Determine the Prevalence and Fluoroquinolone Resistance Pattern of *Escherichia coli* ST131 among Clinical Isolates from Vaginitis Patients

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Abstract

Background: The most interesting lineage of *Escherichia coli* is the sequence type 131 (ST131), a pandemic clone that is responsible for the high incidence of *E. coli* infections outside the intestine; in addition, it contributes to the global dissemination of multiantibiotic resistance. **Aims:** To determine the prevalence of *E. coli* st131, along with their fluoroquinolone resistance patterns and their phylogenetic background. **Materials and Methods:** A total of 150 high vaginal swabs were taken from female patients with aerobic vaginitis and were screened for the isolation and identification of *E. coli* ST131 using polymerase chain reaction technology that targets ST131-specific sequence polymorphisms in both the *mdh* and *gyr*B genes. An antibiotic sensitivity test was performed to determine resistance patterns. **Results:** Twenty-seven (18%) *E. coli* isolates were obtained from women with aerobic vaginitis, including 11 (40.7%) isolates belonging to sequence type 131. Phylogenetic analysis showed that the majority belonged to phylogroup B_2 (90.9%), and one isolate belonged to phylogroup B_1 (9.09%). All the isolates were resistant to ciprofloxacin and norfloxacin, and levofloxacin, whereas they were all sensitive to gatifloxacin and moxifloxacin. **Conclusions:** The presence of *E. coli* ST131 among *E. coli* isolates from patients with aerobic vaginitis, and the confirmation of their high resistance to most fluoroquinolones included in this study reflect their ability to adapt to different sites, which increases their risk as a pathogen.

Keywords: Fluoroquinolones, phylogenetic, ST131, vaginitis

Introduction

Extraintestinal infections caused by *Escherichia coli* lead to massive morbidity and mortality and increase the cost of health care. The *E. coli* sequence type ST131 recently represents the predominant extraintestinal pathogen and belongs to the most virulent phylogenetic group B2, and the majority of its strains are serologically typed as O25:H4, specifically O25b.[1,2] All *E. coli* sequence type131 isolates have high allelic diversity of the *fim*H gene; the most predominant one is *fim*H30, which includes two important sublineages, H30-R and H30-Rx.[2] in 2008 *E. coli* ST131 was identified in Asia, Europe, and North America as an important pathogen causing various infections in humans[2,3]; it is becoming the predominant extraintestinal pathogenic *E. coli* clone which drives multidrug resistance that rapidly emerged globally, which led to complicated their management, specifically, fluoroquinolone and trimethoprim–sulfamethoxazole resistance limit outpatient treatment options, whereas extended-spectrum cephalosporin resistance limits treatment options in the hospital. This causes delays in appropriate therapy, higher costs, and increased use of "last resort" antimicrobials (e.g., carbapenems).[1,3] However, high-level fluoroquinolone resistance usually involves multiple point mutations within the "quinolone resistance-determining regions" of *gyr*A (DNA gyrase)

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DOI: 10.4103/MJBL.MJBL_414_23

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Submission: 10-Apr-2023 **Accepted:** 04-Jun-2023 **Published:** 09-May-2024

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How to cite this article: Nasser NI, Abdul-Razzaq MS, Al-Hili NM. Determine the prevalence and fluoroquinolone resistance pattern of *Escherichia coli* ST131 among clinical isolates from vaginitis patients. Med J Babylon 2024;21:8-14.

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and *par*C (topoisomerase), which encode the targets of fluoroquinolones. These mutations arise spontaneously and are vertically transmitted.[3] This study aimed to investigate the prevalence of ST131 and fluoroquinolone resistance profiles in a collection of 150 high vaginal swabs from clinical *E. coli* isolates collected at the Maternity and Children Hospital in Babil Province.

Materials and Methods

Study design and bacterial isolates

Clinical examinations of 150 female patients with aerobic vaginitis admitted to Babylon Teaching Hospital for Women and Children during a period of three months (from July 2021 to September 2021) were performed by clinicians, and the high vaginal and cervical swab were collected by using sterile swabs and then inoculated on, MacConkey agar (Himedia/India), blood agar (Himedia/India), and Eosin methylene blue (Himedia/India). All cultures were incubated for 24 h at 37°C, colony morphology and hemolytic reactions were checked for the precharacterization of *E. coli* pure strains after the incubations; the definitive identifications of these strains were performed by using polymerase chain reaction (PCR) technique for detection of 16S rDNA gene for *E. coli*, The forward primers 5′-GATTAGATACCCTGGTAG-3′ and reverse primer 5′-AGTCACTTAACCATACAACCC-3′. According to Magray *et al.*,[4] the reaction mixture and reaction steps for PCR were accomplished.

