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Prevalence of Asymptomatic Bacteriuria in HIV Positive Patients and The Antibiotic Susceptibility of The Bacterial Isolates

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Abstract—— ***Urinary tract infection remains a major public health problem in developing countries, where there are limited health-care services. Its prevalence is fueled by human immunodeficiency virus (HIV) infection. The emergence of antimicrobial resistance is now widespread and poses a serious clinical threat. This cross sectional study was conducted during January-April/ 2023 to determine the prevalence, etiology and antimicrobial susceptibility pattern of isolates among HIV positive individuals compared to healthy controls. A total 180 mid-stream urine and blood samples were collected from 100 previously confirmed HIV positive patients attending Benghazi center for infectious diseases and immunology for their routine investigations, and 80 age and gender matched healthy subjects (control). The study consisted of 97 females, and males 83, with age ranging from 20 to 47. About 50 ml of clean–catch midstream urine was collected from each patient into sterile screw–capped universal container. The specimen was mixed, labeled and transported to the laboratory for processing. The bacterial species were identified by conventional laboratory methods according to the international standards. The results of the study revealed that, the prevalence evaluated by the number of organisms which showed significant growth on the culture among HIV positive subjects was 28%, while in the control group it was 35%, but no significant differences in the prevalence among both groups. No significant differences in the prevalence of UTI according to the age groups among both HIV positive subjects and control subjects. The prevalence of UTI was higher in females than males in general, with significant differences among HIV positive subjects, but with no significant differences among the control group. Totally 56 bacterial species were isolated from the subjects of the study, 67.9% were gram negative, 32.1% were gram positive. Among HIV positive subjects 21 (75%) were gram negative and 7 (25%) were gram positive, among the control group 17 (60.7%) were gram negative and 11 (39.3%) were gram negative, among the gram negative species, E. coli was the most predominant bacterial etiology among the study population (33.9%), followed by K. pneumonia it was (25%) equally in both groups, P. aeruginosa was (8.9%), which was more seen in HIV positive subjects than control. while among gram positive species, S. aureus was the most common pathogen (17.9%) equally found in both groups, S. agalactiae was (5.4%), more frequently in HIV subjects than control. S. saprophyticus was (3.6%), S. pyogenes was (4.5%) seen in the control group only with no significant differences in the distribution of the isolated species. The isolated uropathogens from HIV positive subjects were tested for their sensitivity to 12 antimicrobial agents, the results of antimicrobial susceptibility testing revealed that most species were resistant to more than three commonly used antimicrobial agents.***

Keywords- HIV, ***Bacteriuria in HIV, Patients, Antibiotic***

I. INTRODUCTION

UTI is defined as the presence of some pathogenic microorganism that induces a local or systemic inflammatory response. The infection can affect any structure of the urinary tract including the renal parenchyma, pelvic, ureters, bladder, and urethra. In general, infections located above the ureterovesical junction are considered as high infections and below this junction are considered as low. High-location UTIs are considered more severe but less frequent, and their exact location is often difficult to determine. UTI is considered the most common bacterial infection, estimated to affect more than 150 million people annually (Foxman, 2002). From 15 to 50 years of age, UTI is practically nonexistent in men, while in women, it has a prevalence that can reach up to 3% of the population (González-Chamorro *et al*., 2012). Approximately, one in three women suffers an uncomplicated UTI before the age of 24 years old, and 30–44% develop a recurrent disease (Foxman *et al*., 2000). Positive urine culture is defined when it shows a bacterial colony count of greater than or equal to 103 colony-forming units per µl of a typical urinary tract organism (Nicolle, 2008). They are the most frequent community-acquired infections in the world and the most common pathogens are *E. coli* (Magliano *et al*., 2012). Urinary tract infections, both symptomatic and asymptomatic, are serious public healthcare problems decreasing the quality of life and leading to work absence (Olowe *et al*., 2015).

Most microorganisms colonize the colon, perianal region and periurethral and introitus region in females can cause urinary tract infections (Debalke *et al*., 2014). The frequency and severity of UTI is determined by local uroepithelial defense system and pathogenic factors of the micro-organisms. The common route of spread is ascension

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and the rate depends on effectiveness of the commensal flora in preventing colonization, local trauma such as in sexual activity and urethral massage, abnormalities of the urinary tract, diagnostic procedures, high vaginal pH and micro-organism virulence factors. The virulence factors which ensure micro-organism survival include fimbriae, motility, glycocalyx-mediated adherence, urease production, production of haemolysin, somatic antigen expression and synthesis of aerobactin and enterobactin (Johnson *et al*., 2017). Specific groups of people are at increased risk of urinary tract infection. Vulnerable populations are women, especially during pregnancy, infants, elderly patients and low socioeconomic status (Nicolle, 2008; Nelson and Good, 2015). Also certain conditions may increase susceptibility to infections i.e. spinal cord injuries, urinary catheters, diabetes, multiple sclerosis, immunodeficiency and underlying urologic abnormalities (Mladenovi *et al*., 2015). Vulnerable individuals, apart from being at risk of developing the condition, they are also at risk for recurrences, require long treatment duration and may develop more complex infection like pyelonephritis (Skrzat-klapaczy *et al*., 2018). HIV positive patients are also prone to urinary tract infections. The incidence of urinary tract infections in HIV population is clearly related to infection and immune function, determined by lymphocytes $CD4^+$ cells count (de Gaetano *et al*., 2003). As confirmed by observational studies the incidence of various bacterial infections in HIV-infected patients, including urinary tract infections, is inversely correlated with lymphocyte $CD4^+$ count (Banu and Ramachandrian, 2013). It is therefore interesting to note that although the wide introduction of antiretroviral therapy has dramatically reduced morbidity related to AIDS, non- AIDS defining infections remain an important and frequent clinical problem (de Gaetano *et al*., 2003). This may result from increased frequency of non-HIV related diseases in the HIV population, such as diabetes and glucose metabolism disturbance, liver cirrhosis and metabolic syndrome (Lombo *et al*., 2015; Palmer *et al*., 2016). Moreover the HIV population in Poland is aging, bringing new risk factor for non-HIV related infections. It is therefore crucial to observe the burden of such infections, its outcomes and change in clinical characteristics (Kowalska *et al*., 2016). It is also noted that the spectrum of pathogens is more broad and diverse than in general population including less common microorganisms (Hrbacek *et al*., 2010). Improving knowledge on the prevalence and prognostic factors for urinary infections in HIV patients along with better recognition of the causative pathogens could substantially improve current diagnostic and treatment guidelines translating into better prognosis for HIV- positive patients (Lebovitch and Mydlo, 2008). 1- The prevalence of bacteriuria in people living with HIV (PLHIV) has been documented to be high, and vary from one place to another ranging from 4–25.3%. (Murugesh *et al*., 2014; Skrzat-klapaczy *et al*., 2018). Risk factors for having bacteriuria in PLHIV include sex, and lower CD4 cell count (Fenta *et al*., 2016; Skrzat-klapaczy *et al*., 2018). PLHIV have a diverse and broad spectrum of microbes causing BUTI. The most prevalent bacteria isolates causing bacteriuria in PLHIV were previously noted to be *E. coli, E. faecalis*, and *S. aureus* (Fenta *et al*., 2016; Skrzat-klapaczy *et al*., 2018) Others have found *Enterococcus* spp. as the most common isolates in HIV-infected subjects while control group had *E. coli* (Schönwald *et al*., 1999). In this group of PLHIV, they have uropathogens which demonstrate moderate to high resistance against commonly prescribed antibiotics (Klasinc *et al*., 2017). Multidrug Resistance (MDR) has been demonstrated in 58.3% and in up to 78.4% of isolates causing ABU in PLHIV (Fenta *et al*., 2016). MDR is the public health problem globally particularly in developing countries (Kemajou *et al*., 2016). This high prevalence of MDR isolates is a threat and therefore routine screening for bacteriuria has been recommended for early identification and treatment in order to prevent spread but also for betterment of patients (Skrzat-klapaczy *et al*., 2018). 2- HIV infected patients have higher risk of acquiring kidney disease (acute or chronic) due to the effects HIV virus on the renal system, immune mediated mechanisms, risk of infections and side effects of antiretroviral therapy (ART). HIV infected patients have been shown to have increased risk of UTI (Hamdam *et al*., 2011; Aswani *et al*., 2014), however this risk seem to be inversely proportional to the lymphocytes count or immune function of the patient (Hammar *et al*., 2010; Hirji *et al*., 2012). There has not been a difference in the prevalence of bacterial growth between ART users and non-users, *E. coli* is a predominant isolate (Hirji *et al*., 2012; Al-Rubeaan *et al*., 2013; Aswani *et al*., 2014).

A. Aims of the study:

This study aims to determine the prevalence of UTI among HIV patients and HIV sero-negative subjects including a most common etiological agent.

II. METHODS AND MATERIALS

A. *Study location and design*:

This cross sectional study was conducted at Benghazi center for infectious diseases and immunology, during January-April/ 2023 to determine the prevalence, etiology and antimicrobial susceptibility pattern of isolates among HIV positive individuals compared to healthy controls.

B. Study population and sample size:

A total 180 mid-stream urine and blood samples were collected from 100 previously confirmed HIV positive patients attending Benghazi center for infectious diseases and immunology for their routine investigations, and 80 age and gender matched healthy subjects (control). The study consisted of females, and

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males, with age ranging from 20 to 47 years old. Samples were collected from patients who were not on antibiotic treatment, while those that have had antibiotic treatment 2 months prior to sample collection were excluded. Samples were collected after oral informed consent was given by the patients.

C. Patient education on urine collection:

Prior to urine collection, education of subjects on the procedure for proper, adequate and careful collection of mid-stream urine sample (after voiding the initial stream) was done without prior cleansing or washing of the perineum.

D. Specimen collection and processing:

About 50 ml of clean–catch midstream urine was collected from each patient into sterile screw–capped universal container, containing, few crystals of boric acid as preservative. The specimen was mixed, labeled and transported to the laboratory for processing.

E. Transport and storage of samples:

Once collected, mid-stream specimens of urine were transported to the laboratory without delay.

F. Processing of Urine Specimen:

Microscopic Examination of uncentrifuged sample of urine. Microscopic examination of the un-centrifuged urine sample using x10 and x40 objective lens was done after seeding of MacConkey, cystine lactose electrolyte-deficient agar (CLED) and blood agar media on 90mm Petri dish for culture. Microscopic examination of urine sample was done for recognition of organism morph type and Gram stain reaction.

Seeding of Plate: (Standard Loop Method)

The seeding of plate MacConkey, CLED agar and blood agar was done immediately after macroscopic examination to minimize the contamination of urine samples. Using a commercially available, sterile, standard circular wire loop, charged with mixed un-centrifuged urine sample, pre-incubated CLED agar and MacConkey agar plates were seeded respectively adopting a plating procedure that yielded discrete colonies. The plates were incubated aerobically at 35oC for 18-24 hours, the number of discrete colonies was estimated. After 24 hours of incubation, if similar colonies were found in numbers suggesting significant bacteriuria, cultures of mixed growth were ignored, culture with less than 30 colonies were considered of no significant growth.

Reporting colonial morphology:

A. MacConkey agar:

The colonial morphology on MacConkey as colony size, edge, surface, consistency, odour and the reaction of the bacteria with the medium (Fermintation) were noted. Members of nonfastidious gram-negative bacilli such as Enterobacteriaceae and Pseudomonas spp. were grown on this media

B. CLED medium:

The colonial morphologies of the isolates, colonial reaction with the medium may reveal lactose- fermenters as yellowish discoloration of the initially bluish medium around the areas with growth whereas the non- lactose fermenters or slow fermenters showed no colour change in the medium. Apart from reaction with the medium, other colonial morphologies like the colony size (diameter in mm), shape, colour, outline (circular, entire, wavy, indented), elevation (flat, raised, low convex, dome shaped) translucency (clear and transparent, opaque) mucoid, consistency and odour were noted.

C. Blood agar:

Gram positive bacteria appeared as clear colonies with Beta hemolysis which seen as clear zones around the colonies.

Fig. (I): *Significant growth on MacConkey agar.*

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Fig. (II): *Significant growth on CLED medium.*

Fig. (III): *Significant growth on Blood agar medium.*

A. Gram stain:

Gram stain of the isolates after overnight incubation on Blood agar and CLED were performed to determine whether the Gram reaction, shape and spatial arrangement.

- *Principal:*

Gram staining is a complex and differential staining procedure. Through a series of staining and decolorization steps, organisms in the Domain Bacteria are differentiated according to cell wall composition. Gram-positive bacteria have cell walls that contain thick layers of peptidoglycan (90% of cell wall). These stain purple. Gram-negative bacteria have walls with thin layers of peptidoglycan (10% of wall), and high lipid content. These stain pink. This staining procedure is not used for Archeae or Eukaryotes as both lack peptidoglycan. The performance of the Gram Stain on any sample requires 4 basic steps that include applying a primary stain (crystal violet) to a heat-fixed smear, followed by the addition of a mordant (Gram's Iodine), rapid decolorization with alcohol, acetone, or a mixture of alcohol and acetone and lastly, counterstaining with safranin.

Materials:

- Crystal Violet, the primary stain
- Iodine, the mordant
- A decolorizer made of acetone and alcohol (95%)
- Safranin, the counterstain

*Procedure***:**

1. A suspension of one colony was taken from culture media by a sterile loop , and spread on a clean slide contain a drop of water , they then mixed gently and fixed by heat.
2. The smear covered with crystal violet stain for 30–60 seconds. The stain then washed rapidly
3. Lugol's iodine was used to cover the smear for 30–60 seconds. The stain washed off with clean water.
4. The smear was decolorized rapidly (few seconds) with acetone–alcohol, and washed immediately with clean water.
5. The smear then covered with neutral red stain for 2 minutes.
6. The stain then washed off with clean water, the back of the slide was then wiped clean, and placed in a draining rack for to get dry.
7. The smear was examined microscopically, first with the 40 object to check the stain and to examine the distribution of bacteria, and then with the oil to report the type of bacteria

Interpretation:

Gram-negative bacteria will stain pink/red and Gram-positive bacteria will stain blue/purple.

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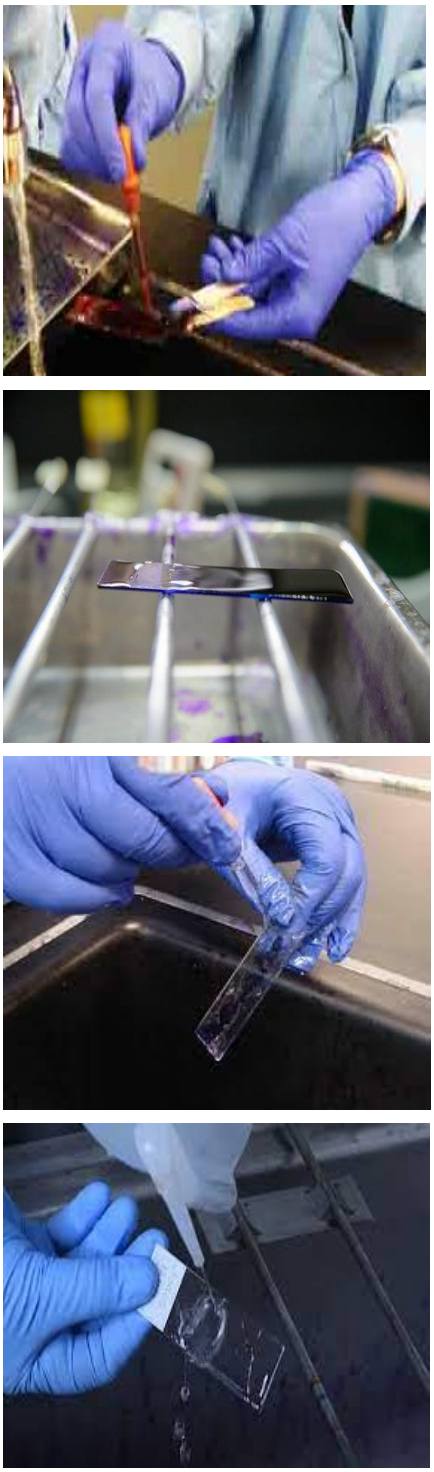

Fig. (IV): *Gram staining technique.*

B. ***Bacterial Identification:***

The biochemical tests were made based on the Gram stain reaction and whether the isolate was lactose fermenting or non-lactose fermenting on CLED medium were carried out.

