



T. C.

KAHRAMANMARAŞ SÜTÇÜ İMAM UNIVERSITY

GRADUATE SCHOOL OF NATURAL AND APPLIED SCIENCES

**ISOLATION OF BACTERIAL PATHOGENS FROM
PATIENTS WITH URINARY TRACT INFECTION IN
ERBIL, IRAQ AND DETERMINATION OF THEIR
ANTIBIOTIC SUSCEPTIBILITY PATTERNS**

MAY ALI SALEH

MASTER'S THESIS

DEPARTMENT OF BIOENGINEERING AND SCIENCES

KAHRAMANMARAŞ - TURKEY 2017

T.C.
KAHRAMANMARAŞ SÜTÇÜ İMAM UNIVERSITY
GRADUATE SCHOOL OF NATURAL AND APPLIED SCIENCES

**ISOLATION OF BACTERIAL PATHOGENS FROM
PATIENTS WITH URINARY TRACT INFECTION IN
ERBIL, IRAQ AND DETERMINATION OF THEIR
ANTIBIOTIC SUSCEPTIBILITY PATTERNS**

MAY ALI SALEH

**A thesis submitted in partial fulfillment of the requirements for the degree
of Master of Science in
Bioengineering and Sciences**

KAHRAMANMARAŞ-2017

M.Sc. thesis entitled “Isolation of bacteria pathogens from patients with urinary tract infections in Erbil, Iraq and determination of their antibiotic susceptibility patterns” and prepared by May Ali Saleh, who is a student at Department of Bioengineering and Sciences, Graduate School of Natural and Applied Sciences, KahramanmaraşSütçü İmam University was certified by all the majority jury members, whose signatures are given below, at the date of 15 / 6 / 2017.

Assist. Prof. Dr. Sabahattin CÖMERTPAY (Supervisor)

Department of Agricultural Biotechnology

KahramanmaraşSütçü İmam University

Assoc. Prof. Dr. E. Banu BÜYÜKÜNAL BAL (Member)

Department of Biology

KahramanmaraşSütçü İmam University

Assist. Prof. Dr. Akın YİĞİN (Member)

Department of Zootechnics and Animal Feeding.

Harran University

I approve that the above signatures related to the members.

Assoc. Prof. Dr. Mustafa ŞEKKELI

Director of Graduate School of Natural and Applied Sciences

KahramanmaraşSütçü İmam University

THESIS NOTIFICATION

I declare and guarantee that the entire information in this document has been obtained and presented in accordance with academic rules and ethical conduct. Based on these rules and conduct, I have fully cited and referenced all materials and results that are not original in this work.

May Ali Saleh



Note: Uses of the reports in this thesis, from original and other sources, tables, figures and photographs without citation, subject to the provisions of Law No.5846 concerning Intellectual and Artistic Works.

ERBİL ŞEHRİ İDRAR YOLU ENFEKSİYONU HASTALARINDAN BAKTERİYAL PATOJENLERİN İZOLASYONU VE ANTİBİYOTİK DİRENÇLERİNİN BELİRLENMESİ

(YÜKSEK LİSANS TEZİ)

MAY ALI SALEH

ÖZET

İdrar yolu enfeksiyonları en sık görülen bakteriyel enfeksiyonlardan biridir. Bu çalışmanın amacı, Erbil şehrinde yaşayan hastalardan alınan örneklerdeki etiyolojik ajanları ve bunların antibiyotik dirençlerini tespit etmektir. Çalışma için klinik olarak idrar yolu enfeksiyonu olduğu şüphesi bulunan 500 bireyden idrar örneği toplanmış ve incelenmiştir. Bakteriyel patojenler standart mikrobiyolojik yöntemlerle izole edilmiş ve antibiyotiklere karşı direnci Kirby-Bauer disk difüzyon yöntemiyle test edilmiştir. Sonuçlarımız, çalışılan hasta örneklerinin büyük çoğunluğunda hastalık yapıcı bakterilerin bulunduğunu göstermiştir. İzole edilen patojenler arasında en yaygın olanı *E. Coli* (44.38%)'dir. Bunu sırasıyla *Staphylococcus aureus* (%19.10), *Staphylococcus epidermidis* (10.39%), *Klebsiella pneumonia* (%6.17), *Pseudomonas aeruginosa* (5.05%), *Proteus mirabilis* (3.37%), *Staphylococcus haemolyticus* (1.96%), *Staphylococcus sarprophyticus* (1.96%), *Enterobacter aerogenes* (1.68%), *Klebsiella oxytoca* (1.12%), *Citrobacter koseri* (0.84%), *Morganella morganii* (0.84%), *Enterobacter cloacae* (0.56%), *Enterococcus faecalis* (0.56%), *Proteus vulgaris* (0.56%), *Serratia fonticola* (0.56%), *Steptocooccus agalactiae* (0.28%), *Citrobacter freundii* (0.28%), ve *Salmonella typhimurium* (0.28%), takip etmektedir. İzole edilen türlerin antibiyotik dirençlerini ölçmek için 10 farklı antibiyotik elde edilen 19 tür bakteri üzerinde denenmiştir. Sonuç olarak, tüm bakteriler Ciprofloxacın, Norfloxacın ve Ceftriaxone hassaslık gösterdiler. Ancak denenilen diğer antibiyotiklere karşı orta seviyeden yükseğe değişen miktarlarda dirençli oldukları gözlemlendi.

Anahtar Kelimeler: Hastalık yapıcı bakteriler, idrar yolu enfeksiyonu, antibiyotik direnci.

Kahramanmaraş Sütçü İmam Üniversitesi
Fen Bilimleri Enstitüsü

Biyomühendislik ve Bilimleri, Haziran, 2017

Danışman: Yrd. Doç. Dr.Sabahattin COMERTPAY

Sayfa sayısı: 69 page

ISOLATION OF BACTERIAL PATHOGENS FROM PATIENTS WITH URINARY TRACT INFECTION IN ERBIL CITY AND DETERMINATION OF THEIR ANTIBIOTIC SUSCEPTIBILITY PATTERNS

(M.Sc. THESIS)

MAY ALI SALEH

ABSTRACT

Urinary tract infections (UTIs) are among the most common bacterial infections in community practice. The aim of this study was to identify the etiologic agents and antibiotic resistance on samples taken from the patients living in Erbil City. A total of 500 urine specimens were collected from patients that were clinically suspected cases of UTIs. The bacterial pathogens were isolated by standard microbiologic methods and antimicrobial susceptibility testing was detected by the Kirby-Bauer disk diffusion method. The results showed significant bacterial pathogens were found in (71.2%) the final results of each patient. The most widespread pathogens isolated were *E. coli*(44.38%) while other bacteria were *Staphylococcus aureus*(19.10%), *Staphylococcus epidermidis* (10.39%),*Klebsiella pneumonia* (6.17%),*Pseudomonas aeruginosa* (5.05%), *Proteus mirabilis* (3.37%), *Staphylococcus haemolyticus* (1.96%), *Staphylococcus saprophyticus* (1.96%), *Enterobacter aerogenes* (1.68%), *Klebsiella oxytoca* (1.12%), *Citrobacter koseri* (0.84%), *Morganella morganii* (0.84%), *Enterobacter cloacae* (0.56%), *Enterococcus faecalis* (0.56%), *Proteus vulgaris* (0.56%), *Serratia fonticola* (0.56%), *Streptococcus agalactiae* (0.28%), *Citrobacter freundii* (0.28%), and *Salmonella typhimurium* (0.28%). 10 different microbial agents have been tried on 19 species of bacteria in order to determine their antibiotic resistance. As a result, it was found that all bacteria species were sensitive to Ciprofloxacin, Norfloxacin and Ceftriaxon while their resistance ranged from high to moderate against the other antibiotics studied.

Key words: Bacterial pathogens, Urinary Tract

Kahramanmaraş Sütçü İmam University
Graduate School of Natural and Applied Sciences
Department of Bioengineering and sciences, June, 2017
Supervisor: Assist. Prof. Dr.Sabahattin COMERTPAY
Page numbers: 69 page

ACKNOWLEDGEMENTS

First of all, I would like to thank God for providing me all ability for preparing master program and developing myself in this aspect. On accomplishment of the present study, I would like to extend my sincere thanks to my supervisor Assist. Prof. Dr. Sabahattin CÖMERTPAY, for serious conductive advice throughout this research work.

Thanks and appreciations to my family, especially my parents, my husband, my brothers and my sisters whose supports, patience and efforts gave me hope, force and enthusiasm while made me strong to prepare and complete the research.

Finally, I would like to thank to Mr. Ali Jalak for their guidance, encouragement and criticism throughout the period of this study.

May Ali SALIH

TABLE OF CONTENTS

	<u>Page No</u>
ÖZET	i
ABSTRACT	ii
ACKNOWLEDGEMENTS	iii
TABLE OF CONTENTS.....	ix
LIST OF TABLES	xiii
LIST OF ABBREVIATIONS	xiv
1. INTRODUCTION	1
1.1.General.....	1
2 LITERATURE REVIEW.....	5
2.1.Urinary Tract Infection.....	5
2.1.1Definition.....	5
2.1.2.Etiology.....	5
2.1.3.Manifestation.....	6
2.2.Sources and Routes of Infection.....	7
2.3.Classification of UTIs.....	7
2.3.1.Uncomplicated UTI	7
2.3.2.ComplicatedUTI.	8
2.3.3.Isolated infection.....	8
2.3.4.Unresolve dinfection	8
2.3.5.Reinfection.....	8
2.3.6.Relapse.....	8
2.4.The Factors Predisposing to UTIs	9
2.4.1.Vesico-Ureteric reflux.....	9
2.4.2.Prostatic hypertrophy.....	9
2.4.3.Diabetes mellitus.....	9
2.4.4.Urolithiasis.....	9
2.4.5.Pregnancy.....	10
2.4.6.Age and gender	10
2.4.7.Genetic factors	10
2.5.Bacterial Virulence Factors.....	11
2.6.General Consideration for Some Common Facultative Anaerobic and Aerobic Bacteria Causing UTIs.....	11

2.6.1. Gram negative bacteria.....	11
2.6.1.1. Escherichia coli.....	11
2.6.1.2. Klebsiella sp.....	11
2.6.1.3. Proteus sp.....	12
2.6.1.4. Pseudomonas aeruginosa.....	12
2.6.1.5. Citrobacter sp.....	12
2.6.1.6. Enterobacter sp.....	13
2.6.1.7. Proteus sp.....	13
2.6.1.8. Morganella morganii.....	13
2.6.1.9. Serratia sp.....	14
2.6.1.10. Salmonella sp.....	14
2.6.2. Gram positive bacteria.....	14
2.6.2.1. Staphylococcus aureus.....	14
2.6.2.2. Staphylococcus epidermidis.....	15
2.6.2.3. Staphylococcus saprophyticus.....	15
2.6.2.4. Staphylococcus haemolyticus.....	15
2.6.2.5. Enterococcus sp.....	16
2.6.2.6. Enterococcus faecalis sp.....	16
2.7. Antimicrobial Drugs and Sensitivity.....	16
3. EXPERIMENTAL STUDY AND METHODOLOGY.....	18
3.1. Materials.....	18
3.1.1. Transport of Urine Specimen.....	18
3.1.1.1. The apparatuses.....	18
3.1.1.2. The equipments.....	19
3.1.1.3. Chemical agents.....	20
3.1.1.4. Culture Media.....	21
3.1.1.4.1. Nutrient agar.....	21
3.1.1.4.2. MacConkey agar.....	22
3.1.1.4.3. Blood agar base.....	22
3.1.1.4.4. Mannitol salt agar.....	22
3.1.1.4.5. Mueller Hinton agar.....	23
3.1.1.4.6. Eosin methylene blue (EMB) agar.....	23
3.1.1.4.7. Triple sugar iron agar.....	24
3.1.1.4.8. Simmon's citrate agar.....	24
3.1.1.4.9. Urea agar base.....	24

3.1.1.4.10. Methyl Red-Voges Proskauer (MR-VP) medium	25
3.1.1.4.11. <i>Salmonella Shigella</i> (S.S.) agar	25
3.1.1.4.12. Xylose-Lysine-Deoxycholate (X.L.D.) agar	25
3.1.1.4.13. Tryptone broth (Tryptone Water).....	25
3.1.1.5. Reagents.....	26
3.1.1.5.1. Kovac's Indole reagent	26
3.1.1.5.2. Methyl red reagent.....	26
3.1.1.5.3. Voges-Proskauer reagents.....	26
3.1.1.5.4. Oxidase reagent	26
3.1.1.5.5. Catalase reagent.....	26
3.1.1.5.6. Nitrate reduction test reagents.....	27
3.1.1.6. Gram stain.....	27
3.1.1.7. Antimicrobial discs	27
3.1.2. Study area	28
3.1.3. Specimens collection.....	28
3.1.4. Specimens collection.....	28
3.1.5. Sterilization of media and materials.....	29
3.1.5.1. Autoclaving.....	29
3.1.5.2. Dry sterilization.....	29
3.1.6. Processing of urine samples	29
3.1.6.1. Urinalysis	29
3.1.6.2. General urine examination.....	29
3.1.6.3. Gram stain smear.....	29
3.1.7. Urine culture	30
3.1.8. Identification of pathogens	30
3.1.8.1. Morphology	30
3.1.8.2. Microscopically.....	30
3.1.8.3. Biochemical tests	30
3.1.8.3.1. Indole test	30
3.1.8.3.2. Methyl Red-Voges -Proskauer tests.....	30
3.1.8.3.3. Citrate utilization test	31
3.1.8.3.4. Urea utilization	31
3.1.8.3.5. Motility test.....	31
3.1.8.3.6. Oxidase test.....	31
3.1.8.3.7. Catalase test.....	32

3.1.8.3.8.Coagulase test	32
3.1.9.Identification of isolated bacteria by Vitek 2 Compact System	32
3.1.9.1.Suspension preparation.....	33
3.1.10.Antimicrobial sensitivity testing	33
4.RESULTS AND DISCUSSIONS	36
4.1 Incidence of Urinary Tract Infections.....	36
4.2 Incidence of The Isolated Pathogens Associated with UTIs.....	37
4.3 Incidence of UTIs in Relation to Sex and Age Groups	39
4.4 Frequency of Clinical Symptoms in Positive Urine Culture Patients.....	40
4.5 Incidence Rate of UTIs According to the Residency of the Patients	40
4.6 Antimicrobial Susceptibility	40
5.CONCLUSIONS	56
5.1.Conclusions	56
5.2.Suggestions	56
REFERENCES.....	58
CURRICULUM VITAE (CV).....	69

LIST OF TABLES

	<u>Page No</u>
Table 3.1 The apparatuses used in the present study.	18
Table 3.2 The equipments used in the present study.	19
Table 3.3 Chemical agents and stains used in the present study.	20
Table 3.4 Culture media used in the present study.	21
Table 3.5 The antibiotics that commonly used in the treatment of UTIs.	27
Table 3.6 Zone-diameter interpretive standers according to (CLSI).	34
Table 4.1 Distribution of patients with UTIs	36
Table 4.2 pathogens obtained from urine culture.	37
Table 4.3 Frequency of UTIs according to the gender.	39
Table 4.4 shows the age and gender distribution of patients suffering from UTIs.	39
Table 4.5 Prevalence of UTI in relation to the residency	40
Table 4.6 Antimicrobial susceptibility of isolates pathogens to amoxicillin	42
Table 4.7 Antimicrobial susceptibility of isolates pathogens to amoxicillin-clavulanic acid.	43
Table 4.8 Antimicrobial susceptibility of isolates pathogens to ciprofloxacin	44
Table 4.9 Antimicrobial susceptibility of isolates pathogens to cefadroxil	45
Table 4.10 Antimicrobial susceptibility of isolates pathogens to cefotaxime.	46
Table 4.11 Antimicrobial susceptibility of isolates pathogens to ceftriaxone	47
Table 4.12 Antimicrobial susceptibility of isolates pathogens to nalidixic acid.	48
Table 4.13 Antimicrobial susceptibility of isolates pathogens to nitrofurantoin	49
Table 4.14 Antimicrobial susceptibility of isolates pathogens to norfloxacin	50
Table 4.15 Antimicrobial susceptibility of isolates pathogens to Chloramphenicol.	51
Table 4.16 Antimicrobial susceptibility of isolates pathogens to Cephalixin.	52

LIST OF ABBREVIATIONS

UTIs:	Urinary Tract Infections
STD:	Sexually Transmitted Disease
HAIs:	Health Care Associated Infections
ICU:	Intensive Care Units
ASB:	Asymptomatic Bacteriuria
<i>E. coli:</i>	<i>Escherichia coli</i>
LTC:	Long-Term Care
APN:	Acute Pyelonephritis
VUR:	Visicoureteric Reflux
UEC:	Uroepithelial Cell
SPA:	Supra Pubic Aspiration
MSU:	Mid-Stream Urine
CNS:	Coagulase-Negative <i>Staphylococcus</i>
MRSH:	Methicillin- Resistant <i>S. Haemolyticus</i>
S.S. agar:	<i>Salmonella shield</i> Agar
X.L.D. agar:	Xylose-Lysine-Desoxycholate
MR-VP:	Methyl Red-Voges Proskauer
EMB agar:	Eosin Methylene Blue
µg:	Microgram
GN ID:	Gram Negative Identification Cards
GP ID:	Gram Positive Identification Cards
CFU:	Colony Forming Unit
UPEC:	Uropathogenic <i>Escherichia Coli</i>
<i>S.aureus:</i>	<i>Staphylococcus Aureus</i>

1 INTRODUCTION

1.1 General

Urinary system is where the blood is purified by the removal of the metabolic waste and excreted as a fluid out of the body through urination. All parts of the urinary tract above the urethra in healthy person's body are sterile (VanDeGaaff, 2011)

Urinary system infection (UTI) is the most common micro bacterial infection all around the world and it accounts for 1-3% of consultation (Davidson practical medicine, 21st Edition). Rectal flora may enter the urinary tract and cause UTI in healthy person (Handley et al., 2002). Up to half of females have UTI and 3% of women have UTI at the age 20, increasing by about 1% in each following decade (Davidson practical medicine, 21st Edition). In male urinary, the disease or the infection is infrequent but that does not include the first year of the life and above the age of 60 (Handley et al., 2002).

