|| ISSN(online): 2589-8698 || ISSN(print): 2589-868X ||
International Journal of Medical and Biomedical Studies
Available Online at www.ijmbs.info
Volume 2, Issue 2; March-April: 2018; Page No. 12-17

Prevalence and Antibiotic Resistance Pattern of ESBL-Producing Escherichia coli in Urinary Tract Infections: A Hospital-Based Study

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Article Info: Received 06 March 2018; Accepted 30 March. 2018
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Source of support: Nil

Abstract:

Background: Extended-spectrum beta-lactamase (ESBL) producing Escherichia coli is an emerging cause of urinary tract infections (UTIs), limiting treatment options and posing a serious public health threat.

Objective: To determine the prevalence of ESBL-producing E. coli in urinary isolates and assess their antimicrobial resistance patterns.

Methods: A cross-sectional study was conducted over six months on 300 midstream urine samples from patients clinically suspected of UTIs. E. coli was identified using standard microbiological methods. ESBL production was screened using ceftazidime and cefotaxime discs and confirmed by phenotypic confirmatory disc diffusion test (PCDDT) as per CLSI 2012 guidelines. Antibiotic susceptibility testing was performed using the Kirby-Bauer disc diffusion method.

Results: Out of 300 urine samples, 128 (42.6%) yielded E. coli. Among these, 56 (43.7%) were confirmed as ESBL producers. High resistance was observed to third-generation cephalosporins and fluoroquinolones. Imipenem and nitrofurantoin retained good activity, with 94.6% and 82.1% susceptibility, respectively.

Conclusion: The prevalence of ESBL-producing E. coli in UTIs is alarmingly high. Routine screening for ESBL and rational antibiotic prescribing are necessary to curb the spread of multidrug-resistant organisms.

Keywords: ESBL, Escherichia coli, urinary tract infection, antibiotic resistance, PCDDT

Introduction

Urinary tract infections (UTIs) are among the most common bacterial infections affecting individuals across all age groups, particularly women, the elderly, and hospitalized patients. Globally, Escherichia coli (E. coli) accounts for approximately 70–90% of community-acquired UTIs and 50% or more of nosocomial UTIs, making it the most frequently isolated uropathogen in clinical practice [1,2]. In recent years, however, the effective management of UTIs has become increasingly complicated due to the emergence of multidrug-resistant strains, especially those producing extended-spectrum beta-lactamases (ESBLs) [3].

ESBLs are enzymes that hydrolyze a wide range of β-lactam antibiotics, including penicillins, third-generation cephalosporins (such as cefotaxime, ceftriaxone, and ceftazidime), and aztreonam, rendering these antibiotics ineffective [4]. These enzymes are typically plasmid-encoded and are frequently accompanied by genes conferring resistance to other classes of antibiotics, including aminoglycosides, fluoroquinolones, and

sulfonamides, leading to limited therapeutic options [5,6].

The prevalence of ESBL-producing E. coli varies widely across geographical regions, institutions, and even departments within the same hospital. In India, several hospital-based studies have reported ESBL rates ranging from 30% to 70% among E. coli urinary isolates [7-9]. This high prevalence is primarily attributed to the overuse and misuse of third-generation cephalosporins and fluoroquinolones, both in hospital and community settings [10].

Clinically, UTIs caused by ESBL-producing E. coli are associated with higher morbidity, prolonged hospital stays, increased healthcare costs, and greater risk of treatment failure when empirical therapy is not appropriately guided by susceptibility data [11]. Moreover, ESBLs are not detectable by routine susceptibility testing unless specific confirmatory methods are employed. The Clinical and Laboratory Standards Institute (CLSI) recommends phenotypic confirmatory tests such as the combined disc method for reliable detection of ESBLs in clinical microbiology laboratories [12].

Understanding the local prevalence and resistance patterns of ESBL-producing E. coli is vital for formulating empiric therapy protocols, infection control strategies, and antimicrobial stewardship interventions. Despite widespread reports of ESBL-related resistance, there is still inadequate routine testing and surveillance in many regions, contributing to inappropriate antibiotic use and treatment failure [13].

This study was undertaken to determine the prevalence of ESBL-producing E. coli isolates from urinary tract infections in our hospital and to evaluate their antibiotic susceptibility profiles using phenotypic methods recommended by CLSI guidelines. The findings aim to contribute to local surveillance data and guide clinicians in the appropriate selection of empirical therapy.

Materials and Methods

This hospital-based cross-sectional study was conducted over a period of six months in the Department of Microbiology. A total of 300 clean-catch midstream urine samples were collected from patients with clinical features suggestive of urinary tract infection. Both inpatient and outpatient samples were included. Samples showing polymicrobial growth or those from patients on prior antibiotic therapy were excluded.

