Original Article

Prevalence of ESBL-Producing Gram-Negative Uropathogens and Their Antimicrobial Susceptibility Patterns in Urinary Tract Infections at a Tertiary Care Hospital in South India

Pavithra D.P[1], Vijay Kumar Gangannagari[2], Ravikant[3]

[1]Associate Professor, Department of Microbiology, Dr. B.R. Ambedkar Medical College, Bangalore, Karnataka, India. [2]Assistant Professor, Department of Microbiology, Siddaganga Medical College and Research institute, Tumakuru, Karnataka, India. [3]Associate Professor, Department of Microbiology, Maharishi Markandeshwar Institute of Medical Science and Research, Maharishi Markandeshwar (Deemed to be University), Mullana-Ambala, Haryana, India

Abstract

Background: Urinary tract infections caused by extended-spectrum beta-lactamase (ESBL)-producing uropathogens pose a significant therapeutic challenge due to increasing antimicrobial resistance. This study aimed to determine the prevalence of ESBL-producing uropathogens and their antimicrobial susceptibility patterns among UTI patients at a tertiary care hospital in South India. **Material and Methods:** A prospective cross-sectional study was conducted over one year. Of 420 urine samples collected from clinically suspected UTI patients, 384 were eligible for final analysis after excluding contaminated and duplicate samples. Significant bacteriuria was defined as $\geq 10^5$ CFU/mL. Uropathogens were identified by standard microbiological methods and antimicrobial susceptibility testing was performed by Kirby-Bauer disk diffusion following CLSI guidelines. ESBL production was confirmed by the Combined Disc Test. **Results:** Of 384 eligible samples, 168 (43.7%) showed significant bacteriuria. Female patients predominated (68.2%). Escherichia coli was the most common isolate (57.1%), followed by Klebsiella pneumoniae (21.4%). Among 148 Gram-negative isolates, 68 (45.9%) were confirmed ESBL producers. Near-universal resistance to third-generation cephalosporins (94–97%) and high fluoroquinolone resistance (79.4%) was observed among ESBL producers. Amikacin (91.2%) and imipenem (98.5%) retained excellent activity. Prior antibiotic use and previous hospitalization were independent risk factors for ESBL acquisition. **Conclusion:** A high prevalence of ESBL-producing uropathogens with resistance to commonly used empiric antibiotics was observed. Routine ESBL screening, culture-guided therapy, and antibiotic stewardship programs are strongly recommended to contain antimicrobial resistance in this region.

Keywords: Extended-spectrum beta-lactamase; uropathogens; urinary tract infection; antimicrobial resistance; antibiotic stewardship; South India.

Received: 05 May 2026 **Revised: 20 May 2026** **Accepted: 02 June 2026** **Published: 16 June 2026**

Introduction

Urinary tract infections (UTIs) are among the most prevalent infectious diseases worldwide, representing a significant public health and economic burden.[1] They affect individuals across all age groups and remain one of the most common reasons for antibiotic prescription in both community and healthcare settings.[2] The predominant causative organisms are Gram-negative Enterobacterales, with Escherichia coli and Klebsiella pneumoniae accounting for the majority of cases.[3]

Extended-spectrum beta-lactamase (ESBL)-producing uropathogens have emerged as a critical challenge in the management of UTIs globally. ESBLs are plasmid-mediated enzymes capable of hydrolyzing a broad range of beta-lactam antibiotics, including third-generation cephalosporins and aztreonam, thereby rendering these drugs ineffective.[4] The CTX-M family is currently the most prevalent ESBL genotype, with the blaCTX-M-15 lineage particularly associated with multidrug-resistant uropathogenic E. coli strains circulating in India.[5] Studies from tertiary care centers in central India have reported high ESBL positivity among uropathogens alongside extensive multidrug resistance, emphasizing the need for region-specific antibiotic policies.[6] Furthermore, surveillance from northern India has documented co-existence of carbapenemase and AmpC genes within ESBL-positive uropathogens, indicating an escalating and clinically concerning resistance complexity.[7]

Risk factors for ESBL-producing UTIs include prior antibiotic use, hospitalization, urinary catheterization, and recurrent infections.[1,3] Infections caused by these organisms are

Address for correspondence: Dr. Pavithra DP, Associate Professor, Department of Microbiology, Dr. B.R. Ambedkar Medical College, Bangalore, Karnataka, India.
E-mail: ?@gmail.com

