Research and Reports in Urology

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Quinolone Resistance and Virulence Genes Prevalence of Uropathogenic *Escherichia coli* Isolated from Kidney Transplant Recipients with Urinary Tract Infections in Tehran, Iran

Ghazaleh Talebi[1,*], Mehrzad Sadredinamin[2,*], Mojdeh Hakemi-Vala[3], Atefeh Najafikhah[4], Amin Alizadeh[5]

[1]Department of Biology, Science and Research Branch, Islamic Azad University, Tehran, Iran; [2]Department of Microbiology, School of Medicine, Shahid Beheshti University of Medical Sciences (SBMU), Tehran, Iran; [3]Infectious Diseases and Tropical Research Center, Shahid Beheshti University of Medical Sciences (SBMU), Tehran, Iran; [4]Department of Microbiology, North Tehran Branch, Islamic Azad University, Tehran, Iran; [5]School of Medicine, Shahid Beheshti University of Medical Sciences (SBMU), Tehran, Iran

*These authors contributed equally to this work

Correspondence: Mojdeh Hakemi-Vala, Email m.hakemivala@sbmu.ac.ir; mojdeh_hakemi@yahoo.com

Background: Kidney transplantation is the last suggested treatment option for patients with end-stage kidney disease. Uropathogenic *Escherichia coli* (UPEC) is a leading cause of urinary tract infections (UTIs) in transplant recipients. The rising resistance rates to therapeutic antibiotics and the presence of various virulence factors in *E. coli* pose serious concerns for this vulnerable population. This study aimed to investigate quinolone resistance and key virulence genes in UPEC isolates from kidney transplant recipients.

Materials and methods: Fifty *E. coli* isolates were collected from kidney transplant recipients diagnosed with UTIs who were referred to Yekta, Gholhak, and Labbafi Nezhad Hospital laboratories in Tehran, Iran, from 2022 to 2024. Antimicrobial susceptibility testing (AST) was performed using the Kirby–Bauer disc diffusion method. Additionally, polymerase chain reaction (PCR) was conducted to detect the presence of *ompT, fimH, hlyA, aac (6')-Ib, qnrA*, and *qnrB* genes.

Results: The resistance rates for the following antibiotics were as follows: ampicillin (94%), amoxicillin-clavulanate (54%), ampicillin-sulbactam (64%), piperacillin-tazobactam (54%), cefazolin (88%), cefepime (70%), cefotaxime (80%), cefoxitin (48%), cefpodoxime (80%), doripenem (16%), ertapenem (20%), meropenem (72%), imipenem (18%), gentamicin (34%), tobramycin (44%), amikacin (28%), ciprofloxacin (62%), trimethoprim/sulfamethoxazole (70%), nitrofurantoin (20%), and fosfomycin (86%). Additionally, the frequencies of quinolone-associated resistance genes were reported, with *aac (6')-Ib* at 30%, *qnrA* at 18%, and *qnrB* at 8%. The distribution of virulence genes includes *ompT* at 40%, *fimH* at 82%, and *hlyA* at 10%.

Discussion and conclusion: The rising resistance rates to ciprofloxacin, combined with the higher prevalence of a specific virulence gene in *E. coli* isolates from kidney transplant recipients with UTIs, underscore the need for phenotypic and molecular antimicrobial susceptibility testing. Additionally, analyzing virulence genes is crucial for developing effective treatment strategies and preventing *E. coli* infections in UTI patients.

Keywords: *Escherichia coli*, multidrug resistance, kidney transplantation, urinary tract infections, Quinolon

Introduction

Kidney transplantation is the final treatment option for individuals with end-stage kidney disease, significantly enhancing their survival rates and quality of life. However, infections that occur post-transplant pose a significant challenge, potentially leading to serious complications and increased mortality.[1] Urinary tract infections (UTIs) are especially concerning because they are associated with an increased risk of graft dysfunction and higher healthcare costs.[2] Various factors increase the risk of UTIs in kidney transplant recipients, such as pre-transplant renal impairment, diabetes

https://doi.org/10.2147/RRU.S577044
Received: 27 October 2025
Accepted: 30 January 2026
Published: 7 February 2026

mellitus, older age, and a history of recurrent UTIs.[1] UTIs can lead to sepsis, and recent data indicate that even a single UTI episode may negatively affect graft function, in addition to the risk of progressing to septicemia.[3,4]

Among the bacteria responsible for UTIs, uropathogenic *Escherichia coli* (UPEC) is the most common cause of UTIs, accounting for 90% of community-acquired and 50% of hospital-acquired infections.[5] This bacterium has developed several virulence factors, such as *fimH* (type 1 fimbriae), *hlyA* (hemolysin), and *ompT* (outer membrane protein T). These factors play crucial roles in adhesion, invasion, and resistance to the host's immune defenses, contributing to the severity of UTIs.[6,7] Due to the diverse phylogenetic backgrounds of *E. coli*, the presence and expression of these virulence factors can vary significantly.[8]

