International Journal of Community Medicine and Public Health
Das P et al. Int J Community Med Public Health. 2025 Jan;12(1):193-200
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20244017

Original Research Article

Escherichia coli in West Bengal: active surveillance of uropathogenicity, ESBL, MDR and XDR

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Received: 24 September 2024
Revised: 22 November 2024
Accepted: 02 December 2024

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ABSTRACT

Background: Urinary tract infection (UTI) is one of the *Escherichia coli* (*E. coli*) induced extraintestinal disorders that dramatically raise morbidity and mortality rates. Treatment ineffectiveness resulted from the emergence of multidrug resistance (MDR), extensive-drug resistance (XDR), and extended-spectrum β-lactamases (ESBL). This study in West Bengal, India, explored the prevalence of uropathogenic, ESBL, MDR, and XDR *E. coli* strains.
Methods: It was a cross-sectional study. Examining 77 clinical isolates from diverse regions of West Bengal, the research identified ESBL, AmpC, metallo-β-lactamase (MBL), MDR, and XRD production through phenotypic methods and polymerase chain reaction (PCR).
Results: Disc agar diffusion (DAD) test detected 84.4% as ESBL producers, with 31.19% AmpC, 6.49% MBL, and 12.98% carbapenem-resistant isolates. 87.01% and 2.95% isolates were suspected to be MDR and XDR. The presence of specific 16S rRNA identifies all isolates as *E. coli*. Confirmation via PCR revealed ESBL genes in 92.2% of isolates, predominantly bla_{CTX-M} (75.3%), bla_{TEM} (62.3%), and bla_{SHV} (22.1%). Uropathogenicity was confirmed in 80.5% of isolates, all of which co-occurred with ESBL genes.
Conclusions: The study underscores the common occurrence of uropathogenic and antibiotic-resistant *E. coli* in tertiary care settings, emphasizing the need for robust antimicrobial stewardship and strict infection control practices to address the proliferation of drug-resistant pathogens.

Keywords: *Escherichia coli*, Extended-spectrum β-lactamase, Extensive drug resistance, Multi-drug-resistance, Uropathogenicity, West Bengal

INTRODUCTION

Escherichia coli (*E. coli*) is the most prevalent facultative anaerobic enteric bacteria commonly found in the environment, food, and gastrointestinal tract of humans and animals.[1] The most frequent etiologic agent, Uropathogenic *Escherichia coli* (UPEC), is currently accountable for over 90% of all Urinary tract infections (UTIs) that occur worldwide.[2] Most cases of *E. Coli* infection occur when patients use community care services, are hospitalized, or consume vegetables contaminated by inadequately cleaned water or biocide compounds used during cultivation.[1,3] The recent development of antimicrobial resistance in *E. coli* is of particular concern due to its ability to spread easily and its potential to cause serious infections.[4,5] The global community has reported multi-drug resistance (MDR) and extensive drug resistance (XDR) *E. Coli* resistance to most first-line antimicrobial drugs, including cotrimoxazole, ampicillin, and nitrofurantoin.[3] MDR is facilitated by a wide range of intricately interconnected processes, including target protection, enzyme inactivation, and drug efflux.[6,7] Production of extended-spectrum β-lactamase (ESBL) is one of the major reasons causing MDR.[6] Unfortunately, Enterobacteriaceae that produce β-lactamases exhibit resistance to most

commonly used antibiotic classes, including aminoglycosides, trimethoprim-sulfamethoxazole, and fluoroquinolones.[8] This poses a great deal of difficulties and leaves very few therapeutic options in a clinical setting.[8,9] Therefore, monitoring these Enterobacteriaceae that produce β-lactamase is crucial for providing clinical care.[8]

Whole genome sequencing (WGS) is gradually taking the place of phenotypic antimicrobial susceptibility testing in many affluent nations' anti-microbial resistance (AMR) surveillance systems.[10] However, surveillance systems are still not well established in many low- and middle-income countries because of their high expenses.[11] Research has indicated that the disc diffusion method (DAD) is an economical approach that can produce outcomes that are on par with other phenotypic techniques for assessing antibiotic susceptibility, including broth microdilution or agar dilution methods.[12]

