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Molecular and Phenotypic Patterns of Antibiotic-Resistant *E. coli* in Jordan

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Abstract

Antibiotic resistance accounts for over 50% of Escherichia coli (*E. coli*) infections, mediated by extended-spectrum β-lactamases (ESBLs), AmpC β-lactamases, carbapenemase, and other mechanisms. Data on AmpC, carbapenemase, aminoglycosides, and quinolones resistance of *E. coli* in Jordan are minimal. This study aimed to determine the molecular and phenotypic prevalence of antibiotic-resistant *E. coli* in Jordan. Methods: 153 *E. coli* isolates collected from multiple Jordanian hospitals were tested for species identification, antibiotic susceptibility, and resistance genes. Results: 153 *E. coli* isolates were collected with a mean age of 47.09 ± 25.32. Most samples were collected from the emergency department (29.7%) and urine samples were the major source (82.9%). For AmpC detection, 67 (57.8%) samples were resistant to cefoxitin, 13 (23.2%) were positive for AmpC disk test, all tested samples were negative for CMY-1 gene, while 15 (14.4%) samples were positive for CMY-2 gene. Regarding aminoglycoside resistance, 54 (38 %) strains were resistant to gentamycin, 3 (2.1%) were resistant to amikacin, and 94 (61.4%) samples had aac6'-Ib-cr gene. For fluoroquinolones resistance, 92 (65.7 %) isolates were resistant to ciprofloxacin, 65 (47.1%) were resistant to levofloxacin, and 102 (98%) isolates were positive for the gyrA gene. Finally, 3 (2%) isolates were resistant to imipenem and meropenem, however, carbapenemase genes including KPC, OXA-48, IMP, and VIM genes were negative in all samples. Conclusion: Understanding the molecular and phenotypic characteristics of antibiotic-resistant *E. coli* will help to guide proper antibiotic therapy and combat microbial resistance in Jordan.

Keywords: *E. coli*, AmpC beta lactamas, Carbapenemases, Fluoroquinolones, Aminoglycosides, CMY, aac6'-Ib-cr, GyrA.

Abbreviations: Escherichia coli (*E. coli*) , N-acetyltransferases (aacs), Clinical & Laboratory Standards Institute (CLSI), intra-abdominal infection (IAI), Multidrug-resistant (MDR)

INTRODUCTION

Escherichia coli (*E. coli*) is a Gram-negative, facultative anaerobic lactose fermenting bacillus that belongs to the Enterobacteriaceae family. It is the major bacteria that resides in the human large intestine [1]. It can cause a wide range of infections including both intestinal and extraintestinal infections [2] and poses a particularly serious public health problem worldwide affecting different ages and body systems.

The β-lactams, fluoroquinolones, and aminoglycosides are antimicrobial agents used to treat *E. coli* infections [3]. Unfortunately, *E. coli* has developed a variety of mechanisms for resisting antibiotics. *E. coli* resistance is listed as a critical priority pathogen, according to the World Health Organization with millions of deaths associated with *E. coli* resistance in 2019 worldwide [4].

Producing Beta-lactamase enzymes, including AmpC β-lactamases and ccarbapenemase, is the most common resistance mechanism in *E. coli.* AmpC β-lactamases belong to class C in the Amber classification [5]. It confers resistance to Cephalosporins and Cephamycins and is not inhibited by clavulanic acid [6]. It is mostly plasmid mediated through genes including the CMY, FOX, and DHA families. This mechanism of resistance is associated with the failure of multiple antibiotic treatments and contributes to carbapenem resistance with structure mutation [7]. Carbapenem resistance is caused by the production of carbapenemase which is plasmid-mediated through genes including KPC enzyme, OXA-48 type enzymes, metallo VIM, IMP, and NDM metallo-b-lactamases [8].

Fluoroquinolones or β- lactam antimicrobials were the first drugs of choice for treatment [9]. *E. coli* developed different mechanisms for fluoroquinolone resistance including point mutation in DNA gyrase [10].

Aminoglycoside antimicrobials are particularly used for treatment of severe *E. coli* infections [11] and work by blocking protein synthesis in bacteria [12]. The most common mechanism of resistance for these antimicrobials is aminoglycoside-modifying enzymes which are mediated by different classes of genes including N-acetyltransferases (aacs) [13].

The aim of the present study was to characterize phenotypes and genotypes of clinical isolates of *E. coli* in Jordan in order to investigate resistance mechanisms of beta-lactam, aminoglycoside, and fluoroquinolones antibiotics.

