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Phenotypic And Molecular Characterization Of Esbl-Producing Escherichia Coli In Urinary Tract Infections At A Tertiary Care Centre In India

Dr. Sultan Ahmad[1], Dr. Amit Kumar Singh[2], Dr. Soni Sinha[3], Dr. Nashra Afaq[4], Dr. Rajesh Yadav[5], Dr. Rubeena Bano[6], Dr Nadeem Ahmad[7], Dr. Noor Jahan[8]

[1]Assistant Professor, Department of Microbiology, Prasad Institute of Medical Sciences, Uttar Pradesh, India.
[2]Assistant Professor, Department of Microbiology, Prasad Institute of Medical Sciences, Uttar Pradesh, India.
[3]Assistant Professor, Department of Microbiology , Prasad Institute of Medical Sciences, Uttar Pradesh, India.
[4]Assistant Professor, Department of Microbiology and Central Research Laboratory, Rama Medical College Hospital and Research Centre, Uttar Pradesh, India.
[5]Professor & Head, Department of Microbiology, Prasad Institute of Medical Sciences, Uttar Pradesh, India
[6]Associate Professor, Department of Physiology, College of Medicine, Taibah University, Madinah KSA.
[7]Associate Professor, Department of Family and Community Medicine and Medical Education College of Medicine , Taibah University ,Madinah KSA
[8]Professor, Department of Microbiology, Integral Institute of Medical Sciences & Research, Uttar Pradesh, India

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*Corresponding Author:

Dr. Noor Jahan
Professor, Department of Microbiology, Integral Institute of Medical Sciences & Research, Uttar Pradesh, India

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A B S T R A C T

Background: Extended-spectrum beta-lactamase (ESBL)-producing Escherichia coli has emerged as a major uropathogen with significant therapeutic challenges. This study aimed to investigate the prevalence, antimicrobial resistance pattern, and molecular characteristics of ESBL-producing E. coli isolated from urine samples in a tertiary care hospital.

Aim and Objective: To study the phenotypic and molecular characterization of ESBL-producing *Escherichia coli* in urinary tract infections at a tertiary care centre.

Material and Methods: A total of 836 urine samples were processed; 366 (49%) yielded *E. coli* isolates. Phenotypic screening for ESBL was done using ceftazidime-clavulanic acid disk synergy test. PCR was performed for detection of blaTEM, blaCTX-M, and blaSHV genes in ESBL-positive isolates.

Results: Out of 366 *E. coli* isolates, 82 (22.5%) were ESBL producers. Among them, 53.7% carried blaTEM, 35.4% blaCTX-M, and 10.9% blaSHV. Female predominance was seen (70.5%). Most isolates showed 100% susceptibility to meropenem, nitrofurantoin, fosfomycin, and ceftazidime-clavulanate, while high resistance was noted against ceftriaxone and ceftazidime.

Conclusion: The high frequency of ESBL-producing *E. coli* among urinary isolates emphasizes the need for routine molecular detection and prudent antibiotic use in clinical settings.

Keywords: E. coli, ESBL, blaTEM, blaCTX-, blaSHV, urinary tract infection, PCR

INTRODUCTION

Urinary tract infections (UTIs) are among the most frequent bacterial infections encountered globally, affecting both hospitalized and community-based populations. *Escherichia coli* accounts for up to 80% of uncomplicated UTIs and remains the principal causative agent, especially among females due to anatomical predispositions [1].

The global rise of antimicrobial resistance has severely complicated UTI management. One of the critical contributors to this crisis is the emergence and spread of extended-spectrum β-lactamase (ESBL)-producing organisms, notably *E. coli*. ESBLs are enzymes capable of hydrolyzing penicillins, third-generation cephalosporins, and aztreonam, rendering many β-lactam antibiotics ineffective [2,3]. These enzymes are often encoded by transferable plasmids, facilitating rapid dissemination among Enterobacteriaceae [4].

Among the different types of ESBLs, the most frequently detected genes are TEM (Temoneira), SHV (Sulfhydryl Variable), and CTX-M (cefotaximase), with CTX-M variants currently dominating the epidemiological landscape worldwide [5,6]. The detection and characterization of these enzymes are crucial for epidemiological surveillance and guiding antimicrobial therapy.

India has reported a high prevalence of ESBL-producing organisms, particularly in tertiary care hospitals, raising serious concerns about treatment failures, prolonged hospital stays, and increased healthcare costs [7,8]. The overuse and misuse of antibiotics in the community and hospital settings are key drivers behind this resistance pattern [9,10].

Phenotypic detection methods like the combined disk test and double-disk synergy test (DDST) have been widely used in clinical laboratories. However, these methods lack the specificity and sensitivity offered by molecular techniques. Polymerase Chain Reaction (PCR)-based detection of bla_{TEM}, bla_{SHV}, and bla_{CTX-M} genes offers accurate and rapid results, enabling effective infection control and epidemiological monitoring [11-14].

