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Antibiotic Resistance Patterns and Molecular Detection of Class 1 Integron and Some Gene Cassettes of Uropathogenic *Klebsiella pneumoniae*

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Abstract

The emergence of multidrug-resistant *Klebsiella pneumoniae* is a major obstacle in the treatment of urinary tract infections (UTIs), and integron-mediated gene transfer contributes to this problem. This cross-sectional study included *K. pneumoniae* isolates obtained from urine samples of 200 patients diagnosed with UTI. Isolates were identified using culture and biochemical tests, and antimicrobial susceptibility was determined by disc diffusion against 14 antibiotics, including ampicillin, amoxicillin, cefotaxime, ceftriaxone, cephalexin, tetracycline, chloramphenicol, nalidixic acid, trimethoprim, ciprofloxacin, gentamicin, amikacin, and imipenem according to CLSI guidelines. Detection of class 1 integron integrase gene *intI1* and gene cassettes *dfrA5, dfrA7*, and *dfrA25* was performed by polymerase chain reaction using gene-specific primers that amplify target DNA sequences. Multidrug-resistance was observed in 94% of isolates, with high resistance to cephalexin (100%), ampicillin (99%), amoxicillin (99.5%), and cefotaxime (93%), whereas lower resistance was noted to imipenem (17%), gentamicin (21%), amikacin (21.5%), and ciprofloxacin (26%) ($P < 0.05$). PCR analysis showed that 31% of isolates carried the class 1 integron gene, while *dfrA5, dfrA7*, and *dfrA25* were detected in 10%, 17%, and 12% of isolates, respectively, with *dfrA7* being the most frequent cassette associated with integron-positive isolates. These findings indicate a high burden of multidrug-resistant *K. pneumoniae* in UTIs and highlight the role of class 1 integrons in the dissemination of resistance genes, emphasizing the need for continuous molecular surveillance and rational antibiotic use.

Keywords: *K. pneumoniae*, UTIs, Resistance Patterns, Antibiotics, *IntI1* gene

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INTRODUCTION

Urinary tract infection (UTI) is one of the most common bacterial infections in both community and hospital settings.[1] Although *Escherichia coli* accounts for nearly 80% of UTIs, *Klebsiella pneumoniae* is an important secondary pathogen associated with recurrent and complicated infections.[2] The increasing prevalence of antimicrobial-resistant *K. pneumoniae* has made empirical therapy more difficult, posing a serious challenge for clinicians.[3] Excessive and inappropriate antibiotic use, together with horizontal gene transfer mediated by mobile genetic elements such as plasmids, transposons, and integrons, has accelerated the spread of multidrug-resistance among *Enterobacteriaceae*.[4]

Integrons are genetic platforms that capture and express antibiotic resistance genes through site specific recombination. They typically consist of an integrase gene (*intI*), an attachment site, and a promoter that drives expression of integrated gene cassettes.[4,5] Among different integron classes, class 1 integrons are the most prevalent in clinical Gram-negative bacteria and contain variable regions with resistance gene cassettes located between conserved 5' and 3' segments.[6-9] These gene cassettes encode resistance to multiple antibiotic classes, and their expression depends on the integron promoter.[10,11] More than 130 gene cassettes have been described, including members of the *aadA* and *dfrA* families that confer resistance to streptomycin and trimethoprim, respectively.[12,13]

Although integrons and their gene cassettes have been widely reported in *E. coli*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* isolates,[14] information on their distribution in uropathogenic *K. pneumoniae* from Iraq remains limited.[15] Increasing rates of recurrent UTIs and treatment failure in Dhi Qar city highlight the need for local molecular surveillance. Therefore, this study aimed to determine the prevalence of class 1 integrons and selected resistance gene cassettes and to evaluate their association with antibiotic resistance patterns in clinical *K. pneumoniae* isolates from patients with UTIs in northern Dhi Qar, Iraq.

