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Isolation and identification of bacteria from burn wound infection in Burn and plastic sergery Hospital in Tripoli

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Dedication

To my greatfather , who did not spare on some days and to mygreat mother give me all tenderness and love .

To my dear husband who supported me true my life

To my sisters and brothers

To everyone who learn me a letter.

Acknowledgement

First of all, I give my thanks to my ***Allah****, who gaveme power, inspiration and helped my through my life and to complete this research. I would like to thank my supervisor* ***prof. Salha F. Ben-Gweirif,*** *a professor of bacteriology, for her great support before and during my research and it was a great pleasure for me.I am also grateful to microbiology stuff in Aljalaa analysis laboratory, Benghazi and a great.*

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A S T	Aspartate Transaminase
D C A	*Deoxycholate Citrate Agar*
C L E D	Cystine lactose electrolyte deficient agar
R T A	Rod Trfic Accidents
ID	idem
I C U B	Intensive care units
M R S A	Mithicillin-resistants S. aureus
M B S	Male burn S
X L D	Xylose Lysine Deoxycholate agar
O T	Operation theatres
S P SS	statistical package for social science

Abstract

The aim of the study was to determine the prevalenve of bacteria in burn wound infection and its sensitivity to the commonly used antibiotic. Also to the impact of environmental conditions and the risk factors associated with infection burns during a period of 2012 to 2013 . Atotal of 133 specimens were collected from burn patient. (burn and plastic sergery hospital) in the city of tripoli in Libyan. samples from the inanimate environment were also examined by taking swabs from different areas to detect the bacteria that may be found in the surrounding environment of patients. The isolated bacteria were*Acinetobacterbaumannii* isolates (37.6%), followed by *Pseudomonas aeruginosa*(23.3%), *Klebsiella pneumonia* (8.3%), *Staphylococcus aureus* (7.5%), *Staphylococcus haemolyticus* (5.3%), *Enterobacter cloacae* (4.5%), and *Staphylococcus epidermidis* (3.8%) , *Enterobacteraerogenes*,*Escherichia coli* and *Proteus mirabilis* isolates (3.0%), followed the lowest causative agents of burn wound infection were *Staphylococcus saprophyticus* isolates (.8%). The isolated bacteria were sensitive to Colisten ,Amikacin , Ciprofloxacin, Gentamicin, However no effect was observed by Penicilln, Tetracycline, Erythromycin andCefipeme .

this study showed that *Acinetobacterbaumannii* was the most counstive agent causes burn infeaction in burn and plastic sergery hospital in tripoli. the gram-positive bacteria *satphylococcusaureus* was the most caustive agent was burn wound infection.

CHAPTER ONE

INTRODUCTION

1.1- Introduction

A burn breaches a vital barrier to infection the skin and leaves the body vulnerable to bacterial invasion. A burn is initially free from microorganisms but soon becomes colonized by skin commensally faecal microbes and by airborne, exogenous sources of bacteria.(skoll *et al*., 1998) the skin one of the largest in the body. per forms numerous vital functions including fluid homeostasis thermoregulation, immunologic function, neurosensory functions, and metabolic functions (eg., vitamin d); the skin also provides primary protection against infection by acting as a physical barrier, when this barrier is damaged, pathogens have a direct route to infiltrate the boody, possibly resulting in infection(Murray *et al*, 2011) , the burned patient may suffer many complications either as are sult of direct effect of the burn injury such as wound infection, bacteremia and septicemia or indirectly du to malnutrition, immobility for long time, reduced immunity, urinary tract complication.

Walls, floors and ceilings play an important role for the spread and as source of infection from inanimate environment, hydrotherapy pools and associated are other important sources of infection. Awhole range of techniques is used, inciuding many invasive Procedures, all requiring strict hygienic precaution .many infection control procedures are controversial and opinions are changing . in most units minimal protective clothing is now worn (theatre dresses for general wear, and disposable plastic aprons for direct patient care) masks have been abandoned in recent years without evidence of an increase in infection in the units .Occasional outbreak of antibiotic -resistant hospital strains of bacteriaoccur in a burn unit and many cause its temporary closue. Such an occurrence serves to emphasize the need for rigid adherence to infection control policies and procedures(Ayliffe, 1975)It is now estimated that about 75% of the mortality following burn injuries is

related to infections. The pattern of infection differs from hospital to hospital; the varied bacterial flora of infected wound may change considerably during the healing period (Rajput et al., 2008). When a hole is created on the skin, microorganisms, usually the opportunistic organisms, invade the holes and multiply leading to a delay in the healing process and finally infectious condition. The spectrum of infection ranges from asymptomatic colonization to bacteraemia and death (Abubakar, 2009).Age of the patient, extent of injury, and depth of burn in combination with microbial factors such as type and number of organisms, enzyme and toxin production, colonization of the burn wound site, systemic dissemination of the colonizing organisms (Pruitt, 1984).

Moreover the larger area of tissue is exposed for a longer time that renders patients prone to invasive bacterial sepsis. In extensive burns when the organisms proliferate in the eschar, and when the density exceeds 100,000 organisms per Gram of tissues, they spread to the blood and cause a lethal bacteremia. Therapy of burn wound infections is therefore aimed at keeping the or ganisms burden below 100,000 per gram of tissues which increases the chances of successful skin grafting. (Medical, 2005) (Order SE, Mason)Microorganisms may also be transferred to a patient’s skin surface via contact with contaminated external environmental surfaces, water, fomites, air, hydrotherapy treatment, and the soiled hands of health care worker(Mayhall,2003 and Church, 2006).

1.2- The Aim of the study

the aim of this study.

1. Isolation and identification of causative bacteria of burn wound infection in the burn hospital in the Tripoli
2. To study the sensitivity test of isolated bacteria to different antibiotic .

CHAPTER TWO

REVIEW OF LITERATURE

2-Review of literature

2-1 Historical background

Infection is an important cause of mortality in burns. Rapidly emerging nosocomial pathogens and the problem of multi-drug resistance necessitates periodic review of isolation patterns and antibiogram in the burn ward. (mehta *et at*, 2007). Physicians have searched for and formulated a myriad of treatments for burns over the centuries but these treatments mostly were of littlebenefit to the victims mainly because the fundamental understanding of the patho-physiological impact of burns was not known yet. A wide variety of therapies for burns have been described since ancient times (Artz,1970) but the idea of collecting burn patients in a special place is relatively new, and emerged in Scotland during the 19th century. Syme established the first burn unit in Edinburgh in 1843. he argued that mixing burn patients with postoperative patients would make him "chargeable with the highest degree of culpable recklessness." This experiment was relatively short-lived, however, since burn patients were transferred to one of the "Sheds" in 1848 to make way for an increased number of mechanical trauma casualties from railway accidents (Wallace, 1987). Another Scottish hospital, the Glasgow Royal Infirmary, had by 1933 accumulated 100 years of experience with over 10,000 burn patients, having established a separate burn ward midway through that period in 1883.

2.2-Struture and function of the skin

The skin is an organ system with multiple function , including protection of tissues from external microbial invasion. Its keratinized stratified epithelium prevents direct microbial invasion under normal condition of surface temperature and humidit , and its normal flora ,pH and chemical defenses tend to inhibit colonization by many pathogens however the skin is subject to repeated minor traumas that are often unnoticed but that destroys its integrity and aiiow organism to gain access to its deeper layers from the external envieoment 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 .,2002).

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 regenenrates quickly after damage if the basal layer is intact (Spence *et al*,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 and elasitn fibers , provides a defense against infection and helps deep wounds heal due to activity of component (Spence *et al*., 1987).

2.2.3- The hypoderma ,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 *et al* , 1987and Nester et al., 2004).

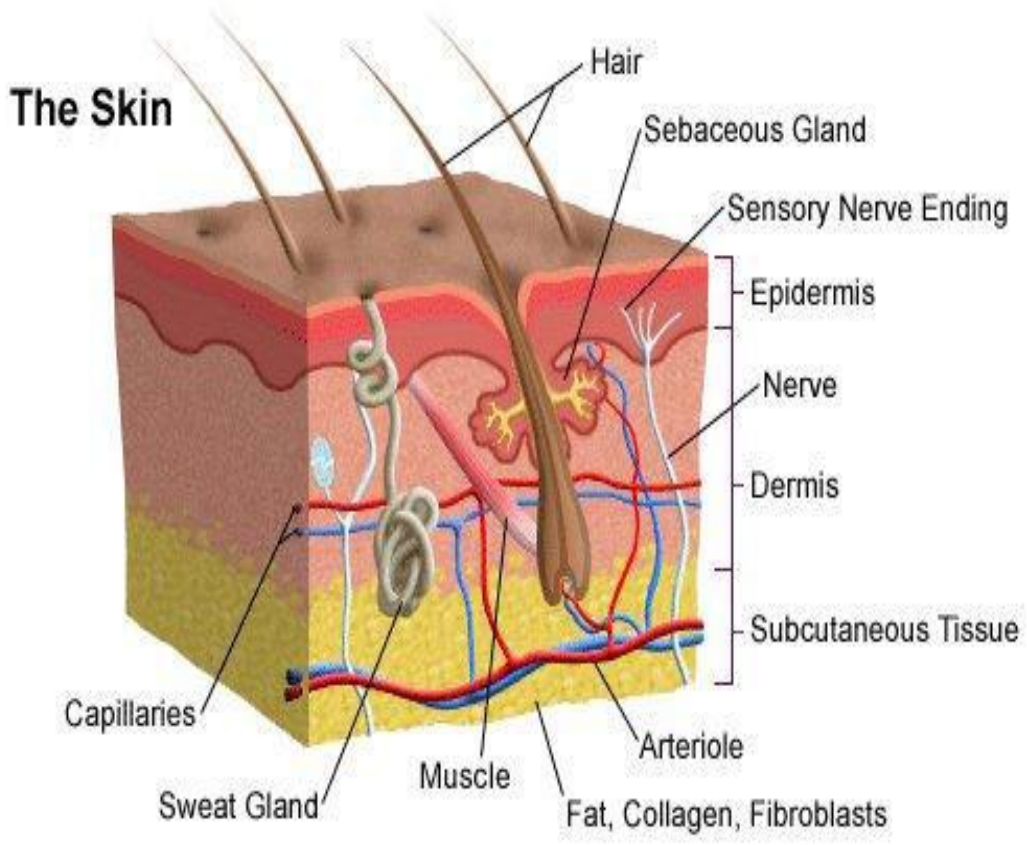


Fig.(1): Anatomy of the skin

2.3-Normal flora of the skin .

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 (Medical, 2005)as seen in (table 1.**)**

1. *Staphylococcus epidermidis*
2. *Staphylococcus aureus*(in small number).
3. *Micrococcus species*.
4. Nonpathogenic Neisseria species.
5. Alpha-hemolytic and nonhemolytic *streptococci*
6. Diphtheroids.
7. Propionibacterium species.
8. Propstreptococcus species.

9.Small numbers of other organisms (*candida species*, *Acinetobacterspecies, etc)*

Table 1. Normal flora of the skin

2.4-Wound infection

When the protective skin barrier broken as result of burns , puncture wound ,surgical procedure, 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 infection (Burton *et al* , 2004).

2.5-Causes of burn:

Burns can be caused by bry heat (like fire), wet heat (such as steam or hot liquids), radiation , friction , hot objects ,sun rays , electricity, or chemicals. thermal burn are the most common type, it occur when hot metals, liquid , steam ,or flames come in contact with the skin . they are frequenty the result of fires, rod trafic accidents (R.T.A), playing with matches , improperly use of gasoline, space heaters , and electrical equipments malfunctions. Also burn to airways can caused by inhaling smoke, superheated air, or toxic fumes often in apoorly close and confined space (John *et al* ., 1996).

2.6-Risk groups of the population

Children comprise 45% of the total burns unit workload the great major, therr quarters, are young children. scalds are particularly prominent in the very young , affecting about 79% of the under 5 year old young boys are one and half times more likely to be scalded than little girls .the population data for the city of Birmingham suggests that children in the under than 5 years old group are 36 times more likely to be scalded than are adults of working age .children aged between 6 months and 2 years are at the greatest risk of suffering thermal injury due to their developing mobility and starting to explore the environment school childern have about three times higher risk of scalding than adults of working age. in birmrngham 1980 one child in every 143 is likely to have been detained in the burn unit with a serious burn or scald before entering school at age of five (Birachall ., 1988)

2.7- Degree of burn

When the skin is exposed to excessive heat as from fire , electricity , hot object or corrosive chemicals, the resulting tissue damaged (surface area and depth). (Field *et a.l*,1998). burn can be classified according to depth into :

2.7.1 First degree (superficial)

Burn , affect only the epidermis causing reddening of skin , edema , pain which usually resolves in 48-72 hour the damage of epithelium peels off within 5- 10 days

2.7.2 Second degree (partial thickness)

Burns affect epidermis and part of dermis causing reddening of the skin, acute pain and edema in and around the affected area , if heat destruction involves epidermis and upper third laye of the dermis the burn is classified as superficial dermal burn , but if the heat destruction involves epidermis and most of dermis as well the burn is classified deep dermal burn (Arturson , 1985).

2.7.3 Third dehree (full thickness)

Burns involve destruction of all skin elements; it destroys the full thickness of the skin epidermis and the whole dermis, and may extend up to muscles and bones . the burned area become waxy white in colour and dry and skin lose it sensation. In thin case

The burned wound should be ultimately treated surgically by skin grafting or flap (Jacson ., 1982)

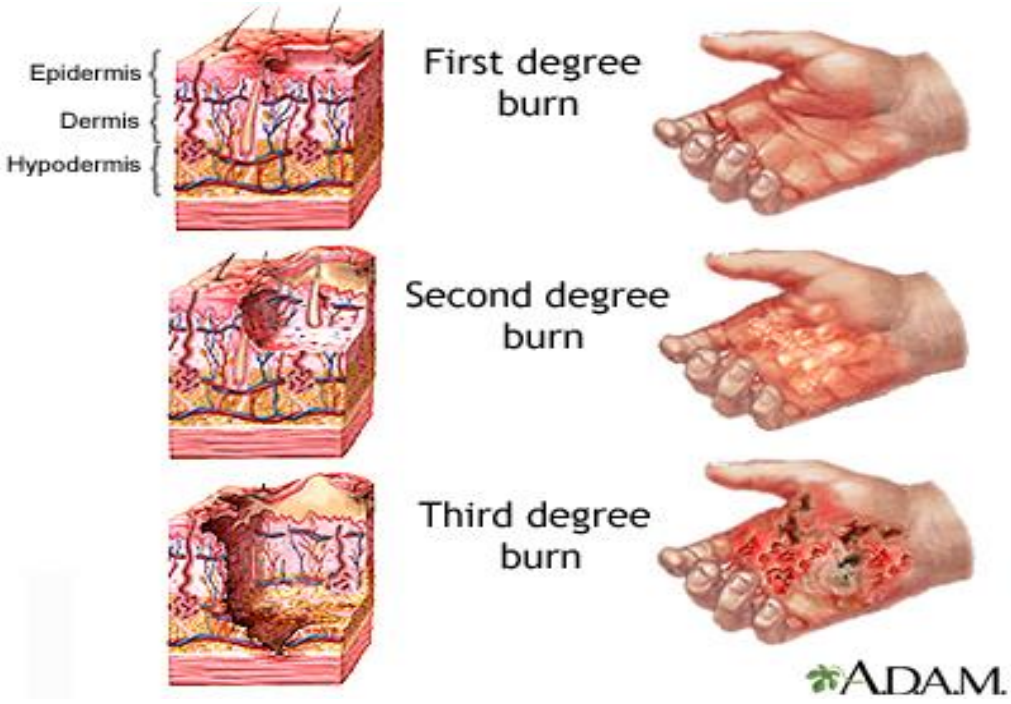


Fig2: Degree of burn

2.8-Mechanism of burn injury :

2.8.1-Energy transfer from heat sources to the body .

Thermal injury occurs as a result of energy transfer from a heat source. Transfer of energy depends upon the following factors that can not be measured directly in a thermally injured patient.

1. The rate of absorption of heat which is dependent initially upon the function of peripheral circulation
2. Presence or absence of insulation such as hair , cornified layer of surface epithelium, natural skin oil's etc.
3. The total water content of the tissue .
4. The amount of surface pigmentation .
5. The presence or absence of clothing of different kinds (Costa,1996).

2.8.2-Heat exposure versus duration

Due to the body's capacity to diffuse and dissipate heat very rapidly , the temperature of the body surface can be increased to a rather high degree without much local tissue damage. Also increased in skin temperature of moderate degree for long periods of time ordinarily do not result in significant skin damage. A temperature greater then 51^0C will destroy tissues very rapidly , but above 70^0C total tissue destruction will occur within seconds. Below 44^0C will result only in the epidermis cellular damage(Gosta ., 1996).

2.9-Effects of burn injury

A sudden increase in the surface of the body temperature results in both local effects and systemic effects.

2.9.1- Local affects

Mostof local effects produced in case of burns could be summarized in the following points.

1. Aperiod of rapid local edema formation due to local haemodynamic changes and micro vascualtion responses(vasodilatation and increased vascular permeability)

2. The above changes maight lead to local tissue ischemia and death

3.large water losses by evaporation from the burn wound , resulting in significant body water loss, protein and salt loss

4.The thermal injury initiates an inflammatory response and release of local inflamatory mediators (histamine, kallikein , prostaglandins and leukotrines). (Gosta , 1996).

2.9 .2-Systemic effects

The burned patient may suffer many complications either as aresult of direct effect of the burn injury such as wound infection , bacteremia and septicemia , or indirectly due to malnutrition , immobility for long tim, reduced immunity urinary tract

2.10-Burn infection

Burn are common throughout the world , they result from accidents in the home , in industry , and in travel . the application of heat may cause partial or complete destruction of skin which one of the most important natural barriers to infection . The frequency of burn increases every year. Being more frequent in the cold season , young adults are the most commonly affected followed by infants and children . scald accidents were the commonest cause of thermal injuries followed by flame burns (El-gallal , 1998) . One of the causes of burn in children is prolonged transition from one social

system to another with profound reflection on the political , economic and social life of all people. there has been an un controlled demographic displacement of population with people moving from the villages toward larger cities, and many of the families and social groups involved live in conditions below the average standard of living (Belba , 1998) .

The particular problem of burn infection was recognized by early civilization and overthe centuries it continues to be the major cause of death in burn patients (Berger, 1998) in a retrospective survey covering a 15-year period (1978 - 1993).(El-gallal *et al*,. 1998).found that 71 % of hospitalized burn patients ' deaths were due to wound sepsis, septicaemia and complications of septicaemia . *Staphylococcus aureus* and other gram - positive cocci isolated from infected sites were the dominant organisms causing infection in the first week of hospitalization .

In the second week and subsequently Gram - negative bacteria , especially *Pseudomonas aeruginosa* dominated the picture and were responsible for most of the complication of septicaemia .the problem of cross-infection in burn units was recognized by cruickshould (1935) when he described the acquisition of *streptococcus pyogenes* in patients after admission to hospital. he also showed that the organisms were frequently present in the air .investigation into the spread of *Strept pyogenes* and later *Staphylococcus aureus* continued throughout and following the second world war (Belba , 1998) . In the 1950 and 1960 infection came to be recognized as the major cause of death in patients with extensive burn caused by bacteria which had formerly been dismissed by many aas harmless commensals in 1997 shriners Burne institute had designed a workabout nosocomial infections in pediatric patients with burns , and

they stated that Infection remains a cause of significant morbidity and death for patients with burns . Burns are sterile immediately after infection but liable to be colonized rapidly by bacteria . burn wound infection remains a serious complication unique to the burn recipient. the methode for managing thermal have evolved during the past 50 years .(Wu et al , 1994)

2.11- Complications of burn infection

The examination of the burned area usually reveals a sterile surface after the 24-36 hours. but infections occasionally occur . the term infection is generally used to mean deposition and multiplication of bacteria and other micro organisms in tissues or surfaces of the body where they can cause adverse effects.(Lowbort *et al* 1954)

2.11.1- Bacteraemia

Bacteraemia is the presence of bacteria in the blood it is the principal means by which local infection spread to distant organs (referred to as haematogenous) bacteraemia is typically transient rather than continuous due to a vigorous immune system response when bacteria are detected in the blood.

There are three types of bacteraemia, transient, intermittent and continuous according to several basic entry mechanisms of bacteria into the blood stream.

2.11.2-septicemia

Septicemia is defined as active growth of organisms in blood it is a clinical syndrome characterized by fever chills, malaise,

tachycardia, hyperventilation and toxicity or prostration which results when circulating bacteria multiply at a rate that exceeds removal by phagocytes ((Muir *et al* ,1987).