The dissemination of ESBL genes has also been linked to mobile genetic elements like integrons, transposons, and insertion sequences, increasing their public health relevance [15,16].

In rural India, where empirical therapy is common, delayed or inaccurate detection of resistance genes further aggravates the problem. Compounding this challenge is the limited availability of molecular diagnostic tools in secondary care centers [17].

Therefore, the present study was undertaken to determine the prevalence of ESBL-producing *E. coli* in urinary isolates and to characterize the presence of blaTEM, blaCTX-M, and blaSHVgenes using PCR, alongside their antibiotic susceptibility patterns.

MATERIALS AND METHODS

This was a cross-sectional, observational study conducted over a 12-month period at a tertiary care hospital in the Department of Microbiology. A total of 836 non-repetitive urine samples were collected and processed for microbiological analysis.

Inclusion Criteria

1. Patients of all age groups with clinically suspected urinary tract infections.
2. Indoor and outdoor patients attending the hospital.
3. Patients who provided informed consent.

Exclusion Criteria

1. Patients who received antibiotics within 48 hours before urine sample collection.
2. Duplicate isolates from the same patient.
3. Non-*E. coli* urinary isolates.

Sample Collection and Processing

Urine samples were collected in sterile containers using midstream clean-catch technique. Samples were cultured on MacConkey and Blood agar and incubated aerobically at 37°C for 24 hours. Isolated organisms were identified using standard biochemical methods.

Phenotypic Screening of ESBL

Isolates of *E. coli* were subjected to the phenotypic confirmatory test using the Combined Disk Test (CDT) with ceftazidime (30 µg) and ceftazidime-clavulanic acid (30/10 µg) as per CLSI guidelines (2021). A ≥5 mm increase in zone diameter with the combination disk was interpreted as ESBL positive.

Antimicrobial Susceptibility Testing

Antibiotic susceptibility testing was done using the Kirby-Bauer disk diffusion method on Mueller-Hinton agar. Antibiotics tested included ampicillin-sulbactam, gentamicin, cefoxitin, amikacin, ciprofloxacin, meropenem, ceftazidime, ceftriaxone, nitrofurantoin, tigecycline, piperacillin-tazobactam, fosfomycin, and ceftazidime-clavulanate.

Molecular Characterization by PCR

DNA was extracted from ESBL-positive isolates using the boiling method. PCR was used to detect the presence of blaTEM, blaCTX-M, and blaSHVgenes using specific primers.

PCR cycling conditions for each gene were standardized as follows:
TEM: Initial denaturation at 98°C for 5 min; 35 cycles of denaturation at 98°C for 30s, annealing at 51°C for 30s, extension at 72°C for 30s; final extension at 72°C for 5 min.
CTX-M and SHV: Similar conditions with annealing at 55°C.
Amplicons were visualized on 1.5% agarose gel electrophoresis using ethidium bromide staining.

RESULTS
Throughout the learning period a total 836 urine samples were collected and processed. Out of 836 which 366 (49%) were showing Isolates of *E. coli* isolates. In which 82 (22.5%) were phenotypically identified as ESBL producers and 284 (77.6%) were Non-ESBL (Fig. 1). Among 82 ESBL producing strains, 26 (31.7%) belong to Outdoor patients and 56 (68.3%) belong to Indoor patients (Fig.2).

