Ministry of Higher Education
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University of Al-Qadisiyah
College of Pharmacy

The antibiotic susceptibility of *S. aureus* isolates from patients with burn wound infection in Al-Diwaniyah Teaching hospital/ burns unit

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بسم الله الرحمن الرحيم

(وقُـــــــــل ربي زدني علـــــــــمـــاً)

صدق الله العلي العظيم

سورة طه اية (١١٤)

Dedication:

To our families
To our supervisor
To our professors

Certification :

This is certify that this research was prepared under my supervision at the University of AL-Qadisiyah , College of Pharmacy , as a partial requirement for the degree of Bachelor in Pharmacy

Signature:

Advisor’s Name: Lec. Saba Abood Ali

Date: / / 2018

List of Subjects

Abstract

Background: Burns provide a suitable site for bacterial multiplication and are more persistent richer sources of infection than surgical wounds. *Staphylococcus aureus* is one of the most frequently isolated pathogens in both community and hospital practices.

Objectives : objective of this study was to address the prevalence and antibiotic susceptibility patterns of *S. aureus* isolated from burn wound infections in Al-diwaniyah teaching hospital / burns unit.

Results : Out of 114 patients, bacterial infection was observed in 95(83.3%) of which, 66 (69.5%) had *S. aureus* infection. Overall prevalence of *S. aureus* isolation was 57.8%. Most of them were sensitive to vancomycin, clindamycin, Kanamycin and Erythromycin, but highly resistant to penicillin G. All isolates were found to be multi drug resistant, and one isolate was resistant to all the tested drugs.

Conclusion: The current study is highly important and informative for the high level of multi-drug resistant *S. aureus* isolates in burn patients. Finally, strict consideration for *S. aureus* infection and proper usage of antibiotic policy are recommended in decreasing the incidence and occurrence of multidrug resistant *S. aureus* infections in in Al-diwaniyah -Teaching Hospital/ burns unit.

Keywords: *Staphylococcus aureus*, burn wound infection, drug sensitivity

Chapter One
Introduction

1.Introduction:

Burns are one of the most common and devastating forms of trauma. Patients with serious thermal injury need immediate specialized care to reduce morbidity and mortality. Data from the National Center for Injury Prevention and Control in the United States show that approximately 2 million fires are reported each year which result in 1.2 million people with burn injuries [1] Moderate to severe burn injuries requiring hospitalization account for approximately 100,000 of these cases, and about 5,000 patients die each year from burn-related complications [2]. In Canada, the estimated numbers of burn victims and deaths in serious cases are proportionally smaller on a per capita basis [3].

The survival rates for burn patients have improved substantially in the past few decades due to advances in modern medical care in specialized burn centers. Improved outcomes for severely burned patients have been attributed to medical advances in fluid resuscitation, nutritional support, pulmonary care; burn wound care, and infection control practices. As a result, burn-related deaths, depending on the extent of injury, have been halved within the past 40 years [4]. In patients with severe burns over more than 40% of the total body surface area (TBSA), 75% of all deaths are currently related to sepsis from burn wound infection or other infection complications and/or inhalation injury [5]. Initially, the burned area is considered free of major microbial contamination.

There are several studies [52] that indicate the rates of bacterial infection in burns: *Staphylococcus aureus (*22.9%) , *Pseudomonas aeruginosa* (20.9%) , *Pseudomonas species* (7.2%) , *Escherichia coli* (6.7%) , *Group D Streptococcus* (5%) , *Enterococcus faecalis* (4.2%). Since *S. aureus* infection is the biggest percentage in burns wound infection, we chose to be the subject of research due to its importance in burns.

