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"Detection and Antimicrobial resistance patterns of extended-spectrum Beta-lactamase producing *Escherichia coli* among Urinary tract infection patients"

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ABSTRACT

Introduction: Urinary Tract Infections (UTIs) are common bacterial infections, often nosocomial, affecting any part of the urinary system. *Escherichia coli*, a Gram-negative bacillus, is the predominant uropathogen. UTI is an infection which is occur in any part of urinary tract most of UTIs involves the lower urinary tract includes bladder and urethra, Although women are more suspected than mens due tothe anatomical structure of their urinary tract so that bacterium can easily move there and develop infection, The emergence of Extended-Spectrum Beta-Lactamase (ESBL) producing *E. coli* has become a critical concern due to its ability to confer multi-drug resistance (MDR), rendering many standard antibiotics ineffective and complicating treatment.

Objectives: The study was designed to determine the frequency of *E. coli* isolates in symptomatic UTI patients; assess associated risk factors; evaluation of the antibiotic susceptibility pattern of the clinical isolates and identification of the proportion of ESBL-producing *E. coli* among them.

Methods: This retrospective study was conducted on 100 patients with symptoms of UTI. Urine samples were collected aseptically and processed in the bacteriology laboratory. Semi-quantitative culture was performed on CLED agar using the standard loop method. Bacterial identification was done using standard biochemical tests and the automated VITEK-2 system. Antibiotic susceptibility testing (AST) was performed by the Kirby-Bauer disk diffusion method, and ESBL production was phenotypically confirmed using the Double Disc Synergy Test (DDST).

Results: Out of 100 samples, 78 showed significant bacteriuria ($\geq 10^5$ CFU/mL) with *E. coli*. The frequency of ESBL-producing *E. coli* was 39 out of these 78 isolates (50%), while the remaining 39 (50%) were non-ESBL producers. The ESBL isolates demonstrated high levels of resistance to third-generation cephalosporins, cefepime, and co-trimoxazole, but remained largely susceptible to carbapenems, nitrofurantoin, and aminoglycosides.

Conclusion: The study reveals a high prevalence (50%) of ESBL-producing *E. coli* among UTI patients in our setting. The significant level of multi-drug resistance observed necessitates routine screening for ESBL production in all *E. coli* isolates. These findings are crucial for guiding appropriate empiric antibiotic therapy and implementing effective infection control strategies to curb the spread of these resistant pathogens.

Index Terms: ESBL, UTI, *Escherichia coli*, Antibiotic Resistance, MDR, DDST.

1. INTRODUCTION

An estimated 150 million people are afflicted with urinary tract infections (UTIs) each year, making it one of the most common bacterial illnesses in the world [1]. Any portion of the urinary tract, including the urethra, bladder, ureters, and kidneys, may be infected [2]. Particularly in susceptible groups like infants and children, UTIs can result in severe side effects such pyelonephritis, renal insufficiency, hypertension, and chronic kidney disease if they are not identified early and treated appropriately [10].

About 80–85% of UTI infections worldwide are caused by Gram-negative bacteria belonging to the Enterobacteriaceae family [2]. The most often isolated bacteria among these is Escherichia coli, which causes 70–90% of community-acquired UTIs and a sizable percentage of hospital-acquired UTIs [9, 10]. Females are particularly vulnerable to recurring UTIs due to the anatomy of the female urethra [3].

Historically, antibiotic medication has been the mainstay of UTI care. However, the quick development and spread of antibiotic resistance is seriously undermining these treatments' efficacy. The generation of beta-lactamase enzymes is the most common mechanism of resistance to beta-lactam drugs among Enterobacteriaceae [1, 3, 14,17]. These enzymes make medicines like cephalosporins and penicillins ineffective by hydrolyzing their beta-lactam ring [3, 15].

The challenge of antimicrobial resistance intensified significantly with the emergence of Extended-Spectrum Beta-Lactamases (ESBLs), first documented in 1983 [1, 2]. By hydrolyzing oxyimino-cephalosporins and monobactams, these enzymes render key antibiotics ineffective, though they do not typically compromise cephamycins or carbapenems [1, 2, 11]. ESBLs are frequently produced by common uropathogens, most notably *Escherichia coli* and *Klebsiella pneumoniae* [1, 13]. Consequently, the management of urinary tract infections has evolved from a straightforward task into a significant therapeutic hurdle, marked by rising rates of morbidity and mortality [3, 16]. This review aims to synthesize current knowledge on ESBL-producing *E. coli* in UTIs, addressing the underlying mechanisms, approaches to detection, and the subsequent strain on public health.

