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Phenotypic detection of ESBL production and colistin resistance in *Escherichia coli* from urinary tract infections in Kano Metropolis, Nigeria

Aisha Mohammed and Bashir Ado Kurawa

Department of Pharmaceutical Microbiology and Biotechnology, Faculty of Pharmaceutical Sciences, Bayero University, Kano, Nigeria

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ABSTRACT

Background: Antimicrobial-resistant bacteria, particularly *Escherichia coli* producing extended-spectrum β-lactamases (ESBLs) and the mcr-1 gene, can cause urinary tract infections (UTIs) that may be fatal due to the lack of effective treatment options. This study aimed to determine the co-occurrence of colistin resistance genes in ESBL-producing *E. coli* and to assess their antibiotic susceptibility patterns in suspected UTI cases at Aminu Kano Teaching Hospital, Kano, Nigeria.

***Methods*:** Seventy-one (71) *E. coli* isolates obtained from patients with suspected UTIs were studied. The identity of the isolates was confirmed using standard biochemical tests. Antibiotic susceptibility testing was performed using the Kirby–Bauer disc diffusion method. Screening for ESBL production and colistin resistance was conducted using Clinical and Laboratory Standards Institute (CLSI) breakpoints. Suspected ESBL producers were confirmed using the double-disc synergy test. Standard antibiotic discs—Augmentin (AMC, 30 µg; Oxoid, England), Ceftazidime (CAZ, 30 µg; Oxoid, England), and Cefotaxime (CTX, 30 µg; Oxoid, England)—were used for screening and confirmation.

Results: Screening for ESBL production indicated that 67.6% of isolates were suspected ESBL producers, while the double-disc synergy test confirmed 22.9% as ESBL-producing *E. coli*. Co-production of ESBLs and the colistin resistance gene was observed in 9.1% of isolates. Antimicrobial susceptibility testing revealed 100% resistance to Augmentin, ceftriaxone, ceftazidime, and cefotaxime; resistance to gentamicin was 90.1%, chloramphenicol 72.7%, nitrofurantoin 54.5%, ciprofloxacin 90.9%, and cotrimoxazole 90.9%. All isolates were 100% sensitive to imipenem.

Conclusion: ESBL-producing *E. coli* are present in Aminu Kano Teaching Hospital and exhibit high resistance to commonly prescribed antibiotics, underscoring the need for continuous surveillance and prudent antibiotic use.

***Keywords*:** Colistin, *Escherichia coli*, Extended-spectrum β-lactamase, Imipenem, Urinary tract infection

1. INTRODUCTION

Urinary tract infections (UTIs) are among the most common infectious diseases worldwide, affecting approximately 250 million people each year [1]. Gram-negative pathogens, primarily members of the Enterobacteriaceae family—most notably *Escherichia coli*, followed by *Klebsiella pneumoniae* and Proteus mirabilis—are the predominant causative agents [2]. Studies have demonstrated that antibiotic use influences both the development and emergence of multidrug-resistant microorganisms. Uropathogenic E. coli has increasingly shown resistance to most antibiotics, particularly cephalosporins, due to the production of enzymes known as extended-spectrum β-lactamases (ESBLs) [3–5]. ESBLs are β-lactamases capable of hydrolysing oxyimino cephalosporins and are inhibited by β-lactamase inhibitors [5]. The misuse and overuse of antibiotics have contributed to a global increase in ESBL-producing E. coli [7]. In many regions, 10%–40% of *E. coli* and *K. pneumoniae* strains produce ESBLs [8]. In Nigeria, prevalence rates range from 5% to 44.3% [9–13]. With