2.12-Bacterial infection

The major challenge for a burn team is nosocomial infection in burn patients, which is known to cause over 50% of burn deaths. most studies on infection in burn patients focus on burn wound infection Ekrani and Kalartar (2007) reported thay *pseudomonas aeruginosa* remain the most important microorganism responsible for burn wound infection floued by *Staphylococcus aurous* (20.2%), similar results was observed by (mehta *et at*., 2007) who found that *pseudomonas aeruginosa* was the most common organism isolated from burn wound infection (51.5%); followed by Acinetobacter spp(14.28%), *staph. aureus* (11.15%), klebsiella spp,and *proteus* spp(2.3%). in contrast Macedo and Santos (2005) reported that prevalence rate of s. *aureus* was (28.4%) of all pathogens isolated from burn wound infection. Murray and cunha . 2011).reported that the prevelance of s.*aureus* to was hiyher than klebsiella.spp burn wound infection. in addition in study `of Abubakar (2009) the common pathogens isolation from burn wound *are staphylococcus aureus* (75%), pseudomonas aeruginosa (25%), streptococcus pyogenes (20%) and avrious coliform bacilli (5%) in Iran , shakibaie et al ., (2008) found that 77 (64.2%) out of 120 burn infection patients were males while 43 (35.8%) were females . most of the burn infection patients aged between 11 to 20 year old .

Alwan *et al* .,(2011) reported that study was carried out to determine the bacterial isolates and study their antimicrobial susceptibility in case of burned wound infections. 70 burn wound swabs were taken from patients, who presented invasive burn wound infection from both sex and average

age of 3-58 years, *Pseudomonas aeruginosa* was found to be the most common isolate (48.9%) followed by *Staphylococcus aureus* (24.4%), *Citrobacter braakii* (13.3%), *Enterobacter spp*. (11.1%), *Coagulase*-negative Staphylococci (11.1%), Proteus vulgaris (6.66%), *Corynebacterium spp*. (6.66%), *Micrococcus* (6.66%), *Proteus mirabilis*(4.44%), *Enterococcus faecalis* (4.44%), *E.coli* (4.44%), *Klebsiella spp*. (2.22%), *Bacillus spp*. (2.22%), *Serratia macerscens* (2.22%) and Serratia rubidia (2.22%).

Antimicrobial susceptibility testing was carried out to the bacterial isolates against 8 antibiotics, in which ciprofloxacin was found to be the most effective drug against most of the Gram-negative and Gram-positive isolates followed by amikacin, while chloramphenicol and gentamicin were less sensitive to few isolates as well as as doxycycline, as compared with the othertwo, mentioned previously. Oxacillin was the worst at all .Chaudry *et al* (1993) In the present study the mostcommonly isolated organisms fromburned patients were *P. aeruginosa*followed by *S. aureus*, *C. braakii* and*Enterobacter spp*.

Another study by Arslan *et al*(1999) reported that Pseudomonas species was the commonestpathogen isolated (23.33 %) from burn wound followed by *S. aureus* (15.33 %), *Klebsiella spp*. (3.33 %) and *Proteus*

species(8 %).Mahmoud (2009) reported that *Enterobacter spp*. is the main isolate (8.66 %) from burn wound sample,Micrococcus spp (3.33%) and *E. coli* (4.66 %). Microbial infection is one of the major serious complications in woundpatients, the results of the present study showed that 35 (23.33%) burn wound swabs revealed *P. aeruginosa* , this goes toconfirm that *P. aeruginosa* is a major factor in the etiology ofwound infectionin contrast Obritsch *et al* ,(2004) reported that the rate of gram negative bacterial isolation from burn wound was more than twicethat

gram- positive and they noticed that *Klebsiella spp*. was the pathogen less isolated constituting 3.33% followed by *P. aeruginosa* (23.33 %) and *S. aureus* (15.33%) . Sewunet*et al* (2013) a total of 50 burn patients who either visited or were admitted to the Burn Center during the data collection period were included in the study. Both blood and wound swab samples were collected from all study subjects. Of the total study participants, females accounted for 20(40%) and males accounted for 30 (60%), whereas the age ranged from 7 years to 55 years with the mean and median ages of 26.24 years and 24.5 years respectively.The magnitude of bacteremia among burn patients at the center was 21(42%). Five different bacterial species were isolated; Coagulase negative Staphylococci and *Staphylococcus aureus* were most common. The distribution of these isolates ranged between Coagulase negative *Staphylococci,* 9(42.8%), *S. aureus,* 8(38.2%), *Bacillus spps* 2(9.52%), and both *Klebsellapneumoniae*and *Pseudomonas aeruginosa*, 2(4.8%).

Another studies by Anuradha *et al* (2008) and Merlin *et al*(2009). On the other hand, isolates from wound swabs were analyzed separately and hence the most common isolates from the wound swab include *S. aureus* (34.04%), followed by *P. aeruginosa* (31.5%), *Coagulase negativestaphylococci* (12.76%), *Proteus mirabilis* (8.5%), *Proteus vulgaris* (8.5%), *K. pneumoniae* (2.1%), and *Providencia spps* (2.1%). Although a number of studies have been conducted on burn wound infection and bacterial profile, nearly all of them are retrospective studies which made comparison of findings of this study to those findings difficult; however, these studies remain optional for comparison. Comparison of bacterial isolates with other studies may also be difficult because of geographical variations, drug policies, infection control policies and the like.

Ghaffar *et al*., (2002) who found that burn wound infection in males was 189(62.4%) while burn wound infection in females 114 (37.6%) In a similar study Macedo and Santos (2005) found that burn wound infection in males 120(59.1%) was more than burn wound infection in females 83 (40.9%). .This may be due to that males are exposed more to burns and wear loose fitting clothes like dhoti, lungi and phiran which catch fire easily also mostly restaurant workers are males engaged in cooking. Naqvi ., (2007) Bagdonas *et al*., (2004), Elsayed *et al*., (2003) and Rahbar *et al*., (2005) who found that the most prevalentbacteria among burn patients was *S. aureus*. Inthe other hand, AL-Akayleh, (1999) andSharma *et al*., (2006) found that the most prevalence isolated bacteria from burn wound patients were *P. aeruginosa*, *Klebsiella*, *S.aureus, P. mirabilis*, while the least prevalence isolated bacteria was *E.coli*. With Ghaffar *et al*., (2002) in India who found flame burns were the most common types in burn infection patients. Kerosene was the main accelerant accounted for burns. This is probably because kerosene is cheap and easily accessible and more use of keroseneestove and kerosene lamp by the people of low socioeconomic status in rural area where obsolete and unsafe uses of fire forcooking and light are still prevalent. Elsayed *et al.,* (2003) who found that the most *S. aureus* is a versatile human pathogen. It was the predominant cause of burn wound infection in pre antibiotic era and still persists as an important pathogen, strongly considered as a major cause of nosocomial infection. Interestingly the frequency of infection has increased duringlast three decades. Burn units have becomemajor reservoir for *S. aureus* that has thespecial characteristics for spreading quicklyin a hospital Vougiouklakis*et al* (2005) who observed out of 100 study cases that 18 cases and 7 cases died within 6 hours and 6 to12 hours respectively. 4 cases died within 12 to24 hours. 27 cases died within 1 to 3 days, 23cases between 3-5 days, 14 cases in between 5to7 days and only 7 cases died after 7 days from

time of injury. The highest period of survival was observed in 6% cases belonging to category of within 3 weeks. Another studies by Aggarwal and Chandra (1970)had highest period of survival of about 3 weeks in 2 cases out of total 100 study cases. Singh et al (2003) Regarding isolation rates of organisms from our Burn ward, it was decreased for Pseudomonas species, *S. aureus* and Proteus species whereas it was increased for *Klebsiella species* . This changing trend in burn bacteriology In contrast to this Sengupta *et al* (2001) showed that In contrast to this, there was a significant rise in the isolation rate of *Acinetobacter species* over the last five to eight years in our burn unit.

Vivian *et al* (1981) reported *Acinetobacter species* are emerging as an important cause of nosocomial infection in burn units. There are a number of factors which may contribute to this increase like its presence as a normal skin commensal and its easy spread due to multi drug resistance in a hospital setting. Agnihotri *et a.,l* (2004) The change in the pattern of bacterial resistance in the burn unit has importance both for clinical settings and epidemiological purpose. We saw a significantly high percentage resistance among gram-negative bacilli to aminoglycosides like gentamicin and amikacin, ciprofloxacin, carbenicillin, tobramycin, amoxicillin, cefotaxime and ceftriaxone. This alarming trend was seen for both Enterobacteriaceae group and for Pseudomonas species. similar report Singh *et al*., (2003) of multi drug resistant gram-negative bacilli was also reported by Singh et al . In comparison, imipenem and combination drugs like cefoperazone,sulbactum and ceftazidime ,clavulanic acid were found to be effective. This could be due to the reason that these are reserve drugs and used as last options for multi drug resistant bacteria in our hospital settings. For gram-positive cocci a significantly high resistance was seen only for netilmicin. Nevertheless, other antimicrobials tested also showed

high percentage resistance. However, newer drugs like vancomycin and linezolid were shown to behighly effective.Such high antimicrobial resistance is probably promoted due to selective pressure exerted on bacteria due to numerous reasons like non adherence to hospital antibiotic policy, and excessive and indiscriminate use of broad-spectrum antibiotics. These multi drug resistant strains establish themselves in the hospital environment in areas like sinks, taps, railing, mattress, toilets and thereby spread from one patient to another. To conclude, routine microbiological surveillance and careful in vitro testing prior to antibiotic use and strict adherence to hospital antibiotic policy may help in the prevention and treatment of multi-drug resistant pathogens in burn infection.

2.13-The bacterial causing infection in burn wound

2.13.1 -*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 ,(Dayoub , 1995)

is a bacterium that is a member of the Firmicutes, and is frequently found in the human respiratory tract and on the skin. Although S. aureus is not always pathogenic, it is a common cause of skin infections (e.g. boils), respiratory disease (e.g. sinusitis), and food poisoning. Disease-associated strains often promote infections by producing potent protein toxins, and expressing cell-surface proteins that bind and inactivate antibodies. The emergence of antibiotic-resistant forms of pathogenic *S. aureus* (e.g. MRSA) is a worldwide problem in clinical medicine.(Ogston ,1984).

2.13.2-*Streptococcus spp.*

Gram- positive, anaerobic, often pathogenic bacteria having an ovoid or spherical appearance and occurring in pairs or chains, When cultured on blood agar species can be classified as Beta-hemolytic colonies are surrounded by a zone complete haemolysis. Alpha-hemolytic coloniesare surrounded by a green-brown colour. Non-hemolytic colonies showneither typical alpha- nor beta haemolysis(Geis, 2006 and Cheesbrough,1984) .

The main of medical importance *S. pyogene s*this species causes acute sore throat, scarlet fever, earinfection, puerperal sepsis, skin infection, and septicemia. *S. agalactiae* this species causes neonatal septicemia, pneumonia, meningitis, septicabortion, and puerperal sepsis. Enterococci can cause urinary tract andulcers and wound infection, occasionally endocarditis and meningitis.Viridians Streptococci can cause infective occasionally endocarditis, dentalcaries, and abdominal and brain abscesses. Streptococcus spp. Rarely appears in cosmetics products. Their presence in a cosmetic would be aresult of employee failure to follow good sanitary practices for example, by placing a hand in to a product or container (Geis, 2006 and Cheesbrough,1984).

2.13.3-*Pseudomonas spp.*

Gram negative , strictly aerobic, have characteristics of being straight or slightly curved gram negative bacilli. They all use the tricarboxylic acid cycle to oxidize substrates to carbon dioxide. The organisms are infectors of wounds and burns. They also cause pneumonia in patients who take immune suppressivedrugs. In cosmetics, the organism has been implicated in eye infections and loss of sight. When

found in a cosmetic manufacturing plant, they usually arise from failure to control and monitor water system, formation of biofilm in the equipment, ineffective or infrequent sanitization and deadlags (short lengths of pipes with closed or dead end) or other sources of stagnant products (Geis, 2006).

2.13.4- *Acinetobacter spp*.

Gram-negative, non-motile and non-fermentative bacteria belonging to the family *Moraxellaceae*. *Acinetobacter* is also commonly found as a harmless coloniser on the skin of healthy people and usually poses very few risks. *Acinetobacte*r infections acquired in the community are very rare and most strains found outside hospitals are sensitive to antibiotics, while Acinetobacter poses few risks to healthy individuals, a few species, particularly *Acinetobacter baumannii*, can cause serious infections, mainly in very ill hospital patients. The most common Acinetobacter infections include pneumonia, bacteraemia (blood stream infection), meningitis , wound infections, and urinary tract infections. 'Hospital-adapted' strains of Acinetobacter are sometimes resistant to antibiotics and are increasingly difficult to treat.(Ryan and Ray2004) .

2.13.5 -*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 but also in adult (collee et al ., 1989)

2.13.6- *proteus spp.*

The main species of medical is : proteus 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).

2.13.7-*Klebsiella spp.*

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 (Murray et al .,1998)

2.13.8- *Enterobacter spp.*

Gram negative rods this organisms are found mostly in soil and dry surface thay are not generally considered human pathogens unless they are directly introduced into the bloodstream some species include enterobacter agglomerans can cause avariety of necroses . thuscausing a variety of necroses. They are not generally considered humanpathogens unless they are directly introduced into the bloodstream.(Geis, 2006).

2.14-Router of infection

Infection remains one of the major challenges in the management of the burned patients. it continues to account for 22 to 68% deaths in thermal injury, despite improvements in care (Merrel *et al* , 1989 and Weber *et al*,1997) hospitals are the ideal environment to develop bacterial resistance, that is considered to be a serious problem. moreover burns become infected, because the environment at the site of the wound is ideal for the proliferation of infecting (Rokas *et al* , 2004).many bacteria tend to be difficult to control with antibiotics because of their resistance. Multi- drug resistant bacteria are wide spread in hospital environment.infection occurs in deep partial- or full- thickness burn that has not been surgically excised .Infection of the burn wound is associated with change in wound appearance or character, such as rapid eschar separation, dark brown in colour, or black.the onset of infection starts by a rise in temperature, general malaise, reddening of the wound edges and presenct discharge(Ellsworth *et al*, 1971 and Glen , 2003)

2-15-Sources of infection

Walls, floors and ceilings play an important role for the spread and as source of infection from inanimate environment (Ayliffe *et al* , 1967), sinks, baths, hydrotherapy pools and associated equipment are other important sources of infection (Ayliffe *et al* , 1974). infection may also originate from the hands or respiratory tracts of first aiders and the patients may acquire infection from bacteria present in bed and linen.fromthese sources organisms may be transferred through the air to other patients (cross infection) or transmitted indirectly on the hands and clothes of nurses and doctors (Muir *et al* , 1987). the patients own flora, especially coliform bacilli and anaerobic, sporing and non-sporing,

bacteria may contaminate the patients burn wound (endogenous infection) (Field *et al* , 1998).

2.16-Treatment in burn

Burn are the most serious injuries a person can suffer and they are perhaps the only injury that requires special treatment by a team of medical and nursing personnel possessing specific skills and experience.

2.16.1-first aid treatment in burn.

The treatment of burn caused by heat or electricity starts with the application of cold water in order to cool destroyed tissues and to minimize damage to them. this method is useful only for minor or second degree burns but if it is used in extensive or third degree burns it will aggravate the state of shock (Rosendery , 2002)

The burned area should be continued to cool about 5 minutes or unit the pain diminishes, then dry gently with clean towel and dress it with a clean dry cloth. remove watches, bracelets, rings, belts, from the affected area befor it begins to swell. never apply butter, oil or grease to the wound. Analgesic drugs and ointments should be used (Ioannovich *et al* .,1999 and Germenis, 2002)

2.16.2-**Treatment of burn patients in the emergency department**

Treatment of burned patients in emergency department includes checking of respiratory passages to ensure that the patient is properly ventilated , and giving intravenous fluid replacement.the greatest problem associated with major burn is fluid loss,where water, plasma and electrolytes are released from the surface of burned wound.

Advancements in the treatment of shock have reduced the mortality rate in the first 48 hours form 50% to around 5%.

However since effective methods of preventing hypovolumic shock have been introduced, infection has come to be recognized as the most common cause of death in extensively burned patients a large burn is very susceptible to contamination with bacteria from the environment, not only the cause of the large area of necrotic tissues, but the dehydration, electrolyte imbalance hypo-proteinemia and loss of immunoglobulins, all of which diminish resistance to infection. therefore wherever the burned patient is nursed, a strategy for the prevention of infection is essential and must be carried out alongside life-saving measures(Cason , 1981)

2.17-Prevention of burn wound infection

2.17.1-Prophylactic antibiotics

Prophylactic practice has been abandoned in many burn units previously all patients admitted to burn unit received antibiotics as prophylaxis (Muir *et al*, 1987). Many agents have been tried, but the most effective are those with a broad spectrum of antibacterial activity, lack of developing resistance, low toxicity, and active penetration of the wound (John , 1978)

The management of in burns has been directed towards attempting to prevent bacterial colonzation, not only by peotective isolation and interruption of cross infection, but also by the utilization of both topical and systematic antimicrobials.the ideal local antibiotic should have a high degree of antimicrobial activity, against a broad spectrum of bacteria including *pseudomonas aeruginosa, staph aureus* and *streptococcus pyogenes,* also the antibiotic should be simple to use and acceptable to both patients and nursing staff. the success of topical application of antimicrobial agents in preventing septic complication in burn patients is

in sharp contrast to the result for the routine administration of systematic antibiotics for prophylaxis, whic doesn't lead to reduction in the colonization of burns. topical therapy to prevent and remove bacterial colonization of burn was used since 1966. All patients admitted acutely have been given a 3-5days course of antibiotic effective against clostridium tetani, staphylococci and *streptococcus pyogenes*, these antibiotic included penicillin, Erythromycin, Clindamycin,Cloxacillin and lucloxacillin(John , 1978).

2.17.2-Protection from other sources of infection

Convalescent patients should not touch other patients bed or exchange magazines and the risk of cross infection should be explained to them.

sterile linen may be used on the bed or burned areas placed on sterile dressing towels the bed should be changed completely with clean line once daily, cotton boilable blankets are preferable and these are changed at least once a week all pillows, mattresses, bowls, bedpans, trays should be sterilized before use by patient and after use. The room should be air conditioned(Towner , 1997).

2.18-Prognosis of burn infection

A number of factors influence the prognosis of burn infection, which include the type, depth., and site of burns and associated injuries, also the response of the patient to injury, and subsequent treatment, and the treating staff and the number and type of organisms that may colonize the wound (Gosta , 1996).

2.18.1-Types of burn wound

Scalds are effectively thermal injuries occurring at or below 100^0 C where flame burns reach many hundreds or even thousands of degrees Celsius. Chemical burns are a different group which may pose their own

peculiar metabolite problems and progress for some time after the initial exposure. Electrical burn is one of the most devastating injury that may affect multiple organ systems (Gosta , 1996).

2.18.2-Depth

Depth of burn has a significant influence on prognosis the superficial or partial thickness burn to some extent can be considered self limiting within 10-14 days of injury. full-thickness burn heal naturally by fibrous repair but the patient is exposed to complications such as invasive sepsis that leads to lengthier process of prognosis (Gosta , 1996).

2.18.3- Site of burn

The region of the body affected by the burn injury may also affect prognosis largely because of the injuries or complication associated within the site as the following

A. Deep injuries involving muscles may result in renal impairment
B. burns of the perineum and surrounding area are more likely to be infected by *pseudomonas aeruginosa* and causing septicemia
C. Burn of chest may restrict the tidal volume during breathing
D. Circumferential burn of limbs can affect the blood supply to distal areas
E. Flame burn of the face are often associated with thermal damage to the upper airways or chemical to the lower air ways (Gosta , 1996).

2.18.4-The personal response to the injury

Extremes of age have been recognized to be associated with worse prognosis.

A. Pre-existing diseases have worse prognosis, pregnancy in burn victim increases the risk of death pregnant mother and increase the risk to the baby.
B. If the burn injury is severe or the patient resistance is low.
C. Partial or complete failure of one or more body system , this failure is associated with a mortality of over 70%. And has greater effect on prognosis than either age or size of burn alone.
D. Untreated hypovolumia will lead to death of the burned patient who has got large burns(Gosta , 1996)..

CHAPTER THREE

MATERIALS AND METHOOD

3-Materials and methods

3.1-Patients

Over the period of four months (January to December 2012) a total of 133 burned patients were admitted to the burn unit of burn and plastic sergery hospital Tripoli .

