A DISSERTATION ON COMPARATIVE STUDY OF SINGLE DOSE PROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POSTOPERATIVE ANTIBIOTICS IN PREVENTION OF SSI

A PROSPECTIVE STUDY

Dissertation Submitted to

THE TAMIL NADU

DR. M.G.R. MEDICAL UNIVERSITY CHENNAI

In partial fulfillment of the regulations for

the award of the degree of

M.S. GENERAL SURGERY (Branch 1)

CHENGALPATTU MEDICAL COLLEGE

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

APRIL 2016

DECLARATION

I hereby declare that this dissertation entitled **"COMPARATIVE STUDY OF SINGLE DOSE PROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POSTOPERATIVE ANTIBIOTCS IN PREVENTION OF SSI"** was prepared by me under the direct guidance and supervision of **Prof. DR.S. SURESH M.S.,** CHENGALPATTU MEDICAL COLLEGE. The dissertation is submitted to the Dr. M.G.R. Medical University in partial fulfillment of the University regulations for the award of MS degree in General Surgery, Examination to be held in April 2016.

Place: Chengalpattu **Dr. K. Alex Franklin**

Date:

CERTIFICATE

This is to certify that **Dr.K. Alex Franklin** post graduate student (2013-2016) in the department of General surgery, CHENGALPATTU MEDICAL COLLEGE has done this dissertation titled **"COMPARATIVE STUDY OF SINGLE DOSE PROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POSTOPERATIVE ANTIBIOTCS IN PREVENTION OF SSI"** under the direct guidance and supervision of guide Prof .DR.S.SURESH M.S., in partial fulfillment of the regulations laid down by the **Tamilnadu Dr.M.G.R. Medical University**, Chennai, for M.S., General Surgery Degree Examination.

Prof. DR.K. MUTHURAJ M.S.,
Professor & HOD
Dept. of General Surgery
Chengalpattu Medical College

Prof. DR. K.MUTHURAJ M.S
DEAN
Chengalpattu Medical College

BONAFIED CERTIFICATE BY GUIDE

This is to certify that **Dr. K.Alex Franklin** post graduate student (2013-2016) in the department of General surgery, CHENGALPATTU MEDICAL COLLEGE has done this dissertation titled **"COMPARATIVE STUDY OF SINGLE DOSE PROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POSTOPERATIVE ANTIBIOTCS IN PREVENTION OF SSI"** under the direct guidance and supervision of guide Prof .DR.S.SURESH M.S., in partial fulfillment of the regulations laid down by the **Tamilnadu Dr.M.G.R. Medical University**, Chennai, for M.S., General Surgery Degree Examination.

Place : Chengalpattu

Date :

Dr. S. SURESH M.S.,

Professor of Surgery,

Department of General Surgery,

Chengalpattu Medical College.

ACKNOWLEDGEMENT

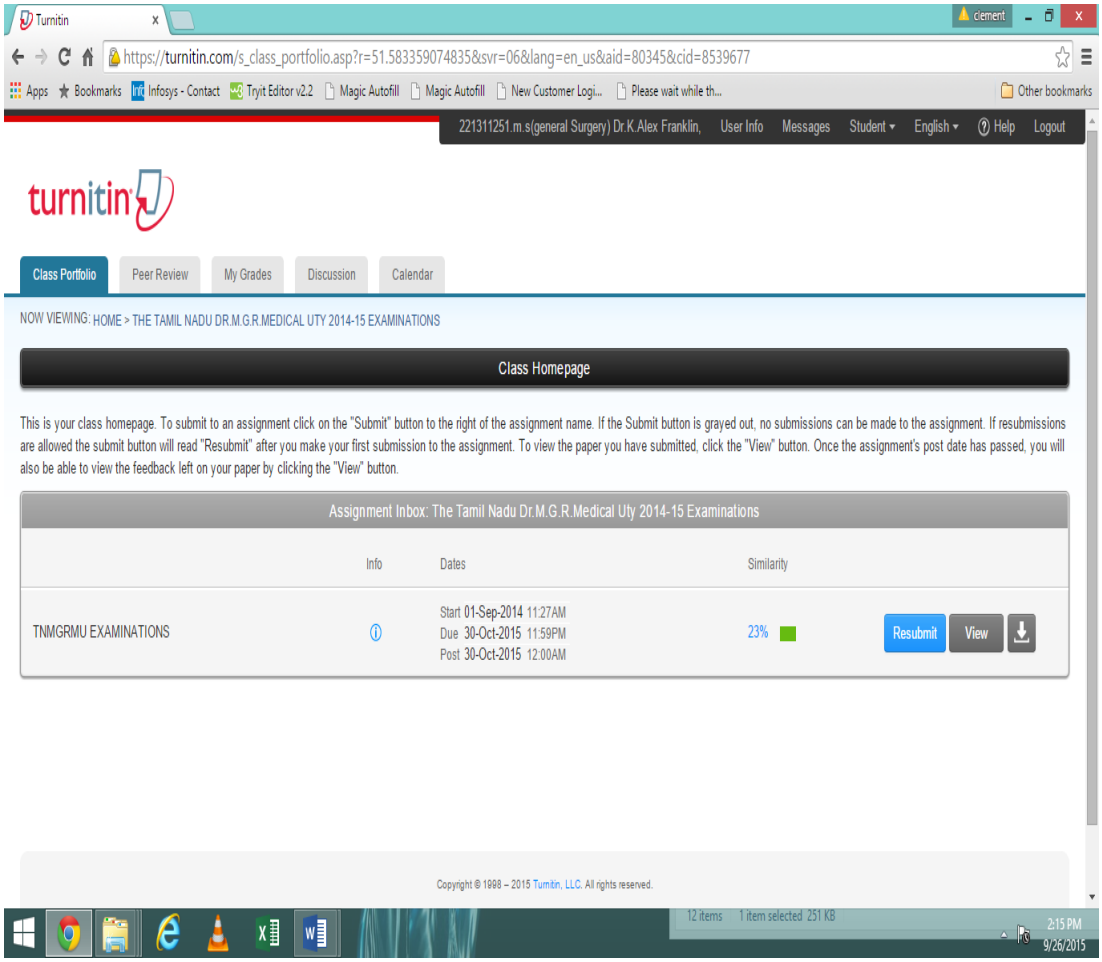
At the outset, it is with a sense of accomplishment and deep gratitude that I dedicate this dissertation to all those who have been instrumental in its completion.

First and foremost I express my heartful thanks to my esteemed and respected HOD Department of General Surgery, CHENGALPATTU MEDICAL COLLEGE Prof. Dr. K. Muthuraj M.S., and my guide Prof.Dr.S.Suresh M.S., Had it not been for his whole hearted support throughout the period of this study, extending from his vast knowledge, invaluable advice and constant motivation, I truly would not have been able to complete this dissertation topic in its present form.

I sincerely thank my Professors, Dr. C.Srinivasan M.S., and Dr.T. Ragupathy M.S.,. I am greatly indebted to my Assistant Professors Dr. P. Sankarlingam M.S., and Dr. M. Sabrena M.S., for giving me practical suggestions and permitting me to carry out this study in their patients.

I dedicate this work to my parents and my wife Dr. R. Mary Stella for her constant encouragement and support. I am deeply indebted to all the teaching staff and my fellow postgraduates for their helpful attitude and valuable suggestions in every stage of my study.

Lastly, I thank the ethics committee for permitting me to do this study and more importantly I thank all my patients involved for their kind help and co operation.


Turnitin
https://turnitin.com/s_class_portfolio.asp?r=51.583359074835&svr=06&lang=en_us&aid=80345&cid=8539677
221311251.m.s(general Surgery) Dr.K.Alex Franklin,
User Info
Messages
Student
English
Help
Logout
turnitin
Class Portfolio
Peer Review
My Grades
Discussion
Calendar
NOW VIEWING: HOME > THE TAMIL NADU DR.M.G.R.MEDICAL UTY 2014-15 EXAMINATIONS
Class Homepage
This is your class homepage. To submit to an assignment click on the "Submit" button to the right of the assignment name. If the Submit button is grayed out, no submissions can be made to the assignment. If resubmissions are allowed the submit button will read "Resubmit" after you make your first submission to the assignment. To view the paper you have submitted, click the "View" button. Once the assignment's post date has passed, you will also be able to view the feedback left on your paper by clicking the "View" button.
Assignment Inbox: The Tamil Nadu Dr.M.G.R.Medical Uty 2014-15 Examinations
Info
Dates
Similarity
TNMGRMU EXAMINATIONS
Start 01-Sep-2014 11:27AM
Due 30-Oct-2015 11:59PM
Post 30-Oct-2015 12:00AM
23%
Resubmit
View
Copyright © 1998 – 2015 Turnitin, LLC. All rights reserved.
12 items
1 item selected 251 KB
2:15 PM
9/26/2015

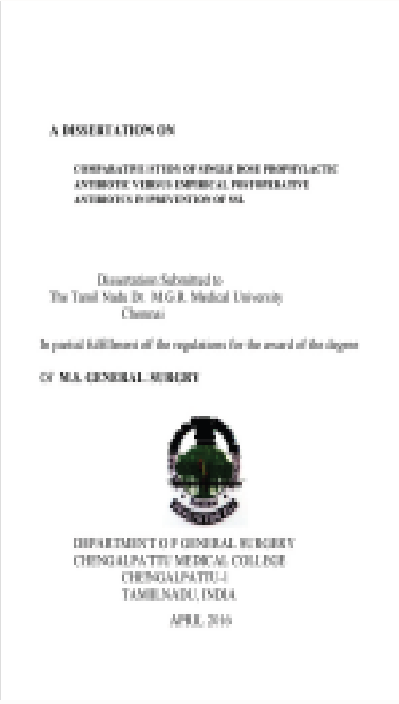
turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	221311251.m.s(general Surgery) D...
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	COMPARATIVE STUDY OF SINGLE..
File name:	SUS_EMPIRICAL_POSTOPERATIV...
File size:	199K
Page count:	110
Word count:	20,775
Character count:	112,175
Submission date:	23-Sep-2015 12:48AM
Submission ID:	572587908



A DISSERTATION ON

COMPARATIVE STUDY OF SINGLE DOSE PROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POSTOPERATIVE ANTIBIOTICS IN PREVENTION OF SSI

Dissertation Submitted to
The Tamil Nadu Dr. M.G.R. Medical University
Chennai

In partial fulfillment of the regulations for the award of the degree

OF M.S. GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY
CHENGALPATTU MEDICAL COLLEGE
CHENGALPATTU
TAMILNADU, INDIA
APRIL 2016

INSTITUTIONAL ETHICS COMMITTEE

CHENGALPATTU MEDICAL COLLEGE , CHENGALPATTU

APPROVAL OF ETHICAL COMMITTEE

To

Alex Franklin K,
2nd Year PG student (General Surgery),
Chengalpattu Medical College,
Chengalpattu

Dear Dr.

The Institutional Ethical Committee of Chengalpattu Medical College reviewed and discussed your application to conduct the clinical / dissertation work entitled

COMPARATIVE STUDY OF SINGLE DOSE PROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POSTOPERATIVE ANTIBIOTICS IN PREVENTION OF SSI..

ON 19.02.2015

The following documents reviewed

1. Trial protocol, dated _____version no
2. Patient information sheet and informed consent form in English and / or vernacular language.
3. Investigators Brochure, dated _____version
4. Principal Investigators current CV
5. Investigators undertaking

The following members of the Ethics committee were present at the meeting held on

Date 19.02.2015 Time 11.00 am Place Chengalpattu Medical College

Approved James Roni. Chairman Ethics Committee

19/2/15 Member secretary of Ethics Committee.

Name of each member with designation:-

Clinical Members

1. Dr.K.Srinivasagalu MD.,
   Prof & HOD of Medicine, CHMC

2. Dr.C.Srinivasan MS.,
   Prof & HOD of Surgery, CHMC

Biological Scientist

3. Dr.K.Baskaran MD.,
   Asso Prof of Pharmacology, CHMC

Non Clinical Member

4. Dr.P.Parasakthi MD
   Prof & HOD of Forensic Medicine,CHMC

5. Member from Nongovernmental
   Voluntary Organisation : Mr.P.Durairaj

6. Philosopher : Mr.K.S.Ramprasad

7. Lawyer : Lr. I. M. Karimala Basha

8. Layperson : Mr.Dilli

We approve the clinical trial to be conducted in its presented form

The Institutional Ethics Committee expects to be informed about the progress of the study and any SAE occurring in the course of the study, any changes in protocol and patient information / informed consent and asks to provide copy of final report.

Yours sincerely

19/2/15

Member secretary, Ethics Committee

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF ABBREVATIONS USED:

BT	**:**	Bleeding Time
CDC	**:**	Center for Disease Control
CMV	**:**	Cytomegalovirus
CNS	**:**	Central Nervous System
CO_2	**:**	Carbondioxide
CSF	**:**	Cerebro Spinal Fluid
CT	**:**	Clotting Time
CVS	**:**	Cardiovascular System
CXR	**:**	Chest X-ray
DC	**:**	Differential Count
DM	**:**	Diabetes Mellitus
DNA	**:**	Deoxyribonucleic Acid
ESR	**:**	Erythrocyte Sedimentation Rate
FBS	**:**	Fasting Blood Sugar
Hb	**:**	Hemoglobin
HIV	**:**	Human Immuno Deficiency Virus
Ht	**:**	Hypertension
IV	**:**	Intra Venous
LFT	**:**	Liver Function Test
MRSA	**:**	Methicillin Resistance Staphylococcus Aureus
PA	**:**	Per Abdomen

RBS	**:**	Random Blood Sugar
RR	**:**	Respiratory Rate
RNA	**:**	Ribonucleic Acid
RS	**:**	Respiratory System
SPO_2	**:**	Partial Pressure of Oxygen
SSI	**:**	Surgical Site Infection
TC	**:**	Total Count
URTI	**:**	Upper Respiratory Tract Infection
USG	**:**	Ultrasound
UTI	**:**	Urinary Tract Infection

LIST OF FIGURES

ABSTRACT

TITLE: A DISSERTATION ON COMPARATIVE STUDY OF SINGLE DOSPROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POST OPERATIVE ANTIBIOTICS IN PREVENTION OF SSI

AIM OF THE STUDY

To compare the efficacy of single dose antibiotic prophylaxis versus empirical post operative prophylaxis in prevention of SSI.

MATERIALS AND METHODS:

This study includes 100 clean and clean contaminated cases randomized to groups of 50 each. The study group will receive a single dose of antibiotic preoperatively while the control group will receive 3 to 5 days of empirical antibiotic therapy.

All the clean class 1 cases in the study group were given a single dose of 1gm of inj. Ceftriaxone at the time of induction or 30 minutes before skin incision in case the procedure is prolonged for more than 3 hrs a second dose was given.

They received no further antibiotics i.v or oral. All the cases in the control group received 5 days of inj. Cefotaxime 1Gm iv BD for 5 days. The incidence of SSI was noted and analysed.

All the class 2 cases in study group received inj. Ceftriaxone 1gm and inj. Metronidazole 500 mg iv 30 minutes before the skin incision. In case the procedure was extended beyond 3 hrs a second dose was given. They received no further antibiotics i.v or oral. All the cases in the control group received inj. Cefotaxime 1gm iv BD along with

inj. Metronidazole 500 mg i.v TDS for 5 days. In case of underweight or obese patients the dose was adjusted according to their body weight.

All the cases were followed up at 8th POD,15th POD, 30th POD and later at 3 months and 6 months. Any wound related complications noted and data obtained. The incidence of SSI in both the groups was calculated and results analysed.

CONCLUSION:

Based on my study I would like to conclude that it is recommendable to use single dose antibiotic prophylaxis using appropriate antibiotics for all class I and Class II cases, as per the study results there is no significant difference in incidence of SSI when compared to the traditional regimes with the added advantage of significant reduction in hospital stay, with its resultant savings in resources. In addition as the use of antibiotics is reduced it further results in increased cost effectiveness and reduces the incidence of complications due to antibiotic overuse.

INTRODUCTION

Surgical site infections (SSI) are one of the most common causes of postoperative morbidity. The introduction of antibiotics in 20th century led to great improvement in surgical outcomes. From being a dreaded event surgeries have become an accepted part of modern day life due to the advent of antiseptic techniques and more importantly the advent of antibiotics.

But with rampant antibiotic use came its own set of problems like the rise in incidence of antibiotic resistant strains (MRSA) and rise in incidence of allergies and other complications of antibiotic use. The recent incidence of mass casualty due to tainted antibiotic in a mass sterilization camp being one of them.

This leads one to reexamine our current postoperative antibiotic use. Compared to western countries where the stress has shifted to improved asceptic precautions and better techniques there by shifting the focus away from post operative antibiotics, but in our set up we are still stuck in prolonged post operative antibiotic regimes. This is perhaps due to lack of significant clinical trials especially in the government set up showing the efficacy of antibiotic prophylaxis alone in preventing incidence of SSI's. This study aims to fill that lacunae and there by aid our gradual shift away from over reliance on antibiotics in prevention of SSI.

In modern surgical care antibiotics are known to account for about 20% of total expenses during hospitalization. In our country where the proportion of health budget to GDP is one of the lowest in the world, the amount of savings that can be obtained by reducing our over reliance on antibiotics will be enormous.

AIM OF THE STUDY

To compare the efficacy of single dose antibiotic prophylaxis versus empirical post operative prophylaxis in prevention of SSI.

REVIEW OF LITERATURE:

HISTORICAL CONTEXT:

The knowledge of infections and wound management have been known to man since time immemorial. The knowledge that the ancients learned has been recorded in their ancient writings. Egyptian Papyruses mention about use of salves and ointments in management of wounds.

In the bible references to wound care in the form of drainage of pus and application of local salves like vinegar and wine have been recorded in both the old and new testament. Among the Greeks Hippocrates taught the use of wine and vinegar in wound irrigation.

Later Roman surgeon Galen recognized that the localization of pus in wounds followed by drainage signaled the beginning of wound healing leading to his famous saying. *Pus bonum et laudible.* Sadly this was misunderstood in the dark ages of Europe and harmful practices like inoculation of feces in wounds to promote pus formation had developed.

Advances in wound healing in Europe had to wait until renaissance before rational thought and keen observation by figure like Ambroise Pare, Theodoric of cervia and others for the reintroduction of antisepsis and abandonment of harmful practices like cauterization and blood letting.

But medicine had to wait till 19th century for Ignac Semmelweis who showed that the simple practice of washing hands before conducting deliveries.

He was able to reduce puerperal sepsis from above 10 percent to less than 2 percent. Later Louis Pasteur with his germ theory and Anton Von Leeuwenhoek with his microscope Led to increase in knowledge about microbes and their role in wound infection.

Later Joseph Lister applied this knowledge and introduced the concept of antiseptic technique by introducing the concept of washing hands, instruments and sutures in carbolic acid. Later the discovery of steam sterilization by Earnrst von Bergmann in 1907 and its subsequent adoption all over the world led to improvement in surgical outcomes. Later the introduction of gloves and newer techniques by Halsted led to further improvement. But it was the discovery of antibiotics especially penicillin by Sir Alexander Fleming in 1928 and its subsequent use that surgery became truly modern and safe.

With the advent of modern anesthesia and newer antibiotics we are now performing surgeries that could only be dreamt of in the past. The days of certain death following fecal peritonitis are now past but challenges still remain. While the role of antibiotic prophylaxis in contaminated cases is accepted but the role of prolonged antibiotics in clean surgeries remain controversial.

Definition and classification of Surgical Site Infection

Surgical site infections are defined as infections of the tissues, organs and spaces exposed by surgeons during the performance of an invasive procedure. SSI can be classified into incisional and organ / space infection. Incisional SSI is further divided into deep and superficial categories.

Even though study of Microbial density in tissue can be performed but for practical purposes the decision on starting antibiotics and the intended agents are decided based on clinical classification. The classification gives an idea of bacterial contamination during the procedure.

To be classified as Superficial incisional wound infection itmust meet the following criteria. Infection must occur at the incision site within 30 days after surgery and must involvethe skin or subcutaneous tissue abovefascial layers and any one of the following.[13]

1. There is purulent drainage from the incision or collection located above the fascial plane.
2. If the wound is opened deliberately by the surgeon, unless the exudate is culture negative.
3. If Organism is isolated from the culture of fluid aseptically obtained from the wound closed primarily.

To be classified as Deep surgical wound infection it must meet the following criteria. Infection must occurs at the operative site within 30 days after the surgery if no prosthesis was placed or within 6 months to l year if an implant had been placed, and the infection involves tissues or spaces at or beneath the fascial plane along with any of the following

1. The wound dehisces spontaneously or is opened deliberately by the surgeon when patient has persistent fever (>38^{o} c) and or localized tenderness or pain, unless the wound is culture negative.
2. Evidence of an abscess or infection directly under the incision is seen on

direct examination, during surgery or on histopathological examination.

3. The Surgeon declares infection.

Surgical wounds are classified based on the assumed magnitude of bacterial load at the time of operation as clean, clean contaminated, contaminated and dirty. [14]

CLASSIFICATION [15]:

Class I / Clean: This catergory **i**ncludes those surgeries in which no contamination is present; only microflora from skin potentially contaminates the wound. An uninfected surgical wound in which no inflammation is encountered and the respiratory, alimentary, genital, or uninfected urinary tract is not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Class ID wounds are similar except that a prosthetic device (eg. Mesh or valve) is inserted.

Class II/Clean-Contaminated: It includes those surgeries in which a hollow viscus with indigenous bacterial flora is opened under controlled circumstances without significant spillage of the contents. A surgical wound in which the respiratory, alimentary, genital or urinary tracts are entered under controlled conditions and without unusual contamination, Specifically operations involving the billiary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in technique is encountered.

