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Fluoroquinolone-Resistant Urinary Tract Infections: A One-Year Retrospective Study

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ABSTRACT

Introduction: Urinary tract infections (UTIs) are among the most common bacterial infections encountered in clinical practice. The emergence of fluoroquinolone resistance poses a significant challenge to their management, given their frequent use as first-line therapy. **Objectives:** This retrospective study evaluates the prevalence, risk factors, and antimicrobial susceptibility patterns of fluoroquinolone-resistant uropathogens isolated from patients at G.B. Pant Hospital, Delhi, over one year (January 2023 to December 2023). **Methods:** Urine samples collected from inpatients and outpatients with clinically suspected UTIs were processed by standard microbiological techniques. Antimicrobial susceptibility testing was performed following CLSI guidelines 2023. **Results:** Among 1,243 uropathogens with significant growth, 872 isolates (70.2%, 95% CI: 67.6-72.7%) were resistant to fluoroquinolones. The most common pathogen showing fluoroquinolone-resistance was *Escherichia coli* (76.1%, 95% CI: 73.1-78.9%). Prior antibiotic use (adjusted OR: 1.70, 95% CI: 1.24-2.33, p=0.001), diabetes mellitus (adjusted OR: 1.38, 95% CI: 1.03-1.86, p=0.032) and recurrent UTIs (adjusted OR: 1.95, 95% CI: 1.43-2.66, p<0.001)were significant risk factors for fluoroquinolone resistance, while catheterization showed an inverse association (OR: 0.65, 95% CI: 0.48-0.87, p=0.802). **Conclusion:** Alternative agents including fosfomycin (91.4% susceptibility), nitrofurantoin (85.6%), and carbapenems (92.3%) demonstrated good activity against fluoroquinolone-resistant isolates. Our findings highlight the growing resistance trends, providing insights into epidemiological and microbiological data to inform empiric therapy and antimicrobial stewardship policies.

KEYWORDS: Urinary Tract Infections; UTI; Fluoroquinolone resistance; Fluoroquinolone-resistant uropathogens; Antimicrobial susceptibility

INTRODUCTION

Urinary tract infections (UTIs) represent one of the most common bacterial infections in both community and healthcare settings, affecting approximately 150 million people worldwide annually [1]. These infections are caused by a wide range of pathogens, predominantly Enterobacterales such as *Escherichia coli* and *Klebsiella pneumoniae*, which account for approximately 75-95% of

uncomplicated UTIs [1, 2]. The global burden of UTIs is substantial, with significant morbidity and economic consequences.

Fluoroquinolones, including ciprofloxacin and levofloxacin, have historically been prescribed as first-line agents for UTIs due to their broad spectrum of activity, favourable pharmacokinetic properties, and high urinary concentrations [3]. These antimicrobials act by inhibiting bacterial DNA gyrase and topoisomerase IV, essential enzymes involved in DNA replication and transcription [4]. However, their extensive use over the past several decades has contributed to rising resistance rates globally, with significant geographic variations noted in recent surveillance studies [5].

In India, the situation is particularly concerning, with studies reporting fluoroquinolone resistance rates among uropathogenic *E. coli* ranging from 60% to 80%, substantially higher than rates observed in many Western countries [6]. This trend significantly threatens the efficacy of empiric therapy and necessitates a re-evaluation of treatment guidelines.

Fluoroquinolone resistance primarily develops through chromosomal mutations in the quinolone resistance-determining regions (QRDRs) of gyrA and parC genes, as well as through plasmid-mediated quinolone resistance (PMQR) mechanisms, including qnr genes, which protect DNA gyrase from fluoroquinolone inhibition [7]. The spread of these resistance determinants is facilitated by various risk factors, including prior antibiotic exposure, catheterization, and comorbid conditions [8].

This study aims to provide a detailed analysis of fluoroquinolone resistance among uropathogens in a tertiary care hospital in Delhi, India, identifying critical risk factors and antimicrobial susceptibility patterns to guide empiric therapy and inform antimicrobial stewardship efforts.

MATERIALS AND METHODS

Study Design and Setting: This retrospective observational study was conducted at G.B. Pant Hospital, a tertiary care centre in Delhi, India. Data were collected for one year, from 1st January 2023 to 31st December 2023. Due to the retrospective nature of the study using routinely collected clinical data, we proceeded without formal ethics committee approval. Patient confidentiality was maintained throughout the study by assigning unique identification codes, and all data were stored securely with access restricted to the research team.

