

**“A COMPARATIVE STUDY OF SINGLE DOSE
PREOPERATIVE CEFTRIAZONE AND ROUTINE
CONVENTIONAL POSTOPERATIVE PROPHYLAXIS IN
ELECTIVE GENERAL SURGICAL CASES”**

A DISSERTATION SUBMITTED TO



THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

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



M.S. GENERAL SURGERY – BRANCH I

DEPARTMENT OF GENERAL SURGERY

REGISTER NUMBER : 221711312

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

MAY 2020

CERTIFICATE

Certified that this is the bonafide dissertation done by **DR.NAVEEN KUMAR.M** and submitted in partial fulfilment of the requirement for the Degree of M.S. General Surgery, Branch I of the Tamilnadu Dr. M.G.R. Medical University, Chennai.

DATE:

UNIT CHIEF

DATE:

PROFESSOR & HOD

DEPARTMENT OF GENERAL SURGERY

DATE:

DEAN

COIMBATORE MEDICAL COLLEGE

COIMBATORE – 641014

**INSTITUTIONAL HUMAN ETHICS COMMITTEE
COIMBATORE MEDICAL COLLEGE, COIMBATOR - 14**

EC Reg No. ECR/892/Inst/TN/2016
Telephone No: 0422 - 2574375/76
Fax : 0422 - 2574377

CERTIFICATE OF APPROVAL

To
Dr.Naveen Kumar M
Post Graduate,
Department of General Surgery,
Coimbatore Medical College & Hospital
Coimbatore -18.

Dear **Dr.Naveen Kumar M**

The Institutional Ethics Committee of Coimbatore Medical College, reviewed and discussed your application for approval of the proposal entitled "**A Comparative study of single dose pre - operative Ceftriaxone versus Routine Conventional Post - operative Prophylaxis in Elective General Surgical Cases in Coimbatore Medical College, Coimbatore.**"No.0103/2017.

The following members of Ethics Committee were present in the meeting held on 28.11.2017, conducted at MM - II Seminar Hall, Coimbatore Medical College Hospital Coimbatore-18

1	Dr.S.Ramalingam MD, Dean, PSG IMS&R, Cbe	Chairman
2	Dr.Usha MD., Professor of General Medicine, CMCH, Cbe	Member Secretary
3	Dr.R.Manonmani MD., Professor of O&G, CMCH, Cbe	Clinicians
4	Dr.N.Renganathan MS., Professor of General Surgery, CMCH,Cbe	Clinicians
5	Dr.Sudha Ramalingam MD., Professor of SPM, PSG IMS&R, Cbe	Clinicians
6	Dr.R. Shanmugavadivu MD., Professor of Physiology, CMC, Cbe	Basic Medical Scientist
7	Dr.N. Shanthy MD., Professor of Pharmacology, CMC, Cbe	Basic Medical Scientist
8	Dr.A.Dhanalakshmi MD., Assoc. Professor of Pathology, CMC,Cbe	Basic Medical Scientist
9	Dr.L.Madhan MD., Professor of Pharmacology, CMC, Cbe	Basic Medical Scientist
10	Dr.N.Paramasivan MD., Professor of Pharmacology, Sri Ramakrishna Dental College, Coimbatore	Basic Medical Scientist
11	Mrs.A.Sharmila BA., BL., Advocate	Legal Expert
12	Dr.K.P.Sampath Kumar M.Pharm, Ph.D., Asst. Prof. of Pharmacy, CMC, Cbe	Scientific Member
13	Dr.G.Vani Ganesh M.Sc.,Ph.D., Tutor in Medical Surgical Nursing, CMCH, Cbe	Scientific Member
14	Mr.V. Balasubramani MA,MA,MBA,LLB,M.Phil,PG.D.M, DLLAL, Chief Executive, Avinashilingam JSS Self Finance Courses, Cbe	Social Worker
15	Mr.V.A.Shahul Hameed, +2	Lay-Person

We approve the Proposal to be conducted in its presented form.

Sd/Chairman & Other Members

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

Member Secretary
**INSTITUTIONAL HUMAN ETHICS COMMITTEE
COIMBATORE MEDICAL COLLEGE
COIMBATORE - 641 014.**

Urkund Analysis Result

Analysed Document: NAVEEN THESIS EDITED.docx (D56885839)
Submitted: 10/11/2019 6:35:00 PM
Submitted By: nicksongerrald1129@gmail.com
Significance: 2 %

Sources included in the report:

<https://academic.oup.com/bja/article/119/1/13/3897055>
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4702440/>
<https://www.egms.de/static/en/journals/dgkh/2006-1/dgkh000015.shtml>
<https://academic.oup.com/jac/article-pdf/41/5/501/9837952/410501.pdf>
<https://www.ncbi.nlm.nih.gov/pubmed/15082963>
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5738868/>
<https://www.elsevier.es/en-revista-cirugia-espanola-english-edition--436-articulo-prevention-surgical-site-infection-in-52173507714001719>
<https://cbc.org.br/wp-content/uploads/2017/03/032017WJS.pdf>
[https://www.rjpbcs.com/pdf/2017_8\(2\)\[122\].pdf](https://www.rjpbcs.com/pdf/2017_8(2)[122].pdf)
<https://www.science.gov/topicpages/s/surgical+site+infection.html>
<https://ijbamr.com/pdf/December%202016%20343-354.pdf.pdf>