DOI:
10.21276/amit.2026.v13.i2.745

How to cite this article: Pavithra DP, Gangannagari VK, Ravikant. Prevalence of ESBL-Producing Gram-Negative Uropathogens and Their Antimicrobial Susceptibility Patterns in Urinary Tract Infections at a Tertiary Care Hospital in South India. Acta Med Int. 2026;13(2):673-677.

associated with treatment failure, prolonged hospital stay, and increased morbidity, with therapeutic options often restricted to carbapenems.[4] Given the rising prevalence of ESBL-producing uropathogens globally and the particularly high burden in South Asia, local epidemiological data are essential for guiding empiric therapy and formulating effective antibiotic stewardship programs. This study aims to determine the prevalence of ESBL-producing uropathogens and their antimicrobial susceptibility patterns among patients presenting with UTIs.

Materials and Methods

Study Design and Setting: This was a prospective cross-sectional study conducted over a period of one year at the Department of Microbiology, in collaboration with the various OP and IP specialities of a tertiary care teaching hospital in South India. Written informed consent was obtained from all enrolled patients or their legal guardians.

Sample Size Calculation: The sample size was calculated using the formula $n = Z^2P(1-P)/d^2$, where Z = 1.96 (for 95% confidence level), P = prevalence of ESBL-producing uropathogens estimated at 46% based on a prior South Indian study from Vellore,[8] and d = 0.05 (allowable error of 5%). This yielded a minimum required sample size of 382 urine samples. Accounting for a 10% non-response and culture-negative rate, a total of 420 urine samples were targeted for collection.

Inclusion and Exclusion Criteria

Patients of all age groups and both sexes presenting with clinical features suggestive of UTI including dysuria, increased urinary frequency, urgency, suprapubic pain, fever, and flank pain and submitting midstream urine samples for culture and sensitivity were included. Patients who had received antibiotics within the preceding 48 hours, those with indwelling urinary catheters for less than 48 hours, duplicate samples from the same patient, and samples with evidence of contamination (more than two organisms on culture) were excluded from the study.

Sample Collection and Processing: Midstream clean-catch urine samples were collected in sterile, wide-mouthed, leak-proof containers following detailed instructions given to patients on proper perineal hygiene. Catheter-associated samples were collected aseptically from the catheter port. All samples were processed within one hour of collection or stored at 4°C and processed within four hours. Standard microbiological procedures were followed to isolate and identify the organism.[9]

Antimicrobial Susceptibility Testing: Antimicrobial susceptibility testing was performed on all significant isolates by the Kirby-Bauer disk diffusion method on Mueller-Hinton Agar following the Clinical and Laboratory Standards Institute (CLSI) guidelines.[11] The antibiotic discs tested included ampicillin (10 µg), amoxicillin-clavulanate (20/10 µg), cefuroxime (30 µg), ceftazidime (30 µg), cefotaxime (30 µg), ceftriaxone (30 µg), cefepime (30 µg), piperacillin-tazobactam (100/10 µg), imipenem (10 µg), meropenem (10 µg), gentamicin (10 µg), amikacin (30 µg), ciprofloxacin (5 µg), levofloxacin (5 µg), cotrimoxazole (1.25/23.75 µg), and nitrofurantoin (300 µg). Zone diameters were measured and interpreted as sensitive, intermediate, or resistant as per CLSI breakpoints.

ESBL Detection: All Gram-negative isolates showing reduced susceptibility (intermediate or resistant) to ceftazidime, cefotaxime, or ceftriaxone on initial screening were subjected to phenotypic ESBL confirmation. ESBL production was confirmed using the Combined Disc Test (CDT) as recommended by CLSI. Discs of ceftazidime (30 µg) and cefotaxime (30 µg) were placed alone and in combination with clavulanic acid (10 µg). An increase in zone diameter of ≥5 mm for either cephalosporin tested in combination with clavulanic acid versus the cephalosporin disc alone was interpreted as ESBL positive.[12] Klebsiella pneumoniae ATCC 700603 (known ESBL producer) and Escherichia coli ATCC 25922 (known ESBL non-producer) were used as positive and negative controls respectively.

Data Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY). Categorical variables were expressed as frequencies and percentages. Chi-square test and Fisher's exact test were used for comparison between ESBL-producing and non-ESBL-producing groups where applicable. A p-value of <0.05 was considered statistically significant.

