Observational Study

Medicine

OPEN

Study on extended-spectrum beta-lactamases genes and drug resistance in patients with urinary tract infection of enterohemorrhagic *Escherichia coli* after bladder cancer surgery

Yanzhi Lu, MM[a], Jinman Li, MM[a], Xuewen Qin, MM[a,*]

Abstract

To explore of the detection of enterohemorrhagic *Escherichia coli* with extended-spectrum beta-lactamase (ESBLs) in patients with urinary tract infections (UTIs) after bladder cancer surgery, and analysis of their genotypic distribution and drug resistance. From February 2022 to February 2024, patients who underwent bladder cancer surgery at our hospital were collected. Among them, those who developed UTIs with enterohemorrhagic *E coli* postoperatively had their urine specimens isolated and cultured, resulting in 87 strains of enterohemorrhagic *E coli*. Cultures were conducted on the obtained enterohemorrhagic *E coli* samples, ESBLs production was screened, and drug sensitivity tests were performed to investigate the resistance rate and antibacterial effects. Additionally, genotypic testing was conducted. This study successfully isolated 87 strains of *E coli*, among which 49 strains (56.32%) were found to produce ESBLs after screening. The resistance rates of these ESBL-producing *E coli* to cefotaxime and ampicillin were relatively high (93.88% and 97.96%, respectively), while the resistance rate to imipenem was the lowest (2.04%). Genotypic testing revealed that among the 49 strains of ESBL-producing *E coli*, the detection rate of blaCTX-M-14 was the highest at 53.06%, followed by bla-TEM at 30.61%. The detection rates of bla-SHV (4.08%), bla-OXA (2.04%), blaCTX-M-3 (2.04%), blaCTX-M-15 (2.04%), as well as combinations of several genotypes (blaCTX-M-3 + bla-TEM, blaCTX-M-14 + bla-TEM, blaCTX-M-15 + bla-TEM, all with a detection rate of 2.04%), were relatively low. Strains carrying the bla-TEM genotype exhibited 100% resistance rates to ampicillin and tetracycline. Strains carrying the blaCTX-M-14 genotype showed a 100% resistance rate to ampicillin and a 96.15% resistance rate to cefotaxime. Bladder cancer patients with postoperative complications of *E coli* urinary tract infection have a detection rate of 56.32% for ESBL-producing *E coli*. The detected ESBL-producing strains show a high resistance rate to ampicillin and cefotaxime, with the lowest resistance rate observed against imipenem. Genotypic analysis reveals that blaCTX-M-14 and bla-TEM are the main ESBL genes, with blaCTX-M-14 having the highest detection rate.

Keywords: bladder cancer, E coli, extended-spectrum β-lactamases, resistance, urinary tract infection

1. Introduction

In recent years, the emergence of extended-spectrum β-lactamases (ESBLs) has led to increasingly severe drug resistance, posing significant challenges to clinical treatment.[1–3] ESBLs can hydrolyze a variety of β-lactam antibiotics, rendering *Escherichia coli*, which is normally sensitive to these drugs, resistant.[4] Despite some progress in the study of ESBL-producing *E coli*, research on patients with postoperative urinary tract infections (UTIs) after bladder cancer surgery remains insufficient.[5,6]

Bladder cancer has a significant incidence and is associated with substantial harm, with surgery being the primary treatment method.[7] While surgical treatment for bladder cancer is crucial, it can result in damage to the bladder wall and urethral mucosa, among other tissues. Prolonged postoperative catheterization provides conditions conducive to the growth of pathogens, increasing the risk of UTIs.[8,9] *E coli* has become the predominant pathogen causing such infections. In recent years, the increasing resistance of *E coli* to multiple drugs has become more prominent, especially with the emergence of ESBLs, exacerbating the problem of drug resistance in *E coli*.[10,11] ESBLs exhibit a wide range of subtypes, including CTX-M, TEM, SHV, and OXA types, among others.[12,13] Previous studies have touched upon the antimicrobial resistance and genotypes of *E coli*, but they have predominantly focused on general urinary tract infection patients or the general population. Research specifically targeting the unique patient population of post-bladder cancer surgery remains

The authors have no funding and conflicts of interest to disclose.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

[a] Department of Clinical Laboratory, Chifeng City Cancer Hospital, Chifeng, Neimenggu, China.

** Correspondence: Xuewen Qin, Department of Clinical Laboratory, Chifeng City Cancer Hospital, Chifeng 024000, Neimenggu, China (e-mail: luyanzhi7001@163.com).*

How to cite this article: Lu Y, Li J, Qin X. Study on extended-spectrum beta-lactamases genes and drug resistance in patients with urinary tract infection of enterohemorrhagic Escherichia coli after bladder cancer surgery. Medicine 2025;104:17(e42098).

