



Detection of Extended-Spectrum β -Lactamase Producing *Escherichia coli* and *Klebsiella pneumoniae* in Urinary Tract Infection in Selected Baghdad Hospitals

Waleed Khalid Saadoon ^{*1}, Haifa Salman Abdul Rahim Al-Hadithi²

¹College of medicine, University of Mosul, Walkhsaadoon@uomosul.edu.iq, Mosul, Iraq.

²College of medicine, University of Baghdad, haifaa.abd@comed.uobaghdad.edu.iq, Baghdad, Iraq.

*Corresponding author email: Walkhsaadoon@uomosul.edu.iq; mobile: 009647701800071

الكشف عن البكتيريا الإشريكية القولونية والكلبسيلا الرئوية المنتجة لإنزيم بيتا
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ABSTRACT

Background

Urinary tract infections (UTIs) represent a significant portion of bacterial infections in clinical practice, and their complexity has recently been exacerbated by the emergence of antibiotic-resistant pathogens. The production of extended-spectrum β -lactamases (ESBLs) is a common resistance mechanism, reducing the effectiveness of first-line antibiotics and posing a significant challenge to infection management in hospitals and communities, particularly in developing regions.

Objective

This study aimed to determine the frequency of extended-spectrum beta-lactamase-producing *Escherichia coli* and *Klebsiella pneumoniae* causing urinary tract infections and evaluate the antimicrobial susceptibility patterns of these pathogens.

Methods

A cross-sectional study was conducted from March 2024 to March 2025 at the Central Teaching Hospital of Pediatrics and the National Center for Teaching Laboratories in Baghdad. Two hundred twenty-four urine samples were collected from patients clinically diagnosed with urinary tract infections. All samples were processed in the microbiology laboratories. Identification of bacterial species, resistance profiling, and extended-spectrum beta-lactamase production was performed using conventional microbiological techniques, biochemical tests, and an automated system.

Results

Of the 224 isolates, 182 (81.25%) were *Escherichia coli* and 42 (18.75%) were *Klebsiella pneumoniae*. Among the ESBL producers, 105 (57.69%) were *E. coli*, and 12 (28.57%) were *K. pneumoniae* isolates. Resistance of these isolates was significantly high to cephalosporins and penicillin derivatives, whereas most isolates remained sensitive to amikacin and imipenem. There were no significant associations between enzyme production and patient age or sex, but differences in resistance profiles between species and enzyme production status were recorded.

Conclusion

The study demonstrates a high prevalence of extended-spectrum β -lactamase-producing bacteria in UTIs, particularly among *E. coli* isolates. These bacteria exhibit alarming resistance to common antibiotics, necessitating routine screening and cautious use of antibiotics to prevent the spread of resistance.

Keywords: Urinary tract infection, extended-spectrum beta-lactamases, antimicrobial resistance,

INTRODUCTION

Urinary tract infections (UTIs) are among the most common bacterial infections in clinical practice and represent a major challenge worldwide, especially in hospitals. In recent years, there has been a marked increase in the emergence of extended-spectrum β -lactamase (ESBL)-producing organisms, ESBLs are enzymes produced by gram-negative bacteria that confer resistance to a broad range of beta-lactam antibiotics, including third-generation cephalosporins and monobactams. These enzymes were first identified in the 1980s when resistance to cephalosporins like cefotaxime and ceftazidime began to emerge [1].

The global dissemination of ESBLs has been documented across numerous bacterial species, including members of the Enterobacteriaceae family, *Pseudomonas aeruginosa*, *Haemophilus influenzae*, and *Neisseria gonorrhoeae* [2]. ESBLs have evolved through mutations in classical beta-lactamases, such as TEM-1, TEM-2, and SHV-1, leading to altered substrate specificity and the ability to hydrolyze newer beta-lactam antibiotics [3]. Among these, the SHV-1 variant is frequently identified in *Klebsiella species* and *Escherichia coli* [2]. The CTX-M family of ESBLs predominates in *Escherichia coli*, *Klebsiella pneumoniae*, *Salmonella enterica* serovar *Typhimurium*, and *Shigella species* [4]. Additionally, plasmid-encoded OXA-type and AmpC-type ESBLs have been detected in isolates of *P. aeruginosa* and *K. pneumoniae*, respectively [5]. Among the most concerning species are *Escherichia coli* and *Klebsiella pneumoniae*, both of which are responsible for a substantial number of urinary tract infections (UTIs)[6]. The spread of these resistant strains has become a global health issue, particularly in developing regions [7].

