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Molecular Profile of Aminoglycoside, Fluoroquinolone, and Class 1 Integron Genes among Gentamicin-Resistant *Escherichia coli* in Najaf City, Iraq

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ABSTRACT

Escherichia coli has a major cause of women urinary tract infection, which it harbours various kinds of drug resistance-associated genes. So, the current study examined the prevalence and frequency of genes. These genes are responsible for the resistance of aminoglycoside and fluoroquinolone drugs in uropathogenic gentamicin-resistant *E. coli* isolated from urinary tract infections among women. Six hundred urine specimens were tested. The data revealed 348 (58%) and 70 (11.66%) had gram-positive and gram-negative, respectively. The other 182 (30.33%) were found without any growth. A total of 600 clinical specimens were 167(27.833%) identified as *E.coli* isolate according to biochemical tests and Vitek-2 System. The phenotypic gentamicin-resistant screening (MIC and disk diffusion) revealed out of 167(27.833%) *E.coli* isolates were 25(4.166%) gentamicin-resistance. Antibacterial agents susceptibility of 25 gentamicin-resistant *E.coli* isolates showed concern level of resistance among different categories of antibacterial agents, ranged from high resistance 25(100%) for nalidixic acid to less rate of resistance 4/25(16%) by imipenem drug. Molecular data have demonstrated the prevalence of associated resistance genes for both aminoglycosides and fluoroquinolones. Among 25 gentamicin-resistant *E.coli* isolates 24/25(96%) were harbours for the genes gyr-B, aac(6')-Ib-cr, strA/B, and 23/25(92%) of isolates were harbouring for the genes gyrA, qnrS, and aacC-2. In contrast, qnr-B, aac(6′)/aph(2′), and aph(3)lla were identified in 20/25(80%), 11/25(44%) and 8/25(32%) respectively. At the same respect, aacC-1, qnrA, and qnrC genes were no detect in the current study. However, 24/25(96%) of isolates were carrying the class 1integron (intel-1) gene.

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INTRODUCTION

Urinary tract infection (UTIs) is one of the infectious diseases that have a high frequency and prevalence among different groups of society. Uropathogenic *Escherichia coli* have a significant role in most hospital-acquired infections as well as It is the principal cause of various clinical specimens, especially urine (Abad *et al.*, 2019).

Antimicrobial resistance has been a cause of concern and alarm over the period time worldwide, especially in developed and developing countries (Rather *et al.*, 2017). Aminoglycosides drugs are significant antimicrobial agents for the treating of several infections in humans. At the same respect,

gentamicin has shown marked as perfect curative options for UTIs treatment (Goodlet *et al.*, 2018).

Quinolones drugs are broadly applied to heal UTIs generated via *E. coli*. This expanded application of quinolones becomes led to enhanced resistance in *E. coli*. Target alteration and modifications in the permeability of the cell membrane can give quinolone resistance. Moreover,plasmid-mediated quinolones genes such as qnrA,qnrB, and qnrS can promote resistance to these drugs (Düzgün *et al.*, 2019).

Recently, traditional drug resistances and increase of multi-drug resistance (MDR) organisms in UTIs are related to higher averages of unsuitable empirical medication due to weakening drug covering. *E. coli*, like other gram-negative bacteria, the aminoglycosides resistance usually are mediated mainly through enzyme production; it changes or modifies antibiotics either by adenylation, acetylation or phosphorylation and maybe by efflux pump mechanism (Reygaert, 2018).

MATERIALS AND METHODS

Specimens collection

The contemporary study was associated with 600 non-duplicate women patients undergoing UTIs of both Medical Al-Sader City and Al-Hakim General Hospitals as well as leading clinical laboratories in Najaf City, from January to June of 2019. A clean catching midstream urine specimens collected in a sterile urine container and carried directly to the Advance Medical Bacteria Laboratory, Department of Biology, Faculty of Science, University of Kufa, Iraq.

