



Scientific Research

University of Baghdad

AL-Kindy College of Medicine

Bacterial and fungal infections in burn and wound

A project Submitted to AL-Kindy College of Medicine In partial fulfilment of the requirement of a project module/ 3rd stage

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2023/1444

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صدق الله العظيم

سورة الإسراء {85}

Abstract:

- **Background:**

- The burn and wound provide a suitable site for bacterial multiplication.
- Burn injury is a major public health problem in many countries of the world.
- Infection is the most common cause of death and serious problems following thermal injury.
- Extensive burns contribute to immunosuppression and this renders such patients prone to invasive bacterial and fungal infections.
- Burn and wound infections can be classified on the basis of the causative organism, the depth of invasion, and the tissue response for treatment.

- **Objectives:**

- 1- To determine the type of bacteria & fungal that cause wound and burn infections.
- 2- To know the best of antibiotics used for treatment wound and burn infections.

- **Study design:**

This was a retrospective study.

- **Methods:**

A retrospective study with a sample size of 200 patients both inpatient and outpatient referred to the laboratory of Al-Kindy Teaching Hospital from (2nd of October 2022 to the 1st of May 2023) to assess burn and wound infections. Ethical approval and permissions to collect samples were obtained from the Al-Kindy College of Medicine and Al-Kindy Teaching Hospital.

- **Results:**

(200) samples of burn and wound infections were found (140) positive was 89 for burn and 51 for wound. Males held the higher percentage (56%) in burn infections and (63%) in wound infections compared to females (44%) and (37%) respectively. Then, analyzing the percentage of pathogens involved burn and wound infections, the study had found that the highest percentage was for *Pseudomonas aeruginosa* (41%) followed by *Klebsiella pneumonia* (30%) in burn infection, and *Staphylococcus aureus* (25%) followed by *Pseudomonas aeruginosa* (21%).

- **Conclusions:**

The highest rate of infections with bacteria was recorded for *Pseudomonas aeruginosa* in burn infections and for *Staphylococcus aureus* in wound infection. There was a different ratio between sensitivity and resistance to the types of antibiotics. *Pseudomonas aeruginosa* was highly sensitive to Colistin sulfate (64.8%), then to Lincomycin and Norfloxacin (24.3%) Ciprofloxacin (13.5%). while it was highly resistant to Cefepime (78.3%), Ciprofloxacin (75.6%), Amikacin (64.8%) and Aztreonam (56.7%), and *Staphylococcus aureus* was highly

sensitive to Colistin sulfate (85.7%), Clindamycin and Tetracycline (28.2%), While it was highly resistant to Amikacin and Cefepime (71.4%), Azithromycin, Aztreonam, Ciprofloxacin and Tetracycline (57.1%).

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INTRODUCTION

1. Introduction:

The burn & wound represents a susceptible site for opportunistic colonization by organisms of endogenous and exogenous origin. Patient factors such as age, extent of injury, and depth of burn in combination with microbial factors such as type and number of organisms, enzyme and toxin production, and motility determine the likelihood of invasive burn and wound infections ⁽¹⁾.

In the past, the predominant pathogens were bacterial, but with advancements in burn & wound care and the introduction of topical mafenide in 1964, the epidemiology of burn & wound infections has shifted such that fungal pathogens are now more common ⁽²⁾.

An increase in fungal colonization has been observed because of the widespread use of topical antibiotic agents. Male gender, older age, lower extremity burn, scald burn, full-thickness burn, delay in treatment, and pre-existing diabetes place patients at increased risk of infection. Fungal wound infections pose a special challenge and cause substantial morbidity ⁽³⁾.

Most of the study found that the most causative agents followed the fungi were the endogenous bacteria, which will lead us to think that despite the fact that endogenous bacteria are beneficial to the host in their natural habitat (e.g., prevent the overgrowth of opportunistic pathogens and colonization by antibiotic resistant bacteria), many of these microorganisms are potentially pathogenic. Consequently, any disturbance in the relationship between the host and the normal micro flora may make the host more susceptible to infection. Such disturbance was likely to occur when endogenous bacteria are presented with an opportunity to colonize a new habitat, an example of which was the: wound and burn infections ⁽¹²⁾.

Causes of burn and wound infections relate to the loss of the protective barrier of the skin and thrombosis of the subcutaneous blood vessels. The resulting avascular wound bed makes an excellent medium, which can support the growth of microorganisms as well as prevent the penetration of systemically administered antimicrobial drugs ⁽⁷⁾.

Generally, microorganisms routinely isolated from burn and wound Include aerobic organisms like *Staphylococcus aureus*, *Streptococcus pyogenes*, *E. coli*, *Klebsiella Spp.*, *Proteus* etc., anaerobic organisms like *Bacteroides Fragilis*, *Peptostreptococcus*, *Propionibacterium Spp.*, *Fusobacterium Spp* and fungi like *Aspergillus niger*, *Candida Spp* and *Zygomycetes* ⁽⁵⁾.

It has been found that the distribution of various species of bacteria from burn and wound surfaces was similar to that from blood Specimens ⁽⁶⁾.

Regarding the relationship between gender, and burn and wound infections, females have been observed to have more prominent hormonal and cell mediated immune responses compared with males, so maintenance of immune integrity in females

following injury was due, at least in part, to the absence of immunosuppressive effects by androgenic hormones ⁽¹³⁾.

With advancements in burn care over the last 50 years, infections is now the leading cause of death after extensive burn injuries. Multiple study over the last decade have shown that 42%–65% of deaths in burn victims are attributable to infections ⁽¹⁰⁾.

The results of a study found that in the first days of the post burn hospitalization, more susceptible, Gram-positive organisms predominate, whereas later more resistant Gram-negative organisms are found ⁽⁹⁾.

Before starting to talk about wound infection, we should mention that there's two types of wounds (acute and chronic) If wound healing fails to progress through an orderly and timely sequence of events (i.e., months or years), then may defined as being chronic which is more exposed to be infected with some microorganisms. The presence of devitalized (non-viable) tissue, inadequate local tissue perfusion and unregulated (chronic) in amatory activity are all involved in the infections of the chronic wounds ⁽¹²⁾.

Some wound infections are caused by marine bacteria, which was mostly seen in persons with a history of seawater or seafood exposure ⁽⁸⁾.

Most common causative agents of:

a) Burn infections ^{(10)(11):}

- 1-*Pseudomonas aeruginosa*
- 2-*Staphylococcus aureus*
- 3-*Escherichia coli*
- 4-*Acinetobacter baumannii*
- 5-*Klebsiella pneumoniae*.

Pseudomonas aeruginosa produces a number of cell-associated (adhesins, alginate, pili, flagella, and lipopolysaccharide) and extracellular (elastase, exoenzyme S, exotoxin A, hemolysins, iron-binding proteins, leukocidins, and proteases) virulence factors that mediate a number of processes, including adhesion, nutrient acquisition, immune system evasion, leukocyte killing, tissue destruction, and bloodstream invasion, it also carries many intrinsic and acquired antimicrobial resistance traits that make infected burn and wound difficult to treat, which makes it the predominant pathogen for burn infections ⁽¹⁴⁾.

b) Wound infections ^{(22):}

- 1-*Staphylococcus aureus*
- 2-*Proteus mirabilis*

- 3-*Pseudomonas aeruginosa*
- 4-*Klebsiella aerogenes*
- 5-*Escherichia coli*

Staphylococcus aureus also has a diverse array of virulence factors that facilitate adherence to host tissues, immune system evasion, and destruction of host cells and tissues, including (coagulase, protein A, leukocidins, hemolysins, and superantigens). Resistance to methicillin in *Staphylococcus aureus*, and more recently emergence of resistance to glycopeptides and oxazolidinones, also complicate the treatment of burn and wound infections ⁽¹⁴⁾.

Risk factors:

The risk of burn and wound infections is directly correlated to the extent of the burn and wound and is related to the impaired resistance resulting from disruption of the skin's mechanical integrity and generalized immunosuppression ⁽⁴⁾.

a) Burn infections ⁽⁹⁾⁽¹⁷⁾⁽¹⁸⁾:

- 1-Delay for surgery due to patient medical instability
- 2-Lack of surgical facility
- 3-Poverty, overcrowding and lack of proper safety measures.
- 4-Multidrug-resistant bacterial pathogens
- 5-Hospitalization and it's duration (hospital-associated infections especially ventilator-associated pneumonia)

b) Wound infections ⁽²¹⁾:

- 1-Older age
- 2-Diabetes.
- 3-Immune system disorders, cancer, human immunodeficiency virus infection, and malnutrition.
- 4-Paralysis or other limited mobility (wheelchairs, confined to bed)
- 5-Hospitalization (organisms that are resistant to antibiotic).

Complications of:

a) Burn infections ⁽¹⁶⁾:

- 1-Cellulitis (most common)
- 2-Pneumonia

- 3-Sepsis
- 4-Toxic shock syndrome
- 5-Urinary tract infections (UTI)
- 6-Bloodstream infections (BSI).

b) Wound infections ⁽¹⁵⁾:

- 1-Cellulitis
- 2-Sepsis
- 3-Osteomyelitis
- 4-Hematomas
- 5-Bacteremia
- 6-Abscess formation

Methodology

2. Methodology:

-Study Design:

Retrospective design.

-Setting and duration:

The study was conducted in Al-Kindy College of Medicine (from 2nd of October 2022 to the 1st of May 2023).

-Study population and sampling procedure:

The study sample included patients admitted to Al-Kindy Hospital. Out of total number of patients, only a convenient sample of 200 patients was selected and they agreed to participate in the current study.

-Method of data collection:

A retrospective study consists of 200 patients referred to Al-Kindy Teaching Hospital for burn / wound culture and antibiotic susceptibility examination (from 2nd of October 2022 to the 1st of May 2023). Ethical approval and permissions to collect samples were obtained from the Al-Kindy College of Medicine and Al-Kindy Teaching Hospital.

The survey included the following types of questions:

Age, Gender, Type of infection, infectious pathogen, sensitivity and resistance to antibiotics and time of healing.

-Statistical analysis:

It was done by using IBM/SPSS version 25.0 (Statistical Package for social science) computer software. For sample description, a number and percentage were presented of in figures. P value less than or equal to 0.05 was considered statistically significant.

Results

3. Results:

Total number of samples (200) collected from Al-Kindy hospital; the positive samples were (140); was 89 for burn and 51 of wound. The majority of patients in burn infections were males (56.2%) compared to females (43.8%) as well as in wound infections where the majority of patients were also males (62.8%) compared to females (37.2%) represented in figure (1) (a & b).