2. ESBL PRODUCING ESCHERICHIA COLI AND MECHANISM OF RESISTANCE

Escherichia coli is a versatile microorganism, existing as a commensal inhabitant of the human and animal gastrointestinal tract while also being readily isolated from environmental reservoirs such as water and soil [12]. In its pathogenic role, *E. coli* employs an array of virulence factors that facilitate extraintestinal infections. A key mechanism by which this pathogen evades antibiotic therapy is the production of beta-lactamase enzymes [3, 14].

Beta-lactam antibiotics, including penicillins and cephalosporins, exert their bactericidal activity by inhibiting cell wall synthesis. This is achieved through the covalent binding of the drug to penicillin-binding proteins (PBPs), essential transpeptidases involved in peptidoglycan cross-linking [14, 15]. Extended-Spectrum Beta-Lactamases (ESBLs) are evolved variants of narrow-spectrum beta-lactamases (e.g., TEM-1, SHV-1, and OXA). The acquisition of specific mutations has extended their hydrolytic capacity to include a broader range of beta-lactam substrates [13, 14]. Consequently, ESBL-producing *E. coli* circumvents antibiotic action by producing enzymes that hydrolyze the beta-lactam ring—a structural component critical for drug activity—via the addition of a water molecule, rendering the antibiotic incapable of binding to its PBP target [15].

The genes responsible for encoding ESBLs are predominantly plasmid-borne. These extrachromosomal DNA elements serve as highly efficient vehicles for horizontal gene transfer, facilitating the dissemination of resistance traits not only between different bacterial strains but also across species. This plasmid-mediated mechanism is a primary driver of multidrug resistance, as the relevant plasmids frequently harbor additional resistance determinants against other clinically important antibiotic classes, including aminoglycosides, trimethoprim-sulfamethoxazole, and fluoroquinolones [12, 15]. As a result, ESBL-producing organisms are often multidrug-resistant (MDR), a term defined as acquired resistance to at least three distinct antimicrobial classes [12].

The major ESBL families are phylogenetically classified into three main groups: TEM, SHV, and CTX-M. Although the TEM and SHV types were historically dominant, the epidemiological landscape has shifted dramatically. Variants of the CTX-M family have now emerged as the most prevalent and clinically significant ESBL types worldwide, representing a dominant lineage in both hospital and community settings [8, 13].

3. RISK FACTORS AND EPIDEMIOLOGY

- The incidence of UTIs caused by ESBL-producing organisms has risen significantly around the globe, posing a serious issue in both patients admitted to hospitals and those in the community[1,3, 11]. This spread is driven by various critical risk factors:
- **Prolonged Hospitalization and ICU Stays:** Extended exposure to the hospital environment increases the risk of acquiring MDR organisms [11, 13].
- **Long-term and Indiscriminate Antibiotic Use:** The empirical and symptomatic use of broad-spectrum antibiotics, without a proper diagnosis, alters the normal intestinal flora and creates selective pressure, favoring the growth of resistant bacteria [11, 12].
- **Underlying Medical Conditions:** Patients with severe illness, diabetes mellitus, and renal insufficiency are at higher risk [13].
- **Instrumentation:** Urinary catheterization provides a direct route for bacteria to enter the urinary tract [13].
- **Residence in Long-Term Care Facilities:** Nursing homes can be reservoirs for MDR organisms [13].

- **Poor Sanitation and Hygienic Practices:** Inadequate sanitation facilitates the fecal-oral transmission of resistant bacteria, especially in community settings [11].

In India, the prevalence of ESBL-producing uropathogens is strikingly high, with reported figures between 39.5% and 58% [11, 12]. This high resistance rate is particularly worrisome given its spread beyond adults; ESBL-producing *E. coli* is now increasingly isolated from pediatric populations, including young children and infants [7, 8]. The clinical implications are profound, as pediatric upper UTIs (pyelonephritis) are a major risk factor for long-term sequelae such as renal scarring and hypertension [10]. This escalating resistance, combined with a heavy disease burden, is a key contributor to the estimated 700,000 global deaths annually resulting from antimicrobial-resistant infections [17].

