Qurina Scientific Journal – QSJ

ISSN: 2959- 7463

Identification and antimicrobial resistance of E-coli from clinical samples isolated from patients at Al-Jalaa hospital for trauma and surgery, Benghazi- Libya

[1] Mohamed Mustafa Abdelaziz [2] Asma Ahmed Mohammed, [3]Abdulmunem Mohammed Abdulmunem,[4]Dareen El shareef Jadullah.

[1]lecturer at Faculty of medical Sciences, Qurina International University-Benghazi - Libya. https://orcid.org/000-0002-4468 E-mail: mohamedmustafaabdelaziz@qiu.edu.ly.
[2]Lecturer at Faculty of pharmacy, Qurina International University, Benghazi– Libya, orcid.org/0009-0007-2064-2608 Email: asmaahmadmohammedaltargi@qiu.edu.ly.
[3]lecturer at Faculty of medical Sciences, Qurina International University-Benghazi - Libya. https://orcid.org/0009-0005-7902-1334 E-mail: menemobed@qiu.edu.ly.
[4]Lecturer at Faculty of pharmacy, Qurina International University, Benghazi– Libya, orcid.org/0009-0007-2064-2608 Email: dareenelshareef@qiu.edu.ly

Received: April 15, 2025 Revised: May 6, 2025 Accepted: May 26, 2025 Published online: Jul 10, 2025

Abstract— *Escherichia coli (E-coli)* is the most extensively studied prokaryotic model organism in biotechnology and microbiology and is a major pathogen of increasing importance due to the rise in antibiotic resistance. **Aim:** This study was aimed to identify the prevalence and antimicrobial susceptibility pattern of *E-coli* isolated from patients in Al-Jalaa hospital for trauma & surgery - Benghazi - Libya. **Methods:** A total of 108 samples were randomly selected from different wards and outpatient department. The bacterial identification and sensitivity were carried out using BD Phoenix system. Significant bacterial growth on culture of the specimens was processed for identification on the basis of colony morphology, Gram staining, and biochemical reactions. Using the Kirby-Bauer disk diffusion method, the antimicrobial susceptibility test (AST) was conducted. Version 28 of IBM SPSS software was utilized to analyze the data. **Results:** our results show that *E-coli* found in 83 patients' sample, *E-coli* was found to be resistant to majority of antibiotics, whereas sensitive to both Amikacine (43) and Imipenem (36). **Conclusion:** Our study shows that *E-coli* found resistant to the majority of antibiotics used in anti-sensitivity test as it's a play a major role in infection in hospitals and consider a big health issue. Also, the use of broad-spectrum antibiotic irrationally increases without proper plan, lack of surveillance, suitable antibiotics detection through the period of management and infection control, lead to failure of management process. A further evaluation astudy required to study the prevalence and the resistance of *E-coli* toward antibiotics.

***Keywords*-; Escherichia coli, Antibiotics, Antibiotic resistance, Isolation and Identification.**

1.INTRODUCTION

1.1. E-coli:

Theodor Escherich (1857-1911), a German pediatrician, identified the bacteria *Escherichia coli* in 1885 when he isolated it from baby excrement. *E. coli* is a gram-negative, non-sporulating, rod-shaped, facultative anaerobic, and coliform bacteria from the *Escherichia* genus that is often found in the environment, foods, and the lower guts of warm-blooded animals [(1,2)]. It is the most extensively studied prokaryotic model organism in biotechnology and microbiology. It can thrive for extended periods of time in feces, soil, and water and is often used as a water contamination indicator organism. The bacterium multiplies rapidly in fresh feces under aerobic conditions for 2-3 days, but it gradually decreases in number.

E. coli is a gram-negative, straight, rod-shaped, non-sporing, non-acid fast bacillus that can exist alone or in pairs. Cells are typically rod-shaped, measuring 1-3 μm × 0.4-0.7

µm (micrometer), 1 µm in length, 0.35 µm in width, and 0.6-0.7 µm in volume. It is motile due to the peritrichous flagellar structure, and only a few strains are non-motile. Although *E. coli* grows best around 37°C, certain laboratory strains can multiply at temperatures as high as 49°C. In good settings, reproduction can occur in as little as 20 minutes [3,4].

Polysaccharide capsules have been observed in several *E. coli* strains recovered from extra-intestinal infections. Negative staining processes provide a bright halo over a black backdrop, making it possible to see the *E. coli* capsules clearly. They have a thin cell wall that contains only one or two layers of peptidoglycan [5]. It colonizes a newborn's gastrointestinal (GI) tract within hours after delivery and even helps to maintain digestive health. Several strains of *E. coli* have been identified as good and effective probiotics, and are being used in pharmaceuticals. It is a true facultative anaerobic chemoorganotroph, capable of both respiratory and fermentative metabolisms.