In India, the prevalence of ESBL, AmpC, and metallo-β-lactamase (MBL) producing *E. coli* has been increasing steadily in recent years.[8] This is of particular concern as ESBL-producing *E. coli* can cause severe infections, including urinary tract infections, bacteremia, childhood diarrhea, septicemia, hospital-acquired pneumonia, intra-abdominal abscess, brain abscess, device-related infections, and meningitis.[13] The goal of the current investigation was to ascertain the prevalence of MDR, XDR, and ESBL *E. coli* in our geographic region because there has been no documentation addressing its prevalence concerning UTIs.

METHODS

Study design

This cross-sectional study involves patients who had UTIs who received medical treatment across primary, secondary, and tertiary care facilities in West Bengal.

Study population

From 2020 to 2022, people with recurrent UTIs who were treated at primary, secondary, and tertiary healthcare facilities in West Bengal were included in the study. Patients with bacterial infections other than UTIs were excluded.

Sample size calculation

For estimating the prevalence, the sample size was calculated using Scalex Calculator considering the expected prevalence of 95% from the pilot study conducted in our laboratory in 2020 and the potential loss/attrition of 5% with the absolute precision of ±5%.[14] Formula used for sample size calculation is as follows:

$$n = Z^2P(1-P)/d^2$$

Where, n=sample size, Z=Z score for level of significance, P=expected prevalence (if the expected prevalence is 20%, then P=0.2), and d=precision (if the precision is 5%, then d=0.05).

Collection of bacterial strains

From 2020 to 2022, pre-cultured clinical samples were obtained from different UTI patients at different hospitals and private laboratories located in West Bengal, including North 24 Pargana, South 24 Pargana, East Midnapur, West Midnapur, Howrah, Durgapur, Murshidabad and Kolkata. The sample source of these isolates was urine. The collected samples were streaked onto MacConkey agar and then incubated at 37˚C for 24 h and stored in the refrigerator at 4˚C for future usage. In addition, reference strains *E. coli* ATCC 25922, ATCC 35218, *Klebsiella pneumoniae* ATCC 700603, and ATCC 25955 were used for comparison.

Identifications of bacterial strains

Identification of bacterial isolates was carried out using Gram staining, conventional biochemical methods (IMViC testing), and the presence of *E. coli*-specific 16s rRNA.[15,16]

Antibiotic susceptibility testing

Following guidelines from the Clinical Laboratory Standard Institute (CLSI), all isolates that were verified to be *E. coli* were tested using the Kirby-Bauer disc agar diffusion (DAD) method for antibiotic sensitivity to a panel of 16 different drugs. The concentrations of each disc's antibiotics were amoxicillin-clavulanic acid 20/10 µg (AMC), amoxicillin 10 µg (AMX), azithromycin 15 µg (AZI), cefoxitin 30 µg (FOX), cefotaxime 30 µg (CTX), chloramphenicol 30 µg (CHL), levofloxacin 5 µg (LEV), cotrimoxazole 1.25/23.75 µg (COT), imipenem 10 µg (IMP), fosfomycin 200 µg (FOS), amikacin 30µg (AMK), tetracycline 30 µg (TET), nitrofurantoin 300µg (NTF), cefixime 5 µg (CFX), ceftazidime 30 µg (CTZ) and aztreonam 30 µg (AZT). The sizes of the inhibition zones surrounding the discs impregnated with antibiotics were measured and compared to the Clinical and Laboratory Standard Institute (CLSI) clinical breakpoints after rounding off to the nearest integer, to determine if each bacterial isolate is resistant, intermediate, or susceptible.