Instances where selected sources appear:

14

DECLARATION

I solemnly declare that the dissertation titled “**A COMPARATIVE STUDY OF SINGLE DOSE PREOPERATIVE CEFTRIAZONE AND ROUTINE CONVENTIONAL POSTOPERATIVE PROPHYLAXIS IN ELECTIVE GENERAL SURGICAL CASES**” was done by me from 2018 onwards under the guidance and supervision of **DR. V. LEKSHMINARAYANI , M.S , D.G.O.**

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfilment of the requirement for the award of M.S Degree in General Surgery (Branch I).

PLACE:

DR. NAVEEN KUMAR.M

DATE:

REGISTER No: 221711312

ACKNOWLEDGEMENT

I owe my reverential gratitude and humble thanks to Lord God Almighty for all his mercy, for being with me and showering abundant blessing upon me throughout the course of the study.

I am obliged to record my immense gratitude to **DR. B. ASOKAN MCh**, The Dean, Coimbatore Medical College Hospital for providing all the facilities to conduct the studies.

I express my deep sense of gratitude and heartfelt thanks to **Prof. DR. A.NIRMALA, M.S**, Head of Department of General Surgery for his dynamic guidance, constant help and encouragement throughout the study.

I express my respectful gratitude and indebtedness to my guide **Prof .DR. V. LEKSHMINARAYANI , M.S, D.G.O.** for her valuable guidance and support.

I would like to express my sincere thanks to Prof Dr.Srinivasan M.S, Prof Dr. V.S. Venkadesan, M.S,D.A, Prof Dr.Narayanamoorthy ,M.S , Prof Dr. D.N. Renganathan, M.S, Prof DR. V. Elango , M.S.

I deeply thank Dr.Jayakumar M.S., Dr.Radhika M.S., Dr.P.Sumitra M.S Assistant professors of surgery and Dr.Ramyalakshmi

MS PG, Dr. Vivek MS PG, Dr Santhosh MS PG for all the needful help they have provided for the study.

I acknowledge my gratitude to our Registrar Dr.Ravi M.S and all my assistant professors of Department of surgery for their encouragement and support.

I am thankful to **The ETHICAL COMMITTEE** of Coimbatore Medical College for permitting me to proceed with this dissertation.

Lastly I am grateful to all the patients whose cooperation made this work possible.

DATE:

SIGNATURE OF THE CANDIDATE

PLACE:

Dr. NAVEEN KUMAR.M

TABLE OF CONTENTS

S.NO	CONTENTS	PAGE NO
1.	INTRODUCTION	3
2.	REVIEW OF LITERATURE	5
3.	MATERIALS & METHODS	51
4.	OBSERVATION AND ANALYSIS	55
5.	CONCLUSION	92
6.	RECOMMENDATION	93
7.	BIBLIOGRAPHY	
8.	ANNEXURES	
9.	CONSENT FORM	
	PROFORMA	
	MASTER CHART	

ABBREVIATIONS

BT	:	Bleeding Time
CDC	:	Centres for Disease Control
CXR	:	Chest X-ray
CT	:	Clotting Time
CMV	:	Cytomegalovirus
CO2	:	Carbon dioxide
CVS	:	Cardiovascular System
DC	:	Differential Count
DM	:	Diabetes Mellitus
ESR	:	Erythrocyte Sedimentation Rate
FBS	:	Fasting Blood Sugar
GIT	:	Gastrointestinal Tract
GUT	:	Genitourinary Tract
Hb	:	Haemoglobin
HT	:	Hypertension
HIV	:	Human Immuno Deficiency Virus

IV	:	Intra Venous
LFT	:	Liver Function Test
MRSA	:	Methicillin Resistance Staphylococcus Aureus
MIC	:	Minimum inhibitory concentration
PA	:	Per Abdomen
RBS	:	Random Blood Sugar
RS	:	Respiratory System
RR	:	Respiratory Rate
SSI	:	Surgical Site Infection
SPO2	:	Partial Pressure of Oxygen
TC	:	Total Count
UTI	:	Urinary Tract Infection
USG	:	Ultrasound
URTI	:	Upper Respiratory Tract Infection

ABSTRACT

TITLE: A COMPARATIVE STUDY OF SINGLE DOSE PREOPERATIVE CEFTRIAZONE AND ROUTINE CONVENTIONAL POST OPERATIVE PROPHYLAXIS IN ELECTIVE GENERAL SURGICAL CASES

AIM:

To assess the efficacy of single dose preoperative CEFTRIAZONE compared to multiple doses of postoperative prophylaxis in reducing surgical site infection and to prevent unnecessary development of antimicrobial resistance.

MATERIALS AND METHODS:

This study includes 50 clean cases randomized to two category of 25 each, like clean group without implant (e.g. Hydrocele) and clean group with implant (inguinal hernia).