Although the majority of *E. coli* strains are harmless, some serotypes can cause diarrhea if taken through contaminated food or drink, while others can cause urinary tract infections (UTIs), anemia, lung or kidney diseases. However, certain strains have evolved into pathogenic *E. coli* by acquiring virulence factors via plasmids, transposons, bacteriophages, and/or pathogenicity islands. The pathogenic strain of *E. coli* can be classified based on serogroups, pathogenicity mechanisms, clinical symptoms, and virulence factors [6-9]

1.2 Identification of *E-coli* in the Clinical laboratory:

1.2.1. Structure:

E. coli is a Gram-negative bacillus that measures 1-3 µm (0.4-0.7 µm) in size. Bacilli might be found in pairs or singly. They have peritrichous flagella, which makes them motile. Some strains are not motile. Some strains of *E. coli* may be fimbriated. The fimbriae are type I (hemagglutinating and mannose-sensitive), and they are seen in both motile and nonmotile strains. Some *E. coli* strains recovered from extraintestinal infections feature polysaccharide capsules. They do not produce any spores [10].

1.2.2. Isolation and Identification:

E. coli is an aerobic and facultative anaerobe. It develops at temperatures ranging from 10 to 40°C (optimal 37°C) with a pH of 7.2. The bacterium can grow on a variety of media, including Mueller-Hinton agar, nutrient agar, blood agar, and MacConkey agar. Primary isolation can be performed on nutrition and blood agar. After 18 hours of incubation at 37°C, *E. coli* on nutritional agar develops big, circular, low convex, greyish white, moist colonies that are smooth, opaque, or partially translucent. These smooth colonies are easily emulsified in saline. The rough or R types result in rough colonies with an uneven, dull surface. These colonies are frequently autoagglutinable in saline. Smooth to rough variation (S-R variation) is caused by repeated sub-culturing and is linked to the loss of surface antigens as well as pathogenicity. *E. coli* ferments lactose in MacConkey medium, resulting in bright pink flat colonies. Many strains, particularly those isolated from pathologic circumstances, form beta-hemolytic colonies on blood agar. They do not develop on selective medium like DCA (deoxycholate citrate agar) or SS (Salmonella- Shigella) agar, which are used to culture salmonellae and shigella. Finally, *E. coli* in liquid broth culture causes cloudy growth with a deposit that disperses completely when shaken [10].

1.2.3. *E-coli* biochemical reactions:

E. coli shows following reactions:

- *E. coli* ferments lactose, glucose, mannitol, maltose, and many other sugars with the production of acid and gas. They do not ferment sucrose. Some strains of *E. coli* are late lactose or non-lactose fermenters.
- They do not liquefy gelatin, do not produce hydrogen sulfide (H_2S), or do not utilize urea. Some variant strains of *E. coli* produce H_2S.
- The "IMViC" tests, which include indole, methyl red (MR), Voges-Proskauer (VP), and citrate consumption, are commonly used biochemical assays for classifying enterobacteria. *E. coli* is indole and MR positive but VP and citrate negative (IMViC++--).
- Some strains of *E. coli* are late lactose or non-lactose fermenters. Ability to produce H_2S (positive variants) and utilize citrate by *E-coli* are controlled by transmissible plasmids [10].

1.3.1. Epidemiology of *E-coli* Infections:

In tropical countries, Enteropathogenic E-coli (EPEC) is an important cause of childhood diarrhea. Enterotoxogenic *E-coli* (ETEC) causes 11-15% of cases of traveler's diarrhea in persons visiting developing countries and 30-45% of cases of traveler's diarrhea among those visiting Mexico. Enteroaggregative *E-coli* (EAggEC) causes 30% of cases of traveler's diarrhea. *E-coli* neonatal meningitis has a death rate

of up to 10%, and the majority of survivors have neurological or developmental issues. *E-coli* bacteremia causes the same mortality and morbidity as other aerobic gram-negative bacteria. *E-coli* UTI is more frequent in women than in men due to anatomical variations and changes during sexual development, pregnancy, and childbirth. Men over the age of 45 with prostatic enlargement are more likely to develop a UTI due to bladder stasis. *E-coli* UTI in neonates is more prevalent in boys than in girls, but circumcision lowers the risk [(11)].

1.3.2. Clinical Manifestations of *E-coli*:

E-coli are a type of gram-negative bacteria that live in healthy people's intestines, but some strains can infect the digestive system, urinary tract, and other regions of the body. Intestinal infections can produce severe or bloody diarrhoea, as well as abdominal pain. Many *E. coli* infections affecting places other than the digestive system originate in persons who are ill, are in a health care facility, or have taken antibiotics.

E. coli can cause infections outside the gut if it is torn or destroyed, such as by an injury or an illness like inflammatory bowel disease. Then, the bacteria may leave the intestine and spread to nearby structures that have no defences against them or they may enter the bloodstream. One strain produces a toxin that causes brief watery diarrhoea. This disorder (called traveller's diarrhoea) usually occurs in travellers who consume contaminated food or water in areas where water is not adequately purified. People with traveller's diarrhoea have abdominal cramping and watery diarrhoea and sometimes nausea and vomiting. Symptoms are often mild and remit within 3 to 5 days. Certain *E. coli* strains release toxins that harm the colon and cause severe inflammation (colitis). These strains are collectively known as enterohemorrhagic *E. coli*. Haemolytic-uremic syndrome is a complication that develops in about 5 to 10% of people (mainly children under 5 years and adults over 60 years) about 1 week after symptoms begin. In this syndrome, red blood cells are destroyed (called haemolysis), and kidney failure occurs, causing toxic substances to build up in the blood (called uraemia). This complication is a common cause of chronic kidney disease in children [(12)].