Gram negative bacteria:

A. *Oxidase test:*

Principal:

The oxidase test is used to identify bacteria that produce cytochrome c oxidase, an enzyme of the bacterial electron transport chain. When present, the cytochrome c oxidase oxidizes the reagent (tetramethyl-p-phenylenediamine dihydrochloride)to indophenols, a purple or dark blue color end product. When the enzyme is not present, the reagent remains reduced and is colorless.

Materials:

Freshly prepared Kovács oxidase reagent (1% tetra-methyl-p-phenylenediamine dihydrochloride, in water).

Procedure:

1. Using a loop a well-isolated colony was picked from a fresh bacterial plate and rubbed onto a small piece of filter paper.
2. 1 or 2 drops of 1% Kovács oxidase reagent was added on the organism smear.
3. The change of the color is observed.

Result interpretation

- Oxidase positive: color changes to dark purple within 5 to 10 seconds.
- Oxidase negative: color does not change or it takes longer than 2 minute
- The test is positive for *P. aeruginosa* and negative for Enterobacteriaceae.

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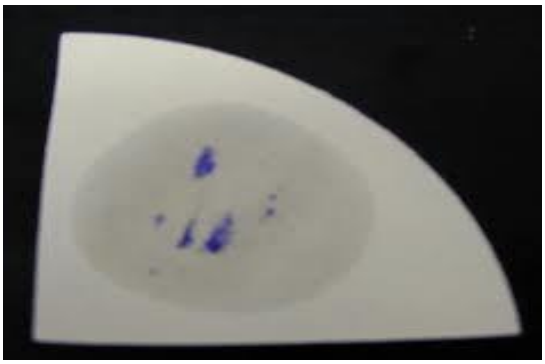

Fig. (V): *Oxidase test.*

B. Levine EMB (eosin methylene blue):

Principal:

EMB agar is an example of a selective and differential medium. This means that only some bacteria will grow on this agar and that the appearance of those that do grow will be different. In particular, EMB agar inhibits the growth of Gram-positive bacteria and helps differentiate some of the Gram-negative rods.

Procedure

1. A freshly prepared EMB agar is obtained by melting the agar in Petri plate and allowed to solidify at room temperature.
2. A clear colonies was selected and inoculated by streaking on the EMB agar plates.
3. The plate is incubated upside down for least 48 hours.
4. After the incubation period, any color changes were recorded.

Interpretation

EMB agar contains lactose and the dyes eosin and methylene blue. The fermentation oflactose by some Gram-negative rods produces acidic products that react with the dyes to produce colored colonies. *E. coli* colonies produce a green, metallic sheen. *Pseudomonas aeruginosa* colonies are colorless indicating no fermentation

Fig. (VI): *Growth of E. coli on EMB agar.*

C. API (Analytical Profile Index) 20E:

API is a biochemical panel (bioMérieux Inc)for identification and differentiation of members of the family Enterobacteriaceae.

Principle

The API range provides a standardized, miniaturized version of existing identification techniques, which up until now were complicated to perform and difficult to read. In the API 20E, the plastic strip holds twenty mini-test chambers containing dehydrated media having chemically-defined compositions for each test. They usually detect enzymatic activity, mostly related to fermentation of carbohydrate or catabolism of proteins or amino acids by the inoculated organisms.

A bacterial suspension is used to rehydrate each of the wells and the strips are incubated. During incubation, metabolism produces color changes that are either spontaneous or revealed by the addition of reagents. All positive and negative test results are compiled to obtain a profile number,

which is then compared with profile numbers in a commercial codebook (or online) to determine the identification of the bacterial species.

Materials:

- API E20 test kit (bioMérieux Inc)
- API NaCl 0.85 % Medium or API Suspension Medium
- API 20 E reagent kit
- Zn reagent
- Mineral oil

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- API 20 E Analytical Profile Index

Procedure:

1. A single isolated colony (from a pure culture) was picked and a suspension of it was made in sterile distilled water.
2. API20E biochemical test strip was prepared which contains dehydrated bacterial media/bio-chemical reagents in 20 separate compartments.
3. Using a pasteur pipette, the compartments were filled up to the brim with the bacterial suspension.
4. A sterile oil was added into the ADH (arginine-dihydrolase), LDC (lysine decarboxylase), ODC (ornithine decarboxylase), H2S (sulfate Production) and URE (urease) compartments.
5. Some drops of water were added in the tray and the API Test strip was added and the tray was closed.
6. The tray was marked with identification number, date and our initial identifications.
7. The tray was incubated at 37^{o}C for 18 to 24 hours.

Interpretation

1. For some of the compartments, the colour change can be read straightway after 24 hours but for some reagents must be added to them before interpretation.
2. The following reagents were added to these specific compartments:
    - TDA (tryptophane deaminase): one drop of Ferric Chloride.
    - IND (Indol ring): one drop of Kovacs reagent.
    - VP (Voges–Proskauer): one drop of 40 % KOH (VP reagent 1) and One drop of VP Reagent 2 (α-Naphthol).
3. The API reading scale (color chart) was recorded by marking each test as positive or negative on the lid of the tray. The wells were marked off into triplets by black triangles, for which scores were allocated.
4. The scores for the positive wells only were added in each triplet.
5. Three test reactions were added together at a time to give a 7-digit number, which can then be looked up in the codebook. The highest score possible for a triplet is 7 (the sum of 1, 2 and 4) and the lowest is 0.
6. The organisms were identified by using API catalog.

Table (I): *API E20 results interpretation*

Test	Code	Negative	Positive
β-galactosidase	ONPG	Colorless	Yellow
Arginie Dihydrolysis	ADH	Yellow	Red-orange
Lysine Decarboxylase	LDC	Yellow	Red-orange
Ornithine Decarboxylase	ODC	Yellow	Red-orange
Citrate Utilization	CIT	Yellow	Green-blue
Hydrogen Sulfide	H2S	Colorless	Black sediment
Urease production	URE	Yellow	Red-orange
Tryptophan Deaminase	TDA	Yellow	Dark brown
Indole production	IND	Yellow ring	Red ring
Acetone production	VP	Colorless	Pink-red
Gel Hydrolysis	GEL	No pigment	Black pigments
Glucose	GLU	Blue	Yellow
Manitol	MAN	Blue	Yellow
Insonitol	INO	Blue	Yellow
Sorbitol	SOR	Blue	Yellow
Rhaminose	RHA	Blue	Yellow
Sucrose	SAC	Blue	Yellow
Melibiose	MEL	Blue	Yellow
Amayloid	AMY	Blue	Yellow
Arabinose	ARA	Blue	Yellow

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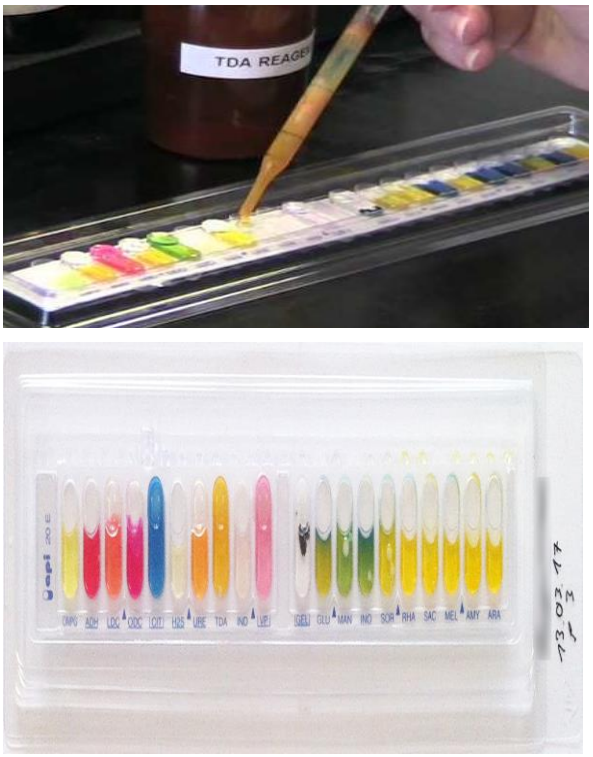

Fig. (VII): *API E20 tests for Enterobacteriaceae.*

Gram positive cocci:

A. Hemolysis on blood agar:

Principal:

Hemolysis is determined by streaking for isolation on a blood agar plate. In clinical settings, this might also include several stabs of the inoculum into the agar to encourage any anaerobic versions of the enzymes to digest blood cells. After incubation overnight, the medium is inspected for telltale signs of alpha- or beta-hemolysis . If the medium is discolored or darkened after growth, the organism has demonstrated alpha-hemolysis. If the medium has been cleared under growth, the organism is beta-hemolytic . No discernible change in the color of the medium constitutes gamma-hemolysis blood agar is a differential medium for detecting haemolysis (destruction of red blood cells) by cytolytic toxins secreted by certain bacteria, such as certain strains of Bacillus, Streptococcus, Enterococcus, Staphylococcus and Aerococcus.

Procedure:

- Using an inoculating loop, the medium was inoculated with bacterium specimen.
- Place the inoculated tube into the 35-37 C incubator for 24 hours.
- Retrieve the incubated culture from the incubator.
- Observe the medium surrounding colonies in the plate.

The culture showing a darkening or discoloration of the medium in the vicinity of growth demonstrates alpha-hemolysis. Cultures showing clear halos around colonies and under growth is exhibiting beta-hemolysis**.**

Fig. (VIII): *Hemolysis test of gram positive isolates.*

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B. Catalase Test:

Principal:

Catalase is the enzyme that breaks down hydrogen peroxide (H_2O_2) into H_2O and O_2. The oxygen is given off as bubbles in the liquid. The catalase test is primarily used to differentiate between gram-positive cocci. Members of the genus *Staphylococcus* are catalase-positive, and members of the genera *Streptococcus* and *Enterococcus* are catalase-negative.

Materials:

Catalase reagent (Hydrogen peroxide H202 3%).

Procedure:

- From an overnight growth on the culture, using a disposable loop a colony was carefully removed and placed in a tube.
- About 2-3 ml of 3% hydrogen peroxide was poured into a clean test tube.
- Using a sterile wooden stick or glass rod, several colonies of the test organism was immersed in the H2O2 solution.
- Immediate active gas bubbling was considered a positive result.

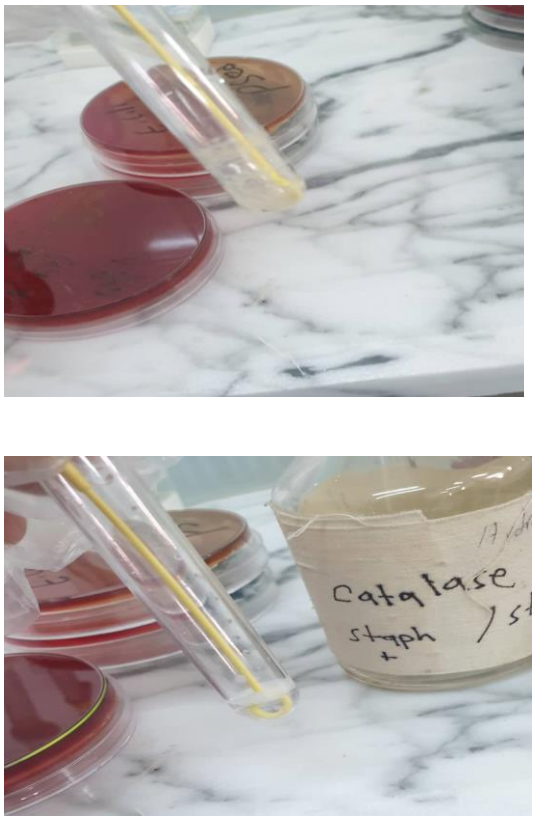

Fig. (IX): *Catalase test.*

*Interpretation***:**

Positive results: Any bubbling from a transferred colony indicates a positive test (Staphylococcus spp).

Negative results: The absence of bubbling from a transferred colony indicates a negative test (Streptococcus spp.).

C. Coagulase Test:

Principal:

Coagulase is an enzymatic protein that is a thermostable thrombin-like substance, which converts fibrinogen into fibrin resulting in clotting or clumping. In *S. aureus*, two different forms of coagulase are found; free coagulase and bound coagulase. The coagulase test is used to detect free coagulase or/and bound coagulase.

Materials:

- Deionized water
- Coagulase reagent (rabbit or human plasma).

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Procedure:

1. About 10 µl of deionized water or physiological saline was added to a slide.
2. Several colonies from a fresh culture were collected with an inoculating loop and were emulsified into the water to obtain a smooth milk-colored suspension**.**
3. A drop of a rabbit or human plasma was added to the slide, and the clumping was observed immediately during 10 seconds.

Interpretation:

Positive results: the demonstration of the agglutination of the bacterial cells after the plasma is added indicating the presence of *Staphylococcus auerus*.

Negative results: The lack of agglutination indicating the presence of other gram positive isolates**.**

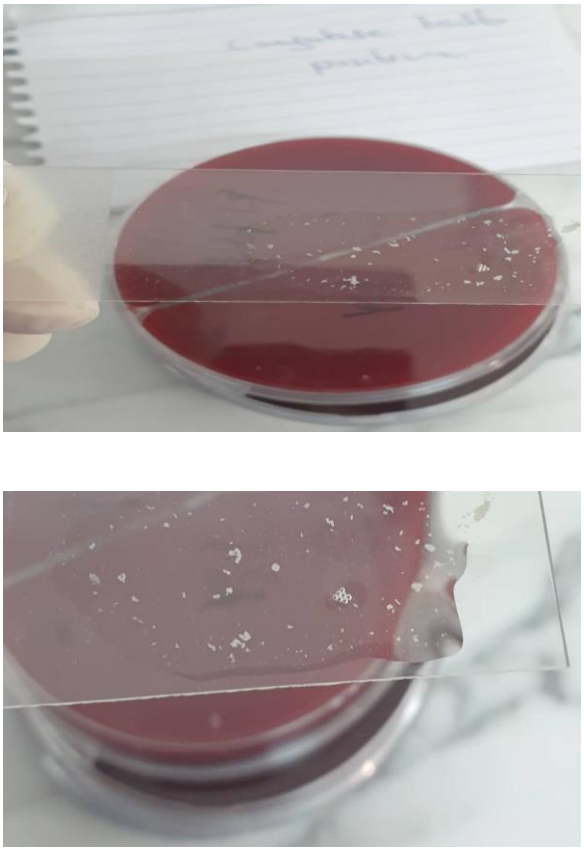

Fig. (X): ***Coagulase positive produced by S. aureus.***

D. Deoxyribonuclease (DNase) Test :

Principal:

The test is used to determine the ability of an organism to hydrolyze DNA. DNase agar is a differential medium that tests the ability of an organism to produce an exo-enzyme, called deoxyribonuclease. DNase are extracellular endonucleases that cleave DNA and release free nucleotides and phosphate. DNase agar contains nutrients for the bacteria, DNA, and mostly methyl green as an indicator. Methyl green is a cation which binds to the negatively-charged DNA.

Materials:

- Freshly prepared DNase agar plates.
- HCL solution 1N

Procedure:

1. Using a sterile loop, the DNase agar is inoculated with the test organism by streaking a pure colonies on the surface of the plate.
2. The plate was incubated at 35-37°C for 24 hours.
3. After incubation the color change in DNase with methyl green was noticed.
4. The surface of agar was flooded with 1N HCL solution and the excess acid was tipped.
5. The reagent was allowed to absorb into the plate.
6. The clear zones around the colonies were observed within 5 minutes.

Interpretation:

Positive results: discoloration of the medium around the test organism.

Negative results**: If no degradation of DNA occurs, the medium remain green**

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Fig. (XI): *DNase test.*

E. Mannitol Salt Agar:

Principal:

Mannitol Salt Agar is a selective medium used for isolating pathogenic staphylococci from clinical samples, food and other materials of sanitary importance.

Pancreatic digest of casein, peptic digest of animal tissue and beef extract provide amino acids, nitrogen, carbon, vitamins and minerals for organisms growth. Mannitol is the fermentable carbohydrate. The high salt content of 7.5% inhibits most bacteria other than staphylococci. Phenol red is the pH indicator.

Materials:

- Mannitol powder.
- Deionized water

Procedure:

1. About 111 g of the mannitol powder was suspended in 1 liter of distilled or deionized water and mixed well.
2. The suspension was heated to boil for 1 minute with shaking frequently until completely dissolved.
3. The mixture was sterilized in autoclave at 121°C for 15 minutes.
4. The content of the bottle a was melt in water bath at 100°C (loosing the cap partially removed) until completely dissolved.
5. The content was cooled at 45-50°C and mixed well to avoid foam formation and aseptically distributed into Petri dishes**.**
6. Plates were inoculated by the direct streaking of pure culture to be examined over the agar surface.
7. Incubated aerobically at 35 ± 2°C for 24-48 hours.