All the bacterial isolates that are not susceptible (i.e., intermediate and resistant) to third-generation cephalosporins (cefotaxime and ceftazidime), cefoxitin, and imipenem were expected to have a phenotype reflecting ESBL- or AmpC or MBL production. Suspected beta-lactamase-producers were discriminated by the combination disc test (CDT) according to CLSI and European Committee on Antimicrobial Susceptibility Testing (EUCAST) guidelines using a second panel of 12 antibiotic-impregnated discs.

According to CLSI guidelines ceftazidime (30 µg), ceftazidime-clavulanate (30/10 µg), ceftazidime-phenylboronic acid (30/300 µg), ceftazidime-phenylboronic acid-clavulanate (30/300/10 µg) and imipenem (10 µg) discs were used to determine ESBL and AmpC producer.[17] In accordance with the EUCAST standards, suspected beta-lactamase-producing bacteria were distinguished using another 2 panels of eight antibiotic-impregnated discs, with 4 discs in each panel. First panel contains discs impregnated with cefotaxime (30 µg) (CTX), cefotaxime/clavulanic acid (30/10 µg) (CEC), cefotaxime/cloxacillin (30/200 µg) (CTC), and cefotaxime/clavulanic acid/cloxacillin (30/10/200 µg) (CCC). Antibiotic-impregnated discs in the second panel were meropenem (10 µg) (MRP), meropenem-phenylboronic acid (10/200 µg) (MRB), meropenem-cloxacillin (10/200 µg) (MCL), meropenem-EDTA (10/750 µg) (MRE). The results of these CDT were interpreted according to Shoorashetty et al and EUCAST guidelines.[17]

E. coli ATCC 25922, ATCC 35218, *Klebsiella pneumoniae* ATCC 700603, and ATCC 25955 were used as quality control strains.

Genotypic characterization of E. coli, ESBL, and uropathogenic genes by PCR

The *E. coli* gene (16S rRNA), ESBL gene (bla_{TEM}, bla_{SHV}, bla_{CTX-M}), and uropathogenic gene (UPEC c3509) were detected in collected *E. coli* samples isolated by polymerase chain reaction (PCR) and confirmed by agarose gel electrophoresis. Briefly, bacterial genomic DNA was extracted from the isolates using the Himedia DNA isolation kit and measured by Spectrophotometer (260/280) for its purity and concentration. PCR was performed in 25 µl PCR volume using 5 µl of DNA template, 1 µl of each gene-specific primer (bla_{TEM}: F-TTGGGTGCACGAGTGGGTTA, R-TAATTGTTGCCGGGAAGCTA, T_a - 55˚C; bla_{SHV}: F-AGGATTGACTGCCTTTTTG, R-ATTTGCTGATTTCGCTCG, T_a - 53˚C; bla_{CTX-M}: F-CGATGTGCAGTACCAGTAA, R-TAGGTCACCAGAACCAGCGG, T_a - 53˚C; *E. coli* 16S RNA: F- GGAAGAAGCTTGCTTCTTTGCTGAC, R-AGCCCGGGGATTTCACATCTGACTTA, T_a - 60˚C; UPEC c3509: F- ACAATCCGCCACCATCCA, R-ACAATCCGCCACCATCCA, T_a - 58˚C), and 12.5µl of Hi-Chrom PCR master mix (Himedia).[2,16,18,19] The products were placed for electrophoresis. The cycling parameters were as follows: 95°C for 2 minutes followed by 45 cycles of 95°C for 1 minute, specific annealing temperature (T_a) for 30 sec, and 72˚C for 1 minute, with a final extension at 72°C for 10 minute.

5 µl of the PCR product was mixed with 1 µl loading buffer and separated on a 2% agarose gel containing 0.5 µg/ml of ethidium bromide in TAE buffer. UV light was used to visualize amplified products (Figure 6-10).

Data analysis

The prevalence proportion of bacteria resistant to each tested antibiotic was, expressed as a percentage and the Clopper-Pearson interval reflecting 95% binomial proportions were used to calculate confidence intervals using SPSS software. The fraction of samples containing bacteria that are resistant or intermediate to antimicrobials was determined by dividing the number of samples with non-susceptible bacteria by the total number of samples collected.