UTI is a kind of infection that is resulted from any kind of bacteria going into the urinal system and growing anywhere in the urinary tract. The urinary tract includes organs which collect and keep the urine and release it outside of the body, and that system comprises the kidneys, ureters, bladder and urethra. UTIs are among the most widespread micro bacterial infections in human beings. Both the community and the private hospitals have reported the disease in all age groups in both male and female and young patients (Faried, 2012).

UTIs are less common in men, since the urethral length prevents bacterial colonization in the bladder and thanks to the antibacterial activity of prostatic fluid. Other clinical factors including anatomical variations, hormonal influences, and behavior of patterns may cause UTIs (Hooton, 2000; Hindi *et al.*, 2013).

UTIs remain the most common bacterial infection in childhood; if it is not diagnosed and treated quickly, it can lead to dangerous problems such as the renal scarring and kidney failure (Al-Madani *et al.*, 2007).

Even though a big amount of etiology is involved with Urinary system infection, *E. coli* and other coliforms account for a huge part of naturally acquired

urinary system bacterial infections. They are the most repeated reason for the cause of nosocomial infections in a lot of hospitals. Bacteriological investigations of UTI cannot be done without an antibiotic sensitivity test of the isolate. The bacteria causing UTI are different in their susceptibility to antimicrobials from different places and times (Vergidis and Patel, 2012).

The microorganisms that cause UTIs are common in stool. When bacteria move through the urethra to urinary tract and start to multiply, its increasing number will cause UTI. Although the urinary system is designed to keep out such microscopic agents that invade the bladder, these defenses sometimes fail. When that takes action, bacteria will grow into a fully-grown infection in the urinary tract. The most frequently found way to UTIs is the ascension of the bacteria through the urethra to the bladder (Kasper *et al.*, 2005).

The diagnosis of urinary tract diseases is based on the symptoms felt by the patient, pyuria, bacteriuria, blood analysis, and radiation or ultrasound exams. Pyuria and bacteriuria are very important not just to diagnose, but also to assess the effectiveness of treatment in Japanese standards. For assessing drug efficiency, pyuria and bacteriuria were changed to harmonize with the guidelines of the American and European guidelines (Bergan, 1997).

UTIs are very common diseases caused by bacterial infections which lead patients to obtain medicare. It has been estimated that in the United States, 7 million outpatient visits, one million visits to the emergency department and 100,000 admissions to hospital occur every year as a result of UTIs (Forbes *et al.*, 2007; Chamberlain, 2009).

UTI can influence the lower and sometimes also the upper urinary tracts. The term inflammation of the bladder infection is used to identify the cystitis and is distinguished by symptoms such as dysuria, frequency, urgency, and supra pubic pain. The existence of symptoms of lower urinary tract infection does not exclude upper urinary tract infection, which often presents in most of the cases of UTI (Prakash and Saxena, 2013).

Cystitis refers to inflammation of bladder, patients suffering from cystitis complaining of dysuria, urgency and frequency. These symptoms are due to inflammation and also multiplication of bacteria in the urine and urethra. In fact, a

complex cystitis is produced by irritation of the surface of the mucous membrane of the urethra and bladder (Ryan *et al.*, 2004). Uncomplicated cystitis is the most common UTIs and is responsible of 95% of all the symptoms of a UTI (Linhares *et al.*, 2013).

Urethritis is common to patients in both sexes. It is associated with urinary tract infection in many cases and occasionally with bacterial prostatitis. Male urethritis is commonly defined as a sexually transmitted disease (STD) and is associated with urethral discharge (Probert, 2009).

A health care associated infection (HAIs) is the most common complication from hospitalized patients, and it increases the hospitalization, mortality, morbidity and additional costs associated with the disease (Geffers and Gastmeier, 2011). Urinary tract infections are commonly found in Hospital patients. It has been reported that in hospitals in the United States, between adults and children outside of an intense kind of care units (ICU), the urinary tract is a widespread site of HAIs, representing a 36% of the cases of infection, followed by surgical site infection (20%), and bloodstream infections and pneumonia (11%) and other infections (22%) (Klevens *et al.*, 2007).

Cure for UTI cases sometimes begins with experimentation. Medication is based on the knowledge specified from the antimicrobial resistance pattern of the bacteria in the urine (Wilson and Gaido, 2004). However, disorderly employed antibiotics have contributed to a large percentage of the resistant bacterial infection. Consequently, the spread of antimicrobial resistance among the diseases of urinary pathogens has been growing all over the world (Beyene and Tsegaye, 2011).

Many strains of bacteria were developed that cause UTI usually resistant to various types of antimicrobial agents such as amoxicillin, ampicillin (Sweih *et al.*, 2005). Since ampicillin and amoxicillin have been regarded as the main stay of oral treatment for community acquired UTI in many years so this caused increase in the resistance to this antimicrobial. Early treatment of UTI with the suitable antibiotics can reduce the rate of mortality, morbidity and any renal injury from acute UTI (Francesco *et al.*, 2007).

There are many different kinds of microorganisms which can infect the urinary tract, but bacteria are the most common microorganisms responsible for UTI

(Maskell, 1995; Kunin and Calvin, 1997), and the majority is gram negative bacilli. *E.coli* causes approximately 80% of acute infection in patients without urologic abnormalities or calculi. Other gram-negative rods, such as *Klebsiella*, *Enterobacter*, *Proteus*, *Serratia*, and *Pseudomonas* cause the remaining part of uncomplicated infection but they participated largely in causing recurrent infection, calculi or obstruction. *Proteus* and *Pseudomonas* play a major role in hospital infections and are most likely to be associated with urinary tract abnormalities (Stamm and Turck, 1991).

Gram-positive cocci can also cause UTI such as *Staphylococcus epidermidis*, *Staphylococcus aureus*, *Staphylococcus haemolyticus*, and *Enterococcus faecalis* can cause acute urinary symptoms (Moyheanglam et al., 1988). Gram-positive bacteria are found more often as etiologic agents of UTI. Symptoms associated with uncomplicated UTI caused by Gram-positive pathogens are similar to those caused by Gram-negative organisms and usually include dysuria, urinary frequency, urinary urgency, and/or suprapubic pain. Fever, chills, flank pain, and/or nausea are suggestive of upper urinary tract (kidney) involvement (Hooton, 2012)

2 LITERATURE REVIEW

UTI causes a great deal of discomfort and inconvenience to the patient and in some cases responsible for the asymptomatic or the appearance of more serious cases, such as sepsis and death. UTI is considered one of the most important causes of the imbalance and it can infect people in both sexes at all ages (Abbas and Naji, 2010).

Urinary System Infection are the second most common infections in community practice, and it is estimated that they affect up to 150 million individuals per year (Mishra et al., 2013). There are limited data on the real impact of UTI in the developing world. However, in the United States, UTI has been found to be responsible for 7 million physician visits and more than 100,000 hospital admissions each year, costing the global economy in excess of one billion US dollars (Renuart et al., 2013).

2.1 Urinary Tract Infection

2.1.1 Definition

Ouno et al. (2013), in Kenya demonstrated that UTI defined as a condition caused by pathogenic organism invasion of the urinary system which leads to an inflammation in the urothelium. The proliferation of bacteria in the urinary tract leads to UTIs. A urinary tract infection (UTI) is an inflammation in any part of your urinary system your kidneys, ureters, bladder and urethra. Most infections involve the lower urinary tract the bladder and the urethra (Faried, 2012).

2.1.2 Etiology

Jha and Bapat (2005) in Kathmandu, Nepal showed that among five hundred samples of patient's urine from five hospitals collected from January 2005 to April 2005, 244 samples were found to be positive. The most common bacterial isolate was *E. coli* (49%), followed by *Staphylococcus aureas* (23%), *Proteus sp.* (3.6%), *Klebsiella sp.* (9.71%), *Pseudomonas aeruginosa* (0.8%) and *Citrobacter sp.* (2.8%). The study showed that UTI was more common in females of younger age groups as compared to males.

Arslan et al. in (2005) conducted a study on a total of 611 Gram-negative isolates; 321 were separated from uncomplicated UTI and 290 were isolated from complicated UTI, in order to determine the risk factors for community-acquired ciprofloxacin resistant *E. coli* strains isolated from UTIs in Turkey. *E. coli* was the causative agent in 90 % of the uncomplicated UTI and in 78 % of the complicated UTI. Moreover, 17 % of *E. coli* separated from uncomplicated cases and 38% of *E. coli* isolated from complicated UTI was found to be resistant to ciprofloxacin.

2.1.3 Manifestation

There is a wide spectrum of effects in the UTI ranging from patients who are completely asymptomatic to those with symptoms referable to the urethra, bladder, and those who have acute pyelonephritis with acute flank pain, tenderness and high Temperature as well as with the signs and symptoms of urethritis and cystitis (Schrier, 2007).

Clinical manifestations of UTIs are variable, about 50% of infection did not produce recognizable illness and are discovered incidentally during a general medical examination. The symptoms of UTIs in infants including fever and vomiting. The manifestation in old children and adult, when the symptoms present often suggest the diagnosis (Ryan et al., 2004)

UTI is the most common infectious diseases in countries that are in the beginning of developing by clinicians with a seamed annual global incidence of at least 250 million. UTI affects all age groups, but females are more susceptible than males, because female have a short urethra, and they do not have the capability of prostatic secretion (Sharma and Bidwai, 2013).

Hamdan *et al.* in (2011) mentioned that UTI is not only a common infection, but also its range of clinical effect varies from asymptomatic bacteriuria (ASB) to cystitis and pyelonephritis. Females with ASB are most likely to deliver low weight birth or pre-mature infants during pregnancy.

UTIs are most common in elderly males that have an enlargement of the prostate gland and the infection usually follows instrumentation of catheter drainage (Melekos and Kurt, 2000).

2.2 Sources and Routes of Infection

Davis and Flood defined (2011) that, in most patients, pathogen microorganisms go into the urinary tract through the urethra into the bladder; this is known as the ascending way and pathogens initially adhere and grow, they subsequently colonize in urothelium of the distal urethra.

Sexual intercourse is also an important reason in the ascent of bacteria to the bladder. This was proved by study carried out by Okonko *et al.* (2010), which showed that incidence of UTIs in sexually active women was more common compared to other group of women, because sexual intercourse caused bacteria to be pushed into the urethra.

Hematogenous spread accounts for fewer than 5 percent of documented UTI. Infections, especially of *Staphylococcus aureus*, *Salmonella sp.* and *Mycobacterium tuberculosis*, can occasionally reach the kidney through the blood (Forbes *et al.*, 2007).

2.3 Classification of UTIs

Dulczak and Kirk (2005), described that the upper tract infections are located in ureters collecting system and parenchyma such as pyelonephritis. They also defined pyelonephritis as diffuse pyogenic infection of parenchyma of kidney with signs and symptoms including fever over 38.5°C, chills along with flank pain, tenderness with Pouria and positive urine culture.

National Collaborating Centre for female's and young Children's Health, London: RCOG Press; 2007. We can classify symptomizes of UTI to:

- Lower UTI or cystitis
- Upper UTI or acute pyelonephritis (APN)

But Al-Badr and Al-Shaikh (2013) in Saudi Arabia investigated that the main classification of UTIs are classified into six categories

2.3.1 Uncomplicated UTI

Mazzulli (2012), in Canada defined that the not-complicated UTI occurs in healthy patients with a structurally and functionally normal urinary tracts. He also

mentioned the uncomplicated UTIs include infections in the lower part of the urinary tract or system (cystitis) and the upper part of the urinary tract or system (pyelonephritis).

2.3.2 Complicated UTI

Study carried out by Neal (2008), in U.S.A. demonstrated that complicated UTI occurs in patients with underlying anatomical or functional abnormal urinary tract due to intrinsic or extrinsic factors and usually needed a prolonged course of antibiotics.

2.3.3 Isolated infection

Isolated infection is a term used to describe UTIs as a first infection which isolated from patients, which can be treated once, and does not appear in less than six months. Isolated infections can affect about 25-40% of young women (Al-Badr and Al-Shaikh, 2013).

2.3.4 Unresolved infection

Sometimes UTI is caused by unresolved infection because of inadequate antimicrobial therapy, either because of resistance of bacteria to antibiotics that cannot kill it or it is because the patient does not take the medication in the correct way (Kavaler, 2006).

2.3.5 Reinfection

Reinfection is characterized by the same or different microorganisms grow during any period of time. Reinfection occurred where it appeared that has been no growth after the infection has been treated. Ninety-five percent of UTIs are due to reinfections (Chang and Shortliffe, 2006).

2.3.6 Relapse

Relapse UTIs occurs in 5% to 10% of women with a persistence microorganism of the same species causes an UTI within two weeks of completing antimicrobial therapy (Schrier, 2007).

2.4 The Factors Predisposing to UTIs

There are many factors that are causing UTIs. Some of them are given below:

2.4.1 Vesico-Ureteric reflux

Administration of VUR is argumentative and includes long-term antibiotic protection of UTI, surgical ways for the VUR, or surveillance only. VUR Partially has a role in the development and progression of pyelonephritis. Childhood VUR is five times more common in girls than boys and it has a genetic background (Mattoo, 2012; Chen *et al.*, 2013).

2.4.2 Prostatic hypertrophy

The incidence of UTI in men in their fifth decade and older is very common due to enlargement of the prostate gland. This can cause obstruction to the flow of urine with deficient voiding resulting in remaining urine in the bladder. Prostatic hypertrophy recognized as a risk factor for UTI (William *et al.*, 2006).

2.4.3 Diabetes mellitus

Hoepelman *et al.* (2003), in Netherland found that both gender with diabetes mellitus associated with increased risk of acute pyelonephritis or acute cystitis. Also they can see that the adherence of *Escherichia coli* to uroepithelial cells of females with diabetes mellitus is increased compared with females without diabetes mellitus, and as we can see that the glycosuria promoted the growth of various strain of *Escherichia coli*. Some data are available related to the role of diabetes mellitus as a risky factor that effects the development of antimicrobial resistance of the pathogens (Bonadio *et al.*, 2006).

2.4.4 Urolithiasis

The relation between urinary stones and UTI is frequent. Kidney stones can act as a place in which microorganisms can escape antibiotics and cause recurrent UTI (Chamberlain, 2009). Infection by urease enzymes producing organisms such as *Proteus mirabilis* can stimulate the failure of urea into ammonia and carbon dioxide.

The ammonia produced from this kind of interaction raise the pH of the urine and cause formation of kidney stones (Coker *et al.*, 2000).

2.4.5 Pregnancy

Anyone can get a urinary tract infection, but it is most common in women, especially if they are pregnant. Partly due to the pressure of the pregnant uterus from the infant that affects the ureters, increases the bladder volume and decreases the bladder tones, elevating the risk of urinary stasis. This effect may also be due to the humoral and immunological changes that happen normally during pregnancy (Emiru *et al.*, 2013).

2.4.6 Age and gender

Prakash and Saxena (2013), in India showed that from a total of 288 urine samples collected from patients of the age ranging from 15 to ≥ 48 years, 140 were females while 148 were males. Prevalence of UTI was 53.83% in patients, and the UTI prevalence was conspicuously higher in females 73.57% than males 35.14%. They also found that the highest prevalence of UTI in males was ≥ 48 years (71.15%), while in females the highest susceptible age group of 26-36 years (90.69%).

2.4.7 Genetic factors

The research has demonstrated that women with certain antigens in the blood (called lewis group) are most susceptible to cystitis. The cells lining the urinary tract appeared to be more receptors to which bacteria can adhere. It has been shown that non-secreting blood group antigens are at risk of Recurrent UTI. Other glycosaminoglycan layer may be insufficiency, these layers have the proteins that will snare the bacteria and evacuate it, occur on the bladders surface. If the layer is not intact or sometimes the proteins are not viscous enough, the bacteria will be able to sit in the bladder and proliferate. Mutations in the genes of the integrated host immune responses (interferon receptor, etc.) may also affect the susceptibility to UTI (Kasper *et al.*, 2005).

2.5 Bacterial Virulence Factors

The type 1 fimbriae promote bacteria during colonization process to attachment to mannosylated receptors that found on the UEC and overrun the UEC within the urinary system. Interactions mediated by these adhesions can induce a number of factors that can directly influence the consequence of an UTI; also, the presence of type 1 fimbriae may facilitate adherence and colonization to mucosa of the host in bladder (Mulvey, 2002).