Interpretation:

Positive results: *S. aureus* cultivates with yellow or white colonies surrounded by a yellow zone.

Negative results: Staphylococci form small colorless to red colonies with no color change to the medium**.**

Fig. (XII): *Mannitol agar test*

F. Novobiocin test:

Principle

Novobiocin is an antibiotic interfering with the unpackaging and repackaging of DNA during DNA replication and the bacterial cell cycle. Novobiocin binds to DNA gyrase, and blocks adenosine triphosphatase (ATPase) activity. Susceptibility to novobiocin is determined by placing a novobiocin-impregnated paper disk on a agar plate seeded with the organism under investigation. As the organism multiplies during incubation to produce a lawn of confluent growth, cells are exposed to the antibiotic diffusing into the agar from the paper disk. If the bacteria are susceptible to novobiocin, there will be a formation of visible zone of inhibition around the disk, representing an area where the antibiotic concentration has prevented bacterial growth. No zone of inhibition around the disk represents that organism is resistant to the anitibiotic.

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Materials:

- Tryptic soy broth
- Blood agar plate
- Novobiocin disk

Procedure:

1. From pure culture incubated for 18-72 hours, a suspension of the test isolate is prepared by adding pure culture in tryptic soy broth equal to a McFarland 0.5 standard or equivalent.
2. A sterile swab is Immersed into the suspension and rotated against the side of the tube above the fluid level to remove excess inoculum.
3. Using the expressed swab, blood agar plate was inoculated by streaking the swab over the entire agar surface and repeated in 2 planes.
4. The agar surface allowed to dry not more than 15 minutes before applying a Novobiocin Disk.
5. With a sterile swab, a lawn of growth was prepared over the entire plate by swabbing over the entire plate in 3 directions and around the edge of the plate.
6. Using alcohol-dipped and flamed forceps, the novobiocin antibiotic disc aseptically applied to the surface of each inoculated plate.
7. With the sterile forceps the disks were gently pressed down to ensure that they adhere to the agar surface.
8. The plates were incubated aerobically for 18 to 24 hours at 35 to 37°C.
9. The diameter of the zone of inhibition was measured using sliding calipers or a metric ruler.

Interpretation:

Positive results: A zone of inhibition greater than 16mm indicates that the organism is sensitive to the antibiotic.

Negative results: A zone of inhibition less than or equal to 16mm is indicative of novobiocin resistance

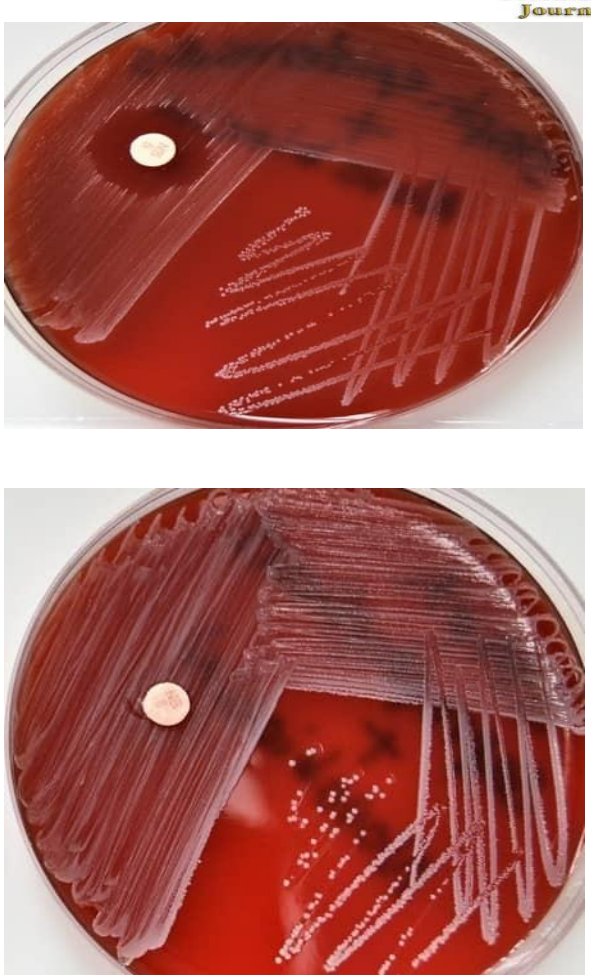

Fig. (XIII): *Novopiocin susceptibility test.*

G. CAMP (Christie, Atkinson, Munch, and Peterson) test :

Principal:

Certain organisms (including group B streptococci) produce a diffusible extracellular hemolytic heat-stable protein (CAMP factor) that acts synergistically with the beta-lysin of *S. aureus* to cause enhanced lysis of red blood cells. The group B streptococci are streaked perpendicular to a streak of *S. aureus* on sheep blood agar. A positive reaction appears as an arrowhead zone of hemolysis adjacent to the place where the two streak lines come into proximity The hemolytic activity of the beta-hemolysin produced by most strains of *S. aureus* is enhanced by extracellular protein produced by group B streptococci.

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Procedure:

1. A beta-lysin–producing strain of *S. aureus* was streaked down the center of a sheep blood agar plate.
2. The streptococcal species was streaked 3 to 4 cm long the S. *aureus.*
3. The test organisms was streaked across the plate perpendicular to the *aureus* streak within 2 mm.
4. The plate was incubated at 35°-37°C in ambient air for 18-24 hours.

*Interpretation***:**

Positive: Group B streptococci like S. *agalactiae* and a few other beta-streptococci produce an enhancement of the ß-lysin activity of the *aureus* strain.

Negative: No enhancement of hemolysis.

Fig. (XIV): *CAMP test.*

H. Enzyme latex test:

Principal:

The BBL™ Streptocard™ Enzyme Latex Test is a latex test system for the qualitative identification of Lancefield streptococcal groups A, B, C, D, F and G. Majority of pathogenic streptococci possess specific carbohydrate antigens, which permit the classification of streptococci into groups. These streptococcal group antigens are extracted from the streptococcal cell wall in a liquid form, and reacted with group specific antibodies. In this test latex particles are sensitized with group specific antibody and will agglutinate in the presence of homologous antigen. In the absence of such antigen, the latex particles will remain in a smooth suspension. The use of a patented enzymatic extraction in the BBL™ Streptocard ™ Enzyme Latex Test procedure considerably shortens the time required for antigen extraction and improves the antigen yield.

Materials

- Test Latex (A, B, C,D,F, G)
- Extraction Enzyme (lyophylized)
- Control
- Reaction Cards
- Mixing Sticks

Procedure:

1. Samples for identification allowed to grow on a blood agar plate 16-24 h at 35 ± 2°C.
2. About 0.4 mL of BBL™ Extraction Enzyme was dispensed into clean, labeled test tube for each specimen to be tested.
3. 2-5 similar clear colonies were selected with a microbiological loop and emulsify in the BBL™ Extraction Enzyme until a slightly turbid suspension was obtained.
4. The tubers were incubated for a total of 10 minutes at 37° ± 2°C in a water bath or heat block . After 5 min incubation the tubes were removed and mixed by shaking for 2-3 sec, then continued the incubation.
5. The tubes were removed and allow to cool to room temperature.
6. 1 drop from was dispensed for each test latex to be tested onto a separate circle on the reaction card.
7. Using a Pasteur pipette 1 drop of extract was added to each of the six test circles.
8. With the mixing sticks provided, the mixture was spread over the entire area of the circle, using a separate stick for each.
9. The card was rocked manually for up to 1 min and observed for agglutination under normal lighting conditions.

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Interpretation:

Positive Result: An obvious agglutination of the blue latex particles occurs within 1 min with a single Test Latex.

Negative Result: No agglutination occurs within 1 min.

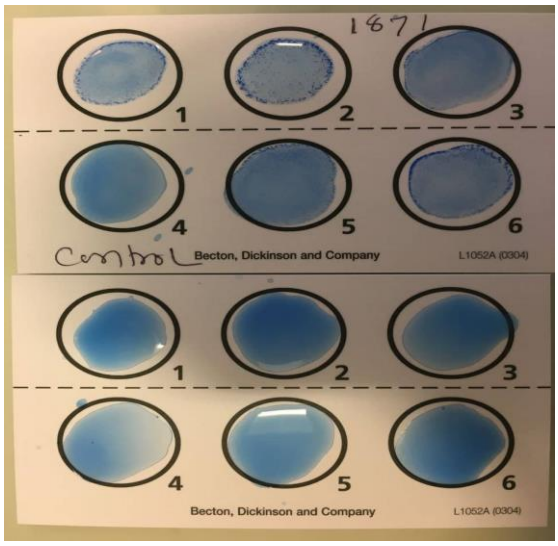

Fig. (XV): *BB Streptocard enzyme latex test.*

Antimicrobial Susceptibility Testing

The antibiotic susceptibility testing for each of the isolates was done to determine the bacterial susceptibility to some antibiotics in vitro, based on the disk diffusion method using Mueller-Hinton agar and blood agar according to the guidelines by the Clinical and Laboratory Standards Institute (CLSI).

Materials:

- Mueller-Hinton agar
- Blood agar plate
- Susceptibility disks (Oxoid, England).

Procedure:

1. The suspension of the isolate (test organism) from the purity plate on Blood agar or MacConkey was prepared in sterile peptone water to match with 0.5 McFarland turbidity standard.
2. The resulting suspension was inoculated evenly over the surface of pre-incubated Mueller-Hinton agar and Blood agar plate and anti-microbial disks (based on Gram-stain reaction) which have equilibrated with the room temperature
3. Susceptibility disks were placed gently with equal distance to another, six (6) disks per 90mm Petri dish antibiotics used were described in the table (3-6).
4. The plates were incubated aerobically at 35C° overnight and were read the next day.
5. The drugs / antibiotic disks (Oxoid England) zone diameters were measured to the nearest millimeter and isolates were classified as susceptible, intermediate or resistant according to CLSI – specified interpretative criteria.

Table (II): *Oxoid antimicrobial susceptibility discs.*

Antimicrobial agent	**Disk conc.**
Amikacin	30 µg
Amoxicillin/clavulanic acid	30µg
Cefoxitin	30µg
Ceftazidime	30µg
Ciprofloxacin	5µg
Gentamicin	10 µg
Imipenem	10µg
Kanamycin	30 µg
Naldixic acid	30 µg
Nitrofurantoin	200 µg
Tetracycline	30µg
Trimethoprim/sulfamethoxazole	75µg

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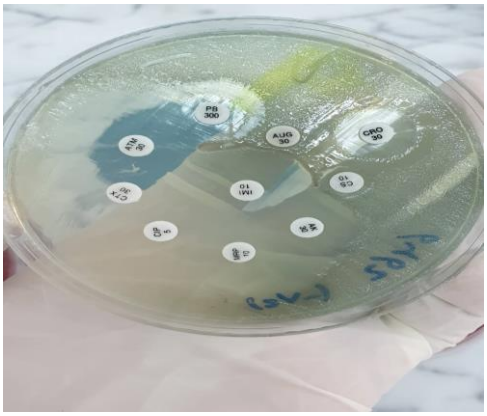

Fig. (XVI): *Antibiotic susceptibility testing.*

Statistical analysis:

Descriptive statistics were used to determine the prevalence of urinary tract infection, The differences in prevalence of urinary tract infection among different age groups were evaluated by Chi-square test, and among different gender were evaluated by Mann-Whitney test, with significance level of 0.05 using SPSS software version 26.

[1] RESULTS AND DISCUSSION

A. Bacterial identification of gram negative species:

A.1. Colony morphology:

Escherichia coli :

On CLED agar: Appeared as large 1.5-2.0mm, yellowish, moist, low convex, entire edge lactose fermenting colonies.

On MacConkey agar: Appeared as flat, dry, pink, non-mucoid colonies with a surrounding darker pink area of precipitated bile salts due to lowered pH of the agar below 6.8, figure (XVII) showing growth and colony morphology on CLED and MacConkey agar.

Klebsiella pneumoniae:

On CLED: Large 2.0-2.5mm, yellowish, moist, luxuriant mucoid, high convex.

On MacConkey: Appeared large, mucoid, and pink, with pink-red pigment usually diffusing into the surrounding agar due to lowered pH of the agar below 6.8, figure (XVIII) showing Growth and colony morphology of *Klebsiella pneumoniae* on CLED and MacConkey agar.

Pseudomonas aeruginosa:

On CLED agar: Fruity-smelling, large 1.0-1.5mm, greyish-coloured moist, low convex, entire edge non-lactose fermenting colonies on CLED agar.

On MacConkey agar: Forms flat translucent and smooth colonies with regular margins that are between 2 -3 mm in diameter, figure (XIX) showing the growth and colony morphology of *P. aeruginosa* on CLED and MacConkey agar.

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Fig. (XVII): *Growth and colony morphology of E. coli on CLED and MacConkey agar.*

Fig. (XVIII): *Growth and colony morphology of K. pneumoniae on CLED and MacConkey agar*

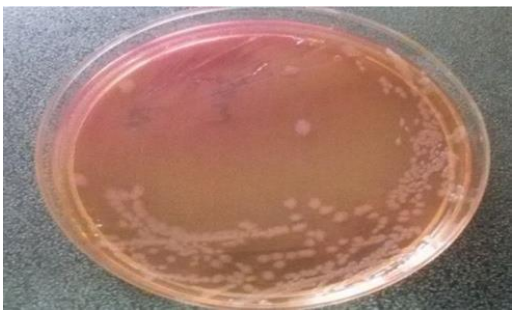

Fig. (XIX): *Growth and colony morphology of P. aeruginosa on CLED and MacConkey agar*

Gram Stain and motility:

Escherichia coli :

Gram negative motile non spore forming rods shaped, figure (XX) showing the microscopic features of *E. coli*.

Klebsiella pneumoniae:

Gram negative non motile encapsulated rod figure (XXI) showing the microscopic features of *K. pneumoniae*.

***Pseudomonas aeruginosa*.**

Gram-negative, encapsulated, motile rod-shaped, figure (XXII) showing microscopic features of *P. aeruginosa*.

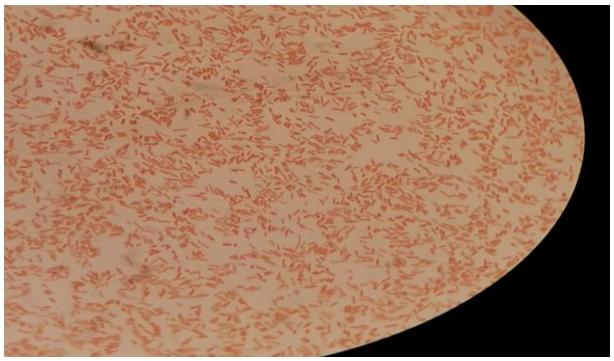

Fig. (XX): *Microscopic features of E. coli*

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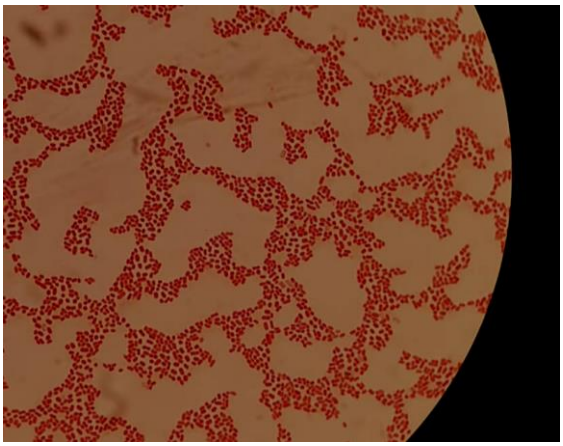

Fig. (XXI): *Microscopic features of K. pneumoniae.*

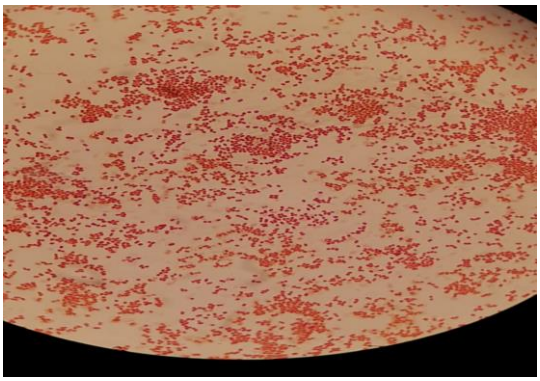

Fig. (XXII): *Microscopic features of P. aeruginosa.*

Biochemical reactions:

Biochemical reactions of Gram negative isolates are listed in the table (4-1). Oxidase test was used to differentiate *P. aeruginosa*. from other gram negative isolates, *P. aeruginosa*. was positive to Oxidase, other isolates were members of enterobacteriace and differentiated by API panel and the characteristic tests were listed in table (IV).