The World Health Organization (WHO) has classified fluoroquinolones as a critical group of antibiotics frequently prescribed to kidney transplant patients who have been infected with *E. coli*. However, increased antibiotic resistance has restricted their use.[9–11] Resistance to quinolones in *E. coli* isolates occurs through the protection of DNA gyrase and topoisomerase IV from the action of quinolone antibiotics. This resistance is mediated by plasmid-borne quinolone resistance genes such as *qnrA* and *qnrB*.[11] Additionally, the aminoglycoside-modifying enzyme, encoded by the *aac(6')-Ib* gene, contributes to this resistance by acetylating fluoroquinolones, which reduces susceptibility to these antibiotics.[12]

Given the infections that occur as a result of transplantation cause high mortality, further research is essential to understand the contributing factors associated with antibiotic resistance development and severity of UTIs in transplant patients.[8] Accordingly, this study aims to investigate the prevalence of key virulence genes and quinolone-associated resistance genes in *E. coli* isolates from patients with post-kidney transplant UTIs.

Methods

In this study, all kidneys were donated voluntarily with written informed consent, and that these were conducted in accordance with the Declaration of Istanbul.

Urine Collection

Also, urine collection was done as a part of the routine hospital laboratory procedure for all patients with UTIs signs. All urine samples were collected in sterile containers based on mid-stream clean-catch method.

Bacterial Isolation and Identification

This study was conducted using 50 non-duplicate *E. coli* isolates obtained from kidney transplant patients suffering from UTIs referred to Yekta and Gholhak laboratories and Labbafi Nezhad Hospital in Tehran, Iran, from February 2022 to May 2024. In this study, urine samples were collected in sterile containers. The urine samples were then cultured on three media, including blood Agar, EMB Agar, and MacConkey Agar. Subsequently, biochemical tests were performed on Gram-negative isolates suspected to be Enterobacteriaceae. The isolates, which had been detected as *E. coli*, were later transferred to the microbiology research laboratory at Shahid Beheshti University of Medical Sciences, Tehran, Iran. This study was approved by the ethics committee of Shahid Beheshti University of Medical Sciences (ethical approval code: IR.SBMU.RETECH.REC.1403.533). The isolates were confirmed using Gram staining and standard biochemical tests, including Catalase, Oxidase, IMViC (Indole, Methyl Red, Voges-Proskauer, and Citrate Utilization), Triple Sugar Iron Agar (TSI), Urease, Motility and Oxidative Fermentation tests on pure isolates on Blood Agar and MacConkey Agar.[13] They were stored in a 10% glycerol and TSB solution at −70°C for further evaluation.

Antimicrobial Susceptibility Test

Antimicrobial susceptibility testing was conducted using the Kirby–Bauer disc diffusion method according to Clinical Laboratory and Standards Institute (CLSI) guidelines,[14] and *E. coli* ATCC 25922 was used as a control strain. The antibiotics used in this study included ampicillin (10 µg), ampicillin-sulbactam (10/10 µg), amoxicillin-clavlunate (20/10 µg), cefotaxime (30 µg), cefoxitine (30 µg), cefepime (30 µg), cefazolin (30 µg), imipenem (10 µg), meropenem (10 µg), ertapenem (10 µg), doripenem (10 µg), amikacin (30 µg), tobramycin (10 µg), gentamicin (10 µg), piperacillin/

tazobactam (100/10 µg), ciprofloxacin (5 µg), fosfomycin (200 µg), nitrofurantoin (300 µg), trimethoprim (5 µg), cefpodoxime (10 µg) purchased from Mast group (Merseyside, UK) and Rosco (Taastrup, Denmark) company.

DNA Extraction

Bacterial DNA was extracted using the boiling method. Briefly, pure *E. coli* colonies were harvested from overnight cultures and washed with sterile distilled water. The cell pellet was re-suspended in sterile distilled water, vortexed, and then boiled at 100°C for 10 minutes. The lysate was immediately cooled on ice for 5 minutes and centrifuged to remove cellular debris.[15] The supernatant containing genomic DNA was collected and stored at −70°C for further molecular investigation.

Detection of Virulence Factors, and Fluoroquinolones Resistant Genes

As described in Table 1, PCR with specific primers was utilized to screen for *hlyA, fimH, ompT, aac(6′)Ib, qnrA*, and *qnrB* genes. The PCR reaction was carried out in a total volume of 25 µL that contained 1.5X Taq PCR Master Mix, 1 µM of each primer, and 2 µL of template DNA. The PCR products were subjected to electrophoresis on a 1.5% agarose gel. In this study, three *E. coli* isolates were used as controls, including ATCC 25922, an *E. coli* strain that carries both the *hlyA* and *fimH* genes and dedicated by Dr. Shiva MirKalantari (Iran University of Medical Sciences) along an *qnrA*+ *E. coli* isolate from our previous research.[15]

Sequencing

Sanger sequencing was conducted by the Metabion company to confirm the presence of *ompT, aac(6')Ib*, and *qnrB* genes in a clinical isolate, which harbored each of these genes. Further nucleotide analyses were performed using Chromas 1.45 software and BLAST in NCBI.

