# ETIOLOGY AND RISK FACTORS OF BACTERIAL WOUND INFECTIONS

BY

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## DECLARATION

This dissertation is my original work and has not been presented for a degree anywhere else to the best of my knowledge. No part of this document should be reproduced without permission of the author and/or University of Nairobi.

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*To Almighty God* for all that I am and I have, praise be His Holy name.

## DEDICATION

This dissertation is dedicated to all clinicians and microbiologists, in particular those working at KNH. It is also dedicated to researchers who will find it very useful as a resource material.

## ABSTRACT

**Background**: Kenyatta National Hospital (KNH) is a referral center serving patients from Kenya and beyond. There are several departments among them orthopedics which houses many patients with wounds, some of which are infected thereby increasing morbidity and mortality. This research focused on etiology and risk factors of bacterial wound infections in the orthopedics wards.

**Objective:** To assess the factors that contribute to wound infections. The specific factors assessed were prevalence of aerobic bacteria, use of antibiotics and clinical practices among the nurses when dressing wounds.

**Methods:** A descriptive research design was used and target populations were nurses and hospitalized patients in the department of orthopedics at KNH. Sixteen nurses and one hundred and fifteen patients were selected using simple random sampling and convenience sampling techniques respectively. Data was collected using a questionnaire and specimens taken from wounds analyzed in microbiology laboratories of UON and KNH.

**Results:** The prevalence of bacteria isolated was; *Pseudomonas* spp. *(42.6%), Proteus* spp. *(33.9%), Staphylococcus aureus (33%), Klebsiella* spp. *(7.9%), Streptococcus faecalis (6.1%), Enterbacter* spp. *(2.6%), Alcaligenes* spp. *(1.7%), Citrobacter freundii (0.9%), Serratia* spp. *(0.9%),* and *Acinetobacter baumanii (0.9%).*

The sensitivity patterns were as follows: *Pseudomonas* spp.; Pipril/Tozabactam (89.9%), Meropenem (75.5%), Gentamycin (55.1%), Amikacin (73.5%), Ceftazidime (82.6%), Ceftriaxone (30.6%), Ticarcillin/Clavulonic acid (65.3%) and Piperacillin (83.7%).

*Proteus* spp.; Ceftazidime (89.7%), Ceftriaxone (79.5%), Ciprofloxacin (87.2%), Augmentin (76.9%), Cefuroxime (61.5%), Piperacillin (48.7%), Gentamycin (46.2%)

Minocycline (51.1%).

*Staphylococcus aureus* portrayed the following sensitivity pattern; Minocycline (86.8%), Clindamycin (84.2%), Ciprofloxacin (62.2%), Gentamycin (60.5%), Oxacillin (55.3%), Erythromycin (44.7%), Augmentin (39.5%) and Amoxicillin (13.2%). Irrational prescribing was widespread where in most cases duration of administration of the drug was not indicated. In some cases, the nurses were observed not to follow recommended methods of managing wounds.

**Conclusion:** Management of wounds in orthopedics at KNH was deficient in several aspects that require to be improved.

**Recommendations**: Use of aseptic techniques should be given more attention when dressing wounds and rational use of antibiotics should be encouraged in orthopedic wards at KNH.

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## LIST OF ABBREVIATIONS

BA - Blood Agar

CBA –Chocolate Blood Agar

DNA-Deoxy ribonucleic acid

FDA- Food and Drug Administration

KNH-Kenyatta National Hospital

MIC - Minimum Inhibitory Concentration

MIU-Motility Indole Urea

MOH-Ministry of health

MRSA-Methicillin resistant *Staphylococcus aureus*

NCCL-The National Committee for Clinical Laboratory Standards

PAST-Performance standard for Antimicrobial Testing

PIP- Povidone iodine

RNA-Ribonucleic acid

SPSS-Statistical package for social sciences

UON-University of Nairobi

WHO-World health organization

# DEFINITION OF TERMS

**Aerobe-** A microbe that requires the presence of oxygen for life and growth.

**Anaerobe-** A microbe that is able to live and grow in absence of free oxygen.

**Antibiotic-** A Substance produced by or derived from a living organism or artificially synthesized that destroys or inhibits the growth of other microorganism.

**Bacteria** - Microorganisms which lack distinct nuclear membrane and have cell wall of unique composition.

**Confounding variable-** A variable that affects relationship between dependent and independent variables.

**Culture-** Population of microorganism usually bacteria grown in solid or liquid laboratory medium.

**Dependent variable-** A variable that does not depend on other variables to get effected.

**Descriptive research design-**Studies, which are concerned with describing the characteristics of a particular individual.

**Etiology-** The cause of a specific disease.

**Flagellum-** Fine long whip like structure attached to certain types of bacteria responsible for locomotion.

**Gram-negative organism-** A microbe that stains red with safranin or neutral red.

**Gram-positive organism-** A microbe that stains blue with crystal violet.

**Incubation-** A process of development of bacterial culture.

**Independent variable-** A variable that does not depend on any other variable to be effected.

**Inoculation-** Introduction of a small quantity of bacteria on a culture plate.

**Morbidity** - State of being diseased.

**Mortality-** Number of deaths due to a disease.

**Nosocomial infection-**An infection whose development is favored by hospital environment.

**Orthopedics** -The science or practice of correcting deformities caused by disease or damage to the bones and joints of the skeleton.

**Pathogen-**A microorganism such as bacterium that infects an animal or man and produces a disease.

**Pathogenic-** Capable of causing a disease.

**Petri dish-**A flat shallow circular glass or plastic dish with a pillow like box lid used to hold solid agar or gelatin media for culturing bacteria.

**Pili-**Hair like projections present on the surface of certain bacteria which are test involved in adhesion of bacteria to other cells and in transfer of DNA.

**Prevalence-** Number of occurrences of microorganisms in a population of patients.

**Sample-**A representative portion of the target population.

**Sensitivity test disc-** Round paper disc impregnated with a known quantity of antibiotic, which is placed on bacteria culture to test for sensitivity.

**Sepsis-** The putrefactive destruction of tissues by disease causing bacteria or their toxins.

**Target population-** Collection of all subjects of interest.

# CHAPTER ONE: INTRODUCTION

## 1.1 Background

### 1.1.1 Introduction

Wound infection is not a modern phenomenon [1]. As early, as 14-37 A.D there is documentary evidence that Cornelius Celsius (Roman Physician) described the four principle signs of inflammation and used antiseptic solution. Another Roman physician Claudius Gallen (130 – 200A.D) had such an influence on the management of wounds that he is still thought of by many today as the father of surgery. It should be remembered that he and some of his followers instigated the laudable pus theory, which incorrectly considered the development of pus in a wound as a positive part of healing.

### 1.1.2 History and organization structure of Kenyatta National Hospital

Kenyatta National Hospital (KNH) was established in 1901 as a native civil hospital with two ward bed facilities [2]. The hospital expanded its services to cater for Africans and Asians between 1922 and 1937. In 1952, it was renamed king George the VI and following Kenyan independence in 1963, the hospital was yet again renamed Kenyatta National Hospital in honour of the founding president of the Republic of Kenya, Mzee Jomo Kenyatta. Since independence, the hospital has continued to develop and in 1987, it became a state corporation. The hospital is at the apex of referral system in Kenya. It provides specialized medical care, training and research and other services that fall under its mandates in Kenya and the region. It is the second largest hospital in Africa with a bed capacity of 1800 and a staff complement of 6213.

The Board of management is responsible for development of policies [2]. The executive director is responsible for day-to-day running of the hospital and has two deputies namely; Deputy Director's clinical services and administrative services. The former is incharge of clinical departments, which includes public health, nutrition, occupational therapy, pharmacy, radiology, laboratory medicine, Obstetric and Gynaecology, Dentistry, Surgery, Anesthesia/ICU, Medicine, Dermatology, Pediatrics, Medical social work, Medical records, Physiotherapy and Orthopedics.

The deputy director administrative service is responsible for the departments of Personnel and Training, Finance, Hospital engineering, Supplies and Procurement and Administration [2]. Other departments not under the above two departments include; Quality assurance unit, Legal unit, Public relations, internal audit, Planning and data and Chief Nurse.

### 1.1.3 Wound infections

The characteristics of infected tissue includes; flimsy friable granulation, superficial bridging within the wound, spontaneous bleeding or bleeding on light contact, pain or discomfort within the wound, delayed healing or wound exudates, pus secretion and vasculitis or inflammation in the tissue surrounding the wound [3].

The development of a wound infection depends on the complex interplay of many factors, namely: type of organism, antibiotic use and clinical practices amongst hospital staff [1]. If the integrity and protective mechanisms are damaged, organisms of different types will enter the wound and initiate an inflammatory response. This may be characterized by the classic signs of

redness, pain, swelling and fever. This process ultimately aims at restoring homeostasis.

The potential for infection depends on a number of patient variables such as the state of hydration, immune status, nutrition and existing medical conditions as well as extrinsic factors related to pre, intra and post-operative care if the patient has undergone surgery [1]. This often makes it difficult to predict which wounds will become infected and therefore the prevention of wound infection should be a primary management objective for all Health Care practitioners.

The 2002 survey report by the nosocomial infection surveillance done in United Kingdom, which covered the period between October 1997 and September 2001, indicated that the incidence of hospital-acquired infection (HAI) related to surgical wounds was 10% [1]. These infections complicate sickness; because they cause anxiety, increase patient discomfort and can lead to death.

Health Care facilities whether hospitals, nursing homes or outpatient facilities can be dangerous places for the acquisitions of infections. The most common types of nosocomial infections are wound infections, respiratory infections, genito-urinary infections as well as gastrointestinal infections [4]. These infections are often caused by breaches of infection control practices and procedures, unclear and non-sterile environmental surfaces and/or ill employees [4].

At KNH, the department of orthopedics is among the largest consumers of finances due to the high cost of materials used and prolonged hospitalization of patients. Most of the wounds become septic during hospitalization. It is important therefore to find out the underlying contributing factors.

## 1.2. Statement of the research problem

Wound infection in hospitals is common all over the world. The duration taken to control these infections as well as their severity is crucial for the well-being of patients, staff and hospital establishment. These infections lead to increased patients morbidity and mortality and their economical well-being diminishes. The hospital staff is usually at risk of contracting the infections. Wound infections are common at KNH. In addition, many resources both financial and human are channeled towards controlling these infections. Prevalence of drug resistance is also common among aerobic organisms. It is important therefore to find the root cause of these problems.

## 1.3. Goal of the study

To improve wound management at KNH.

## 1.4. Objectives of the study

### 1.4.1. General Objective

To determine the etiology and risk factors of bacterial wound infections at KNH.

### 1.4.2. Specific Objectives

(1) To determine the prevalence of aerobic bacteria in infected wounds at KNH.

(2) To find out whether antibiotics are rationally used in treatment of wound infections at KNH.

(3) To investigate whether recommended clinical practices that prevent wound infections are applied at KNH.

**Staff in department of Orthopedics**

They will use the report to reflect on their strengths and weaknesses on how they manage wounds. In return they will become more effective and improve quality of their services.

**Top Management of KNH**

They will use the report to assess the quality of care at KNH as regards wound infections. As a result, remedial measures will be put in place to correct any mistakes arising from omission or commission.

**Other Clinical Departments at KNH**

The clinicians will benefit greatly and preferably put remedial measures necessary to treat and prevent wound infections. They will use the report to take corrective measures and implement proper rules and regulations that govern wound management. This will lead to reduced mortality, morbidity and minimize wastage of antibiotics.

**Departments in the College of Health Sciences, UON**

The report will give an insight into the actual practices regarding wound care at KNH. In turn, they will research ways and means to improve management of patients and teach students how to do things correctly.

**Other Hospital and Clinics**

Both public and private hospitals will use the report to improve quality of care for their patients.

**The Government of Kenya**

The report will enlighten policy makers in the MOH to make more resources available to the institution and performance appraisal of staff instituted.

**The Students**

Both Postgraduate and Undergraduates will use it as reference material for further research.

**Clinicians and laboratory staff**

They will use the report to manage patients in a better way, come up with a protocol that is effective in the use of antibiotics, and thereby improve quality of care.

## 1.7 Limitations of the study

Antiseptics and hospital environment may have been contaminated with microorganisms and could have given rise to erroneous impression. Bias, arising from missing information in the records and recall problems among patients may have contributed to incomplete information. Lack of adequate facilities prevented the identification of all the bacteria genera or species isolated. The dressing materials, instruments and gloves used were presumed to be sterile according to information from the sterile production unit.

# .CHAPTER TWO: LITERATURE REVIEW

## 2.1 Etiology of bacterial wound infections

Potential wound pathogens include bacteria, fungi, viruses and parasites [5]. Bacteria comprises of both Gram-positive and Gram-negative. *Staphylococcus aureus, Streptococcus pyogenes, Streptococcus feacalis, Clostridium tetani, Clostridium perfringens,* and *Actinomycetes* are examples of Gram-positive group. Gram-negative organisms include *Pseudomonas aeruginosa, Proteus* spp., *Escherichia coli, Bacteroides* spp., *Klebsiella* spp., and *Pasturella* spp. *Mycobacterium tuberculosis* may be found ocasionally. Among the fungi *Histoplasma dubosii, Blastomyces dermatidis, Candida albicans* and *Cryptococcus neoformans* are occasionally found.

The development of wound infection depends on the integrity and protective function of the skin [6]. It has been shown that wound infection is universal and the type of bacteria varies with geographical location, resident flora of the skin, clothing at the site of wound and time between wound dressing [7]. In recent years, there has been a growing prevalence of Gram-negative organisms, which have almost replaced *Staphylococcus aureus* in nosocomial infections [7]. Of the Gram-negative bacilli, *Pseudomonas aeruginosa* has been of particular interest and the incidence in wound infection is increasing [8]. It has also been observed that 28% of healthy people in hospital environments are carriers for *Pseudomonas aeruginosa* [9]. Studies have been done in various parts of the world concerning bacterial wound pathogens and some of them are analyzed below.

*Giacometti et al., [10] in a study done at Salvatore hospital in Italy found that Staphylococcus aureus* was the most prevalent organism isolated from wounds followed

by *Pseudomonas aeruginosa*, *Escherichia coli*, *Staphylococcus epidermidis* and *Enterococcus faecalis*. Most pure cultures yielded *Staphylococcus aureus*, *Pseudomonas aeruginosa* and *Enterobacteriaceae*. Polymicrobial infections involved a similar spectrum of pathogens and frequently involved Gram-positive and Gram-negative organisms, especially *Staphylococcus aureus* together with *Pseudomonas aeruginosa*.

A retrospective study carried out at Deemed University in India showed that a single etiologic agent was identified in most (37%) wound specimens; multiple agents were found in 10% while the rest of the samples (53%) were culture-negative [11]. The most common pathogen isolated was *Staphylococcus aureus* followed by *Pseudomonas aeruginosa*, *Escherichia coli*, *Enterococcus* spp., *Klebsiella pneumoniae* and *Acinetobacter* spp.

Shamim *et al.*, [12] in a study done at Rawalpindi in Pakistan found that *Staphylococcus aureus* was the most common organism isolated from wounds followed by *Escherichia coli*, *Klebsiella* spp., *Pseudomonas aeruginosa*, *Proteus* spp., and *Acinetobacter* spp.

*Staphylococcus aureus* was the predominant microorganism isolated from wounds according to a study carried out in two Health Care institutions at Ile-ife in Nigeria, followed by *Escherichia coli*, *Pseudomonas aeruginosa* and *Staphylococcus epidermidis* [13]. The diversity of the microorganisms and the high incidence of polymicrobial flora in this study gave credence to the value of identifying one or more bacterial pathogens from wound cultures in order to enhance proper management. *Staphylococcus aureus* was found to be the most prevalent wound isolate at King

Khalid hospital in Saudi Arabia followed by *Escherichia coli*, *Pseudomonas aeruginosa*, and *Klebsiella* spp., [14].

Rastergar *et al.*, [15] in a study done at Tehran in Iran found that *Pseudomonas aeruginosa* accounted for 73.2% of total isolates from wounds followed by *Staphylococcus aureus*. Other organisms such as *Acinetobacter* spp., and *Enterobacter* spp., were rare.