The P fimbriae or mannose resistant pili are the second other common virulence factor, which has a significant role in pathogenesis of ascending UTIs and uncomplicated pyelonephritis in human. As the receptor for P fimbriae is the globodides of glycolipid component present on renal tubular cells. They are termed mannose resistant because during the haemagglutination of human red cells that is not altered by mannose (Davis and Flood, 2011).

2.6 General Consideration for Some Common Facultative Anaerobic and Aerobic Bacteria Causing UTIs

2.6.1 Gram negative bacteria

2.6.1.1 *Escherichia coli*

This species is considered as the most popular causative agent in 90% of community-acquired UTIs and up to half of nosocomial UTIs. UPEC, which is separated from simple community-acquired UTIs, explained a variety of virulence factors that boost the effective colonization of the urinary tract (Toval *et al.*, 2014).

Gupta *et al.* in (2001), reported that the vision of agents causing UTI has remained relatively constant. *E. coli* accounted for 75% to 90% of cases; *Staphylococcus saprophyticus* accounted for 5% to 15% (particularly in younger female); *Enterococcus sp.*, *Klebsiella sp.* and *Proteus mirabilis*, accounted for the remaining 5% to 10%.

2.6.1.2 *Klebsiella sp.*

Klebsiella is one of the Enterobacteriaceae families of bacteria. This can be recognized as a rod-shaped, not movable, gram-negative bacterium. *Klebsiella sp.* known for its polysaccharide capsule that surrounds the whole organism, *Klebsiella*

pneumoniae, making the treatment difficult. The capsule is associated with resistance to most antibiotics (Ristucci, Patricia; Cunha, Burke July 1984). All the genus of *Klebsiella* show two types of antigens, lipopolysaccharide (O-antigens) and capsular polysaccharide (K antigen) (Cullor, 1996). *Klebsiella* is also seen in the urine and is the origin of some urinary tract infections acquired in hospitals (nosocomial infection), especially in certain at-risk services like surgical services, long stays or intensive care. (Ronald, 2003).

2.6.1.3 *Proteus sp.*

Proteus mirabilis causes 90% of proteus infections. *Proteus mirabilis* is an important etiological agent of UTI. *Proteus mirabilis* typically causes uncomplicated UTIs in healthy women and less repeatedly in children. Less often, *Proteus mirabilis* can cause complicated UTIs in persons with structural, physiologic, or neurogenic disorders of the urinary tract requiring catheterization (Goldman and Green, 2009; Bahashwan and EI-Shafey, 2013; Pandey *et al.*, 2013).

2.6.1.4 *Pseudomonas aeruginosa*

This genus consists of Gram-negative, motile, aerobic rods some of which produce pigments. *Pseudomonas aeruginosa* (an important opportunist) is the major human pathogen of its genus. *Pseudomonas aeruginosa* has a characteristic grape-like or corn taco-like odor, that produces a green diffusible pigment when grow on solid media. This organism is usually resistant to the most commonly used antimicrobial therapy, it can resist many chemical disinfectants, and commonly present in moist environments in hospital. It causes disease in patients with abnormal host fortification (Brooks *et al.*, 2007).

2.6.1.5 *Citrobacter sp.*

The genus *Citrobacter* is distinct groups of aerobic, Gram-negative bacilli from the *Enterobacteriaceae* family. *Citrobacter* species account for 1 to 2% of nosocomial infection. UTI causes by *Citrobacter* species have been described in 5 to 12% of bacteriuria isolated in adults.

Citrobacter freundii and *Citrobacter koseri* (formerly *C. diversus*) cause the majority of human *Citrobacter* infection, which are similar epidemiologically and clinically to *Enterobacter* and *Acinetobacter* infections. It is generally a saprophyte in the gastrointestinal tract and they are considered opportunists which can be found

in a variety of infections, particularly UTIs. *Citrobacter freundii* is generally more resistant to antibiotics than *Citrobacter koseri*. (Kasper *et al.*, 2005; Metri *et al.*, 2013).

2.6.1.6 *Enterobacter sp.*

Enterobacter sp. is Gram-negative bacilli. *Enterobacter sp.*, in particularly *Enterobacter aerogenes* and *Enterobacter cloacae*, caused many serious life-threatening medical complications associated with nosocomial infection and damage, among which UTIs. They are morphologically and biochemically similar to *kleibsella sp.*, the difference is that they are motile (Mohammed, 2005).

2.6.1.7 *Proteus sp.*

Proteus is a genus of Gram-negative bacteria belonging to the family of Enterobacteriaceae. This species is among the commonly involved pathogens in nosocomial as well as community acquired infections. Different species of *Proteus* are encountered in human infection. *Proteus sp.* are differentiable from most other genera by their capability to swarm across an agar surface, this phenomenon is due to the swimming cells in the broth to swarmer cells in agar and is linked with the elongation of the cells and increase the synthesis of flagellin. *Proteus mirabilis* causes 90% of proteus infections. *Proteus mirabilis* is an important etiological agent of UTI. *Proteus mirabilis* typically causes uncomplicated UTIs in females with good health and less common in young children and infants. Less often, *Proteus mirabilis* can be causing complicated UTIs in people with structural, physiologic, or neurologic disorders of the urinary tract requiring catheterization (Goldman and Green, 2009; Bahashwan and EI-Shafey, 2013; Pandey *et al.*, 2013).

2.6.1.8 *Morganella morganii*

Morganella morganii (formerly *Proteus morganii*) is a Gram-negative motile rod. *Morganella morganii* strains produce individual colonies that do not have the ability to swarming-type growth; it can be found human and animal intestines. The only disease principally associated with *Morganella morganii* is UTIs (Goldman and Green, 2009). Dishvarian (2010), in Baghdad, Iraq confirmed that *Morganella morganii* causes 6.6% of UTI from patients in ages between 1-65 years old in both gender.

2.6.1.9 *Serratia sp.*

Serratia sp. is Gram-negative rods, whose natural habitat is the intestinal tract of human beings. *Serratia sp.* is nosocomial and profiteer pathogens responsible for a wide range of infections. *Serratia sp.* can cause UTI. *Serratia marcescens* is a common opportunistic pathogen in patients in hospital patients, it is often re-double resistant to aminoglycosides and penicillins, you can treat the infections with third-generation cephalosporins. *Serratia sp.* is generally resistant to cephradine, cefuroxime and amoxicillin but sensitive to ciprofloxacin, imipenem, ceftazidime and ceftazidime (Gillespie and Hawkey, 2006; Brooks *et al.*, 2007).

2.6.1.10 *Salmonella sp.*

Haematogenous infection of the urinary system is restricted to a few relatively not common microbes, such as *Salmonella sp.*, *Salmonella typhi* and *paratyphi*, are often isolated from the urine in the early stages of typhoid fever and paratyphoid fever. Urine test should be carried out in suspected cases of *Salmonella* infection. Also urine samples taken from the patient's contacts should be cultured in the medium to exclude specific injury (Probert, 2009).

2.6.2 Gram positive bacteria

2.6.2.1 *Staphylococcus aureus*

It is a species of facultative anaerobic Gram-positive bacteria, and it can be called as "golden staph". The bacteria are sometimes referred to as *S. aureus* or *Staph aureus*. *Staphylococcus* should not be bemused with the similarly named and medically relevant genus *Streptococcus*. *S. aureus* shows asgrape-like clusters when examinant it through a microscope, it will show that it has large, round, golden-yellow

Colonies, often with hemolysis, when it's growing on blood agar plates.

S. aureus reproduces asexually by binary fission. The two daughter cells will not be fully separated and remain attached to each other (Matthews, 1997) and that is why the cells are observed in clusters. *S. aureus* is catalase-positive (meaning it can produce the enzyme catalase). Catalase converts hydrogen peroxide (H₂O₂) to water and oxygen. Catalase-activity tests are used sometimes to distinguish staphylococci from enterococci and streptococci. Previously, *S. aureus* was differentiated from

other staphylococci by the coagulase test. However, it has been known that not all *S. aureus* are coagulase-positive and that incorrect species identification might and will impact effective cure and control measures. (Ryan, 2004).

2.6.2.2 *Staphylococcus epidermidis*

One of the most frequently encountered microorganisms associated with acute UTI in young sexually active female out patients is *Staphylococcus epidermidis* (Martinau *et al.*, 2000). Nowadays, they are proven to be urinary pathogens in both sexes, and it act as opportunistic pathogens, causing infection to patients with defective resistance, urinary tract abnormalities or often instrumental manipulation, and frequency of this organism in UTI varies from 0-26% (Montague *et al.*, 1995).

2.6.2.3 *Staphylococcus saprophyticus*

Staphylococcus saprophyticus is Gram-positive bacterium and that is one of the most frequently encountered pathogens associated with acute UTIs, particularly among young sexually active women. It is accounting for 5-15% of community-acquired episodes. In men, it is may also develop nonspecific urethritis. they found the difference of *Staphylococcus saprophyticus* from other urinary coagulase-negative staphylococci (i.e., *Staphylococcus epidermidis*) by its uniform resistance to novobiocin (Martineau *et al.*, 2000). It causes 7.22% of UTIs in females (Kolawole *et al.*, 2009).

2.6.2.4 *Staphylococcus haemolyticus*

Staphylococcus haemolyticus is identified as coagulase-negative staphylococci (CNS), and it doesn't harm usually commensalism are very necessary. Pathogens in such category are considered in clinical microbiology laboratories across the globe. *Staphylococcus haemolyticus* has been reported to be the second most common isolated CNS and has been isolated in UTIs. Methicillin- resistant *S. haemolyticus* (MRSH) seemed to show even higher frequencies of cross resistance. Cross resistance was most common in the MRSH, for example, rates of SXT, ciprofloxacin, and gentamicin were 64%, 50.5%, and 72.3% respectively. Multiple antibiotic resistance genes it was probably found in *Staphylococcus haemolyticus*. Most oxacillin resistance was seen in *Staphylococcus haemolyticus* (John and Harvin, 2007). Lewis *et al.* in (2013) confirmed that *Staphylococcus haemolyticus* causes 1.0% of UTI cases.

2.6.2.5 *Enterococcus sp.*

Enterococcus sp. is a type of Gram-positive bacterium causes almost exclusively infection in hospitalized patients with significant compromise of their defenses, and the primary sites are the urinary tract. *Enterococcus faecalis* is one of the most common pathogens in UTIs. *Enterococcus faecalis* is the pathogen that is most effective in the genus *Enterococcus*, causing about 95% of enterococcal infections. It is often multiply resistant to fluoroquinolone (Yasufuku *et al.*, 2011). It causes 6.2% UTIs in both genders (Shaaban *et al.*, 2012).

2.6.2.6 *Enterococcus faecalis sp.*

Enterococcus faecalis sp. is a type of Gram-positive bacterium, commensal bacterium nests the gastrointestinal tracts of human beings and other living mammals. Like other species that are in the genus *Enterococcus*, *E. faecalis* can cause infections that threaten human lives, and that happens especially in the nosocomial (hospital) environment, where the normally high levels of antibiotic resistance found in *E. faecalis* contribute to its pathogenicity (Ryan and Ray, 2004).

2.7 Antimicrobial Drugs and Sensitivity

A lot of new studies have been reported that the resistance profiles of pathogens to antimicrobial agents that normally used to cure UTI. Increasing resistance of bacterial pathogens is of worldwide concern. Pathogens may be resistant to a single or to multiple antibiotics. The treatment of UTIs varies depending on the patient's sex, age, proportion of infection, underlying disease, infecting agents and whether there is involvement of the lower urinary tract or upper (Mandal *et al.*, 2012).

Antimicrobial drugs used in treatment of UTI should compete and support natural healing by eliminating the offending microorganisms, with regard to certain factors; these are the general condition of the patient, type and localization of infection, type of microorganisms and type of the drug used. The treatment should be according to sensitivity testing (Parson, 1988).

Pathogens become resistant to antimicrobial drugs by many factors and mechanisms, the commonest being (Cheesbrough, 2006; Probert, 2009):

- a. The Product of beta-lactamase enzymes that devastate the *beta*-lactam ring of penicillin's and cephalosporin's (the most common and well-known form of resistance).
- b. Production of acetylating, adenylating and phosphorylating enzymes that disrupt antimicrobials like aminoglycosides and chloramphenicol.
- c. Changing the permeability of the outside membrane of the bacterial cell wall, as happens in resistance to tetracycline's (common form and a way to find the resistance in *P. aeruginosa*).
- d. Production of metabolic pathways that go above and beyond the site of antimicrobial action and act as in resistance to sulfonamides and trimethoprim.
- e. Transferable resistance is presented and showed with additional chromosomal plasmid-mediated factor. It is most common and known cause of resistance and can cause multiple resistances

3 EXPERIMENTAL STUDY AND METHODOLOGY

3.1 Materials

3.1.1 Transport of Urine Specimen

- a. All the containers that are used to transfer the samples were sterilized.
- b. The containers were able to resist any leaking to decrease from the loss of samples and to protect the samples from any Contamination
- c. Specimen containers were not used again.
- d. Transporting tubes were compatible with automated systems and work forms and instruments used by the lab.

3.1.1.1 *The apparatuses*

The apparatuses used for preparing the appropriate experiments in the present study that we can see in table (3.1).

Table 3.1 The apparatuses used in the present study.

Name	Company	Country
Bunzen bernor	Flamefast	England
Vaccum pump Filtration	Rocker Scientific Co.	Taiwan
Table lamp	Turned	Turkey
Refrigerator	BEKO	Turkey
Digital timer	Hengyi Industry Co.	China
UV lamp trolley air purification	DHgate	China
Camera	Sony	Japan
Centrifuge	Kokusan	Japan
Microscope	Genex Laboratories	U.S.A.
pH meter	HANNA	U.K
Vitek 2 Compact system	Biomeriux	French
Electronic balance	PSAW	India
Microbiology safety cabinat	DLabTech	Korea

Water distillator	DLabTech	Korea
Micropipette different volumes	Eppendorf	Germany
Autoclave	Witeg laborotechnik	Germany
Incubator	Memmert	Germany
Magnetic stirrer hot	IKA Laborotechnik	Germany
Water bath	Memmert	Germany
Electric oven	Melag	Germany

3.1.1.2 *The equipments*

The tools and equipments used for preparing the convenient experiments in the present study are shown in table (3.2).

Table 3.2 The equipments used in the present study.

Name	Company	Country
Cotton	NCPI	Iraq
Urine cup	AFCO	Jordan
Loop	HiMedia	India
Forceps	Vantage	Pakistan
Uripath	Plasmatic	U.K
Durham tube	Marienfeld	Germany
Cover slide	Beromed GmbH	Germany
Filter papers (0.4) microns	Schleicher&Schuell	Germany
Conical flask different size	Bro3.3	Germany
Beaker different size	Bro3.3	Germany
Graduated cylinder	Bro3.3	Germany
Microscope slide	Beromed GmbH	Germany
Disposable swab	Citotest	China
Pediatric urine collector	Citotest	China
Wooden stick	Citotest	China
Centrifuge tube	Citotest	China
Vernier	Anyi Instrument Co.	China

Micropipette tips	Plasti Lab	Lebanon
Aluminum foil	Sanita	Lebanon
Petri dishes	Plasti Lab	Lebanon
Thermos	KST	U.S.A.
Parafilm	Bemis	U.S.A.

3.1.1.3 *Chemical agents*

The chemical agents used for preparing the convenient experiments in the present study that you can see in table (3.3).

Table 3.3 Chemical agents and stains used in the present study.

Name	Company	Country
Normal saline	Pioneer	Iraq
Gram stain	Syrbio	S.A.R.
Oil emmersion	HiMedia	India
Tetra methyl-p-phenylene diamine dihydrochloride	HiMedia	India
Kovac's Indole reagent	HiMedia	India
Urea crystal	HiMedia	India
Hydrogen peroxide	BDH	England
Sulfuric acid (H ₂ SO ₄)	BDH	England
Glacial acetic acid	BDH	England
Potassium hydroxide	BDH	England
Dihydrate barium chloride (BaCl ₂ .2H ₂ O)	BDH	England
Ethanol 95%	BDH	England
Zinc powder	Fluka	Switzerland
α-naphthol	Fluka	Switzerland
Methyl red	Fluka	Switzerland
Sulfanilic acid	Fluka	Germany
alpha-naphthylamine	Fluka	Germany

3.1.1.3. Culture Media

The culture media used for identification of bacterial uropathogens in the present study that we can see in table (3.4).

Table 3.4 Culture media used in the present study.

Culture Media	Company	Country
Nutrient agar	HiMedia	India
MacConkey agar	HiMedia	India
Blood agar base	HiMedia	India
Mueller Hinton agar	HiMedia	India
Eosin methylene blue (EMB) agar	HiMedia	India
Mannitol salt agar	HiMedia	India
Methyl Red-Voges Proskauer (MR-VP) medium	HiMedia	India
Salmonella Shigella (S.S.) agar	Lab M	England
Urea agar base	HiMedia	India
Simmon's citrate agar	HiMedia	India
Triple sugar iron agar	HiMedia	India
Xylose-Lysine-Desoxycholate (X.L.D.) agar	Oxoid	England
Nitrate broth	HiMedia	India
Motility test medium	HiMedia	India
Tryptone broth	HiMedia	India

*The composition and methods of preparation of different media used in this study are shown below, performed as instructed by the industrialist.