Inclusion criteria of patients

The female patients included in this study were subjected to clinical and laboratory examinations after sample collection. The clinical sign of aerobic vaginitis was a red vaginal wall, small erosions or ulcerations may be present, itching, yellowish discharge relatively thick, and mucoid. However, the laboratory diagnosis included deficiency in lactobacilli, presence of cocci or coarse bacilli bacteria, presence of parabasal or immature epithelial cells, vaginal leucocytes (plus their granular aspect), and increased pH.[5]

Screening of vaginal samples for *E. coli* ST131 clone

Initially, the *E. coli* isolates were subjected to phylogenetic analysis to determine the four main phylogenetic groups (A, B1, B2, and D) based on the presence of *chu*A, *yja*A, the gene, and *TspE4*.C2 DNA fragment, the primers used for the detection of *chu*A were forward (5′-ATGGTACCGGACGAACCAAC-3) and reverse (3′-TGCCGCCACTACCAAAGACA-5′), *yja*A forward (5′-CAAACGTGAAGTGTCAGGAG-3′) and reverse (3′-AATGCGTTCCTCAACCTGTG-5′), and *TspE4*. C2 forward (5′-CACTATTCGTAAGGTCATCC-3′) and reverse (3′-AGTTTATCGCTGCGGGTCGC-5′), PCR conditions described previously.[6] The B_2 isolates were subjected to further typing of ST131 clones by PCR, based on the presence of ST131-specific single nucleotide polymorphisms in both *mdh* and *gyr*B genes, The primers used for the detection of *mdh* single nucleotide polymorphisms (SNPs) were mdh36_forward (5-GTTTAACGTTAACGCCGGT-3) and mdh36_reverse (5-GGTAACACCAGAGTGACCA-3), and the primers used for the detection of *gyr*B SNPs were gyrB47_forward (5-CGCGATAAGCGCGAC-3) and gyrB47_reverse (5-ACCGTCTTTTTCGGTGGAA-3). Amplification was performed according to a previously described method.[7] All *E. coli* isolates were tested using the disc diffusion method to determine their susceptibility to fluoroquinolone antibiotics, including ciprofloxacin (10 mg), norfloxacin (10 mg), levofloxacin (10 mg), gatifloxacin (3 mg), and moxifloxacin (5 mg), according to the Clinical and Laboratory Standards Institute (CLSI; M02-A10).[8]

Results

Among 150 women patients that participated in the current study, the high vaginal swab samples obtained from them showed the presence of aerobic bacteria in 58 (38.6%) of cultures, 27 (18%) of them gave a positive result in culture for *E. coli* and confirmed the result by using 16s rDNA, while the rest of 31 (20.6%) of the samples yielded positive results for other Gram-negative and Gram-positive bacteria and 92 (61.3%) of samples gave negative results in culture, as mentioned in Figures 1 and 2.

All isolates of *E. coli* were analyzed to determine the phylogenetic groups using a triplicate PCR test based on the presence or absence of (*chu*A, *yja*A, and *TspE4*. C2). The results revealed that the majority of isolates belonged to B_2 phylogroup 21 (77.7%), which included three (11.1%) belonging to subgroup B_{22}, and 18 (66.6%) belonging to the B_{23} subgroup, while the rest of the isolates were distributed among the phylogroup A subgroup A_0 3 (11.1%) and phylogroup B_1 3 (11.1%), and the absence of phylogenetic subgroups (A_1, D_1, and D_2) in the vaginal samples, as shown in Figures 3 and 4.