In each category a single dose of preoperative ceftriazone is given to study group whereas 3 to 5 days of routine empirical antibiotic prophylaxis is given to control group.

Single shot of 1 gm Ceftriazone is given before half an hour of skin incision or at induction time for all clean class 1 cases in study

group and in case if the procedure is prolonged for more than 3 hrs a second dose was given. No further administration of antibiotics is considered in study group.

Routine empirical antibiotic prophylaxis is given to control group of inj. Cefotaxime 1Gm IV BD and inj Metronidazole 500mg IV TDS for 3- 5 days.

The surgical site was inspected daily from second post operative day and was followed up at 8th POD, 15th POD, and 30th POD and later at 3 months and 6 months based on the following criteria for SSI. Complications related to SSI are noted and data is obtained and the results were analysed with both groups.

CONCLUSION:

Based on my study I would like to conclude that it is recommendable to use single dose preoperative CEFTRIAXONE prophylaxis for all class I, as per the study results there is a 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 infection (SSI) is defined by Centre for Disease Control and Prevention (CDC) as proliferation of microorganisms developing in a surgical incision site within skin and subcutaneous fat (superficial), musculo-fascial layers (deep), or in an organ or cavity. Surgical site infection is a common malady caused due to health care associated infections causing significant postoperative morbidity and mortality.

The introduction of antibiotics in 20th century led to great improvement in surgical outcomes. In the interest of promoting cost-effective surgical practice as well as reducing the development of bacterial resistance to antimicrobial agents, several surgical centres in many countries have adopted this practice of using a "single dose pre-operative prophylactic antibiotic(s)" to prevent surgical site infections in suitable surgical patients.

Surveillance of surgical site infections shown to a powerful preventive measure. A multidisciplinary team is therefore necessary to produce a clinically and statistically significant improvement in rates of SSI. Strategies for preventing SSI helps in reducing the patient's

morbidity, mortality, duration of hospital stay and save cost for healthcare institutions. A forefront of these measure is antimicrobial prophylaxis, which shown to be effective at reducing the risk of surgical site infections.

The purpose of this study was to compare the rate of surgical site infection in Patients receiving a single dose pre-operative prophylactic antibiotic preferably CEFTRIAXONE with that in patients receiving prolonged post-operative prophylactic antibiotics as per current practice.

REVIEW OF LITERATURE

HISTORICAL CONCEPTS :

- Egyptians like Ebers papyrus and Edwin Smith papyrus framed out various information in the wound management and wound healing.
- Hippocrates described the use of wine and vinegar to irrigate open, infected wounds before delayed primary or secondary wound closure and wound dressing.
- Galen's pivotal role in drainage of Pus, localised infection (suppuration) in wounds.
- Ambroise Pare doyen role in Wound Dressing.
- Koch and his POSTULATES,
 1. The infective organism to be found in considerable numbers (septic foci)
 2. Cultivate the organism in pure form
 3. Cultivated organism should produce similar lesions on injecting into another host
- Ignaz Semmelweis demonstrated simple act of hand washing between performing post-mortem examinations and entering the delivery room.

- Joseph Lister and Loius Pasteur) revolutionised ASEPTIC TECHNIQUE.
- Zauberkugel enlightened the concept of “magic bullet”
- Depage introduced debridement of wound and delayed wound closure based on microbial culture and sensitivity.
- Alexander Fleming the man behind the discovery of Penicillin.

WOUND CLASSIFICATION

CLASSIFICATION OF OPERATIVE WOUND BASED ON DEGREE OF MICROBIAL OCONTAMINATION:

S.NO	CLASS	TYPE	EXAMPLES
1	I	Clean	Purely elective, wounds primarily closed GIT/GUT not entered(no break)
2	II	Clean - Contaminated	Clean emergency ,GIT/GUT entered without major contamination (minor break)
3	III	Contaminated	Open wounds, gross contamination from GIT, penetrating trauma <4hrs old (major break)
4	IV	Dirty - Infected	Old wounds with dead tissue, penetrating trauma >4hrs or perforated viscera

WOUND HEALING

Wound healing is the body's response to tissue injury and it is an essential and primitive process common to all multicellular organisms which starts a predictable sequence of events that follows a set pathway resulting in wound healing.

TYPE OF HEALING

1. HEALING BY PRIMARY INTENTION

Most of the wounds heal by primary intention, in which the wound edges are brought together (apposed) and held in a place by mechanical means for a short time after injury (adhesive strips, staples & sutures), which allows time to heal and produce enough strength to withstand stress without any support.

It is also the way most surgical wounds heal. The main goal is to achieve healing in such a way with minimal odema, no discharge and with minimal scar formation.

2. HEALING BY SECONDARY INTENTION

Occurs in wound which is infected, discharging pus or wound with excessive skin loss. Such wounds heal with an ugly scar by contraction or granulation.

3. HEALING BY TERTIARY INTENTION (DELAYED PRIMARY CLOSURE)

Often performed in contaminated wounds, does not retard wound strength. Thus delayed closure may decrease wound morbidity without impairing wound strength.