Table (IV): *Biochemical reactions of Gram negative isolates.*

Biochemical	***E. coli***	***K. pneumonia***	***P. aeruginosa***
Fermentation	+	+	-
Oxidase	-	-	+
Urease	-	+	-
Citrate utilization	-	+	+
Indole production	+	-	-
H2S	-	-	-

Bacterial identification of gram positive species:

Gram Stain and motility: Staphylococcus aureus:

Showed deep golden yellow, spherical, smooth, raised, and glistening colonies in clusters in two planes, showed Beta-hemolytic when cultivated for 24 hours in aerobic atmosphere, 37°C on sheep blood agar (figure XXIII).

***Staphylococcus saprophyticus*:**

Pin-point 0.2-0.5mm, creamy colonies non hemolytic on Blood agar, figure (XXIV) showing the growth and colony morphology of *S. saprophyticus*.

***Streptococcus agalactiae*:**

Group B S. agalactiae isolates showed a small zone of beta hemolysis on Blood agar, figure (XXV) showing the growth and colony morphology of *S. agalactiae*.

***Streptococcus pyogenes*:**

S. pyogenes colonies are dome-shaped with a smooth or moist surface and clear margins. They display a white-greyish color and have a diameter of > 0.5 mm, and are surrounded by a zone of β-hemolysis that is often two to four times as large as the colony diameter as shown in figure (XXVI).

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Fig. (XXIII): Growth and colony morphology of S. aureus.

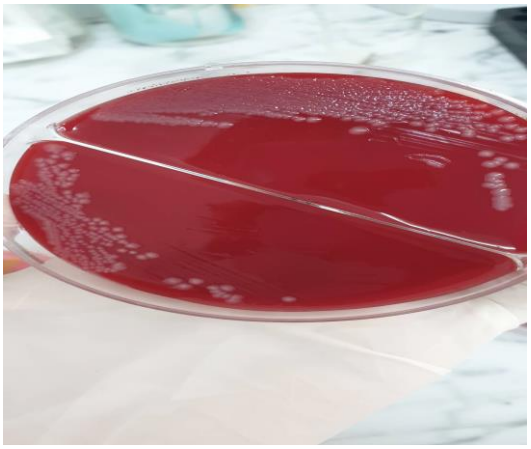


Fig. (XXIV): *Growth and colony morphology of S. saprophyticus.*

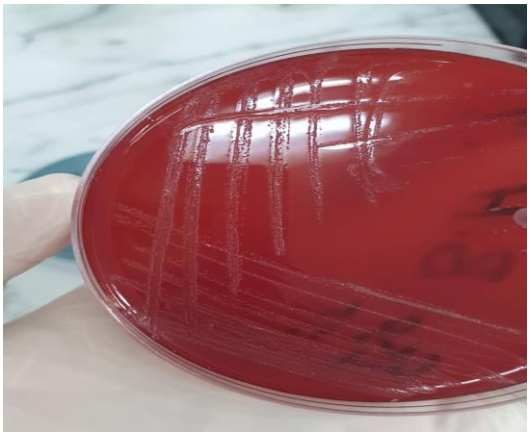
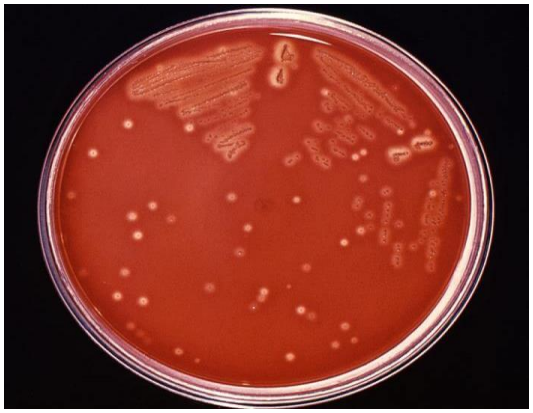


Fig. (XXV): *Growth and colony morphology of S. agalactiae*



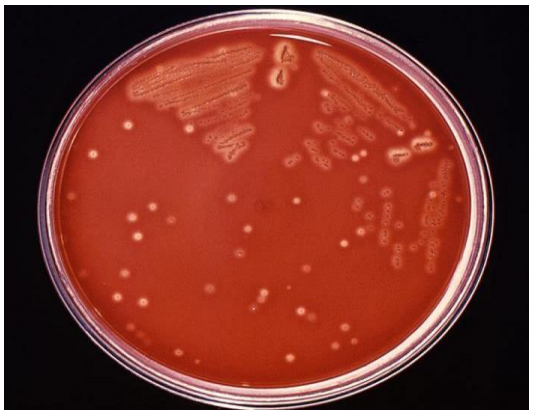


Fig. (XXVI): *Growth and colony morphology of S. pyogenes.*

Gram stain and motility: Staphylococcus aureus:

S. aureus showed round, convex, and 1-4 mm in diameter with a sharp border and showed deep golden yellow, spherical, smooth, raised, and glistening colonies in clusters in two planes and Beta-hemolytic when cultivated for 24 hours in aerobic atmosphere (XXVII) showing the microscopic features of *S. aureus***.**

***Staphylococcus saprophyticus*:**

Gram-positive, capsulated singly, in pairs, or in a short chain of 3-4 bacteria, Irregular grape like clusters of cells. Non-Flagellated, Non-Motile and Non-Sporing, figure (XVIII) showing the microscopic features of *S. saprophyticus*

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***Streptococcus agalactiae*:**

Gram-positive cocci 0.6-1.2 µm in diameter that form short chains, non motile, non sporing capsulated bacteria, figure (XXIX) showing the microscopic features of *S. agalactiae*.

***Streptococcus pyogenes*:**

S. pyogenes appears as Gram-positive cocci, arranged in short chains, diplococci and single cocci, as shown in figure (XXX)

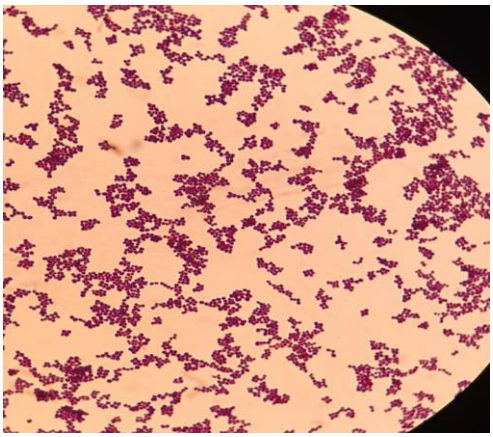

Fig. (XXVII): *Microscopic features of S. aureus.*

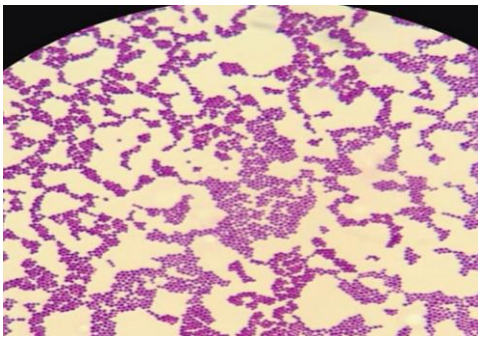

Fig.(XVIII): *Microscopic features of S. saprophyticus.*

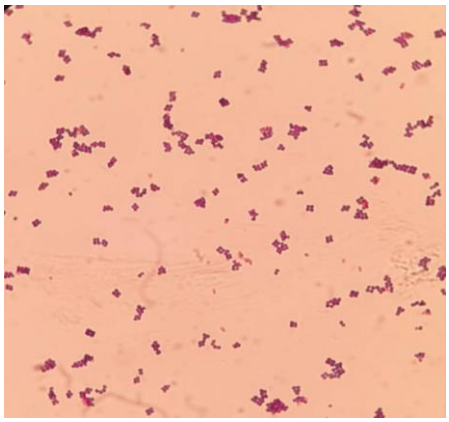

Fig.(XXIX): *Microscopic features of S. agalactiae*

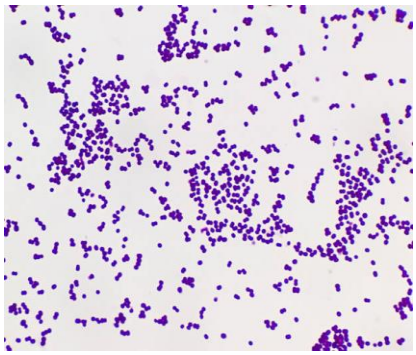

Fig. (XIX): Microscopic features of S. pyogene *Biochemical reactions:*

Staphylococcus aureus*:*

- Catalase and coagulase positive, produced golden yellow pigment on mannitol agar.

Staphylococcus saprophyticus:

- Catalase positive, coagulase negative, resistant to novobiocin

.***Streptococcus agalactiae:***

- Showed catalase and coagulase negative, CAMP positive.

Streptococcus pyogenes:

Catalase negaive, sensitive to bacitricin.

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A. Evaluation of Demographic data:

***Study populations*:**

The study comprised of 180 subjects, 100 (55.6%) were HIV positive subjects, 80 (44.4) were HIV negative healthy subjects (control), the distribution of the study subjects is shown in table (V) and figure (XXX).

Table (V): *The distribution of study subjects.*

Study subjects	Frequency	Percent
HIV positive	100	55.6
Control	80	44.4
Total	180	100

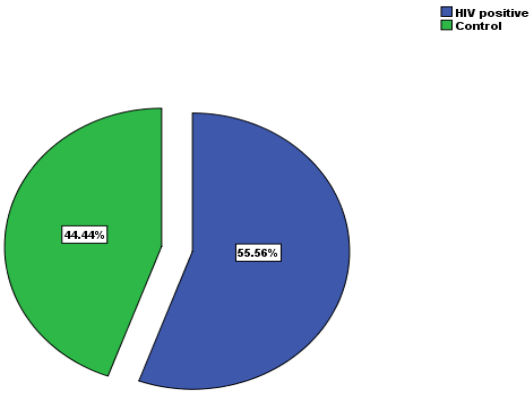

Fig. (XXX): *The distribution of study subjects.*

Age:

Among the total population the mean age was (27.34 ±7.29), ranging between 20-47 years old, among the HIV positive subjects mean age was (25.8± 6.66), ranging between 20-47 years old, among the control subjects, the mean age was (29.3± 7.6 2) ranging between 20-45 years old, figure (XXXI) is showing the age means of the study subjects.

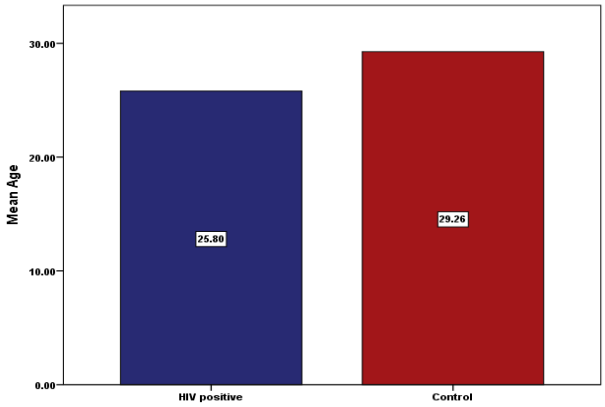

Fig. (XXXI): *Mean age among the study subjects.*

***Age groups*:**

The age group ≤ 25 was the predominant age group in both study groups, (66% and 46.3%) respectively, followed by the age group from 26-30 years old (17%) in HIV positive subjects, but in control group it was followed by age group between 31-40 years old (30%) the distribution of the study subjects according to their age groups is shown in table (VI) and figure (XXXII).

Table (VI): *The distribution of study subjects according to their age groups*.

Age group	HIV positive		Control	
	Frequency	Percent	Frequency	Percent
≤ 25	66	66	37	46.3
26-30	17	17	9	11.3
31-40	9	9	24	30
≥ 41	8	8	10	12.5
Total	100	100	80	100

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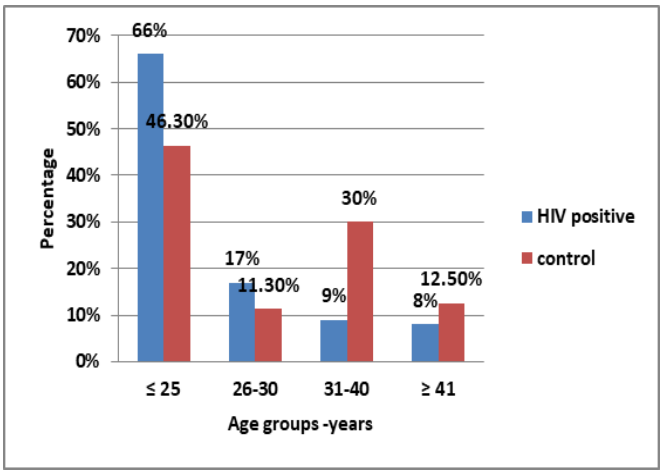

Fig (XXXII): *The distribution of study subjects according to their age groups.*

Gender:

HIV positive group was comprised of 55% females, and 45% males, the control group was approximately matched, it was comprised of 52.5% females and 47.5% males, the distribution of the study subjects according to their gender is shown in table (VII) and figure (XXXIII).

Table (VI): *The distribution of study subjects according to their gender.*

Gender	HIV positive		Control	
	Frequency	Percent	Frequency	Percent
Female	55	55	42	52.5
Male	45	45	38	47.5
Total	100	100	80	100

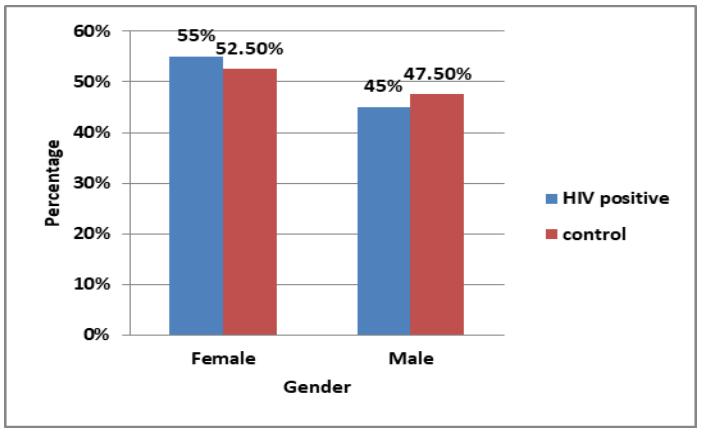

Fig. (XXXIII): *The distribution of HIV positive cases according to their gender.*

***Prevalence of UTI*:**

The prevalence of UTI was evaluated by the number of organisms which showed significant growth on the culture. The prevalence of UTI among HIV positive subjects was 28%, while in the control group it was 35%, but no significant differences in the prevalence among both groups according to Mann-Whitney test (P-value)> 0.05 , as shown in table (VIII) and figure (XXXIV).

Table (VIII): *Prevalence of UTI.*

Culture Growth	Prevalence in HIV positive		Prevalence in Control		Mann-Whitney test (P-value)
	Frequency	Percentage	Frequency	Percentage	
No growth	72	72	52	65	0.315 (NS)
Growth	28	28	28	35	
Total	72	72	80	100	

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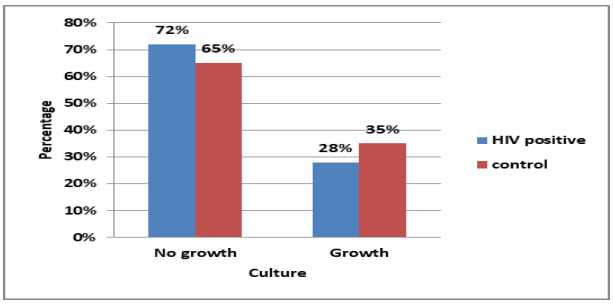

Fig. (XXXIV): *Prevalence of UTI.*

Prevalence according to age groups:

No significant differences in the prevalence of UTI according to the age groups among both HIV positive subjects and control subjects according to Chi-square test (p-value)> 0.05, a shown in table (IX).