MATERIALS AND METHODS

Sample collection

This cross-sectional study was conducted from December 1, 2023, to March 24, 2024, at Al-Rifai General Hospital, Dhi Qar, Iraq. Urine samples were obtained once from 200 patients clinically suspected of urinary tract infection and confirmed by laboratory diagnosis. Diagnosis was based on compatible clinical symptoms (dysuria, urgency, frequency, fever, chills, or flank pain) together with significant bacteriuria ($\geq 10^5$ CFU/mL of a single predominant organism), considering patient age and clinical context.

Patients receiving antibiotics prescribed by their physicians were included, and individuals with other chronic diseases or concurrent bacterial infections were excluded. Written informed consent was obtained from all participants before sample collection. Midstream clean catch urine samples were collected from toilet trained patients in sterile screw cap containers, whereas urine from infants under two years of age was collected using sterile adhesive urine collection bags.[7]

Identification of bacterial isolates

Urine samples were cultured immediately on blood agar and MacConkey agar plates to isolate *Enterobacteriaceae* and assess lactose fermentation. Plates were incubated aerobically at 37 °C for 24 hrs, and colonies were examined for morphology, pigmentation, and hemolysis. Representative lactose-fermenting colonies suggestive of *K. pneumoniae* were subcultured on nutrient agar to obtain pure isolates. Identification was confirmed using standard biochemical tests in accordance with established microbiological procedures.[2,16]

Antimicrobial susceptibility testing

Antimicrobial susceptibility of confirmed uropathogenic *K. pneumoniae* isolates was determined by the Kirby-Bauer disc diffusion method on Mueller-Hinton agar following Clinical and Laboratory Standards Institute (CLSI) guidelines. Antibiotic discs representing drugs routinely tested for *Enterobacteriaceae* were applied as listed in Table 1, and plates were

Table 1. Primers sequences and PCR annealing temperature of *dfrA5, dfrA7* & *dfrA25*

Genes	Primers sequences (5'-3')	Annealing	Molecular weight
dfrA5	Forward: GCCGGAATTCTAACCCAGGACGAGTAC	56 °C for 30 sec	510 bp
	Reverse: GTCGAATTCTGACCCAGGAACGAGTTTC		
dfrA7	Forward: TCGAATTCTAACCCAGGACGAGTGGGC	54 °C for 35 sec	277 bp
	Reverse: GTCGAATTCTGGGCGCGTTAGTTCCCCG		
dfrA25	Forward: CTGGAATTGGTAACCCAGGAAGCGCAC	50 °C for 35 sec	770 bp
	Reverse: TCGAAATCTGGGTCCAGGAACGAGTGCC		

incubated at 37 °C for 18-24 hrs. Zone diameters were measured and interpreted as susceptible, intermediate, or resistant according to CLSI breakpoints.[2,16]

DNA extraction

Genomic DNA was extracted from confirmed *Klebsiella pneumoniae* isolates using a modified boiling method.[16] Briefly, 300 µL of bacterial suspension adjusted to 0.5 McFarland turbidity was mixed with sterile distilled water in a 1.5 mL microcentrifuge tube, vortexed, and heated at 100 °C for 10 min in a water bath. The lysate was centrifuged at 14,000 × g for 10 min, and the clear supernatant containing genomic DNA was collected and stored at -20 °C until PCR analysis.

PCR for integron gene detection

The presence of the class 1 integron integrase gene *intI1* was detected by polymerase chain reaction using primers F: AGTGGGTGGCGAATGAGTG and R: TGTTCTTGTATCGGCAGGTG.[16] Each 25 µL reaction mixture contained 12.5 µL of 2× PCR Master Mix (Amplicon, Denmark), 0.3 µL of each primer (20 pmol/µL), 2.5 µL of template DNA, and nuclease free water to volume. Amplification was performed

Table 2. Age and gender properties of UTI patients

Character	Data
Age range	1-65 years
Age mean	32.7
Standard deviation	± 11.3
Standard error	0.799
Gender	**N (%)**
Females	162 (81%)*
Males	38 (19%)
Total number	200