4. LABORATORY DETECTION OF ESBL PRODUCTION

Prompt detection of ESBL-producing organisms is critical for guiding therapy and infection control. While urine culture remains the gold standard for UTI diagnosis, conventional antimicrobial susceptibility testing (AST) is hampered by a turnaround time of 48–72 hours [5, 6]. The Cica Beta Test, a chromogenic test based on the HMRZ-86 substrate, offers a more rapid alternative for detecting beta-lactamase activity directly from colonies. Its combination with clavulanic acid, an ESBL inhibitor, enables quick preliminary identification of ESBL production [5, 6]. Several phenotypic methods are routinely employed in microbiology laboratories for the confirmation of ESBL-producing *E. coli* [4]:

1. **Screening Test:** The initial step involves performing a standard disc diffusion test. Resistance to any of the oxyimino-cephalosporins (e.g., cefpodoxime, ceftazidime, cefotaxime, ceftriaxone) or aztreonam suggests possible ESBL production.
2. **Double Disc Synergy Test (DDST):** This is a simple and widely used method. Discs containing an oxyimino-cephalosporin (e.g., ceftazidime) and a disc containing amoxicillin-clavulanic acid (which inhibits ESBLs) are placed at a specific distance on a lawn of the test bacterium. A clear extension or "keyhole" of the inhibition zone towards the clavulanate-containing disc indicates synergy and confirms ESBL production.
3. **Inhibitory Potentiated Disc Diffusion (IPDD):** This method compares the inhibition zone of an antibiotic disc alone versus the same antibiotic disc combined with clavulanic acid. An increase of ≥5mm in the zone diameter for the combination disc confirms ESBL production.
4. **E-test (Strips):** This is a confirmatory test using a strip with a gradient of an oxyimino-cephalosporin (e.g., ceftazidime) on one end and a gradient of the same antibiotic plus clavulanic acid on the other. A ratio of the MIC for the antibiotic alone to the MIC for the antibiotic plus clavulanic acid of ≥8, or the presence of a deformation zone, confirms ESBL production.

AIM:

The primary goal of this research is to detect and characterize Extended-Spectrum Beta-Lactamase (ESBL) producing *Escherichia coli* isolated from patients presenting with urinary tract infections.

OBJECTIVES:

1. To estimate the frequency of *E. coli* as the causative organism in patients clinically diagnosed with a UTI.
2. To evaluate the various host-related and clinical risk factors that contribute to the acquisition of ESBL-producing *E. coli*.
3. To ascertain the antimicrobial resistance patterns of the isolated *E. coli* against a panel of commonly used antibiotics.
4. To specifically identify the proportion of *E. coli* isolates that are ESBL-producers using standard microbiological techniques.

MATERIALS AND METHODS

Study Design and Setting: This was a retrospective study conducted in the Department of Microbiology at SRMS Institute of Medical Sciences, Bareilly. The duration of the study was six months, from November 2023 to May 2024.

Sample Size: A total of 100 patients were included in the study.

Inclusion Criteria: The study enrolled patients presenting with symptoms indicative of a urinary tract infection (UTI), such as burning micturition, urgency, increased frequency, hesitancy, pain during urination, hematuria, and fever. All types of urine samples, including clean-catch midstream urine (MSU), catheter specimen of urine (CSU), and suprapubic aspirates, were considered for analysis.

Ethical Consideration: Prior approval for the study was obtained from the Institutional Human Ethics Committee. Informed written consent was secured from all participants, and their confidentiality was maintained throughout the research process.

METHODOLOGY

Sample Collection and Transport: Urine samples were collected from symptomatic patients in sterile, wide-mouthed, screw-capped universal containers. The collected specimens were promptly transported to the Bacteriology Laboratory. In the event of an anticipated delay, the samples were refrigerated at 4°C to prevent bacterial overgrowth.

Sample Processing [15]: Upon receipt in the laboratory, all urine samples were processed for culture using conventional bacteriological techniques.

- **Urine Culture:** A semi-quantitative culture method was performed using a standard loop technique on Cystine Lactose Electrolyte Deficient (CLED) agar [15]. A standard 4mm nichrome loop, calibrated to hold 0.005 mL of urine, was used to inoculate the sample onto the CLED agar plate. The plates were then incubated aerobically at 37°C for 18 to 24 hours. A colony count yielding 500 or more colonies, corresponding to $\geq 10^5$ colony-forming units per milliliter (CFU/mL), was considered indicative of significant bacteriuria.
- **Identification of Isolates:** Growth of uropathogens was identified following standard microbiological techniques [15]. The identification process involved:

1. **Colony Morphology:** Observing the characteristic appearance of colonies on CLED agar.
2. **Gram Staining:** Performing Gram staining to determine the bacterial cell morphology and Gram reaction.
3. **Motility Test:** Assessing motility using the hanging drop preparation.
4. **Biochemical Tests:** A battery of biochemical tests was employed, including catalase, oxidase, indole, citrate utilization, urease production, oxidative-fermentative (OF) glucose test, and Triple Sugar Iron (TSI) agar test. Sugar fermentation tests were also conducted.
5. **Automated Identification:** For definitive identification, the isolates were further processed using the automated VITEK-2 Compact system (bioMérieux) with GN (Gram-Negative) identification cards (REF-21341), strictly adhering to the manufacturer's instructions.

