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Antimicrobial Resistance Patterns among Uropathogens Isolated from Positive Urine Cultures: A Cross-Sectional Study

Ibrahim O. Eswaey[1,*], Mohamed A. Alghoj[2], Mohamed Abu Sneana[3,4], Hadeel Izeit[5,6], Johnny M. Jwely[7]

[1]Department of Medical Laboratories, Faculty of Medical Technology, Sabratha University, Libya.

[2]Department of Diabetes and Endocrinology, Faculty of Medicine, University of Zawia, Libya.

[3]Department of Biological Sciences/Biomedical Sciences, Libyan Academy for postgraduate,Janzur, Libya.

[4]The Multidisciplinary Clinic of Zawia, Libya.

[5]Department of Genetic, Faculty of Science, University of Misurata, Libya.

[6]Department of Biological Sciences/Biomedical Sciences, Libyan Academy Misrata, Libya.

[7]Department of Botany, Faculty of Science, University of Zintan, Libya.

*Corresponding author: Ibrahim O. Eswaey, Email: ibrahimomer.elgrady88@gmail.com

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Abstract

Background: Urinary tract infections (UTIs) stand as one of the most frequently encountered bacterial infections worldwide, yet the growing challenge of antimicrobial resistance has made treatment increasingly difficult. This study set out to examine which bacteria are most commonly responsible for UTIs in our region and how susceptible they remain to available antibiotics.

Materials and Methods: We analyzed 150 urine samples that tested positive for bacterial growth, collected between 2022 and 2024 from various private clinics in Zawia, Libya. Standard laboratory techniques were employed to identify the bacteria and test their susceptibility to 13 different antimicrobial agents. We then organized our findings according to bacterial species, patient characteristics, and resistance profiles.

Results: *Escherichia coli* emerged as the leading culprit, accounting for 58 isolates (38.7%), followed by *Klebsiella* species (36 isolates, 24.0%), *Proteus mirabilis* (12 isolates, 8.0%), *Staphylococcus aureus* (11 isolates, 7.3%), and *Pseudomonas* species (10 isolates, 6.7%). Women represented nearly three-quarters of cases (72.7%). The highest resistance rates were observed with amoxicillin (88.7%) and ampicillin (85.3%), while carbapenems specifically imipenem showed the lowest resistance at just 4.7%. Notably, 43.3% of *E. coli* and *Klebsiella* isolates displayed the extended-spectrum beta-lactamase (ESBL) phenotype, evidenced by their resistance to third-generation cephalosporins. More than 40% of *E. coli* isolates were resistant to fluoroquinolones.

Conclusions: Our findings reveal troubling levels of antimicrobial resistance, particularly against first-line treatment options, highlighting the pressing need for improved antibiotic stewardship and locally tailored empirical therapy guidelines. Carbapenems and amikacin continue to represent effective choices for severe infections, though their careful use is essential to maintain their effectiveness.

Keywords: Urinary tract infection, antimicrobial resistance, ESBL, *E. coli*, *Klebsiella*, empirical therapy.

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INTRODUCTION

Urinary tract infections rank among the most common bacterial infections clinicians face, affecting millions of people each year across healthcare settings worldwide.[1] These infections span a spectrum from simple bladder

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infections to potentially life-threatening bloodstream infections, with successful treatment hinging critically on selecting the appropriate antibiotic.[2] The rise and spread of antimicrobial resistance (AMR) among the bacteria causing these infections has complicated treatment decisions considerably, contributing to increased illness, deaths, and healthcare expenditures.[3]

Escherichia coli continues to dominate as the primary cause of UTIs, whether acquired in the community or during hospital stays, responsible for 75-90% of uncomplicated cases.[4] Nevertheless, other members of the Enterobacteriaceae family such as *Klebsiella pneumoniae* and *Proteus mirabilis*, along with Gram-positive organisms like *Staphylococcus aureus*, account for substantial portions of complicated UTIs and those associated with catheter use.[5] The worldwide dissemination of extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae has severely undermined the effectiveness of third-generation cephalosporins drugs long considered frontline options for kidney infections.[6]

Surveillance data specific to particular regions prove invaluable for guiding treatment decisions when culture results are not yet available, since resistance patterns differ markedly depending on geography, healthcare environment, and patient population.[7] The World Health Organization has classified AMR among the ten most significant threats to global public health, making ongoing local monitoring of resistance trends essential.[8] This study offers current data on which organisms are causing UTIs in our area and how resistant they have become, with the goal of informing evidence-based treatment recommendations for our region.

MATERIALS AND METHODS

Study Design and Setting

We carried out a cross-sectional study examining positive urine cultures from patients showing clinical signs of UTIs who presented at tertiary care facilities between January 2022 and December 2024.

Sample Collection and Processing

Outpatients with symptoms provided midstream clean-catch urine samples, while hospitalized patients contributed catheterized specimens. Laboratory staff processed these samples within two hours, plating them on Cystine Lactose Electrolyte Deficient (CLED) agar and blood agar. After incubating at 35-37°C for 24-48 hours under aerobic conditions, technicians identified bacteria based on their appearance, Gram stain characteristics, and biochemical testing including catalase, oxidase, and API 20E/NE systems.[9] *Staphylococcus aureus* was confirmed through coagulase testing.