***Corresponding author: Email:** amohammed.phb@buk.edu.ng **; Phone: +2348035897992**

changes in the aetiology of UTIs and in the antibiotic resistance patterns of uropathogens over recent years, therapeutic options for treating UTIs have become increasingly limited. Colistin is considered a last-resort antibiotic for infections caused by multidrug-resistant Gram-negative bacteria [14]. However, its effectiveness is threatened by the emergence of resistance mediated by mobile colistin resistance (mcr) genes [15]. Although the clinical significance of ESBLs and mcr genes is well established, there is limited knowledge regarding the prevalence and distribution of mcr genes among ESBL-producing colistin-resistant isolates in low-income countries such as Nigeria [16]. ESBL-producing bacteria are associated with increased mortality, prolonged hospital stays, and higher healthcare costs [17]. Unfortunately, colistin-resistant infections have now spread globally, largely due to its overuse and misuse in both human and veterinary medicine. Alarmingly, resistance can develop even in the absence of prior colistin exposure, leaving clinicians unprepared to manage such cases [18]. Polymyxins, including colistin, are often the only remaining treatment option for life-threatening infections caused by multidrug-resistant Gram-negative bacteria [19]. Reports indicate an increasing frequency of colistin resistance in Enterobacteriaceae. Because multidrug-resistant bacteria can develop colistin resistance through mutation or adaptive processes, clinicians must remain vigilant. The scientific community, healthcare specialists, government authorities, and public–private partnerships have all advocated for restricting colistin use to last-resort situations in order to slow resistance development [14]. Globally, ESBL-producing pathogens are a growing concern [20]. The co-production of ESBL and colistin resistance genes has significant therapeutic implications, as such isolates often exhibit resistance to multiple antibiotic classes, severely limiting treatment options. This increases the risk of treatment failure and adverse patient outcomes. Molecular characterisation of colistin- and ESBL-resistance genes is crucial to understanding the genetic mechanisms underlying resistance and its spread [21]. Investigating the prevalence, genetic diversity, and potential transmission mechanisms of mcr genes in colistin-resistant isolates is essential for improving infection control, antimicrobial stewardship, and treatment strategies [22]. Therefore, this study aimed to determine the co-production of colistin resistance genes in ESBL-producing E. coli from suspected UTI cases at Aminu Kano Teaching Hospital using phenotypic techniques. The findings will be valuable for guiding empirical therapy and optimising the management of UTI patients, as well as supporting the development of stronger antibiotic stewardship programmes.

2. MATERIALS AND METHODS

2.1 Materials

Mueller Hinton Agar(Bio-Rad), Nutrient Agar (HiMedia), Oxoid Antibiotic discs, MacConkey Agar, Kovacs indole reagent, Simmon's Citrate Agar Petri dishes, Oven, Incubator,

2.2 Methods

2.2.1 Sample Collection and Analysis

The study was conducted on 71 isolates of uropathogenic *Escherichia* coli obtained from patients with suspected UTIs at Aminu Kano Teaching Hospital between August and December 2024, following approval from the hospital's Ethical Committee. Urine microscopy was performed by placing a drop of uncentrifuged urine on a clean glass slide, carefully placing a coverslip over it, and examining it under high power (40×) to determine significant pyuria. The samples were inoculated onto Cysteine Lactose Electrolyte Deficient (CLED) agar and incubated at 37°C for 18–24 hours. Discrete colonies were picked and subjected to Gram staining. All isolates were identified to the species level using standard biochemical tests [23].

2.2.2 Antibiotic Susceptibility Testing

Antibiotic susceptibility was determined using the Kirby–Bauer CLSI-modified Disc Agar Diffusion (DAD) technique. One millilitre (1.0 ml) of a standardised overnight culture of each isolate (containing 1.5×10^8 CFU/ml) was used to flood the surface of Mueller–Hinton Agar (MHA) plates. Excess inoculum was drained off and the plates were allowed to dry with the Petri dish lid in place. Standard antibiotic discs were aseptically placed at appropriate equidistances on the inoculated MHA plates and allowed to stand for 1 hour. The plates (prepared in duplicate for each isolate) were then incubated at 37°C for 18 hours. The diameters of the inhibition zones produced by each antibiotic disc were measured and recorded [24]. The following Oxoid antibiotic discs were used: amoxicillin/clavulanic acid (20/10 µg), ceftriaxone (CRO, 30 µg), cefotaxime (CTX, 30 µg), ceftazidime (CAZ, 30 µg), ciprofloxacin (CIP, 10 µg), chloramphenicol (C, 30 µg), gentamicin (CN, 10 µg), cotrimoxazole (STX, 1.25/23.75 µg), imipenem (10 µg), and nitrofurantoin (300 µg).

