BMC Research Notes

RESEARCH NOTE Open Access

Antibiotic resistance and virulence patterns of O25 and O16 serogroups in uropathogenic *Escherichia coli*

Seyedamirmehdi Hejazi Dehaghani[1], Haider Qassim Raheem[2*], Mohammad Latifpour[3,4] and Mehrdad Hallaji[5*]

Abstract

Objective This study investigates antibiotic resistance patterns, virulence factors, and phylogenetic groups of O25 and O16 serogroups in uropathogenic *Escherichia coli* (UPEC) isolates from kidney transplant recipients (KTPs) and non-KTPs. The presence of serogroups O25 and O16, resistance genes (e.g., bla_{CTX-M}, bla_{TEM}), and virulence factors (e.g., *fimH*, *PAI*) were determined using PCR. Phylogenetic groups were identified via quadruplex PCR, and genetic diversity was assessed using ERIC-PCR.

Results A total of 111 *E. coli* isolates were examined in the present study. The O-serotyping results indicated that 18% and 3.6% of isolates were positive for O25 and O16 serogroup, respectively. In serogroup O25, the highest resistance rates were observed for nalidixic acid and cotrimoxazole, whereas in serogroup O16, the highest resistance rates were against cotrimoxazole and ampicillin-sulbactam. ESBL production was identified in 30% of O25 and 25% of O16 isolates. O25 isolates belonged to phylogenetic group B2, whereas O16 isolates were grouped in B1. ERIC-PCR revealed significant genetic diversity among isolates. The O25 serogroup is prevalent and closely associated with high antibiotic resistance and virulence, suggesting its critical role in UTI pathogenesis in transplant patients. These findings underscore the importance of monitoring resistance patterns and developing targeted therapeutic and preventive strategies for managing UPEC infections.

Keywords O serogroup, UPEC, ESBL, ERIC-PCR, Phylogenetic groups

Introduction

Renal transplantation is one of the primary and most effective treatment methods for patients with advanced and chronic kidney failure [1, 2]. Urinary tract infections (UTIs) are the most common bacterial infections among these patients. The prevalence of these infections varies globally, ranging from 35 to 79%, and approximately 60% of hospital-acquired septicemias in kidney transplant recipients are attributed to UTIs [3]. The most common pathogens responsible for UTIs in these patients are Enterobacterales, *Enterococcus* spp., *Staphylococcus* spp., and *Pseudomonas* spp [4–6]. The primary causative agent of UTIs is uropathogenic *Escherichia coli* (UPEC). *E. coli*

*Correspondence:
Haider Qassim Raheem
haider.qassim@uobabylon.edu.iq; haiderbio412@gmail.com
Mehrdad Hallaji
m.hallaji@resident.mui.ac.ir
[1]School of Medicine, Padova University, Padova, Italy
[2]DNA Research Center, University of Babylon, Babylon, Iraq
[3]Cultural and Research Affairs, Fatemeh Al-Zahra (S) Hospital, Ahvaz, Iran
[4]Department of Microbiology, Javad Al-Aemeh Specialized and Subspecialized Laboratory, Ahvaz, Iran
[5]Department of Microbiology, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran

© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by-nc-nd/4.0/.

strains are typically identified through serological typing of surface antigens, including flagellar (H), lipopolysaccharide (O), and, in some cases, capsular (K) antigens [7]. A total of 174 O serogroups have been described for *E. coli*. O serogroups of UPEC strains are associated with specific virulence factor profiles unique to each strain. Previous studies have reported that serogroups O1, O2, O4, O6, O7, O8, O15, O16, O18, O21, O22, O25, O75, and O83 are frequently expressed in UPEC clones [8].

Treating these infections often requires antibiotic therapy; however, antibiotic-resistant strains tend to cause more severe and prolonged infections than antibiotic-sensitive strains. Numerous studies have demonstrated a rising trend in antibiotic resistance among UPEC strains [9]. The global dissemination of multidrug-resistant (MDR) bacterial strains has emerged as a critical public health concern. Several recent investigations have reported the emergence of MDR bacterial pathogens from various origins, highlighting the increasing necessity for the proper use of antibiotics. Additionally, the routine application of antimicrobial susceptibility testing is essential to determine the appropriate antibiotic of choice, as well as to screen for emerging MDR strains. Given the high prevalence of antimicrobial resistance in UPEC strains, analyzing their antibiotic resistance patterns and identifying MDR and extended-spectrum beta-lactamase (ESBL)-producing strains are crucial steps toward reducing treatment costs and expediting patient recovery [10–15].