1.3.3. Treatment of E-coli:

Treatment depends on both the strain and the illness itself. The first step in caring for a patient with an *E. coli*-caused intestinal problem is to treat the symptoms. Patients suffering from diarrheal disease can experience significant distress. Experts prescribe rehydration and antidiarrheal medications as the primary treatments for mild illness. When tolerated, oral rehydration is advised as the first-line therapy for all patients with diarrheal disease and is as effective as intravenous hydration. If patients are unable to accept oral intake, intravenous hydration is indicated. Anti-motility medications, such as bismuth-subsalicylate and loperamide, are used to relieve distressing symptoms.

Antibiotics are not indicated as a first-line treatment for *E. coli*-caused diarrhea for the majority of patients due to the adverse side effects and relationship with antibiotic resistance. Antibiotics may be appropriate for patients with severe disease (e.g., more than six stools per day, fever, dehydration that requires hospitalization, diarrhea lasting more than seven days, or bloody diarrhea). The Infectious Diseases Society of America (IDSA) and the International Society of Travel Medicine (ISTM) currently propose rifaximin, azithromycin, and ciprofloxacin as treatments for *E. coli* diarrheal disease. Antibiotics are not advised for patients suspected of having EHEC/STEC, particularly in children and the elderly, because they increase the risk of hemolytic uremic syndrome [(13)].

1.3.4. Antibiotic resistance:

1.3.4.1. Resistance to beta-lactams antibiotics:

Resistance to β-lactams is conferred by many genes in *E. coli*, both human and animal-derived. Some genes, such blaTEM-1, are found in *E. coli* from animals but only produce narrow-spectrum β-lactamases capable of inactivating penicillin and aminopenicillin. However, in recent years, genes coding for ESBLs/AmpCs have appeared in *E. coli* from people and animals. Recently, genes coding for carbapenems have been found in animal-derived *E. coli* [(14)].

1.3.4.2. Resistance to quinolones and fluoroquinolones:

Quinolones and fluoroquinolones are effective antibacterial medicines for treating a variety of infections in people and animals. They are known to be effective against almost all germs. Resistance of some strains of *E-coli* to these antimicrobial agents is usually due to mutations, namely, the genes for DNA gyrase and topoisomerase IV, but other mechanisms such as reduced permeability of the outer membrane, protection of the target structures, or up-regulated efflux pumps may also play a role [(15)].

1.3.4.3. Resistance to Aminoglycosides:

Aminoglycosides inhibit translation in a wide range of pathogens, including Gram-negative and Gram-positive bacteria. Two key difficulties may restrict the therapeutic efficacy of these crucial molecules: the first is their toxicity.

The second issue is the spread of bacterial resistance connected to the use of aminoglycosides, which has occurred worldwide. *E. coli* resistance to aminoglycosides is caused by target mutations in the 16S RNA and/or the S5 and S12 ribosomal proteins. The target site of aminoglycosides can also be modified by methylation of residues G1405 and A1408 of site A of the 16S RNA, resulting in high resistance to amikacin, tobramycin, gentamicin, and netilmicin. Enzymes also inactivate aminoglycosides by modifying the molecules, preventing them from reaching or binding to the target site. There are now three types of aminoglycoside-modifying enzymes: acetyltransferases, nucleotidyltransferases, and phosphotransferases [16, 17].

1.3.4.4. Resistance to Tetracyclines:

Because of the selection pressure caused by the extensive use of tetracyclines, many bacteria, including *E. coli*, have developed tetracycline resistance. According to the tetracycline resistance gene nomenclature center, nine tetracycline efflux genes [tet(A), tet(B), tet(C), tet(D), tet(E), tet(G), tet(J), tet(L), and tet(Y)], two tetracycline resistance genes encoding ribosome protective proteins [tet(M) and tet(W)], and one gene coding for an oxidoreductase that inactivates tetracyclines [tet(X)] have been identified in *E. coli*. The primary mechanisms of tetracycline resistance in *E. coli* are active efflux by proteins of the main facilitator superfamily, and ribosome protection [18].

1.3.4.5. Resistance to Sulfonamides and Trimethoprim:

Sulfonamides and trimethoprim have been used for decades in humans. Acquired resistance mechanisms have been frequently identified in *E-coli*, mainly due to (i) mutational modifications in the genes encoding the target enzymes, namely, the dihydropteroate synthase or dihydrofolate reductase, respectively, or (ii) the acquisition of sul genes encoding dihydropteorate synthetases that are insensitive to sulfonamides or dfr genes encoding dihydrofolate reductases that are insensitive to trimethoprim [19].