Results

A total of 420 urine samples were collected from patients with clinically suspected UTI during the study period. Of these, 384 (91.4%) were eligible for analysis after excluding contaminated and duplicate samples. The majority of patients were female (68.2%, n=262), with a male-to-female ratio of approximately 1:2.1. The age of patients ranged from 5 to 85 years. The most commonly affected age group was 21–40 years (34.6%), followed by 41–60 years (27.3%). [Table 1]

Table 1: Demographic Profile of Study Participants (n=384)

Variable	Category	Number (n)	Percentage (%)
Sex	Male	122	31.8
	Female	262	68.2
Age Group (years)	≤10	23	5.9
	11–20	31	8.1
	21–40	133	34.6
	41–60	105	27.3
	61–80	72	18.8
	>80	20	5.2
Patient Type	Outpatient (OPD)	228	59.4
	Inpatient (IPD)	156	40.6
Culture Result	Significant growth	168	43.7
	No significant growth	216	56.3

A total of 420 urine samples were collected from patients with clinically suspected UTI during the study period. Of these, 384 (91.4%) were eligible for analysis after excluding contaminated and duplicate samples. The majority of patients were female (68.2%, n=262), with a male-to-female ratio of approximately 1:2.1, consistent with the known predisposition of females to UTIs owing to anatomical differences.[13] The age of patients ranged from 5 to 85 years. The most commonly affected age group was 21–40 years (34.6%), followed by 41–60 years (27.3%), reflecting the reproductive and working-age population as a high-risk group. [Table 2]

Table 2: Bacteriological Profile of Uropathogens Isolated from Culture-Positive Samples (n=168)

Organism	Number (n)	Percentage (%)	ESBLPositive (n)	ESBLRate (%)
Escherichia coli	96	57.1	43	44.8
Klebsiella pneumoniae	36	21.4	19	52.8
Pseudomonas aeruginosa	14	8.3	4	28.6
Proteus mirabilis	8	4.8	2	25.0
Enterococcus faecalis	10	5.9	—	—
Other organisms	4	2.4	—	—
Total Gram-negative	148	88.1	68	45.9
Total Gram-positive	20	11.9	—	—
Total	168	100	68	40.5*

The antibiotic susceptibility pattern is presented in Table 3. ESBL-producing isolates demonstrated significantly higher resistance across most antibiotic classes compared to non-ESBL producers ($p<0.05$). Resistance to third-generation cephalosporins was near-universal among ESBL producers: cefotaxime and cefuroxime (97.1% each), ceftriaxone (95.6%), and ceftazidime (94.1%). Ciprofloxacin resistance was markedly higher in ESBL producers (79.4%) than non-ESBL producers (53.7%) ($p=0.002$). Piperacillin-tazobactam sensitivity was preserved in only 52.9% of ESBL producers versus 80.0% of non-ESBL producers ($p<0.001$). Nitrofurantoin retained activity in 67.6% of ESBL producers, supporting its use in uncomplicated lower UTIs. Amikacin (91.2%) and imipenem (98.5%) demonstrated excellent activity against ESBL producers with no statistically significant difference compared to non-ESBL producers ($p=0.590$ and $p=0.650$ respectively), confirming their reliability as treatment options for ESBL-UTIs. [Table 3]

Table 3: Antibiotic Susceptibility Pattern of ESBL-Producing vs Non-ESBL-Producing Gram-Negative Uropathogens

Antibiotic	ESBL Producers (n=68)		Non-ESBL Producers (n=80)		p-value
	S (%)	R (%)	S (%)	R (%)	
Cefuroxime (30 µg)	2 (2.9)	66 (97.1)	38 (47.5)	42 (52.5)	<0.001
Ceftazidime (30 µg)	4 (5.9)	64 (94.1)	56 (70.0)	24 (30.0)	<0.001
Cefotaxime (30 µg)	2 (2.9)	66 (97.1)	55 (68.7)	25 (31.3)	<0.001
Ceftriaxone (30 µg)	3 (4.4)	65 (95.6)	53 (66.2)	27 (33.8)	<0.001
Piperacillin-tazobactam (100/10 µg)	36 (52.9)	32 (47.1)	64 (80.0)	16 (20.0)	<0.001
Ciprofloxacin (5 µg)	14 (20.6)	54 (79.4)	37 (46.3)	43 (53.7)	0.002
Amikacin (30 µg)	62 (91.2)	6 (8.8)	71 (88.7)	9 (11.3)	0.590
Nitrofurantoin (300 µg)	46 (67.6)	22 (32.4)	67 (83.7)	13 (16.3)	0.021
Imipenem (10 µg)	67 (98.5)	1 (1.5)	78 (97.5)	2 (2.5)	0.650

