



The Relationship Between ABO Blood Group Phenotypes and Carriage of BlaCTX-M, BlaTEM, and BlaSHV ESBL Genes in Urinary Escherichia Coli Isolated among Female Patients

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ABSTRACT

β -lactam resistance due to the production of β -lactamases, more importantly, extended- spectrum β -lactamases by gram negative bacteria such as Escherichia coli, which has been highly implicated, is a serious health challenge. The primary aim of this study is to characterize ESBL producers among cephalosporin resistant Escherichia coli isolated from the urine samples of the female patients who visited the Microbiology Laboratory Unit of Federal Medical Centre, Owerri between February 2023 and August 2023 and investigate the relationship between the ESBL genes – blaCTX-M, blaTEM, and blaSHV and their ABO blood group phenotypes. During these six months, 57 E. coli samples isolated from female urine specimens showed resistance to cephalosporin following the laboratory's routine antimicrobial testing procedure. For this study, pure cultures of these isolates were obtained and subjected to biochemical tests to confirm they were E. coli. ESBL was detected by screening test using the standard Minimum Inhibitory Concentration (MIC) of the disk diffusion method where all the E. coli isolates were tested against an indicator cephalosporins (ceftriaxone 30 μ g) on Mueller-Hinton agar medium. ESBL was confirmed by the use of combination disk test. Of the 57 samples, only 43 samples were confirmed to be ESBL producer. Blood samples were collected from those patients who harboured ESBL producing E. coli for their ABO blood group phenotype. Polymerase chain reaction was carried out for the detection of the specific ESBL genes. Results showed overwhelming molecular dominance of blaCTX-M, there was high rate of multi-gene resistance and absence of association with ABO blood group.

KEYWORDS: ESBL Genes, E. coli, ABO Blood Group, PCR, blaCTX-M

1. INTRODUCTION

Urinary Tract Infections (UTIs) represent one of the most common bacterial infections worldwide, imposing a significant burden on healthcare systems and disproportionately afflicting women due to anatomical and physiological factors such as the shorter distance to the bladder in women compared to men. [15, 21]. Furthermore, the urethral opening in women is close to the rectum. Urogenital manipulations associated with daily living or medical interventions facilitate the movement of bacteria to the urethra [20]. Escherichia coli (E. coli) is the predominant causative agent of uncomplicated and complicated UTIs, accounting for a substantial majority of community and hospital-acquired cases [49, 19]. The management of these infections has been severely complicated by the rapid and global emergence of antimicrobial resistance, particularly the rise of extended-spectrum β -lactamase (ESBL)-producing Enterobacteriaceae such as Escherichia coli.

There is no generally acceptable or precise definition of ESBLs. A commonly used working definition is that the ESBLs are β -lactamases capable of conferring bacterial resistance to the penicillins, first-, second-, and third-generation cephalosporins, and aztreonam (but not the cephamycins or carbapenems) by hydrolysis of these antibiotics, and which are inhibited by β -lactamase inhibitors such as clavulanic acid [36, 11]. ESBLs are enzymes that confer resistance to a broad range of critically important beta-lactam antibiotics, including penicillins, third-generation cephalosporins, and aztreonam. Among the numerous ESBL families, the CTX-M-type enzymes have achieved global dominance, effectively replacing the earlier TEM and SHV variants in many regions. The genes encoding these enzymes, notably blaCTX-M, blaTEM, and blaSHV, are often located on highly mobile plasmids, facilitating their rapid dissemination within and between bacterial species [33]. In the antimicrobial resistance (AMR) era, the evolving resistance caused by ESBL-producing E. coli is associated with higher rates of treatment failure, prolonged illness, increased healthcare costs, and greater

mortality, making them a paramount public health concern [24, 45]. Antimicrobial resistance (AMR) has a negative impact on achieving Sustainable Development Goals (SDG), food safety, and food security [24, 31].

In the search for risk factors for infections with resistant organisms, host genetics has emerged as a potential area of interest. The ABO blood group system, a genetically determined polymorphism of red blood cells and mucosal surfaces, has been implicated in susceptibility to various infectious diseases. (2, 19). The surface glycans that determine ABO status are also expressed on the uroepithelial lining, where they can serve as receptors or attachment sites for pathogens [30, 46]. Epidemiological studies have suggested links between ABO blood groups and susceptibility to infections caused by *Helicobacter pylori*, Norovirus, and *Vibrio cholerae* [14]. It is likely that these antigens could also influence the initial adhesion and colonization of uropathogenic *E. coli* (UPEC), potentially creating a host environment that is more or less permissive to strains carrying specific virulence or resistance determinants.