Class III/Contaminated: Include those open accidental wounds encountered early after injury, those with extensive introduction of bacteria into a normally

sterile area of the body.

Open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (e.g., open cardiac massage) or gross spillage from the gastrointestinal tract, and incisions in which acute, nonpurulent inflammation is encountered are included in this category.

Class IV/Dirty-Infected: Include traumatic wounds in which a significant delay in treatment has occurred and in which necrotic tissue is present, those created in presence of overt infection.

Old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation.

WOUND HEALING

Any injury surgical or otherwise starts a predictable sequence of events that follows a set pathway resulting in wound healing. Those phases are inflammatory phase, the fibroplastic phase and remodeling phase.

INFLAMMATORY PHASE

This phase prepares the area for healing and immobilizes the wound by causing the wound to swell and become painful, resulting in restriction of movement.

Inflammation is a normal and needed prerequisite to healing. Changes in vascular flow cause the clinical symptoms that are used to detect an

inflammatory response. The vast majority of specialized cells involved in this phase of healing process come from blood.[17]

Blood vessels that had traversed the wound before injury are severed at the time of injury and it results in exudation of whole blood into the wound, which on coagulation, seals the damaged vessels and lymphatic channels that drain the area in order to close the wound, and it prevents further bleeding. The release of histamine and other vasodilator agents by the injured tissues cause the undamaged vessels to dilate. Histamine causes short acting vasodilatation in nearby intact vessels and it is this combination of whole blood exudate from damaged blood vessels and serous transudate that creates a red, hot, swollen and painful local environment. Bradykinins, that are derived from plasma in the area of the injury, contribute to more prolonged vascular permeability. Certain types of prostaglandins further contribute to long-term vasodilatation. The fibrin plugs that clot in the wound also form in the lymphatic vessels. Blocking the lymphatic flow seals the wound and also helps to stop the spread of infection. The lymphatics remain closed until later in the healing process.[18]

The Mast cells also release hyaluronic acid and other proteoglycans into the mix of chemicals accumulating in the wound and these bind with the wound fluid to create a non-flowing gel that gradually slows down leakage and fluid loss. The inflammatory oedema that is formed as a result fills up the spaces in the wound and surrounds the damaged structures and binds them together.

The soluble fibrinogen circulates in the blood and provides the material to form the insoluble fibrin clot during blood coagulation. Fibrinogen responds to infection and other short-term inflammatory stressors as it is an active phase

reactant.

Secondary wound care is needed to address the changes made by induced vasodilatation, which varies in relation to the severity of the wound. This transudate can be diminished by following a regimen of rest, ice, compression and elevation. For healing to commence, the wound needs to be decontaminated by phagocytosis, and neovascularisation must then progress.

Phagocytosis

Within blood vessels adjacent to the wound, WBC's start to adhere to the capillary walls. Chemical changes in the wound attract these cells and induce them to slip through the enlarged capillary pores following which they migrate to the site of the injury. The main target of this phase is to prevent infection and to rid the wound of infective agents.

The first WBC's to reach the wound are polymorphonuclear leucocytes. These cells begin the process of phagocytosis by attaching to bacteria and phagocytosing them.

Within a few days following the injury, another type of phagocyte will predominate in the wound until all the signs of inflammation cease. This cell, the macrophage, plays two important roles in the repair process. Macrophages originate from specific WBC's called monocytes. Their role is to phagocytose the pathogens, either as stationary or mobile cells, and to stimulate other immune cells especially lymphocytes to respond to the triggering pathogen. As a scavenger cell, the macrophage not only phagocytoses bacteria but also disposes of necrotic tissue in the wound. Soon, fibroblasts responding to the chemical signals issued by macrophages provide a structural framework for

many tissues, playing a critical role in wound healing. They responding to the stimulation secrete the precursors of all the components of the extracellular matrix, predominantly the ground substance and a variety of fibers.[19]

The macrophage influences repair by chemically recruiting the fibroblastic repair cells. It is the local platelet-derived growth factor that is released from the platelets during clotting and from macrophages that signals the fibroblasts.

Neovascularisation is growth of new blood vessels, as healing will not proceed without new functioning blood vessels to supply oxygen and nourishment to the injured tissue. It is likely that it is the macrophages that signal this vascular regeneration to start. Patent blood vessels in the wound area develop small ‘sprouts’ that grow into the wound area and it is from these buds that eventually come into contact and anastamose with other arteriolar or venous buds to form a functioning capillary loop. The capillary sprouts, during the early stages, lack full thickness walls, which renders them delicate and easily disrupted, this neccesitates immobilization during this phase to permit the vascular regrowth and prevent the formation of microhaemorrhages. At the conclusion of this phase, fibrinolysin in blood vessels is formed to assist in dissolving clots and the lymphatic channels open in to assist in resolving the wound oedema.

Under ideal conditions, these events all happen within the first four days after injury and the main objective of treatment is to minimize all the factors that can prevent or prolong inflammation.

FIBROPLASTIC PHASE

When the inflammatory phase is completed, rebuilding can commence. This phase is named after the primary cell that aides scar production, the fibroblast. Many different cells are also involved in the inflammatory phase, but fewer cell types operate during the fibroplastic phase, which lasts for about three weeks. During this phase the wound is resurfaced resulting in strength being imparted to the wound. The fibroblasts originate from the mesenchymal cells located in connective tissue around blood vessels and fat. In response to chemotactic signals, fibroblast precursors transform into cells with migratory ability.

These fibroblasts follow the fibrin meshwork created earlier in the wound fluid environment, which enveloped all injured structures, and thus they have access to all depths of the wound. Once in place, the fibroblasts initiates synthesis of the collagen molecule. During this phase, three processes occur concurrently to achieve coalescence and closure. These processes are epithelialisation, wound contraction and collagen production.

Epithelialisation

The factors important to tissue survival are phagocytosis, blood flow and the provision of a surface covering. These events happen early in the healing process. The provision of even a single cell layer will provide protection from the invading organisms. Within hours of injury, the undamaged epithelial cells at the wound margin start to reproduce. This epithelial mitosis causes accelerated reproduction and leads to formation of a ridge around the periphery

of the wound. These new cells are true epithelial cells and therefore this multiplication represents a regeneration process. The surviving epidermal structures like hair shafts and sweat glands also give rise to epithelial mitoses. Once the wound bed is viable and a decent blood supply is available, the migration of these new cells begins, with those coming from the periphery moving in and those from appendages moving out. These migratory cells continue to remain attached to the parent cells and this movement causes tension on the normal skin around the wound edge. The advancing edge of the epithelium seeks out oxygen-rich tissue, moist tissue.

If the epithelial edge meets eschar, foreign material like sutures or blood clots, it then plunges under it to maintain contact with the vascular loop network in the wound. If the necrotic tissue or the wound is too extensive or if oxygen availability is poor, then epithelial migration cannot proceed. If sufficient capillary circulation does not exist to maintain epithelial integrity then wound dehiscence can occur.

Dehiscence is the phenomenon of premature bursting or splitting of a wound along natural or surgical suture lines. This is a complication of surgery that occurs secondary to poor wound healing.

When epithelial cells migrating from one direction meet similar migratory cells from another direction, contact inhibition results in cessation of movement. Although clean, approximated wounds are clinically resurfaced within 48 hours, larger open wounds require prolonged period of repair. Several weeks are required for this thin initial covering to become multilayered and to differentiate into various strata of the normal epidermis.

Wound Contraction

Epithelialisation closes the wound surface, but it is contraction that pulls the entire wound together, thereby shrinking the defect. A successful contraction results in a smaller wound that can be repaired by scar formation. Minimizing the area to be healed is highly beneficial in certain tissues with fixed, deep structures that are covered by mobile, loose skin.

Wound contraction may be harmful in those areas that require every millimeter of skin and tissue length, like the hands and face. Allowing uncontrolled contraction here is a potential problem as it will distort the topography of the skin and cause the tissues to be drawn abnormally towards the site of healing, causing disfigurement and discomfort.

A specialized cell called the myofibroblast is involved in the contraction process. In terms of differentiation, myofibroblast lies between a fibroblast and a smooth muscle cell. Myofibroblasts attach to the skin margins, pull the entire epidermal layer inward, and are a feature in the hypertrophic scars. Control of wound contraction and scar formation at the time of wound formation is useful in order to control the direction of wound contraction and thus prevent the distortion.

Collagen Production

The conclusion of wound healing process is collagen production, which is essential if the wound healing is to occur. Migratory fibroblasts are now present throughout the wound and this stimulates the fibroblasts to synthesize and secrete collagen. The buildup of lactic acid influences the amount of collagen produced. Adequate supplies of oxygen, ascorbic acid and other

cofactors such as zinc, iron and copper are needed to help create the proper background for fibroplasia, which is the production of fibrous tissue, usually implying an abnormal increase of non-neoplastic fibrous tissue.[20]

The fibroblast synthesizes three polypeptide chains that coil to form a right-handed helix. These spiraled chains (procollagen) are then extruded from the fibroblast into extracellular space. Once extruded, the triple-helical molecule undergoes cleavage at specific terminal sites to become tropocollagen. Tropocollagens then associate spontaneously in an overlapping array to eventually convolve with other tropocollagen molecules to form a collagen fibril. These filaments lay disorganized in the wound and are in a gelatinous state. There is little strength inherent in this collagen mass, which requires cross links and other bonding to be formed before the wound durability or tensile strength can be achieved.

Fibroblast also synthesizes glycosaminoglycans (GAG), which fill in the space between and around the collagen. This GAG ground substance, combined with water, Provides lubrication and acts as a spacer between moving collagen fibres. New crosslinks that are then formed convert mobile tissue into immobile tissue. The relationship between GAG ground substance and collagen dictates scar architecture. A bulky, rough, tender, red scar is then visible and palpable. Oedema, infection and rough handling can then cause the wound to become re-inflamed.

REMODELLING PHASE

Synthesis-lysis Balance

Despite the fact that collagen synthesis continues to occur at a high rate, no further increase in the scar mass occurs. At this point, new collagen is created and the old collagen is broken down in a balanced fashion as a result of the action of the action of enzyme collagenase. Collagen turnover is then accelerated as old fibrous tissue is removed and new fibrous tissue is formed. This process continues until the remodelling phase ends at completion of six months to a year, depending on the state of the injury. It should be remembered at this point that the speed of collagen synthesis and laying down of new collagen is age-related and its speed decreases with advancing years.

Collagen fiber orientation

During the remodelling phase, collagen turnover allows the randomly deposited scar tissue to be arranged, in both lateral and linear orientation. Scar tissue is non-elastic and it attempts to mimic the characteristics of the tissue that is being healed. The tissue structure induces the collagen weave so that dense tissues induce a dense, highly cross-linked scar, while more pliable tissues induce a loose, coiled, less crosslinked scar. A scar can adapt through remodelling forces of synthesis and lysis.

Sometimes the process does not function as expected and resulting in unwanted scar tissue in the form of a hypertrophic or keloid scars.

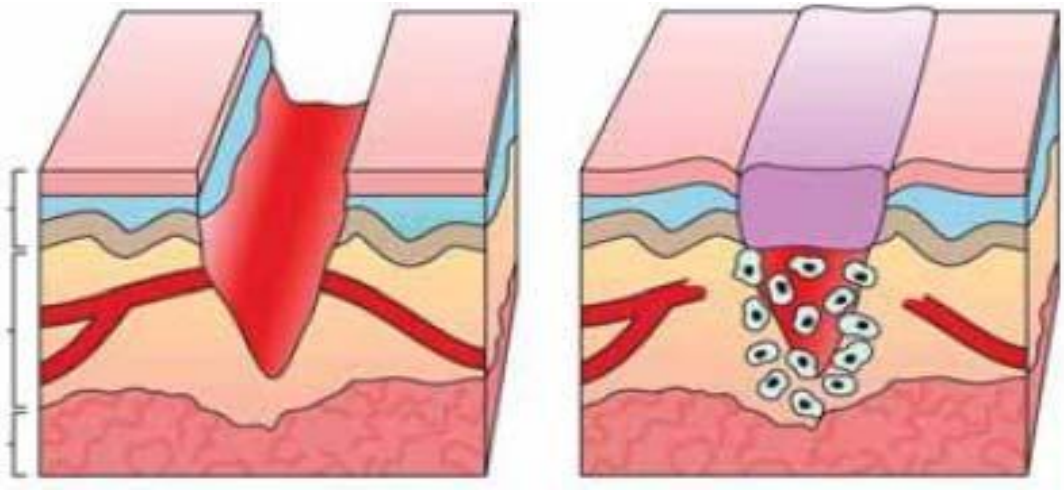
Types of Wound Healing :

Surgical wounds may heal by either primary intention, secondary intention or tertiary intention (delayed primary).

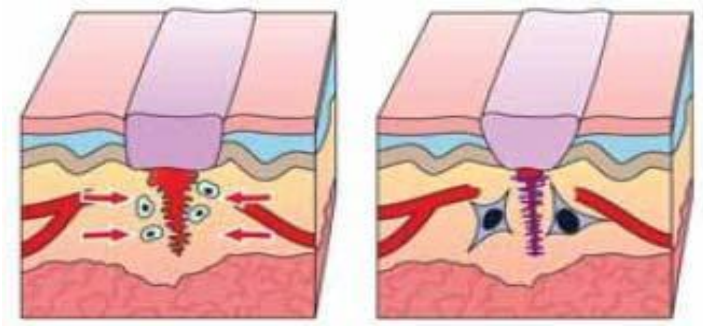
Healing by Primary intention

Most heal by primary intention, here the wound edges are brought together (apposed) and then held in together by mechanical means (adhesive strips, staples or sutures), allowing the wound time to heal and then develop enough tensile strength to withstand stress without support. The goal of surgery is to achieve healing by such means so that there is minimal oedema, no serous discharge or infection, no separation of the wound edges and with very minimal scar formation.

Haemostasis and Inflammatory Phase :



FibroplasticPhase :



RemodellingPhase :

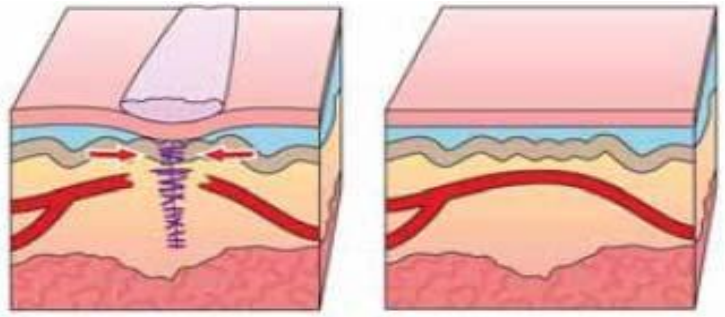


Fig 1: Phases of Wound Healing

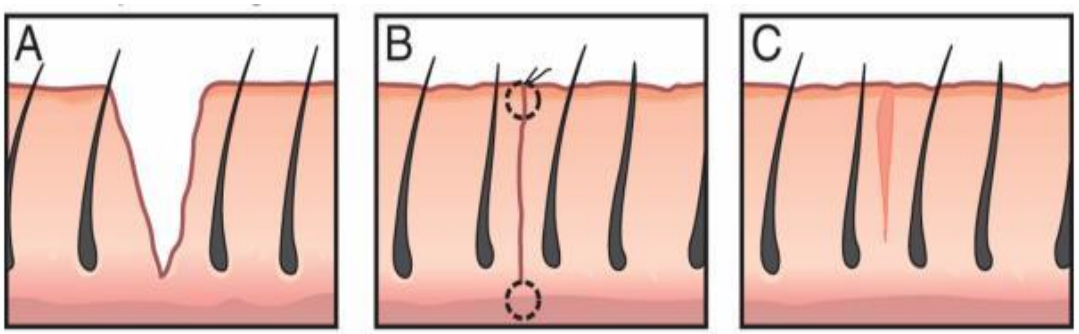
Healing by Secondary intention

Healing by secondary intention happens when the wound is left open, because of the presence of infection, excessive trauma or skin loss, and the wound edges come together naturally by means of granulation and contraction.[21]

Healing by Tertiary intention

On occasions surgical incisions are allowed to heal by delayed primary intention where non-viable tissue are removed and the wound is initially left open. Wound edges are then brought together at about 4-6 days, before granulation tissue is visible. This method is often used after traumatic injury or dirty surgery.[22]

Healing by Primary intention



Healing by Secondary intention

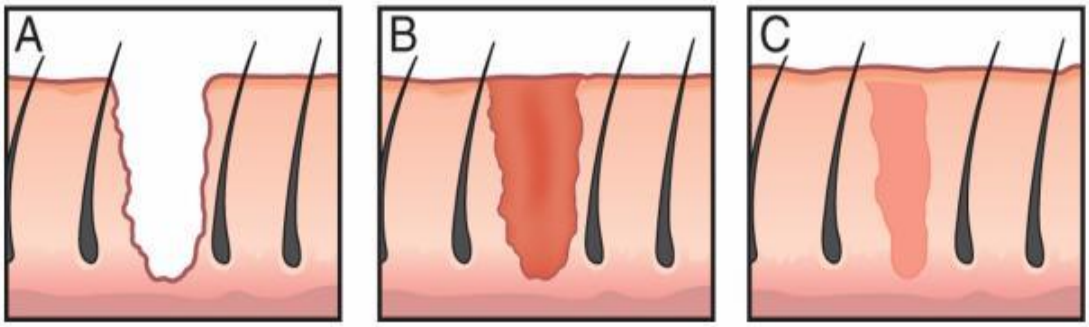


Fig 2: Types of Wound Healing

Factors Affecting Wound Healing[23]

The factors that may adversely affect the wound healing can be conveniently considered in two categories: factors, which locally affect wound repair (local factor) and the systematic abnormalities which have remote effects on the wound (systemic factors).

Local Factors

The local factors which have been implicated in the failure of wound healing are

1. Surgical technique.
2. Blood supply.
3. Mechanical stress.
4. Suture materials.
5. Suture technique.
6. Infection
7. Radiation

Surgical Technique[24]

The most important local factor in pathogenesis of wound complications is performance of the surgeon. Indeed, this is the single most important factor in the success or failure of wound healing. One might then expect that surgeons in training would experience a higher incidence of complications than qualified senior surgeons and there is some evidence to support the case.

The essentials of good surgical technique include gentle handling of the tissues, securing meticulous hemostasis, the prevention of any dead space in the wound, and the avoidance of tissue necrosis resulting from excessive use of surgical diathermy or strangulation of the tissues by the ligatures. The presence of one or more of these variables constitutes a barrier to the processes of cellular repair and they are the factors leading to propagate wound infection. Ischemic tissue, a wound hematoma,or a collection of serum in the wound is excellent media for the subsequent growth of bacteria.

The relative merits of surgical diathermy compared to suture ligation in wound hemostasis remain controversial but there is probably very little difference between the two methods as far as they affect the wound healing both may cause problems if they are used incorrectly. Diathermy should be used sparingly and precisely. Ligatures on blood vessels should not strangulate adjacent tissues. Fine suture materials can be used for most blood vessels and absorbable sutures can be used for vessels in the subcutaneous tissues.

Hematomas or collections of serum usually occur when ever dead space exists in the wound. Dead space is created in surgeries involving the reflection of skin or tissue flaps and in obese patients and it must be said that a potential dead space is virtually unavoidable in certain surgeries. However, dead space can be reduced or obliterated either by applying external mechanical pressure or by the use of wound drains. In obese patients, there is often a large potential dead space in the subcutaneous tissue and suture obliteration or drainage of this layer is advisable in these subjects.

Other local factors affecting wound healing such as blood supply of the wound and the presence or absence of mechanical tension may also be results of surgical technique and these are considered below.

Blood Supply

A good blood supply is a basic factor in the success of wound repair; it is essential for the supply of oxygen and other nutrients required in the cellular and biochemical processes of repair and it is necessary for the removal of wound metabolites.

Disease may lead to impaired blood supply of the wound. This is most frequently encountered in the surgical treatment of atherosclerotic arterial insufficiency of the lower limb. Any factor causing mechanical tension in the wound will have adverse effects on the blood supply. Extrinsic forces cause wound tension by distracting the wound edges. In the simple example of a sutured skin wound, the elastic pull of the unwounded skin on either side of the incision exerts a lateral pull on the wound edges. In wounds of the hollow viscera and the abdominal wall, wound tension is also derived from the pressure within the lumen of the viscus or hollow cavity; the tension occurring in the wound is directly related to this pressure and the radius of the lumen according to the law of Laplace.

The simple suture of wound therefore will inevitably result in wound tension and adverse effects on blood flow. It is difficult to state in quantitative terms the point at which such tension becomes harmful; the avoidance of tension in wound closure is a matter of surgical experience or expertise rather

than a measurable parameter. Intrinsic wound tension or the buildup of pressure within the volume of the wound contents following suture. Some degree of swelling of the wound is a normal feature of the early phase of repair. It results from the inflammatory response which is a feature of the first few days in all wounds and the surgeon should allow for such changes by ensuring that his sutures are not tied too tightly. More serious problems of intrinsic wound tension occur in the presence of wound infection, hematomas and collections of serum. These factors may cause an injurious rise in tissue pressure within the relatively inelastic confines of the wound. The presence of ischemic tissue in the wound initiates a vicious circle whereby the ischemic tissue results in tissue swelling and the tissue swelling lead to a further reduction in blood supply of the wound.

Mechanical Stress

The extrinsic forces affecting wound tension may cause wound disruption or it may be a consequence of excessive movement of the wound edges. In the former case, the tension at the suture or wound interface created by the extrinsic forces becomes so greater that the sutures simply cut out through the wound edges, less commonly the suture material may break or the knots may slip.

General surgeons are familiar with the effects of mechanical stress on abdominal wound healing a sharp rise in intra abdominal pressure caused by coughing or gaseous distension of the intestine may result in the abdominal wound disruption.