Received: 22 May 2024 / Received in final form: 20 January 2025 / Accepted: 30 January 2025

http://dx.doi.org/10.1097/MD.0000000000042098

limited.[14] Furthermore, there is a notable lack of in-depth studies exploring the relationship between the distribution of ESBL-producing *E coli* genotypes and antimicrobial resistance. In light of this, this study aims to investigate the specific circumstances of UTIs caused by ESBL-producing *E coli* in patients following bladder cancer surgery. It seeks to reveal the detection rate, genotype distribution, and antimicrobial resistance of ESBL-producing *E coli* among these patients. It is hoped that through this series of studies, robust support can be provided for clinically rational drug use and the reduction of resistant bacterial strains.

2. Materials and methods

2.1. General data

This study was approved by the Ethics Committee of Chifeng City Cancer Hospital. From February 2022 to February 2024, a total of 87 strains of *E coli* were isolated from urine specimens of patients who underwent bladder cancer surgery at our hospital and subsequently developed UTIs caused by *E coli*. During the study period, a total of 211 patients underwent bladder cancer surgery at our hospital, including 145 patients (68.72%) who received radical cystectomy and 66 patients (31.28%) who underwent transurethral resection of bladder tumor (TURBT). Among them, 120 patients were diagnosed with postoperative urinary tract infection, and urine samples from these patients were collected. Based on inclusion and exclusion criteria, 87 patients with *E coli*-positive urine cultures were ultimately included in the study. Among the 120 patients diagnosed with postoperative UTIs, 87 cases (72.5%) were caused by *E coli*, while 33 cases (27.5%) were due to other pathogens, including Klebsiella spp. (12 cases, 10.0%), Enterococcus spp. (9 cases, 7.5%), Pseudomonas aeruginosa (7 cases, 5.8%), and other bacteria (5 cases, 4.2%).

2.1.1. Inclusion criteria. All patients were confirmed as bladder cancer by pathological examination and received surgical treatment in our hospital. patients were complicated with urinary tract infection after operation. After urine culture and *E coli* culture, the results were positive, and urine bacteria ≥ 105/mL; white blood cell count in 10/field.

Urine analysis and culture were performed based on the following clinical indications: the presence of urinary tract infection-related symptoms such as fever, dysuria, or cloudy urine; laboratory evidence such as elevated white blood cell count suggesting infection; abnormal findings in imaging studies such as urinary retention or obstruction; and routine postoperative monitoring for patients with a high risk of infection, such as those with long-term indwelling catheters or urinary diversion procedures.

2.1.2. Exclusion criteria. There was contamination in the samples, and there were 3 or more pathogens in the urine culture results (15 patients). Combined with other infectious diseases (10 patients). There were blood and immune dysfunction (8 patients). A total of 33 patients were excluded based on these criteria.

2.2. Methods

Sample collection: Urine samples were collected for analysis and culture from patients who met the clinical indications for suspected UTIs, which included the presence of typical symptoms, laboratory abnormalities, or findings on imaging studies. Additionally, for patients undergoing high-risk surgical procedures such as radical cystectomy or ileal conduit, urine culture was performed as part of routine infection surveillance. Urine samples were collected from patients as follows: For patients with an ileal conduit or those catheterized after radical cystectomy, urine samples were obtained using sterile techniques directly from the catheter or conduit to ensure accuracy and avoid contamination. For patients who underwent transurethral resection of bladder tumor (TURBT) or those not requiring long-term catheterization, midstream urine samples were collected following standard procedures. A precise volume of 10μL of the urine sample was inoculated onto blood agar plates and incubated at 35°C in a 5% CO_2 environment for 20 hours. The VITEK 2 Compact fully automated microbial identification and susceptibility analysis system was used to identify bacteria in the urine specimens to confirm the presence of *E coli* infection. Ensure that each patient provided only non-repetitive specimens.

Screening for ESBL-producing strains: Firstly, individual *E coli* colonies are selected from each sample and inoculated into MH broth, followed by incubation under identical conditions for 8 hours. Subsequently, the bacterial suspensions of the strains are diluted with sterile physiological saline to a turbidity of 0.5 McFarland units and evenly spread on MH agar plates. Then, diffusion tests are performed using antimicrobial sensitivity test paper discs (containing amoxicillin/clavulanic acid and cefotaxime) within specific temperature and time parameters (35°C, 16–18 hours). Based on the size of inhibition zones (≤27 mm), further confirmatory tests are conducted if necessary.

Extended-spectrum beta-lactamases confirmation test: In the testing of inhibition zone diameters, 2 schemes are used: the combination of cefotetan and clavulanic acid, and the combination of ceftriaxone and clavulanic acid. Carefully observe that when clavulanic acid is added, if there is a significant difference in the inhibition zone diameter compared to when clavulanic acid is not added, specifically if the difference in diameter reaches or exceeds 5 mm for any 1 or both pairs of discs, then it can be concluded that the strain is ESBL-producing.