All specimens were cultured and streaking on different media included blood agar (Oxoid, England), MacConkey agar (Oxoid, England), chromagar orientation (CHROMagar^{TM}, France) till reached a single colony. The petri-dish were incubated under aerobic conditions overnight at 37°C. Moreover, a negative growth incubated for two days. The present investigation depended on specimens that concentration bacteria at least 10^5 colony-forming unit (cfu) per ml. Isolates of *E.coli* were diagnosis based on the characters of microscopic, the colour of Chromagar Orientation media., IMViC tests, motility and oxidase tests (MacFaddin, 2000). Vitek-2 system (bioMèrieux France) used to confirm the diagnosis.

Screening of gentamicin-resistant *E.coli* isolates using minimal inhibitory concentration (MIC) strip and disk diffusion

Phenotypically, all isolates of uropathogenic *E.coli* procedure were investigated and screened. These were to detect gentamycin-resistant *E.coli*. For detecting these *E.coli* isolates, both gentamicin MIC strip (Liofilchem®, MTS, Italy), covering of 0.016-266 μg/mL and gentamicin disk (10μg) (Bioanalyse, Turkey) were used. It was employed on sterile media of Mueller Hinton agar (England) The suspension of all tested isolates were achieved based on 0.5 McFarland standard. The MIC is recorded at the point where the edge of the inhibition ellipse touches with the Strip further the results were described via the instructions guide of the Clinical Laboratory Standards Institute (CLSI, 2018). At the same time, all these achieved at the same moment beside the procedure of disk diffusion; furthermore, all plates were incubated at the same conditions. The strain of *E.coli* ATCC 25922 employed as the negative control.

Antibacterial Agents susceptibility

The present study covered testing susceptibility profile of various commercial classes of antibacterial agents (Bioanalyse, Turkey) against all 25 isolates of gentamycin-resistant *E.coli* according to Kirby-Bauer procedure using sterile media of Mueller Hinton agar (Oxoid, England), (Bauer *et al.*, 1966). The resistance, intermediate and sensitive of isolates were expressed according to guide instructions of the clinical and Laboratory Standards Institute (CLSI, 2018). Disks of antibacterial agents and their concentration marked in the Table 5.

Total DNA extraction

The entire genomic DNA of 25 isolates of gentamycin-resistant *E.coli* was extracted after 24 hours of liquid growth for these pathogens using a kit of the total genomic DNA extraction (iNtRoN, Biotech. Inc., Korea), wherever the extraction was completed according to the instructions of manufacture company. The DNA was saved below -20^{o}C situation employing deep freezing apparatus, till executed PCR to the investigation of gyr-A, gyr-B, aac(6')-Ib -cr, qnrA, qnrB, qnrC, qnrS, aacC-1, aacC-2, strA/B, aac(6')/aph(2'), aph(3)lla and Int-1 genes by specific primers and requirements listed in Table 1 and Table 2. The system of gel document (Cleaver, United Kindom), applied to examine and separate the migration of PCR products using 1% agarose (iNtRoN, Biotech. Inc., Korea), after staining the gel with 0.5 μg/ml ethidium bromide.

RESULTS

Specimens collection and *E.coli* identification

The present study was involved 600 no duplicate urine specimens taken from woman patients suffering from UTI, who attended to main hospitals and clinical laboratories in Najaf city-Iraq, through the

period the time five months (from January to June of 2019). Results in Table 3 were demonstrated that the percentage of the bacterial growth was 348 (58%) and 70(11.66%) to both Gram-negative and gram-positive bacteria, while no bacterial growth was 182(30.33%).

According to microscopic features, culture growth on MacConkey Agar, Chromagar Orientation, biochemical tests and finally all suspected *E. coli* isolates were confirmed using the Vitek-2 system, the data showed the number and percentage of this pathogen reached to 167(27. 833%) isolates.