Suture Materials[25]

The choice of suture material in primary wound closure may have a significant bearing on the success of the subsequent wound repair. There have been striking developments in the manufacture of sutures in recent years and there is now an extensive range of naturally occurring and synthetic sutures. It has been suggested that the ideal suture may be defined as follows:

1. It should hold the tissues in apposition for as long as the natural forces are insufficient to resist separation or stretching of the wound edges.
2. It should handle easily and knot securely.
3. It should provoke minimal tissue reaction and it should be quickly absorbed so that the infection is not encouraged and it should not result in sinus formation.

Suture Technique[26]

There are general aspects of suture technique which need to be observed in the primary closure of all wounds and there are certain technical aspects which are peculiar to particular tissues or wounds. The general aspects include the careful apposition of the wounds. The general aspects include the careful apposition of the wound edges, the avoidance of strangulation of the tissues, the selection of suture materials which are sufficiently strong to provide adequate mechanical support to the wound and secure knotting of the wound sutures. Sutures should be inserted some distance away from the wound edges. The edges of the wound are weakened by collagenolysis for several days following wound closure and suture may cut out if they are too close to the wound edges.

Knot security is provided by the ‘surgeon’s knot’ or square knot and this should always be used in preference to a ‘granny knot’. Monofilament nylon and polypropylene have poor knotting characteristics and at least five ‘throws’ should be used to prevent knot slipping when these suture materials are used.

Radiation

Problems of wound healing resulting from ionizing irradiation chiefly occur in the management of skin wounds in previously irradiated tissues. These problems are frequently encountered in the surgical treatment of recurrent malignant disease of the chest wall or head and neck.

Infection

Bacterial infection is the most common complication of wound healing and it encountered in every surgical specialty. Multiple factors are involved in the pathogenesis of wound infection and the effects of infection are divers. Classical wound infection occurring in wounds closed by primary suture may simply be a source of significant morbidity but infection in vascular operations, plastic surgery and orthopaedic surgery may have disastrous consequences.

SYSTEMIC FACTORS[27]

Systemic factors which may affect wound healing are

1. Age
2. Malnutrition
3. Vitamin deficiency
4. Zinc deficiency

5. Trauma, hypovolemia and hypoxia
6. Anemia
7. Uremia
8. Malignant disease
9. Jaundice
10. Corticosteroid drugs
11. Cytotoxic and antimetabolite drugs

Age

It is a common finding in studies of wound healing that complications are more prevalent in elderly patient: abdominal wound dehiscence is more common and has been shown that such patients also have a significantly higher incidence of dehiscence of colonic or colorectal anastomoses. The fact is, however, that surgical patients also have a higher incidence of malnutrition, major operations, vitamin deficiencies and various other systemic abnormalities and it is difficult to conclude that age alone is a factor affecting wound healing.

Malnutrition

Many surgeons believe that malnutrition is the most important systemic factor affecting wound healing. Malnutrition has selective effects on certain tissues and wounds in different tissues may have a greater or lesser susceptibility to the effects of malnutrition. Malnutrition in surgical patients involves the deprivation of both protein and calories. Existing tissue collagen cannot be utilized in the local repair of wounds but it can be broken down and there is evidence that the amino acids of tissue collagen are reutilized in wound healing.

The amino acid methionine may have a key role in wound repair and that the adverse effects of malnutrition on wound healing may be reversed by the administration of methionine alone. Methionine is involved in the synthesis of the sulphatemucopolysaccharides of wound tissue and methionine and cystine are essential nutrients for the survival of fibroblasts in tissue culture. A deficiency of methionine would therefore provide a neat explanation for the failure of synthesis of collagen and mucopolsaccharides in malnourished subjects.

Vitamin Deficiency

Ascorbic acid deficiency is a significant factor in the healing of wound in surgical patients. Surgical trauma causes a fall in leucocyte ascorbic acid levels but these changes are unrelated to the severity of surgical trauma, blood loss, or blood transfusion and they may simply be obligatory features of the metabolic response to trauma; some of the ascorbic acid lost from leucocytes may be utilized in the surgical wound. The lowest levels of leucocyte ascorbic acid are found in the elderly and there may be a case for the use of ascorbic acid supplements in elderly patients.

Deficiencies of other vitamins probably have little relevance to wound healing in surgical patients. Vitamin A has a stabilizing effect on lysosomal membranes and it is alleged that it may reverse in inhibitory effects of corticosteroids on wound tissue. Vitamin E has an inhibitory effect on wound repair in experimental animals but there is no evidence that either vitamin affects wound healing in man.

Vitamin D is an essential in calcium metabolism and in the formation of new bone. In rickets, severe deficiency of the vitamin interferes with bone growth and a soft collagenous matrix of osteoid tissue is laid down instead of calcified bone. However, deficiencies of vitamin D are rarely encountered in civilized communities and there is no evidence that vitamin D deficiency affects the healing on bone in surgical patients.

Zinc Deficiency

It has been suggested that a deficiency of zinc has adverse effects on the healing of wounds in man. Zinc is required for several enzymatic reactions in the human body and it stabilizes lysosomal and cell membranes probably by inhibition of lipid peroxidases; the enzymes DNA – polymearse, reverse transcriptase and lysyl oxidase and zinc dependent. A deficiency of zinc has adverse effects on cell multiplication, fibroplasias, collagen synthesis and the epithelial covering of wounds but significant levels of zinc deficiency affecting wound healing in man are probably found only in severe burns, and in the management of intestinal fistulas. Patients required prolonged parenteral nutrition therapy in the absence of a normal diet should be given zinc supplements but the recommended dosage of zinc should not be exceeded since high serum levels of zinc are associated with toxic effects.

Trauma hypovolemia and hypoxia

Numerous studies have suggested that trauma, hypovolemia and hypoxia have important systemic effects on wound healing. Chassin and his colleagues measured the breaking strength of abdominal wound in rats, and they found that

the additional trauma of an extensive skin incision resulted in impaired healing of the abdominal wound. Zederfeldt suggested that the effects of remote trauma and hypovolemia on the breaking strength of abdominal wounds in rabbits and he reported that both factors had adverse effects on wound healing. Zedereldt suggested that the effects of trauma and hypovolemia on wound healing had a common basis and that tissue hypoxia was the final common factor affecting wound repair. Oxygen is an essential factor in the hydroxylation of the amino acid proline and lysine during the synthesis of collagen and further experimental studies have confirmed that a low tissue PO2 has adverse effects on wound healing. Further possible effect of remote trauma and wound healing is the adverse effect it may on the immunological defenses.

It has been shown that remote trauma and hypovolemia increased the susceptibility of experimental animals to staphylococcal and pseudomonas infections. However, according to Conolly and his colleagues, tissue hypoxia may be the most likely explanation for the susceptibility of traumatized or shocked animals to wound infection. Finally it has been suggested that postoperative changes in ascorbic acid availability may be a significant factor in the pathogenesis of wound failure in traumatized subjects but this seems unlikely since there is no significant correlation between the severity of trauma and postoperative changes in leucocyte ascorbic acid.

Several clinical studies have supported the thesis that major trauma and hypovolaemia affect wound healing. It has been shown that the incidence of wound complications increases in relation to the severity of abdominal trauma and there is a correlation between the volume of blood transfused or blood loss

during surgical operations and the incidence of abdominal wound dehiscence and dehiscence of colonic anastomoses. All the same, these observations do not prove the tissue hypoxia is the final factor causing these complications of wound healing and there may be other factors involved. Indeed, in experimental studies of the effects of trauma on the healing of colonic anastomoses, it was found that the postoperative intraperitoneal infection was the factor responsible for an increased incidence of anastomotic dehiscence in traumatized animals.

It has been suggested that the normal process of wound repair is inhibited by low wound oxygen tensions and that wound healing may be improved by increasing the oxygen supply to the wound. In tissue cultures, the optimal growth of fibroblasts is achieved at a SpO_2 of 60 mm of Hg, but Silver has reported that the proliferation of fibroblasts occurs in vivo at a tissue oxygen tension of 15 mm of Hg. In studies of the tissue fluid accumulating in wound chambers and sponges implanted in the subcutaneous tissue of experimental animals, Hunt and his colleagues found tissue SpO_2 levels which were significantly lower than 15 mm Hg and they suggested that hypoxia may be a rate limiting factor in the synthesis of collagen during the normal process of wound repair. Indeed it was shown that synthesis of collagen during the normal process of wound repair. Indeed it was shown that increased inspired oxygen tensions were associated with increased collagen synthesis in the simulated wounds in these experiments and it has also been reported that oxygen therapy in experimental animals results in an increase in the breaking strength of sutured skin incisions. Thus we now have the concept of ‘super-normal’ wound healing, but it should be noted that this is based to a large extent on biochemical observations of stimulated wounds, where implanted

tissue chambers and the tissue capillary oxygen gradients or conditions of oxygenation in these wounds may be quite different from those in sutured surgical incisions.

Anemia

Studies of surgical patients have suggested that anaemia may be involved in the pathogenesis of wound complications; an increased incidence of dehiscence of abdominal wounds and colonic anastomoses has been reported in anaemic patients. However, it is possible that other factor such as malnutrition or the type of surgery performed in anaemic patients were actually responsible for the failure of wound healing.

Malignant Disease

The significance of malignant disease as a systemic factor affecting wound healing is uncertain. Wolf suggested that it has no effect on healing and the results of clinical studies of abdominal wound healing by White and his colleagues Ellis and Heddle appear to support his contention. However, Reitamo and Moeller reported that there was a two-fold increase in the incidence of wound dehiscence in surgical patients suffering from malignant disease and Irvin and Goligher found that the dehiscence of colonic and colorectal anastomoses occurred frequently in patients with incurable malignancy. Again, it has been shown that jaundice resulting from malignant obstruction of the bile duct is associated with a high incidence of abdominal wound dehiscence, irrespective of the degree of biochemical disturbance associated with the jaundice.

Jaundice

In retrospective clinical studies of abdominal wound healing, Reitamo and Moller and Keill and other found no convincing evidence that jaundice was involved in the pathogenesis of abdominal wound dehiscence. However, these studies included very small numbers of jaundiced patients and in a careful prospective study of abdominal wound healing. Ellis and Heddle found that jaundiced patients had a significantly higher incidence of wound dehiscence and incisional hernia compared with anicteric patients undergoing laparotomy for various abdominal disorders.

Bayer and Ellis investigated the effects of jaundice on wound healing in the rat following ligation of the common bile duct and they reported that obstructive jaundice was associated with delay in the appearance of wound fibroblasts and new blood vessels and that there was a significant reduction in the breaking strength of abdominal wounds. Than et al., reported that there was evidence of reduced collagen synthesis in the tissues of jaundiced patients; measurements of the enzyme prolyl hydroxylase in skin biopsies of jaundiced patients reveal that the accumulation of collagen in abdominal wounds was delayed in jaundice patients but this biochemical abnormality was not accompanied by changes in mechanical strength or rupture stress of abdominal wounds. The effects of jaundice on wound healing in humans have been reexamined recently in a retrospective clinical study of 48 jaundiced patients undergoing abdominal surgery; abdominal wound healing in these patients was compared with the findings in 281 anicteric patients undergoing elective surgery for gallstones. Forty six of the 48 jaundiced patients had extra-hepatic

biliary obstruction and it was found that they had a significantly higher incidence of abdominal failure compared with anicteric patients ; wound dehiscence or incisional hernia occurred in 27.1% of jaundice patients and in 4.3% of anicteric patients. However, there was no correlation between wound dehiscence or incisional hernia and the depth of jaundice or plasma bilirubin, preoperative liver enzymes or plasma albumin levels and the factor which did seem to determine the outcome of abdominal wound healing was the presence or absence of malignant disease. Wound dehiscence or incision hernia occurred in 12 of 22 patients with malignant disease (59.1%) but these complications did not occur in 26 patients with jaundice resulting from benign pathology.

The findings of experimental studies indicate that jaundice has adverse effects on wound repair but the clinical significance of these findings is uncertain and it appears that other factors may account for the failure of wound healing in surgical patients; jaundiced patients with malignant disease are particularly at risk of complications of wound healing. The type of surgery involved in the management of patients with malignant disease may be a factor in the pathogenesis of wound complications.

Corticosteroids

According to Ehrilch and Hunt, the effects of steroids on wound repair may be related to the stabilizing effect of cortisone on lysosomal membranes. Experimental studies have shown that the effects of steroids on wound tissue are reversed by the administration of vitamin A which stabilizes lysosomal membranes. Laboratory studies of Sandberge have suggested that steroid therapy affects wound healing only when it is used before surgery or in the

early postoperative period; steroid therapy commenced several days after surgery has little or no effect on the healing of wound healing in experimental animals.

Clinical studies of wound healing in surgical patients have shown that wound dehiscence and sepsis are more frequent complications in patients receiving steroids at the time of operation. However, it is by no means certain that steroids are responsible for these complications for the patients receiving steroid therapy are frequency those who have serious disease, malnutrition and other factors which may affect wound healing.

Cytotoxic and Anti – Metabolite Drugs

There is increasing use of cytotoxic and anti – metabolic durg in medicine, transplantations surgery and in the management of malignant disease.

It seems probable by the nature of their action that the therapeutic use of these agents would interfere with wound repair and experimental studies in laboratory animals have produced some evidence to support this connection.

'Cut well, sew well, heal well' is an axiom favored by surgeons but it appears that there is rather more involved in the healing of surgical wounds. Surgical technique and other local factors are undoubtedly of much greater importance than the influence of systemic factors in the success or failure of wound repair, but we can nevertheless identify groups of patients who are at risk of wound complications by virtue of the presence of various systemic abnormalities. Unfortunately, with the probable exceptions malnutrition and

uraemia, we remain in the unhappy position of being unable to define the precise significance of the systemic abnormalities or to offer specific therapy which may remove the threat of wound complications. Further progress may depend on the development of improved techniques of local wound care.

The effect of bacterial infection on wound healing

The biochemistry of wound infection is complex. Delayed epithelial growth and migration, cellular necrosis and microvascular thrombosis are histological features of infected wounds and they result from the combined effect of bacterial toxins and the hostile chemical environment of the infected wound. The principle of biochemical abnormality in infected wound seems to be a disturbance of collagen metabolism, there is a constant process of synthesis and lysis of collagen in all wounds and to a lesser extent in unwounded tissue and this process may be affected in several ways by the presence of bacterial infection. Firstly, there is exaggerated lysis of wound collagen by collagenolytic enzymes, some of these are lysosomal enzymes present in polymorphonuclear lymphocytes in the infected wounds, others are enzymes which are normally present in tissues.[31]

The second factor, which may affect collagen metabolism, is disturbance of collagen synthesis in infected wound. Fibroblasts engaged in synthesis of wounds collagen must complete with other cells for available nutrients within the wound and in the presence of infection the metabolism of bacteria and inflammatory cells may utilize oxygen and other wound nutrients to the extent that the metabolism of fibroblasts is impaired. The net result of the changes in collagen metabolism is that the collagen content of the wound is reduced, the

process is not confined to the wound alone, it extends through the wound edges into unwounded tissues, the wound edges becomes soft and mechanically weak, and wound sutures will cut out the softened tissue resulting in the disruption of wounds closed by primary suture.

Etiology of Surgical Site Infection

No single factor is responsible for surgical site infection. Several factors are involved and the relative contribution of these factors varies greatly in different types of surgery. The vast majority of wound infections are endogenous. They are self-infections resulting from contamination of wound by bacteria carried by the host either on the body surface or more commonly within hollow viscera. A smaller proportion of wound infections are exogenous. They are cross infections by bacteria derived from another source and they may occur in the operating room or in the hospital ward.

Wound infection may be primary or secondary. Primary wound infection is the result of bacterial contamination of the wound occurring during surgery. Secondary wound infection occurs within the postoperative environment when bacteria gain access to the wound either through the wound suture line or through another portal such as a drainage tube or drainage track. The majority of wound infections are primary of type.

SURGICAL MICROBIOLOGY[32]

Surgical infections are usually caused by bacteria, but fungal and viral infections can also occur especially as post operative infections in immune-compromised hosts.

Bacteria

Bacteria can be classified according to staining characteristic with Gram stain (positive or negative), shape (cocci, rods, spirals) and sensitivity to Oxygen (aerobic, facultative, anaerobic) or according to the combination of these characters.

Gram positive cocci

Staphylococci and some streptococci species are the Gram positive cocci of interest to surgeons because of their ability to cause primary surgical infections and post operative infections. Staphylococci may be coagulase positive or coagulase negative.

Staphylococcus aureus is the most common pathogen isolated from wound infections. A major factor in its pathogenicity is coagulase productions, although the mechanism by which coagulase production increases virulence is not known. Most coagulase positive staphylococci should be resistant to penicillin and require treatment by a penicillinase resistant antibiotic. Extensive use of penicillinase resistant Beta lactam antibiotics during past 2 decades has encouraged emergence of Methicillin resistant staphylococcus aureus (MRSA). Coagulase negative staphylococci are the most common organisms recovered in nosocomial bacteremia and are frequently associated with clinically significant infections of intravascular devices.

Surgically important members of the genus streptococci include S.pyogenes, pneumoniae and the viridians group which includes S.mulleri, S.salaivarium. Streptococci are classified according to Lancefield classification and ability to cause hemolysis on blood agar, alpha hemolysis a zone of green

discoloration around colonies containing intact red blood cells, beta hemolysis, complete clearing of the area around colonies and destructions of red blood cells; and gamma hemolysis.

Group A streptococci can cause infections of almost any organ although skin, subcutaneous tissue and pharynx are the most frequently affected areas. Streptococci are important pathogens because of their ability to cause post operative infections including cellulitis, wound infection, endocarditis, urinary tract infection and bacteremia. Enterococci are commonly recovered as a part of the normal flora of the gastrointestinal tract and the vagina. Enterococcal bacteremia has a poor prognosis in combination with intra abdominal or pelvic infections and is found most often in patients who here been hospitalized for long time.

Aerobic and facultative anaerobic gram negative bacilli[33]

Numerous gram negative rods that can cause human disease have been identified, but only a few are of surgical importance. The genera Escherhia, Klebsiella, Proteus, Enterobacter, frequently can be cultured from patients with intra abdominal and pelvic peritonitis and abscess, post operative wound infection, pneumonia and urinary tract infection.

Pseudomonas aeurginosia is the species responsible for most surgical infections. They are frequently found in immunologically compromised patients, especially if they have been hospitalized for some time. Because of its resistance to single antibiotic therapy, Pseudomonas infections are frequently treated with a combination of two antibiotics.

Anaerobic Bacteria[33]

Anaerobic bacteria require reduced oxygen for growth. Virtually all anaerobic infections arise endogenously. The cell wall of anaerobic bacteria is important in abscess formation. The genus Clostridium is most virulent of all anaerobes. C.Dfficile cause pseudomembranous colitis and occurs in patients on antimicrobial therapy.

Fungi

Fungi are the most primitive eukaryote organism and are classified as protists. Because of their cell wall similarity to mammalian cells they are not sensitive to antibacterial agents, and many antifungal agents are toxic to human cells.

In surgical patients opportunists cause most infections. Candida albicans and other candida species are by far the most common. They cause infection in patients treated with broad spectrum antibiotics and steroids. These infection can be treated by stopping antimicrobial, correcting host defences and therapy with amphotericin B or one of the azole antifungal agents.

Viruses[34]

Viruses are obligate intracellular parasites and are distinguished by their having either DNA or RNA. CMV causes most viral infections in organ transplant recipients. Hepatitis B virus, hepatitis C virus and human immunodeficiency virus (HIV) are of importance of surgeons because of the possibility that they can become infected from patient exposure and that patients can potentially be infected from physicians who harbor the viruses.

PHYSIOLOGY AND PATHO PHYSIOLOGY OF SURGICAL SITE INFECTION

Physiology[35]

The unique feature of all surgical infection is tissue necrosis. In post traumatic surgical infection tissue necrosis is induced by technical or other physical trauma, while in primary surgical infection tissue the pathophysiological process induces necrosis. Inflammation is the response to tissue necrosis, leading to the events visible at surface, which were well described by Celsius and refined by Galen as rubor, tumor, calor, dolor and functiolaesa. These symptoms describe the host response and when controlled and properly regulated, result in elimination of necrotic material and prepare the way for tissue repair.

The magnitude of inflammatory response and of its symptoms is dependent on the burden of tissue injury and on the number and pathogenicity of invading organisms. If toxins or other bacterial products continuously destroy tissue, or exceed the capability of the host to confine the challenge of body integrity, the inflammatory process will continue and may then result in multisystem malfunction.

Local Phase of Infections

Surgical infections take a relatively uniform course once initiated. Macrophages may not be capable of phagocytosing all dead cells and remaining

necrotic tissue is an excellent medium for bacterial growth. Bacterial in turn release toxins and invade the surrounding tissue, which causes the host to respond with further inflammation in an attempt to confine the infection.

If tissue injury and number of bacteria exceed the capability of the host to terminate an infection locally an abscess may form. During infections inflammatory process spread centrifugally and fibrin deposition occur to confine the infection faster than bacterial toxins can destroy the tissue, and a pyogenic membrane is formed. An abscess is characterized by high pressure, low pH and low oxygen tension and is an ideal environment for multiplication of anaerobic bacteria[36]. Antibiotics poorly permeate it. The best treatment of an abscess is drainage. The local host defenses are so intensely concentrated around the abscess capsule that additional antibiotic therapy, except for the period immediately before and during drainage is rarely indicated.

Systemic Phase of Infection

When local circumscription of infection is not possible, either by bacteria or by abscess formation micro organisms eventually invade the blood stream and may reach distant organs. Non toxin producing, mostly non multiplying bacteria can sometimes be isolated by blood culture, but these cause no or only mild systemic symptoms, bacteremia may however progress to systemic disease especially in immune compromised and post-operative patients.