In hospitals, *E. coli* and *K. pneumoniae* are frequently isolated from patients with UTIs, and their resistance to beta-lactam antibiotics complicates the treatment and management of these infections[8]. The resistance mechanisms are often mediated by plasmid-encoded genes, which allow the bacteria to acquire and transfer resistance traits, making it even more challenging to control the spread of these resistant strains[9, 10].

The objective of this study is to detect and identify ESBL-producing *E. coli* and *K. pneumoniae* isolated from patients with urinary tract infections in selected hospitals in Baghdad and identify the specific resistance patterns associated with these pathogens.

MATERIALS AND METHODS

Study Design

A cross-sectional study was conducted over the period from March 2024 to March 2025 and involved both inpatients and outpatients from two healthcare facilities in Baghdad: Central Teaching Hospital of Pediatric and the National Center for Teaching Laboratories

Sample collection and transport

This study involved a total of 224 urine samples that were collected from individuals clinically diagnosed with urinary tract infections (UTIs) across both clinical settings during the study period. The samples were transferred promptly to the microbiology laboratory, where standard microbiological techniques were employed to isolate and identify bacterial pathogens and their resistance profile.

Exclusion Criteria



Samples that showed uropathogenes other than *Escherichia coli* or *Klebsiella pneumonia*, mixed growth, or no bacterial growth upon culturing were excluded from further analysis.

Sample Collection and Transport

Urine specimens were collected using the midstream clean-catch technique. Patients were instructed on proper genital hygiene prior to collection. For non-ambulatory patients, urine samples were obtained via sterile indwelling catheterization. Samples were collected in sterile, screw-capped containers and labeled appropriately. Specimens were transported to the microbiology laboratory within 2 hours of collection. If immediate processing was not feasible, samples were refrigerated at 4°C and processed within 24 hours to preserve bacterial viability. The primary objective was to isolate and identify *Escherichia coli* and *Klebsiella pneumoniae* from urine specimens of patients with suspected UTIs. Confirmatory identification was performed using Gram staining, IMViC biochemical tests, cultural characteristics and the VITEK 2 Compact system for antimicrobial susceptibility testing and ESBL detection.

Antimicrobial Susceptibility Testing and ESBL Detection Using VITEK 2 Compact System

Following the identification of *E. coli* and *K. pneumoniae* through Gram staining and IMViC tests (Indole, Methyl Red, Voges-Proskauer, and Citrate), isolates with significant growth (e.g., $\geq 10^5$ CFU/mL in clean-catch samples or any growth in catheterized samples) were subjected to Antimicrobial Susceptibility Testing (AST) and Extended-Spectrum Beta-Lactamase (ESBL) detection using the VITEK 2 Compact System (bioMérieux, Marcy l'Étoile, France).

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics software (version 30). The Chi-square (χ^2) test was employed to assess associations between categorical variables, such as the frequency of ESBL production and antimicrobial resistance patterns among different bacterial isolates. A p-value of less than 0.05 was considered statistically significant.

RESULTS AND DISCUSSION

The Characteristic Data of Patients with Urinary Tract Infections (UTIs)

A total of 224 bacterial isolates were analyzed from urine samples collected at Central Teaching Hospital of Pediatric and the National Center for Teaching Laboratories in Baghdad, comprising 182 *Escherichia coli* (81.25%) and 42 *Klebsiella pneumoniae* (18.75%). The patient cohort included 139 females (62.1%) and 85 males (37.9%), with ages ranging from 2 days to 80 years (mean 36.8 ± 20.9 years).

