University of Benghazi

Faculty of Science

Department of Botany

Bacterial Post-operative Wound Infections In Caesarean Sections and Gynaecology In Jomhoriya Hospital-Benghazi

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Dedication

I dedicate this work to:

- My father, who did not spare on some days and to my mother that give me all tenderness and love.
- My husband for his co- operation and support during the study.
- My sister Dr. Huda who guide me, my brothers, and my sisters for their continuous encouragement and patience .
- To everyone who learn me a letter.

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List of abbreviation

Surgical Site Infection	SSI
Anno Domini	AD
Centers for Disease Control	CDC
Limus Milk	LM
Surgical Wound Infection	SWI
Methicillin-resistant *S. aureus*	MRSA
Topical negative pressure	TNP
Vacuum –assisted closure	VAC
Carbon dioxide	CO_2
Hydrogen peroxide	H_2O_2
Microorganism	M.O
Hydrogen Sulfied	H_2S
Operation Theatre	O.T

P-dimethylaminobenzaldehyde	DMABA
Cystine lactose electrolyte deficient agar	CLED
Deoxycholate Citrate agar	DCA
Xylose Lysine Deoxycholate agar	XLD
Deoxyribonuclease	DNase
Sulfide Indole Motility Medium	SIM
Triple Sugar Iron agar	TSI
Human Immunodeficiency Virus	HIV

Abstract

The primary aim of this study was to determine the prevalence of bacteria in post-operative wound infection and its sensitivity to the commonly used antibiotic. During a period of four months between April to July 2010. Examination of wounds, with cultures of all suspicious wounds using standard bacteriological methods was performed. Of a total of 351 specimens were collected from caesarean sections and Gynecology in Jomhoriya hospital - Benghazi in Libyan, 221 (63%), became infected. Samples from the inanimate environment were also examined by taking pre moisted swabs from different areas to detect the bacteria that may be found in the surrounding environment of patients. The commonest causative organism was *Staphylococcus aureus* (19.9%), followed by *staphylococcus epidermidis* (10.6%), *klebsiella pneumonia* (9.4%), *Acinetobacter baumanni* (6%), *E.Coli* (4%), *pseudomona aeruginosa* (3.1%), *streptococcus agalactia* (3.1.3%), *Proteus mirbilis (2.3%)*, *S.haemolyticus* (0.9%) and *streptococcus viridaus* (0.3%). The sensitivity pattern of bacterial isolated from patients in postoperative wound infection, the organism were sensitive to ciprofloxacin, gentamicin, imipenem, Amikacin and chlorophenicol with ciprofloxacin showing the highest

percentage sensitivity, while Ampicillin, Pencillin, Amoxicillin, cephaloxin and colstin sulphat were showing the low percentage sensitivity. Factors associated with wound infection included obesity, pre-existing illness, duration of hospital stay, metabolic disease and wound contamination are considered as independent risk factors for wound infections.

CHAPTER ONE

Introduction

The term surgical site infection (SSI) was in introduced in 1992 to replace the previous term surgical wound infection. SSIs are defined as infections occurring within 30 days after a surgical operation (or within one year if an implant is left in place after the procedure) and affecting either the incision or deep tissue at the operation site. These infections may be superficial, deep incisional infections, infections involving organs or body spaces (Owens and Stoessel, 2008).

Postoperative wound infection results from bacterial contamination during or after a surgical procedure (Pradhan and Agrawal, 2009). SSIs are primarily caused by Gram positive organisms from the patient's own flora, which may be found on the skin, mucous membranes, or hollow viscera during surgical procedures. However, other organism can be introduced from inadequately sterilized contaminated surgical instruments, contaminated traumatic injuries, the operating room environment, or because of poor surgical technique (Jarvis and Marton, 1992, Gould and Chamberlain, 1994 and Rapp, 2000). Sources of infection also include: pathogens from the environment that can contaminate the wound through soil, clothing, and other foreign material. Examples of such infections

include contamination of a penetrating stab wound to the abdomen by colonic flora, contamination of a clean surgical wound in the operating room with *S. aureus* spread from the flora of a perineal carrier, and introduction of spores of *Clostridium tetani* into the tissues on a splinter (Ryan, 2004).

Classification of sources of infection included primary: acquired from community or endogenous and secondary: acquired from operating theatre or ward (nosocomial) or from contamination at surgery (Bailely and Loves, 2004). Surgical wound infection is a common postoperative complication and causes significant postoperative morbidity and mortality, prolongs hospital stay, and adds between 10 % and 20 % to hospital cost. Any purulent discharge from a closed surgical incision, together with signs of inflammation of the surrounding tissue should be considered as wound infection, irrespective of whether microorganisms can be cultured. Infection can occur at an incision are within 30 days of an operation.

However the development of wound infection depends on the integrity and protective function of the skin (Nandi *et al.,* 1999). The skin may be inhabited by bacteria that become resident there (commensal bacteria). They are of great value in preventing colonization by true pathogens by

competing for binding sites (receptors), competing for nutrients and secreting toxic substance to invading bacteria (Hunter, 1991). A pathogenic microorganism is defined as one that cause or is capable of causing disease. The infectious process can, in general, be devided into several stages: Entry into the host, with evasion of host primary defenses; adhesion of the microorganism to host cells; propagation of the organism; damage to host cells by bacterial toxins or an inflammatory response of the host; and evasion of host secondary defenses (Harvey ***et al.,*** 2007). Any breach of the skin surface, whether accidental or surgical, provides an open door for bacterial infection. Wound infection may be trivial, with simple local erythema, swelling and tenderness, or there may be pus formation, fever, wound dehiscence and delayed healing, or the infection may extend to cause local thrombophlebitis, lymphangitis and even septicemia, shock and sometimes death (Duerden ***et al***., 1990).

There are many factors that are thought to affect the susceptibility of any wound to infection, some of which strongly predispose to wound infection. These factors include pre-existing illness, length of operation, wound class, and wound contamination. Other factors such as extremes of age, malignancy, metabolic diseases, malnutrition, immunosuppression, cigarette smoking, remote site infection, emergency procedures, and long

duration of preoperative hospitalization are not considered as independent risk factors for wound infections (Sawyer and Pruett, 1994 and Garibaldi *et al.,* 1991).

1.2- Aim of the study

Postoperative wound infections are serious problems to many of people and frequently the person exposure to bacterial infection. Since, there is no study done in postoperative Caesarean and Gynaecology Wound Infection. Therefore the aim of this study:

1- Isolation and identification causative bacteria SSI.

2- To study the percent of bacteria that cause wound infection in Jomhoriya Hospital.

3- To study the sensitivity test of isolated bacteria to different Antibiotics.

CHAPTER TWO

2- Review of literature

2.1- Historical background

Wound infection is not a modern phenomenon. As early as 14-37Anno Domini (AD) there is documentary evidence that Cornelius Celsius (a Roman physician) described the four principal signs of inflammation and used 'antiseptic' solutions. Another Roman physician, Claudius Galen (130-200 AD) had such an influence on the management of wounds that he is still thought of by many today as the 'father of surgery'. It should also be remembered that he and some of his followers instigated the 'laudable pus' theory, which incorrectly considered the development of pus in a wound as a positive part of the healing process (Bibbings, 1984).Further historical references are listed in table 1 (Ellis, 1994 and Beilman and Dunn, 2009).

2.2- Structure and function of the skin

The skin is an organ system with multiple functions, including protection of the tissues from external microbial invasion. Its keratinized stratified epithelium prevents direct microbial invasion under normal condition of surface temperature, humidity, its normal flora, pH, chemical defenses tend to inhibit colonization by many pathogens.

Table 1: Historical background (1510 – 1994)

Ambrose pare (1510 – 1590)	Encouraged wounds to suppurate
Semmelweiss (1818-1865), Pasteur (1822 – 1895) and lister (1827 – 1912)	Accepted germ theory and introduced antiseptics
Florence Nightingale (1894)	Not in bacteriology but looking into drains (for smells) is the thing needed. Held farm belief in the benefit of hand –washing and strict hygiene
Mary Ayton (1985)	Defined terminology in current use for wound infection
Vincent Falanga (1994)	Identified the concept of critical colonization with fresh insights into chronic wound healing and non healing wounds

However, the skin is subject to repeated minor traumas that are often unnoticed but that destroys its integrity and allow organism to gain access to its deeper layers from the external environment. The surface is also penetrated by ducts of pilosebaceous untis and sweat glands, and microbial invasion can occur along these routes, particularly if the ducts are obstructed (Ryan, 2004 and Patel *et al*., 2000).

The skin consist of three main layers (Fig.1)

2.2.1- Epidermis:

Thin outer layer, nonvascular, and consist of stratified squamous epithelium which are a protective, coating that limits fluid loss, and contains melanin which colors the skin, contains only 10% water the skin regenerates quickly after damage if the basal layer is intact (Spence and Mason, 1987).

2.2.2- The Dermis:

Complex layer, contains blood vessels and sensory receptors for temperature, pain and pressure, contains hair follicles, sebaceous gland and ducts of sweat gland, provides mechanical strength because many collagen

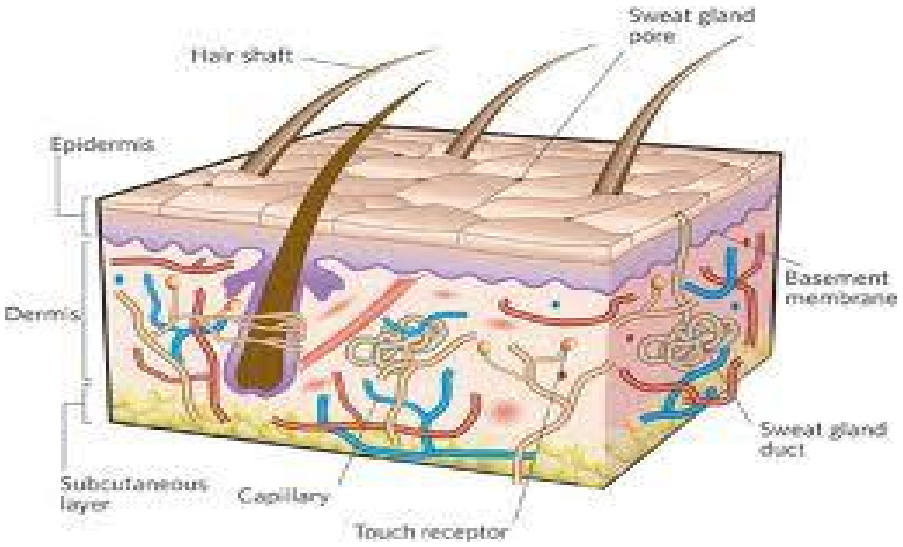

Figure (1): Anatomy of the skin

and elastin fibers, provides a defense against infection and helps deep wounds heal due to the activity of component (Spence and Mason, 1987).

2.2.3- The hypoderms, subcutaneous tissue:

This is below skin, and contains fat, with muscle and bone beneath, and may contain roots of hair follicles and sweat glands (Spence and Mason, 1987 and Nester et al., 2004).

2.3- Normal skin flora

The average human has approximately 1.8m of skin, colonized by vast numbers of bacteria, the quantity and type of these varying by site and by individual (table 2). In general, bacterial numbers tend to by lowest in cool, dry peripheral sites (hands and face), and highest in moist central sites (groin and axillae). Gram-positive organisms predominate, including micrococcaceae and coryneforms. Gram-negative organisms, such as the enterobacteriaceae, are generally confined to skin site adjacent to major reservoirs such as the gastrointestinal tract; Acinetobacter spp. are an exception, being found on up to one-quarter of individuals, most frequently isolated from the groin, axillae and toe webs (Sage, 2003).

Table 2: Normal skin bacteria Flora (Brooks *et al*.,1995).

Skin
1.*Staphylococcus epidermidis.*
2.*Staphylococcus aureus* (in small number).
3.Micrococcus species.
*4.*Non pathogenic Neisseria species.
*5.*Alpha-hemolytic and non hemolytic streptococci .
6.Diphtheroids.
7.Propionibacterium species.
8.Peptostreptococcus species.
9.Small numbers of other organisms(Candida species, Acinetobacter species, etc)

Most skin commensals, including Acinetobacter spp., are organisms of low pathogenicity, causing disease only in immunocompromised patients or when inoculated into sterile sites. All, however, share a propensity to develop multiple antibiotic resistances. On occasions, and mucous membranes of normal individuals become colonized with well-recognized pathogens, thus: some 10-20% of the population are colonized with *S. aureus.* Overt infection, such as recurrent boils and carbuncles, may follow (Sage, 2003). Patients with chronic skin disease such as eczema or psoriasis are universally colonized with *S. aureus,* and are significant disseminators of such organisms (Sage, 2003).

2.4- Wound infection

When the protective skin barrier is broken as a result of burns, puncture wound, surgical procedures, or bites, opportunistic indigenous microflora and environmental bacteria can invade and cause local or deep tissue infection. The pathogens may spread via blood or lymph, causing serious systemic infections (Burton and Engelkirk, 2004).

2.5- Surgical wound infections

Most surgical infections are caused by the patient's own organisms, but some arise exogenously, often by cross-infection. In orthopedic units, most exogenous infections are due to Staphylococci, but in gastrointestinal units, Gram -negative bacilli and anaerobes more common. The situation may change from time to time as a result of ecological movements in the microbial flora within the unit, associated with selective pressures of antibiotic used. Specimens from infected lesions always be sent to the laboratory, since the identity of the isolate may have epidemiological significance as well as being of importance in the management of the patient. The microbiologist always be informed of any therapy as this may affect the interpretation of bacteriological tests (Slack, 1997 and Inglis, 1996). The US Centers for Disease control (CDC) definition states that only infections occurring within 30 days of surgery (or within a year in the case of implants) be classified as surgical site infections (SSIs). Wound infections have been subdivided according to the following clinically related subgroups: A etiology: in a primary infection, the wound is the primary site of infection, the whereas a secondary infection arises following a complication that is not directly related to the wound; Time:

an early infection presents within 30 days of a surgical procedure, whereas an infection is described as intermediate if it occurs between one and three months afterwards and late if it present more than three months after surgery; Severity: a wound infection is described as minor if there is discharge without cellulites or deep tissue destruction, and major if the discharge of pus is associated with tissue breakdown, partial or total dehiscence of the deep fascial layers of the wound, or if systemic illness is present (Peel, 1992 and Oluwatosin, 2005).

2.6- Classification of surgical wounds

Surgical and traumatic wounds are classified according to the extent of potential contamination and thus, the risk of infection. These criteria carry important implications regarding surgical treatment and chemoprophylaxis (Ryan, 2004). Four categories of surgical wounds are recognized, differing in their liability to develop infections

2.6.1- Clean wounds:

Are due to elective surgery that does not involve entering the gastroin-testinal, genitor-urinary or respiratory tract. Infection rates of under 2% are the norm, with *S. aureus* as the commonest infecting organism. Such

infection is often exogenous and airborne, in contrast to the endogenous infections that predominate in the other categories of wound (Duerden *et al.,* 1990).