3.1.1.3.1 Nutrient agar

Used for dilution of bacterial culture and cultivation of culture isolates. It can be done as instructed by the manufacturer by suspending 28 grams in 1000 mL distilled water then mixed thoroughly, slowly and gently exposed to heat till it reaches a boiling status to dissolve the medium fully. Dispense it as desired and sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes. Cooled it to

45°C–50°C and mixed well before pouring into sterile Petri dishes. This medium was stored at 4°C.

3.1.1.3.2 MacConkey agar

MacConkey agar is a differential and selective medium used to isolation of Gram-negative organisms and also used to separate between lactose-fermenter and non-lactose-fermenter bacteria. Lactose-fermenting bacteria will be shown as red to pink colonies. Lactose non-fermenting bacteria will be shown as colorless or transparent colonies (Atlas, 2010).

This medium was prepared by suspending 49.53 grams of dehydrated medium in 1000 mL of distilled water and it was gently heated while stirring until it gets boiled to dissolve the medium completely. Then it is sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes. Overheating was avoided. It was cooled to 45-50°C and mixed well before pouring into sterile Petri plates. The surface of the medium was dried when inoculated. This medium was stored at 4°C.

3.1.1.3.3 Blood agar base

It is a different and enriched media supported with blood, promote the growth of most Gram-positive and Gram-negative bacteria are also used for the right way, cultivation, and detection of hemolytic activity of staphylococci, streptococci, and other fastidious microorganisms (Atlas, 2010).

This medium was made by suspending 40.0 g in 1000 mL of distilled water and sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes. Then, it was cooled to 50°C and %5 sterile defibrinated blood was aseptically added to it. It was mixed carefully and poured into sterile Petri dishes. The medium was stored at 4°C.

3.1.1.3.4 Mannitol salt agar

Mannitol salt agar contains the carbohydrate mannitol and 7.5% NaCl, which is inhibitory to most bacteria because they cannot survive in this level of salinity. It is used for isolation and finding the difference of pathogenic staphylococci bacteria, especially *Staphylococcus aureus*. It ferments mannitol and it might produce acid, which turns the pH indicator from red to yellow. Another staphylococcal species

may grow, but they cannot ferment mannitol, so no color change occurs (Alexander and Strete, 2001).

This medium was prepared by suspending 111.02 grams in 1000 mL of distilled water, then gently heated while stirring until to boiling to dissolve the medium totally and sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes. When the medium has cooled to 50–55°C, it was mixed well and dispensed aseptically in sterile Petri dishes. This medium was stored at 4°C.

3.1.1.3.5 Mueller Hinton agar

Used in the study of bacterial sensitivity to antibiotics (Stacher, *et al.*, 1993). This medium is used for antibiotic disk diffusion sensitivity testing by the Bauer-Kirby method, as well as observing fluorescent pigment that is produced by *Pseudomonas aeruginosa*. It is prepared by suspending 38.0 grams in 1000 mL of distilled water and distributed into tube or flasks. They were gently heated and brought to boiling then sterilized by autoclaving at 15 lbs pressure (121 °C) for 15 minutes. After autoclaving, it was poured into sterile Petri dishes and stored at 4°C (Cavalieri, *et al.*, 2005).

3.1.1.3.6 Eosin methylene blue (EMB) agar

Used for isolation and differentiation of *E.coli*, coliform bacterium *E.coli* appear as colonies will a green metallic sheen or blue black to brown color. (Atlas, *et al.*, 1995).

EMB agar is prepared by suspending 35.96 grams in 1000 mL of distilled water and mixing until suspension is uniform. It is heated to its boiling point to dissolve the medium totally and sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes. Too much heating was avoided. Then the medium was cooled to 45-50°C and shaken in order to oxidize the methylene blue and to suspend the flocculent impetuous. After mixing, it was dispensed aseptically in sterile Petri dishes. The medium was stored at 4°C.

3.1.1.3.7 Triple sugar iron agar

Triple sugar iron agar can be used to define Gram-negative enteric bacilli on the basis of dextrose, lactose and sucrose fermentation, gas production and hydrogen sulphide production (Forbes *et al.*, 2007).

This medium is made as a shallow agar slant with a deep butt, and that provides both aerobic and anaerobic growth environments. It is prepared by suspending 64.52 g in 1000 mL of distilled water and the medium was heated to till it reached to a boiling status to dissolve the medium completely. Then it was mixed well and distributed into test tubes, sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes. It was allowed to set in sloped form in tube with a butt about 1 inch long. The medium was stored at 4°C.

3.1.1.3.8 Simmon's citrate agar

This medium was used to test the ability of microorganism to utilize citrate as the sole carbon and energy and ammonia salt as the sole source of nitrogen.

Simmon's citrate agar you can prepare it by suspending 24.28 g of the medium in 1000 mL of distilled water and mixing well and distributing it in flasks. It was then heated to disband it completely and sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes. It was cooled and distributed in the tubes in an oblique position, this medium was stored at 4°C.

3.1.1.3.9 Urea agar base

This medium was used for the identification organisms that possess the enzyme ureases. It can be prepared by suspending 24.01g of dehydrated urea agar foundation in 950 ml cold water that must be distilled and boiled to dissolve the medium completely. This solution was then sterilized by autoclaving at 15 lbs pressure (115°C) for 20 minutes and cooled to about 50°C and aseptically added 50 ml of filter sterile 40% urea solution per 950 ml basal medium and mixed well, dispensed it into sterile tubes and allow setting in the slanting position. Do not heat it too much or reheat the medium because urea has an easy way to decomposes. This medium was stored at 4°C.

3.1.1.3.10 Methyl Red-Voges Proskauer (MR-VP) medium

MR-VP Medium is better for the rendering of the Methyl Red and Voges-Proskauer tests in differentiation of the enteric bacteria and to improve it (Goldman and Green, 2009).

This medium was prepared by suspending 17 g in 1000 ml of water and boiled to disband the medium completely. This solution was then sterilized by autoclaving at 15 lbs pressure (121°C) for 20 minutes. This medium was stored at 4°C.

3.1.1.3.11 *Salmonella Shigella* (S.S.) agar

S.S. agar is a differential selective media used for the isolation of *Salmonella sp.* and some *Shigella sp.* from pathological specimens (Chessbrough 1992, Perilla *et al.*, 2003).

S.S. agar was prepared by suspending 63.02 g in 1000 ml and then it was boiled with frequent agitation to dissolve the medium completely. It was autoclaved or overheated because heating it too much might have destroyed selectivity of the medium. It was then cooled to about 50°C by mixing. The medium was poured into sterile Petri plates. This medium was stored at 4°C.

3.1.1.3.12 Xylose-Lysine-Deoxycholate (X.L.D.) agar

It was a selective medium for the isolation of Gram-negative bacteria from clinical specimens because this media contains deoxycholate, which is inhibitory to Gram-positive bacteria (Alexander and Strete, 2001).

It was prepared by suspending 53g in 1 liter of distilled water and heating until the medium boils to frequent agitation. It was not overheated or autoclaved. The mixture was transferred to a water bath at 50°C, and as soon as it was cooled down it was poured into sterile Petri dishes and stored at 4°C.

3.1.1.3.13 Tryptone broth (Tryptone Water)

This can be used for the detection of indole production by coliforms because it contains a great deal of tryptophan (Benson, 2001).

It was made by suspending 15 grams in 1000 mL of distilled water and heating if necessary to disband the medium completely. Then it was sterilized by autoclaving at 15 lbs pressure (121°C) for 15 minutes.

3.1.1.4 *Reagents*

3.1.1.4.1 **Kovac's Indole reagent**

Formulation per 100 mL

P-Dimethyl amino benzaldehyde 5 gm

Hydrochloric acid, concentrated 25 ml

Amyl alcohol 75 ml

3.1.1.4.2 **Methyl red reagent**

Formulation per 100 mL

Methyl red 0.02gram

Ethanol 95% 60.0 ml

Sterile deionized water 40.0 ml

3.1.1.4.3 **Voges-Proskauer reagents**

It can consist two solutions:

- a. Solution A is made by dissolving 6 grams of α -naphthol into 100 ml of 95% ethyl alcohol.
- b. Solution B is made by dissolving 16 grams of potassium hydroxide into 100 ml of distilled water.

3.1.1.4.4 **Oxidase reagent**

It is consisting of dissolving one gram of tetra-methyl p-phenylene-diamine in 100 ml distilled water.

3.1.1.4.5 **Catalase reagent**

It is consisting of 3% Hydrogen peroxide.

3.1.1.4.6 Nitrate reduction test reagents

This reagents used for developing the nitrate reduction tests, it consists of following (Perilla *et al.*, 2003):

- a. Nitrate reagent solution A is made by dissolving 0.5 grams of sulfanilic acid in 30 ml of glacial acetic acid, then adding 100 ml of distilled water. It should be Stored at room temperature in dark place or room.
- b. Nitrate reagent solution B is made by disbanding 0.1 grams of alpha-naphthylamine in 100 ml of boiling distilled water. Cooling to room temperature then adding this suspension to 30 ml of glacial acetic acid.
- c. Zinc powder.

3.1.1.5 Gram stain

It is used in the identification of bacterial shape and the typing of bacteria into the Gram-negative and Gram-positive bacteria.

3.1.1.6 Antimicrobial discs

In vitro tests that measure the growth of an isolated microbe and bacteria in the presence of particular kind of drug or drugs (antibiotics) in order to predict the in vivo success or failure of antibiotic therapy

The results - guide the choice of antibiotics (+ clinical information and experience). The antibiotics that commonly used in the treatment of UTIs and used in this study as shown in table (3.5).

Table 3.5 The antibiotics that commonly used in the treatment of UTIs

Antibiotics discs	Symbol	Conc. (μg)	Company/country
Ciprofloxacin	CIP	10	Bioanalyse/Turkey
Ceftriaxone	CRO	10	Bioanalyse/Turkey
Chloramphenicol	C	30	Samara/ Iraq
Cefadroxil	CFR	30	Bioanalyse/Turkey
Amoxicillin-clavulanic acid	AMC	30	Bioanalyse/ Turkey
Cefotaxime	CTX	10	Bioanalyse/Turkey
Nalidixic acid	NA	30	Bioanalyse/Turkey

Nitrofurantoin	F	100	Bioanalyse/Turkey
Norfloxacin	NOR	10	Bioanalyse/Turkey
Amoxicillin	AX	25	Bioanalyse/Turkey
Cephalexin	CL	30	Oxoid/Basingstoke

3.1.2 Study area

This study got carried out in Erbil, also known as Hewler is the capital state of Northern Iraq. It is located in northwestern side of Baghdad, with a population of approximately 1.61 million. The city of Erbil hosts a number of hospitals so is there a number of cases more satisfactory for study.

3.1.3 Specimens collection

A total of 500 patients with clinical symptoms of UTI referred from patients attending Arbil General Hospital in Arbil city from 1/11/2015 to 1/6/2016. Samples had been taken before antibiotic use. A questionnaire list was already prepared to report all information's concerning the patient's clinical symptoms, age, sex, geographical distribution and level of education.

3.1.4 Specimens collection

When collecting urine samples, precautions should be taken to avoid pollution by bacteria from the distal urethra. The patients were asked to clean their external genitalia. They were asked to discard the first part of the urine and to collect the mid-stream in the sterilized container. The clean catch midstream urine samples that obtained after proper cleaning of the perianal and urethral were analyzed within 2 hrs. of collection, or be kept refrigerated at 4°C until delivery to the laboratory in order to ensure that the pathogenic organisms present in the urine were isolated and also to avoid contamination and overpopulation of the pathogenic organisms. All samples were coded and processed at the time of collection according to the standard method.

3.1.5 Sterilization of media and materials

3.1.5.1 Autoclaving

All media were sterilized by autoclaving at 121°C and under 1.5 bar for 15-20 minutes, except for sugar containing media which were sterilized just for 10 minutes.

3.1.5.2 Dry sterilization

All glassware were washed then the glassware's were sterilized by a hot air oven at 200°C for two hours.

3.1.6 Processing of urine samples

3.1.6.1 Urinalysis

Urinalysis is very important in the diagnosis of urologic conditions can alert and warn the physician to the presence and appearance of systemic disease that's affecting the kidneys such as calculi, urinary tract infection (UTI), and malignancy.

3.1.6.2 General urine examination

The microscopic exam was done on the urine that has been centrifuged to focus the substances in it at the bottom of a tube. The liquid at the top of the tube was then discarded and then the drops of fluid remaining were examined by a microscope for detection of pus cells, erythrocytes, casts, crystals and other such as mucous, bacteria and parasites.

3.1.6.3 Gram stain smear

A staining technique was used to classify bacteria. A drop of an uncentrifuged urine was swelling mixed and it was placed on a slide and then the drop was allowed to dry and should not be spread. Later it was heated, fixed and stained. They were examined under an oil-immersion lens for the presence or absence of microorganisms and polymorphonuclear leukocytes. One or more microorganism's cells per oil-immersion field normally implies that there are 10 or more micro bacteria per milliliter in the specimen and presence of one or many leukocytes per oil-immersion is another indication of UTI (Vandepitte *et al.*, 2003).

3.1.7 Urine culture

The agars were prepared to the bacteriological and biochemical tests using a standard procedure. It was done after the plates were kept in the incubator at 35-37°C for 18-24 hours. The number of isolated bacterial colonies was multiplied by 1000 for the estimation of bacterial colony forming/ml of the urine samples. A specimen was positive for UTI if an organism was cultured at a concentration of 10⁴ CFU/ml. If no growth appeared they were further incubator for another 24 hours before regarded as a negative.

3.1.8 Identification of pathogens

3.1.8.1 Morphology

The isolated bacterial colonies were identified according to the morphology, pigment production, fermentation and haemolysis on the blood agar.

3.1.8.2 Microscopically

The bacterial isolates were further classified by Gram-staining to Gram-negative and Gram-positive bacilli and cocci.

3.1.8.3 Biochemical tests

The predominant isolated bacterial colonies were tested for their biochemical characteristics as follows:

3.1.8.3.1 Indole test

Tryptophane broth was inoculated with the test organism ability to breakdown the amino acid tryptophan by the enzyme tryptophanase to form indole. The isolates were inoculated into tryptone broth and then we it was placed in the incubator at 35°C for 24-48 hours. After incubation, only five drops of Kovac's reagent were added to the broth. The positive result would be a development of a red/pink ring on top of the media. Appearance of no red layer was a negative result.

3.1.8.3.2 Methyl Red-Voges -Proskauer tests

2 glucose broths with inoculating loop were inoculated. It was left for 48 hours of incubation, then just a few drops of MR were added to one tube, and VP reagents to the other tube.

A MR- test for acid end products from glucose fermentation was performed. If the tube turned red then that was considered as a positive result and if the tube turned yellow the result was negative.

A VP- test for action production from glucose fermentation. Positive result would be red after I added VP reagents and negative result would be no change of colour.

3.1.8.3.3 Citrate utilization test

The test culture was vaccinated on the surface of a slant simmon's citrate agar tube, and then vaccinated at 35°C for 24-48 hours. A positive test was represented by the colour of agar changed from green to blue when the organism was vaccinated with citrate as it is the sole source of carbon.

3.1.8.3.4 Urea utilization

The colony that was isolated inoculated on the surface of urea agar slant tube and incubated at 35°C for 24-48 hours. Urease producing organism reduced urea to ammonia and produce pink color.

3.1.8.3.5 Motility test

To help differentiate species of bacteria that motile this test must be used. An isolate was inoculated into a tube containing motility test medium with a sterile transfer straight needle, by making only one stab down the center of tube to about half the depth of the medium and got placed in the incubation at the temperature of 35°C for 24-48 hours. After incubation, if microorganisms bacterium is motile there will be growing and going out away from the stab line that we made, which means that test is positive. So, if a bacterium is not motile there will only be growing along the stab line and never growing out of it.

3.1.8.3.6 Oxidase test

Some bacteria produce oxidase enzyme so oxidase test was used to detect these bacteria. A filter paper was placed inside a sterile plastic disposable Petri dish and moistened with several drops of fresh oxidase reagent, and then a small portion of colony of the test organism (preferably not more than 24 hours old) was removed

with a wooden stick and rubbed on the filter paper. The positive reaction observed by a change in the color to blue or purple within ten seconds, while the result was no change of colour meant negative.

3.1.8.3.7 Catalase test

Some bacteria produce catalase enzymes, so this kind tests was used to determine the organism's ability to breakdown hydrogen peroxide (H₂O₂) into oxygen and water by action of the enzyme catalase. Using sterile loop remove a several colonies of the test organisms (preferably not more than 24 hours old and do not taken it from blood agar medium because red blood cells contain catalase) and immerse in the hydrogen peroxide solution. The evolution of bubbles of gas indicates a positive result.

3.1.8.3.8 Coagulase test

Coagulase test was used to test the ability of organism to produce coagulase enzyme. It is useful to differentiate between *Staphylococcus aureus* (which is positive) from coagulase-negative staphylococci (which is negative). The test was done by two ways: slide and tube test method, tube test used in our study.

Take a test tube and carried out by emulsify many colonies in 0.5 ml of rabbit plasma, and then incubated at 35°C for 4 hours. If no clot shown, the tube was placed incubation again at room temperature overnight. A positive coagulase was represented by any degree of clotting.