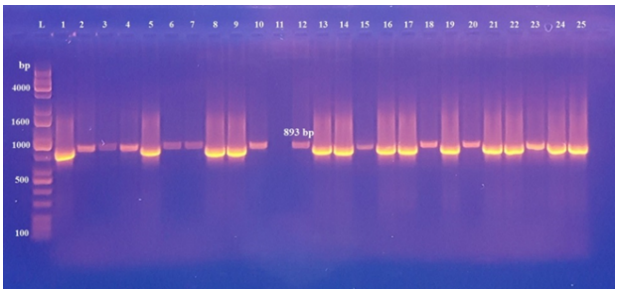

Figure 1: PCR result of StrA/B geneof 25 gentamicin-resistant *E.coli* isolates

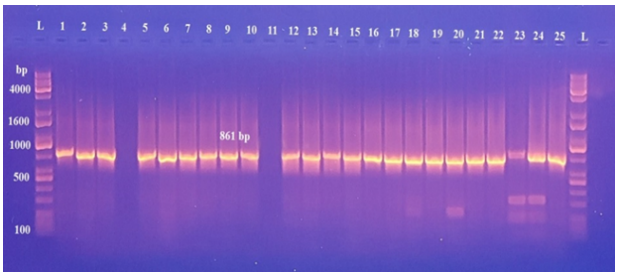

Figure 2: PCR result of aacC-2 gene of 25 gentamicin-resistant *E.coli* isolates

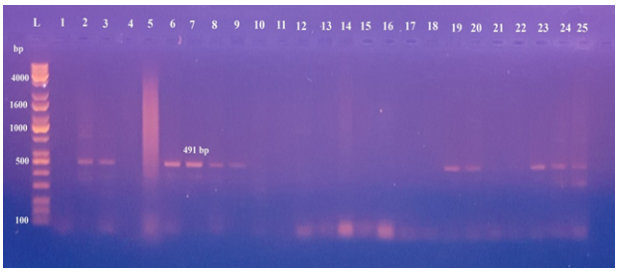

Figure 3: PCR result of aph(6)-(2) gene of 25 gentamicin-resistant *E.coli* isolates

Phenotypic detection of gentamicin-resistant *E.coli* isolates according to disc diffusion method and MIC strip

According to the results of phenotypic gentamicin susceptibility using gentamicin disk and MIC strip revealed thoroughly 167(27. 833%) isolates from uropathogen *E.coli* were 25(4.166%) gentamicin-resistant for both disk gentamicin disc at 10μg con-

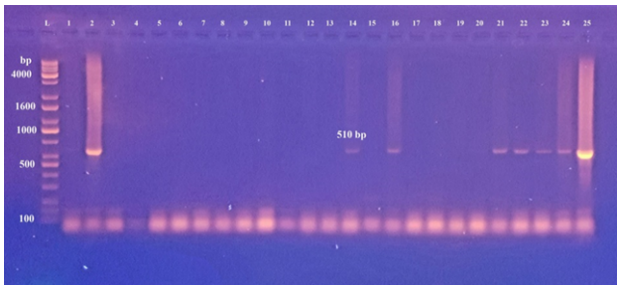

Figure 4: PCR result of aph(3)lla gene of 25 gentamicin-resistant *E.coli* isolates

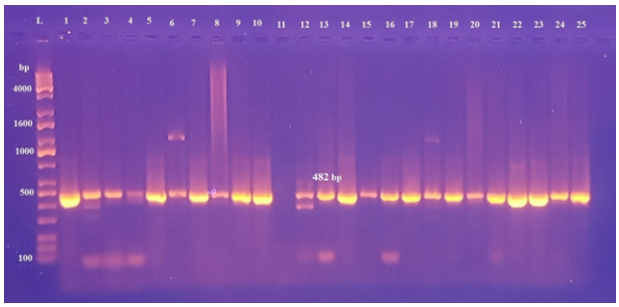

Figure 5: PCR result of aac(6')-Ib-crgene of 25 gentamicin-resistant *E.coli* isolates