2.6.2- Clean contaminated or potentially contaminated wounds:

Occur when a site which has a resident bacterial flora is entered without significant spillage, as in an uncomplicated appendectomy or an elective cholecystectomy. Infection rate of 5 –10% are reported in this group (Duerden *et al*., 1990).

2.6.3- Contaminated wounds:

Occur when there is significant spillage of bacterial flora-e.g. from operations on the intestinal tract. The degree of contamination during the procedure will influence the sepsis rate, which is often in the range 15-20%. The enterobacteria and aerobes predominated in these infections (Duerden *et al*., 1990).

2.6.4- Dirty or infected wounds:

Occur when surgery involves a perforated viscus, or is to drain an abscess, or when devitalised tissue must be removed after trauma. Infection rates of greater than 30% are common in this group and antibiotic therapy

is an essential part of the treatment (Cruse and Foord, 1980 and Duerden *et al.,* 1990 and Culver *et al.,* 1991)

2.7- Factors Contributing to Wound Infection

Various factors, in addition to those indicated previously, contribute to the probability of a wound becoming infected. The contaminating dose of microorganisms and their virulence can be critical and, other things being equal, the chance of infection developing increases progressively with the contaminating dose. The physical and physiologic condition of the wound also influences the probability of infection. Areas of necrosis, vascular strangulation from excessively tight sutures, hematomas, excessive edema, poor blood supply, poor oxygenation all compromise normal defense mechanisms, substantially reduce the dose of organisms needed to initiate infection. Thus, removal of necrotic tissue, the surgeon's skill, gentleness, attention to detail are major factors in preventing the development of infection (Ryan, 2004). The general health, nutritional status, and ability of patients to mount an inflammatory response are also major determinants of whether a wound infection develops. Infection rates are higher in the elderly, the obese, individuals with uncontrolled diabetes, and those on immunosuppressive or corticosteroid therapy. Nutritional deficiencies enhance the risk of infection, and new approaches to avoid protein–calorie

malnutrition in patients with severe burns, for example, have led to substantial reductions in serious clinical infections. There is strong evidence that the critical period determining whether contamination of surgical wounds proceeds to infection lies within the first 3 hours after contamination. For this reason, prophylactic chemotherapy of some surgical wounds and procedures can be restricted to the operative and immediate preoperative period. There is general agreement that extending such prophylaxis beyond 24 hours increases the chance of complications without reducing the risk of infection (Ryan, 2004).

2.8- Pathogenesis

By the end of an operation, bacteria and other microorganisms contaminate all surgical wounds, but only a small number of patients actually develop a clinical infection (Fry, 2003). Infection does not develop in most patients because their defense mechanisms effectively eliminate the contaminating organisms at the surgical site. Whether a potential infection occurs depends on several factors, with the most important being:

number of bacteria entering the wound; type and virulence (ability to cause infection) of the bacteria; host defense mechanisms (e.g., effectiveness of inflammatory response and status of the immune system); and external

factors, such as being in the hospital several days before surgery or the operation lasting more than 4 hours (Krizek and Robson, 1975).

Two factors that can help minimize the number of organisms entering the wound are the skill and experience of the surgeon and use of good surgical technique. Both are important because if a surgical site is contaminated with more than (100,000) organisms per gram of tissue, the risk of SSI is markedly increased (Krizek and Robson, 1975). The dose required for infection can be even lower, however, if foreign material is present at the site (e.g., only about 100 staphylococci are enough if silk suture is used for closure or to control bleeding) (James and Macleod, 1961). While the type and virulence of the bacteria cannot be controlled, the other factors can to a large extent. For example, tissue injury caused by making the wound incision triggers a chain of events, called the inflammatory response, that take place even before bacterial contamination occurs. The effectiveness of the inflammatory response to mobilize patient defense mechanisms (e.g., activation of various types of white blood cells that contain and destroy the bacteria before infection can occur) depends to large extent on the patient's general health, age, obesity, smoking, some chronic diseases and the status of the immune system (Nichols, 2001).

2.9- The bacterial causing infection in surgical wound

2.9.1- Staphylococcus aureus

It is a Gram-positive non motile, non capsulate coccus occurring singly, in pairs, in short chains or in irregular cluster. Producing lactic acid but not gas. On initial isolation the organism typically produces a golden yellow pigment on mannitol salt agar, and the colonies are usually opaque, circular, and smooth. The organism grows well on blood agar, nutrient agar. Some strains are beta-haemolytic when grown aerobically also grow well carbon dioxide enriched atmosphere. Also grow anaerobically, but less well. The production of coagulase and DNAse whereas others *Staphylococci* are coagulase and DNAs Negative ***S. aureus*** is carried in the nose of 40% or more of healthy people. This species causes: Abscesses, boils, styes, impetigo. It may also cause secondary infections of wounds and skin disorders, cross-infections in hospital, septicaemia, endocarditis, osteomyelitis, pneumonia, empyema, mastitis, food-poisoning, scalded skin syndrome in young children and toxic shock syndrome (Cheesbrough, 1984 and Actor, 2007).

2.9.2- Streptococcus pyogens

Streptococcus pyogens (Lancefield Group A) Gram positive streptocpcci, non motile, capsulate. In fluid cultures, long chine is formed whereas in pus and preparations made from solid cultures the cocci tend to form short chains or may break up and be seen in pairs or singly. Beta-haemolytic on blood agar colonies are surrounded by a zone of complete haemolysis, usually less than 1mm in diameter, grey-white or colourless and sensitivity to bacitracin. Grow aerobically and anaerobically. This species causes: Acute sore throat, scarlet fever caused by erythrogenic toxin producing strains, ear infections, puerperal sepsis, skin infections, septicaemia and occasionally endocarditis (Cheesbrough, 1984 and Awetz *et al*., 1987).

2.9.3- Enterococci

Enterococci (Lancefield Group D) Gram positive streptococci, non motile, capsulate. On blood agar, enterococci colonies may be non-hemolytic, beta-hemolytic, or alpha-hemolytic. They can be identified as enterococci by rapid litmus milk (LM) reduction test. On macConkey agar, enterococci produce distinctive small dark red colonies. This group causes:

Urinary tract infections, infections of ulcers and wounds, occasionally endocarditisa and meningitis (Cheesbrougy, 1984 and Elmishad, 2002).

2.9.4- Anaerobic Streptococci

Most of the pathogenic Gram positive anaerobic streptococci belong to the genera Peptostreptococcus can cause septicaemia, puerperal sepsis, and bone and joint infections. Many strains are proteolytic and gas (H_2S) producing which gives infected material and cultures very unpleasant smell. On blood agar, the colonies are very small, shiny, and non-hemolytic (Cheesbrougy, 1984).

2.9.5- Clostridium spp.

The main species of medical importance are: *C. perfringens* (formerly *C. welchii*), *C. botulinum*. These species causes: Gas gangrene, food poisoning, puerperal infection, septicaemia, tetanus, very occasionally *C. botulinum* infects wounds. Gram positive rod, strict anaerobes or facultative anaerobes. *C. perfringens*: Non-motile and capsulated, the organism appears as thick brick-shaped rods, *C. tetani*: Motile and non-capsulated, long thin rods with round spore at one end, *C. botulinum*:

Motile pleomorphic rods with oval subterminal spores. On blood agar, large, beta-hemolytic colonies are produced (Cheesbrough, 1984).

2.9.6- Pseudomonas aeruginosa

Gram negative motile rods, bipolar stining, oxidase positive, it is an obligatory aerobe and is usually recognized by the yellow-green pyocyanin pigment it produces. ***P. aeruginosa*** usually produces large, flat, haemolytic colonies on blood agar. Also grows well on nutrient agar, MacConkey agar and other media containing bile salt. This species causes: Skin infections especially at burn sites, wounds, urinary infection, respiratory infections, external ear infections, eye infections, septicaemia (Cheesbrough, 1984).

2.9.7- Proteus spp.

The main species of medical importance is: ***P. mirabilis.*** Occasionally infections are also caused by ***P. vulgaris.*** These are actively motile, non-capsulate, Gram negative pleomorphic rods, non-lactose fermenting, swarming on blood agar, however, is inhibited on media containing bile salts such as MacConkey agar, DCA, XLD agar, and SS agar. ***Proteus*** rapidly hydrolyzes urea. These species causes: Urinary infections,

abdominal infection, wound infection, septicaemia, occasionally meningitis and chest infections (Cheesbrough, 1984).

2.9.8- Escherichia coli

E. coli is a Gram negative usually motile rod, non-sporing, some strains are capsulated; grow well aerobe and facultative anaerobes. On blood agar the colonies may appear mucoid and some strains are haemolytic. Whereas, on MacConkey agar, most *E. coli* strains produce lactose fermenting colonies. An important biochemical feature of most *E. coli* strains is the production of indole from peptone water containing tryptophan. *E. coli* causes*:* Urinary infections, wound infections, bacteraemia, meningitis especially of the newborn and diarrhoeal disease especially in infants but also in adult (Collee *et al*., 1989).

2.9.9- Bacteroides spp

Bacteroides spp. are Gram negative, pleomorphic rods, non-motile, non-sporing. They often stain palely or unevenly, obligate anaerobeses, on blood agar produces brown to black haemolytic colonies. *Bacteroides spp.* Causes: Genitourinary infections, appendicitis infections, wound infections, bacteraemia. Also abscesses of brain, long and liver (Cheesbrough, 1984).

2.9.10- Klebsiella spp.

Klebsiella spp. Are Gram negative rod, non-motile, capsulated, aerobes and facultative anaerobes, produce large and usually mucoid colonies. On blood agar large grey white. Whereas MacConkey agar most strains produce lactose fermenting appear mucoid pink colonies and CLED agar appear mucoid yellow colonies. The organism gives a positive urease test. Klebsiella spp. Causes: Chest infection, urinary infection, septicaemia, meningitis, peritonitis and wound infections (Murray et al., 1998).

2.9.11- Pasteurella spp.

The mean species of medical importance is *P. multocida*, Gram negative, non-motile coccobacillus, Oxidase positive, indole positive, virulent strains are capsulated. Most strains show bipolar staining when stained by the Giemsa technique. On blood agar, *P. multocida* colonies are small, non-haemolytic, translucent and sometimes coloured blue. Can cause respiratory infections, bacteraemia, meningitis, abscesses, ulcers, arthritis and osteomyelitis (Cheesbrough, 1984).

2.10- Recognition of wound infection

The inflammatory response is a protective mechanism that aims to neutralise and destroy any toxic agents at the site of an injury and restore tissue homeostasis (Coller, 2003). There are a number of indicators of infection, these include the classic signs related to the inflammatory process and further more subtle changes as highlighted by Cutting and Harding (Cutting and Harding, 1994).The classic signs of infection include:

-Localised erythema

-Localised pain

-Localised heat

-Cellulitus

-Oedema.

Further criteria include:

-Abscess

-Discharge which may be viscous in nature, discoloured and purulent

-Delayed healing not previously anticipated

-Discolouration of tissues both within and at the wound margins

-Friable, bleeding granulation tissue despite gentle handling of and the non adhesive nature of wound management materials used

-Unexpected pain and/or tenderness either at the time of dressing change or reported by the patient as associated specifically with the wound even when the wound dressing is in place

-Abnormal smell

-Wound breakdown associated with wound pocketing/bridging at base of wound, ie when a wound that was assessed as healing starts to develop strips of granulation tissue in the base as opposed to a uniform spread of granulation tissue across the whole of the wound bed.

The above criteria should be used as discriminating factors when the 'classic' signs of wound infection do not appear to be present but the presence of a wound infection is suspected, usually as a result of a delay in wound healing that was not anticipated from the patient's medical history or knowledge of the patient's wound.

2.11- Surgical site infection

Since skin is normally colonised by a range of microorganisms that could cause infection, defining an SSI requires evidence of clinical signs and symptoms of infection rather than microbiological evidence alon. SSIs frequently only affect the superficial tissues, but some more serious an SSI requires evidence of clinical signs and symptoms of infection rather than microbiological infections affect the deeper tissues or other parts of the body manipulated during the procedure. The majority of SSIs become apparent within 30 days of an operative procedure and most often between the 5^{th} And 10^{th} postoperative days. However, where a prosthetic implant is used, SSIs affecting the deeper tissues may occur several months after the operation. Although the outcome measure for SSI used by many studies is based on standard definitions such as those described by the Centers for Disease Control and Prevention or the Surgical Site Infection Surveillance Service, other valid measures based on clinical signs and symptoms have been described such as the Southampton and Asepsis methods (Horan *et al.,* 1992, Ridgeway *et al.,* 2005, Bailey *et al.,* 1992 and Wilson *et al.,* 1986).

The CDC definition describes three levels of SSI

• Superficial incisional, affecting the skin and subcutaneous tissue. These infections may be indicated by localised (Celsian) signs such as redness, pain, heat or swelling at the site of the insicion or by the drainage of pus.

• Deep incisional, affecting the fascial and muscle layers. These infections may be indicated by the presence of pus or an abcess, fever with tenderness of the wound, or a separation of the edges of the incision exposing the deeper tissues.

• Organ or space infection, which involves any part of the anatomy other than the incision that is opened or manipulated during the surgical procedure, for example joint or peritoneum. These infections may be indicated by the drainage of pus or the formation of an abscess detected by histopathological or radiological examination or during pre- operation. Organ infection is not included within the scope of this guideline. In addition, there may also be microbiological evidence of wound infection from cultures obtained aseptically from wound fluid or tissue. However, since skin sites are normally colonised by a variety of organisms, positive wound cultures in the absence of clinical signs are rarely indicative of SSI. Some studies reported infections that affect any part of the incision,

whereas other studies focus only on those that affect the deeper tissues as these may be considered to be more important and their definition less subjective (Bailey *et al.,* 1992).

Onch and Adedeji (2004) reported that two hundred and fifty-four patients were recruited and 19 had post-operative wound infection.Thirty-six bacterial isolates were recovered. *S. aureus* was the commonest in 16 cases (44%), *B. fragilis* 4(11%), *E. coli* 4(11%), proteus spp.4 (11%). Others were Pseudomonas spp., Klebsiella spp. and Peptostreptococcus. Cephalosporine were found to be the most potent against *S. aureus* while the anaerobes responded favourably to metronidazole.

Simillary Chia *et al.,* (1993) reported that *S. aureus* was the most common organism isolated from post-operative wound infection (POWI).This study covers 6.639 major operations in Ka-ndang Kerbau Hospital , Singapore over a 12-month period,of which 2.489 were caesarean sections and 4.150 were gynaecological operations. Of 150 cases had wound infection, therefore the overall wound infection rate was 2.26%.The highest wound infection rate occurred in hystere-ctomies and the lowest in laparoscopies. There was a good correlation between monthly caesarean wound infection rate and number of caesarean sections. *S. aureus* was (58%) were followed by Streptococcus spp. (10.5%), Klebsiella spp.