MATERIAL AND METHODS

Patients and bacterial isolates

Between 2017 to 2019, a total of 153 non duplicated clinical isolates were collected from two hospitals, including Prince Hamza Hospital and Islamic Hospital in Jordan. All methods were carried out in accordance with relevant guidelines and regulations. Institutional review boards from Hashemite university approved the research as well as the licensing committees in each of Islamic Hospital and Prince Hamza Hospital approved the study. All isolates were collected from patients with suspected *E. coli* infections. Isolates were processed for identification by traditional microbiological procedures including culture on MacConkey agar, Gram stain, and standard biochemical tests. Moreover, a Gram-negative identification card (BioMerieux, France) using the Vitek 2 compact system was carried out to confirm *E. coli* species.

Antibiotic susceptibility tests

Antibiotic susceptibility testing was

performed using the standard disk diffusion method for different classes of antibiotics and zones of inhibition were determined and interpreted according to the last recommendation of Clinical & Laboratory Standards Institute (CLSI).

Screening and confirmation tests for *E. coli* producing AmpC and carbapenemase enzymes

AmpC screening was performed with cefoxitin disk 30 µg [14] while confirmation testing was performed by AmpC disk test [15] while carbapenemase producing stains were confirmed using imipenem 10 µg and meropenem 10 µg disks with or without EDTA (a metallo-carbapemanse inhibitor) performed using the double disk potentiation method. An increase of ≥5 mm of the inhibition zones of EDTA with imipenem or meropenem compared to imipenem and meropenem alone without EDTA was considered positive [16].

Molecular characterization of AmpC, carbapenemase, aminoglycosides and fluoroquinolones resistant genes:

DNA extraction was performed following the procedure recommended by the manufacturer (Qiagen, Hilden, Germany). DNA concentration and purity were checked using Quvet as outlined by the manufacturer. AmpC encoding genes (CMY-1, CMY-2), carbapenemase encoding genes (IMP, VIM, NDM, OXA-48), aminoglycosides encoding gene (aac6'-Ib-cr) and quinolones encoding genes (gyrA, parC) were detected using uniplex polymerase chain using specific and universal primers and protocols described previously that detect different variants of each gene [17-19]. PCR products were electrophoresed and visualized under UV transillumination. The fluoroquinolones resistant samples were sequenced for ParaC.

Statistical analysis

The Statistical analysis used was SPSS version 24. P value less than or equal to 0.5 was considered statistically significant. Descriptive analysis was used to calculate the prevalence of variables, mean, and standard deviation. Chi-square was performed to detect associations between variables.

RESULTS

Demographic characteristics of patients with *E. coli* isolates

153 *E. coli* isolates were predominantly collected from females (75.8%) with ages ranging from 1 month to 100 years (mean 47.09 ± Standard deviation 25.32) from Prince Hamza Hospital (23%) and Islamic Hospital (77%). Most samples were collected from the emergency department. Urine samples were a major source for samples (82.9%) (Table 1).

Table 1: Demographic of patients with *E. coli* isolates (n = 153).

Variable	Category	Number of *E. coli (%)*
Hospital	Islamic Hospital	118 (77%)
	Prince Hamzah Hospital	35 (23%)
Age (years)	≤20	23 (15.8%)
	21 to 40.9	40 (27.4%)
	41 to 60.9	26 (17.8%)
	61 to 80.9	41 (28.1%)
	>80.9	16 (11%)

Variable	Category	Number of *E. coli (%)*
	Not available	7
Gender	Male	37 (24.2%)
	Female	116 (75.8%)
Department	ICU	16 (21.6%)
	Emergency	22 (29.7%)
	Medicine	6 (8.1%)
	Pediatric	13 (17.6%)
	Surgery	7 (9.7%)
	Urology	2 (2.7%)
	Others	8 (10.8%)
	Not available	79
Type of samples	Blood	5 (3.4%)
	Urine	121 (82.9%)
	Sputum	4 (2.7%)
	Wound	7 (4.8%)
	Others	9 (6.2%)
	Not available	7

Phenotypic and molecular tests of *E. coli*-producing AmpC

About 67 (57.8%) samples were resistant to cefoxitin according to the AmpC screening test, and only 13 (23.2%) samples were positive for the AmpC disk test. AmpC genes were tested for the presence of CMY-1 or CMY-2. All tested samples (104 samples) were negative for the presence of the CMY-1 gene, while 13 (23.2%) samples were positive for the CMY-2 gene (Table 2).