PHASES OF WOUND HEALING:

There are essentially 3 phase of wound healing

- LAG PHASE / INFLAMMATORY OR EXUDATIVE PHASE
- PROLIFERATIVE OR GRANULATION PHASE
- WOUND CONTRACTION (MATRIX FORMATION) OR REMODELLING PHASE

INFLAMMATION / EXUDATIVE PHASE (2-5 DAYS)

It occurs immediately after injury and lasts for several days. Tissue injury causes disruption of blood vessels and extravasation of blood constituents. The blood clot which is formed re-establishes haemostasis and provides extracellular matrix for cell migration. Entered Platelets which not only initiate the formation of a haemostatic plug but also secrete several mediators of wound healing such as platelet derived growth factor and numerous vasoactive mediators and chemotactic factors which are generated by the coagulation and activated complement pathway helping in recruiting inflammatory leukocytes to the site of injury.

In first 5 – 6 hours after injury- neutrophils enter the wound and helps in process of phagocytosis.

Monocytes then infiltrate the wound by 24 – 48 hours in response to specific chemo attractants such as TGF- β and initiate the formation of granulation tissue.

Macrophages bind to specific proteins of the extracellular matrix by their integrin receptors, an action that stimulates phagocytosis of micro organisms and fragments of extra cellular matrix. Cytokines which are released by the macrophages like CSF-1, TNF α , TGF- α , IL-1 helps in the initiation and propagation of the new tissue formation.

EPITHELIALIZATION :

During this period, epithelial cells proliferate at the epidermal-dermal junction, which then migrates towards the midline reforming a thin epidermal layer under the surface of the clot in sutured surgical wounds .Epithelial migration begins within the first 24 hours of the injury and may be completed as early as 72 hours in healthy individuals.

Hence closure of the wound is not the only function of epithelial cells in the inflammatory phase.

PROLIFERATION OF GRANULATION TISSUE (2 days – 3 weeks)

Numerous growth factors, chemotactic and activating factors produced during the inflammatory phase are concerned in the initiation

and development of granulations tissues, which lasts for about 4 – 21 days after injury.

Granulation tissue comprise of a loose matrix of fibrin, fibronectin, collagen and glycosaminoglycans (hyaluronic acid) and containing macrophages, fibroblasts and in growing blood vessels. During this phase the wound begins to gain tensile strength, but it is during this early period that wound dehiscence and evisceration most commonly occurs.

FORMATION OF GRANULATION TISSUE :

Growth factors mainly PDGF and TGF β stimulate the fibroblasts to proliferate and migrate into the wound space. The structural molecules to the newly formed extracellular matrix, termed the provisional matrix, contribute to the formation of granulation tissue by providing a conduit for cell migration.

NEOVASCULARIZATION :

Formation of new blood vessels which is necessary to sustain the newly formed granulation tissue. Angiogenesis is a complex process which relies on extracellular matrix on the wound bed as well as migration and mitogenic stimulation of endothelial cells.

WOUND CONTRACTION OR REMODELLING PHASE

It is during the second week of wound healing the fibroblasts assume a myofibroblasts phenotype which are characterized by a large

bundles of actin containing microfilaments disposed along the plasma membrane of cells and by cell – and cell – matrix linkage

When the granulation tissue begins to remodel, its vascularity decreases as the amount of collagen increases. Scar maturation occurs over the next few months which define further remodelling. Collagen which is produced from fibroblasts is initially laid down in a vertical manner; but then gradually oriented to align across the defect, thereby increasing the wound strength. In addition collagen type III, which is initially laid down in the immature scar is replaced with the more mature collagen type I

Collagen and wound healing:

Collagen remodelling occurs during the transition from granulation tissue to scar is dependent on continued synthesis and catabolism of collagen in the wound is controlled by matrix metalloproteinase (MMP).

A healed skin will never achieve the tensile strength found previously in undamaged skin. Following trauma only 10% of original tensile strength is regained 1 week and by third week 29% of strength is gained where fibrillar collagen has accumulated relatively rapidly and has been remodelled by contraction of the wound. Nevertheless wounds never attain the same tensile strength as uninjured skin. A maximal strength scar is only 70 % as strong as normal skin