(9.5), Enterobacter spp. (5.7%), *P. aeruginosa* (4.8%), *E. coli* (3.8%), Proteus spp. (2.9%) and Acinetobacter spp. (2.9%).

In addition in study of Banjara *et al.*, (2002) showed that, rate of surgical wound infection (SWI) was 4.7% (189/3988).Were collected and processed at Microbiology Laboratory Tribhuvan University Teaching Hospital. The highest infection rate was *S. aureus* (24.9%), *E. coli* (23.9%), *P. aeruginosa* (19.8%), *K. pneumoniae* (11.7), Acinetobacter spp. (7.6%), *Citrobacter fruendii (*4.6%), *P. mirabilis* (2.5%), *Citrobacter diversus* (1.0%), *S. epidermidis* (1.0%), *Streptococcus faecalis* (1.0%), *K. oxytoca* (1.0%), *Proteus vulgaris* (0.5%) and *Aeromonas hydrophila* (0.5%).

A total of 350 swab specimens from postoperative wounds consenting patients in Central Hospital, Benin City, were screened for the presence of aerobic pathogens and *Candida albicans.* The 348 specimens (96.4%) yielded pathogens in the following order: *S. aureus* (35%), *P. aeruginosa* (26%), *E. coli* (13%),*Candida albicans* (9.3%), *K. aeruginosa* (7.4%), Proteus spp. (7.4%) and Streptococcus spp. (1.9%).The highest infection was seen in the age group 51 years and above, followed by 41-50, 21-30 and 11-20, while 0-10 years gave the lowest incidence. A gradual decline in resistance to infection among patients in the age group 51 and above could be responsible for the high prevalence rate (100%) observed in this

study. The anti-microbial susceptibility test indicated that there were differences in the sensitivity and resistance patterns of the isolates (Isibor *et al*., 2008).

Another study by Giacometti *et al*., (2000) included 676 surgery patients. Bacterial pathogens were isolated from 614 individuals. Among the common pathogens was *S. aureus* (191 patients, 28.2%), *P. aeruginosa* (170 patients, 25.2%), *E. coli* (53 patients, 7.8%), *S. epidermidis* (48 patients, 7.1%) and *Enterococcus faecalis* (38 patients, 5.6%).

The occurrence in 2441 postoperative wounds were studied during 15 month-period from January 1987 to March 1988 at Songklanagarind Hospital to deter-mine the postoperative wound infection rate.There were 159 wounds that became infected. *S. aureus* remain the most important microorganism responsible for POWI (36%), *P. aeruginosa* (24%), *E. coli* (16%), *K. pneumonia* (11%) and *Proteus mirabilis* (5%). Yielding an overall infection rate of 6.5%.When categorized operation by traditional wound classificantion, infection occurred in 3.6% of clean wound 8.4% of clean-contaminated wound 11.8% of contaminated wound and 31.0% of dirty or infected wound (Jamulitrat *et al*., 1988).

Tourmousoglou *et al.,* (2008) reported that 898 patients were enrolled and electively operated in a General surgey Clinic in Athens, Greece. Overall, 402 patients underwent a clean and 496 patients underwevt a clean-contaminted operation. A total of 17 SSIs (4.2%) were observed in clean and 64 SSIs (12.9%) in clean-contaminted operations. *S. aureus* was the commonest microorganism isolated, followed by *E. coli* and *P. aeruginosa*.

In contrast Anupurba *et al.,* (2006) reported that prevalence rate of *P. aeruginosa* was (32%) of overall 301 the pathogens isolated from POWI at SS Hospital, Varanasi, India. Antibiotic susceptibility was determined by the disc diffusion method where cefoperazone was found to be most effective (74%) followed by ciprofloxacin (58%) and ceftazidime (54%). Simillarly Masaadeh and Jaran, (2009) recovered *P. aeruginosa* in POWI. During a period of six months between February to December, 2005, 115 specimens were collected from King Abdullah University Hospital, Princess Basma Hospital, Princess and no growth (1.7%). The microorganisms were sensitive to amikacin, gentamicin, ciprofloxacin and aztreonam.

During 702 surgical patients, 80 (11.4%) developed an SSI. The most frequently isolated pathogens were *P. aeruginosa* (29.5% of isolates),

S. aureus (11.5% of isolates), and *E. coli* (10.3% of isolates). Ninety percent of *S. aureus* isolates were methicillin resistant, 91% of *P. aeruginosa* isolates were ceftazidime resistant and 38% of *E.coli* isolates were cefotaxime resistant (Thu *et al.,* 2006).

Whereas Bakir et al., (1995) reported that the commonest causative organismis were coagulase-negative *Staphylococci* (21.7%), *S. aureus* (19.7%), *E. coli* (19.7%) Enterobacter spp. (17.6%) and Pseudomonas spp. (10.7%). A prospective study of POWI was carried out over a two year period in Cumhuriyet University Medicine Faculty Hospital in Sivas,Tukey. Of a total of 4146 surgical wounds, 188 (4.53%), became infected.

Brook and Frazier, (1999) studied the bacterial growth in the infected site. Sixty isolates were recovered: 38 aerobes and 22 anaerobes. The predominant aerobes were *E. coli* (n =8) and Proteus spp. (n=7). The predominant anaerobes were *Bacteroides fragilis* group (n=9) and Peptostreptococcus spp. (n=6) isolates.

Owens and Stoessel, (2008) reported that incidence of SSIs may be as high as 20%, depending on the surgical procedure, the surveillance criteria used, and the quality of data collection. In many SSIs, the responsible

pathogens originate from the patient's endogenous flora. The causative pathogens depend on the type of surgery; the most commonly isolated organisms are *S. aureus*, coagulase-negative Staphylococci, Enterococcus spp. and *E. coli*.

The factors affecting the incidence of POWI included four various factor (age, preoperative stay, shaving and the surgeon) were shown to have a statistically significant association with the development of wound infection. A strong association between the individual surgeon and the development of a wound infection was demonstrated and this supports the need for routine surgical audit (Mishriki ***et al.***, 1990).

Study by Henderson and Love, (1994) showed that, the women delivered by primary caesarean section had significantly higher rates of endometritis, deep surgical wound infection and bacteraemia than those delivered by secondary section. All type of post-caesarean infection, except asymptomatic bacteriuria, caused the duration of the post-partum hospital stay to be significantly increased.

Study of post-operative infection in 124 patients under caesarean section, showed that 39 (31.5%) patients developed a total of 45infection. Wound infection was found in 14.5% developed a post-operative urinary

tract infection, found the risk factors predisposing to post-caesarean infection were obesity and low socioeconomic status (Parrott *et al*., 1989).

Another studies by Moir-Bussy *et al.*, (1984) and Pelle *et al*., (1986) showed that, a prospective incidence into wound infection after caesarean section was carried out. Factor associated with wound infection included size of hospital, obesity, time in labor, number of vaginal examinations and various operative procedures. The occurrence most risk factors associated with the development of a surgical site infection: body mass index, age, blood loss, method of wound closure, antimicrobial prophylaxis, wound classification, duration of operation, malignant neoplasm, emergency procedures. These results suggest that caesarean section is associated with high infectious morbidity, the extent of which would have been considerably underestimated without post-discharge monitoring. Almost all women with wound problems were treated with antibiotics, regardless of how minor the problem, with 97% being prescribed in the community. This indicates a requirement for local review of antibiotic prescribing practice (Garcia *et al.*, 1997, Johnson *et al.,* 2006 and Ward *et al.,* 2008).

2.12- Defenses against Infection

A macroorganism manifests defensive reactions against invasion by microorganisms in two forms: as specific, acquired immunity and as nonspecific, innate resistance.

2.12.1- Nonspecific Defense Mechanisms

2.12.1.1- Primary defenses**:**

The main factors in the first line of defense against infection are mechanical, accompanied by some humoral and cellular factors. These defenses represent an attempt on the part of the host organism to prevent microorganisms from colonizing its skin and mucosa and thus stave off a generalized invasion.

2.12.1.2- Secondary defenses**:**

The second line of defense consists of humoral and cellular factors in the blood and tissues, the most important of which are the professional phagocytes.

2.12.1.3- Phagocytosis:

"Professional" phagocytosis is realized by polymorphonuclear, neutrophilic, eosinophilic granulocytes also known as microphages and by

mononuclear phagocytes (macrophages). Microphages contain both primary granules, which are lysosomes containing lysosomal enzymes and cationic peptides, and secondary granules. Phagocytes are capable of ingestion of both particulate matter (phagocytosis) and solute matter (pinocytosis). Receptors on the phagocyte membrane initiate contact (Fig. 2). Particles adhering to the membrane are engulfed, ingested and deposited in a membrane-bound vacuole, the so-called phagosome, which then fuses with lysosomes to form the phagolysosome.

2.12.2- Specific Defense Mechanisms

Specific immunity, based on antibodies and specifically reactive T lymphocytes, is acquired in a process of immune system stimulation by the corresponding microbial antigens. Humoral immunity is based on antitoxins, opsonins, microbicidal antibodies, neutralizing antibodies, etc. Cellular immunity is based on cytotoxic T lymphocytes (T killer cells) and T helper cells.

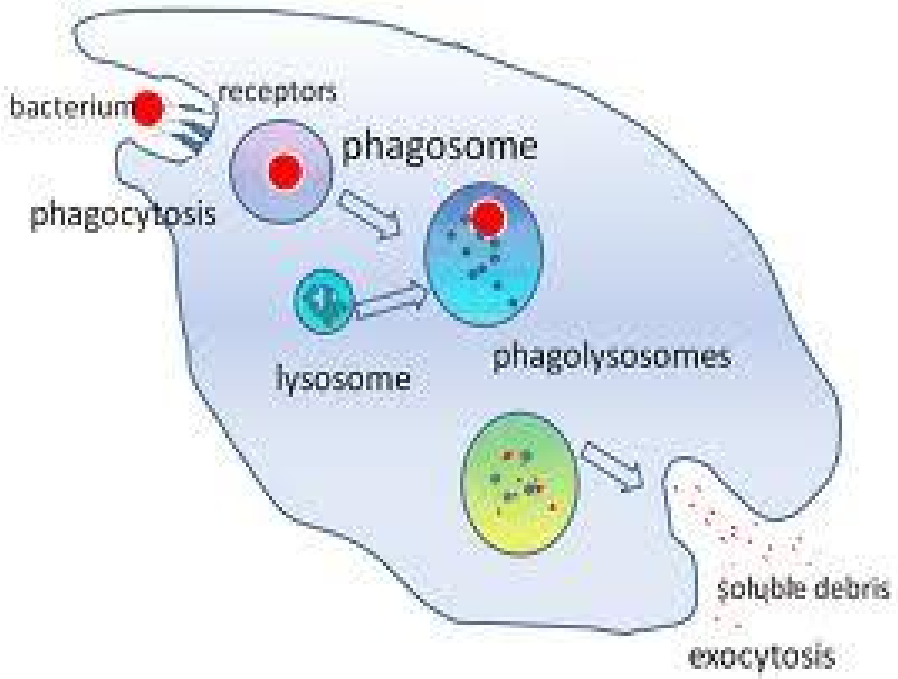

Figure (2): Phagocytosis of bacteria

2.13- Defects in Immune Defenses

Hosts with defects in their specific and/or nonspecific immune defenses are prone to infection.

2.13.1- Primary defects: - Congenital defects in the complement-dependent phagocytosis system are rare, as are B and T lymphocyte defects.

2.13.2- Secondary defects: - Such effects are acquired, and they are much more frequent.

Examples include malnutrition, very old and very young hosts, metabolic disturbances (diabetes, alcoholism), autoimmune diseases, malignancies (above all lymphomas and leukemias), immune system infections (HIV), severe primary diseases of parenchymatous organs, injury of skin or mucosa, immunosuppressive therapy with corticosteroids, cytostatics and immunosuppressants, and radiotherapy. One result of progress in modern medicine is that increasing numbers of patients with secondary immune defects are now receiving hospital treatment. Such “problem patients” are frequently infected by opportunistic bacteria that would not present a serious threat to normal immune defenses. Often, the pathogens involved (“problem bacteria”) have developed a resistance to

numerous antibiotics, resulting in difficult courses of antibiotic treatment in this patient category (Drutz and Millis, 1987 and Kayser et al., 2005)

2.14- Treatment

Once a diagnosis of wound infection has been confirmed and antibiotic sensitivities identified, appropriate management regimens should be considered, with a high priority given to reducing the risk of cross infection. It is important to treat the patient as a whole and not the infection alone, so management strategies must be based on data derived from an holistic assessment of the needs of the individual (Collier, 2001). The main treatment objective will be to reduce rather than eradicate the bacterial burden within the wound margins. In addition to antibiotic therapy, there are two main generic groups of wound management products that have the potential to reduce the bacterial burden in the wound, these are compounds containing silver or iodine (Collier, 2003).

2.14.1- Antibiotic therapy:-

Antibiotics are chemical substances produced by a micro-organism that have the capacity, in dilute solutions, to selectively inhibit the growth of or to kill other micro-organisms (Cooper and Lawrence, 1996) Whereas it is

now generally accepted that systemic antibiotics are essential for the management of clinically infected wounds, the choice of antibiotic to be used is not always apparent. Only after a comprehensive assessment process including consideration of patient characteristics, the results of microbiological investigations and the identification of both the nature and location of the wound, can the most appropriate antibiotic be identified. The routine use of topical antibiotics is not justified for colonised or infected wounds (Anon, 1991). In addition, a recent systematic review of antimicrobial agents has concluded that systemic or topical antimicrobials are not generally indicated for the management of chronic wound infections (Omeara *et al*., 2001). However, there may be some values in the prophylactic use of topical antimicrobials for the initial management of acute cellulitus, whilst awaiting clarification of antibiotic sensitivity and the establishment of a therapeutic regimen.

Resistance to antibiotics has become a serious problem in recent years particularly with the rise of epidemic strains of Methicillin Resistant *S. aureus* (MRSA). The overuse of broad-spectrum antibiotics will only serve to exacerbate the situation. It could therefore be argued that all antibiotic use should be based on known sensitivities.

2.14.2- Iodine:-

Iodine is an element that has antiseptic properties. It is active against a number of pathogens. In the past its use has been limited by the fact that elemental iodine can be absorbed systemically, is almost insoluble and can be an irritant to the skin. In wound management iodine is used in two forms: Cadexomer iodine- a polysaccharide starch lattice containing 0.9% elemental iodine that is released on exposure to wound exudate. PVP-1 (Povidone iodine) - an iodophor composed of elemental iodine and a synthetic polymer. Both have different physical characteristics that relate to the component parts and the iodine concentration of available iodine that is released when in use. Clinically iodine is indicated for wound cleansing, wound bed preparation (the stimulation and influence of specific cells involved with the immune system) and the prevention and management of wound infection (Jones and Milton, 2000).