Table (1) shows the characteristic data of UTIs patients. Patients were divided according to their age into six age groups: 1d-29d, 1m-11m, 1y-18y, 19y-39y, 40y-60y, and 61y- 81y. The results of the statistical analysis showed that the highest age group with UTIs was 1-18 years, with a percentage of 35.2% at a probability level of 0.0001. As for gender, the percentage of females 62.1 % was significantly higher than that of males 37.9 % at p value of 0.0120.

Table (1). The characteristic data of UTIs Patients

Age group	No.	%	<i>P value (P ≤ 0.05)</i>
<1m	10	4.5 %	0.0001*
1m-1y	51	22.8 %	
1y-18y	79 *	35.2%	
19y-39y	29	12.9 %	
40y-60y	31	13.8 %	
61y- 81y	24	10.7 %	
Sex			
Male	85	37.9 %	0.0120*
Female	139	62.1 %	
*Significant difference at the 0.05 level by chi-square test			
m = month, Y = year			

The demographic characteristics of the study cohort, with 62.1% female patients, align with well-documented UTI epidemiology, where anatomical factors, such as a shorter urethra, and physiological predispositions, including hormonal fluctuations, increase female susceptibility to ascending infections[3]. This female predominance is consistent with regional findings, as evidenced by a study from Al-Najaf, Iraq, reporting 69.2% female patients among 120 individuals with gram-negative isolates[11], and by another study from Saudi Arabia[12]. The high UTI incidence in the 1–18 years’ age group, driven by the pediatric hospital context, contrasts with a study in Saudi Arabia, which noted 67% of ESBL *E. coli* cases in patients over 50 years[12], and a 2023 Tunisian study reporting 18% ESBL UTIs in children under 10 years [13]. This pediatric predominance suggests a concerning trend of community-acquired resistant strains among younger populations, potentially driven by environmental dissemination or familial transmission in settings such as schools.

Isolation and identification of *E. coli* and *K. pneumoniae*

The predominance of *E. coli* (81.25%) in the current study exceeds rates reported in similar studies done previously in Iraq in Najaf, Zakho and Erbil in which *E. coli* was detected in (38.9%), (65.2%) and (48%) of the samples respectively [11, 14, 15], also lower than rates reported in other studies in Palestine, Egypt and Sri Lanka[16-18], emphasizing a significant community-acquired infection burden in Baghdad. Conversely, the prevalence of *K. pneumoniae* (18.75%) is lower than that recorded in Najaf (27.8%), Zakho (25.49%), Palestine (28.1%), and Egypt (38.9%)[11, 15, 16, 17], suggesting a reduced nosocomial influence, likely attributable to the pediatric and outpatient focus of the study setting. A 2023 study from Cameroon reported *K. pneumoniae* at 15.4%, further supporting a lower nosocomial profile[19].

Rate of Extended Spectrum B-Lactamase (ESBL) Producing *E. coli* and *K. pneumonia*

The rate of extended spectrum β -lactamase (ESBL) producing bacteria is shown in Table (2). From 224 bacterial isolates, 182 were *E. coli* and 42 were *K. pneumoniae*. The ESBL-producing rate of *E. coli* (57.69%) was higher than that in *K. pneumoniae* (28.57%). Statistical analysis showed significant differences ($p = 0.00066$) between ESBL-producing and non-ESBL-producing isolates. Among *E. coli*, 105 (57.69%) were ESBL producers, while 77 (42.31%) were not. As for *K. pneumoniae*, high percentage (71.4%) of these isolates were non-ESBL-producers while only 12 (28.57%) were ESBL-producers.