PHASES OF WOUND HEALING

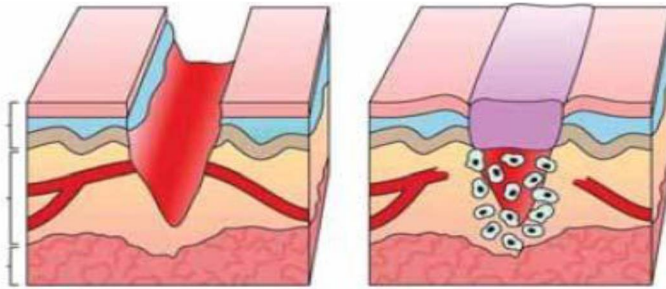


Fig 1. Haemostasis and Inflammatory Phase

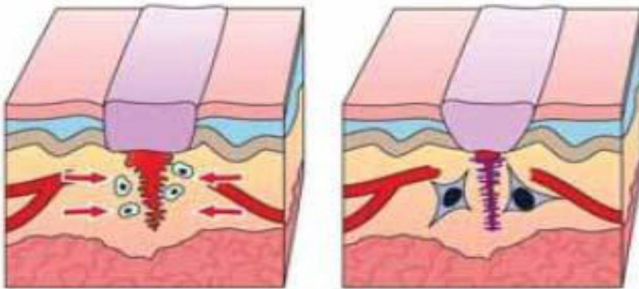


Fig 2. Fibroblastic Phase

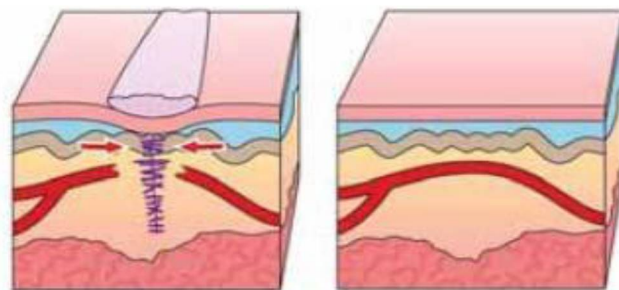


Fig 3. Remodelling Phase

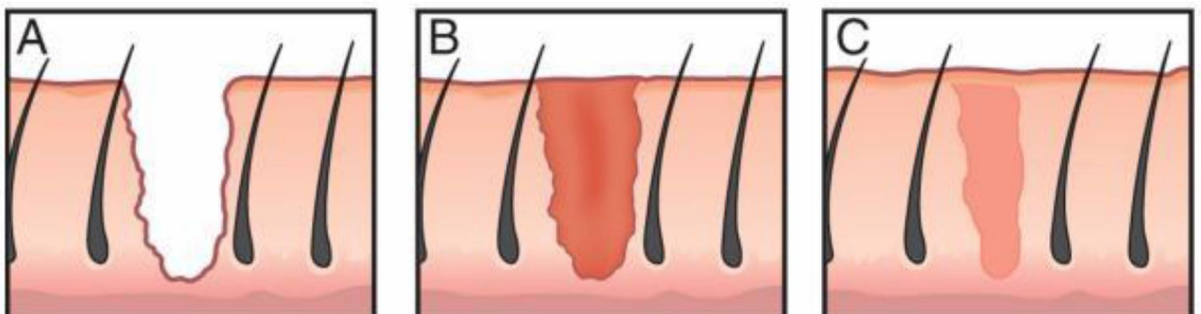


Fig 4 . Types of Wound Healing

FACTORS AFFECTING WOUND HEALING

The factors that may affect wound healing considered into two categories

- Systemic factors
- Local factors

SYSTEMIC FACTORS :

Much less is known on the role of systemic factors that affect wound healing and it co-exist in surgical patients. The following factors have been implicated in wound healing

- **Age** : Elderly patients heal more slowly and with less scarring. Sutures should be left in place longer to allow for slower gain in the tensile strength. Although the aged heal surgical incisions without complications.
- **Malnutrition** : Many surgeons believe that malnutrition is the most important systemic factor affecting wound healing. It is due to defect in fibroblast and collagen synthesis .Histological studies have suggested that reduced plasma colloid osmotic pressure and tissue edema may be important factors in pathogenesis of wound failure in malnutrition.
- **Vitamin deficiency** : Role of ascorbic acid in connective tissue metabolism and wound repair has held the attention of surgeons for more than 30 years. Studies show that ascorbic acid deficiency

affect collagen synthesis and also inhibit synthesis of mucopolysaccharides.

- **Zinc deficiency** : It affects cell multiplication , fibroblasia , collagen synthesis and epithelial covering of wounds .Maximum zinc deficiency noted in severe Burns
- **Steroids** : Corticosteroids causes prolonged monocytopenia which prevents macrophage migration into the wound and thus affects the wound healing cascade. The in vitro effects of steroid treatment are to depress fibroblast proliferation and inhibit procollagen and matrix protein synthesis.
- **Cytotoxic and Anti – Metabolite Drugs**
- **Irradiation** : Depletes dermal fibroblasts and decreasing the proliferative potential of endothelium. It also depresses bone marrow derived elements, virtually eliminating macrophages.
- **Cytotoxics** : Cytotoxic treatment decreases circulating white cells and impairs the formation of granulation tissue in the wound chamber.
- **Trauma and hypovolaemia** : Major trauma and hypovolaemia affect wound healing

- **Hypoxia**
- **Diabetes** : Microangiopathy, neuropathy and decreased phagocytosis and neutrophil chemotaxis affect healing in diabetes.
- **Uraemia**
- **Malignant disease / jaundice**

LOCAL FACTORS :

- | | |
|----------------------|------------------|
| • Surgical technique | Blood supply |
| • Mechanical stress | Suture technique |
| • Suture materials | Infection |

1. Surgical technique

Perhaps the performance of the surgeon like good surgical technique, adequate haemostasis, meticulous handling of tissues plays a major role in wound healing.

2. Blood supply

Rich blood supply is essential for wound repair. Extrinsic forces distracting the wound edges cause wound tension. Intrinsic wound tension results from an increase in the volume of the wound contents following sutures. It can also occur in the presence of wound infection, haematomas and seromas.