3.2-Collection of samples and isolation of bacteria

The samples were collected within 24 hr of admission by using cotton wool swabs taken from the burned area from patient admitted to the hospital.whenever possible the specimens were transported to the laboratory where they were cultured onto blood agar and macConkey agar and incubated aerobically at 37^0C for 48 h, then inspected and processed further to identify any isolated.(figure 3)

Fig (3):Collection of sample

3.3- Isolation of pathogenic bacteria of Environmental

A sterile cotton swabs moistened in sterile normal saline were usedto collect environmental samples from the floors, doors, sinks, incubators, and other instruments in the units in burns. the area of the swab was approximately 10 sq. cm (Ness, 1994).One hundred and thirty sample samples were taken Hospital in Tripoli from patient`s rooms, dressing rooms, halls, Bathroom Department burns and toilets,(ICU), different places such as walls, beds, wheelchairs ,trolley, halls, floors, doors, Air Conditioners and patients instruments.

3.4- Gram stain and microscopic examination

The Gram stain used to identify pathogens by their Gram reaction (Gram positive or Gram negative) also it's combined with the morphology (cocci or bacilli) and arrangement of the bacteria.A thin smear of the bacterial suspension was made and allowed to air dry then the dried smear was fixed by passing the the slide three or four times through the flame .The slide was then covered with crystal violet stain for one minut, then washed with tap water and covered with lugol,s iodin for one minut. It was decolorized with acetone alcohol and immediately washed by tab water then covered with safranine then washed with water and allowed to dry and examined microscopically with the oil lens.

3.5-Biochemicaltests

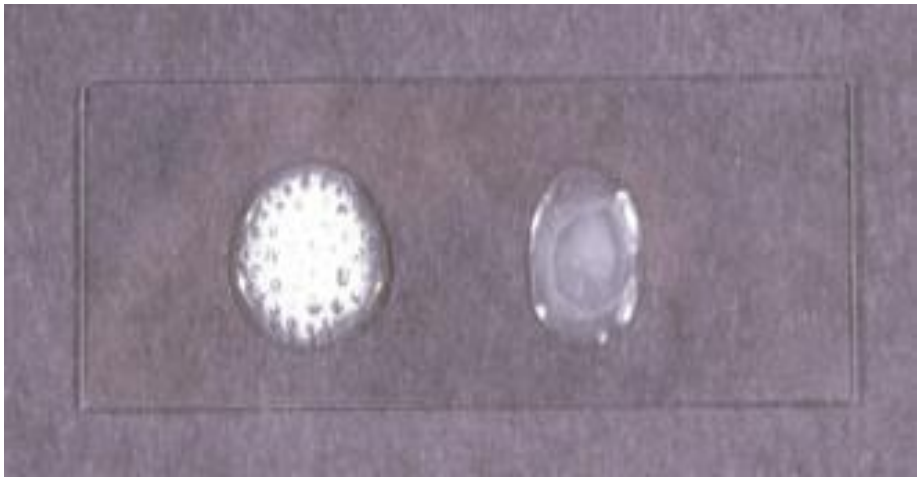
3.5.1-Catalase test

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 as streptococci. the teat is performed by taking a few colonies of the test organism, using a sterile wooden stick or glass rod and immerse it in the hydrogen

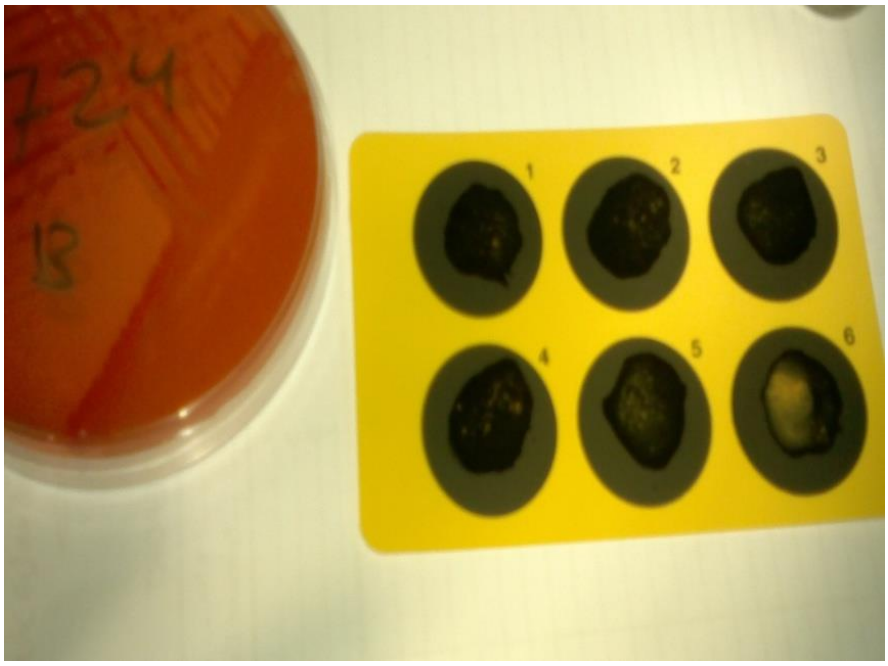
peroxide solution. bubbles of oxygen will be seen in the slid when catalase is produced by the organism.fig(4)

3.5.2-Coagulase test

Coagulase test is used to identify and differentiate *Staphylococcus aureus* from coagulase negative Staphylococci. Coagulates causes plasma to clot by converting fibrogen to fibrin. A small amount of colony/colonies of the tested organism is emulsified in a sterile test tube containing the 0.5 ml of 1:10 diluted plasma in normal saline then incubate at 35-37°C and examined each half hour up to 4-6 h Examine for a clot as a Positive result.(Fig 5).



Fig(4):Catalase test



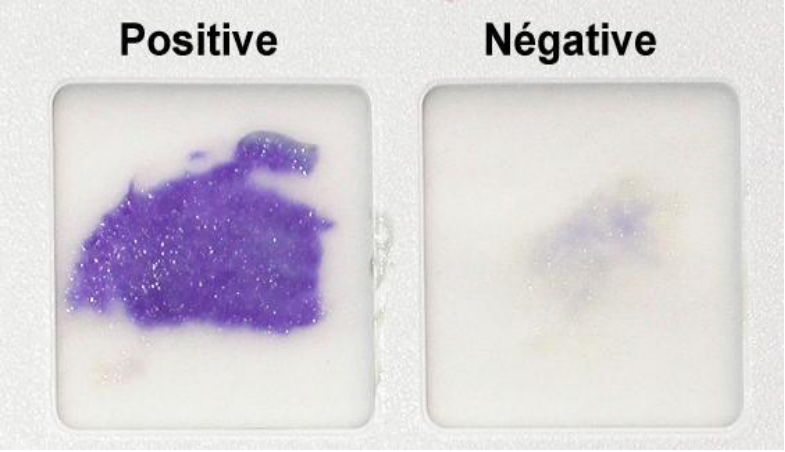
Fig(5):Coagulase test

3.5.3-Oxidase test

The Oxidase test is used in identification of Pseudomonas Neisseria, Moraxella, Campylobacter and Pasteurella species all of which produce cytochrome oxidase enzyme. The solution was prepared by dissolving 0.1g of tetramethyl-p-phenylenendiamine dihydrochloride in 10 ml 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 soaked filter paper looked for the development of blue-purple colour within a few seconds as shown in colourPositive oxidase control *:paseudomonas aeruginosa* and Negative oxdiase control : *E.coli* fig(6)

3.6-Antibiotic sensitivity tests

Isolated bacteria were tested against various antibiotics Gentamicin and Ciprofloxacin, Imipenem Vancomycin, Erythromycin, Tetracycline, Amoxicillin, Ampicillin and Penicillin. Bacterial growth were streaked on Muller Hinton agar, antibiotic discs were placed on the media, than incubated at 37 for 24h.fig(7)



Fig(6): Oxidase test

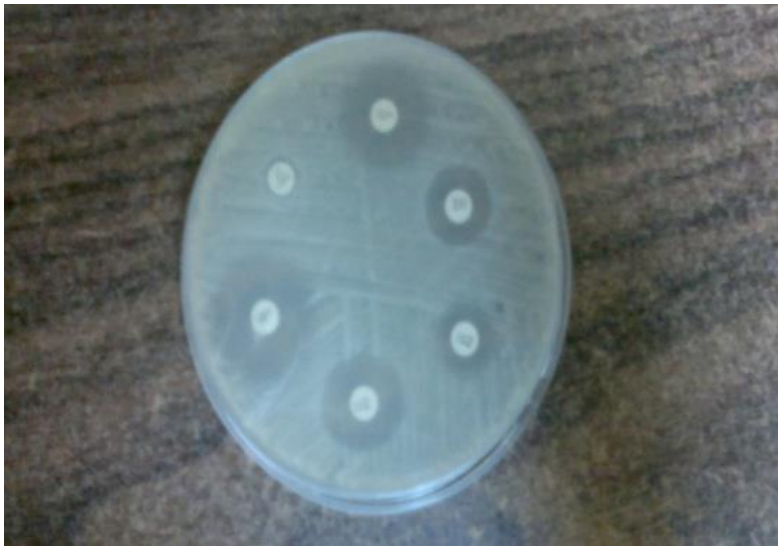


Fig (7): Antibiotic sensitivity tests

3.7-BDphoenix system

The Phoenix System (BD Biosciences, Sparks, Maryland, USA) is a fully automated identification (ID) and susceptibility test (AST) system. The identification portion of the system is based upon numerical taxonomy utilizing multiple probabilities to obtain an answer. Bacteria were confirmed by BD phoenix system in burn Hospital laboratory, Tripoli. fig (8)

3.8- Statistical analysis

Data were analyzed using statistical package for social science (spss) version 18 .descriptive statistics, as 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 from of tables and figures,were the figures done by Microsoft Excel 2003

Fig.(8):BD phoenix

CHAPTER FOUR

RESULTS

4_Results

A total of 133 burned patients were examined during the period of this study. positive cases samples showed 100 gram positive bacteria and 28 garam negative bacteria and 4 not done .

4.1- Identification of bacterial isolates

4.1.1-*Staphylococcus spp.*

In study that staphylococcus was the most prevent bacterial wound infection isolated. It produces circular and smooth colonies on blood agar (fig.10), nutrient agar. All samples examined microscopically and showed gram positive cocci and the production catalas positive(fig.4) presence of *S.aureus* was confirmed by the coagulase positive (fig.5)*s.aureus*(fig .10) gave orange colonies on MacConkey agar (fig. 9) wher as other staphylococci are coagulase negative *S.epidermidis* (fig .11) . *S.haemolyticus*(fig .12 and *S. saprophyticus*(fig .13) growth was confirmed by using BD phoenix.

Fig.(9): *S.aureus* on macConkey agar

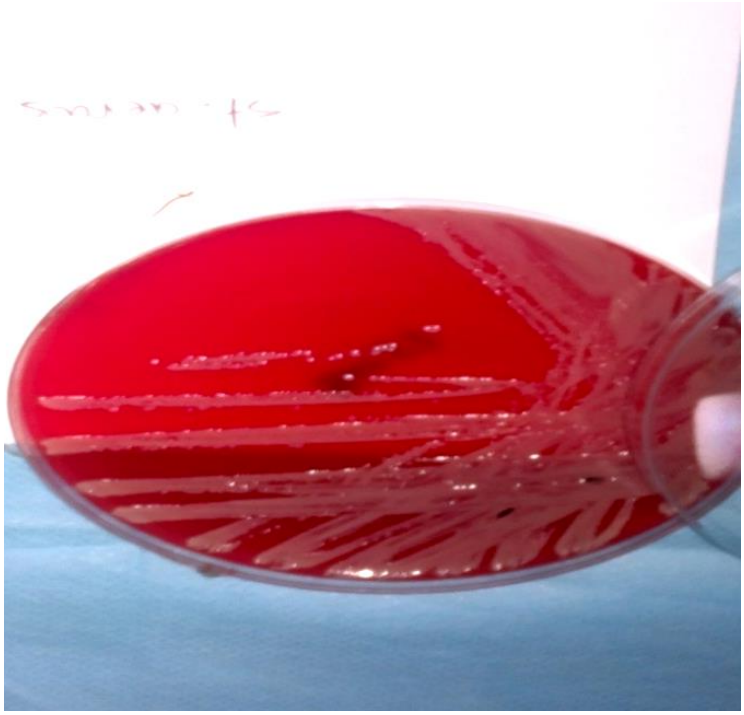


Fig.(10) *S.aureus* on blood agar

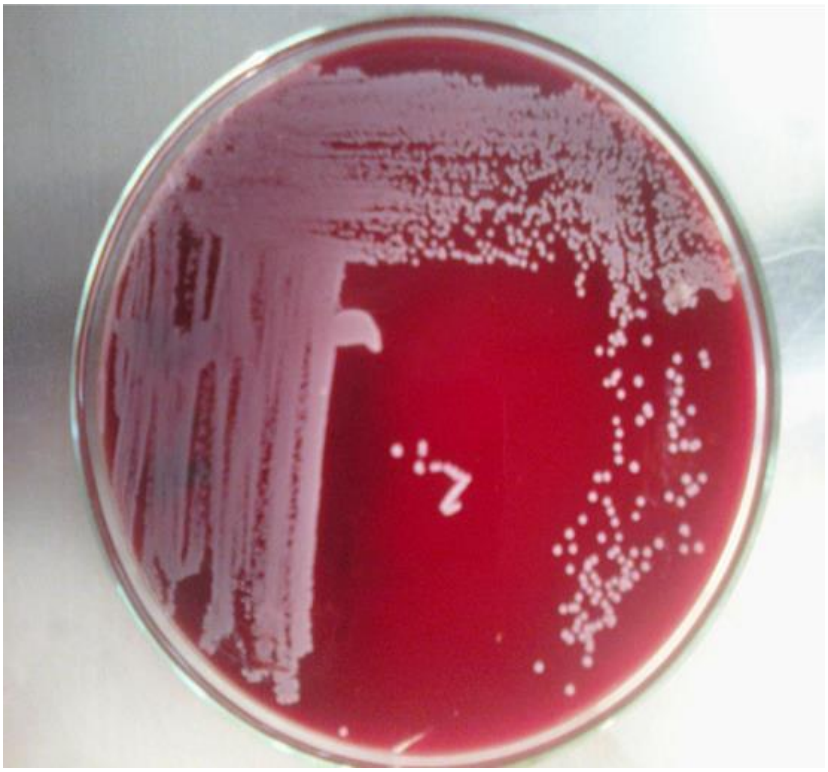
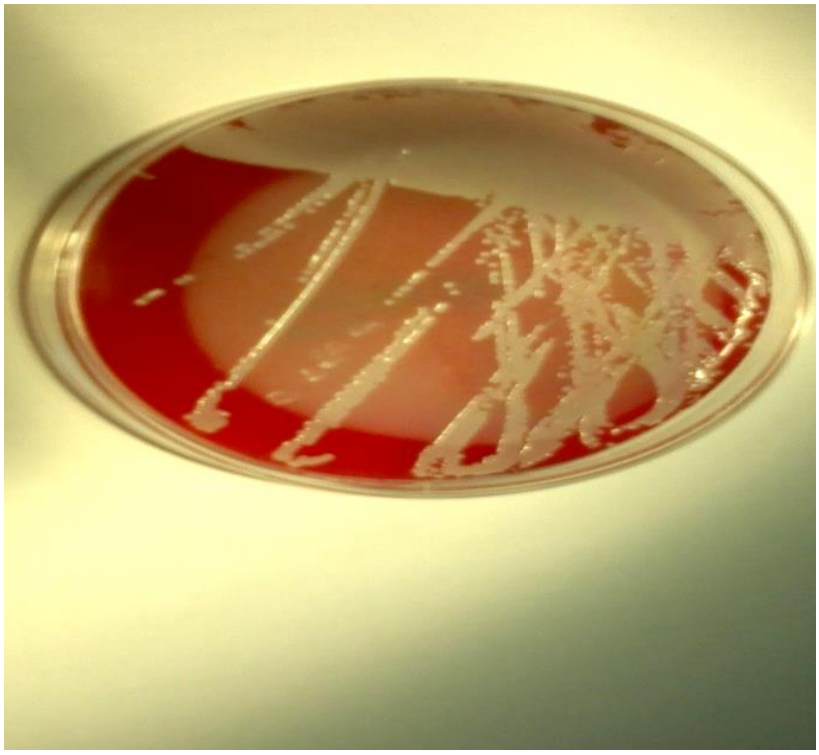
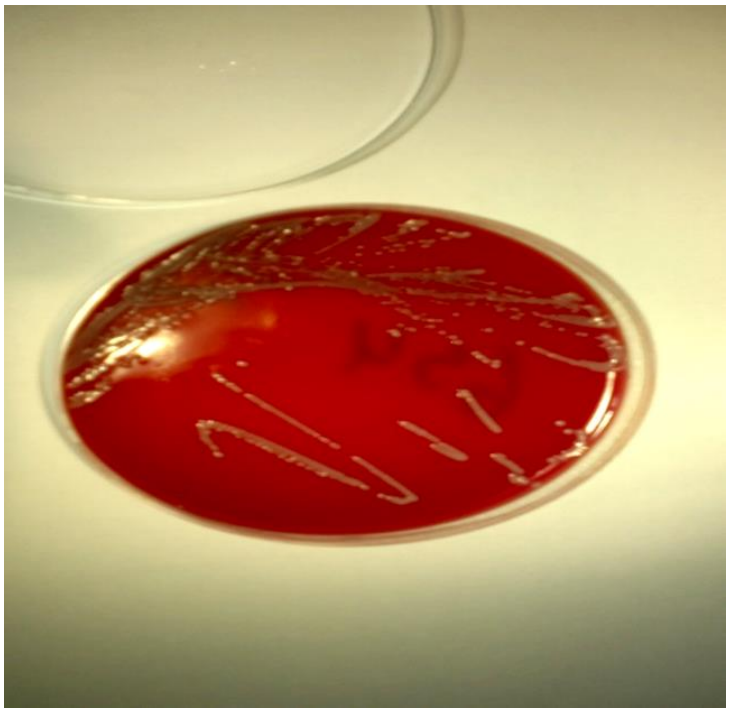


Fig (11): *S.epidermidis* on blood agar



Fig(12).*S. haemolyticus* on blood agar.



Fig(13).*S. saprophyticus* on blood agar.

4.1.2- Identification of *Pseudomonas aeruginosa*

In this study showed that *pseudomonas aeruginosa* Gram negative rods.It produces large, flat hemolytic colonies on blood agar.(fig 14)

the biochemical tests showed oxidase positive All isolates was confirmed by BD phoenix system(fig.14, 15)

4.1.3- Iaentification of *Acinetobacter paumannii*

In this study showed that *Acinetobacter paumannii* was as Gram-negative .on MacConkey agar small cream colonies was observed .the identification of this bacteria was confirmed by BDphonex system. (Fig 16, fig 17.)