However, the specific relationship between ABO blood group phenotype and the carriage of particular ESBL genes in uropathogenic *E. coli* remains largely unexplored, especially in the Nigerian context. At the Federal Medical Centre (FMC), Owerri, the escalating challenge of antimicrobial resistance necessitates a deeper understanding of local epidemiological patterns and risk factors.

Therefore, this study aims to investigate the relationship between ABO blood group phenotype and the carriage of blaCTX-M, blaTEM, and blaSHV ESBL genes in urinary *Escherichia coli* isolated from female patients at FMC Owerri. Determining whether such an association exists could provide valuable insights into host-pathogen interactions, contribute to the identification of at-risk populations, and inform more personalized infection prevention and empirical treatment strategies in our local setting.

2. MATERIALS AND METHODS

This was a cross-sectional study. The study samples included clinically significant *E. coli* isolated from the urine samples of female patients who visited the Microbiology Laboratory Unit of Federal Medical Centre, Owerri, Imo State Nigeria, between February 2023 and August 2023, and who had their urine samples tested for urinary tract infections.

Ethical Approval was obtained from the Chairman of the Ethical Committee of the Federal Medical Centre, Owerri. Further permission was obtained from the head of the Microbiology Unit of the laboratory and the Medical Laboratory Scientist in charge of urine culture.

Written informed consent and oral consent of the patients were sought. The inclusive criteria were patients whose urine culture showed the growth of *E. coli*, a lactose fermenter which produces acid, turns the colonies and the surrounding medium pink/red on MacConkey agar and which had shown antimicrobial resistance to cephalosporins. The study population also agreed to be enrolled in the study. Exclusive criteria were those cultures which showed clinically insignificant growth of *E. coli*, isolates that were sensitive to cephalosporin and patients that refused to be included in the study.

2.1 Pure Culture

To obtain a pure culture, a sterile wire loop was used to pick a single, well isolated colony which looked like *E. coli* (pink on MacConkey agar) from the primary culture plate. The colony was sub-cultured on a new, freshly prepared Blood agar (Merck Germany) plate using the quadrant streak method to ensure proper separation. The plate was incubated aerobically at 35-37°C for 16-18h hours, and the plate showed growth of one type of colony looking alike in shape, size, colour, and texture therefore confirming purity of the culture. Confirmation tests were carried out to authenticate that it was *E. coli*. Gram stain showed gram-negative rods, and two different biochemical tests namely, indole and oxidase showed positive and negative, respectively.

2.2 ESBL Detection Re-Testing

ESBL was detected again, by a screening test using the Minimum Inhibitory Concentration of the disk diffusion method. All the *E. coli* isolated were tested against an indicator cephalosporin (ceftriaxone 30µg) on Mueller-Hinton agar medium (Merck, Germany) following the Clinical and Laboratory Standard Institute. For quality control, the *E. coli* ATCC 25922 standard strain was used. The isolate is suspected to be an ESBL producer if the zone of inhibition is ≤ 19 mm.

2.3 ESBL Confirmation

The presence of *E. coli* which is an ESBL producer, is confirmed by the use of combination disk test. A standardized *E. coli* suspension was prepared (0.5 McFarland standard) and lawn cultured on Mueller-Hinton agar medium. Four disks were placed which were the disks of cefotaxime (CTX, 30µg) and ceftazidime (CAZ, 30µg) placed alone and the combination disks of cefotaxime + clavulanic acid (CTX/CLA, 30/10µg) and ceftazidime + clavulanic acid (CAZ/CLA, 30/10µg). The plates were cultured aerobically at 35°C. An increase in the zone diameter of ≥ 5 mm for the combination disks versus the antibiotic disk alone confirms the presence of an ESBL.