If the condition is present in association with multiplication of bacteria in the blood stream, then a serious state of infection termed sepsis or septicemia

ensure. SIRS (systemic inflammatory response syndromes) is the clinical symptomatic state resulting from host response to septicemia. Septic shock is a state of acute circulatory failure identified by presence of persistent arterial hypotension despite adequate fluid resuscitation without other identifiable causes. Septic shock is the most severe manifestation of infection, occurring in approximately 40% of patients with severe sepsis, it has an attendant mortality rate of 60% to 80%[37]. If sepsis is not treated immediately patient may die immediately of septic shock or later following multisystem organ failure.

Pathogenesis of Wound Infection[38,39]

Factors involved in the pathogenesis of wound infection are

1. Nature of Surgery
2. Exogenous infection (cross-infection)
3. Infecting organism
4. Host resistance

Nature of Surgery

There is a significant relationship between the different types of surgery and risk of wound infection. Surgical operations may be conveniently classified under the headings contaminated, clean - contaminated, and clean according to the actual or potential degree of bacterial contamination of the wound. Contaminated surgery refers to the operations which are undertaken in the presence of established sepsis. Thus, operations for peritonitis, perforated appendicitis and drainage of abscesses are included in this category and wound

infection may occur in 40-60% of such cases. Clean contaminated surgery refers to operations in which the surgical procedure includes exposure of the wound to bacterial contamination. Operations on the hollow viscera are included in this category ; operations on the biliary tract, gastrointestinal surgery and the surgery of the urinary tract. Widely differing accounts of the incidence of wound sepsis in clean contaminated operations have been descried. The incidence overall is probably of the order 10-20% but as one would expect, operations on the colon and the rectum are associated with the highest incidence of wound infection and the infection rates in excess of 50% have been reported in some series of colorectal operations.

In clean surgery, there are no special septic hazards inherent in the surgical procedure and wound infection results either from contamination by organisms from the patient's own skin surface or by exogenous contamination from the environment. Included in this category are most plastic, neurosurgical, orthopaedic and cardiovascular operations as well as breast surgery, herniorraphy and a variety of minor surgical procedures in general surgical practice. Infection rates of 2-4% have been reported in such operations.

Exogenous Infection

Cross infection assumes less importance than endogenous infection in the statistics of wound infection but it is a major cause of infection in clean surgery. Exogenous infection may occur in the operating room during exposure of the wound. The presence of bacteria in the wound at the end of the operation results in a five fold increase in the incidence of wound infection and the longer the wound is exposed, the more contamination is likely to occur; lengthy

operations are associated with an increased incidence of wound sepsis. Cross infection also occurs in the surgical ward and ward design is a significant factor in the pathogenesis of wound infection. Infection is more common in the traditional open type of ward than in modern surgical units which include patient's segregation and positive pressure ventilation. Patients are exposed to risk of cross infection in the surgical ward ; bacterial colonization of the wound may occur following surgery and the patient may acquire pathogenic strains of hospital bacteria following his admission to the hospital. In the later instance, these organisms may subsequently contaminate the wound during the surgical operation.

The risk of contaminating the patients wound occurs briefly within the operating room and the evidence or lack of it suggests that probably very few cases of wound infection are attributable to this factor. This is no doubt largely due to the insistence upon high standards of asepsis within the operating room environment.

The density of airborne contamination is a consequence of dispersal or shedding of bacteria by the operating room staff and it is thus affected by the number of staff in the operating room, the type of clothing worn, the activity or movement of the staff, and the nature of the ventilation system. Staff harboring pathogenic bacteria, present a special hazard and several outbreaks of staphylococcal wound infection have been attributed to this source.

The surgeon and his scrubbed assistants contribute to the bacterial contamination of the operating room and they present the added risk to the patient of direct bacterial inoculation of the surgical incision. Fortunately, this

mode of wound contamination appears to be an unusual cause of wound infection although it has been shown that wet operating gowns permit the transfer of bacteria from the surgeon's skin and that 5% surgeons gloves are perforated by the end of the surgical operation[40].

Infecting Organisms[41]

Most bacteria recovered from the surgical wounds are opportunistic pathogens; they are commensal organisms normally found in the hollow viscera or on the skin surface and they give rise to wound infections when they are inoculated in the wound in sufficient numbers.

Most endogenous infections result from spillage of bacteria into the wound during operations on the biliary tract or gastrointestinal tract; and the different types of wound infection in abdominal surgery reflects the differing bacteria flora of the abdominal viscera. Infections following gastric surgery are caused by the oral commensals, diptheroid species and occasionally coliform bacteria. The bacterial content of the stomach increases in pathological states of hypochlorhydria or pyloric obstruction. Wound infections following biliary surgery are usually caused by aerobic bacteria including Escherichia coli, Streptococcus faecalis and Klebsiellaaerogenes. In contrast, the colon the rectum contain large numbers of nonsporing anaerobic organisms in addition to aerobic bacteria and this is reflected by the types of wound infection which complicate the surgery of the colon and the rectum.

Frequently, endogenous infections are mixed infections involving more than one bacterial species and there is evidence that bacterial synergy occurs

when wounds are contaminated with aerobic bacteria and nonsporing anaerobes. The bacteriology of exogenous infections is less predictable but in most instances the infecting organism is an opportunistic pathogen which reaches the wound by airborne contamination. Usually such infections are caused by a single bacterial species.

Minority of surgical wound infections are caused by pathogenic bacteria, and most of these are staphylococcal. Certain phage types of staphylococcus aureus have been responsible for several minor epidemics of wound infection. These infections tend to be severe and they may have very serious consequences in clean surgical operations. Thus the repair of hernias, corrective or cosmetic plastic surgery and open orthopedic operations on joints or long bones may result in failure and infections in cardiovascular surgery may result in hospital deaths.

Host Resistance

Local factors affecting host resistance include any factor which interferes with or compromises the local inflammatory response to bacterial invasion of the tissues. In normal circumstances, the inoculation of bacteria into the tissues followed by a brisk vascular and cellular reaction; arteriolar dilation is followed by the stasis of capillary blood flow, the axial flow of capillary blood becomes disorganized and the emigration of white blood cells occurs though the capillary walls. Polymorphonuclear leucocytes appear within the wound, macrophages follow and phagocytosis of bacteria occurs.

Any factor which interferes with the blood supply will affect the local inflammatory response and favour bacterial growth. This may occur in several ways. In some cases, extensive tissue destruction is responsible particularly in traumatic wounds. In other cases, surgical technique is at fault. The surgeon may unwittingly produce tissue necrosis either by rough handling of the tissues or by strangulation of tissues during the knotting of ligatures or by excessive use of surgical diathermy.

Other local factors which affect host resistance are haematomas, seromas, and foreign bodies. Collections of blood or serum tend to occur when there is dead space in the wound. The most common foreign bodies are sutures and certain suture materials are more likely to propagate wound infections. Generally, bulky braided suture materials are more likely to cause trouble than fine monofilament sutures. Tissue fluid is drawn into space between the multiple fibers of braided sutures by a process of capillary attraction and bacteria may also be deposited in this location safe from the wound macrophages and free to multiply.

Wound drains also behave as foreign bodies in surgical wounds and they may adversely affect the host resistance. Drains are used either to remove blood or serum which may collect in dead space or to provide a route of escape for potentially infective material in contaminated wounds. Drains may be open or closed. In open drainage, the wound is protected only by wound dressing or a drainage collection bag and the disadvantages of this system are that the wound may be bathed in drain effluent and that secondary infection of the wound may occur through the drain site. This type of drainage system is frequently used in

heavily contaminated wounds and the risk of secondary infection is an accepted hazard. In closed drainage systems, the drainage is connected to a collecting receptacle and the drainage may be aided by the use of vacuum suction. The risk of secondary infection of the wound is reduced as such drains are commonly used to remove collections of blood or serum rather than to drain infected material. In clean surgical operations, wound drainage may be desirable if collections of blood or serum are probable, but the drainage system should be closed; increased rates of wound sepsis have been reported when open drainage systems are used in clean surgical operations.

Other conditions associated with an increased susceptibility to postoperative sepsis include malnutrition, uraemia, jaundice, lymphatic leukemia, diabetes and chronic granulomatous disease. Protein calorie malnutrition is frequently encountered in patient undergoing surgery for malignant disease or inflammatory disorders and recent studies have suggested that it is directly responsible for the increased incidence of septic complications in such cases; serious septic complications are common when preoperative weight loss exceed 20% of body weight. Malnutrition may be associated with impairement both of B and T cell mediated immune functions but the nature of the defect in host resistance is complex and it is almost certainly multifactorial. Moreover, the effects of acute and chronic starvation on the host defences may be quite different. Vitamin deficiency, particularly ascorbic acid deficiency may have important effects on the host susceptibility to infection; deficiency has adverse effects on phagocytosis and the migration of macrophages.

Systemic drug treatment may affect the host defences. The corticosteroid and cytotoxic drugs depress the immune response to infection and they may interfere with the early phase of wound repair. Steroids have cytotoxic effects on the reticuloendothelial system. They delay cellular repair and they have adverse effects on phagocytosis and it would be surprising perhaps if they did not propagate wound infection.

Similar problems are encountered in determining the effect of age on host resistance. Elderly patients have a higher incidence of wound infection, but these patients also have a higher incidence of malignant disease, malnutrition, major operations and heavily contaminated wounds. The total avoidance of wound infection in surgical operations is a desirable goal but one which currently remains beyond our reach. Nevertheless, the incidence of infection may be significantly reduced with care and attention to the factors which are involved in its pathogenesis and infection in clean surgery should be encountered very rarely.

RISK FACTORS FOR DEVELOPMENT OF SURGICAL SITE INFECTIONS[42]

Patient Factor

Older age

Immunosuppression

Obesity

Diabetes Mellitus

Chronic inflammatory process

Malnutrition

Peripheral vascular disease

Anaemia

Radiation

Chronic skin disease

Recent operation

Local Factor

Poor skin preparation

Contamination of instruments

Inadequate antibiotic prophylaxis

Prolonged procedure

Local tissue necrosis

Hypoxia, Hypothermia

Microbial Factors

Prolonged hospitalization

Toxin secretion

Resistance to clear form

Methods used in Prevention of Surgical Site Infection[43]

1. Endogenous infections - Reduce bacterial content of hollow viscera.

Prevent access of bacteria to wound

Mechanical cleansing of wound

Prophylactic antibiotics

2. Exogenous infection - Aseptic technique

Design of surgical wards

Isolation of infected patients

Non-woven operating room clothing

Laminar flow operating room ventilation

Prophylactic systemic antibodies

3. Host resistance - Meticulous surgical technique

Delayed primary suture of contaminated wounds

The measures which may lead to a reduced incidence of wound infection are summarized above and to a large extent they follow naturally from the identification of the factors which cause infection.

Endogenous infection

Contamination of the surgical wound by the host's own bacteria resulting in the endogenous infection and it is a problem which is chiefly encountered in the surgical operations on the hollow viscera. The prevention of

wound infection is therefore concerned with the prevention of wound contamination or with the use of techniques which may prevent the infective sequel of wound contamination.

Wound contamination may be limited either by achieving a temporary reduction in the bacterial content of the hollow viscera and skin or by using mechanical methods which prevent bacterial access to the wound. Most of the evidence suggests that former method is more effective in practice.

Antiseptic preparation of the skin is a necessary prelude to the surgical incision and it results in a temporary reduction in the numbers of viable organisms resident in the skin; effect of skin preparation is partly due to the mechanical washing and partly due to the antimicrobial properties of the antiseptic wash. Complete sterilization of the skin is impossible but a satisfactory reduction in the skin flora is achieved with a 0.5% solution of chlorhexidine in 70% alcohol or l% iodine in 70% alcohol.

The prevention of bacterial access to the wound by mechanical methods has proved to be reliable. In a controlled clinical trial, Raahave found that disposable plastic wound drapes reduced the extent of endogenous and exogenous wound contamination. Disposable adhesive plastic skin drapes are commonly used to prevent the endogenous contamination of wound by skin organisms. The infective sequel of wound contamination may be avoided either by mechanical cleansing of the wound or by the use of antimicrobial agents. Mechanical cleansing of the wound is achieved by irrigation usually with a normal saline solution. The actual technique of irrigation may involve gravity flow, bulb syringe irrigation or a pressurized pulsating jet lavage.

Antimicrobial agents may be used locally by topical application or systematically in the prevention of infection in contaminated or potentially contaminated wounds. Topical agents may be either antiseptic solutions or antibiotics. Antiseptic solutions have generally proved to be ineffective with possible exception of povidoneiodine. Systemic antibiotics are effective in the prevention of wound infection when therapeutic blood levels are achieved during the surgical operation; treatment started as prophylactic measure after the operation is probably of little value. Systemic treatment may be used either on a short term or on a long term basis. There are two distinct disadvantages associated with the prophylactic use of antibiotics. First, it has been shown that increased use of antibiotics results in an increased incidence of antibiotic resistant organisms in the hospital environment and this is inevitable consequence of long term antibiotic therapy. However, there is no evidence that short term therapy is associated with this risk. The second problem is the hazard of pseudomembranous colitis. The factors involved in the pathogenesis of this disease are obscure but it is associated with broad spectrum antibiotics lincomycin and clindamycin have been associated with a particularly high incidence of this disease but no broad spectrum antibiotic regimen may be exempted from this complication. Recent research has suggested that pseudomembranous colitis results from the suppression of the normal bowel flora and overgrowth of toxigenic strain of clostridium difficle.

Exogenous infection

Cross infection may be avoided by attention to various aspects of operating room and sterilized surgical materials, disinfection of skin and use of

no-touch techniques in the dressing of surgical wounds all of which are designed to prevent the transfer of bacteria to the surgical wound. The available evidence suggests that such measures are relatively effective in prevention of wound infection and air borne bacterial contamination of the surgical wound appears to be more important causes of wound infection.

It has been shown that traditional open ‘nightingale’ wards are associated with the higher incidence of wound sepsis compared with wards based on race-track principle. In the latter type of ward, clean and dirty areas are physically separated, air currents are controlled by positive pressure ventilation and patients are segregated in single rooms or in small units.

Patients who have clinical infections caused by the pathogenic bacteria such as staphylococcus aureus, Shigella or Salmonella must be isolated and barrier nursed. Ideally the general hospital should include an infectious disease unit in which such cases can be nursed.

Cross infection or endogenous contamination of the surgical wounds may both occur postoperatively in the surgical ward. The wound is vulnerable to contamination through the suture line for 4-5 days and it should be protected during this period. Exceptions to this rule are wounds of the face or neck and perineal wounds. Wounds of the face or neck have an exceedingly rich blood supply. They heal rapidly and septic complications are rare. The anatomy of the perineum makes perineal wounds dressing a difficulty and rather pointless exercise but septic complications in wounds closed by primary suture are surprisingly uncommon. There is now an enormous range of wound dressings but the choice really depends on the type of wound and its location. Dressings

should be dry and occlusive: ideally they should also be non adherent so that fibrin coagulum of the wound suture line is undisturbed if early removal of the dressing is necessary. However, wound dressings should not be disturbed until sutures are removed unless there is a valid reason for an earlier inspection.

In the operating room, cross infection is chiefly determined by the shedding or air borne dispersal of the bacteria by the operating room personnel. The staff in the operating room should be limited to an optimal number and unnecessary movement or talking should be discouraged. The use of nonporous or non-woven fabric clothing and operating gowns results in reduced bacterial dispersal compared with the woven cotton materials.

Steri-Drape Absorbent Prevention Fabric creates a barrier to inhibit fluid strike-through, reducing the need for multiple drapes and decreases chances of exogenous infection. Less draping means less time in application and removal. Fewer drapes to dispose off, therefore saving time and money.

The standard ventilation system in the operating room is a plenum system providing 15-20 air changes per hours. The air is partially filtered, humidified, heated or cooled and it is pumped into the operating room. If the air flow is turbulent; air currents do not provide special protection of the surgical wounds and bacterial particles are slowly and inefficiently removed from the operating room. Recent developments in the techniques of operating room ventilation involve the use of highly filtered air in special operating enclosure or wound isolators or laminar flow ventilation systems. In the Charnley enclosure, the surgical team operates in a clean room within the operating theater. The room is ventilated with highly filtered air and bacterial emission by

the operating team is reduced, by the use of special protective clothing and breath exhaust systems. The principle of laminar air flow is to eliminate turbulent recirculation of air at the operating site or wound and this may be achieved by vertical or horizontal air streams with a rate of air change of 600-700 times per hours. Vertical and horizontal laminar flow systems are probably quite similar in efficiency but horizontal systems have advantages in the cost and ease of operation. Ventilated wound isolators are even more economical but access to the patient is restricted by these devices and they are suitable only for a limited number of operating techniques and exposures[44].

The possibility that intraoperative contamination of the wound may be an unimportant factor in the pathogenesis of wound infection may seem difficult to accept but cross infection is chiefly encountered in clean surgery and existing rates of wound sepsis in the surgery are very low; they are certainly much lower than the incidence of air borne contamination in clean wounds. The incidence of wound sepsis may be no more than l-2% in clean operations and surgical technique host resistance factors may play a much greater part in the pathogenesis of wound sepsis by comparison with cases of endogenous wound infection.

Host Resistance

Local factors affecting host resistance are mainly related to surgical technique; infection is likely to occur in the presence of dead or devitalized tissue, foreign materials, wound hematomas or dead space.

Grossly ischemic or devitalized tissue is most frequently encountered in traumatic wounds or in the amputation of limbs for peripheral arterial insufficiency. In such cases, surgeon must be certain that all the dead tissues are excised and the blood supply of the final wound is adequate. Wound hematomas or collections of serum in the wound usually result from presence of dead space, and later may be consequence of unnecessary dissection or reflection of flaps comprising the skin and the subcutaneous tissue. In the presence of dead space, a closed system of suction drain is used to empty the space of blood or serum. The drains should be inserted through separate puncture incisions rather than through the wound itself. Dead space also occurs in the subcutaneous wounds of very obese patients and closed suction drainage of such wounds may also be desirable. However, this may be unnecessary if the tissues are carefully approximated with fine sutures of absorbable suture material or monofilament polypropylene.

All sutures, prosthetic implants and wound drains behave as foreign bodies and they propagate wound infection. In most cases, the use of foreign materials is unavoidable, but the surgeon may be responsible for some cases of wound infection by the injudicious use of wound drains or certain types of suture material. There is a temptation for to surgeon to use braided materials because they are easier to handle than monofilament sutures, but braided materials have a greater tendency to propagate wound infection and monofilament sutures should be used in contaminated wounds[45]; monofilament nylon, steel, or polypropylene cause little tissue reaction and persistent sepsis or wound sinuses are rarely encountered with such sutures. The use of wound drains should have a rational base. It is acceptable practice to use drains to

remove collections of blood or serum, but they are also used by some surgeons to prevent wound infection in contaminated operations. Whenever wound drainage is employed, the drains should be closed and they should emerge through incision separate from surgical wound.

Clinical Features and Treatment of Surgical Site Infection

The clinical signs and symptoms of wound infection are varied and they depend on several factors including the location or distance of the infected focus from the skin surface, the nature of infecting organisms and host resistance. The classical signs of infection i.e. heat, redness, swelling, pain and loss of function may or may not be present. In most of the cases the diagnoses is finally with the discharge of pus from the wound either spontaneously or by deliberate opening by surgeon[46]. The peak incidence of onset of symptoms and signs of wound infection occur 3-10 days after surgery.

Mild, moderate or severe fever is usually present but significant toxemia is unusual. In superficial wound infection limited to the skin and subcutaneous tissue, signs of infections are usually immediately apparent on examination of the wound. The surrounding skin is edematous and red. The wound is exquisitely tender on palpation and a purulent discharge may be present. The diagnosis is confirmed by gently separating the edges of the skin incision with a sinus forceps and pus is released from the subcutaneous tissue. In deep wound infection arising beneath the fascial layers, clinical signs of infection may be absent on examination of wound apart perhaps from some tenderness on palpation, presence of unexplained fever in such cases often prompts a search elsewhere for other possible foci of infection[47].

The nature of the pus discharge may provide a clue to the species of infecting organisms. Staphylococcal infection traditionally produces creamy yellow pus, pseudomonas pus has a characteristic odour and it may cause green or blue staining of the wound dressing. Proteus infections have a fishy odour and infections following intestinal surgeries which are frequently mixed infections involving bacteroid species and aerobic coliforms produce pus which looks and smells like liquid faeces[48].

In majority of cases, the treatment of wound infection is a relatively simple matter and consists of providing adequate drainage of the infected wound. When pus is already discharging though the skin, the drainage tracts are gently stretched with a sinus forceps. The sinus forceps is pointed in various directions deep to the skin so that all foci of infection are drained. It is rarely necessary to open the wound widely or to conduct a formal wound exploration under anaesthesia, although this is occasionally necessary in cases of deep wound sepsis located beneath the facial layers.

Aggressive soft tissue infections are rare, difficult to diagnose, and require immediate surgical intervention plus administration of antimicrobial agents. Failure to do so results in an extremely high mortality rate 80 to 100%, even with rapid recognition and intervention, current mortality rates remain approximately 30 to 50%[49].

Role of Laboratory in Infection Diagnosis

A variety of laboratory tests may be helpful in determining the timing of therapeutic intervention in patients with proven or suspected infection. The

basic procedures usually include a naked eye examination of the specimen, microscopic examination of Gram stain, and culture on aerobic and anaerobic blood agar plates, on MacConkey's agar and in cooked meat broth.

Generally surgical infections are characterized by leukocytosis. Affected patients may have some degree of coagulopathy, glucose intolerance in septic patients, and may be seen with hypoglycemia. Ascending colangitis in infected patients with hyperbilirubinemia should be considered. From the point of view of surgical intervention, laboratory helps in defining laws of infection in isolating a specific organism or a group of organism and providing data that supports the worthiness of antimicrobial treatment in terms of insuring both the killing of organism and minimum toxicity from the dry closure. Gram stain is a simple procedure which pathogenic agents can be predicted and can guide as for empirical therapy.