Overall, 27 *E. coli* isolates were screened to determine the prevalence of ST131 in the high vaginal samples by using the PCR technique to determine CNP in the mdh (mdh36) and gyr (gyrB47), as shown in Figure 5, indicating that the ST131 prevalence rate was 11 (40.7%) among the clinical

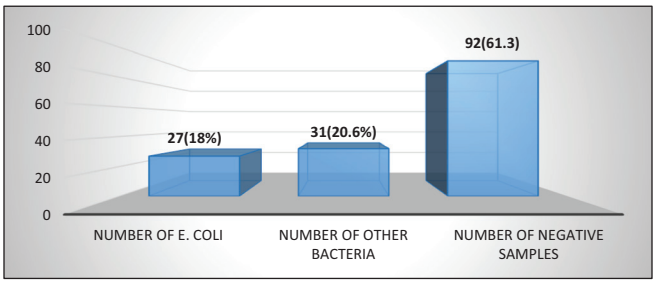

Figure 1: Prevalence of clinical isolates of *E. coli* among collected samples

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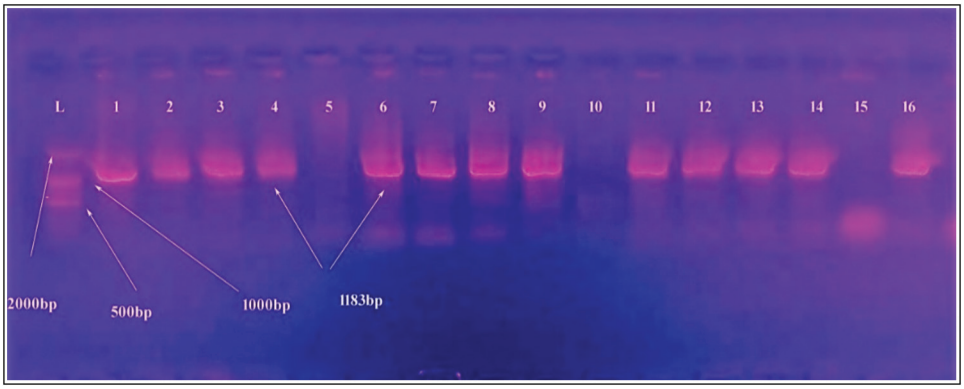

Figure 2: Gel electrophoresis of PCR product (1183 bp) for *Escherichia coli* 1.8% of ethidium bromide-stained agarose gel. Lane (L) 1 kb plus DNA ladder other lanes polymerase chain reaction product for 16S rDNA genes