Biochemical Identification: Standard biochemical tests were performed for the identification of *Escherichia coli*. Key tests included the catalase test, oxidase test, indole test, and others as per standard protocols [15].

Antibiotic Susceptibility Testing (AST): Antibiotic susceptibility testing was conducted on Mueller-Hinton agar using the Kirby-Bauer disk diffusion method. The selection of antibiotic discs and the interpretation of susceptibility patterns (Sensitive, Intermediate, Resistant) were performed in accordance with the Clinical and Laboratory Standards Institute (CLSI) guidelines 2023 [16]. *Escherichia coli* ATCC 25922 was used as the quality control strain throughout the study. Furthermore, the Minimum Inhibitory Concentration (MIC) for specific antibiotics was determined using the VITEK-2 Compact system with GN card 235 (bioMérieux), following the manufacturer's instructions.

The following antibiotic discs (with concentrations) were used for the Kirby-Bauer method:

- **Cephalosporins:** Cefotaxime (30 µg), Ceftriaxone (30 µg), Cefepime (30 µg), Ceftazidime (30 µg), Cefpodoxime (10 µg), Cephalothin (30 µg).
- **Fluoroquinolones:** Ciprofloxacin (5 µg), Levofloxacin (5 µg), Ofloxacin (5 µg), Norfloxacin (10 µg).
- **Aminoglycosides:** Amikacin (30 µg), Gentamicin (120 µg), Tobramycin (10 µg), Netilmicin (30 µg).
- **Carbapenems:** Meropenem (10 µg), Imipenem (10 µg).
- **Others:** Doxycycline (30 µg), Chloramphenicol (30 µg), Cotrimoxazole (25 µg), Aztreonam (30 µg), Nitrofurantoin (300 µg), Colistin (10 µg), Polymyxin-B (300 units).
- **Beta-Lactam Combination Agents:** Cefoperazone+Sulbactam (50/10 µg), Amoxicillin+Clavulanic Acid (20/10 µg), Piperacillin+Tazobactam (100/10 µg).

Procedure for AST:

1. A sterile cotton swab was used to inoculate the Mueller-Hinton agar plate with the test organism to create a uniform bacterial lawn.
2. After inoculation, the antibiotic discs were placed aseptically onto the surface of the agar using a disc dispenser or sterile forceps.
3. The plates were incubated at 37°C for 18-24 hours.
4. Following incubation, the diameters of the zones of inhibition around each disc were measured in millimeters.
5. The measured zone diameters were compared with the CLSI 2023 interpretive chart to determine the organism's susceptibility category (Sensitive, Intermediate, or Resistant) for each antibiotic.

Phenotypic Screening for ESBL Production [7,8,15]: Screening for the production of Extended-Spectrum Beta-Lactamase (ESBL) enzymes was performed using the Double Disc Synergy Test (DDST).

- **Procedure for DDST:** A lawn of the test bacterial strain was cultured on Mueller-Hinton agar. Discs containing later-generation cephalosporins (e.g., Ceftazidime 30 µg, Cefotaxime 30 µg) were placed at a distance of 20-30 mm (center-to-center) from a disc containing Amoxicillin-Clavulanic Acid (20/10 µg). The plates were incubated overnight at 37°C.
- **Interpretation:** The test was considered positive for ESBL production if the inhibition zone around any of the cephalosporin discs was observed to be extended or distorted towards the disc containing clavulanic acid (the inhibitor). This synergy indicates that the clavulanic acid has inhibited the ESBL enzyme, restoring the susceptibility of the cephalosporin [7,8,15].

RESULTS AND DISCUSSION

A total of 100 urine samples from clinically suspected cases of UTI were enrolled in this study.

Culture Positivity: Out of 100 samples, 78 (78%) showed significant growth (≥10^5 CFU/mL), while 22 (22%) were sterile (Table 1).

