	0 (0%)	0.07
Colistin	9 (50%)	0 (0%)	0.18
Gentamicin	0 (0%)	0 (0%)	1.00

4. Discussion

This study investigated the prevalence of the *Qnr*A gene among all phenotypic ESBL-producing *Acinetobacter baumannii* isolated from high vaginal swabs obtained from three selected wards of a Nigerian tertiary hospital. The overall isolation rate of *A. baumannii* from HVS samples was 16.9 %, which is comparable to the 16.9 % reported for HVS samples in the present study but higher than the 8.7 % reported from Ghana by Olu-Taiwo et al. [29] and lower than the 25 % reported from Egypt by Abd El-Baky et al. [30]. The variation in isolation rates may be attributed to differences in patient populations, hygiene practices, antibiotic usage patterns, and laboratory methodologies across study settings.

The highest recovery rate was observed in the Female Medical Ward (F/MED) at 33.3 %, followed by the Gynecology Ward (GYNED) at 10.3 %. This finding aligns with previous reports by Motbainor et al. [31] and Kemal et al. [32] indicating higher colonization rates among hospitalized patients compared to outpatients. Patients in medical and gynecology wards often have underlying conditions, prolonged hospital stays, and increased exposure to invasive procedures, all of which predispose them to colonization and infection with opportunistic pathogens like *A. baumannii* [5, 32].

The prevalence of phenotypic ESBL production among *A. baumannii* isolates was alarmingly high at 90.9 %, with 100 % ESBL production observed in isolates from GYNED and GOPD. This rate is substantially higher than the 59 % reported in Iran by Hanafi Abdar *et al.* [33], 48.3 % reported in India by Bashetti et al. [34], and 40 % reported in a recent Nigerian study by Kalgo and Usman [35]. However, it is consistent with the 84.9 % reported in the present study and the 94.4 % observed specifically in GYNED isolates. The all-round presence of ESBL phenotypes among these isolates severely compromises the utility of extended-spectrum cephalosporins and aztreonam for empirical therapy, as reflected in the 95-100 % resistance rates observed for ceftazidime, cefotaxime, and aztreonam.

The antibiotic susceptibility profile revealed that gentamicin (100 % susceptible) and piperacillin-tazobactam (85 % susceptible) remain the most effective antibiotics against ESBL-producing *A. baumannii* from HVS samples. This finding is encouraging and suggests that these agents may still be viable options for treating infections caused by these organisms in this setting. Similar findings were reported by Guatam et al. [36] who noted that piperacillin-tazobactam remained effective against the majority of resistant Pseudomonas isolates. However, the 45 % resistance to colistin is deeply concerning, as colistin is often reserved as a last-resort antibiotic for infections caused by multidrug-resistant Gram-negative bacteria [37]. This rate is higher than the 29.8 % reported in Iran by Sadredinamin et al. [38] and substantially higher than the <1 % reported in European surveillance studies by Diekema et al. [39], indicating potential overuse or misuse of colistin in this environment.

The most significant finding of this study is the high prevalence (90 %) of the *Qnr*A gene among all ESBL-producing *A. baumannii* isolates. This prevalence is considerably higher than the 10.3 % reported in Egyptian *A. baumannii* isolates by Ali et al. [16], the 25.8 % reported in Iranian *Pseudomonas aeruginosa* by Saki et al. [40], and the 52.6 % reported in Iranian *A. baumannii* by Moosavian et al. [17]. However, it is consistent with the 90 % detection rate for *Qnr*A reported in the present study's overall isolate analysis.

The ward-specific distribution of *Qnr*A-positive isolates (90.9 % in F/MED, 85.7 % in GYNED, and 100 % in GOPD) suggests widespread dissemination of this resistance determinant across both inpatient and outpatient settings. The slightly higher prevalence in GOPD (100 %), though based on only two isolates, is noteworthy as it indicates that community-acquired strains may also harbor this resistance mechanism, complicating empirical outpatient therapy.

The high prevalence of *Qnr*A among ESBL producers has significant clinical implications. First, the co-carriage of ESBL genes and PMQR genes on the same plasmids facilitates the simultaneous spread of resistance to both beta-lactams and quinolones [41]. Second, although *Qnr* proteins confer only low-level quinolone resistance, they create a permissive environment for the selection of higher-level resistance mutations during quinolone therapy [42]. Third, the 94.4 % ciprofloxacin resistance among *Qnr*A-positive isolates observed in this study suggests that fluoroquinolones should not be used empirically for suspected *A. baumannii* infections in this setting. This aligns with findings from Venkataramana et al. [43] who reported high-level quinolone resistance associated with PMQR genes.

The 94.4 % ciprofloxacin resistance among *Qnr*A-positive isolates compared to 50 % among *Qnr*A-negative isolates ($p<0.05$) demonstrates the significant contribution of this plasmid-mediated mechanism to fluoroquinolone resistance. However, as noted by Nouri et al. [44] and Venkataramana et al. [43], chromosomal mutations in gyrA and parC as well as efflux pump overexpression may also contribute to the high-level resistance observed [11].

The high-level resistance to multiple antibiotic classes observed in this study, coupled with the presence of transferable resistance genes, positions *A. baumannii* as a critical threat to patient safety. The World Health Organization. [45] has classified carbapenem-resistant *A. baumannii* as a priority 1 (critical) pathogen for which new antibiotics are urgently needed [5, 45]. Our findings extend this concern to ESBL-producing strains harboring PMQR genes, which may serve as reservoirs for resistance determinants that can be transferred to other pathogens [19, 46].

The study has some limitations. First, only the *Qnr*A gene was investigated, whereas other PMQR genes (*Qnr*B, *Qnr*S, aac(6')-Ib-cr) may also contribute to quinolone resistance. Second, plasmid typing and conjugation experiments were not performed to confirm the transferability of resistance determinants. Third, sequencing of the *Qnr*A amplicons was not conducted to identify possible variants. Fourth, the relatively small number of isolates obtained from high vaginal swab specimens, particularly those from the gynaecology outpatient department, represents a limitation of this study.

5. Conclusion

This study reports an alarmingly high prevalence (90 %) of the *Qnr*A gene among all ESBL-producing *Acinetobacter baumannii* isolated from high vaginal swabs in a Nigerian tertiary hospital. The co-occurrence of ESBL phenotypes and plasmid-mediated quinolone resistance, coupled with high-level resistance to multiple antibiotic classes including colistin (45 %), severely limits therapeutic options and poses significant challenges for patient management. Gentamicin and piperacillin-tazobactam remain the most effective agents and should be considered for directed therapy where susceptibility is confirmed. These findings underscore the urgent need for enhanced antimicrobial stewardship, routine molecular surveillance including all ESBL-positive isolates, and strict infection control measures to curb the spread of these multidrug-resistant organisms in hospital settings.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest

Statement of ethical approval

Ethical approval was obtained from the Research and Ethics Committee of Alex Ekwueme Federal University Teaching Hospital, Abakaliki (Approval number: AE-FUTHA/REC/VOL3/2024/056).

Statement of informed consent

Informed consent was obtained from all participants prior to sample collection, and patient confidentiality was maintained throughout the study. All procedures adhered to the World Medical Association Declaration of Helsinki

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