Drug sensitivity test: Analysis is conducted using the VITEK 2 Compact fully automated microbial identification and drug sensitivity analysis system. Analysis is performed using drug sensitivity discs, with materials purchased from the British company Oxoid. The drug sensitivity discs cover a variety of antimicrobial drugs, including penicillins, cephalosporins, monobactams, aminoglycosides, quinolones, and carbapenems, allowing for a comprehensive assessment of the sample's sensitivity to different drugs. To ensure the accuracy of the test, standard quality control strains, such as *E coli* ATCC 25922, are used. Based on the results of the drug sensitivity test, particular attention is paid to the sensitivity and resistance characteristics of *E coli* to various antibacterial drugs. The resistance rate is calculated as (number of resistant strains/total number of strains) × 100%.

ESBL-producing *E coli* resistant gene detection: Firstly, culture and isolate ESBL-producing *E coli* strains to ensure their viability and quantity. Subsequently, use a bacterial nucleic acid extraction kit to extract total DNA. Take 2 μL of the DNA extract as a template and use multiplex PCR technology to detect genotypes such as bla-TEM, blaCTX-M, bla-SHV, bla-OXA, blaCTX-M-14, blaCTX-M-15, and blaCTX-M-3. Primers are synthesized by Shanghai Bioengineering Co., Ltd. The polymerase chain reaction (PCR) is utilized to amplify the target genes. A 25 μL PCR reaction system is constructed. The amplification process begins with a pre-denaturation at 95°C for 3 minutes to prepare for subsequent DNA replication. A 32-cycle amplification program is designed. In the denaturation stage, the temperature is rapidly raised to 95°C for 30 seconds to denature the double-stranded DNA into single strands. The annealing stage lowers the temperature to 56°C for 30 seconds to allow the primers to bind to the complementary sequences of the template DNA. During the extension stage, the temperature is maintained at 70°C for 40 seconds to allow DNA polymerase to synthesize new DNA strands guided by primers. After the entire cycle, another extension is performed at 72°C for 10 minutes to ensure complete replication of all DNA fragments.

Following PCR, the products are separated by agarose gel electrophoresis with a concentration of 1 g/dL and stained with ethidium bromide to visualize DNA fragments of different sizes under UV light. The amplified products are recovered using a DNA gel recovery kit and then sequenced. After sequencing, the obtained sequences are aligned against the GenBank database to determine the genetic typing of the strain based on the alignment results.

2.3. Management of patients

Management strategies for patients were tailored based on the pathogen type (ESBL or non-ESBL-producing *E coli*) and the severity of UTIs. The specific approaches were as follows:

2.3.1. Management of patients with ESBL-producing strains. *Antibiotic selection*: patients infected with ESBL-producing *E coli* were treated with carbapenems (e.g., imipenem or meropenem) as first-line antibiotics due to their effectiveness against ESBL strains. For patients with mild infections or contraindications to carbapenem use, piperacillin/tazobactam or ceftazidime/avibactam was used as an alternative.

Treatment duration: The duration of treatment varied between 7 and 14 days, depending on the clinical severity and response to therapy.

Monitoring: Inflammatory markers such as white blood cell count and C-reactive protein levels were regularly monitored to assess treatment efficacy and guide adjustments.

2.3.2. Management of patients with non-ESBL-producing strains. *Antibiotic selection*: narrow-spectrum antibiotics, such as cefazolin or ampicillin, were primarily used for non-ESBL infections. Aminoglycosides (e.g., amikacin) were considered for cases sensitive to this class of antibiotics.

Treatment duration: The treatment duration ranged from 5 to 7 days for uncomplicated infections and up to 10 days for complicated cases.

Monitoring: Similar to ESBL strains, inflammatory markers and clinical symptoms were closely observed to ensure infection resolution.

2.3.3. General management for all patients.. Risk mitigation: For patients with ileal conduits or long-term indwelling catheters, additional measures were taken to prevent catheter-associated infections. These included sterile handling practices and regular catheter replacement protocols.

Multidisciplinary collaboration: Complex cases involving recurrent infections or treatment resistance were managed collaboratively by a team of urologists, infectious disease specialists, and clinical laboratory staff to optimize treatment outcomes and minimize resistance.

2.4. Antibiotic prophylaxis protocol

A standardized protocol for antibiotic prophylaxis was implemented for all patients undergoing bladder cancer surgery. The details are as follows:

2.4.1. Preoperative antibiotics. All patients received prophylactic antibiotics 30 to 60 minutes prior to the incision to ensure optimal blood levels during surgery.

First-line choice: Cefazolin (2g intravenously).

Alternative choice: Aztreonam or Vancomycin for patients allergic to cephalosporins.