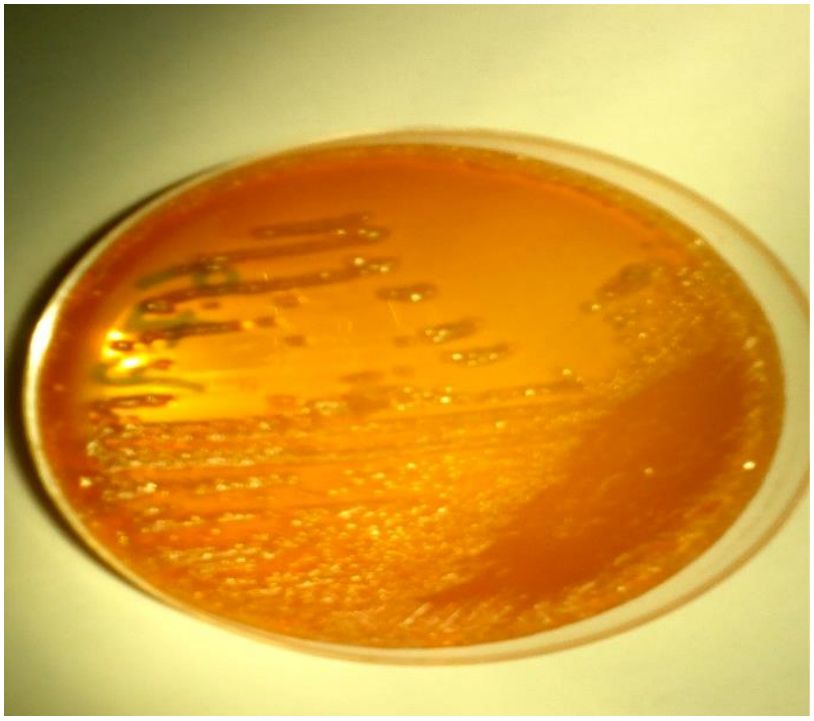


Fig.(14) *P.aeruginosa* on MacConkey agar

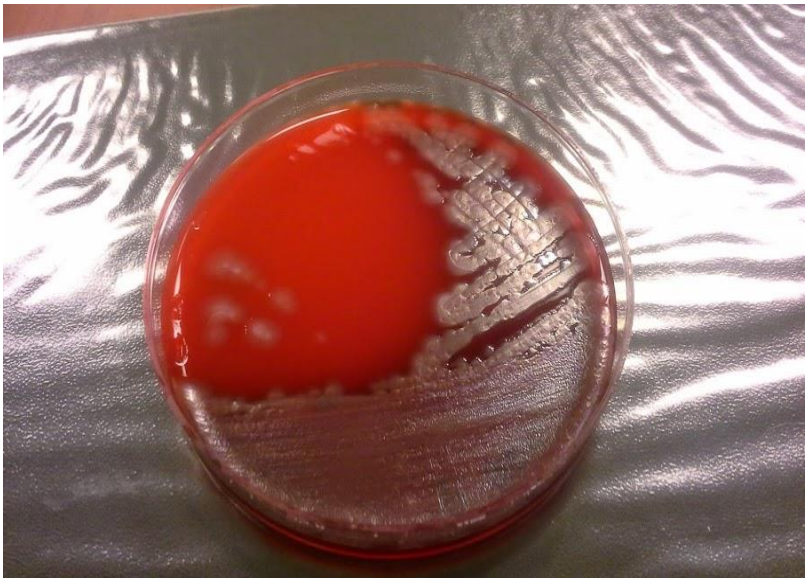
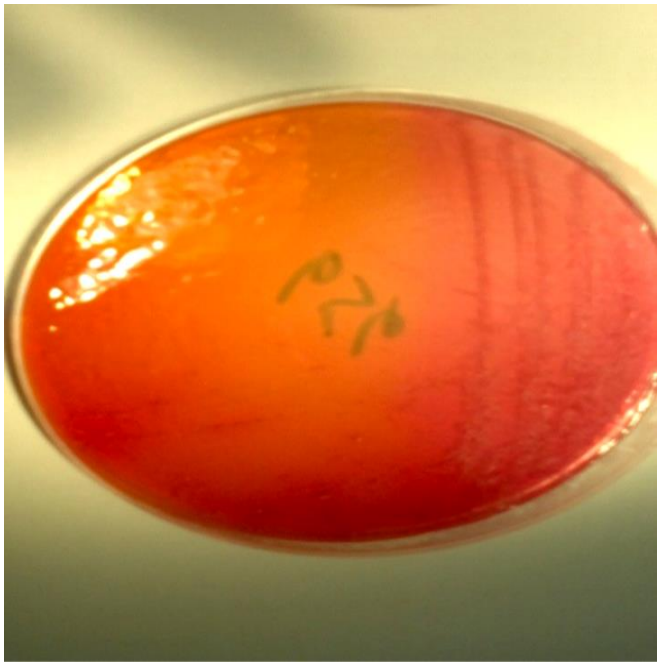


Fig.(15).*P.aeruginosa* on blood

Fig(16). *Acinetobacter paumannii* on blood agar.



Fig(17). *Acinetobacterpaumannii*iMacConkey agar

4.1.4- Idaentification of *Klebsiella pneumonia*

By Gram stain showed the isolates was Gram - negative rods. However Klebsilla sp . gav lactose fermenting mucoid pink colonies on macconkey agar (fig 18).and with biochemical reactions it produced negative oxidase test. All samples examined microscopically and showed gram negative rods Other *Klebsiella pneumonia* confirmed by using BD phoenix system.

4.1.5-*Escherichia coli*

E.coli produces smooth pink colonies on MacConky agr. All samples examined gave lactose fermenting colonies on MacConkey agar (fig19) the bacteria was Gram-negative rod shaped bacteria. *E.coli* growth was confirmed by using BD phoenix system.

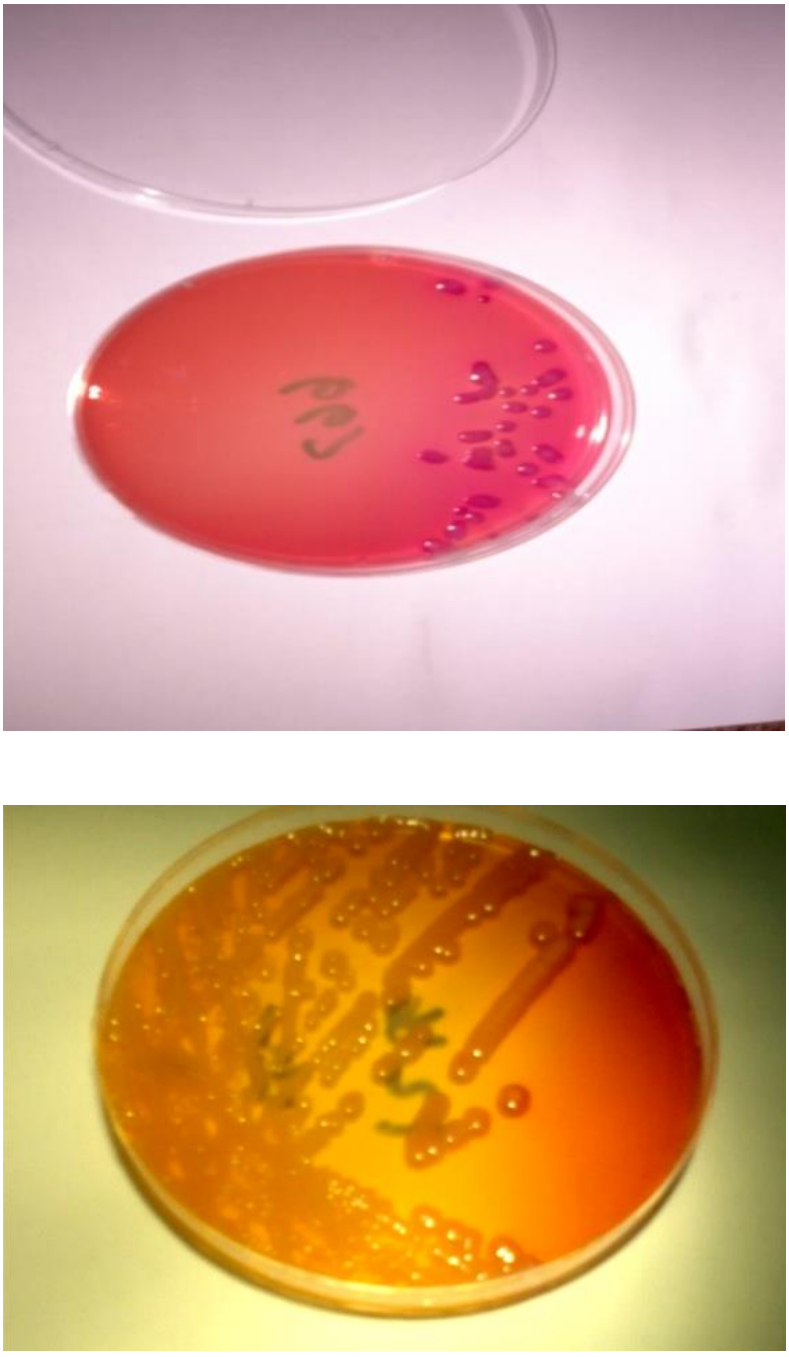


Fig.(18)*K.pneumonia* on MacConkey agar

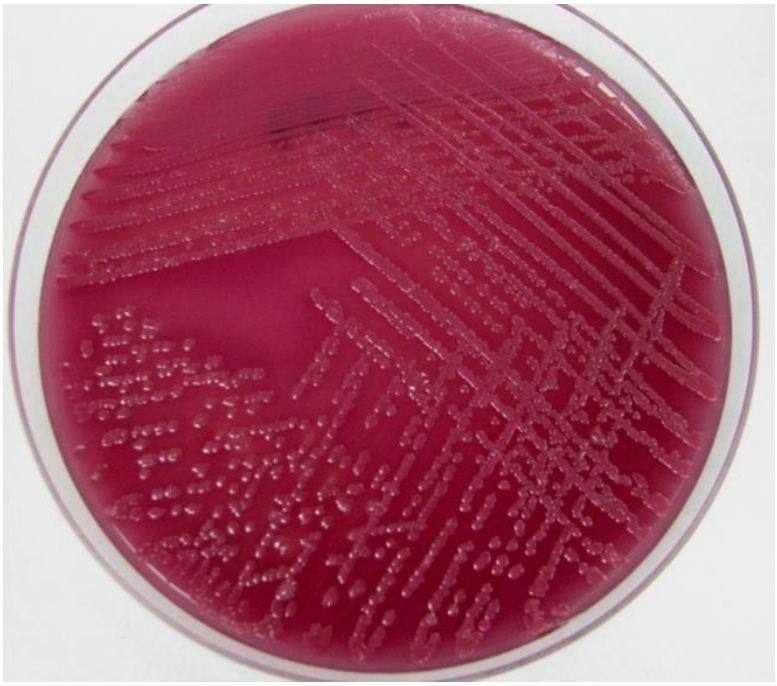


Fig.(19). *Esherichia coli*

4.1.6-*Enterobacterspp*

The Gram stain of Enterobacter appeared as Gram - negative rods.culture media showed that small smooth colonies on MacConky agar .all species was confirmed by BD phoenix system (fig .20)

4.1.7-*Proteus spp*

Was produced swarming growth on blood agar (fig 21) Microscopic examination of positive cultures showed gram negative rods. Confirmed biochemically by producing negative oxidase test, positive indol test,and confirmed by using BD phoenix system.

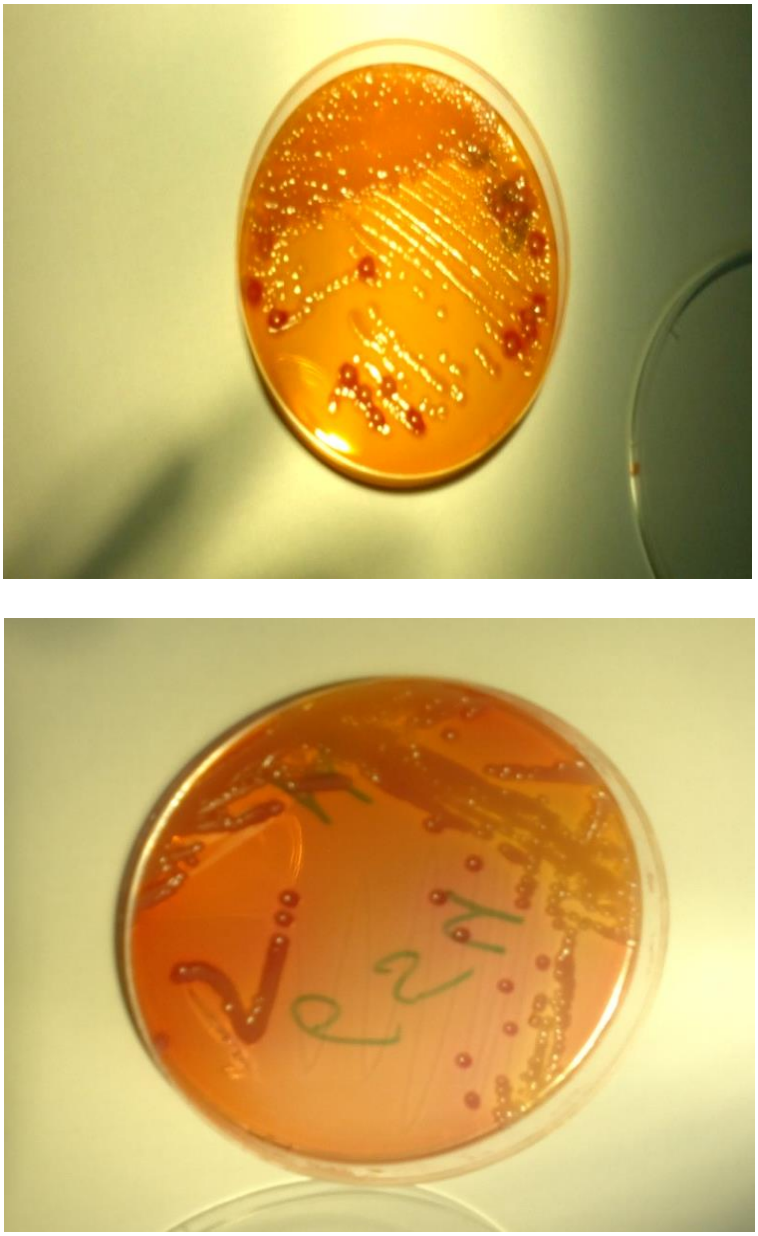


Fig (20) *Enterobacter spp*

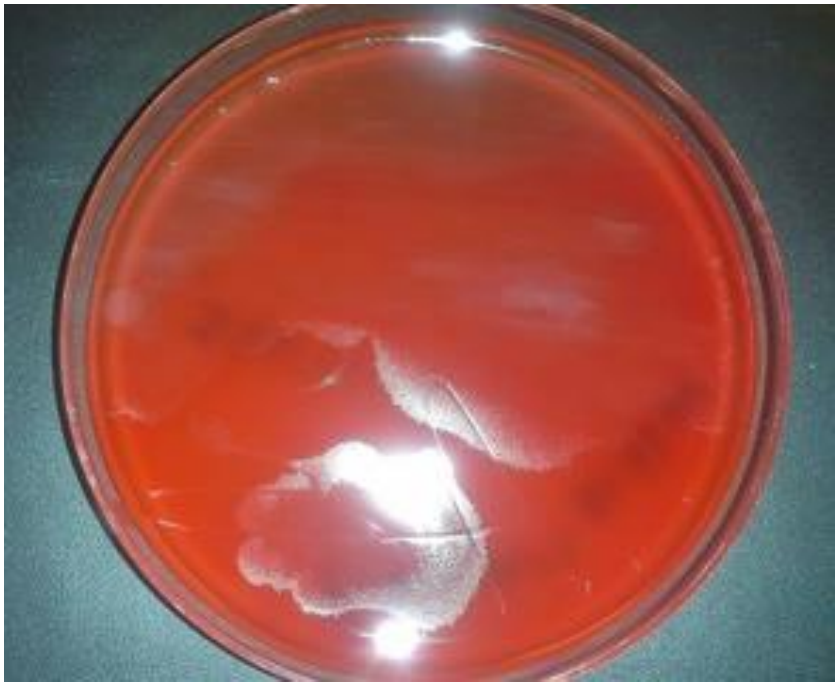


Fig.(21).*P.mirabilis* on blood agar

4.2_ Distribution of patients

4.2.1- Distribution of bacterial according Gram-negative and Gram-positive bacteria

Table (2) showed the prevalence of Gram-negative bacteri was higher than Gram positive bacteria reach to (75.2%) The most prevalence bacteria was *Acinetobacter baumannii* (37.6%) followed by *Pseudomonas aeruginosa* (23.3%) followed by *Klebsiella pneumonia* reach to (8.3%) Lowest bacteria was among observed by *Escherichia coli* and *Proteus mirabilis* reach to (3.0%); However the infection Gram-positive bacteria reach to (24.8%) ,The most prevalence bacteria was *Staphylococcus aureus* reach to (7.5%) followed by *Staphylococcus haemolyticus* (5.3%) followed by *Enterobacter cloacae* (4.5%) followed by *Staphylococcus epidermidis* (3.8%) followed by *Enterobacter aerogenes* (3.0%) Lowest bacteria was among observed by *Staphylococcus saprophyticus* reach to (.8%) (fig.22)

4.2.2-Distribution of bacteria growth in wound sample according to the age.

Table (3) showed that different bacteria was isolated according to age groups the most common bacteria was *Acinetobacter baumannii* which showed associated with age of 15-30 years represent around (18.o%) followed by *Pseudomonas aeruginosa* (8.3%) in the age groups less than 15 year followed by *Klebsiella pneumonia* (4.5%) in the age groups 15 - 30. Followed by *Staphylococcus aureus*(4.5%) among 30-45 year. *Staphylococcus haemolyticus* (2.3%) less than 15 year,*Enterobacter cloacae*(3.8%). In the age groups 15 -30,the other bacteria were shown fluctuation numbers associated with burn and are *Staphylococcus epidermidis, Proteus mirabilis, Enterobacter aerogenes ,Staphylococcus saprophyticus* ,Eschirechia coliand (fig. 23).

Table2: - Distribution of bacterial according Gram-negative and Gram-positive bacteria

Pathogenic	**Gram**		**Total** No. %
	Gram –Ve No. %	**Gram +Ve** No. %	
Pseudomonas aeruginosa	31 23.3%	0 .0%	31 23.3%
Staphylococcus haemolyticus	0 .0%	7 5.3%	7 5.3%
Staphylococcus aureus	0 .0%	10 7.5%	10 7.5%
Klebsiella pneumonia	11 8.3%	0 .0%	11 8.3%
Escherichia coli	4 3.0%	0 .0%	4 3.0%
Acinetobacter baumannii	50 37.6%	0 .0%	50 37.6%
Enterobacter aerogenes	4 3.0%	0 .0%	4 3.0%
Proteus mirabilis	4 3.0%	0 .0%	4 3.0%
Enterobacter cloacae	6 4.5%	0 .0%	6 4.5%
Staphylococcus saprophyticus	0 .0%	1 .8%	1 .8%
Staphylococcus epidermidis	0 .0%	5 3.8%	5 3.8%
Total	100 82.7%	23 17.4%	133 100.0%

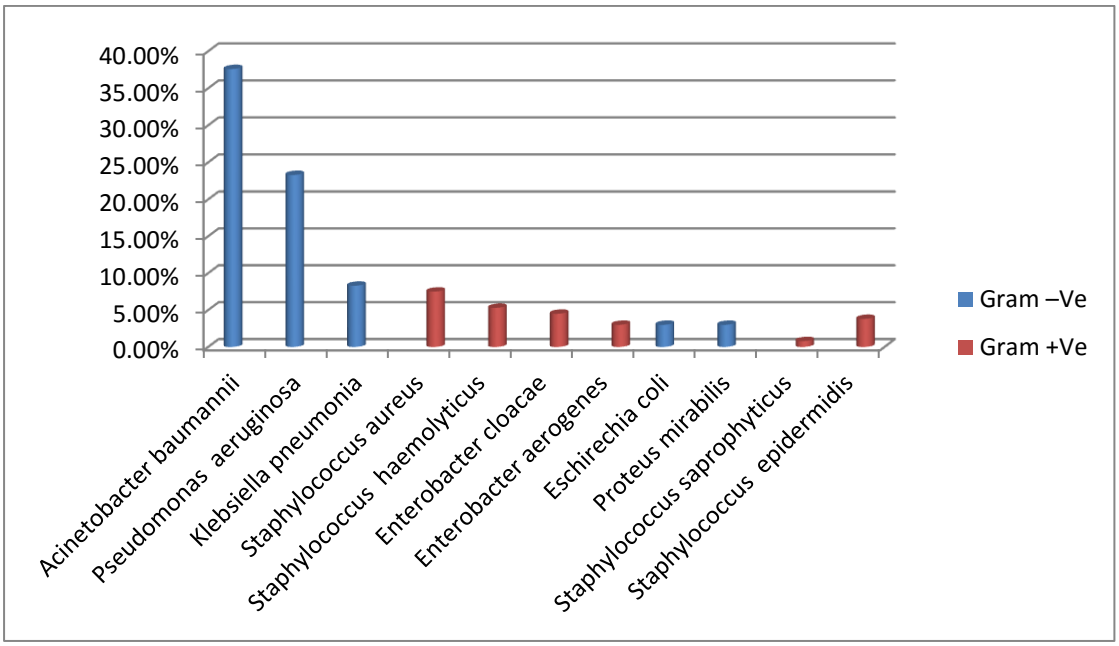


Fig (22- Distribution of bacterial according Gram-negative and Gram-positive bacteria .