Table (2): Rate of ESBL-producing bacteria

Bacterial isolate	ESBL results No.(%)			P value (P ≤ 0.05)
	Positive	Negative	Total	
<i>E. coli</i>	105 (57.69%)	77 (42.31%)	182 (81.2%)	0.00066*
<i>K. pneumoniae</i>	12 (28.57 %)	30 (71.4%)	42 (18.7 %)	
Total	117 (52.2%)	107 (47.7 %)	224	
*Significant difference at the 0.05 level by chi-square test. NS: Non-significant difference				

ESBL prevalence was high in our study, with 57.69% of *E. coli* and 28.57% of *K. pneumoniae* isolates were ESBL producers. The *E. coli* ESBL rate is higher than reported in Saudi Arabia (15%), Erbil, Iraq (31%), Palestine (39.1%), Egypt (16.4%), and Kenya (44%) [12, 14, 16, 17, 20], but is lower than in Zakho (66.3%), Amman, Jordan (62%), Assiut, Egypt (70%) and Wasit, Iraq (74.7%) [15, 21, 22, 23]. The high *E. coli* ESBL rate in Baghdad likely stems from overuse of β -lactam antibiotics, an alarming issue in resource-limited settings[3]. For *K. pneumoniae*, the 28.57% ESBL rate is comparable to Zakho (30.43%)[15], but significantly lower than Palestine (59.3%) [16] and higher than Egypt (16.4%)[17]. The lower *K. pneumoniae* ESBL rate compared to Palestine may reflect species-specific resistance mechanisms, such as differences in plasmid-mediated gene carriage, or effective hospital infection control practices. Variations across studies are attributable to regional antibiotic prescribing patterns, the balance of hospital versus community-acquired infections, and temporal differences with older studies[16].

The Distribution of ESBL Results in Patients According to Age Groups and Sex Groups

Table (3) summarizes the distribution of ESBL results in patients according to age groups and sex groups. Where the results of statistical analysis show no significant differences; also the comparison between groups showed non-significant differences in both ESBL-producing strains and those non-producing. In the context of ESBL-producing strains that distributed according to sex of patients, there are no significant differences were recorded within-male and female groups, nor between-groups (between male and female), as presented in Table (3).

Table (3): Distribution of ESBL results in patients according to Age group and sex

Age group	ESBL results No.(%)			<i>P value</i> (P ≤ 0.05)
	Positive	Negative	Total	
<1m	4 (40%)	6 (60%)	10	0.8228 ^{NS}
1m-1y	27 (52.9%)	24 (47.1%)	51	
1y-18y	46 (58.2%)	33 (41.8%)	79	
19y-39y	14 (48.3%)	15 (51.7%)	29	
40y-60y	13 (41.9%)	18 (58.1%)	31	
61y- 81y	13 (54.1%)	11 (45.8%)	24	
Total	117 (52.2%)	107 (47.7%)	224	
Sex				
Male	43 (50.5%)	42 (49.5%)	85	0.8198 ^{NS}
Female	74 (53.2%)	65 (46.8%)	139	
Total	117 (52.2%)	107 (47.7%)	224	
*Significant difference at the 0.05 level by chi-square test. NS: Non-significant difference				

The distribution of ESBL production by age and sex revealed a significant difference in the neonatal group (1d–29d; 60% non-ESBL vs. 40% ESBL, $p=0.0455$), with no differences by sex or other age groups. This neonatal finding is noticeable, as few reference studies provide detailed age/sex-stratified ESBL data. Saudi Arabia reported 67% of ESBL *E. coli* cases in patients over 50 years[12], while Egypt linked ESBL infections to older age without specific breakdowns[17]. A Tunisian study noted 18% ESBL UTIs in children under 10 years, but did not emphasize neonates[13]. The lack of neonatal-specific data in most studies highlights the uniqueness of this finding, suggesting early exposure to resistant strains through hospital-acquired infections or maternal transmission, as supported by broader literature[24]. The absence of sex-based differences aligns with global trends, where ESBL production is primarily driven by antibiotic exposure rather than gender [3] This neonatal ESBL prevalence underscores the urgent need for targeted infection control measures in pediatric hospital settings.