Moreover, the levels of antibiotic resistance, virulence factors, and phylogenetic group distribution of *E. coli* vary based on serotypes, necessitating further detailed studies in this area [16, 17]. To understand the role and significance of *E. coli* O25 and O16 serogroups in the development of UTIs in both transplant and non-transplant patients, this study investigates the antibiotic resistance patterns, virulence factors, and phylogenetic groups of O25 and O16 serogroups in uropathogenic *E. coli* (UPEC) isolates from kidney transplant recipients (KTPs) and non-KTPs.

Materials and methods

Bacterial isolates

As previously described, a total of 111 non-repetitive UPEC isolates were collected from patients in this case-control study, including 65 isolates from non-KTPs (control group) and 46 isolates from KTPs ($n = 46$). The samples were obtained from two nephrology private clinics and a laboratory affiliated with Isfahan University of Medical Sciences (IUMS) between June and October 2019. In this retrospective descriptive study, all isolates were identified using standard microbiological and biochemical tests, with confirmation established in a prior study conducted by our team [18, 19].

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing and phenotypic evaluations were conducted using the disc diffusion technique in accordance with Clinical & Laboratory Standards Institute (CLSI) guidelines [20]. To identify ESBL-producing strains, the double-disk synergy test (DDST) was employed as a phenotypic approach, following CLSI recommendations [20]. All strains had been previously characterized [19]. In this assay, 16 antibiotics (BD BBL™ Sensi-Disc™) were previously tested, including amoxicillin/clavulanic acid, piperacillin/tazobactam, cefoxitin, ceftazidime, cefepime, cefixime, imipenem, meropenem, trimethoprim/sulfamethoxazole, ciprofloxacin, ofloxacin, nalidixic acid, norfloxacin, nitrofurantoin, gentamicin, and amikacin [19]. *Escherichia coli* ATCC® 25,922™ was considered the quality control strain for these susceptibility tests [21].

Screening of *E. coli* strains of O-serogroup

Genomic DNA was extracted from fresh colonies, and PCR was performed to detect the presence of O16 and O25 genes using specific primers, as previously described [22].

Molecular detection of resistance and virulence genes

The presence of ESBLs genes, including $bla_{\text{CTX-M}}$, bla_{TEM}, bla_{SHV}, PMQR (*qnrA*, *qnrA* and *qnrS*), efflux pumps (*acrB*, *acrA* and *tolC*), integrons (*Int2* and *Int2*) and virulence genes (*papG I*, *II*, *III*, *sfaDE*, *afaBC*, *fimH*, *iutA*, *chuA*, *PAI*, *hlyA*, and *cnf1*) was determined using PCR assays as previously described [18, 19].

Phylogenetic analysis

All UPEC isolates were categorized into phylogenetic groups using quadruplex PCR, following the method by Clermont et al. [23].

ERIC-PCR

The ERIC-PCR technique was utilized to examine O25 and O16 UPEC isolates, with the primer sequence for the study having been previously reported [24]. The ERIC patterns were analyzed using GelJ software, version 2.0, as previously described. Isolates demonstrating a similarity coefficient of 80% or higher were grouped and classified as belonging to the same genotypes [25, 26].

Statistical analysis

Statistical analysis was performed using SPSS™ software, version 16 (IBM Corp., USA). Categorical variables were presented as frequencies and percentages. The Fisher's exact test or Chi-square (χ^2) test was applied to evaluate significant differences. A *P*-value of less than 0.05 was regarded as statistically significant.

Results

A total of 111 *E. coli* isolates were examined in the present study. The O-serotyping results indicated that 18% (20/111) and 3.6% (4/111) of the examined strains were positive for serogroup O25 and serogroup O16, respectively.

Antibiotic resistance patterns in serogroups O25 and O16

The antibiotic resistance patterns of the two serogroups were analyzed and summarized in Tables 1 and 2. In serogroup O25, the highest resistance rates were observed for nalidixic acid and cotrimoxazole. However, all isolates were 100% sensitive to amikacin, meropenem, and imipenem. In serogroup O16, the highest resistance rates were against cotrimoxazole and ampicillin-sulbactam (75%), while all isolates were 100% sensitive to amikacin, gentamicin, norfloxacin, ciprofloxacin, meropenem, imipenem, cefepime, and piperacillin-tazobactam.

Statistical analysis revealed a significant correlation between the presence of O16 and O25 genes and resistance to ciprofloxacin and norfloxacin. Additionally, 30% of the isolates in serogroup O25 and 25% of the isolates in serogroup O16 were identified as ESBL producers.