Table(3)- Distribution of bacteria growth in wound sample according to the age

PATHOGEN * AGE.GR Crosstabulation

		AGE.GR					
		< 15 year	15 - 30	30 - 45	45 - 60	>= 60 year	Total
PATHOGEN	Pseudomonas aeruginosa	11	7	10	3	0	31
		8.3%	5.3%	7.5%	2.3%	.0%	23.3%
	Staphylococcus haemolyticus	3	1	2	0	1	7
		2.3%	.8%	1.5%	.0%	.8%	5.3%
	Staphylococcus aureus	3	1	6	0	0	10
		2.3%	.8%	4.5%	.0%	.0%	7.5%
	Klebsiella pneumonia	0	6	3	1	1	11
		.0%	4.5%	2.3%	.8%	.8%	8.3%
	Eschreichia coli	2	1	1	0	0	4
		1.5%	.8%	.8%	.0%	.0%	3.0%
	Acinetobacter paumannii	8	24	11	1	6	50
		6.0%	18.0%	8.3%	.8%	4.5%	37.6%
	Enterobacter aerogenes	1	3	0	0	0	4
		.8%	2.3%	.0%	.0%	.0%	3.0%
	Proteus mirabilis	1	1	0	1	1	4
		.8%	.8%	.0%	.8%	.8%	3.0%
	Enterobacter cloacae	0	5	1	0	0	6
		.0%	3.8%	.8%	.0%	.0%	4.5%
	Staphylococcus saprophyticus	1	0	0	0	0	1
		.8%	.0%	.0%	.0%	.0%	.8%
	Staphylococcus epidermidis	1	2	2	0	0	5
		.8%	1.5%	1.5%	.0%	.0%	3.8%
Total		31	51	36	6	9	133
		23.3%	38.3%	27.1%	4.5%	6.8%	100.0%

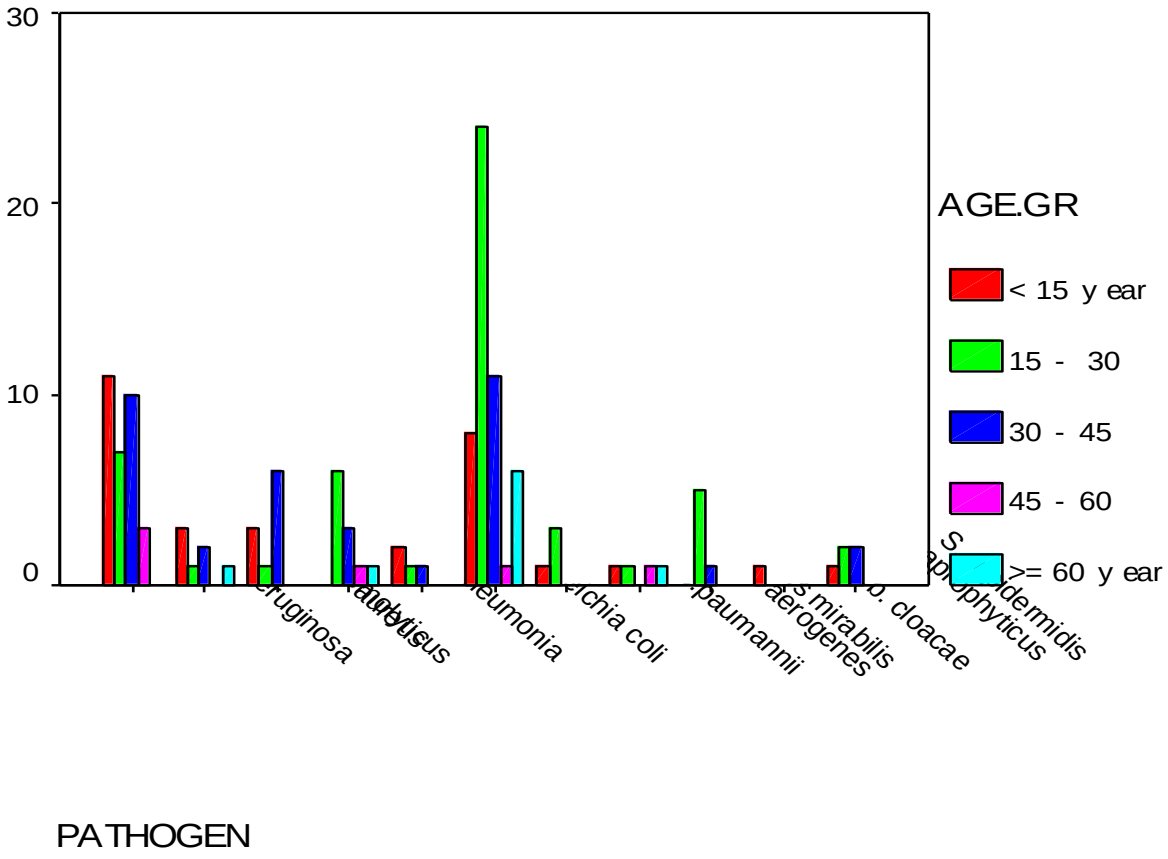


Fig.(23): Distribution of bacteria growth in wound sample according to the age

4.2.3- Distribution of bacteria according to the nationality.

Table(4) Showed that the highest bacterial infection among Libyan was *Acinetobacter baumannii* (35.3%) this bacteria were also shown to have high prevalence among Non-Libyan (2.3%) followed by *Pseudomonas aeruginosa* (19.5%) among libyan, *Klebsiella pneumonia* (7.5) *Staphylococcus aureus* (6.0%) followed by *Staphylococcus haemolyticus* reach to(4.5%) among Libyan followed by *Enterobacter cloacae* (4.5%) . followed by *Staphylococcus epidermidis*reach to (3.8%) and the lowest bacteria by *Enterobacter aerogenes,Proteus mirabilis,Eschirechia coli* reach to (3.0%) among libyan. Followed by *Staphylococcus saprophyticus* reach to (.8%).(fig.24)

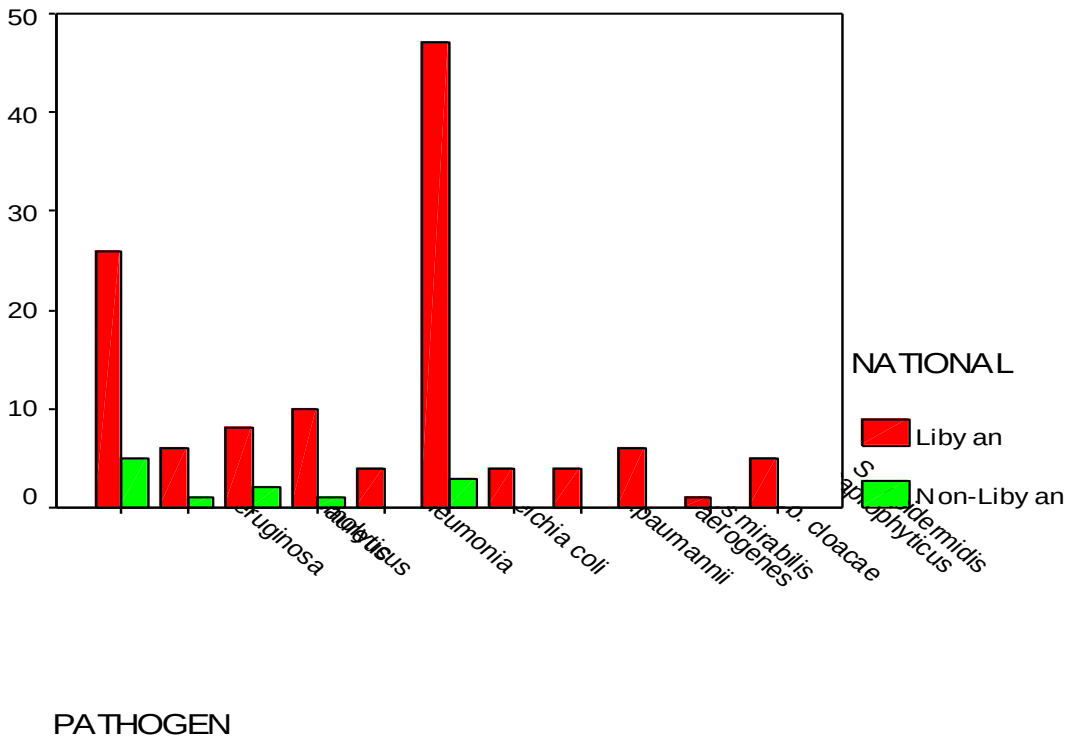
4.2.4- Distribution of isolated bacterial according to the residence

Table(5). showed that SSI of in tripoli residence was the most prevalent bacteria *Acinetobacter baumannii* (28.6%) this bacteria were also shown to have high prevalence among outside tripoli (9.0%). followed by *Pseudomonas aeruginosa*(12.8%) among Tripoli (10.5%) among outside tropoli . followed by *klebsiella pneumonia* (5.3%) among outside Tripoli (3.0%) in tripoli , followed by *Staphylococcus aureus* (4.5%) among Tripoli (3.0%) outside Tripoli followed by *Staphylococcus haemolyticus* reach to (3.8%) in Tripoli (1.5%) outside Tripoli followed by *Enterobacter cloacae* reach to (4.5%) in tripoli followed by *Staphylococcus epidermidis* reach to (3.8%) in Tripoli, followed *Eschirechia coli* and *Enterobacter aerogenes* reach to (1.5%) in both the Tripoli and Outside Tripoli. followed by *Proteus mirabilis* reach to (3.0%) in Tripoli. the lowest bacteria by*Staphylococcus saprophyticus* reach to (.8%).(fig .25)

Table(4)- Distribution of bacteria according to the nationality.

PATHOGEN * NATIONAL Crosstabulation

		NATIONAL		
		Libyan	Non-Libyan	Total
PATHOGEN	Pseudomonas aeruginosa	26 19.5%	5 3.8%	31 23.3%
	Staphylococcus haemolyticus	6 4.5%	1 .8%	7 5.3%
	Staphylococcus aureus	8 6.0%	2 1.5%	10 7.5%
	Klebsiella pneumonia	10 7.5%	1 .8%	11 8.3%
	Eschreichia coli	4 3.0%	0 .0%	4 3.0%
	Acinetobacter paumannii	47 35.3%	3 2.3%	50 37.6%
	Enterobacter aerogenes	4 3.0%	0 .0%	4 3.0%
	Proteus mirabilis	4 3.0%	0 .0%	4 3.0%
	Enterobacter cloacae	6 4.5%	0 .0%	6 4.5%
	Staphylococcus saprophyticus	1 .8%	0 .0%	1 .8%
	Staphylococcus epidermidis	5 3.8%	0 .0%	5 3.8%
Total		121 91.0%	12 9.0%	133 100.0%

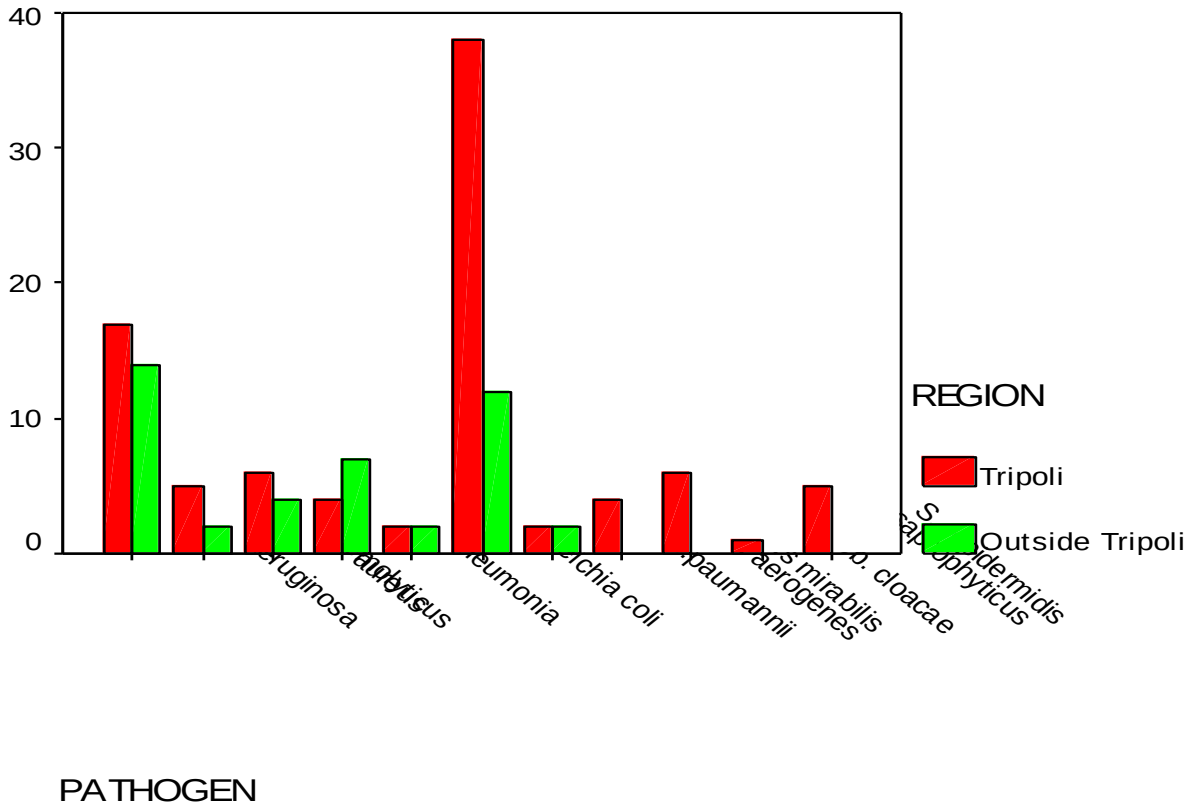


Fig(24).: Distribution of bacteria according to the nationality.

Table(5)- Distribution of isolated bacterial according to the residence

PATHOGEN * REGION Crosstabulation

		REGION		
		Tripoli	Outside Tripoli	Total
PATHOGEN	Pseudomonas aeruginosa	17 12.8%	14 10.5%	31 23.3%
	Staphylococcus haemolyticus	5 3.8%	2 1.5%	7 5.3%
	Staphylococcus aureus	6 4.5%	4 3.0%	10 7.5%
	Klebsiella pneumonia	4 3.0%	7 5.3%	11 8.3%
	Eschreichia coli	2 1.5%	2 1.5%	4 3.0%
	Acinetobacter paumannii	38 28.6%	12 9.0%	50 37.6%
	Enterobacter aerogenes	2 1.5%	2 1.5%	4 3.0%
	Proteus mirabilis	4 3.0%	0 .0%	4 3.0%
	Enterobacter cloacae	6 4.5%	0 .0%	6 4.5%
	Staphylococcus saprophyticus	1 .8%	0 .0%	1 .8%
	Staphylococcus epidermidis	5 3.8%	0 .0%	5 3.8%
Total		90 67.7%	43 32.3%	133 100.0%



Fig(25).:)- Distribution of isolated bacterial according to the residence.

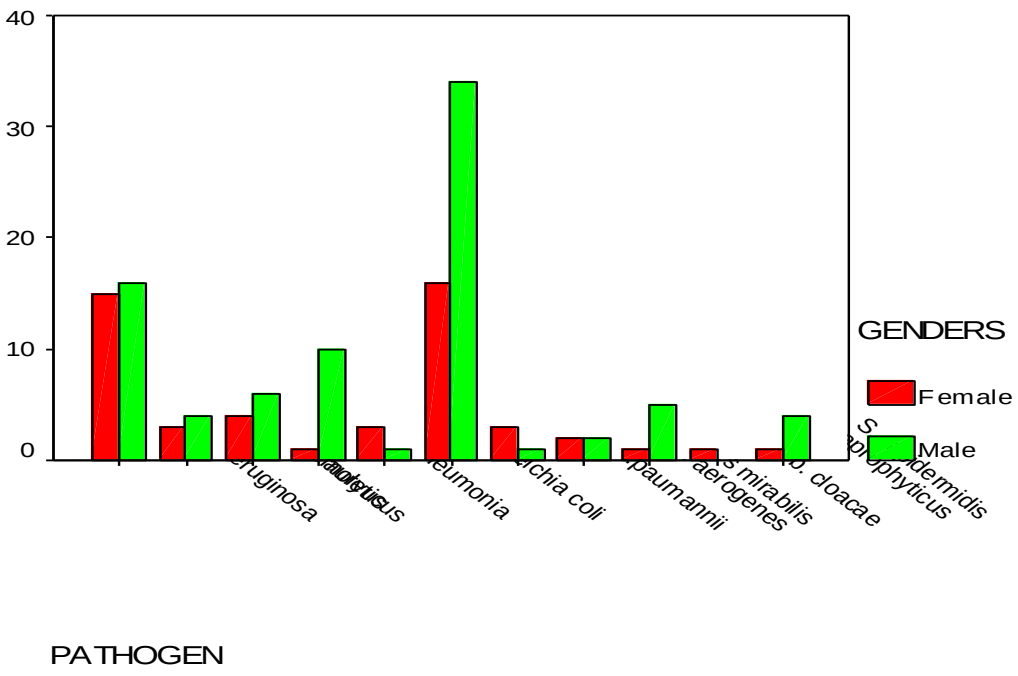
4.2.5- 4.2.5-Distribution of isolated bacteria according to the sex:

Table(6).Showed that the Microbial infection was investigated among genders in which the highest bacterial infection among male was *Acinetobacter baumannii* which found (25.6%) this bacteria were also shown to have high prevalence among female (12.0%) followed by *Pseudomonas aeruginosa* which found (12.0%) this bacteria were also shown to have high prevalence among female (11.3%). followed by male *klebsiella pneumonia* (7.5%) and female (.8%) followed by *Staphylococcus aureus* among male (4.5%) and female (3.0%) followed by *Staphylococcus haemolyticus* reach to (3.0%) in male and reach to (2.3%) in female followed by *Enterobacter cloacae* reach to (3.8%) in male and reach to (0.8%) in female followed by *Staphylococcus epidermidis* reach to (3.0%) in male and reach to (0.8%) in female followed by*Proteus mirabilis* reach to (1.5%) in both male and female .follwed by *Eschirechia coli* and *Enterobacter aerogenes*reach to (2.3%) in female and reach to (0.8%) in male the lowest bactria by *Staphylococcus sprophyticus*reach to (.8%) in female .(fig.26)

Table(6)- 4.2.5-Distribution of isolated bacteria according to the sex

PATHOGEN * GENDERS Crosstabulation

		GENDERS		
		Female	Male	Total
PATHOGEN	Pseudomonas aeruginosa	15 11.3%	16 12.0%	31 23.3%
	Staphylococcus haemolyticus	3 2.3%	4 3.0%	7 5.3%
	Staphylococcus aureus	4 3.0%	6 4.5%	10 7.5%
	Klebsiella pneumonia	1 .8%	10 7.5%	11 8.3%
	Eschreichia coli	3 2.3%	1 .8%	4 3.0%
	Acinetobacter paumannii	16 12.0%	34 25.6%	50 37.6%
	Enterobacter aerogenes	3 2.3%	1 .8%	4 3.0%
	Proteus mirabilis	2 1.5%	2 1.5%	4 3.0%
	Enterobacter cloacae	1 .8%	5 3.8%	6 4.5%
	Staphylococcus saprophyticus	1 .8%	0 .0%	1 .8%
	Staphylococcus epidermidis	1 .8%	4 3.0%	5 3.8%
Total		50 37.6%	83 62.4%	133 100.0%



Fig(26).: 4.2.5-Distribution of isolated bacteria according to the sex.

4.2.6-Distribution of isolated bacteria according to the years.

Table(7) Showed the The largest number of burned patients admitted to tripoli burn unit during the period of this study was during 2013 by bacteria *Acinetobacter baumannii* (18.1%).followed by *Pseudomonas aeruginosa* (12.8%) followed by *Staphylococcus aureus* reach to (5.3%). followed by *Klebsiella pneumonia* reach to (4.5%). followed by *Staphylococcus haemolyticus* reach to (3.0%). Followed by similar infection with *Staphylococcus epidermidis* and *Enterobacter cloacae* and *Eschirechia coli*reach to (2.3%) the lowest bacteria by *Proteus mirabilis*reach to (1.5%) and followed by *Enterobacter aerogenes* reach to (.8%).showed during 2012 the most common bacteria *was Acinetobacter baumannii* reach to (19.5%). followed by *Pseudomonas aeruginosa* reach to (10.5%) followed by *Klebsiella pneumonia* reach to (3.8%) followed by similar infection with *Staphylococcus aureus*, *Staphylococcus haemolyticus ,Enterobacter cloacae,Enterobacter aerogenes*reach to (2.3%), followed by *Staphylococcus epidermidis*, *Proteus mirabilis*reach to (1.5%) the lowest bacteria by *Staphylococcus saprophyticus* and *Eschirechia coli* reach to (.8%).(fig.27)

Table (7)- Distribution of isolated bacteria according to the years.