Wound swab from the local site of suspected infection should be cultured and blood cultures should also be sent along. The cases in which prophylactic antibiotic is administered, timely estimation of serum level should be done. The specimen should be inoculated on to two plates of blood agar, one for incubation in 37^0C aerobically, preferably in air plus 5-l0%, Co_2, the other for incubation anaerobically in nitrogen / hydrogen pulse 5-10% Co_2. The agar plate also has antibiotic walls to identify sensitivity. The culture plates are examined after overnight incubations at 37° C for 18-24 hours. If no growth, plate should be reincubated for another 24 hours[50]. Most surgical infectious can be managed well by using standard disc diffusion antibiotic susceptibility data and providing dosage of standard amount of antibiotics as required. Recent

investigations such as accessing blood, CSF and urine by countercurrent immunoelectrophoresis or using latex agglutinations test for the presence of antigens of pathogens such as streptococcus pneumoniae, hemophilus influenza or niesseria meningitides. Other techniques such as gas liquid chromatography are used to identify footprints that are short chain fatty acids of anaerobes.

The Pathogenic Bacteria Responsible for Surgical Infection:

Surgical infections are usually caused by bacteria but fungal and mixed infections can also occur especially as postoperative infection in immune compromised hosts. Most bacterial infections are due to organisms that are part of the patient's endogenous flora bacteria that are normal residents of skin or gastrointestinal tract. The various selected features of bacteria in surgical infection is as shown in the table below[51]:

Table 1 : Organisms causing surgical site infection			
Organism	**Frequency of organism seen in Surgical Infection**	**Likelihood of Single-Pathogen Surgical Infection**	**Type of Surgical Infection**
AEROBIC BACTERIA Gram-positive Cocci Staphylococcus aureus	High	High	Skin and wound abscess, infected I.V. catheter site, bacteremia, endocarditis, infected prosthetic device, pneumonia, postneurosurgery meningitis, osteomyelitis, infected joint
Staphylococcus epidermis	Moderate	Low	Usually mixed infection but can cause bacteremia, ventriculoperitoneal shunt infection, endocarditis, skin infection.
Streptococcus pneumoniae	Moderate	High	Pneumonia, bacteremia, infected joint.
Enterococci	High	Low	Usually mixed infection - wound and intraabdominal abscess, endocarditis, urinary tract infection (UTI).
Other Streptococcus Species	Moderate	Low	Usually mixed infection-skin and woundinfection, intraabdominal abscess.
Gram – negative Cocci Neisseria gonorrhoeae	Low	Moderate	Tubo-ovarian abscess mixed infection with anaerobes, enteric bacilli and Chlamydia is common
Neisseria meningitides	Low	High	Bacteremia, pneumonia (especially group Y)
Branhamellacatarrhis	Low	Moderate	Pneumonia (usually community acquired)

Gram-positive bacilli Bacillus species (especially cereus)	Low	High	Usuallycontaminant may cause bacteremia, endophthalmitis
JK-Dinhtheroids	Low	High	Bacteremia
Gram-negative bacilli Escherichia coli	High	Moderate	Bacteremia, UTI, pneumonia; often in mixed infection wound, intraabdominal and pelvic abscess
Other Enterobacteriaceae KlebsiellaEnterobacter,	High	Low	Mixed infection such as wound, intra- abdominal and pelvic abscess; occasional bacteremia. UTI and pneumonia
ANAEROBIC BACTERIA Gram-positive Cocci Peptococcus,	Moderate	Low	Mixed infection, genitourinary infections, fasciitis
Gram - Positive bacilli	High	Moderate to Low	Usually mixed infection (wound, intraabdominal) gas gangrene, occasional devastating sepsis in genitourinary infection
Clostridium tetani	Low	High	Causes tetanus
Clostridium botulinum	Low	High	Causes wound botulism
Gram-negative bacilli	High	Moderate	Usually mixed infection
Other Bacteroides species	High	Low	Mixed infection
Fusobacteria	Moderate	Low	Mixed infection

ANTIBIOTICS

Role of Antibiotics in Infection Management

The use of antimicrobials or antibiotics in surgical infections has come in a long way in prophylactic therapeutic management. The role of antimicrobial therapy is to prevent or treat infections by reducing or eliminating pathogenic organism until the host's own defenses can get rid of the last pathogen. The basic consideration in choosing antimicrobial is efficacy, toxicity and cost effectiveness. Effective antimicrobial agent must be active against the pathogens causing the infections and must be able to reach the site of infections in adequate concentration and in particular time.

All antibiotics have potential toxicity. Toxic effects may be idiosyncratic such as allergy or the rare instance of bone marrow aplasia caused by chloramphenicol or result in damage to tissue and organs as renal toxicity or ototoxicity seen with aminoglycosides and amphotericin B. Antimicrobial agents also exert selective pressure on the microbial ecology of hospital that leads to resistant microbes lost in the final consideration in the selection of antimicrobials. Determining antimicrobial costs include more than just the cost of the drug, the drug administration charges, nursing time, intravenous fluids and lines and monitoring costs must also be added to drug costs.

Distribution of Antimicrobial Agents[52]

Successful treatment of localized infections with systemic antimicrobial agents requires that an adequate concentration of antibiotics be delivered to the site of infection ideally the tissue concentration of antibiotics should exceed the minimum

inhibitory concentration. Tissue penetration depends on protein binding of antibiotics. Only the unbound form of antibiotic will pass through capillary wall or act to inhibit the bacterial growth. Lipid solubility is also an important factor in tissue penetration.

Blood

Rapidity of excretion and protein binding are two main determinants of blood concentrations of antimicrobial agents. Those that are highly protein bound are not excreted rapidly as those with a low binding affinity and thus have longer half lives. Efficacy of pencillins, Cephalosporins and other antibiotics that affect bacterial cell wall synthesis depend on the time during which serum levels are above the minimum inhibitory concentration rather than a peak serum concentration.

Urine

Most commonly used antibiotics are excreted principally in the urine and achieves high urinary concentrations upto 50-200 times their serum concentrations. Notable exceptions are erythromycin and chloramphenicol. Since concentrating ability is severely compromised in patients with renal disease infectious of urinary tract are more difficult to treat in these patients.

Bile

Beside urine, only bile has concentrations of antibiotics higher than found in serum. The biliary concentration of the penicillins especially nafacillin, piperacillin and azlocillin, cephalosporins, especially cefazolin, cefamandole, cefamide, cefoperazone and cefadroxil frequently are several times that of serum.

Intestinal fluids and Tissues

High prolonged serum concentration and low protein binding favor diffusion of antibiotics from serum into extra vascular tissue. Absolute tissue level may not accurately reflect the therapeutic potential because tissue may bind with antibiotic and thus be unavailable for binding to bacteria.

Principles of Antibiotic Therapy

1. The organism should be sensitive to antibiotic chosen.
2. Antibiotics should be in dose that ensures adequate peak concentration and tissue penetration.
3. The Antibiotics should come in contact with the organism.
4. Frequency of administrative is based on the half life and the route of eliminations of the antibiotics
5. Choose a bactericidal antibiotic when appropriate.
6. Use synergistic therapy when appropriate
7. Avoid antagonistic combination of antibiotics
8. Choose the most appropriate and narrow spectrum antibiotic
9. Adverse effects should be evaluated and risk benefit balanced.
10. Ensure proper duration of therapy to ensure eradication of pathogenic organism.

In general if a single effective, nontoxic drug is used to prevent infection by a specific microorganism or to eradicate an early infection, then chemoprophylaxis frequently is successful[53].

Prophylactic Antibiotics[54]

Ever since antibiotics became available they have been used to prevent infection in surgical practice. It has greatly evolved and grained much attention in the last 25 years. The objective of most antibiotic prophylaxis is to achieve a high tissue level of an appropriate choice of antibiotic and they have defined more clearly the value of techniques in reducing post operative wound infection.

Selection and Administration of Prophylactic Antibiotic

An appropriate prophylactic antibiotic should be[55]

1. Effective against microorganisms anticipated to cause infection.
2. Achieve adequate local tissue levels.
3. Cause minimal side effects
4. Be relatively inexpensive.

The microbial content of the wound and the hospital environment may influence the choice of antibiotic but coverage should primarily target those organisms known to cause post operative infection. In general, a first generation or third generation cephalosporin fulfills these criteria and is regarded as sufficient prophylaxis for the majority of clean and clean contaminated surgeries.

Timing of Prophylactic Antibiotic Agents[56]

It has been observed in laboratory that the effectiveness of antimicrobial

agents in preventing infection diminishes as the time between contamination and the initial administration of the antimicrobial agent is lengthened. Timing of administration is critical. The drug should be administered within 30 minutes and certainly within 2 hours of the time of incisions. The first dose should always be given before the skin incision is performed. For longer procedures, re-administration of drug is indicated at internals of one or two times the half life of the drug. This ensures adequate tissue levels throughout the duration of the procedure. For clean procedures, only single dose with long half life in high dose is preferred. The duration of administration is extended only in special circumstances such as gross contamination secondary to ruptured viscus or trauma[57].

Prophylactic Agents[58]

The ideal prophylactic antibiotic needs to achieve a balance between safety and efficacy. Some commonly used agents are

Beta - Lactam Antibiotics

The most common and largest class of antibiotics in current usage the term is derived from the presence of a unique four member beta-lactam ring in all agents in this class. These include penicillin, cephalosporins, the monobactams and the thiocyanins.

Penicillins

These are the oldest group of beta-lactams. It was first extracted from the penicilliumnotatum. With molecular manipulation on the original nucleus using modern biochemical techniques a large number of enhancements and alterations to bacterial sensitivity have been achieved.

Cephalosporins

These are the largest group of beta-lactams in common usage the natural compound is procedure by the fungus cephalosporium. Cephalosporins have developed into series of generation with each generation representing a broadening of the antibiotic spectrum and activity. The agent within a given generation possesses similar antibacterial characteristics.

First generation cephalosporins include cephalothin, cefazolin and cephalexin. These are most active against gram positive organisms like staphylococcus and streptococcus and are generally ineffective against anaerobes and many gram negative organisms.

Second generation cephalosporins include cefoxitin, ceforoxime, cefatetan and cefaclor. These possess an increased activity over gram negative organisms, although their activity against gram positive organisms is less than the first generation, they are also effective against anaerobes.

Third generation cephalosporins have been most heavily developed in recent years. These include cefotaxime, ceftizoxime, ceftriaxone etc., These are beta lactamase resistant, thus have enhanced activity against aerobic gram negative bacteria they possess little activity over anaerobes.

Forth generation cephalosporins include cefipime and cefpirome which have broader activity and are effective against gram positive as well as gram negative organisms.

Vancomycin: Glycopeptide is most active against Gram-positive bacteria and has proved most effect against MRSA. It is effective against C.difficile and given orally in cases of pseudomembranous colitis.

Carbapenems:Meropenem, ertapenem and imepenem are members of this group. They are stable to beta lactamases and have useful broad spectrum anaerobic as well as Gram positive activity.

Imidazoles: Metronidazole is most widely used member and is active against allanaerobic bacteria. Infection with anaerobic cocci and strains of Bacteroids and Clostridia can be treated or prevented by its use.

Other agents used include Aminoglycosides Tetracyclins

Quinolones

METRONIDAZOLE

Metronidazole is a nitroimidazole antiprotozoal drug that also has potent antibacterial activity against anaerobes, including bacteroides and *Clostridium* species. It is well absorbed after oral administration, is widely distributed in tissues, and reaches serum levels of 4–6 mcg/mL after a 250-mg oral dose. Metronidazole can also be given intravenously or by rectal suppository. The drug penetrates well into the cerebrospinal fluid and brain, reaching levels similar to those in serum.

Metronidazole is metabolized in the liver and may accumulate in hepatic insufficiency. Metronidazole is indicated for treatment of anaerobic or mixed intra-abdominal infections, vaginitis (trichomonas infection, bacterial vaginosis), *C difficile* colitis, and brain abscess. The typical dosage is 500 mg three times daily orally or intravenously (30 mg/kg/d). Vaginitis may respond to a single 2-g dose. A vaginal gel is available for topical use. Adverse effects include nausea, diarrhea, stomatitis, and peripheral neuropathy with prolonged

use. Metronidazole has a disulfiram-like effect, and patients should be instructed to avoid alcohol. Although teratogenic in some animals, metronidazole has not been associated with this effect in humans.

MATERIALS AND METHODS:

This study includes 100 clean and clean contaminated cases randomized to groups of 50 each. The study group will receive a single dose of antibiotic preoperatively while the control group will receive 3 to 5 days of empirical antibiotic therapy.

All the clean class 1 cases in the study group were given a single dose of 1gm of inj. Ceftriaxone at the time of induction or 30 minutes before skin incision in case the procedure is prolonged for more than 3 hrs a second dose was given.

They received no further antibiotics i.v or oral. All the cases in the control group received 5 days of inj. Cefotaxime 1Gm iv BD for 5 days. The incidence of SSI was noted and analysed.

All the class 2 cases in study group received inj. Ceftriaxone 1gm and inj. Metronidazole 500 mg iv 30 minutes before the skin incision. In case the procedure was extended beyond 3 hrs a second dose was given. They received no further antibiotics i.v or oral. All the cases in the control group received inj. Cefotaxime 1gm iv BD along with inj. Metronidazole 500 mg i.v TDS for 5 days. In case of underweight or obese patients the dose was adjusted according to their body weight.

All the cases were followed up at 8th POD,15th POD, 30th POD and later at 3 months and 6 months. Any wound related complications noted and data obtained. The incidence of SSI in both the groups was calculated and results analysed.

INCLUSION CRITERIA:

Clean and clean contaminated cases in department of general surgery**.**

EXCLUSION CRITERIA:

1. Contaminated cases are excluded.
2. Those patients who do not consent are excluded.
3. Patients below 18 yrs age were excluded.
4. Pregnant patients were excluded.

RESULTS:

OBSERVATIONS

CLASS I:

AGE:

Graph 1:

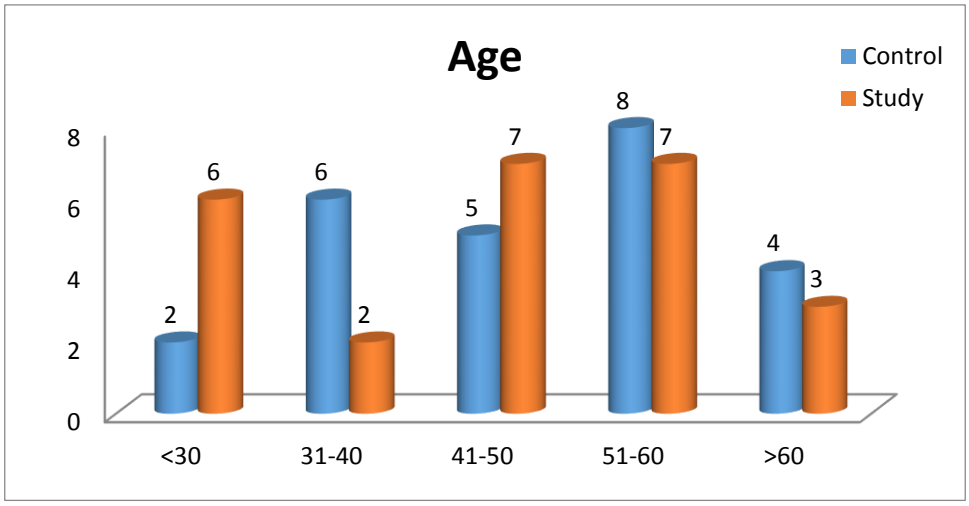


Table 2:

Age_r	Control	Study	Chi sq	p
<30	2	6	4.54	0.33
31-40	6	2		
41-50	5	7		
51-60	8	7		
>60	4	3		
Total	25	25		

In my study the age distribution of the patients varied from less than 30 years to more than 60 years. The most common age group was 50-60 years. There was no significant difference between the control and study group based on age as borne out by the tables and p value of 0.33 which is not significant.

SEX:

Graph 2:

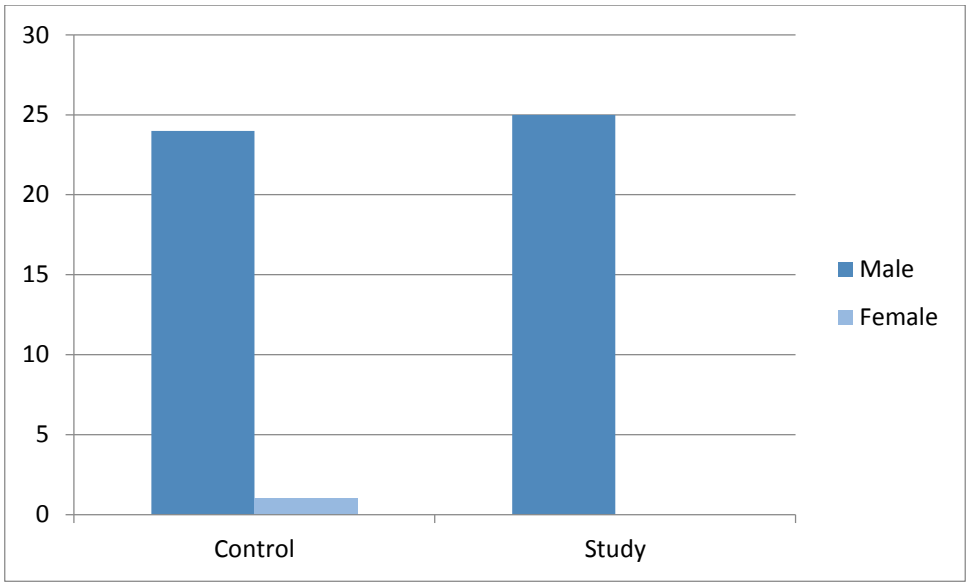


Table 3:

SEX	Control	Study
Male	24	25
Female	1	0
Total	25	25

SEX	Control	Study	Total	Chisq	P
Male	24 (96%)	25	49	1.02	0.3
Female	1 (4%)	0	1		
Total	25	25	50		

In the above study most of the patients were male as inguinal hernia is more common in males. Again there was no significant difference between both the groups in sex wise distribution of cases as borne out by the p value of 0.3 which is not significant.

SIDE OF HERNIA:

Graph 3:

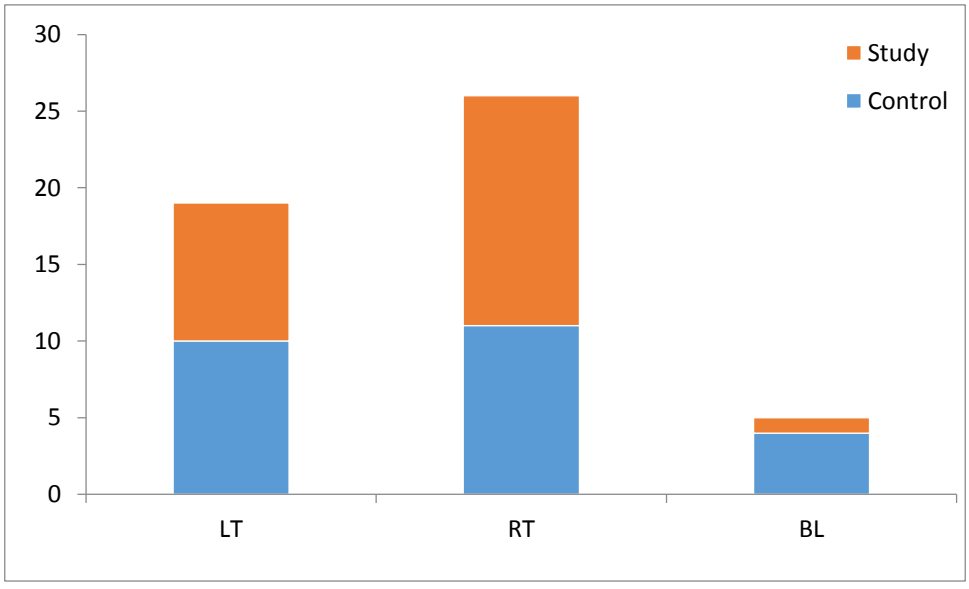


Table 4:

HERNIA	Control	Study
LT	10	9
RT	11	15
BL	4	1
Total	25	25

HERNIA	Control	Study	Total	Chisq	P
LT	10	9	19	2.46	0.2
RT	11	15	26		
BL	4	1	5		
Total	25	25	50		

In this study there was an even distribution of cases based on the side of hernia. There was a slight preponderance of right sided hernia overall. But again as borne out by p value of 0.2 which is not significant, there is no significant discrepancy in distribution of cases based on side of hernia between the groups.

FEVER:

Graph 4:

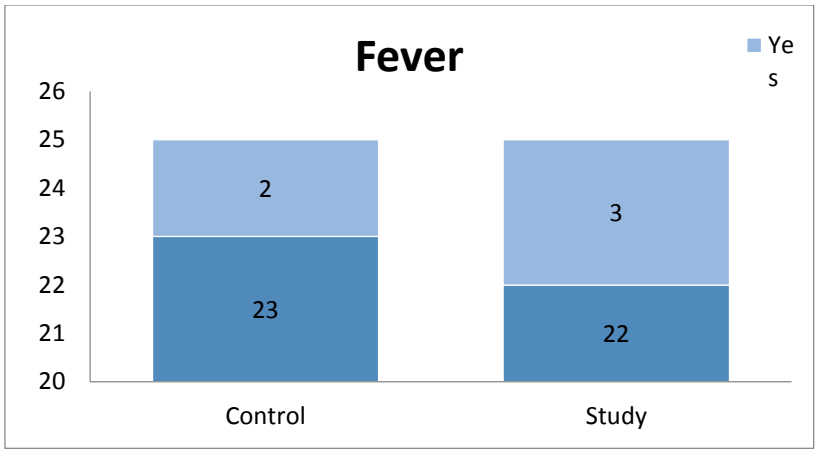


Table 5:

FEVER	Control	Study
No	23	22
Yes	2	3
Total	25	25

FEVER	Control	Study	Total	Chisq	P
No	23	22	45	0.22	0.6
Yes	2	3	5		
Total	25	25	50		

In the above study 0.5% of control and 0.75% of study group pts developed fever secondary to wound infection. Again there is no significant

difference in incidence of post operative fever between the groups as shown by the p value of 0.6 which is not significant.