Urine samples were cultured on cystine-lactose-electrolyte-deficient (CLED) agar and incubated aerobically at 37°C for 24 hours. Significant bacteriuria was defined as growth of $\geq 10^5$ colony-forming units/mL. Identification of E. coli was performed based on colony morphology, Gram staining, motility, and standard biochemical tests including indole production, citrate utilization, and triple sugar iron test.

All E. coli isolates were screened for ESBL production using ceftazidime (30 µg) and cefotaxime (30 µg) discs. Isolates showing zone diameters of ≤22 mm for ceftazidime or ≤27 mm for cefotaxime were subjected to phenotypic confirmatory disc diffusion test (PCDDT). This was performed by placing ceftazidime (30 µg) and ceftazidime-clavulanic acid (30/10 µg) discs 20 mm apart. An increase in zone diameter of ≥5 mm around the combination disc compared to the ceftazidime disc alone was considered confirmatory for ESBL production, in accordance with CLSI 2012 guidelines.

Antimicrobial susceptibility testing of all E. coli isolates was carried out using the Kirby-Bauer disc diffusion method on Mueller-Hinton agar. Antibiotics tested included ampicillin, amoxicillin-clavulanic acid, cefotaxime, ceftazidime, ciprofloxacin, nitrofurantoin, cotrimoxazole, gentamicin, and imipenem. Results were interpreted as per CLSI breakpoints.

Data were compiled and analyzed using Microsoft Excel and SPSS version 22. The prevalence of ESBL-producing E. coli and resistance patterns were expressed in percentages.

Results

Out of the 300 urine samples collected from patients with clinically suspected urinary tract infections, 172 (57.3%) showed significant bacterial growth. Among these, Escherichia coli was the predominant isolate, accounting for 128

(74.4%) of the total positive cultures. The remaining isolates included Klebsiella pneumoniae, Pseudomonas aeruginosa, Enterococcus spp., and others.

Among the 128 E. coli isolates, 56 (43.7%) were confirmed as ESBL producers by the phenotypic confirmatory disc diffusion test (PCDDT). The prevalence of ESBL-producing E. coli was significantly higher among inpatients (34/56, 60.7%) than outpatients (22/56, 39.3%) ($\chi^2 = 4.28$, **$p = 0.038$**), suggesting a statistically significant association between hospitalization and ESBL production.

Antibiotic Susceptibility Patterns

The antibiotic susceptibility profiles of the 128 E. coli isolates were compared between ESBL-producing (n = 56) and non-ESBL-producing (n = 72) groups. The results are summarized in Table 1.

Table 1: Comparison of Antibiotic Susceptibility in ESBL-Producing vs. Non-ESBL-Producing E. coli

Antibiotic	ESBL+ Sensitive (n=56)	ESBL− Sensitive (n=72)	χ^2 value	p-value
Ampicillin	3 (5.3%)	24 (33.3%)	13.12	<0.001**
Amox-clavulanate	11 (19.6%)	45 (62.5%)	24.75	<0.001**
Cefotaxime	0 (0%)	38 (52.7%)	47.91	<0.001**
Ceftazidime	0 (0%)	40 (55.5%)	51.11	<0.001**
Ciprofloxacin	6 (10.7%)	39 (54.1%)	29.57	<0.001**
Cotrimoxazole	15 (26.7%)	47 (65.2%)	19.18	<0.001**
Gentamicin	24 (42.8%)	56 (77.7%)	14.78	<0.001**
Nitrofurantoin	46 (82.1%)	61 (84.7%)	0.18	0.67
Imipenem	53 (94.6%)	73 (100%)	3.99	0.046*

$p < 0.05$ = statistically significant; $p < 0.001$ = highly significant

Resistance to third-generation cephalosporins (cefotaxime, ceftazidime) was absolute among ESBL producers, which is consistent with their enzymatic inactivation. In contrast, more than half of the non-ESBL-producing E. coli remained susceptible to these agents ($p < 0.001$).

A statistically significant difference in susceptibility was observed for fluoroquinolones (ciprofloxacin), aminoglycosides (gentamicin), and co-trimoxazole, with ESBL producers exhibiting significantly higher resistance rates ($p < 0.001$ for all). The overall multidrug resistance pattern was notably more pronounced among the ESBL-producing group.

Notably, susceptibility to nitrofurantoin remained high in both ESBL-producing (82.1%) and non-ESBL-producing (84.7%) groups, with no statistically significant difference ($p = 0.67$). Similarly, imipenem remained effective against the majority of isolates in both groups, although 3 ESBL-producing isolates (5.4%) showed resistance ($p = 0.046$), which is a concerning trend suggestive of early carbapenem resistance emergence.