3. Mechanical stress

Wound disruption may be caused by the extrinsic forces affecting wound tension or it may be a consequence of excessive movement of the wound edges e.g. cutting through of sutures, slipping of knots.

4. Suture technique

The general aspects of suture techniques include, appropriate suture material with good knotting capability providing strong mechanical support to wound. Suture bite must be taken at a distance away of the wound edges.

The retention of subcuticular stitch using absorbable suture has been performed by using a variety of methods and have been associated with problems such as ‘dog ears’ and a bulky knot that becomes difficult to buy. The latter can also be responsible for wound gaping especially of small wound. Retention knots can be complicated by an irritative granulomatous reaction with ulceration through the skin as the knot may also be a possible nidus for infection and can lead to delayed wound healing.

Relation of suture with SSI

The presence of foreign body in the wound in form of suture enhances the susceptibility of surrounding tissues to infection. Bacteria adhere to the suture and form a biofilm under which the bacteria

propagate. Fowler in 1965 recommended the use of suture materials after being treated with 1/2,000 solution of chlorhexidine before use. Recently used substance for impregnating the suture is triclosan.

Characteristics of an ideal suture

Sterile, Minimal tissue injury, Easy to handle or pliability, Holds securely when knotted (i.e. no fraying or cutting), High tensile strength, Favourable absorption profile, Resistant to infection Memory, Breaking strength, Capillarity, Knot strength. Extensibility, Co efficient of friction

5. Infection

Presence of bacterial contamination affects wound healing

SURGICAL SITE INFECTION

DEFINITION:

Infection that occurs in an operative site. An infection of the subcutaneous tissue only (the most common “wound infection”) is termed a superficial SSI. One that involves the muscular and fascial layers and includes a partial or complete fascial dehiscence is a deep SSI.

AETIOLOGY: Primary infection-wound is the primary site of infection and in secondary infection occurs as a complication following and it’s not related to the wound directly.

TIME: Early infection occurs within 30days of the surgical procedure, when it occurs between 1 to 3 months it is described as intermediate infection, and when it presents more than 3 months of surgery it is termed as late infection.

SEVERITY: Minor if there is discharge without cellulitis or deep tissue involvement and it’s considered as major when pus discharge associated with tissue breakdown, partial or total destruction of deep fascial layers of wound or involvement with systemic illness

CLASSIFICATION OF SURGICAL SITE INFECTIONS

S.NO	TYPE OF WOUND	DEFINITION	TIMING	CLINICAL FEATURES
1	Superficial Incisional SSI	Involving Skin and Subcutaneous tissue	Within a month after the operation	<ul style="list-style-type: none"> • Pus • Culture positive for organism • Pain, swelling, redness
2	Deep Incisional SSI	Involving Deep tissues	Within a month after the operation (within 1 year if implant in place)	<ul style="list-style-type: none"> • Pus deep from incision(not from organ/space) • Wound dehiscence • Pain, redness, fever
3	Organ / Space SSI	involving organs or spaces	Within a month (within 1 year if implant in place)	<ul style="list-style-type: none"> • Drain tube purulent discharge • Culture positivity for organism • Clinical, radiological evidence of abscess

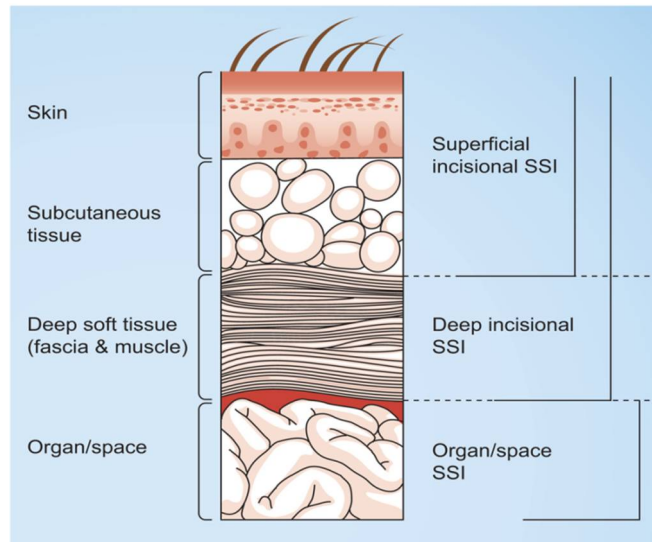


Fig 5. Infographic of the classification of Surgical site infection.

ETIOLOGY OF SSI

No single factor is responsible for surgical site infection. Numerous factors are involved and their contribution varies greatly in different types of surgery. The majority of wound infections are endogenous. They are self-infectious which results from wound contaminating by bacteria carried by the host either on the body surface or more commonly within hollow viscera. A minor proportion of wound infections are exogenous which 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 which is the result of bacterial contamination of the wound occurring during surgery.

- Secondary wound infection occurs within the postoperative environment where the 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

PHYSIOLOGY AND PATHO PHYSIOLOGY OF SURGICAL SITE INFECTION

The unique feature of all surgical infection is tissue necrosis. In primary surgical infection tissue necrosis is the pathophysiological process major role whereas in post traumatic surgical infection tissue necrosis is induced by technical or other physical trauma. The response to tissue necrosis is evident Inflammation leading to the events visible at surface, described by Celsius and refined by Galen as rubor, tumor, calor, dolor and functiolaesa.