1.3.4.6. Resistance to Polymyxins:

Colistin is widely used in medicine; however, due to significant worries that colistin resistance could be transmitted from animals to humans, stringent limits on the use of colistin have been established in Europe under the auspices of the European Medicines Agency. In April 2017, a prohibition on colistin as a growth promoter became applicable in China. Colistin is active against a variety of Enterobacteriaceae species, including *E. coli*, however Proteus spp. and Serratia spp. are intrinsically resistant. Colistin resistance can be caused by either chromosomal gene mutations or acquired resistance genes [20-22].

1.4. Aim of the study:

This study aimed was to identify the prevalence and antimicrobial susceptibility pattern of *E-coli* bacteria isolated from patients in AL-Jalaa teaching hospital for trauma & surgery - Benghazi - Libya.

2. METHODOLOGY

2.1. Ethical approval:

This study was approved from Qurina International University – Faculty of pharmacy, and AL-Jala hospital for trauma & surgery, consent was taken from patients before the study.

2.2. Study design:

This was a cross-sectional descriptive study was done in inpatients admitted to surgical wards and out patients of AL-Jala hospital for trauma & surgery, from January 2023 to April 2023.

2.3. Sample collections and isolation:

A total of 108 samples were randomly selected from different wards (female surgical ward A, male surgical ward A, intensive care unit, burn shock room, and outpatient department at AL-Jala hospital for trauma & surgery). Samples collected were taken from both genders with different ages. Specimen isolates were obtained from (urine, swap, tip of folly catheter, Endotracheal tube, and blood). All the culture and sensitivity reports of E-coli from hospitalized patients and outpatient departments were analyzed. The bacterial identification and sensitivity were carried out using the BD Phoenix system (BD Diagnostics, Sparks, MD, USA) & the guidelines of the Clinical and Laboratory Standard Institute were used in the laboratory.

Inoculation in culture media and aerobic incubation at 37°C were performed following normal microbiological procedures. Significant bacterial growth on the culture of the specimens was used to identify E. coli using colony morphology, Gram staining, and biochemical responses. The Kirby-Bauer disk diffusion method was used to perform an antimicrobial susceptibility test (AST). The Kirby-Bauer disk diffusion method was used to perform an antimicrobial susceptibility test (AST). Antibiotics used for assessing bacterial susceptibility included (Amoxicillin, Imipenem, Ticarcillin-

Clavulanic Acid, Cefeimpe, Ertapenem, Chloramphenicol, Ciprofloxacin, Tetracycline, Ceftriaxone, Nitrofuranntoin, Gentamicin, Vancomycin, Cephalothin, Nalidixic Acid, Penicillin, Cefoxitin, Clindamycin, Amoxicillin-Clavulanate, Piperacillin, Kanamycin).

2.4. Statistical analysis:

IBM SPSS software version 28 was used for data analysis. Data was comprised of gender, samples from patients, and wards of hospitals as frequencies and percentages. Chi square test was employed to determine the difference between each variable in the study.

3. RESULTS

A total 108 isolated samples were obtained from inpatients in surgical and out patients department in AL-Jala hospital for trauma & surgery, as found 83 samples were positive in the presence of E-coli growth.

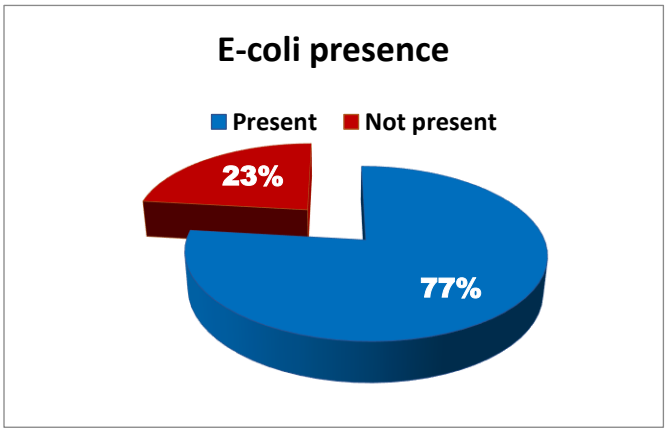

Figure (1): The presence of E-coli in the samples.

More than half of the samples obtained from outpatient department, while 9.6% from male surgical ward A, sharing the same percent 6 % burn surgical shock room and female surgical ward A, 4.8% in chest surgical ward and last intensive care unit with 14.5%. As seen in table (1).

Table (1): The department frequency and percent

Department	Frequency	Percent
Out-patient departement (OPD)	49	59%
Burn surgical shock room (BSSR)	5	6%
Intensive care unit (ICU)	12	14.5%
Female surgical ward (FSW)	5	6%
Male surgical ward (MSW)	8	9.6%
Chest surgical ward (ChSW)	4	4.8%
Total	**83**	**100%**

As seen in table and figure (2), Which describe the gender of patients enrolled in the study as 35% of them was male while female taking the rest percent with 65 %.