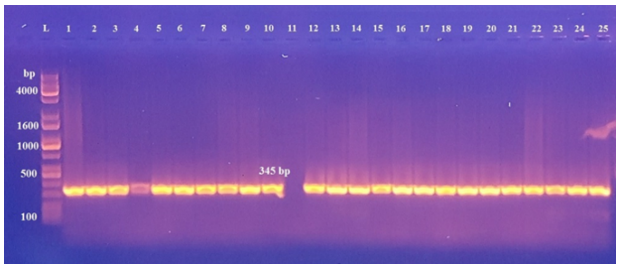

Figure 6: PCR result of gyr-B gene of 25 gentamicin-resistant *E.coli* isolates

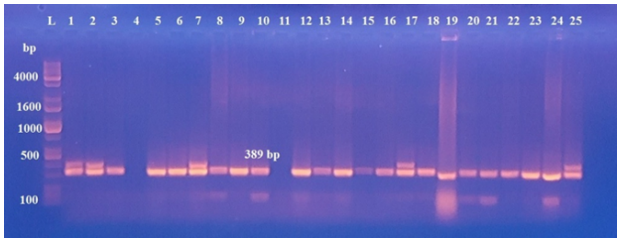

Figure 7: PCR result of gyr-A gene of 25 gentamicin-resistant *E.coli* isolates

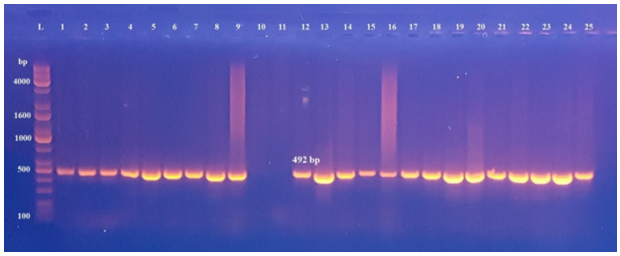

Figure 8: PCR result of qnrS gene of 25 gentamicin-resistant *E.coli* isolates

Table 1: Oligonucleotides of primer used in presentwork

Gene	Name	Sequence (5´to 3´)	Product size (bp)	Reference
gyrA	gyrA-F	ATGGCTGAATTACCTCAATC	398	(Sierra *et al.*, 2002)
	gyrA-R	GTGTGATTTTAGTCATACGC		
gyrB	gyrB-F	CAAACTGGCGGACTGTCAGG	345	(Ling *et al.*, 2003)
	gyrB-R	TTCCGGCATCTGACGATAGA		
aac(6')-Ib -cr	aac(6')-Ib -cr -F	TTGCGATGCTCTATGAGTGGCTA	482	(Kim *et al.*, 2009)
	aac(6')-Ib -cr -R	CTCGAATGCCTGGCGTGTTT		
qnr A	qnr -F	GATAAAGTTTTTCAGCAAGAGG	593	(Jacoby *et al.*, 2003)
	qnr -R	ATCCAGATCCGCAAAGGTTA		
qnr B	qnr B-F	ATGACGCCATTACTGTATAA	560	(Jacoby *et al.*, 2006)
	qnr B-R	GATCGCAATGTGTGAAGTTT		
qnr C	qnr C-F	GGGTTGTACATTTATTGAATC	447	(Wang *et al.*, 2009)
	qnr C-R	TCCACTTTACGAGGTTCT		
qnrS	qnrS-F	ATGGAAACCTACAATCATAC	492	(Afzal *et al.*, 2013)
	qnrS-R	AAAAACACCTCGACTTAAGT		
aacC-1	aacC-1-F	ATGGGCATCATTCGCACATGTAGG	873	(Hujer *et al.*, 2006)
	aacC-1-R	TTAGGTGGCGGTACTTGGGTC		
aacC-2	aacC-2-F	ATGCATACGCGGAAGGCAATAAC	861	(Hujer *et al.*, 2006)
	aacC-2-R	CTAACCGGAAGGCTCGCAAG		
strA/B	strA-F	ATGGTGGACCCTAAAACTCT	893	(Duran *et al.*, 2012)
	strA-R	CGTCTAGGATCGAGACAAAG		
aac(6′)/aph(2′)	aac(6′)/aph(2′)-F	GAAGTACGCAGAAGAGA	491	(Duran *et al.*, 2012)
	aac(6′)/aph(2′)-R	ACATGGCAAGCTCTAGGA		
aph(3)lla	aph(3)lla-F	GAACAAGATGGATTGCACGC	510	(Jaja *et al.*, 2019)
	aph(3)lla-R	GCTCTTCAGCAATATCACGG		
Intl-1	Int1-F	CAGTGGACATAAGCCTGTTC	160	(Xicohtencatl-Cortes *et al.*, 2019)
	Int1-R	CCCGAGGCATAGACTGTA		