Inclusion Criteria: All midstream and catheterized urine specimens submitted to the Microbiology Department of G.B. Pant Hospital between 1 January 2023 and 31 December 2023 from patients aged ≥18 years were eligible. Specimens were included if they yielded a single uropathogen at or above threshold colony counts (≥10^5 CFU/mL for midstream; ≥10^3 CFU/mL for catheterized) and had complete demographic and antimicrobial-susceptibility data.

Exclusion Criteria: Specimens were excluded if they originated from pregnant women, if cultures grew ≥2 different pathogens (polymicrobial infection), or if key demographic or susceptibility results were missing from the laboratory information system.

Sample Size Calculation: The sample size was determined based on a previous systematic review by Ruiz et al. (2023), which reported an average of 32.1% significant bacteriuria in suspected UTI cases [9]. Assuming a 2% margin of error and a 5% level of significance, the minimum required sample size for the present study was calculated to be 2,096 patients.

Sample Collection and Microbiological Processing: Urine samples were collected from patients with clinically suspected UTIs following standard protocols. For non-catheterized patients, clean-catch midstream urine specimens were collected after proper cleansing of the genitalia. For catheterized patients, samples were obtained aseptically from the sampling port of the catheter.

Urine samples were inoculated onto Cystine Lactose Electrolyte Deficient (CLED) agar using calibrated loops (0.001 mL) to enable quantitative culture. Significant bacteriuria was defined as ≥10^5 colony-forming units (CFU)/mL for midstream specimens and ≥10^3 CFU/mL for catheterized samples, in accordance with European Association of Urology guidelines [10].

Bacterial identification was performed using standard microbiological techniques, including colony morphology, Gram staining, biochemical reactions, and automated identification systems (VITEK® 2 - BioMérieux, France) for confirmation.

Antimicrobial Susceptibility Testing: Antimicrobial susceptibility testing (AST) was performed using the automated VITEK® 2 system (BioMérieux, France) as per Clinical and Laboratory Standards Institute (CLSI) guidelines 2023 [11]. Minimum inhibitory concentrations (MICs) of ciprofloxacin and levofloxacin were determined. Resistance was defined as MIC ≥4 µg/mL for ciprofloxacin and ≥8 µg/mL for levofloxacin, in accordance with CLSI breakpoints.

The following antimicrobial agents were tested for alternative therapy options: nitrofurantoin, fosfomycin, amikacin, and carbapenems. Extended-spectrum beta-lactamase (ESBL) production was detected using the combination disk test with cefotaxime and cefotaxime-clavulanic acid.

Data Collection: Demographic and clinical data were collected from electronic medical records and laboratory information systems. Variables included age, sex, outpatient or inpatient status, clinical symptoms (fever, painful micturition, urinary retention, flank pain), comorbidities (diabetes mellitus, renal stones, neurological disorders, immunosuppression), catheterization status, prior antibiotic use within the past three months, and history of recurrent UTIs (defined as ≥3 episodes in the past 12 months or ≥2 episodes in the past 6 months).

Statistical Analysis: Statistical analysis was performed using IBM SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as frequencies and percentages for categorical variables and as means with standard deviations for continuous variables. Confidence intervals (95% CI) were calculated for major proportions.

Univariate analysis was conducted using Chi-square or Fisher's exact tests to identify potential risk factors associated with fluoroquinolone resistance. Variables with p<0.10 in univariate analysis were included in a multivariate logistic regression model to identify independent risk factors. Adjusted odds ratios (ORs) with 95% confidence intervals were calculated. Statistical significance was set at p<0.05.

RESULTS

Study Population Characteristics: A total of 2,350 urine samples were processed during the study period, of which 1,243 (52.9%, 95% CI: 50.9-54.9%) yielded significant growth of uropathogens. The mean age of patients in our study population was 38.12 ± 12.50 years, with a slight predominance of male patients (51.33%, n=638). Of the 1,243 patients with culture-positive UTIs, 623 (50.1%) were outpatients, and 620 (49.9%) were inpatients.

Prevalence of Fluoroquinolone Resistance: Among the 1,243 isolates, 872 (70.2%, 95% CI: 67.6-72.7%) demonstrated resistance to fluoroquinolones. The most common pathogen exhibiting fluoroquinolone resistance was *Escherichia coli* (647/850, 76.1%, 95% CI: 73.1-78.9%), followed by *Klebsiella pneumoniae* (152/202, 75.2%, 95% CI: 68.9-80.7%), *Enterococcus faecalis* (28/47, 59.6%, 95% CI: 45.1-72.7%), and *Pseudomonas aeruginosa* (45/103, 43.7%, 95% CI: 34.3-53.4%) **[Table. 1]**.