Antimicrobial Susceptibility Testing

Using the Kirby-Bauer disc diffusion method following Clinical and Laboratory Standards Institute (CLSI) protocols,[10] we tested susceptibility to: amoxicillin (AX, 25µg), ampicillin (AM, 10µg), amoxicillin-clavulanic acid (AMC, 20/10µg), ciprofloxacin (CIP, 5µg), ceftriaxone (CRO, 30µg), ceftazidime (CAZ, 30µg), cefotaxime (CTX, 30µg), nalidixic acid (NA, 30µg), nitrofurantoin (F, 300µg), gentamicin (GN, 10µg), amikacin (AK, 30µg), imipenem (IPM, 10µg), trimethoprim-sulfamethoxazole (SXT, 1.25/23.75µg), and levofloxacin (LEV, 5µg). Interpretation of zone diameters as susceptible, intermediate, or resistant followed CLSI 2023 breakpoints.[11]

Detection of ESBL Production

We identified ESBL production phenotypically using the combination disc method, where an increase of at least 5mm in zone diameter for ceftazidime or cefotaxime when combined with clavulanic acid compared to the antibiotic alone confirmed ESBL presence.[12]

Statistical Analysis

We employed descriptive statistics to calculate frequencies and percentages for bacterial species, patient demographics, and resistance patterns, analyzing data with SPSS version 26.0 (IBM Corp., USA). Chi-square tests compared resistance rates across bacterial species, with statistical significance set at $p<0.05$.

RESULTS

Demographic Characteristics and Pathogen Distribution

Our analysis included 150 unique urine samples with significant bacterial growth exceeding 10^5 CFU/mL. Consistent with established UTI epidemiology,[13] women comprised the majority (109/150, 72.7%). Patient ages ranged from 1 to 91 years (mean: 32.4 ± 21.7 years), with children under 18 representing 28.7% of our sample.

E. coli predominated (58 isolates, 38.7%), trailed by *Klebsiella* species (36 isolates, 24.0%), *Proteus mirabilis* (12 isolates, 8.0%), *Staphylococcus aureus* (11 isolates, 7.3%), and *Pseudomonas* species (10 isolates, 6.7%). The remainder included *Enterobacter* species (3 isolates, 2.0%) (Table 1).

Antimicrobial Resistance Patterns

Table 2 presents resistance rates across all isolates. Penicillins showed the weakest performance, with 88.7% resistance to amoxicillin and 85.3% to ampicillin. Amoxicillin-clavulanic acid demonstrated somewhat better activity but still faced 62.0% resistance. Among cephalosporins, ceftriaxone resistance reached 46.7%, while ceftazidime and cefotaxime resistance stood at 41.3% and 39.3%, respectively.

Fluoroquinolones exhibited worrisome resistance levels: 44.7% for ciprofloxacin and 38.0% for levofloxacin. Aminoglycosides showed mixed results gentamicin resistance was 32.0%, whereas amikacin maintained excellent activity with only 8.7% resistance. Nitrofurantoin performed well overall (18.7% resistance). Imipenem, our sole carbapenem test agent, proved most effective with 95.3% susceptibility. Trimethoprim-sulfamethoxazole resistance was elevated at 42.7%.

Table 1: Distribution of uropathogens by patient demographics.

Bacteria	Total (n=150)	Female n (%)	Male n (%)	Mean Age (years)
E. coli	58 (38.7)	44 (75.9)	14 (24.1)	28.3 ± 19.4
Klebsiella spp.	36 (24.0)	28 (77.8)	8 (22.2)	31.5 ± 22.8
P. mirabilis	12 (8.0)	7 (58.3)	5 (41.7)	26.7 ± 18.9
S. aureus	11 (7.3)	5 (45.5)	6 (54.5)	25.6 ± 15.3
Pseudomonas spp.	10 (6.7)	6 (60.0)	4 (40.0)	38.2 ± 24.1

Table 2: Overall antimicrobial resistance rates (n=150).

Antimicrobial	Resistant n (%)	Susceptible n (%)
Amoxicillin (AX)	133 (88.7)	17 (11.3)
Ampicillin (AM)	128 (85.3)	22 (14.7)
Augmentin (AMC)	93 (62.0)	57 (38.0)
Ciprofloxacin (CIP)	67 (44.7)	83 (55.3)
Ceftriaxone (CRO)	70 (46.7)	80 (53.3)
Ceftazidime (CAZ)	62 (41.3)	88 (58.7)
Cefotaxime (CTX)	59 (39.3)	91 (60.7)
Trimethoprim-sulfamethoxazole (SXT)	64 (42.7)	86 (57.3)
Levofloxacin (LEV)	57 (38.0)	93 (62.0)
Gentamicin (GN)	48 (32.0)	102 (68.0)
Nitrofurantoin (F)	28 (18.7)	122 (81.3)
Amikacin (AK)	13 (8.7)	137 (91.3)
Imipenem (IPM)	7 (4.7)	143 (95.3)

Pathogen-Specific Resistance Profiles

E. coli Resistance Patterns

E. coli showed substantial resistance to penicillins: 94.8% to amoxicillin and 89.7% to ampicillin. We detected the ESBL phenotype in 25 isolates (43.1%), manifesting as resistance to ceftriaxone (48.3%), ceftazidime (41.4%), and cefotaxime (44.8%). Fluoroquinolone resistance was notable, affecting 46.6% of isolates against ciprofloxacin and 39.7% against levofloxacin. Encouragingly, 82.8% remained susceptible to nitrofurantoin and 94.8% to imipenem.