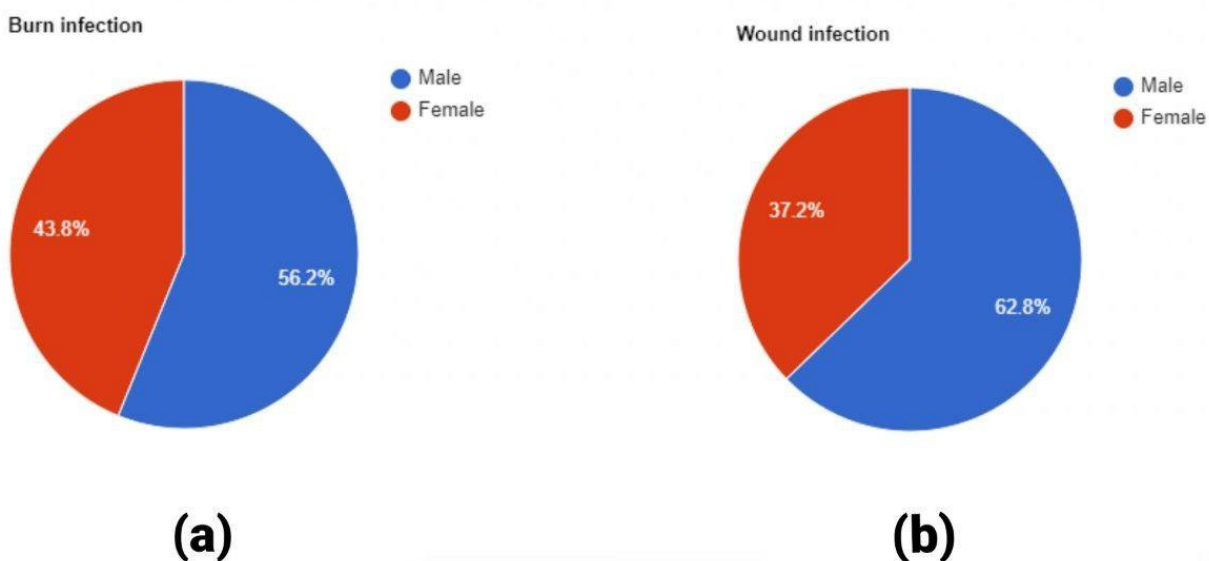


Figure (1): Distribution of (a) burn infections relation with gender (b) wound infections relation with gender.

The bacterial profile shows that the most common types of bacteria that recorded a higher rate of burn infections are: *Pseudomonas aeruginosa* was found to be the dominant bacteria with the frequency rate of (41.5%), the second most prevalent isolate was *Klebsiella pneumoniae* (30.3%), followed by *Acinetobacter baumannii* (16.8%) and *Pseudomonas fluorescens* (13.4%). As shown in figure (2).

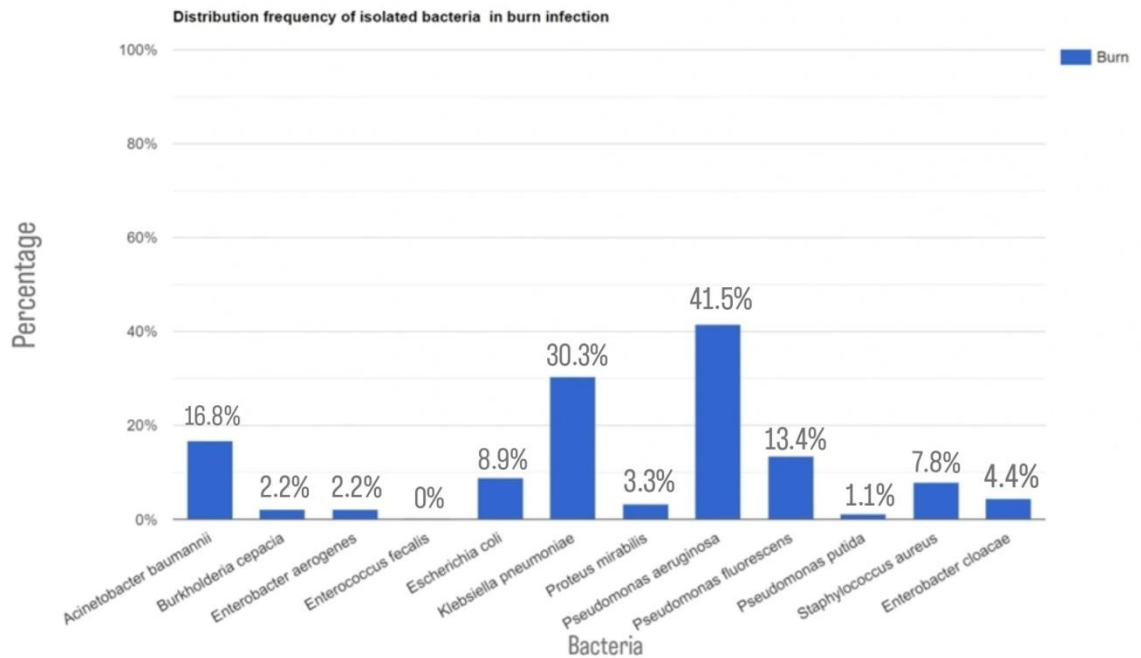


Figure (2): Distribution frequency of isolated bacteria in burn infection.

-While in figure (3), the most common types of bacteria that recorded a higher rate of wound infections are: *Staphylococcus aureus* was found to be the dominant bacteria with the frequency rate of (25.4%), the second most prevalent isolate was *Pseudomonas aeruginosa* (21.5%), followed by *Acinetobacter baumannii* (19.6%) and *Escherichia coli* (15.6%).

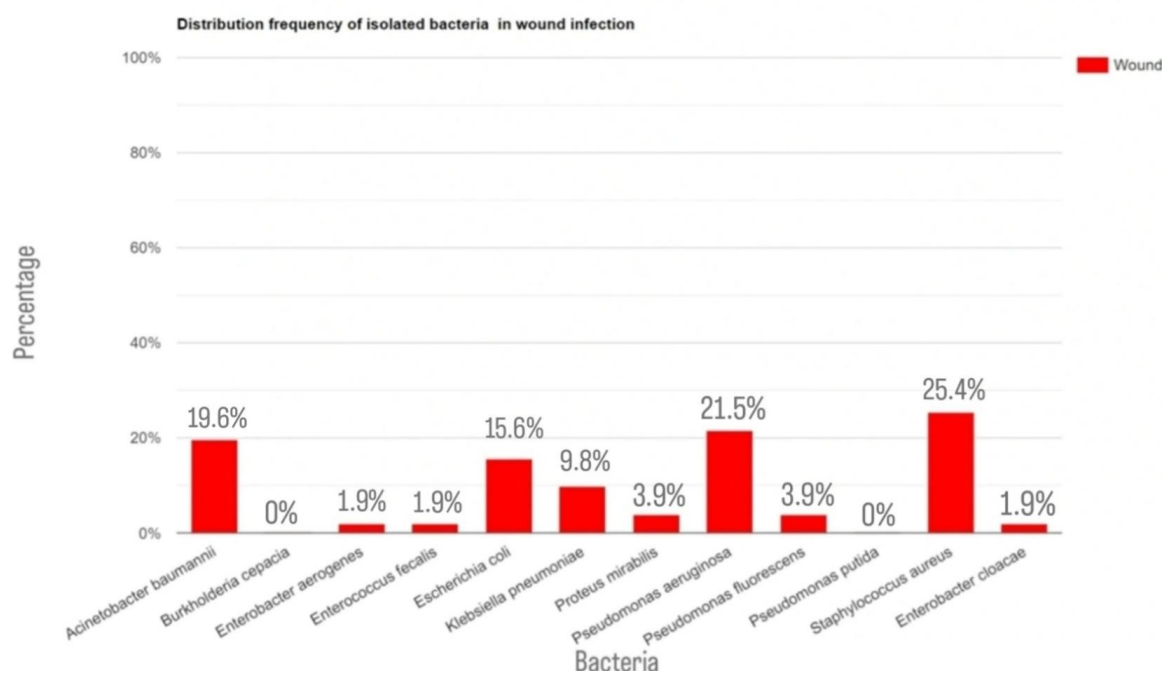


Figure (3): Distribution frequency of isolated bacteria in wound infection.

-The study has been relying on 12 types of antibiotics (Amikacin, Amoxicillin clavulanic acid, Azithromycin, Aztreonam, Cefepime, Ceftriaxone, Ciprofloxacin, Clindamycin, Colistin sulfate, Lincomycin, Norfloxacin and Tetracycline) which are most commonly used in the treatment of burn and wound infections.

-Regarding antibiotic susceptibility in burn infection, *Pseudomonas aeruginosa* was highly sensitive to Colistin sulfate (64.8%), then to Lincomycin and Norfloxacin (24.3%) Ciprofloxacin (13.5%), and it's less sensitive to Amikacin (10.8%), Aztreonam (5.4%) and Azithromycin (2.7%). while it was highly resistant to Cefepime (78.3%), Ciprofloxacin (75.6%), Amikacin (64.8%) and Aztreonam (56.7%), but it's less resistant to Colistin sulfate (2.7%) and Clindamycin (8.1%), then Amoxicillin clavulanic acid (13.5%) and Aztreonam (16.2%), followed by Ceftriaxone (18.9%) and Tetracycline (24.3%) (Fig. 4)

Antibiotics sensitivity p value: 0.122

Antibiotics resistance p value: 0.006

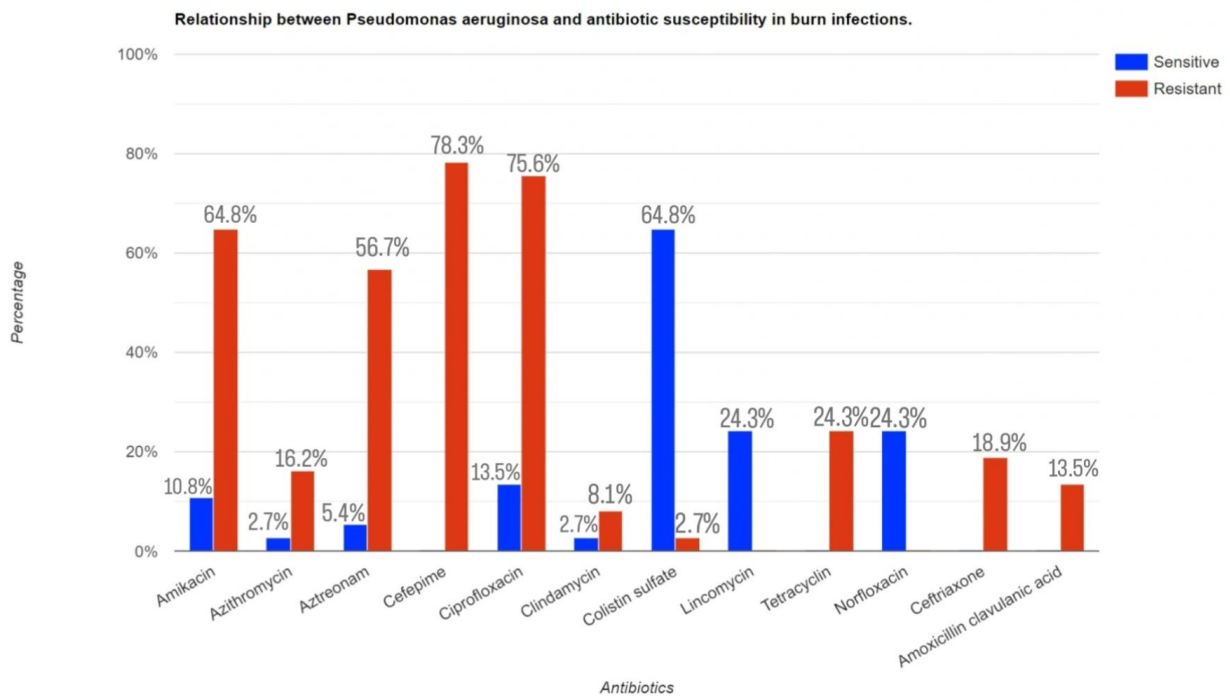


Figure (4): Relationship between *Pseudomonas aeruginosa* and antibiotic susceptibility in burn infections.