Distribution of antibiotic resistance genes

The frequency distribution of antibiotic resistance genes demonstrated that in serogroup O25, the most prevalent genes were $bla_{\text{CTX-M}}$ (55%) for ESBL, *qnrB* (40%) for PMQR, *acrB* (100%) for efflux pumps, and *Int1* (90%) for integrons. In comparison, serogroup O16 predominantly exhibited bla_{TEM} (50%) for ESBL, *acrB* (100%) and *tolC* (100%) for efflux pumps, and *Int2* (25%) for integrons. Notably, PMQR genes were not detected in any of the isolates belonging to serogroup O16. Statistical analysis revealed no significant correlation between the presence of $bla_{\text{CTX-M}}$ and *Int1* genes and serogroup O25.

Table 1 Distribution of virulence-related genes and antibiotic resistance genes in O25 and O16 serogroup

Variable		O serotype/ *n*(%)		*p*-value
		O25 (*n* = 20; %)	O16 (*n* = 4; %)	
Miscellaneous	*pai*	15 (75)	1 (25)	0.058
Adhesions	*sfa*	9 (45)	3(75)	0.284
	pap G I	5 (25)	2 (50)	0.326
	pap G II	0	0	1.000
	pap G III	5 (25)	2 (50)	0.326
	afa	2 (10)	0	0.518
	fimH	20 (100)	4 (100)	1.000
Toxins	*hyl A*	1 (5)	1 (25)	0.196
	cnf	0	0	1.000
Siderophores	*iutA*	12 (60)	2 (50)	0.717
Phylogenetic groups	B2	13 (65)	1 (25)	0.147
	D	0	1 (25)	0.025
	A	3 (15)	0	0.418
	B1	1 (5)	2 (50)	0.015
	C	1 (5)	0	0.655
	E	0	0	1.000
	F	0	0	1.000
	Unknown	2 (10)	0	0.518
Antibiotic resistance genes	Oxa-48	1 (5)	0	0.655
	$bla_{\text{CTX-M}}$	11 (55)	0	0.049
	bla_{TEM}	8 (40)	2 (50)	0.717
	bla_{SHV}	0	0	1.000
	qnrA	0	0	1.000
	qnrB	8 (40)	0	0.129
	qnrS	7 (35)	0	0.169
	Int1	18 (90)	0	< 0.001
	Int2	3 (15)	1 (25)	0.632
	acrA	17 (85)	3 (75)	0.632
	acrB	20 (100)	4 (100)	1.000
	tolC	19 (95)	4 (100)	0.655
	ESBL	6 (30)	1 (25)	0.844

ESBL: Extended-spectrum beta-lactamase

Table 2 Antibiotic resistance profile of uropathogenic *Escherichia coli* isolates based on O25 and O16 serogroup

Antimicrobial category	Antibiotics	O25 *n* = 20; (%)		O16 *n* = 4; (%)		*p*-value
		R	S	*R*	S	
Penicillins + b-lactamase inhibitors	Amoxicillin/Clavulanic	3 (15)	15 (75)	3 (75)	1 (25)	0.113
Antipseudomonal penicillins + b-lactamase inhibitors	Piperacillin-tazobactam	2 (10)	18 (90)	0	4 (100)	0.518
Cephamycins	Cefoxitin	6 (30)	14 (70)	1 (25)	3 (75)	0.844
Extended-spectrum cephalosporins	Ceftazidim	10 (50)	10 (50)	2 (50)	2 (50)	1.000
	Cefepime	4(20)	14 (70)	0	4 (100)	0.220
	Cefixim	9 (45)	11 (55)	2 (50)	2 (50)	0.858
Carbapenem	Imipenem	0	20 (100)	0	4 (100)	1.000
	Meropenem	0	20 (100)	0	4 (100)	1.000
Sulfonamides	Trimethoprim/ sulfamethoxazole	13(65)	7 (35)	3 (75)	1 (25)	0.705
Quinolones	Ciprofloxacin	11 (55)	8 (40)	0	4 (100)	0.035
	Ofloxacin	12 (60)	8 (40)	1(25)	3 (75)	0.209
	Nalidixic acid acid	15 (75)	5 (25)	0	2 (50)	0.651
	Norfloxacin	12 (60)	8 (40)	0	4 (100)	0.032
Nitrofurans	Nitrofurantoin	2 (10)	17 (85)	1 (25)	3 (75)	0.676
Aminoglycosides	Gentamicin	7 (35)	13 (65)	0	4 (100)	0.169
	Amikacin	0	19 (95)	0	4 (100)	0.655

R: Resistant S: Sensitive; n: Number

Virulence factors

In serogroup O25, the most prevalent virulence factors were *fimH* (100%) and *PAI* (75%), whereas *papGII* and *cnf* genes were not detected in any isolates. In serogroup O16, the most prevalent virulence factors were *fimH* (100%) and *sfa* (75%), while *papGII*, *afa*, and *cnf* genes were absent in all isolates. No statistically significant correlation was found between the presence of O16 and O25 genes and virulence factors.