SWELLING:

Graph 5:

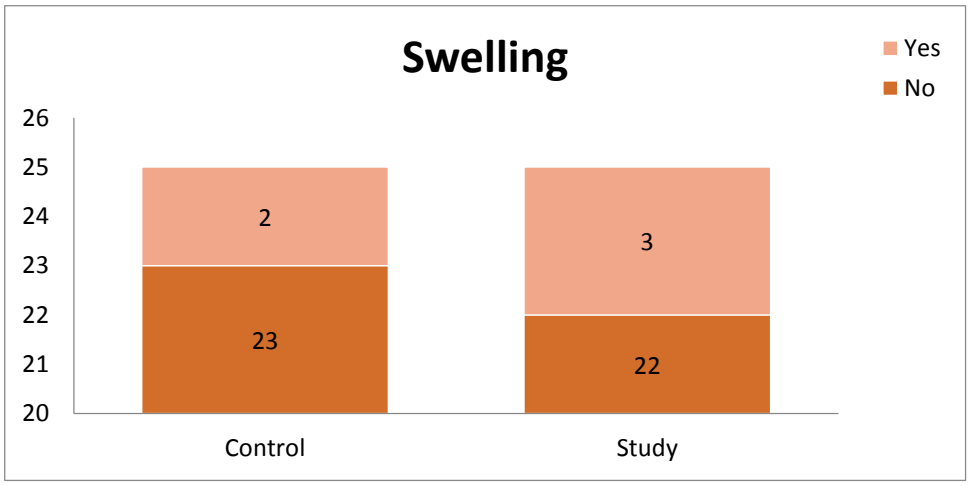


Table 6:

SWELLING	Control	Study
No	23	22
Yes	2	3
Total	25	25

SWELLING	Control	Study	Total	Chisq	P
No	23	22	45	0.22	0.6
Yes	2	3	5		
Total	25	25	50		

In my study the incidence of post operative swelling of operative site

presumably due to SSI was 2 in control group and 3 in study group. But the difference was significant as evidenced by the p value of 0.6.

PAIN:

Graph 6:

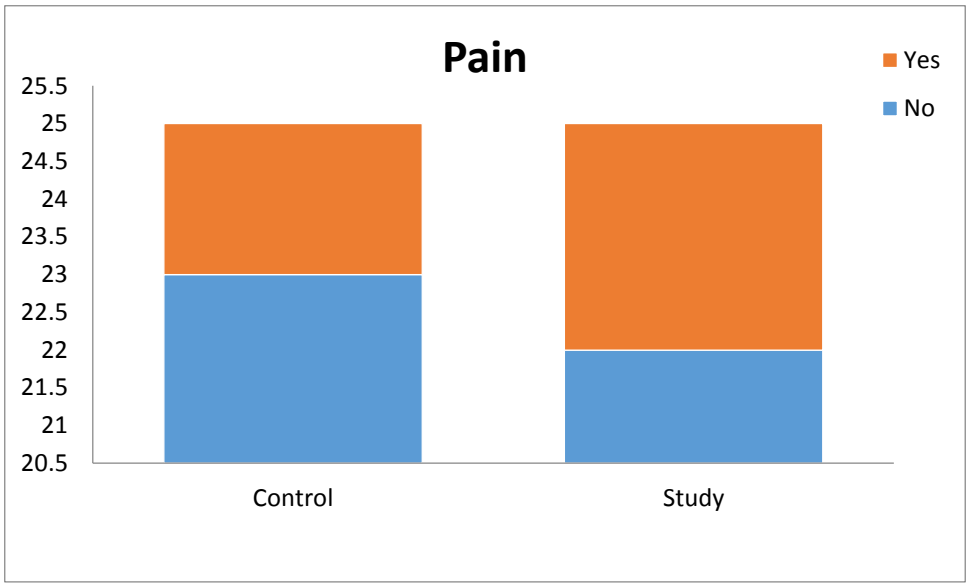


Table 7:

PAIN	Control	Study
No	23	22
Yes	2	3
Total	25	25

PAIN	Control	Study	Total	Chisq	P
No	23	22	45	0.22	0.6
Yes	2	3	5		
Total	25	25	50		

In my study the incidence of post operative swelling of operative site secondary to infection was 0.5% in control group and 0.75% in study group. Again there is no significant difference in incidence between the two groups as shown by p value of 0.6 which is not significant.

WOUND DISCHARGE:

Graph 7:

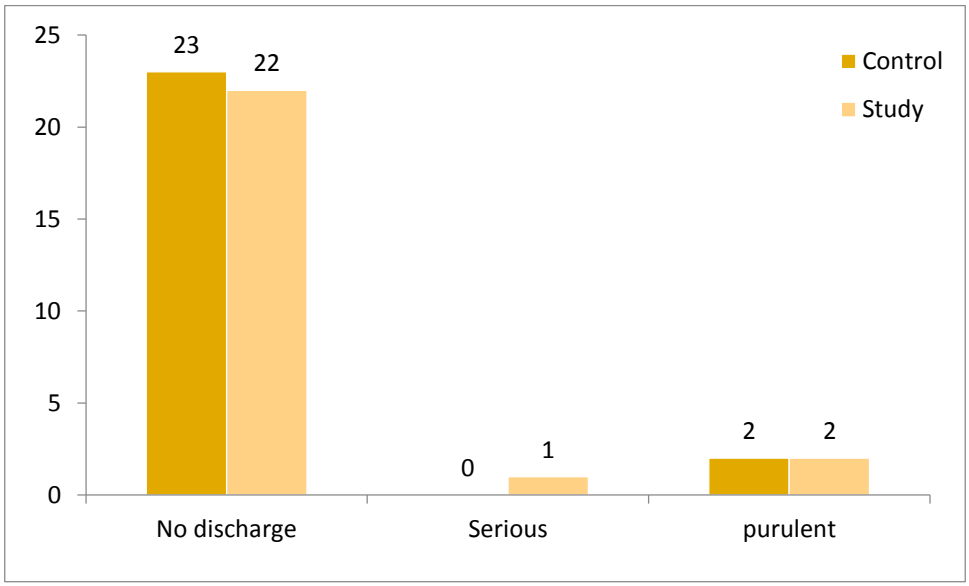


Table 8:

Wound Discharge	Control	Study
No discharge	23	22
Serious	0	1
purulent	2	2
Total	25	25

Wound Discharge	Control	Study	Total	Chi sq	P
No discharge	23	22	44	0.2	0.7
Serious	0	1	2		
purulent	2	2	4		
Total	25	25	50		

In the present study 2 patients in the control group and 2 patients in the study group developed purulent discharge. In addition one patient in the study group developed serous discharge. The difference is not significant as shown by the p value of 0.7.

ORGANISM:

Graph 8:

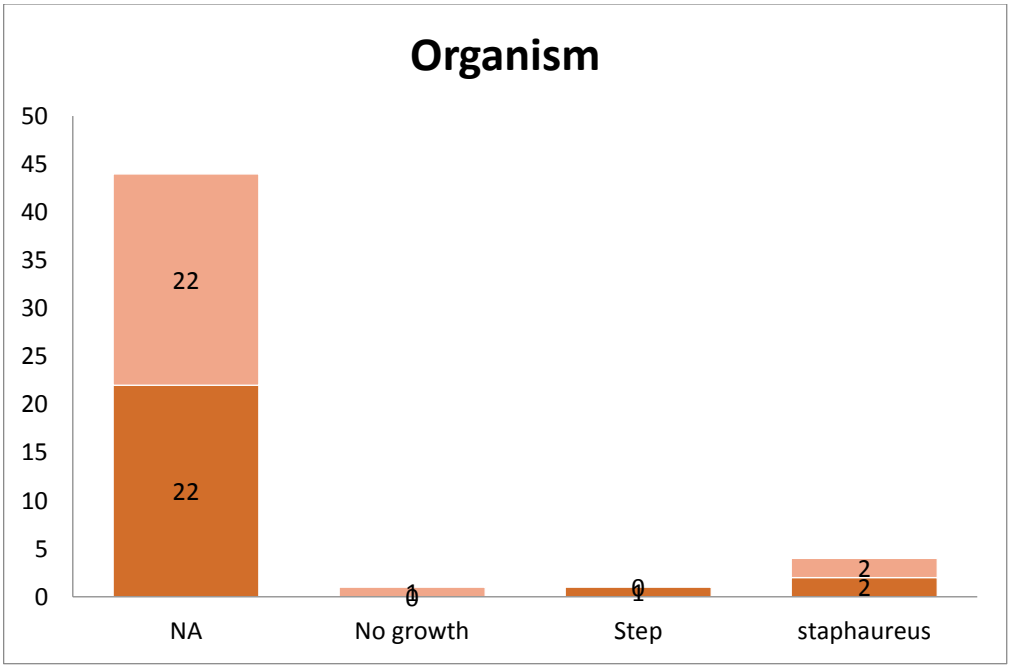


Table 9:

Organism	Control	Study
NA	23	22
No growth	0	1
Strep	1	0
Staph aureus	1	2
Total	25	25

Organism	Control	Study	Total	Chisq	P
NA	23	22	44	0.35	0.83
No growth	0	1	2		
Strep	1	0	1		
Staph aureus	1	2	4		
Total	25	25	50		

All the patients with wound discharge had pus culture and sensitivity of the discharge done. There was a predominance of staph aureus grown in the culture, of the 5 pts with organisms grown 4 were staph. Aureus. Group A streptococcal sp was grown in one case and there was no growth in another case.

This is in line with other studies where the predominant organism grown is staph aureus. Most of the strains were sensitive to cephalosporins except for one that was sensitive to piperacillin tazobactum.

SSI:

Graph 9:

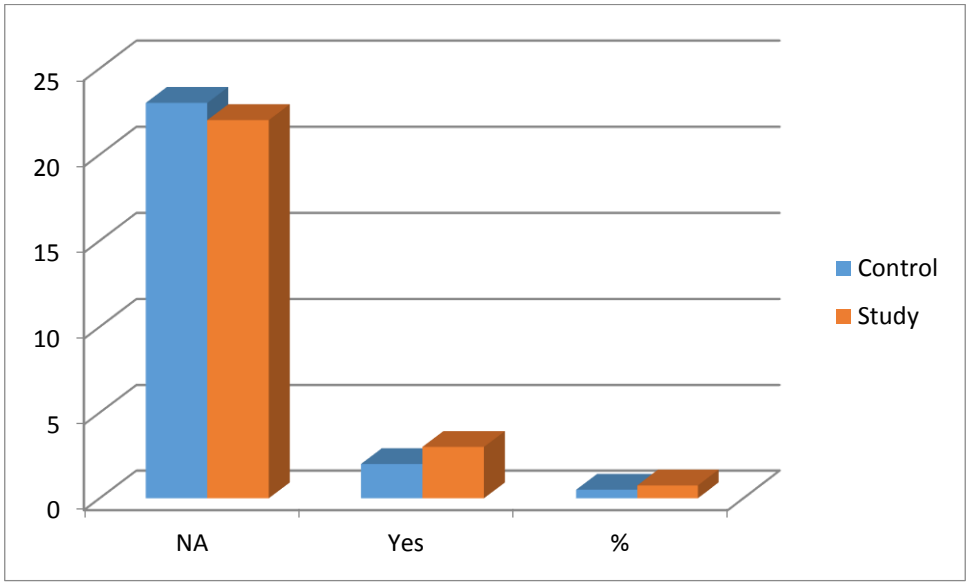


Table 10 :

SSI	Control	Study
NA	23	22
Yes	2	3
%	0.5	0.75
total	25	25

SSI	Control	Study	Total	Chisq	P
NA	23	22	45	0.22	0.6
Yes	2	3	5		
Total	25	25	50		

In the present study 2 out of control and 3 out of study group patients developed SSI's. All of them developed superficial SSI and none had deep SSI. The incidence of SSI in the present study was 0.5% in the control group and 0.75% in the study group. The difference is not significant as shown by the p value of 0.6.

MANAGEMENT:

Graph 10:

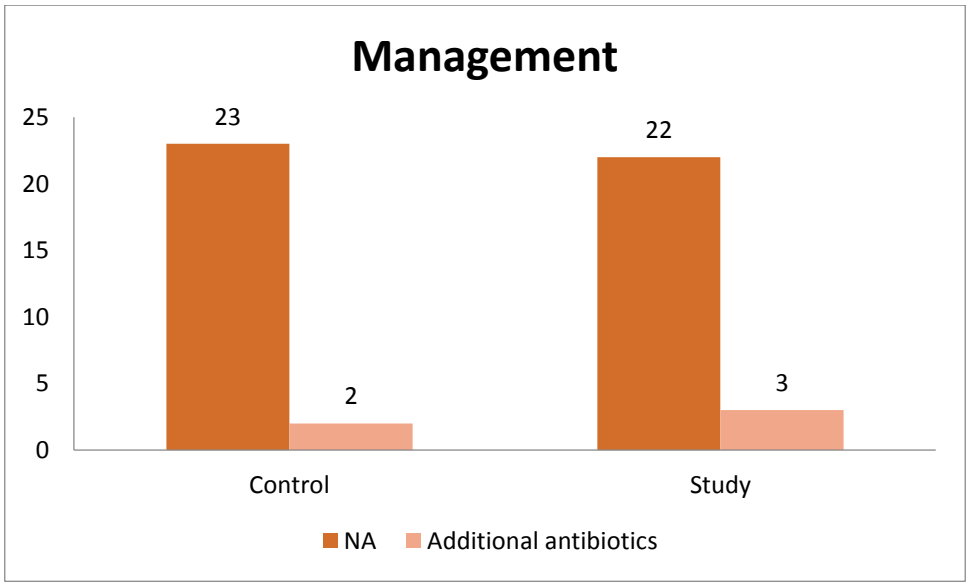


Table 11:

Management	Control	Study
NA	23	22
Additional antibiotics	2	3
Total	25	25

Management	Control	Study	Total	Chisq	P
NA	23	22	45	0.28	0.6
Antibiotics	2	3	5		
Total	25	25	50		

In the present study 2 patients in the control group and 3 patients in the study group developed SSI. All of them were managed with additional antibiotics. They were initially started on broad spectrum iv antibiotic like piperacillin tazobactum and based on culture and sensitivity reports antibiotics were changed. None of the patients in the study required any further intervention.

HOSPITAL STAY:

Graph 11:

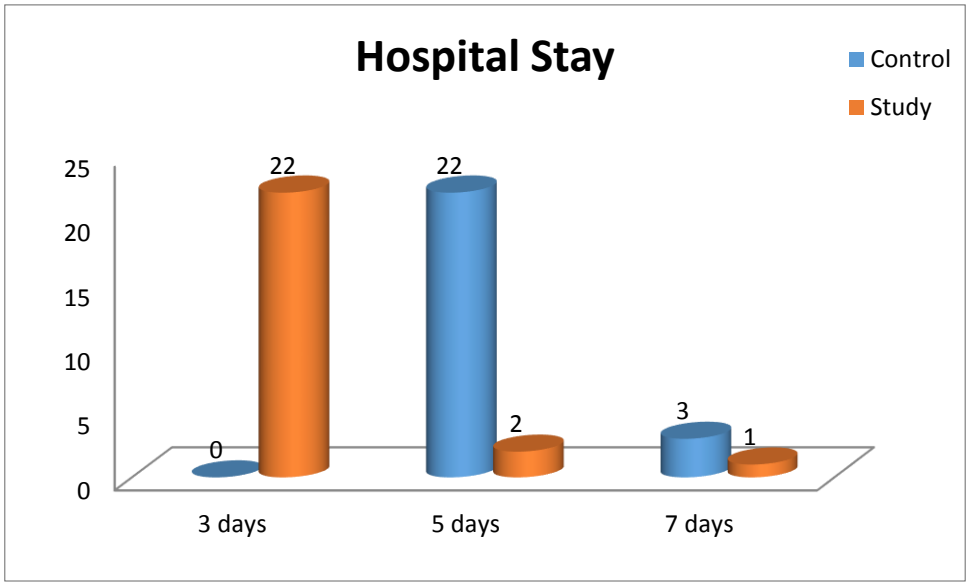


Table 12:

Hospital stay	Control	Study
3 days	0	22
5 days	22	2
7 days	3	1
Mean stay	5.24 days	3.32 days
Total	25	25

Hospital stay	Control	Study	Total	Chi sq	p	Correlation	
3	0	22	22	39.67	**0.0001**	-85%	
5	22	2	24				
7	3	1	4				
Total	25	25	50				

In the present study the mean duration of stay of patients in the control group was 5.24 days while it was 3.32 days for the patients in the control group. There was an significant reduction in hospital stay of -85% and the p value of 0.0001 again signifies significant association.

CLASS II(APPENDICECTOMY):

AGE:

Graph 12:

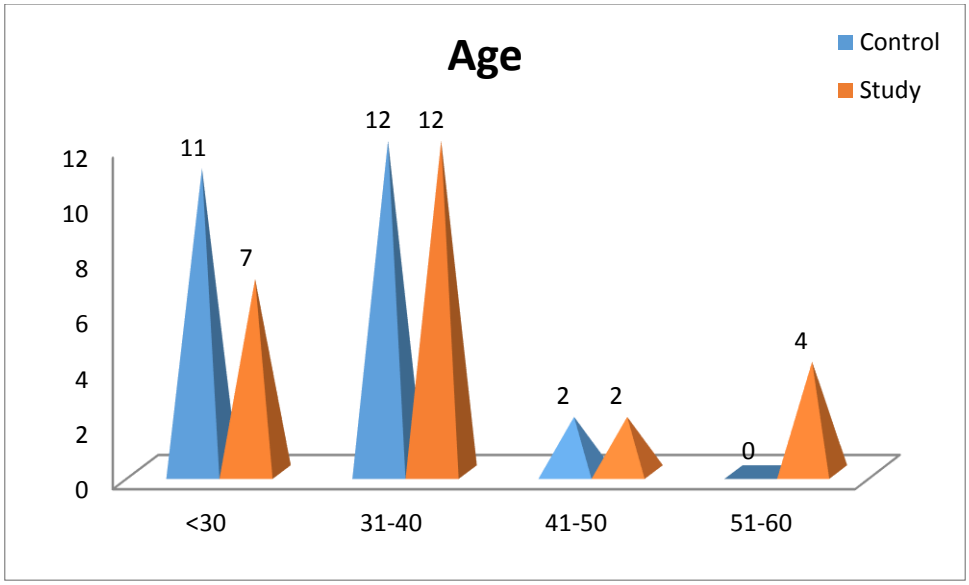


Table 13:

Age_r	Control	Study
<30	11	7
31-40	12	12
41-50	2	2
51-60	0	4
Total	25	25

Age_r	Control	Study	Total	Chi sq	p
<30	11	7	18		
31-40	12	12	24		
41-50	2	2	4	4.89	0.18
51-60	0	4	4		
Total	25	25	50		

In the present study the youngest patient was 19 years old and the oldest pt was 60 yrs old. Most of the patients were in the 31-40 age group there was no significant difference in age wise distribution of cases between the two groups as borne out by the p value of 0.18 which is not significant.

SEX:

Graph 13:

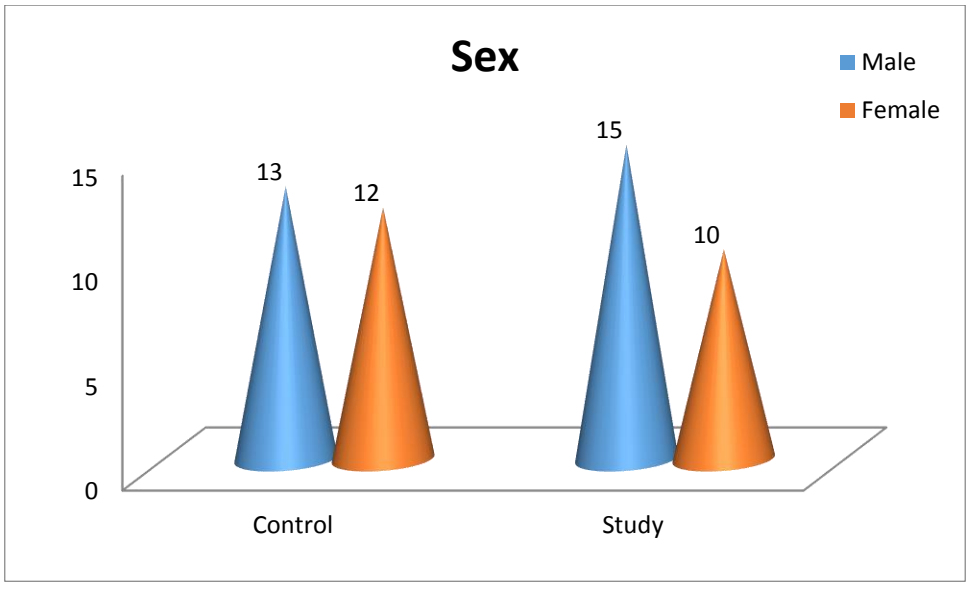


Table 14:

SEX	Control	Study
Male	13	15
Female	12	10
Total	25	25

Sex	Control	Study	Total	Chi sq	P
Male	13	15	28	0.32	0.5
Female	12	10	22		
Total	25	25	50		

In the present study there was a slight preponderance of male pts at 28 to 22. There was no significant difference in sex wise distribution of cases as made out by p value of 0.5 which is not significant.