RESULTS

For the expected prevalence of 95%, the required sample size was calculated to be 77 for the margin of error or absolute precision of ±5% in estimating the prevalence with 95% confidence and considering the potential loss/attrition of 5%. In this investigation, 77 patients with a mean age of 46.37037±11.4928 were included. The majority of participants were female, accounting for 62.34% of the samples. While 68.83% reported using shared toilets, 35.06% experienced more than two episodes of infections in six months. For sanitation, the majority were using groundwater. The characteristics of the patients that were part of this investigation are summarized in Table 1. All these 77 precultured clinical samples were confirmed to be *E. coli* by IMViC screening and the presence of *E. coli*-specific 16S RNA gene (Figure 6A).

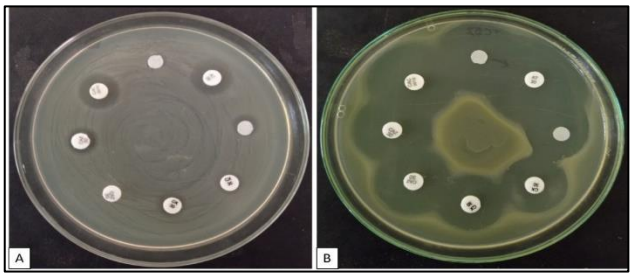

Figure 1: Antibiogram of *E. coli* isolate Kirby-Bauer disc diffusion method: A) multi-drug resistant *E. coli* isolate; B) drug-sensitive *E. coli* isolate.

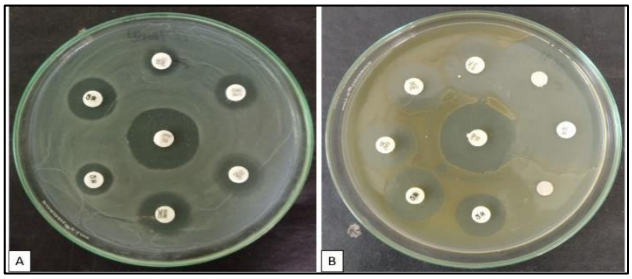

Figure 2: Phenotypic detection of ESBL-AmpC producing *E. coli* according to Shoorashetty et al: A) ESBL-AmpC co-producing *E. coli* isolate; B) ESBL-AmpC negative *E. coli* isolate.[23]

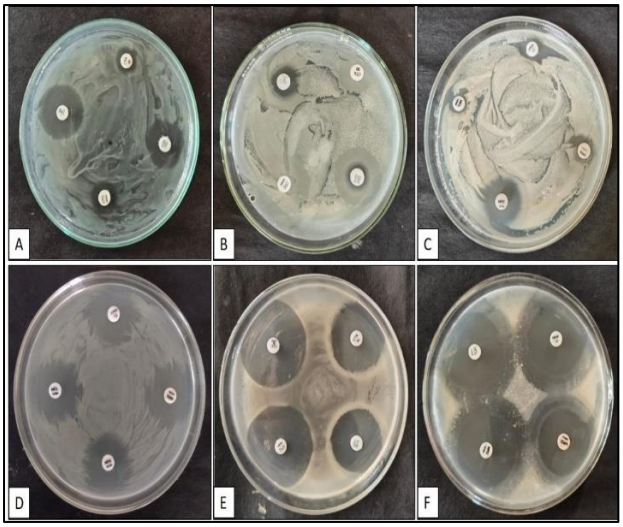

Figure 3: Phenotypic detection of ESBL, AmpC, MBL producers and carbapenem-resistant E. coli according to EUCAST guidelines: A) detection of ESBL; B) Detection of ESBL-AmpC co-producer; C) detection of MBL; D) detection of carbapenem resistance; E) detection of ESBL-AmpC negative isolate; F) detection of carbapenem sensetive isolate.