Phylogenetic group distribution

Among the O25 isolates, 65% were classified into phylogenetic group B2 followed by phylogenetic group A (15%). In contrast, 50% of the O16 isolates belonged to phylogenetic group B1.

ERIC-PCR

The ERIC-PCR dendrogram is presented in Fig. 1. The number of bands observed in the electrophoresis of ERIC-PCR products ranged from 5 to 11, with DNA fragment sizes varying between 200 bp and 2 kb. The ERIC-PCR typing results revealed that the 24 UPEC isolates from O25 and O16 serogroups were classified into five distinct cluster based on an 80% cut-off, while one isolate was identified as a singleton. In this method, the most frequent cluster was cluster D, comprising seven isolates, followed by clusters A (5 isolates), E (5 isolates), and B (4 isolates), respectively. The least frequent cluster was cluster C, with only two isolates.

Discussion

The serogroups of UPEC are closely associated with specific virulence factors contributing to the pathogenicity of this bacterium. Previous studies have demonstrated a significant correlation between UPEC serogroups and their associated strains [27]. In the present study, PCR analysis revealed 18% carried the O25 gene, and 3.6% carried the O16 gene. Similarly, in a study conducted by Momtaz et al. [7] on 123 *E. coli* isolates from patients with UTIs, O25 and O16 were identified as the most prevalent serogroups, with frequencies of 26.1% and 10.6%, respectively. Al-Saadi et al., [28] in a study conducted in Iraq, reported an O16 prevalence of 16% among 300 isolates, a higher percentage than observed in our study, likely due to the larger sample size. Conversely, a study by Rashki et al. [29] in Zabol reported an O16 frequency of approximately 4.2% among 100 *E. coli* isolates. A similar study conducted in Iraq documented O25 and O16 serogroup frequencies of 24.4% and 1.1%, respectively [30]. These percentages are lower than those in our study, especially for O16. The observed variations may stem from differences in the UPEC strains isolated or geographical differences. In general, O25 has been identified as the predominant UPEC serogroup in numerous studies. For example, Askari et al. reported that among 120 UPEC isolates, 55.8% carried the O25 gene, while only one isolate carried O16. Another study by Tajbakhsh et al. [31] reported O25 as the most prevalent serogroup, with a frequency of 26.66% among UPEC isolates. Similarly, Dehkordi et al. [32] found that O25 was the most