-Regarding antibiotic susceptibility in burn infection, *Klebsiella pneumoniae* was highly sensitive to Colistin sulfate (62.9%), then Lincomycin and Norfloxacin (29.6%), followed by Amikacin and Ciprofloxacin (14.8%), but it's less sensitive to Azithromycin, Cefepime, Tetracycline and Amoxicillin clavulanic acid (3.7%), followed by Aztreonam (7.4%). While it was highly resistant to Amikacin (85.1%), then Cefepime (81.4%), Aztreonam and Ciprofloxacin (77.7%), followed by Ceftriaxone and Amoxicillin clavulanic acid (54.2%), but it's less resistance to Colistin sulfate (7.4%), followed by Azithromycin and Tetracycline (44.4%) (Fig .5)

Antibiotics sensitivity p value: 0.047

Antibiotics resistance p value: 0.001

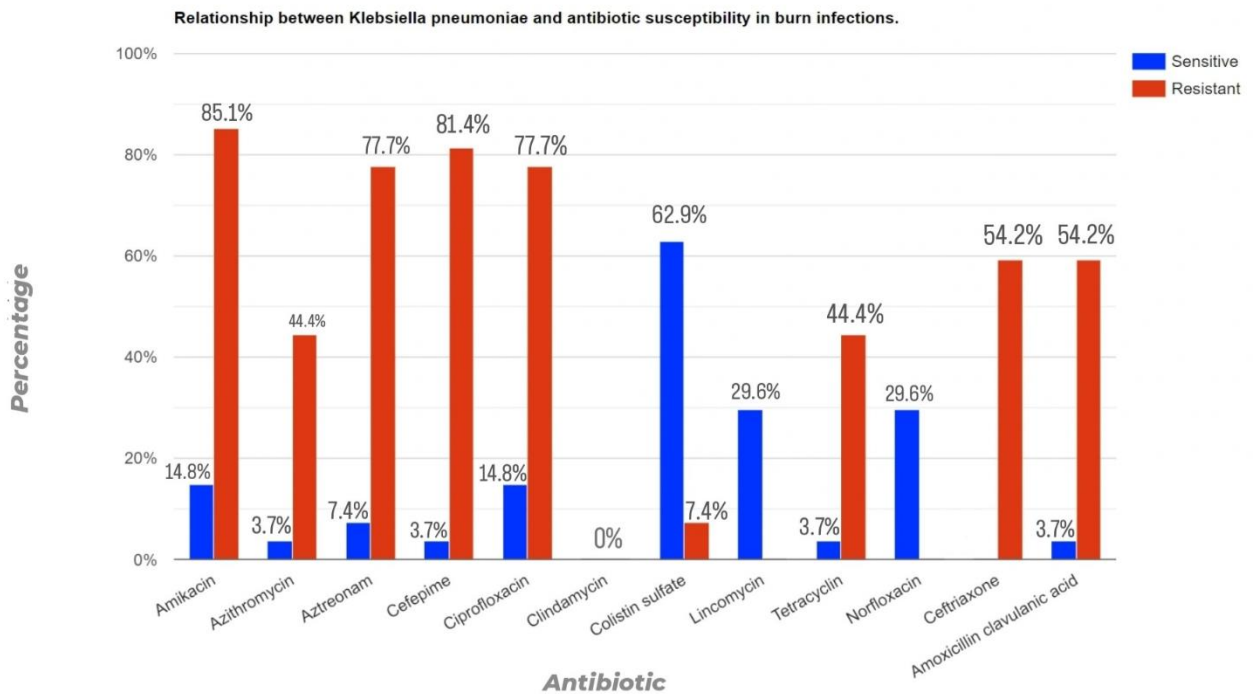


Figure (5): Relationship between *Klebsiella pneumoniae* and antibiotic susceptibility in burn infections.

-Regarding antibiotic susceptibility in burn infection, *Acinetobacter baumannii* was highly sensitive to Colistin sulfate (60%), then Lincomycin and Norfloxacin (26.6%), but it's less sensitive to Aztreonam (6.6%), followed by Ciprofloxacin (13.3%). While it's highly resistant to Cefepime (80%), Ciprofloxacin (73.3%), then Amikacin (53.3%), and Aztreonam (40%), but it's less resistant to Azithromycin and Amoxicillin clavulanic acid (13.3%), then Tetracycline (26.6%), followed by Ceftriaxone (33.3%) (Fig. 6).

Antibiotics sensitivity p value: 0.095

Antibiotics resistance p value: 0.006

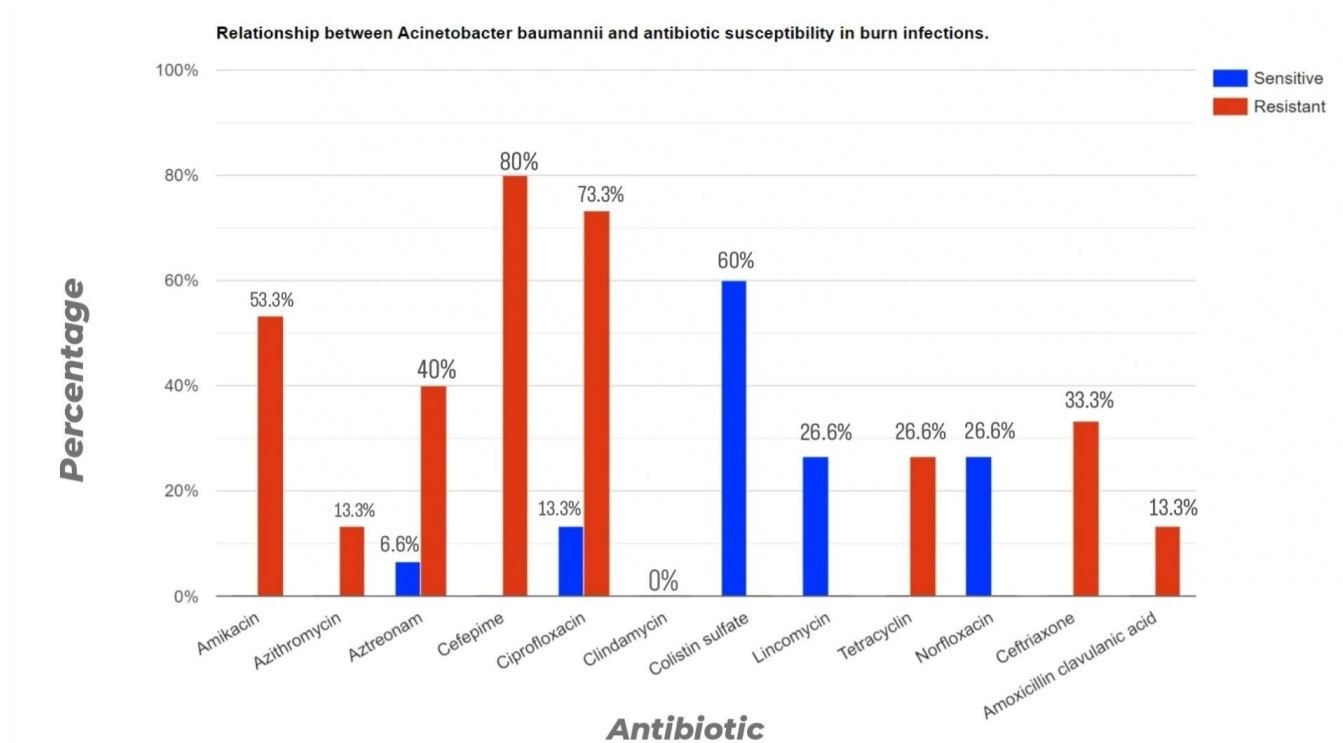


Figure (6): Relationship between *Acinetobacter baumannii* and antibiotic susceptibility in burn infections.

- Regarding antibiotic susceptibility in burn infection, *Pseudomonas fluorescens* was highly sensitive to Colistin sulfate (100%), but it's less sensitive to Amikacin, Clindamycin and Tetracycline (8.3%). While it was highly resistant to Cefepime (100%), then Amikacin (91.6%), followed by Aztreonam (75%), but it's less resistant to Tetracycline and Ceftriaxone (33.3%), then Azithromycin and Amoxicillin clavulanic acid (50%), followed by Ciprofloxacin (66.6%) (Fig. 7).

Antibiotics sensitivity p value: 0.391

Antibiotics resistance p value: 0.001

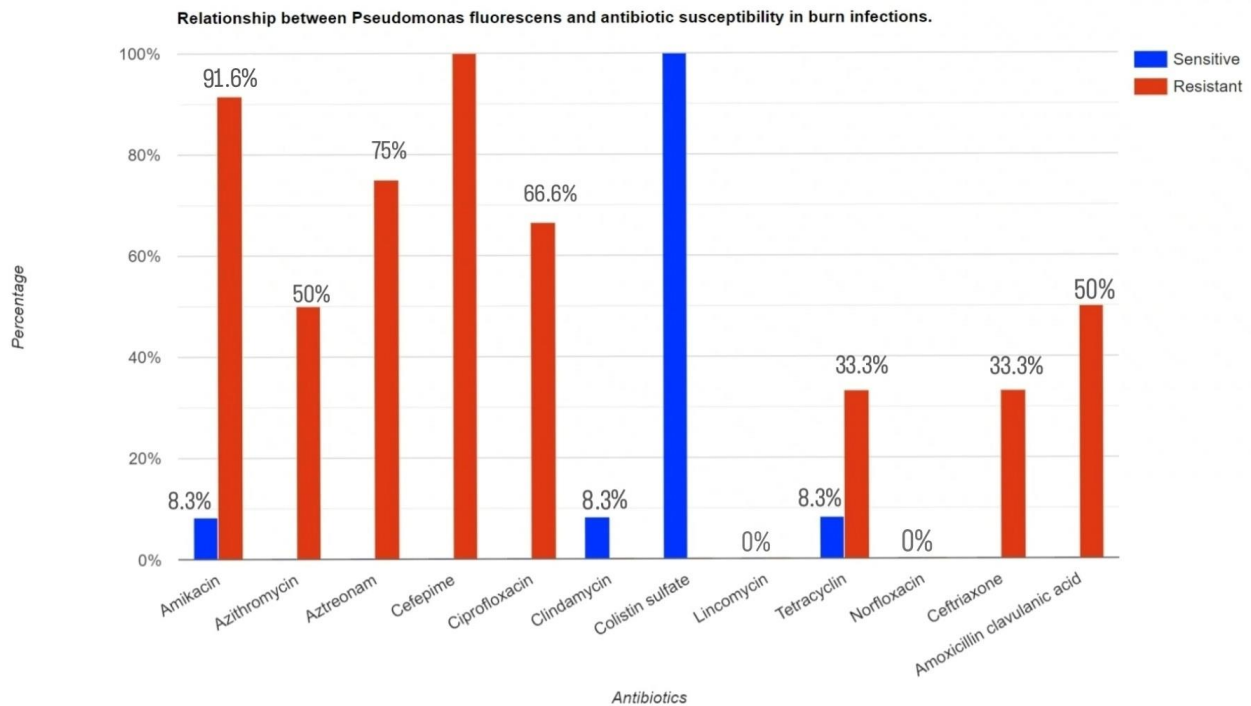


Figure (7): Relationship between *Pseudomonas fluorescens* and antibiotic susceptibility in burn infections.