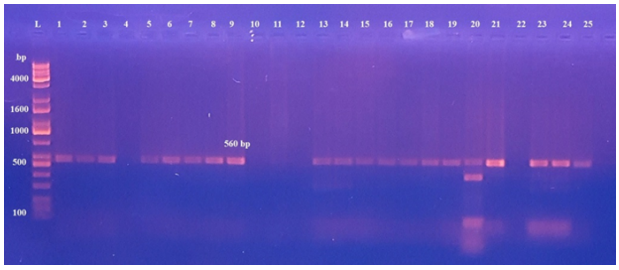

Figure 9: PCR result of qnrB gene of 25 gentamicin-resistant *E.coli* isolates

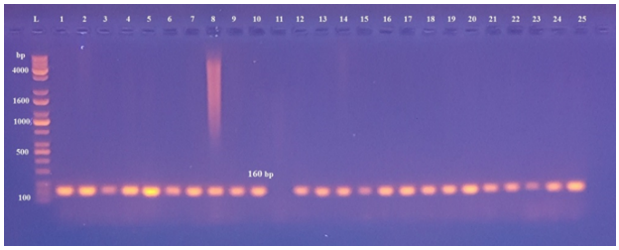

Figure 10: PCR result of Intl-1 gene of 25 gentamicin-resistant *E.coli* isolates

centration and MIC strip with concentration beyond the breakpoint values depending on CLSI (2018). At the same respect, other *E.coli* isolates which gentamicin-sensitive were excluded from the current study (Table 4).

Antibacterial agents susceptibility of gentamicin-resistant *E.coli* isolates

Depending on the results of 20 antibacterial agents susceptibility in the Table 5, most gentamicin-resistant *E.coli* isolates were high resistance against most various antibiotics categories used in the current study. At same respect, data of antibacterial agents susceptibility showed that uropathogenic

Table 2: Conditions of PCR which used in the present study

PCR gene	Temperature (c) / Time					Cycle number
	Initial denaturation	Cycling condition			Final extension	
		Denaturation	Annealing	Extension		
gyr A	94 ºC/5 min	94 ºC/45 sec	50 ºC/45 sec	72 ºC/1 min	72 ºC/5 min	35
gyr B	94 ºC/5 min	94 ºC/1 min	62 ºC/1 min	72 ºC/2 min	72 ºC/1 min	35
aac(6′)-Ib -cr	94 °C/4 min	94 °C/45 sec	55 ºC/45 sec	72 °C/45 sec	72 °C/5 min	35
qnrA	94 °C/5 min	94 °C/1 min	57 °C/40 sec	72 °C/1 min	72 °C/7 min	35
qnrB	95 °C/5 min	94 °C/45 sec	53 ºC/45 sec	72 °C/1 min	72 °C/5 min	35
qnrC	94 °C/5 min	94 °C/40 sec	50 ºC/40 sec	72 °C/40 sec	72 °C/5 min	35
qnrS	94 ºC/5 min	94 ºC/40 sec	55 ºC/40 sec	72 ºC/40 sec	72 ºC/5 min	35
aacC-1	94 ºC/5 min	94 ºC/1 min	55 ºC/1 min	72 ºC/2 min	72 ºC/5 min	35
aacC-2	94 ºC/5 min	94 ºC/1 min	55 ºC/1 min	72 ºC/2 min	72 ºC/5 min	35
strA/B	94 ºC/10 min	94 ºC/1 min	54 ºC/1 min	72 ºC/1 min	72 ºC/10 min	35
aac(6′)/aph (2′)	94 ºC/5 min	94 ºC/1 min	54 ºC/1 min	72 ºC/1 min	72 ºC/7 min	35
aph(3)lla	94 ºC/5 min	94 ºC/30 sec	50 ºC/30 sec	72 ºC/1.5 min	72 ºC/5 min	30
Intel-1	94 ºC/5 min	94 ºC/35 sec	55 ºC/40 sec	72 ºC/35 sec	72 ºC/5 min	35