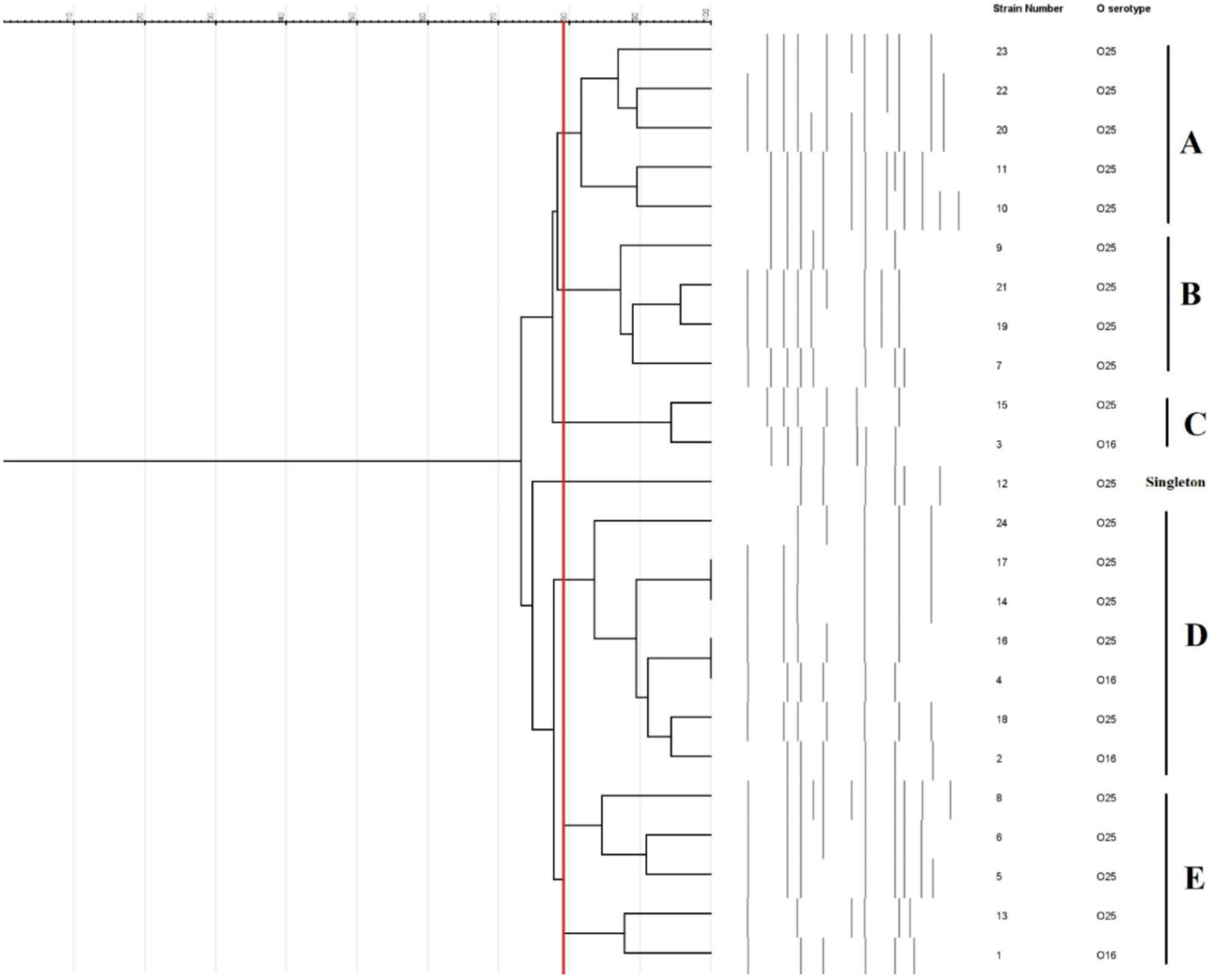

Fig. 1 ERIC-PCR dendrogram of O25 and O16 serogroup uropathogenic *Escherichia coli* isolates. The ERIC-PCR typing results revealed that the 24 UPEC isolates from O25 and O16 serogroups were classified into five distinct cluster (A-E), while one isolate was identified as a singleton

frequent serogroup among UPEC isolates, with a frequency of 29.4%.

The analysis of antibiotic resistance patterns in serogroups O25 and O16 revealed that the highest levels of resistance in both groups were against nalidixic acid, cotrimoxazole, and amoxicillin-clavulanic acid. Conversely, O25 showed the greatest sensitivity to carbapenems and amikacin, whereas all O16 isolates were sensitive to cephalosporins, carbapenems, piperacillin-tazobactam, aminoglycosides, and fluoroquinolones. Our findings align with those of Moradpour et al. in Rasht (northern Iran), where the highest resistance for O25 and O16 serogroups was reported against nalidixic acid and cotrimoxazole [33]. The lowest resistance in O25 was observed against nitrofurantoin and amikacin, while O16 isolates showed minimal resistance to imipenem and nitrofurantoin. Noie Oskouie et al. similarly reported that all UPEC serogroups showed the lowest resistance to imipenem, amikacin, and nitrofurantoin, consistent with our findings [34]. Another study by Jasim Mohammed et al. reported the lowest antibiotic resistance among all serogroups against imipenem and amikacin. The highest resistance was against amoxicillin-clavulanic acid and cotrimoxazole, results closely resembling our findings [30].

In a study by Momtaz et al., antibiotic resistance patterns in different serogroups were examined, and they reported that all O25 and O16 isolates were sensitive to nitrofurantoin and cotrimoxazole [7]. However, in our study, the resistance to cotrimoxazole was considerably higher. Moreover, resistance among O25 and O16 serogroups to most antibiotics was higher in our study, which could be attributed to the increasing prevalence of antibiotic resistance and the spread of resistant strains. In contrast to the present study, Mohammed et al. reported that O8 and O25 were the most frequent serogroups among

the isolates examined. Their analysis of the relationship between serogroups and antibiotic resistance in UPEC strains from Iraqi patients showed that isolates belonging to serogroups O75 and O18 exhibited the lowest resistance, whereas O4 and O21 isolates demonstrated the highest resistance [30].

Our results underscore the urgent need for updated treatment guidelines, especially in regions with high resistance rates, to prioritize the use of more effective antibiotics such as carbapenems, amikacin, and nitrofurantoin. The sensitivity of O16 isolates to cephalosporins, carbapenems, and fluoroquinolones further supports their potential as first-line treatments for infections caused by these serogroups.

On the other hand, *E. coli* isolates were found to harbor antibiotic resistance genes, including $bla_{\text{CTX-M}}$, bla_{TEM}, bla_{SHV}, *qnrB*, *qnrS*, *Int1*, and *Int2*.