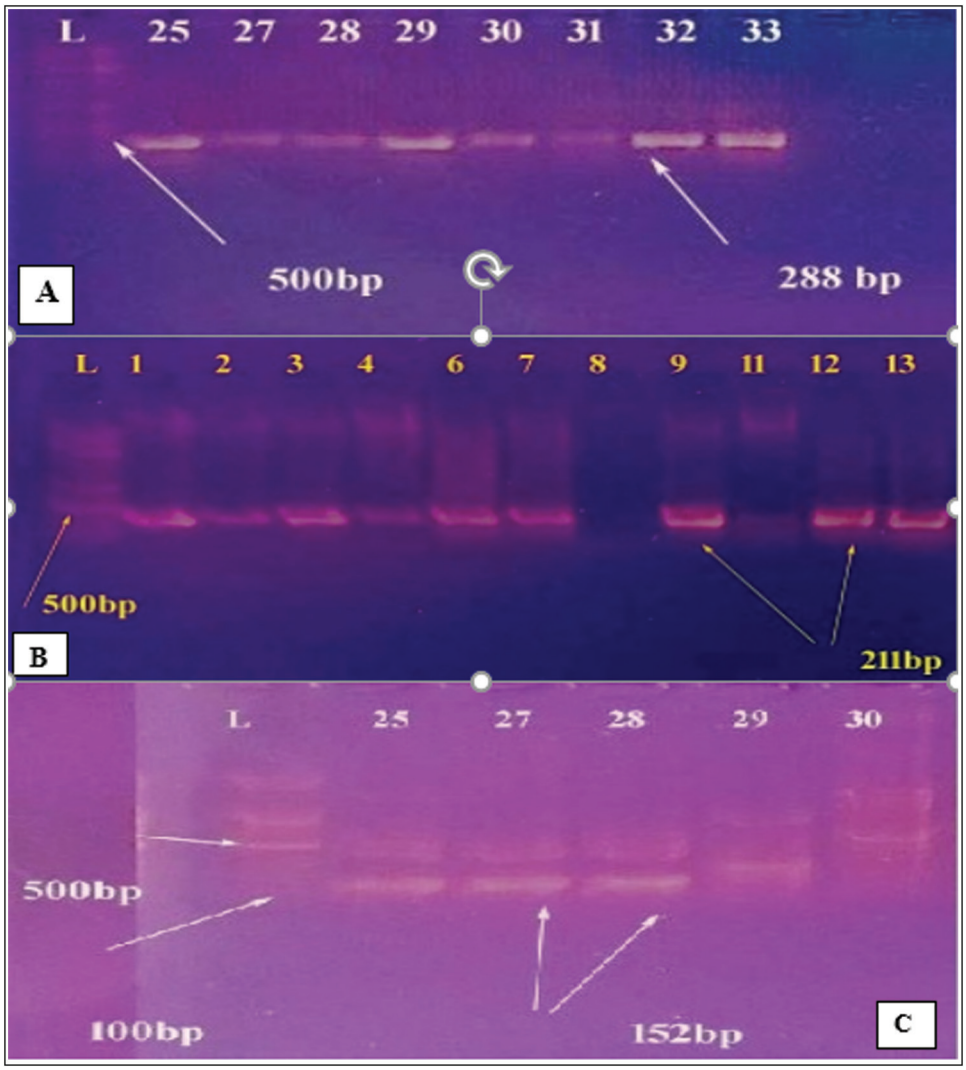

Figure 3: Gel electrophoresis of polymerase chain reaction (PCR) product for (A) chuA (288 bp), (B) yjaA (211 bp), (C) TspE4C2 (152 bp) for *Escherichia coli* 1.8% of ethidium bromide-stained agarose gel. Lane (L) 1 kb plus DNA ladder other lanes PCR product for target genes

isolates of *E. coli*, as shown in Table 1. Most of them belonged to the phylogenetic subgroup B_{23} 10 (90.9%), while one isolate belonged to phylogenetic group B_1 1 (9.01%).

For all 27 bacterial isolates, an antimicrobial susceptibility test was performed as per CLSI 2019 recommendations and interpretations. All isolates belonging to the genus *E. coli* were subjected to a sensitivity test for fluoroquinolone

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antibiotics according to CLSI 2019 recommendations and interpretations. The results indicated that the majority of the isolates were resistant to ciprofloxacin at a rate of 21 (77.7%). The isolates also showed high resistance to both levofloxacin and norfloxacin, but at a lower rate than ciprofloxacin 19 (70.3%); however, gatifloxacin and moxifloxacin antibiotics showed the lowest resistance rate (7.4%). For the isolates of *E. coli* ST131, the percentage of resistance to ciprofloxacin, levofloxacin, and norfloxacin was 11 (100%), while all isolates were sensitive to gatifloxacin and moxifloxacin 11 (100%), as listed in Table 2.

Dissection

Vaginitis is a serious problem and a source of real nuisance and concern for women because most women have suffered from this disease at some point in their lives, the data on bacterial causes and risk factors vary from one study to another from one place to another worldwide, and there is no apparent reason for that. This study focused on determining the prevalence of one type of bacteria, *E. coli*, as a cause of vaginitis in women. A total of 27 (18%) of the 150 high vaginal swab samples. This result is similar to what was mentioned in other research, which reported (16.8%),[9] while it was higher than the percentages mentioned in the study of[10,11] which represent (2.2%, 6.29%), but lower than the result of[12-14] which indicated that the prevalence of *E. coli* was (29.9%, 20.4%, and 25.8%), respectively. The vaginal flora of women of reproductive age is dominated by a few lactobacilli species. These lactobacilli maintain the acidic PH of the vagina by producing lactic acid, hydrogen peroxide, and bacteriocin, which inhibit the growth of potential pathogens.[15] However, low

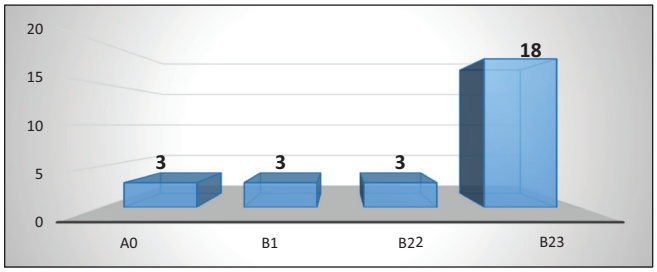

Figure 4: The distribution of phylogenetic groups among *E. coli* isolates depends on the presence or absence of gene *chu*A, *yja*A& *TspE4*.C2

Table 1: The prevalence of *E. coli* sequence type 131 among *E. coli* isolates

E. coli	St131		Non-St131		
No	11		16		
%	40.7%		59.2%		
Polygenetic groups	B1	B2	A	B1	B2
No	1	10	3	2	11
%	9%	90.9%	18.%	12.5%	6.7%