Table 1 Primer Sequences, Product Sizes, and Annealing Temperatures for Quinolone Resistance and Virulence Genes in Clinical Isolates of *E. coli*

Primer Name	Sequence	Target Gene	Product Size (bp)	Accession Number	Annealing Temperature (°C)	Reference
hlyA- F	AACAAGGATAAGCACTGTTCTGGCT	*hlyA*	1177	Not available	63	[16]
hlyA- R	ACCATATAAGCGGTCATTCCCGTCA					
fimH-F	TGCAGAACGGATAAGCCGTGG	*fimH*	506	PQ676531	63	[17]
fimH-R	GCAGTCACCTGCCCTCCGGTA					
ompT-F	TTTGATGCCCCAGATATCTATCGG	*ompT*	236	PQ676532	63	[18]
ompT-R	GGCTTTCCTGATATCCGGCCATG					
aac(6′)Ib-F	TTGCGATGCTCTATGAGTGGCTA	*aac(6′)Ib*	482	PQ676530	58	[19]
aac(6′)Ib-R	CTCGAATGCCTGGCGTGTTT					
qnrA-F	GGGTATGGATATTATTGATAAAG	*qnrA*	661	Not Available	58	[20]
qnrA-R	CTAATCCGGCAGCACTATTA					
qnrB-F	GGAATAGAAATTCGCCACTG	*qnrB*	263	PQ676533	57	[21]
qnrB-R	TTTGCTGTTCGCCAGTCGAA					

Statistical Analysis

Descriptive statistics were utilized in this study using GraphPad Prism software version 9.4.1 to assess the frequency of antibiotic resistance and virulence genes. Prevalence estimates are provided as n (%) with 95% confidence intervals (CIs) using the Wilson score method. No comparative analyses were performed.

Results

Bacterial Identification and Antimicrobial Susceptibility Results

All *E. coli* colonies were identified and confirmed after visualization of Gram-negative bacilli, Oxidase (-), TSI A/A gas (+), H 2 S (-), IMViC (+ + - -), Urease (-), and Motility (+). Antimicrobial susceptibility testing of isolates which have been interpreted according to the CLSI guideline was displayed that the highest resistance rate was for ampicillin (94%, CI: 83.5–98.0), followed by cefazolin (88%, CI: 76.2–94.4), fosfomycin (86%, CI: 73.4–93.1), cefotaxime (80%, CI: 66.8–88.9), cefpodoxime (80%, CI: 66.8–88.9), meropenem (72%, CI: 58.1–82.5), trimethoprim/sulfamethoxazole (70%, CI: 56.2–80.9), cefepime (70%, CI: 56.2–80.9), ampicillin-sulbactam (64%, CI: 50.1–75-9), ciprofloxacin (62%, CI: 48.2–74.1), amoxicillin-clavulanate (54%, CI: 40.4–67.0), piperacillin-tazobactam (54%, CI: 40.4–67.0), cefoxitin (48%, CI: 34.8–61.5), tobramycin (44%, CI: 31.2–57.7), gentamicin (34%, CI: 22.4–47.8), amikacin (28%, CI: 17.5–41.7), ertapenem (20%, CI: 11.2–33.0), nitrofurantoin (20%, CI: 11.2–33.0), imipenem (18%, CI: 9.8–30.8), doripenem (1%, CI 0.0–7.1), as illustrated in Figure 1.

Identification of Resistance and Virulence Genes by PCR

PCR results demonstrated that among *E. coli* isolates from kidney transplant recipients with UTI, *fimH* had the highest frequency (82%, CI: 69.2–90.2), followed by *ompT* (40%, CI:27.6–53.8), *aac (6′) Ib* (30%, CI: 19.1–43.8), *qnrA* (18%, CI: 9.8–30.8), *hlyA* (10%, CI: 4.3–21.4), and *qnrB* (8%, CI: 3.2–18.8), which were at a lower level (Figure 2). Representative PCR-gel electrophoresis images are provided in a Supplementary Figure 1.

The simultaneous presence of invasive and resistance genes was observed in several isolates. Notably, 28% (CI: 17.5–41.7) of the isolates carried both *aac (6′) Ib* and *fimH*, 20% (CI: 11.2–33.0) had *aac (6′) Ib* and *ompT*, and 6% (CI: 2.1–16.2) demonstrated *aac (6′) Ib* and *hlyA*. Moreover, 16% (CI: 8.3–28.5) of *E. coli* isolates harbored both *qnrA* and