The prevalence of these critical antibiotic resistance genes was observed to be higher in O25 strains compared to O16 strains. Research conducted on antibiotic resistance in O25 strains supports our findings, indicating that resistance levels and the presence of associated resistance genes are more prominent in O25 strains [35, 36].

Moreover, studies have shown that *E. coli* O25 and O16 possess a wide array of virulence factors located on plasmids, which are highly transmissible and have achieved global dissemination. Previous investigations have identified O25-B2-ST131 *E. coli*, characterized by resistance genes and significant virulence potential, as a globally distributed strain [37, 38].

The present study aimed to employ the ERIC-PCR technique for the genetic classification of UPEC isolates obtained from UTIs. The ERIC-PCR method was applied for genotyping 24 UPEC isolates belonging to serogroups O25 and O16. Approximately 91.6% (22 out of 24) of the isolates exhibited unique genotypes, indicating relatively high genotypic diversity within serogroups O16 and O25. Additionally, *E. coli* isolates from the same serogroup were distributed across distinct clusters (ERIC types), further highlighting the genotypic diversity among these isolates. Consistent with the findings of Mirzaiyan and colleagues in Iran [39], this study also demonstrated significant diversity among O25 serogroup isolates [40].

Conclusion

Similar to most other studies, the O25 serogroup was found to be highly prevalent in this study. This serogroup is likely to play a significant role in the pathogenesis of UTIs and the antibiotic resistance of uropathogenic *E. coli* strain. Moreover, ERIC-PCR indicating relatively high genotypic diversity within serogroups O16 and O25. These findings emphasize the need for continuous monitoring of antibiotic resistance patterns and the development of targeted therapeutic and preventive strategies. Future research should focus onevaluating their epidemiological trends in different patient populations to inform more effective management of UPEC infections.

Limitations

ERIC-PCR was employed in this study to evaluate genetic diversity. Although ERIC-PCR findings revealed considerable genetic diversity among the isolates, it is evident that more advanced and precise molecular methods, such as MLST (multilocus sequence typing) and PFGE (pulsed-field gel electrophoresis), are required to provide deeper insights into the genetic relationships of these isolates.

Acknowledgements

The authors would like to thank Isfahan University of Medical Sciences for providing the facilities for conducting the research and especially thanks to guidance and advice from Professor Seyed Asghar Havaei.

Author contributions

Conceived and designed the experiments: MH; Performed the experiments: MH, ML, and SAMHD, performed statistical and spatial analyses and interpreted all the results. MH, HQR, and SAMHD, contributed to the writing of the manuscript and revised the final version manuscript: HQR, SAMHD, ML and MH. All authors read and approved the final manuscript. Mehrdad Halaji and Haider Qassim Raheem contributed equally to this work.

Funding

Self-funding.

Data availability

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

Declarations

Ethics approval and consent to participate

This study was confirmed and permitted by the Ethics Committee of Isfahan University of Medical Sciences (IR.MUI.MED.REC.1398.202). These patients routinely referred to the microbiology laboratory and a urine sample was taken and then we only used bacteria isolates obtained from urine culture. A written informed consent was obtained from all participants. In this study, we didn't use samples from minors. The study was performed in accordance with the Declaration of Helsinki. The study used bacteria isolated from clinical samples in the clinical microbiology laboratory.

Competing interests

The authors declare no competing interests.

Received: 3 February 2025 / Accepted: 18 March 2025
Published online: 10 April 2025

References

1. Ferreira VM, Sarcinelli CCS. Alvarenga ads: admissions within the first year after kidney transplantation: evaluation of risk factors, causes, and outcomes. Brazilian J Transplantation. 2024;27:e4724.
2. Laibi H, Jewad A, Salih M. Role of routine markers in the diagnosis of patients with chronic kidney disease. J Biomed Biochem. 2023;2:28–35.
3. Hosseinpour M, Pezeshgi A, Mahdiabadi MZ, Sabzghabaei F, Hajishah H, Mahdavynia S. Prevalence and risk factors of urinary tract infection in kidney recipients: a meta-analysis study. BMC Nephrol. 2023;24:284.
4. Pinchera B, Trucillo E, D'Agostino A, Gentile I. Urinary tract infections in kidney transplant patients: an open Challenge-Update on epidemiology, risk factors and management. Microorganisms. 2024;12.