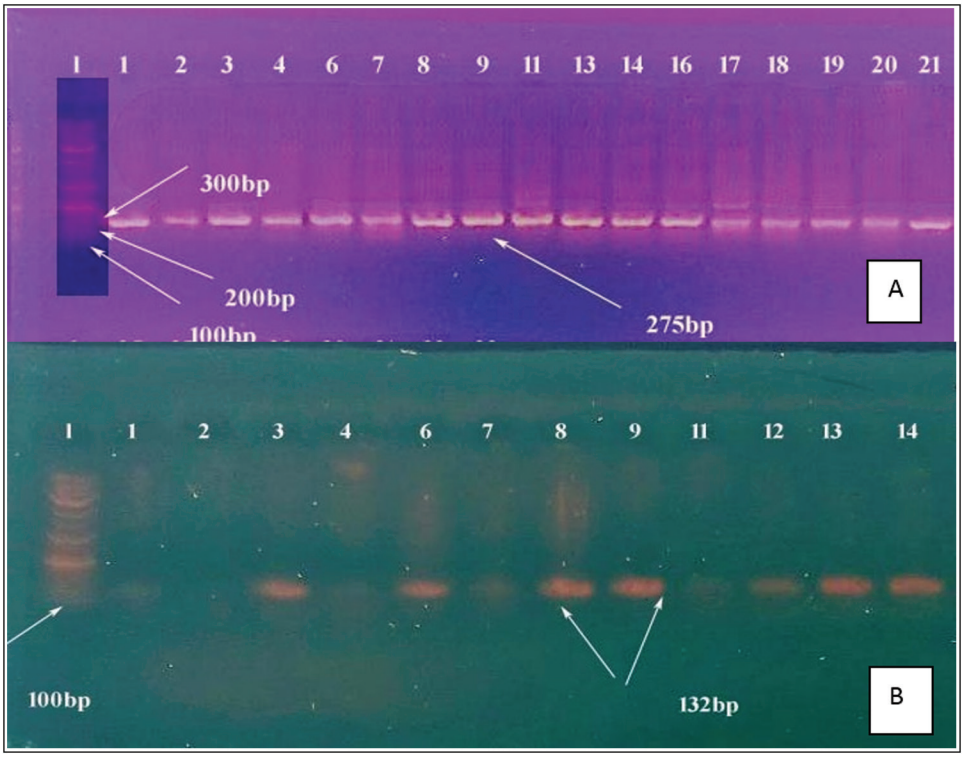

Figure 5: Gel electrophoresis of polymerase chain reaction (PCR) product for (A) mdh36 (275 bp), (B) gyrB47 (132 bp), for *Escherichia coli* ST131 1.8% of ethidium bromide-stained agarose gel. Lane (L) 1 kb plus DNA ladder other lanes PCR product for target genes

Table 2: The prevalence of fluoroquinolones resistance among *E. coli* ST131 and *E. coli* non-ST131

Antibiotic		Ciprofloxacin		Norfloxacin		Levofloxacin		Gatifloxacin		Moxifloxacin	
Concentration		10 mg		10 mg		10 mg		3 mg		5 mg	
Interpretations		R	S	R	S	R	S	R	S	R	S
E. coli isolates	ST131 *n* = (11)	11 (100%)	**0**	11 (100%)	**0**	11 (100%)	**0**	**0**	11 (100%)	11 (100%)	11 (100%)
	Non-ST132 *n* = (16)	10 (62.5%)	6 (37.5%)	8 (50%)	8 (50%)	8 (50%)	8 (50%)	2 (12.5%)	14 (87.5%)	2 (12.5%)	14 (87.5%)
	Total *n* = (27)	21 (77.7%)	6 (22.2%)	19 (70.3%)	8 (29.6%)	19 (70.3%)	8 (29.6%)	2 (7.40%)	25 (92.5%)	2 (7.40%)	25 (92.5%)