A study carried out at an Indian medical college hospital showed that *Pseudomonas* spp., were the most prevalent organisms in infected wounds followed by *Acinetobacter* spp., *Escherichia coli*, *Klebsiella pneumoniae*, *Klebsiella oxytoca*, *Proteus vulgaris*, *Proteus mirabilis*, *Enterobacter* spp., and *Citrobacter* spp., [16].

Oguntibeju *et al.*, [7] at Lagos teaching hospital in Nigeria observed that *Pseudomonas aeruginosa* was the most prevalent organism isolated from wounds followed by *Staphylococcus aureus*, *Klebsiella* spp., *Escherichia coli*, Atypical *coliforms*, *Proteus* spp., *Streptococcus pyogenes* and *Enterococcus faecalis*. *Pseudomonas aeruginosa* infection was higher in females than males with a ratio 3:2 and was found more among young and elderly debilitated patients.

Thanni *et al.*, [17] in a study carried out in a Nigerian tertiary hospital observed that the most common isolates from wounds were *Pseudomonas* spp., and *Staphylococcus aureus* followed by *Klebsiella* spp., *Proteus* spp. and *Escherichia coli*. *Streptococci* and *Enterococci* were rare. *Pseudomonas* spp., accounted for 33% of isolates in the adult wards, while *Staphylococcus aureus* was 21% and *Escherichia coli* 8%. A similar study done at Yirga Alem hospital in Ethiopia showed that Gram-negative organisms were

more prevalent than Gram-positive organisms in wounds and the isolates were *Staphylococcus aureus, Escherichia coli, Proteus* spp., and *Pseudomonas* spp., [18] .

Richard *et al.*, [19] working in USA isolated organisms from infected wounds that included *Pseudomonas* spp., *Staphylococcus aureus, Enterococcus, Proteus* spp., *Staphylococcus epidermidis*, Fungi, *Klebsiella* spp., and *Corynaebacterium* in that order of prevalence. *Pseudomonas aeruginosa* causes a variety of soft tissue infections and was found to be the most common pathogen in osteomyelitis in children, and had equal prevalence with *Staphylococcus aureus* in leg ulcers [20, 21].

Kehinde *et al.*, [22] in a study done at Ibadan in Nigeria found that wound infections were significantly more frequent in children and adolescents than in adults. *Klebsiella* spp., were the pathogens most commonly isolated, constituting 34.4% and were closely followed by *Pseudomonas aeruginosa* and *Staphylococcus aureus*. The rate of isolation of Gram-negative organisms was more than twice that of Gram-positive organisms.

Alireza *et al.*, [23] in study done at Taleghani burn hospital in Iran found that the most prevalent microorganism causing wound infections was *Pseudomonas aeruginosa* followed by *Staphylococcus aureus, Acinetobacter baumanni, Escherichia coli, Proteus mirabilis*, coagulase negative *Staphylococcus, Enterobacter aerogenes, Klebsiella pneumoniae, Enterococcus* spp., *Citrobacter freundii, Serratia marcescens, Alcaligenes* spp., and *Streptococcus pyogenes*.

Tiwari *et al.*, [24] in a study done in India found that the prevalence of wound isolates in descending order was as follows; *Staphylococcus aureus, Pseudomonas aeruginosa, Escherichia coli, Enterococcus* spp., *Klebsiella pneumoniae, Acinetobacter* spp.,

*Streptococcus* spp., *Proteus mirabilis, Enterobacter* spp., *Candida albicans, Proteus vulgaris, Corynebacterium* spp., *Citrobacter diversus, Klebsiella oxytoca, Morganella morganii, Providencia* spp., and *Citrobacter freundii.*

From the above quoted studies, it is apparent that the most common bacterial wound pathogens are *Pseudomonas aeruginosa, Escherichia coli, Klebsiella* spp., *Proteus* spp., *Staphylococcus aureus, Enterococcus, Streptococcus pyogenes* and *Acinetobacter baumanni.* It is prudent therefore to describe some of these organisms further.

*Pseudomonas aeruginosa* is a Gram-negative rod measuring 0.5 to 0.8μm by 1.5 to 3.0μm and almost all strains are motile by means of a single polar flagellum [25]. The bacterium is ubiquitous in soil and water and on surfaces in contact with water or soil. Its metabolism is oxidative and never fermentative, but it will grow in the absence of oxygen if nitrate is available as a respiratory electron acceptor. It has several virulence factors such as fimbriae, lipolysacchride capsule, enzymes and toxins. It can adapt to minimal nutritional requirements and has widespread occurrence in a variety of habitats.

Diseases caused by *Pseudomonas aeruginosa* include endocarditis, respiratory infections, bacteraemia and septicemia, central nervous system infections, ear infections, eye infections, bone and joint infections, urinary tract infections, gastrointestinal infections, skin and soft tissue infections including wound infections, pyoderma and dermatitis [25]. Within the hospital, *Pseudomonas aeruginosa* may be found in numerous reservoirs such as disinfectants, respiratory equipment, food, sink, taps and mops. Furthermore, it is constantly re-introduced into the hospital environment on fruits, vegetables as well as by visitors and patients transferred from other facilities. Spread occurs from patient to patient on the hands of hospital personnel, by direct

contact with contaminated reservoirs and by the ingestion of contaminated foods and water. Observing proper isolation procedures, aseptic techniques and careful cleaning of instruments can control the spread.

*Escherichia coli* is the most frequently encountered member of the *Enterobacteriacea* in the normal colonic flora and a leading cause of opportunistic infections [26]. Virulent factors include alpha hemolysins, siderophores, aerobactin, capsular polysaccharide, toxins and pili. The organism causes several diseases such as urinary tract infection, intestinal infections, meningitis and wound infections.

*Klebsiella* spp., are Gram-negative non-motile capsulated rods [5]. They are found in the intestinal tract of humans and animals, plants, soil and water. *Kebsiella pneumoniae* can be found as a commensal in the mouth and upper respiratory tract and in most environments in hospital and elsewhere. Virulence factors are pili and capsule. Pathogenicity includes chest infections, urinary tract infections, wound infections and peritonitis, as well as septicemia and meningitis.

*Staphylococcus aureus* is widely distributed in the environment and forms part of normal flora of the skin, upper respiratory tract and intestinal tract [5]. It is Gram-positive and the different strains possess several virulence factors such as; coagulase, leucocidin, Dnase, Hyaluronidase, lipase, staphylokinase, exfoliatin, enterotoxin B and Beta-lactamase. It is responsible for a wide range of infections such as skin infections, wound infections, conjunctivitis, septicemia, endocarditis, osteomyelitis, pneumonia, empyema, mastitis, food poisoning, scalded skin syndrome and toxic shock syndrome.

*Proteus* spp., are part of normal human gastrointestinal flora and exists in water and soil as saprophytic organisms [26]. The principal virulence determinants are lipopolysacchride, pili, urease activity and capsule. Diseases caused by *Proteus* spp., are urinary tract infections, sepsis, and wound infections.

*Enterococci* are Gram-positive cocci, which are primarily found in the intestine [26]. They are capable of growing in high concentrations of bile and sodium chloride. Some *Enterococci* are beta hemolytic, but most produce non-hemolytic or alpha hemolytic colonies that are larger than those of other *Streptococci. Enterococci* cause opportunistic urinary tract infections and occasionally wound and soft tissue infections. There is often an associated bacteriaemia, which can result in the development of endocarditis on previously damaged cardiac valves.

**2.2 Rational drug use**

Antibiotics are among the most commonly prescribed drugs today [27]. At KNH antibiotics commonly used in orthopedics department are cefuroxime, cloxacillin, gentamycin, benzyl penicillin, ampicillin, flucloxacillin and augmentin (amoxicillin/clavulanic acid). Rational use of antibiotics is extremely important since misuse can adversely affect the patient, cause emergence of resistance and increase the cost of health care. Prescribing an antibiotic comprises several phases as follows:-

i. Perception of need- is an antibiotic necessary;
ii. Choice of antibiotic-which is the most appropriate antibiotic;
iii. Choice of regimen- what dose, route, frequency and duration are needed;
iv. Monitoring if the treatment is effective.

The successful outcome of therapy would depend very much on the choice of the antibacterial agent. In the process of selecting an antibiotic, four main factors

considered are the etiological agent, site of infection, the patient and the type of antibiotic.

### 2.2.1 Duration of Treatment

Except for a few conditions, the optimum duration of antibiotic treatment is unknown [27]. Many antibiotics are prescribed for a duration of 5-7 days. Nevertheless, it is reasonable to discontinue therapy even after shorter period if the patient's symptoms have resolved. There are however certain infections where prolonged treatment is necessary.

### 2.2.2 Causes of Non Response to Antibiotics

A patient may fail to respond to antibiotics for a number of reasons, which include:

i. The etiological agent being resistant to the antibiotic;
ii. The diagnosis is incorrect;
iii. The choice of antibiotic is correct but the dose and / or route of administration is wrong;
iv. The antibiotic cannot reach the site of infection;
v. There is collection of pus that should be drained surgically or a foreign body/devitalized tissue that should be removed;
vi. There is secondary infection;
vii. Non-compliance of the host
viii. Use of substandard drugs.

The rates of non response to antibiotics and methodological differences of carrying out studies in published reports and local surveillance data should be available to assist clinicians in the development of antibiotic guidelines [28]. Antibiotic use is one of the

most important factors for the development and spread of resistance in hospitals, as well as in the community [29]. The WHO has established antibiotic use as a priority in its campaign for the rational use of medications. Antibiotics account for a significant proportion of total hospital drug expenditure [30]. Furthermore, it is estimated that 50% of all physicians' orders for antibiotics are for wrong drug, or an inappropriate dosage or duration [31]. In addition, inadequate antibiotic use increases costs by increasing the length of hospitalization [30, 31].

Control of antibiotic use is recommended by WHO in order to prevent emergence of resistance, minimize wastage and reduce the morbidity arising from side effects [31]. One tool to address this problem is the elaboration of therapeutic and prophylactic protocols developed by examining each hospital's most prevalent infection, together with the local rate of bacterial resistance [32]. An infection at the surgical site is one of the most important types of nosocomial infections and many studies have shown that the use of antibiotic prophylaxis in some surgical procedures can reduce these infections

In spite of extensive knowledge about effectiveness of antibiotic prophylaxis, their administration is often inappropriate [29]. The proper use of antibiotic prophylaxis in surgical procedures require the consideration of several factors. Effectiveness depends on the current selection and application of the following items: appropriate antibiotics choice, timing of initial administration, dose administered and postoperative drug use.

Several studies have demonstrated that a remarkable amount of antibiotic use in hospitalized patients is inappropriate [33]. Filius *et al.*, [28] in his study found that 54% of hospitalized patients received antibiotics. This figure is similar to the 61.4% prevalence seen in a Greek hospital, as described by Gikas *et al.*, [34].

When Filius *et al.*, [28] at Erasmus medical center in Netherlands evaluated the appropriateness of therapeutic use, it was found that 27% of the patients were treated incorrectly. One of the most important problems detected in this study was the large number of patients who received antibiotic combinations (43%) particularly with the use of aminoglycosides, which are known for their severe side effects.

Gayel *et al.*, [35] in a study carried out in eighteen hospitals in Turkey found that, 44% of antimicrobial prescriptions were made for surgical prophylaxis and 52.4% of them were inappropriate. This group found that inappropriate prescription ratios were increased because of the long duration of administration and wrong selection of antimicrobials. In addition, combination drugs were found to be 33% of total antimicrobials prescribed.

### 2.2.3 Antibacterial susceptibility

*Pseudomonas aeruginosa* is generally sensitive to ticarcillin, piperacillin, amikacin, gentamycin and tobramycin [5]. A study carried out in Brazil by Santosh *et al.*, [36] showed that *Pseudomonas aeruginosa* was sensitive to amikacin, cefotaxime, ceftazidime, ceftizoxime, ciprofloxacin, gentamycin, and piperacillin but resistant to ampicillin, cefuroxime, clindamycin, cotrimoxazole, erythromycin, norfloxacin, penicillin and tetracycline.

Mehta *et al.*, [37] in a study done in India, observed that *Pseudomonas* spp., were moderately resistant to piperacillin whereas resistance was more marked against amikacin, gentamicin, ciprofloxacin, carbenicillin, tobramycin and ceftazidime. On the other hand, the organisms were found to be more sensitive to newer antimicrobials such

as, *ceftazidime/clavulanic acid, cefoperazone/sulbactum and imipenem* but 50% were resistant to cefepime.

Serap *et al.*, [38] working at Instanbul in Turkey realized that thirty-six percent of *Pseudomonas* spp., isolated were resistant to more than one group of antibiotics. The sensitivity was above 60% for ciprofloxacin, amikacin, ceftazidime, meropenem, imipenem and piperacillin/tozabactam. The sensitivity ranged between 40-59% for cefepime, cefoperazone/sulbactam and tobramycin. The majority of carbapenem resistant isolates were susceptible to ciprofloxacin and amikacin. A study done in Iran showed that similar organisms were absolutely sensitive to gentamycin, amikacin, ciprofloxacin, cephalothin, carbenicillin, ceftazidime and tobramycin [23].

*Staphylococcus aureus* has several strains and some of which produce pencillinases and are resistant to benzyl penicillin [5]. Other strains resistant to methicilin and flucloxacillin have also been encountered and some of these are sensitive to vancomycin. The organisms may occasionally be susceptible to rifampicin, sodium fusidate, tetracycline, aminoglycoside and minocin. Quinoxpristine and dalfopristine are active against MRSA [39]. Cefuroxime and cefamadole have greater activity against the organism compared to other cephalosporins [39]. Paul *et al.*, [40] observed that 50% of *Staphylococcus aureus* isolates were resistant to oxacillin. *Staphylococcus aureus* was found by Santosh *et al.*, [36] to be highly sensitive to amikacin, cefuroxime and clindamycin but resistant to co-trimoxazole, erythromycin, penicillin and tetracycline. In another study, the organism was sensitive to azithromycin and perfloxacin [22]. A sensitivity range of 70-90% to gentamycin, cephalothin, ciprofloxacin, cephalexin, clindamycin, ceftazidine and cotrimoxazole was observed in a study done in Iran but the organism portrayed moderate sensitivity to oxacillin (58%) [23].

*Proteus* spp., are usually resistant to benzyl penicillin, penicillinases resistant penicillins, erythromycin, clindamycin, vancomycin, ampicillin, cephalothin and tetracycline [8]. On the other hand, they are susceptible to cefotetan, ceftazidime, imepenem, gentamycin, amikacin and ciprofloxacin [8]. Twenty-five percent of the organisms are resistant to piperacillin, chloramphenicol and co-trimoxazole [26]. *Proteus* spp., were found to be sensitive to cefotaxime but resistant to several drugs including, amikacin, ampicillin, cotrimoxazole, ciprofloxacin, gentamycin, tetracycline, ceftazidime, clindamycin, erythromycin, penicillin and piperacillin by Santosh *et al.*, [36]. In a study done in Nigeria, Kehinde *et al.*, [22] observed that *Proteus* spp., were highly sensitive to gentamycin, ceftriaxone, ceftazidime and perfloxacin.

*Escherichia coli* is often resistant to several drugs such as benzyl pencillin, pencillinase –resistant penicillins, erythromycin, clindamycin and vancomycin [5]. Approximately twenty-five percent of the strains are usually resistant to ampicillin, cephalothin, aztreonam, gentamycin, amikacin, tetracycline, chloramphenicol, ciprofloxacin and cotrimoxazole but sensitivity to cefotetan, ceftazidime, and impenem is usually high [26]. In a past study amikacin, cefotaxime, ceftizoxime cefuroxime were found to be active against *Escherichia coli* but ampicillin, cefuroxime, cotrimoxazole, gentamycin, norfloxacin, and tetracycline were not [36]. A study carried out in Nigeria showed that ceftazidime and ceftriaxone had absolute activity against *Escherichia coli* [22]. Giacometti *et al.*, [10] observed that the organism had over 50% sensitivity to ampicillin, cefazolin, amoxicillin-clavulanic acid, ceftriaxone, imipenem, ciprofloxacin and netilmicin.