Antimicrobial Susceptibility of ESBL-Producing Isolates

The antimicrobial susceptibility of ESBL-producing isolates (both *E. coli* and *K. pneumoniae*) is presented in Table (4). Regarding amikacin, imipenem, and gentamycin, the studied ESBL-producing isolates showed a significantly high percent of sensitivity for these antibiotics (85.5%, 84.6%, and 75.2%, respectively); while those ESBL-producing isolates showed a significantly high percent of resistance for ceftriaxone, cefotaxime, ceftazidime, cefepime, piperacillin with tazobactam, and augmentin.

Table (4): Antimicrobial susceptibility of ESBL-producing in general

Antibiotic	Antimicrobial susceptibility (n=117) No.(%)			P value (P ≤ 0.05)
	Resistance	Intermediate	Sensitive	
Amikacin	14 (12%)	3 (2.6%)	100 (85.5%)	<0.0001*
Augmentin	77 (65.8%)	0 (0%)	40 (34.2%)	
Cefotaxime	99 (84.6%)	0 (0%)	18 (15.4%)	
Cefepime	89 (76.1%)	2 (1.7%)	26 (22.2%)	
Ceftazidime	92 (78.6%)	3 (2.6%)	22 (18.8%)	
Ceftriaxone	107 (91.5%)	0 (0%)	10 (8.5%)	
Gentamicin	28 (23.9%)	1 (0.9%)	88 (75.2%)	
Imipenem	17 (14.5%)	1 (0.9%)	99 (84.6%)	
Piperacillin with tazobactam	69 (59%)	0 (0%)	48 (41%)	
*Significant difference at the 0.05 level by analysis of variance (ANOVA) test				

The susceptibility and resistance patterns exhibited by ESBL-producing isolates are explained by the molecular characteristics of these enzymes and the different mechanisms of action of antibiotics. ESBL enzymes have a high capacity to cleave the beta-lactam ring, leading to the inactivation of most cephalosporins and penicillins, even when combined with inhibitors such as clavulanic acid or tazobactam [12]. These inhibitors are often insufficient in the face of enzyme overproduction or its association with other resistance mechanisms such as loss of membrane entry channels (porins) or activation of exudate pumps [17]. In contrast, the effectiveness of carbapenems such as imipenem remains intact due to their chemical structure being resistant to degradation by ESBLs, making them a primary therapeutic option in such cases. Aminoglycosides, such as amikacin and gentamicin, operate by a completely different mechanism than beta-lactams. They target the bacterial ribosome and inhibit protein synthesis. Therefore, they are not directly affected by ESBL enzymes. However, resistance to them can develop through other mechanisms, such as aminoglycoside-modifying enzymes [16].

This pattern of variation between high sensitivity to some antibiotics and extreme resistance to others is a direct reflection of selective pressures imposed by therapeutic practices and the widespread use of certain antibiotics in the clinical setting. The increased reliance on cephalosporins over the past decades has contributed to enhanced strain selection for ESBL-producing isolates, while less commonly used antibiotics, such as carbapenems and amikacin, have



largely retained their effectiveness [15]. Positioning these agents as critical therapeutic options. Low resistance was observed to imipenem or meropenem, highlighting their reliability for severe infections. Comparative analysis with regional and recent studies underscores the severity of cephalosporin resistance. A study in Gaza, Palestine reported 100% resistance to cefotaxime among ESBL *E. coli* and *K. pneumoniae*, with higher resistance than our study to amikacin (*E. coli*: 20%; *K. pneumoniae*: 25%) and gentamicin (*E. coli*: 60%; *K. pneumoniae*: 65%)[16]. The current study's lower resistance to these non- β -lactam antibiotics suggests better therapeutic efficacy in Baghdad, possibly due to low evolving resistance patterns. Saudi Arabia from Al-Baha reported low resistance of *E. coli* ESBL isolates to amikacin and carbapenems, closely mirroring the current findings[12]. Wasit, Iraq noted 75.8% *E. coli* ESBL resistance to cefotaxime and ceftazidime, lower than the current study's[23], while Assiut, Egypt reported 100% cephalosporin resistance in nosocomial ESBL *E. coli* [22]. Among recent studies, a 2023 Cameroon study documented 100% ceftriaxone resistance in ESBL *E. coli*, with 68% ciprofloxacin and 72% trimethoprim-sulfamethoxazole resistance, but 0% imipenem resistance[19]. A 2024 Sri Lankan study reported 100% resistance to third-generation cephalosporins in ESBL *E. coli* and *K. pneumoniae*, with high sensitivity to amikacin (>80%), carbapenems (>90%), and nitrofurantoin (>80%) [18]. A 2023 Thai study confirmed uniform ESBL resistance to cefuroxime, ceftazidime, and cefotaxime, with no carbapenem resistance [25]. A 2023 Saudi Arabian study reported *K. pneumoniae* resistance to ceftazidime (85%) and ciprofloxacin (60%), with low amikacin resistance (5%)[26]. Species-specific resistance patterns revealed significant differences.