Table 3: Number and percentage of growth and no growth among 600 urine specimens

Status	Number (%)
Gram-negative	348 (58%)
Gram-positive	70(11.66%)
No growth	182(30.33%)
Total	600 (100%)

Table 4: Phenotypic detection of gentamicin-resistant *E.coli* isolates according to disc diffusion method and MIC strip

Number (%) of total specimens	Number (%) of *E.coli*	Number (%) of gentamicin-resistant *E.coli*	Number(%) of gentamicin-sensitive *E.coli*
600(100%)	167(27.833%)	25(4.166%)	142(23.666%)

Table 5: Antibiogram profile ofgentamicin-resistant *E.coli* isolates

Antibacterial agent	Symbol (μg)	Resistance	Intermediate	Sensitive
Ampicillin	AM (10)	24(96%)	0(0%)	1(4%)
Cefoxitin	FOX(30)	22(88%)	1(4%)	2(8%)
Ceftriaxone	CRO(30)	23(92%)	1(4%)	1(4%)
Aztreonam	ATM(30)	20(80%)	0(0%)	5(20%)
Imipenem	IPM(10)	4(16%)	0(0%)	21(84%)
Amikacin	AK(10)	24(96%)	0(0%)	1(4%)
Netilmicin	NET(10)	17(68%)	2(8%)	6(24%)
Tobramycin	TOB(10)	23(92%)	1(4%)	1(4%)
Streptomycin	S (10)	24(96%)	0(0%)	1(4%)
Ofloxacin	OFX(10)	10(40%)	0(0%)	15(60%)
Norfloxacin	NOR (10)	10(40%)	1(4%)	14(56%)
Nalidixic acid	NA(30)	25(100%)	0(0%)	0(0%)
Ciprofloxacin	CIP (10)	17(68%)	0(0%)	8(32%)
Levofloxacin	LEV (5)	9(36%)	2(8%)	14(56%)
Chloramphenicol	C (30)	20(80%)	1(4%)	4(16%)
Nitrofurantion	F"(300)	23(92%)	0(0%)	2(8%)
Trimethoprin	TMP(5)	24(96%)	0(0%)	1(4%)
Trimethoprin/ Sulphamethoxazole	SXT(25)	21(84%)	2(8%)	2(8%)
Tetracycline	TE(30)	23(92%)	1(4%)	1(4%)
Doxycycline	DO(30)	19(76%)	0(0%)	6(24%)

gentamicin-resistant *E.coli* isolates regarded as multidrug resistance (MDR) through all isolates were resisted to three classes of antibacterial agents and above. However, these pathogens revealed a high effect on beta-lactam drugs (except imipenem) where resistance rates were 24(96%), 23(92%), 22(88%) and 20(80%) to Ampicillin, ceftriaxone, cefoxitin and aztreonam respectively. While imipenem drug was the best in inhibition the bacterial growth, the resistance rate was 4(16%) and this lowest rate among all antibacterial agents which used in current work.

Although all 25 *E.coli* isolates were resistant to gentamicin, the data in the Table 5 proved different resistance rates among aminoglycosides drugs. They were high resistance reached to 24(96%) in both amikacin and streptomycin, 23(92%) in tobramycin, while they were low resistance reached to 10(40%) in both ofloxacin and norfloxacin. At the same time, moderate resistance to netilmicin reached to17(68%).