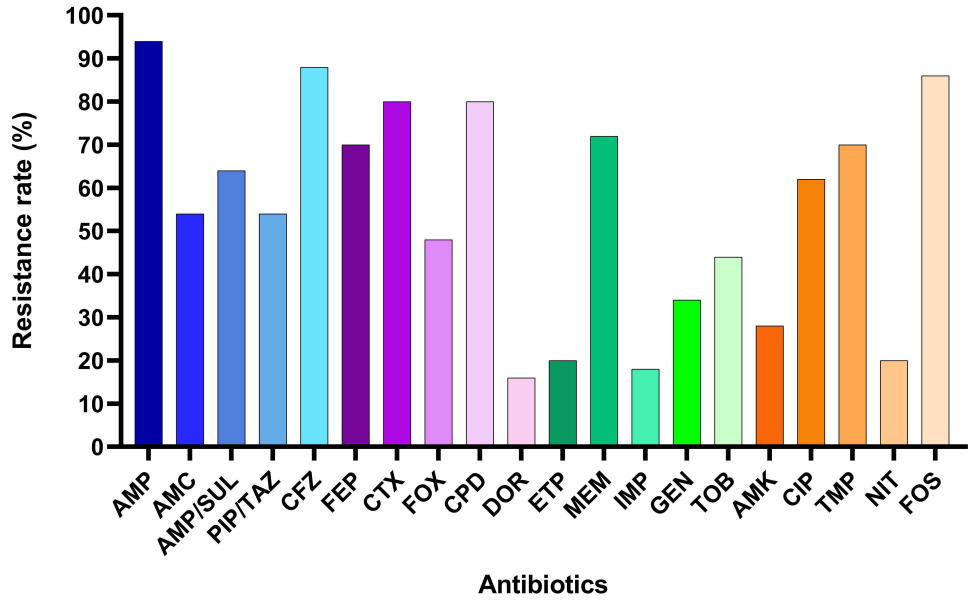

Figure 1 Antibiotic resistance rate in *E. coli* isolates from kidney transplantation with urinary tract infections. Resistance was assessed by disk diffusion method.
Abbreviations: AMP, Ampicillin; AMC, Amoxiclav; AMP/SUL, Ampicillin/Sulbactam; PIP/TAZ, Piperacillin/Tazobactam; CFZ, Cefazolin; FEP, Cefepime (fourth-generation cephalosporisn); CTX, Cefotaxime; FOX, Cefoxitin; CPD, Cefpodoxime; DOR, Doripenem; ETP, Ertapenem; MEM, Meropenem; IMP, Imipenem; GEN, Gentamicin; TOB, Tobramycin; AMK, Amikacin; CIP, Ciprofloxacin; TMP, Trimethoprim; NIT, Nitrofurantoin; FOS, Fosfomycin.

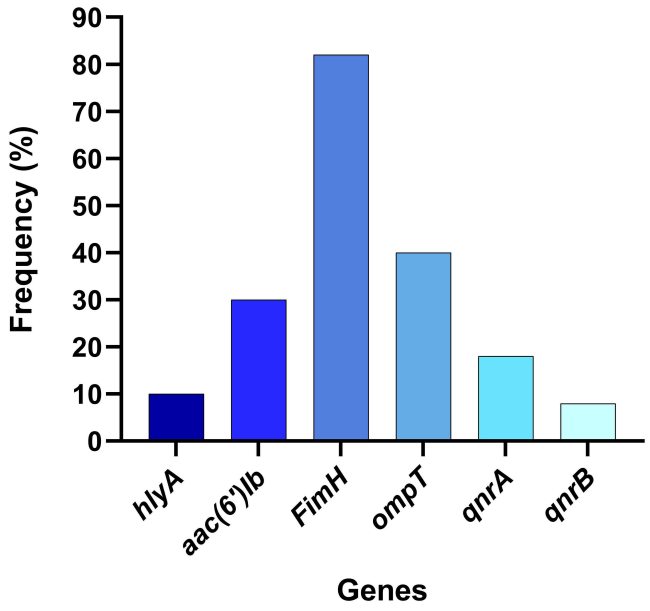

Figure 2 Frequency of quinolone resistance and virulence genes detected by PCR among *E. coli* isolates from kidney transplantation with urinary tract infections.

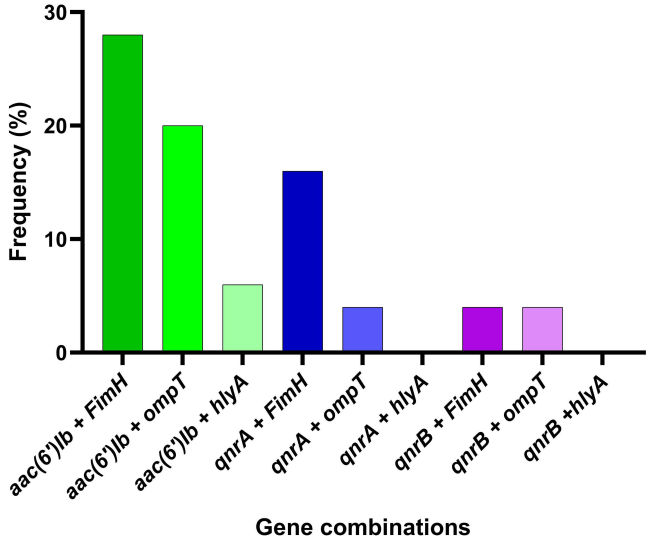

Figure 3 Co-occurrence of resistance and virulence genes in *E. coli* isolates from kidney transplantation with urinary tract infections.

fimH, while 4% (CI: 1.1–13.5) had *qnrA* and *ompT*. None of the isolates concurrently exhibited *qnrA* and *hlyA, qnrB* and *hlyA* (CI: 0.0–7.1). Additionally, 4% (CI: 1.1–13.5) of isolates showed the presence of *qnrB* and *fimH*, and 4% (CI: 1.1–13.5) co-harbored *qnrB* and *ompT* (Figure 3).