*Klebsiella* spp., portrays sensitivity to cefotetan ceftazidime, imepenem, astronain and ciprofloxacin [8]. Twenty-five percent of the organisms are generally resistant to cotrimoxazole, chloramphenicol, tetracycline, amikacin, gentamycin, cephalothin and piperacillin [26]. Absolute resistance against benzyl penicillin, penicillinase resistant penicillins, erythromycin, clindamycin, vancomycin and ampicillin is common. *Klebsiella pneumoniae* was found to be susceptible to amikacin, ampicillin, cefotaxime, cefuroxime, cotrimoxazole, ciprofloxacin, gentamycin, norfloxacin and tetracycline but resistant against ceftazidime, clindamycin, erythromycin, penicillin and piperacillin [36]. In a past study, ceftazidime and perfloxacin showed above average activity against *Klebsiella* spp., while gentamycin and ceftriaxone were ineffective [22].

*Enterococci* are generally sensitive to vancomycin, piperacillin, imipenem and ciprofloxacin but resistant to ampicillin, erythromycin and chloramphenicol [26]. The organism is often resistant to penicillinase resistant penicillins, cephalothin, cefotetan, aztreonam and gentamycin.

*Acinetobacter baumanii* showed absolute sensitivity to gentamycin, cephalothin, amikacin, carbenicillin ,ceftazidime , cephalexin and ciprofloxacin in a past study [23].

**2.3 Clinical Practices**

Hand washing remains the most effective method of preventing the spread of microorganisms and should be routinely performed [41]. Several well-designed studies have shown that patient contact result in contamination of health care workers' hands by pathogens. For example, hospital staff dressing wounds containing methicillin resistant *Staphylococcus aureus* has an 80% chance of carrying the organism on their hands for up to three hours [42]. Another study by Louise *et al.*, [43] showed that patient nurse

interactions in an intensive care unit resulted in transmission of *Klebsiella* spp., to the nurse's hands, even after minimal contact such as touching a patient's shoulder.The Organisms remained on hands for up to 150 minutes and hand washing removed them. Mathematical modelling suggests that even small increments in hand hygiene may be highly effective in controlling endemic methicillin resistant *Staphylococcus aureus*. The risk of transfer on carers' hands is proportional to the power of the number of times a patient is touched [44]. Given that chance plays a strong part in events on a small ward, it is apparent that even small increments of frequency of effective hand hygiene should reduce the risk of infection.

Gloves should be worn when touching blood, body fluids, secretions, excretions and contaminated items. They should also be used before touching mucus membrane and non-intact skin [41].Gloves should be changed after tasks and procedures for every patient [45]. They should be removed promptly after use before touching non-contaminated environments, surfaces and before going to another patient. Hands should be washed subsequently. Masks, eye protection and face shields should be worn to protect the mucus membrane of the eye, nose and mouth during procedures and patients care activities that are likely to expose the health care worker through splashes or sprays of blood, body fluids, secretions or excretions. Gowns should be worn to protect skin and avoid contamination of clothing during splashes of blood or body fluids. Used patient care equipment should be handled in a cautious manner to prevent the contamination of clothing and transfer of microbes to other patients or environments. Environmental surfaces, beds, beds rails, bedside equipment should be clean and disinfected regularly [4].

## 2.4 Confounders

Other factors that may affect wound infections include:-

**Contaminated antiseptics and disinfectants**

Some organisms can multiply in disinfectants and antiseptics. These solutions may act as reservoirs and a source of infections since they are used regularly for cleaning the floor, bathrooms and sinks. *Serratia marcescens* has been implicated in outbreak of infections due to contaminated chlorhexidine while *Pseudomonas aeruginosa* is known to be a common contaminant [47, 22]. Anderson [46] discussed two reports of povidone-iodine stock solution (10%) contaminated with *Pseudomonas* spp. The contamination apparently occurred during production of the povidone-iodine solution. The bacteria remained viable for several weeks and eventually caused infections.

**Visitors**

Visitors in hospital often visit patients. They bring various items such as foodstuffs, clothing's and personal effects. These can be sources of infections

**Hospital linen**

Bed sheets, mattresses and bedcovers may provide suitable environment for multiplication of microorganism. Patients can easily obtain infection from these sources.

# CHAPTER THREE: MATERIALS AND METHODS

## 3.1 Area of Study

The area of study was KNH, and Wards 6A, 6B, 6C and 6D were included because different types of wounds were well represented. Only aerobes were considered since pure anaerobes do not cause many problems due to their high susceptibility to drugs. Specimens were analyzed in the microbiology laboratories located at the departments of Medical Microbiology, UON and KNH.

## 3.2 Research Design

The study made use of a cross sectional research design where the researcher went out to the population of interest, collecting specimens of pus, reading patient's records, interviewing and observing the respondents to note some of the issues related to the problem under study. This was appropriate because the respondents were examined in the hospital environment, which was conducive.

## 3.3 Target Population

Two sets of population used for the study were patients and staff of wards 6A, 6B, 6C and 6D.

### Patients

All patients in orthopedic wards with septic wounds were eligible.

### Staff

All nurses who were involved in wound management.

### 3.3.1 Inclusion Criteria

**Patients**

Potential participants met the following criteria: -

- Of legal age or have a guardian willing to volunteer information.
- Able and willing to sign consent form directly or through proxy.
- Possess an infected wound.

**Staff**

All nurses working in the department of orthopedics.

### 3.3.2 Exclusion Criteria

**Patients**

All hospitalized patients with infected wounds but refused to give consent.

Patients who were unconscious or confused or those already using antibiotics.

**Staff**

Staff members who were not nurses.

## 3.4 Ethical Considerations

Prior to the study, the protocol and informed consent forms were availed and clearance sought from Department of medical microbiology, UON and Ethical and Research committee of Kenyatta National Hospital. Informed consent was obtained from the patients and confidentiality was maintained on all information and data collected.

## 3.5 Sampling

### 3.5.1 Sampling technique

Kenyatta National Hospital orthopedics department was the focus of the study. Hospitalized patients were sampled using convenience sampling method while simple random sampling was applied to the staff.

### 3.5.2 Sample Size

Appropriate sample sizes for both the patients and staff were as follows.

**Sample Size for Patients**

According to the information obtained from staff, it was estimated that 5-11% of orthopedic patients suffer from infected wounds. Thus, the sample size at 95% confidence level was obtained using the formula;

$$N=\frac{Z^2pq}{d^2}$$

[48]

Where
Z = Standard normal deviation which corresponds to 95% CI
P = Proportion of the target population with infected wounds
q = 1-p
d = degree of freedom = 0.05

Thus:
$Z = 1.96, \quad p = \frac{.05+.11}{2} = 0.08, q = 1- 0.08 = 0.92$

$$n = \frac{1.96^2 \times 0.08 \times 0.92}{0.05^2} = 113.2 = 113$$

**Sample Size for Staff**

Twenty percent of the nurses were considered for the study as follows:-

**Table 3.1 Staff sample**

| Category | Total Number | Proportion | Sample Size | Approx |
|---|---|---|---|---|
| Nurses | 72 | 0.2 | 14.4 | 14 |

## 3.6 Data Collection Method

### 3.6.1 Clinical practices and rational use of antibiotics

**Staff**

The researcher and/or the research assistants observed the nurses from the beginning of the dressing exercise and then followed them up to the end of the day's session. Each nurse was observed at a time and only once during the study period. All the variables of interest were noted and entered in a form (Appendix IVB).

**Patient**

The researcher and/or research assistants approached each patient who met the inclusion criteria and after introduction and getting consent (Appendix III), went ahead to ask the relevant questions. More information was obtained from the records available in the wards especially issues on prescribed drugs. Each study subject was interviewed only once during the study period. All the issues of interest were noted and entered in a questionnaire (Appendix IV A).

### 3.6.2 Laboratory Procedures

**Specimen Collection**

The researcher and/or research assistants collected specimens of pus using sterile cotton swabs from patients at the end of interview. Special care was taken to avoid contaminating the specimen with commensal organisms from the skin. The specimens were collected before an antiseptic dressing was applied, then labeled and delivered to the laboratory within one hour and processing commenced immediately.

**Control Strains**

*Staphylococcus aureus*

Oxford strain NCTC 6571 or ATCC 25923, for controlling all drugs except polymyxins against pathogens from specimens of pus.

*Escherichia coli*

NCTC 10418 or ATCC 25922 for controlling all drugs against *Enterobacteriaceae* pathogens from pus.

*Pseudomonas aeruginosa*

NCTC 10662 or ATCC27853 for controlling drugs against *Pseudomonas* spp.

**Inoculation of culture media**

The media was prepared and poured in petri dishes up to a depth of 4mm and allowed to cool. Aseptic techniques were used during inoculation where a main inoculum was applied to a small area of the media in a plate and spread using a sterile wire loop to provide for single colonies. Blood Agar, Chocolate Blood Agar and MacConkey media were used. All inoculated plates were labeled using a grease pencil and incubated at $37^0$C for 12 hours. Proper humidity was maintained and carbon dioxide jar was used incase of CBA and BA plates.

**Identification of the organisms**

Identification of the organisms was done using the recommended standard procedures (Appendix I). BACTEC system was used to identify some of the organisms that could not be identified using the common biochemical methods.

**Sensitivity Testing Technique**

Kirby and Bauer Disc Diffusion sensitivity test was used for the study [5]. The test organisms were inoculated on Muller Hinton Agar plates except for *Streptococcus pyogenes* where Blood Agar was used. Appropriate sensitivity discs were placed on the media and the drug activity was shown by zones of inhibition of growth around the discs. The diameter of the zone was compared to the standard measurements [49]. Categorization was done as either resistant, intermediate or sensitive (Appendix II).

Drugs tested were augmentin, oxacillin, amoxicillin, cefuroxime, clindamycin, gentamycin, flucloxacillin, ciprofloxacin, tetracycline,erythromycin, amikacin,chloramphenicol, ceftazidime, ceftriaxone,minocycline, meropenem, pipril/tozabactam, ticarcillin/clavulonic acid, vancomycin, nalidixic acid, streptomycin, nitrofurantoin and piperacillin. These drugs were used in different combinations for various organisms. Standardized disks designated as KNH1, KNH2, KNH3, KNH4 and KNH5 that contained drugs commonly used at KNH were used. The control organisms were subjected to similar procedure as the test organisms in order to verify the viability of the drugs. All the measurements of the zones of inhibition were accurate at 95% confidence level.

## 3.7 Data analysis

Data was analyzed using SPSS and tabulated in form of percentages and figures as shown in Chapter four.

# CHAPTER FOUR: RESULTS

## 4.1 Assessment done on the staff members

### 4.1.1 Gender

All the sixteen staff members assessed were nurses from the four wards; 6A, 6B, 6C and 6D (Table 4.1). Out of those, only one representing 6.3% was a male. Fifteen nurses representing 93.7% were females, an indication that the profession is tilted in their favour. No doctor was found to be involved in routine dressing of wounds and therefore they were not assessed.

**Table 4.1 Gender distribution of staff**

| Gender | Frequency | Percent |
|---|---|---|
| Male | 1 | 6.3 |
| Female | 15 | 93.7 |
| Total | 16 | 100 |

### 4.1.2 Hand washing during dressing of patients' wounds

Fifteen nurses were observed washing hands during the activity (Table 4.2). This represented 93.8%. One member representing 6.3% did not wash the hands. Hand washing is very crucial in the course of dressing wounds. Most of the nurses observed seemed to be conscious of this fact. Hence most of them passed in this regard, an indication that they practised according to the recommended standards.

**Table 4.2 Washing of hands**

| Observation | Frequency | Percent |
|---|---|---|
| Yes | 15 | 93.75 |
| No | 1 | 6.25 |
| Total | 16 | 100 |

### 4.1.3 Timing of hand washing

Thirteen staff members representing 86.7% were found to wash hands after finishing the dressing exercise (Table 4.3). Two nurses representing 13.3% washed their hands after dressing some patients. Observations were made after following up each nurse from the beginning of dressing exercise until completion or after dressing more than one patient.

**Table 4.3 Timing of washing hands**

| Timing of washing hands | Frequency | Percent |
|---|---|---|
| After dressing wound in some patients | 2 | 13.3 |
| After dressing all the wounds | 13 | 86.7 |
| Total | 15 | 100 |

### 4.1.4 Use of gloves

Gloves should always be worn during dressing of wounds. All the sixteen members assessed representing 100% wore gloves during the exercise. Gloves prevented direct contact between the clinician and the patient. This was very crucial since organisms are easily transmitted through direct contact.

### 4.1.5 Timing of changing gloves

Fourteen nurses representing 87.5% were seen to change gloves only when they became soiled with materials from the patients' wounds (Table 4.4). Two of them representing 12.5% removed the gloves only after the entire activity was over. Nobody was observed changing gloves after dressing the wound in every patient during the entire session of the day.

**Table 4.4 Timing of changing gloves**

| Timing of changing gloves | Frequency | Percent |
|---|---|---|
| At the end of the dressing session | 2 | 12.5 |
| When they become soiled | 14 | 87.5 |
| Total | 16 | 100 |

### 4.1.6 Methods used to clean wounds

Fifteen staff members used gauzes or cotton wool soaked in antiseptic solution to clean wounds (Table 4.5). This represented 93.8% of those assessed. Only one person representing 6.3% used irrigation method.

**Table 4.5 Methods of cleaning wounds**

| Methods of cleaning wounds | Frequency | Percent |
|---|---|---|
| Irrigation with antiseptic solution | 1 | 6.25 |
| Swabbing with gauze/cotton wool soaked in antiseptic solution | 15 | 93.75 |
| Total | 16 | 100 |

### 4.1.7 Use of facemask by the staff

Most nurses wore facemask during the exercise (Table 4.6). Twelve nurses representing 75% used facemasks and four representing 25% did not.

**Table 4.6 Use of facemask**

| Observation | Frequency | Percent |
|---|---|---|
| Yes | 12 | 75 |
| No | 4 | 25 |
| Total | 16 | 100 |

Wearing facemask during wound dressing was important. It protected the clinician from inhaling microbes from infected surfaces. In addition, exhalation of microorganisms by the clinician to the patient was minimised.

### 4.1.8 Opening of the wounds

Out of the sixteen cases assessed, the nurse opened 50% of the wounds alone while the nurse and patient opened the other 50% jointly (Table 4.7). Staff members requested the patients to start opening wounds while attending other patients. In most cases patients used bare hands and did not wash immediately. This was a very dangerous practice. The patients unknowingly soiled their hands as well as the beddings.

**Table 4.7 Opening of wounds**

| Observation | Frequency | Percent |
|---|---|---|
| Nurse(s) | 8 | 50.0 |
| Both patient and nurse(s) | 8 | 50.0 |
| Total | 16 | 100 |

### 4.1.9 Frequency of dressing wounds

Changing of dressing materials was very crucial in wound management. The spent materials could form source of infection and may worsen the prognosis of the condition. According to the study, 75% of the wounds were dressed on alternate days, 12.5% anytime and 12.5% on daily basis (Table 4.8). Patients were seen occasionally requesting the nurses to replace the dressings. Generally, the frequency of wound dressing was quite arbitrary and often based on how bad the wound was perceived. It was difficult to establish how frequently the dressing should be changed.

**Table 4.8 Frequency of dressing wounds**

| Observation | Frequency | Percent |
|---|---|---|
| Daily | 2 | 12.5 |
| On alternate days | 12 | 75.0 |
| Any time | 2 | 12.5 |
| Total | 16 | 100 |

### 4.1.10 Cleansing agent(s) used

Some of antiseptics were used at the same rate (Table 4.9). Preference for use of savlon, normal saline, rifamycin and a mixture of hydrogen peroxide and normal saline was 6.3% in each case. Hydrogen peroxide and betadine equalled at 12.5%. Preference of mixtures was higher, where a mixture of betadine and normal saline as well as hydrogen peroxide and normal saline were at par (25%). It was difficult to establish which criterion was used to choose an antiseptic. On further investigation,

the researcher found that there was no established protocol. Choosing solely depended on what was available and what the nurses preferred.