3.1.9 Identification of isolated bacteria by Vitek 2 Compact System

This system conciliates the colorimetric reagent cards that were placed in the incubation and interpreted automatically. Two identification cards GN ID (is Gram negative identification cards), GP ID (is Gram positive identification cards) were used. A principle of an automated microbiology system utilizing based on a growth technology and it's the Vitek 2 Compact System

All of the steps that were required were performed automatically for identification and antimicrobial susceptibility testing after primary inoculum has been prepared and standardized. By using a sterile stick, a pure culture of sufficient

number of overnight incubation was transferred and the isolated (Ligozzi *et al.*, 2002).

3.1.9.1 *Suspension preparation*

Bacteria were suspended in 3 mL of sterile (0.45% NaCl) saline with pH (4.5-7.0) in the clear plastic polystyrene test tube. The turbidity was adjusted according to the tables provided by manufacturer recommendation McFarland turbidity range (0.5-0.63 for Gram-positive and Gram-negative), and the turbidity was measured by using a turbidity meter called the DensiChek.

The test tube that contains the bacterial suspension was placed into a rack they call it (cassette), and the identification card got placed in the neighboring slot while they inserted the transfer tube into the corresponding suspension tube and after reading the barcode of the cards, the filled cassette was placed manually.

3.1.10 **Antimicrobial sensitivity testing**

The determination susceptibility of the entire isolated organisms to selected antibiotics which were normally used to treat pathogens were performed as previously described (Bauer *et al.*, 1966). All of the isolated pathogens bacteria were placed into appropriate media for antibiotic susceptibility. Disc according to the recommendations of the Clinical and Laboratory Standards Institute (CLSI, 2011). Diffusion test was carried out Pass through the numbers of steps as follows (Cavalieri *et al.*, 2005):

- a. The tops of 4 to 5 well-isolated uniform colonies (colonies must not be older than 18–24 hours) were touched with an inoculating loop and used to inoculate the tube containig 4 to 5 ml of sterile normal saline and mixed well. The tube which contained bacterial suspension was compared with the turbidity of 0.5 McFarland standard solutions, and the density of the test suspension that the standard is adjusted visually by putting more microorganisms' bacteria or normal saline, and then the turbidity of the suspensions was compared by placing the tubes with black lines in front of a file card.
- b. All tests are tempted to be done on Mueller-Hinton agar plates, and before used it should be warm to room temperature. By dipping a sterile cotton swap the plates were inoculated into bacterial suspension that were visually

equivalent turbidity to 0.5 McFarland standard solutions; the excess of inoculums was removed by rotating and pressing the swab firmly against the tubes side above the level of the suspension.

- c. The swab then was used to streak over the dry surface of Mueller-Hinton agar plates in three directions and this will ensure that the inoculum is evenly distributed. The inoculums plates was left to dry with the lid closed for at least 15 minutes at room temperature.
- d. The antimicrobial disks were stored at -20°C when not in use; the disks were allowed to reach room temperature before being opened. Various antibiotic discs were placed on the surface of the agar medium by softly pressing using sterile forceps on the top of the discs. The plates were incubated in an inverted position at 35°C for 18 to 24 hours.
- e. The next day, by counting the measurement of zone of inhibition produced by antimicrobial inhibition of bacterial growth the plates are read. Inhibition zones were measured in millimeter (mm) by using a vernier caliper.
- f. Each zone size interpreted by referring to an interpretative chart table (table 3.7) which presently is recommended by CLSI guidelines, into sensitive, intermediate and resistant.

Table 3.6 Zone-diameter interpretive standers according to (CLSI).

Antimicrobial agents	Disc content	Zone of inhibition diameter (mm)		
		Resistant	Intermediate	Sensitive
Ciprofloxacin (CIP)	10 µg	≤ 15	16-20	≥ 21
Ceftriaxone (CRO)	10 µg	≤ 13	14-20	≥ 21
Cefadroxil (CFR)	30 µg	≤ 14	15-17	≥ 18
Amoxicillin/clavulanic acid (AMC) (When testing staphylococci)	30 µg	≤ 19	---	≥ 20

Amoxicillin/clavulanic acid (AMC) (when testing other organisms)	30 µg	≤ 13	14-17	≥ 18
Cefotaxime (CTX)	10 µg	≤ 14	15-22	≥ 23
Nalidixic acid (Na)	30 µg	≤ 13	14-18	≥ 19
Nitrofurantoin (F)	100 µg	≤ 14	15-16	≥ 17
Norfloxacin (NOR)	10 µg	≤ 12	13-16	≥ 17
Amoxicillin (AX) (when testing staphylococci)	25 µg	≤ 19	---	≥ 20
Amoxicillin (AX) (when testing other organisms)	25 µg	≤ 13	14-17	≥ 18
Chloramphenicol (C)	30 µg	≤ 12	13-17	≥ 18
Cephalexin (Cl)	30 µg	≤ 18	15-17	≥ 18

4 RESULTS AND DISCUSSIONS

4.1 Incidence of Urinary Tract Infections

UTI is a very common disorder, approximately 0.4% to 3.2% million person seeks professional medical assistance annually in united states (Haley *et a*, 1985).

Urinary tract infection and damage is much more widely spread in adults than in children and younger aged humans, but about a percentage of 1-2% of children do get UTI. The UTI in children shouldn't be ignored because they are more likely to be earnest than those in adults.

Our result shown that, out of 500 urine specimens collected from patients complaining of signs and symptoms of UTIs attending Erbil general hospital laboratory in Erbil city, 356 (71.2%) were positive for bacterial infections (their colony count was equal or more than 10⁴ CFU/ml), while 144 (28.8 %) samples showed culture negative, as shown in table (4.1).

Table 4.1 Distribution of patients with UTIs

Samples (No. =500)	Cultures results	
	Positive No. (%)	Negative No. (%)
	356 (71.2%)	144 (28.8 %)

The pattern of pathogens encountered in this study correlates well with many studies conducted in different countries either in the regional or international settings. For example, Mezal *et al.* (2011) in Basrah, Iraq, reported that only (72.6%) of samples showed positive culture. Ali *et al.* (2014), in Kalar, Iraq reported that only (69.8%) of samples is positive culture . Kolawole *et al.* in (2009), studied that among 300 urine samples only (60%) showed culture positive. Mishra *et al.* in (2013), reported that among 1245 samples only (80%) of samples were positive for bacterial infections.

The percentage variable differences could be assigned to the variation in the size of the sample or in the technical procedure or in relation to the survey area , the remaining 144 specimens (28.8%) as shown in table (4.1) did not give any sign of the growth of bacteria even after 48 hours of incubation, this could be due to UTI

caused by agents other than bacteria such as viruses, fungi, anaerobic bacteria and other bacterial causes that cannot be isolated by traditional methods used in this study and may need special media for their growth or improper use of antibiotics.

4.2 Incidence of The Isolated Pathogens Associated with UTIs

Table (4.2) shows percentage of pathogens isolates from samples that were collected (500 samples); from which 356 strains of the microorganism's bacteria belonging to 19 species were isolated. The percentage of isolates were as follows: The most common pathogens isolated were *Escherichia coli* 158(44.38%) while other bacteria were *Staphylococcus aureus* 68 (19.10%), *Staphylococcus epidermidis* 37 (10.39%), *Klebsiella pneumonia* 22 (6.17%), *Pseudomonas aeruginosa* 18 (5.05%), *Proteus mirabilis* 12 (3.37%), *Staphylococcus haemolyticus* 7 (1.96%), *Staphylococcus saprophyticus* 7 (1.96%), *Enterobacter aerogenes* 6 (1.68%), *Klebsiella oxytoca* 4 (1.12%), *Citrobacter koseri* 3 (0.84%), *Morganella morganii* 3 (0.84%), *Enterobacter cloacae* 2 (0.56%), *Enterococcus faecalis* 2 (0.56%), *Proteus vulgaris* 2 (0.56%), *Serratia fonticola* 2 (0.56%), *Streptococcus agalactiae* 1 (0.28%), *Citrobacter freundii* 1 (0.28%), and *Salmonella typhimurium* 1 (0.28%)

E. coli appears to be the most common pathogen causing the dieades in this study which was isolated from 158 urine samples representing (44.38%) of the total, approximately similar to what was obtained by Dishvarian, 2010; Beyene and Tsegaye, 2011; Hanan et al.,2012. These represented 39.7%, 33.3%, 27.2% of *E. coli* in their isolated samples, respectively.

Table 4.2 pathogens obtained from urine culture.

No.	Isolated pathogens	Number	Percentage
1	<i>Escherichia coli</i>	158	44.38%
2	<i>Staphylococcus aureus</i>	68	19.10%
3	<i>Staphylococcus epidermidis</i>	37	10.39%
4	<i>Klebsiella pneumonia</i>	22	6.17%
5	<i>Pseudomonas aeruginosa</i>	18	5.05%
6	<i>Proteus mirabilis</i>	12	3.37%
7	<i>Staphylococcus haemolyticus</i>	7	1.96%

8	<i>Staphylococcus saprophyticus</i>	7	1.96%
9	<i>Enterobacter aerogenes</i>	6	1.68%
10	<i>Klebsiella oxytoca</i>	4	1.12%
11	<i>Citrobacter koseri</i>	3	0.84%
12	<i>Morganella morganii</i>	3	0.84%
13	<i>Enterobacter cloacae</i>	2	0.56%
14	<i>Enterococcus faecalis</i>	2	0.56%
15	<i>Proteus vulgaris</i>	2	0.56%
16	<i>Serratia fonticola</i>	2	0.56%
17	<i>Streptococcus agalactiae</i>	1	0.28%
18	<i>Citrobacter freundii</i>	1	0.28%
19	<i>Salmonella typhimurium</i>	1	0.28%
	Total	356	100%

The high percentage ratios of *Staphylococcus sp* could be due to the prevalence of these organisms in the anterior urethra and genital tract of both sexes and also it is an opportunistic pathogen which can cause disease when the bacteria change the location (from skin to urinary tract).

The recovery rates of *Klebsiella sp.* from cases of UTIs in this study were *Klebsiella pneumonia* (6.3%) and *Klebsiella oxytoca* (1.43), Al-Sehlawi in (2010) reported that the percentage of *Klebsiella sp.* in urine samples was (6.8%). In our opinion, the presence of this organism in high numbers in the gastrointestinal tract and the virulence factors exhibited by the microorganism like presence of prominent capsule may play a role in precipitating infection by such pathogenic organisms (Goldman and Green, 2009).

In the present study, the rate of isolates of *Pseudomonas aeruginosa* was 5.05%, while Mezal *et al.* in 2011 reported 8.3%. The majority of *Pseudomonas aeruginosa* could have been isolated from patients who are immunocompromised or in the phase after surgery or under catheterization procedures (Brooks *et al.*, 2007).

They might have some differences between newer studies and other studies due to the passage of time for the study, geographical variation, difference among the

female and male ratio, various personal, educational and overall socioeconomic status, availability of medical facilities, and method of collection of urine samples.

4.3 Incidence of UTIs in Relation to Sex and Age Groups

In this study 356 (71.2%) patients out of 500 were showed to be urine culture positive. This study shows a high incidence of UTI in females than males, there were 149 (41.85%) males and 207 (58.35%) females in patients with urine positive culture (Table4.3). Reported data concerning this field of study regularly shows high incidence of UTIs in females.

The high incidence of UTIs in females might be as a result of a variety of factors such as anatomical shape of the urethra in females (the short and wide female urethra) (3-4 cm length) and its proximity to the anus (Kolawole et al., 2009), wherefore bacteria from the rectum can easily travel up the urethra and cause infections. Also, pregnancy is one of the factors that increase the incidence of UTIs in women (Emiru et al., 2013).

Table 4.3 Frequency of UTIs according to the gender.

Bacterial infection	Gender. No. (%)	
	Male	Female
Infected	149 (41.85%)	207 (58.35%)
Non-infected	53(36.80%)	91(63.19%)

In our study, it was found that the incidence of UTIs among female patients in the age group of 13 to 25 years much higher than males in the same age group. Increasing incidence of UTI in females is associated with menstruation period started and high sexual activity. In the age group of 24 to 34 years, the incidence of UTI in females was high compared to the females in other age groups.

Table 4.4 shows the age and gender distribution of patients suffering from UTIs.

Age	Total No. (%) of infected male and female	Infected male No. (%)	Infected female No. (%)
0-12	43 (12.07)	25 (7.02)	19 (5.33)
13-23	56 (15.73)	10 (2.80)	42 (11.79)

24-34	68 (19.10)	28 (7.86)	48 (13.48)
35-45	65 (18.25)	20 (5.61)	40 (11.23)
46-56	55 (15.44)	13 (3.65)	31 (8.70)
57-67	36 (10.11)	23 (6.46)	19 (5.33)
68 \geq	33 (9.26)	30 (8.42)	8 (2.24)
Total	356	149	207

4.4 Frequency of Clinical Symptoms in Positive Urine Culture Patients

There is a significant relationship between the presence of the symptoms and bacterial infection. In this study, after taking information from the patient about symptoms who suffer from it, it was found that the percentage of Supra pubic pain was 78.09%, dysuria 75.21%, frequency 73.49%, burning 84.26%, urgency 38.29%, strong smell to urine 28.93%, cloudy urine 45.19%, flank pain 25.50%, fevers 6.6% and nausea and vomiting 5.15%. Symptoms of UTI include: a Strong urge to urinate multiple times, even right after the bladder is just emptied and with pain and during urinating you might get a burning sensation and pain in the pelvic area or back, cloudy or might have blood urine, which might have a strange odour (Akram *et al.*, 2007).

4.5 Incidence Rate of UTIs According to the Residency of the Patients

The incidence rate of UTIs at urban was 42.97 % and 57.03 % in rural. The most probable explanation, this might be due to poor diagnostic facilities available in the health centers in rural areas and also the lower socioeconomic, hygienic standards and the absence of a proper sewage disposal system.

Table 4.5 Prevalence of UTI in relation to the residency

Residency	Number	Percentage
Urban	153	42.97%
Rural	203	57.03%
Total	356	100%

4.6 Antimicrobial Susceptibility

In settings for health care, a few extra studies on antimicrobial susceptibility survey can facilitate to provide completely workable information of the resistance

pattern (Abdulrazzaq, 2013). Continuous monitoring of sensitivity patterns is very important, since this sensitivity is affected by the wide use of antimicrobials and this lead to the evolution of new resistant strains.

It is of importance to have updated information about the sensitivity of the urinary pathogens to the available drugs. ideally, choice of treatment should be made after the sensitivities of the causative organisms had been determined in vitro. The treatment of life threatening infections should be recommendation before sensitivity tests have been carried out (Brumfitt and Hamilton-miller,1985). continuous monitoring of sensitivity patterns is important, since this sensitivity if effected by the wide use of antimicrobials and this lead to the emergence of new resistant strains (Thomas et al., 1977)

In these study the results of the disk diffusion testing for the antibiotic susceptibility are shown in table (4.6), most of our bacterial isolates were highly resistant to amoxicillin, we found that *Escherichia coli* (94.93%), *Staphylococcus aureus* (85.29%), *Staphylococcus epidermidis* (67.56%), *Klebsiella pneumonia* (86.36%), *Pseudomonas aeruginosa* (77.77%), *Proteus mirabilis* (83.33%), *Staphylococcus haemolyticus* (100%), *Staphylococcus saprophyticus* (85.71%), *Enterobacter aerogenes* (50%), *Klebsiella oxytoca* (100%), *Citrobacter koseri* (75%), *Morganella morganii* (75%), *Enterobacter cloacae* (100%), *Enterococcus faecalis* (50%), *Proteus vulgaris* (100%), *Serratia fonticola* (50%), *Citrobacter freundii* (100%), *Salmonella typhimurium* (100%). Were *Enterococcus faecalis* (50%), *Serratia fonticola* (50%), *Streptococcus agalactiae* (100%) found sensitive to amoxicillin.