2.2.3 Extended-Spectrum Beta-Lactamase (ESBL) Screening Test

Screening for ESBL production was carried out by disc diffusion, following CLSI (2016) guidelines. Resistance to cefotaxime, ceftriaxone, and ceftazidime was assessed. From pure overnight cultures grown on MacConkey agar, a bacterial suspension equivalent to a 0.5 McFarland standard (1.5×10^8 CFU/ml) was prepared in nutrient broth. Using a sterile cotton swab, the bacteria were spread on Mueller–Hinton agar to obtain a lawn culture. After allowing the plates to dry, antibiotic discs were placed on the surface, and the plates were incubated at 37°C for 18–24 hours. The diameters of the inhibition zones around the discs were measured and recorded. Resistance breakpoints were defined as follows: cefotaxime (30 µg) ≤ 27 mm, ceftriaxone (30 µg) ≤ 25 mm, and ceftazidime (30 µg) (breakpoint not provided in original but implied). Resistance to at least one of these antibiotics was considered positive in the screening test for possible ESBL production [25].

2.2.4 Confirmation of ESBL Production by Double Disc Synergy Test (DDST)

The Double Disc Synergy Test was performed using three antibiotics: amoxicillin–clavulanic acid (20/10 µg), cefotaxime (30 µg), and ceftazidime (30 µg). The cephalosporin discs were placed 25 mm (centre-to-centre) from the amoxicillin–clavulanic acid disc on Mueller–Hinton Agar. Enhancement of the inhibition zone towards the clavulanate disc after 24 hours of incubation at 37°C was interpreted as indicative of ESBL production [26].

2.2.5 Phenotypic Detection of Colistin Resistance

The colistin agar dilution method, following CLSI guidelines, was used for phenotypic detection of colistin resistance at a concentration of 3 µg/ml of colistin sulphate [27]. Using an analytical balance, 3 mg of colistin sulphate powder was aseptically weighed and dissolved in 500 ml of distilled water. Separately, 38 g of Mueller–Hinton agar powder was suspended in 500 ml of distilled water, boiled on a hot plate until fully dissolved, and autoclaved at 121°C for 15 minutes. After cooling, the colistin sulphate stock solution was thoroughly mixed into the agar before aseptically pouring 20 ml into sterile Petri dishes, which were then allowed to solidify at room temperature. Using sterile swabs dipped in bacterial inocula adjusted to the 0.5 McFarland standard, spots of approximately 20 mm in diameter were created on the agar surface. Following 18–20 hours of inverted incubation at 35°C, the presence of growth (one or more colonies) was interpreted as indicative of resistance.

2.3 Statistical Analysis

The Chi-square test was used to compare categorical data, using SPSS Statistics (version 17, IBM Corporation, NY, USA). A p-value of < 0.05 was considered statistically significant.

3. RESULTS

A total of 500 suspected urine samples were collected from Aminu Kano Teaching Hospital. Figure 1 shows the distribution of samples by gender. Samples obtained from male patients were 106 (21.2%), while those obtained from female patients were 394 (78.8%). There was a significant difference between genders at the sampling site ($p = 0.0178$), with the female population showing a higher rate of suspected urinary tract infections (UTIs).

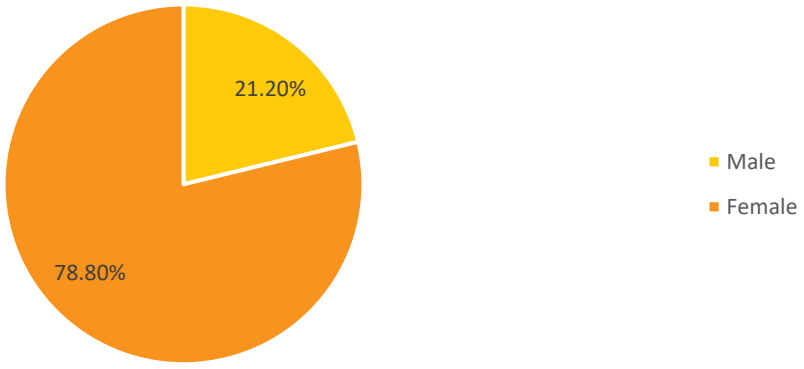

Figure 1: Distribution of samples based on gender

Of the 500 samples collected, 307 showed growth of organisms. A total of 71 *Escherichia coli* isolates were obtained, making it the most frequently isolated organism. This was followed by *Klebsiella pneumoniae*, with 68 isolates. Table 1 below shows the distribution of isolates at the sampling site.