2.4 ABO Blood Group Phenotype

About 1ml of blood samples were collected by venipuncture from the confirmed carriers of ESBL producing *E. coli* into ethylene di-amine tetracetic acid (EDTA) anti-coagulated tubes for their ABO blood grouping. Red cell phenotyping was carried out using standard tube techniques as described

by Judd (1994) and Brecher (2002). For ABO blood grouping, a drop of Biorad Seralone anti-A, anti-B, and anti-AB (Bio Rad Medical Diagnostics, Germany) each was placed in clean test tubes labelled 1, 2, and 3. To each tube was added, a drop of 5% red blood cell suspension in saline. The contents were gently mixed by tilting back and forth and centrifuged for 30 seconds at 1000g. The cell buttons were re-suspended and observed for agglutination. Agglutination of tested red cells constituted positive results and indicated that the red cells contain the group specific antigen. A smooth cell suspension after re-suspension followed by a microscopic confirmation constituted negative test results.

2.5 Polymerase Chain Reaction for the Detection of the Specific ESBL Genes

Genotypic method using PCR (Polymerase Chain Reaction) was used for the detection of the resistant genes. Genomic DNA was extracted from the pure *E. coli* culture. Specific primers designed to bind to and amplify a unique segment of the genes *bla*CTX-M, *bla*TEM, and *bla*SHIV were used. The PCR product was run on an agarose gel. A band of expected size indicated the presence of the gene.

Step 1: DNA Extraction: The boiling method was employed for the extraction of pure *E. coli* DNA from overnight cultures [5]. A loop of *E. coli* cells was collected from cultures that had been incubated for 18 h on MacConkey agar plates and then suspended in brain heart infusion broth for an additional 18 to 24 h at 37°C. Upon completion of the incubation period, 1.5 ml of the broth sample was subjected to centrifugation at 13,000 rpm for 10 min using BIO RAD micro centrifuge. The supernatant was then discarded, and the resulting pellets were resuspended in 200 µl of Tris-EDTA buffer (TE buffer) and washed twice. Subsequently, 200 µl of TE buffer was added to the pellets, and vortexed to ascertain thorough homogenization. The vortexed mixture was boiled in a dry bath at 100 °C for 10 min. Subsequently, the boiled pellets were kept at -20 °C for 2min. Thereafter, the mixture was centrifuged at 13,000 rpm for 10 min. The supernatant, which contained the DNA, was carefully pipetted to a separate tube using a micropipette and stored at -20 °C for later use. The DNA template for polymerase chain reaction (PCR) analysis was approximately 2 µl of the isolate supernatant [6].

STEP 2: Polymerase Chain Reaction (PCR): The ESBLs encoding genes (*bla*SHV, *bla*TEM, and *bla*CTX-M) were amplified individually on a PCR thermocycler (Qiagen, CA) using the specific primers (Metabion Co, Germany). The Table 1 displays the list of primer sequences. The amplification reaction was performed in a final volume of 20 µL containing Master Mix (Bioneer, South Korea), primers at concentrations of 10 pM, 50–100 ng of extracted DNA templates, and ddH₂O. The PCR conditions for the amplifications were as follows, 5 min at 95°C for the initial denaturation step; 30 cycles of 30 sec at 95°C for DNA denaturation, 30 sec for primer annealing. The temperature depended on the sequences of primers (primer extension at 72 °C for 1 min) and a final extension of 5 min at 72°C. For each run, the PCR program was run with a cycle number of 35 without the use of DNA template as negative control. Electrophoresis was used to separate the amplification products in a 1.5% agarose gel prepared in prepared in 1X TBE(Tris/Boric/EDTA) buffer. After electrophoresis, amplification products were visualized under an ultra-violet trans-illuminator light after staining with safe stain (Thermo Fisher Scientific, India).

Table 1. The Primer Sequences of the ESBL Genes Amplified by the PCR [41].

	Primers	Sequences 5'→3'	Size (bp)	
SHV	F	TCAGCGAAAAACACCTTG	471	
	R	CCCGCAGATAAATCACCA		
TEM	F	GAGTATTCAACATTTCCTGTGC	861	
	R	TAATCAGTGAGGCACCTATCTC		
CTX-M	F	TTTGCGATGTGCAGTACCAGTAA	544	
	R	CGATATCGTTGGTGGTGCCATA		

F: Forward, R: Reverse

2.6 Statistical Analysis

Statistical Analysis was performed using SPSS™ version 21.0 (IBM Corp., USA). Using descriptive statistics Chi-Square or Fisher's exact tests were used to analyse the data where appropriate. A p-value of <0.05 was considered statistically significant.

3. RESULTS AND DISCUSSION

The main objective of the present study was to characterize the molecular profile of ESBL-producing isolates and investigate any potential relationship with the ABO blood group system.