A dominant type of resistance, based on the DAD results (Figure 1-3), was found to amoxicillin and amoxicillin-clavulanate conjugates with a 95% CI of 93.0-100.0 followed by ceftazidime (92.2%; 95% CI: 83.8-97.1), cefotaxime (92.2%; 95% CI: 83.8-97.1), cefixime (75.32%; 95% CI: 64.2-84.4), aztreonam (75.32%; 95% CI: 64.2-84.4), nitrofurantoin (61.03%; 95% CI: 49.2-72.0) and azithromycin (55.84%; 95% CI: 44.1-67.2). A maximum of the samples was susceptible to imipenem leaving only 12.98% (95% CI: 6.4-22.6) of resistance to it (Table 2) (Figure 4). All these isolates were resistant to more than one antibiotic but only 67 (87.01%; 95% CI: 77.4-93.6) showed resistance to at least one agent in three different antimicrobial categories. In addition, 2 (2.59%; 95% CI: 0.3-9.1) of them showed sensitivity to only one or two antimicrobial classes while resistant to all others.

The phenotypic profile of all the non-susceptible isolates to third-generation cephalosporins is shown in Figure 5. From all the samples tested 65 (84.41%; 95% CI: 74.4-91.7) were ESBL, 24 (31.16%; 95% CI: 21.1-42.7) were AmpC and 5 (6.49%; 95% CI: 2.1-14.5) were MBL producers. The CDT method also revealed that 23 (29.87%; 95% CI: 20.0-41.1) isolates were co-producers of ESBL and AmpC.

Table 1: Demographic and clinical characteristics of the patients.

Parameters	Frequency	Percentage/Mean±SD#
Age (Mean±SD)		46.37037±11.4928
Gender (%)		
Male	29	37.66
Female	48	62.34
Number of family members (Mean±SD)		5.185±2.82
Family income (Mean±SD)		6707.41±3817.11
Education (%)		
Nil	9	11.69
Primary	3	3.90
Middle	38	49.35
High	16	20.78
Graduate and above	11	14.29
Occupation (%)		
Household	41	53.25
Service	28	36.36
Other	8	10.39
Sanitation habit (%)		
Use of common toilet	53	68.83
Use of personal toilet	24	31.17
Source of water for sanitation (%)		
Supplied water	24	31.17
Groundwater	48	62.34
Pond water	5	6.49
Recurrence of UTI (%)		
2 infections in 6 months	50	64.94
More than 2 infections in 6 months	27	35.06

#SD= Standard Deviation.

Table 2: Distribution of inhibition zone diameters of *E. coli* (n=77) isolated on Muller Hinton agar.

ATB	%	95%	Distribution (numbers) for inhibition zone diameters in mm																									
	R	CI	0	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	>30
TET	38.96	28.0-50.8	15	4	7	2	2				3	2		1	1	1	1		11		6		2		2		9	9
AMK	33.76	23.4-45.4	5		1	3	4		5	1	7	3	10	2	7		2		4		11	1	1		6	1	2	1
NTF	61.03	49.2-72.0	17	2	4	3	1		8		12	2	10	2	9	1	3		1		1						1	
FOS	31.16	21.1-42.7	8		8		3		5		1	1	1	1	1	2	4		3		4	4	9		6	1	10	5
AMX	98.70	93.0-100.(	42	1	3	5	7	6	4	8	1					1												
CFX	75.32	64.2-84.4	29	7	14	3	2		3				3		3		5		5	1	3							
AZI	55.84	44.1-67.2	16		8	1	6	3	9		12	2	3		2	1	5		2		1	2		1			3	
LEV	24.67	15.6-35.8	9	1	1	2	3	2	1		3		4		5	1	5		6	3	3	3	1	1	1		9	13
IMP	12.98	6.4-22.6	4		2		2			2							5	1	5	5	4	1	9	3	6		4	24
COT	29.87	20.0-41.4	19	1	3				4	1	2	2	3		3		3	2	6		6	3	5		5		6	3
CHL	23.37	14.5-34.4	3		2	10	1		2	1	2		1		10	4	4		5	3	6	2	5	3	8	1	4	
CTZ	92.20	83.8-97.1	35	5	5		8	3	4		1	1	9		2	1		1	2									
CTX	92.20	83.8-97.1	18	4	4	4	3	1	1	1	1	5	6	3	2	2	2	11	3	3			2					
AZT	75.32	64.2-84.4	15	2	3	3	8	1	7	4	7	4	4		2	1	2	1	1		4	1	4	1		2		
AMC	98.70	93.0-100.(	42	4	12		9	1	3	5	1														1			
FOX	33.76	23.4-45.4	11	1	1	4	1		4	3	1	4	1	1	2	4	8	7	6	4	6	4	3		1	1		