*P-value (0.006) compared with males

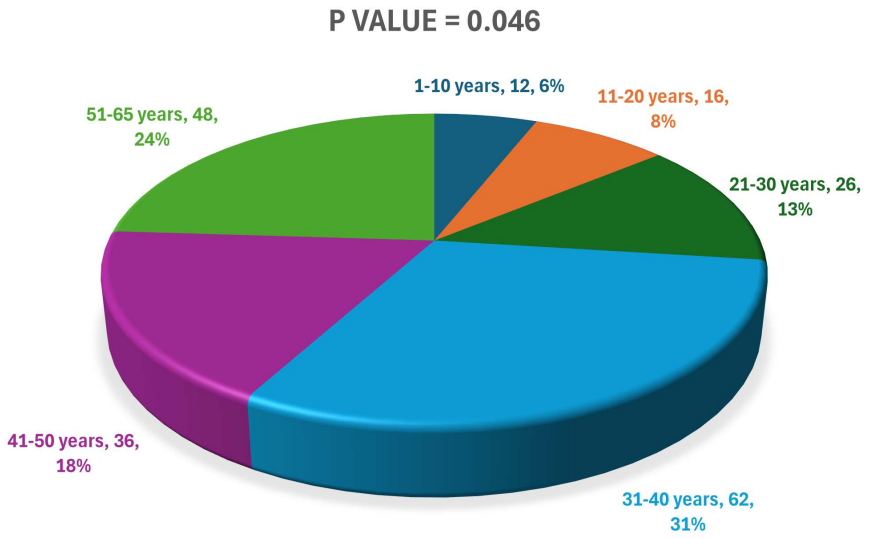

Figure 1. Frequency of *K. pneumoniae* infection according to patients age groups

Table 3. Results of antibiotic sensitivity test of uropathogenic *Klebsiella pneumoniae*

Antibiotic agents	Resistance N (%)	Sensitivity N (%)	χ^2	P-value
Chloramphenicol	72 (36)	128 (64)	7.53	0.012
Tetracycline	180 (90)	20 (10)	13.8	0.0018
Lincomycin	134 (67)	66 (33)	17.76	0.010
Ampicillin	198 (99)	2 (1)	26.1	<0.0001
Amoxicillin	199 (99.5)	1 (0.5)	29.7	<0.0001
Cefotaxime	186 (93)	14 (7)	13.14	0.0001
Ceftriaxone	98 (48)	72 (36)	12.4	0.021
Cephalexin	200 (100)	0 (0)	0.075	0.61
Nalidixic acid	108 (54)	92 (46)	1.08	0.111
Trimethoprim	102 (51)	98 (49)	0.035	0.822
Ciprofloxacin	52 (26)	148 (74)	9.40	0.029
Amikacin	43 (21.5)	158 (78.5)	14.1	0.030
Gentamicin	42 (21)	158 (79)	13.6	0.028
Imipenem	34 (17)	166 (83)	11.94	0.005
MDR	188 (94)	12 (6)	15.85	<0.0001

in a thermal cycler with initial denaturation at 94 °C for 4 min, followed by 35 cycles of denaturation at 94 °C for 40 sec, annealing at 58 °C for 30 sec, and extension at 72 °C for 40 sec, with a final extension at 72 °C for 5 min. PCR products were separated by electrophoresis on a 1.5% agarose gel stained with ethidium bromide and visualised under UV illumination. The presence of an amplicon of expected size confirmed detection of the *intI1* gene.

PCR for gene cassette array detection

Detection of trimethoprim resistance gene cassettes *dfrA5*, *dfrA7*, and *dfrA25* was performed by polymerase chain reaction using gene specific primers designed in this study. PCR assays were carried out using genomic DNA from confirmed *K. pneumoniae* isolates, particularly those positive for the class 1 integron-integrase gene (*intI1*). Reaction mixtures and cycling conditions were identical to those used for *intI1* amplification, except for gene specific annealing temperatures as listed in Table 1. Amplified products were analyzed by agarose gel electrophoresis, and the presence of bands of expected size was considered positive for the respective gene cassette.