Chapter Two
Literature review

2. Literature review:-

2.1 Burn wound infection in Iraq:

Wound infections are common problems in burn units in hospitals of Iraq and mostly originate from nosocomial contamination.The large number of patients exposed to burns in Iraq for men because of the social situation or during work in factories or other places that require the presence of fire either for women is due to the fact that they are more likely to burn because of domestic work such as cooking in addition to psychological cases such as suicide fire so much recently.The number of dead in the fire due to the reasons mentioned above. The factors that affectthe rate of burning are sex and age where the rate of burns in men more than in women As for age , researchers noticed an increase in the rate of burns in ages ranging from 20 to 45 years.The results show that the percentage of female fire is higher than that of men. [1],[2]

2.2 Types of the burns:-

2.2.1 Friction burns:

When a hard object rubs off some of your skin, you have what's called a friction burn. It's both an abrasion (scrape) and a heat burn. These are common in motorcycle and bike accidents. Carpet burn is another type of friction burn. [26]

2.2.2 Cold burns:

Also called "frostbite," cold burns cause damage to your skin by freezing it. You can get frostbite by being outside in freezing temperatures. It can also happen when your skin comes into direct contact with something very cold for a prolonged period of time. [27]

2.2.3 Thermal burns:

Touching a very hot object raises the temperature of your skin to the point that your skin cells start dying. Very hot metals, scalding liquids, and flames all cause thermal burns. Steam can, too. [27]

2.2.4 Radiation burns:

sunburn is a type of radiation burn. Other sources of radiation, like X-rays or radiation therapy to treat cancer, can also cause these. [28]

2.2.5 Chemical burns:

Strong acids, solvents or detergents that touch your skin can cause it to burn. [29]

2.2.6 Electrical burns:

If you come into contact with an electrical current, you can get this type of burn. [30]

2.3 Causative agent:-

Burns are caused by a variety of external sources classified as thermal (heat-related), chemical, electrical, and radiation [33] .In the United States, the most common causes of burns are: fire or flame (44%), scalds (33%), hot objects (9%), electricity (4%), and chemicals (3%) [33]. Most (69%) burn injuries occur at home or at work (9%)[33] and most are accidental, with 2% due to assault by another, and 1–2% resulting from a suicide attempt[34]. These sources can cause inhalation injury to the airway and/or lungs, occurring in about 6% [34].

Burn injuries occur more commonly among the poor[33]. Smoking and alcoholism are other risk factor[35]. Fire-related burns are generally more common in colder climates[35]. Specific risk factors in the developing world include cooking with open fires or on the floor [36].

2.4 classification of the burns:-

2.4.1 First-degree burns:

Cause minimal skin damage. They are also called “superficial burns” because they affect the outermost layer of skin. Signs of a first-degree burn include:

- redness
- minor inflammation, or swelling
- pain
- dry, peeling skin occurs as the burn heals

Since this burn affects the top layer of skin, the signs and symptoms disappear once the skin cells shed. First-degree burns usually heal within 7 to 10 days without scarring.[32]

2.4.2 Second-degree burns:

Second-degree burns are more serious because the damage extends beyond the top layer of skin. This type burn causes the skin to blister and become extremely red and sore.

Some blisters pop open, giving the burn a wet or weeping appearance. Over time, thick, soft, scab-like tissue called fibrinous exudate may develop over the wound.

Due to the delicate nature of these wounds, keeping the area clean and bandaging it properly is required to prevent infection. This also helps the burn heal quicker.[32]

to three weeks without scarring, but often with pigment changes to the skin.

The worse the blisters are, the longer the burn will take to heal. In some severe cases, skin grafting is required to fix the damage. Skin grafting takes healthy skin from another area of the body and moves it to the site of the burned skin. [32]

As with first-degree burns, avoid cotton balls and questionable home remedies. Treatments for a mild second-degree burn generally include:

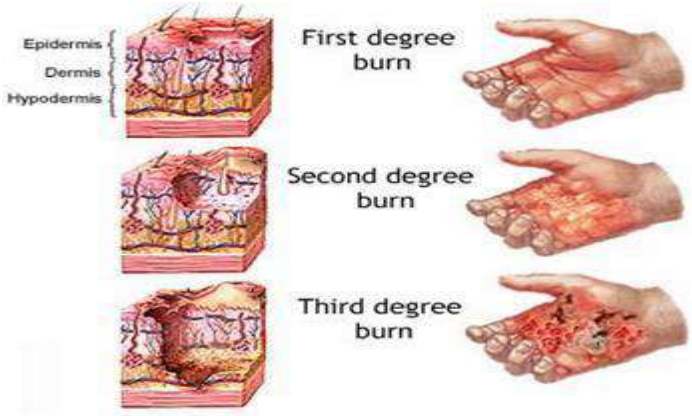
- running the skin under cool water for 15 minutes or longer
- taking over-the-counter pain medication (acetaminophen or ibuprofen)
- applying antibiotic cream to blisters