2.14.3- Silver:-

Recently a number of dressings containing silver have become available, although silver and silver compounds have been routinely used in clinical practice as bactericidals for over a century. Silver interferes with the bacterial electron transport system and inhibits the multiplication of the

bacteria. However, to achieve this, silver ions have to be able to enter a cell. The chemical bonding of silver with a sulphonamide antimicrobial - sulphadiazine - has resulted in the development of a safe broad-spectrum agent for topical use (eg Flamazine). In this formulation silver is released slowly from the transport medium in concentrations that are selectively toxic to micro-organisms such as bacteria and fungi. This type of silver product has been used successfully in the management of acute and chronic wounds (Jones and Milton, 2000). Products that can sustain the interaction of silver with micro-organisms in the exuding wound are likely to be more effective in preventing/controlling local infection as potentially more silver ions will be available to enter bacterial cells. This assumes that the concentration of silver in the solution is both correct and maintained.

2.14.4- Further interventions:-

Other appropriate wound management interventions that can be considered to help reduce the bacterial burden on the wound surface include autolytic or enzymatic debridement, surgical debridement, maggot therapy and the use of topical negative pressure (TNP) for example, vacuum-assisted closure (VAC), in conjunction with the use of appropriate secondary dressings as required (Collier,2002).

2.15- Prevention Of Surgical Site Infections

The most critical factors in the prevention of postoperative infections, although difficult to quantify, are the sound judgment and proper technique of the surgeon and surgical team, as well as the general health and disease state of patient (Nichols, 2001). Other factors influence the development of postoperative surgical site infection, especially in clean surgical procedures for which the infection rate (<3%) is related to airborne exogenous microorganisms (Nichols, 2004).

In 1999, the CDC's Health Care Infection Control Practices Advisory Committee published revised guidelines for the prevention of infections. These guidelines delve extensively into the literature concerning perioperative factors associated with postoperative infections (Mangram et al., 1999). Many factors proven to influence surgical site infections, such as length of preoperative stay, duration of operation, preoperative cleansing and shaving techniques, use of abdominal drains, presence of remote infection, and adequate control of serum glucose levels perioperatively are among others authoritatively reviewed. Lacking are studies that offer no recommendations (unresolved issue) on such factors as preoperative application of mupirocin to the nares, enhancement of nutritional support

and enhancement of wound space oxygenation. The subject of operating room or patient temperature is not considered in these CDC guidelines (Nichols, 2004).

CHAPTER THREE

3- Material and Methods

3-1- Patients

Over the period of four months (April to July 2010) a total 351 samples collected from patient were admitted to the units of caesarean section, Gynaecology and outpatient department (opd) in Jomhoriya hospital – Benghazi. Thire were with ages ranging from 18 years to 76 years, each patient fill in the question.

3.2- Collection of sample and isolation of bacteria

The samples were collected within third day of operation with sterile cotton wool swab taken from the surgical wound area from patient admitted to the hospital (Masaadeh *et al.*, 2009).

Whenever possible the specimens were transported to the laboratory where they were cultured on chocolate agar CO_2 (Fig.3), on blood agar anaerobically (Fig.4) and on macConkey agar. All incubated at 37^0C for overnight (18-24hr) (Joffe *et al.*, 1978). Identification of isolated bacteria was confirmed by using BD Phoenix system and biochemical tests.

Figure (3): Chocolate agar plate in CO_2 jar

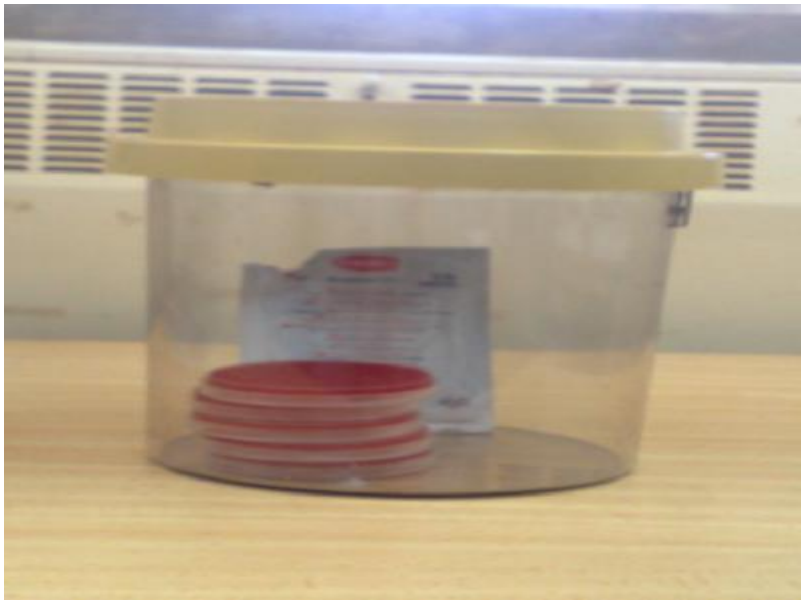

Figure (4): Blood agar plates in anaerobic jar

3.3- Environment

The samples were taken after cleaning and disinfection procedures, also air cultures were done before routine medical staff activities with patients and after finishing and examined for total microbial presence and for the occurrence of pathogenic or opportunistic microbial species. Isolated from the unit and O.T environment including the liquids used, floors, walls bathrooms, tables, anesthesia machine, anesthesia mask, air conditioner and air. The air in the operating theatre and in the dressing room was also sampled using settle plates, exposed for half an hour each, before incubation. In the past the procedure frequently adopted for determining the relative numbers and species of microorganisms present in air has been to expose open plates of culture medium for given periods of time and if blood agar is used, the occurrenence in the air of pathogenic Staphylococci and Streptococci can be determined. The method has proved valuable in demonstrating the presence of such organisms in the air and dust of hospital wards. Such findings have also thrown light on cross infection in hospital (Cruickshank *et al.,* 1976). All samples were cultured on blood agar and MacConkey agar plates, incubated aerobically 37^0C for at least 48 hours. Any isolates were then identified to the species level according to standard methods (Dujuid, 1989).

3.4-Gram stain and microscopic examination

A colony was taken from each culture growth, emulsified, maked a thin preparation on slide and then dried using gentle heat. All fixed dried smears were covered with crystal violet stain for 30-60 seconds, rapidly washed off the stain with filtered tap water, covered with Iodine stain for 30-60 seconds, washed off the iodine stain with filtered tap water, decolorized rapidly (few seconds) with acetone –alcohol, washed immediately with filtered tap water, covered the smears with neutral red stain for two minutes, washed, wiped the slides back, stayed in a draining rack for the smear to air –dry and then examined microscopically.

3.5-Catalase test

Catalase is an enzyme that decomposes hydrogen peroxide into water and oxygen (Cheesbrough, 1984). The catalase test is used to differentiate Staphylococci from non catalase producing bacteria such Streptococci. The test is performed by taking few colonies of the test organism, using a sterile wooden stick and immerses it in the hydrogen peroxide solution. Look for immedi- ate bubbling as shown in figure. Bubbles of oxygen will be seen in the tube test when catalase is produced by the organism (positive test). No release of bubble (negative test) (Fig. 5) (Cheesbrough, 1984).

3.6- Coagulase test

This test is used to differentiate *S. aureus* which produces the enzyme coagulase from Staphylococcus spp. which do not produce coagulase. Coagulase causes plasma to clot by converting fibrinogen to fibrin. Two types of coagulase are produced by most strains of *S. aureus.*

3.6.1- Free coagulase test:-

A small amount of the colony of the organism to be tested is emulsified in a sterile test tube containing 0.5ml of 1:10 diluted plasma in normal saline then in incubated at 37^0C and followed each half hour and reading intervals up to 6 hours and again overnight. Clot formation in positive test is observed by a gently tilting the tube (Fig.6).

3.6.2- Bound coagulase (slide test):-

The test is performed by adding a drop of distilled water on a clean slide to make a heavy suspension, then adding a drop of plasma and mixed thorough and observed for clumping in the positive test. Clumps indicate a negative test (Cheesbrough, 1984).

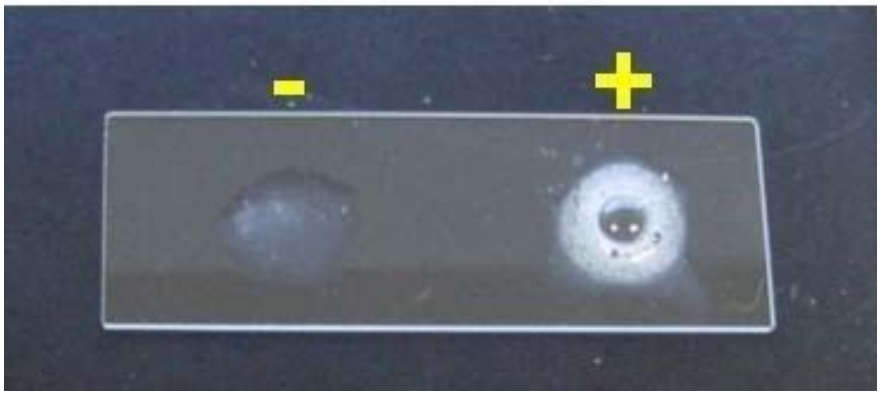

Figure (5): Catalase test

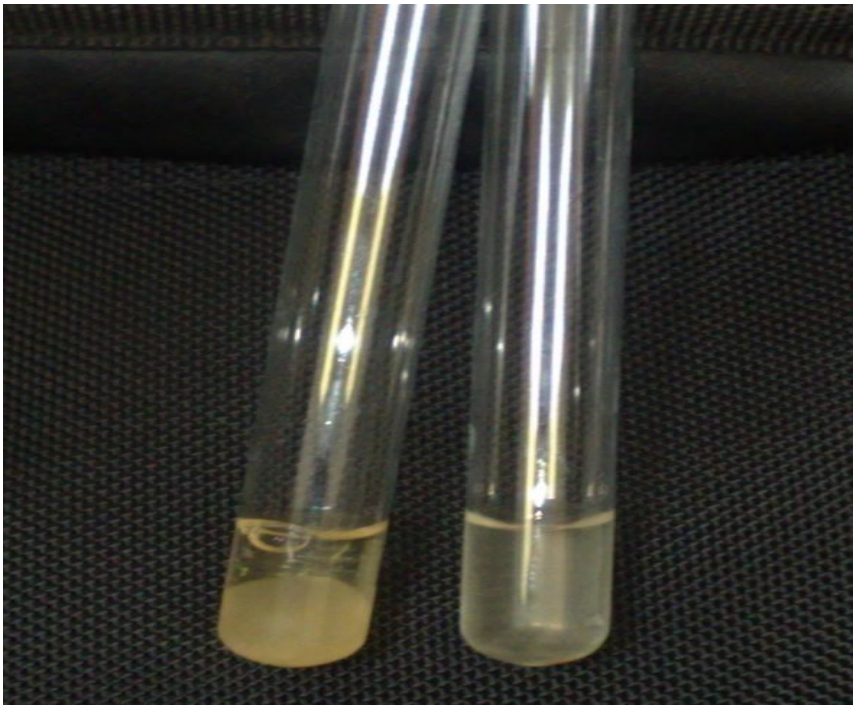

Figure (6): Coagulase test

3.7- Oxidase test (cytochrome oxidase)

The oxidase test is used to assist in the identification of Pseudomonas, Neisseria, Vibrio and Pasteurella species, all of which produce oxidase enzyme.The solution was prepared by dissolving 0.1g of tetramethyl-p-phenylenediamine dihydrochloride (Fig.7) in 10ml of distilled water. Placed apiece of filter paper in a clean Petri dish and added 2 or 3 drops of freshly prepared oxidase reagent. A few colonies are picked up and immediately smeared on to the soaked filter paper looked for the development of a blue-purple colour within a few seconds as shown in colour (Fig.8).

Positive oxidase control: *P. aeruginosa*

Negative oxidase control: *E .coli*

3.8-Biochemical tests

3.8.1-Urease test:

Testing for urease enzyme activity is important in differentiating enterobacteria proteus strains *and* klebsiella strains are strong urease producer. Whereas *E.coli* does not produce urease.

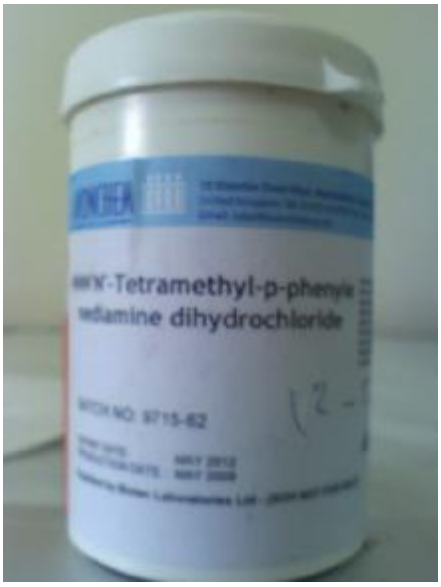

Figure (7): Tetramethyl-p-phenylenediamine dihydrochloride

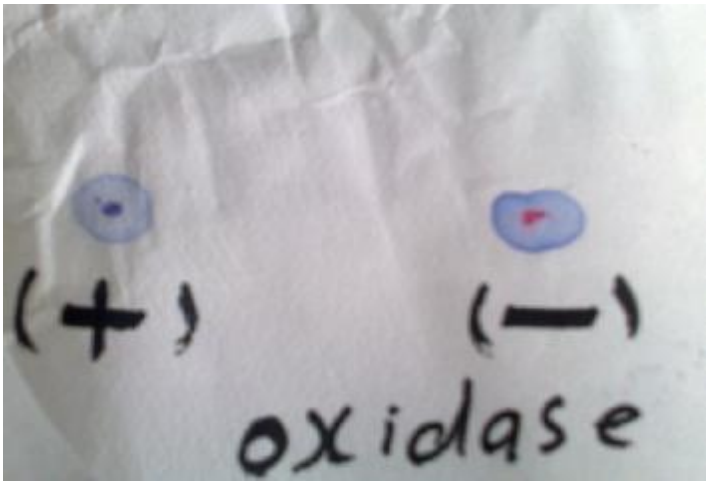

Figure (8): Oxidase test

The test organism is cultured in a medium which contains urea and the indicator phenol red. If the strain is urease –producing the enzyme will break down the urea (by hydrolysis) to give ammonia and carbon dioxide (CO_2) with the release of ammonia, the medium becomes alkaline as shown figure by a change in colour of the indicator to red-pink (Fig.9) (Cheesbrough, 1984).