Table 2. Distribution Of ABO Blood Group Among the Study Population (n =43)

BLOOD GROUP	FREQUENCY (n)	PERCENTAGE (%)
O	21	48.8
A	12	27.9
B	6	13.9
AB	4	9.3

The findings in Table 2 reveals that blood group O was the most dominant blood group among the participants, constituting 21 out of 43 individuals (48.8%). This shows that nearly half of the study population belonged to this blood group. The other blood groups, blood group A was the second most common, represented by 12 individuals, accounting for 27.9% of the cohort. This was followed by blood group B (6 individuals, 13.9%) and blood group AB, which was the least common (4 individuals, 9.3%).

The initial demographic and phenotypic characterization of our study cohort, as summarized in Table 2, reveals a unique distribution of ABO blood groups. The most striking finding from this baseline data is the predominance of Blood Group O, which accounted for almost half of the study population (48.8%, n=21). This blood group was followed by Blood Group A (27.9%, n=12), with Blood Groups B (13.9%, n=6) and AB (9.3%, n=4) being the least represented. This observed hierarchy—O > A > B > AB—is consistent with the well-established seroprevalence patterns reported in numerous populations worldwide, particularly in those of Nigeria [43, 1, 34, 3, 35] African [27, 16, 17, 48] and European descent [47]. However, high frequencies of blood group A have been reported in USA and Morrocco [10]. This alignment suggests that our study cohort is, from a blood group perspective, representative of the broader population from which it was drawn, thereby improving the fact that our subsequent findings concerning ESBL genes can be generalized. The high prevalence of Blood Group O in our cohort is not merely a demographic footnote; it carries significant biological and epidemiological implications. The relationship between blood groups and intestinal microbe has been reported [14]. Blood group antigens are not only present on red blood cells but also expressed on the surface of mucosal epithelial cells, where they can act as receptors or co-receptors for various bacterial pathogens [30]. Increased number of patients in blood group AB with infections caused by *Escherichia coli* and *Klebsiella pneumonia* has been reported and it concluded that an individual's blood group may be a significant factor in the host-response to bacterial invasion and influence the development of infection with some gram-negative bacilli [40]. The O antigen, or the absence of A and B antigens in individuals with Blood Group O, has been linked in previous literature to altered susceptibility to certain infections. For instance, some studies have reported that individuals with Blood Group O may have a higher risk of infection with *Helicobacter pylori* [32] or more severe manifestations of *Vibrio cholerae* and Norovirus [7].

In the context of our study, this high frequency of Blood Group O gives a significant sub-population for a concrete comparative analysis. When we later investigate the distribution of ESBL genes like blaCTX-M, the fact that almost half of our isolates come from Blood Group O individuals allows for a statistically meaningful comparison against non-O groups. Should a significant association be found, the pre-existing biological plausibility regarding blood group antigens and bacterial attachment would provide a compelling framework for the result interpretation. Conversely, the finding of no association would be equally strong, suggesting that the mechanisms of ESBL-producing bacterial colonization and infection are independent of the ABO blood group interface. Summarily for Table 2, the blood group profile of our study population establishes a representative baseline and sets the stage for a meaningful investigation into the potential interplay between host genetic factors, like ABO status, and the molecular determinants of antimicrobial resistance.

TABLE 3. Prevalence and Distribution of ESBL Genes among the Study Population (n=43)

ESBL GENE	FREQUENCY (n)	PERCENTAGE (%)
blaCTX-M	31	70.1
blaTEM	23	53.5
blaSHV	8	18.6

Considering Table 3, the Prevalence and Distribution of ESBL Genes, all 43 isolates were phenotypically confirmed as ESBL producers prior to PCR. The molecular analysis revealed a high prevalence of ESBL-encoding genes. The blaCTX-M gene was the most dominant, detected in 31 out of 43 isolates (81.4%). This was followed by the blaTEM gene where 23 isolates (51.2%) were detected and finally, the blaSHV gene was the least common, present in only 8 isolates (20.9%).