Table 1: Overall Culture Positivity Rate (n=100)

Urine Culture Result	Number of Samples	Percentage (%)
Significant Growth	78	78%
Sterile	22	22%
Total	**100**	**100%**

Frequency of ESBL Producing *E. coli*: Among the 78 culture-positive samples, all were identified as *Escherichia coli*. The frequency of ESBL-producing *E. coli* was 39 out of 78 isolates (50%), and the frequency of non-ESBL *E. coli* was also 39 out of 78 isolates (50%), as shown in Table 2.

Table 2: Frequency of ESBL and Non-ESBL *E. coli* Isolates (n=78)

Type of Isolate	Number of Isolates	Percentage (%)
ESBL *Escherichia coli*	39	50%
Non-ESBL *Escherichia coli*	39	50%
Total Isolates	**78**	**100%**

Antibiotic Susceptibility Pattern of ESBL-producing *E. coli*: The antibiogram of the 39 ESBL-producing *E. coli* isolates is presented in Table 3. These isolates demonstrated complete resistance (100%) to several antibiotics, including cefotaxime, ceftriaxone, cefepime, ceftazidime, cefpodoxime, cephalothin, co-trimoxazole, cefoperazone+sulbactam, and amoxicillin+clavulanic acid. However, they showed high susceptibility to carbapenems (meropenem, imipenem), nitrofurantoin, colistin, and piperacillin+tazobactam. Variable susceptibility was noted for fluoroquinolones and aminoglycosides (with some isolates showing intermediate susceptibility).

Table 3: Antibiogram of ESBL-producing *Escherichia coli* Isolates (n=39)

Antibiotic	Resistant (R)	Intermediate (I)	Susceptible (S)
Cefotaxime	39 (100%)	0	0
Ceftriaxone	39 (100%)	0	0

Antibiotic	Resistant (R)	Intermediate (I)	Susceptible (S)
Cefepime	39 (100%)	0	0
Ceftazidime	39 (100%)	0	0
Cefpodoxime	39 (100%)	0	0
Cephalothin	39 (100%)	0	0
Ciprofloxacin	0	0	39 (100%)
Levofloxacin	0	0	39 (100%)
Ofloxacin	0	0	39 (100%)
Norfloxacin	0	0	39 (100%)
Amikacin	0	10 (25.6%)	29 (74.4%)
Gentamicin	0	8 (20.5%)	31 (79.5%)
Tobramycin	0	9 (23.1%)	30 (76.9%)
Netilmicin	0	7 (17.9%)	32 (82.1%)
Meropenem	0	0	39 (100%)
Imipenem	0	0	39 (100%)
Doxycycline	0	0	39 (100%)
Co-trimoxazole	39 (100%)	0	0
Cefoperazone + Sulbactam	39 (100%)	0	0
Amoxicillin + Clavulanic Acid	39 (100%)	0	0
Piperacillin + Tazobactam	0	0	39 (100%)
Nitrofurantoin	0	0	39 (100%)
Colistin	0	0	39 (100%)

Antibiotic Susceptibility Pattern of Non-ESBL *E. coli*: The antibiogram of the 39 non-ESBL *E. coli* isolates is shown in Table 4. While a majority were susceptible to many drugs, a subset of 8 isolates (20.5%) exhibited resistance to a panel of antibiotics

including ceftriaxone, cefepime, ceftazidime, co-trimoxazole, cefoperazone+sulbactam, amoxicillin+clavulanic acid, aztreonam, and colistin, indicating the presence of other resistance mechanisms.

Table 4: Antibiogram of Non-ESBL *Escherichia coli* Isolates (n=39)

Antibiotic	Resistant (R)	Intermediate (I)	Susceptible (S)
Cefotaxime	0	0	39 (100%)
Ceftriaxone	8 (20.5%)	0	31 (79.5%)
Cefepime	8 (20.5%)	0	31 (79.5%)
Ceftazidime	8 (20.5%)	0	31 (79.5%)
Cefpodoxime	0	0	39 (100%)
Cephalothin	0	0	39 (100%)
Ciprofloxacin	0	0	39 (100%)
Levofloxacin	0	0	39 (100%)
Ofloxacin	0	0	39 (100%)
Norfloxacin	0	0	39 (100%)
Amikacin	0	5 (12.8%)	34 (87.2%)
Gentamicin	0	6 (15.4%)	33 (84.6%)
Tobramycin	0	4 (10.3%)	35 (89.7%)
Netilmicin	0	3 (7.7%)	36 (92.3%)
Meropenem	0	0	39 (100%)
Imipenem	0	0	39 (100%)
Doxycycline	0	8 (20.5%)	31 (79.5%)
Co-trimoxazole	8 (20.5%)	0	31 (79.5%)
Cefoperazone + Sulbactam	8 (20.5%)	0	31 (79.5%)
Amoxicillin + Clavulanic Acid	8 (20.5%)	0	31 (79.5%)