3.8.2-Citrate utilization test:

This test is one of several techniques used to assist in the identification of enterobacteria. The test is based on the ability of an organism to use citrate as it's only source of carbon and ammonia as it's only source of nitrogen.

The tests organism is cultured in medium which contains sodium citrate, an ammonium salt, and indicator bromo-thymol blue. Growth in the medium is shown figure by turbidity and a change in colour of the indicator from light green to blue, due to the alkaline reaction, following citrate utilization (Fig.10) (Cheesbrough, 1984).

Figure (9): Urease test

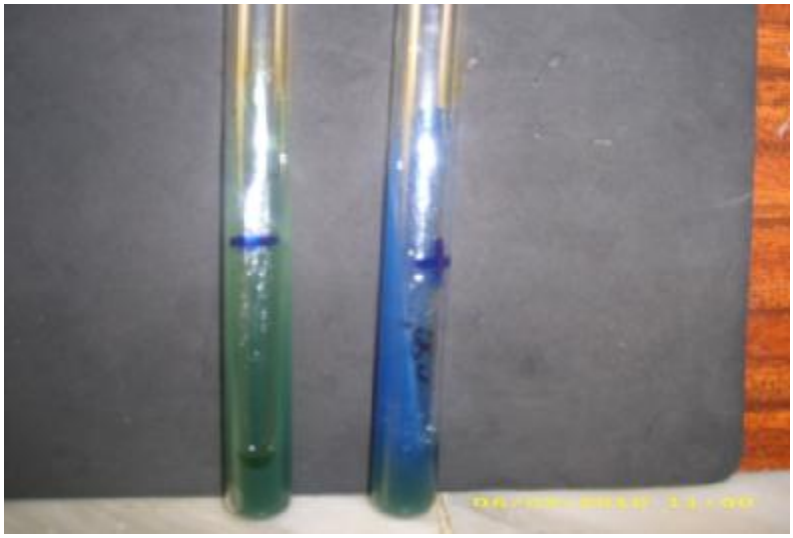

Figure (10): Citrate test

3.8.3- Sulfide Indole Motility Medium (SIM)

SIM medium is a combination differential medium that tests three different parameters, which are represented by the three letters in the name: sulfur reduction, indole production and motility.

SIM medium contains nutrients, iron, and sodium thiosulfate. One of the nutrients is peptone, which contains amino acids, including tryptophan. If an organism can reduce sulfur to hydrogen sulfide, the hydrogen sulfide will combine with the iron to form ferric sulfide, which is a black precipitate. If there is any blackening of the medium, it indicates the reduction of sulfur and is a positive result (Cheesbrough, 1984).

The sulfur and motility test results should be determined before you perform the indole test.

Some bacteria possess the ability to produce the enzyme tryptophanase, which hydrolyzes tryptophan. The end products of this hydrolyzation are indole, pyruvic acid, and ammonia, by way of deamination. The Kovac's reagent that you add to the SIM medium to test for indole contains hydrochloric acid, dimethylaminobenzaldehyde (DMABA), and n-amyl alcohol. DMABA reacts with indole to produce a red quinoidal compound.

If the reagent turns red, the indole test is positive (Fig. 11) (Cheesbrough, 1984).

3.8.4- Triple sugar iron agar (TSI):-

Is a differential medium that contains lactose, sucrose, a small amount of glucose (dextrose), ferrous sulfate, and the pH indicator phenol red. It is used to differentiate enterics based on the ability to reduce sulfur and ferment carbohydrates. As with the phenol red fermentation broths, if an organism can ferment any of the three sugars present in the medium, the medium will turn yellow. If an organism can only ferment dextrose, the small amount of dextrose in the medium is used by the organism within the first ten hours of incubation. After that time, the reaction that produced acid reverts in the aerobic areas of the slant, and the medium in those areas turns red, indicating alkaline conditions. The anaerobic areas of the slant, such as the butt, will not revert to an alkaline state, and they will remain yellow (Fig.12)

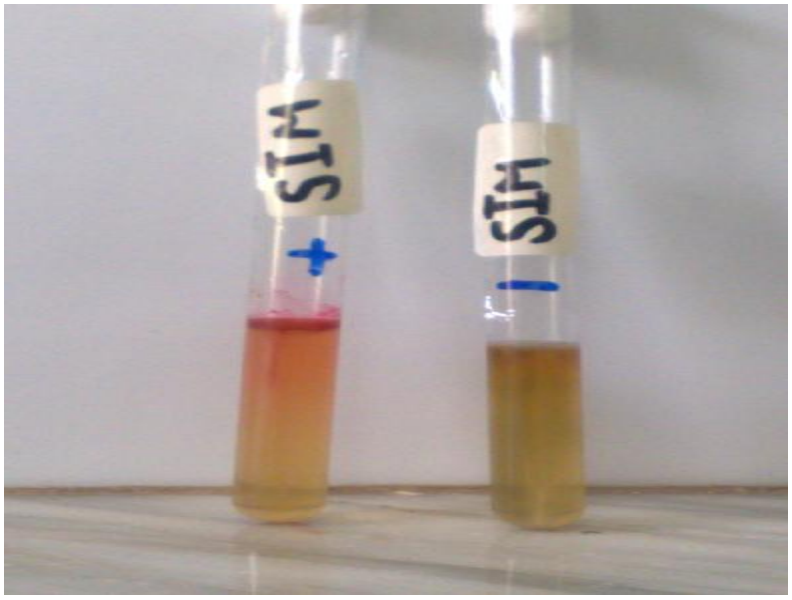

Figure (11): SIM test

Figure (12): TSI agar

3.9- Antibiotic sensitivity tests

All isolated organisms were tested against various antibiotics shown in table 3. Using the Kirby-Bauer method. About 3 to 5 colonies from each bacterial growth were striked aseptically onto Muller Hinton media, antibiotic discs were placed in a constant distances on the media, then the media were incubated at 37 C for 24 hours.

3.10- BD phoenix system

Some bacterial growths were confirmed by using BD Phoenix system in the Jomhoriya Laboratory, Benghazi (Fig.13).

3.11- Statistical analysis

Data were analyzed using statistical package for social science (spss) version 18. Descriptive statistics, as mean, standard deviation, median and mode were used. Inferential statistics were used when needed, as t-test to find the difference between the means of the two group, and Chi-square (2x) to find the difference in distribution of the variables between the two group, p-value were considered significant when ≤ 0.05.

Data were presented in form of tables and figures, were the figures done by Microsoft Excel 2003.

Table 3: The antibiotics used in sensitivity tests in study and their concentration:-

Antibiotic	Disk potency 10µg	Manu factory oxide Ltd,Englad,UK
Amikacin	30µg	oxide Ltd,Englad,UK
Ceftriaxone	30µg	oxide Ltd,Englad,UK
Augmentin	30µg	oxide Ltd,Englad,UK
Ciprofloxacin	5µg	oxide Ltd,Englad,UK
Gentamicin	10µg	oxide Ltd,Englad,UK
Amoxycillin	25µg	oxide Ltd,Englad,UK
Chloramphenicol	30µg	oxide Ltd,Englad,UK
Ampicillin	10µg	oxide Ltd,Englad,UK
Tetracycline	30µg	oxide Ltd,Englad,UK
Vancomycin	5µg	oxide Ltd,Englad,UK
Imipenem	10µg	oxide Ltd,Englad,UK
Erythromycin	15µg	oxide Ltd,Englad,UK
Penicillin G	10µg	oxide Ltd,Englad,UK

Figure (13): BD Phoenix

CHAPTER FOUR

4.1-Results

A total of three handured fifty one samples were examined during the period of this study . Positive cases samples showed 134 Gram positive bacteria and 87 Gram negative bacteria.

4.1.1- Identification of bacterial isolates

4.1.1.1- Identification of Staphylococcus spp.

This study showed that Staphylococcus was the most prevent bacterial wound infection isolated. It produces circular and smooth colonies on blood agar, nutrient agar. All samples examined microscopically and showed gram positive cocci and the production catalase positive (Fig.14) Presence of *S. aureus* was confirmed by the coagulase positive (Fig.15). S. aureus gave orange colonies on MacConkey agar (fig.16) Where as others staphylococci are coagulase negative *S.epidermidis ,S.hominisand* and *S.haemolyticus* growth was confirmed by using BD phoenix .

Figure (14): Catalase positive.

Figure (15): Coagulase positive.

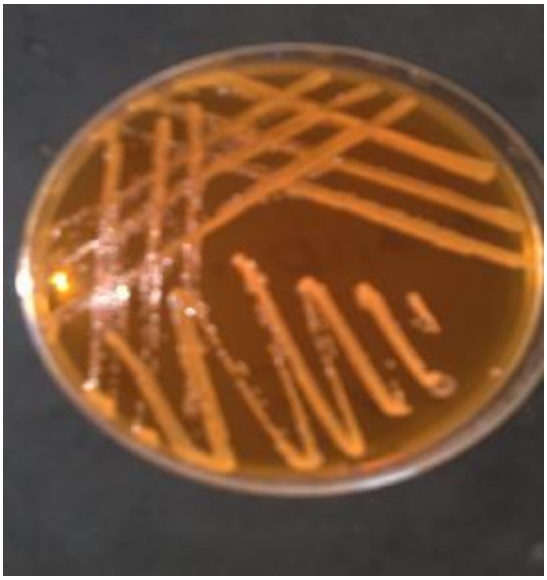

Figure (16): Growth of ***Staphylococcus aureus*** on macConkey agar

4.1.1.2- Identification of *pseudomonas aeruginosa:*

In this study showed that ***p. aeruginosa*** was Gram negative rods. It produces large, flat hemolytic colonies on blood agar.

Also grows well on nutrient agar and macconkey agar, produces the yellow green pyocyanin pigment (Fig.17) and positive oxidase (Fig.18). ***P.aeruginosa*** growth was confirmed using biochemically tests (Fig.19) or by BD phoenix system.

4.1.1.3- Identification of *Klebsiella pneumonia*

Klebsilla sp. was appeared in some cases in this study. However Klebsilla sp. gave lactose fermenting mucoid pink colonies on MacConkey agar (Fig. 20) , and with biochemical reactions it produced negative oxidase test, negative indol test and positive urease test (Fig. 21). All samples examined microscopically and showed gram negative rods. Other ***K. pneumonia*** confirmed by using BD phoenix system.

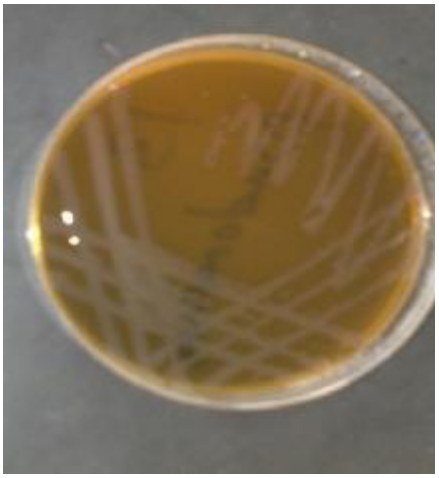

Figure (17): Growth of *P.aeruginosa* On MacConkey agar.

Figure (18): Oxidase positive.

Figure (19): Biochemical test of *P.aeruginosa*

Figure (20): Growth of *K. pneumonia* on MacConkey agar

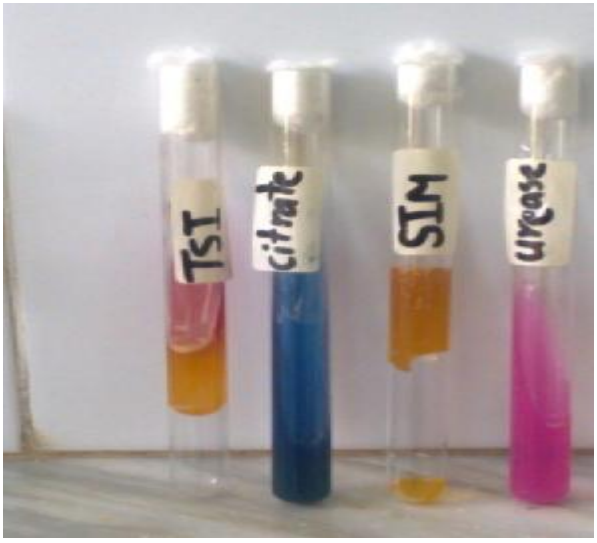

Figure (21): Biochemical test of *K. pneumonia*

4.1.1.4- Identification of *E.coli*:

It produces smooth pink colonies on MacConkey agar. All samples examined gave lactose fermenting colonies on MacConkey agar (Fig.22). All samples examined microscopically and showed Gram negative rods *E.coli* growths were confirmed biochemically (Fig. 23) when it produce positive Indol test, a negative oxidase test and negative urease test. *E.coli* growth was confirmed by using BD phoenix system.

4.1.1.5- Identification of *Proteus mirabilis*

Proteus sp. was produced swarming growth on blood agar (Fig. 24). Microscopic examination of positive cultures showed Gram negative rods. Confirmed biochemically by producing negative oxidase test, positive indol test, positive urease test after four hours incubation at 37^{o} C (Fig. 25) and confirmed by using BD phoenix system.

Figure (22): Growth of *E. coli* on MacConkey agar

Figure (23): Biochemical test of *E.coli*

Figure (24): Growth of *P. mirabilis* on blood agar

Figure (25): Biochemical test of *P. mirabilis*

4.1.1.6- Identification Streptococcus spp.

Only twelve streptococcus spp. Was isolated in this study Gram positive streptococci .Streptococci growth were confirmed biochemically when produce negative catalase test.

This study showed those *S.viridans* Alph-haemolytic on chocolate agar resistance Optechen (Fig. 26), while *S. agalactiae* Beta-haemolytic on blood agar resistance Bactracin (Fig. 27).