Our result showed that most of the isolates bacterial pathogens are resistant to the amoxicillin-clavulanic acid *Escherichia coli* (96.20%), *Staphylococcus aureus* (82.35%), *Staphylococcus epidermidis* (86.48%), *Klebsiella pneumonia*(45.45%), *Pseudomonas aeruginosa* (94.44%), *Proteus mirabilis* (58.33%), *Staphylococcus haemolyticus* (100%), *Staphylococcus saprophyticus* (85.71%), *Enterobacter aerogenes* (50%), *Klebsiella oxytoca* (100%), *Citrobacter koseri* (100%), *Morganella morganii* (75%), *Enterobacter cloacae* (100%), *Enterococcus faecalis* (50%), (100%), *Serratia fonticola* (50%), *Citrobacter freundii* (100%), *Salmonella typhimurium* (100%), we found that were resistant to the amoxicillin-clavulanic acid, while *Klebsiella pneumonia* (50%), *Enterobacter aerogenes* (50%), *Enterococcus*

faecalis (50%), *Serratia fonticol* (50%), *Streptococcus agalactiae* (100%), were sensitive to amoxicillin-clavulanic acid (Table 4.7). The most bacteria were showed high resistance to amoxicillin and amoxicillin-clavulanic acid because bacteria develop mechanisms to resist the drug

Table 4.6 Antimicrobial susceptibility of isolates pathogens to amoxicillin

No.	Isolated pathogens	Number	amoxicillin No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	150(94.93%)	2(1.26%)	6(3.79%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	58(85.29%)	0 (00)	10(14.70%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	25(67.56%)	0 (00)	12(32.43%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	19(86.36%)	1(4.54%)	2 (9.09%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	14 (77.77%)	3 (16.66%)	1 (5.55%)
6	<i>Proteus mirabilis</i>	12(3.37%)	10 (83.33%)	0(00)	2 (16.66%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	7 (100%)	0 (00)	0 (00)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	6 (85.71%)	0 (00)	1 (14.28%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	3 (50%)	0(00)	3 (50%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	4(100%)	0(00)	0(00)
11	<i>Citrobacter koseri</i>	3(0.84%)	2 (75%)	0 (00)	1 (25%)
12	<i>Morganella morganii</i>	3(0.84%)	2 (75%)	0(00)	1 (25%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	2(100%)	0(00)	0(00)
14	<i>Enterococcus faecalis</i>	2(0.56%)	1(50%)	0 (00)	1(50%)
15	<i>Proteus vulgaris</i>	2(0.56%)	2(100%)	0(00)	0(00)
16	<i>Serratia fonticola</i>	2(0.56%)	1 (50%)	0(00)	1 (50%)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0 (00)	0 (00)	1 (100%)
18	<i>Citrobacter freundii</i>	1(0.28%)	1 (100%)	0 (00)	0 (00)
19	<i>Salmonella typhimurium</i>	1(0.28%)	1 (100%)	0 (00)	0 (00)

Table 4.7 Antimicrobial susceptibility of isolates pathogens to amoxicillin-clavulanic acid

No	Isolated pathogens	Number	amoxicillin-clavulanic acid No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	152(96.20%)	6(3.79%)	0 (00)
2	<i>Staphylococcus aureus</i>	68(19.10%)	56(82.35%)	0 (00)	12(17.64%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	32(86.48%)	1 (2.70%)	4(10.81%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	10(45.45%)	1(4.54%)	11 (50%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	17 (94.44%)	0 (00)	1 (5.55%)
6	<i>Proteus mirabilis</i>	12(3.37%)	7 (58.33%)	3(2.42%)	2 (16.66%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	7 (100%)	0 (00)	0 (00)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	6 (85.71%)	0 (00)	1 (14.28%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	3 (50%)	0(00)	3 (50%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	4(100%)	0(00)	0(00)
11	<i>Citrobacter koseri</i>	3(0.84%)	3 (100%)	0 (00)	0 (00)
12	<i>Morganella morganii</i>	3(0.84%)	2 (75%)	0(00)	1 (25%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	2(100%)	0(00)	0(00)
14	<i>Enterococcus faecalis</i>	2(0.56%)	1(50%)	0 (00)	1(50%)
15	<i>Proteus vulgaris</i>	2(0.56%)	2(100%)	0(00)	0(00)
16	<i>Serratia fonticola</i>	2(0.56%)	1 (50%)	0(00)	1 (50%)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0 (00)	0 (00)	1 (100%)
18	<i>Citrobacter freundii</i>	1(0.28%)	1 (100%)	0 (00)	0 (00)
19	<i>Salmonella yphimurium</i>	1(0.28%)	1 (100%)	0 (00)	0 (00)

We showed in the Table (4.8), the antibiotic susceptibility tests for isolated bacterial pathogens are found that *Staphylococcus haemolyticus* (42.85%), *Enterobacter cloacae* (50%), *Proteus vulgaris* (50%) were resistance for ciprofloxacin, but *Escherichia coli* (62.02%), *Staphylococcus aureus* (57.35%), *Staphylococcus epidermidis* (56.75%), *Klebsiella pneumonia* (86.36%), *Pseudomonas aeruginosa* (55.55%), *Proteus mirabilis* (50%), *Staphylococcus saprophyticus* (71.42%), *Enterobacter aerogenes* (83.33%), *Klebsiella oxytoca* (75%), *Citrobacter koseri* (66.66%), *Enterobacter cloacae* (50%), *Enterococcus faecalis* (100%), *Proteus vulgaris* (50%), *Serratia fonticola* (100%), *Streptococcus*

agalactiae (100%), *Citrobacter freundii* (100%), *Salmonella typhimurium* (100%) were sensitive for the same antibiotic.

The results of present study showed that the resistance percentage of isolates bacterial pathogens for cefadroxil were as following; *Escherichia coli* (62.65%), *Pseudomonas aeruginosa* (66.66%), *Enterobacter aerogenes* (50%), *Klebsiella oxytoca* (50%), *Citrobacter koseri* (66.67%), *Morganella morganii* (75%), *Enterobacter cloacae* (50%), *Proteus vulgaris* (100%), *Serratia fonticola* (50%), *Citrobacter freundii* (100%), *Salmonella typhimurium* (100%) while the sensitive percentage rate of isolates bacterial pathogens to same antibiotic were as following; *Staphylococcus aureus* (66.17%), *Staphylococcus epidermidis* (56.75%), *Enterobacter aerogenes* (50%), *Enterococcus faecalis* (50%), *Serratia fonticola* (50%) were intermediate for cefadroxil (Table 4.9).

Table 4.8 Antimicrobial susceptibility of isolates pathogens to ciprofloxacin

No.	Isolated pathogens	Number	ciprofloxacin No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	55(34.82%)	5(3.16%)	98(62.02%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	13(19.11%)	16(23.52%)	39(57.35%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	9(24.32%)	7(18.92%)	21(56.75%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	1(4.45%)	2(9.09%)	19(86.36%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	4(22.22%)	4(22.22%)	10(55.55%)
6	<i>Proteus mirabilis</i>	12(3.37%)	3(25%)	3(25%)	6(50%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	3(42.85%)	1(14.28%)	3(42.85%)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	2(28.57%)	0(00)	5(71.42%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	1(16.66%)	0(00)	5(83.33%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	0(00)	1(25%)	3(75%)
11	<i>Citrobacter koseri</i>	3(0.84%)	1(33.33%)	0(00)	2(66.66%)
12	<i>Morganella morganii</i>	3(0.84%)	1(33.33%)	1(33.33%)	1(33.33%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	1(50%)	0(00)	1(50%)
14	<i>Enterococcus faecalis</i>	2(0.56%)	0(00)	0(00)	2(100%)
15	<i>Proteus vulgaris</i>	2(0.56%)	1(50%)	0(00)	1(50%)
16	<i>Serratia fonticola</i>	2(0.56%)	0(00)	0(00)	2(100%)

17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	0(00)	1(100%)
18	<i>Citrobacter freundii</i>	1(0.28%)	0(00)	0(00)	1(100%)
19	<i>Salmonella typhimurium</i>	1(0.28%)	0(00)	0(00)	1(100%)

Table 4.9 Antimicrobial susceptibility of isolates pathogens to cefadroxil

No.	Isolated pathogens	Number	cefadroxil No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	99(62.65%)	32(20.25%)	27(17.09%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	11(16.17%)	12(17.64%)	45(66.17%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	10(27.03%)	6(16.22%)	21(56.75%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	8(36.36%)	4(18.18%)	10(45.45%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	12(66.66%)	0(00)	6(33.33%)
6	<i>Proteus mirabilis</i>	12(3.37%)	3(25%)	7(58.33%)	2(16.67%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	3(42.86%)	2(28.57%)	2(28.57%)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	3(42.86%)	1(14.28%)	3(42.86%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	3(50%)	0(00)	3(50%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	2(50%)	1(25%)	1(25%)
11	<i>Citrobacter koseri</i>	3(0.84%)	2(66.67%)	1(33.33%)	0(00)
12	<i>Morganella morganii</i>	3(0.84%)	2(75%)	1(25%)	0(00)
13	<i>Enterobacter cloacae</i>	2(0.56%)	1(50%)	1(50%)	0(00)
14	<i>Enterococcus faecalis</i>	2(0.56%)	0(00)	1(50%)	1(50%)
15	<i>Proteus vulgaris</i>	2(0.56%)	2(100%)	0(00)	0(00)
16	<i>Serratia fonticola</i>	2(0.56%)	1(50%)	0(00)	1(50%)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	1(100%)	0(00)
18	<i>Citrobacter freundii</i>	1(0.28%)	1(100%)	0(00)	0(00)
19	<i>Salmonella typhimurium</i>	1(0.28%)	1(100%)	0(00)	0(00)

In our study, the percentage of resistance to cefotaxime is showed in table (4.9) for isolates bacterial pathogens as following; *Staphylococcus aureus* (63.24%), *Staphylococcus epidermidis* (89.19%), *Klebsiella pneumonia* (50%), *Pseudomonas aeruginosa* (88.90%), *Proteus mirabilis* (50%), *Staphylococcus haemolyticus* (100%), *Staphylococcus saprophyticus* (71.43%), *Enterobacter aerogenes* (83.33%),

Klebsiella oxytoca (50%), *Citrobacter koseri* (75%), *Enterobacter cloacae* (100%), *Proteus vulgaris* (50%), *Serratia fonticola* (50%), *Citrobacter freundii* (100%), *Salmonella typhimurium* (100%) while the percentage of sensitive of isolates bacterial pathogens to same antibiotic were as following; *Morganella morganii* (75%), *Enterococcus faecalis* (50%), *Proteus vulgaris* (50%). the most bacteria were showed high resistance to cefotaxime because bacteria develop mechanisms to resist the drug

The resistance rate to ceftriaxone were found to be (67.09%) of *Escherichia coli*, *Staphylococcus aureus* (72.05%), *Pseudomonas aeruginosa* (55.55%), *Staphylococcus haemolyticus* (85.71%), *Staphylococcus saprophyticus* (71.43%), *Enterobacter aerogenes* (50%), *Klebsiella oxytoca* (75%), *Morganella morganii* (75%), *Enterobacter cloacae* (50%), *Proteus vulgaris* (50%), *Citrobacter freundii* (100). While the sensitive rates to same antibiotic is *Proteus mirabilis* (75%), *Enterococcus faecalis* (100%), *Proteus vulgaris* (50%), *Serratia fonticola* (50%), *Streptococcus agalactiae* (50%), *Salmonella typhimurium* (100%) (Table 4.10)

Table 4.10 Antimicrobial susceptibility of isolates pathogens to cefotaxime

No.	Isolated pathogens	Number	cefotaxime No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	76(48.10%)	53(33.54%)	29(18.35%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	43(63.24%)	14(20.59%)	11(16.18%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	33(89.19%)	2(5.41%)	2(5.41%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	11(50%)	10(45.45%)	1(4.55%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	16(88.90%)	1(5.55%)	1(5.55%)
6	<i>Proteus mirabilis</i>	12(3.37%)	6(50%)	2(16.67%)	4(33.33%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	7(100%)	0(00)	0(00)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	5(71.43%)	2(28.57%)	0(00)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	5(83.33%)	1(16.67%)	0(00)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	2(50%)	1(25%)	1(25%)
11	<i>Citrobacter koseri</i>	3(0.84%)	2(75%)	1(25%)	0(00)
12	<i>Morganella morganii</i>	3(0.84%)	1(25%)	0(00)	2(75%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	2(100%)	0(00)	0(00)

14	<i>Enterococcus faecalis</i>	2(0.56%)	0(00)	1(50%)	1(50%)
15	<i>Proteus vulgaris</i>	2(0.56%)	1(50%)	0(00)	1(50%)
16	<i>Serratia fonticola</i>	2(0.56%)	1(50%)	1(50%)	0(00)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	1(100%)	0(00)
18	<i>Citrobacter freundii</i>	1(0.28%)	1(100%)	0(00)	0(00)
19	<i>Salmonella typhimurium</i>	1(0.28%)	1(100%)	0(00)	0(00)

Table 4.11 Antimicrobial susceptibility of isolates pathogens to ceftriaxone

No	Isolated pathogens	Number	ceftriaxone No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	106(67.09%)	4(2.53%)	78(49.37%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	49(72.05%)	11(16.18%)	8(11.76%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	16(43.24%)	15(40.54%)	6(16.22%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	8 (36.36%)	5 (22.73%)	9 (40.91%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	10(55.55%)	5(27.78%)	3(16.67%)
6	<i>Proteus mirabilis</i>	12(3.37%)	2(16.67%)	1(8.33%)	9(75%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	6(85.71%)	1(14.29%)	0(00)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	5(71.43%)	2(28.57%)	0(00)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	3(50%)	2(33.33%)	1(16.67%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	3(75%)	0(00)	1(25%)
11	<i>Citrobacter koseri</i>	3(0.84%)	1(33.33%)	1(33.33%)	1(33.33%)
12	<i>Morganella morganii</i>	3(0.84%)	2(75%)	0(00)	1(25%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	1(50%)	1(50%)	0(00)
14	<i>Enterococcus faecalis</i>	2(0.56%)	0(00)	0(00)	2(100%)
15	<i>Proteus vulgaris</i>	2(0.56%)	1(50%)	0(00)	1(50%)
16	<i>Serratia fonticola</i>	2(0.56%)	0(00)	1(50%)	1(50%)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	1(50%)	1(50%)
18	<i>Citrobacter freundii</i>	1(0.28%)	1(100%)	0(00)	0(00)
19	<i>Salmonella</i>	1(0.28%)	0(100%)	0(00)	1(100%)

	<i>typhimurium</i>			
--	--------------------	--	--	--

As showed in table (4.11) the antimicrobial sensitivity pattern of our bacterial isolates we found that *Escherichia coli* (62.65%), *Staphylococcus aureus* (73.53%), *Staphylococcus epidermidis* (75.67%), *Pseudomonas aeruginosa* (77.78%), *Proteus mirabilis* (58.33%), *Staphylococcus haemolyticus* (85.71%), *Staphylococcus saprophyticus* (71.43%), *Morganella morganii* (66.67%), *Enterococcus faecalis* (100%), *Proteus vulgaris* (50%), *Serratia fonticola* (100%), *Streptococcus agalactiae* (100%), *Salmonella typhimurium* (100%) were resistances to nalidixic acid, while another isolates such as *Klebsiella pneumonia* (54.54%), *Enterobacter aerogenes* (50%), *Klebsiella oxytoca* (75%), *Citrobacter koseri* (66.67%), *Proteus vulgaris* (50%), *Citrobacter freundii* (100%) were sensitive to nalidixic acid, but only *Enterobacter cloacae* was (100%) intermediate to same antibiotic.

In our study based on results obtained from susceptibility testing (table 4.12), we found that *Klebsiella pneumonia* (68.18%), *Pseudomonas aeruginosa* (83.33%), *Proteus mirabilis* (91.67%), *Enterobacter aerogenes* (83.33%), *Klebsiella oxytoca* (75%), *Citrobacter koseri* (66.67%), *Morganella morganii* (66.67%), *Enterobacter cloacae* (100%), *Proteus vulgaris* (100%), *Citrobacter freundii* (100%), *Salmonella typhimurium* (100%) were resistant to nitrofurantoin, whereas we found that *Escherichia coli* (62.02%), *Staphylococcus aureus* (55.88%), *Staphylococcus epidermidis* (51.35%), *Staphylococcus haemolyticus* (57.14%), *Staphylococcus saprophyticus* (57.14%), *Enterococcus faecalis* (100%), *Streptococcus agalactiae* (100%) were sensitive to nitrofurantoin.

Table 4.12 Antimicrobial susceptibility of isolates pathogens to nalidixic acid

No.	Isolated pathogens	Number	nalidixic acid No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	99(62.65%)	19(12.03%)	40(25.32%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	50(73.53%)	12(17.65%)	6(8.82%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	28(75.67%)	6(16.22%)	3(8.10%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	9 (40.91%)	1 (4.55%)	12 (54.54%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	14(77.78%)	3(16.66%)	1(5.56%)
6	<i>Proteus mirabilis</i>	12(3.37%)	7(58.33%)	3(25%)	2(16.67%)

7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	6(85.71%)	0(00)	1(14.29%)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	5(71.43%)	1(14.29%)	1(14.29%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	2(33.33%)	1(16.67%)	3(50%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	1(25%)	0(00)	3(75%)
11	<i>Citrobacter koseri</i>	3(0.84%)	1(33.33%)	0(00)	2(66.67%)
12	<i>Morganella morganii</i>	3(0.84%)	2(66.67%)	0(00)	1(33.33%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	0(00)	2(100%)	0(00)
14	<i>Enterococcus faecalis</i>	2(0.56%)	2(100%)	0(00)	0(00)
15	<i>Proteus vulgaris</i>	2(0.56%)	1(50%)	0(00)	1(50%)
16	<i>Serratia fonticola</i>	2(0.56%)	2(100%)	0(00)	0(00)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	1(100%)	0(00)	0(00)
18	<i>Citrobacter freundii</i>	1(0.28%)	0(00)	0(00)	1(100%)
19	<i>Salmonella typhimurium</i>	1(0.28%)	1(100%)	0(00)	0(00)

Table 4.13 Antimicrobial susceptibility of isolates pathogens to nitrofurantoin

No.	Isolated pathogens	Number	nitrofurantoin No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	16(10.13%)	44(27.85%)	98(62.02%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	10(14.71%)	20(29.41%)	38(55.88%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	6(16.22%)	12(32.43%)	19(51.35%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	15 (68.18%)	2 (9.09%)	5 (22.73%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	15(83.33%)	0(00)	3(16.67%)
6	<i>Proteus mirabilis</i>	12(3.37%)	11(91.67%)	1(8.33%)	0(00)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	1(14.28%)	2(28.57%)	4(57.14%)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	2(28.57%)	1(14.28%)	4(57.14%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	5(83.33%)	0(00)	1(16.67%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	3(75%)	1(25%)	0(00)
11	<i>Citrobacter koseri</i>	3(0.84%)	2(66.67%)	1(33.33%)	0(00)
12	<i>Morganella morganii</i>	3(0.84%)	2(66.67%)	0(00)	1(33.33%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	2(100%)	0(00)	0(00)

14	<i>Enterococcus faecalis</i>	2(0.56%)	0(00)	0(00)	2(100%)
15	<i>Proteus vulgaris</i>	2(0.56%)	2(100%)	0(00)	0(00)
16	<i>Serratia fonticola</i>	2(0.56%)	0(00)	2(100%)	0(00)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	0(00)	1(100%)
18	<i>Citrobacter freundii</i>	1(0.28%)	1(100%)	0(00)	0(00)
19	<i>Salmonella typhimurium</i>	1(0.28%)	1(100%)	0(00)	0(00)

As shown in table (4.13), we found that (57.14%) of *Staphylococcus haemolyticus*, (50%) of *Enterobacter cloacae*, (50%) of *Proteus vulgaris* were Resistant to norfloxacin, while another isolates found that (59.49%) of *Escherichia coli*, (60.29%) of *Staphylococcus aureus*, (86.36%) of *Klebsiella pneumonia*, (55.55%) of *Pseudomonas aeruginosa*, (50%) of *Proteus mirabilis*, (71.42%) of *Staphylococcus saprophyticus*, (66.67%) of *Enterobacter aerogenes*, (75%) of *Klebsiella oxytoca*, (66.67%) of *Citrobacter koseri*, (66.67%) of *Morganella morganii*, (50%) of *Enterobacter cloacae*, (100%) of *Enterococcus faecalis*, (50%) of *Proteus vulgaris*, (100%) of *Serratia fonticola*, (100%) of *Streptococcus agalactiae*, (100%) of *Citrobacter freundii* were Sensitive for norfloxacin.