In accordance with the Clinical Laboratory Standards Institute (CLSI) human clinical break points, pink, light blue, and light green fields represent numbers of isolates with inhibition zone diameters for resistant, intermediate and susceptible Enterobacteriaceae, respectively. TET: tetracycline, AMK: amikacin, NFT: nitrofurantoin, FOS: Fosfomycin, AMX: amoxicillin, CFX: cefixime, AZI: azithromycin, LEV: levofloxacin, IMP: imipenem, COT: cotrimoxazole, CHL: chloramphenicol, CTZ: ceftazidime, CTX: cefotaxime, AZT: aztreonam, AMC: amoxicillin-clavulanic acid, FOX: cefoxitin. ATB: Antibiotic, R: resistance, CI: Confidence intervals were calculated as 95% binomial proportions and presented as Clopper-Pearson interval.

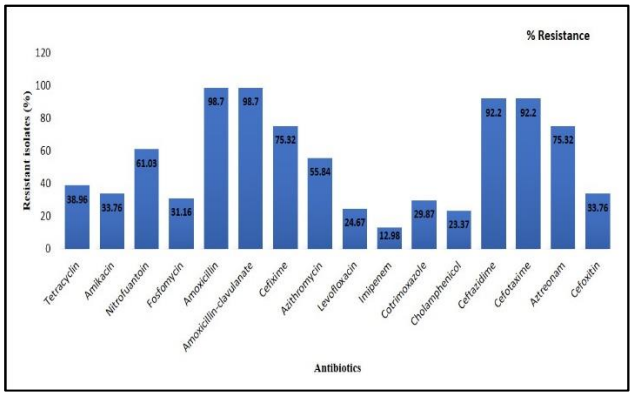

Figure 4: Antibiotic resistance pattern of *E. coli* isolates.

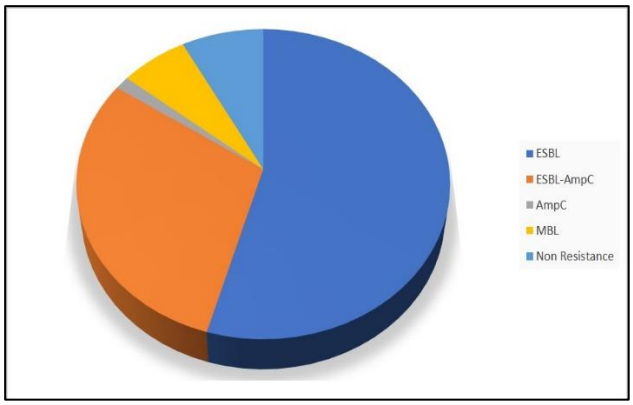

Figure 5: Prevalence of β-lactamases and their co-existence in *E. coli*.

All the collected isolates were confirmed to be *Escherichia coli* by genotypic sequencing 16s rRNA. Occurrences of ESBL-specific genes were found in 71 (92.2%; 95% CI: 83.8-97.1) isolates. The most common ESBL encoding gene detected was bla_{CTX-M} (75.3%; 95% CI: 64.2-84.4) followed by bla_{TEM} (62.3%; 95% CI: 50.6-73.1) and bla_{SHV}. (22.1%; 95% CI: 13.4-33.0) (Figure 6 B-D). Among these 77 clinical isolates, 62 (80.5%; 95% CI: 69.9-88.7) samples were harboring the uropathogenic gene UPEC c3509 (Figure 6E).