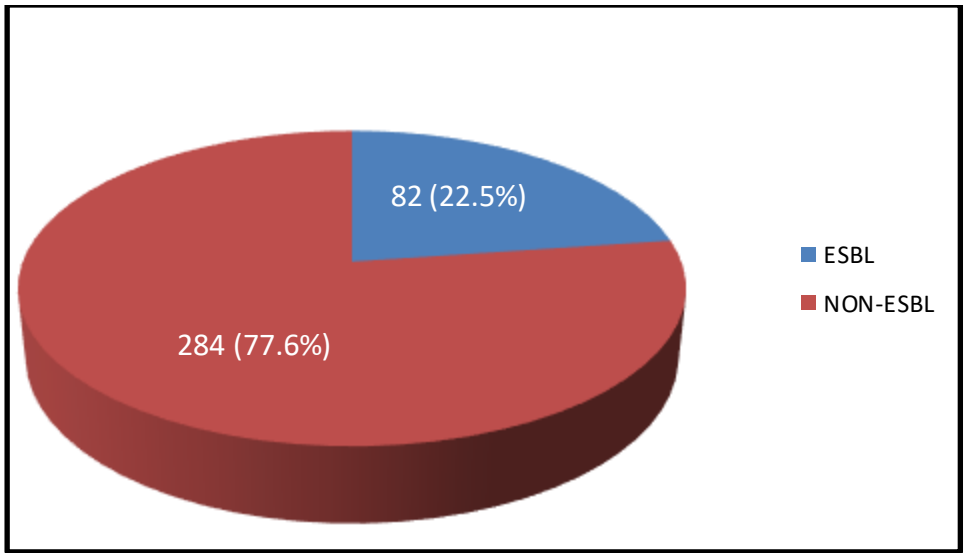

Figure 1 Distribution of *E.coli* Isolates among ESBL / Non-ESBL

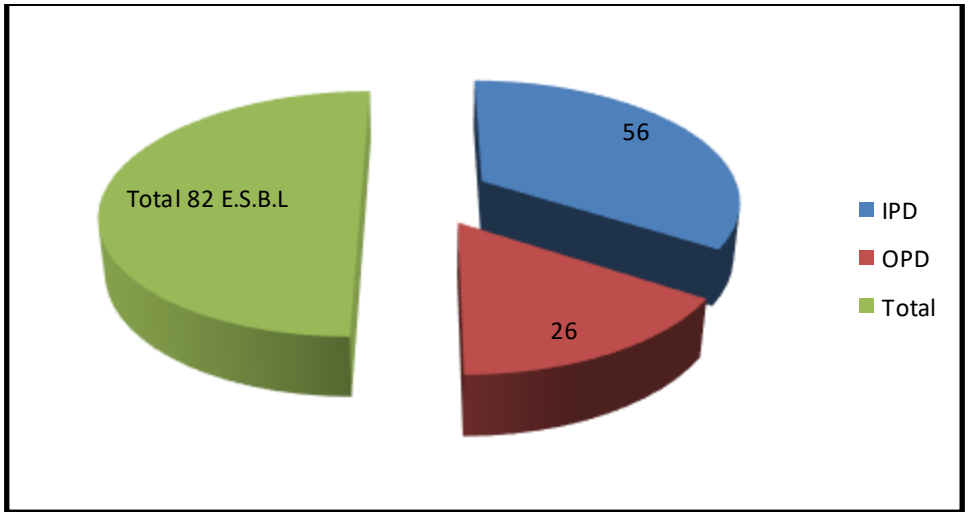

Figure 2: Distribution of E.S.B.L producing E.*coli* Isolates among IPD/OPD Patients

Table 1 shows that Out of 366 patients who were included in this study 108 (29.5%) were Male & 258 (70.5%) were Female patients.

Table 1 Distribution of patients according to Gender (Male/ Female)

Distribution of patients according to Gender (Male/ Female)	No. of patients	Percentage %
Male	**108**	**29.5**
Female	**258**	**70.5**
Total	**366**	**100**

Table 2 shows that the largest percentage of cases (24.1%) occurs among patients aged 21 to 30 years, while the lowest percentage (0.9%) occurs among patients aged 80 and above.

Table 3: Distribution of Patients according to Age in tubular form

Distribution of patients according to age group	No. of patients	Percentage (%)
0-10	37	10.2
11-20	40	10.7
21-30	88	24.1
31-40	54	15.1
41-50	50	13.5
51-60	56	15.3
61-70	23	6.3
71-80	14	3.9
81-90	3	0.9
Total	366	100

Table 3 shows the distribution of patients according to their residence 36.4% of the patients were from urban areas, while 63.6% of the patients were from rural areas.

Table 4 Distribution of Patients according to their residence status in tubular form

Distribution of patients according to their residence status	No. of Patients	Percentage %
Urban	133	36.4
Rural	233	63.6
Total	366	100

Table 4 shows the distribution of patients according to their educational status. It was found that majority (74.4%) patients were illiterate followed by, 15.3% patients pre primary, 6% patients primary school, 2.7% patients high school and 1.6% patients were found to be graduate or above.

Table 5: Distribution of patients according to their educational status

Educational status	No. of Patients	Percentage %
Graduate and above	6	1.6
High School	10	2.7
Primary School	22	6
Pre primary	56	15.3
Illiterate	272	74.4
Total	366	100

Table 5 shows the distribution of patients based on socioeconomic status. A total of 6% of the 366 patients were upper class, followed by middle class patients (25.2%) and lower class patients (68.8%).

Table 6: Distribution of Patients according to their Socio-economical status

Socio economical status	No. of Patients	Percentage %
Upper	22	6
Middle	92	25.2
Lower	252	68.8
Total	366	100

Table 7 shows the distribution of patients according to their Susceptibility of ESBL Isolates to various Antibiotics (N=82) status.