The magnitude of inflammatory response and its symptoms is dependent on the amount of tissue injury, the number and pathogenicity of invading organisms. If bacterial products and toxins or other products generated 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 travel a uniform course once initiated. Macrophages which are produced may not be capable of phagocytosing all dead cells and remaining necrotic tissue acts as an excellent medium for bacterial growth. Bacteria release toxins which invade the surrounding tissue, causing the host to respond with further inflammation in order to confine the infection.

During infections inflammatory process will spread centrifugally and fibrin deposition occur to confine the infection faster than bacterial toxins can destroy the tissue, and a pyogenic membrane is formed. If tissue injury and number of bacteria exceeds the capability of the host to terminate an infection locally an abscess may form. Antibiotics poorly permeate it. The best treatment of an abscess is drainage.

SYSTEMIC PHASE OF INFECTION

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

When multiplication of bacteria started in the blood stream, then a serious state of infection termed sepsis or septicaemia ensure. **SIRS (systemic inflammatory response syndromes)** is the clinical symptomatic state resulting from host response to septicaemia. Septic shock, a state of acute circulatory failure characterized by presence of persistent arterial hypotension inspite of 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 a mortality rate of 60% to 80%. If sepsis is not treated immediately patient may die immediately of septic shock or later following multisystem organ failure.

PATHOGENESIS OF SSI's:

Microbial contamination of the surgical site is an essential precursor of SSI. The risk of SSI can be conceptualised according to the following relationship:

$$\frac{\text{Dose of Bacterial Contamination} \times \text{Virulence of Microorganism}}{\text{Resistance of Patient to Infection}} = \text{Risk of SSI}$$

FACTORS INVOLVED IN THE PATHOGENESIS OF WOUND INFECTION :

- Nature of Surgery
- Infecting organism (microbiological spectrum)
- Exogenous infection (cross-infection)
- Host resistance

1. NATURE OF SURGERY

A significant relationship exists between the different types of surgery and risk of wound infection. Surgical operations may be classified as contaminated, clean - contaminated, and clean according to the actual or potential degree of bacterial contamination of the wound.

CLEAN SURGERY:

There are no special septic hazards inherent in the surgical procedure. Wound infection occurs either from organisms contaminating from the patient's own skin surface or by exogenously from the environment. Surgeries included in this category are most plastic, neurosurgical , orthopaedic (elective) and cardiovascular operations as well as breast surgery, hernial surgery and a variety of minor surgical procedures in general surgical practice. Infection rates incidence is 2-4% in such operations.

CLEAN CONTAMINATED SURGERY:

Clean contaminated surgery refers to operations in which the surgical procedure includes exposure of the wound to bacterial contamination. Operations on the biliary tract, gastrointestinal surgery and the surgery of the urinary tract without unusual contamination or minor technique break. The infection rates incidence overall is 10-20% but as one would expect, colorectal operations the infection rates in excess of 50% have been reported in some series.

CONTAMINATED SURGERY:

Refers to the operations which are conducted in the presence of established sepsis. Thus, operations for peritonitis, perforated appendicitis and drainage of abscesses are included in this category .The incidence of infection is 40-60%

2. INFECTING ORGANISM (MICROBIOLOGICAL SPECTRUM)

According to **national nosocomial infection surveillance (NNIS)**, the pathogens reported for SSI occurrence hasn't changed markedly since last decade. Staphylococcus aureus, Coagulase negative staphylococci, Enterococcus species and Escherchia coli remains the most frequent pathogens. Antimicrobial resistant pathogens like MRSA or Candida

albicans are emerging SSI caused by resistant pathogens and Candida species reflects ill and immuno-compromised patients.

Outbreaks or clusters of SSI are caused by unusual pathogens, like *Rhizopus oryzae*, *Clostridium perfringens*, *Rhodococcus bronchialis*, *Nocardia farcinia*, *Legionella pneumophila* and *dermoffini* and *Pseudomonas multivorans*. These are rare outbreaks occurs in a contaminated dressings and elastic bandages or contaminated disinfectant solutions.

3. EXOGENOUS INFECTION (CROSS-INFECTION)

The role of Cross infection is less comparing to endogenous infection in the statistics of wound infection, but it plays a major role of infection in clean surgery. It may occur in the operating room during exposure of the wound. Bacteria presence in the wound at the end of the operation results in a fivefold increase in the incidence of wound infection and also the longer the wound is exposed, the more contamination set in and lengthy operations are associated with an increased incidence of wound sepsis. Infection is more common in traditional open type of ward than in modern surgical units which include patient's segregation and positive pressure ventilation.

The risk of wound contamination 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.

The increased density of airborne contamination is due to shedding of bacteria or dispersal by the operating room staff and hence 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.

The operating surgeon and the assistants also contribute to the bacterial contamination of the operating room and they present the additional risk to the patient of direct bacterial inoculation of the surgical incision. Also shown that wet operating gowns allows the transfer of bacteria from the surgeon's skin and that 5% surgeons gloves are perforated by the end of the surgical operation.