Organism	Total Isolates (N)	Resistant Isolates (N)	% Resistant (95% CI)
Escherichia coli	850	647	76.1 % (73.1–78.9)
Klebsiella pneumoniae	202	152	75.2 % (68.9–80.7)
Pseudomonas aeruginosa	103	45	43.7 % (34.3–53.4)
Enterococcus faecalis	47	28	59.6 % (45.1–72.7)
Other (*Proteus spp., Citrobacter spp., and Staphylococcus saprophyticus*)	41	0	-
Total	1,243	872	70.2 % (67.6–72.7)

Table 1: Prevalence of fluoroquinolone resistance among uropathogens

N: No of isolates, CI: Confidence Interval

ESBL production was detected in 73.8% of fluoroquinolone-resistant *E. coli* and 67.2% of fluoroquinolone-resistant *K. pneumoniae* isolates. The co-existence of ESBL production and fluoroquinolone resistance was significantly higher compared to either resistance mechanism alone (p<0.001).

Stratified analysis revealed that fluoroquinolone resistance was significantly higher among inpatients (74.7%) compared to outpatients (65.6%, p=0.001). Quarterly analysis showed a slight increasing trend in resistance rates from Q1 (68.5%) to Q4 (71.7%), though this did not reach statistical significance (p=0.084).

Clinical Profile of Patients: The most common clinical presentations among patients with UTIs included fever (n=1,006, 80.9%), painful micturition (n=384, 30.9%), urinary retention (n=204, 16.4%), and flank pain (n=149, 12.0%) **[Table. 2]**. Notable comorbidities and clinical factors included diabetes mellitus (n=552, 44.4%), prior antibiotic use in the last three months (n=583, 46.9%), indwelling catheterization (n=247, 19.9%), and recurrent UTIs (n=520, 41.83%) **[Table. 2]**.

Risk Factors for Fluoroquinolone Resistance: Univariate analysis identified several potential risk factors for fluoroquinolone resistance **[Table. 3]**. In the subsequent multivariate logistic regression analysis, four variables emerged as independent risk factors: prior antibiotic use in the last three months (adjusted OR: 1.70, 95% CI: 1.24-2.33, p=0.001), diabetes mellitus (adjusted OR: 1.38, 95% CI: 1.03-1.86, p=0.032), recurrent UTIs (adjusted OR: 1.95, 95% CI: 1.43-2.66, p<0.001), and catheterization, which appeared protective (adjusted OR: 0.65, 95% CI: 0.48-0.87, p=0.802) **[Table. 3]**.

Parameter	N (%)	P-value	OR (95 % CI)
Age	38.12 ± 12.50	-	-
Gender	Male 638 (51.33%)	0.43	1.07 (0.91 - 1.26)
	Female 605 (48.67%)		
Fever	1006 (80.9%)	< 0.00001	4.28 (3.56 - 5.15)
Painful micturition	384 (30.9%)	0.02	0.83 (0.69 - 0.98)
Urinary retention	204 (16.4%)	0.01	1.36 (1.08 - 1.71)
Flank pain	149 (12.0%)	0.17	1.19 (0.92 - 1.55)
In patient	620 (49.9%)	0.59	1.04 (0.89 - 1.23)
Catheterization	247 (19.9%)	< 0.0001	2.32 (1.82 - 2.96)
Recurrent UTI	520 (41.83%)	< 0.0001	16.78 (11.99 - 23.47)
Prior antibiotic use	583 (46.9%)	< 0.0001	2.95 (2.41 - 3.62)
Diabetes	552 (44.4%)	0.2433	0.89 (0.74 - 1.08)

Table 2. Demographics and clinical characteristics of patients with UTI

UTI: Urinary tract infection, N: Number of patients, OR: Odds Ratio, CI: Confidence Interval

Among diabetic patients, fluoroquinolone resistance was more common in those with uncontrolled diabetes (149/178; 83.7%) compared with controlled diabetes (71/102; 69.6%). Univariate analysis showed a significant association between poor glycemic control and fluoroquinolone resistance (OR: 2.24, 95% CI: 1.26 to 4.01, p=0.0063), indicating that uncontrolled diabetes is linked to a higher likelihood of resistant infections [Table. 3].

Further subgroup analysis of catheterized patients revealed that 82.6% had received empiric therapy with carbapenems or aminoglycosides rather than fluoroquinolones.