**Table 4.9 Antiseptic(s) used**

| Agents | Frequency | Percent |
|---|---|---|
| Savlon | 1 | 6.3 |
| Hydrogen peroxide | 2 | 12.5 |
| Betadine | 2 | 12.5 |
| Normal saline | 1 | 6.3 |
| Rifocin | 1 | 6.3 |
| Hydrogen peroxide + betadine | 4 | 25.0 |
| Betadine + normal saline | 4 | 25.0 |
| Hydrogen peroxide + normal saline | 1 | 6.3 |
| Total | 16 | 100 |

The concentrations of antiseptics were difficult to establish. Mixing was often done in a kidney dish. In other cases pouring was done on the wound in turns. Rationale could not be established and was absolutely the prerogative of the nurse.

### 4.1.11 Use of forceps to hold gauze during dressing

Forceps were used to hold gauze when cleaning the wounds and placing the final gauze soaked in antiseptic solution on the wound (Table 4.10). In 93.8% of cases, forceps were used but in 6.3% they were not.

**Table 4.10 use of forceps**

| Observation | Frequency | Percent |
|---|---|---|
| Yes | 15 | 93.75 |
| No | 1 | 6.25 |
| Total | 16 | 100 |

### 4.1.12 Sterility of the forceps

In 26.7% of cases, forceps used were sterile (Table 4.11). This percentage represented the initial stages of the activity. All the nurses were provided with sterile dressing kit composed of a kidney dish, gauzes, cotton wool, forceps and antiseptics. Subsequently, the same forceps were used on other patients. Use of unsterile forceps was observed in 73.3% of cases.

**Table 4.11 Sterility of forceps**

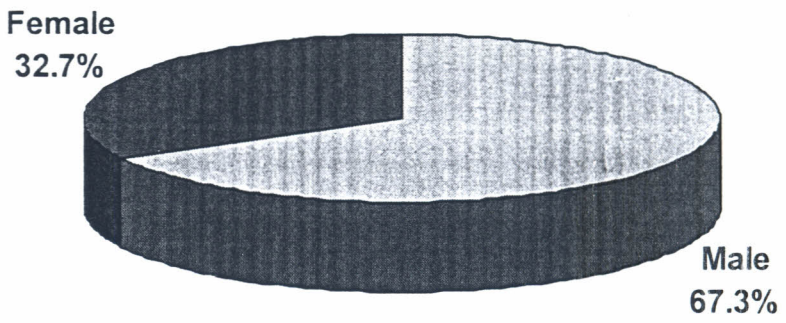
| Observation | Frequency | Percent |
|---|---|---|
| Yes | 4 | 26.7 |
| No | 11 | 73.3 |
| Total | 15 | 100 |

## 4.2 Patients Results

### 4.2.1 Gender distribution

67.3% of the study subjects were males, and 32.7% were females (Fig 1). This significant difference showed that males were probably more predisposed to traumatic episodes than females. Their nature of work coupled with frequent travelling makes them more amenable especially to accidents.

**Fig 1 Gender distribution of study patients**

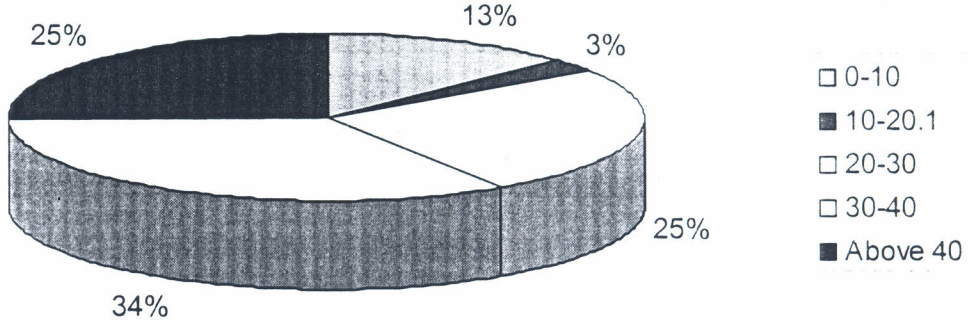



### 4.2.2 Age distribution of study patients

Most patients were in their productive age (Fig 2). Only a small percentage (13.0%) represented the children. Age distribution was as follows; above 40 years (25 %), 30-40 years (34 %), 20-30 years (25 %), and 10-20 years (3 %). Their hospitalization was

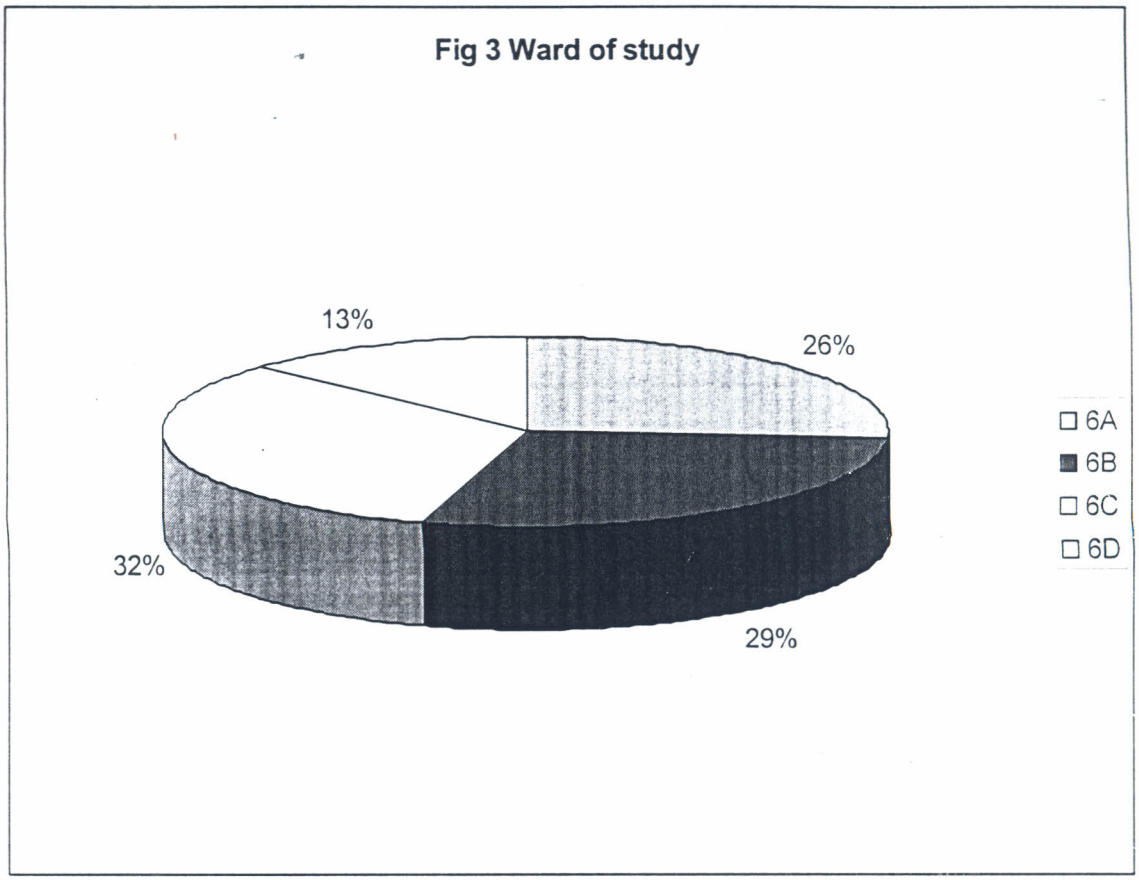
a grim reality of how traumatic episodes, especially accidents adversely affected productivity and social fabric.

**Fig 2 Age distribution in years**




### 4.2.3 Ward of study

All the four wards in orthopedic department were well represented (Fig 3). For the adult wards, 6A, 6C and 6D, the patients constituted 26%, 28.7% and 32.2% respectively. Children formed the minority at 13.0%.




### 4.2.5 Cause(s) of the wound

Road traffic accidents were the main causes of injuries contributing 71.3% of cases followed by infections at 20.0% (Table 4.12). Other contributors were assault (2.6%), falling and accidental cuts (4.3%) while accident together with cellulitis constituted 1.7%.

**Table 4.12 Wound etiology**

| Cause | Frequency | Percent |
|---|---|---|
| Road traffic accidents | 82 | 71.3 |
| Cellulitis /osteomyelitis | 23 | 20.0 |
| Assault | 3 | 2.6 |
| Falling and Accidental cuts | 5 | 4.3 |
| Road traffic Accidents + cellulitis | 2 | 1.7 |
| Total | 115 | 99.9 |

### 4.2.6 Duration of hospitalisation in weeks

40.9% of the patients had been in the hospital for over four weeks, 20.9% between 3-4 weeks, 14.8% for 2-3 weeks, 13.9% for 1-2 weeks and only a minority 9.6% were admitted probably with already infected wounds (Table 4.13). It is prudent to deduce that the source of the organisms was mainly hospital.

**Table 4.13 Duration of hospitalisation in weeks**

| Weeks | Frequency | Percent |
|---|---|---|
| 0-1 | 11 | 9.6 |
| 1-2 | 16 | 13.9 |
| 2-3 | 17 | 14.8 |
| 3-4 | 24 | 20.9 |
| >4 | 47 | 40.9 |
| Total | 115 | 100 |

### 4.2.7 Reason(s) for hospitalisation

Most of the subjects were inpatients because the damage inflicted home-based care was not tenable. The situation was worsened by infection admissions were due to delayed healing, 3.5% as a result of delayed procedures delayed healing and delayed procedures gave rise to 9.6%, while other factors like financial constraints contributed 1.7% (Table 4.14).

**Table 4.14 Reason(s) for hospitalisation**

| Reason | Frequency | Percent |
|---|---|---|
| Delayed procedures | 4 | 3.5 |
| Delayed healing | 98 | 85.2 |
| Delayed procedures and delayed healing | 11 | 9.6 |
| Other factors | 2 | 1.7 |
| Total | 115 | 100 |

## 4.3 Analysis of bacteria isolated

### 4.3.1. Prevalence of organisms isolated

Twelve different organisms were isolated (Table 4.15). The total number of isolates was one hundred and sixty seven. From 115 specimens collected, the prevalence was as follows: *Pseudomonas* spp. (42.6%), *Proteus* spp. (33.9%), *Staphylococcus aureus* (33.04%), *Escherichia coli* (13.2%), *Klebsiella* spp. (7.9%), *Streptococcus faecalis* (6.1%), *Enterobacter* spp. (2.6%), *Streptococcus pyogenes* (1.7%), *Citrobacter freundii* (0.9%), *Serratia* spp. (0.9%), and *Acinetobacter baumanii* (0.9 %).

**Table 4.15 Prevalence of bacteria**

| Organism | Frequency | Percent | % Prevalence |
|---|---|---|---|
| *Pseudomonas* spp. | 49 | 29.3 | 42.6 |
| *Proteus* spp. | 39 | 23.4 | 33.9 |
| *Staphylococcus aureus* | 38 | 22.8 | 33.0 |
| *Escherichia Coli* | 15 | 9 | 13.1 |
| *Klebsiella* spp. | 9 | 5.4 | 7.9 |
| *Streptococcus faecalis* | 7 | 4.2 | 6.1 |
| *Enterobacter* spp. | 3 | 1.8 | 2.6 |
| *Streptococcus pyogenes* | 2 | 1.2 | 1.7 |
| *Alcaligenes* spp. | 2 | 1.2 | 1.7 |
| *Citrobacter freundii* | 1 | 0.6 | 0.9 |
| *Serratia* spp. | 1 | 0.6 | 0.9 |
| *Acinetobacter baumanii* | 1 | 0.6 | 0.9 |
| Total | 167 | 100.1 | 144.64 |

### 4.3.2 Number of organisms isolated per specimen

47.8% of the specimens yielded only one organism and 13.9% had none (Table 4.16). Two, three and four organisms were found in 25.2%, 5.2% and 7.8% of the specimens respectively.

**Table 4.16 Number of organisms isolated per patient**

| Number | Frequency | Percent |
|---|---|---|
| None | 16 | 13.9 |
| One | 55 | 47.8 |
| Two | 29 | 25.2 |
| Three | 6 | 5.2 |
| Four | 9 | 7.8 |
| Total | 115 | 99.9 |

The organism(s) isolated per patient are shown in Table 4.17. 20.9% of the specimens yielded *Staphylococcus aureus* alone while 13.9% had *Pseudomonas* spp., only. *Proteus* spp., alone was isolated in 5.2% of the cases. Notable combinations were: *Pseudomonas* spp. + *Proteus* spp. (9.6%), *Proteus* spp. + *Staphylococcus aureus*

(3.5%), *Pseudomonas* spp. + *Escherichia coli* (3.5%), *Proteus* spp. + *Peudomonas* spp. + *Staphylococcus aureus* + *Streptococus pyogenes* (4.3%) and *Proteus* spp. + *Escherichia coli* (2.6%). Other combinations were rare. Multi-infected wounds are very difficult to manage since the organisms respond to drugs differently. A multi-drug therapy is usually preferred. This scenario may explain the reason why patients were not responding well to antibiotics since most of them were on one drug.

**Table 4.17 Organism(s) isolated per patient**

| | Frequency | % |
|---|---|---|
| No organism isolated | 16 | 13.9 |
| *Pseudomonas* spp. | 16 | 13.9 |
| *Proteus* spp. | 6 | 5.2 |
| *Klebsiella* spp. | 2 | 1.7 |
| *Staphylococcus aureus* | 24 | 20.9 |
| *Streptococcus pyogenes* | 1 | .9 |
| *Citrobacter freundii* | 1 | .9 |
| *Alcaligenes* spp. | 2 | 1.7 |
| *Serratia* spp. | 1 | .9 |
| *Enterobacter* spp. | 1 | .9 |
| *Acinetobacter baumanii* | 1 | .9 |
| *Pseudomonas* spp. + *Proteus* spp. | 11 | 9.6 |
| *Klebsiella* spp. + *Staphylococcus aureus* | 1 | .9 |
| *Pseudomonas* spp. + *Escherichia coli* | 1 | .9 |
| *Pseudomonas* spp. + *Proteus* spp. | 1 | .9 |
| *Proteus* spp. + *Staphylococcus aureus* | 4 | 3.5 |
| *Proteus* spp. + *Eschericia coli* + *Streptococcus faecalis* | 1 | .9 |
| *Pseudomonas* spp. + *Escherichia coli* | 4 | 3.5 |
| *Pseudomonas* spp. + *Klebsiella* spp. + *Enterobacter* spp. | 1 | .9 |
| *Escherichia coli* +*Streptococcus faecalis* | 1 | .9 |
| *Pseudomonas* spp.+ *Staphylococcus aureus* | 2 | 1.7 |
| *Pseudomonas* spp.+*Proteus* spp. + *Staphylococcus aureus* + *Streptococcus faecalis* | 5 | 4.3 |
| *Klebsiella* spp. +*Enterobacter* spp. | 1 | .9 |
| *Pseudomonas* spp.+ *Proteus* spp. + *Staphylococcus aureus* + *Streptococcus pyogenes* | 1 | .9 |
| *Pseudomonas* spp.+*Proteus* spp.+*K lebsiella* spp. | 1 | .9 |
| *Proteus* spp. + *Escherichia coli* | 3 | 2.6 |
| *Pseudomonas* spp. + *Proteus* spp. + *Escherichia coli* + *Klebsiella* spp. | 2 | 1.7 |
| *Pseudomonas* spp. + *Proteus* spp. + *Klebsiella* spp. | 1 | .9 |
| *Pseudomonas* spp. + *Proteus* spp. + *Escherichia coli* | 2 | 1.7 |
| *Pseudomonas* spp. + *Proteus* spp. + *Escherichia coli* + *Staphylococcus aureus* | 1 | .9 |
| Total | 115 | 99.4 |

## 4.4 Use of antibiotics

### 4.4.1 Types of antibiotics used

A total of 13 different types of anticrobial agents were prescribed for the patients included in the study (Table 4.18). The prevalence of prescribing was flucloxacillin (37.9%), gentamycin (14.6%), ceftriaxone (9.7%), cefuroxime (9.7%), augmentin (6.8%), amoxicillin (3.9%), ciprofloxacin (2.9%), ceftazidime (1.9%), cloxacillin (1.9%), antituberculous drugs (1.9%), chloramphenicol (1.0%) and erythromycin (1.0 %).