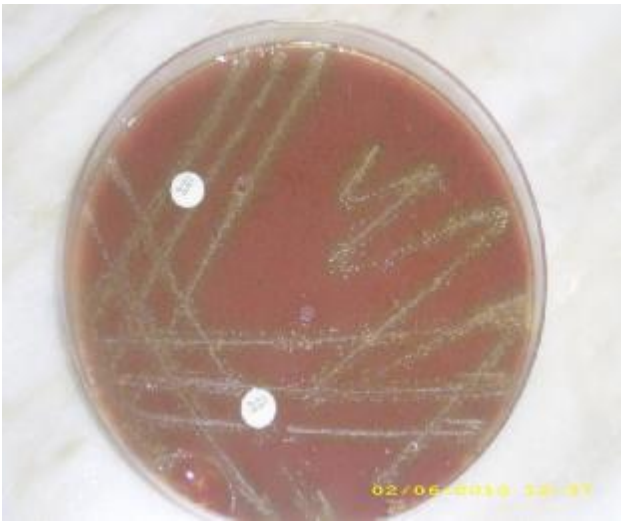

Figure (26): Growth of S. viridians on chocolate agar (Optchin resistant)

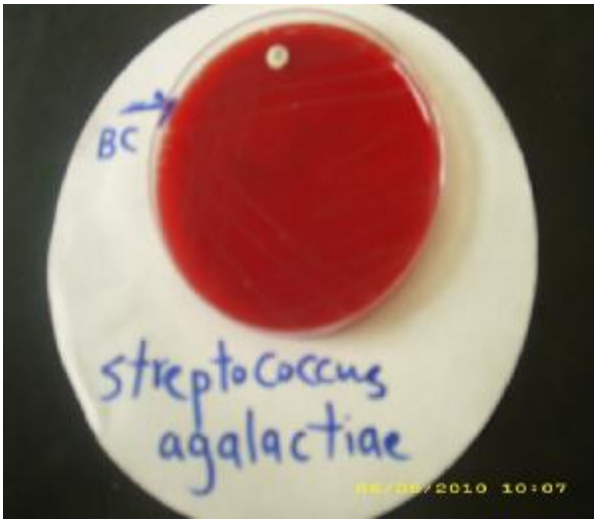

Figure (27): Growth of *S. agalactiae* on blood agar (bacitracin resistant)

4.1.2- Distribution of patients

4.1.2.1- Distribution of patients According to bacterial growth and nationality:

Table 4 showed that the wound infection reach to 62.9% (214 of 341 patients) among Libyan nationality ,while the wound infection among non Libyan patient reach up to the 70% (7 of 10 patients), (Fig.28) .

4.1.2.2- Distribution of patients According to bacterial growth and residence:

This study Showed that SSI of in Benghazi residence was 59.5% (169 0f 284) while the SSI reach to 77.6% (52 of 67) among resident outside of Benghazi city(Table 5) (Fig. 29)

Table4: Distribution of patients according to bacterial growth and nationality.

Nationality	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
Libyan	214	62.9	127	37.2	341	100
Non-Libyan	7	70	3	30	10	100
Total	221	63	130	37	351	100

X^2 = 0.219 df=1 p= 0.640(Not Significant difference

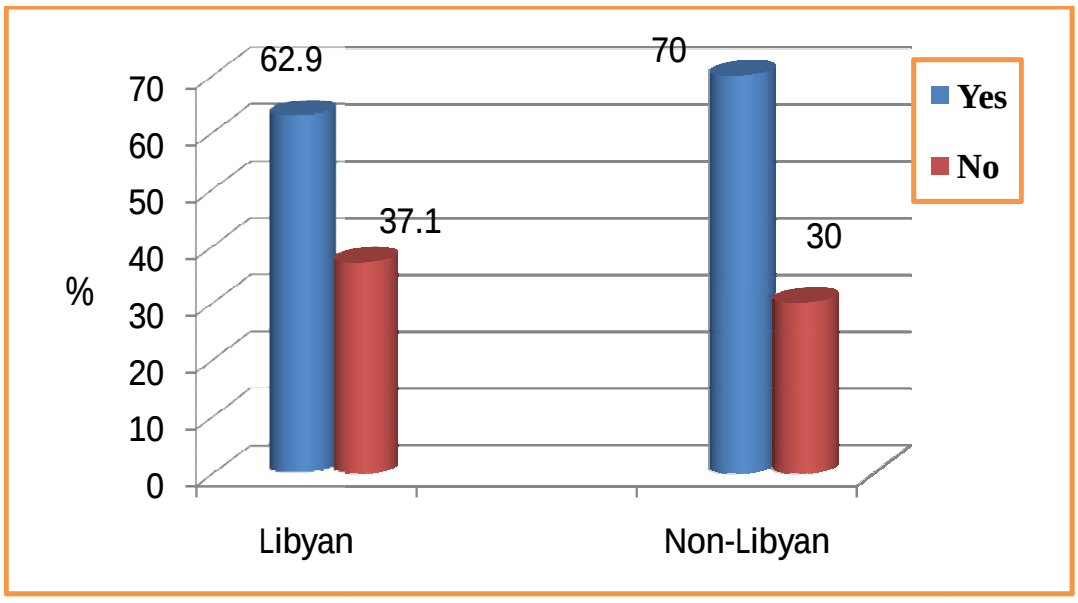

Figure (28): Distribution of patients according to bacterial growth and nationality.

Table5: Distribution of patients according to bacterial growth and residence.

Residence	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
Benghazi	169	59.5	115	40.5	284	100
Outside-Benghazi	52	77.6	15	22.4	67	100
Total	221	63	130	37	351	100

X^2 = 7.620 df=1 p= 0.006(Significant difference)

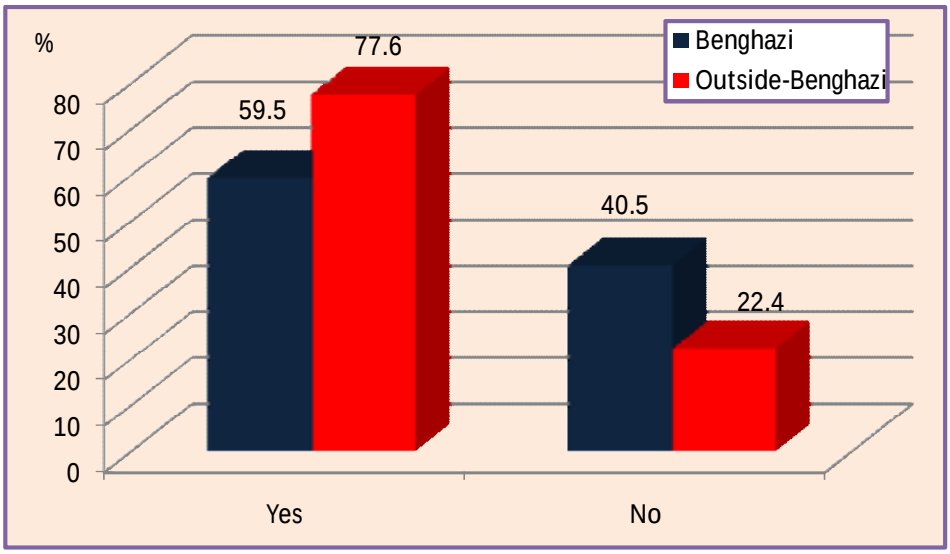

Figure (29): Distribution of patients according to bacterial growth and residence.

4.1.2.3- Distribution of patients According to bacterial growth and age:

Table 6, Fig.30 showed the relationship between bacterial growth and the age of the patients. The age groups were divided in to six categories : equal to or less 20 years, 21-30, 31-40, 41-51, 51-60 years and above .The results showed that the highest overall infection rate was in the age range from 51-60 years (80%), while the lowest range was in the age group of 60 years and above (50%).Were infected rate in the age range from equal to or less 20 years (61.5%), rate in the age range from 21-30 years (61.3%), rate in the age range from 31-40 years (66.2%) and rate in the age range from 41-50 years (62.5%).

4.1.2.4- Distribution of patients According to bacterial growth and duration of hospital stay:

Duration of hospital stay was also studied. Table 7, Fig. 31 the percent of infected wound in patients stayed for one week reach to 61%, wherever the infected wound reach to 73.1% in patients stayed for 14 days. However the percent of infected wound increased by the increase of duration hospital stay which react after three weeks to 80%. While the infected wound reach

Table6: Distribution of patients according to bacterial growth and age.

Age / year	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
≤ 20	5	61.5	8	38.5	13	100
21 – 30	76	61.3	48	38.7	124	100
31 – 40	104	66.2	53	33.8	157	100
41 – 50	30	62.5	18	37.5	48	100
51 – 60	4	80	1	20	5	100
>60	2	50	2	50	4	100
Total	221	63	130	37	351	100

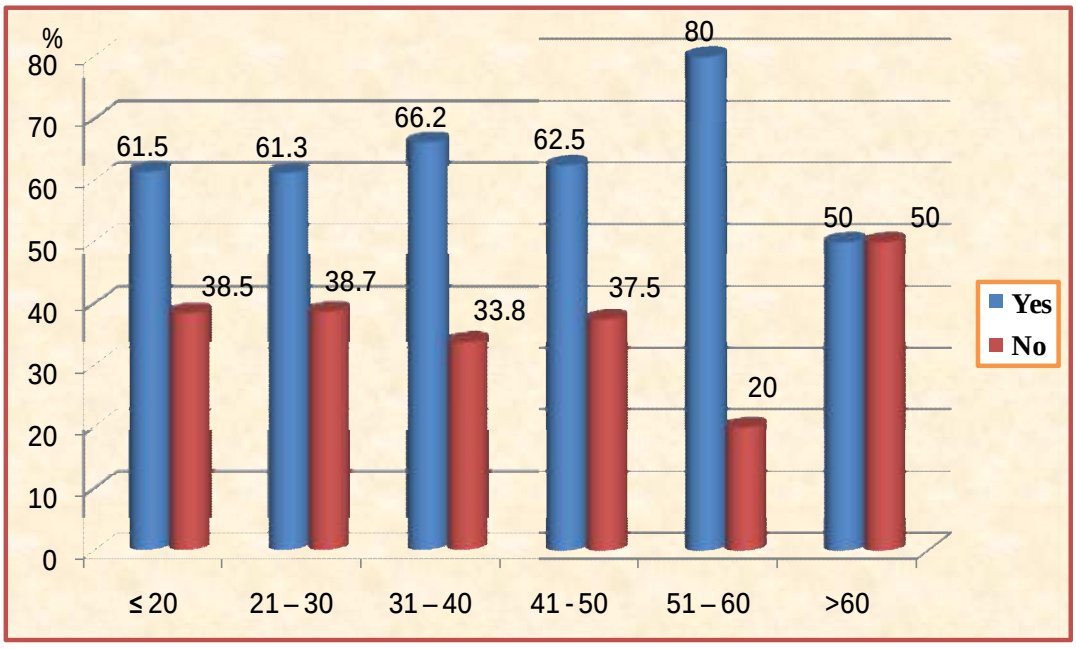

Figure (30): Distribution of patients according to bacterial growth and age.

Table7: Distribution of patients according to bacterial growth and duration of hospital stay.

Duration of hospital stay/days	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
1 – 7	177	61	113	39	290	100
8 – 14	38	73.1	14	26.9	52	100
15- 21	4	80	1	20	5	100
22 – 28	1	50	1	50	2	100
≥ 29	1	50	1	50	2	100
Total	221	63	130	37	351	100

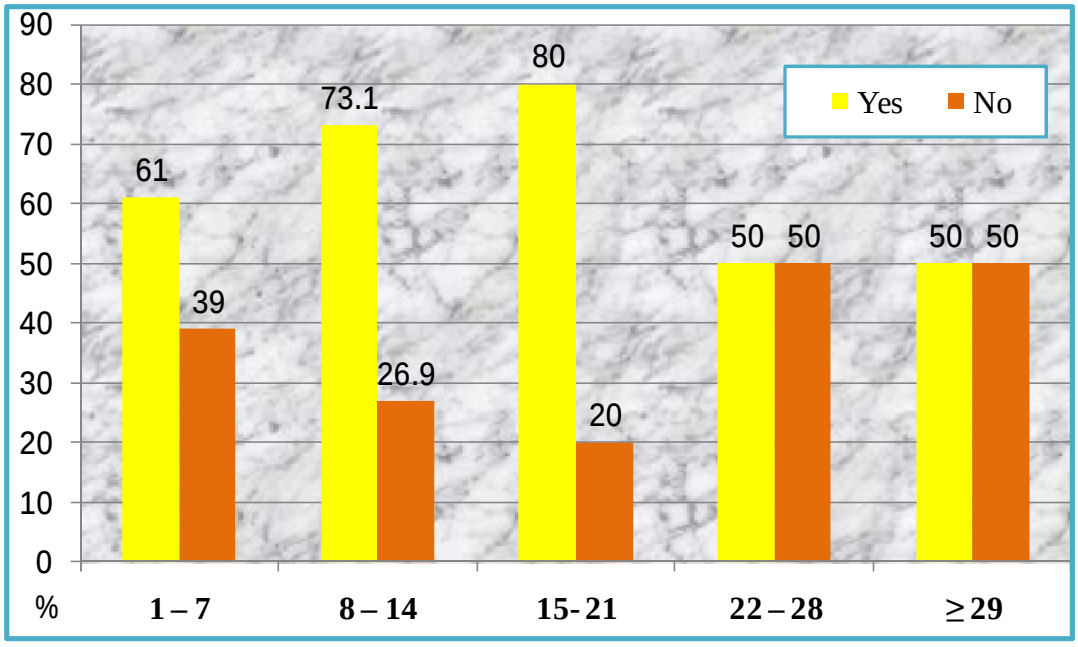

Figure (31): Distribution of patients according to bacterial growth and duration of hospital stay.

to 50% in patients stayed for 22-28 days, this rate was similar with equal to or more than 29 days.

4.1.2.5- Distribution of patients According to bacterial growth and body weight:

Table 8. showed that one patient, 1 (100%) developed in SSIs, her body weight range from 90 kg and above followed by 78.7% (37 of 47 patients) their bodies weight range from 81-90 kg, 74.6% (88 of 118 patients) their bodies weight range from 71-80 kg, 62.5% (80 of 128 patients) their bodies weight range from 61-70 kg. However only two patients their body weight range equal to or less 50kg, had SSIs reach to 50%. While the lowest SSIs were in the body weight range from 51-60 kg, the infection rate reach to 25.5% (14 of 50 patients).

4.1.2.6- Distribution of patients According to bacterial growth and other health problems:

Seventy of 351 patients health problems during this period of study, while two hundred eighty one had no health problems. Table 9 showed that the incidence of SSIs in thyroid diseases reach to 100% (6 of 6), 84.6% in patients with diabetic, 82.9% in patients with hypertensive and in anemic patients the SSI reach to 75% .

Table8: Distribution of patients according to bacterial growth and body weight.

Body weight/kg	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
≤ 50	1	50	1	50	2	00
51 – 60	14	25.5	41	74.5	55	100
61 – 70	80	62.5	48	37.5	128	100
71 – 80	88	74.6	30	25.4	118	100
81- 90	37	78.7	10	21.3	47	100
>90	1	100	0	0	1	100
Total	221	63	130	37	351	100

Table 9: Distribution of patients according to bacterial growth and other health problems.

Other health problems	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
No other health problem	163	58	118	42	281	100
Hypertension	29	82.9	6	17.1	35	100
Diabetic	11	84.6	2	15.4	13	100
Thyroid disease	6	100	0	0	6	100
Anemia	12	75	4	25	16	100
Total	221	63	130	37	351	100

X^2 = 16.037 df=4 p= 0.003(Significant difference)

4.1.2.7- Distribution of patients According to bacterial growth and of condition of the wounds:

Table 10, Fig 32 showed the condition of the wounds included in this study which classified as, clean wounds, contaminated wounds and dirty wounds. Dirty wound was observed in sixty eight patients and the infection rate reach to 98.5%. However contaminated wounds observed in 93 patients and the infection rate reach to 95.7%. The rate of clean wounds was higher among the patients and become infected during this study reach to 34.2%.