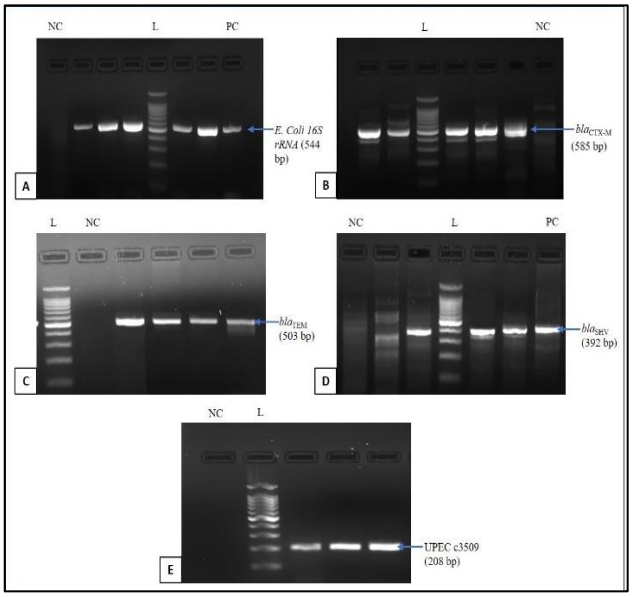

Figure 6: Gel picture of amplified PCR product: A) *E. Coli* 16S rRNA., NC: Negative control: K. pneumoniae ATCC 25955 ; L: Ladder; PC: Positive control: E. coli ATCC 25922; B) blaCTX-M, L- ladder, NC-negative control *E. coli* ATCC 25922; C) bla_{TEM}, NC-negative control *E. coli* ATCC 25922, L- ladder; D) bla_{SHV}, NC- negative control *E. coli* ATCC 25922, L-ladder, PC- positive control *K. pneumoniae* ATCC 700603; E) UPEC c3509, NC- negative control *E. coli* ATCC 25922, L- ladder.

DISCUSSION

Antimicrobial resistance is spreading throughout the world, endangering both efficient infection prevention and treatment as well as global public health.[20] *Acinetobacter sp.* showed the highest levels of resistance, followed by *E. coli*, according to the European Antimicrobial Resistance Surveillance Network and the U.S. National Healthcare Safety Network.[21] According to reports from multiple research conducted in Indian hospitals, the prevalence of *E. Coli* that produces ESBL is higher than that of AmpC and carbapenemase.[22] In South India, 53% of patients suffering from community-acquired bacteremia carry drug-resistant *E. coli*.[23] Among MDR Gram-negative bacteria isolated from neonatal septicemia in tertiary institutions, the most frequent ones are *Acinetobacter*, *Klebsiella* and *E. coli*.[24] Likewise, the most common microorganisms responsible for pediatric urinary tract infections (UTIs) are *E. Coli* and *Klebsiella* Sp.[25,26] One of the most prevalent infectious disorders worldwide is urinary tract infection, which can be brought on by both Gram-positive and Gram-negative bacteria.[27] When compared to Gram-positive bacteria, studies discovered that Gram-negative bacteria were more common culprits in UTIs.[28] *E. coli* is the major pathogen responsible for UTIs, according to numerous research.[29] cases of UTIs increased by 60.40% from 1990 to 2019 worldwide.[30]