Results of resistance in fluoroquinolones drugs, *E.coli* were more effect by nalidixic acid 25(100%), while the resistance by levofloxacin was 9(36%) compared with ciprofloxacin 17(68%). Also, 25 gentamicin-resistant *E.coli* isolates were proved high resistance rates among other antimicrobial agents including chloramphenicol, nitrofurantoin, Trimethoprim, Trimethoprim/Sulphamethoxazole, Tetracycline and Doxycycline reached to 20(80%), 23(92%), 24(96%), 21(84%), 23(92%) and 19(76%) respectively.

Molecular assay

Detection of aminoglycoside, fluoroquinolones and Integron class 1 genes

One of the principal aim of this work was to detect the frequency of some aminoglycoside genes, using specific primers. The results of PCR revealed accurate positive bands at variable numbers, for strA/B, aacC-2, aac(6′)/aph(2′) and aph(3)lla genes they were 24/25(96%), 23/25(92%) 11(44%) and 8(32%) respectively. There was no found aacC-1 gene in this work (Figure 1, Figure 2, Figure 3 and Figure 4).

This study also focused on investigation and distribution of genes which were responsible for the resistance of both quinolones and fluoroquinolones drugs among 25 isolates of gentamicin-resistant *E.coli* which isolated from the urine of non-duplicate women. The data in Figure 5and Figure 6 revealed that 24/25(96%) were harbours for the genes gyr-B, and aac(6')-Ib-cr, as well as 23/25(92%) of isolates, were harbours for the genes gyr-A, and qnrS,

(Figure 7 and Figure 8), while qnrB, was identified in 20/25(80%), (Figure 9). At the same respect, qnrA and qnrC genes were no detect in the current study. However, 24/25(96%) of isolates were carrying the intel-1 gene (Figure 10).

DISCUSSION

Specimens collection and *E.coli* identification

UTIs are the common persistent infection in women usually generated by bacteria. The current study appeared *E. coli* isolates remained the most prevalent species isolated from UTIs. Data of this study was following the findings of numerous articles from various sections of the world (Kulkarni *et al.*, 2017).

Domestically, several studies have been conducted on the bacterial causes of urinary tract infections, all of which showed that the frequency of *E. coli* is the highest between gram-negative and positive bacteria. These results were near to a study carries out in Iraq (Erbil) found the rate of *E.coli* isolates was 44.6 % and it is highest amongst other bacteria causes which isolated from a pregnant woman suffering from UTIs (Mohammad *et al.*, 2018).

Antibacterial agents susceptibility of gentamicin-resistant *E.coli* isolates

MDR *E. coli* becomes expanded in the recent years reasonably because of the increasing and wrong application of antimicrobials agents(Kulkarni *et al.*,2017). The susceptibility results are shown in Table 5 revealed high resistance to *E. coli* isolates for different groups of antibiotics, especially those drugs used in UTIs (aminoglycosides, Fluoroquinolones, and sulfonamides drugs), which constitute a source of reduced human health, especially the elderly or those with weak immunity. Therefore, a realistic plan and solutions must be put in place to limit its spread. However, the results of this study are closely related and somewhat compatible with previous local studies (Almamoori *et al*., 2019). The study also showed the sensitivity of bacteria to imipenem. Despite its high efficacy, it is used only in critical illness cases which reduces the exposure of bacteria to this antagonist and thus reduces the chance of a mutation.

Molecular assay

Detection of aminoglycoside, fluoroquinolones and Integron class 1 genes

Aminoglycoside resistance is expanding year after year. It is significant and dangerous rate; this is so, not because of their capability to create chronic diseases but also because they are capable of building resistance to conventional drugs. globally and locally, Found an abundance of articles about resistance to aminoglycosides in *E.coli* isolates. (Chaudhary and Payasi, 2014; Fasugba *et al.*, 2015; Tawfeeq *et al.*, 2017).

Previous articles have done through Ho *et al.* (2010) observes the appearance of aacC-2 gene were a rate of 84.1% and 75.5% from *E.coli* isolated from human and animal, respectively. A further article by Dias-Goncalves *et al.* (2015) manifested about 80% of the gentamicin-resistant *E. coli* isolates were carried out the aacC-2 gene. However, this gene was found and detected in a study that done in Iraq on *E.coli* isolates and other Enterobacteriaceae (Tawfeeq *et al.*, 2017).