In Table 3, the molecular analysis of the 43 ESBL-producing isolates revealed a clear hierarchy in the prevalence of ESBL genes. The blaCTX-M gene was the most predominant, identified in 31 isolates (70.1%). This was followed by the blaTEM gene, found in 23 isolates (53.5%). The blaSHV gene was the least common, present in only 8 isolates (18.6%). The blaCTX-M as the dominating gene in the present study agrees with the previous research done worldwide [8, 22, 13, 37]. However, The recent findings contradict the findings of [25, 41, 38, 4] carried out in Iran where blaTEM genes were the most prevalent while the blaSHV was the least discovered among ESBL-Producing *Escherichia coli* Isolated from Thalassemia Patients in Erbil, Iraq. This result is not an anomaly but rather a reflection of a well-documented worldwide pandemic of CTX-M-type enzymes. Since the early 2000s, CTX-

M enzymes have progressively replaced the classical TEM and SHV derivatives as the most prevalent ESBLs in both hospital and community settings globally [22, 13]. The dominance of CTX- M gene in our study, conducted at Federal Medical Centre, Owerri, confirms that this facility is experiencing the same trend. The success of blaCTX-M is often linked to its location on highly mobile genetic elements, such as plasmids, which sometimes carry additional resistance genes for other antibiotic classes (for example, fluoroquinolones, aminoglycosides), accelerating their rapid spread and persistence across bacterial populations [23, 29]. In this research, the blaTEM gene was the second most common ESBL determinant, found in 23 isolates (53.5%). While its prevalence is substantial, it is notable that it is lower than blaCTX-M. TEM-type enzymes were historically the first ESBLs to be identified. Their continued high frequency indicates their persistent circulation in the bacterial population [13]. It is common to find blaTEM genes co-harboured with blaCTX-M on the same plasmid, a genetic arrangement that can confer a fitness advantage or a broader resistance profile, a finding supported by the high rate of co-occurrence shown in our Table 4.

In contrast, the blaSHV gene was the least prevalent, identified in only 8 isolates (18.6%). This is consistent with the general observation that SHV-type ESBLs are more typically associated with nosocomial outbreaks of *Klebsiella pneumoniae* [26] and are less frequently found in community-acquired *E. coli* isolates, which often dominate urinary tract infection samples. The lower prevalence of blaSHV in our study could reflect the specific bacterial species in our cohort or the local antibiotic selection pressures that favour blaCTX-M and blaTEM carrying strains.

Table 4. Distribution and Co-occurrence of ESBL Genes (n=43)

Genetic Profile	Frequency (n)	Percentage (%)
blaCTX-M (only)	10	23.3
blaTEM (only)	6	14.0
blaSHV (only)	2	4.6
blaCTX-M + blaTEM	15	34.9
blaCTX-M + blaSHV	5	11.6
blaTEM + blaSHV	1	2.3
blaCTX-M + blaTEM + bla SHV	4	9.3

The Table 4 shows the distribution and co-occurrence of ESBL Genes. In this table, the genes often appear together. More than half of the samples (58.1%) had more than one ESBL gene. The most common combination was blaCTX-M + blaTEM. This pair was found in 15 samples (34.9%), making it the most frequent genetic profile overall. blaCTX-M is the most important gene. It was present in most samples, either alone (10 samples) or in combination with other genes. blaSHV is the least common. It was rarely found alone (only 2 samples) and was the least frequent gene in combinations. In short, the bacteria in this study typically carried multiple resistance genes, with blaCTX-M and blaTEM being the dominant pair.

In Table 4, the analysis of gene co-occurrence revealed that a majority of isolates (58.1%) harbored more than one ESBL gene. The most frequent genetic profile was the co-carriage of blaCTX-M and blaTEM, found in 15 isolates (34.9%). The blaCTX-M-only profile was the second most common (10 isolates, 23.3%). In contrast, profiles involving blaSHV without blaCTX-M were rare, with blaTEM+blaSHV being the least common combination (1 isolate, 2.3%). This demonstrates a landscape of multi-gene resistance dominated by blaCTX-M. The analysis of ESBL gene co-occurrence in this study reveals a complex and concerning resistance landscape that extends beyond the simple presence of these enzymes. The most salient finding is that a majority of the clinical isolates (58.1%) are carriers of more than one ESBL gene. This high frequency of co-carriage indicates a significant accumulation of resistance determinants within the bacterial population at Federal Medical Centre, Owerri, a phenomenon with profound implications for public health and clinical management.