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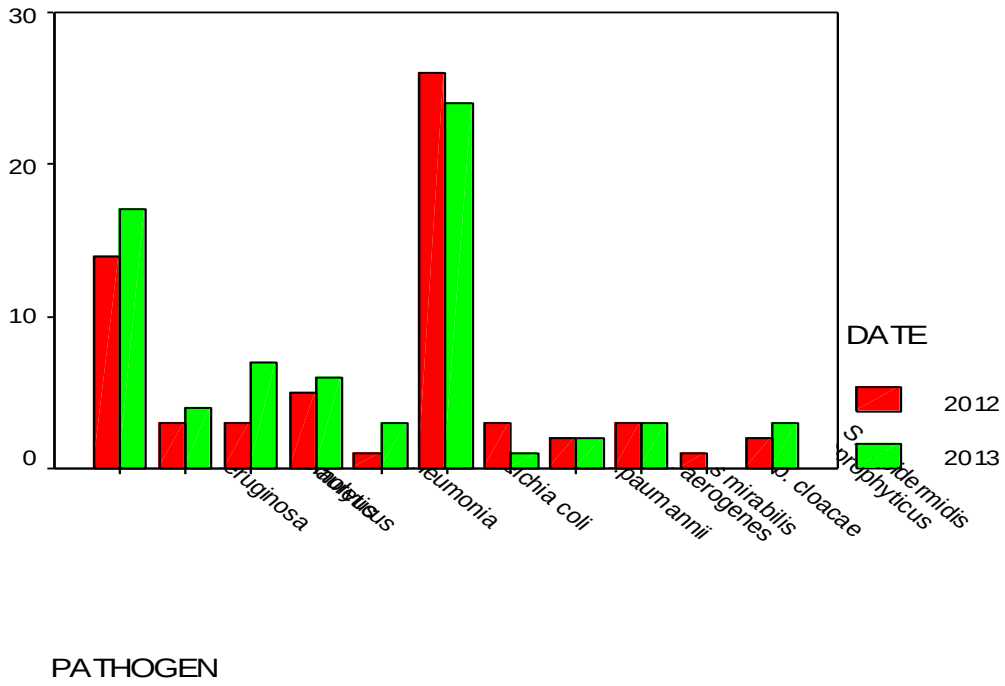
PATHOGEN * DATE Crosstabulation

		DATE		
		2012	2013	Total
PATHOGEN	Pseudomonas aeruginosa	14 10.5%	17 12.8%	31 23.3%
	Staphylococcus haemolyticus	3 2.3%	4 3.0%	7 5.3%
	Staphylococcus aureus	3 2.3%	7 5.3%	10 7.5%
	Klebsiella pneumonia	5 3.8%	6 4.5%	11 8.3%
	Eschreichia coli	1 .8%	3 2.3%	4 3.0%
	Acinetobacter paumannii	26 19.5%	24 18.0%	50 37.6%
	Enterobacter aerogenes	3 2.3%	1 .8%	4 3.0%
	Proteus mirabilis	2 1.5%	2 1.5%	4 3.0%
	Enterobacter cloacae	3 2.3%	3 2.3%	6 4.5%
	Staphylococcus saprophyticus	1 .8%	0 .0%	1 .8%
	Staphylococcus epidermidis	2 1.5%	3 2.3%	5 3.8%
Total		63 47.4%	70 52.6%	133 100.0%



Fig(27).: Distribution of isolated bacteria according to the years.

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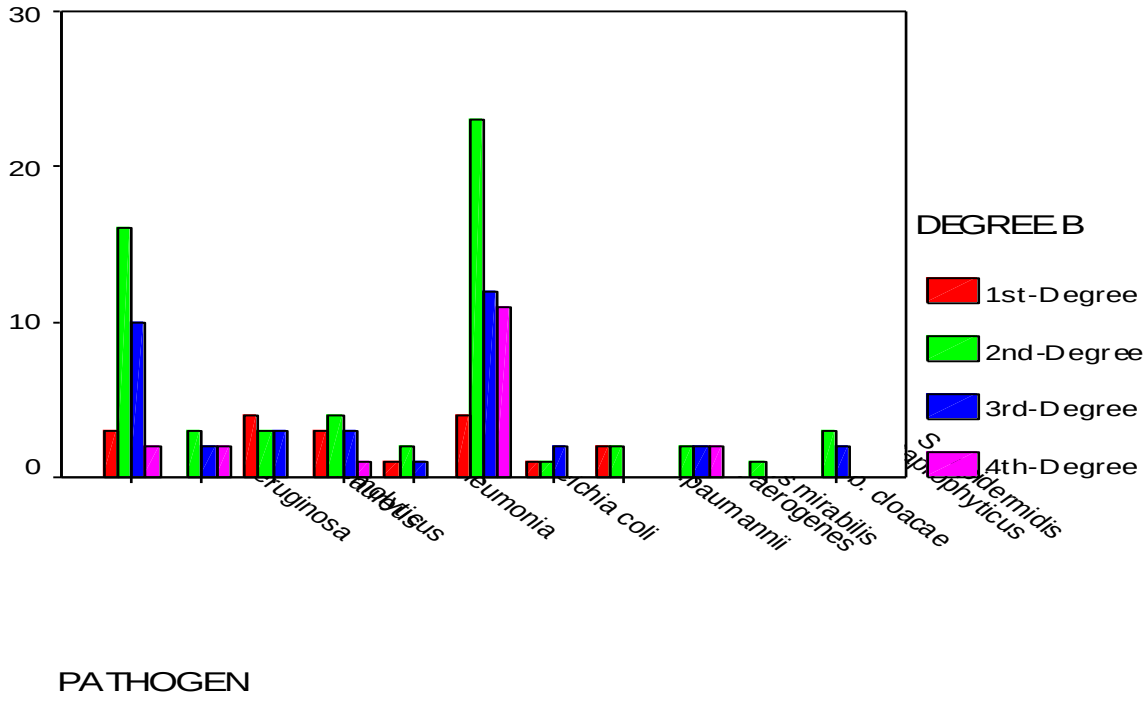
4.2.7- Distribution of bacterial according to the degree of burn

Table (8). showed that the most prevalent bacteria among second - degree burns in care units patients. was *Acinetobacter baumannii* reach to (17.3%). The lowest percentage was observed (3.0%)in first-degree. followed by *pseudomonas aeruginosa* (12.0%) insecond – degreeand the lowest (1.5%) in fourth-degree ,and followed by *Klebsiella pneumonia* (3.0%) in second – degree and the lowest (o.8%) in fourth-degree. followed by *Staphylococcus aureus* (3.0%) in first- degree and was observed in both second - degree and third-degree reach to (2.3%). followed by *Staphylococcus haemolyticus* reach to (2.3%) in second-degree and the lowest (1.5%) in both third-degree and fourth-degree . followed by *Enterobacter cloacae* reach to (1.5%) in both second-degree and third-degree and fourth-degree followed by *Staphylococcus epidermidis* reach to (2.3%) in second-degree and lowest (1.5%) in third-degree . followed by *Enterobacter aerogenes* reach t o(1.5%) in third-degree and lowest (0.8%) in both first - degree and second-degree . followed by Proteus mirabilis reach t o(1.5%) in first- degree and second-degree followed by Escherichia coli reach to (1.5%) in second-degree and lowest (0.8%) in both first - degree and first- degree the lowest bacteri by *Staphylococcus saprophyticus* reach to (0.8%) in second-degree .(fig.28)

Table(8)- Distribution of bacterial according to the degree of burn.

PATHOGEN * DEGREE.B Crosstabulation

		DEGREE.B				
		1st-Degree	2nd-Degree	3rd-Degree	4th-Degree	Total
PATHOGEN	Pseudomonas aeruginosa	3	16	10	2	31
		2.3%	12.0%	7.5%	1.5%	23.3%
	Staphylococcus haemolyticus	0	3	2	2	7
		.0%	2.3%	1.5%	1.5%	5.3%
	Staphylococcus aureus	4	3	3	0	10
		3.0%	2.3%	2.3%	.0%	7.5%
	Klebsiella pneumonia	3	4	3	1	11
		2.3%	3.0%	2.3%	.8%	8.3%
	Eschreichia coli	1	2	1	0	4
		.8%	1.5%	.8%	.0%	3.0%
	Acinetobacter paumannii	4	23	12	11	50
		3.0%	17.3%	9.0%	8.3%	37.6%
	Enterobacter aerogenes	1	1	2	0	4
		.8%	.8%	1.5%	.0%	3.0%
	Proteus mirabilis	2	2	0	0	4
		1.5%	1.5%	.0%	.0%	3.0%
	Enterobacter cloacae	0	2	2	2	6
		.0%	1.5%	1.5%	1.5%	4.5%
	Staphylococcus saprophyticus	0	1	0	0	1
		.0%	.8%	.0%	.0%	.8%
	Staphylococcus epidermidis	0	3	2	0	5
		.0%	2.3%	1.5%	.0%	3.8%
Total		18	60	37	18	133
		13.5%	45.1%	27.8%	13.5%	100.0%



Fig(28).: Distribution of bacterial according to the degree of burn

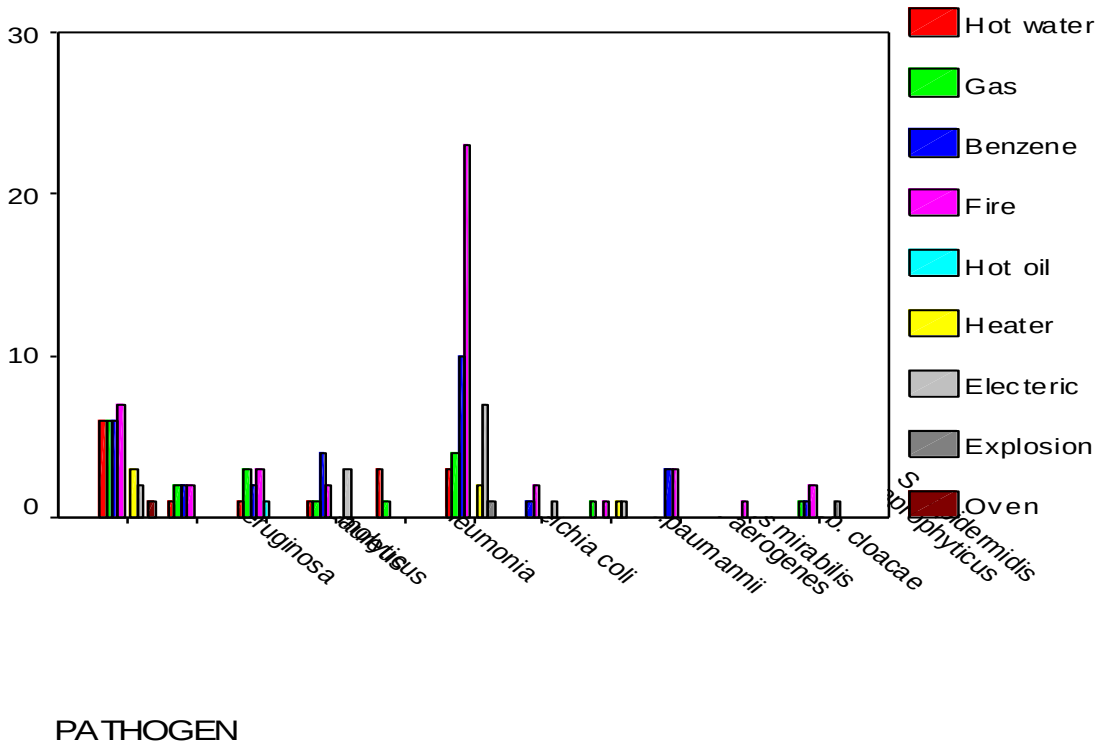
4.2.8- Distribution of isolated bacteria according to causes of burn

Table(9)Shows in this study type of causes of burn the most causes was by fire reach to (34.6%) the most common bacteria was *Acinetobacter baumannii* reach to (17.3%) and the lowest bacteria by *Staphylococcus saprophyticus* and *Proteus mirabilis* reach to (0.8%). the followed causes of benzene reach to (21.8%) the most bacteria by *Acinetobacter baumannii* reach to (7.5%) and the lowest bacteria by *Staphylococcusepidermidis* and *Enterobacter aerogenes* reach to (0.8%).followed by cases Gas reach to (14.3%) the most bacteria by *Pseudomonas aeruginosa* reach to (4.5%) and the lowest bacteria by *Staphylococcus epidermidis* and *Proteus mirabilis* and *Klebsiella pneumonia* and *Eschirechia coli* reach to (0.8%) followed by cases hot water reach to (11.3%) the most bacteria by *Pseudomonas aeruginosa* (4.5%) the lowest bacteria by *Staphylococcus haemolyticus* and *Staphylococcusaureus* and *Klebsiella pneumonia* reach to (0.8%).and followed cases by Electrical reach to (4.5%) the most bacteria by *Acinetobacter baumannii* reach to (5.3%) and the lowest bacteria by *Enterobacter aerogenes* and *Proteus mirabilis* reach to (0.8%).the followed cases by heater reach to (4.5%) the most bacteria by *Pseudomonas aeruginosa* (2.3%) and lowest bacteria by Proteus mirabilis reach to (0.8%) the followed cases by Explosion reach to (1.5%) and the most bacteria by *Acinetobacter baumannii* and *Staphylococcus saprophyticusin* both reach to (0.8%). the lowest causes by hot oil reach to (.8%) the most bacteria by *Staphylococcus aureus* (0.8%) and causes oven reach to (0.8%) the most bactria *Pseudomonas aeruginosa* reach to (0.8%) .(fig. 29)

Table(9)- Distribution of isolated bacteria according to causes of burn

PATHOGEN * REASON Crosstabulation

		REASON									
		Hot water	Gas	Benzene	Fire	Hot oil	Heater	Electeric	Explosion	Oven	Total
PATHOGEN	Pseudomonas aeruginosa	6	6	6	7	0	3	2	0	1	31
		4.5%	4.5%	4.5%	5.3%	.0%	2.3%	1.5%	.0%	.8%	23.3%
	Staphylococcus haemolyticus	1	2	2	2	0	0	0	0	0	7
		.8%	1.5%	1.5%	1.5%	.0%	.0%	.0%	.0%	.0%	5.3%
	Staphylococcus aureus	1	3	2	3	1	0	0	0	0	10
		.8%	2.3%	1.5%	2.3%	.8%	.0%	.0%	.0%	.0%	7.5%
	Klebsiella pneumonia	1	1	4	2	0	0	3	0	0	11
		.8%	.8%	3.0%	1.5%	.0%	.0%	2.3%	.0%	.0%	8.3%
	Eschreichia coli	3	1	0	0	0	0	0	0	0	4
		2.3%	.8%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	3.0%
	Acinetobacter paumannii	3	4	10	23	0	2	7	1	0	50
		2.3%	3.0%	7.5%	17.3%	.0%	1.5%	5.3%	.8%	.0%	37.6%
	Enterobacter aerogenes	0	0	1	2	0	0	1	0	0	4
		.0%	.0%	.8%	1.5%	.0%	.0%	.8%	.0%	.0%	3.0%
	Proteus mirabilis	0	1	0	1	0	1	1	0	0	4
		.0%	.8%	.0%	.8%	.0%	.8%	.8%	.0%	.0%	3.0%
	Enterobacter cloacae	0	0	3	3	0	0	0	0	0	6
		.0%	.0%	2.3%	2.3%	.0%	.0%	.0%	.0%	.0%	4.5%
	Staphylococcus saprophyticus	0	0	0	1	0	0	0	0	0	1
		.0%	.0%	.0%	.8%	.0%	.0%	.0%	.0%	.0%	.8%
	Staphylococcus epidermidis	0	1	1	2	0	0	0	1	0	5
		.0%	.8%	.8%	1.5%	.0%	.0%	.0%	.8%	.0%	3.8%
Total		15	19	29	46	1	6	14	2	1	133
		11.3%	14.3%	21.8%	34.6%	.8%	4.5%	10.5%	1.5%	.8%	100.0%



Fig(29).: Distribution of bacterial growth in wound sample according to causes.

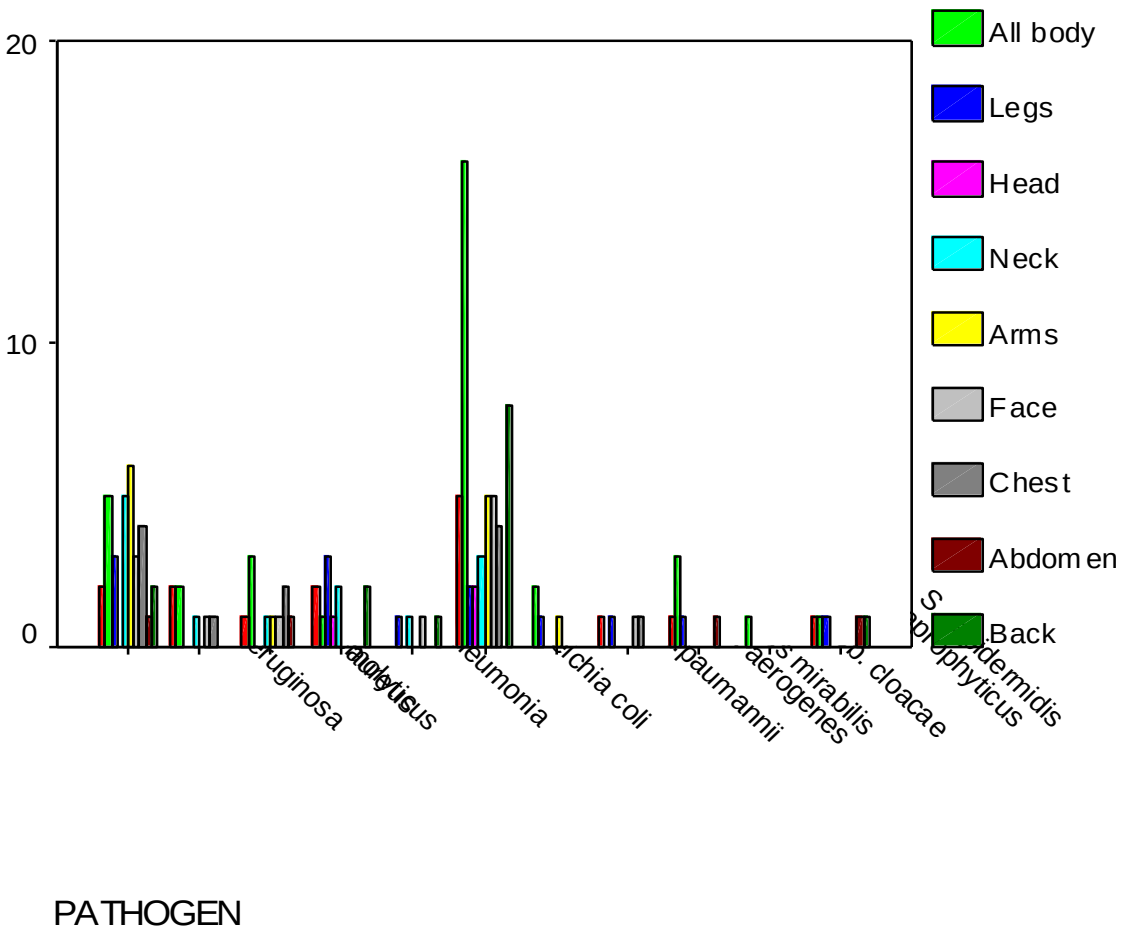
4.2.9- Distribution of bacteria growth in wound sample according to site of burn

Table (10). showed the most common site of burn was all body reach to (25.6%) and the most bacteria isolated by *Acinetobacter baumannii* reach to (12.0%) and the lowest bacteria by *Staphylococcus saprophyticus* and *Staphylococcus epidermidis* and *Klebsiella pneumonia* reach to (0.8%).the followed site by hands reach to (11.3%) the most bacteria isolated by *Acinetobacter baumannii* reach to (3.8%) the lowest bacteria by *Staphylococcus epidermidis* and *Enterobacter cloacae* and *Proteus mirabilis* and *Staphylococcus aureus* reach to (.8%) . the followed site by back reach to (10.5).the most bacteria isolated by (6.0%) and the lowest bacteria by *Escherichia coli* and *Staphylococcus epidermidis* reach to (0.8%) . the followed site by arms and legs and neck reach to (9.8%). the most bacteria isolated in arms by *Pseudomonas aeruginosa* (4.5%) and the lowest bacteria by *Staphylococcus aureus* and *Enterobacter aerogenes*(0.8%) and the most bacteria in legs by *Pseudomonas aeruginosa* and*Klebsiella pneumonia* reach to (2.3%) and the lowest bacteria by *Escherichia coli* and *Enterobacter aerogenes*, *Proteus mirabilis* ,*Enterobacter cloacae*,*Staphylococcus epidermidis* reach to (0.8%). (fig. 30)