2.4.3 Third-degree burns

Third -degree burns are the most severe. They cause the most damage, extending through very layer of skin. There is a misconception that third-degree burns are the most painful. However, with this type of burn the damage is so extensive that there may not be any pain because of nerve damage. [32]

Depending on the cause, the symptoms third-degree burns can exhibit include:

- waxy and white color
- char
- dark brown color
- raised and leathery texture
- blisters that do not develop



Picture (2-1) : The degrees of the burns

2.5 *Staphylococcus aureus:-*

In humans, *S. aureus* is part of the normal microbiota present in the upper respiratory tract, and on skin and in the gut mucosa. *S. aureus*, along with similar species that can colonize and act symbiotically but can cause disease if they begin to take over the tissues they have colonized or invade other tissues,

have been called "pathobionts".[37]

2.5.1 Bacterial Characteristics:-

S. aureus is a facultative anaerobic, gram-positive coccal (round) bacterium also known as "golden staph". *S. aureus* appears as staphylococci (grape-like clusters) when viewed through a microscope, and has large, round, golden-yellow colonies, often with hemolysis, when grown on blood agar plates , *S. aureus* reproduces asexually by binary fission. Complete separation of the daughter cells is mediated by *S. aureus* autolysin, and in its absence or targeted inhibition, the daughter cells remain attached to one another and appear as

clusters [38]. *S. aureus* is catalase-positive (meaning it can produce the enzyme catalase). Catalase converts hydrogen peroxide (H_2O_2) to water and oxygen. Catalase-activity tests are sometimes used to distinguish staphylococci from enterococci and streptococci. Previously, *S. aureus* was differentiated from other staphylococci by the coagulase test. However, not all *S. aureus* strains are coagulase-positive and incorrect species identification can impact effective treatment and control measures[39].

2.5.2 Role of *S. aureus* in burns :

Infection of burn wounds with *S. aureus* increases the risk of sepsis and multi organ failure. Furthermore, infection of burn wounds can lead to hypertrophic scarring.[40]

A hypertrophic scar is a widespread red, raised, sometimes itchy scar that remains within the borders of the injury' these scars can be disfiguring and even painful [40].

If such scars are situated across joints, the almost inevitable contractures can impair function and result in painful fissures.[41]

Burn scars may have a dramatic influence on a patient's quality of life. They have been associated with anxiety, social avoidance, and depression, a disruption in normal daily activities, the onset of sleep disturbances, and all of the consequent difficulties in returning to normal life after physical rehabilitation [41].

2.5.3 Virulence factors:-

2.5.3.1 Enzymes:

S. aureus produces various enzymes such as coagulase (bound and free coagulases) which clots plasma and coats the bacterial cell, probably to prevent phagocytosis. Hyaluronidase (also known as spreading factor) breaks down hyaluronic acid and helps in spreading it. *S. aureus* also produces deoxyribonuclease, which breaks down the DNA , lipase to digest lipids, staphylokinase to dissolve fibrin and aid in spread, and beta-lactamase for drug resistance.[41]

2.5.3.2 Toxins:

Depending on the strain, *S. aureus* is capable of secreting several exotoxins, which can be Categorized into three groups. Many of these toxins are associated with specific diseases.[42]

2.5.3.3 Superantigens :

Antigens known as superantigens can induce toxic shock syndrome (TSS). This group includes the toxins TSST-1, and enterotoxin type B, which causes TSS associated with tampon use. Toxic shock syndrome is characterized by fever, erythematous rash, low blood pressure, shock, multiple organ failure, and skin peeling.