2.4.2. Postoperative antibiotics. Low-risk patients (e.g., those undergoing transurethral resection of bladder tumor [TURBT]): Postoperative cefazolin was continued for 24 hours.

High-risk patients (e.g., those undergoing radical cystectomy or ileal conduit): Postoperative antibiotics were extended to 48 to 72 hours, and adjustments were made based on clinical indicators.

2.5. Observed indexes

To cultivate the collected *E coli* samples, screen for ESBL-producing strains, and conduct drug sensitivity experiments to explore their resistance rates and inhibition conditions, and perform genotype detection.

2.6. Statistical processing

The study used SPSS 26.0 software (Chicago) for statistical analysis. The count data in the study were expressed as the number of cases/percentage, and the measurement data were expressed as ($\bar{\chi} \pm s$).

3. Results

3.1. Bacterial culture results and ESBL screening results

In this study, a total of 87 strains of *E coli* were extracted, and after ESBL screening, 49 strains (56.32%) were found to produce ESBLs.

3.2. Drug resistance of the 49 ESBL-producing E coli strains

The resistance rates of the 49 ESBL-producing *E coli* strains to ampicillin and cefotaxime are relatively high, at 97.96% and 93.88% respectively, while the resistance rate to imipenem is the lowest, at only 2.04%. Following imipenem are furantoin (6.12%) and ciprofloxacin (8.16%). See Table 1.

3.3. Distribution of ESBL-producing E coli genotypes in 49 strains

In 49 strains of ESBL-producing *E coli*, the detection rate of blaCTX-M-14 was the highest at 53.06%, followed by bla-TEM at 30.61%. Detection rates of bla-SHV (4.08%), bla-OXA (2.04%), blaCTX-M-3 (2.04%), blaCTX-M-15 (2.04%), and combinations of several genotypes (blaCTX-M-3 + bla-TEM, blaCTX-M-14 + bla-TEM, blaCTX-M-15 + bla-TEM, all with a detection rate of 2.04%) were lower. See Table 2.

Table 1

Drug resistance of the 49 ESBL-producing *E coli* strains.

Antibiotic	Number of Resistant Strains	Resistance Rate (%)	Average Inhibition Zone Diameter (mm)
Amoxicillin	48	97.96	–
Cefazolin	46	93.88	–
Ceftriaxone	30	61.22	5.11 ± 0.98
Cefotetan	29	59.18	6.65 ± 1.59
Cefoperazone	23	46.94	11.25 ± 2.11
Piperacillin	5	10.20	18.45 ± 2.65
Suprax	4	8.16	18.11 ± 1.22
Levofloxacin	22	44.90	8.98 ± 1.35
Ciprofloxacin	31	63.27	16.56 ± 2.56
Levofloxacin	28	57.14	8.25 ± 2.02
Amikacin	33	67.35	4.98 ± 1.03
Amikacin	21	42.86	9.12 ± 3.03
Nitrofurantoin	3	6.12	17.46 ± 2.45
Imipenem	1	2.04	22.12 ± 3.11
Sulfamethoxazole	34	69.39	5.02 ± 0.98
Minocycline	29	59.18	9.12 ± 1.06

ESBL = extended-spectrum beta-lactamase.

3.4. Drug resistance rate of bla-TEM genotype

The strain carrying the bla-TEM genotype exhibits a resistance rate of 100% to ampicillin and minocycline, and a resistance rate of 93.33% to cefotaxime. It shows a high level of resistance. See Table 3.

3.5. Drug resistance rate of blaCTX-M-14 genotype

The strain carrying the blaCTX-M-14 genotype exhibits a resistance rate of 100% to ampicillin and 96.15% to ceftriaxone, demonstrating a high level of resistance. See Table 4.

3.6. Non-ESBL E coli antibiotic resistance distribution

Among the 87 isolated *E coli* strains, 38 strains (43.68%) were identified as non-ESBL-producing. The antibiotic resistance rates for these non-ESBL strains were as follows: 45.37% to ampicillin, 28.57% to cefotaxime, 10.53% to ciprofloxacin, and 2.63% to nitrofurantoin. Resistance to imipenem was not observed. A comparative analysis indicates that non-ESBL strains exhibit significantly lower resistance rates compared to ESBL-producing strains, particularly against β-lactam antibiotics (see Table 5).

4. Discussion

Escherichia coli is a key pathogen responsible for various clinical infectious diseases, with UTIs being particularly prominent.[15] The high recurrence rate and increasing antibiotic resistance of this bacterium have imposed a heavy burden on public health.[16] Patients with bladder cancer often undergo invasive procedures such as indwelling catheterization during surgery, which increases the risk of UTIs.[17,18] However, compared to research in other fields, there is a relative scarcity of reports on postoperative UTIs caused by ESBL-producing *E coli* in bladder cancer patients in China. Therefore, there is an urgent need in clinical practice to strengthen the monitoring of such bacteria. Based on the aforementioned research background, this study aims to comprehensively analyze the detection status of ESBL-positive *E coli* in postoperative bladder cancer patients with concurrent UTIs, and to analyze their genotype distribution and antibiotic resistance.