Antibiotic	Resistant (R)	Intermediate (I)	Susceptible (S)
Piperacillin + Tazobactam	0	0	39 (100%)
Aztreonam	8 (20.5%)	0	31 (79.5%)
Nitrofurantoin	0	0	39 (100%)
Colistin	8 (20.5%)	0	31 (79.5%)

RESULTS AND DISCUSSION

In this retrospective study conducted at the Department of Microbiology, SRMS Hospital, Bareilly, we investigated the prevalence of ESBL-producing *Escherichia coli* in urinary tract infections and assessed their antibiotic susceptibility patterns. Among 100 patients, 78 (78%) demonstrated culture-proven UTIs, all caused by *E. coli*. Alarmingly, 39 isolates (50%) were confirmed as ESBL producers.

This 50% prevalence rate is a striking finding that reflects the growing threat of antimicrobial resistance in India, correlating with previously documented ranges of 39.5% to 58% [11,12,13]. Our results corroborate the work of Ejaz et al., who reported a high proportion of ESBL-mediated UTIs with associated increases in fatality [1]. The clinical ramifications are substantial; as noted by Lakshminarayana et al. and Adabara et al., such infections are linked to extended hospital stays, elevated morbidity and mortality, and considerable financial burden on healthcare systems [2,3].

The ESBL-producing isolates exhibited a predictable resistance profile (Table 3), characterized by complete resistance to all tested penicillins and cephalosporins, including third- and fourth-generation agents. Universal resistance to co-trimoxazole and beta-lactam combination drugs (amoxicillin-clavulanate, cefoperazone-sulbactam) was also observed, severely limiting empirical treatment options. Encouragingly, susceptibility to carbapenems (meropenem, imipenem), piperacillin-tazobactam, and nitrofurantoin was retained, providing clinicians with reliable therapeutic alternatives. As Harwalkar et al. emphasized, beta-lactamase production is the most common mechanism of beta-lactam resistance, underscoring the critical need for accurate detection methods such as the DDST used in this study [4].

A noteworthy finding was the multidrug resistance observed in a subset of non-ESBL *E. coli* isolates (8 of 39), which demonstrated resistance to agents including aztreonam and colistin. This points to the involvement of alternative resistance mechanisms, such as AmpC beta-lactamases or plasmid-mediated colistin resistance (mcr genes), further complicating treatment. These results corroborate the assertion by Ehsan et al. that antibiotic-resistant UTIs are the most prevalent clinical infections, with uropathogenic *E. coli* implicated in 70-90% of cases [10].

Based on our findings, we advocate for routine ESBL screening of all *E. coli* isolates, consistent with prior recommendations [8,12]. The local resistance patterns elucidated in this study serve as a critical resource for clinicians devising empiric treatment regimens. Furthermore, as suggested by Sayida et al., identification of patients at risk for ESBL-producing infections can refine empiric therapy choices [8]. The adoption of rapid diagnostic methods, including the Cica Beta Test described by Yoshimura et al., is strongly recommended; early detection facilitates immediate appropriate treatment, potentially averting complications such as renal scarring that result from delayed therapy [5,10]. Finally, the higher incidence of UTIs in females observed in our cohort, in agreement with Fitriawati et al., underscores the role of anatomical factors in UTI pathogenesis [9].

CONCLUSION

This study aimed to detect ESBL-producing *Escherichia coli* in urinary tract infections and found that ESBL producers and non-producers occur with equal frequency among symptomatic patients.

ESBL production is a key mechanism of antimicrobial resistance, frequently conferring multidrug resistance (MDR) and promoting the spread of MDR organisms. Given the escalating prevalence of ESBL-producing strains, routine antimicrobial susceptibility testing is essential for detection and containment.

We recommend that all isolates with reduced susceptibility to third-generation cephalosporins undergo routine ESBL screening. Additionally, our findings emphasize the necessity for improved rapid diagnostic methods to enable early detection and prompt treatment, thereby mitigating the clinical impact of these infections.

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