a part in the pathogenesis of TSS. Other strains of *S. aureus* can produce an enterotoxin that is the causative agent of a type of gastroenteritis. This form of gastroenteritis is self-limiting, characterized by vomiting and diarrhea one to six hours after ingestion of the toxin, with recovery in eight to 24 hours. Symptoms include nausea, vomiting, diarrhea, and major abdominal pain.[43][44]

2.5.3.4 Exfoliative toxins :

Exfoliative toxins are exotoxins implicated in the disease staphylococcal scalded skin syndrome (SSSS), which occurs most commonly in infants and young children. It also may occur as epidemics in hospital nurseries. The protease activity of the exfoliative toxins causes peeling of the skin observed with SSSS.[44]

2.5.3.5 Other toxins:

Staphylococcal toxins that act on cell membranes include alpha toxin, beta toxin, delta toxin, and several bicomponent toxins. The bicomponent toxin PVL is associated with severe necrotizing pneumonia in children.[45][46]

2.6 Defensive Mechanism of *S. aureus*:-

Resistance mechanisms include enzymatic inactivation of the antibiotic (penicillinase and aminoglycoside-modification enzymes), alteration of the target with decreased affinity for the antibiotic (notable examples being penicillin-binding protein 2a of methicillin-resistant S. aureus and D-Ala-D-Lac of peptidoglycan precursors of vancomycin-resistant strains), trapping of the antibiotic (for vancomycin and possibly daptomycin) and efflux pumps (fluoroquinolones and tetracycline). Complex genetic arrays (staphylococcal chromosomal cassette mec elements or the vanA operon) have been acquired by S. aureus through horizontal gene transfer, while resistance to other antibiotics, including some of the most recent ones (e.g., fluoroquinolones, linezolid and daptomycin) have developed through spontaneous mutations and positive selection. Detection of the resistance mechanisms and their genetic basis is an important support to antibiotic susceptibility surveillance in S. aureus [42].

2.7 pathogenicity of *S. aureus:-*

Strains of *S. aureus* are known to enter through the breaks in the skin to cause localized infections or spread via blood to cause more generalized infections like that of blood (sepsis), bone (osteomyelitis), brain (meningitis), lungs (pneumonia) etc. Individuals with a compromised immune system are particularly vulnerable.[43]

2.8 Diagnosis of *S. aureus*:

In the case of the staphylococci, the main clinically relevant factor is the differentiation between the contagious *S. aureus* and other staphylococci. On one hand, selective agars like modified Baird-Parker agar have been used successfully for the detection and identification of *S. aureus* and other coagulase-positive staphylococci [45] However, such agars may not allow the detection of other microorganisms and can be used only for a targeted search of *S. aureus*. On the other hand, several tests are currently used to differentiate *S. aureus* from other staphylococci in primary nonselective cultures.[44] Testing for the presence and type of hemolysis on blood agar plates represents a first simple and rapid method. Unfortunately, this criterion is not very sensitive[44]. Among the three coagulase-positive staphylococci, *S. aureus* is the only one with hemolytic activity that is regularly encountered in clinical milk samples. Therefore, a combination of

hemolysis and coagulase activities seems to represent an optimal criterion for the identification of *S. aureus* in cultures from milk samples[44]. The presence of a DNase activity is often used as a surrogate marker for the identification of coagulase-positive staphylococci and particularly of *S. aureus* in milk samples. However, the specificity of this criterion or of the more laborious test for thermonucleases is not entirely satisfactory [44]

2.9 Prevention of *S. aureus* infection:

Prophylactic antimicrobial therapy is recommended only for coverage of the immediate perioperative period around excision or grafting of the burn wound. Infection control programs need to document and report burn wound infections according to recent classification systems. The incidence of infections reported among burn patients has been found closely related to the person who is assessing the patient for infection. On the basis of the infection control assessment, using the CDC's definitions, individual researcher's rates can be compared with the pooled means from prevoius prospective studies, especially those using multivariable analysis to assess independent risk factors for infections. Preparation of burn unit-specific antibiograms will reveal effective topical antimicrobial agents. Surveillance for surgical site infections and reporting of these rates to surgeons has been shown to reduce the rates of infection [23].