This research revealed that up to 56.32% of strains have been confirmed to possess the ability to produce ESBLs. This result is attributed to the interplay of various factors. Increased invasive procedures in patients elevate the risk of infection, while excessive antibiotic usage provides conditions for resistant bacteria to thrive. Moreover, the transmission of resistant bacteria within hospital environments exacerbates this trend.[19,20] Previous studies have confirmed that patients who undergo bladder cancer surgery, particularly radical cystectomy, experience a higher incidence of UTIs compared to the general population. This is likely due to the combination of surgical factors, catheterization, and the immunosuppressive effects of the procedure. However, this study did not include a comparison group of nonsurgical patients for urinary tract infection, as its primary focus was on postoperative infections caused by ESBL-producing *E coli*. The absence of a control group of nonsurgical patients is a limitation of the study. Further analysis revealed that the resistance rates of ESBL-producing *E coli* to ampicillin and cefotaxime were as high as 97.96% and 93.88%, respectively. These 2 drugs, representing β-lactam antibiotics, were expected to exert potent antibacterial effects. However, their inhibitory effects on ESBL-producing *E coli* were inadequate. The analysis indicated that ESBLs can precisely hydrolyze the β-lactam ring, rendering the drugs inactive against bacterial growth, thereby resulting in the emergence of high resistance rates.[21–23] Imipenem exhibited strong inhibitory effects on ESBL-producing *E coli*, with a resistance rate of only 2.04%. Imipenem's unique antimicrobial mechanism allows for direct destruction of bacterial cell walls, resulting in rapid bactericidal effects.[24] Therefore, imipenem can be considered as the preferred drug. Additionally, studies have found relatively low resistance rates for meropenem and sulbactam, at 6.12% and 8.16% respectively. These 2 drugs have their own characteristics and can precisely target specific sites of ESBL-producing *E coli* for effective treatment.[25,26] In certain specific circumstances, they may serve as alternative treatment options. In summary, ESBL-producing *E coli* exhibit relatively low resistance rates to carbapenem antibiotics, especially the representative drug imipenem. This suggests that when prescribing medications clinically, the resistant characteristics of bacteria should be fully considered to develop personalized treatment plans.

The results of this study demonstrated that among ESBL-producing *E coli*, the detection rate of the blaCTX-M-14 gene is the highest, reaching 53.06%, followed by the bla-TEM gene with a detection rate of 30.61%. In comparison, the detection rates of other genotypes appear to be lower. This data reveals the predominant distribution of specific genotypes in resistant *E coli*. The high detection rate of the blaCTX-M-14 gene suggests that this genotype may possess strong dissemination capability, rapidly spreading within bacterial populations through mechanisms such as horizontal gene transfer.[27,28] In addition, the blaCTX-M-14 genotype may confer bacteria with resistance to multiple antibiotics, giving them a survival advantage in clinical settings. Moreover, the unique physiological status and environmental factors of postoperative bladder cancer patients, such as antibiotic treatment received and hospital environment, may also promote the dissemination and spread of the blaCTX-M-14

Table 2

Distribution of ESBL-producing *E coli* genotypes in 49 strains.

Genotype	Number of strains	Detection rate (%)
bla-TEM	15	30.61
bla-SHV	2	4.08
bla-OXA	1	2.04
blaCTX-M-14	26	53.06
blaCTX-M-3	1	2.04
blaCTX-M-15	1	2.04
blaCTX-M-3 + bla-TEM	1	2.04
blaCTX-M-14 + bla-TEM	1	2.04
blaCTX-M-15 + bla-TEM	1	2.04

ESBL = extended-spectrum beta-lactamase.

Table 3

Drug resistance rate of bla-TEM genotype (n = 15).

Antibiotic	Number of resistant strains	Resistance rate (%)
Amoxicillin	15	100.00
Cefazolin	14	93.33
Ceftriaxone	9	60.00
Cefotetan	10	66.67
Cefoperazone	7	46.67
Piperacillin	2	13.33
Suprax	1	6.67
Levofloxacin	6	40.00
Ciprofloxacin	8	53.33
Levofloxacin	7	46.67
Amikacin	11	73.33
Amikacin	8	53.33
Nitrofurantoin	1	6.67
Imipenem	1	6.67
Sulfamethoxazole	12	80.00
Minocycline	15	100.00

Table 4

Drug resistance rate of blaCTX-M-14 genotype (n = 26).