Table 7: Distribution of Antimicrobial susceptibility of ESBL Isolates (N=82) status

ANTIMICROBIAL	CATEGORY	NUMBER	PERCENTAGE
AMPICILLIN/SULBACTUM	RESISTANT	58	70.7%
	SUSCEPTIBLE	24	29.3%
GENTAMICIN	RESISTANT	30	36.6%
	SUSCEPTIBLE	52	63.4%
CEFOXITIN	RESISTANT	40	48.8%
	SUSCEPTIBLE	42	51.2%
AMIKACIN	RESISTANT	14	17.1%
	SUSCEPTIBLE	68	82.9%
CIPROFLOXACIN	RESISTANT	40	48.8%
	SUSCEPTIBLE	42	51.2%
MEROPENAM	RESISTANT	0	0
	SUSCEPTIBLE	82	100%
CEFTAZIDIME	RESISTANT	82	100%
	SUSCEPTIBLE	0	0
CEFTAZIDIME/CLAVULANIC ACID	RESISTANT	0	0
	SUSCEPTIBLE	82	100%
PIPERACILLIN-TAZOBACTUM	RESISTANT	12	14.6%
	SUSCEPTIBLE	70	85.4%
CEFTRIAXONE	RESISTANT	82	100%
	SUSCEPTIBLE	0	0
NITROFURANTOIN	RESISTANT	0	0
	SUSCEPTIBLE	82	100%
TIGECYCILINE	RESISTANT	2	2.4%
	SUSCEPTIBLE	80	97.6%
FOSFOMYCINE	RESISTANT	0	0
	SUSCEPTIBLE	82	100%

Table 8: Association of Antimicrobial Susceptibility with Demographic Characteristics.

VARIABLE	CATEGORY	ANTIMICROBIAL		CHI SQUARE	P VALUE
		AMPICILLIN			
AGE GROUP		RESISTANT	SUSCEPTIBLE		

	5-10YEARS	7	0		
	10-20YEARS	9	2		
	20-30YEARS	17	5		
	30-40YEARS	13	0		
	40-50YEARS	9	1	6.35	0.499
	50-60YEARS	9	3		
	60-70YEARS	4	1		
	70-80YEARS	2	0		
GENDER	MALE	26	4	0.0641	0.800
	FEMALE	44	8		
TYPEOF ADMISSION	IPD	48	8	0.0172	0.896
	OPD	22	4		

VARIABLE	**CATEGORY**	**ANTIMICROBIAL**		**CHI SQUARE**	**P VALUE**
AGEGROUP		**AMPICILLIN/SULBACTUM**			
		RESISTANT	SUSCEPTIBLE		
	5-10YEARS	6	1		
	10-20YEARS	5	6		
	20-30YEARS	14	8		
	30-40YEARS	13	0	12.5	0.086
	40-50YEARS	8	2		
	50-60YEARS	7	5		
	60-70YEARS	3	2		
	70-80YEARS	2	0		
GENDER	MALE	24	6		
	FEMALE	34	18	1.96	0.161
TYPEOF ADMISSION	IPD	38	18		
	OPD	20	6	0.705	0.401
VARIABLE	CATEGORY	ANTIMICROBIAL		CHI SQUARE	P VALUE
AGEGROUP		GENTAMICIN			
		RESISTANT	SUSCEPTIBLE		
	5-10YEARS	1	6		
	10-20YEARS	6	5		
	20-30YEARS	6	16		
	30-40YEARS	5	8	6.03	0.536
	40-50YEARS	4	6		
	50-60YEARS	6	6		
	60-70YEARS	2	3		
	70-80YEARS	0	2		
GENDER	MALE	8	22		
	FEMALE	22	30	2.01	0.157

TYPEOF ADMISSION	IPD	20	36		
	OPD	10	16	0.0578	0.810
VARIABLE	CATEGORY	ANTIMICROBIAL		CHI SQUARE	P VALUE
		CEFOXITIN			
AGEGROUP		RESISTANT	SUSCEPTIBLE		
	5-10YEARS	4	3		
	10-20YEARS	7	4		
	20-30YEARS	11	11		
	30-40YEARS	6	7		
	40-50YEARS	4	6	1.92	0.964
	50-60YEARS	5	7		
	60-70YEARS	2	3		
	70-80YEARS	1	1		
GENDER	MALE	8	22	9.26	0.002*
	FEMALE	32	20		
TYPEOF ADMISSION	IPD	30	26	1.62	0.203
	OPD	10	16		
VARIABLE	CATEGORY	ANTIMICROBIAL		CHI SQUARE	PVALUE
		AMIKACIN			
AGEGROUP		RESISTANT	SUSCEPTIBLE		
	5-10YEARS	1	6		
	10-20YEARS	1	10		
	20-30YEARS	4	18		
	30-40YEARS	4	9		
	40-50YEARS	1	9	3.07	0.878
	50-60YEARS	2	10		
	60-70YEARS	1	4		
	70-80YEARS	0	2		
GENDER	MALE	4	26	0.467	0.494
	FEMALE	10	42		
TYPEOF ADMISSION	IPD	7	49	2.61	0.106
	OPD	7	19		
VARIABLE	CATEGORY	ANTIMICROBIAL		CHI SQUARE	PVALUE
		CIPROFLOXACIN			
AGEGROUP		RESISTANT	SUSCEPTIBLE		
	5-10YEARS	3	4		
	10-20YEARS	7	4		