Table (IX): *Prevalence of UTI according to age groups.*

Age	**No growth**	**Growth**	**Total**	**Chi-square (P- value)**
Prevalence among HIV positive				
≤ 25	47 (47%)	19 (19%)	66 (100%)	0.95 (NS)
26-30	13 (13%)	4 (4%)	17 (100%)	
31-40	6 (6%)	4 (3%)	9 (100%)	
≥ 41	6 (6%)	2 (2%)	8 (100%)	
Total	72 (72%)	28 (28%)	100 (100%)	
Prevalence among control				
≤ 25	29 (78.4%)	8 (21.6%)	37 (100%)	0.125 (NS)
26-30	5 (55.6%)	4 (44.4%)	9 (100%)	
31-40	12 (50%)	12 (50%)	24 (100%)	
≥ 41	6 (60%)	4 (40%)	10 (100%)	
Total	52 (65%)	28 (35%)	80(100%)	

Prevalence according to gender:

The prevalence of UTI was higher in females than males in general, with significant differences among HIV positive subjects (p-value < 0.05), but with no significant differences among the control group (p-value > 0.05), as shown in table (X).

Table (X): *The prevalence of UTI among HIV positive patients according to their gender.*

Gender	No growth	Growth	Total	Chi-square (P- value)
Prevalence among HIV positive				
Female	35 (35%)	20 (20%)	55 (100%)	0.023(S)
Male	37 (37%)	8 (8%)	45 (100%)	
Total	72 (72%)	28 (28%)	100 (100%)	
Prevalence among control				
Female	24 (24.7%)	18 (18.6%)	42 (100%)	0.094 (NS)
Male	28 (33.7)	10 (12%)	38 (100%)	
Total	52 (28.9%)	28 (15%)	80 (100%)	

Gram reaction results:

Totally 56 bacterial species were isolated from the subjects of the study, 67.9% were gram negative, 32.1% were gram positive. Among HIV positive subjects 21 (75%) were gram negative and 7 (25%) were gram positive, among the control group 17 (60.7%) were gram negative and 11 (39.3%) were gram negative, the distribution of the isolated species according to the gram reaction is shown in table (4-8) and figure (XI).

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Table (XI): *The distribution of bacterial isolates according to Gram reaction.*

Gram reaction	No.	Percent
All subjects		
Gram (-)	38	67.9
Gram (+)	18	32.1
Total	56	100
HIV positive		
Gram (-)	21	75
Gram (+)	7	25
Total	28	100
Control		
Gram (-)	17	60.7
Gram (+)	11	39.3
Total	28	100

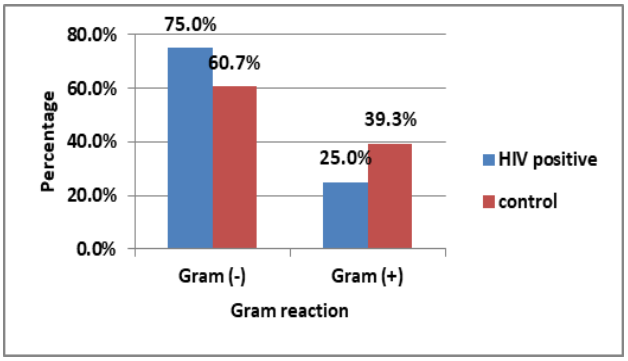

Fig. (XXXV): *The distribution of bacterial isolates according to Gram reaction.*

Bacterial Etiologies:

Among the gram negative species, E. coli was the most predominant bacterial etiology among the study population (33.9%), followed by K. pneumonia it was (25%) equally in both groups, P. aeruginosa was (8.9%), which was more seen in HIV positive subjects than control. while among gram positive species, S. aureus was the most common pathogen (17.9%) equally found in both groups, S. agalactiae was (5.4%), more frequently in HIV subjects than control. S. saprophyticus was (3.6%), S. pyogenes was (4.5%) seen in the control group only.According to the chi-square test no significant differences in the distribution of the bacterial species among the study populations, as represented in table (XII) and figure (XXXVI).

Table (XII): *The results of bacteria identifications.*

Uropathogens	**HIV positive**		**Control**		***Total***	
	No.	**Percent**	**No.**	**Percent**	**No.**	**Percent**
E. coli	10	17.9%	9	16.1%	19	33.9%
K. pneumonia.	7	12.5%	7	12.5%	14	25%
P. aeruginosa	4	7.1%	1	1.8%	5	8.9%
S. aureus	5	8.9%	5	8.9%	10	17.9%
S. agalactiae	2	3.6%	1	1.8%	3	5.4%
S. saprophyticus	-	-	2	3.6%	2	3.6%
S. pyogenes	-	-	3	5.4%	3	5.4%
Total	28	50%	28	50%	*56*	*100%*
Chi-square (P-value)			0.055 (NS)			

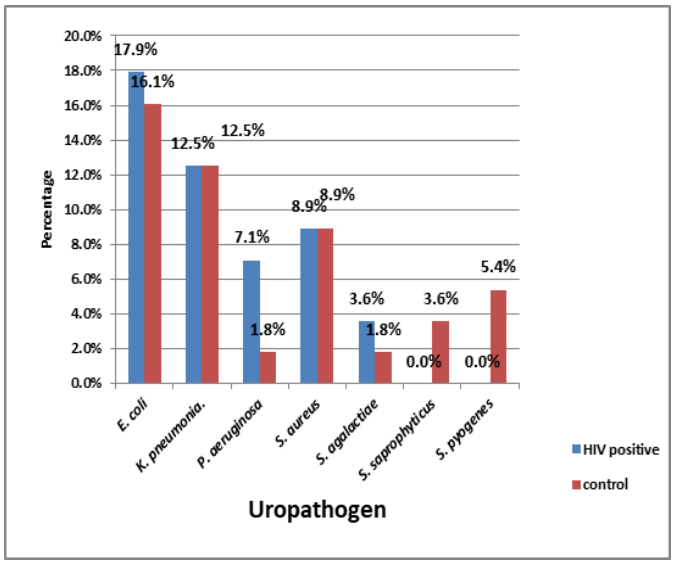

Fig. (XXXVI): *The results of bacteria identifications.*

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Antimicrobial susceptibility testing:

Gram negative bacteria:

The isolated uropathogens were tested for their sensitivity to 11 antimicrobial agents, the results of antimicrobial susceptibility testing revealed that for 21 isolated gram negative bacteria most isolates were resistant to the tested antibiotics and their resistance profile were as following: Tetracycline and Sulfamethoxazole (95.2%), Kanamycin (90.5%), Amoxicillin-clavulanic acid (Augmentin) (85.71%), Meropenem, Ceftazidime and Cefoxitin (76.19%), Amikacin and Ciprofloxacin (71.43%), Imipenem (57.1%) and Nitrofurantoin (38.1%). The gram negative isolates were somewhat susceptible to Nitrofurantoin by (61.9%). The detailed antimicrobial susceptibility testing is described in the table (XIII) and figure (XXXVII).

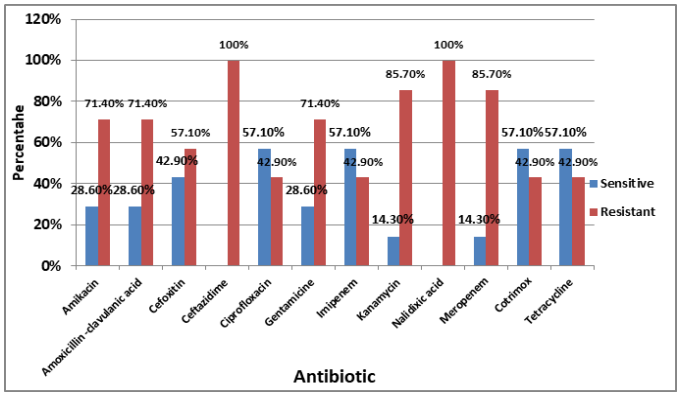

Fig. (XXXVIII): Antibiotic susceptibility testing for Gram positive isolates

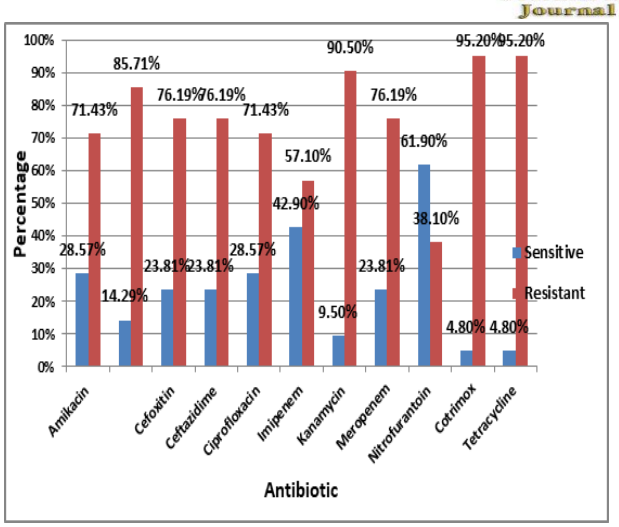

Fig. (XXXVII): *Antibiotic susceptibility testing for Gram negative isolates*

Ceftazidime and Naldixic acid (100%), Kanamycin and Meropenem (85.7%), Amikacin, Amoxicillin -clavulanic acid (Augmentin) and Gentamicine (71.4%). Cefoxitin (57.1%), Ciprofloxacin and Imipenem, Kanamycin, Sulfamethoxazole and Tetracycline (42.9%), About 67.86 % of the gram positive isolates showed multidrug resistance profile. The detailed antimicrobial susceptibility testing for gram positive bacteria is described in the table (XIV) and figure (XXXVII).

Gram positive Bacteria:

The results of antimicrobial susceptibility testing of 12 antimicrobial agents revealed that for 7 isolated gram positive bacteria most isolates were resistant to all tested antimicrobial agents and their resistance profile were as following:

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Table (XIII): Antibiotic susceptibility testing for gram negative isolates

Antibiotic	E. coli (10 isolates)		Klebseilla spp. (7 isolates)		Pseudomonas spp. (4 isolates)		Total (21 isolates)	
	S	R	S	R	S	R	S	R
Amikacin	3 (30%)	7 (70%)	3 (42.86%)	4 (57.14%)	-	4 (100%)	6 (28.57%)	15 (71.43%)
Amoxicillin - clavulanic acid	-	10 (100%)	3 (42.86%)	4 (57.14%)	-	4 (100%)	3 (14.29%)	18 (85.71%)
Cefoxitin	3 (30%)	7 (70%)	2 (28.57%)	5 (71.43%)	-	4 (100%)	5 (23.81%)	16 (76.19%)
Ceftazidime	3 (30%)	7 (70%)	-	7 (100%)	2 (50%)	2 (50%)	5 (23.81%)	16 (76.19%)
Ciprofloxacin	2 (20%)	8 (80%)	2 (28.57%)	5 (71.43%)	2 (50%)	2 (50%)	6 (28.57%)	15 (71.43%)
Imipenem	7 (70%)	3 (30%)	1 (14.3%)	6 (85.7%)	1 (25%)	3 (75%)	9 (42.9%)	12 (57.1%)
Kanamycin	2 (20%)	8 (80%)	-	7 (100%)	-	4 (100%)	2 (9.5%)	19 (90.5%)
Meropenem	-	10 (100%)	4 (57.14%)	3 (42.86%)	1 (25%)	3 (75%)	5 (23.81%)	16 (76.19%)
Nitrofurantoin	7 (70%)	3 (30%)	2 (28.57%)	5 (71.43%)	2 (50%)	2 (50%)	13 (61.9%)	8 (38.1%)
Co-trimox	-	10 (100%)	-	7 (100%)	1 (25%)	3 (75%)	1 (4.8%)	20 (95.2%)
Tetracycline	-	10 (100%)	-	7 (100%)	1 (25%)	3 (75%)	1 (4.8%)	20 (95.2%)
Multidrug susceptibility							58 (23%)	194 (77%)

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Table (XIV): Antibiotic susceptibility testing for Gram negative isolates

Antibiotic	*S. aureus (5 isolates)*		*S. agalactiae (2 isolate)*		*Total (7 isolates)*	
	S	*R*	*S*	*R*	*S*	*R*
Amikacin	*2 (40%)*	*3 (60%)*	*-*	*2 (100%)*	*2 (28.6%)*	*5 (71.4%)*
Amoxicillin -clavulanic acid	*2 (40%)*	*3 (60%)*	*-*	*2 (100%)*	*2 (28.6%)*	*5 (71.4%)*
Cefoxitin	*1 (20%)*	*4 (80%)*	*2 (100%)*	*-*	*3 (42.9%)*	*4 (57.1%)*
Ceftazidime		*5 (100%)*	*-*	*2 (100%)*	*-*	*7 (100%)*
Ciprofloxacin	*2 (40%)*	*3 (60%)*	*2 (100%)*	*-*	*4 (57.1%)*	*3 (42.9%)*
Gentamicine	*1 (20%)*	*4 (80%)*	*-*	*2 (100%)*	*2 (28.6%)*	*5 (71.4%)*
Imipenem	*1 (20%)*	*4 (80%)*	*2 (100%)*	*-*	*4 (57.1%)*	*3 (42.9%)*
Kanamycin	*-*	*5 (100%)*	*1 (50%)*	*1 (50%)*	*1 (14.3%)*	*6 (85.7%)*
Nalidixic acid	*-*	*5 (100%)*	*-*	*2 (100%)*	*-*	*7 (100%)*
Meropenem	*1 (20%)*	*4 (80%)*	*-*	*2 (100%)*	*1 (14.3%)*	*6 (85.7%)*
Cotrimox	*3 (60%)*	*2 (40%)*	*1 (50%)*	*1 (50%)*	*4 (57.1%)*	*3 (42.9%)*
Tetracycline	*2 (40%)*	*3 (60%)*	*-*	*2 (100%)*	*4 (57.1%)*	*3 (42.9%)*
Multidrug susceptibility					*27 (32.14%)*	*57 (67.86%)*

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Urinary tract infection (UTI) is caused by the bacterial invasion and multiplication in the organs of the urinary tract system (Ifeanyichukwu et al., 2013). The frequency of UTI is gradually increasing amongst HIV-infected patients as an opportunistic infection. This is due to the unique pathogenesis of the virus, which decreases the $CD4^+$ cells, and as such, the individual's immune system can no longer fight against invading commensal organisms (Debalke et al., 2014; Olowe et al., 2015). E. coli, Proteus spp., Klebsiella spp., P.aeruginosa, Enterococcus spp., and S. aureus are the most causative agent of UTI in people living with HIV (Alemu et al., 2013; Olutosin et al., 2016). The health consequences of UTI among HIV-infected patients can be grave, resulting in acute and chronic kidney diseases (Akinbami et al., 2013), infertility, cancer, sepsis, and neurologic complication, which lead to urinary stasis (Charanchi et al., 2012; Rashmi et al., 2013). Some of the patients may substantially suffer from financial burden not only because of the recurrence of UTI but also due to the use of expensive antimicrobials, longer duration hospitalization, adverse drug effects, and unsatisfactory therapeutic options (Akinbami et al., 2013; Maniga et al., 2015). This study was to compare the prevalence of asymptomatic bacteriuria among HIV and healthy subjects and to evaluate the antimicrobial susceptibility of the bacteria isolated from HIV positive subjects, the comprised of 180 subjects, 100 (55.6%) were HIV positive subjects, 80 (44.4%) were HIV negative healthy subjects (control), among the HIV positive subjects mean age was (25.8± 6.66), ranging between 20-47 years old, while among the control subjects, the mean age was (29.3± 7.6 2) ranging between 20-45 years old, HIV positive group was comprised of 55% females, and 45% males, the control group was approximately matched, it was comprised of 52.5% females and 47.5% males.