Table(10)- Distribution of bacteria growth in wound sample according to site of burn

PATHOGEN * SITE Crosstabulation

		SITE										
		Hands	All body	Legs	Head	Neck	Arms	Face	Chest	Abdomen	Back	Total
PATHOGEN	Pseudomonas aeruginosa	2	5	3	0	5	6	3	4	1	2	31
		1.5%	3.8%	2.3%	.0%	3.8%	4.5%	2.3%	3.0%	.8%	1.5%	23.3%
	Staphylococcus haemolyticus	2	2	0	0	1	0	1	1	0	0	7
		1.5%	1.5%	.0%	.0%	.8%	.0%	.8%	.8%	.0%	.0%	5.3%
	Staphylococcus aureus	1	3	0	0	1	1	1	2	1	0	10
		.8%	2.3%	.0%	.0%	.8%	.8%	.8%	1.5%	.8%	.0%	7.5%
	Klebsiella pneumonia	2	1	3	1	2	0	0	0	0	2	11
		1.5%	.8%	2.3%	.8%	1.5%	.0%	.0%	.0%	.0%	1.5%	8.3%
	Eschreichia coli	0	0	1	0	1	0	1	0	0	1	4
		.0%	.0%	.8%	.0%	.8%	.0%	.8%	.0%	.0%	.8%	3.0%
	Acinetobacter paumannii	5	16	2	2	3	5	5	4	0	8	50
		3.8%	12.0%	1.5%	1.5%	2.3%	3.8%	3.8%	3.0%	.0%	6.0%	37.6%
	Enterobacter aerogenes	0	2	1	0	0	1	0	0	0	0	4
		.0%	1.5%	.8%	.0%	.0%	.8%	.0%	.0%	.0%	.0%	3.0%
	Proteus mirabilis	1	0	1	0	0	0	1	1	0	0	4
		.8%	.0%	.8%	.0%	.0%	.0%	.8%	.8%	.0%	.0%	3.0%
	Enterobacter cloacae	1	3	1	0	0	0	0	0	1	0	6
		.8%	2.3%	.8%	.0%	.0%	.0%	.0%	.0%	.8%	.0%	4.5%
	Staphylococcus saprophyticus	0	1	0	0	0	0	0	0	0	0	1
		.0%	.8%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	.0%	.8%
	Staphylococcus epidermidis	1	1	1	0	0	0	0	0	1	1	5
		.8%	.8%	.8%	.0%	.0%	.0%	.0%	.0%	.8%	.8%	3.8%
Total		15	34	13	3	13	13	12	12	4	14	133
		11.3%	25.6%	9.8%	2.3%	9.8%	9.8%	9.0%	9.0%	3.0%	10.5%	100.0%



Fig(30).: Distribution of bacteria growth in wound sample according to site of burn

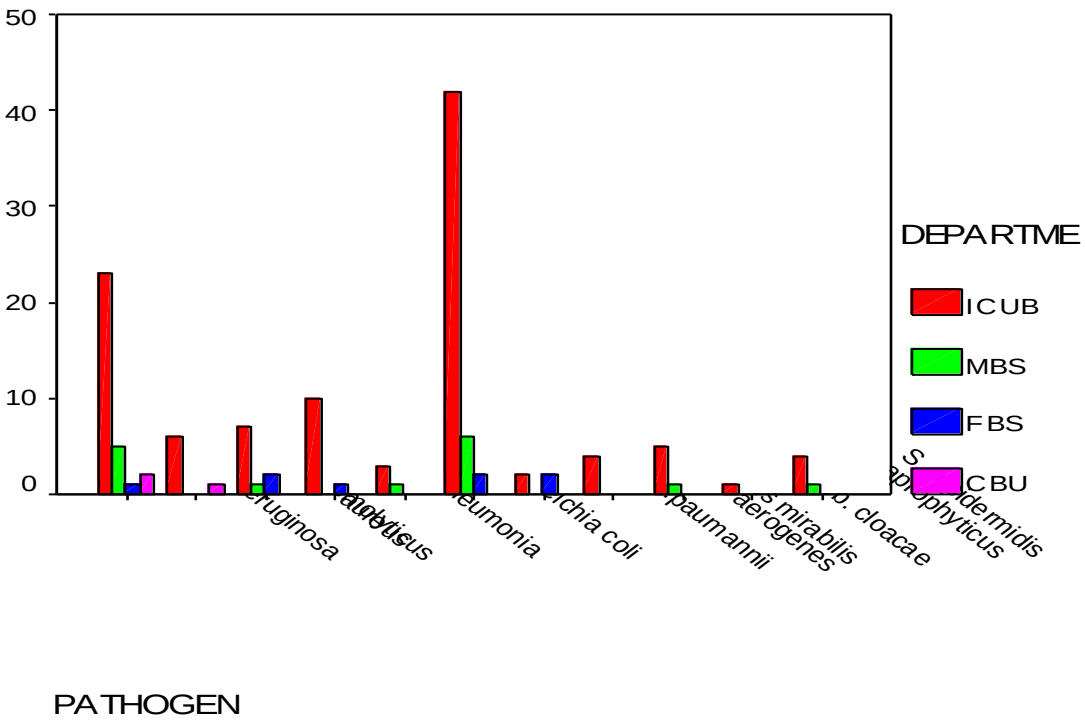
4.2.10- Distribution of bacteria growth in wound sample according to department admitted to burn

Table (11). Showed the distribution of Bacterial Growth from burn wound of The high frequency between departments was observed in ICUB unit the most bacteria was *Acinetobacter baumannii* reach to (31.6%) followed by *Pseudomonas aeruginosa* reach to (17.3%) followed by *Klebsiellapneumonia* (7.5%) *Staphylococcus aureus* (5.3%),*Staphylococcushaemolyticus*(4.5%) ,*Enterobacter cloacae* (3.8%).*Proteus mirabilisandStaphylococcusepidermidis*reach to (3.0%) *Enterobacteraerogenes* (1.5%), the lowest bacteria by *Staphylococcus saprophyticus* (0.8%). While in MBS unit the high percentage was observed by *Acinetobacter baumannii*reach to (4.5%) followed by*Pseudomonas aeruginosa* (3.8%) followed by The lowest percentage was *Staphylococcus aureus,Escherichia coli , Enterobacter cloacae, Staphylococcusepidermidis* (0.8%) While in FBS unit the the high percentage was observed by *Acinetobacter baumannii* and *Enterobacter aerogenes and Staphylococcus aureus*reach to (1.5%) the lowest percentage was *Pseudomonas aeruginosa, ,Klebsiella pneumonia* reach to (0.8%). Finally the lower bacterial was observed in CBU was observed by *Pseudomonas aeruginosa* reach to (1.5%) and *Staphylococcusepidermidis*reach to (0.8%) .(fig .31)

Table(11)- Distribution of bacteria growth in wound sample according to department admitted to burn.

PATHOGEN * DEPARTME Crosstabulation

		DEPARTME				
		ICUB	MBS	FBS	CBU	Total
PATHOGEN	Pseudomonas aeruginosa	23 17.3%	5 3.8%	1 .8%	2 1.5%	31 23.3%
	Staphylococcus haemolyticus	6 4.5%	0 .0%	0 .0%	1 .8%	7 5.3%
	Staphylococcus aureus	7 5.3%	1 .8%	2 1.5%	0 .0%	10 7.5%
	Klebsiella pneumonia	10 7.5%	0 .0%	1 .8%	0 .0%	11 8.3%
	Eschreichia coli	3 2.3%	1 .8%	0 .0%	0 .0%	4 3.0%
	Acinetobacter paumannii	42 31.6%	6 4.5%	2 1.5%	0 .0%	50 37.6%
	Enterobacter aerogenes	2 1.5%	0 .0%	2 1.5%	0 .0%	4 3.0%
	Proteus mirabilis	4 3.0%	0 .0%	0 .0%	0 .0%	4 3.0%
	Enterobacter cloacae	5 3.8%	1 .8%	0 .0%	0 .0%	6 4.5%
	Staphylococcus saprophyticus	1 .8%	0 .0%	0 .0%	0 .0%	1 .8%
	Staphylococcus epidermidis	4 3.0%	1 .8%	0 .0%	0 .0%	5 3.8%
Total		107 80.5%	15 11.3%	8 6.0%	3 2.3%	133 100.0%



Fig(31).: Distribution of bacteria growth in wound sample according to department admitted to burn

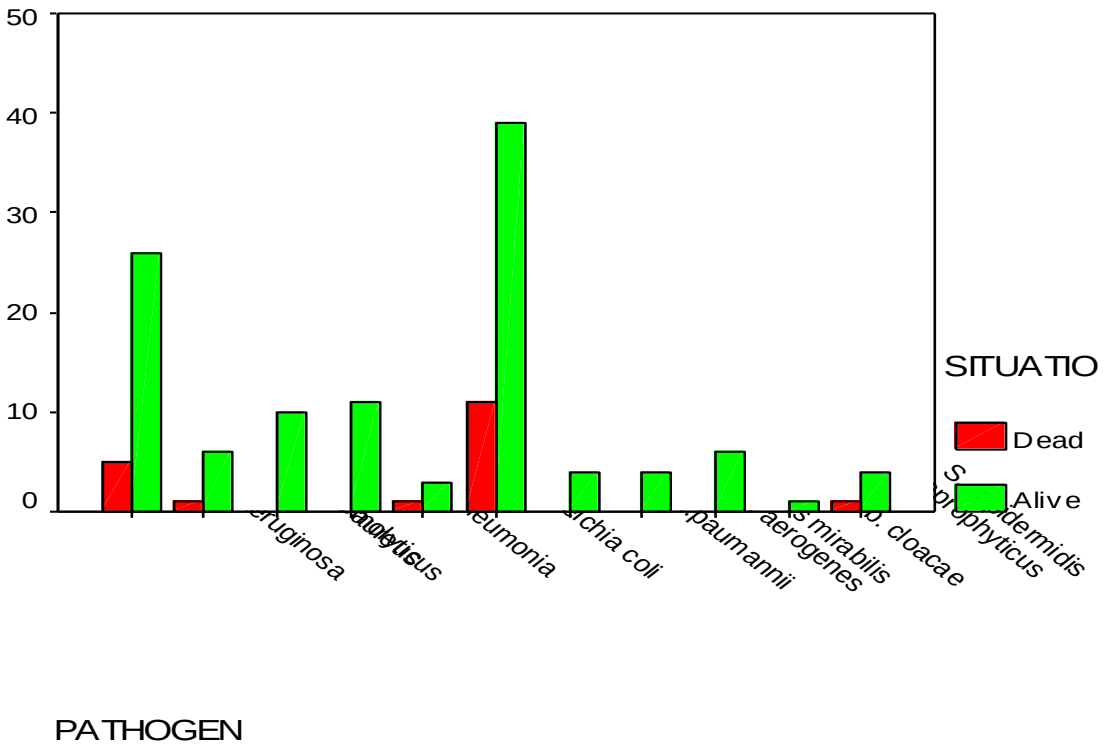
4.2.11- Distribution of bacteria growth in wound sample according to both live and death

Table(12). showed that died 14.3%. the most common bacteria was *Acinetobacter baumannii* reach to (8.3%) followed by *Pseudomonas aeruginosa* (3.8%) similar infection with *Staphylococcus haemolyticus*, *Eschirechia coli*, *Staphylococcus epidermidis* reach to (0.8%). and showed that alive 85.7%. the most common bacteria was *Acinetobacter baumannii* reach to (29.3%) followed by *Pseudomonas aeruginosa* (19.5%) followed by *Klebsiella pneumonia* (8.3%) followed by *Staphylococcus aureus* (7.5%) followed by*Enterobacter cloacae* ,*Staphylococcus haemolyticus* (4.5%) and similar infection with *Enterobacter aerogenes*,*Proteus mirabilis*, *Staphylococcus epidermidis* reach to (3.0%) *Eschirechiacoli* reach to (2.3) the lowest bacteria by *Staphylococcus saprophyticus*, reach to (0.8%).the followed site in neck reach to (9.8%) the most bacteria isolated by Pseudomonas aeruginosa (3.8%) and the lowest bacteria by *Staphylococcus haemolyticus* ,*Staphylococcus aureus* reach to (0.8%). and the followed site by face and chest reach by (9.0%).similar infection with *Acinetobacter baumannii* and the lowest bacteria by *Proteus mirabilis* and *Staphylococcus haemolyticus* rech to (0.8%) . the followed site by abdomen reach to (3.0%) the most bacteria isolated by *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Enterobacter cloacae* reach to (.8%). the lowest site head reach to by (2.3%) the most bacteria isolated by (1.5%) and the lowest bacteria isolated by *Klebsiella pneumonia* (0.8%) . (fig . 32)

Table (12)- Distribution of bacteria growth in wound sample according to both live and death

PATHOGEN * SITUATIO Crosstabulation

		SITUATIO		
		Dead	Alive	Total
PATHOGEN	Pseudomonas aeruginosa	5 3.8%	26 19.5%	31 23.3%
	Staphylococcus haemolyticus	1 .8%	6 4.5%	7 5.3%
	Staphylococcus aureus	0 .0%	10 7.5%	10 7.5%
	Klebsiella pneumonia	0 .0%	11 8.3%	11 8.3%
	Eschreichia coli	1 .8%	3 2.3%	4 3.0%
	Acinetobacter paumannii	11 8.3%	39 29.3%	50 37.6%
	Enterobacter aerogenes	0 .0%	4 3.0%	4 3.0%
	Proteus mirabilis	0 .0%	4 3.0%	4 3.0%
	Enterobacter cloacae	0 .0%	6 4.5%	6 4.5%
	Staphylococcus saprophyticus	0 .0%	1 .8%	1 .8%
	Staphylococcus epidermidis	1 .8%	4 3.0%	5 3.8%
Total		19 14.3%	114 85.7%	133 100.0%



Fig(32).: Distribution of bacteria growth in wound sample according to both live and death .

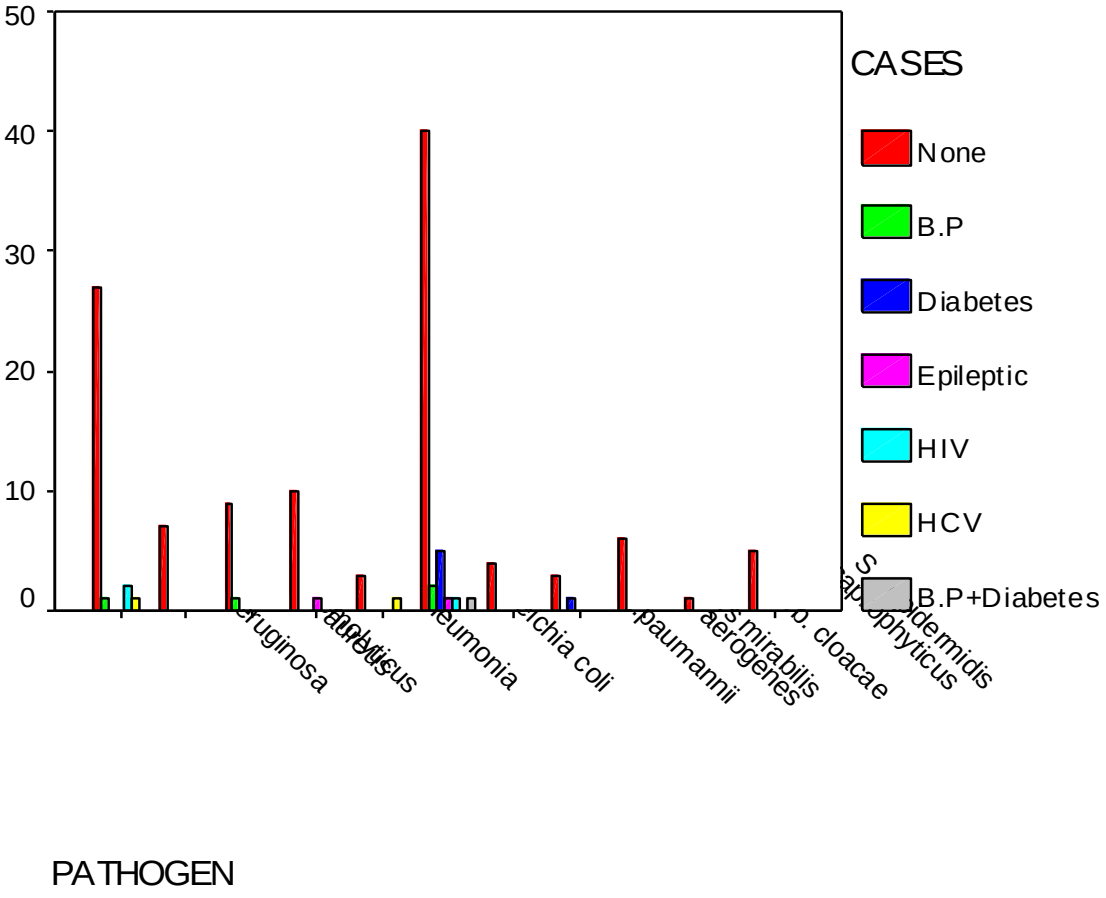
4.2.12- Distribution of bacteria growth in wound sample according to past medical history.

Table (13). showed that 18 of patients health problems during this period of study while 115 had no health problems. study showed that the most prevalent bacteria among the diabetic patients, *Acinetobacter baumannii* reach to (3.8%) followed by*Proteus mirabilis*reach to (0.8%). similarly prevalent bacteria among the blood pressure was *Acinetobacter baumannii* reach to(1.5%) followed by *Pseudomonas aeruginosa*, *Staphylococcus aureus* reach to (0.8%) however patients with human immunodeficiency Virus (HIV) the most prevalence bacteria was *Pseudomonas aeruginosa* reach to (1.5%) followed by *Acinetobacter baumannii* (0.8%) however patients with Epileptic the most prevalence bacteria was *Acinetobacter baumannii* and *Klebsiella pneumonia* reach to (0 .8%) .however patients with diabetic and blood pressure the most prevalence bacteria was reach to*Acinetobacter baumannii* (0.8%) (fig. 33)

Table(13)- Distribution of bacteria growth in wound sample according to past medical history.

PATHOGEN * CASES Crosstabulation

		CASES							
		None	B.P	Diabetes	Epileptic	HIV	HCV	B.P+Diabetes	Total
PATHOGEN	Pseudomonas aeruginosa	27	1	0	0	2	1	0	31
		20.3%	.8%	.0%	.0%	1.5%	.8%	.0%	23.3%
	Staphylococcus haemolyticus	7	0	0	0	0	0	0	7
		5.3%	.0%	.0%	.0%	.0%	.0%	.0%	5.3%
	Staphylococcus aureus	9	1	0	0	0	0	0	10
		6.8%	.8%	.0%	.0%	.0%	.0%	.0%	7.5%
	Klebsiella pneumonia	10	0	0	1	0	0	0	11
		7.5%	.0%	.0%	.8%	.0%	.0%	.0%	8.3%
	Eschreichia coli	3	0	0	0	0	1	0	4
		2.3%	.0%	.0%	.0%	.0%	.8%	.0%	3.0%
	Acinetobacter paumannii	40	2	5	1	1	0	1	50
		30.1%	1.5%	3.8%	.8%	.8%	.0%	.8%	37.6%
	Enterobacter aerogenes	4	0	0	0	0	0	0	4
		3.0%	.0%	.0%	.0%	.0%	.0%	.0%	3.0%
	Proteus mirabilis	3	0	1	0	0	0	0	4
		2.3%	.0%	.8%	.0%	.0%	.0%	.0%	3.0%
	Enterobacter cloacae	6	0	0	0	0	0	0	6
		4.5%	.0%	.0%	.0%	.0%	.0%	.0%	4.5%
	Staphylococcus saprophyticus	1	0	0	0	0	0	0	1
		.8%	.0%	.0%	.0%	.0%	.0%	.0%	.8%
	Staphylococcus epidermidis	5	0	0	0	0	0	0	5
		3.8%	.0%	.0%	.0%	.0%	.0%	.0%	3.8%
Total		115	4	6	2	3	2	1	133
		86.5%	3.0%	4.5%	1.5%	2.3%	1.5%	.8%	100.0%



Fig(33).: Distribution of bacteria growth in wound sample according to past medical history.