Discussion

Kidney transplant recipients are at increased risk of UTIs due to the long-term use of immunosuppressive drugs. Among the various bacteria, *E. coli* is the most common pathogen associated with these infections and is often linked to multidrug resistance (MDR).[22] In the current study, the resistance rates to ampicillin and ampicillin/sulbactam within the β-lactam class were higher than those for other antibiotics in this group. Additionally, the resistance rate to ciprofloxacin, a fluoroquinolone, was notably high, exceeding 60%. These findings are consistent with previous research.[23,24] However, other epidemiological investigations reported a lower frequency of fluoroquinolone resistance.[25,26] The discrepancies

https://doi.org/10.2147/RRU.S577044

between these findings may be attributed to variations in geographical regions, antibiotic prescribing patterns, and infection control policies in their respective countries.

In addition to epidemiological factors, genetic mechanisms also play a critical role in the development of fluoroquinolone resistance patterns. The emergence of antibiotic resistance can be associated with genes that confer resistance, including plasmid-mediated determinants that specifically affect fluoroquinolone susceptibility.[27]

The findings revealed that the prevalence of each of the *qnrA, qnrB*, and *aac (6')-Ib* genes stood at 18%, 8%, and 30%, respectively. Raheem et al reported a pattern comparable to that observed in the present study. Their research highlighted that the *qnr* gene was detected in 18% of UPEC isolates.[28] Similarly, other studies have reported a low prevalence of *qnr* genes in clinical isolates.[29,30] Given that 30% of the isolated strains harbored the *aac (6')-Ib* gene, it is likely that this gene can contribute to the development of fluoroquinolone resistance.[31] In the similar investigation conducted by Esmaeel et al, the *aac (6')-Ib* gene was identified as the most common fluoroquinolone resistance gene. However, the prevalence of *qnr* genes was found to be higher than what we reported.[32] Additionally, Badamchi et al documented that the *aac(6')-Ib* gene was present in 24% of *E. coli* isolates from patients in Iran.[33]

Research indicates that antibiotic-resistant genes exhibit varying prevalence across different studies and geographical regions. This variability can be linked to antibiotic usage patterns and different consumption regimes.[34] In addition to antimicrobial resistance, bacterial virulence factors significantly influence the severity and clinical outcome of urinary tract infections.[35]

Various virulence factors contribute to the pathogenicity of *E. coli*, which can be detected through PCR.[36] The gene coding for FimH adhesion is recognized as one of the consequential factors in the virulence of *E. coli* in UTIs, facilitating pathogenicity through the colonization, invasion, and persistence of UPEC in the bladder.[37] Our findings indicate a higher frequency of *fimH* gene than other examined genes, suggesting that this gene plays a crucial role in *E. coli* infections causing UTI. The distribution of this gene in our strains aligns with previously published data.[38,39] However, other studies have noted a higher percentage of *fimH* PCR-positive strains in patients with UTIs.[40–42] This discrepancy can be explained by the occurrence of mutations and the failure to detect them.[39] In this investigation, the frequencies of the *hlyA* and *ompT* genes were 10% and 40%, respectively. However, the prevalence of these virulence factors varies across other studies, which differences in sample sources, geographical regions, and host clinical conditions can explain this discrepancy.[43,44] Furthermore, our findings indicated a considerable number of isolates carrying both invasive and resistance genes simultaneously, which aligns with previous studies.[45,46] Isolates that concurrently harbored both types of genes may cause more severe infections or exhibit a poor response to treatment. Additionally, the co-existence of invasive and resistance genes on a plasmid could pose a heightened threat to *E. coli* infection control, as they can be co-transferred and spread to other isolates.

Conclusion

The current study reveals significant levels of antibiotic resistance and the distribution of virulence genes in *E. coli* isolates from kidney transplant recipients with UTIs. To the best of our knowledge, the current study represents the first report of the simultaneous occurrence of plasmid-mediated quinolone resistance and invasive genes in *E. coli* isolates from UTIs in kidney transplant patients, which may be associated with poor therapeutic outcomes. High phenotypic and molecular resistance rates to therapeutic antibiotics, such as ciprofloxacin, increase the risk of treatment failure in *E. coli* UTI infections. The high prevalence of *fimH*, which is associated with bacterial adhesion, colonization, and persistence of *E. coli* in the urinary tract, also emphasizes the role of this gene in the development of *E. coli* infection in UTI patients. Collectively, these findings highlight the critical need to apply both phenotypic antimicrobial susceptibility testing and molecular screening for antibiotic resistance, along with analysis of virulence genes, to develop effective treatment strategies and prevent *E. coli* infections in patients with UTIs. Further multicenter, longitudinal studies are required to examine the clinical impact of these genetic profiles across a broader range of clinical isolates to determine treatment outcomes and inform evidence-based therapeutic guidelines.