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levels of lactobacilli in the vagina or the presence of a mixed microbiome may affect the balance of the vaginal ecosystem, making it vulnerable to infection by opportunistic bacteria such as *E. coli*, which is often described as a series of colonization events, starting with the alimentary canal, followed by entrance to the vagina, which can establish a vaginitis infection and may progress to the urethral meatus and then to the bladder, possibly the kidneys.[16] This progress is reasonable given the proximity of the urethra to the vaginal and anal openings in females. Longitudinal examinations have indicated that the colonization of the intestines and vagina by these bacteria may be the reason for recurrent urinary tract infections.[17] the majority of *E. coli* isolates belonged to the phylogenetic group B2, while the rest of the isolates belong to A and B1 phylogenetic groups; this result was in the same line as another study which revealed that B_2 phylogroup was the dominant one (51%, 52%, 66.7%, and 76%), respectively.[18-21] This means that most of the isolates are among the EXPECs, and this explains their ability to colonize the epithelial cells lining the vagina and cause vaginitis because the ExPEC isolates usually belong to phylogenetic group B_2 and, to a lesser extent to group D, which possesses virulence attributes and specific fitness that allows it to grow in other niches outside the intestine and confer an enhanced ability to cause a broad spectrum of disease.[22] the predominance of group B2 among clinical *E. coli* isolates may be attributed to the fact that most virulence factors and antibiotic resistance genes existed jointly within this group, and this could increase survival fitness in the extraintestinal sites, as recognized by many researchers. All *E. coli* isolates were examined to determine the proportion of serial type 131, in addition to their phylogenetic background. The ratio was (40.7%), the majority of isolates belonged to phylogroup B_2, and one isolate was within phylogroup B_1. The results of this study are in contrast with the results of other studies, including research performed on 199 isolates of *E. coli* reported that the sequence type 131 represents 46 (23.1%)[7] that have been identified that 54 ST131 isolates estimated 17% out of all isolates,[23] it has been reported that in 83 (11.6%) of the 700 clinical *E. coli* isolates were identified as ST131,[24] Muller and his contributor revealed that the clone ST131 accounted for 17.5%.[25] While the results of Al-Guranie and Al-Mayahie[3] indicated that out of 112 *E. coli* isolates, 35 (31.25%) were positive for *E. coli* ST131. These contradictory reports reflect the variability in ST131 prevalence according to country or population. One possible interpretation is that the global spread of ST131 relies more on bacterial factors than on host characteristics.[26] This bacterium has become a point of interest worldwide since it was identified in 2008 because of the clinical and public health implications that have been observed from the high prevalence of ST131, which may be due to the ability of this pathogen to cause different and serious or fatal extraintestinal infections.[27,28] It is thought that the possible explanation for ST131's remarkable success as an emerging pathogen, which includes a phylogenetic background in addition to diverse virulence characteristics and their wide antimicrobial resistance capability. The majority of ST131 isolates are from phylogenetic group B2, which is epidemiologically and experimentally associated with extraintestinal infections.[29] There are also other suggestions regarding the global spread of this clone,[30] including the role of bacteriophages and plasmids, which make an important contribution by accessing the genome with new adaptive traits and tools that modify the genome structure, leading to the modification of transcriptional regulation.[31] This resistance plus virulence combination may give ST131 a competitive advantage over other *E. coli*, promoting its clonal expansion and dominance within niches traditionally occupied by less virulent and/or more susceptible clones.[23] Antibiotic resistance is a major global concern. *Escherichia coli* is a bacterial species that has shown advanced patterns of resistance to a wide range of antibiotics, the most important of which is a fluoroquinolone, which was associated with the emergence of 131. In this study, the highest rates of resistance were observed for ciprofloxacin, norfloxacin, and levofloxacin, and the lowest rates of resistance to gatifloxacin and moxifloxacin. In addition, all isolates of *E. coli* ST131 were resistant to ciprofloxacin, norfloxacin, and levofloxacin, which was comparable with the results of several studies worldwide,[32-38] while all the isolates were susceptible to gatifloxacin and moxifloxacin. Since the mechanism of action of fluoroquinolones is the same in all species, we ruled out that the cause of resistance is a mutation in the quinolone-resistance determining region of (gyrA and

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parC) genes, but may be due to the indiscriminate and widespread use of antibiotics without a prescription and for an imperfect period leading to heightened levels of antibiotic resistance and decreased effectiveness. This has resulted in an alarming level of antibiotic resistance in the pathogen.[7,29]

Conclusion

The presence of *E. coli* ST131 among *E. coli* isolates from patients with vaginitis, and the confirmation of their high resistance to most fluoroquinolones included in this study reflect their ability to adapt to different sites, which increases their risk as a pathogen.

Ethical approval

The necessary ethical approval was obtained through verbal consent. This study was approved by the Committee of Publication Ethics at the College of Medicine, Babylon Province, Iraq, under reference no: BMS\0203\016.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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