	20-30YEARS	10	12		
	30-40YEARS	8	5		
	40-50YEARS	4	6	2.72	0.910
	50-60YEARS	5	7		
	60-70YEARS	2	3		
	70-80YEARS	1	1		
GENDER	MALE	10	20	4.52	0.034*
	FEMALE	30	22		
TYPE OF ADMISSION	IPD	26	30	0.391	0.532
	OPD	14	12		
VARIABLE	CATEGORY	ANTIMICROBIAL		CHI SQUARE	P VALUE
		PIPERACILLIN/TAZOBACTUM			
AGEGROUP		RESISTANT	SUSCEPTIBLE		
	5-10YEARS	2	5		
	10-20YEARS	0	11		
	20-30YEARS	4	18		
	30-40YEARS	1	12		
	40-50YEARS	1	9	6.03	0.537
	50-60YEARS	2	10		
	60-70YEARS	1	4		
	70-80YEARS	1	1		
GENDER	MALE	4	26	0.0641	0.800
	FEMALE	8	44		
TYPEOF ADMISSION	IPD	7	49	0.644	0.422
	OPD	5	21		
VARIABLE	CATEGORY	ANTIMICROBIAL		CHI SQUARE	P VALUE
		TIGICYCILINE			
AGEGROUP		RESISTANT	SUSCEPTIBLE		
	5-10YEARS	0	7		
	10-20YEARS	0	11		
	20-30YEARS	1	21		
	30-40YEARS	0	13		
	40-50YEARS	0	10	3.36	0.850
	50-60YEARS	1	11		
	60-70YEARS	0	5		
	70-80YEARS	0	2		
GENDER	MALE	0	30	1.18	0.277
	FEMALE	2	50		
TYPE OF ADMISSION	IPD	1	55	0.317	0.574
	OPD	1	25		

MOLECULAR RESULTS
POLYMERASE CHAIN REACTION (PCR)
PCR was used to amplify the TEM, CTX-M, and SHV gene sequences.

STEP	Program TEM				
	Time	Temperature	Time	Temperature	Cycles
Initial denaturation	5 min.	98 ºC	5 min	98ºC -	
Denaturation	30 s	98 ºC	28 s	98º C	
Annealing	30 s	51 ºC	29 s	55 ºC	35
Extension	30 s	72º C	30 s	72º C	
Final Extension	5 min.	72 ºC	5 min.	72 ºC	

Table 9: The PCR cycling conditions to amplify TEM gene fragments.
***bla*-TEM gene Results**

TARGET GENE	PRIMER	LENGTH
*bla*TEM	Forward-5: ATGAGTATTCAACATTTCCGTG-3 Reverse-5: TTACCAATGCTTAATCAGTGAG-3	861

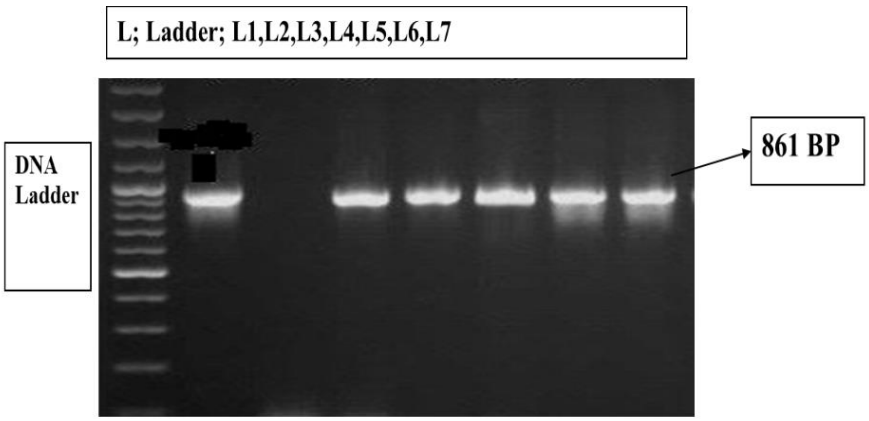

Figure 3: Amplified DNA with PCR for *bla*TEM gene

L corresponds to the DNA Ladder.
L1 corresponds to the positive.
Control: L2 corresponds to Negative control *bla*TEM gene.
L3-L7 are the sample positive for *bla*TEM gene.