The infection control literature indicates that precise, written definitions are essential to accurately identify hospital-acquired infections. It has been suggested that because of discrepancies between the surgeon's assessment and infection control assessment, burn patients are over-treated with antimicrobial agents and antimicrobial use could possibly be decreased if more precise definitions of infection were used in clinical practice [24]. significantly associated with nosocomial infection in the logistic regression model of risk factors for infection, as identified by either set of criteria. Decreased use of invasive devices, and improved aseptic technique when inserting devices could decrease the rates of nosocomial infections in burn units. CDC has developed evidence-based guidelines for preventing central venous catheter- associated BSIs(Reference 25) Thus, wherever possible, use of indwelling devices should be minimized and these devices should be removed when no longer needed.

2.10 Treatment of *S. aureus* in burn wound infection:-

Most antimicrobial therapy prescribed for burn patients is administered topically. Antibiotic resistant microorganisms have been associated with infections of burn wounds [21] .Risk factors for acquisition of antibiotic resistant organisms include receipt of antibiotics prior to development of infection and extended duration of hospitalization. Burn centers should routinely determine and track the specific pattern of burn microbial flora and trends in the nasocomial spread of these pathogens [22]. The main antibiotics used to treat *S. aureus* in burns wound infections are

penicillin (10 units), erythromycin (15 µg), chloramphenicol (30 µg), clindamycin (2 µg), vancomycin (30 µg) ,Augmentin (30 µg) ,Polymyxin-B (300 µg) ,Amikacin (30 µg) Cephalothin ,(30 µg) Methicillin ,(10 µg) and ,Kanamycin (30 µg).[22]

Chapter Three
Material and Method

3. Material and method:

3.1 Study Population

A total number of 114 burned patients in AL-Diwaniyah Teaching Hospital / Burn Unit were involve.

The studygroup comprised of males and females with age range from 1 year to 65 years, during a period since (15/August to 15/November 2018).

3.2 Collection of Samples

pus swab specimens was collected from each patient .The sample collection, culturing, staining and sensitivity tests were performed according to the WHO standard microbiological diagnosis for *S. aureus* [53]. The specimens were collected by the attending physician and health officer using sterile applicator stick with cotton swabs moistened with normal saline and test tubes.The sample is transported to microbiological laboratory in hospital for further processing , with the information of each patient (the name, age and gender).

3.3 Culturing of the samples:-

The swabs were then inoculated on blood agar and mannitol salt agar (MSA) plate and incubated at 37ºc for 24 hrs. After the plate had been left at room temperature for pigment formation, colonies were selected and checked for gram stain and coagulase test was done to differentiate *S. aureus* from other *staphylococci spp.[53]*

3.4 Identification:-

Coagulase test is used to differentiate *Staphylococcus aureus* (positive) from other types of *Staphylococci.* [54]

3.4.1 Coagulase test:-

A slide coagulase test is run with a negative control to rule out autoagglutination. Two drops of saline are put onto the slide labeled with sample number, Test (T) and control (C). The two saline drops are emulsified with the test organism using a wire loop, straight wire, or wooden stick. A drop of plasma is placed on the inoculated saline drop corresponding to test, and mixed well, then the slide is rocked gently for about 10 seconds.[54]