**Table 4.18 Types of drugs used**

| Drug | Total | | Daily dosage | | Duration | |
|---|---|---|---|---|---|---|
| | No | % | Correct | Incorrect | Indicated | Not indicated |
| Benzyl penicillin | 7 | 6.8 | 7 | 0 | 1 | 6 |
| Flucloxacillin | 39 | 37.9 | 39 | 0 | 13 | 26 |
| Gentamycin | 15 | 14.6 | 15 | 0 | 1 | 14 |
| Ceftriaxone | 10 | 9.7 | 10 | 0 | 2 | 8 |
| Ceftazidime | 2 | 1.9 | 2 | 0 | 1 | 1 |
| Cloxacillin | 2 | 1.9 | 2 | 0 | 0 | 2 |
| Cefuroxime | 10 | 9.7 | 10 | 0 | 7 | 3 |
| Antituberculous | 2 | 1.9 | 2 | 0 | 1 | 1 |
| Augmentin | 7 | 6.8 | 7 | 0 | 4 | 3 |
| Chloramphenicol | 1 | 1.0 | 1 | 0 | 0 | 1 |
| Ciprofloxacin | 3 | 2.9 | 3 | 0 | 1 | 2 |
| Amoxicillin | 4 | 3.9 | 4 | 0 | 1 | 3 |
| Erythromycin | 1 | 1.0 | 1 | 0 | 0 | 1 |
| Total | 103 | 100 | 103 | 0 | 32<br>30.76% | 71<br>68.26% |

In all the treatment sheets, correct daily dosage was indicated. Some of the patients were on more than one type of drug and the total number of drugs prescribed was 103. In the majority of cases (68.26%), the duration of administration of the drugs was not indicated.

### 4.4.2 Number of antibiotics used per patient

Thirty six and a half percent of the patients under study were not on any antibiotic while the majority, (63.5%) were on at least one antibiotic (Table 4.19). Forty two point six percent were on one drug, 17.4% on two, and 1.7% on three and four drugs respectively.

**Table 4.19 Number of antibiotics prescribed per patient**

| Number of different antibiotics per patient | Frequency | Percent |
|---|---|---|
| 1 | 49 | 42.6 |
| 2 | 20 | 17.4 |
| 3 | 2 | 1.7 |
| 4 | 2 | 1.7 |
| None | 42 | 36.5 |
| Total | 115 | 99.9 |

The records showed that most prescriptions were aimed at only one organism or at most two. Due to absence of prior culture and sensitivity results, it was not possible to establish the criteria used in prescribing. Empirical prescribing carried the day.

### 4.4.3 Antimicrobial susceptibility of isolated bacteria

***Proteus* spp.**

A total of thirty-nine isolates of *Proteus* spp., were obtained (Table 4.20). Of these, 76.9% were found to be sensitive to augmentin, 20% were resistant and 2.6% categorised as intermediate. Cefuroxime was found to be effective in 61.5% of isolates, but 35.9% were resistant. Forty six point two percent were sensitive to gentamycin, the majority were resistant (51.3%) and 2.6% were intermediate. Ciprofloxacin was found to be quite effective (87.2%) and only a small proportion (12.8%) was resistant.

**Table 4.20 Antimicrobial susceptibility of *Proteus* spp.**

| Drug | Count/Percentage | | | |
|---|---|---|---|---|
| | Resistant | Intermediate | Sensitive | Total |
| Augmentin | 8 (20.5%) | 1 (2.6%) | 30 (76.9%) | 39 (100%) |
| Cefuroxime | 14 (35.9%) | 1 (2.6%) | 24 (61.5%) | 39 (100%) |
| Gentamycin | 20 (51.3%) | 1 (2.6%) | 18 (46.2%) | 39 (100%) |
| Ciprofloxacin | 5 (12.8%) | 0 | 34 (87.2%) | 39 (100%) |
| Minocycline | 36 (92.3%) | 1 (2.6%) | 2 (5.1%) | 39 (100%) |
| Piperacillin | 18 (46.2%) | 2 (5.1%) | 19 (48.7%) | 39 (100%) |
| Ceftazidime | 4 (10.3%) | 0 | 35 (89.7%) | 39 (100%) |
| Ceftriaxone | 5 (12.8%) | 3 (7.7%) | 31 (79.5%) | 39 (100%) |

Most of the organisms were resistant to Minocycline (92.3%) while 5.1% were sensitive and 2.1% intermediate acting. Piperacillin showed 48.7% sensitivity, but 46.2% were resistant. Ceftazidime was quite effective (89.7%) and only 10.3% were resistant. For ceftriaxone, 79.5% were sensitive, 12.8% resistant and 7.7% intermediate.

***Escherichia coli***

Fifteen isolates of *Escherichia coli* were obtained (Table 4.21). The organism was found to be highly sensitive to ciprofloxacin and ceftazidime (93.3%) followed by ceftriaxone (86.7%), augmentin and gentamycin (73.3%), minocycline and cefuroxime (53.3%) and finally piperacillin (40.0%).Resistance was high against piperacillin (60%) followed by minocycline (40%), gentamycin (26.7%), cefuroxime (20.0%), ciprofloxacin and ceftriaxone (6.7%). Most of the isolates showed intermediate sensitivity especially to ceftazidime, ceftriaxone, minocycline and augmentin where a rate of 6.7% was obtained. Cefuroxime showed 26.7% intermediate sensitivity. According to this analysis, the

most effective drugs in descending order include; ciprofloxacin and ceftazidime, ceftriaxone, augmentin and gentamycin, minocycline and cefuroxime and piperacillin

**Table 4.21 Antimicrobial susceptibility of *Escherichia coli***

| Drug | Count/Percentage | | | |
|---|---|---|---|---|
| | Resistant | Intermediate | Sensitive | Total |
| Augmentin | 3 (20.0%) | 1 (6.7%) | 11 (73.3%) | 15 (100%) |
| Cefuroxime | 3 (20.0%) | 4 (26.7%) | 8 (53.3%) | 15 (100%) |
| Gentamycin | 4 (26.7%) | 0 | 11 (73.3%) | 15 (100%) |
| Ciprofloxacin | 1 (6.7%) | 0 | 14 (93.3%) | 15 (100%) |
| Minocycline | 6 (40.0%) | 1 (6.7%) | 8 (53.3%) | 15 (100%) |
| Piperacillin | 9 (60%) | 0 | 6 (40.0%) | 15 (100%) |
| Ceftazidime | 0 | 1 (6.7%) | 14 (93.3%) | 15 (100%) |
| Ceftriaxone | 1 (6.7%) | 1 (6.7%) | 13 (86.7%) | 15 (100%) |

***Pseudomonas* spp.**

*Pseudomonas* spp., were found to be the most common organisms (Table 4.22). A total of forty-nine isolates were obtained that showed sensitivity to most drugs. Out of the eight drugs tested, the sensitivity pattern was of the order; pipril/tozabactam (89.8%), piperacillin (83.7%), ceftazidime (77.5%), meropenem (75.5%), amikacin (73.5%), ticarcillin/clavulonic acid (65.3%), gentamycin (55.1%) and ceftriaxone (30.6%). Resistance was highest against ceftriaxone (46.8%), followed by gentamycin (42.9%), ticarcillin/clavulonic acid (27.7%), amikacin and meropenem (22.4%), Piperacillin (16.3%) and ceftazidime (15.2%). Drugs that showed intermediate activity were meropenem (2.2%), gentamycin (2.2%), ceftazidime (2.2%), amikacin and ticarcillin/clavulonic acid (4.3%) and ceftriaxone (21.3%).

**Table 4.22 Antimicrobial susceptibility of *Pseudomonas* spp.**

| Drug | Count/Percentage | | | |
|---|---|---|---|---|
| | Resistant | Intermediate | Sensitive | Total |
| Pipril/Tozabactam | 5 (10.2%) | 0 | 44 (89.8%) | 49 (100%) |
| Meropenem | 11 (22.4%) | 1 (2.2%) | 37 (75.5%) | 49 (100%) |
| Gentamycin | 21 (42.9%) | 1 (2 %) | 27 (55.1%) | 49 (100%) |
| Amikacin | 11 (22.4%) | 2 (4.3%) | 36 (73.5%) | 49 (100%) |
| Ceftazidime | 10 (20.4%) | 1 (2%) | 38 (77.5%) | 49 (100%) |
| Ceftriaxone | 22 (44.8%) | 12 (24.5%) | 15 (30.6%) | 49 (100%) |
| Ticarcillin/clavulonic acid | 13 (26.5%) | 4 (8.2%) | 32 (65.3%) | 49 (100%) |
| Piperacillin | 8 (16.3%) | 0 | 41 (83.7%) | 49 (100%) |

***Klebsiella* spp.**

*Klebsiella* spp., were shown to exhibit significant resistance against several drugs (Table 4.23). In descending order they were; minocycline (66.7%), augmentin and piperacillin (55.6%), cefuroxime (44.4%), gentamycin (22.2%), ceftazidime (22.2%), ceftriaxone (22.2%) and ciprofloxacin (22.2%).

**Table 4.23 Antimicrobial susceptibility of *Klebsiella* spp.**

| Drug | Count/Percentage | | | |
|---|---|---|---|---|
| | Resistant | Intermediate | Sensitive | Total |
| Augmentin | 5 (55.6%) | 0 | 4 (44.4%) | 9 (100%) |
| Cefuroxime | 4 (44.4%) | 2 (22.2%) | 3 (33.3%) | 9 (100%) |
| Gentamycin | 2 (22.2%) | 0 | 7 (77.9) | 9 (100%) |
| Ciprofloxacin | 2 (22.2%) | 0 | 7 (77.8%) | 9 (100%) |
| Minocycline | 6 (66.7%) | 1 (11.1%) | 2 (22.2%) | 9 (100%) |
| Piperacillin | 5 (55.6%) | 1 (11.1%) | 3 (33.3%) | 9 (100%) |
| Ceftazidime | 2 (22.2%) | 0 | 7 (77.8%) | 9 (100%) |
| Ceftriaxone | 2 (22.2%) | 0 | 7 (77.8%) | 9 (100%) |

Among the nine isolates, some drugs exhibited good activity. They included ceftriaxone (77.8%), ceftazidime (77.8%), ciprofloxacin (77.8%) and gentamycin (77.9%). Intermediate activity was also portrayed by cefuroxime (22.2%), minocycline (11.1%) and piperacillin (11.1%).

***Staphylococcus aureus***

*Staphylococcus aureus* was found to be the second most common organism isolated from infected wounds and showed significant resistance to several drugs, which are commonly used (Table 4.24). They were amoxicillin (86.3%), augmentin (60.5%), erythromycin (52.6%), oxacillin (44.7%), gentamycin (39.5%), cefuroxime (39.5%), clindamycin (15.8%) and minocycline (10.5%). Insignificant numbers showed intermediate sensitivity. Drugs that showed above average activity against the organism were clindamycin, (84.2%), minocycline (86.8%), ciprofloxacin (60.5%), gentamycin (60.5%),

**Table 4.24 Antimicrobial susceptibility of *Staphylococcus aureus***

| | Count/Percentage | | | |
|---|---|---|---|---|
| Drug | Resistant | Intermediate | Sensitive | Total |
| Amoxicillin | 33 (86.3%) | 0 | 5 (13.2%) | 38 (100%) |
| Augmentin | 23 (60.5%) | 0 | 15 (39.5%) | 38 (100%) |
| Oxacillin | 17 (44.7%) | 0 | 21 (55.3%) | 38 (100%) |
| Erythromycin | 20 (52.6%) | 1 (2.6%) | 17 (44.7%) | 38 (100%) |
| Gentamycin | 15 (39.5%) | 0 | 23 (60.5%) | 38 (100%) |
| Ciprofloxacin | 14 (36.8%) | 1 (2.6%) | 23 (60.5%) | 38 (100%) |
| Minocycline | 4 (10.5%) | 1 (2.6%) | 33 (86.8%) | 38 (100%) |
| Cefuroxime | 15 (39.5%) | 1 (2.6%) | 22 (57.9%) | 38 (100%) |
| Clindamycin | 3 (15.8%) | 0 | 16 (84.2%) | 19 (100%) |

oxacillin (55.3%), cefuroxime (57.9%), erythromycin (44.7%), augmentin (39.5%) and amoxicillin (13.2%). Several strains were probably beta lactamase producing which explains why penicillins were largely ineffective.

***Streptococcus pyogenes***

*Streptococcus pyogenes* occur rarely in wounds and Table 4.25 attest to this fact. Out of 115 specimens taken only two had this organism. It was found to be 100% sensitive to amoxicillin, cefuroxime, augmentin, erythromycin, chloramphenicol, ofloxacin and ceftriaxone.

**Table 4.25 Antimicrobial susceptibility of *Streptococcus pyogenes***

| | Count/Percentage | | | |
|---|---|---|---|---|
| Drug | Resistant | Intermediate | Sensitive | Total |
| Amoxicillin | 0 | 0 | 2 (100%) | 2 (100%) |
| Cefuroxime | 0 | 0 | 2 (100%) | 2 (100%) |
| Augmentin | 0 | 0 | 2 (100%) | 2 (100%) |
| Erythromycin | 0 | 0 | 2 (100%) | 2 (100%) |
| Chloramphenicol | 0 | 0 | 2 (100%) | 2 (100%) |
| Ofloxacin | 0 | 0 | 2 (100%) | 2 (100%) |
| Tetracycline | 1 (50%) | 0 | 1 (50%) | 2 (100%) |
| Ceftriaxone | 0 | 0 | 2 (100%) | 2 (100%) |

Tetracycline showed 50% effectiveness and 50% resistance. It was a proof that the organism was not a serious problem when targeted for eradication since most of the drugs are common and cheap.

***Streptococcus faecalis***

*Streptococcus faecalis* was highly sensitive to penicillins (Table 4.26). According to the study, a total of seven organisms were isolated. They were found to vary in their sensitivity to drugs, among them amoxicillin (100%), augmentin (85.7%),

nitrofurantoin (71.4%), streptomycin (57.1%), vancomycin (57.1%), ciprofloxacin (28.6%) and gentamycin (28.6%).

**Table 4.26 Antimicrobial susceptibility of *Streptococcus faecalis***

| Drug | Count/Percentage | | | |
|---|---|---|---|---|
| | Resistant | Intermediate | Sensitive | Total |
| Vancomycin | 2 (28.6%) | 1 (14.3%) | 4 (57.1%) | 7 (100%) |
| Amoxicillin | 0 | 0 | 7 (100%) | 7 (100%) |
| Augmentin | 1 (14.3%) | 0 | 6 (85.7%) | 7 (100%) |
| Ciprofloxacin | 4 (57.1%) | 1 (14.3%) | 2 (28.6%) | 7 (100%) |
| Gentamycin | 3 (42.9%) | 2 (28.6%) | 2 (28.6%) | 7 (100%) |
| Streptomycin | 2 (28.6%) | 1 (14.3%) | 4 (57.1%) | 7 (100%) |
| Nitrofurantoin | 2 (28.6%) | 0 | 5 (71.4%) | 7 (100%) |
| Nalidixic acid | 7 (100%) | 0 | 0 | 7 (100%) |

It showed significant resistance against several drugs including nalidixic acid (100%), ciprofloxacin (57.1%), gentamycin (42.9%), nitrofurantoin (28.6%), streptomycin (28.6%), vancomycin (28.6%), augmentin (14.3%). Intermediate activity was shown by vancomycin (14.3%), ciprofloxacin (14.3%), gentamycin (28.6%) and streptomycin (14.3%).

***Enterobacter* spp.**

Only three isolates were found. This reflected the scarcity of the organism in the wounds. Sensitivity was 100% to ciprofloxacin, piperacillin, ceftazidime, and ceftriaxone. Variable sensitivity patterns were exhibited by other drugs such as augmentin (33.3%) and gentamycin (66.7%). Resistance was highest against augmentin (66.7%) followed by cefuroxime (33.3%), minocycline (33.3%) and gentamycin (33.3%). Intermediate activity was shown by cefuroxime (66.7%) and minocycline (66.7%).