4.1.2.8- Distribution of patients According to bacterial growth and type of operation:

There were 351 major operations during the survey, of which 293 were caesarean sections and 58 were gynecological operation, of these 221 cases had wounds infection. The high rate of infection was observed in caesarean sections reach to 67.6%. Followed by hysterectomy 61.9% (13 of 21 patients), myomectomy 26.9% (7 of 26 patients), ectopic pregnancy 22.2% (2 of 9 patients). However only one patient lapratomy had infected and one patient of cystectomy was reported with no bacterial growth (table11).

Table10: Distribution of patients according to bacterial growth and of condition of the wounds.

Condition of the wound	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
Contaminated	89	95.7	4	4.3	93	100
Clean	65	34.2	125	65.8	190	100
Dirty	67	98.5	1	1.5	68	100
Total	221	63	130	37	351	100

$X^2 = 146.98$ df=2 p= 0.000(Significant difference)

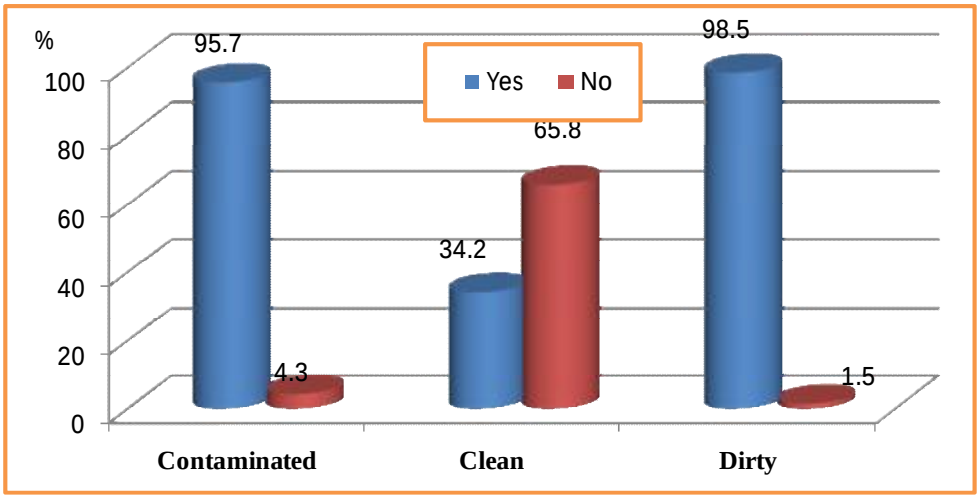

Figure (32): Distribution of patients according to bacterial growth and of condition of the wounds

Table 11: Distribution of patients according to bacterial growth and type of operation.

Type of operation	Bacterial growth					
	Yes		No		Total	
	No.	%	No.	%	No.	%
C/S	198	67.6	95	32.4	293	100
Hysterectomy	13	61.9	8	38.1	21	100
Myomectomy	7	26.9	19	73.1	26	100
Ectopic pregnancy	2	22.2	7	77.9	9	100
Cystectomy	0	0	1	100	1	10
Lapratomy	1	100	0	0	1	100
Total	221	63	130	37	351	100

4.1.2.9- Distribution of bacteria isolated from the environment:

Table (12) showed that the *S.aureus* was the most bacteria isolated from the air, bed lines ,floor. Sporing bacilli were prevalent in different areas of the inanimate environment, *S.epidermidis* was isolated from the O.T air conditione, O.T air room, wall and rooms floor of the unit. However *Acinetobacter baumanni* was isolated from the O.T table, suceton baby machine, rooms floor and rooms air of the unit.

4.1.2.10- Distribution of patients According to type of bacteria isolated from infected wounds:

Table13 showed that the most causative agent of post operation infections was *S.aureus* 70 isolates (19.9%) , following *S.epidermidis* 37 isolates (10.7%), *Klebisella pneumonia* 33 isolates (9.4%), *Acinetobacter baumanni* 21 isolates (6%), *E. coli* 14 isolates (4%), *S. hominis* 12 isolates (3.4%), *P.aeruginosa* 11 isolates (3.1%), *Streptococcus agalactiae* 11 isolates(3.1%), *Proteus mirabilis* 8 isolates (2.3%) and *S.haemolyticus* 3 isolates (0.9%). The lowest causative agents of post operation infections were *streptococcus viridans* 1 isolates (0.3%) (Table13) (Fig.33).

Table12: The bacteria isolated from the unit inanimate environment :-

Sampled location	**organism isolated**
Unit O.T	
O.T table	*Acinetobacter baumanni*
Anesthesia machine	No growth
Anesthesia mask	No growth
O.T air condiationer	*S.epidermidis*
O.T air room	*S.epidermidis*
Disinfection liquid used	No growth
O.T room floor	*S.aureus*
O.T bed	No growth
Sucetion baby machine	*Acinetobacter baumanni*
Ward	
Wall corner	*Bacillus spores*
Disinfectant liquids used	No growth
Bathroom (toilet seat)	*Bacillus spores ,S.aureus,S.epidermides*
Bed line	*S.aureus*
Walls	*Bacillus spores,S.epidermides*
Holes	*Bacillus spores*
Rooms air	*S.aureus , Acinetobacter baumanni*
Rooms floor	*S.aureus ,S.epidermides, Acinetobacter ssp.*

Table 13: Distribution of patients according to type of bacteria isolated from infected wound.

Type of bacteria	No.	%
E-Coli	**14**	**4**
S.hominis	**12**	**3.4**
Acinetobacter baumanni	**21**	**6**
S.epidermidis	**37**	**10.5**
Streptococcus agalactiae	**11**	**3.1**
Staph aureus	**70**	**19.9**
Klebsella pneumonia	**33**	**9.4**
Streptococcus viridians	**1**	**0.3**
S. haemolyticus	**3**	**0.9**
Proteus Mirabilis	**8**	**2.3**
Pseudomonas aeruginosa	**11**	**3.1**

Percentage from 351.

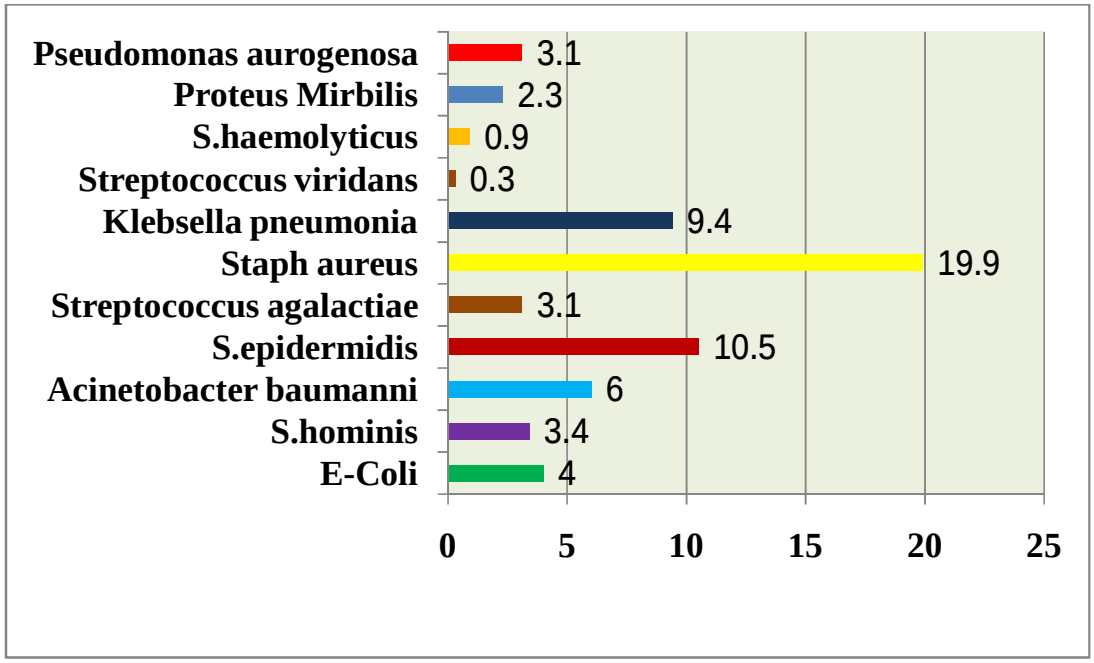

Figure (33): Distribution of patients according to type of bacteria isolated from infected wound.

4.1.2.11- Distribution of patients according to susceptibility of antibiotics.

Table (14) showed the sensitivity pattern of bacterial isolated from patients in post operation wound infections to different antibiotic used in Jomhoriya hospital –Benghazi (Fig.34 and Fig. 35). The antibacterial effect of ciprofloxacin reach to (69.2%) followed imipenem (57.1%), gentamicin (55.1%), amikacin (32.6%), chlorophenicol (31.7%), ceftrixone (21.6%), Augmentin (12.1%), vancomycin (10.9%), tetracycline (10%), erythromycin (8.4%), colstin sulphate (1.8%), amoxicillin (1.7%), penicillin (1.1%), cephaloxin (0,8%) and Ampicillin (0.5%), ciprofloxacin showed the highest activity against most bacteria isolated in this study.

Table: 14 Distribution of patients according to susceptibility of antibiotics.

Susceptibility of antibiotics	Sensitive		Resistance		Intermediate		Total	
	No.	%	No.	%	No.	%	No.	%
Ampicillin	1	0.5	190	94.5	10	5	201	100
Amoxicillin	3	1.7	169	90.8	14	7.5	186	100
Augmantin	25	12.1	138	66.7	44	21.3	207	100
Chlorophenicol	45	31.7	66	46.5	31	21.8	142	100
Ciprofloxicin	146	69.2	29	13.7	36	17.1	211	100
Tetracycllin	18	10	124	68.9	38	21.1	180	100
Gentamycin	114	55.1	41	19.8	52	25.1	207	100
Ceftrixone	43	21.6	101	50.8	55	27.6	199	100
Amikacin	60	32.6	45	24.5	79	42.9	184	100
Colstin sulphat	2	1.8	97	89	10	9.2	109	100
vacomycin	11	10.9	61	60.4	29	28.7	101	100
Pencillin	1	1.1	87	94.6	4	4.3	92	100
Impereum	52	57.1	18	19.8	21	23.1	91	100
Cephaloxin	1	0.8	119	92.2	9	7	129	100
Erythromycin	9	8.4	88	82.2	10	9.4	107	100

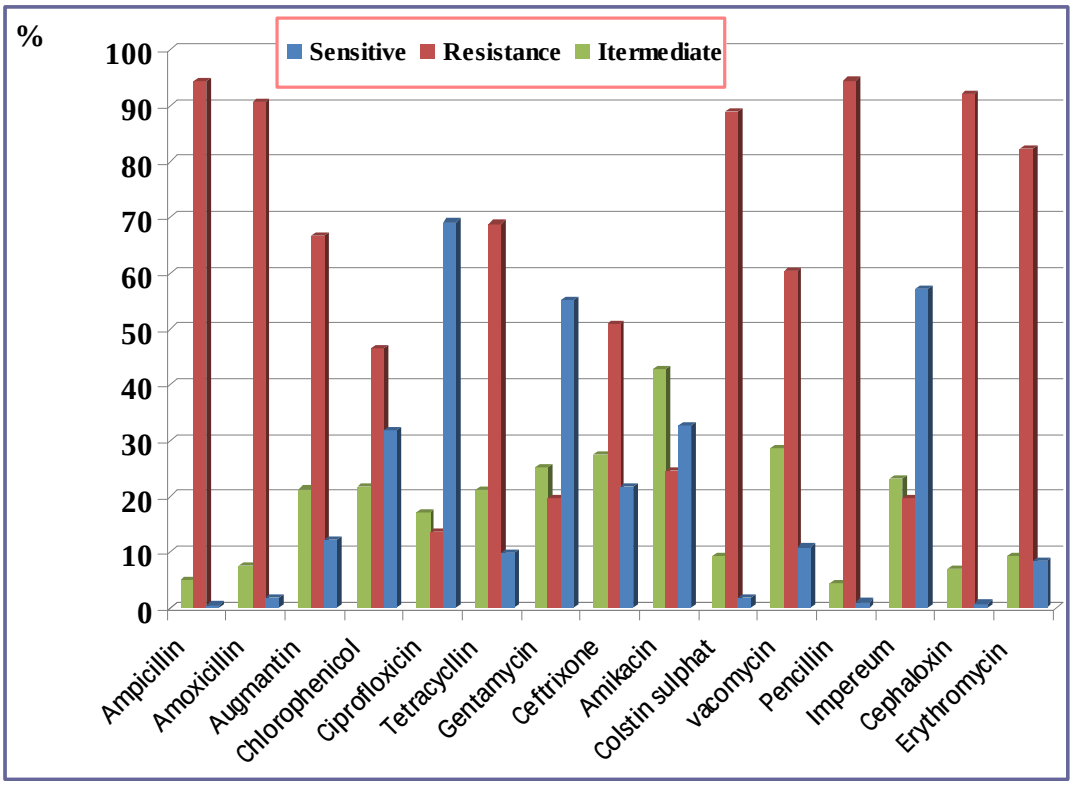

Figure (34): Distribution of patients according to susceptibility of antibiotics.

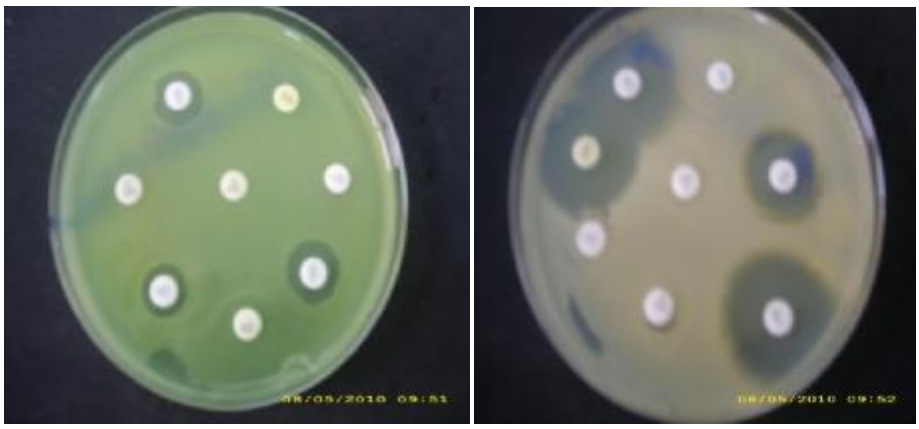

Figure (35): Susceptibility test

4.2- Discussion

Surgical wound infection is a common post operative complication and causes significant post operative morbidity and mortality, prolongs hospital stay and adds between 10% and 20% to hospital costs .Although the total elimination of wound infection is not possible, a reduction in the infection rate to minimal level could have significant benefits in terms of both patient comfort and medical resources used (Nandi *et al.*,1999).