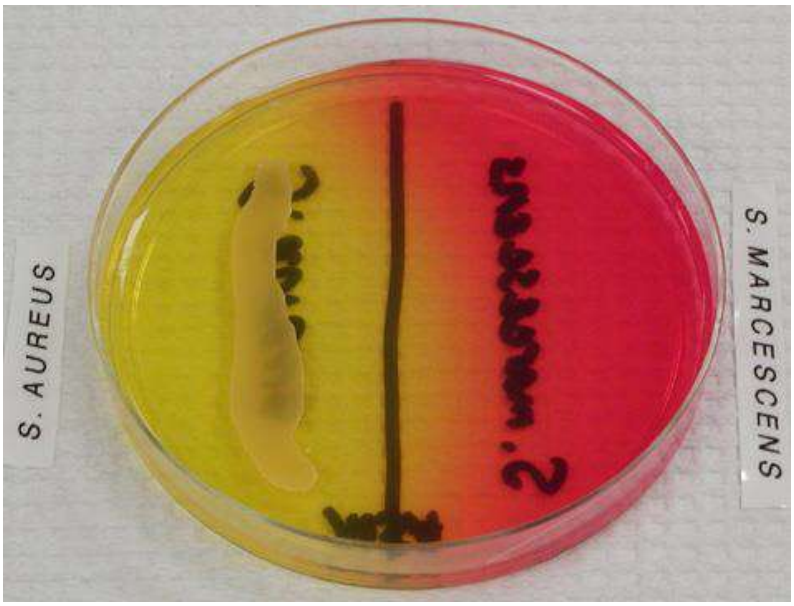
- If 'positive', macroscopic clumping would be observed in the plasma within 10 seconds, this is indicated the presence of *Staphylococcus aureus* .
- If 'negative', no clumping will be observed.[54] See (picture 3-1)

Picture (3-1) Coagulase Test

3.4.2 Mannitol Salt Agar (MSA) :-

Mannitol Salt Agar (MSA) is a selective and differential medium. The high concentration of salt (7.5%) selects for members of the genus *Staphylococcus*, since they can tolerate high saline levels. Organisms from other genera may grow, but they typically grow very weakly. [55],[56].

MSA also contains the sugar mannitol and the pH indicator phenol red. If an organism can ferment mannitol, an acidic byproduct is formed that will cause the phenol red in the agar to turn yellow. Most pathogenic staphylococci, such as *Staphylococcus aureus*, will ferment mannitol. Most non-pathogenic staphylococci will not ferment mannitol. [55]



Picture (3-2) : Mannitol Salt Agar

3.5 Evaluation of S. aureus by Antibiotic Sensitivity Pattern:

Antibiotic sensitivity test (disc diffusion method):

Antimicrobial susceptibility testing was performed by both disk diffusion methods according to NCCLS guidelines [56,57].

The following antimicrobial agents were used for disk diffusion tests by local laboratories: penicillin (10 units), erythromycin (15 µg), chloramphenicol (30 µg), clindamycin (2 µg), vancomycin (30 µg) Augmentin (30 µg) Polymyxin-B (300 µg) Amikacin (30 µg) Cephalothin (30 µg) Methicillin (10 µg) and Kanamycin (30 µg).

by the Kirby- Bauer disk-diffusion technique. Small discs containing antibiotics are placed onto a plate upon which *S. aureus* are growing. If the bacteria are sensitive to the antibiotic, a clear ring, or zone of inhibition, is seen around the disc indicating poor growth.[56]

Chapter Four
Results

4. The Results:-

4.1 The prevalence of burned patients according to age and gender:-

The present study included a collection of 114 swap samples from AL-Diwaniyah- Teaching Hospital during the period from (15-August to 15-November 2018). The females were 56 (49.1%) while the males were 58 (50.9%).

Show th Table (4-1)

Table(4-1): Distribution of 114 burned patient according to age and gender

Age	Gender		Total
	Male	Female	
< 20 years	22(19.3%)	24(21.1%)	46(40.4%)
20 – 45 years	31(27.2%)	28(24.6%)	59(51.8%)
> 45 years	5(4.4%)	4(3.5%)	9(7.9%)
Total	58(50.9%)	56(49.1%)	114(100.0%)

4.2 The prevalence of positive culture among age group:-

Out of 114 burn wound pus swab specimens processed, 95(83.3%) grew organism on culture. From the 95 isolates:

- 66 (69.5%) showed significant positive *S. aureus* culture.
- 29(18.5%) showed other organisms.
- 19(12%) showed no infection

the females were slightly infected than males with percentage 35(53.0%) out of the 66 positive cultures and the males were 31(47.0%).