5. Hemati S, Halimi S, Jabalameli F, Emaneini M, Beigverdi R. Phylogenetic group, antibiotic resistance, virulence gene, and genetic diversity of Escherichia coli causing bloodstream infections in Iran. Front Microbiol. 2024;15:1426510.
6. Salman A. Antibacterial effect of Onion`s infusion and Garlic`s infusion on Escherichia coli isolated from urine samples. J Biomed Biochem. 2022;1.
7. Momtaz H, Karimian A, Madani M, et al. Uropathogenic Escherichia coli in Iran: serogroup distributions, virulence factors and antimicrobial resistance properties. Ann Clin Microbiol Antimicrob. 2013;12:8.
8. Terlizzi ME, Gribaudo G, Maffei ME. UroPathogenic Escherichia coli (UPEC) infections: virulence factors, bladder responses, antibiotic, and Non-antibiotic antimicrobial strategies. Front Microbiol. 2017;8:1566.
9. Biswas S, Bal M, Pati S, Rana R, Dixit S, Ranjit M. Antibiotic resistance in toxigenic E. coli: a severe threat to global health. Discover Med. 2024;1:72.
10. Nasrollahian S, Graham JP, Halaji M. A review of the mechanisms that confer antibiotic resistance in pathotypes of E. coli. Front Cell Infect Microbiol. 2024;14:1387497.
11. Herrera-Espejo S, Rubio A, Ceballos-Romero L, et al. Detection of possible resistance mechanisms in uropathogenic Escherichia coli strains isolated from kidney transplant recipients based on whole genome sequencing. Biomolecules. 2025;15:260.
12. Shafiq M, Zeng M, Permana B, et al. Coexistence of Bla (NDM-5) and tet(X4) in international high-risk Escherichia coli clone ST648 of human origin in China. Front Microbiol. 2022;13:1031688.
13. Eid HM, Algammal AM, Elfeil WK, Youssef FM, Harb SM, Abd-Allah EM. Prevalence, molecular typing, and antimicrobial resistance of bacterial pathogens isolated from ducks. Vet World. 2019;12:677–83.
14. Algammal AM, Wahdan A, Elhaig MM. Potential efficiency of conventional and advanced approaches used to detect Mycobacterium Bovis in cattle. Microb Pathog. 2019;134:103574.
15. Elashkar E, Alfaraj R, El-Borady OM, Amer MM, Algammal AM, El-Demerdash AS. Novel silver nanoparticle-based biomaterials for combating Klebsiella pneumoniae biofilms. Front Microbiol. 2024;15:1507274.
16. Kakian F, Shahini Shams Abadi M, Gholipour A, et al. Evaluating the prevalence of virulence genes of Escherichia coli in patients affected by urinary tract infection. Gene Rep. 2019;16:100433.
17. Zhao X, Lv Y, Adam FEA et al. Comparison of antimicrobial resistance, virulence genes, phylogroups, and biofilm formation of Escherichia coli isolated from intensive farming and Free-Range sheep. Front Microbiol. 2021;12.
18. Halaji M, Shahidi S, Ataei B, Atapour A, Feizi A, Havaei SA. Molecular epidemiology of bla(CTX-M) gene-producing uropathogenic Escherichia coli among Iranian kidney transplant patients: clonal dissemination of CC131 and CC10. Ann Clin Microbiol Antimicrob. 2021;20:65.
19. Halaji M, Shahidi S, Atapour A, Ataei B, Feizi A, Havaei SA. Characterization of Extended-Spectrum β-Lactamase-Producing uropathogenic Escherichia coli among Iranian kidney transplant patients. Infect Drug Resist. 2020;13:1429–37.
20. Wayne P. Clinical and Laboratory Standards Institute: Performance standards for antimicrobial susceptibility testing: 33th informational supplement CLSI document M100-S20 2023.
21. Alhamadani Y, Oudah AS. Study of the bacterial sensitivity to different antibiotics which are isolated from patients with UTI using Kirby- Bauer method. J Biomed Biochem. 2022;1:1–5.
22. Sadeghi A, Halaji M, Fayyazi A, Havaei SA. Characterization of Plasmid-Mediated quinolone resistance and serogroup distributions of uropathogenic Escherichia coli among Iranian kidney transplant patients. Biomed Res Int. 2020;2020:2850183.
23. Clermont O, Christenson JK, Denamur E, Gordon DM. The Clermont Escherichia coli phylo-typing method revisited: improvement of specificity and detection of new phylo-groups. Environ Microbiol Rep. 2013;5:58–65.
24. Sechi LA, Zanetti S, Dupré I, Delogu G, Fadda G. Enterobacterial repetitive intergenic consensus sequences as molecular targets for typing of Mycobacterium tuberculosis strains. J Clin Microbiol. 1998;36:128–32.