Table 1 Distribution of isolates at the sampling site

Organism	Number of Occurrence	Percentage (%)
E. coli	71	36.8
K. pneumoniae	68	35.2
K. oxytoca	4	2.1
Pr. mirabilis	15	7.8
P. aeruginosa	16	8.3
E. faecalis	8	4.1
S.saprophyticus	6	3.1
S. aureus	5	2.6
No growth	307	
Total	500	

Based on the CLSI breakpoints for screening ESBL production using ceftriaxone, ceftazidime, and cefotaxime, Table 2 below shows the distribution of isolates as suspected ESBLs and non-ESBLs, with 67.6% of *E. coli* suspected to be ESBL producers.

Table 2 Distribution of Suspected ESBLs producing *E. coli* based on CLSI breakpoint

Isolates	Number of isolates (%)
Suspected ESBLs producers	48 (67.6)
Non ESBLs producers	23 (32.4)
Total	71

Table 3 shows percentage distribution of confirmed ESBL producers using the Double Disc Synergy Test. ESBL production was confirmed in 22.9% of *E. coli* isolates.

Table 3 Distribution of Confirmed ESBLs and Non-ESBLs *E. coli* using Double Disc Synergy Test

Isolates	Number of isolates (%)
Confirmed ESBLs producers	11 (22.9)
Non ESBLs producers	37 (77.1)
Total	48

The antimicrobial susceptibility pattern of ESBL-producing *E. coli* is presented in Table 5, with 100% of the isolates resistant to ceftazidime, cefotaxime, augmentin, and ceftriaxone. All isolates were sensitive to imipenem. Resistance rates to gentamicin, chloramphenicol, nitrofurantoin, ciprofloxacin, and cotrimoxazole were 90.1%, 72.7%, 54.5%, 90.9%, and 90.9%, respectively.

Table 6: Antimicrobial Susceptibility Pattern of ESBLs producing *E. coli* (n=11)

	Organism (*E.coli*)	
Antibiotic	Sensitive (%)	Resistant (%)
Gentamicin (10µg)	1 (9.1)	10 (90.1)
Ceftazidime (30µg)	0 (0)	11 (100)
Chloramphenicol(30µg)	3 (27.3)	8 (72.7)
Cefotaxime(30µg)	0 (0)	11 (100)
Nitrofurantoin(300µg)	5 (45.5)	6 (54.5)
Ciprofloxacin (5µg)	1(9.1)	10 (90.9)
Augmentin (30µg)	0 (0)	11 (100)
Imipenem (10µg)	11 (100)	0 (0)
Ceftriaxone(30µg)	0 (0)	11 (100)
Cotrimoxazole(1.25/23.75µg)	1 (9.1)	10 (90.9)

Based on the colistin and ESBL phenotypic data, Figure 2 shows 1 (9.1%) of the isolates coproduced colistin and ESBL resistance, whereas 90.9% (10) did not coproduce.