Table 2: Phenotypic and molecular tests for detection of *E. coli*-producing AmpC (n = 153).

`		Number of positive (%)	Number of negative (%)	Not available
AmpC screening test	Cefoxitin	67 (57.8%)	49 (42.2%)	37
AmpC disks test		13 (23.2%)	43 (76.8 %)	48
AmpC resistance genes	CMY-1	0 (0%)	104 (100%)	
	CMY-2	15 (14.4%)	89 (85.6%)	1

Phenotypic and molecular tests for detection of aminoglycoside resistance in *E. coli*

In aminoglycoside-resistant isolates, 54 (38 %) of the *E. coli* strains were resistant to gentamycin, while 3 (2.1%) isolates were resistant to amikacin. Aminoglycoside resistance was mediated by the aac6'-Ib-cr gene in 94 samples (61.4%) (Table 3).

Table 3: Phenotypic and molecular tests for detection of aminoglycoside resistance in *E. coli* (n = 153).

`		Number of positive (%)	Number of negative (%)	Number of Intermediate (%)	Not available
Aminoglycosides screening test	Gentamycin	54 (38 %)	86 (60.6%)	2 (1.4%)	11
	Amikacin	3 (2.1%)	138 (95.2%)	4 (2.6%)	8
Aminoglycosides resistance genes	aac6'-Ib-cr gene	94 (61.4%)	59 (38.6%)	-	13

Phenotypic and molecular tests for detection of fluoroquinolone resistance in *E. coli*

It was found that 92 (65.7 %) isolates were resistant to ciprofloxacin, and 65 (47.1%) were resistant to levofloxacin. The gyrA gene was present in 102 (98%) isolates (Table 4).

Table 4: Phenotypic and molecular tests for detection of fluoroquinolones resistance in *E. coli* (n = 153).

`		Number of positive (%)	Number of negative (%)	Number of Intermediate (%)	Not available
Fluoroquinolones screening test	Ciprofloxacin	92 (65.7%)	47 (33.6%)	1 (0.7%)	13
	Levofloxacin	65 (47.1%)	69 (50%)	4 (2.9)	15
Fluoroquinolones resistance genes	GyrA	102 (98%)	4 (2%)		

Phenotypic and molecular tests for detection of carbapenemase resistance in *E. coli*

Most isolates were sensitive to carbapenem antibiotics except three samples that were resistant to imipenem and meropenem. All suspected samples were negative for the presence of carbapenemase genes including KPC, OXA-48, IMP, and VIM genes (Table 5).

Table 5: Phenotypic and molecular tests for detection of carbapenemase resistance in *E. coli* (n = 153).

		Number of positive (%)	Number of negative (%)	Number of Intermediate (%)	Not available
Carbapenemase screening test	Imipenem	3 (2%)	143 (98%)	-	7
	Meropenem	3 (2%)	135 (98%)	-	15
Carbapenemase resistance genes	IMP	0%	100%		
	KPC	0%	100%		
	VIM	0%	100%		
	OXA-48	0%	100%		

DISCUSSION

Multiple studies have indicated the widespread distribution of *E. coli* in Jordan. *E. coli* accounted for 32.4% and 37.4% of all isolates from Jordan University Hospital inpatients and outpatients respectively [20]. It was the most common cause of UTIs in children under 14 years old [21] and community-acquired-UTIs [22,23]. About 70% of UTI isolates and 46% of intra-abdominal infection (IAI) isolates from Jordan and Lebanon [24] and UTI in both diabetic and non-diabetic patients [25] were attributed to *E. coli.* It was also associated with diarrheal infections [20,26-30], neonatal meningitis and sepsis [31] otitis media [32], bacteremia [33,34], serious bacterial infections in the first 90 days of life [35], post-operative wound infections [36] and post-trauma infection in Syrian refugees treated in Jordan [37]. Also, 59% to 65.3% of infant's intestines were colonized with *E. coli* [38,39].