Abbreviations

aac (6′)-Ib, Aminoglycoside acetyltransferase (6′)-Ib; AST, Antimicrobial Susceptibility Testing; ATCC, American Type Culture Collection; CI, Confidence interval; CLSI, Clinical and Laboratory Standards Institute; *E. coli, Escherichia coli; fimH*, Fimbrial adhesin gene; *hlyA*, Hemolysin A; IMViC, Indole, Methyl red, Voges-Proskauer, Citrate; MDR, Multi Drug Resistant; NCBI, National Center for Biotechnology Information; *ompT*, Outer membrane protease T; PCR, Polymerase Chain Reaction; *qnrA*, quinolone resistance gene A; *qnrB*, quinolone resistance gene B; TSI, Triple Sugar Iron Agar; UPEC, Uropathogenic *Escherichia coli*; UTI, Urinary Tract Infection.

Data Sharing Statement

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

Ethics Approval and Informed Consent

This study was approved by the ethics committee of Shahid Beheshti University of Medical Sciences (ethical approval code: IR.SBMU.RETECH.REC.1403.533).

Acknowledgments

Authors like to say their thanks to infectious diseases and tropical research center Shahid Beheshti University of Medical Sciences for their support. Also, the authors wish to thank Dr. Shiva Mirkalantari, Microbiology Department, Iran University of Medical Sciences, Tehran, Iran, for kindly providing an *E. coli* isolate harbored *hlyA* and *fimH* genes used in this study.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study is financially supported by Infectious diseases and tropical research center, Shahid Beheshti University of Medical Sciences (SBMU) Tehran, Iran.

Disclosure

There is no conflict of interest to declare.

References

1. Fiorentino M, Pesce F, Schena A, Simone S, Castellano G, Gesualdo L. Updates on urinary tract infections in kidney transplantation. *J Nephrol*. 2019;32(5):751–761. doi:10.1007/s40620-019-00585-3
2. Espinar MJ, Miranda IM, Costa-de-Oliveira S, Rocha R, Rodrigues AG, Pina-Vaz C. Urinary tract infections in kidney transplant patients due to *Escherichia coli* and *Klebsiella pneumoniae*-producing extended-spectrum β-lactamases: risk factors and molecular epidemiology. *PLoS One*. 2015;10(8):e0134737. doi:10.1371/journal.pone.0134737
3. Ariza-Heredia EJ, Beam EN, Lesnick TG, Cosio FG, Kremers WK, Razonable RR. Impact of urinary tract infection on allograft function after kidney transplantation. *Clin Transplant*. 2014;28(6):683–690. doi:10.1111/ctr.12366
4. Sánchez MPR, Rubio DCA, Luna IM, et al. Impact of complicated urinary tract infection on renal graft function. In: *Transplantation Proceedings*. Elsevier; 2020.
5. He XL, Wang Q, Peng L, et al. Role of uropathogenic *Escherichia coli* outer membrane protein T in pathogenesis of urinary tract infection. *Pathog Dis*. 2015;73(3):ftv006. doi:10.1093/femspd/ftv006
6. Lüthje P, Brauner A. Virulence factors of uropathogenic *E. coli* and their interaction with the host. *Adv Microbial Physiol*. 2014;65:337–372.
7. Kalu M, Jorth P, Wong-Beringer A. Comparison of phenotypic and genetic traits of ESBL-producing UPEC strains causing recurrent or single episode UTI in postmenopausal women. *Ann Clinic Microbiol Antimicrob*. 2025;24:11. doi:10.1186/s12941-025-00779-7
8. Clermont O, Christenson JK, Denamur E, Gordon DM. The Clermont E scherichia coli phylo-typing method revisited: improvement of specificity and detection of new phylo-groups. *Environ Microbiol Rep*. 2013;5(1):58–65. doi:10.1111/1758-2229.12019