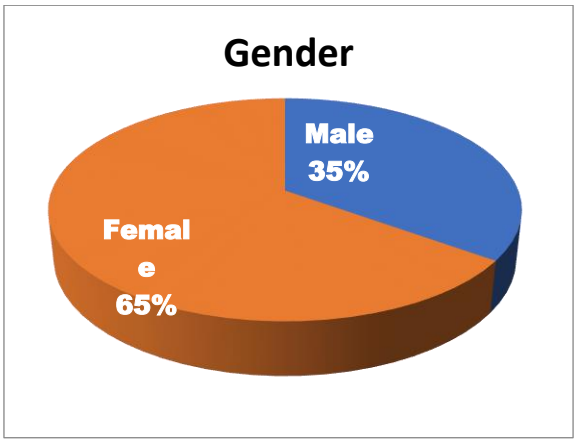

Figure (2): The gender distibution.

Figure (3) shows source of samples as most of the samples were urine sample with 45.8%, second swaps from site of the wound with 43.4%, third was catheter tip with 4.8%, then blood with 3.3%, and Endotracheal tube (ETT) is 2.4%.

ISSN: 2959- 7463

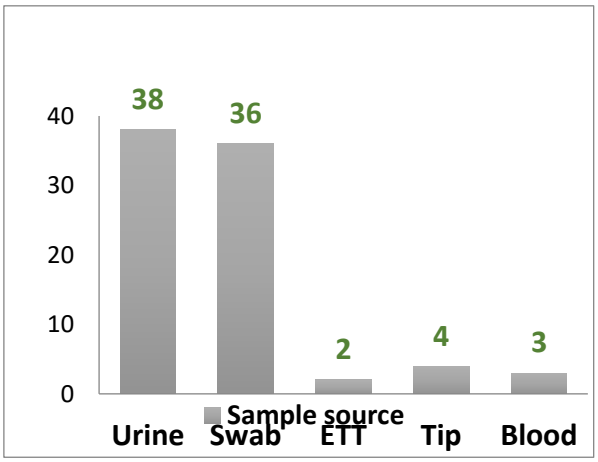

Figure (3): The sample source distribution.

From the following table (2) and figure (4) we notice that E-coli was resistant to majority of antibiotics, whereas sensitive to both Amikacine (43) and Imipenem (36).

Table (2):Antimicrobial susceptibility pattern of *E-coli* to different antibiotics

Antibiotic	Resistant	Intermediate	Sensitive
Ampicilin (AMP)	44	3	**36**
Cephalexin (CL)	78	2	**3**
Cefepime (FEP)	76	0	**7**
Ertapenem (ETP)	81	1	**1**
Ceftazidime (CZ)	79	0	**4**
Ciprofloxacine (CIP)	54	0	**29**
Tetracycline (TE)	75	0	**8**
Levofloxacin (LEV)	72	0	**11**
Trimethoprim-sulfamethoxazole (SXT)	80	0	**3**
Cefotaxime (CTX)	70	1	**12**
Ceftriaxone (CRO)	81	0	**2**
Aztreonam (ATM)	81	0	**2**
Nitrofurantoin (FA)	70	1	**12**
Nalidixic acid (NA)	76	0	**7**
Gentamicin (GN)	72	0	**11**
Cefoxitin (CN)	76	0	**7**
Oflatoxin (OFX)	61	0	**22**
Imipenem (IPM)	72	3	**8**
Amikacine (AK)	38	2	**43**
Ceftazidime (CAZ)	79	0	**4**
Doxycycline (DO)	65	5	**13**