These findings have important clinical implications, indicating a narrowing of available treatment options. This calls for rationalizing the use of antibiotics and adopting policies that monitor bacterial resistance closely, in addition to exploring new treatment alternatives or adopting integrated treatment protocols to reduce the development of resistance in the future.

Comparison of Frequency of Antibiotic Susceptibility Pattern Between ESBL-Producing Isolates of *E. Coli* and *K. Pneumoniae*

The results in present study show, cefotaxime exhibited a significantly higher proportion of sensitive isolates in *E. coli* compared to *K. pneumoniae*. In contrast, cefepime showed a greater resistance rate among *E. coli* isolates, while *K. pneumoniae* isolates were more frequently categorized as intermediate or sensitive. For ceftriaxone, a significantly higher percentage of *E. coli* isolates were resistant. Regarding gentamicin, *K. pneumoniae* demonstrated a notably higher resistance rate, whereas *E. coli* showed a higher proportion of sensitive isolates. Antibiotics resistance patterns between isolates of *K. pneumoniae* and *E. coli* in general are displayed in Figure (1). The results of statistical analysis reveal no-significant differences ($p>0.05$) between *E. coli* and *K. pneumoniae* with regard to antibiotic susceptibility patterns for the studied antibiotics.

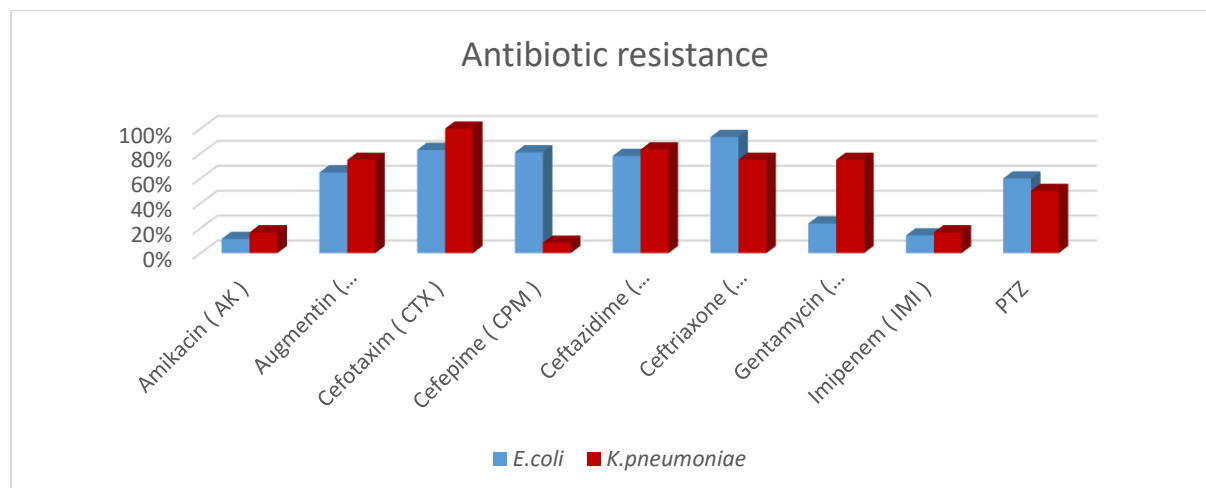


Figure (1). Comparison of antibiotics resistance between ESBL producing isolates of *K. pneumoniae* and *E. coli*.