9. Strohaeker J, Aschke V, Koenigsrainer A, Nadalin S, Bachmann R. Urinary tract infections in kidney transplant recipients—is there a need for antibiotic stewardship? *J Clin Med*. 2021;11(1):226. doi:10.3390/jcm11010226
10. Cuypers WL, Jacobs J, Wong V, Klemm EJ, Deborggraeve S, Van Puyvelde S. Fluoroquinolone resistance in Salmonella: insights by whole-genome sequencing. *Microbial Genomics*. 2018;4(7):e000195. doi:10.1099/mgen.0.000195
11. Gomaa Elsayed A, E MF, Abdellatif Alsayed M, Ahmed ME, M ESZ, Mofreh mohamed M. Study of plasmid mediated quinolone resistance genes among *Escherichia coli* and *Klebsiella pneumoniae* isolated from pediatric patients with sepsis. *Sci Rep*. 2024;14(1):11849. doi:10.1038/s41598-024-61357-z
12. Rodríguez-Martínez JM, Machuca J, Cano ME, Calvo J, Martínez-Martínez L, Pascual A. Plasmid-mediated quinolone resistance: two decades on. *Drug Resist Updates*. 2016;29:13–29. doi:10.1016/j.drup.2016.09.001
13. PM T. *Bailey & Scott's Diagnostic Microbiology*. St Louis, Missouri: Elsevier; 2014.
14. Clinical and Laboratory Standards Institute. *Performance Standards for Antimicrobial Susceptibility Testing, 33rd Edition: CLSI Supplement M100*. Wayne, PA: CLSI; 2024.
15. Zahedi Z, Vala MH, Bejestani FB. Contribution of GyrA and QnrA genes in ciprofloxacin resistant Ecsherichia coli isolates from patients with urinary tract infections of Imam Khomeini Hospital of Tehran. *Arch Pediatr Infect Dis*. 2018;6(4):1–7.
16. Raeispour M, Ranjbar R. Antibiotic resistance, virulence factors and genotyping of Uropathogenic *Escherichia coli* strains. *Antimicrob Resist Infect Control*. 2018;7:118. doi:10.1186/s13756-018-0411-4
17. Haghighatpanah M, Mojtahedi A. Characterization of antibiotic resistance and virulence factors of *Escherichia coli* strains isolated from Iranian inpatients with urinary tract infections. *Infect Drug Resist*. 2019;12:2747–2754. doi:10.2147/IDR.S219696
18. Desloges I, Taylor JA, Leclerc JM, et al. Identification and characterization of OmpT-like proteases in uropathogenic *Escherichia coli* clinical isolates. *Microbiologyopen*. 2019;8(11):e915. doi:10.1002/mbo3.915
19. Hu X, Xu B, Yang Y, et al. A high throughput multiplex PCR assay for simultaneous detection of seven aminoglycoside-resistance genes in Enterobacteriaceae. *BMC Microbiol*. 2013;13:58. doi:10.1186/1471-2180-13-58
20. Horri M, Gholami-Ahangaran M. Phenotypic and genotypic characterization of antibiotic resistance in *Escherichia coli* strains isolated from broiler chickens with suspected Colibacillosis in Isfahan Province, Iran. *Infect Epidemiol Microbiol*. 2022;8(3):193–201. doi:10.52547/iem.8.3.193
21. Calayag AMB, Widmer KW, Rivera WL. Antimicrobial susceptibility and frequency of bla and qnr genes in Salmonella enterica Isolated from Slaughtered Pigs. *Antibiotics*. 2021;10(12). doi:10.3390/antibiotics10121442
22. Yuan X, Liu T, Wu D, Wan Q. Epidemiology, susceptibility, and risk factors for acquisition of MDR/XDR Gram-negative bacteria among kidney transplant recipients with urinary tract infections. *Infect Drug Resist*. 2018;11:707–715. doi:10.2147/IDR.S163979
23. Ahmed SA, Flayyih MT. Detection of afa and yqi in Ciprofloxacin Resistant Uropathogenic Echerichia coli. *Ibn AL-Haitham J Pure Appl Sci*. 2025;38(2):52–62.
24. Hemmati H, Taher M, Shahbazi AE, Malekzadegan Y. Prevalence and antimicrobial resistance pattern of uropathogenic *Escherichia coli* at a Tertiary Care Hospital: a Retrospective Study. *J Med Bacteriol*. 2024. doi:10.18502/jmb.v12i2.15624
25. García-Meniño I, García V, Lumbreras-Iglesias P, Fernández J, Mora A. Fluoroquinolone resistance in complicated urinary tract infections: association with the increased occurrence and diversity of *Escherichia coli* of clonal complex 131, together with ST1193. *Front Cell Infect Microbiol*. 2024;14:1351618. doi:10.3389/fcimb.2024.1351618
26. Chreim S, Hosseini SM, Medlej A, Tarhini M. The evaluation of antimicrobial resistance rates in infections caused by uropathogenic *Escherichia coli* strains collected from the south of Lebanon. *Iranian J Microbiol*. 2025;17(2):261. doi:10.18502/ijm.v17i2.18386
27. Shetty SS, Deekshit VK, Jazeela K, et al. Plasmid-mediated fluoroquinolone resistance associated with extra-intestinal *Escherichia coli* isolates from hospital samples. *Indian J Med Res*. 2019;149(2):192–198. doi:10.4103/ijmr.IJMR_2092_17
28. Qasim Raheem H, Al-Maliki L, Alaa Abdolzahra M, Shafi Hussein T. Prevalence of Extended-Spectrum B-Lactamase (ESBL) and Quinolone Resistance (qnr) genes among cytotoxic necrotizing Factor-1-Producing Uropathogenic *Escherichia coli* in Babylon, Iraq. *Infect Epidemiol Microbiol*. 2023;9(3):209–218. doi:10.61186/iem.9.3.209
29. Malekzadegan Y, Rastegar E, Moradi M, Heidari H, Sedigh Ebrahim-Saraie H. Prevalence of quinolone-resistant uropathogenic *Escherichia coli* in a tertiary care hospital in south Iran. *Infect Drug Resist*. 2019;12:1683–1689. doi:10.2147/IDR.S206966
30. Amiri M, Jajarmi M, Ghanbarpour R. Prevalence of resistance to quinolone and fluoroquinolone antibiotics and screening of qnr genes among *Escherichia coli* isolates from urinary tract infection. *Int J Enteric Pathog*. 2017;5(4):100–105. doi:10.15171/ijep.2017.24
31. Liang S, Cai W, Mao R, et al. Three simple and cost-effective assays for AAC (6′)-Ib-cr enzyme activity. *Front Microbiol*. 2025;16:1513425. doi:10.3389/fmicb.2025.1513425
32. Esmaeel NE, Gerges MA, Hosny TA, Ali AR, Gebriel MG. Detection of chromosomal and plasmid-mediated quinolone resistance among *Escherichia coli* Isolated from urinary tract infection cases; Zagazig University Hospitals, Egypt. *Infect Drug Resist*. 2020;13:413–421. doi:10.2147/IDR.S240013
33. Badamchi A, Javadinia S, Farahani R, Solgi H, Tabatabaei A. Molecular detection of plasmid-mediated quinolone resistant genes in uropathogenic *E. coli* from tertiary referral hospital in Tehran, Iran. *Arch Pharmacol Therap*. 2019;1(1):19–24.
34. Javadi B, Kafshdouzan K, Chashmi SHE, Pazhand O. Plasmid-mediated quinolone resistance in *Escherichia coli* isolates from commercial broiler chickens in Semnan, Iran. *Iranian J Microbiol*. 2024;16(2):193. doi:10.18502/ijm.v16i2.15352
35. Bunduki GK, Heinz E, Phiri VS, Noah P, Feasey N, Musaya J. Virulence factors and antimicrobial resistance of uropathogenic *Escherichia coli* (UPEC) isolated from urinary tract infections: a systematic review and meta-analysis. *BMC Infect Dis*. 2021;21(1):753. doi:10.1186/s12879-021-06435-7
36. Valadbeigi H, Esmaeeli E, Ghafourian S, Maleki A, Sadeghifard N. Molecular analysis of uropathogenic *E. coli* isolates from urinary tract infections. *Infect Disorders-Drug Targets*. 2019;19(3):322–326.
37. Derakhshandeh S, Shahrokhi N, Khalaj V, et al. Surface display of uropathogenic *Escherichia coli* FimH in Lactococcus lactis: in vitro characterization of recombinant bacteria and its protectivity in animal model. *Microb Pathogenesis*. 2020;141:103974. doi:10.1016/j.micpath.2020.103974
38. Pourzare M, Derakhshan S, Roshani D. Distribution of uropathogenic virulence genes in *Escherichia coli* isolated from children with urinary tract infection in Sanandaj, Iran. *Arch Pediatr Infect Dis*. 2017;5(3):e41995.