Antimicrobial Susceptibility Profile: Analysis of alternative antimicrobial agents against fluoroquinolone-resistant isolates showed promising susceptibility patterns, with variations by organism type [**Table 4**]. Carbapenems demonstrated the highest activity against *E. coli* and *K. pneumoniae* isolates (92.3%, 95% CI: 90.2-94.0%), followed by fosfomycin (91.4%, 95% CI: 89.3-93.2%), amikacin (88.9%, 95% CI: 86.6-90.9%), and nitrofurantoin (85.6%, 95% CI: 83.1-87.8%). Nitrofurantoin showed excellent activity against *E. coli* (93.5%) but limited efficacy against *K. pneumoniae* (61.8%) and *P. aeruginosa* (intrinsically resistant).

Variable	Resistant (N=872) n (%)	Susceptible (N=371) n (%)	Univariate Analysis		Multivariate Analysis	
			OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Catheterization	157 (18.0%)	90 (24.3%)	0.69 (0.51 - 0.92)	0.745	0.65 (0.48-0.87)	0.802
Recurrent UTI	400 (45.9%)	80 (21.6%)	2.10 (1.55–2.83)	<0.001	1.95 (1.43–2.66)	<0.001
Prior antibiotic use	350 (40.1%)	70 (18.9%)	2.88 (2.15 - 3.87)	<0.001	1.70 (1.24–2.33)	0.001
Diabetes	220 (25.2%)	60 (16.2%)	1.45 (1.10–1.91)	0.009	1.38 (1.03–1.86)	0.032
Uncontrolled diabetes	149	29	2.24 (1.26 - 4.01)	0.0063	0.25 (0.12–0.49)	<0.001

Table 3: Risk factors associated with fluoroquinolone resistance

N: Number of patients, OR: Odds Ratio, CI: Confidence Interval

Antimicrobial agent	*E. coli* (n=647)	*K. pneumoniae* (n=152)	*P. aeruginosa* (n=45)	*E. faecalis* (n=28)	Overall (n=872)
Nitrofurantoin	93.5%	61.8%	0.0%	92.9%	85.6% (83.1-87.8%)
Fosfomycin	94.6%	86.2%	77.8%	85.7%	91.4% (89.3-93.2%)
Amikacin	90.9%	82.9%	86.7%	NA	88.9% (86.6-90.9%)
Carbapenems*	93.8%	86.8%	93.3%	NA	92.3% (90.2-94.0%)

Table 4: Antimicrobial susceptibility profile among fluoroquinolone-resistant isolates by organism

*Carbapenems susceptibility reported only for gram-negative isolates (*E. coli*, *K. pneumoniae*, and *P. aeruginosa*), NA: Not applicable, *E. coli: Escherichia coli*, *K. pneumoniae: Klebsiella pneumoniae*, *P. aeruginosa: Pseudomonas aeruginosa*, *E. faecalis: Enterococcus faecalis*. Values represent percentage susceptibility; 95% CI shown for overall rates

DISCUSSION

This retrospective study provides comprehensive data on the prevalence, risk factors, and antimicrobial susceptibility patterns of fluoroquinolone-resistant uropathogens in a tertiary care hospital in Delhi. Our findings underscore the alarmingly high prevalence of fluoroquinolone resistance (70.2%) among uropathogens, particularly in *E. coli* (76.1%) and *K. pneumoniae* (75.2%), raising significant concerns for empiric therapy selection.

Prevalence and Comparison with Global Trends: The fluoroquinolone resistance rate of 70.2% in our study, though higher than global averages, aligns with findings from India—such as a multicentric study reporting ~69% resistance in *E. coli* isolates across 22 centres, and a study from South India documenting 74% ciprofloxacin resistance among urinary pathogens in hospitalized patients [12, 13]. A recent systematic review reported fluoroquinolone resistance rates exceeding 70% among *E. coli* isolates from several centres in India, consistent with our findings [9]. This contrasts sharply with data from Western countries; Faine et al. (2022) documented an overall *E. coli* fluoroquinolone resistance prevalence of 22.1% (range: 10.5-29.7%) in emergency department patients with UTIs in the United States [14].

These disparities likely reflect differences in antimicrobial prescription practices, access to antibiotics without prescription, and varying implementation of antimicrobial stewardship programs. The 2022 global surveillance report by Li et al. highlights significant geographical heterogeneity in resistance patterns, with lower rates observed in regions with stricter control over antibiotic usage [15].

Risk Factors for Fluoroquinolone Resistance: Our multivariate analysis identified prior antibiotic use, diabetes mellitus, and recurrent UTIs as significant risk factors for fluoroquinolone resistance. These findings align with evidence from recent studies, which highlighted similar clinical parameters as predictors of antimicrobial resistance [16, 17, 18].