***Alcaligenes* spp.**

These organisms were rare and isolated only from two specimens. In addition most of the drugs were 100% effective, among them being gentamycin, ciprofloxacin, piperacillin, ceftazidime and ceftriaxone. *Alcaligenes* spp., exhibited 100% resistance against augmentin and minocycline. Cefuroxime was 50% sensitive and 50% moderately active.

***Citrobacter freundii***

Only one isolate was obtained that showed 100% sensitivity to augmentin, cefuroxime, gentamycin, ciprofloxacin, minocycline, piperacillin, ceftazidime and ceftriaxone.

***Serratia* spp.**

Only one organism was isolated, which showed absolute sensitivity to augmentin, ciprofloxacin, minocycline, piperacillin, ceftazidime and gentamycin. Cefuroxime was ineffective and minocycline showed intermediate sensitivity.

***Acinetobacter baumanii***

One organism was isolated that showed absolute sensitivity to augmentin, gentamycin, ciprofloxacin, minocycline, piperacillin and ceftazidime. It was resistant to ceftriaxone and cefuroxime.

# CHAPTER FIVE: DISCUSSION, CONCLUSION AND RECOMMENDATIONS

## 5.1 DISCUSSION

### 5.1.1 Age and Gender

Wound infections are universal and all age groups and both sexes are susceptible. Physical injury is known to raise circulating levels of stress hormones, notably corticosterones, which are potent suppressors of immune function and would be expected to increase susceptibility to infection following injury [50, 51].More males than females were admitted with infected wounds. This is probably due to the roles played by men which involve risky ventures.

### 5.1.2 Washing of hands during dressing of wounds

Hand washing is an important prerequisite in wound infection control. Unfortunately, most of the nurses washed their hands at the end of the exercise. *Staphylococcus aureus* is a normal flora of the skin and possibility of transmission through contact was high. Patients who assisted in opening the wounds posed a danger to themselves and to others. By soiling their hands and clothing, the risk of subsequent infections could be increased. The hands of staff are the commonest vehicles by which microorganisms are transmitted between patients and hand washing is accepted as the single most important measure in infection control [52]. Not surprisingly, hospital staff believes that they wash their hands more often than they actually do, and they overestimate the duration of hand washing [53]. In a study of nurses' practices, hands were only cleaned after 30% of patient

contacts and after 50% of activities likely to result in heavy contamination [54]. Poorer hand washing performance was related to increasing nursing workload and the reduced availability of hand decontaminating agents [54]. At many hospitals and clinics, particularly in developing countries, washing basins are poorly accessible and the unavailability of soap, sprays, and hand towels is a regular annoying occurrence [55]. It is difficult to provide clear guidelines on how often hands should be washed but preferably hands should be decontaminated before each patient contact [56].

### 5.1.3 Use of gloves

Although most nurses used gloves during wound dressing, the frequency of changing them was not commendable. New gloves were worn at the beginning of the dressing exercise and subsequently when they became soiled, instead of after attending each patient. The consequence of all these malpractices was increased risk of transmitting microorganisms between patients and staff members. Several wound pathogens can remain viable outside the body for a long time therefore enhancing their prevalence.

Gloves are a useful additional means of reducing nosocomial infection, but they supplement rather than replace hand washing. Possible microbial contamination of hands and transmission of infection has been reported despite gloves being worn [63]. Not surprisingly, health care workers who wash their hands more often are also more likely to wear gloves [57]. Single use gloves should never be washed, resterilised, or disinfected, and gloves must be changed after each patient encounter [52]. For gloves to be used appropriately they must be readily available.

### 5.1.4 Use of facemask

Most of the nurses wore facemasks when dressing wounds during the study but it has never been shown that wearing surgical facemasks decreases wound infections. When originally introduced, the primary function of the surgical mask was to prevent the migration of microorganisms residing in the nose and mouth of members to the open wound of the patient. However, it is now recognized that most bacteria dispersed by talking and sneezing are harmless to wounds [58]. The prevailing opinion that masks are useful in preventing surgical site infection has been challenged [58]. Orr reported a 50% decrease in wound infections when masks were worn, but the study was criticized for lack of proper controls [59]. Tunevall, using better controls, confirmed the earlier findings of lack of clear benefit from wearing masks [60]. Thus while masks may be used to protect the nurses from airborne infections, they have not been proved to protect the patient [59].

### 5.1.5 Use of antiseptics

Antiseptics are widely used in wound management. Their effectiveness depends on the type of wound and any other factor that might cause their deterioration. Hydrogen peroxide acts by releasing nascent oxygen but it is short acting because the release occurs rapidly [61]. It is the substance released by active neutrophils and it is an active microbicide when applied in close contact with most microorganisms [61]. However, the ubiquitous enzyme catalase often destroys it before it reaches organisms in wounds. Effervescence helps cleanse wounds mechanically [61]. During the study, the concentration of hydrogen peroxide used could not be established. This was because the

containers had no labels and occasionally the nurses used to add water when washing wounds. In 12.5% of the cases hydrogen peroxide alone and in 25% a mixture of hydrogen peroxide and betadine were used. Mixing made it even more difficult to establish the concentration and therefore its effectiveness was doubtful.

A three-percent solution of hydrogen peroxide is commonly used as a wound antiseptic and it demonstrates *in vitro* broad-spectrum efficacy with greatest activity against Gram-positive bacteria [62]. The presence of catalase in these bacteria makes dilutions below three percent less effective [62]. In a similar fashion, catalase present in tissues can render hydrogen peroxide even less bactericidal *in vivo* [63]. Although hydrogen peroxide is commonly used, surprisingly few studies have been conducted to examine its effect on the wound healing process and its efficacy as a wound antiseptic.

Betadine (Povidone-iodine) kills Gram-positive and Gram-negative bacteria, fungi, viruses, protozoa and yeasts and it takes 6-8 h for the skin bacterial population to return to normal [64]. It occurs in aqueous solution and should be applied twice daily on an infected wound. Betadine was used to clean the wounds. In addition, the inner gauze was soaked into the solution before placing it on the wound occasionally. The minimum period for a repeat dressing was 24 h. In some cases, dressings were changed after two days or more. Its effectiveness was quite doubtful especially considering that in some patients (37.5%), it was used for dressing.

Povidone Iodine is a useful bacteriostatic and bactericidal agent shown to be effective against MRSA and other pathogens in *in vitro* and clinical studies [64]. The slow release from the iodophor and cadexomer versions is intended to optimize activity and reduce

toxicity. The use of PVP-I as a pre-surgical skin antiseptic is unquestioned although its value in wound antisepsis is subject to debate [65, 66]. Dressings containing iodine in slow-release formulations are considered safe and effective [66, 68]. The FDA maintains that PVP-I in 5% to 10% solution does not adversely affect healing, although a later review of published data disputes this view [69]. Povidone iodine at a concentration of 0.001% is bactericidal and non-cytotoxic to fibroblasts *in vitro* [70]. Iodine as the PVP-I iodophor is available in a range of concentrations as medicated dressings, solutions and ointments, powders and sprays, and incise drapes. The cadexomer is a polysaccharide starch lattice containing 0.9% elemental iodine that is released on exposure to exudates and has antimicrobial activity for up to 3 days [67]. It has been extensively evaluated in a variety of acute and chronic wounds and found to be safe and effective in reducing bioburden[68].

The PVP-cadexomer-containing dressings provide sustained release of low levels of free iodine in the presence of moisture [71]. Consequently, for best effect, modern iodinated dressings should only be used on exuding wounds. The microbicidal action spectrum of povidone-iodine is broad. Unlike local antibiotics and other antiseptic substances, no resistance develops. The high degree of bactericidal efficiency in respect of highly resistant Gram-positive pathogenic micro-organisms, such as methicillin-resistant *Staphylococcus aureus* and *Enterococcus* strains, is particularly significant for hospital hygiene. On the basis of available research, it appears that povidone-iodine should be promoted by nurses and other health-care professionals involved in wound care as a means of preventing and treating infection in a range of acute and chronic wounds [72].Overall observations from animal models have indicated cytotoxicity against

leukocytes, fibroblasts and keratinocytes, but human studies suggest that PVP-I reduces bacterial load, decreases infection rates and promotes healing [73].

Furthermore, histological assessment indicated lack of cytotoxicity because PVP-I induced less changes in microvessels and dendrocytes [74]. Additionally, a report of the ability of iodine released from a dressing to modulate the secretion of cytokines by human macrophages *in vitro* has provided another justification of its role in promoting healing [75].

Savlon(Chlorhexidine) has doubtful value although it was used by 6% of the nurses. A study involving six trials used four per cent chlorhexidine gluconate and compared chlorhexidine with placebo [76]. Bathing with chlorhexidine did not reduce surgical site infection rate. Although chlorhexidine is useful in disinfecting intact skin and cleaning dirty traumatized wounds, this agent should not be used on clean healing wounds because it can further increase patients' morbidity [77].

Sodium chloride isotonic solution was used to wash very dirty wounds by 12.5% of the staff and occasionally alternately with betadine. It has no antibacterial activity and was only meant to keep the tissues moist and clean. A study done in Brazil observed that saline solution was the irrigation product of choice (85.4%) followed by antiseptic solution, Ringer's lactate, distilled water, antibiotic solution and other products [72]. Manual irrigation was the most preferred than pulsed irrigation by participants.

Saline is the only topical treatment used on most open or infected postoperative wounds. Treatment with saline alone significantly increases microbial titers and should not be

relied upon to completely reduce bacterial contamination, although it removes debris, foreign materials and clots, which often contain bacteria [78]. An *in vitro* study showed that saline irrigation reduced colony counts of *Staphylococcus aureus*, *Staphylococcus epidermidis* and *Escherichia coli* by 12%–56%.

**Use of sterile equipments**

Equipments used during wound dressing must be sterile to avoid cross transmission. Use of contaminated forceps and kidney dishes in 68.8% of cases was disastrous. The isolation of organisms with similar antimicrobial susceptibility suggested related sources of infection.

### 5.1.2 Use of Antimicrobial drugs

Factors that influence the effectiveness of drugs are route of administration, susceptibility of the organism to antimicrobial agent and ability to reach optimal concentration at the site of action [5]. Bacteria can resist antimicrobial agents through several mechanisms such as

i. Changing Proteins and other components of bacterial cells, which antimicrobials use as binding sites. Changes of genetic origin are associated with resistance to amikacin, gentamycin, clindamycin and erythromycin.

ii. Production of enzymes that destroy or inactivate antimicrobials. The genes that code for the production of resistant enzymes are carried on plasmids and can be transferred from one species or genus of

bacterium to another. Examples include beta lactamases, which inactivate beta-lactam antibiotics such as amoxicillin, piperacillin, ceftrixone, ceftazidime and cefuroxime that were encountered during the study. Some Gram-negative bacteria produce acetylating, adenylating and phosphorylating enzymes that are capable of inactivating aminoglycosides. Chloramphenicol is inactivated by enzyme acetyltransferase.

iii. Changing to other metabolic systems not affected by the antimicrobial agent being used. This mechanism of resistance is found in some sulphonamides resistant bacteria. In the study, no sulphonamide was used.

iv. Altering the permeability of their cell wall membrane, making it difficult for antimicrobials to enter. This type of resistance is found in bacteria resistant to polymyxins and tetracycline. The cell wall of streptococci forms a natural barrier to aminoglycosides.

**Aminoglycosides**

They act by binding to 30s sub unit and prevent protein synthesis [79]. Streptomycin causes misleading by distorting the conformation of the mRNA so that the wrong amino acyl – tRNA binds to the cordon. At high concentrations, streptomycin fixes to the ribosomes and complexes with the mRNA. F-met – tRNA initiates protein synthesis under these circumstances but protein synthesis does not continue beyond initiation. After about 5 minutes, the permanently altered ribosome's fall off the mRNA. If the

altered ribosome complexes with a new piece of mRNA, it will block reading of the mRNA. This polysomal blockade completely halts protein synthesis.

Acquired aminoglysides resistance is most commonly due to the presence of a plasmid (R factor) that encodes an enzyme, which inactivates aminoglycosides by attaching acetyl phosphate, or adenyl group to the modifying enzyme, which is located in the cytoplasmic membrane [79]. Here the enzyme attaches a group to the aminoglycoside during transport and the inactivated antibiotic is released into the cytoplasm. This has a secondary effect of slowing antibiotic transport. Two other modes of resistance to aminoglycoside have been described. In the first mode *Pseudomonas aeruginosa* and occasionally enteric bacteria undergo a smooth to rough lipopolysacharide transition that greatly reduces the ability of aminoglycoside to pass through the outer membrane. In the second mode, *Enterococcus* and *Pseudomonas* experience a mutation in the 12S protein gene causing the gene to lose its ability to recognize and avidly bind the drug.

Aminoglycosides encountered during the study were gentamycin, streptomycin and amikacin. Gentamycin was found to be the second most commonly prescribed drug, probably because it is cheap and easily available. No patient was on amikacin, which is more expensive. In all susceptibility tests, gentamycin was found to be reasonably effective. Above average sensitivity was observed with *Escherichia coli, Pseudomonas* spp., *Klebsiella* spp., *Staphylococcus aureus, Enterobacter* spp., *Alcaligenes* spp., *Citrobacter freundi, Serratia* spp., and *Acinetobacter baumanii.* According to past studies, aminoglycosides were found to be active against these organisms [5, 36, 12, 22, 23, 38]. These drugs therefore have an important role in the management of wound

infections at KNH. *Proteus* spp., and *Enterococcus* were largely resistant to aminoglycosides tested. The mechanisms of resistance described above may have played a substantial role and therefore judicious use of the drug should be observed.

Out of the fourteen treatment sheets none had any duration of administration of aminoglycosides indicated, a pointer that irrational use was highly likely. Under use may augment resistance while over use predisposes patients to side effects such as vestibular and auditory damage, nephrotoxicity, nausea, vomiting and hypomagnesia [60]. Use of aminoglycosides should be very rational; otherwise the prevalence of resistance may continue to rise.

**Beta lactam antibiotics**

They include the penicillins and cephalosporins and act by inhibiting cell wall synthesis [79]. The beta lactam ring acts as an analogue of acyl-D-alanine and binds tightly to the active site of transpeptidase enzymes that catalyze the transpeptidation of murnac units within the cell wall. Transpeptidation is important in cross-linking the murei sauculus and provides it with the tensile strength needed to resist osmotic lysis.

Resistance against beta lactam antibiotics is due to several reasons [79]. First, changes in the permeability of bacterium may occur where the drug fails to penetrate. Secondly, some organisms produce beta lactamases that destroy the ring. The third major mechanism of resistance involves changing the affinity of PBS for beta lactam antibiotics. Finally, the antibiotic may fail to induce autolysis. This renders the antibiotic bacteriostatic rather than bactericidal.

Several beta-lactam antibiotics were used in the study. The penicillin's tested were augmentin, piperacillin, amoxicillin, meropenem, ticarcillin and oxacillin. All these are semi synthetic penicillins and differed in their activities among different organisms. Augmentin showed above average activity against *Enterobacteriaceae* except *Klebsiella* spp., which showed 44.4% sensitivity. *Staphylococcus aureus* seemed to exhibit more than one method of resistance since 60.5% of the isolates were resistant. *Streptococcus pyogenes* and *Streptococcus feacalis* were sensitive to augmentin. Amoxicillin had similar pattern to that of augmentin. Oxacillin showed moderate activity against *Staphylococcus aureus* (55.3%).

The antipseudomonal penicillins; Piperacillin, pipril/tozobactram, ticarcillin/clavuconic acid and meropenem exhibited good activity against *Pseudomonas* spp., with sensitivity above 68%. More common organisms such as *Proteus* spp., *Escherichia coli* and *Klebsiella* spp., were quite resistant to piperacillin. However, rare members of *Enterobacteriaceae* such as *Enterobacter* spp., *Alcaligenes* spp., and *Serratia* spp., were quite sensitive.

Flucloxacillin was the most commonly prescribed drug while amoxicillin and benzyl penicillin were occasionally used. From the results obtained, their relevance was highly questionable. The *Staphylococcus aureus* isolated was significantly resistant to penicillins tested. Augmentin still offered hope due to its activity against *Enterobacteriaceae*, which were quite prevalent.