Prevalence of UTI:

About 180 morning clean catch midstream urine samples were collected from patients attending Benghazi center for infectious diseases and immunolog, Significant asymptomatic bacteriuria was observed in 56/180 (31.1%).The prevalence of asymptomatic bacteriuria among HIV positive subjects was 28%, while in the control group it was 35%, but no significant differences in the prevalence among both groups, other studies reported that, patients with HIV had a UTI more frequently than the controls (Schönwald et al., 1999). Cultures counts less than 30 colonies of urine samples were not considered significant to define a UTI. However, in immunocompromised patients, this cannot be ignored. In this work the prevalence was relatively higher than reported in an older study in which the prevalence of asymptomatic bacteriuria in HIV positive patients attending BCIDI was (7.3%) (Buzayan and Taher, 2009). Other related studies showed varying urinary tract infection prevalence rates at different parts of the world. In a study conducted in Ethiopia the prevalence was (18%) (Marami et al., 2019), in India, the prevalence rate of urinary tract infection in HIV positive patients was (4.0%) (Banu and Ramachandrian, 2013). in Poland the prevalence was (23.2%) (Skrzat-Klapaczyńska et al., 2018), in Tanzania the prevalence of bacteriuria was (12.3%) (Ngowi et al., 2021). While higher prevalence rates of asymptomatic UTI were reported in Croatia (41%) (Schowald et al., 2009), in Uganda the prevalence was (32.2%) (Odoki et al., 2019) in India the prevalence was (77.5%) (Xavier et al., 2015), in South Africa the prevalence was (48.7%) (Benson et al., 2012). The divergence in the prevalence rates of urinary tract infections could be due to the difference in the immune levels/ HIV status of the patients who had participated in the studies which might have contributed to the increase or decrease in the prevalence rates of urinary tract infections in different geographic locations. The variation in prevalence rate of urinary tract infections in HIV infected persons from one geographical area to another could also be attributed to differences in UTI perception, mode of screening, compounding risk factors such as age, host behavioral factors and parity (Ogbukagu et al., 2016). In addition, due to differences in study methods, designs population, prevalence rates can differ from place to place. Also, the difference in the disease stage among patients can also contribute to the differences in prevalence rates. The difference in prevalence rates goes to also suggest that geographical location could be a pre disposing cofounding factor in the prevalence of urinary tract infections in HIV positive patients.

Prevalence according to gender:

The healthy urinary tract, like other body systems, is normally able to resist bacterial infections; numerous studies have indicated that the frequency of urinary tract infections is greater in women than in men. The prevalence of asymptomatic bacteriuria according to gender, was significantly higher in the females 20% (n = 20/100), than in the male patients 8% (n = 8/100) among HIV subjects, but the difference was not significant among the healthy subjects, this finding is agree with numerous previous studies This finding concurs with several other studies including the studies from India (Banu and Ramachandrian, 2013), Uganda (Odoki et al., 2019), Ethiopia (Marami et al., 2019) Poland (Skrzat-Klapaczyńska et al., 2018) and Kenya (Kayima et al., 1996).The reason for having high prevalence in females could be related to the close proximity of the urethral to the anus but also the short and wide urethra. This follows the trend of normal healthy individuals where females are at higher risk of being infected with urinary tract infections due to their short, straight urethra. The close proximity of the female urethra to the anus, autoinfection, incontinence, poor hygiene and bad toilet habits, have all been reported as factors that influence higher prevalence rate of urinary tract infections in females. The large intestines and the perinea area serve as reservoir for pathogenic bacteria. Different studies have indicated that women who are prone to urinary tract infections possess epithelial cells with significantly more receptors for uropathogenic bacteria than healthy controls (Debalke et al., 2014). This observation is supported by the reports of

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(Ogbukagu et al., 2016). However, these findings are in contrast to the study by Inyang – Etoh et al., who recorded higher prevalence rate 28.6% of urinary tract infections in males and 23.8% in female patients in Calabar, even though it was not statistically significant (Inyang–Etoh et al, 2009). Spence also reported higher urinary tract infection prevalence rate of 15.9% in males (Spence et al., 1996).

Prevalence according to age groups:

The prevalence of asymptomatic bacteriuria among different age groups showed no significant differences other studies reported that, the prevalence of urinary tract infections decreaes during middle age but rises in older adults the prevalence of urinary tract infections increase substantially (Rowe and Juthani–Mehta, 2013). Other studies done in Uganda and Ethiopia showed age to be a significant predictor of UTI (Odoki et al., 2019; Marami et al., 2019). Another study in Nigeria stated that, urinary tract infections occurred highest in age group 26 – 38 years (Ogbukagu, et al., 2016). In another study it was also observed that the prevalence rate of UTI in HIV seropositive individuals was highest in age group 24 – 30 years and least in age group 44 years and above (Kemajou et al, 2016). The prevalence of asymptomatic bacteriuria in younger age groups was almost twice as high as what was found in the older age group (Olowe et al., 2015). A significant association was also found between age and the presence of bacteriuria with younger individuals at higher risk (Olowe et al., 2015). However, this is in contrast to findings in South East Nigeria indicating that age group 60 – above had the highest prevalence of 100% followed by age group 30 – 44 (44.9%) (Kanu et al., 2016). In another related study carried out in Nigeria, age group 46 and above had the highest prevalence rate of urinary tract infections (Bigwan and Wakjissa, 2013).

The bacterial etiology:

In the present study about 28 uropathogens were identified, 21/28 (75%) were gram negative bacteria, while only 7/28 (25%) were gram positive pathogens. This finding also collaborate with most previous studies which implicating gram negative rods as the main cause of urinary tract infections in Ethiopia (Fenta et al., 2016; Ogbukagu, et al., 2016; Marami et al., 2019), in Italy, (Magliano et al., 2012), in Poland (Skrzat-Klapaczyńska1et al., 2018), in Uganda (Odoki et al., 2019), Nigeria (Olowe et al., 2015). This might be due to the presence of unique structures in gram negative bacteria used attachment to uroepithelial cells and prevent them from urinary lavage allowing for multiplication and tissue invasion resulting in invasive infections and pyelonephritis (Ifeanyichukwu et al., 2013). Other studies in Tanzania (Ngowi et al., 2021), in India (Prakash and Saxena, 2013), Nigeria (Ochada et al., 2014) in Pakistan (Bano et al., 2012) revealed that gram positive isolates were the most predominant isolated uropathogens. In this study among the 56 pathogens the mostly identified bacteria Among the gram negative species, E. coli was the most predominant bacterial etiology among the study population (33.9%), followed by K. pneumonia it was (25%) equally in both groups, P. aeruginosa was (8.9%), which was more seen in HIV positive subjects than control. while among gram positive species, S. aureus was the most common pathogen (17.9%) equally found in both groups, S. agalactiae was (5.4%), more frequently in HIV subjects than control. S. saprophyticus was (3.6%), S. pyogenes was (4.5%). Comparable results were noticed in the previous study conducted by (Buzayan and Taher, 2009). were E. coli was seen in (32%) of cases followed by K. pneumoniae (24%); K. terrigena (12%); S. aureus (12%), S. epidermidis (8%), and (4%) each of S.saprophyticus; E. aerogenes; Citrobacter koseri; P. mirabilis (Buzayan and Taher, 2009). The greater prevalence of members of enterobacteriaceae group in this study, especially the coliforms proved that a high percentage of urinary tract infections in our cohort may be due to fecal contamination and poor hygiene. In other related studies, gram negative bacteria were more prevalent than gram positive bacteria. This finding was comparable with other findings done in Ethiopia (Fenta et al., 2016; Ogbukagu, et al., 2016; Marami et al., 2019).

The most predominant isolated bacteria was E. coli. The preponderance of E. coli could be due to the presence of a unique structure that helps these bacteria for attachment to the uroepithelial cells, allowing for multiplication and tissue invasion, E. coli predominance may be due to E. coli is the most common microorganism in the vaginal and rectal area (Ali and Gholamreza, 2009), The high prevalence of E. coli in the female gender could be due to the close proximity of the anus to the vagina. This high possibility of UTIs in females is due to the inherent virulence of E. coli for urinary tract colonization such as its abilities to adhere to the urinary tract and also association with other microorganisms moving from the perineum areas contaminated with fecal microbes to the moist warmth environment of the female genitalia (Andabati and Byamugisha, 2010) The second most predominant uropathogen is Klebsiella spp. The most likely cause is that HIV-positive patients are at higher risk for infections due to hospital associated pathogens such as Klebsiella spp. This is due to more frequent necessity of hospitalization for patients with immunodeficiency and thus more frequent infections requiring hospitalization. This findings were in agreement with studies conducted in Ethiopia (Fenta et al., 2016; Ogbukagu, et al., 2016; Marami et al., 2019), in Italy, (Magliano et al., 2012), in Poland (Skrzat-Klapaczyńskal et al., 2018), in Uganda (Odoki et al., 2019), Nigeria (Olowe et al., 2015; Essien et al., 2015). In contrast, this study was inconsistent with the finding reported in Nigeria in which the predominant isolates were S. aureus (Ifeanyichukwu et al., 2013), in Ghana the predominant isolates were S. aureus and S. saprophyticus (Barnie et al., 2019). Most of the isolates reported from India were P. aeruginosa (Xavier, et al., 2015). In Croatia, Enterococcus species were the most frequent isolate in HIV patients, in South Africa, the most common organism isolated was P.aeruginosa (Bereczky et al., 2001). This variation in the

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type of bacteria isolates might be due to differences in sample size, specimen collection technique, sample processing, and personal and environmental hygiene (Adebayo and Salman, 2014; Marami,et al., 2019). Large scale studies revealed that, theses variations in the bacterial agents of UTI and percentage for uro-pathogens may be as a result of the very low level of immune suppression as well as the socio-cultural differences between the subjects (Anochie et al., 2001; Soje et al., 2006).

Antibacterial Susceptibility:

Antimicrobial resistance is a major clinical problem in treating infections caused by different bacterial pathogens and has increased over the years. In This study, the isolated uropathogens were tested for their sensitivity to 11 antimicrobial agents, the results of antimicrobial susceptibility testing revealed that for 21 isolated gram negative bacteria most isolates were resistant to the tested antibiotics and their resistance profile were as following: Tetracycline and Sulfamethoxazole (95.2%), Kanamycin (90.5%), Amoxicillin-clavulanic acid (Augmentin) (85.71%), Meropenem, Ceftazidime and Cefoxitin (76.19%), Amikacin and Ciprofloxacin (71.43%), Imipenem (57.1%) and Nitrofurantoin (38.1%). The gram negative isolates were somewhat susceptible to Nitrofurantoin by (61.9%). The results of antimicrobial susceptibility testing of 12 antimicrobial agents revealed that for 7 isolated gram positive bacteria most isolates were resistant to all tested antimicrobial agents and their resistance profile were as following: Ceftazidime and Naldixic acid (100%), Kanamycin and Meropenem (85.7%), Amikacin, Amoxicillin -clavulanic acid (Augmentin) and Gentamicine (71.4%). Cefoxitin (57.1%), Ciprofloxacin and Imipenem, Kanamycin, Sulfamethoxazole and Tetracycline (42.9%), this findings were in agreement with (Ngowi et al, 2021) who found that most of the gram positives bacteria were sensitive to nitrofurantoin (88.2%) while demonstrating resistance to ciprofloxacin and erythromycin by (77.8%) (and 60%) respectively. This is in disagreement with one of the Ethiopian studies that found a very high sensitivity (100%) to nitrofurantoin and erythromycin (Debalke et al., 2014). From this study, most of the bacterial isolates were resistant to the commonly used antibiotics like Augmentin and Sulfamethoxazole, and showed intermediate resistance to third generation cephalosporin (Cefoxitin and Ceftazidime), gram positive showed week susceptibility to Cefoxitin and Imipenem in addition to very poor susceptibility to Ciprofloxacin and Nitrofurantoin by gram negative isolates Theses findings were supported by previous study from Libya, in which all bacterial isolates showed high resistance rates to ampicillin, nitrofurantoin, amoxicillin/clavulanic acid and Trimethoprim/ sulfamethoxazole (Buzayan and Taher, 2009). This finding is in agreement with a study conducted by (Oladeinde et al., 2011) who found that Nalidixic acid, sulphamethoxazole–trimetoprim, amoxicillin–clavulanate showed very poor activity. And disagree The susceptibility profile indicates reported in the study conducted in Tanzania (Ngowi et al., 2021). In which high sensitivity to nitrofurantoin was noted, this is in consonance with the report of (Samuel et al.,2012) from Nigeria, (Msaki et al., 2012) and (Festo et al., 2011) in north-western Tanzania. These resistance profile may be due to long term use of these drugs over the years. Also, prescription of antibiotics without laboratory guidance as well as over the counter sales of antibiotics without prescription (Okeke et al., 1999; Omoregie and Eghafona, 2009). In this study most of the isolates were resistant to cotrimoxazole. The resistance to cotrimoxazole may be due to the fact that this drug is widely used for prophylaxis against *opportunistic infections associated with HIV (Lyamuya et al., 2011), this drug incorporated into the current drug management of* HIV/AIDS (Soje et al., 2006). This trend may also be a reflection of the changes in antibiotic sensitivity pattern recently noted in UTI in association with other morbidities that has been ascribed to wide spread self-medication and indiscriminate use of antibiotics practice that are more likely to be more with patients having HIV/AIDS (Ibadin et al., 2006; Aiyegoro et al., 2007). Multidrug resistance has serious implications on the health outcome of HIV-infected patients (Rashmi et al., 2013; Murugesh et al., 2014). It is quite alarming to note that almost (72.6 %) of gram positive and (71.7%) of gram negative isolates were found to be resistant to two or more antimicrobials. This was higher compared to the finding reported in Mysore, India (28%) (Murugesh et al., 2014) but it was lower than a report from Gondar, Ethiopia (95%) (Alemu et al., 2013). The high rate of resistance seen to the most commonly prescribed antibiotics in this study might be due to easy availability in the community, very cheap in terms of cost and subject to misuse.

iv. CONCLUSION

Significantly high prevalence of asymptomatic urinary tract bacteriuria (28%), than recorded in previous report less (7%). The prevalence of asymptomatic bacteriuria is significantly higher in female than males in HIV positive subjects. There was no statistical significant differences between different age groups. Among 28 samples that showed significant growth of bacterial isolates 21 (75%) samples were gram negative bacteria, while only 7 (25%) were gram positive pathogens. Gram negative rods (Enterobacteriacea) were the most predominant isolate. Twenty eight different uropathogens were isolated they are: *E. coli*, *K. pneumonia*, *P. aeruginosa*, *S. saprophyticus* and *Staphylococcus aureus*. Most isolates showed multidrug resistance profile, including commonly used antimicrobial agents (Augmentin), Quinolones, (Ciprofloxacin) and third generation cephalosporin (Ceftazidime).

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References

[1] Abdul K. and Abbas MD. (2004). Diseases of immunity. In: Kumar V., Abbas AK., Fausto N., eds. Robbin and Cotran pathologic Basis of Diseases ,7th ed. Philadelphia: Saunders, 245-258.

[2] Abelson B., Sun D., Que L., Nebel RA., Baker D., Popiel P., et al. (2018). Sex differences in lower urinary tract biology and physiology. Biol. Sex Diff., 9(1):45.

[3] Address T. (2018). 5,000 HIV infections reported in Libya in 2018. The Address 2018.

[4] Adebayo RA., Oladoyin AM., Irinonye OO. (2003).Comprehensive care for people living with HIV/AIDS; issues and problems of social integration in Nigeria. Nig J Med., 12(1):12-1.

[5] Adedoyin OT., Oyeyemi BO. and Aiyedehin OV. (2003). Screening of febrile children on hospital admission for urinary tract infection (UTI). Afr J clin and Exp Micro., 4(1):56-62.

[6] Aiyegoro OA., Igbinosa OO., Ogunmwonyi IN., Odjadjaro E., Igbinosa OE. and Okoh AI. (2007). Incidence of urinary tract infections among children and adolescents in Ile-Ife, Nigeria. Afr J Microbiol Res. 1:13-9.

[7] Akadri, AA., and Odelola, OI. (2020). Determinants of Asymptomatic Bacteriuria in HIV-positive and Negative Pregnant Women in Sagamu, South-West Nigeria. West Afr J med., 37(1), 1–6.

[8] Akinbami A., Bode-Shojobi I., Ajibola S. et al. (2013). Prevalence of asymptomatic bacteriuria in HIV infected patients in a Tertiary Hospital in Lagos, Nigeria. World J. AIDS, 3(2):105–110.

[9] Alemu A., Dagnew M., Alem M., and Gizachew M., (2013). Uropathogenic bacterial isolates and their antimicrobial susceptibility patterns among HIV/AIDS patients attending Gondar University Specialized Hospital, Gondar, Northwest Ethiopia", J Microbiol Res Rev., 1(4) 42–51.

[10] Ali J. and Gholamreza I. (2009). Asymptomatic urinary tract infection in pregnant women". Iran J Pathol.; 4:105–108.

[11] Alpers CE. (2004). The kidney. In: Kumar V, Abbas AK, Fausto N, eds. Robbins and Cotran Pathologic Basis of Disease, 7th ed. Philadelphia: Saunders, 955-19.