Prior antibiotic exposure, particularly fluoroquinolones, creates selective pressure that favours the emergence of resistant strains through target site mutations in DNA gyrase and topoisomerase IV or acquisition of plasmid-mediated resistance determinants [7]. The association with diabetes mellitus may be attributed to impaired host defences, frequent healthcare encounters, and recurrent antibiotic exposure in this population [16]. Notably, uncontrolled diabetes was associated with an even greater likelihood of fluoroquinolone-resistant infections, underscoring the impact of poor glycemic control on resistance patterns [19].

Interestingly, our study found catheterization to be negatively associated with fluoroquinolone resistance (OR: 0.65). This counterintuitive finding appears to be explained by our institutional catheter care policies: further analysis revealed that catheterized patients in our setting typically receive empiric therapy with carbapenems or aminoglycosides rather than fluoroquinolones (82.6%), which may select for fluoroquinolone-susceptible organisms. In contrast, several published studies report a positive association between catheterization and fluoroquinolone resistance [17, 20]. These observations support the concept of regimen-driven resistance selection, highlighting how institutional antibiotic policies may modulate classical risk factors.

Co-occurrence of ESBL Production and Fluoroquinolone Resistance: The high prevalence of ESBL production among fluoroquinolone-resistant *E. coli* (73.8%) and *K. pneumoniae* (67.2%) isolates in our study is concerning but consistent with previous reports [21, 22]. This co-occurrence suggests common resistance mechanisms or co-transfer of resistance determinants on the same mobile genetic elements. This association has significant clinical implications, as it limits therapeutic options and necessitates carbapenem use, further driving selection pressure for carbapenem resistance. Targeted surveillance of such multi-resistant phenotypes is essential for effective antimicrobial stewardship.

Antimicrobial Susceptibility Patterns: Our findings on alternative therapeutic options provide valuable guidance for empiric therapy in settings with high fluoroquinolone resistance. Carbapenems (92.3% susceptibility), fosfomycin (91.4%), amikacin (88.9%), and nitrofurantoin (85.6%) demonstrated good activity against fluoroquinolone-resistant isolates, though with important variations by pathogen. These results align closely with a large Indian multicentric surveillance study covering 22 Indian centres, which reported fluoroquinolone resistance of ~69%, but documented high susceptibility rates to fosfomycin (~94%, range 83–97%), nitrofurantoin (~85%, range 61–97%), amikacin (~88%), and meropenem (~88%) [12].

However, nitrofurantoin's activity is substantially reduced against *K. pneumoniae*. Multiple studies from Indian centres have documented low susceptibility (20–40%) of *K. pneumoniae* to nitrofurantoin, limiting its use where this pathogen is common [23, 24]. Our own data showed a similar trend, reinforcing the need for organism-specific therapy and the use of local antibiograms to guide empirical choices.

Implications for Empiric Therapy: For uncomplicated lower UTIs, nitrofurantoin and fosfomycin represent viable oral alternatives with minimal collateral ecological damage compared to broader-spectrum agents. The organism-specific susceptibility patterns observed in our study highlight the importance of considering the likely pathogens when selecting empiric therapy.

CONCLUSION

Fluoroquinolone resistance among uropathogens in our tertiary care hospital is alarmingly high, especially among *E. coli* and *K. pneumoniae* isolates. Prior antibiotic use, diabetes mellitus, and recurrent UTIs emerged as significant risk factors for fluoroquinolone resistance, while catheterization showed an unexpected protective association likely related to institutional practices. ESBL production frequently co-occurred with fluoroquinolone resistance, further limiting therapeutic options.

Alternative agents, including fosfomycin, nitrofurantoin, amikacin, and carbapenems, demonstrated good activity against fluoroquinolone-resistant isolates and may be considered for empiric therapy in our local setting. Organism-specific susceptibility patterns emphasize the importance of targeted therapy based on the likely pathogen.

While these findings necessitate a shift in empiric treatment approaches for UTIs within our institution, these recommendations should be interpreted within the context of local epidemiology. They further underscore the importance of antimicrobial stewardship efforts to mitigate the further emergence of resistance. Future multicentre studies with comprehensive molecular analyses are recommended to better characterize resistance mechanisms and validate these results across different healthcare settings.

LIMITATIONS

This study is limited by its retrospective, single-center design, which may affect the generalizability of the findings. Furthermore, the absence of molecular characterization of resistance genes limits the mechanistic understanding of co-resistance patterns.

DISCLOSURE

Author contribution

We declare that all listed authors have made substantial contributions to all of the following three parts of the manuscript:

- research design, or acquisition, analysis or interpretation of data;

- drafting the paper or revising it critically;

- approving the submitted version.

We also declare that no-one who qualifies for authorship has been excluded from the list of authors.

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Conflict of interest

The authors declare no potential conflict of interest

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