This study showed that the overall infection rate was slightly higher among Libyan than and non Libyan patients. Also this study showed that SSI in Benghazi residence was lower compared with of resident outside of Benghazi city which reach to77.6%. The increased rate of infection among patients out of Benghazi may be due to different of environmental condition and lifestyle.

The overall wound infection rate reach to 63% among the patients during this period of study. Similar result was observed by Thu *et al.,* (2006) who reported that the wound infection rate reach to (68.8%), while the rate of infection in this study was relatively high compared to the infection reported by Johnson *et al.*(11.2%), *Chia et al*., (2.26%), *Banjara et al.,* (4.7%) and Jamulitrat *et al*. (6.5%). This discrepancy may be due to

many factors such as patient and hospital characteristics, criteria used for diagnosing and method of survey. The factor that influence wound infection includes host factors and agent factors. Host factors are the resistance of host to infection. These include local and systemic resistance of host. Agent factors include dose of bacterial contamination and pathogencity.

This study showed that increasing age was slightly identified as a risk specific to this category of operations. Similar study was observed by Masaadeh and Jaran (2009) who reported that the overall infection rate was slightly influence by age. In contrast Johnson *et al.,* (2006) and Mishriki *et al.,* (1990) reported that increasing of the patients age increase the risk of surgical sit infection (SSI).

The rate of bacterial infection increased with the in duration of stay in the hospital. Similar results were observed by Garibaldi *et al.*, (1991) and Sawyer and Pruett, (1994) who found that the rate infection increased by the duration of stay in the hospital. Also this study showed that obesity increased the risk of SSI among the patients which reach to 78.7%. Similar studies have suggested that obesity may increase the risk of SSI (Moir-Bussy *et al*., 1985, Pelle *et al.,* 1986 and Johnson *et al.*, 2006).

The rate of bacterial infection increased in the patients with different health problems. Thyroid and diabetic patients possessed the high rate of infection reach to 84.6% and 100% respectively. Similar results were observed by (Garcia *et al*., 1997, Johnson *et al*., 2006 and Ward *et al*., 2008).

Also Liaeo *et al*., *(*2006) and Hjortrup *et al*., (1985) reported that diabetic patients possess the high risk of wound infection. The high risk of diabetic patients may be due to the:

Macro vascular and micro vascular disease. Plaque easily forms in the circulatory systems of patients with macro vascular disease producing a high carriage rate of organisms. In patient with microangiopathy, subsequent decreased nutrition and oxygen delivery to peripheral tissue can reduce the body's ability to resist infection (Giardino *et al*.1997). However reported that poor blood sugar control will impair the leukocyt's ability for chemotaxis, adherence, phagocytosis and intracellular elimination of microorganism (Mowat and Baum, 1971, Delamaire *et al*., 1997 and Sima *et al*., 1988). Suggested that Twigg *et al*., 2001 in diabetic patients, delayed wound healing is a result of defective fibroblast proliferation and impaired synthesis of collagen.

This study showed that the wound infection rate for dirty wounds (98.5%) was more than clean wounds(34.2%) similar results were observed by Whyte *et al.,* (1991), Haley *et al.,* (1985), Garcia *et al.,* (1997), Tourmousogbs *et al.,* (2008) and Jamulitrat *et al*., (1988), they reported that the dirty wound and contamination wound strongly predispose to wound infection.

The rate of infection in caesarean sections was higher compared to gynaecology. This may be due to as the majority of the former were emergency caesarean sections which involved higher risk of contamination from prolonged labour.

S. aureus was the predominant bacteria isolated in this study. The percent of *S.aureus* reach to 19.9% any all bacteria isolated. Similarly Chia *et al*., (1993) reported that *S.aureus* was the most common organism isolated from (*POWI*) (58%). In addition study of Banjara *etal*., (2002) showed that the highest infection rate was *S.aureus* (24.9%). Another study by Giacometti *et al., (*2000) reported that *S.aureus* remain the most important Bacteria responsible for postoperative wound infection (28.2%).

S.aureus a common nosocomial contaminant and epidemics have been traced to many items in the hospital environment. Patients who are

hospitalized for extended periods are frequently colonized by this organism and are at increased risk of developing infection. The most serious infections include malignant, external otitis, pneumonia, septicemia and endocarditis, as well as an important cause of surgical wound infection.

S. epidermidis the secondary bacteria cause wound infection reach to 10.5% flowed *K. pneumonia*. However Bakir *et al*., (2004) reported that the commonest causative organism is were coagulase- negative staphylococci (21.7%). In contrast Anupurba *et al*., (2006) reported that prevalence rate *P. aeruginosa* was (32%) of all the pathogens isolated form POWI. Similarly Masaadeh and Jaran, (2009) recovered *P. aeruginosa* in POWI (27.8%). The occurrence *p. aeruginosa* was higher in young groups than in the other groups (29.5%) (Thu *etal.,* 2006) .

S.aureus was found in 19.9%of patients, also found in air, bed lines, floor and bathroom (toilet seat). This may indicated that the source of *S. aureus* infection in this unit is more likely to be of environmental source. The environmental source for this organism was identified taking multiple environmental swabs. Often contemporaneously with their isolation from a patient, thus confirming the close relationship between environment and patient particularly in such areas of very high infective risk. This agrees the

microbial flora colonizing wounds and their resistance patterns to selected locally available topical systemic agents.

All species were tested for susceptibility to antibiotics, using the disc diffusion method and BD phoenix system. Study of sensitivity test to antibiotics showed that all bacteria were sensitive (69.2%) to ciprofloxacin, (32.6%) to Amikacine, (55.1%) to Gentamycin, (57.1%) to Imipenem. Similar study represented that all bacterial growth were sensitive to ciprofloxacin, Gentamycin and Amikacin (Anupurba *et al*., 2006 and Masaadeh and Jaran, 2009)

Our study showed high resistance rate to Ampicillin, Amoxicillin, pencillin, cephaloxin and colstine sulphate, the phenomena which may contributed to the frequent and miss use of the antibiotic without medical prescription .This result was in agreement with that reported by Gold *et al*., (1996), Janda *et al.,* (1997) and Marc and Struelens, (1998) .

Multiple drug resistant (MDR) bacterial infections are being increasingly reported from all parts of the world. Multi resistant microbes are an important cause of hospital-acquired infection.

Infections associated with such organisms can pose a serious threat to vulnerable patients.

Generally make frequent use of antimicrobial agents, resulting in great likelihood of resistance and multi drug resistance.

4.3- Conclusion

1- This study shows that there is an increased rate of incidence of bacteria in postoperative wound infection.

2- The most causative agent of post operation infection were *S.aureus*, followed by *S.epidermidis, klebsiella pneumonia, Acinetobacter baumanni, E.coli, S. hominis, pseudomona aurogenosa, Streptococcus agalactiae, proteus mirbilis, S.haemolyticus and streptococcus viridians*. This is in agreement with survey studies carried out in various hospitals.

3- The infection appears to be common in hospitals with relaxed hygienic measures and is dependent on obesity, metabolic diseases, wound class, pre-existing illness and duration of stay in the hospital.

4- The reason for this increase in postoperative infection rate with prolonged preoperative hospitalization is primarily due to colonization of patients with hospital-acquired resistance microorganism. The development of multi drug resistance may be reduced by appropriate pre and postoperative antibiotic therapy.

5- The inappropriate usage of antimicrobials in surgical preoperative prophylaxis is still a problem and a close collaboration between surgeons and microbiologists is needed.

6- On the basis of our results, antimicrobial agents or drug combinations with wider spectra of activity and stronger resistance to enzymatic degradation are desirable for per operative prophylaxis or treatment of surgical infection.

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APPENDIX I

Questionnaire form

No. of patient: No. of file:

Name of patient: Age:

Nationality:

Residence:

Type of operation:

Duration of hospital stay:

Obesity (weight):

Wound classification:

Other health problem:

Bacterial isolated from infected wound:

Susceptibility of antibiotic:

APPENDIX II

Blood agar Base (Oxoide CM55, England)

Formula	gram per litre
'Lab-lemco' poder	10.0 g/L
Pepton	10.0 g/L
Sodium Chloride	5.0g/L
Agar	15.0 g/L

DIRECTIONS

Suspend 40g in 1litre of distilled water. Bring to the boil to dissolve completely. Sterilize by autoclaving at 121^0C for 15min. Cool to 45-50^0C for blood agar add 7% sterile defibrinated blood.

Macconkey agar without salt (Oxoid CM 0507, England)

Formula	gram per litre
Peptone	20.0 g/L
Lactose	10.0 g/L
Bile salts	5.0 g/L
Neutral red	0.075 g/L
Agar	12.0g/L

DIRECTION:-

Suspend 47g in litre of distilled water. Bring to the boil to dissolve completely. Sterilize by autoclaving at 121^0C for 15minutes mix wall before pouring. Dry surface of the gel before inoculation.

Muller Hinton Agar (M.H.A) (Oxoid –Basing stoke, UK)

Formula	gram per litre
Meat infusion	6.0g/L
Casein Hydrolyte	17.og/L
Starch	1.5 g/L
Agar No.1	10.0 g/L

DIRECTION:-

The medium was mad by dissolving 38g in one litre of distilled water and sterilized by autoclaving at 121^0C for 15 minute. The sterilized medium was poured in to 90mm diameter sterile petri dishes to a depth of 4mm(about 25ml per plate).

Simmons citrate Agar (oxoid CM 0155, England)

Formula	gram per litre
Magnesium	0.2g/L
Ammonium dihydrogen phosphate	0.2g/L
Sodium ammonium phosphate	0.8g/L
Sodium citrate, tribsic	2.0g/L
Sodium chloride	5.0g/L
Bromothymol blue	0.08g/L
Agar	15.0g/L

Direction:-

Suspend 23g in1 litre of distilled water. Bring to the boil to dissolve completely. Sterilize by autoclaving at 121^0C for 15minutes.

Urea Agar Base (LAB.MTM) LAB130

Formula	gram per litre
Pepton	1.0g/L
Glucose	1.0g/L
Sodium chloride	5.0g/L
Disodium phosphate	1.2g/L
Potassium dihydrogen phosphate	0.8g/L
Phenol red	0.012g/L
Agar No.1	15.0g/L

Directions :-

Weight 2.1grams of powder, dispense in 95ml of distilled water. Allow to soak for 10minutes, swirl to mix, then sterilise by autoclaving at 121^0C for15minutes. Allow to cool to 47^0C,add 5ml sterile urea bottle and distribute in to sterile bottles and allow to set in the sloped position.

Sulfide Indole Motility (S.I.M) (Oxoid CM 0435, England)

Formula	gram per litre
Magnesium sulphate	0.2g/L
Ammonium dihydrogen phosphate	0.2g/L
Sodium ammonium phosphate	0.8 g/L
Sodium citrate ,tribsic	2.0g/L
Sodium chloride	5.0 g/L
Bromothymol blue	0.08 g/L
Agar	15.0g/L

Direction:-

Suspend 30g in1 litre of distilled water and boil to dissolve the medium completely. Dispense in to final containers and Sterilize by autoclaving at 121^0C for 15min.

Triple sugar Iron Agar LAB 53 (LAB.MTM)

Formula	gram per litre
Beef Extract	3.0 g/L
Yeast Extract	3.0
Balanced peptone No.1	20.0
Sodium chloride	5.0
Lactose	10.0
Glucose	1.0
Ferric citrate	0 .3
Sodium thiosulphate	0.3
Phenol red	0.025
Agar No.2	12.0

Direction:-

Weight 65grams of powder ,dispersal in 1litre of distilled water.

Allow to shoak for 10 minute, swirl to max then bring to the boil with frequent swirling.

Distribute in to tube and sterilize at 121^0C For 15minute.

Allow to set as aslope nsuring.

Oxidase reagent:

Contents: to make 10ml

Tetramethyl-p-phenylnendiamine dihydrochloride	0.1g
Distilled water	10 ml

Preparation:

The chemical was dissolved in the water and the reagent was used immediately.

Kovacs indol reagent:

Contents: to make about 40 ml

4-Dimethylaminobenzaldhyde	2g
Isoamyl alcohol	30ml
Hydrochloricacid,concentrated	10 ml

Preparation:

(1) The 4-Dimethylaminobenzaldhyde was dissolved in the Isoamyl alcohol.

(2) The concentrated Hydrochloric acid was then added and mixed well.

ملخص البحث

الهدف الاساسى من هذ ه الدراسة كان تحديد البكتيريا المنتشرة في عدوى الجروح بعد العمليات وحساسيتها للمضادات الحيوية الشائعة الاستعمال . خلال فترة أربعة أشهر بين ابريل الي يوليو 2010 . تم فحص الجروح بزراعة كل الجروح المشتبه فيها باستخدام طرق بكتريولوجية قياسية. من مجموع 351 عينة جمعت من العمليات القيصريه وامراض النساء بمستشفي الجمهورية – بنغازي \ ليبيا ، 221 عينة اصبحت ملوثه. أيضا شملت الدر اسة فحص البيئة المحيطة بالمريض وذلك باخذ عينات من مختلف المناطق المحيطة بالمريض.

كانت أكثر الجراثيم انتشارا بين المرضى و ذلك بنسبة (19.9%)، *Staphylococcus aureus* (9.4%)، *Klebsiella pneumonia* (10.6%)، *Staphalococcus epidrmidis* يلى ذلك *Acinetobacter baumanni* (6%)، E.coli (4%)، *pseudomona aeuroginosa* (3.1%)، *Streptococcus agalactia* (3.1%)، *proteus mirbilis* (2.3%)، *Staphylococcus haemolyticus* (0.9%)، *Strptococcus Viridans* (0.3%). في هذه الدراسة كانت كل الانواع المعزولة حساسة للسيبروفلوكساسين، جنتاميسين، امبينيم، اميكاسين والكلووفينوكول. وكان سيبروفلوكساسين أ علي نسبة حساسية بينما امبيسلين وبنسلين وآموكسلين وسيفالوكسين والكولوستين أ قل حساسية. اوضحت النتائج أن العوامل الم تعلقة بعدوى الجروح تشمل السمنة ،حالة المريض قبل العملية ، مدة مكوث المريض في المستشفي،الأمراض الميتابولزمية، درجة تلوث الجرح.

كلية العلوم

قسم النبات

العدوى البكتيرية لجروح العمليات القيصرية وأمراض النساء في مستشفى الجمهورية- بنغازي / ليبيا

اعداد

منى نوح عبدالله البدري

إشراف

أ.د.صالحة فرج بن جويرف

مشروع بحث مقدم كجزء من متطلبات الحصول على درجة الماجستير في علم النبات

خريف (2011- 2012 ف)