[12] Al-Rubeaan KA., Moharram O., Al-Naqeb D., Hassan A. and Rafiullah MR. (2013). Prevalence of urinary tract infection and risk factors among Saudi patients with diabetes". World J Urol., 39(3): 573-578.

[13] Andabati G. and Byamugisha J. (2010). Microbial aetiology and sensitivity of asymptomatic Bacteriuria among ante-natal mothers in Mulago hospital, Uganda. Afr Health Sci., 10 (4): 349–352.

[14] Anochie JC., Nkanginieme KEO. and Eke PC. (2001). The influence of instruction about method of urine collection and storage on prevalence of urinary tract infection. Nig J Paediatr.; 28: 39-42.

[15] Arienzo A., Celliti V., Ferrante V., Losito F., Stalio O. et al. (2016). on behalf of GREAT Network A pilot clinical trial on a new point-of-care test for the diagnosis and fast management of urinary tract infections in the emergency department". Int J Clini Med Mic.1:107.

[16] Aswani SM., Chandrashekar U., Shivashankara K. and Pruthvi B. (2014): Clinical profile of urinary tract infections in diabetics and non-diabetics. Aust Med J., 7(1): 29- 34.

[17] Augosto MA. (2007). Urinary tract infections. In: Mahon CR, Lehman DC, Manuselis G, eds". Diagnostic Microbiology, 3rd ed. New Delhi: Saunders. 1023-1024.

[18] Awolude, OA., Adesina, OA., Oladokun, A., Mutiu, WB., and Adewole, IF. (2010). Asymptomatic bacteriuria among HIV positive pregnant women. Virulence, 1(3), 130–133.

[19] Bano K., Khan J., Begum RH. et al. (2012). Patterns of antibiotic sensitivity of bacterial pathogens among urinary tract infections (UTI) patients in a Pakistani population, African J Microbiol Res., 6:414–420.

[20] Banu A. and Ramachandrian J. (2013). Asymptomatic bacteriuria in HIV positive individuals in a tertiary care hospital. J HIV Hum Reprod.;1 (2):2–5.

[21] Barnie P., Akwetey S., Swallah M., Acheampong D. and Kwakye-Nuako G. (2019). Occurrence and distribution of bacterial uropathogens among antiretroviral therapy users and non-users, Cape Coast Teaching Hospital

[22] Barrons R. and Tassone D. (2008). Use of Lactobacillus probiotics for bacterial genitourinary infections in women: a review. Clin Ther., 30(3):453-468.

[23] Beerepoot MA., Geerlings SE., van Haarst EP., van Charante NM., Ter-Riet G. (2013) Nonantibiotic prophylaxis for recurrent urinary tract infections: A systematic review and meta-analysis of randomized controlled trials. J Urol.,190(6):1981-1989.

[24] Benson I., Chibuzo O., Kenn A., Ramalivhana N., Toshio H. and Anthony K. (2012) Uropathogens isolated from HIV-infected patients from Limpopo Province, South Africa. Afr J Biotechnol., 11:10598–10604.

[25] Bent S, Nallamothu BK, Simel DL, Fihn SD, Saint S. (2002). Does this woman have an acute uncomplicated urinary tract infection? JAMA. 287(20):2701-2710.

[26] Bereczky BZ., Chetty P., Akhtar SS. and Abel-Goad EH. (2001). Unusual urological presentation of HIV/AIDS at king Edward VIII hospital, South-Africa. South Afri J HIV Med;. 18-20.

[27] Bigwan EI and Wakjissa FD (2013). Prevalence of Urinary Tract Infections among HIV Patients Attending a Non – Governmental Health Facility in Jos, Plateau State, Nigeria. IJBAR.; 4. 8.

[28] Bryce A., Hay AD., Lane IF., Thornton HV., Wooton M. and Costelloe C. (2016). Global prevalence of antibiotic resistance in paediatric urinary tract infections caused by Escherichia coli and association with routine use of antibiotics in primary care: systematic review and meta analysis. BMJ., 15:352:i939.

[29] Charanchi S, Kudi A, Tahir F. (2012). Antimicrobial sensitivity patterns of urogenital bacterial isolates among HIV positive patients in the Federal Medical Centre in Gombe. IJID . 2012;10(1):1-6

[30] Charanchi S., Kudi A. and Tahir F. (2012). Antimicrobial sensitivity patterns of urogenital bacterial isolates among HIV positive patients in the Federal Medical Centre in Gombe," IJID, 10(1):1–6.

[31] Cheesbourgh M, ed. District Laboratory Practices in the tropical countries. Low priced ed., Part 2. New York: Cambridge University Press. 2002:62-115.

[32] Cihlar T, Fordyce M. (2016). Current status and prospects of HIV treatment. Curr Opin Virol., 18:50-56.

[33] Coetzer E. (2004). Urinary tract infection in adults". CME., 22(4):182-188.

[34] Collee JG., Fraser AG., Marmion BP. and Simmons A. Mackie & McCartney Practical Medical Microbiology,14th ed., India: Raj Kamal Electric Press, 2006: 84-90.

[35] Croxen MA, Finlay BB. (2010). Molecular mechanisms of Escherichia coli pathogenicity. Nature Reviews. Microbiology, 8(1):26-38.

[36] Culligan EP, Sleator RD, Marchesi JR, Hill C. (2014). Metagenomics and novel gene discovery: Promise and potential for novel therapeutics. Virulence., 5(3):399-412.

[37] Daw AM., Sifennasr NEM., Draha AM., Daw AA., Ahmed MO., Mokhtar ES., El-Bouzedi AH., Daw IM, Adam SI. and Warrag S. (2019). In association with Libyan Study Group of Hepatitis and HIV. Spatiotemporal analysis and epidemiological characterization of the human immunodeficiency virus (HIV) in Libya within a twenty five year period: 1993-2017. AIDS Res Ther., 25;16(1):14.

[38] de Gaetano K., Tumbarello M., Tacconelli E., Bertagnolio S., Rabagliati R., Scoppettuolo G. et al. (2003). Impact of highly active antiretroviral therapy (HAART) on the incidence of bacterial infections in HIV-infected subjects. J Chemother., 15(1):60–65.

[39] Debalke S., Cheneke W., Tassew H. and Awol M. (2014). Urinary tract infection among antiretroviral therapy users and nonusers in Jimma University Specialized Hospital, Jimma, Ethiopia. Int J Microbiol., 6: 968716

[40] Drekonja DM., Rector TS., Cutting A. and Johnson JR. (2013). Urinary tract infection in male veterans: Treatment patterns and outcomes. JAMA., 173(1):62-68.

[41] Duffau P, Ozanne A, Bonnet F, Lazaro E, Cazanave C, Blanco P, et al. (2018). Multimorbidity, age-related comorbidities and mortality: Association of activation, senescence and inflammation markers in HIV adults. AIDS., 32(12):1651-1660.

[42] Elder JS. (2004). Urologic disorders in infants. In: Behrman RE, Kliegman RM, Jenson HB, eds. Neslon text book of Pediatrics, 17th ed. Philadelphia: Saunders,:1783-1826.

[43] El-Gadi S., Abudher A. and Sammud M. (2008). HIV-related knowledge and stigma among high school students in Libya. Int J STD AIDS.,19(3):178–183.

[44] Elhodairi MA., Mutwakil GA., Almahdi W. and Fulton J. (2008). HIV/AIDS in Libya. Sebha Med J.;7(2):36–43..

[45] Essien UC., Iheukwumere CC., Davou GI., Sheyin Z., Okolie CE, Ede ER and Ekwenifu AI (2015). Prevalence and Predictors of Asymptomatic UTI among HIV Positive Patients in Jos". Inter J Curr Microbiol App Sci., 4, 454.

[46] Fenta GM., Legesse MH., and Weldearegay GM. (2016). Bacteriuria and their antibiotic susceptibility patterns among people living with HIV attending Tikur Anbessa Specialized and Zewditu Memorial Hospital ART clinics, Addis Ababa, Ethiopia". J Bacteriol Parasitol., 07(05).

[47] Festo E., Kidenya BR., Hokororo A. and Mshana SE. (2011). Predictors of urinary tract infection among febrile children attending at Bugando Medical Centre, Northwestern Tanzania. Archives Clin Microbiol., 2 (5): 3823/239.

[48] Finer G. and Landau D. (2004). Pathogenesis of urinary tract infections with normal female anatomy. The Lancet infect dis., 4(10): 631-635.

[49] Florescu MC, Miles CD, Florescu DF. What do we know about adenovirus in renal transplantation? Nephrol, Dialysis, Transplant., 2013;28(8):2003-2010.

[50] Foxman B, Barlow R, D'Arcy H, Gillespie B, Sobel JD. (2000). Urinary tract infection: Self-reported incidence and associated costs. Ann Epidemiol., 10(8):509-515. -7

[51] Foxman B. (2000). Epidemiology of urinary tract infections: Incidence, morbidity, and economic costs. Am J M., 113(Suppl. 1A):5S-13S.

[52] Foxman B. (2010). The epidemiology of urinary tract infection. Nature Reviews. Urology.,7(12):653-660.

[53] Foxman B. (2014). Urinary tract infection syndromes: Occurrence, recurrence, bacteriology, risk factors, and disease burden. Infect. Dis. Clin. N. Am., 28(1):1-13.

[54] Goddard J., Turner AN., Cumming AD., et al. (2006). Kidney and urinary tract disease". In: Boon NA, Colledge NR, Stewart AD. eds. Davidson Principles and Practice of Medicine, 20th ed. Edinburgh: Churchill Livingstone:467.

[55] Gökengin D., Doroudi F., Tohme J, et al. (2016). HIV/AIDS: trends in the Middle East and North Africa region. Int J Infect Dis.,44:66–73.

[56] González-Chamorro F, Palacios R, Alcover J, Campos J, Borrego F, Dámaso D. (2012). Urinary tract infections and their prevention. Actas Urológicas Españolas., 36(1):48-53.

[57] Gottschick C, Deng ZL, Vital M, Masur C, Abels C, Pieper DH, et al. (2017). The urinary microbiota of men and women and its changes in women during bacterial vaginosis and antibiotic treatment. Microbiome. 5(1):99.

[58] Grabe M., Bartoletti R., Bjerklund Johansen TE., Cai T., Cek M., Koves B., Naber KG., Pickard RS., Tenke P., Wagenlehner F. and Wullt B. (2015). Guidelines on urological infections. Available from: http://uroweb.org/ [Accessed 31st April 2023].

[59] Hamarsheh O. (2020). HIV/AIDS in Palestine: a growing concern. Int J Infect Dis.;90:18–20.

[60] Hamdam HZ., Ziad AH., Ali SK. and Adam I. (2011). Epidemiology of urinary tract infections and antibiotics sensitivity among pregnant women at Khartoum North Hospital". Ann Clin Microbiol Antimicrob., 10:2.

[61] Hammar N., Farahmand B., Gran M., Joelson S. and Andersson SW. (2010). Incidence of urinary tract infection in patients with type 2 diabetes. Experience from adverse event reporting in clinical trials". Pharmacoepidemiol Drug Saf., 19(12):1287-1292.

[62] Harper M. and Fowlis G. (2007). Management of urinary tract infections in men. Trends in Urol Gynecol Sexual Health. 2007;12:30-35.

[63] Harrison LS. (2007). Staphylococci. In: Mahon CR, Lehman DC, Manuselis G, eds. Textbook of Diagnostic Microbiology, 3rd ed. New Delhi: Saunders, 367-381.

[64] Hawkey PM. and Lewis DA, eds. (1989). Medical bacteriology a practical approach". 1st ed. Oxford: © IRL Press., 15-16.

[65] Heinzelmann M., Mercer-Jones. MA. and Passmore JC. (1999). Neutrophils and renal failure", American journal of kidney diseases; 34(2): 384-399.

[66] Hirji I., Guo Z., Andersson SW., Hammar N. and Gomez-Caminero A. (2012). Incidence of urinary tract infection among patients with type 2 diabetes in the UK General Practice Research Database (GPRD). J Diabetes Complications., 26(6): 513-516.

[67] Hoepelman A., van Buren M, van den Broek J Borleffs JC. (1992). Bacteriuria in men infected with HIV-1 is related to their immune status (CD4+ cell count). AIDS., 6(2):179-84.

[68] Hooton TM, Bradley SF, Cardenas DD, Colgan R, Geerlings SE, Rice JC, et al. (2010). Diagnosis, prevention, and treatment of catheter-associated urinary tract infection in adults: 2009 International Clinical Practice Guidelines from the Infectious Diseases Society of Am. Clin Infect Dis., 50(5):625-663.

[69] Hrbacek J., Konopasek P., Eis V., Hamsikova E., Tachezy R., Pokorny J. et al., (2010). Urologic complications of HIV infection. Cas Lek Cesk., 149(3):115-119.

[70] Hyun G, Lowe FC. (2003). AIDS and the urologist. The Urologic Clinics of North America. 30(1):101-109.

[71] Ibadin OM., Onunu A. and Ukoh GM. (2006). Urinary tract infection in adolescent/ young adult Nigerians with acquired immune deficiency Disease in Benin City. J Med Biomed Res.; 5(2):55-60.

[72] IfeanyI I. Nwakeze E., Chika E., Oji A., Udu-Ibiam E., Afiukwa N. and Ngwu J. (2013). Frequency and antibiogram of uropathogens isolated from urine samples of HIV infected patients on antiretroviral therapy, Am. J. BioSci; 50:50–53.

[73] Ingersoll MA. (2017). Sex differences shape the response to infectious diseases. PLoS Pathogens. 13(12):e1006688.

[74] Inyang-Etoh PC., Udofa GC. Alaribe AA. and Udonwa NE. (2009). Asymptomatic bacteriuria in patients on antiretroviral drug therapy in calabar. J Med Sci., 9(6):270–275.

[75] Jahnukainen T., Chen M. and Celsi G. (2005). Mechanisms of renal damage owing to infection. Pedi nephrol., 20(8):1043-1053.

[76] Johnson L., Dorrington RE. and Moolla H. (2017). HIV epidemic drivers in South Africa: A model-based evaluation of factors accounting for inter-provincial differences in HIV prevalence and incidence trends. S Afr J HIV Med., 18(1), a695.

[77] Jombo GTA., Egah DZ. and Ayeni JA. (2005). Bacteriology of urinary tract infection among patients with acquired immunodeficiency syndrome in Jos, Nigeria. Nig J Med.; 14(2):422-4.

[78] Kanu AM., Mgbajiaka N. and Abadom N, (2016). Prevalence of Urinary Tract Infection among HIV Patients in Aba State". IJID., 45(1):229

[79] Kayima JK., Otieno LS., Twahir A. and Njenga E. (1996). Asymptomatic bacteriuria among diabetics attending Kenyatta National Hospital. East Afr Med J., 73(8):524–526.

[80] Kemajou TS., Ajugwo AO., Oshoma CE. and Oi E. (2016). Antibiotic resistance of bacterial isolates from HIV positive patients with Urinary Tract Infection (UTI) in Portharcourt, Nigeria. J AIDS Clin Res., 7 (8):8–11.

[81] Kennedy T. (2003). Urinary tract infection. In: Rudolph CD, Rudolph AM, eds. Rudolph's Paediatrics, 21st ed. New York: McGraw Hill. 1667-1673.

[82] Kim VH. (1991). Disorders of the kidney and urinary tract, In: Stanfield P, Brueton M, Chan M, eds. Disease of children in the subtropics and tropics. 4th ed. London: Arnold, 1991:784-804

[83] Klasinc R., Rieger A., Presterl E., Wrba T. and Diab M.(2017). Epidemiology of urinary tract infections in HIV-positive patients at a tertiary care hospital in Central Europe (2011–2016). Infect Disord Targets, 18(3):199–206.

[84] Kowalska JD., Kubicka J., Siwak E., Pulik P., Firlag-Burkacka E., Horban A. et al., (2016). Factors associated with the first antiretroviral therapy modification in older HIV-1 positive patients. AIDS Res Ther., 13:2.

[85] Kwan DJ. and Lowe FC. (1992). Acquired immunodeficiency syndrome. A venereal disease. The Urolo Clin North Amer., 19(1):13-24

[86] Lebovitch S. and Mydlo JH. (2008). HIV-AIDS: urologic considerations. Urol Clin North Am., 35(1):59-68; vi.

[87] Lee LK., Dinneen MD. and Ahmad S. (2001). The urologist and the patient infected with human immunodeficiency virus or with acquired immunodeficiency syndrome. BJU International, 88(6):500-510.