FEVER:

Graph 14:

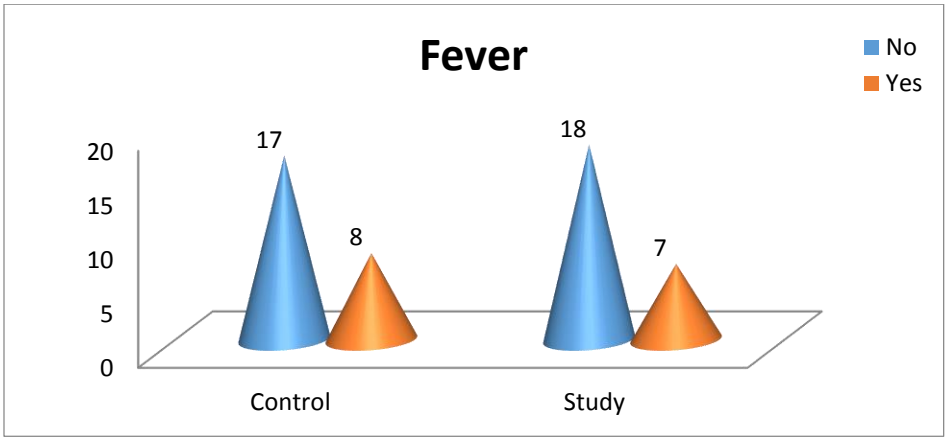


Table 15:

Fever	Control	Study
No	17	18
Yes	8	7
Total	25	25

Fever	Control	Study	Total	Chi sq	P
No	17	18	34	0.09	0.7
Yes	8	7	16		
Total	25	25	50		

In this study 8 out of control group and 7 out of study group developed post operative fever. There was no significant difference in occurrence of fever between the groups as shown by the p value of 0.7 which is not significant.

SWELLING:

Graph 15:

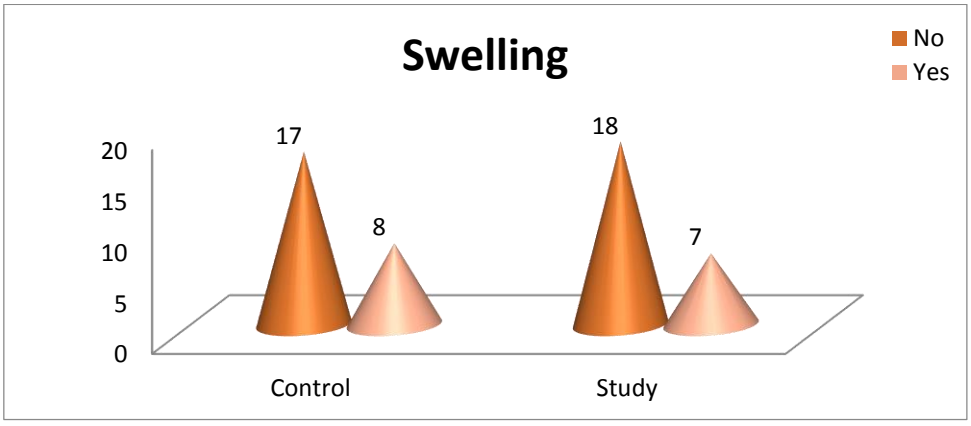


Table 16:

Swelling	Control	Study
No	17	18
Yes	8	7
Total	25	25

Swelling	Control	Study	Total	Chi sq	p
No	17	18	35	0.09	0.7
Yes	8	7	15		
Total	25	25	50		

In this study 8 out of control group and 7 out of study group developed swelling of the surgical site. There is no significant difference between the groups as shown by the p value of 0.7 which is not significant.

PAIN:

Graph 16:

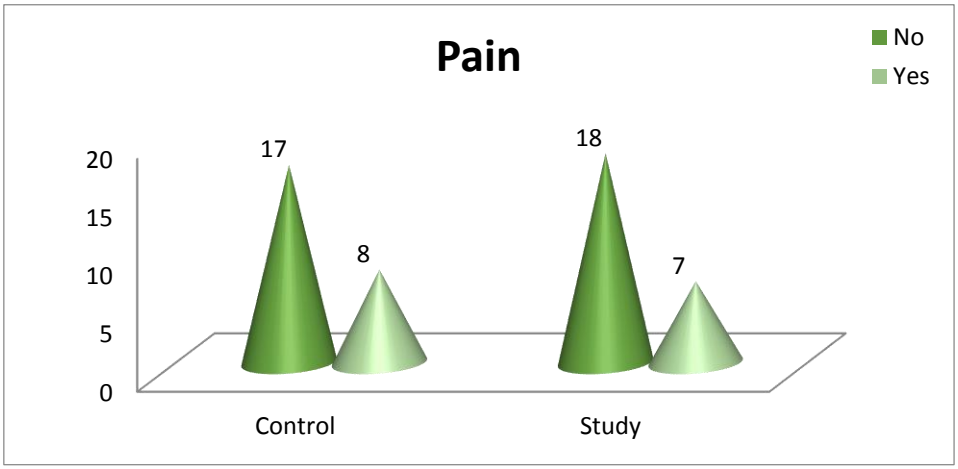


Table 17:

Pain	Control	Study
No	17	18
Yes	8	7
Total	25	25

Pain	Control	Study	Total	Chi sq	P
No	17	18	34	0.09	0.7
Yes	8	7	16		
Total	25	25	50		

In this study 8 out of control and 7 out of study group patients developed significant post operative pain in the surgical site. There was no significant differencde in the incidence of pain between both the groups as made out by p value of 0.7 which is not significant.

WOUND DISCHARGE:

Graph 17:

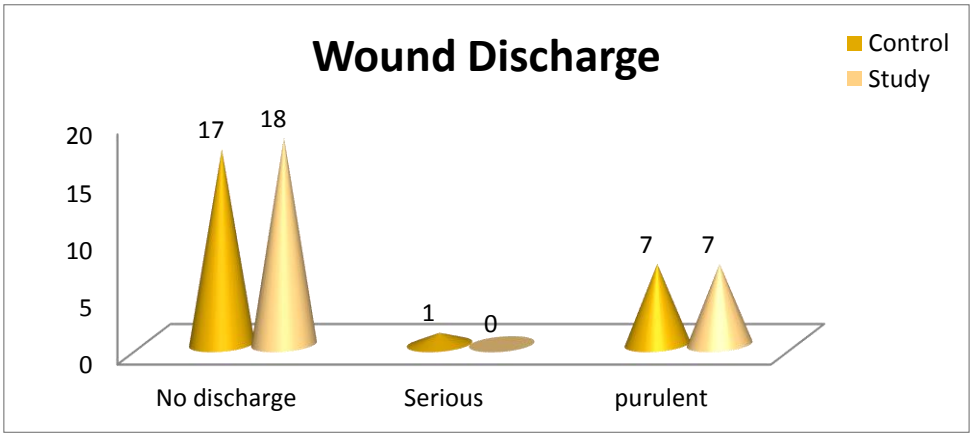


Table 18:

Wound Discharge	Control	Study
No discharge	17	18
Serious	1	0
purulent	7	7
Total	25	25

Wound Discharge	Control	Study	Total	Chi sq	P
No discharge	17	18	33	1.02	0.59
Serious	1	0	1		
purulent	7	7	14		
Total	25	25	50		

In this study 8 patients in the control group and 7 patients in the study group developed wound discharge. Of these majority of them developed purulent discharge except for 1 patient in the control group who developed serous discharge. There was no significant difference between the two groups in the occurrence of wound discharge as evidenced by the p value of 0.59 which is not significant.

ORGANISM:

Graph 18:

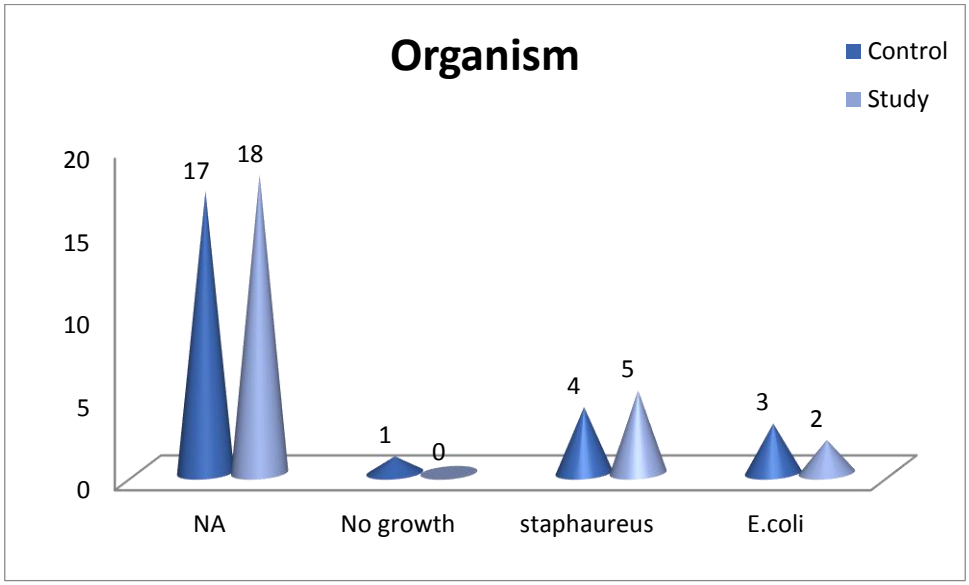


Table 19

Organism	Control	Study
NA	17	18
No growth	1	0
Staph aureus	4	5
E.coli	3	2
Total	25	25

Organism	Control	Study	Total	Chi sq	p
NA	17	18	33	1.33	0.71
No Growth	1	0	2		
s. aureus	4	5	10		
E.coli	3	2	5		
Total	25	25	50		

All the patients with wound discharge in the study had pus culture and sensitivity done. The predominantly grown organism was staph aureus 10 patients were infected by it followed by E.coli 5 patients were infected by it. All the strains were sensitive to cephalosporins and all the E.coli isolates were sensitive to Amikacin in addition . There was no significant difference between both the groups as shown by the p value of 0.71 which is not significant.

SSI:

Graph 19:

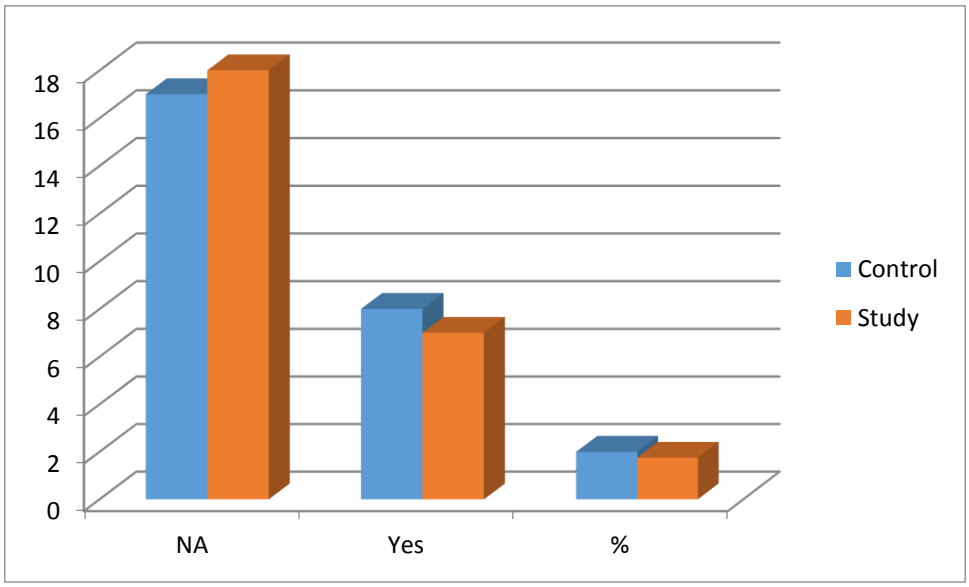


Table 20:

SSI	Control	Study
NA	17	18
Yes	8	7
%	2	1.75
total	25	25

SSI	Control	Study	Total	Chi sq	P
No	17	18	35	0.09	0.7
Yes	8	7	15		
Total	25	25	50		

In this present study 8 out of 25 pts in control group and 7 out of 25 pts in study group developed SSI. The incidence was 2%in control group and 1.75% in study group. There was no significant difference between the groups in the incidence of SSI between the groups it is also made out by the p value of 0.7 which is not significant.

MANAGEMENT:

Graph 20:

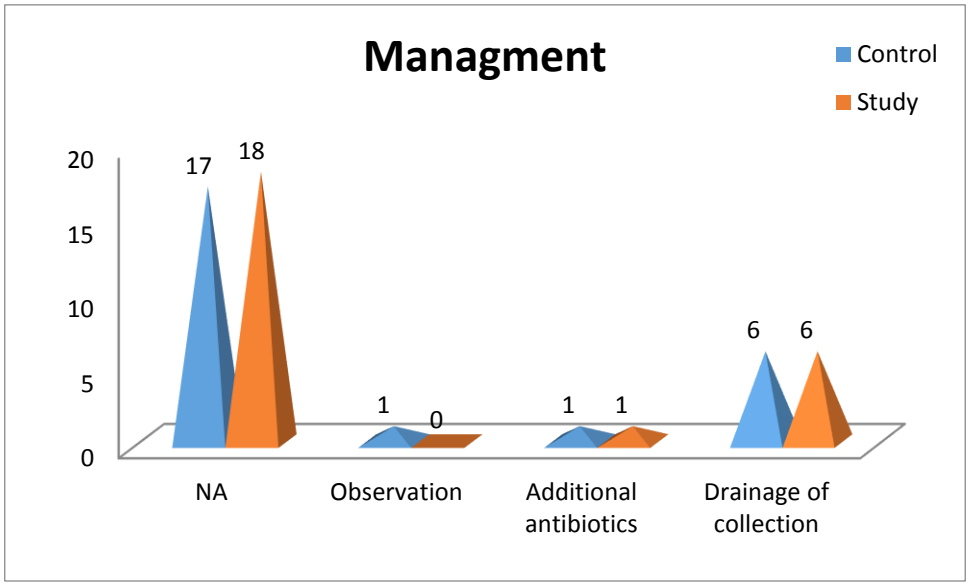


Table 21:

Management	Control	Study
NA	17	18
Observation	1	0
Additional antibiotics	1	1
Drainage of collection	6	6
Total	25	25

Management	Control	Study	Total	Chi sq	P
NA	17	18	35	1.02	0.79
Observation	1	0	1		
Additional antibiotics	1	1	6		
Drainage of collection	6	6	8		
Total	25	25	50		

Out of 8 patients who developed SSI in control group 6 patients were managed by drainage of infection while 1 patients infection resolved with antibiotics alone and 1 patient had minor infection that resolved spontaneously.

In the study group of the 7 patients with SSI 6 required drainage of collection while 1 patients infection resolved with antibiotics alone. There was no significant difference between the groups as shown by the p value of 0.79 which is not significant.

HOSPITAL STAY:

Graph 21:

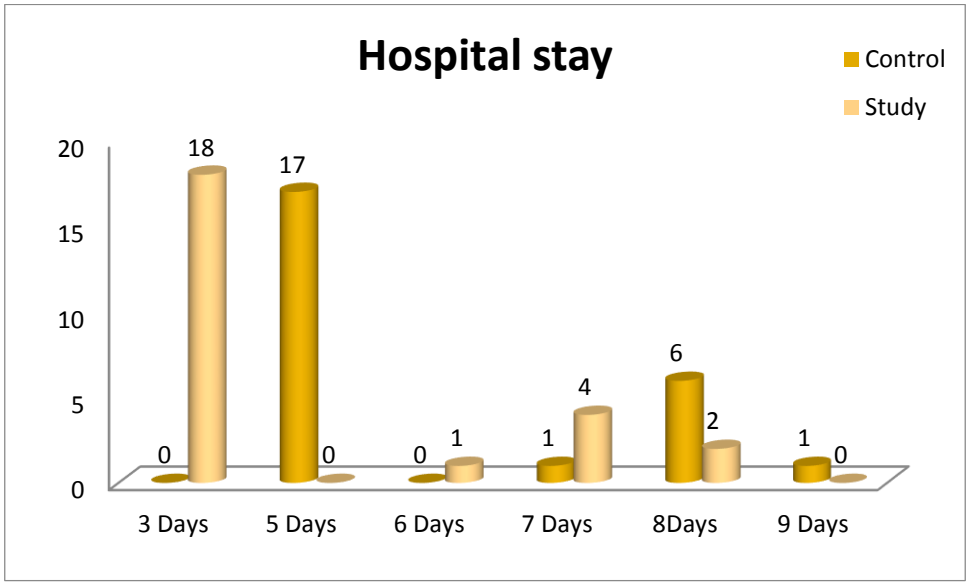


Table 22:

Hospital stay	Control	Study
3 Days	0	18
5 Days	17	0
6 Days	0	1
7 Days	1	4
8Days	6	2
9 Days	1	0
Mean stay	5.96 Days	3.52 Days
Total	25	25

Hospital stay	Control	Study	Total	Chi sq	P	Correlation	P
3 days	0	18	18	37.14	**0.0001**	-0.6	**0.001**
5 days	17	0	17				
6 days	0	1	1				
7 days	1	4	5				
8 days	6	2	8				
9 days	1	0	1				
Total	25	25	50				

In this present study the mean hospital stay was 5.96 days in the control group where as it was 3.52 days in the study group. On further analysis there was an 60% reduction in hospital stay associated with the study group. It is also borne out by the p value of 0.001 which is very significant.

DISCUSSION

The present study was conducted in the Department of General surgery Govt Chengalpattu Medical College from the period of November 2014 to August 2015. Proper institutional ethical committee clearance was obtained.

The study involved 100 cases of clean and clean contaminated cases randomized to groups of 50 each i.e. 25 study and control cases in each group. Cases were randomized to both the groups based on their presentation alternate cases were placed in both the groups.

For ease of comparison clean cases were restricted to Hernioplasty and clean contaminated cases were restricted to Appendicectomy. All the clean

cases in the study group received a single dose of inj. Ceftriaxone 1 gm i.v. given 30 minutes before the skin incision.

They received no further antibiotics i.v or oral. All the cases in the control group of clean group received 5 days of inj. Cefotxime 1 gm i.v. BD. All the cases in the study limb of clean contaminated group received a single dose of inj. Ceftriaxone 1 gm i.v and inj. Metronidazole 500 mg i.v 30 mins before skin incision. They received no further antibiotics i.v or oral with the exception of a single repeat dose if the surgery duration exceeded 3 hrs.

All the cases in the control group received 5 days of inj. Cefotaxime 1gm i.v BD and inj. Metronidazole 500 mg i.v TDS. The incidence of SSI was noted and results analysed.

INCIDENCE OF SSI IN CLEAN SURGERIES:

In my study the incidence of SSI was 0.5% in the control group and 0.75% in the study group which is not statistically significant as evidenced by the p value of 0.6 which is not significant. The incidence of SSI is comparable to the occurrence in other studies of similar nature.

Table 23:

study	%
Cruse et al.[61],	7%
Anne et al.,	0.59%
Muhammed Sharif et al.[69],	5.4%
Present Study	0.75%

DURATION OF HOSPITAL STAY:

In the present study the mean duration of stay of patients in the control group was 5.24 days while it was 3.32 days for the patients in the control group. There was an significant reduction in hospital stay of -85% and the p value of 0.0001 again signifies significant association.

CLEAN CONTAMINATED CASES:

In my study 8 out of 25 pts in control group and 7 out of 25 pts in study group developed SSI. The incidence was 2%in control group and 1.75% in study group. There was no significant difference between the groups in the incidence of SSI between the groups it is also made out by the p value of 0.7 which is not significant.

Table 24:

STUDY	%
Cruse et al.[61],	18%
Anne et al.,	2.6%
Muhammed Sharif et al.[69],	11.4%
Present Study	2.0%

The incidence of SSI in the study is comparable to the incidence in other studies of similar nature.

HOSPITAL STAY:

In my study the mean hospital stay was 5.96 days in the control group where as it was 3.52 days in the study group. On further analysis there was an 60% reduction in hospital stay associated with the study group. It is also borne out by the p value of 0.001 which is very significant.

CONCLUSION:

The study on prophylactic antibiotic for clean and clean-contaminated surgeries has led me to this conclusion.

1. Surgical site infection is the condition that may increase the morbidity and hospital stay of the patient. In severe cases may lead to loss of hospital resources, emergence of resistant bacteria, or may even lead to death of patient due to sepsis.

2. Risk factors for development of SSI, should be identified if present and patient factors like anemia, DM are to be corrected prior to surgery.

3. Local factors and microbial factors should be borne in surgeons mind and appropriate steps taken to avoid them.

4. When surgical site infection is diagnosed, wound / pus swab should be sent for culture sensitivity and antibiotic started on the basis of sensitivity to organism cultured.

5. Adequate drainage of pus in case of major infection should be encouraged, by release of one or more suture and planned for secondary closure once infection is controlled.

6. Surgical site infection with hospital acquired infection should be reduced by providing proper nursing case and proper surgical wards.

7. Prophylactic antibiotic with third generation cephalosporins should be given to patients and when appropriate additional antibiotics like Metronidazole can be added based on anticipated contaminants.

8. When given antibiotic prophylaxis must be given at least 30 minutes before skin incision and dose repeated if duration of surgery is prolonged.

9 Misuse of antibiotics should be avoided as it may lead to increased cost burden on patients, and increase the emergence of resistant microorganisms and also increase side effects seen with antibiotic usage.