Table 4.14 Antimicrobial susceptibility of isolates pathogens to norfloxacin

No	Isolated pathogens	Number	norfloxacin No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	49(31.01%)	15(9.49%)	94(59.49%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	21(30.88%)	6(8.82%)	41(60.29%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	14(37.83%)	7(18.92%)	16(43.24%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	3 (13.64%)	0 (00)	19 (86.36%)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	6(33.33%)	2(11.11%)	10(55.55%)
6	<i>Proteus mirabilis</i>	12(3.37%)	2(16.66%)	4(33.33%)	6(50%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	4(57.14%)	0(00)	3(42.86%)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	2(28.57%)	0(00)	5(71.42%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	2(33.33%)	0(00)	4(66.67%)

10	<i>Klebsiella oxytoca</i>	4(1.12%)	1(25%)	0(00)	3(75%)
11	<i>Citrobacter koseri</i>	3(0.84%)	1(33.33%)	0(00)	2(66.67%)
12	<i>Morganella morganii</i>	3(0.84%)	1(33.33%)	0(00)	2(66.67%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	1(50%)	0(00)	1(50%)
14	<i>Enterococcus faecalis</i>	2(0.56%)	0(00)	0(00)	2(100%)
15	<i>Proteus vulgaris</i>	2(0.56%)	1(50%)	0(00)	1(50%)
16	<i>Serratia fonticola</i>	2(0.56%)	0(00)	0(00)	2(100%)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	0(00)	1(100%)
18	<i>Citrobacter freundii</i>	1(0.28%)	0(00)	0(00)	1(100%)
19	<i>Salmonella typhimurium</i>	1(0.28%)	0(00)	1(100%)	0(00)

As shown in table (4.14), we found that *Staphylococcus epidermidis* (51.35%), *Klebsiella pneumonia* (86.36%), *Pseudomonas aeruginosa* (55.55%), *Proteus mirabilis* (50%), *Staphylococcus saprophyticus* (57.14%), *Enterobacter aerogenes* (66.67%), *Klebsiella oxytoca* (50%), *Citrobacter koseri* (66.67%), *Enterobacter cloacae* (100%), *Enterococcus faecalis* (50%), *Proteus vulgaris* (50%), *Serratia fonticola* (100%), *Citrobacter freundii* (100%), were Resistant to Chloramphenicol, while another isolates found that *Escherichia coli* (54.43%), *Proteus mirabilis* (50%), *Staphylococcus haemolyticus* (57.14%), *Enterococcus faecalis* (50%), *Proteus vulgaris* (50%), *Salmonella typhimurium* (100%) were Sensitive to Chloramphenicol.

Table 4.15 Antimicrobial susceptibility of isolates pathogens to Chloramphenicol

No.	Isolated pathogens	Number	Chloramphenicol No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	54(34.17%)	18(11.39%)	86(54.43%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	30(44.11%)	8(11.76%)	30(44.11%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	19(51.35%)	7(18.91%)	11(29.73%)
4	<i>Klebsiella pneumonia</i>	22(6.17%)	19(86.36%)	3(13.63%)	0(00)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	10(55.55%)	8(44.44%)	0(00)
6	<i>Proteus mirabilis</i>	12(3.37%)	6(50%)	0(00)	6(50%)

7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	3(42.86%)	0(00)	4(57.14%)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	4(57.14%)	0(00)	3(42.86%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	4(66.67%)	1(16.67%)	1(16.67%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	2(50%)	1(25%)	1(25%)
11	<i>Citrobacter koseri</i>	3(0.84%)	2(66.67%)	1(33.33%)	0(00)
12	<i>Morganella morganii</i>	3(0.84%)	1(33.33%)	1(33.33%)	1(33.33%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	2(100%)	0(00)	0(00)
14	<i>Enterococcus faecalis</i>	2(0.56%)	1(50%)	0(00)	1(50%)
15	<i>Proteus vulgaris</i>	2(0.56%)	1(50%)	0(00)	1(50%)
16	<i>Serratia fonticola</i>	2(0.56%)	2(100%)	0(00)	0(00)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	1(100%)	0(00)
18	<i>Citrobacter freundii</i>	1(0.28%)	1(100%)	0(00)	0(00)
19	<i>Salmonella typhimurium</i>	1(0.28%)	0(00)	0(00)	1(100%)

In (table 4.15) can see the resistant of bacteria to Cephalixin; *Klebsiella pneumonia* (86.36%), *Pseudomonas aeruginosa* (77.78%), *Staphylococcus haemolyticus* (57.14%), *Klebsiella oxytoca* (100%), *Citrobacter koseri* (100%), *Morganella morganii* (66.67%), *Enterobacter cloacae* (50%), *Enterococcus faecalis* (50%), *Serratia fonticola* (50%), *Citrobacter freundii* (100%), *Salmonella typhimurium* (100%), and the Sensitive to the same antibiotic is; *Escherichia coli* (50.63%), *Staphylococcus aureus* (50%), *Proteus mirabilis* (58.33%), *Staphylococcus saprophyticus* (57.14%), *Enterobacter aerogenes* (66.67%), *Enterobacter cloacae* (50%), *Proteus vulgaris* (100%), *Streptococcus agalactiae* (100%).

Table 4.16 Antimicrobial susceptibility of isolates pathogens to Cephalixin

No	Isolated pathogens	Number	Cephalixin No. (%)		
			Resistant	Intermediate	Sensitive
1	<i>Escherichia coli</i>	158(44.38%)	64(40.50%)	14(8.86%)	80(50.63%)
2	<i>Staphylococcus aureus</i>	68(19.10%)	31(45.58%)	3(4.41%)	34(50%)
3	<i>Staphylococcus epidermidis</i>	37(10.39%)	17(45.94%)	8(21.62%)	12(32.43%)

4	<i>Klebsiella pneumonia</i>	22(6.17%)	19(86.36%)	3(13.63%)	0(00)
5	<i>Pseudomonas aeruginosa</i>	18(5.05%)	14(77.78%)	4(22.22%)	0(00)
6	<i>Proteus mirabilis</i>	12(3.37%)	2(16.67%)	3(25%)	7(58.33%)
7	<i>Staphylococcus haemolyticus</i>	7(1.96%)	4(57.14%)	0(00)	3(42.85%)
8	<i>Staphylococcus saprophyticus</i>	7(1.96%)	2(28.57%)	1(14.28%)	4(57.14%)
9	<i>Enterobacter aerogenes</i>	6(1.68%)	2(33.33%)	0(00)	4(66.67%)
10	<i>Klebsiella oxytoca</i>	4(1.12%)	4(100%)	0(00)	0(00)
11	<i>Citrobacter koseri</i>	3(0.84%)	3(100%)	0(00)	0(00)
12	<i>Morganella morganii</i>	3(0.84%)	2(66.67%)	0(00)	1(33.33%)
13	<i>Enterobacter cloacae</i>	2(0.56%)	1(50%)	0(00)	1(50%)
14	<i>Enterococcus faecalis</i>	2(0.56%)	1(50%)	1(50%)	0(00)
15	<i>Proteus vulgaris</i>	2(0.56%)	0(00)	0(00)	2(100%)
16	<i>Serratia fonticola</i>	2(0.56%)	1(50%)	1(50%)	0(00)
17	<i>Streptococcus agalactiae</i>	1(0.28%)	0(00)	0(00)	1(100%)
18	<i>Citrobacter freundii</i>	1(0.28%)	1(100%)	0(00)	0(00)
19	<i>Salmonella typhimurium</i>	1(0.28%)	1(100%)	0(00)	0(00)

In the city of Erbil, there is no control on taking antibiotics and giving them to the patients without a physician's prescription, as well as the occurrence of an infection in any organ of a patient, patients take antibiotics without knowing anything about antibiotic susceptibility or the side effects. This leads to killing commensal bacteria and the emergence of different types of antibiotic resistant bacteria.

5 CONCLUSIONS

5.1 Conclusions

According to this study, we concluded the followings:

1. The females appear to be more at risk to infection than the male sex.
2. *E. coli* was among the commonest pathogenic bacteria isolated with an incidence rate (44.38%).
3. Depending on the academic achievement, the uneducated person shows a higher percentage of the infection than the educated one
4. According to the residency, UTIs were more frequent in rural than urban area, urban was 42.97 % and 57.03 % in rural.
5. It is quite alarming to note that most of the isolates included in this study were found resistant to four or more antibiotics Amoxicillin, Amoxicillin clavulanic acid, Chloramphenicol.

5.2 Suggestions

I suggest the followings:

1. When feeling any supra pubic pain, burning during urination should consult the doctor and make the necessary tests.
2. All physicians should give proper treatment depending on culture and susceptibility test.
3. Awareness of the people about the misuse of antimicrobial agents must be increased because the misuse may lead to complications of UTI and emergence of different antimicrobial resistant bacteria.
4. All laboratory staff should know the proper way for collecting urine specimen and enforcement it, and they must be more accurate in doing culture and antibiotic susceptibility test.
5. Education of the family about self-hygiene control and ways of management of UTI should be adopted.
6. We also recommend the people should have the simple knowledge about Urinary system because it is most expected disease especially in poor place such as villages.

7. Also, taking care about the water level in the body by drinking the water and juice in order to filter the body fluid from salt and other minerals is suggested.
8. Lastly, we would like to highlight the significance of the physician to detect and diagnose patients with UTI and determine the appropriate treatment.



REFERENCES

- Abbas, A. F., and Naji, S. A., 2010. Determination of The Bacterial Types That Cause Urinary Tract Infection in Diyala Provence. *Diyala Journal for Pure Sciences*, Vol. 6, No. 2
- Arslan, H., Azap, O. K., Ergonul, O., and Timurkaynak, F., 2005. Risk factors for ciprofloxacin resistance among *Escherichia coli* strains isolated from community-acquired urinary tract infections in Turkey. *Journal of Antimicrobial Chemotherapy*, 56, 914–918.
- Abdulrazzaq, G. M., 2013. Pattern of antibiotic sensitivity and resistance of uropathogens among pediatric patients with urinary tract infection. *Iraq J. Pharm.* Vol., 13, No. 1.
- Akram, M., Shahid, M., and Khan, A. U., 2007. Etiology and antibiotic resistance patterns of community-acquired urinary tract infections in JNMC Hospital Aligarh, India. *Annals of Clinical Microbiology and Antimicrobials*, 6:4.
- Al-Badr, A., and Al-Shaikh, G., 2013. Recurrent Urinary Tract Infections Management in Women. *Sultan Qaboos University Med. J.*, Vol. 13, Iss. 3, pp. 359-367.
- Alexander, S. K., and Strete, D., 2001. *Microbiology: A Photographic Atlas for the Laboratory*. Benjamin Cummings, an imprint of Addison Wesley Longman, Inc., USA, pp. 35-92.
- Al-Madani, T. A., Hashim, J. M., and Al-Deen, D. S., 2007. Prevalence of bacterial agents causing urinary tract infections in children below 5 years of age and their antibiotic sensitivity. *Thi-Qar Medical Journal (TQMJ)*, Vol. 1, No.1 :(75-82).
- Al-Sehlawi, Z. S. R., 2010. A Bacteriological Study on Urinary Tract Infections Associated with Catheterization of Hospitalized Patients. *Medical Journal of Babylon*, Vol. 18, No.2.
- Atlas, M.R.; Parks, L.C. and Brown, A.E. (1995). *Laboratory manual of experimental microbiology*. Mosby. Yearbook.Inc.

- Atlas, R. M., 2010. Handbook of Microbiological Media. 4th edition. Taylor and Francis Group, LLC, USA, pp. 228,990.
- Bahashwan, S. A., and El-Shafey, H. M., 2013. Antimicrobial resistance patterns of proteus isolates from clinical specimens. European Scientific Journal, Vol. 9, No. 27.
- Brumfitl, W., Hamilton –Miller, M.T. (1985). Development of bacterial resistance during treatment of UTI, a constant clinical challenge .in:Recent advice in treatment of UTI.Role society of medicine services international congress and symposium series,No.97.
- Bauer, A.W., Kirby, W.M., Sherris, J.C., Turck, M., 1966. Antibiotic susceptibility testing by a standardized single disk method. Am. J. Clin. Pathol., 45(4):493–496.
- Benson, J. H., 2001. Benson: Microbiological applications Laboratory manual in general Microbiology. 8th edition. McGraw-Hill Companies, Inc., New York; p. 173.
- Bergan, T., 1997. Urinary Tract Infections. Infectiology. Basel, Karger, Vol. 1, p. 19.
- Beyene, G., and Tsegaye, W., 2011. Bacterial Uropathogens in Urinary tract Infection and Antibiotic Susceptibility Pattern in Jimma University Specialized Hospital, South west Ethiopia. Ethiop. J. Health Sci., Vol. 21, No. 2.
- Bonadio, M., Costarelli, S., Morelli, G., and Tartaglia, T., 2006. The influence of diabetes mellitus on the spectrum of uropathogens and the antimicrobial resistance in elderly adult patients with urinary tract infection. BMC Infectious Diseases, Vol.6, No.54.
- Brooks, G. F., Carroll, K. C., Butel, J. S., and Morse, S. A., 2007. Jawetz, Melnick & Adelberg's Medical Microbiology. 24th ed. McGraw-Hill Companies, Inc., New York; pp. 25-264.
- Cavaliere, S. J., Rankin, I. D., Harbeck, R. J., Sautter, R. L., McCarter, Y. S., Sharp, S. E., Ortez, J. H., and Speigel, C. A., 2005. Manual of Antimicrobial Susceptibility Testing. American Society for Microbiology, p. 40-48.

- Chamberlain, N.L., 2009. The Big Picture Medical Microbiology. McGraw-Hill Companies, Inc., New York; p. 341-345.
- Chang, S. T., and Shortliffe, L. D., 2006. Pediatric Urinary Tract Infections. *Pediatr. Clin. N. Am.*, 53, 379-400.
- Cheesbrough M., 2006. District Laboratory Practice in Tropical Countries. 2nd edition. Part 2. Cambridge University Press, New York; pp. 46 – 189.
- Chen, M. J., Cheng, H. L., and Chiou, Y. Y., 2013. Risk Factors for Renal Scarring and Deterioration of Renal Function in Primary Vesico-Ureteral Reflux *Children: A Long-Term Follow-Up Retrospective Cohort Study. *PLoS One*, 8(2): e57954.
- Chessbrugh, M.(1992). Medical Laboratory Manual for Tropical Countries. Vol 2. Butter worth Heine mann. Tropical Health Technol.,214-216.
- Coker, C., Carrie, A. P., and Harry, L. T., 2000. Pathogenesis of *Proteus mirabilis* in urinary tract infection, *J. Microb. And Inf.*, 2(12): 1497-1505.
- Cullor, J. (1996). Endotoxin and disease in food animals. *Comp. Cont. Ed. Food Animal*. 18:31-38
- Davis N. F., and Flood, H. D., 2011. The Pathogenesis of Urinary Tract Infections, Clinical Management of Complicated Urinary Tract Infection. Dr. Ahmad Nikibakhsh (Ed.), ISBN: 978-953-307-393-4, InTech, pp. 101-120.
- Dishvarian, J. A., 2010. Detection of some bacterial infection in urinary tract and their antibiotic sensitivity. *International Journal for Sciences and Technology*, Vol. 5, No. 3.
- Dulczak, S., Kirk, J., 2005. Overview of the evaluation, diagnosis, and management of urinary tract infections in infants and children. *Urol Nurs*, 25(3):185-191.
- Emiru, T., Beyene, G., Tsegaye, W., and Melaku, S., 2013. Associated risk factors of urinary tract infection among pregnant women at Felege Hiwot Referral Hospital, Bahir Dar, North West Ethiopia. *BMC Research Notes*, 6: 292.
- Fareid, M. A., 2012. Frequency and Susceptibility Profile of Bacteria Causing Urinary Tract Infections among Women. *New York Science Journal*, 5 (2).