In our study, we found 87% of the patients, taking primary, secondary, and tertiary care treatment, are infected with MDR-*E. coli*. with the highest resistance to amoxicillin, third-generation cephalosporins, cephamycins, monobactams, macrolides, and nitrofurans. A study conducted in Mumbai, India, showed that cases of MDR gram-negative bacteria increased steadily from 2012-2014. An estimated 700,000 fatalities globally occur due to MDR bacteria each year, and by 2050, that number is expected to rise to 10 million deaths.[31] In addition, 2 of the total 77 cases were found to be extensively drug-resistant (XDR) leaving only one or two antimicrobials in the treatment regimen. Production of ESBL, AmpC, and MBL are the major reasons behind this multi-drug resistance. According to several additional Indian research, the prevalence of ESBLs is between 60 and 89%.[8,32,33] The prevalence, according to our current investigation, is 92.2% (genotypically). In line with earlier research from India, 31.16% and 6.49% of the *E. coli* in our investigation produced Amp C and MBL respectively.[34] Co-existence of ESBL and AmpC was observed in 29.81% of isolates and 12.98% of *E. coli* samples exhibit carbapenem resistance.

ESBL producers also showed cross-resistance to aztreonam, cefixime, nitrofurantoin, azithromycin, and tetracycline. The most prevalent gene for producing ESBL in *E. coli* was $bla_{\text{CTX-M}}$, followed by bla_{TEM} and bla_{SHV} respectively. Cefotaxime (and ceftriaxone) are significantly preferred substrates by $bla_{\text{CTX-M}}$ over ceftazidime.[35,36] However, in our investigation, we found equivalent resistance to both ceftazidime and cefotaxime. Some previous studies evidenced that isolates harboring a $bla_{\text{CTX-M-32}}$, a member of $bla_{\text{CTX-M-1}}$, exhibit resistance to both ceftazidime and cefotaxime in a similar manner.[37]

Many individuals experience UTIs, which are frequent bacterial infections.[38] Women are more likely than males to get a UTI and ESBL-positive *E. coli* frequently cause complicated UTIs.[39] The use of common toilets and contaminated groundwater worsens the situation by spreading the infection. In our investigation, 80.5% of clinical isolates were uropathogenic and all of them were ESBL positive. This finding is also in concordance with some previous studies conducted in India.[40] Of all cases, 35.06% experienced more than 2 episodes of infection within 6 months, and all of them were infected with *E. coli* harbouring more than one ESBL-producing gene.

The initial two years of the study took place during the COVID-19 pandemic, a time when healthcare facilities were overcrowded, elevating the risk of infection transmission. Conversely, some individuals adhered to the quarantine measures, avoided social gatherings and practiced proper sanitation. Consequently, the probability of infection transmission was decreased. As a result, the findings from this period may not fully reflect the current situation. However, this investigation sheds light on the emergence of antimicrobial resistant uropathogenic *E. coli* strains and their associated risks.

CONCLUSION

The present study highlights the significant incidence of ESBL, AmpC-producing and carbapenem-resistant E. Coli isolates across West Bengal, India. Concerns are raised about the situation by the fact that these resistant organisms are also cross-resistant (MDR or XDR) to the common medications utilized in clinical settings. The increase in carbapenem resistance is very alarming as carbapenem is considered to be the strongest antimicrobial till date. Genotypic analysis is always found to be the gold standard though phenotypic analysis is done frequently in low- and middle-income countries. In most Indian hospitals, however, it is not a routine procedure to screen for and report multidrug-resistant pathogens. Thus, a collaboration of microbiologists and physicians is strongly advised to monitor patterns of pathogen resistance. The management and prevention of the spread of these resistant bacteria in the hospital setting and community could be aided by selecting the course of treatment, upholding appropriate cleanliness, and conducting epidemiological surveys.

Funding: This research was funded by the Council of Industrial and Scientific Research (CSIR), grant number 08/0749(0001)/2019-EMR-I
Conflict of interest: None declared
Ethical approval: The study was approved by the Department of Physiology, University of Calcutta (Ref. No.: IHEC/SSM/P79/2019)

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Cite this article as: Das P, Ghorai T, Shaw K, Mazumder S. *Escherichia coli* in West Bengal: active surveillance of uropathogenicity, ESBL, MDR and XDR. Int J Community Med Public Health 2025;12:193-200.