This study demonstrated significant levels of extended-spectrum β -lactamase (ESBL)-producing *Escherichia coli* and *Klebsiella pneumoniae* in samples from patients with urinary tract infections (UTIs) from selected Baghdad hospitals. The present findings underscore the growing resistance of these bacteria to common antibiotics, suggesting an urgent need to improve treatment and monitoring strategies in hospitals.

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Conflict of interests.

There are non-conflicts of interest.

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الخلاصة

المقدمة

تمثل التهابات المسالك البولية (UTIs) جزءًا كبيرًا من الالتهابات البكتيرية في الممارسة السريرية، وقد تقاوم تعقيدها مؤخرًا بسبب ظهور مسببات الأمراض المقاومة للمضادات الحيوية. يُعد إنتاج بيتا لاكتامازات واسعة الطيف (ESBLs) آلية مقاومة شائعة، مما يقلل من فعالية المضادات الحيوية من الخط الأول ويشكل تحديًا كبيرًا لإدارة العدوى في المستشفيات والمجتمعات، وخاصة في المناطق النامية. هدفت هذه الدراسة إلى تحديد وتيرة البكتيريا المنتجة لبيتا لاكتامازات واسعة الطيف في التهابات المسالك البولية وتقييم أنماط حساسية مضادات الميكروبات لمسببات الأمراض المعزولة.

الهدف

هدفت هذه الدراسة إلى تحديد مدى انتشار البكتيريا المنتجة لـ ESBL في التهابات المسالك البولية وتقييم أنماط حساسيتها للمضادات الميكروبية.

طرق العمل

أجريت دراسة مقطعية من مارس 2024 إلى مارس 2025 في مستشفى الأطفال التعليمي المركزي والمركز الوطني للمختبرات التعليمية في بغداد. تم جمع مائتين وأربع وعشرون عينة ادرار من مرضى تم تشخيصهم سريريًا بالتهابات المسالك البولية. تمت معالجة جميع العينات في مختبرات الأحياء الدقيقة. تم تحديد الأنواع البكتيرية، وتحديد أنماط المقاومة، وإنتاج بيتا لاكتاماز واسع الطيف باستخدام تقنيات ميكروبيولوجية تقليدية، واختبارات كيميائية حيوية، ونظام آلي.

النتائج

من بين 224 عينة، كانت 182 عينة (81.25%) من الإشريكية القولونية، و42 عينة (18.75%) من الكلبسيلا الرئوية. ومن بين منتجي ESBL، كانت 105 عينات (57.69%) من الإشريكية القولونية، و12 عينة (28.57%) من الكلبسيلا الرئوية. أظهرت هذه العينات مقاومة عالية بشكل ملحوظ للسيفالوسبورينات ومشتقات البنسلين، بينما ظلت معظمها حساسة للأميكاسين والإيميبينيم. لم تُلاحظ أي ارتباطات مهمة بين إنتاج الإنزيم وعمر المريض أو جنسه، ولكن سُجلت اختلافات في أنماط المقاومة بين الأنواع وحالة إنتاج الإنزيم.

الاستنتاج

أظهرت الدراسة انتشارًا واسعًا للبكتيريا المنتجة لإنزيم بيتا لاكتاماز واسع الطيف في التهابات المسالك البولية، وخاصةً بين عزلات الإشريكية القولونية. تُظهر هذه البكتيريا مقاومة مُقلقةً للمضادات الحيوية الشائعة، مما يستلزم إجراء فحص دوري واستخدامًا حذرًا للمضادات الحيوية لمنع انتشار المقاومة.

الكلمات المفتاحية: عدوى المسالك البولية، بيتا لاكتامازات واسعة الطيف، مقاومة مضادات الميكروبات، الإشريكية القولونية، الكلبسيلا الرئوية