The most prevalent genetic profile was the simultaneous presence of blaCTX-M and blaTEM, accounting for over a third of all isolates (34.9%). This is not a random occurrence but likely reflects a successful evolutionary strategy. These genes are often co-located on the same mobile genetic elements, such as conjugative plasmids [12, 44]. This genetic linkage creates a horizontal gene transfer where selection pressure from one cephalosporin can simultaneously promote the spread of both genes. Furthermore, the combination of blaCTX-M, which confers high-level resistance to cefotaxime and ceftriaxone, with the broader spectrum of blaTEM, may provide a synergistic advantage, ensuring bacterial survival against a wider array of beta-lactam antibiotics [28]. The finding that the blaCTX-M + blaTEM combination was more common than blaCTX-M alone underscores that multi-gene resistance is the rule rather than the exception in this setting [42]. In a clear contrast, the low prevalence of blaSHV and its rare occurrence in profiles without blaCTX-M (e.g., blaTEM+blaSHV was only 2.3%) suggest it plays a more minor and likely dependent role in this particular bacterial population. This may reflect the specific epidemiology of our isolates, which were primarily from community-acquired urinary *E. coli* infections in Owerri, a context where SHV-type ESBLs are less commonly the dominant genotype compared to hospital-based *Klebsiella* outbreaks [36].

Table 5. Cross Tabulation for the Association of ESBL Genes with ABO Blood Groups (n=43)

Blood Group	Total Isolates	blaCTX-M Positive (n, %)	blaTEM Positive (n, %)	blaSHV Positive (n, %)
O	21	16 (76.2%)	11 (52.4%)	4 (19.0%)
A	12	8 (66.7%)	7 (58.3 %)	2 (16.7%)
B	6	4 (66.7%)	3 (50.0%)	1 (16.7%)
AB	4	3 (75.0%)	2 (50.0%)	1 (25.0%)
Total	43	31 (70.1%)	23 (53.3%)	8(18.6%)

The association of ESBL Genes with ABO Blood Groups is presented in Table 5. The analysis reveals that the blaCTX-M gene was the most prevalent across all blood groups, with its highest occurrence in Blood Group O (16 out of 21 isolates, 76.2%) and Blood Group AB (3 out of 4 isolates, 75.0%). The blaTEM gene was also widely distributed, showing a relatively high and consistent prevalence across groups O (52.4%), A (58.3%), and B (50.0%). In contrast, the blaSHV gene was the least common in all groups, with a low and fairly uniform prevalence ranging from 16.7% to 25.0%. Visually, no blood group demonstrated a uniquely low susceptibility to any specific ESBL gene. The prevalence of all three genes was consistently present in every blood group, with blaCTX-M being the dominant genotype regardless of the host's ABO phenotype.

In Table 5, the blaCTX-M gene was the most prevalent ESBL gene across all blood groups, with its highest frequency in group O (76.2%). The prevalence of blaTEM and blaSHV was lower and relatively consistent across all groups. No blood group was free from any of the tested ESBL genes, indicating a wide distribution of these resistance determinants throughout the patient population. The investigation into a potential association between ABO blood group and the carriage of specific ESBL genes reveals a landscape of resistance that appears largely independent of this particular host genetic factor. The most notable finding from this study is the ubiquitous and dominant presence of the blaCTX-M gene across all blood groups, ranging from 66.7% to 76.2%. This consistency strongly suggests that the forces driving the success of blaCTX-M such as its location on highly mobile genetic elements and potent hydrolysis of third-generation cephalosporins—are so powerful that they outweigh any potential, minor influence of ABO blood group antigens on infection susceptibility [39].

4. CONCLUSION

In conclusion, while the ABO blood group system influences susceptibility to several pathogens, our data suggest it does not play a major role in determining the genetic profile of ESBL-producing *E. coli* in female patients with urinary tract infections at FMC Owerri. For urinary tract infections caused by *E. coli*, other virulence factors, such as P-fimbriae, play a more direct role in attachment to the uroepithelium than ABO antigens, which may explain the lack of a strong association in our findings [9]. The resistance landscape remains dominated by the pervasive blaCTX-M gene, a challenge that requires broad-spectrum public health and clinical interventions. Furthermore, the high rate of ESBL producers, dominated by blaCTX-M, signals a significant public health challenge for Owerri and similar regions. It underscores the urgent need for robust infection prevention and control measures within the hospital to prevent cross-transmission. It also highlights the consequences of antibiotic misuse in both the community and hospital settings, which drives the selection and propagation of these resistant clones.

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