Cephalosporins tested were cefuroxime, ceftazidime and ceftriaxone. Ceftazidime offered the greatest hope with all exposed organisms showing high sensitivity of over 80%. Cefuroxime was moderately active against *Escherichia coli*, *Staphylococcus*

*aureus*, *Enterobacter* spp., *Alcaligenes* spp., *Proteus* spp., and *Klebsiella* spp. *Streptococcus pyogenes* and *Citrobacter* spp., showed high sensitivity to cefuroxime. *Serratia* spp., and *Acinetobacter baumanii* were virtually resistant. Although ceftazidime was highly effective, only 1.94% of patients were on the drug. The cost involved could have been the limiting factor. Cefuroxime ranked third as the most widely preferred drug by prescribers, but its effectiveness was not laudable.

Ceftriaxone had the same ranking as cefuroxime in prescription. It was found to be highly active against most members of *Enterobacteriaceae* family. *Pseudomonas* spp., showed significant resistance of 46.8%.

Studies carried out at Rawalpidi in Pakistan showed that different organisms had variable susceptibility to beta lactam antibiotics [12]. *Staphylococcus aureus* was sensitive to cefaclor, cefuroxime, augmentin, cefotaxime and ceftazidime but resistant to amoxicillin and piperacillin [12, 14]. *Escherichia coli* and *Klebsiella* spp., were highly sensitive to ceftizoxime and ceftriaxone while *Pseudomonas* spp., and *Proteus* spp. were resistant to amoxicillin and augmentin [12, 14, 80] suggesting that susceptibility patterns vary depending on the region.

*Proteus* spp., from KNH depicted different sensitivity patterns, where amoxicillin and augmentin had above average activity. Third generation cephalosporins (ceftazidime and ceftriaxone) had tremendous activity against *Escherichia coli* and *Klebsiella* spp. *Staphylococcus aureus* was more resistant against amoxicillin, augmentin and cefuroxime. Ceftazidime was equally effective against *Pseudomonas* spp., from both places. Therefore, the results from Pakistan study [12] and KNH were comparable.

**Quinolones**

Nalidixic acid and other fluoroquinolones bind the A subunit of DNA gyrase to form phosphodiester bonds [79]. This kills the bacterium by interfering with DNA replication. Resistance to quinolone antibiotics is due to mutation of the gyrA gene.

Ciprofloxacin was found to be effective against all members of *Enterobacteriaceae* family isolated during the study. It showed above 50% activity against *Staphylococcus aureus*. *Streptococcus faecalis* showed marked resistance to both ciprofloxacin and nalidixic acid. Only nitrofurantoin was effective against *Streptococcus faecalis* resembling observation made in a past study by Abdulla *et al.*, 2006 [81]. Sensitivity to ofloxacin by *Streptococcus pyogenes* was absolute.

Ciprofloxacin is cheap and easily available. Because most of the wound pathogens are sensitive, it should be regularly used. Unfortunately, it ranked seventh among the most prescribed drugs in orthopedic wards.

Ciprofloxacin and ofloxacin were found to have above average activity against *Escherichia coli*, *Klebsiella* spp., *Proteus* spp., and *Staphylococcus aureus* [82]. This suggested that resistance against quinolones was low and they should often be used to treat wound infections [82, 83].

**Tetracyclines**

Tetracycline binds to 30S ribosomal fragment and allows an aminoacyl-t-RNA to come to the A site [79]. The aminoacyl-t-RNA is not able to bind stably to the A site and elongation of the peptide is blocked. Resistance occurs through a plasmid transfer that

encodes an inducible active reflux system. This system does not affect the entry of tetracyclines instead; they are transported normally and then rapidly pumped back out of the bacterium. Most of the *Enterobacteriaceae* members were largely resistant to minocycline, luckily the drug was discontinued due to hepatotoxicity. *Streptococcus pyogenes* was 50% resistant to tetracycline.

Among the drugs prescribed, tetracycline did not feature. Its usefulness in wound infections was not recognized. The side effects coupled with interactions makes it not patient friendly [39]. Tetracycline was found to be moderately active against *Klebsiella* spp., and *Staphylococcus aureus* in a past study [14].

**Macrolides**

Erythromycin binds reversibly to free ribosomes but does not bind to polysones [79]. It remains bound to the ribosome during initiation and allows a short peptide to be formed but then it blocks any further synthesis. Both translocation and elongation are blocked. The blocked complex is unstable, so the ribosomal fragments are released from mRNA. These fragments can reassemble a new mRNA but their function will once again be blocked. Bacteria resist the action of macrolides by two mechanisms. First, many bacteria experience a mutation of a chromosomal gene encoding the L4 or the L12 protein in the 50S ribosomal fragment. Second some bacteria carry R factor that encodes an enzyme which dimethylates 23S rRNA in the 50s ribosomal fragment. Because of either of these mechanisms, the 50s fragment fails to avidly recognize the antibiotic. When the mechanism is due to an R factor, the bacterium is resistant to all macrolides and lincosamides.

Only *Staphyococcus aureus* was exposed to erythromycin during the study. Most of the isolates (52.6%) resisted the drug. High sensitivity was shown to clindamycin (84.2%), an indication that probably the first mechanism described above was predominant. Erythromycin was rarely prescribed and was present in only one treatment sheet. The drug is cheap and easily available but has several side effects. In addition, its absorption is usually not complete and tends to cause many gastrointestinal disturbances [39].

It was quite effective against *Streptococcus pyogenes* with sensitivity of 100%. Similar sensitivity patterns to erythromycin were observed in a past study [12].

**Chloramphenicol**

Chloramphenicol is a bacteriostatic agent that inhibits the action of peptidyl transferase and does not allow a peptide bond to form [79]. Because of the bacteriostatic action, it is generally not administered in combination with a bacteriocidal antibiotic. Resistance is caused by an R factor containing resistance genes to both chloramphenicol and tetracyclines. *Streptococcus pyogenes* was 100% sensitive to chloramphenicol. The drug is quite toxic and has been implicated in the development of irreversible aplastic anemia and hence it is rarely used [39]. In addition, enteric bacteria and *Pseudomonas* spp., possess R factor, which makes them quite resistant. It is probably for this reason that the drug is not often used bearing in mind that most wound pathogens are of enteric origin. Out of 103 drugs prescribed, only one was chloramphenicol. The drug was effective against *Streptococcus feacalis* according to a study done by Shampa *et al.*; [82].

### 5.1.3 Prevalence of bacteria isolated

Wound pathogens are generally spread from one person to another through contact with contaminated hands or surfaces. In addition, most bacteria can remain viable on inanimate objects for a long time. Therefore observing proper aseptic techniques is important for their effective control. Failure to wash hands, use of unsterile equipments and contaminated hospital linen contributed to their spread.

*Pseudomonas* spp., finds numerous reservoirs, for example disinfectants, respiratory equipments, food, sink, taps and mops. Furthermore, they are constantly re-introduced into the hospital environment on fruits, plants, and vegetables as well as by visitors and patients transferred from other facilities [11]. Prevalence of *Pseudomonas* spp., was the highest at 42.6%. Other studies carried out in Iran, USA, Nigeria and India [7, 15, 16, 17, 19, 23] yielded similar results to the one obtained in the study suggesting that the organism is widely spread across countries. The organism can remain outside the body for long and may be easily carried by staff.

*Staphylococcus aureus* is a normal resident of the nares and bowel of 30-50% of the general population [26]. The organism is carried by about 90% of the hospital clinical staff and is unusually resistant to drying.

Microbiological analysis revealed that *Staphylococcus aureus* was the second leading etiologic agent of wound infection at KNH. This is similar to reports of studies from Nigeria, Iran, and USA [7, 15, 17, 19, 23]. The presence of *Staphylococcus aureus* in nasal pathways has been identified as an important risk factor for the acquisition of *Staphylococcus aureus* infection, although this may depend on an array of either factors

that may be environmental or patient-related [82]. The postulated sequence of events leading to infection is initiated with *Staphylococcus aureus* nasal carriage, which is then disseminated via the hands to other body sites where infection can occur through breaks in the dermal surfaces [13].

*Enterobacteriaceae* members are primary inhabitants of the lower gastrointestinal tract of man and animals [8]. The most predominant organism was *Proteus* spp., followed by *Escherichia coli*, *Klebsiella* spp., *Enterobacter* spp., *Serratia* spp., *Alcaligene* spp., *Acinetobacter* spp., and *Citrobacter freundii*. Total cumulative prevalence of all these organisms was 62.06%, which was the highest and resembled similar result obtained in previous studies [10, 11, 17]. The risk of patients contracting other diseases caused by these organisms was high.

*Streptococcus faecalis* is carried in the gastrointestinal tract, oral cavity, gall bladder, urethra and vagina [5]. It is an opportunistic pathogen and preferentially causes diseases in patients who are debilitated or immune compromised. It was the sixth most prevalent organism isolated. The mode of spread was mainly through contact especially with linen soiled with human faeces. This was possible since most patients were bed ridden and had bedsores. On defecation, they soiled the linen, thus enhancing contact with open wounds.

*Streptococcus pyogenes* is widely distributed in nature, such as in water, dust, vegetables, milk and milk products [5]. The mode of spread is mainly exhalation. The small percentage of the organisms isolated implies that they are not common wound pathogens.

*Pseudomonas* spp., *Staphylococcus aureus*, *Proteus* spp., *Klebsiella* spp., and *Escherichia coli* are the most common wound pathogens according to several studies carried out in different parts of the world [12, 14, 15]. Rare wound pathogens were *Streptococci*, *Peptostreptococci*, *Acinetobacter* spp., *Enterobacter* spp., and *Citrobacter* spp.,[16].

According to different studies, uniformity of prevalence's does not exist [12]. The only notable issue was that wound pathogens were mainly enteric organisms as well as skin flora. However, specimens' from KNH had relatively higher percentage of *Pseudomonas* spp., suggesting that the hospital environment may be highly contaminated.

**5.2 Conclusions**

The results showed that the management of wounds was deficient in orthopaedic wards at KNH. The clinical malpractices as exhibited by lack of use of recommended protocols may be the most important contributors to wound infections. Variables that correlated negatively include; misuse of gloves, use of facemasks and hand washing during dressing of wounds. In addition most of the instruments used especially the forceps were often non-sterile. The overall effect would be spreading of bacteria from one patient to the other.

Use of antibiotics correlated negatively to the recommended standards. Empirical prescribing coupled with inadequate information, especially regarding duration of use contributed to irrational drug use.

## 5.3 Recommendations

### 5.3.1 Infection control

a) Wounds should be thoroughly cleaned and all necrotic tissues removed. Cleaning should be done with suitable antiseptics. The concentrations of the antiseptic must be appropriate and mixing should be avoided. Irrigation method where the antiseptics are poured freely on the wound is recommended and using gauze for cleaning discouraged. Antiseptics should be stored in clean and well-labelled containers.

b) The nurses should preferably wash their hands before the dressing exercise and subsequently after every patient contact. A suitable disinfectant should be used. This will minimise the bacteria burden on the skin of the nurses thereby reducing risk of transmission.

c) Patients should be turned regularly to prevent development of bedsores, which were common and may serve as reservoirs of infection. Infected bedsores may predispose patients to other systemic infections such as septicaemia, which may complicate wound management. Sharing of beds among patients should be discouraged since it may enhance transmission of bacteria between patients. Beddings should be disinfected frequently because they may serve as reservoirs of bacteria.

d) All instruments used during wound dressing must be sterile. There should be a sterile dressing kit for each patient. Sharing of the instruments must be avoided.

e) Gloves should be worn during the dressing exercise before every patient contact. Only nurses should handle the soiled dressing materials. Patients must never participate in opening or closing the wounds. A no-touch technique for examining and changing dressings should be adopted. The used materials should be kept in a container that should be covered. The soiled materials should be removed from the ward immediately after the dressing exercise is over and subsequently destroyed.

f) Prolonged hospitalization should be avoided whenever possible. Procedures should be done on the patients without delay to avoid congestion in the wards. This will reduce the chances of transmission between patients.

g) Further research should be carried out to assess the extent of contamination of hospital environment, especially beddings, water sinks and floor. This is important because the level of wound infections may be reduced through decontamination of these materials and surfaces.

### 5.3.2 Rational use of drugs

a) All prescribed drugs must have the duration of administration indicated to avoid misuse. When the drug is been administered, a clear mark should be put on the treatment sheet to avoid a repeat or skipping the dose.

b) Empirical prescribing should be minimised. Preferably, sensitivity testing of wound specimen should be done for every patient with infected wound before administration

of drugs. This is because some patients had multiple infections. Therefore, it wa difficult to determine the suitable drug to be used that could eradicate all the bacteri involved without prior antimicrobial susceptibility testing.

c) Treatment guidelines for using antibiotics should be formulated and reviewe occasionally to ensure that any change in sensitivity patterns will be taken care of. They may also enhance supplying of the relevant drugs to the department by the hospital.

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## APPENDICES

## I IDENTIFICATION TESTS

***Gram stain***

It was used to differentiate Gram-positive and Gram-negative organism. The former appears purple while the latter appears pink. The steps were.

1. Fixing the dried smear by passing over a flame three times.
2. Covering the fixed smear with crystal violet for 30-60 seconds.
3. Rapidly washing off the stain with clean water.
4. Tipping off all the water and covering the smear with grams iodine.
5. Washing off the iodine with clean water
6. Decolourising rapidly (few seconds) with acetone alcohol, then washing immediately with clean water.
7. Covering the smear with neutral red stain for two minutes.
8. Washing off the stain with clean water
9. Wiping the back of the slide clear and placing in a draining rack for smear to air dry.
10. Examining the smear microscopically first with 40x objective to check the staining and to see the distribution of materials and then in oil immersion objective to look for bacteria and cells.

***Indole test***

*Method*

i. Using a sterile straight wire, 5ml of sterile medium was inoculated with test organism.

ii. An indole paper strip was placed in the neck of the tube and stopper put. Incubation was done at 35-37$^0$ C overnight.

iii. Indole production was exhibited by reddening of the lower part of the strip.

*Motility*

Spreading of turbidity throughout the medium was a positive proof.

***Catalase tests:***

It was used to differentiate those bacteria that produce enzyme catalase. *Staphylococci* are catalase positive and *Streptococci* catalase negative. Method entailed the following steps:

i. A drop of hydrogen peroxide solutions was put on a glass slide.

ii. Using a sterile wooden stick or glass rod a good growth of test organism was placed in the hydrogen peroxide.

iii. Immediate bubbling denoted a positive result.

***Coagulase test***

The test was used to differentiate *staphylococcus aureus*, which produces, coagulase from *Staphylococcus epidermidis* and *Staphylococcus saprophyticus*, which do not produce coagulase. Both slide and tube tests were employed.

*Slide test*

It was used to test for free coagulase.

*Method*

i. A suspension of organism was made by emulsifying a colony of the organism in normal saline.

ii A drop of plasma was added to one of the suspensions and mixed gently.

Clumping of the organisms occurred within 10 seconds if the organism was *Staphylococcus aureus*.

*Tube test*

It was used to test for bound coagulase and to validate slide negative specimens.

*Method:*

i. The plasma was diluted by 1:10 in physiological saline.

ii. Three small test tubes labelled test, positive and negative control were availed.

iii. 0.5ml of the diluted plasma was pipetted into each tube.

iv. Five drops (about 0.1ml) of the test organism were added to the labelled tube. 5 drops of the *Staphylococcus aureus* culture were added to the tube labelled positive and 5 drops of sterile the tube labelled negative.

*v.* After mixing gently, the three tubes were incubated at 35-37$^0$ C. Clotting was examined after one hour. If no clotting occurred, examination was done at 30 minutes intervals for up to 6hrs. Clotting denoted *staphylococcus aureus.*

***Oxidase test***

It was used to identify *Pseudomonas* spp., from other Gram-negative organisms.