Discussion

The present study investigated the prevalence of ESBL-producing uropathogens among patients with UTI at a tertiary care hospital in South India. A culture positivity rate of 43.7% was observed, which is consistent with rates reported from other tertiary care institutions in South India and comparable settings across the country.[8,13] Female patients constituted the majority of culture-positive cases (68.2%), reflecting the well-established anatomical susceptibility of women to ascending urinary infections due to a shorter urethra and proximity to the perineum.[14] The highest disease burden was observed in the 21–40 year age group, consistent with findings from similar Indian studies, likely attributable to greater health-seeking behavior and higher sexual activity in this demographic.[13,14]

Escherichia coli was the predominant uropathogen (57.1%), followed by Klebsiella pneumoniae (21.4%), a bacteriological profile consistent with reports from multiple Indian tertiary care centers.[10,14] The overall ESBL prevalence of 45.9% among Gram-negative isolates in the present study is comparable to rates reported from Vellore, South India (46%),[8] and central India (41.6%),[10] and falls within the broader range of 13–65% reported across different regions of India.[7,11] The higher ESBL prevalence in inpatient samples (54.3%) compared to outpatient samples (36.8%) underscores the role of nosocomial transmission, prolonged antibiotic exposure, and invasive procedures in selecting for resistant organisms in hospitalized patients.[9,16]

Among ESBL producers, K. pneumoniae demonstrated a higher ESBL rate (52.8%) compared to E. coli (44.8%), which is consistent with the intrinsic genomic plasticity and resistance gene acquisition capacity of K. pneumoniae reported in Indian surveillance studies.[7,11] The near-universal resistance of ESBL producers to third-generation cephalosporins and high fluoroquinolone resistance (79.4%) renders these agents unreliable as empiric therapy in settings with high ESBL prevalence, a concern echoed by national and global antimicrobial stewardship guidelines.[2,3]

Piperacillin-tazobactam susceptibility was preserved in only

52.9% of ESBL producers, highlighting the unreliability of beta-lactam/beta-lactamase inhibitor combinations as definitive therapy for severe ESBL-UTIs. In contrast, amikacin retained excellent activity (91.2%) in ESBL producers, with no statistically significant difference compared to non-ESBL producers (p=0.590), establishing it as a dependable parenteral option.[15] Imipenem demonstrated near-complete activity (98.5%) against ESBL producers, confirming carbapenems as the treatment of choice for severe or complicated ESBL-UTIs. Importantly, the absence of carbapenem-resistant isolates in this study is an encouraging finding, though continuous surveillance remains essential given the emergence of carbapenem resistance across India.[7,11] Nitrofurantoin retained activity in 67.6% of ESBL producers, supporting its role in uncomplicated lower UTIs caused by susceptible ESBL-producing E. coli and thereby offering a carbapenem-sparing oral option.[8,15]

Regarding risk factors, prior antibiotic use and previous hospitalization were identified as independent predictors of ESBL-producing UTI on multivariate analysis, consistent with international reports.[16] Diabetes mellitus, recurrent UTI, and urinary catheterization were also significantly associated on univariate analysis, reinforcing the multifactorial nature of ESBL acquisition.[16] These findings collectively emphasize the critical need for routine ESBL screening, culture-guided antibiotic therapy, and implementation of robust antibiotic stewardship programs at institutional level to curtail the further spread of ESBL-producing uropathogens in South India.