-Other types of bacteria were also found in patients with burn infections included the following:

1. *Escherichia coli*
2. *Staphylococcus aureus*
3. *Enterobacter cloacae*
4. *Proteus mirabilis*
5. *Burkholdera cepacia*
6. *Enterobacter aerogenes*
7. *Pseudomonas putida*

-Regarding antibiotic sensitivity in burn infection, *Burkholdera cepacia* was sensitive in an equal proportion for each antibiotic (Amikacin, Cefepime, Ciprofloxacin and Colistin sulfate) in a percentage of (50%). *Enterobacter aerogenes* was highly sensitive for only Colistin sulfate (100 %). *Escherichia coli* was highly sensitive to Colistin sulfate (50%) and Amikacin (37.5%), but it's less sensitive to Cefepime (12.5%), followed by Lincomycin and Norfloxacin (25%). Antibiotics sensitivity p value: 0.048. *Proteus mirabilis* was highly sensitive to Amikacin and Ciprofloxacin (100%), followed by Cefepime (66.6%), but it's less sensitive to

Aztreonam, Ceftriaxone and Amoxicillin clavulanic acid (33.3%). Antibiotics sensitivity p value: 0.043.

-*Pseudomonas putida* was highly sensitive only to Amikacin (100%). *Staphylococcus aureus* was highly sensitive to Colistin sulfate (85.7%), Clindamycin and Tetracycline (28.2%), but it's less sensitive to Amikacin and Ciprofloxacin (14.2%). Antibiotics sensitivity p value: 0.206. *Enterobacter cloacae* was highly sensitive Ciprofloxacin (50%), but it's less sensitive to Amikacin, Cefepime, Colistin sulfate, Lincomycin and Norfloxacin (25%). Antibiotics sensitivity p value: 0.363.

- As for Azithromycin sensitivity in these seven types of bacteria, recorded cases were zero (Fig. 8).

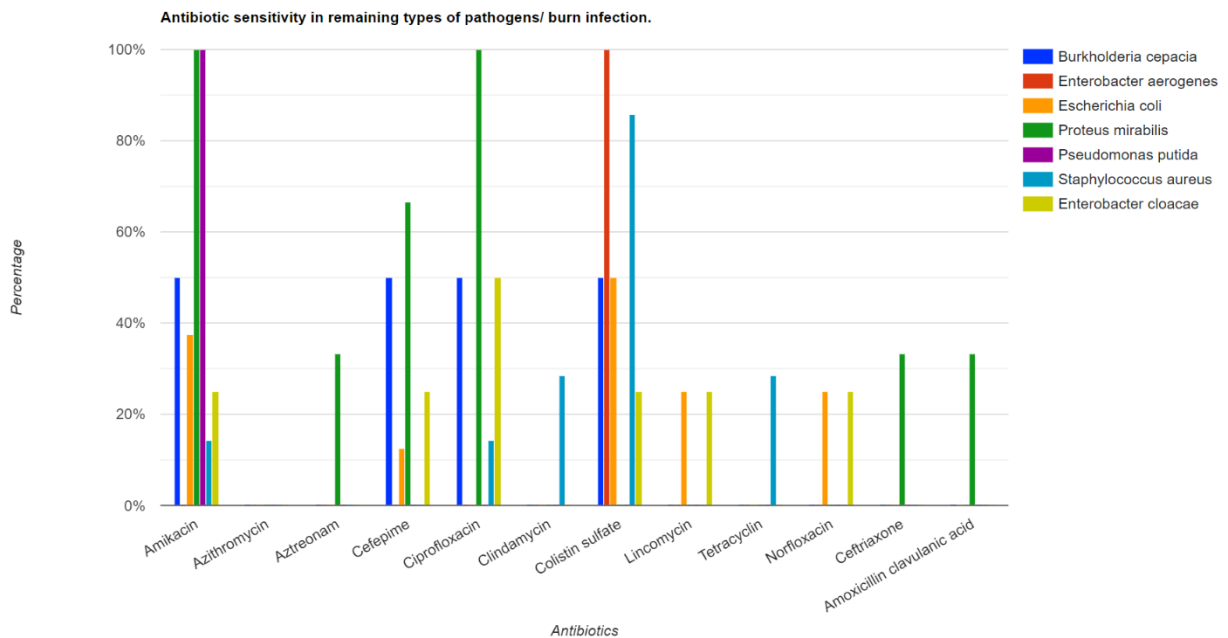


Figure (8): Relationship between antibiotic sensitivity and remaining types of pathogens/ burn infection.

-Regarding antibiotic resistance in burn infection, *Burkholderia cepacia* was resistant in an equal proportion for each antibiotic (Amikacin, Azithromycin, Amoxicillin clavulanic acid, Cefepime, Ciprofloxacin and Tetracycline) in a percentage of (50%). *Enterobacter aerogenes* was resistant to Ciprofloxacin, Ceftriaxone and Amoxicillin clavulanic acid (100%) and Amikacin, Aztreonam, Azithromycin, Cefepime and Tetracycline (50%). Antibiotics resistance p value: 0.08. *Escherichia coli* was highly resistant to Ciprofloxacin (100%), Ceftriaxone (87.5%) and Aztreonam (75%), followed by Amikacin, Azithromycin and Cefepime (62.5%), but

it's less resistant to Amoxicillin clavulanic acid (25%) followed by Tetracycline (37.5%). Antibiotics resistance p value: 0.001. *Proteus mirabilis* was highly resistant to Aztreonam and Colistin sulphate (66.6%), followed by Azithromycin, Tetracycline and Amoxicillin clavulanic acid (33.3%). Antibiotics resistance p value: 0.178.

-*Pseudomonas putida* was highly resistant to both Cefepime and Ciprofloxacin (100%). *Staphylococcus aureus* highly resistant to Amikacin and Cefepime (71.4%), Azithromycin, Aztreonam, Ciprofloxacin and Tetracycline (57.1%), but it's less resistant to Amoxicillin clavulanic acid (14.2%), and Clindamycin (42.8%). Antibiotics resistance p value: 0.001. *Enterobacter cloacae* was highly resistant to Aztreonam (100%), (Amikacin, Azithromycin, Cefepime, Ciprofloxacin, Tetracycline, Ceftriaxone, Amoxicillin clavulanic acid) all were (50%) and Colistin sulphate (25%). Antibiotics resistance p value: 0.015.

- As for Lincomycin and Norfloxacin resistance in these seven types of bacteria, recorded cases were zero (Fig. 9).

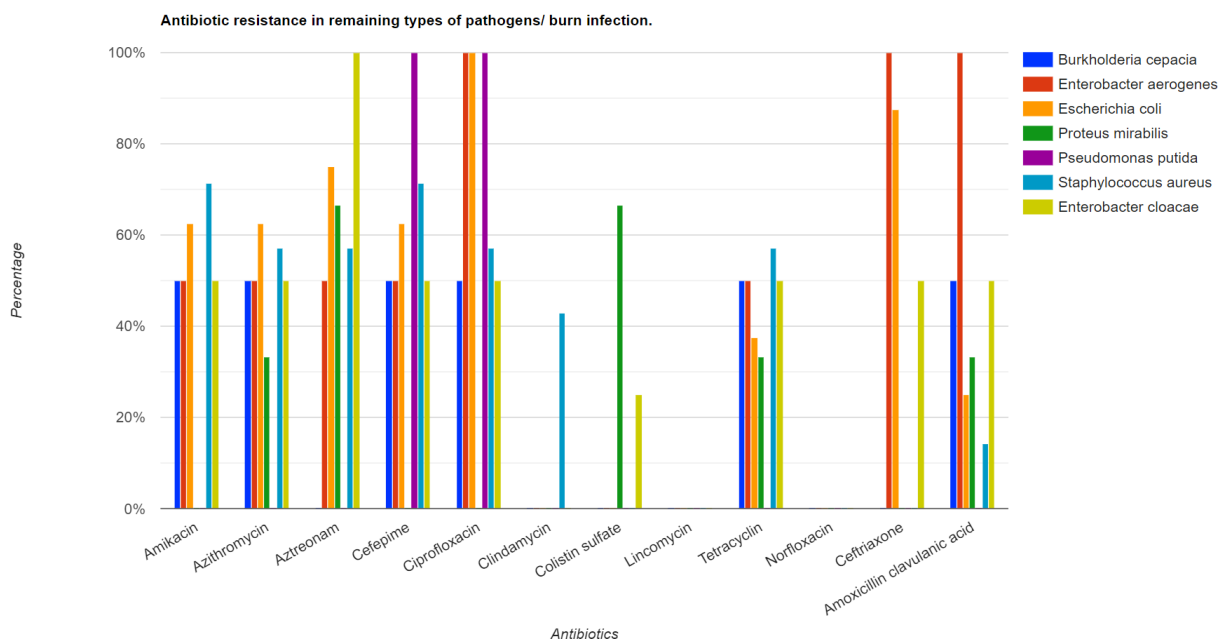


Figure (9): Relationship between antibiotic resistance and remaining types of pathogens/ burn infection.

-Regarding antibiotic susceptibility in wound infection, *Staphylococcus aureus* was highly sensitive to Ciprofloxacin (53.8%), Tetracycline, Lincomycin and

Norfloxacin (30.7%), and less sensitive to Amikacin and Clindamycin (7.6%). While it was highly resistant to Azithromycin (84.6%), Tetracycline (46.1%) and Ciprofloxacin (38.4%), but it's less resistant to Aztreonam and Cefepime (7.6%), Amikacin and Ceftriaxone (15.3%) followed by Clindamycin (23%) (Fig. 10).

Antibiotics sensitivity p value: 0.215

Antibiotics resistance p value: 0.048

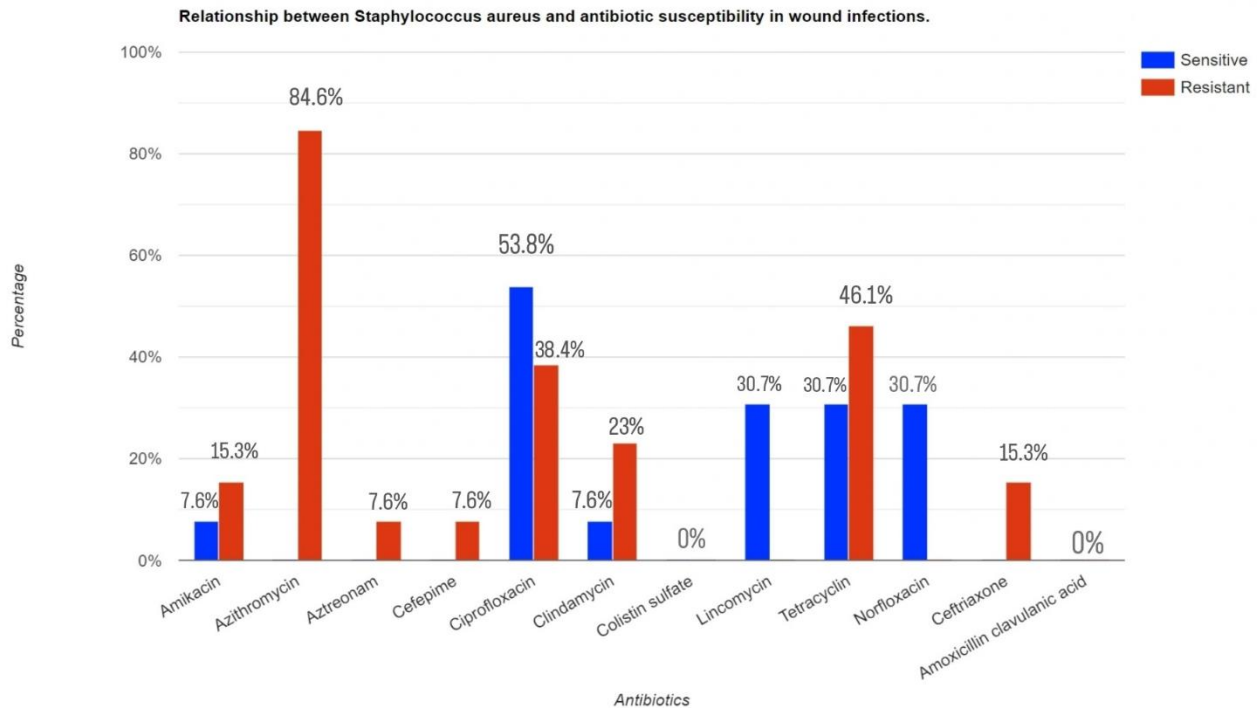


Figure (10): Relationship between *Staphylococcus aureus* and antibiotic susceptibility in wound infections.