Antibiotic	Number of resistant strains	Resistance rate (%)
Amoxicillin	26	100.00
Cefazolin	25	96.15
Ceftriaxone	15	57.69
Cefotetan	14	53.85
Cefoperazone	12	46.15
Piperacillin	3	11.54
Suprax	3	11.54
Levofloxacin	14	53.85
Ciprofloxacin	16	61.54
Levofloxacin	18	69.23
Amikacin	20	76.92
Amikacin	11	42.31
Nitrofurantoin	2	7.69
Sulfamethoxazole	19	73.08
Minocycline	13	50.00

genotype. Although the detection rate of the bla-TEM gene is slightly lower than that of blaCTX-M-14, it still occupies an important position in ESBL-producing *E coli*.[29–31] The widespread distribution and strong resistance of the bla-TEM genotype may be related to its stable genetic characteristics and extensive transmission capability. As for other genotypes such as bla-SHV, bla-OXA, blaCTX-M-3, and blaCTX-M-15, their detection rates are relatively low, possibly influenced by various factors such as their frequency of occurrence in specific environments or conditions and their competitive relationship with other genotypes. Additionally, this study also identified several combinations of genotypes, such as blaCTX-M-3 with bla-TEM, blaCTX-M-14 with bla-TEM, and blaCTX-M-15 with bla-TEM. The emergence of these combinations further increases the bacteria's resistance to antibiotics, posing greater challenges to clinical treatment.

This study further investigated the relationship between different genotypes and antibiotic resistance. The results showed that strains carrying the bla-TEM genotype exhibited a 100% resistance rate to ampicillin and minocycline. Meanwhile, strains carrying the blaCTX-M-14 genotype showed a 100% resistance rate to ampicillin, and a resistance rate of 96.15% to cefazolin. Specifically analyzing the relationship between the bla-TEM genotype and resistance: the bla-TEM genotype encodes a type of β-lactamase enzyme, which possesses remarkable hydrolytic activity capable of precisely disrupting the structure of β-lactam antibiotics, thereby rendering them inactive against bacteria.[32] Benzylpenicillin, as a typical β-lactam antibiotic, cannot effectively withstand the attack of β-lactamase encoded by the bla-TEM genotype, leading to the development of resistance in bacterial strains.[33] Additionally, although the antibacterial mechanism of minocycline differs from that of β-lactam antibiotics, bacterial strains may acquire resistance to different classes of antibiotics through various mechanisms during evolution. Therefore, strains carrying the bla-TEM genotype also possess resistance mechanisms against minocycline.[34] Similar to bla-TEM, the β-lactamase encoded by the blaCTX-M-14 genotype can also hydrolyze benzylpenicillin, rendering it ineffective as an antibacterial agent. Furthermore, cefazolin, as a cephalosporin antibiotic, shares structural similarities with β-lactam antibiotics, making it susceptible to attack by the β-lactamase encoded by the blaCTX-M-14 genotype.[35] The high resistance rate of 96.15% of strains carrying the blaCTX-M-14 genotype to cefazolin fully illustrates this point. Therefore, for the treatment of resistant strains, it is essential to carefully select appropriate antibiotics based on precise results from susceptibility testing.

Table 5

Antibiotic resistance distribution among non-ESBL-producing *E coli* strains.

Antibiotic	Number of resistant non-ESBLs strains	Resistance rate (%)
Ampicillin	17	45.37
Cefotaxime	11	28.57
Ciprofloxacin	4	10.53
Nitrofurantoin	1	2.63
Imipenem	0	0

ESBL = extended-spectrum beta-lactamase.

This study not only focuses on the distribution of genotypes but also extensively analyzes the association between different genotypes and resistance, with particular emphasis on the resistance profiles of bla-TEM and blaCTX-M-14 genotypes to various antibiotics. It provides richer data for the study of resistance mechanisms. However, the study is limited by a relatively small sample size and the incomplete consideration of other factors influencing resistance. Future research will expand the sample size and comprehensively consider multiple factors to more comprehensively reveal resistance mechanisms and optimize control strategies.

5. Limitation

Although this study provides valuable insights into the antibiotic resistance and genotype distribution of ESBL-producing *E coli* in postoperative bladder cancer patients, a major limitation is the lack of a control group of nonsurgical patients. This study focused solely on bladder cancer patients who developed postoperative UTIs, without comparing their infection rates to those in the general population or patients without bladder surgery. Future research should include such comparisons to better understand the specific risks posed by bladder cancer surgery in relation to UTIs. Additionally, this study was limited by its relatively small sample size, and we aim to expand the sample size in future studies to further validate these findings and explore additional factors influencing resistance mechanisms.