Most studies indicated high rates of resistance of *E. coli* isolates from Jordan to ampicillin, cotrimoxazole, carbenicillin, quinolones, aminoglycosides, amoxicillin/clavulanic acid, third-generation cephalosporins and tetracyclines [20,22,27,40-44]. Multidrug-resistant (MDR) *E. coli* accounted for 59.9% [21], 57.3% [45], and 42% [38] of clinical isolates in Jordan. It was found that 71/89 of *E. coli* isolates from children hospitalized with diarrhea showed resistance to 10 antibiotics or more [46]. Fecal *E. coli* in Jordanian people tested against 14 antimicrobial agents showed 51% of the isolates displayed resistance to more than two antibiotics classes, and 19% displayed resistance to more than three antibiotics classes [40]. MDR accounted for 30.6% of *E. coli* intestinal colonization isolates [38]. A large number of samples were collected by the ARMed project from participating countries in the Mediterranean region which suggests higher resistance rates for *E. coli* in Eastern countries compared to other countries in the Mediterranean area with a significant increase in multi-drug resistance observed in Egypt, Jordan and Morocco over 3 years [41,42].

Only 3 *E. coli* samples (4.8%) produced acquired AmpC enzymes with or without ESBLs [47]. One *E. coli* isolate was a carbapenamase producer and was positive for the NDM gene [48], while one isolate was positive for the KPC-2 gene [45], and one isolate, mostly originating from Jordan was positive for OXA-48 [49]. Fifty representative MDR *E. coli* isolates were positive for fluoroquinolones mutated genes (parC and gyrA) [45]. No colistin- or tigecycline-resistant *E. coli* isolates were detected in Jordan.

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الأنماط الجزيئية والمظهرية لبكتيريا الإشريكية القولونية المقاومة للمضادات الحيوية في الأردن

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الملخص

الخلفية : تمثل مقاومة المضادات الحيوية أكثر من 50% من حالات عدوى الإشريكية القولونية (*E. coli*)، وتتوسطها إنزيمات بيتا لاكتاماز واسعة الطيف(ESBLs) ، وبيتا لاكتاماز من نوع AmpC ، والكاربابينيماز ، وآليات أخرى. البيانات المتعلقة بمقاومة بكتيريا *E. coli* للمضادات من نوعAmpC ، والكاربابينيماز ، والأمينوغليكوزيدات، والكينولونات في الأردن قليلة جداً. تهدف هذه الدراسة إلى تحديد الانتشار الجزيئي والمظهري لبكتيريا *E. coli* المقاومة للمضادات الحيوية في الأردن.

الأساليب : تم اختبار 153 عينة من بكتيريا *E. coli* جُمعت من عدة مستشفيات أردنية لتحديد نوع البكتيريا، وحساسيتها للمضادات الحيوية، وجينات المقاومة لديها.

النتائج : تم جمع 153 عينة من بكتيريا *E. coli* بمتوسط عمر 47.09 ± 25.32 سنة. تم جمع معظم العينات من قسم الطوارئ (29.7%) وكانت عينات البول هي المصدر الرئيسي (82.9%). بالنسبة للكشف عنAmpC ، كانت 67 عينة (57.8%) مقاومة للسيفوكسيتين، و13 عينة (23.2%) كانت إيجابية في اختبار قرصAmpC ، وكانت جميع العينات التي تم اختبارها سلبية لجينCMY-1 ، في حين كانت 15 عينة (14.4%) إيجابية لجين CMY-2. فيما يتعلق بمقاومة الأمينوغليكوزيدات، كانت 54 سلالة (38%) مقاومة للجنتاميسين، و3 سلالات (2.1%) مقاومة للأميكاسين، وكانت 94 عينة (61.4%) تحتوي على جينaac6'-Ib-cr. بالنسبة لمقاومة الفلوروكينولونات، كانت 92 عينة (65.7%) مقاومة للسيبروفلوكساسين، و65 عينة (47.1%) مقاومة لليفوفلوكساسين، وكانت 102 عينة (98%) إيجابية لجين gyrA. أخيراً، كانت 3 عينات (2%) مقاومة للإيميبينيم والميروبينيم، ومع ذلك، كانت جينات الكاربابينيماز بما في ذلك KPC و OXA-48 و IMP و VIMسلبية في جميع العينات.

الخلاصة : إن فهم الخصائص الجزيئية والمظهرية لبكتيريا *E. coli* المقاومة للمضادات الحيوية سيساعد في توجيه العلاج المناسب بالمضادات الحيوية ومكافحة المقاومة الميكروبية في الأردن.

الكلمات الدالة: الإشريكية القولونية، بيتا لاكتامات AmpC، كاربابينيمز، فلوروكينولونات، أمينوغليكوزيدات، CMY، aac6'-Ib-cr، GyrA.