25. Sarshar M, Scribano D, Marazzato M, et al. Genetic diversity, phylogroup distribution and virulence gene profile of Pks positive Escherichia coli colonizing human intestinal polyps. Microb Pathog. 2017;112:274–8.
26. Heras J, Domínguez C, Mata E, et al. GelJ–a tool for analyzing DNA fingerprint gel images. BMC Bioinformatics. 2015;16:270.
27. Moradpoor Shamami A, Anvari M, Pourmoshtagh H, Shafighi ST, Seddigh Ebrahim-Saraie H. Serogroup and pathogenicity Island marker distributions among uropathogenic Escherichia coli isolates in Rasht, Iran. Jundishapur J Microbiol. 2023;16:e132754.
28. Al-Sa'ady AT, Mohammad GJ, Hussen BM. Genetic relation and virulence factors of carbapenemase-producing uropathogenic Escherichia coli from urinary tract infections in Iraq. Gene Rep. 2020;21:100911.
29. Rashki A, Rahdar M, Rashki Ghalehnoo Z. Characterization of uropathogenic Escherichia coli: distribution of Adhesin-Encoding genes and O-Serotypes among Ciprofloxacin susceptible and resistant isolates. Jundishapur J Microbiol. 2019;12:e89179.
30. Mohammed EJ, Allami M, Sharifmoghadam MR, Bahreini M. Relationship between antibiotic resistance patterns and O-Serogroups in uropathogenic Escherichia coli strains isolated from Iraqi patients. Jundishapur J Microbiol. 2021;14:e118833.
31. Tajbakhsh E, Ahmadi P, Abedpour-Dehkordi E, Arbab-Soleimani N, Khamesipour F. Biofilm formation, antimicrobial susceptibility, serogroups and virulence genes of uropathogenic E. coli isolated from clinical samples in Iran. Antimicrob Resist Infect Control. 2016;5:11.
32. Safarpoor Dehkordi F, Tavakoli-Far B, Jafariaskari S, et al. Uropathogenic Escherichia coli in the high vaginal swab samples of fertile and infertile women: virulence factors, O-serogroups, and phenotyping and genotyping characterization of antibiotic resistance. New Microbes New Infections. 2020;38:100824.
33. Moradpoor Shamami A, Anvari M, Shafighi ST, Sedigh Ebrahim-Saraie H. Investigation of antibiotic susceptibility pattern and frequency of serogroups O25 and O16 and among uropathogenic Escherichia coli isolates in Rasht City. J Arak Univ Med Sci. 2022;25:3–9.
34. Noie Oskouie A, Hasani A, Ahangarzadeh Rezaee M, Soroush Bar Haghi MH, Hasani A, Soltani E. A Relationship Between O-Serotype, Antibiotic Susceptibility and Biofilm Formation in Uropathogenic Escherichia coli. Microbial drug resistance (Larchmont, NY). 2019;25: 951–958.
35. Anušić M, Marijan T, Mlinarić Džepina A, Tičić V, Gršković L, Vraneš J. A first report on Multidrug-Resistant Escherichia coli O25 ST131 dissemination in an outpatient population in Zagreb, Croatia. Antibiotics. 2025;14:109.
36. Mostaghimi T, Pournajaf A, Bijani A, Mohammadi M, Rajabnia M, Halaji M. Phylogenetic analysis, biofilm formation, antimicrobial resistance and relationship between these characteristics in uropathogenic Escherichia coli. Mol Biol Rep. 2024;51:327.
37. Zhong YM, Liu WE, Meng Q, Li Y. Escherichia coli O25b-ST131 and O16-ST131 causing urinary tract infection in women in Changsha, China: molecular epidemiology and clinical characteristics. Infect Drug Resist. 2019;12:2693–702.
38. Barrios-Villa E, Cortés-Cortés G, Lozano-Zaraín P, et al. Adherent/invasive Escherichia coli (AIEC) isolates from asymptomatic people: new E. coli ST131 O25:H4/H30-Rx virotypes. Ann Clin Microbiol Antimicrob. 2018;17:42.
39. Mirzaeiyan M, Momtaz H, Bamzadeh Z. Typing of uropathogenic Escherichia coli strains isolated from patients with urinary tract infection in Isfahan Province and genetic classification of serogroup O25 isolates by random amplification of polymorphic DNA- polymerase chain reaction method. Navid No. 2018;21:52–62.
40. Montso PK, Bezuidenhout CC, Mienie C, et al. Genetic diversity and whole genome sequence analysis data of multidrug resistant atypical enteropathogenic Escherichia coli O177 strains: an assessment of food safety and public health implications. Int J Food Microbiol. 2022;365:109555.

Publisher's note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.