Statistical analysis

Data were analyzed using Microsoft Excel 2010 and SPSS version 19. Categorical variables were compared using the chi-square test or Fisher

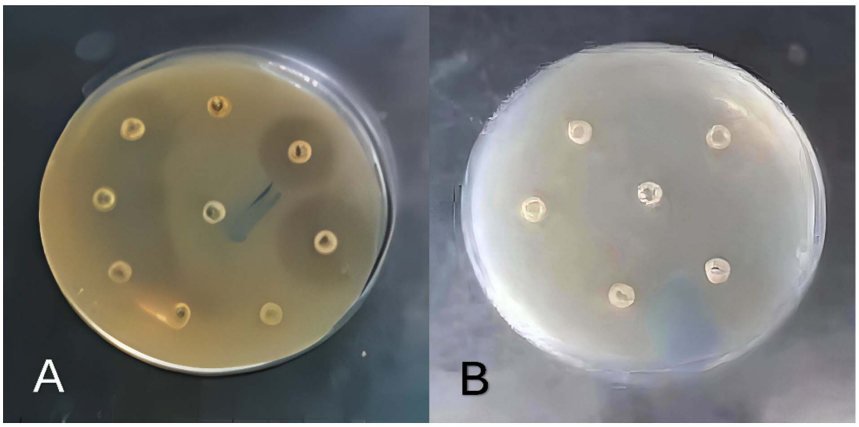

Figure 2. (A) Sensitivity of some isolates to antibiotics; (B) Resistance of some isolates to antibiotics

Table 4. Molecular detection of *IntI1, dfrA5, dfrA7* and *dfrA25* genes

Genes	Positive (%)	Negative (%)	P-value
IntI1	62 (31%)	138 (69%)	0.0251
dfrA5	20 (10%)	180 (90%)	0.0023
dfrA7	34 (17%)	166 (83%)	0.0054
dfrA25	24 (12%)	176 (88%)	0.0031
IntI1-dfrA5	13 (7%)	187 (93%)	0.0019
IntI1-dfrA7	20 (10%)	180 (90%)	0.0023
IntI1-dfrA25	12 (6%)	188 (94%)	<0.0001
IntI1-dfrA5-dfrA7	14 (7.5%)	191 (92.5%)	<0.0021
IntI1-dfrA5-dfrA7-dfrA25	11 (5.5%)	189 (94.5%)	<0.0001
dfrA5-dfrA7	20 (10%)	180 (90%)	0.0023
dfrA5-dfrA7-dfrA25	13 (7%)	187 (93%)	0.0019
dfrA5-dfrA25	12 (6%)	188 (94%)	<0.0001
dfrA7-dfrA25	16 (8%)	184 (92%)	0.0055

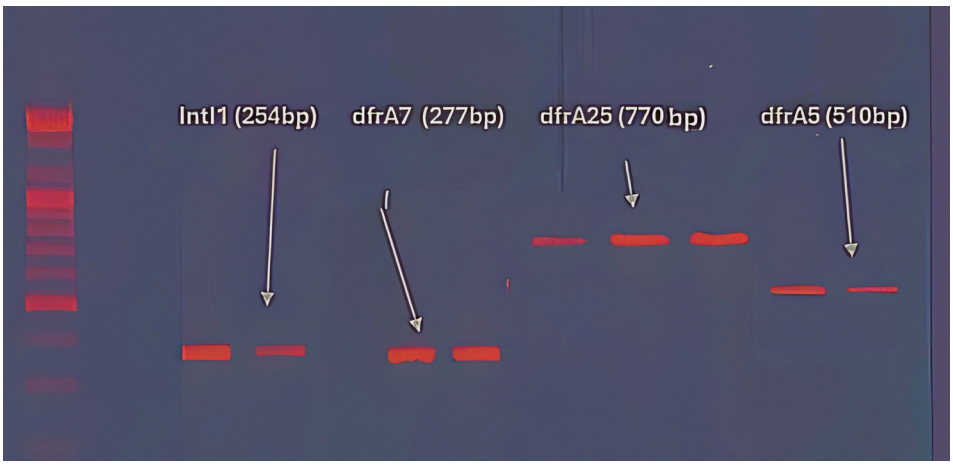

Figure 3. PCR products of primers amplification of *IntI1, dfrA5, dfrA7* and *dfrA25* genes

exact test when appropriate. Continuous variables were analyzed using the Mann-Whitney U test. A P-value less than 0.05 was considered statistically significant.