-Regarding antibiotic susceptibility in wound infection, *Pseudomonas aeruginosa* was highly sensitive to Lincomycin and Norfloxacin (90.9%), and less sensitive to Colistin sulfate and Ciprofloxacin (9%). While it was highly resistant to Aztreonam and Ciprofloxacin (90.9%), followed by Amikacin (72.7%) and Cefepime (63.6%), but it's less resistant Azithromycin and Ceftriaxone (9%) (Fig. 11).

Antibiotics sensitivity p value: 0.423

Antibiotics resistance p value: 0.058

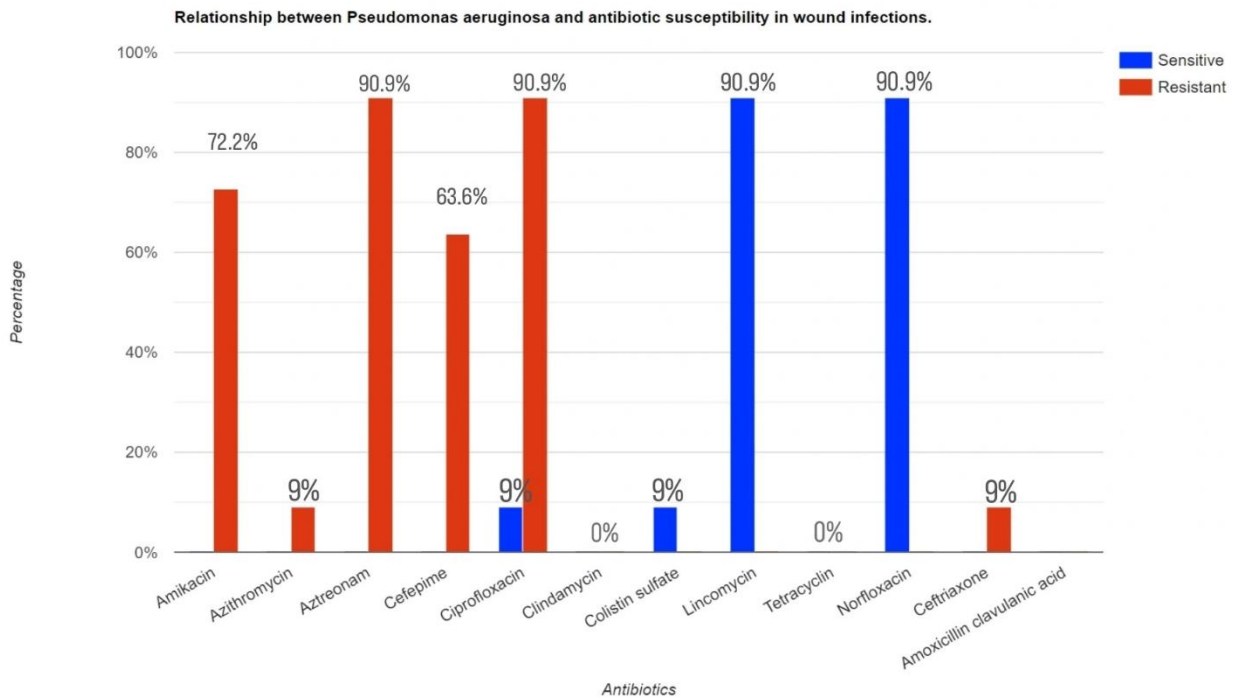


Figure (11): Relationship between *Pseudomonas aeruginosa* and antibiotic susceptibility in wound infections.

-Regarding antibiotic susceptibility in wound infection, *Acinetobacter baumannii* was highly sensitive to Lincomycin and Norfloxacin (60%), but it's less sensitive to Cefepime (10%) and Colistin sulfate (40%). While it was highly resistant to Ciprofloxacin (90%), then Amikacin, Aztreonam and Cefepime (60%), followed by Ceftriaxone (50%) and Amoxicillin clavulanic acid (40%) and less resistant to Azithromycin and Tetracycline (30%) (Fig. 12).

Antibiotics sensitivity p value: 0.071

Antibiotics resistance p value: 0.001

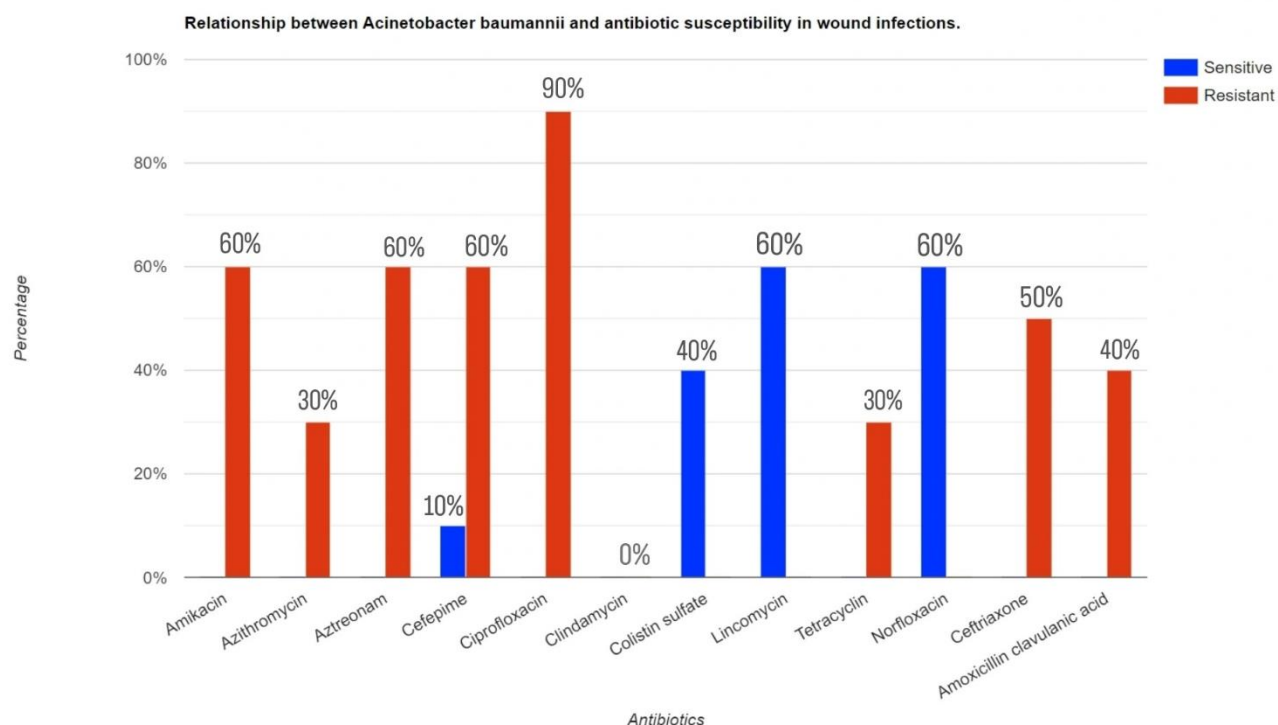


Figure (12): Relationship between *Acinetobacter baumannii* and antibiotic susceptibility in wound infections.

-Regarding antibiotic susceptibility in wound infection, *Escherichia coli* was highly sensitive to Amikacin (50%) and Colistin sulfate (37.5%), but it's less sensitive to Aztreonam, Azithromycin and Tetracycline (12.5%). While it was highly resistant to Ciprofloxacin (100%) then Cefepime and Ceftriaxone (75%), followed by Tetracycline (62.5%), Amikacin and Aztreonam (50%), and it's less resistant to Amoxicillin clavulanic acid (37.5%) followed by Azithromycin (12.5%) (Fig. 13).
 Antibiotics sensitivity p value: 0.189
 Antibiotics resistance p value: 0.002

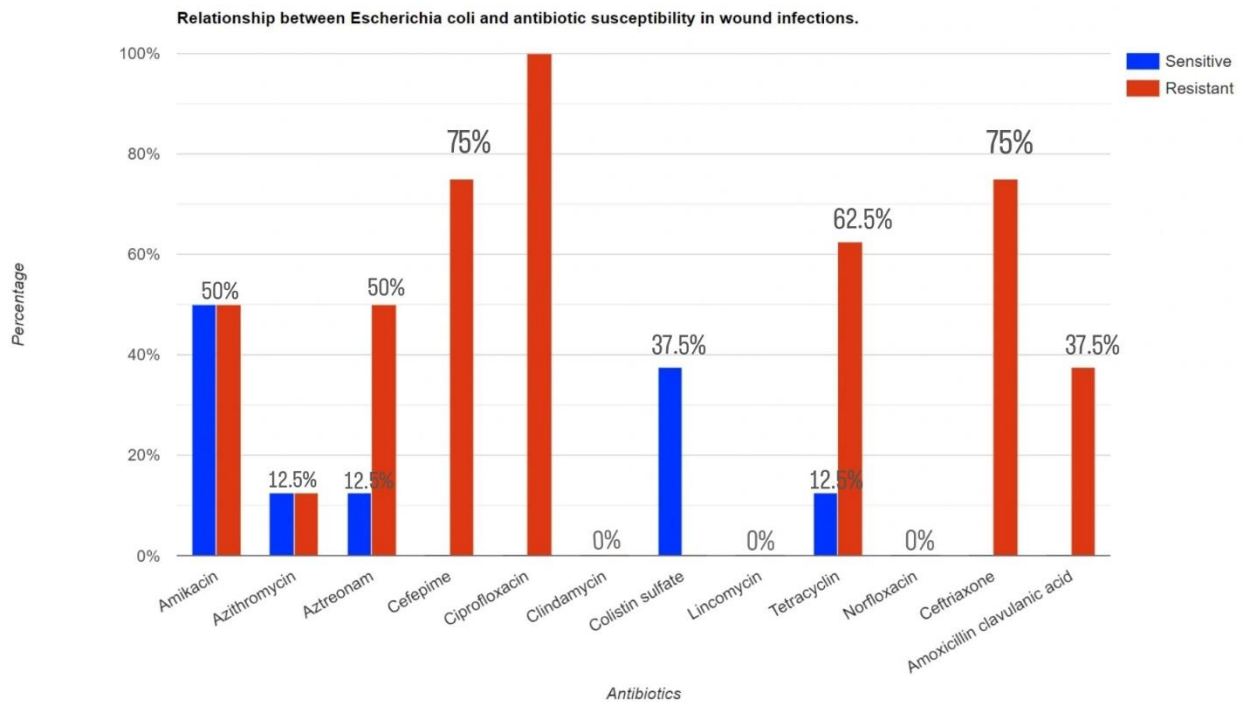


Figure (13): Relationship between *Escherichia coli* and antibiotic susceptibility in wound infections.