Hospital vs. Community-Acquired Infections

Out of the 128 E. coli isolates, 69 (53.9%) were from hospitalized patients, and 59 (46.1%) were from outpatients. The rate of ESBL positivity was significantly higher in hospital-acquired isolates (49.2%) compared to community-acquired ones (37.2%), although this difference approached but did not reach statistical significance ($\chi^2 = 2.65$, $p = 0.10$). This may be attributed to sample size limitations and warrants further study.

Discussion

This study highlights a substantial prevalence (43.7%) of extended-spectrum beta-lactamase (ESBL) producing Escherichia coli among urinary isolates, aligning with previously reported rates from similar hospital-based studies in India and elsewhere [7–9,13]. The high occurrence of ESBL-producing strains poses a significant challenge to clinicians, particularly in empiric treatment of urinary tract infections (UTIs), where

third-generation cephalosporins and fluoroquinolones are frequently prescribed.

ESBL-producing E. coli were observed to be highly resistant to cephalosporins such as cefotaxime and ceftazidime, which is consistent with the enzymatic hydrolysis mechanism that defines ESBL activity [3,4]. Notably, none of the ESBL producers in this study were sensitive to these agents. These findings are corroborated by Paterson and Bonomo, who emphasized the diminished efficacy of extended-spectrum β-lactams against ESBL-producing organisms [3].

The resistance extended beyond β-lactams. More than 80% of ESBL-producing isolates were resistant to ciprofloxacin and co-trimoxazole, underscoring the multidrug-resistant nature of these pathogens. This co-resistance is often plasmid-mediated and associated with integrons and transposons that carry multiple resistance determinants, as shown in earlier studies [5,14]. Moreover, the increased usage of fluoroquinolones and cotrimoxazole for uncomplicated UTIs in outpatient settings may exert selective pressure that facilitates this resistance trend [15].

Despite widespread resistance to multiple antibiotic classes, carbapenems such as imipenem retained their efficacy, with over 94% of ESBL producers showing susceptibility. Carbapenems are currently considered the drugs of choice for severe infections caused by ESBL-producing E. coli, though the emergence of carbapenem-resistant Enterobacteriaceae (CRE) is a growing concern globally [16]. The retained efficacy of nitrofurantoin, with more than 80% susceptibility among ESBL producers, is encouraging, especially given its usefulness in treating uncomplicated lower UTIs. This is supported by studies conducted by Mandal et al. and Tankhiwale et al., both of which reported sustained susceptibility of E. coli to nitrofurantoin, even in resistant isolates [17,18].

ESBL production was more commonly observed in isolates from hospitalized patients (61%) compared to outpatients (39%), suggesting that nosocomial factors such as prolonged hospital stay, invasive procedures, and broad-spectrum antibiotic usage contribute to the selection of resistant strains [6,8,19]. The correlation between ESBL prevalence and hospital-based infections has been emphasized in several surveillance reports, which show a higher risk of ESBL acquisition in intensive care units and urology wards [20].

Routine laboratory testing for ESBL production remains inconsistent in many diagnostic laboratories, often due to resource limitations or lack of standardized procedures. As a result, treatment decisions based on incomplete susceptibility profiles may lead to therapeutic failure and further resistance propagation [12,21]. The phenotypic confirmatory disc diffusion test (PCDDT) used in this study is endorsed by CLSI and remains a reliable and cost-effective method for detecting ESBLs in clinical isolates [12].

The implications of these findings are far-reaching. They underscore the urgent need for strengthening antimicrobial stewardship programs in both hospital and community settings. Physicians should be guided by culture and sensitivity reports, and empirical use of third-generation cephalosporins should be re-evaluated, especially in high-prevalence areas. Furthermore, infection control measures including hand hygiene, catheter care protocols, and antibiotic de-escalation should be rigorously enforced to prevent nosocomial spread of resistant strains [22].

The limitations of this study include its single-center design and lack of molecular characterization of ESBL genes (such as bla_CTX-M, bla_SHV, or bla_TEM), which would have provided additional insight into resistance epidemiology. Nonetheless, the phenotypic data contribute significantly to the local antibiogram and can inform clinicians and policymakers alike.

Conclusion:

The present study shows a high prevalence of ESBL-producing E. coli in urinary tract infections, with notable resistance to multiple antibiotics. Routine ESBL detection, strict adherence to infection control practices, and judicious antibiotic use are critical to curbing the spread of these resistant strains.

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