RESULTS

K. pneumoniae isolates were recovered from urine samples by culture and confirmed by biochemical testing. A total of 200 patients aged 1-65 years were included, with a mean age of 32.7 ± 11.3 years. UTIs were significantly more common in females than males (81% vs 19%, P = 0.006) as shown in Table 2. Age group analysis revealed a higher frequency of infection among patients aged 31-40 years (31%) and 51-65 years (24%), whereas the lowest prevalence was observed in children upto 10 years (6%) (Figure 1).

Antimicrobial susceptibility patterns of *K. pneumoniae* isolates determined by disc diffusion are presented in Figure 2 and Table 3. Significant differences were observed between resistance and susceptibility rates among the tested antibiotics (P < 0.05). High resistance was detected to commonly used antibiotics, particularly cephalexin (100%), ampicillin (99%), amoxicillin (99.5%), and cefotaxime (93%). In contrast, lower resistance rates were observed

Table 5. Distribution antibiotics resistance according to *IntI1, dfrA5, dfrA7* and *dfrA25* genes frequency

Antibiotic agents	Resistance N (%)	Genes				P-value
		IntI1 N (%)	*dfrA5* N (%)	*dfrA7* N (%)	*dfrA25* N (%)	
Chloramph-enicol	72	9 (13%)	1 (1.3%)	3 (4.1)	0 (0%)	0.419
Tetracycline	180	15 (8.3%)	7 (3.8)	7 (3.8)	9 (5%)	0.551
Lincomycin	134	13 (9.7)	3 (2.2%)	9 (7%)	11 (8.2%)	0.472
Ampicillin	198	15 (8%)	4 (2%)	10 (5%)	13 (7%)	0.573
Amoxcillin	199	17 (9%)	5 (3%)	9 (5%)	8 (4%)	0.4444
Cefotaxime	186	6 (3.2)	5 (3%)	9 (5%)	7 (4%)	0.658
Ceftriaxone	98	3 (3%)	0 (0%)	4 (4%)	2 (2%)	0.675
Cephalexin	200	8 (4%)	2 (1%)	8 (4%)	9 (5%)	0.601
Nalidixic acid	108	7 (6.4)	6 (6%)	7 (6.4)	5 (5%)	0.892
Trimethoprim	102	14 (14%)	10 (10%)	11 (11%)	13 (13%)	0.772
Ciprofloxacin	52	4 (8%)	0 (0%)	0 (0%)	2 (4%)	0.104
Amikacin	43	3 (7%)	1 (2.3)	1 (2.3)	6 (14%)	0.096
Gentamicin	42	6 (14.2%)	0 (0%)	3 (7.1)	2 (5%)	0.065
Impenem	34	2 (6%)	1 (3%)	1 (3%)	3 (9%)	0.494

for imipenem (17%), gentamicin (21%), amikacin (21.5%), and ciprofloxacin (26%). Overall, 94% of isolates were classified as multidrug-resistant.

PCR analysis (Figure 3) showed that 31% of *K. pneumoniae* isolates carried the class 1 integron-integrase gene (*intI1*). The trimethoprim resistance gene cassettes *dfrA5*, *dfrA7*, and *dfrA25* were detected at lower frequencies of 10%, 17%, and 12%, respectively (Table 4). Eleven isolates harbored class 1 integrons containing multiple cassette genes (*intI1-dfrA5-dfrA7-dfrA25*), and *dfrA7* was the most frequently detected cassette among integron-positive isolates (20 isolates). Overall, most isolates lacked these cassette genes ($P < 0.05$). Comparison of antibiotic resistance patterns according to gene presence showed no statistically significant differences ($P > 0.05$), although isolates carrying *intI1* tended to exhibit higher resistance rates. No ciprofloxacin resistance was observed among isolates carrying *dfrA5* or *dfrA7*.