6. Conclusion

In summary, among patients with postoperative UTIs caused by *E coli* following bladder cancer surgery, the detection rate of ESBL-positive *E coli* is as high as 56.32%. Further analysis reveals that these ESBL-producing strains exhibit high resistance rates to antibiotics such as benzylpenicillin and cefazolin, with the lowest resistance rate observed against imipenem. Genotypic analysis indicates that blaCTX-M-14 and bla-TEM are the main ESBL genes, with blaCTX-M-14 showing the highest detection rate.

Author contributions

Conceptualization: Yanzhi Lu, Xuewen Qin.
Data curation: Yanzhi Lu, Jinman Li, Xuewen Qin.
Formal analysis: Yanzhi Lu, Xuewen Qin.
Validation: Xuewen Qin.
Visualization: Yanzhi Lu, Xuewen Qin.
Writing – original draft: Yanzhi Lu, Jinman Li, Xuewen Qin.
Writing – review & editing: Yanzhi Lu, Jinman Li, Xuewen Qin.

References

[1] Lu X, Jiang H, Wang D, et al. Early warning models to predict the 90-day urinary tract infection risk after radical cystectomy and urinary diversion for patients with bladder cancer. Front Surg. 2022;8:e782029.

[2] Gayarre Abril P, Subirá Ríos J, Muñiz Suárez L, et al. Urinary tract infection as the main cause of admission in cystectomized patients. Actas Urol Esp (Engl Ed). 2021;45:247–56.
[3] Kao CY, Zhang YZ, Yang DC, et al. Characterization of host and *escherichia coli* strains causing recurrent urinary tract infections based on molecular typing. BMC Microbiol. 2023;23:90–2.
[4] Zhou Y, Zhou Z, Zheng L, et al. Urinary tract infections caused by uropathogenic *Escherichia coli*: mechanisms of infection and treatment options. Int J Mol Sci. 2023;24:e10537.
[5] Rando E, Giovannenze F, Murri R, Sacco E. A review of recent advances in the treatment of adults with complicated urinary tract infection. Expert Rev Clin Pharmacol. 2022;15:1053–66.
[6] Derakhshan S, Ahmadi S, Ahmadi E, Nasseri S, Aghaei A. Characterization of *Escherichia coli* isolated from urinary tract infection and association between virulence expression and antimicrobial susceptibility. BMC Microbiol. 2022;22:89–90.
[7] Quan J, Dai H, Liao W, et al. Etiology and prevalence of ESBLs in adult community-onset urinary tract infections in East China: a prospective multicenter study. J Infect. 2021;83:175–81.
[8] Abalkhail A, AlYami AS, Alrashedi SF, et al. The prevalence of multidrug-resistant *Escherichia coli* producing ESBL among male and female patients with urinary tract infections in Riyadh Region, Saudi Arabia. Healthcare (Basel). 2022;10:1778–80.
[9] Vachvanichsanong P, McNeil EB, Dissaneewate P. Extended-spectrum beta-lactamase *Escherichia coli* and Klebsiella pneumoniae urinary tract infections. Epidemiol Infect. 2020;149:e12.
[10] Soyaltın E, Erfidan G, Kavruk M, et al. Predictors of febrile urinary tract infection caused by extended-spectrum beta-lactamase-producing bacteria. Turk J Pediatr. 2022;64:265–73.
[11] Bush K, Bradford PA. Epidemiology of β-Lactamase-producing pathogens. Clin Microbiol Rev. 2020;33:e00047–19.
[12] Yeh TK, Lin HJ, Liu PY, Wang J-H, Hsueh P-R. Antibiotic resistance in Enterobacter hormaechei. Int J Antimicrob Agents. 2022;60: 106650.
[13] De Angelis G, Del Giacomo P, Posteraro B, Sanguinetti M, Tumbarello M. Molecular mechanisms, epidemiology, and clinical importance of β-lactam resistance in enterobacteriaceae. Int J Mol Sci. 2020;21:5090–2.
[14] Rodríguez-Villodres A, Gil-Marqués ML, Álvarez-Marín R, et al. Extended-spectrum resistance to β-lactams/β-lactamase inhibitors (ESRI) evolved from low-level resistant *Escherichia coli*. J Antimicrob Chemother. 2020;75:77–85.
[15] Solaiman S, Handy E, Brinks T, et al. Extended spectrum β-lactamase activity and cephalosporin resistance in *Escherichia coli* from U.S. Mid-Atlantic surface and reclaimed water. Appl Environ Microbiol. 2022;88:e0083722.
[16] Bitew A, Adane A, Abdeta A. Bacteriological spectrum, extended-spectrum β-lactamase production and antimicrobial resistance pattern among patients with bloodstream infection in Addis Ababa. Sci Rep. 2023;13:2071–3.
[17] Dikoumba AC, Onanga R, Boundenga L, Bignoumba M, Ngoungou E-B, Godreuil S. Prevalence and characterization of extended-spectrum beta-lactamase-producing enterobacteriaceae in Major Hospitals in Gabon. Microb Drug Resist. 2021;27:1525–34.