- Forbes, B. A., Sahm D. F., and Weissfeld, A. S., 2007. *Bailey & Scott's Diagnostic Microbiology*. 12th edition. Mosby, Inc., an Affiliate of Elsevier Inc.; pp. 219-854.
- Francesco, M. A. D., Ravizzola, G., Peroni, L., Negrini, R., and Manca, N., 2007. Urinary tract infections in Brescia, Italy: Etiology of uropathogens and antimicrobial resistance of common uropathogens. *Med. Sci. Monit.*, 13(6): BR136–144.
- Geffers, C., and Gastmeier, P. 2011. Nosocomial infections and multidrug resistance organisms– epidemiological data from KISS. *Dtsch Arztebl Int.*, Vol. 108 (6), pp. 87–93.
- Gillespie, S. H., and Hawkey, P. M., 2006. *Principles and Practice of Clinical Bacteriology*. 2nd edition. John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex PO19 8SQ, England; pp. 383-392.
- Goldman, E., and Green, L. H., 2009. *Practical Handbook of Microbiology*. 2nd edition. Taylor & Francis Group, LLC, pp. 74-687.
- Gupta, K., Hooton, T. M., and Stamm, W. E., 2001. Increasing Antimicrobial Resistance and the Management of Uncomplicated Community-Acquired Urinary Tract Infections. *Ann Intern Med.*, 135:41-50.
- Haley R.W., Culver D.H., White JW, Morgan W.M, and Emori, T G. (1985). The nationwide nosocomial infection rate: A new need for vital statistics. *AmJ Epidemiol*; 121:159. Cited by (walsh,1998).
- Hamdan, H. Z., Ziad, A. H. M., Ali, S. K., and Adam, I., 2011. Epidemiology of urinary tract infections and antibiotics sensitivity among pregnant women at Khartoum North Hospital. *Annals of Clinical Microbiology and Antimicrobials*, 10: 2.
- Handley, M. A., Reingold, A. L., Shiboski, S. and Padian, N. S. (2002) Incidence of acute urinary tract infection in young women and use of male condoms with and without nonoxynol-9 spermicidal. *Epidemiology*, 13(4), 431-6.
- Hindi, N. K. K., Hasson, S. O., and Hindi, S. K. K. 2013. Bacteriological Study of Urinary Tract Infections with Antibiotics Susceptibility to Bacterial Isolates

among Honeymoon Women in Al Qassim Hospital, Babylon Province, Iraq. *British Biotechnology Journal*, 3(3): 332-340.

Hoepelman, A. I. M., Meiland, R., and Geerlings, S. E., 2003. Pathogenesis and management of bacterial urinary tract infections in adult patients with diabetes mellitus. *International Journal of Antimicrobial Agents*, 22: S35-S43.

Hooton, T. M., 2000. Pathogenesis of urinary tract infections: an update. *Journal of Antimicrobial Chemotherapy*, 46 Suppl. S1, 1-7.

Hooton TM. Clinical practice. Uncomplicated urinary tract infection. *N Engl J Med*. 2012; 366:1028–1037.

Jha, N., and Bapat, S.K., 2005. A study of sensitivity and resistance of pathogenic micro organisms causing UTI in Kathmandu valley. *Kathmandu University Medical Journal*, Vol. 3, No. 2, Issue 10,123-129.

John, J. F., and Harvin, A. M., 2007. History and evolution of antibiotic resistance in coagulase-negative staphylococci: Susceptibility profiles of new anti-staphylococcal agents. *Therapeutics and Clinical Risk Management*, 3 (6):1143–1152.

Kasper, D.L., Braunwald, E., Fauci, A.S., Hauser, S. Longo, D.L., and Jameson, L. J., 2005. *Harrison's Principles of Internal Medicine*. 16th edition. McGraw-Hill Companies, Inc., New York; pp.701-1719.

Kavaler, E., 2006. *The Essential Guide to Urinary Tract Problems in Women*. Copernicus Books, an imprint of Springer Science+Business Media, New York; 230-242.

Klevens, R. M., Edwards, J. R., Richards, C. L., Horan, T. C., Gaynes, R. P., Pollock, D. A., and Cardo, D. M., 2007. Estimating health care-associated infections and deaths in U.S. hospitals, 2002. *Public health reports*. Vol. 122 (2):160-6.

Kolawole, A. S., Kolawole, O. M., Kandaki-Olukemi, Y. T., Babatunde, S. K., Durowade, K. A. and Kolawole, C. F., 2009. Prevalence of urinary tract infections (UTI) among patients attending Dalhatu Araf Specialist Hospital,

- Lafia, Nasarawa State, Nigeria. *International Journal of Medicine and Medical Sciences*, Vol.1, (5), pp.163-167.
- Kunin, C.M. (1997). *Role of Host Defense, Detection, Prevention and Management of Urinary tract infection* Philadelphia, Lea and Febiger, Ch.96, 299-300p.
- Lewis, D. A., Gumede, L. Y. E., van der Hoven, L. A., de Gita, G. N., de Kock, E. J., de Lange, T., Maseko, V., Kekana, V., Smuts, F. P., and Perovic, O., 2013. Antimicrobial susceptibility of organisms causing community-acquired urinary tract infections in Gauteng Province, South Africa. *SAMJ*, Vol. 103, No. 6.
- Ligozzi, M., Bernini, C., Bonora, M. G., Fatima, M., Zuliani, J., and Fontana, R., 2002. Evaluation of the VITEK 2 System for Identification and Antimicrobial Susceptibility Testing of Medically Relevant Gram-Positive Cocci. *Journal of Clinical Microbiology*, Vol. 40, No. 5, p. 1681–1686.
- Linhares, I., Raposo, T., Rodrigues, A., and Almeida, A., 2013. Frequency and antimicrobial resistance patterns of bacteria implicated in community urinary tract infections: a ten-year surveillance study (2000–2009). *BMC Infectious Diseases*. Vol. 13, no. 1.
- Mandal, J., Acharya, N. S., Buddhapriya, D., and Parija, S. C., 2012. Antibiotic resistance pattern among common bacterial uropathogens with a special reference to ciprofloxacin resistant *Escherichia coli*. *Indian J. Med. Res.*, 136, pp. 842-849.
- Maskell, R. (1995). Brodening the concept of urinary tract infection, *Brit. Med. J.* 76:2-8.
- Moyhenaglam; Douglas, F.B. and Kenneth, F. (1988). Prevalence of *Gardnerella vaginalis* in the urinary tract, *J. of Clin. Microbiol.* 2:1130-1133.
- Martineau, F., Francois, J.P., Charistian, M., Paul, H.R., Marc, O. and Michel, G.B. (2000). Development of a rapid PCR assay specific for *Staphylococcus saprophyticus* and application to direct detection from urine sample, *J. of Clin. Microbiology*. 38:3280-3284.
- Matthews KR, Roberson J, Gillespie BE, Luther DA, Oliver SP (1997). Identification and Differentiation of Coagulase Negative *Staphylococcus*

- aureus by Polymerase Chain Reaction. *Journal of Food Protection* 60 (6): 688.
- Mattoo, T. K., 2012. Vesicoureteral Reflux and Reflux Nephropathy. *Adv. Chronic Kidney Dis.*, 18(5): 348–354.
- Mazzulli, T., 2012. Diagnosis and management of simple and complicated urinary tract infections (UTIs). *The Canadian Journal of Urology*, 19, Supplement 1.
- Melekos, M.D., and Kurt, G.N., 2000. Complicated urinary tract infection. *Intern. J. of Antimicrob. Age.*, 15: 247-256.
- Mezall, T. J., Ajeel, N. A., and Hasony, H. J., 2011. Antimicrobial resistance of uropathogens in Basrah. *MJBU*, Vol 29, No. 1&2.
- Mohammed, S. M., 2005. Isolation and identification of *Enterobacter* spp. And *Klebsiella pneumonia* from some clinical cases and susceptibility to some antibiotics. M.Sc. Thesis. College of Science. University of Al-Anbar.
- Montague, D.K.; Kenneth, W.A. and Milton, M.L. (1995). Culturs from grnitourinary prostheses at reapportion, therole of *Stphylococcus epidermidis* in periprothetic infection, *J. of Urol.*, 154:387-390.
- Mulvey, M. A., 2002. Adhesion and entry of uropathogenic *Escherichia coli*. *Cellular Microbiology*, 4(5), 257–271.
- Mishra, M. P., Debata, N. K., and Padhy, R. N., 2013. Surveillance of multidrug resistant uropathogenic bacteria in hospitalized patients in Indian. *Asian Pac J Trop Biomed*, 3(4): 315-324.
- Metri, B. C., Jyothi, P., and Peerapur, B. V., 2013. Antibiotic resistance in *Citrobacter spp.* isolated from urinary tract infection. *Urol Ann.*, 5(4): 312–313.
- National Collaborating Centre for Women’s and Children’s Health, Commissioned by the National Institute for Health and Clinical Excellence. Urinary tract infection in children: diagnosis, treatment and long-term management. London: RCOG Press; 2007.
- Neal, D. E., 2008. Complicated Urinary Tract Infections. *Urol. Clin. N. Am.*, 35, 13–22.

- Okonko, I.O., Ijandipe, L. A., Ilusanya, A. O., Donbraye-Emmanuel, O. B., Ejembi, J., Udeze, A. O., Egun, O. C., Fowotade A., and Nkang A.O., 2010. Detection of Urinary Tract Infection (UTI) among pregnant women in Oluyoro Catholic Hospital, Ibadan, South-Western Nigeria. *Malaysian Journal of Microbiology*, Vol. 6 (1), pp. 16-24.
- Ouno, G. A., Korir, S. C., Cheruiyot, J. C., Ratemo, O. D., Mabeya, B. M., Mauti, G. O., Mauti, E. M., and Kiprono, S. J., 2013. Isolation Identification and Characterization of Urinary Tract Infectious Bacteria and the Effect of Different Antibiotics. *Journal of Natural Sciences Research*, 3 (6), 150-159.
- Pandey, J. K., Narayan, A., and Tyagi, S., 2013. Prevalence of *Proteus* species in clinical samples, antibiotic sensitivity pattern and ESBL production. *Int. J. Curr. Microbiol. App. Sci.*, 2 (10): 253-261.
- Parson C.L. (1988). Protocol for treatment of typical urinary tract infection, criteria for antimicrobial selection. *Viol.* 32.22-25.
- Perilla, M. J., Ajello, G., Bopp, C., Elliott, J., Facklam, R., Knapp, J. S., Popovic, T., Wells, J., and Dowell, S. F., 2003. *Manual for the Laboratory Identification and Antimicrobial Susceptibility Testing of Bacterial Pathogens of Public Health Importance in the Developing World*. WHO & CDC, pp. 194-206.
- Prakash, D., and Saxena, R. S., 2013. *Distribution and Antimicrobial Susceptibility Pattern of Bacterial Pathogens Causing Urinary Tract Infection in Urban Community of Meerut City, India*. Hindawi Publishing Corporation, *ISRN Microbiology*, Vol. 2013.
- Probert, J. L., 2009. *An Atlas of Investigation and Diagnosis urology*. Atlas Medical Publishing Ltd., UK; p. 9-21.
- Ronald, A. (2003). The etiology of urinary tract infection. Traditional and emerging pathogens', *Dis Mon*, 49(2), 71-82.
- Ryan KJ, Ray CG (editors). (2004). *Sherris Medical Microbiology (4th Ed.)*. McGraw Hill. ISBN 0-8385-8529-9.
- Ryan, K. J. (2004). Enterobacteriaceae. In K. J. Ryan, & C. G. Ray (Eds.), *Sherris Medical Microbiology: An Introduction to Infectious diseases (4th ed., pp. 343-371)*. USA: McGraw-Hill,

- Renuart, A. J., Goldfarb, D. M., Mokomane, M., Tawanana, E. O., Narasimhamurthy, M., Steenhoff, A. P., and Silverman, J. A., 2013. Microbiology of Urinary Tract Infections in Gaborone, Botswana. *PLoS One*, 8 (3): e57776.
- Ristucci, Patricia; Cunha, Burke (July 1984). "Infection Control". Cambridge University Press on behalf of The Society for Healthcare Epidemiology of America: 343–348.
- Ryan, K. J., Ray, C. G., Champoux, J. J., Drew, W. L., Neidhardt, F. C., and Plorde, J. J., 2004. *Sherris Medical Microbiology an Introduction to Infectious Diseases*. 4th edition. McGraw-Hill Companies, Inc., New York; pp. 294-871.
- Satcher, D; Hughes J.M.; Cohn, M.L.; Macedo, C.G.; Bennet, D.B.; Paganini, J.M. and Escutia, V. (1993). *Laboratory methods for diagnosis of Vibrio cholera*. CDC.USA.
- Schrier, R. W., 2007. *Diseases of the Kidney and Urinary Tract*. 8th edition. Lippincott
- Schrier, R. W., 2007. *Diseases of the Kidney and Urinary Tract*. 8th edition. Lippincott Williams & Wilkins, USA; pp. 832-840.
- Shaaban, M. T., Ghozlan, H. A., and El Maghraby, M. M., 2012. Susceptibility of bacteria infecting urinary tract to some antibiotics and essential oils. *Journal of Applied Pharmaceutical Science*. 02 (04): 90-98.
- Sharma, P. U., and Bidwai, U., 2013. Isolation and identification of bacteria causing urinary tract infections in pregnant women in vidarbha and their drug susceptibility patterns in them. *Int. J. Curr. Microbiol. App. Sci.*, 2 (4): 97-103.
- Swati Banerjee, PhD Associate Professor, Department of Microbiology, Padmashri Dr Vikhe Patil Medical College, and Hospital. (2009). *the Study of Urinary Tract Infections and Antibigram Of Uropathogens In and Around Ahmadnagar, Maharashtra*. 2009
- Sweih, N. A., Jamal, W., and Rotimi, V. O., 2005. Spectrum and Antibiotic Resistance of Uropathogens Isolated from Hospital and Community Patients

with Urinary Tract Infections in Two Large Hospitals in Kuwait. *Med. Princ. Pract.*, 14:401–407.

Stamm, W. E. and Turk, M. (1991). Urinary tract infection, Pyelonephritis and Principle of internal medicine, 12Ed., Wilson, Braunwald, Adams, Peters Dof, Iss Elbacher, Martin (eds) part 5, Section 3, McGraw-Hill International Book Co. 538p.

Toval, F., Köhler, C. D., Vogel, U., Wagenlehner, F., Mellmann, A., Fruth, A., Schmidt, M. A., Karch, H., Bielaszewska, M., and Dobrindt, U., 2014. Characterization of *Escherichia coli* Isolates from Hospital Inpatients or Outpatients with Urinary Tract Infection. *Journal of Clinical Microbiology*, Vol. 52, No. 2, p. 407–418.

Thomas, F.E., Jackson, R. T., Melly, M. and Alford, R.H. (1977). Sequential hospital wide outbreaks of resistant *Serratia* and *Klebsiella* infections, *Arch. Intern. Med.* 137:581.

VanDeGraaff, K. M., Morton, D. A., and Crawly, J. L., 2011. A Photographic Atlas for the Anatomy and Physiology Laboratory. 7th edition. Morton Publishing Company, USA; p. 156.

Vandepitte, J., Verhaegen, J., Engbaek, K., Rohner, P., Piot, P., and Heuck, C. C., 2003. Basic laboratory procedures in clinical bacteriology. 2nd edition. World Health Organization, Geneva; pp. 30-36.

Vergidis P, Patel R. Novel approaches to the diagnosis, prevention, and treatment of medical device-associated infections. *Infect Dis Clin North Am.* 2012 Mar. 26(1):173-86.

William, I., Boswell, T., and Ala Aldeen, D., 2006. Medical Microbiology. Taylor & Francis Group, New York; p.314.

Wilson, M. L., and Gaido, L., 2004. Laboratory Diagnosis of Urinary Tract Infections in Adult Patients. *Clin. Infect Dis.*, 38: 1150–1158.

Yasufuku, T., Shigemura, K., Shirakawa, T., Matsumoto, M., Nakano, Y., Tanaka, K., Arakawa, S., Kawabata, M., and Fujisawa, M., 2011. Mechanisms of and Risk Factors for Fluoroquinolone Resistance in Clinical *Enterococcus*

faecalis Isolates from Patients with Urinary Tract Infections. J. CLIN. Microbiol., Vol. 49, No. 11: p. 3912–3916.



CURRICULUM VITAE (CV)

- **Personal Details**

Name: May Ali Saleh

Surname: AL-HAMADANI

Sex: Female

Status: Married

Date of birth: 28. 01. 1990

Place of birth: Iraq – Erbil

Home Address: Iraq – Erbil – Zanko quarter

Mob: +9647504483125,

E-mail:- hudamayhunar@gmail.com

- ❖ I graduated from CihanUniversity / College of Science / Biology Department, in 2013-2014 year.
- ❖ Now I am Laboratory Assistant - Biology Department in Cihan University

- **Experience**

- ❖ I took English language course in Cambridge Center.
- ❖ I took a course of computer like (Microsoft Office) in IT Technology.
- ❖ I worked in laboratories for two years

- **Hopes:**

- ❖ I hope to get a PhD in Microbiology.
- ❖ Not allowed in the CV section, also CV must be written in third person perspective, please adjust this issue.