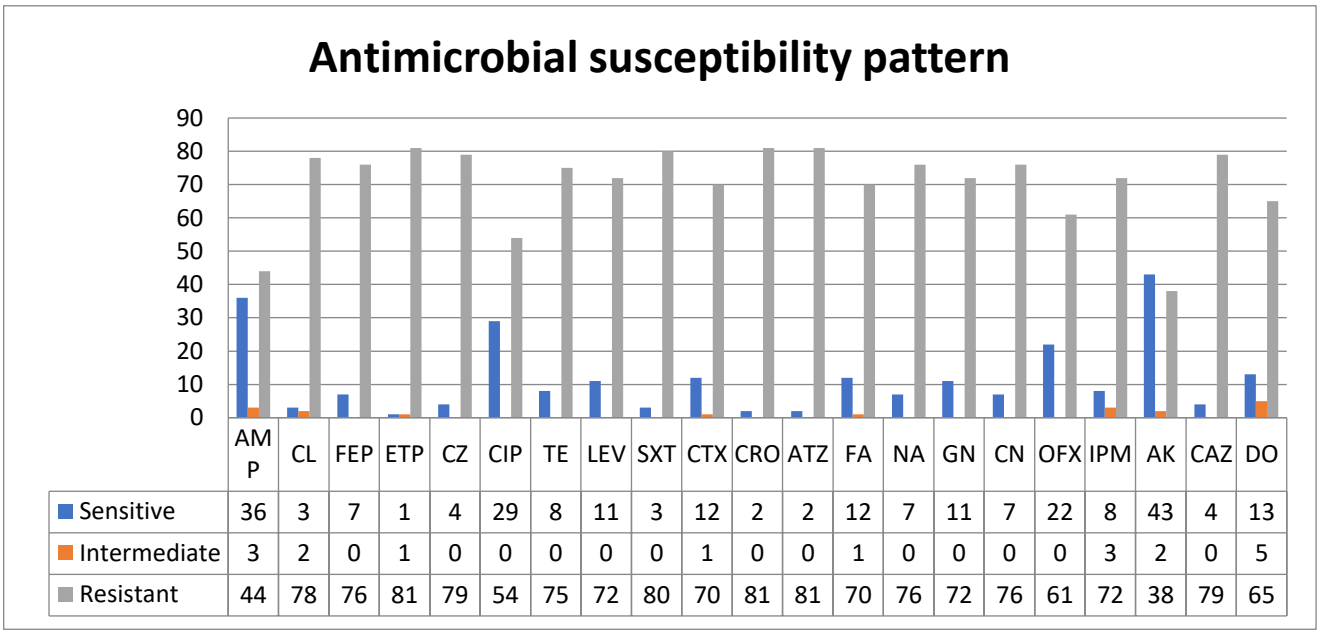

Figure (4):Antimicrobial susceptibility pattern of *E-coli* to different antibiotics.

Table (3): The relation between department & sex.

Cross tabulation of (Department & Sex)				
Department		sex		Total
		Male	**Female**	
Section	Outpatient department	10	39	49
	Burn shocks room	2	3	5
	Intensive care unit	7	5	12
	Male surgical wards A	8	0	8
	Female surgical ward A	0	5	5
	Chest surgical ward	2	2	4
Total		29	54	83

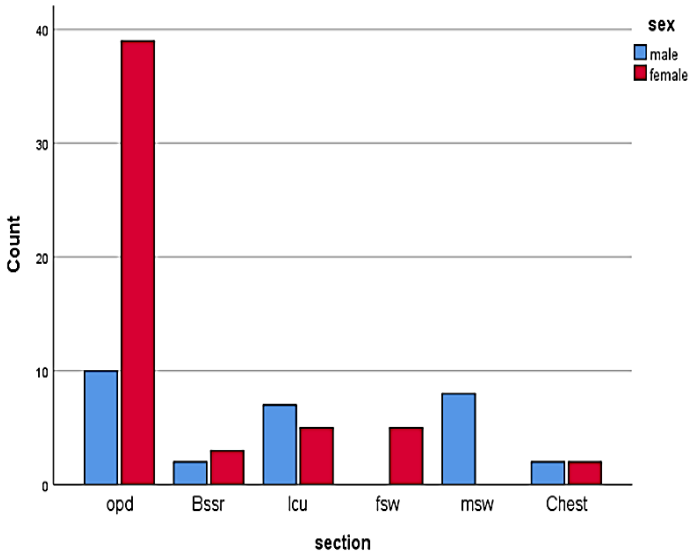

Figure (5): The relation between department and sex curve.

Table (4): The relation between department & sex by chi-Square

Chi-Square Tests			
	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	25.478[a]	5	0.000
N of Valid Cases	83		

From the following tables (5,6) and figure (6) we note that the probability value = 0.000, which is less than the probability value (α =0.05), which indicates the existence of a significant relationship between the two variables (department & sample).

Table (5): relationship between section & sample.

Cross tabulation of (Section & sample)							
		sample					Total
		Urine	Swab	Tip	ETT	BLOOD	
Section	Outpatient department	36	11	1	0	1	49
	Burn shocks room	0	3	2	0	0	5
	Intensive care unit	0	10	0	2	0	12
	Male surgical wards A	0	6	0	0	2	8
	Female surgical wards A	0	4	1	0	0	5
	Chest surgical ward	2	2	0	0	0	4
Total		38	36	4	2	3	83

Table (6): The relationship between section & sample by Chi-Square Tests.

Chi-Square Tests			
	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	75.474[a]	20	0.000
N of Valid Cases	83		

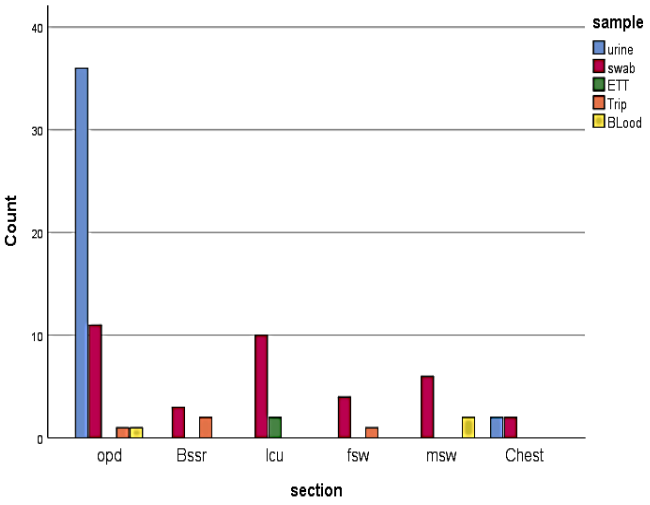

Figure (6): The relationship between section & sample curve.