STEP	Program CTX-M				
	Time	Temperature	Time	Temperature	Cycles
Initial denaturation	5 min.	98 °C	5 min.	98°C	
Denaturation	30 s	98 °C	28 s	98° C	
Annealing	30 s	51 °C	29 s	55°C	35
Extension	30 s	72° C	30 s	72° C	
Final Extension	5 min.	72 ° C	5 min.	72 ° C	

Table 10: The PCR cycling condition to amplify CTX-M gene fragments.
***bla*-CTX gene Results**

TARGET GENE	PRIMER	LENGTH
blaCTX	Forward-5: SCSATGTGCAGYACCAGTAA-3 Reverse-5: CCGCRATATGRTTGGTGGTG-3	544

L1-L9, L10, L11-16, L19, 20

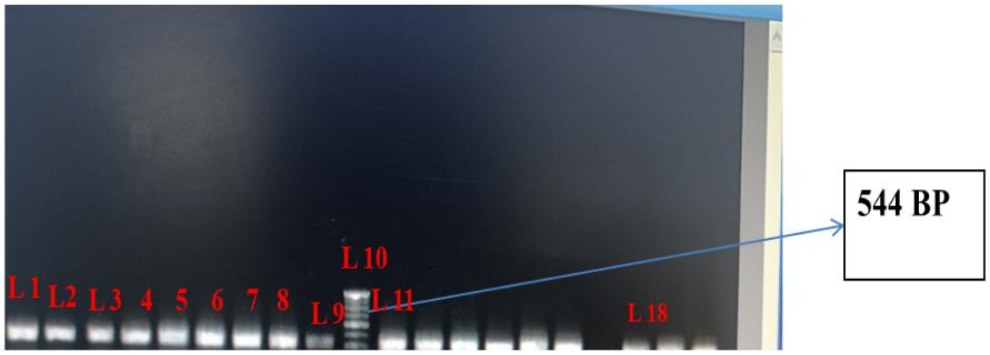

Figure 4: Amplified DNA with PCR for CTX-M gene.

Lane: 1-9 show positive for CTX gene
Lane: 10 DNA Ladder
Line: 11-16 and 19, 20 CTX genes positive
Lane: 17 Negative control for CTX gene
Lane: 18 show positive control for CTX GENE

STEP	Program SHV				
	Time	Temperature	Time	Temperature	Cycles
Initial denaturation	5 min	98 ºC	5 min	98ºC	35
Denatunation	30 s	98 ºC	28 s	98º C	
Annealing	30 s	51 ºC	29 s	55 ºC	
Extension	30 s	72º C	30 s	72º C	
Final extention	5 min.	72º C	5 min.	72 º C	

Table 11: The PCR cycling condition to amplify SHV gene fragments.
***bla* SHV Results**

TARGET GENE	PRIMER	LENGTH
*bla*SHV	Forward-5: TTATCTCCCTGTTAGCCACC-3 Reverse-5: GATTTGCTGATTTCGCTCGG-3	795

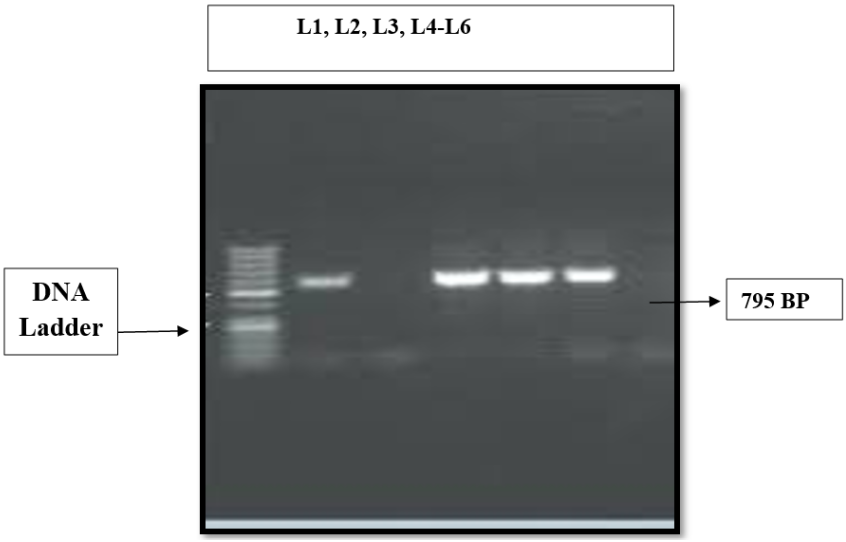

Figure 5: Amplified DNA with PCR for *bla* SHV gene

L1 corresponds to the DNA Ladder.
L2 corresponds to the positive Control.
L3 Corresponds to the Negative Control to *bla*SHV gene;
L4-L6 sample positive for *bla*SHV gene

Table and figure shows that out of 82 ESBL positive patients, gene TEM was seen in 53.7% patients, gene CTX-M was seen in 35.4% patients & gene SHV was observed in 10.9% patients.