10 In a resource deficit nation like ours implementation of single dose antibiotic prophylaxis regimes tailored to the prevalent organisms in the institution can result in enormous savings, as the study shows significant reduction in hospital stay with no significant increase in incidence of SSI and as a single dose of antibiotic is used the cost savings can also be enormous.

RECOMMENDATION:

Based on my study I would like to recommend single dose antibiotic prophylaxis using appropriate antibiotics for all class I and Class II cases, as per the study results there is no significant difference in incidence of SSI when compared to the traditional regimes with the added advantage of significant reduction in hospital stay, with its resultant savings in resources. In addition as the use of antibiotics is reduced it further results in increased cost effectiveness and reduces the incidence of complications due to antibiotic overuse.

BIBLIOGRAPHY

1. Lewis RT, Klein H. Risk factors and post operative sepsis : Significance of preoperative lymphocytopenia. J Surg Res 1975; 26 : 365-371.

2. Strachan CJ, Black JP. Prophylactic use of Cefazolin against sepsis after cholecystectomy British Journal of Medicine 1977; l: 1254-7.

3. Page CP, Bohnen JM, Fletcher JR et al. Antimierobial prophylaxis for surgical wounds: Guidelines for clinical care. Arch Surg 1993; 128 :79-88.

4. Breasted D. The Edwin Smith Surgical Papyrus. University of Chicago : University of Chicago Press ; 1930.

5. Bryan PW. The Papyrus Fbers. London / Washington DC : Government Printing Office ; 1883.

6. Cohen IK, A Brief history of wound healing Yardley, Pa : Oxgord clinical communications Inc ; 1998.

7. Falkow S. Molecular Koch's postulates applied to microbial Pathogenicity. Rev Infect Dis 1988 ; 10 (3) :5274.

8. Nuland SB. The Doctors Plague : Germs, Childbed Fever and the strange story of Ignaz Semmelweis New York : WW Norton and Co 2003.

9. Wangensleen OH, Klinser CF. Some Pre-Listerian and Post-Listerian antiseptic wound practices and the emergence of asepsis. Surg Gynecol Obstet 1973 ; 137:677.

10. Wangensteen OH, Wangensteen SD. Germ theory of infection and disease in Wangenteen OH, Wangensteen SD : the Risk of Surgery from Emperic Craft to Scientific Disipline. Minneapolis: University of Minnesota Press 1978: 387.

11. Taylor EW. Infection in surgical practice, Oxford Medical, Oxford 1992.

12. Seyrnour I, Schwartz, Sheres TS. Principles of Surgery, 6th ed. 151.

13. Howard RJ. Surgical infections. Surg Clin North Am 1988: 68 : 1.

14. Martone WJ, Nichols RL. Recognition, prevention, surveillance and management of surgical site infections. Clin infect Dis 2001 ;33 : 67.

15. Altemeier WA, Buske JF, Pruitt BA Jr (ed) : Manual on Control of Infection in Surgical Patients, 2nd ed. Philadelphia, Lippincoff 1984: 28.

16. Wijetunge DB. Management of acute and traumatic wounds : Main aspects of care in adults and children. Am. J. Surg. 1994 ; 167 : 56S-14S.

17. Atiyesh BS, Amam CA, El Musa KA. Improved scar quality following primary and secondary healing of cutaneous wounds. Aesthetic Plast Surg. 2003 ; 27 : 411-7.

18. Atiyesh BS, Ioannovich J, Al-Amm CA, El Musa KA, Dham R. Improving scar quality a prospective clinical study. Aesthetic Plast Surg. 2002 : 26 : 470-6.

19. Regan MC, Barbul A. The cellular biology of wound healing. Dec. 2007.

20. Wittmann F, Prix N, Mayr S et al., L – arginine improves wound healing after trauma - hemorrhage by increasing collagen synthesis. J Trauma 2005 ; 59 (1) : 162-8.

21. Thomas S. Wound management and dressings, London : Pharmaceutical Press, 1990.

22. Gottrup F. Wound closure techniques. J Wound care 1999 ; 8 (8) : 397-400.

23. Ehren K ranz NT. Surgical wound infection occurrence in class operations, risk stratification for inter hospital comparisons American Journal of Medicine 1981; 70 : 909-41.

24. Culver DH, Horan TC et al. Surgical wound infection rate by wound class, operative procedure and patient risk index. Am J Med I99l ;91 (38) : 1585.

25. Reid MR. Some considerations of the problem of wound healing N Engl J Med 1936 ; 215 : 753.

26. Sterenson JM and Reid MR. The fundamental principles of surgical techniques. Surgical Treatment of the Abdomen. Philadelphia JB Lippincott 1947.

27. Garibaldi RA, Cushing D, Lever T. Risk factors for post operative infection. Ann J Med 1991 ; 9t(38) : 1585.

28. Angus DC, Linde-Zwirble WT, Lidicer J et al. Epidermiology of severe sepses in the united states Crit care Med 2001 : 29 : 1303.

29. Perencevich EN, Sands KE, Lozgrove SE et al. Health and economic impact of surgical site infections diagnosed after hospital discharge merg infect Dis 2003 ; 9 ;196.

30. Morrell S, Saffle JR, Larson C et al. The declining incidence of fatal sepsis following thermal injury. J Trauma l989 ; 29 :1362.

31. Dovi JV, He LK, DePietro LA. Accelerated wound closure in neutrophil-depleted mice. J Leukoc Brol 2003; 73:448.

32. Ahrenholz DH, Simmons RL. Mixed and synergstic infection, in Howard RJ, Simmons RL (eds) : Surgical Infectious diseases, 2nd ed, Norwalk, CT, Appelton and Lange, 1991 ; Chapt. 2, P. 187.

33. Brook I. A 12 year study of aerobic and anaerobic bacteria in intra abdominal and post-surgical abdominal wound infection. Surg Gynecol BOstet 1989; 196: 387.

34. Howard RJ. Viruses, in Howard RJ, (eds) Surgical infectious diseases, 2nd ed NOrwalk, CT, Appelton and Lange, 1991, p. 35.

35. Mims CA, Dimmock NJ, Nash A, Stephen J. Mims. Patho-physiology of infectious diseases, 4th edn. Academic press, London 1995.

36. John M, Kissane. Andersons' Pathology, 9th edition, 1990, volume 1, Inflammation and healing. pg. 84-87.

37. Valles J, Rello J, Ochagavra A. et al. Community acquired blood stream infection in critically ill patients. chest 2003; 123: 1615.

38. Centers for Disease control : Nosocomral infection rates for interhostpial comparison : Limitation and possible solution. Infect Control Hosp Epidemol I 991 : 12 : 609.

39. Bozzotti F, Terno G, et al. Prevention and treatment of central nervous catheter sepsis by exchange via a guide wire. A prospective controlled trial. Ann Surg 1983; 198: 48.

40. Altemeier WA, Burke JF. Manual on control of infection in surgical patients. Philadelphia, JB Lipincott, 1984, p. 307.

41. Howard RJ. Microbes and their pathogenicity. In Simmons, R.L. (eds) : Surgical infection diseases, New York, Appelton- Century - Crafts, 1983, p. 11.

42. Charles F. Brunicardi. Schwartz's Principles of Surgery. 8th edition, 2005, Surgical Infections p. 119, Table 5-7.

43. Olson M, O'Connor M Schwartz ML. Surgical wound infection : A 5 year prospective study of 20, 193 wound at the Minneapolis VA Medical Center. Ann Surg 1984; 1999: 253.

44. Laufman H. Current status of special air handling systems in operating rooms. Med Instrum 1973; 7 : 7.

45. Halsted WS. The employment of fine silk in preference to catgut and the advantage of transfixing tissues and vessels in controlling hemorrhage. JAMA 1913;60:1119.

46. Mangram AJ, Nichols RL. Recognition, preparation, surveillance and management of surgical site infection. Hospital infection control practices advisory Committee. Infect Control Hosp Epidermol 1999; 20:250.

47. Peter J. Morris, Ronald A. Mact. Wound infection, Oxford Text Book of Surgery. Vol. 1 p. 27-43. 1994.

48. Mackie, McCartney. Practical Medical Microbiology : 13th edition, Wound, Skin and deep sepsis p. 619.

49. Bilton BD, Zibai GB, McMillan RW et al. Aggressive Surgical management of necrotizing fascitis serves to decrease mortality : A retrospective study Ann Surg 1998; 64:397.

50. Collee JG, Dugurd JP, Fraser AG. Mackle and McCartney practical medical microbiology: 13th edition : Laboratory strategy in Diagnosis 1989 ; 621-623.

51. Ronald Lede Nichols - 'Bacteriology in Surgery'. Mastery of Surgery. Nyhen and Baker, 2nd edition, vol. 1, 83-93.

52. Good Man and Gilman's. The pharmocological. Basis of therapeutics: tenth edition : Antimicrobial agents: General consideration 2001 ;1164.

53. Schwartz's principles of surgery / Basic considerations, 8th edition wound Healing 2005 ; 226.

54. Keighiey MRB, Burdon DW. Antimicrobial prophylaxis in Surgery. Pitman, London 1979.

55. Bohnen JMA, Solomkin JS et al., Guidelines for clinical care : Anti-infective agents for intra-abdominal infection. A surgical infection Society Policy Statement. Arch Surg 1992 ; 127 :83.

56. Kaiser A B. Antimicrobial prophylaxis in surgery. N Engl J Med 1986;315 : 1121.

57. Jauber MG, Kunz S. Influence of antibiotic dose, dosing interval and duration of therapy on outcome in experimental pneumococcal Meningitis Antimicrob Agents Chemotherapy 1989; 33 : 418-423.

58. The choice of antibacterial drugs Med Lett Drugs Ther 1999; 41 : 95-104.

59. Rao AS, Harsha M. Post operative wound infection. J India Med Assoc 1975 ; 44 :90-3.

60. Chowdary A, Jangale N. Department of Microbiology Gant Medical college, J & S Hospital, Byculla, Mumbai 1997.

61. Cruise PJE and Foord R. A five year prospective study of 23,649 surgical wounds' Archives of surgery 1913 ;107 :206.

62. Lilani, Jangale N. Department of microbiology, Department of surgery, Grand medical college, Byculla, Mumbai Indian J Surg 1997 ;90-3.

63. Mangram AJ, Horan TC. The hospital infection control practices advisory committee Guideline for prevention of SSI, Infect control Hosp Epidemol 1999 ; 20:241-78.

64. Olson MM, Lee JT. Continuous l0 year wound infection surveillance results advantages and unanswered questions Arch Surg 1990 ; 125: 794-803.

65. Funary AP, Zerc KJ, Grunkemeier GC, Starr A. Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. Ann Thorac Surg 1999; 67 :352-360.

66. Hamilton HW, Hamilton KR, Lone FJ. preoperative hair removal. The Canadian Journal of Surgery 1997 ; 20 :269-275.

67. Bull Lowburg, Lilly. Methods of disinfection of hands and operative site. British Journal of Medicine 1964; 2 : 531.

68. Carlson GE, Gonnlanakis C, Tsatsakis A. Pre-incisional single dose ceftriaxone for prophylaxis of surgical wound infection, American Journal of Surgery 1995 ; 170 (4) : 353-5.

69. Mohammed Sharif Auran, Dept. of Surgery, Peoples Medical College and Hospital, Nawabshah, J. of Surg. Pakistan. Jan-March. 2001 ; 16 (1).

CONSENT FORM

For Inclusion in study "**COMPARATIVE STUDY OF SINGLE DOSE PROPHYLACTIC ANTIBIOTIC VERSUS EMPIRICAL POSTOPERATIVE ANTIBIOTICS IN PREVENTION OF SSI".**

I _____ Hosp. No. ____ _____ in my full Senses hereby give my complete consent for _____ or any other Procedure deemed at which is a / and diagnostic procedure / biopsy /Transfusion/operation to be performed on me / my ward _____ age _____ under any anesthesia deemed fit. The nature and risks Involved in the procedure have been explained to me to my satisfaction. For academic and scientific purpose, the operation / procedure may be Televised or photographed.

Date:

Signature / Thumb
Impression
Name:
of patient / Guardian
Designation:

PROFORMA

Name :

Age :

Address :

Socioeconomic Class :

OP/IP No. :
Phone No:

Presenting complaints:

H/O Present illness:

Past History:

Treatment History:

Family History:

Personal History:

O/E Conscious oriented

Febrile yes/no
Pallor yes/no
Pedal edema/no pedal edema,
Icterus yes/no
Clubbing yes/no
Pulse Rate

Height
Weight
Body Mass Index
cyanosis yes/no

CVS :

RS :

CNS :

P/A :

INVESTIGATIONS :

BLOOD INVESTIGATIONS :

HB :

TC :

DC :

ESR :

PLATELET COUNT :

HIV :

HBSAG :

IMAGING :

CXR PA VIEW :

AXR ERECT :

USG ABDOMEN :

CT ABDOMEN :

DIAGNOSIS :

CLASSIFICATION :

CLASS I/CLASS II :

CONTROL/STUDY :

TREATMENT GIVEN :

POST OPERATIVE COMPLICATIONS:

FEVER : Yes/No

SWELLING	:	Yes/No
PAIN	:	Yes/No
WOUND DISCHARGE	:	Yes/No
ORGANISM GROWN	:	
SSI	:	Yes/No
CLASSIFICATION	:	Superficial/Deep
MANAGEMENT	:	
FOLLOWUP	:	

MASTER CHART CLASS 1 CASES (HERNIOPLASTY) CONTROL GROUP

S. No	Ip no	Age	Sex	Inguinal Hernia	Post of complications						Management	Hospital Stay in Days
					Fever	Swelling	Pain	Wound Discharge	Organism	SSI		
1	42553	31	M	LT	1	1	1	1	NA	NA	NA	5
2	43366	32	M	LT	1	1	1	1	NA	NA	NA	5
3	43713	35	M	RT	1	1	1	1	NA	NA	NA	5
4	44202	20	M	LT	1	1	1	1	NA	NA	NA	5
5	44362	52	M	RT	1	1	1	1	NA	NA	NA	5
6	43704	64	M	RT	1	1	1	1	NA	NA	NA	5
7	43707	70	M	RT	1	1	1	1	NA	NA	NA	5
8	43348	30	M	RT	1	1	1	1	NA	NA	NA	5
9	45849	38	M	LT	1	1	1	1	NA	NA	NA	5
10	45833	36	M	LT	1	1	1	1	NA	NA	NA	5
11	45967	49	M	LT	1	1	1	1	NA	NA	NA	5
12	46406	60	M	LT	1	1	1	1	NA	NA	NA	5
13	45841	48	M	B/L	1	1	1	1	NA	NA	NA	5
14	46593	40	M	B/L	2	2	2	3	2	1	2	7

15	47291	48	M	RT	1	1	1	1	NA	NA	NA	5
16	46139	50	M	RT	1	1	1	1	NA	NA	NA	5
17	47333	56	F	RT	1	1	1	1	NA	NA	NA	5
18	47875	56	M	RT	1	1	1	1	NA	NA	NA	5
19	48178	65	M	B/L	1	1	1	1	NA	NA	NA	5
20	51423	60	M	B/L	1	1	1	1	NA	NA	NA	5
21	44587	55	M	LT	2	2	2	3	3	1	2	7
22	4004	60	M	LT	1	1	1	1	NA	NA	NA	5
23	4302	50	M	RT	1	1	1	1	NA	NA	NA	5
24	4091	60	M	RT	1	1	1	1	NA	NA	NA	5
25	4949	62	M	LT	1	1	1	1	NA	NA	NA	5

MASTER CHART CLASS 1 CASES (HERNIOPLASTY) STUDY GROUP

S. No	Ip no	Age	Sex	Inguinal Hernia	Post of complications						Management	Hospital Stay in Days
					Fever	Swelling	Pain	Wound Discharge	Organism	SSI		
1	43327	60	M	RT	1	1	1	1	NA	NA	NA	3
2	43346	23	M	LT	1	1	1	1	NA	NA	NA	3
3	44248	28	M	LT	1	1	1	1	NA	NA	NA	3
4	42166	51	M	LT	1	1	1	1	NA	NA	NA	3
5	44039	35	M	LT	1	1	1	1	NA	NA	NA	3
6	43348	30	M	RT	1	1	1	1	NA	NA	NA	3
7	44238	50	M	RT	1	1	1	1	NA	NA	NA	3
8	43463	45	M	LT	2	2	2	2	1	1	2	5
9	43046	46	M	LT	1	1	1	1	NA	NA	NA	3
10	45715	60	M	RT	1	1	1	1	NA	NA	NA	3
11	46278	66	M	RT	1	1	1	1	NA	NA	NA	3
12	46601	60	M	RT	1	1	1	1	NA	NA	NA	3
13	46809	18	M	RT	1	1	1	1	NA	NA	NA	3
14	47326	41	M	RT	1	1	1	1	NA	NA	NA	3
15	47504	29	M	RT	1	1	1	1	NA	NA	NA	3
16	47638	54	M	RT	2	2	2	3	2	1	2	7

17	49187	25	M	LT	1	1	1	1	NA	NA	NA	3
18	51142	33	M	LT	1	1	1	1	NA	NA	NA	3
19	920	61	M	RT	1	1	1	1	NA	NA	NA	3
20	1967	58	M	RT	2	2	2	3	2	1	2	5
21	3742	65	M	B/L	1	1	1	1	NA	NA	NA	3
22	3860	47	M	LT	1	1	1	1	NA	NA	NA	3
23	4936	58	M	RT	1	1	1	1	NA	NA	NA	3
24	5654	44	M	RT	1	1	1	1	NA	NA	NA	3
25	5490	45	M	RT	1	1	1	1	NA	NA	NA	3

MASTER CHART CLASS 2 (APPENDICECTOMY)CONTROL GROUP

S. No	Ip no	Age	Sex	post of complications					Surgical Site Infection	Management	Hospital Stay in Days
				Fever	Swelling	Pain	Wound Discharge	Organism			
1	45128	26	M	1	1	1	1	NA	NA	NA	5
2	49379	37	F	1	1	1	1	NA	**NA**	NA	5
3	2892	18	**M**	2	2	2	2	**1**	1	1	7
4	5724	28	**M**	1	1	1	1	**NA**	**NA**	NA	5
5	640	19	**M**	2	2	2	3	2	1	2	8
6	5731	20	**M**	1	1	1	1	NA	**NA**	NA	5
7	16117	23	**M**	1	1	1	1	NA	NA	NA	5
8	18647	26	**M**	2	1	2	3	3	1	2	8
9	27698	22	**M**	1	1	1	1	NA	NA	NA	5
10	42699	30	**F**	1	1	1	1	NA	NA	NA	5

11	47012	28	**F**	2	2	2	3	2	1	2	9
12	49444	20	**F**	1	1	1	1	NA	NA	NA	5
13	5590	19	**M**	1	1	1	1	NA	NA	NA	5
14	9151	25	**F**	1	1	1	1	NA	NA	NA	5
15	16307	18	**F**	2	2	2	3	3	1	2	8
16	45114	19	**F**	2	2	2	2	1	1		7
17	48881	19	**F**	1	1	1	1	NA	NA	NA	5
18	8938	22	**F**	1	1	1	1	NA	NA	NA	5
19	14647	18	**M**	2	2	2	3	2	1	3	8
20	25382	18	**M**	1	1	1	1	NA	NA	NA	5
21	26606	30	**F**	1	1	1	1	NA	NA	NA	5
22	29826	23	**M**	2	2	2	3	2	1		

23	31827	40	**F**	1	1	1	2	3	1	2	7
24	11978	24	**F**	1	1	1	1	NA	NA	NA	5
25	16991	20	**M**	2	2	2	3	2	1	3	8

MASTER CHART CLASS 2(APPENDICECTOMY) STUDY GROUP

S. No	**Ip no**	**Age**	**Sex**	**Post of Complications**					**Surgical Site Infection**	**Management**	**Hospital Stay in Days**
				Fever	**Swelling**	**Pain**	**Wound Discharge**	**Organism**			
1	48952	28	F	1	1	1	1	NA	NA	NA	3
2	4998	45	M	1	1	1	1	NA	NA	NA	3

	8										
3	3528	25	F	1	1	1	1	NA	NA	NA	3
4	50161	20	M	2	2	2	3	2	1	2	7
5	972	18	M	1	1	1	1	NA	NA	NA	3
6	5724	23	M	1	1	1	1	NA	NA	NA	3
7	18236	35	F	2	2	2	3	3	1	3	8
8	20754	18	M	1	1	1	1	NA	NA	NA	3
9	2870	27	F	1	1	1	1	NA	NA	NA	3
10	44311	20	M	1	1	1	1	NA	NA	NA	3
11	40986	24	M	2	2	2	3	2	1	3	7
12	2697	21	F	1	1	1	1	NA	NA	NA	3
13	7661	42	M	1	1	1	1	NA	NA	NA	3
14	9083	22	M	2	2	2	3	2	1	3	7
15	21701	18	M	1	1	1	1	NA	NA	NA	3

16	46814	18	M	1	1	1	1	NA	NA	NA	3
17	5025	46	M	2	2	2	3	3	1	3	7
18	9995	18	F	1	1	1	1	NA	NA	NA	3
19	18213	32	M	2	2	2	3	2	1	3	8
20	26252	30	M	1	1	1	1	NA	NA	NA	3
21	27417	45	F	1	1	1	1	NA	NA	NA	3
22	29800	28	F	2	2	2	3	2	1	3	6
23	10853	23	F	1	1	1	1	NA	NA	NA	3
24	14247	30	F	1	1	1	1	NA	NA	NA	3
25	27698	22	M	1	1	1	1	NA	NA	NA	3

KEY WORDS

1. SSI,
2. Surgical Site Infection,
3. Single Dose Antibiotic Prophylaxis,
4. Post operative Antibiotics,
5. Class I surgeries,
6. Class II surgeries,
7. Hernioplasty,
8. Open Appendicectomy
9. Post operative wound Infection.