From the following tables (7,8) and figure (7) we note that the probability value = 0.010, which is less than the probability value (α =0.05), which indicates the existence of a significant relationship between the two variables (sex & sample)

Table (7): The relationship between sex & sample

Cross tabulation of (Sex & sample)							
		sample					**Total**
		Urine	Swab	Tip	ETT	BLOOD	
Sex	Male	7	19	0	1	2	29
	Female	31	17	4	1	1	54
Total		38	36	4	2	3	83

Table (8): The relationship between sex & sample by Chi-Square Tests.

Chi-Square Tests			
	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	13.277[a]	4	0.010
N of Valid Cases	83		

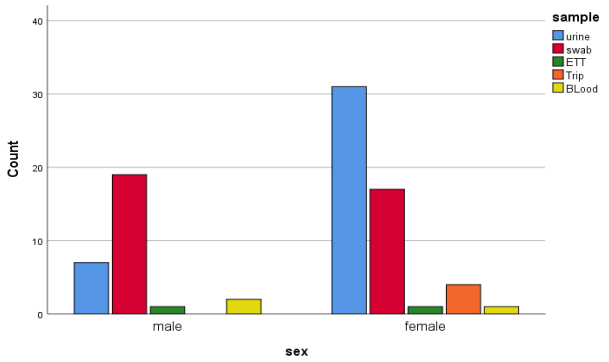

Figure (7): The relationship between sex & sample

Figure (8): The growth of *E-coli* in MacConkey Agar.

As show in the figure (8) the morphology of *E-coli* colonies in MacConkey agar, whereas figure (9) shows the Gram stain as it shown that the *E-coli* is gram negative.

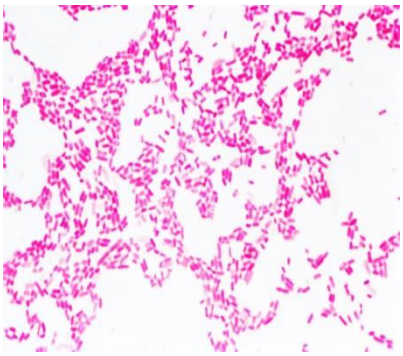

Figure (9): The Gram strain of *E-coli*.

4. DISSCUSSION

This study was based and examine the presence of *E-coli* using clinical samples from different wards and outpatient department in AL-Jalaa hospital. In this study we show that *E-coli* infection is more prevalence in female 65% than male patients with 35%, which the same result with most of the studies such as (Odongo I. et al 2022) [27].

As *E-coli* found resistant to majority of antibiotics used in the study, according to the antibiogram results most of the resistant antibiotics consider broad spectrum ones reflect the un appropriate use of them in treatment of patients. Anti-sensitivity test *E-coli* isolates against commonly used antibiotics showed that the overall resistance to antibiotics was alarmingly higher in Ertapenem, Ceftriaxone, and Aztreonin as found resistant to (81) of patients from (83) for each one of them, followed by Trimethoprim-sulfamethoxazole with (79), which relatively similar to the finding of (Daoud N. et al, 2020)[28].

This study shows that Amikacine is the most potent antibiotics in treating E-coli with (43) of patients in contrast with (Bong et al. 2022) [29] where nitrofurans was the most potent antibiotic used.

5.CONCLUSIONS

Our study shows that *E-coli* found resistant to the majority of antibiotics used in anti-sensitivity test as it is a play a major role in infection in hospitals and consider a big health issue. Also, the use of broad-spectrum antibiotic irrationally increases without proper plan, lack of surveillance, suitable antibiotics detection through the period of management and infection control, lead to failure of management process. A further evaluation and study required to study the prevalence and the resistance of E-coli toward antibiotics.

REFERENCES

1. Feng P, Weagant S, Grant M. Enumeration of Escherichia coli and the Coliform Bacteria. Bacteriological Analytical Manual. 8th ed. USA: FDA/ Center for Food Safety & Applied Nutrition; 2007
2. Campbell NA, Reece JB. Biology. San Francisco: Pearson Education Inc; 2002
3. Facts about E. coli: Dimensions as discussed in bacteria: Diversity of structure of bacteria. Britannica 2015:06-25. Available from: https:// www.britannica.com/science/ bacteria/ The-cell-envelope
4. Bacteria. Microbiology online. Archived from the original on 27 February 2014. Available from: https://web.archive.org/web/20140227212658/DOI:10.1128/AAMCol.1-2011
5. Köhler CD, Dobrindt U. What defines extra intestinal pathogenic Escherichia coli? International Journal of Medical Microbiology. 2011; 301:642-664
6. Enger ED, Ross FC. Concepts in Biology. 10th ed. New York, USA: McGraw-Hill; 2003. Available from: http://hyperphysics.phyastr.gsu.edu/hbase/Biology/ecoli.html
7. Blount ZD. The unexhausted potential of E. coli. eLife. 2015;25(4): e05826. DOI: 10.7554/eLife.05826