Sunde and Norstrom (2005) conducted the study and found the strA/B gene was high frequency among streptomycin-resistant *E.coli* strain. Locally, several reports achieved in Najaf City, Iraq that indicated the frequency of strA/B gene among gram-negative isolated from clinical specimens Locally, many reports delivered in Najaf City, Iraq stated the frequency of aacC-2 gene among gram-negative isolated from clinical and environmental samples (Hayder and Aljanaby, 2019; Tuwaij *et al.*, 2019).

The aac(6′)/aph(2′) and aph(3)lla genes give a high resistance to most agents of aminoglycosides drugs (Chow *et al.*, 2001; Woegerbauer *et al.*, 2015). Results of the current study appeared 11/25(44%) and 8/25(32%) of gentamicin-resistant *E. coli* isolates were carried out the aac(6′)/aph(2′) and aph(3)lla genes respectively. The rate 8/25(32%) of aph(3)lla gene was lower from a study achieved in the same City (Najaf, Iraq) found the frequency of aph(3)lla gene among *E. coli* isolates reach to 90% (Almamoori *et al.*, 2019).

The horizontal transfer of genes by genetic elements like integron or plasmid among microorganisms or chromosomal mutation possesses an essential role in the acquisition of new genes to contribute drug resistance (Düzgün *et al.*, 2019). This may be one of the reasons that explain the vast spreading of fluoroquinolones genes among *E. coli* isolates in the current study.

An earlier article by Abbasi and Ranjbar (2018), data of PCR revealed that among 100 clinical uropathogenic *E.coli* isolates obtained 0%, 25% and 36% for qnrA, qnrB, and qnrS respectively. A study achieved by Ranjbar *et al.* (2018) in Iran found a high frequency of qnrS 92/95 (96.84%)among clinical quinolone-resistant *E.coli* isolates. Data of current work about *the qnrA* gene similar to previous articles, which showed none of this contained qnrA gene (Vaz-Moreira *et al.*, 2016; Conte *et al.*, 2017). However, Among 25 gentamicin-resistant

E.coli isolates 24/25(96%) were harbours for the genes aac(6')-Ib-cr, and this result was congruence with previous local article done by Almamoori *et al.* (2019) showed that 98.3% of clinical uropathogenic *E.coli* isolates contain aac(6')-Ib-cr gene. The primary goal for destroying quinolones drug in isolates of *E.coli* usually by produce DNA gyrase that constituted from two subunits encoding through gyrA and gyrB (Jaktaji and Mohiti, 2010). The results of PCR of current work revealed a high rate of gyrA and gyrB among gentamicin-resistance *E.coli* isolates, however, these data trend to accordance with earlier surveillance (Bhatnagar and Wong, 2019; Hassan *et al.*, 2019).

Integrons can be found inside plasmid or transposons and transport along with them, as well as it promotes the diffusion of antibacterial resistance genes among microorganisms causing dangerous public health effects (Khoramrooz *et al.*, 2016). A high prevalence of intl1in current study may be due to all isolates were clinical and isolated from the patient. A study achieved by Oliveira-Pinto *et al.* (2017) pointed that molecular examination of integrase gene (intl-1) exhibited a higher frequency of class 1 integrons in isolates of uropathogenic *E. coli* reach to 65 % compared with 11.9 % in commensal isolates. However, numerous researches are examining the predominance of integrons in uropathogenic *E. coli* isolates have recorded a notable relationship between antibacterial resistance and integrin. (Khoramrooz *et al.*, 2016).

CONCLUSION

The current research proved that gentamicin-resistant *E.coli* isolated from women infected with UTIs was a significant rate, as well as these isolates, were resistant to multiple commercial antimicrobials agents. In the same respect, imipenem drug was an effect on bacterial growth. A high frequency of aminoglycoside and fluroquinolone genes is a concern in the country.

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