Table 12: Detection of different genes in ESBL producing *E.coli* isolates.

Results	No. of Gene detected	Percentage %
TEM	44	53.7
CTX-M	29	35.4
SHV	9	10.9
Total	82	100

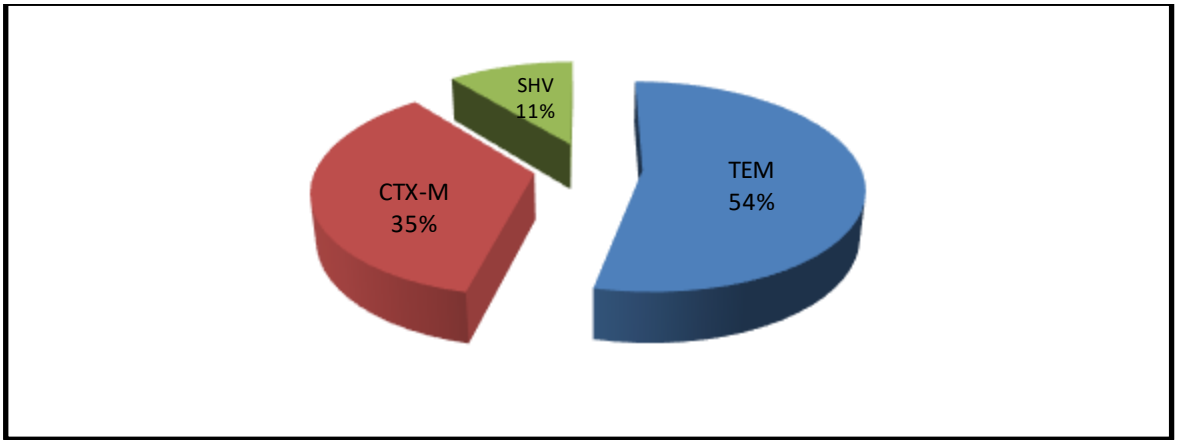

Figure 6: Detection of different genes in ESBL producing *E.coli* isolates.

DISCUSSION

Urinary tract infections (UTIs) remain one of the most common bacterial infections, with Escherichia coli as the predominant uropathogen. The emergence of extended-spectrum beta-lactamase (ESBL)-producing E. coli complicates the empirical management of UTIs due to their resistance to cephalosporins and penicillins. In the present study, 22.5% of the 366 E. coli isolates were found to be ESBL producers, which aligns with findings from studies conducted across India and other parts of the world. For instance, Grover et al. reported an ESBL prevalence of 24% among E. coli isolates in their North Indian cohort [18m, while Taneja et al. found a rate of 20.1% in hospitalized patients [19].

The present study highlights the prevalence and molecular profile of ESBL-producing E. coli in urinary tract infections. A total of 22.5% of E. coli isolates were ESBL positive, which is in concordance with other Indian studies. Gupta et al. [14] reported 26%, and Jain et al. [8] reported 29.3% ESBL prevalence among uropathogens.

The demographic profile of patients in our study reveals a higher prevalence of infections among females (70.5%) compared to males (29.5%), which is consistent with previous findings by Colodner et al., who highlighted female predominance due to shorter urethra and closer proximity to the anal canal [20]. Furthermore, the majority of patients were from rural backgrounds and belonged to the lower socioeconomic class, which has also been associated with higher UTI risks due to poor hygiene and limited healthcare access [21].

The predominance of female patients (70.5%) is consistent with findings by Foxman et al. [1] and Rawat and Nair [11], who observed higher UTI incidence in women due to anatomical and hormonal factors. The highest prevalence was in the 21–30-year age group, similar to findings by Sharma et al. [13] and Singh et al. [7].

Our antimicrobial susceptibility results revealed that ESBL-producing E. coli showed 100% susceptibility to carbapenems (meropenem), nitrofurantoin, fosfomycin, and ceftazidime-clavulanic acid. This is in concordance with previous reports by Paterson and Bonomo, who emphasized the high efficacy of carbapenems against ESBL-producing strains [22]. Similarly, Manoharan et al. and Rodrigues et al. also observed similar susceptibility trends with nitrofurantoin and fosfomycin [23, 24].