-Other types of bacteria were also found in patients with wound infections included the following:

1. *Klebsiella pneumoniae*
2. *Proteus mirabilis*
3. *Pseudomonas fluorescens*
4. *Enterobacter aerogenes*
5. *Enterobacter cloacae*
6. *Enterococcus faecalis*

-Regarding antibiotic sensitivity in wound infection, *Enterobacter aerogenes* was highly sensitive to Amikacin, Aztreonam and Ceftriaxone (100%). *Enterococcus faecalis* was highly sensitive to only Ciprofloxacin (100%). *Klebsiella pneumoniae* was highly sensitive to Amikacin, Cefepime, Ciprofloxacin and Colistin sulphate (40%) and less sensitive to Aztreonam, Azithromycin, Amoxicillin clavulanic acid, Lincomycin and Norfloxacin (20%). Sensitivity p value: 0.035.

-*Proteus mirabilis* was highly sensitive in an equal proportion for each antibiotic (Amikacin, Aztreonam, Azithromycin, Cefepime, Ceftriaxone and Ciprofloxacin) in

a percentage of (50%). *Pseudomonas fluorescens* was sensitive in an equal proportion for each antibiotic (Aztreonam, Lincomycin and Norfloxacin) in a percentage of (50%). *Enterobacter cloacae* was highly sensitive to Amikacin, Aztreonam and Ciprofloxacin (100%).

- As for Clindamycin and Amoxicillin clavulanic acid sensitivity in these six types of bacteria, recorded cases were zero (Fig. 14).

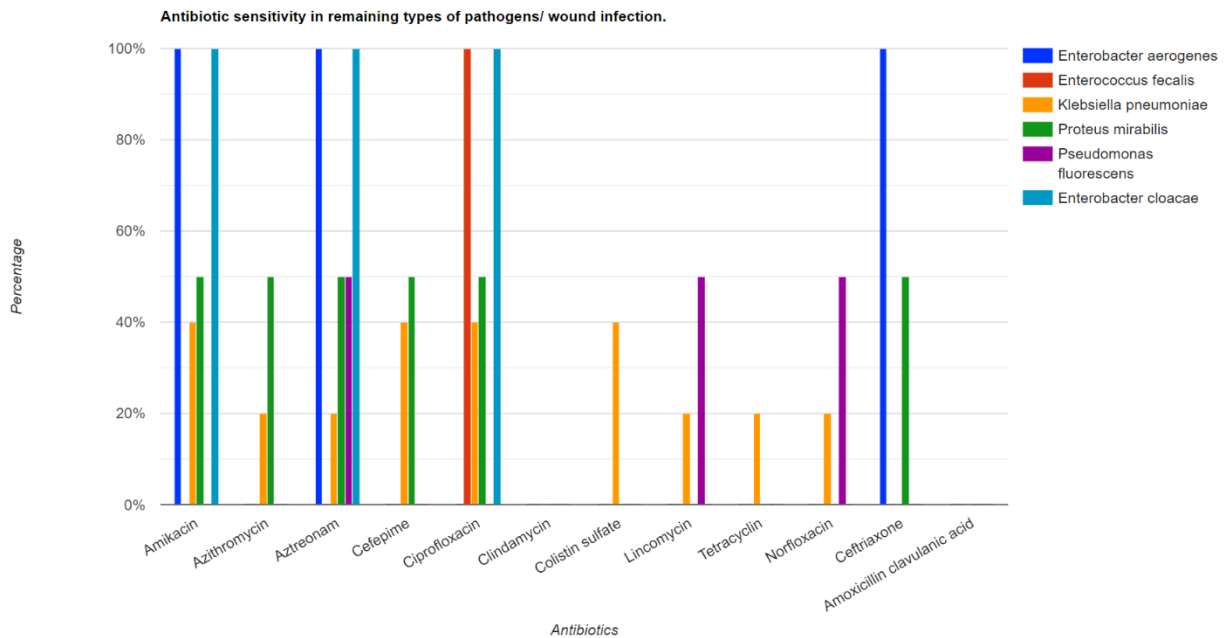


Figure (14): Relationship between antibiotic sensitivity and remaining types of pathogens/ wound infection.

-Regarding antibiotic resistance in wound infection, *Enterobacter aerogenes* was highly resistant to Ciprofloxacin and Tetracycline (100%). *Enterococcus fecalis* was highly resistant to only Tetracycline (100%). *Klebsiella pneumoniae* was highly resistant to Amikacin, Aztreonam, Ceftriaxone, and Ciprofloxacin (60%), followed by Azithromycin, Amoxicillin clavulanic acid and Cefepime (40%) and it's less resistant to Tetracycline (20%). Resistance p value: 0.001

-*Proteus mirabilis* was highly resistant in an equal proportion for each antibiotic (Amikacin, Aztreonam, Ceftriaxone, Ciprofloxacin and Tetracycline) in a percentage of (50%). *Pseudomonas fluorescens* was resistant to Amikacin, Cefepime and Ciprofloxacin (100%) and less resistant to Aztreonam (50%). *Enterobacter cloacae* was highly resistant to Ceftriaxone and Tetracycline (100%).

-As for Clindamycin, Colistin sulfate, Lincomycin and Norfloxacin resistance in these six types of bacteria, recorded cases were zero (Fig. 15).

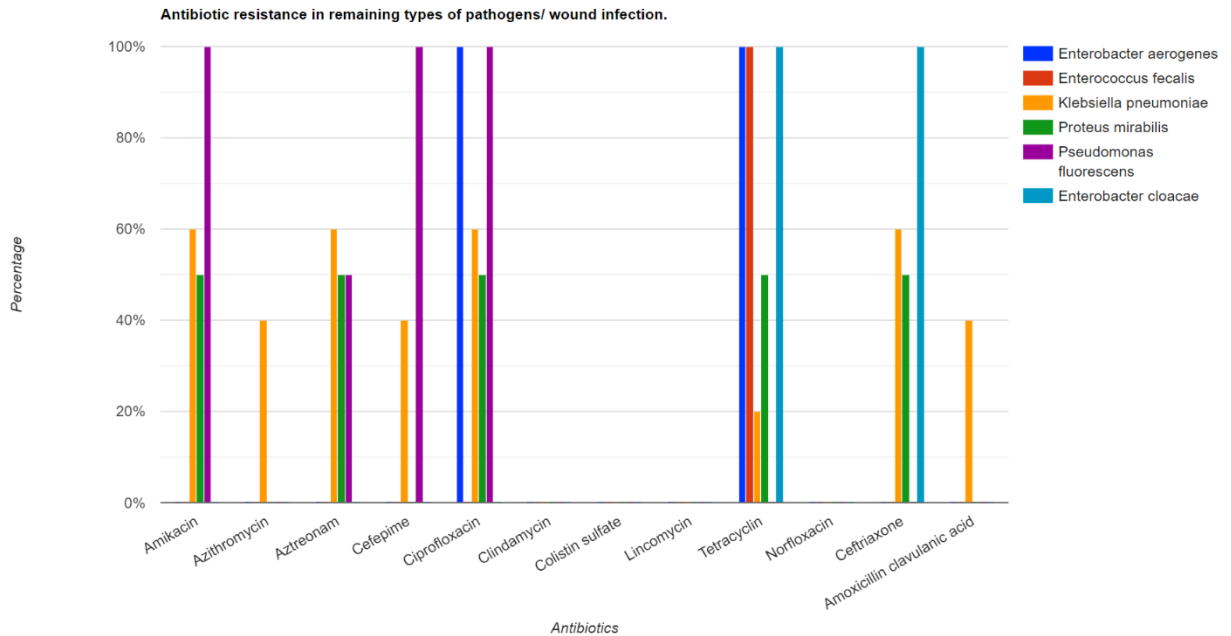


Figure (15): Relationship between antibiotic resistance and remaining types of pathogens/ wound infection.

-It should also be mentioned that we found one fungal infections of unspecified type for a patient with wound infection, as it was accompanied by two other types of bacteria which are, *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*. These bacteria and fungi were resistant to Amikacin, Azithromycin and Ciprofloxacin, while they were sensitive to Lincomycin and Norfloxacin

-Healing time of burn infections pathogens according to their sensitivity to particular antibiotics, the study noticed that the bacteria that recorded the longest healing time (15 days) were *Klebsiella pneumoniae* and *Proteus mirabilis* and the antibiotics that contribute to the healing process of each bacterium were Colistin sulfate and Amikacin respectively, followed by *Acinetobacter baumannii* and *Pseudomonas fluorescens* that recorded 14 days as an average of healing time with effect of Colistin sulfate.

-While the bacteria that recorded the shortest time of healing were *Enterobacter aerogenes* with only 3 days, then come *Staphylococcus aureus*, *Escherichia coli* and

Pseudomonas aeruginosa with 5, 7 and 11 days respectively, all with effect of Colistin sulfate as recorded in figure (16).

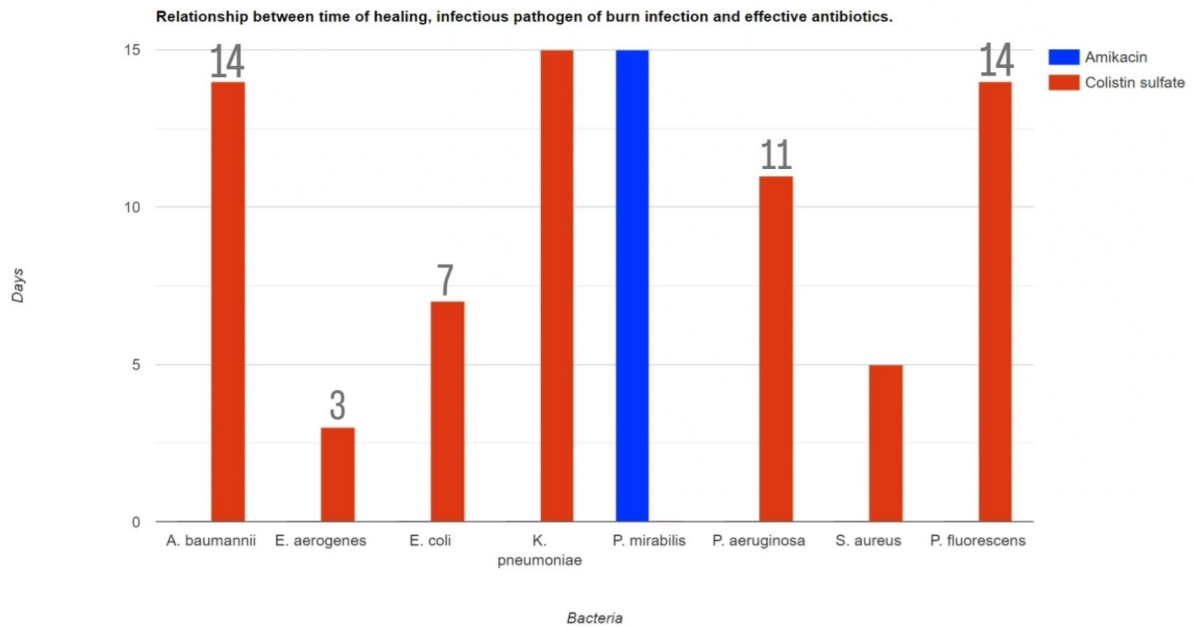


Figure (16): Relationship between time of healing, infectious pathogen of burn infections and effective antibiotics.

-About healing time of wound infections, The study found that the longest healing time was recorded for *Acinetobacter baumannii* with 25 days under Colistin sulfate effect, followed by *Pseudomonas aeruginosa* with 14 days with contribution of Norfloxacin and Lincomycin.