Table(4-2) showed that 15(42.8%) females of age group (less than 20 years) gave a positive bacterial culture . Also the females in age group (20-45) find out 17(48.6%) and the females in age group (more than 45 years) find out 3(8.6%) from 35 positive culture; while in male the age group (less than 20 years) showed 14(45.2%) Also the males in age group (20-45) find out 15(48.4%) and the males in age group (more than 45 years) find out 2(6.4%) from 31 positive culture.

Table(4-2):Distribution of 66 infected patients with S. aureus according to age and gender

Age	*S. aureus* isolates		
	Male	Female	Total
< 20	14(45.2%)	15(42.8%)	29(43.9%)
20 – 45	15(48.4%)	17(48.6%)	32(48.4%)
> 45	2(6.4%)	3(8.6%)	5(7.6%)
Total	31(47.0%)	35(53.0%)	66(100.0%)

4.3 Antimicrobial Sensitivity Tests results :-

Sensitivity of *S. aureus* isolates to (vancomycin is (62) 93.9%) ,(Clindamycin (60) 90.9%), (Kanamycin (57) 86.4%) and (Erythromycin(57) 86.4%) .

4.4Antimicrobial Resistance Tests results :-

The resistance of *S. aureus* isolates above 50% rates was observed to (penicillin (63) 95.5%), (methicillin (51) 77.3%), (polymyxin-B (45) 68.2%) and (chloramphenicol (34) 51.5%). (Table 4-2)

Table(4-3): Antimicrobial susceptibility pattern of isolated S.aureus in burn patients attending in AL-Diwaniyah- Teaching Hospital

Total isolated		Antimicrobial agents test										
		AUG	CEP	CHF	MET	PEN	AMI	VAN	CLI	KAN	ERY	POLM
66	S	44 66.7%	43 65.2%	32 48.5%	15 22.7%	3 4.3%	35 53.0%	62 93.9%	60 90.9%	57 86.4%	57 86.4%	21 31.8%
	R	22 33.3%	23 34.8%	34 51.5%	51 77.3%	63 95.5%	31 47.0%	4 6.1%	6 9.1%	9 13.6%	9 13.6%	45 68.2%

S:sensitivity, R:resistance , AUG: Augmentin , CEP:Cephalothin, CHF:Chloroamphenicol, MET:Methicillin, PEN:Penicillin , AMI: Amikacin , VAN: Vancomycin , CLI:Clindamycin, KAN:Kanamycin, ERY:Erythromycin, POLM: Polymyxin B

Chapter Five
Discussion

5. Discussion:-

The burn wound is considered as one of the major health problems in the world [11] In the present study, *S. aureus* was the most common isolate which is similar to other findings [12 , 13]. In contrast other studies reported that *P. aeruginosa* as a predominant organism [1,14] This could be attributed to differences in geographical location and hygienic measures. At present, the overall prevalence of *S. aureus* infection was high compared to other bacterial isolates in this study. Similar reports were done by Bhat and Vasaikar [15].This may be due to cross infection by the hand of the medical personnel, air and other materials but there was no significant association with age and sex.

Infection is the most important problem in the treatment of burn patients. The bacteriology of burn wounds is often poly-microbial in nature, and the presence of multidrug-resistant organisms is often associated with more severe clinical manifestations and poor response to antimicrobial therapy. Antibiotic sensitivity patterns served as a useful guideline for choosing an appropriate antibiotic. In the present study, drug resistant rate of *S. aureus* isolates were extremely high for penicillin and moderately for methicillin and polymyxin-B. Eke and Rotimi also reported comparable to these finding [16]. Similarly, Bhat and Vasaikar also reported penicillin and methicillin was resistant *S.aureus* isolates in burn wound infections [15]. Yet, the isolates at AL-Diwanyah -Teaching Hospital are highly susceptible to vancomycin, clindamaycin, Kanamycin and Erythromycin. This result is in agreement with Uchenna (2005) and Gebreselassie (2000) studies. [17,18]In the present study, all the *S. aureus* isolates were multi-drug resistant and only one isolate was pane-resistant. This could be due to continuous usage of broad-spectrum antibiotics and non-adherence to a hospital antibiotic policy. Additionally, selective pressure in the hospital wards could also be taken as the most probable factor for

the increased resistance in isolates from the patients.[19]