*Method*

i. A piece of filter paper was placed in a clean Petri dish and 2 – 3 drops of freshly prepared oxidase reagent added.

ii. Using a piece of stick, a colony of the test organism was smeared on the filter paper.

iii. Development of a blue purple colour within a few seconds indicated positive result

*Pseudomonas* spp., is oxidase positive.

***Voges-proskeur (v-p) test***

This test was used to identify *Klebsiella* spp.

*Method:-*

i. 2ml of sterile glucose phosphate peptone water was inoculated with test organism and incubated at 35-37$^0$ C for 48hrs.

ii. A small amount of creatinine was added and mixed well.

iii. 3 ml of sodium hydroxide reagent was added and well shaken.

iv. The bottle cap was removed and left for one hour at room temperature.

The development of pink red colour indicated *Klebsiella pneumoniae.*

***Urease test:***

It was used to identify *Proteus* spp., since they are strong urease producers.

*Method*

Using a straight wire, a tube of MIU was inoculated with a colony of test organism and

i. An indole paper strip was placed in the neck of the tube above the medium. The tube was stoppered and incubated at 35 – 37 $^0$ C overnight

ii. The production of urease changed the colour of paper strip to pink

***Bacitracin test***

It was used to identify *Streptococcus pyogenes.*

*Method*

i. Bacitracin disk was placed on a culture plate inoculated with the organism and incubated at 35-37$^o$ overnight.

ii. A zone of growth inhibition around the disc signified *Streptococcus pyogenes.*

***Esculin test:***

It was used to identify *Streptococcus faecalis.*

*Method*

A suspected organism was inoculated into a solution of esculin reagent in a tube and incubated over night at 35-37°C.

Black coloration of the solution after the incubation depicted presence of *Streptococcus faecalis.*

## II MIC INTERPRETIVE STANDARDS (ug/ml) FOR BACTERIA

| ANTIMICROBIAL AGENT | ORGANISM | DISC CONTENT IN µg | ZONE DIAMETER TO NEAREST MM | | |
|---|---|---|---|---|---|
| Ampicillin and other penicillins | | | R | I | S |
| | *Staphylococcus* spp. | 10 | ≤28 | - | ≥29 |
| | *Enterococcus* | 10 | ≤ 16 | - | ≥17 |
| | *Streptococcus* spp. | 10 | - | - | ≥24 |
| | *Enterobacteriaceae* | 10 | ≤13 | 14-16 | ≥17 |
| Amoxicillin-clavulonic acid | *Enterobacteriaceae* | 20/10 | ≤13 | 14-17 | ≥18 |
| | *Staphylococcus* spp. | 20/10 | ≤19 | - | ≥20 |
| | *Enterococcus* | 20/10 | ≤16 | - | ≥17 |
| | *Streptococcus* spp. | 20/10 | ≤13 | 14-16 | ≥17 |
| Piperacillin | *Enterobacteriaceae* | 100 | ≤19 | 20-22 | ≥23 |
| Cefuroxime | *Enterobacteriaceae* | 30 | ≤14 | 15-17 | ≥18 |
| | *Staphylococcus* spp. | 30 | ≤14 | 15-17 | ≥18 |
| | *Streptococcus* spp. | 30 | - | - | ≥24 |
| Ceftriaxone | *Enterobacteriaceae* | 30 | ≤13 | 14-20 | ≥21 |
| | *Staphylococcus* spp. | 30 | ≤13 | 15-17 | ≥18 |
| | *Streptococcus* spp. | 30 | ≤13 | 25-26 | ≥27 |
| Gentamycin | *Enterobacteriaceae* | 10 | ≤12 | 13-14 | ≥15 |
| | *Staphylococcus* spp. | 10 | ≤12 | 13-14 | ≥15 |
| | *Enterococcus* spp. | 120 | 6 | 7-9 | ≥10 |
| Amikacin | *Enterobacteriaceae* | 30 | ≤14 | 15-16 | ≥17 |
| | *Staphylococcus* spp. | 30 | ≤14 | 15-16 | ≥17 |
| Erythromycin | *Staphylococcus* spp. | 15 | ≤13 | 14-22 | ≥23 |
| | *Streptococcus* spp. | 15 | ≤15 | 16-20 | ≥21 |
| Chloramphenicol | *Enterobacteriaceae* | 30 | ≤12 | 13-17 | ≥18 |
| | *Staphylococcus* spp. | 30 | ≤12 | 13-17 | ≥18 |
| | *Enterococcus* spp. | 30 | ≤12 | 13-17 | ≥18 |
| | *Streptococcus* spp. | 30 | ≤17 | 18-20 | ≥21 |
| Tetracycline | *Enterobacteriaceae* | 30 | ≤14 | 15-18 | ≥19 |
| | *Staphylococcus* spp. | 30 | ≤14 | 15-18 | ≥19 |
| | *Enterococcus* spp. | 30 | ≤14 | 15-18 | ≥19 |
| | *Streptococcus* spp. | 30 | ≤18 | 19-22 | ≥23 |
| Ciprofloxacin | *Enterobacteriaceae* | 5 | ≤15 | 16-20 | ≥21 |
| | *Staphylococcus* spp. | 5 | ≤15 | 16-20 | ≥21 |
| | *Enterococcus* spp. | 5 | ≤15 | 16-20 | ≥21 |
| Oxacillin,Cloxacillin, flucloxacillin | *Staphylococcus* spp. | 1 | ≤10 | 11-12 | ≥13 |

**Source;PSAT(2002)**

## III CONSENT FORM FOR SPECIMEN COLLECTION

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DR. Peter. N. Karimi, Department of Medical Microbiology, UON, Tel: 0722436019

**Introduction**

We are requesting you to accept a specimen to be taken for your wound. The specimen will subsequently be analysed in the laboratory to find out the cause of the infection and the drugs that can be used to manage it. The researcher sponsors the study.

**Purpose of the Research**

Studies have shown that several factors contribute to wound infections. The research will assess how some of the factors affect wound sepsis.

**Your Participation Is Voluntary**

This consent form gives information about the study. Once you understand and agree to take part, we will request you to sign your name or make your mark on this form. We will offer you a copy to keep if need be.

**Risk and /or Discomfort**

You may feel discomfort or pain when taking specimen from the wound. Beside that no significant risk is involved. The information you give will be kept private and confidential.

**Benefits**

You may get no benefit from the test carried out. However, the result obtained from the laboratory may be communicated to the staff members concerned with the management of your wound. As a result you may get treated more effectively. The results will benefit other people in future.

**Cost To You**

There is no cost to you for the laboratory analysis of the specimen.

**Confidentiality**

Effort will be made to keep your personal information confidential. However, disclosure may be made if required by law or hospital administration.

**Research Related Injury**

The possibility of sustaining injury is highly unlikely. If you're injured the researcher in consultation with hospital staff will effect remedial measures.

**Problem or Questions**

If you have any questions about the research study or you have any research related injury, you should contact DR. P.N Karimi at 0722436019.

If you have question about your right as a research participant, you should contact Prof K.M. Bhatt of Kenyatta National Hospital Ethics and Research committee at 2726300.

**Statement of Consent and Signatures**

I have read this form or had it read to me. I have discussed the information with those concerned. All my concerns have been answered. I comprehend that my decision to take part in the study is voluntary. By signing this form, I do not give up any rights that I have as a research participant.

Participant Name/ _____ Participant Signature _____ Date

_____

Thumb Print

Witness Name_____ Witness signature _____

Date_____

UNIVERSITY OF NAIROBI/KNH ETHICS AND RESEARCH COMMITTEE
HOSPITAL ROAD ALONG NGONG ROAD
P.O. BOX 2072300200
NAIROBI
TEL: 2726300 EXT 4354
CHAIRPERSON: PROFESSOR K.M BHATT

DEPARTMENT OF MEDICAL MICROBIOLOGY, UON
P.O. BOX 31967
NAIROBI
TEL: 2719628

Copy To

1) Investigators
2) Study Subjects

## IV DATA COLLECTION INSTRUMENT

CODE _____

### (A) QUESTIONNAIRE FOR PATIENTS

DATE_____

**Introduction Letter**

I am Dr. P.N. Karimi, a student at University of Nairobi, pursuing a Master of Science degree in Medical Microbiology. The purpose of this study is to assess the factors that affect wound infections at Kenyatta National Hospital. All the information you give will be treated confidentially and used for the purpose of study. Kindly oblige.

**SECTION A**

***Personal data***

1. Gender: (i) Male ☐ (ii) Female ☐

2. Age in years
   (i) 0 – 10 ☐
   (ii) 10 – 20 ☐
   (iii) 20 – 30 ☐
   (iv) 30 – 40 ☐
   (v) > 40 ☐

3. Ward of study
   i) 6A
   ii) 6B
   iii) 6C
   iv) 6D

**Section B**

1. Cause of the wound

☐

(i) Accident

(ii) Cellulitis ☐

(iii)Assault ☐

(iii) Others ☐

2. Duration of hospitalisation in weeks.

(i) 0 – 1 ☐

(ii) 1 – 2 ☐

(iii) 2 – 3 ☐

(iv) 3 – 4 ☐

(v) 5< ☐

3. Reason(s) for hospitalisation.

(i) Delayed procedures ☐

(ii) Delayed healing ☐

(iii) Lack of financial resources ☐

(iv) Other(s) specify ☐ ----------

## SECTION C

1. Number of antibiotics used.

a) One
b) Two
c) Three
d) Four

2. Antimicrobial agent used

| DRUG | DAILY DOSAGE | | DURATION | |
|---|---|---|---|---|
| | Correct | Incorrect | Indicated | Not indicated |
| Benzyl penicillin G | | | | |
| Flucloxacillin | | | | |
| Gentamycin | | | | |

| | | | | |
|---|---|---|---|---|
| **Ceftriaxone** | • | | | |
| **Ceftazidime** | | | | |
| **Cloxacillin** | | | | |
| **Cefuroxime** | | | | |
| **Antituberculous drugs** | | | | |
| **Augmentin** | | | | |
| **Chloramphenicol** | | | | |
| **Ciprofloxacin** | | | | |
| **Amoxicillin** | | | | |
| **Erythromycin** | | | | |

## SECTION D

**Prevalence of bacteria in the wound.**

(i) Type of organism isolated

1. *Pseudomonas* spp.
2. *Proteus* spp.
3. *Escherichia coli*
4. *Klebsiella* spp.
5. *Staphylococcus aureus*
6. *Streptococcus faecalis*
7. *Streptococcus pyogenes*
8. *Enterobacter* spp.
9. *Alcaligenes* spp.
10. *Serratia* spp.
11. *Citrobacter* spp.
12. *Acinetobacetr baumanii*
13. Others

(ii) Number of organisms isolated.

(a) One
(b) Two
(c) Three
(d) Four
(e) Five
(f) Six

**SECTION E**

**Antibacterial susceptibility**

(1) ***Proteus*** **spp.**

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

(2) ***Escherichia coli***

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

(3) ***Pseudomonas*** **spp.**

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Piperacillin** | | | |
| **Pipril/Tozabactam** | | | |
| **Meropenem** | | | |
| **Gentamycin** | | | |
| **Amikacin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |
| **Ticarcillin/clavulonic acid** | | | |

(4) ***Klebsiella*** **spp.**

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

(5) *Staphylococcus aureus*

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Amoxicillin** | | | |
| **Augmentin** | | | |
| **Oxacillin** | | | |
| **Erythromycin** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Cefuroxime** | | | |
| **Clindamycin** | | | |

(6) *Streptococcus pyogenes*

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Amoxicillin** | | | |
| **Cefuroxime** | | | |
| **Augmentin** | | | |
| **Erythromycin** | | | |
| **Chloramphenicol** | | | |
| **Ofloxacin** | | | |
| **Tetracycline** | | | |
| **Ceftriaxone** | | | |

(7) *Steptococcus faecalis*

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Vancomycin** | | | |
| **Amoxicillin** | | | |
| **Augmentin** | | | |
| **Ciprofloxacin** | | | |
| **Gentamycin** | | | |
| **Streptomycin** | | | |
| **Nitrofurantoin** | | | |
| **Nalidixic acid** | | | |

(8) **Enterobacter spp.**

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

(9) *Alcaligenes* spp.

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

(10)*Citrobacter freundii*

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

(11) *Serratia* spp.

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

(12) *Acinetobacter baumanii*

| ANTIBIOTIC | RESISTANT | INTERMEDIATE | SENSITIVE |
|---|---|---|---|
| **Augmentin** | | | |
| **Cefuroxime** | | | |
| **Gentamycin** | | | |
| **Ciprofloxacin** | | | |
| **Minocycline** | | | |
| **Piperacillin** | | | |
| **Ceftazidime** | | | |
| **Ceftriaxone** | | | |

## (B) ASSESSMENT FORM FOR STAFF MEMBERS

**Code** ☐

**Date** _____

1. Category of the staff member

   (i) Doctor ☐ (ii) Nurse ☐

2. Gender (i) Male ☐ (ii) Female ☐

3 Did the staff member wash the hands when dressing patient's wounds?

   (i) Yes ☐ (ii) No ☐

4. If the answer in (4) is yes, when were the hands washed?

   (i) Before dressing wound in every patient ☐

   (ii) After dressing the wound in some patients ☐

   (iii) After dressing all the wounds ☐

5. Is the staff member in gloves when attending patients with wounds in the ward?

   (i) Yes ☐ (ii) No ☐

6. If the answer is yes, how often were the gloves changed?

   (i) After attending every patient ☐

   (ii) At the end of the dressing session ☐

   (iii) When they became soiled ☐

7. Which method(s) is/are used to clean wounds?

(i) Irrigation with antiseptic solution ☐

(ii) Swabbing with dry gauze ☐

(iii) Swabbing with dry cotton wool ☐

(iv) Swabbing with gauze or cotton wool soaked in antiseptic solution. ☐

8. Is the staff member always in facemask when dressing wounds?

Yes-------- No----------

9. Who opened the wound?
   (i) Clinician
   (ii) Patient
   (iii) Both patient and clinician

(10) How often is the wound dressing done?

   (i) Daily
   (ii) On alternate days
   (iii) Any time

(11) Which antiseptic is used?
   (i) savlon
   (ii) Hydrogen peroxide
   (iii) Betadine
   (iv) Normal saline
   (v) Rifocin

(12) Did the nurse use a forceps to hold the gauze during dressing?
Yes---------- No----------

(13) Was the forceps sterile?
Yes---------- No----------

**4.0 Work plan**

| Task | Mar | Apri | May | June | July | Aug | Sep | Oct |
|---|---|---|---|---|---|---|---|---|
| Proposal writing | xxx | | | | | | | |
| Proposal defence and approval | | xxx | xxx | | | | | |
| Pilot study | | | | xxx | | | | |
| Data collection | | | | | xxx | xxx | | |
| Data Analysis and report writing | | | | | | | xxx | |
| Defense of Thesis | | | | | | | | xxx |

**Budget**

| Category | No of units | Cost/Unit | Total Cost (Ksh) |
|---|---|---|---|
| *Services* | | | |
| Research Assistants | 3 | 6000 | 18000 |
| Secretarial and Administration | 1 | 5000 | 5000 |
| Photocopy | | 4000 | 4000 |
| Internet Search | | 1000 | 1000 |
| Printing | | 3000 | 3000 |
| Data Analysis | | 10000 | 10000 |
| *Operating Expenses* | | | |
| Printing papers | 6 | 300 | 1800 |
| Ink Cartridge | 1 | 6000 | 6000 |
| Pens | 1 Box | 500 | 500 |
| Pencil | 1 Box | 400 | 400 |
| Other Stationary | | 2000 | 2000 |
| Binding | | 3000 | 3000 |
| *Reagents/Apparatus* | | | |
| Blood Agar culture media | 500gm | 6000 | 6000 |
| MacConkey culture media | 500gm | 5000 | 5000 |
| Muller Hinton | 500gm | 7000 | 7000 |
| Sterile cotton swabs with Amies Transport Media | 120 | 50 | 6000 |
| Glass slides | 3Pkt | 150 | 450 |
| Petri dishes- Disposable | 120 | 50 | 6000 |
| Sensitivity Discs | 4 Pkt | 5000 | 20000 |
| Miscellaneous | | | 20,000 |
| Contingency (15%) | | | 18,772.5 |
| Total | | | 143,922.5 |