Antibiotic resistance pattern analysis is summarized in Table 5. More than 60 different resistance profiles were identified, of which 37 were common patterns. Among isolates carrying the class 1 integron gene *intI1*, the most frequent resistance combinations were AMC-CEP (61 isolates), AMP-AMC-CEP (60 isolates), and AMP-AMC-TRI (60 isolates). Among isolates positive for *dfrA5*, the predominant patterns were AMP-AMC-TRI (14 isolates) and AMP-AMC-CEP (11 isolates). For *dfrA7*-positive isolates, AMC-TET and AMC-CEP were the most common patterns (17 and 16 isolates, respectively), whereas *dfrA25*-positive isolates were mainly associated with AMC-TET and AMP-AMC-CEP resistance profiles (13 and 12 isolates, respectively), as shown in Table 6.

DISCUSSION

The global rise of multidrug-resistant *K. pneumoniae* has made treatment of nosocomial and community-acquired infections increasingly difficult.[17] Mobile genetic elements, particularly integrons, play a major role in the acquisition and dissemination of antimicrobial resistance genes among *Enterobacteriaceae*.[18] In the present study, most isolates showed high resistance to commonly used antibiotics such as cephalexin, ampicillin, amoxicillin, and cefotaxime, whereas lower resistance rates were observed for imipenem, gentamicin, amikacin, and ciprofloxacin. Similar findings have been reported in studies from Iran, Iraq, and India, although resistance rates vary depending on local antibiotic use and infection control practices.[19-21] Misuse and over the counter

Table 6. Antibiotics resistance patterns according to *IntI1, dfrA5, dfrA7* and *dfrA25* genes cassette array frequency

No.	Resistance patterns	*IntI1* (n = 62)	*dfrA5* (n = 20)	*dfrA7* (n = 34)	*dfrA25* (n = 24)
1.	AMC-TET	51	10	17	13
2.	CEP-C	47	3	6	1
3.	LIN-TET	45	4	7	7
4.	AMP-AMC-LIN	44	4	8	5
5.	AMP-AMC-TRI	60	14	9	11
6.	AMC-CEP	61	9	16	9
7.	AMP-AMC-CEP	60	11	12	12
8.	AMC-LIN	44	8	12	3
9.	CEP-AMC-C	32	9	6	5
10.	CEP-LIN	36	10	10	3
11.	CEP-C	47	6	12	9
12.	LIN-C-IME	29	2	0	1
13.	AMP-AMC-CEP-C	51	8	5	6
14.	AMC-CEP-TET-AMP	58	10	7	7
15.	AMP-AMC-C-IME-TRI	32	4	1	2
16.	AMC-CEP-IME-AMP	31	1	4	2
17.	AMC-CEP-GEN-AMP	33	8	2	5
18.	LIN-AMC-CEP-AMP	31	0	3	3
19.	NAL-AMC-CEP-TET	42	7	9	6
20.	AMC-CEP-LIN-AMP,AMI	37	5	1	4
21.	CIPR-AMC-CEP-LIN-GEN	40	0	0	2
22.	AMC-CEP-LIN-C-TET	44	9	10	7
23.	AMC-CEP-LIN-GEN	39	3	5	3
24.	AMC-CEP-C-TET-CIP	41	0	0	2
25.	AMC-CEP-TET-GEN-TRI	37	9	5	9
26.	CIP-NAL-AMC-CEP-GEN	28	4	7	3
27.	NAL-AMC-CEP-LIN-C-TRI	19	3	7	2
28.	AMP-AMC-CEP-LIN-TET-TRI	23	5	2	1
29.	AMC-CEP-LIN-C-TET	19	9	11	6
30.	AMC-CEP-LIN-GEN-AMI	23	6	3	2
31.	AMP-AMC-CEP-LIN-C-TET-AME-TRI	20	2	2	2
32.	AMP-AMC-CEP-LIN-C-GEN	17	5	5	2
33.	AMP-AMC-CEP-C-TET-GEN	22	7	3	4
34.	CIP-NAL-AMP-AMC-CEP-C-TET-AME-CEX	14	0	0	1
35.	AMP-AMC-CEP-LIN-CLR-TET-GEN	10	2	1	3
36.	CIP-NAL-AMP-AMC-CEP-LIN-CLR-C-TRI, CEX-IMP	9	0	0	2
37.	CIP-NAL-AMP-AMC-CEP-LIN-IME-C-TET-TRI-CEF-IMP	5	0	0	3
	Multiple Drug resistance (MDR) 94%	98%	84%	97%	89%