39. Tarchouna M, Ferjani A, Ben-Selma W, Boukadida J. Distribution of uropathogenic virulence genes in *Escherichia coli* isolated from patients with urinary tract infection. *Inter J Infect Dis*. 2013;17(6):e450–e3. doi:10.1016/j.ijid.2013.01.025
40. Chakraborty A, Adhikari P, Shenoy S, Saralaya V. Molecular characterisation of uropathogenic *Escherichia coli* isolates at a tertiary care hospital in South India. *Indian J Med Microbiol*. 2017;35(2):305–310. doi:10.4103/ijmm.IJMM_14_291
41. Cristea VC, Gheorghe I, Czobor Barbu I, et al. Snapshot of phylogenetic groups, virulence, and resistance markers in *Escherichia coli* uropathogenic strains isolated from outpatients with urinary tract infections in Bucharest, Romania. *Biomed Res Int*. 2019;2019(1):5712371. doi:10.1155/2019/5712371
42. Najafi A, Hasanpour M, Askary A, Aziemzadeh M, Hashemi N. Distribution of pathogenicity island markers and virulence factors in new phylogenetic groups of uropathogenic *Escherichia coli* isolates. *Folia Microbiologica*. 2018;63:335–343. doi:10.1007/s12223-017-0570-3
43. Boroumand M, Sharifi A, Ghatei MA, Sadrinasab M. Evaluation of biofilm formation and virulence genes and association with antibiotic resistance patterns of uropathogenic *Escherichia coli* strains in southwestern Iran. *Jundishapur J Microbiol*. 2021;14(9):1–10. doi:10.5812/jjm.117785
44. Boroumand M, Sharifi A, Manzouri L, Khoramrooz SS. *Evaluation of Pap and Sfa Genes Relative Frequency P and S Fimbriae Encoding of Uropathogenic Escherichia coli Isolated From Hospitals and Medical Laboratories*. Yasuj City, Southwest Iran; 2019.
45. Paniagua-Contreras GL, Monroy-Pérez E, Rodríguez-Moctezuma JR, Domínguez-Trejo P, Vaca-Paniagua F, Vaca S. Virulence factors, antibiotic resistance phenotypes and O-serogroups of *Escherichia coli* strains isolated from community-acquired urinary tract infection patients in Mexico. *J Microbiol Immunol Infect*. 2017;50(4):478–485. doi:10.1016/j.jmii.2015.08.005
46. RM AE-B, Ibrahim RA, Mohamed DS, Ahmed EF, Hashem ZS. Prevalence of virulence genes and their association with antimicrobial resistance among pathogenic *E. coli* isolated from Egyptian patients with different clinical infections. *Infect Drug Resist*. 2020;13:1221–1236. doi:10.2147/IDR.S241073