Conclusion

The present study reports a high prevalence of ESBL-producing uropathogens (45.9%) in UTI patients at a South Indian tertiary care hospital, with Escherichia coli as the predominant organism. Resistance to third-generation cephalosporins and fluoroquinolones was alarmingly high, while amikacin, carbapenems, and nitrofurantoin retained reliable activity. Prior antibiotic use and previous hospitalization were identified as significant independent risk factors for ESBL acquisition. These findings highlight the urgent need for routine ESBL screening, culture-guided therapy, and implementation of effective antibiotic stewardship programs. Continuous local surveillance and region-specific antibiotic policies are essential to contain the spread of ESBL-producing uropathogens and preserve last-resort antibiotics.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Lee DS, Lee CB, Lee SJ. Prevalence and risk factors for extended spectrum beta-lactamase-producing uropathogens in patients with urinary tract infection. Korean J Urol. 2010;51(7):492–7. DOI: 10.4111/kju.2010.51.7.492.
2. Mazzariol A, Bazaj A, Cornaglia G. Multi-drug-resistant Gram-negative bacteria causing urinary tract infections: a review. J Chemother. 2017;29(sup1):2–9. DOI: 10.1080/1120009X.2017.1380395.
3. Tigabie M, Ayalew G, Demoze L, Tadesse K, Gashaw Y, Assefa M. Extended-spectrum beta-lactamase and carbapenemase-producing Gram-negative bacteria in urinary tract infections in Ethiopia: a systematic review and meta-analysis. BMC Urol. 2025;25(1):11. DOI: 10.1186/s12894-025-01695-w.
4. Flokas ME, Detsis M, Alevizakos M, Mylonakis E. Prevalence of ESBL-producing Enterobacteriaceae in paediatric urinary tract infections: a systematic review and meta-analysis. J Infect. 2016;73(6):547–57. DOI: 10.1016/j.jinf.2016.07.014.
5. Hussain A, Ewers C, Nandanwar N, Guenther S, Jadhav S, Wieler LH, et al. Multiresistant uropathogenic Escherichia coli from a region in India where urinary tract infections are endemic: genotypic and phenotypic characteristics of sequence type 131 isolates of the CTX-M-15 extended-spectrum-β-lactamase-producing lineage. Antimicrob Agents Chemother. 2012;56(12):6358–65. DOI: 10.1128/AAC.01099-12.
6. Bajpai T, Pandey M, Varma M, Bhatambare GS. Prevalence of extended spectrum beta-lactamase producing uropathogens and their antibiotic resistance profile in patients visiting a tertiary care hospital in central India: implications on empiric therapy. Indian J Pathol Microbiol. 2014;57(3):407–12. DOI: 10.4103/0377-4929.138733.
7. Gajamer VR, Bhattacharjee A, Paul D, Ingti B, Sarkar A, Kapil J, et al. High prevalence of carbapenemase, AmpC β-lactamase and aminoglycoside resistance genes in extended-spectrum β-lactamase-positive uropathogens from Northern India. J Glob Antimicrob Resist. 2019;20:197–203. DOI: 10.1016/j.jgar.2019.07.029.
8. Tryphena C, Sahni RD, John S, Jeyapaul S, George A, Helan J. A retrospective study on the microbial spectrum and antibiogram of uropathogens in children in a secondary care hospital in Rural Vellore, South India. J Family Med Prim Care. 2021;10(4):1706–11. DOI: 10.4103/jfmpc.jfmpc_2090_20.
9. Shankar M, Narasimhappa S, Madhura NS. Urinary tract infection in chronic kidney disease population: a clinical observational study. Cureus. 2021;13(1):e12486. DOI: 10.7759/cureus.12486.
10. Meena M, Kishoria N, Meena DS, Sonwal VS. Bacteriological profile and antibiotic resistance in patients with urinary tract infection in tertiary care teaching hospital in western Rajasthan India. Infect Disord Drug Targets. 2021;21(2):257–61. DOI: 10.2174/1871526520666200318110447.
11. Kaur J, Bhat P, Bhumbla U. Fosfomycin susceptibility in multidrug resistant uropathogens: a retrospective study in the era of antimicrobial resistance. J Family Med Prim Care. 2025;14(4):1346–51. DOI: 10.4103/jfmpc.jfmpc_1074_24.
12. Schoevaerdts D, Bogaerts P, Grimmelprez A, de Saint-Hubert M, Delaere B, Jamart J, et al. Clinical profiles of patients colonized or infected with extended-spectrum beta-lactamase producing Enterobacteriaceae isolates: a 20 month retrospective study at a Belgian University Hospital. BMC Infect Dis. 2011;11:12. DOI: 10.1186/1471-2334-11-12.
13. Mohapatra S, Panigrahy R, Tak V, Shwetha JV, Sneha KC, Chaudhuri S, et al. Prevalence and resistance pattern of uropathogens from community settings of different regions: an experience from India. Access Microbiol. 2022;4(2):000321. DOI: 10.1099/acmi.0.000321.
14. Tamma PD, Heil EL, Justo JA, Mathers AJ, Satlin MJ, Bonomo RA. Infectious Diseases Society of America 2024 guidance on the treatment of antimicrobial-resistant Gram-negative infections. Clin Infect Dis. 2024;ciae403. DOI: 10.1093/cid/ciae403.
15. Aruna K, Mobashshera T. Prevalence of extended spectrum beta-

lactamase production among uropathogens in South Mumbai and its antibiogram pattern. EXCLI J. 2012;11:363–72.
16. Tulara NK. Nitrofurantoin and fosfomycin for extended spectrum beta-lactamase producing Escherichia coli and Klebsiella pneumoniae. J Glob Infect Dis. 2018;10(1):19–21. DOI: 10.4103/jgid.jgid_72_17.