-While the shortest time of healing was recorded for *Escherichia coli* with 6 days followed by *Proteus mirabilis* with 7 days, was affected by Amikacin and Azithromycin respectively According to the recorded information in figure (17).

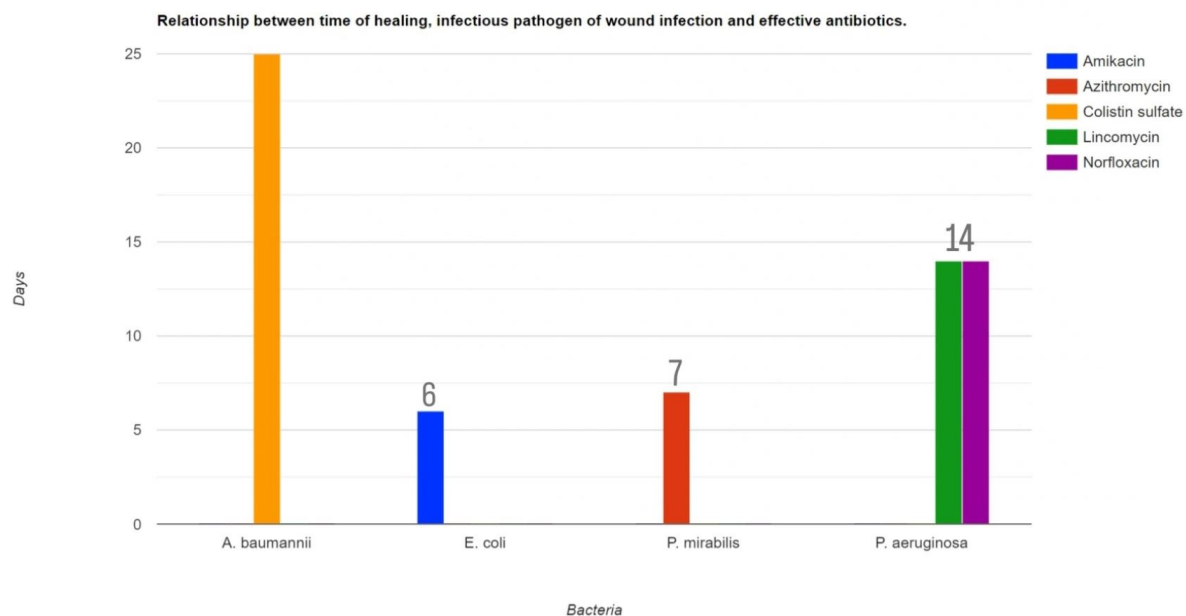


Figure (17): Relationship between time of healing, infectious pathogen of wound infections and effective antibiotics.

Discussion

4. Discussion:

The study observed that the frequency of burn and wound infections was more in males than females in the (140) isolates obtained, (63%) were from male patients while (37%) were from female in wound infections and (56%) were from male patients while (44%) were from female in burn infections. This was backed with other study which reported that these infections were more frequent in males than in females ⁽¹⁷⁾ due to the fact that males are responsible for the most duties outside the home. This was in contrast with other study ⁽²³⁾ in which females were more frequently victims of burn infections than males.

Regarding burn infection, *Pseudomonas aeruginosa* was found to be the dominant bacterium causing these infections which usually cause most severe and life-threatening infections in burn patients ⁽²⁴⁾. This result backed with other study ⁽²⁵⁾. The second most common bacterium was *Klebsiella pneumonia*, this totally backed with other study in military care center in 2010 ⁽²⁶⁾. Then it's followed by *Acinetobacter baumannii* which its high susceptibility was explained by other study because it's a nosocomial infection ⁽²⁷⁾, then *Pseudomonas fluorescence* respectively.

Pseudomonas aeruginosa, results found it was highly sensitive to Colistin sulfate. These results correspond to other study ⁽²⁸⁾, and was highly resistant to Cefepime. This backed with study reported that Cefepime has minimal inhibitory activity against *Pseudomonas aeruginosa* ⁽²⁹⁾.

Regarding *Pseudomonas fluorescence* was also found common in burn infections results, this backed with a study saying that it's the second most gram-negative bacterium cause burn infections ⁽³⁰⁾. Results found this bacterium was highly sensitive to Colistin sulfate which in contrast with the study ⁽³¹⁾. and was highly resistant to Amikacin and Cefepime which backed with the same study applied in Iraq ⁽³¹⁾.

Concerning *Klebsiella pneumonia* was found to be highly sensitive to Colistin sulfate. This backed with other study in which they added silver nanoparticles to Colistin sulfate which makes the bacterium incapable of developing resistance ⁽³²⁾, and was highly resistant to Amikacin, this was confirmed by a study that explained that *Klebsiella* has DNA fragment specified both an acetyltransferase activity and low level of phosphotransferase activity, these two activities were responsible for the high resistance to Amikacin ⁽³³⁾.

Acinetobacter baumannii showed high sensitivity to Colistin sulfate, Lincomycin and Norfloxacin. This does not go with the results of other study which found its highest sensitivity to Colistin, Imipenem and Meropenem ⁽³⁴⁾. Also showed high

resistance to Cefepime and Ciprofloxacin, which backed with other study ensure its full resistance to Ciprofloxacin ⁽³⁵⁾.

Regarding wound infections, *Staphylococcus aureus* was the dominant bacterium causes these infections followed by *Pseudomonas aeruginosa*. This corresponds to other study reported that *Staphylococcus aureus* and *Pseudomonas aeruginosa* were the most common bacteria isolated from chronic wound infections. The co-infections of them together was more virulent than single infections ⁽³⁶⁾.

Then by *Acinetobacter baumannii* which resembled third most common bacterium. This backed with other study that the most common organisms isolated were *Staphylococcus aureus* in the first week and *Acinetobacter baumannii* in the second week ⁽³⁷⁾.

Followed by *Escherichia coli* that explained it's high isolation by other study because *Staphylococcus aureus* was the most common bacterium in wound infections after clean surgery while *Escherichia coli* dominated in cultures from infected wound after contaminated surgery ⁽³⁸⁾.

Regarding *Staphylococcus aureus*, it was found to be highly sensitive to Ciprofloxacin which does not go with the results of other study saying that Ciprofloxacin appears to have limited and usefulness effect in treating *Staphylococcal infections* ⁽³⁹⁾, and was having high resistance to Azithromycin which confirmed by other study ⁽⁴⁰⁾.

Then regarding *Pseudomonas aeruginosa*, the results indicate that it was highly sensitive to Lincomycin and Norfloxacin and highly resistant to Aztreonam and Ciprofloxacin. Which contracts with other study that reported Levofloxacin and Polymycin B as the most sensitive antibiotics and Aztreonam and Ceftazidime as the most resistant antibiotics to these bacteria ⁽⁴¹⁾.

Now regarding *Acinetobacter baumannii*, according to our results it has high sensitivity to Lincomycin and Norfloxacin and high resistance to Ciprofloxacin. The sensitivity was not very specific because *Acinetobacter baumannii* was nosocomial pathogen developmental through time and became less sensitive to known antibiotics but the resistance backed with the study ⁽⁴²⁾.

Considering *Escherichia coli*, it was found highly sensitive to Amikacin which confirmed by other study ⁽⁴³⁾ and almost full resistant to Ciprofloxacin, Cefepime and Cefotaxime which correspond to other study in Pakistan ⁽⁴⁴⁾.

The research showed the time of recovery for different types of burn bacterial pathogens, that the fastest bacterial pathogen to heal was *Enterobacter aerogenes* followed by *Staphylococcus aureus* and the slowest bacteria that takes more than fifteen days was *Klebsiella pneumonia* followed by *Acinetobacter baumannii* and *Pseudomonas aeruginosa* which takes less than fifteen days this was totally backed with this study in military care center ⁽²⁶⁾.

Other information showed in the results that almost all of these bacteria were treated by Colistin sulfate except for *Proteus mirabilis* which was treated by Amikacin. That backed with antimicrobial susceptibility pattern of bacteria causing burn infections with the studies ⁽³²⁾⁽³⁴⁾.

Time of recovery for different types of wound bacterial pathogens, showed that the fastest bacterial pathogen to heal was *Escherichia coli* with three days, that corresponds to the study in rats saying that *E. coli* have the highest healing time rate with 96.4% in wound infections ⁽⁴⁵⁾, and the slowest bacterium to heal was *Acinetobacter baumannii* which took 25 days, this also correlates with the study which reported that the wound was still open after three weeks ⁽⁴⁶⁾.

Other information showed in results that these two bacteria were treated with Colistin sulfate and Amikacin which they have a high sensitivity to these antibiotics as we mentioned previously ⁽⁴³⁾⁽³⁴⁾.

In addition, burn infections remain the most important factor limiting survival in burn intensive care units. Also, it's known that large wound surface impaired immune systems and broad-spectrum antibiotic therapy contribute to the growth of opportunistic fungal species ⁽⁴⁷⁾, this case also showed in result with one patient which had two bacteria isolated with fungi. It's mostly because the wound environment may promote multispecies biofilm formation interaction between bacteria and fungi in wound ⁽⁴⁸⁾, these two bacteria showed sensitivity and resistance to some antibiotics. Although, the nature of fungal infections dictates aggressive treatment to minimize the morbidity association with these infections ⁽⁴⁹⁾.

Conclusions & Recommendation

5. Conclusions:

1. The prevalence of wound and burn infections were higher in males than females.
2. *Staphylococcus aureus* is the most causative agent of wound infections and *Pseudomonas aeruginosa* is most causative agent of burn infection.
3. Effective drugs may be in a clinical study; in burn infections bacteria were highly sensitive to Colistin sulfate and highly resistant to Cefepime while wound infections, the bacteria were highly sensitive to Ciprofloxacin and highly resistant to Azithromycin.
4. In a clinical study; the bacteria found with the longest time of healing in burn infections were *Klebsiella pneumoniae* and *Proteus mirabilis*, and the bacterium with the shortest time of healing was *Enterobacter aerogenes*. While in wound infections; the longest recovery time was recorded by *Acinetobacter baumannii*, and the shortest time recorded by *Escherichia coli*.
5. According to our study, the presence of fungal infections in burn and wound is extremely rare.

5. Recommendations:

- 1- Extended the sample size of the study.
- 2- In the future, it is possible to use nanoparticles as an alternative antimicrobial treatment to burn and wound infections.