[88] Lehman DC. (2007). Biochemical identification of Gram-negative bacteria". In: Mahon CR, Lehman DC, Manuselis G, eds. Textbook of Diagnostic Microbiology, 3rd ed. New Delhi: Saunders, 212-233.

[89] Lehmann LE., Hauser S., Malinka T. et al., (2011). Rapid qualitative urinary tract infection pathogen identiication by SeptiFast real-time PCR. Bereswill S, ed. PLoS One, 6(2):e17146.

[90] Levison ME. and Kaye D. (2013). Treatment of complicated urinary tract infections with an emphasis on drug-resistant gram-negative uropathogens. Curr Infect Dis Rep., 15(2):109-115.

[91] Linhares I., Raposo T., Rodrigues A. and Almeida A. (2015). Incidence and diversity of antimicrobial multidrug resistance proiles of uropathogenic bacteria. BioMed Res Inter., Article ID: 354084.

[92] Lombo B., Alkhalil I., Golden MP., Fotjadhi I., Ravi S., Virata M. et al., (2015). Prevalence of Metabolic Syndrome in Patients with HIV in the Era of Highly Active Antiretroviral Therapy. Conn Med., 79(5):277±81.

[93] Lyamuya E., Moyo S., Komba E., and Haule M. (2011). Prevalence, antimicrobial resistance and associated risk factors for bacteriuria in diabetic women in Dar Es Salaam, Tanzania", Afri J Microbiol Res., 5 (6):683–689.

[94] Mach KE., Wong PK. and Liao JC. (2011). Biosensor diagnosis of urinary tract infections: A path to beter treatment? Trends Pharmacol Sci., 32(6):330-336.

[95] Magliano E., Grazioli V., Deflorio L., Leuci AI., Mattina R., Romano P. et al. (2012). Gender and age-dependent etiology of community-acquired urinary tract infections, Sci. World J., 349597.

[96] Maniga N., Mogaka G., Nyambane L. and Eilu E. (2015). Prevalence and susceptibility pattern of bacterial urinary tract infections among pregnant HIV positive women in Gucha sub country, Kenya, Sp Bact Pathogens J., 1(1):10–15.

[97] Marami D., Balakrishnan S. and Seyoum B. (2019). Prevalence, Antimicrobial Susceptibility Pattern of Bacterial Isolates, and Associated Factors of Urinary Tract Infections among HIV-Positive Patients at Hiwot Fana Specialized University Hospital, Eastern Ethiopia, Can J Infect Dis Med Microbiol., 6: 6780354.

[98] Miles BJ., Melser M., Farah R., Markowitz N. and Fisher E. (1989). The urological manifestations of the acquired immunodeficiency syndrome. J Urol., (3):771-773.

[99] Millán-Rodríguez F., Palou J., Bujons-Tur A., Musquera-Felip M., Sevilla-Cecilia C., Serrallach-Orejas M., et al. (2006). Acute bacterial prostatitis: Two different sub-categories according to a previous manipulation of the lower urinary tract. World J Urol., 24(1):45-50.

[100] Msaki BP., Mshana SE., Hokororo A. et al., (2012). Prevalence and predictors of urinary tract infection and severe malaria among febrile children attending Makongoro health centre in Mwanza city, North-Western Tanzania. Arch Public Health 70, 4.

[101] Munyi ST, Macharia WM, Alrour AJ, Njeru EK. (1998). Screening for urinary tract infection in children with cancer. East Afr med J., 75(5):264-67 .

[102] Murugesh K., Deepa S., Ravindranath C. and Venkatesha D. (2014). Multidrug resistant uropathogens in HIV: are they a threat to community?, Int J Sci Study., 2(3):38–42.

[103] NCCS National Committee for Clinical Standard: Performances standards for antimicrobial susceptibility testing, tenth international supplement M100-S15. Wayne, Pa. The Institute, 2005.

[104] NCDC. GARPR2015. In: Tripoli: National Centre for Disease Control, 2015

[105] Nelson JM. and Good E. (2015). Urinary tract infections and asymptomatic bacteriuria in older adults. Nurse Pract., 40(8):43-48.

[106] Ngowi BN., Sunguya B., Herman A., Chacha A., Maro E., Rugarabamu LF, Bartlett J., Balandya E., Mteta KA., Mmbaga BT. (2021). Prevalence of Multidrug Resistant UTI Among People Living with HIV in Northern Tanzania. Infect Drug Resist., 22(14):1623-1633.

[107] Nicolle LE. (2001). A practical guide to antimicrobial management of complicated urinary tract infection. Drugs & Aging. 18(4):243-254.

[108] Nicolle LE. (2008). Uncomplicated urinary tract infection in adults including uncomplicated pyelonephritis. Urol Clin North Am., 35(1):1-12, v.

[109] Nwokedi E and Dikko A.U. (2006). The physiology and microbial infection of the urogenital tracts , 1st ed. Kano: Hamsa Publication, 70-97.

[110] Ochada N., Nasiru I, Thairu Y., Okanlowan M. and Abdulakeem Y. (2014). Antimicrobial susceptibility pattern of urinary pathogens isolated from two tertiary hospitals in Southwestern Nigeria, Afr J. Clin. Exp. Microbiol., 16(1):12.

[111] Odoki M., Aliero AA, Tibyangye J., Nyabayo Maniga, J., Wampande, EM., Drago Kato C., Agwu E., and Bazira J. (2019). Prevalence of bacterial urinary tract infections and associated factors among patients attending hospitals in Bushenyi District, Uganda. Int J Microbiol., 1–8.

[112] Ogbukagu CM., Anakwenze VN., Ekwealor CC., Ezemba CC. and Ekwealor IA. (2016). Incidence of Urinary Tract Infections amongst Patients Attending Primary Health Centers in Anambra State. Advances in Microbiology. 6. 537.

[113] Ogunro PS., Ogungbamide TO., Elemie PO., Egbewale BE. and Adewole TA. (2006). Plasma selenium concentration and gluthathione peroxidase activity in HIV/AIDS infected patients; a correlation with disease progression. Nig Postgrad Med., 13(1): 1-5.

[114] Ogunshola FT., Okafor UE. and Osinapebi AO. (2005) Aetiology of catheter-associated bacteriuria in Lagos University Teaching Hospital". Nig Postgrad Med J.;12(2):89-92.

[115] Okafor HU., Okoro BA., Ibe BC. and Obi NU. (1993). Prevalence of asymptomatic bacteriuria among nursery school children. Nig J paed., 20:84-8.

[116] Okeke IN., Lamikanra A. and Edelman R. (1999). Socio–economic and behavioural factors leading to acquired bacterial resitance to antibiotics in developing countries. Emerg Infect Dis., 5(1): 18 – 27.

[117] Oladeinde BH., Omoregie R., Olley M. and Anunibe JA. (2011). Urinary tract infection in a rural community of Nigeria. N Am J Med Sci., 3(2):75-7.

[118] Oli AN., Okafor CI., Ibezim EC., Akujiobi CN. and Onwunzo MC. (2010). The prevalence and bacteriology of asymptomatic bacteriuria among antenatal patients in Nnamdi Azikiwe University Teaching Hospital Nnewi; South Eastern Nigeria. Nig J Clin Pract., 13(4):409-412

[119] Olowe OA., Ojo-Johnson BB., Makanjuola OB., Olowe RA. and Mabayoje VO. (2015). Detection of bacteriuria among human immunodeficiency virus seropositive individuals in Osogbo, south-western Nigeria. Eur J Microbiol Immunol., 5(1):126–30.

[120] Olutosin AA., Olubukola A., Oladokun A., Mutiu WB., and Adewole IF.(2016). Asymptomatic bacteriuria among HIV positive pregnant women. Virulence; 1(3): 130–135.

[121] Omoregie R. and Eghafona NO. (2009). Urinary tract infection among asymptomatic HIV patients in Benin City, Nigeria. Br J Biomed Sci., 66(4) 190–193.

[122] Ozumba UC., Mbonu OO. and Njoku-Obi AN. (1993). Bacteriology and antibiotic sensitivity in acute urinary tract infections in Enugu-Nigeria. Orient J Med., 5(2, 3): 67-71.

[123] Palmer CS., Hussain T., Duette G., Weller TJ., Ostrowski M., Sada-Ovalle I. et al., (2016). Regulators of Glucose Metabolism in CD4+ and CD8+ T Cells. Int Rev Immunol.., 35(6):477-88.

[124] Pickard R., Bartoleti R., Bjerklund-Johansen TE., Bonkat G., Bruyère F., Çek M., Grabe M., Tenke P., Wagenlehner F. and Wullt B. (2016). Guidelines Associates: Cai T, Köves B, Pilaz A, Pradere B, Veeraterapillay R. Guidelines on urological infections". Available from: htp://uroweb.org/ [Accessed 31st October 2022].

[125] Prakash D. and Saxena RS. (2013). Distribution and antimicrobial susceptibility pattern of bacterial pathogens causing urinary tract infection in Urban Community of Meerut City, India. ISRN Microbiology, 29;2013:749629.

[126] Rajesh B., Rattan LI. (2004). eds. laboratory diagnosis of important clinical syndromes. In: Essentials of Medical Microbiology, 3rd ed. New Delhi: Jaypee Brothers, :475.

[127] Rashmi K., Ravikumar K., Nimitha J. and Bhagyashree H. (2013). Asymptomatic bacteriuria in HIV/AIDS patients: occurrence and risk associated with low CD4 counts", J Evol Med Dental Sci., 2(19):3358–3366.

[128] RefWorld. (2004). Libya Situation of HIV/AIDS. In: Refworld, eds. Canada: Immigration and Refugee Board of Canada, 2004

[129] Renuart AJ., Goldfarb DM., Mokomane M., Tawanana EO., Narasimhamurthy M., Steenhoff AP., et al. (2013). Microbiology of urinary tract infections in Gaborone, Botswana. PLoS One. 8(3):e57776.

[130] Richard B. and Thomas JR. (2007. Specimen collection, transport and processing; bacteriology. In: Murray PR, Baron EJ, Jørgensen JH ,Landry ML, Pfaller MA, eds. Manual of Clinical Microbiology, 9th ed., vol. I. Washington DC; ASM Press: 291-333.

[131] Roderick DT. (1994). Office Urology". New York: McGraw Hill Book Company, 5-7.

[132] Ronald AR. and Alfa MJ. (1996). Microbiology of genitourinary system". In: Samuel Baron, ed. Medical Microbiology, 4th ed. Wesley: Churchill Livingstone, 1155.

[133] Rowe TA. and Juthani–Mehta M. (2013). Diagnosis and Management of Urinary Tract Infections in Older Adults. Infect. Dis Clin N Am., 28(1): 75–89.

[134] Russo TA., Stapleton A., Wenderoth S., Hooton TM. and Stamm WE. (1995). Chromosomal restriction fragment length polymorphism analysis of Escherichia coli strains causing recurrent urinary tract infections in young women. J Infect Dis., 172(2): 440-445.

[135] Samuel S., Salami T., Adewuyi G., Babatope E. and Ekozien M. (2012). Prevalence of urinary tract infections among a cohort of HIV positive patients accessing care in a rural health centre in Nigeria, J Microbiol Biotechnol., 2(4): 507–510.

[136] Schneeberger C., Kazemier BM and Geerlings SE. (2014). Asymptomatic bacteriuria and urinary tract infections in special patient groups: women with diabetes mellitus and pregnant women. Curr Opin Infect Dis., 27(1):108-114.

[137] Scholz EM. and Kashuba AD. (2021). The lymph node reservoir: Physiology, HIV infection, and antiretroviral therapy. Clin Pharmacol Therap., 109(4):918-927.

[138] Schönwald S., Begovac J. and Skerk V. (1999). Urinary Tract Infections in HIV Disease. Int J antimicrob agents., 11(3-4). 309-11.

[139] Skrzat-Klapaczyńska A., Matøosz B., Bednarska A., Paciorek M., Firląg-Burkacka E., Horban A. et al., (2018). Factors associated with urinary tract infections among HIV-1 infected patients, PLoS ONE., 13(1):1-10.

[140] Sleator RD. (2010). The human superorganism - of microbes and men. Medical Hypotheses. 74(2):214-215.

[141] Sobolewski K., Costello J. and Miller L. (2017). Development of antibiotic stewardship practices targeting urinary tract infections in a hospital with consultant-based infectious disease services. Physical Therapy, 42(8):527-532.

[142] Soje OO., Queena MA., Ojo KK., Adeniyi BA. and Roberts MC. (2006). CTX-M-15 extended spectrum beta-lactamase from Nigerian Klebsiella pneumoniae. J Antimicrob Chemother., 57: 24-30.

[143] Spellerberg B. and Brandt C. (2007). Streptococcus. In: Murray PR, Baron EJ, Pfaller MA, Landry ML, Jorgensen JH, eds. Manual of Clinical Microbiology, 9th ed. Washington DC; ASM Press. 412-429.

[144] Spence MR., Harwell TS. and Jones K. (1996). Asymptomatic bacteriuria in women infected with HIV-1. Int. Conf. AIDS., 11: 283-283.

[145] Srivastara RN. and Bagga A., eds. (2005). Paediatric Nephrology", 4th ed. New Delhi: Jaypee Brothers., 235-264.

[146] Staiman VR. and Lowe FC. (2004). Urologic problems in patients with acquired immune deficiency syndrome. Sci World J., 28(4 suppl 1): 427-7.

[147] Staiman VR. and Lowe FC. (2004). Urologic problems in patients with acquired immunodeficiency syndrome. Sci World J., 4(suppl. 1):427–437.

[148] Stalenhoefa JE., van Dissel JT. and van Nieuwkoop C. (2015). Febrile urinary tract infection in the emergency room. Curr Opin Infect Dis., 28:106-111.

[149] Tambyah PA. and Maki DG. (2000). Catheter-associated urinary tract infection is rarely symptomatic: A prospective study of 1,497 catheterized patients. Arch Inter Med., 160(5):678-682.

[150] Trevino S. and Marsik FJ. (2007). Performance Improvement in the Microbiology Laboratory. In: Mahon CR, Lehman DC, Manuselis G, eds. Textbook of Diagnostic Microbiology, 3rd ed. New Delhi: Saunders, 112-125.

[151] Tsai CC., Lai TM., Lin PP. and Hsieh YM. (2017). Evaluation of lactic acid bacteria isolated from fermented plant products for antagonistic activity against urinary tract pathogen Staphylococcus saprophyticus. Probiotics Antimicrob Proteins, 10 (2). 210-217.

[152] UNAIDS (2020). Country progress report - Libya, Global AIDS Monitoring 2020.

[153] UNAIDS (2020). Regional network of people living with HIV launched in the Middle East and North Africa 2020.

[154] UNAIDS Global AIDS Update 2022. Geneva: Joint United Nations Programme on HIV/AIDS; 2022. Licence: CC BY-NC-SA 3.0 IGO

[155] UNGASS (2012). UNGASS Country progress report 2010-2011.

[156] Vagios S., Hesham H. and Mitchell C. (2020). Understanding the potential of lactobacilli in recurrent UTI prevention. Microbial Pathogenesis, 148:104544.

[157] Vijayakumar N. and Prahald N. (2004). Urinary tract infections in children. In: Nammalwar BR, Vijayakumar M ,eds. Principles and practice of paediatric nephrology,4th ed., New Delhi: Jaypee Brothers, 311-318.

ISSN: 2959- 7463

[158] Wennerstrom M., Hansson S., Jodal U. and Stokland E. (2000). Primary and acquired renal scarring in boys and girls with urinary tract infection. J pediatr., 136(1):30-34.

[159] WHO. WHO provides lifesaving HIV medicines in Benghazi, 2017.

[160] Wong JK. and Yukl SA. (2016).Tissue reservoirs of HIV. Current Opinion in HIV and AIDS, 11(4):362-370.

[161] World Bank. Prevalence of HIV Libya Data. The World Bank The World Bank, 2020.

[162] Xavier TF., Auxilia A. and Kannan M. (2015). Isolation and characterization of UTI pathogens from HIV positive patients of Karur District, Tamil Nadu, India. Int J Curr Microbiol Appl Sci., 4(1), 558–563.

[163] Yepes A., Ávila E., Carreño H., Barreto J., Currea IY., Chaves ES., et al. (2010). Prevalencia de la infección urinaria adquirida en la comunidad en pacientes con HIV/SIDA. RFS Revista Facultad de Salud., 2(1):71-75.