The emergence of worldwide antimicrobial resistance among a wide variety of human, bacterial and fungal burn wound pathogens, particularly nosocomial isolates, limits the available therapeutic options for effective treatment of burn wound infections [20]. Methicillin-resistant *coagulase-negative staphylococci*, vancomycin-resistant *enterococci*, and multi-drug resistant gram-negative bacteria that possess several types of beta-lactamases, including extended spectrum beta-lactamases, *ampC* beta-lactamases, and metallobeta-lactamases, have been emerging as serious pathogens in hospitalized patients [20]. In view of the variety of burn wound isolates seen and their generally increasing antimicrobial resistances regular microbiological surveillance, *in-vitro* testing and monitoring of these parameters would play an important role in guiding the proper empirical antimicrobial therapy in burn patients, preventing multidrug resistance by virtue of using antimicrobials that target specific organisms and decreasing infection-related complications. Empirical usage of broad-spectrum antibiotics was probably the cause of high percentage of multi drug resistant isolates .[21]

The current study is highly important and informative for the high level of multi-drug resistant *S. aureus* isolates in burn patients. This study result may pave a way for providing useful guidelines in choosing to empirical antimicrobial therapy especially in areas where culture facility is not available against *S. aureus* isolate from burn patients. Finally, strict consideration for *s. aureus* infection and proper usage of antibiotic policy are recommended in decreasing the incidence and occurrence of multidrug resistant (MDR) *S. aureus* infections in Al-Diwanyah – Teaching Hospital.

Overuse and misuse of antibiotics can promote the development of antibiotic-resistant bacteria [21] . Every time a person takes antibiotics,

sensitive bacteria (bacteria that antibiotics can still attack) are killed, but resistant bacteria are left to grow and multiply. This is how repeated use of antibiotics can increase the number of drug-resistant bacteria. [13]

Antibiotics are not effective against viral infections like the common cold, flu, most sore throats, bronchitis, and many sinus and ear infections. Widespread use of antibiotics for these illnesses is an example of how overuse of antibiotics can promote the spread of antibiotic resistance. Smart use of antibiotics is key to controlling the spread of resistance

Bacteria can become resistant to antibiotics through several ways. Some bacteria can "neutralize" an antibiotic by changing it in a way that makes it harmless. Others have learned how to pump an antibiotic back outside of the bacteria before it can do any harm. Some bacteria can change their outer structure so the antibiotic has no way to attach to the bacteria it is designed to kill. [11]

After being exposed to antibiotics, sometimes one of the bacteria can survive because it found a way to resist the antibiotic. If even one bacterium becomes resistant to antibiotics, it can then multiply and replace all the bacteria that were killed off. That means that exposure to antibiotics provides selective pressure making the surviving bacteria more likely to be resistant. Bacteria can also become resistant through mutation of their genetic material.[12]

Chapter Six
Conclusion and Recommendations

Conclusion and Recommendations:

1- Burn patients were most commonly infected by bacteria especially with staphylococcus spp.

2- All isolated bacterial pathogens were multi-drug resistant, which is an alarming trend that could be a leading cause for mortality in burn patients.

4- Reports should be documented periodically to evaluate bacterial resistance from time to time.

5- Once bacterial resistance is identified, this should be reported to physicians and central health laboratory.

6- People should be educated to use antibiotics when necessary only as the aggressive unnecessary use of antibiotics could result in resistance

Chapter Seven
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الخلاصة :

توفر الحروق موقعًا مناسبًا لتضاعف البكتيريا وأكثر من ذلك حيث تعتبر
مصادر دائمة غنية بالبكتريا مقارنة بجروح العمليات الجراحية. المكورات العنقودية الذهبية واحدة
من العوامل الممرضة الأكثر عزلة في كل من ممارسات المجتمع والمستشفى.