8. FAQ: E. coli: Good, Bad, & Deadly— NCBI Bookshelf; Retrieved 07 June 2021 from https://www.ncbi.nlm.nih.gov/books/NBK562895. DOI: 10.1128/ AAMCol.1-2011.
9. Kaper J, Nataro J, Mobley H. Pathogenic Escherichia coli. Nature Reviews. Microbiology. 2004; 2:123-140. DOI: org/10.1038/nrmicro818.
10. Parija S. Textbook of Microbiology and Immunology, 2nd edi. Elsevier India; 2012.
11. Medina, M; Castillo-Pino, E. An introduction to the epidemiology and burden of urinary tract infections. Therapeutic Advances in Urology. 02 May 2019.
12. Bush L. Escherichia coli Infections [Internet]. MSD Manual Consumer Version. 2023. Available from: https://www.msdmanuals.com/home/infections/bacterial infections-gram-negative-bacteria/escherichia-coli-infections.
13. Mueller M, Tainter C. Escherichia coli Infection. [Updated 2023 Jul 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK564298/
14. Poirel L, Madec J, Lupo A, Schink A, Kieffer N, Nordmann P, et al. Antimicrobial Resistance inEscherichia coli. Microbiology Spectrum [Internet]. 2018 Jul 27;6(4). Available from: https://doi.org/10.1128/microbiolspec.arba-0026-2017.
15. Hopkins K, Davies R, Threlfall E. Mechanisms of quinolone resistance in Escherichia coli and Salmonella: recent developments. Int J Antimicrob Agents 2005. 25:358–373.
16. Fourmy D, Yoshizawa S, Puglisi J. Paromomycin binding induces a local conformational change in the A-site of 16 S rRNA. J Mol Biol 1998. 277:333–345. http://dx.doi.org/10.1006/jmbi.1997.1551.
17. Bowers D, Tam V. Aminoglycoside pharmacodynamics, p 199–220. In Rotschafer J, Andes D, Rodvold K (ed), Antibiotic Pharmacodynamics. Methods in Pharmacology and Toxicology. Humana Press 2016., New York, NY.
18. Shin S, Shin M, Jung M, Belaynehe K, Yoo H. Prevalence of antimicrobial resistance and transfer of tetracycline resistance genes in Escherichia coli isolates from beef cattle. Appl Environ Microbiol 2015. 81:5560–5566.
19. Van Duijkeren E, Schink A, Roberts M, Wang Y, Schwarz Š. Mechanisms of Bacterial Resistance to Antimicrobial Agents. Microbiology Spectrum. 2018 Apr 6;6(2).
20. Poirel L, Jayol A, Nordmann P. Polymyxins: antibacterial activity, susceptibility testing, and resistance mechanisms encoded by plasmids or chromosomes. Clin Microbiol Rev 2017. 30:557–596 http://dx.doi.org/10.1128/CMR.00064-16.
21. Rhouma M, Beaudry F, Thériault W, Letellier A. Colistin in pig production: chemistry, mechanism of antibacterial action, microbial resistance emergence, and One Health perspectives. Front Microbiol 2016. 7:1789 http://dx.doi.org/10.3389/fmicb.2016.01789.
22. European Medicines Agency. Updated advice on the use of colistin products in animals within the European Union: development of resistance and possible impact on human and animal health 2016. EMA/CVMP/CHMP/231573.1-56.
23. Laupland K, Gregson D, Church D, Ross T, Pitout J. Incidence, risk factors and outcomes of Escherichia coli bloodstream infections in a large Canadian region. Clinical Microbiology and Infection. 2008 Nov 1;14(11):1041–7.
24. Khalid A, Imran R, Ishtiaq H, et al. Prevalence of Escherichia coli in Suspected Urinary Tract Infected Patients and Their Sensitivity Pattern Against Various Antibiotics in Gilgit-Baltistan, Pakistan. Pakistan J. Zool., vol. 46(6), pp. 1783-1788, 2014.
25. Devanand P, Saxena R. Prevalence and antimicrobial susceptibility pattern of Escherichia coli in hospital acquired and community acquired patients related to urinary tract infection in India. Journal of Applied Pharmaceutical Science 2013, 3(08), pp. 124-132.
26. Kibret M, Abera B. Antimicrobial susceptibility patterns of E. coli from clinical sources in northeast Ethiopia. Afr Health Sci. 2011 Aug;11 Suppl 1(Suppl 1): S40-5.
27. Odongo, I., Ssemambo, R., & Kungu, J. M. Prevalence of Escherichia Coli and Its Antimicrobial Susceptibility Profiles among Patients with UTI at Mulago Hospital, Kampala, Uganda. Interdisciplinary perspectives on infectious diseases, 2020, 8042540.
28. Daoud N, Hamdoun M, Hannachi H, Gharsallah C, Mallekh W, Bahri O. Antimicrobial Susceptibility Patterns of Escherichia coli among Tunisian Outpatients with Community-Acquired Urinary Tract Infection (2012-2018). Current Urology [Internet]. 2020 Dec 1;14(4):200–5.
29. Bong C, Low K. Chai L, & Lee C. Prevalence and Diversity of Antibiotic-Resistant Escherichia coli From Anthropogenic-Impacted Larut River. Frontiers in public health 2022. 10, 794513.