Klebsiella Species Resistance Patterns

Klebsiella species displayed even higher cephalosporin resistance than *E. coli*, with 52.8% resistant to ceftriaxone and 47.2% to ceftazidime indicating ESBL production in 44.4% of isolates. Aminoglycoside resistance was also pronounced: 36.1% for gentamicin and 11.1% for amikacin. Fluoroquinolone resistance surpassed 40%. Imipenem susceptibility remained high at 94.4%.

Gram-Positive Pathogens

S. aureus isolates demonstrated 81.8% resistance to penicillins (AX/AM), yet 90.9% remained susceptible to nitrofurantoin. While we did not specifically test for methicillin resistance, the high penicillin resistance suggests clinicians should consider coverage for MRSA in severe cases [14].

ESBL Prevalence

Overall, 43.3% (65/150) of Enterobacteriaceae isolates exhibited the ESBL phenotype. *E. coli* showed a 43.1% ESBL rate, while *Klebsiella* species were slightly higher at 44.4%. These figures exceed the 30% threshold that CLSI identifies as indicating when carbapenems should be considered for serious infections.[15]

DISCUSSION

Our study documents troubling antimicrobial resistance patterns among bacteria causing UTIs, with *E. coli* maintaining its position as the predominant isolate while simultaneously showing high resistance to commonly prescribed antibiotics. The observed resistance rates of 88.7% to amoxicillin and 85.3% to ampicillin effectively rule out these agents for empirical treatment, findings that mirror those from regional surveillance studies across Asia and the Middle East.[16,17]

The 43.3% ESBL prevalence among Enterobacteriaceae constitutes a significant public health concern. Organisms producing ESBLs typically resist multiple drug classes, narrowing treatment options and forcing reliance on carbapenems which in turn drives further resistance development.[18] The particularly elevated resistance to ceftriaxone (46.7%) and cefotaxime (39.3%) in our population suggests that third-generation cephalosporins, traditionally recommended for kidney infections, should be avoided as first-choice empirical therapy in our region.[19]

With 46.6% of *E. coli* isolates resistant to ciprofloxacin, fluoroquinolone resistance in our study exceeds the 20% threshold generally considered acceptable for empirical use,[20] reflecting global trends of increasing quinolone resistance linked to agricultural antibiotic use and chromosomal mutations.[21] This finding indicates fluoroquinolones should be reserved for situations where culture results confirm susceptibility.

Importantly, imipenem and amikacin retained excellent activity (over 90% susceptibility), supporting their role as reliable options for complicated UTIs and pyelonephritis.[22] However, unrestricted carbapenem use risks accelerating resistance emergence. Nitrofurantoin demonstrated favorable activity (81.3% susceptible), particularly against *E. coli* (82.8% susceptible), confirming its value for uncomplicated bladder infections.[23]

The female predominance (72.7%) aligns with anatomical and physiological factors that increase women's UTI risk.[24] The considerable pediatric representation (28.7%) underscores the need for age-appropriate antibiotic stewardship, as children may experience higher treatment failure rates when infected with resistant organisms.[25] This study has some limitations, the single-center design and absence of clinical outcome data constrain its scope. We did not perform molecular characterization of resistance mechanisms, which would have provided deeper

understanding of ESBL genotypes and plasmid-mediated resistance spread.[26] Additionally, while our sample size was relatively modest, our findings correspond well with broader regional patterns.

Clinical Implications

These data strongly support several key initiatives:

1. **Guideline Revision:** Empirical therapy recommendations should prioritize local resistance data rather than relying solely on international guidelines.
2. **AMR Surveillance:** Establishment of a regional surveillance network to track resistance trends quarterly.
3. **Antimicrobial Stewardship:** Limiting broad-spectrum agents to culture-confirmed infections with restricted susceptibility profiles
4. **Rapid Diagnostics:** Implementing molecular ESBL detection to enable targeted therapy within 24 hours.[27]

CONCLUSION

Our study reveals alarmingly high rates of antimicrobial resistance among uropathogens, with ESBL prevalence exceeding 40% and fluoroquinolone resistance rendering these agents unsuitable for empirical therapy. Carbapenems and amikacin remain effective but must be preserved through robust antimicrobial stewardship. Nitrofurantoin retains utility for uncomplicated UTIs caused by *E. coli*. These findings underscore the urgent need for continuous local surveillance, evidence-based empirical therapy guidelines, and intensified antimicrobial stewardship programs to combat the escalating threat of AMR in UTIs.

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