4.2.13- Distribution of bacteria isolated from the environment:

Table (14) showed that *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 Rooms floor of the unit. However *Acinetobacter baumanni* was isolated from the O.T table ,Rooms air of the unit.(fig.34)

Table14 : Distribution of bacteria isolated from the environment:

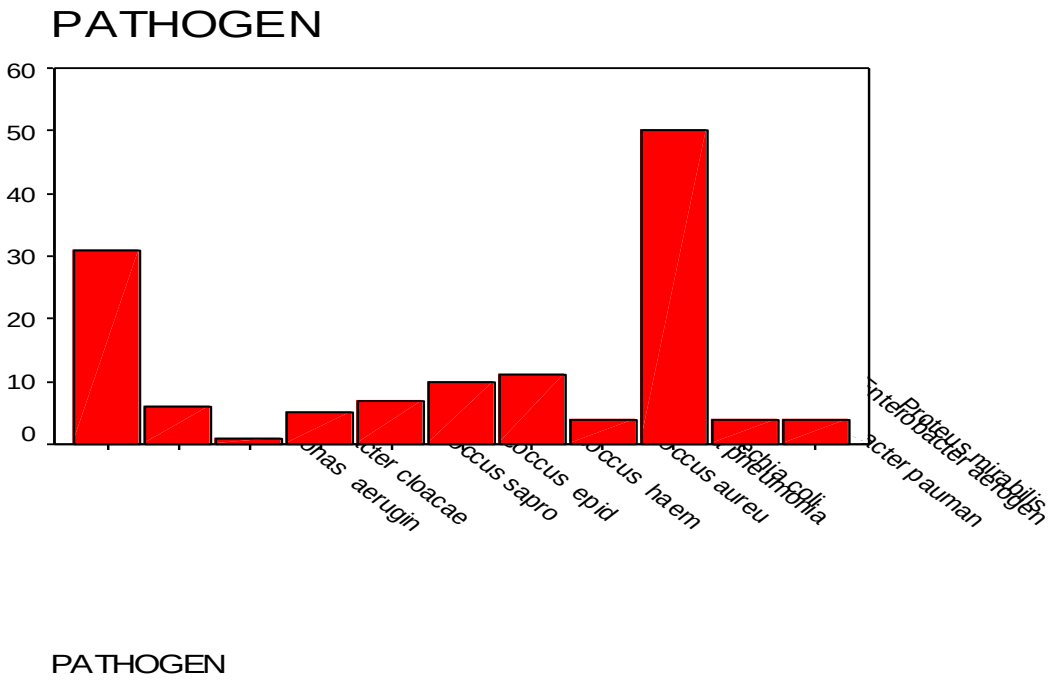
Sampled location	**organism isolated**
Unit O.T	
O.T table	*Acinetobacter baumanni*
Anesthesia machine	No growth
Anesthesiamask	No growth
Operating Lights	No growth
O.T air room	*S. aureus*
O.T room floor	*P.seudomonas spp*
O.T bed	No growth
Ward	
Wall corner	*Bacillus spores*
Disinfectant liquids used	No growth
Bed line	*S. aureus*
Walls	Bacillus spores
Holes	Bacillus spores
Rooms air	S.aureus,Acinetobacter baumanni
Rooms floor	S.aureu, S.epidermides

4.2.14- Distribution of isolated bacteria according to infected wound.

Table (15). showed that the most causative agent of burn by *Pseudomonas aeruginosa* isolates (23.3%), *Klebsiella pneumonia* isolates (8.3%), *Staphylococcus aureus* isolates (7.5%), *Staphylococcus haemolyticus* isolates (5.3%), *Enterobacter cloacae* isolates (4.5%), and *Staphylococcus epidermidis* isolates (3.8%) ,*Enterobacter aerogenes* and *Escherichia coli* and *Proteus mirabilis* isolates (3.0%), followed the lowest causative agents of burn wound infection were *Staphylococcus saprophyticus* isolates (0.8%). (fig . 35)

Table(15)- Distribution of isolated bacteria according to infected wound.

Type of bacteria	No	%
Acinetobacter baumanni	50	37.6%
Pseudomonas aeruginosa	31	23.3%
Klebsiella pneumonia	11	8.3%
Staphylococcus aureus	10	7.5%
Staphylococcus haemolyticus	7	5.3%
Enterobacter cloacae	6	4.5%
Staphylococcus epidermidis	5	3.8%
Escherichia coli	4	3.0%
Enterobacter aerogenes	4	3.0%
Proteus mirabilis	4	3.0%
Staphylococcus saprophyticus	1	.8%
Total	133	100.0%

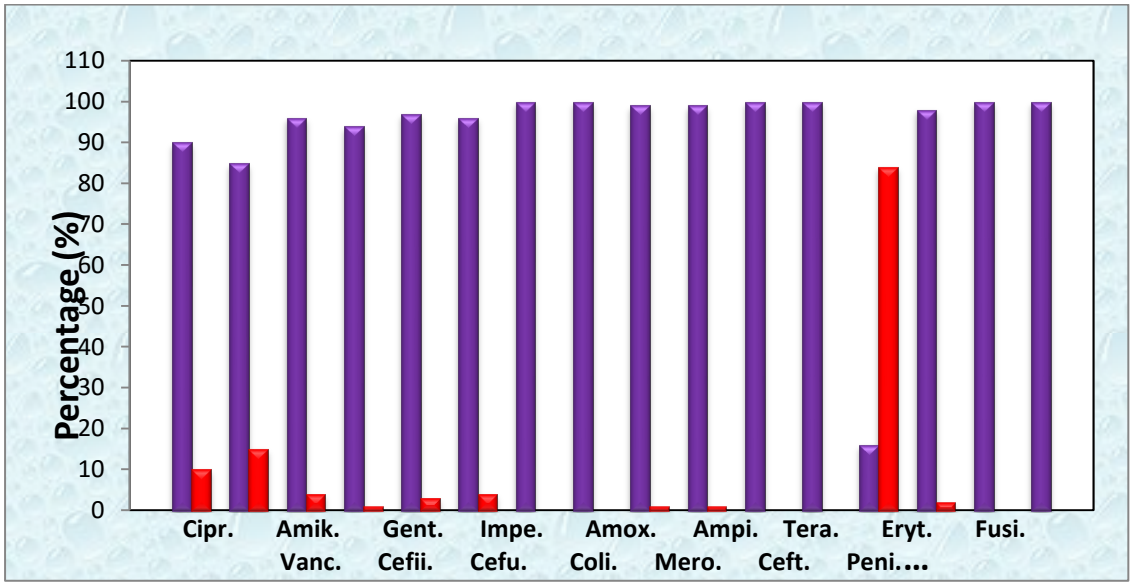


Fig(35).: Distribution of isolated bacteria according to infected wound.

4.2.15- Distribution of ac bacteria cording to susceptibility of antibiotics.

4.2.15.1- sensitivty of the Gram-positive bacteria to antibiotic.

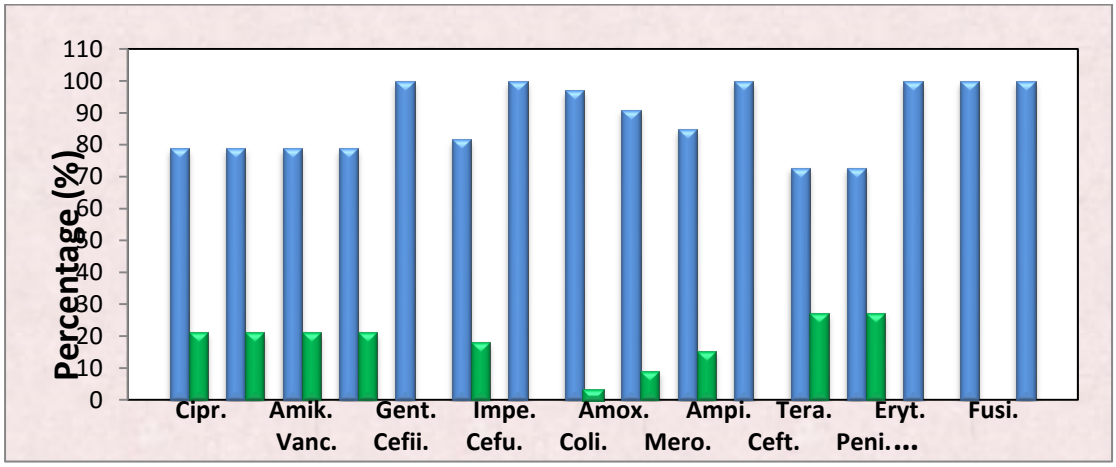
Table (16). showed the antibiotic sensitivity of bacteria Gram – positive from burn wound observed by Ciprofloxacin (72.7%) and Colistin (27.3%), Amikacin and Gentamicin and Imipenem(21.2%) however loweffect was found by Ampicillin (18.2%), Fusidin (9.1%), Erythomycin (3.0%), and no effect was observed by Penicilln, Cefipeme, Amoxicillin, Tetracycline.(fig. 36)


110
100
90
80
70
60
50
40
30
20
10
0
Percentage (%)
Cipr.
Amik.
Vanc.
Gent.
Cefii.
Impe.
Cefu.
Amox.
Coli.
Ampi.
Mero.
Tera.
Ceft.
Eryt.
Peni....
Fusi.

Gram -Ve

4.2.15.2- Sensitivty of the Gram-negative bacteria to antibiotic.

Table (17). showed the antibiotic sensitivity of bacteria Gram negative from burn wound observed by Colistin (84.0%) and Amikacin (15.0%), Ciprofloxacin (10.0%). however low effect was found by Gentamicin and Ampicillin (4.0%), Amoxicillin (3.0%). and no effect was observed by Tetracyclin, Erythromycin, Cefipeme ,Cefuroxime.(fig. 37)


Percentage (%)
110
100
90
80
70
60
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40
30
20
10
0
Cipr.
Amik.
Vanc.
Gent.
Cefii.
Impe.
Cefu.
Amox.
Coli.
Ampi.
Mero.
Tera.
Ceft.
Eryt.
Peni....
Fusi.

Gram +Ve

CHAPTER FIVE

REFRENCES

4.3-Discussion

infections remain the leading cause of death among patients who are hospitalized for burns. The risk of burn wound infection is directly correlated to the extent of the durn and is related to impaired resistance resulting from disruption of the skin mechanical integrity and generalized immune suppression . the overall infection rate was slightly higher among libyan than and non libyan patients. Also this study showed that SSI in outside of Tripoliresidence was lower compared with of resident in Tripoli city which reach to (67.6%).the increased rate of infection among patients in Tripoli may be due to different of environmental condition and lifestyle.

This study showed that Gram-negative bacteria was the cause of the infection most prevalent in the burns sections the percentage of infection reach to (75.2%), bacteria *Acinetobacter baumannii* is the most prevalent cause of infection in burns wound by (36.8%) , similar results were observed by (Babik *et al*.,2008) who reported tha all hospitalized patients were colonized with *Acinetobacter baumannii* reach to (11%).and (Wurtz *et al* 1995) who reportedThe most common pathogen isolated from burn wounds was *Acinetobacter baumannii* .

In the present study isolated bacteria male(62.4%), female (37.6%) In sections of burns, similar results was observed by (Ghaffar *et at*. , 2002) who found that burn wound infection in male was (62.4%) while burn wound infection in female (37.6%). Macedo and santos (2005) found that burn wound infection in male (59.1) was more than burn wound infection in females (40.9%) (Vostrugina *et al*., 2006)

this may be due to that males are exposed more to burns and wear loose filling clothes like dhoti also mostly restaurant workers are males engaged in cooking. In contrast to (Rajupt et al 2008)showed tha burn infection in females reach to (60%) was more than male (40%) in india.

In this study most bacterial infection was among category of age 15-30 year reach to (38.3%) in the Departments of burns followed by category 30-45 year reach to (27.1%). similar results was obtained by Kwong and Chung (1985) found that the age group 19-40 years (55%)were more susceptible to burn wound infection than other age groups.the rate of bacterial infection increased in the patients with different health problems diabetic disease patient possessed the high rate of infection reach to (4.5%).similar results were observed by (Asolaimany, 2013).This study showed that the second degree reach to (45.1%) the most bacteria by *Acinetobacter baumannii* (17.3%) similar results was observed by (Akayleh. 1999) showed that the highest distribution of burn wound infection found in burn patients who had second-degree burn (53%).

This study showed The fire burn was the predominant cause of burn among patientsreach to (34.6%) followed by benzene (21.8%).similar results was observed by (Alghalibi *et al* 2011) reported that fire burns were the most common type in burn reach to (69.5%).Incontrast (Nguyen ., 2008)the 75% of young children burned by hot woter .the present study showed that most site of burn all body reach to (25.6%) followed by hands (11.3%) similar results was observed by (Robins 1990) reported that the large area of the skin, roughly 20%for all body was burn. In the study the most isolated bacteria burn ward by (ICUB) reach to (80.5%)followed by (MBS) reach to (11.3%). similar results was observed by (Singh et al 2003). Incontrast (Sharma 2006) reported that in finland studyfound the the most unit of burn infeaction in children

burn unit reach to 42.2%.the present study showed the (14.3%) cases died after burned . and alive reach to (85.7%) . similar results was observed by (Aggarwal et al 1970) report the in the current study 40% cases died within a few minutes to 24 hours.

The present study showed that most of the bacterial isolates were *Acinetobacter baumannii* isolates (37.6%),followed by *Pseudomonas aeruginosa* isolates (23.3%), *Klebsiella pneumonia* isolates (8.3%), Staphylococcus aureus isolates (7.5%), *Staphylococcus haemolyticus* isolates (5.3%), *Enterobactercloacae* isolates (4.5%), *Staphylococcus epidermidis* isolates (3.8%) ,*Enterobacter aerogenes*,*Escherichia coli*,*Proteus mirabilis* isolates (3.0%), *Staphylococcus saprophyticus* isolates (.8%). similar results was observed by(Sengupta er al 2001) report the isolation bacteria *Acinetobacter species* over the last five to eight years in our burn unit.Similar (Yasemin*et al* . 2013) reportedthat most predominant bacterial isolate was *Acinetobacter baumannii (23.6%)*followed bycoagulase negative*Staphylococci (13.6%), Pseudomonas aeruginosa (12%), Staphylococcus aureus(11.2%), and Escherichia coli (10%)* . . in contrast to (Agnihotri 2004 et al) reported that *pseudomonas species* was the commonest pathogen isolated *pseudomonas aeruginosa*(51.5%) followedAcinetobacter apecies (14.28%) ,*S.aureus* (11.15%) .

The present study showed a Gram-negative bacteria the were sensitivite to Colistin (84.0%) and Amikacin (15.0%), Ciprofloxacin (10.0%). however low effect was found by Gentamicin and Ampicillin (4.0%), Amoxicillin (3.0%). and no effect was observed by Tetracyclin, Erythromycin, Cefipeme ,Cefuroxime. This is similar to a (Orrett et al 2000) showed the sensitivity to Ciprofloxacin (41.7%), and showed the no effect was Amikacin, Augmentine , Gentamicin, Tetracyclin.

The present study showed the antibiotic sensitivity of bacteria Gram positive to Ciprofloxacin (72.7%) and Colistin (27.3%), Amikacin and Gentamicin and Imipenem(21.2%) Ampicillin (18.2%), Fusidin (9.1%), Erythomycin (3.0%), and no effect was observed by Penicilln, Cefipeme, Amoxicillin, Tetracycline.Similar result was observed by Ali and Enayat (2007) showed Gram-positive bacteria property antibiotic sensitivity (Gentamicin, Ciprofloxacin) that showed resistance property in our study.

4.4_ **Conclusion**

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

2-The most causative agent of burn wound infection were *Acinetobacter baumanniif*ollowed by *Pseudomonas aeruginosa*, *Klebsiella pneumonia*, *Staphylococcus aureus*, *Staphylococcu shaemolyticus*, *Enterobacter cloacae*, *Staphylococcus epidermidis*, *Enterobacter aerogenes* and *Escherichia coli*,*Proteus mirabilis*,*Staphylococcus saprophyticus*. byagreement swabs taken from the hospitals in the city oftripoli.

3-This study showed that people who suffer from diabetes are most vulnerable to infection due to opportunistic bacteria to a weakened immune system and physiological changes in the body of diabetics.

4-This study showed that infections are serious problem among burns patients. *Acinetobacter baumannii* has emerged the commonest organism causing infection and is resistant to most of the antibiotics.

5-The inappropriate usage of antimicrobials in burn wound prophylaxis is still a problem and 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 burn infection.

CHAPTER SIX

APPENDIX

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Appendix I

Preparation of culture media and reagents

Blood Agar BASE (infusion agar)

Formula **g/L**

Beef heart, infusion from…….....500.0 g

Tryptose……..10.00g

Sodium Chloride……..5.00g

Agar……..15g

Direction :

Suspend 40g in liter of purified water. bring to boil to dissolve Completely. Sterilize by autoclaving at 121C for 15 min. cool to 45-50C for bloodagar add 7% sterile defibrinated blood.

MacConkey Agar Medium:

Formula g/l

Peptic Digest of Animal Tissue …….15.0g

Casein enzymic hydrolysate……1.5g

Sodium chloride …….....5g

Lactose ……10.0g

Bile salts ……1.50 g

PancreaticDigest of Gelatin ……..17 g

Agar ……..15.00 g

Neutral red ……..0.03 g

Crystal violet ……..0.001

Direction :

Suspend 51.5g of the medium in 1liter of purified water . Heat to boiling with frequent agitation for one minute to completely dissolve the medium autoclaving at 121C for 15min .

Muller Hinton Agar

Formula g/l

Beef infusion form……..300 g

Casein acidhydrolysate……17.50 g

Starch ……..…1.50 g

Agar……..……..17 g

Direction:

Suspend 38.0 g in 1000 ml. distilled water Bring to boil to dissolve completely. Sterilize by autoclaving at 121C for 15 min.

Mannitol Salt Agar Base

Formula **g/L**

Proteose peptone…….…….10.00g

Beef extract……..1.00g

Sodium chloride……..…….75.00g

D.mannitol……...…….10.00g

Phenole red……...……0.025g

Agar…….15.00

Directions :

Suspend 111.0 grams in 100ml distilled water heat to boiling to boiling to dissolve the medium completely sterilize by autoclaving at is lbs pressure 121 C for 15minutes

ملخص البحث

الهدف الاساسى من هذه الدراسة كان عزل وتعريف البكتريا المسببة لعدوي التهابات الحروق وحساسيتها للمضادات الحيوية الشائعة الاستعمال. حيث تم تجميع 133عينة وذلك عن طريق اخذ مسحات من جرح الحروق من المرضى المقيمين فى مستشفي (جراحة الحروق والتجميل – طرابلس) من خلال 2012-2013 ،أيضا شملت الدراسة فحص البيئة المحيطة بالمريض وذلك باخذ عينات من مختلف المناطق المحيطة بالمريض .

وكانت البكتريا السالبة لصبغة جرام Acinetobacter baumannii الاكثر انتشارا بين المرضي وذلك بنسبة.

*Acinetobacter baumannii*isolates(37.6%),followed by *Pseudomonas aeruginosa*(23.3%), *Klebsiella pneumonia* (8.3%), *Staphylococcus aureus* (7.5%), *Staphylococcus haemolyticus* (5.3%), *Enterobacter cloacae* (4.5%), and *Staphylococcus epidermidis*(3.8%) ,*Enterobacter aerogenes* and *Escherichia coli* and *Proteus mirabilis* isolates (3.0%), followed the lowest causative agents of burn wound infection were *Staphylococcus saprophyticus* isolates (.8%).

من خلال هذة الدراسة لوحظ ان هناك تباين لحساسية البكتريا المعزولة للمضادات الحيوية حيث ان اغلب البكتريا السالبة لصيغة جرام كانت ذات حساسية للمضادات الحيوية

Cefipeme, وأكثر مقاومة للمضادات الحيوية Colisten, Amikacin , Ciprofloxacin Tetracyclin, Cefuroxime.بينما كانت الموجبة لصبغة جرام أكثرحساسية للمضادات الحيوية Penicilln, ومقاومة للمضادات الحيوية CiprofloxacinGentamicin, Amoxicillin , Cefipeme.

أظهرت هذه الدراسة أن معظم مسار إنتقال البكتيريا المتعددة المقاومة للمضادات الحيوية كانعبر العدوى المتبادلة وذلك طبقا لنتائج إختبارات المضادات الحيوية بالإضافة إلى بيئة المستشفى التي قد تصبح مصدرا مهما للعدوى وذلك بسبب نظام النظافة الروتيني .

جامعة بنغازي

كلية العلوم

قسم علم النبات

عزل وتعريف البكتيريا من عدوى جرح الحروق في مستشفي جراحة الحروق والتجميل

بنغازى– ليبيا

مقدمة من

مروى محمد الموهوب

إشراف

أ.د.صالحة فرج بن جويرف

الرسالة مقدم كجزء من متطلبات الحصول على درجة (الماجستير)

في علم النبات

ربيع (٢٠١٥)