Antibiotic resistance profiling revealed 100% resistance to ceftriaxone and ceftazidime in ESBL strains. Similar resistance patterns were observed by Paterson and Bonomo [2] and Bradford [3], who emphasized ESBLs' role in hydrolyzing third-generation cephalosporins. However, complete susceptibility to carbapenems (meropenem), nitrofurantoin, and fosfomycin was noted, consistent with findings by Canton and Coque [5] and Jain and Mondal [8].

On the contrary, resistance to ceftriaxone and ceftazidime was universally observed in ESBL producers, which is expected as these are third-generation cephalosporins commonly hydrolyzed by ESBL enzyme [25]. Ciprofloxacin resistance was seen in 48.8% of the isolates, comparable to a study by Gupta et al., where fluoroquinolone resistance exceeded 50% [26]. This growing resistance limits the options for oral outpatient treatment and necessitates stringent antimicrobial stewardship.

Molecular characterization in our study detected the blaTEM gene in 53.7% of isolates, blaCTX-M in 35.4%, and blaSHV in 10.9%. This gene distribution is consistent with several studies across India. For example, Naseer and Sundsfjord documented the predominance of the TEM gene globally [27] . Similarly, Shahid et al. also reported TEM as the most frequently detected gene in their isolates from North India [28] . However, CTX-M genes have been increasingly reported in Western and South Indian studies, suggesting regional genetic variation [29,30].

PCR-based detection showed blaTEM (53.7%) as the predominant gene, followed by bla(35.4%) and blaSHV(10.9%). Similar trends were reported by Bonnet [6], who highlighted the dominance of CTX-M in Europe, but TEM still prevails in South Asia. A study by Shaikh et al. [12] also reported TEM dominance in E. coli from Indian clinical samples.

Interestingly, we observed a statistically significant association of ciprofloxacin and cefoxitin resistance with male gender ($p<0.05$), which has not been extensively explored in prior studies but may reflect gender-based pharmacokinetic or behavioral differences requiring further investigation.

Other molecular studies from India, including that by Singh et al. [17] and Gupta and Datta [14], showed a high prevalence of TEM and CTX-M genes, affirming our findings. Carattoli [15] emphasized the role of plasmids in gene dissemination, which may explain the high occurrence in both community and hospital settings.

The presence of multiple ESBL genes in a single isolate has been noted in various studies including Poirel et al. [16], who found co-expression of TEM and CTX-M in 34% of isolates, possibly due to integrons or transposons. In our study, some strains were positive for more than one gene.

Gender-wise distribution showed statistically significant differences in resistance to cefoxitin and ciprofloxacin, which was also observed in a study by Kumarasamy et al. [9]. They hypothesized that biological and treatment-seeking behavior may influence resistance patterns.

Our results also support the use of ceftazidime-clavulanic acid and piperacillin-tazobactam as empirical therapy, as resistance rates were low (0–14.6%). Similar empirical recommendations were made by the CLSI (2021) and supported by Sharma et al. [13]

Overall, the integration of molecular techniques alongside phenotypic testing enhances diagnostic precision, which is echoed by studies from Shaikh et al. [12], Carattoli [15], and Paterson et al. [2]. PCR not only confirms the presence of genes but also aids in epidemiological surveillance. Thus, this study emphasizes the need for continuous surveillance, rational antibiotic usage, and mandatory molecular characterization in tertiary care centers.

Our results reinforce the urgent need for continuous surveillance of ESBL prevalence and gene patterns. Jain et al. emphasized the critical role of molecular typing in understanding the local epidemiology of antimicrobial resistance [31] . Moreover, Nathisuwan et al. proposed that integrating molecular diagnostics into routine labs can guide appropriate therapy and infection control measures[32].

Our findings underscore the importance of using antibiotics like nitrofurantoin and fosfomycin in outpatient UTI management and reserving carbapenems for complicated or resistant cases. The high susceptibility rates to these drugs are supported by findings from Kaur et al. and Karunasagar et al. [33,34].

Overall, this study adds valuable data to the regional antimicrobial resistance database and supports the need for rapid phenotypic and genotypic screening of ESBL-producing uropathogens to prevent treatment failures and limit resistance spread.

CONCLUSION

This study highlights a significant proportion of ESBL-producing *E. coli* isolates in UTIs, with the blaTEMgene being the most prevalent, followed by blaCTX-M and blaSHV. These strains exhibit high resistance to third-generation cephalosporins but retain susceptibility to meropenem, nitrofurantoin, fosfomycin, and ceftazidime-clavulanate. Regular screening, molecular typing, and effective antibiotic stewardship are essential to curb the spread of resistant strains.

LIMITATIONS

1. This was a single-center study; results may not be generalizable to other regions.
2. Only three ESBL genes were tested; other ESBL or AmpC genes were not included.
3. Genotypic analysis was limited to PCR; sequencing could provide deeper insights.
4. The study lacked follow-up on clinical outcomes post-treatment.

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