[18] Wilkowski P, Hryniewiecka E, Jasińska K, Pączek L, Ciszek M. Breaking antimicrobial resistance: high-dose amoxicillin with clavulanic acid for urinary tract infections due to extended-spectrum beta-lactamase (ESBL)-producing Klebsiella pneumoniae. Ann Transplant. 2023;28:e939258.
[19] Campos-Madueno EI, Moradi M, Eddoubaji Y, et al. Intestinal colonization with multidrug-resistant Enterobacterales: screening, epidemiology, clinical impact, and strategies to decolonize carriers. Eur J Clin Microbiol Infect Dis. 2023;42:229–54.
[20] Kettani Halabi M, Lahlou FA, Diawara I, et al. Antibiotic resistance pattern of extended spectrum beta lactamase producing *Escherichia coli* isolated from patients with urinary tract infection in Morocco. Front Cell Infect Microbiol. 2021;11:e720701.
[21] Cave R, Ter-Stepanyan MM, Kotsinyan N, Mkrtchyan HV. An emerging lineage of uropathogenic extended spectrum β-lactamase *Escherichia coli* ST127. Microbiol Spectr. 2022;10:e0251122.
[22] Sultana KF, Akter A, Saha SR, et al. Bacterial profile, antimicrobial resistance, and molecular detection of ESBL and quinolone resistance gene of uropathogens causing urinary tract infection in the southeastern part of Bangladesh. Braz J Microbiol. 2023;54:803–15.
[23] Gebremedhin MG, Weldu Y, Kahsay AG, Teame G, Adane K. Extended-spectrum β-lactamase and carbapenemase-producing Gram-negative bacteria and associated factors among patients suspected of community and hospital-acquired urinary tract infections at ayder comprehensive specialized hospital, Tigrai, Ethiopia. Infect Drug Resist. 2023;16:4025–37.
[24] Onishi R, Shigemura K, Osawa K, et al. Impact on quinolone resistance of plasmid-mediated quinolone resistance gene and mutations in quinolone resistance-determining regions in extended spectrum beta lactamase-producing Klebsiella pneumoniae isolated from urinary tract infection patients. Pathog Dis. 2022;80:e030.
[25] Xie Z, Jian J, Chen L. Analysis of antimicrobial susceptibility in bacterial pathogens associated with urinary tract infections from Beijing Teaching Hospital in China, 2009-2017. Can J Infect Dis Med Microbiol. 2023;2023:1–8.
[26] Chu LL, Li B, Wu J, et al. Research progress on Rodgersia and predictive analysis on its quality markers. World J Tradit Chin Med. 2023;9:243–57.
[27] Ormeño MA, Ormeñño MJ, Quispe AM, et al. Recurrence of urinary tract infections due to *Escherichia coli* and its association with antimicrobial resistance. Microb Drug Resist. 2022;28:185–90.
[28] Chen R, Wang G, Wang Q, et al. Antimicrobial resistance and molecular epidemiology of carbapenem-resistant *Escherichia coli* from urinary tract infections in Shandong, China. Int Microbiol. 2023;26:1157–66.
[29] Kim B, Kim JH, Lee Y. Virulence factors associated with *Escherichia coli* bacteremia and urinary tract infection. Ann Lab Med. 2022;42:203–12.
[30] Torres-Puig S, García V, Stærk K, et al. "Omics" technologies – what have they told us about uropathogenic *Escherichia coli* fitness and virulence during urinary tract infection? Front Cell Infect Microbiol. 2022;12:e824039.
[31] Hashimoto M, Mao BH, Chiou CS, et al. Association between *Escherichia coli* with NotI-restriction resistance and urinary tract infections. J Microbiol Immunol Infect. 2022;55:686–94.
[32] Chen YC, Lee WC, Chuang YC. Emerging non-antibiotic options targeting uropathogenic mechanisms for recurrent uncomplicated urinary tract infection. Int J Mol Sci. 2023;24:7055–6.
[33] Chaudhary MK, Jadhav I, Banjara MR. Molecular detection of plasmid mediated blaTEM, blaCTX-M,and blaSHV genes in extended spectrum β-lactamase (ESBL) Escherichia coli from clinical samples. Ann Clin Microbiol Antimicrob. 2023;22:33–5.
[34] Alhazmi AH, Alameer KM, Abuageelah BM, et al. Epidemiology and antimicrobial resistance patterns of urinary tract infections: a cross-sectional study from Southwestern Saudi Arabia. Medicina (Kaunas). 2023;59:1411–3.
[35] Zhou Y, Zhang S. Early prediction models for extended-spectrum β-lactamase-producing *Escherichia coli* infection in emergency department: a protocol for systematic review and meta analysis. Medicine (Baltim). 2021;100:e25504.