References

6. **References:**

1. Macedo JL, Santos JB. Bacterial and fungal colonization of burn wounds. *Memórias do Instituto Oswaldo Cruz*. 2005;100:535-9.
2. Nash G, Foley FD, Goodwin MN, Bruck HM, Greenwald KA, Pruitt Jr BA. Fungal burn wound infection. *JJAM* 1971;215:1664–6.
3. Burn wound colonization, infection, and sepsis, Husayn A Ladhani, Charles J Yowler, Jeffrey A Claridge, *Surgical infections* 22 (1), 44-48, 2021.
4. Menon T. A study of burn wound infections and immune response in burns: Thesis. University of Kerala;1984.
5. Lawrence JC, Lilly HA. A quantitative method for investigating the bacteriology of skin: its application to burns. *Br J Exp Pathol*. 1972 Oct;53(5):550-9.
6. Siddique NA. Burn injury is preventable. An analysis of 716 cases in a burn unit. *JCPSP* 1998; 8: 148-52.
7. Weber JM, Sheridan RL, Pasternack MS, Tompkins RG: Nosocomial Infections in Pediatric Patients with Burns: Proposed Definitions and Benchmark Rates. *Am J Infect Control* 1997, 25:195-201.
8. Wound infections caused by *Vibrio vulnificus* and other marine bacteria, JD Oliver, *Epidemiology & Infection* 133 (3), 383-391, 2005.
9. *Clinical Infectious Diseases*, Volume 65, Issue 12, 15 December 2017, Pages 2130–2136.
10. Anne M Lachiewicz, Christopher G Hauck, David J Weber, Bruce A Cairns, David van Duin. Bacterial Infections After Burn Injuries: Impact of Multidrug Resistance. *Clinical Infectious Diseases*, Volume 65, Issue 12, 15 December 2017, Pages 2130–2136.
11. Sathya Bhama, Resmi Rajan, Ramani Bai Joseph Theodor. A study on bacterial profile of burn and wound infections. Year: 2013 | Volume: 15 | Issue: 2 | Page: 54-5.
12. Philip G Bowler. Wound pathophysiology, infection and therapeutic options. Pages 419-427 | Published online: 08 Jul 2009.
13. Patrick J. Offner, MD, MPH; Ernest E. Moore, MD; Walter L. Biffl, MD. Male Gender Is a Risk Factor for Major Infections After Surgery. *Arch Surg*. 1999;134(9):935-940. doi:10.1001/archsurg.134.9.935.

14. Deirdre Church, Sameer Elsayed, Owen Reid, Brent Winston, Robert Lindsay. Burn Wound Infections. Review 1 April 2006.
15. Mate Zabaglo; Tariq Sharman. Postoperative Wound Infection. September 19, 2022.
16. Agnieszka Markiewicz-Gospodarek, Małgorzata Kozioł, Maciej Tobiasz, Jacek Baj, Elżbieta Radzikowska-Büchner, Agata Przekora . Burn Wound Healing: Clinical Complications, Medical Care, Treatment, and Dressing Types: The Current State of Knowledge for Clinical Practice. 2022 Feb; 19(3): 1338.
17. N.A. Al laham, A.A Elmanama, and G.A. Tayh. possible risk factor associated with burn wound colonization of Gaza Strip hospital , palastine. 2013 June 30 :26(2):68-75.
18. Stacy Schiurring and Jess Bell. Assessment of Infection in Burn Injuries. 2016; 17(2): 250-255.
19. Robert A. Weinstein, C. Glen Mayhall. The Epidemiology of Burn Wound Infections: Then and now. Clinical Infectious Diseases, Volume 37, Issue 4, 15 August 2003, Pages 543–550,
20. Sue E. Gardner PhD, RN, Rita A. Frantz PhD, RN, FAAN, Bradley N. Doebbeling MD, MSc, FACP. The validity of the clinical signs and symptoms used to identify localized chronic wound infection. 23 December 2001
21. Janet M. Torpy, MD, Writer; Alison Burke, MA, Illustrator; Richard M. Glass, MD, Editor. Wound Infections. JAMA. 2005;294(16):2122. doi:10.1001/jama.294.16.2122
22. Christopher Aye Egbe (M.Phil), Richard Omoregie (M.Phil), Isaac Ohioenuan Igbarumah (B.MLS), Samson Onemu (M.Phil). Microbiology of Wound Infections and its Associated Risk Factors among Patients of a Tertiary Hospital in Benin City, Nigeria .2011;11(2):109-113.
23. Tigist alebachew, Gizachew yismaw , ayelegn derabe, and zufan sisay. staphylococcus aureus burn wound infection among patient attending yekatit 12 hospital burn unit Addis Ababa ,Ethiopia. Ethiopian journal of health sciences 22 (3), 2012
24. Ian Alan Holder. P. aeruginosa Burn Infections: Pathogenesis and Treatment. Pseudomonas aeruginosa as an Opportunistic Pathogen, 275-295, 1993
25. Jean-Baptiste Ronat, Jabar Kakol, Marwan N Khoury, Mathilde Berthelot, Oliver Yun, Vincent Brown, Richard A Murphy. Highly drug-resistant pathogens

- implicated in burn-associated bacteremia in an Iraqi burn care unit. *PloS one* 9 (8), e101017, 2014
26. Edward F. Keen III a, Brian J. Robinson a, Duane R. Hospenthal a b, Wade K. Aldous a, Steven E. Wolf c, Kevin K. Chung c, Clinton K. Murray. Incidence and bacteriology of burn infections at a military burn center. June 2010, Pages 461-468
 27. Ting Hway Wong, Ban Hock Tan, Moi Lin Ling. Multi-resistant *Acinetobacter baumannii* on a burns unit—clinical risk factors and prognosis. June 2002, Pages 349-357
 28. E. Sans-Serramitjana a, E. Fusté a b, B. Martínez-Garriga a, A. Merlos a, M. Pastor c, J.L. Pedraz c, A. Esquisabel c, D. Bachiller d, T. Vinuesa. Killing effect of nanoencapsulated colistin sulfate on *Pseudomonas aeruginosa* from cystic fibrosis patients. September 2016, Pages 611-618
 29. Luqman Satti, Shahid Abbasi, Tanveer Ahmed Qumar, Muhammad Shoaib Khan, Zahid Ahmed Hashmi. In Vitro Efficacy of Cefepime Against Multi-Drug Resistant *Pseudomonas aeruginosa* – An Alarming Situation in our Setup. 1876-5211 — Volume 1, 2011
 30. Fahimeh Beige, Majid Baseri Salehi, Nima Bahador, Sina Mobasherzadeh. Plasmid mediated antibiotic resistance in isolated bacteria from burned patients. *Jundishapur Journal of Microbiology* 8 (1), 2015
 31. Haider Qassim Raheem, Yasser H. Almawla. Silver Nanoparticles as Antibacterial Action against *Pseudomonas Fluorescens* Isolated from Burn Infection. ISSN:1583-6258, Vol. 25, Issue 4, 2021, Pages. 12578 - 12583
 32. Nadia Wali 1, Aroosh Shabbir, Nadia Wajid, Nasir Abbas & Syed Zeeshan Haider Naqvi. Synergistic efficacy of colistin and silver nanoparticles impregnated human amniotic membrane in a burn wound infected rat model, (2022) 12:6414 Benedetto Caroleo.
 33. M E Tolmasky, M Roberts, M Woloj, J H Crosa. Molecular cloning of amikacin resistance determinants from a *Klebsiella pneumoniae* plasmid. *Antimicrobial agents and chemotherapy* 30 (2), 315-320, 1986, 1 August 1986
 34. Karol Sieniawski, Krzysztof Kaczka, Monika Rucińska, Ludmiła GaGIS, Lech PoMorski. *Acinetobacter baumannii* NOSocomial Infections. 2013, 85, 9, 483–490
 35. Cheng-Hsun Chiu, Hao-Yuan Lee, Li-Yun Tseng, Chyi-Liang Chen Ju-Hsin Chia, Lin-Hui Su, Shu-Ying Liu. Mechanisms of resistance to ciprofloxacin,

- ampicillin/sulbactam and imipenem in *Acinetobacter baumannii* clinical isolates in Taiwan. April 2010, Pages 382-386
36. Raffaele Serra, Raffaele Grande, Lucia Butrico, Alessio Rossi, Ugo Francesco Settimio, Chronic wound infections: the role of *Pseudomonas aeruginosa* and *Staphylococcus aureus*. show all Pages 605-613 | Published online: 08 Mar 2015
 37. Sarita Otta, Jayant Kumar Dash, Bichitrananda Swain. Aerobic bacteriology of burn wound infections. 2015 337-341
 38. Renvall S, Niinikoski J, Aho AJ.. Wound infections in abdominal surgery. A prospective study on 696 operations. *Acta Chirurgica Scandinavica*, 01 Jan 1980, 146(1):25-30
 39. Henry M. Blumberg, David Rimland, Donna J. Carroll, Pamela Terry, I. Kaye Wachsmuth. Rapid Development of Ciprofloxacin Resistance in Methicillin-Susceptible and -Resistant *Staphylococcus aureus*. *The Journal of Infectious Diseases*, Volume 163, Issue 6, June 1991, Pages 1279–1285,
 40. Rashedul Hasan, Mrityunjoy Acharjee, Rashed Noor. Prevalence of vancomycin resistant *Staphylococcus aureus* (VRSA) in methicillin resistant *S. aureus* (MRSA) strains isolated from burn wound infections. June 2016, Pages 49-53
 41. Nagwa M. Atef, Sanaa M. Shanab, Sahar I. Negm & Yasmien A. Abbas. Evaluation of antimicrobial activity of some plant extracts against antibiotic susceptible and resistant bacterial strains causing wound infection. *Bulletin of the national research center* volume 43, Article number: 144 (2019)
 42. Lihua Qi, Hao Li, Chuanfu Zhang, Beibei Liang, Jie Li, Ligui Wang, Xinying Du, Xuelin Liu, Shaofu Qiu and Hongbin Song. Relationship between Antibiotic Resistance, Biofilm Formation, and Biofilm-Specific Resistance in *Acinetobacter baumannii*. *Front. Microbial.*, 12 April 2016
 43. IOANA MOȘ, OTILIA MICLE, MIHAELA ZDRÂNCĂ, MARIANA MUREȘAN, LAURA VICAȘ. ANTIBIOTIC SENSITIVITY OF THE *ESCHERICHIA COLI* STRAINS ISOLATED FROM INFECTED SKIN WOUNDS. *FARMACI* 2010, Vol. 58, 5
 44. Muhammad Asif Habeeb, 1 Yasra Sarwar, 2 Aamir Ali, 3 Muhammad Salman, 4 and Abdul Haque 5. Rapid emergence of ESBL producers in *E. coli* causing urinary and wound infections in Pakistan. 2013 Apr; 29(2): 540–544.
 45. Huifeng Dong, Liangyu Wang, Lin Du, Xing Wang, Qin Li, Xiaoyue Wang, Jie Zhang, Jun Nie, Guiping Ma. Smart polycationic hydrogel dressing for dynamic wound healing. *Small* 18 (25), 2201620, 2022

46. Mitchell G Thompson, Chad C Black, Rebecca L Pavlicek, Cary L Honnold, Matthew C Wise, Yonas A Alamneh, Jay K Moon, Jennifer L Kessler, Yuanzheng Si, Robert Williams, Suleyman Yildirim, Benjamin C Kirkup Jr, Romanza K Green, Eric R Hall, Thomas J Palys, Daniel V Zurawskiv. validation of a novel murine wound model of *Acinetobacter baumannii* infection. *Antimicrobial agents and chemotherapy* 58 (3), 1332-1342, 2014
47. M.F. Struck and J. Gille. *Ann Burns Fire Disasters*. Fungal infections in burns: a comprehensive review. 2013 Sep 30; 26(3): 147–153.
48. Lindsay Kalan, Elizabeth A Grice. Fungi in the wound microbiome. *Advances in wound care* 7 (7), 247-255, 2018.
49. JB Wright, K Lam, D Hansen, RE Burrell. Efficacy of topical silver against fungal burn wound pathogens, *American journal of infection control* 27 (4), 344-350, 1999

Appendices

7. **Appendices:**

- Questionnaire:
 1. Gender
 2. Type of infection
 3. Infectious pathogen
 4. Type of antibiotic
 5. Resistance and sensitivity of antibiotics
 6. Time of healing.
- Information source:
 1. Books
 2. Journal articles
 3. Theses and dissertation
 4. Internet