*Abbreviations: Chloramphenicol (C), Tetracycline (TET), Lincomycin (LIN), Ampicillin (AMP), Amoxicillin (AMC), Cefotaxime (CEF), Ceftriaxone (CEX), Cephalexin (CEP), Nalidixic acid (NAL), Trimethoprim (TRI), Ciprofloxacin (CIP), Amikacin (AME), Gentamicin (GEN), Imipenem (IMP)

availability of antibiotics in many regions, including Iraq, may contribute to the rapid selection and spread of resistant strains.

Molecular analysis showed that 31% of isolates carried the class 1 integron gene *intI1*, supporting the role of integrons in the dissemination of multidrug-resistance. This prevalence was lower than reports from northwest Iran and other regions, where higher integron frequencies have been observed, possibly reflecting differences in antimicrobial pressure, patient populations, and infection control practices.[22] Nevertheless, integron positive isolates in our study tended to show higher resistance rates, consistent with

previous reports demonstrating the association between integrons and MDR phenotypes.[22]

Among the resistance gene cassettes examined, *dfrA7* was the most frequently detected, followed by *dfrA25* and *dfrA5*. These genes encode trimethoprim resistant dihydrofolate reductase enzymes and are commonly found within class 1 integrons of *Enterobacteriaceae*.[13] Similar cassette distributions have been reported in studies from China and Iran, although the predominant cassette types vary geographically.[16,23-27] The higher prevalence of *dfrA7* in our isolates may be related to selective pressure from trimethoprim sulfamethoxazole use or to the presence of highly transmissible mobile genetic elements carrying this cassette. Horizontal transfer of integron associated gene cassettes through plasmids and transposons likely contributes to the persistence and spread of these resistance determinants.

We also identified isolates harboring multiple gene cassettes (*intI1-dfrA5-dfrA7-dfrA25*), indicating accumulation of resistance determinants within single integrons. Such cassette arrays have been reported previously and are associated with increased multidrug-resistance.[28,29] However, the presence of a resistance gene does not always correlate with phenotypic resistance, which may result from weak promoter activity, gene mutations, or regulatory mechanisms affecting gene expression.[30]

Overall, the high prevalence of multidrug-resistant *K. pneumoniae* and the detection of class 1 integrons in this study highlight the importance of continuous surveillance of resistance genes and rational antibiotic use. Strengthening antimicrobial stewardship programs and monitoring integron associated resistance determinants are essential strategies to control the spread of resistant uropathogens.[30]

CONCLUSION

This study demonstrated a high prevalence of multidrug-resistant *K. pneumoniae* among urinary tract infection isolates and confirmed the presence of class 1 integrons and associated resistance gene cassettes. Among the cassette genes examined, *dfrA7* was the most frequently detected, highlighting the contribution of integron-mediated gene transfer to trimethoprim resistance and overall antimicrobial resistance in these isolates. The association between integron carriage and elevated resistance rates underscores the importance of monitoring mobile genetic elements in clinical pathogens. Continuous molecular surveillance of integrons and resistance gene cassettes, together with rational antibiotic use and strengthened antimicrobial stewardship, is essential to limit the spread of resistant *K. pneumoniae*. Further research into novel antimicrobial agents and alternative therapeutic strategies is needed to address the growing threat of antimicrobial resistance.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS' CONTRIBUTION

All authors listed have made a substantial, direct and intellectual contribution to the work, and approved it for publication.

FUNDING

None.

DATA AVAILABILITY

All datasets generated or analyzed during this study are included in the manuscript.

ETHICS STATEMENT

Not applicable.

INFORMED CONSENT

Written informed consent was obtained from the participants before enrolling in the study.

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