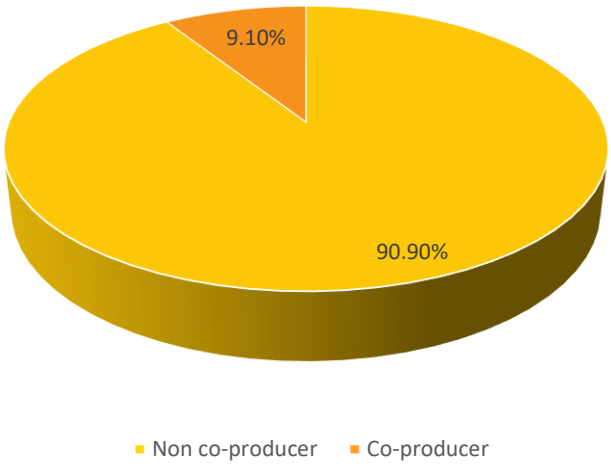

Figure 2: Percentage of phenotypic coproducers

4. DISCUSSION

In this study, a total of 500 urine samples were collected from Aminu Kano Teaching Hospital—106 from males and 394 from females—out of which 193 showed bacterial growth. A significant difference was observed between samples collected from males and females. Previous reports have shown that females are more prone to UTIs than males [20, 28–34]. The higher frequency in females has been attributed to the shorter female urethra and its proximity to the gastrointestinal outlet, which facilitates colonization by enteric flora [35]. *Escherichia coli* was the most prevalent uropathogen identified in this study, consistent with findings from previous reports [36–42]. The distribution of confirmed ESBL-producing *E. coli* was 22.9%, compared to the suspected rate of 67.6%. Similar reductions in confirmed ESBL production relative to suspected cases have been reported, possibly due to the production of multiple β-lactamases that interfere with confirmatory test results [43]. Yushau et al. reported a higher prevalence, with ESBL-producing *E. coli* accounting for 42.6% of isolates [43]. In Pakistan, 56.9% of *E. coli* isolates were ESBL-positive [44], while a study in India found that nearly 40% of urinary *E. coli* isolates were ESBL-positive [45]. Mekki et al. reported 53% ESBL positivity among *E. coli* from patients with urinary tract infections [46], and Ejaz et al. reported a prevalence of 57.4% in Pakistan [47]. Variations in prevalence rates across studies may be due to differences in sample size, population characteristics, or testing protocols. In this study, ESBL-producing isolates exhibited high resistance to both β-lactam and non-β-lactam antibiotics. This pattern suggests cross-resistance induced by ESBL enzymes, affecting susceptibility to non-β-lactam antibiotics such as gentamicin, nitrofurantoin, cotrimoxazole, and ciprofloxacin [48]. Imipenem was the only antibiotic active against 100% of ESBL-producing *E. coli*, followed by nitrofurantoin, which showed activity against 45.5% of isolates. Similar findings have been reported by Chaturvedi et al. [49]. Colistin co-resistance was detected in only one of the eleven phenotypically confirmed ESBL-producing *E. coli* isolates. Previous studies, however, have reported higher rates of colistin resistance. For example, a study in Oyo State, Nigeria, found that 84% of *E. coli* and *Klebsiella* spp. clinical isolates from blood samples of both outpatients and inpatients were colistin-resistant [50], while 43.6% resistance was reported in Lagos [51]. Phenotypic analysis in this study showed co-production of ESBL and colistin resistance in 13.9% of isolates, but no co-production was detected at the molecular level following genetic characterization [52]. This discrepancy may be due to variations in genetic backgrounds among strains, allowing isolates to exhibit phenotypic co-resistance without harboring known colistin-resistance genes.

5. CONCLUSION

This study demonstrated a higher occurrence of UTIs in females compared to males, with *Escherichia coli* being the most frequently isolated organism. *E. coli* isolates from UTIs exhibited high resistance to commonly prescribed antibiotics. Imipenem showed excellent activity against ESBL-positive isolates and is recommended as the treatment of choice for confirmed ESBL producers. The high rate of resistance to empirically prescribed antibiotics, including cotrimoxazole, fluoroquinolones, amoxiclav, and broad-spectrum cephalosporins, underscores the need for routine ESBL screening in clinical laboratories. Antibiogram reports should explicitly

indicate whether an isolate is a confirmed or suspected ESBL producer. Regardless of in vitro susceptibility, clinicians should be aware that ESBL production can render β-lactam antibiotics clinically ineffective. To reduce the prevalence of antimicrobial-resistant bacteria, including ESBL-producing *E. coli*, effective infection control measures—such as barrier precautions and mandatory hand hygiene—must be strictly implemented.

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Conflicts of Interest
The authors declare no conflicts of interest.

Author Contributions
Aisha Mohammed conducted the research and drafted the manuscript; Bashir Ado Kurawa critically reviewed and refined the manuscript

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