OT ZONES:

There are Four zones in an O T complex based on varying degrees of cleanliness, in which the bacteriological count progressively diminishes from the outer to the inner zones (operating area) and is maintained by a differential decreasing positive pressure ventilation gradient from the inner zone to the outer zone.

(1) PROTECTIVE ZONE:

- Change rooms for all medical and paramedical staff with conveniences
- Transfer bay for patient, material & equipments
- Rooms for administrative staff
- Stores & records
- Pre & post-operative rooms
- I.C.U. and P.A.C.U.
- Sterile stores

(2) CLEAN ZONE :

Connects protective zone to aseptic zone and covers other areas such as

- Stores & cleaner room
- Equipment store room
- Maintenance workshop
- Kitchenette (pantry)
- Fire fighting device room
- Emergency exits
- Service room for staff
- Close circuit TV control area

(3) **ASEPTIC ZONE** - Includes operation rooms (sterile)

(4) **DISPOSAL ZONE** - Disposal areas from each OR and corridor lead to disposal zone

4. HOST RESISTANCE

The occurrence of surgical site infection is high when the wound is contaminated with more than **100000 micro-organisms per gram of tissue**.

Micro-organisms cause damage to host by producing **Toxins** by invading the host and **Polysaccharide capsules** which inhibit an defence repose, Phagocytosis .Some strains of Clostridia and Streptococci produce **exotoxins** which disrupts cell membranes or alter the metabolism.

Glycocalyx and associated slime produced by Coag negative staphylococci provides a shield to the bacteria which render phagocytosis.

Endogenous flora remains the source of infection in majority of SSI. The organism which responsible are usually Gram positive aerobic cocci like streptococci, but also includes anaerobic bacteria and gram negative aerobes when perineum or groin in entered.

Seedling from distant foci also can be a source of SSI, particularly in patients with prosthesis or implants during surgery which acts as nidus.

Exogenous source includes surgical personnel- particularly members of surgical team, Operating room, tools and instruments, material which are brought to the surgical field

CLINICAL FEATURES OF SURGICAL SITE INFECTION

5 clinical Signs of infections like,

- Calor - heat
- Rubor - redness
- Tumor - swelling
- Dolor - pain
- Function lease - loss of function may or may not be present.

Classical diagnosis is done with the purulent discharge occurring 3-10 days after surgery from the wound spontaneously or by manipulation by surgeon.

Fever

Signs of Superficial skin infection like wound site inflammation and redness and purulent discharge and deeper infection which are located beneath the facial layers wouldn't be evident with pus discharge but unexplained fever, tender wound provokes the deeper infection.

TREATMENT OF SURGICAL SITE INFECTION

Adequate drainage of the infected wound and administration of antimicrobial agents.

METHODS USED IN PREVENTION OF SURGICAL SITE INFECTION

The surgical technique used will affect the infection rate in many ways, e.g Skin preparation, shaving, wound closure.

- **Skin preparation:** preoperative wash containing chlorhexidine decreases the bacterial count on skin by 80-90%.
- **Shaving:** shaving damages the skin and increases the skin and that the risk of infection increases with the length of time between shaving and surgery. Decreased rates are seen with close to time of surgery
- **Other methods of skin preparation-** shaving, Clipping, chemical depilation.
- **Preoperative antiseptic showering:** decreases skin microbial colony counts
- **Preoperative Hand washing technique-** must be followed by WHO guidelines
- **Skin preparation in operating room**
- **Sterilization**



Fig 6. Hand washing steps.

SSI SURVEILLANCE METHODS

SSI surveillance methods used in SENIC and NNIS systems were designed for monitoring,

Inpatient SSI surveillance, it involves 2 methods

1. **Direct observation**- by surgeon, trained nurse or infection control personnel (most accurate method), but lacks sensitivity.
2. **Indirect observation**- via lab reports, patient records and discussion with primary care providers.

POST DISCHARGE SURVEILLANCE

1. Direct examination of patients wounds during follow up visits
2. Medical records review
3. Patient survey by mail or telephone
4. Integrated health information system (tracking the patients)

Both the direct and indirect methods have been used to detect SSI that complicates outpatient operations.

ANTIBIOTIC PROPHYLAXIS:

INTRODUCTION:

Wound infections are the commonest hospital-acquired infections in surgical patients. They result in increased antibiotic usage, inappropriate usage and prolonged hospitalisation. Appropriate antibiotic prophylaxis can reduce the risk of postoperative wound infections, but additional antibiotic use also favours the emergence of antimicrobial resistance.

DEFINITIONS:

ANTIBIOTIC PROPHYLAXIS refers to administration of a brief course of an antimicrobial agent just before an operation in order to reduce

intraoperative microbial contamination to such a level that will not overwhelm host defence and result in infection.

PERIOPERATIVE PROPHYLAXIS refers to administration of antibiotic in elective surgical procedures in patients without prior infection or signs of inflammation. In order to prevent the occurrence of surgical site infection.

PERIPROCEDURAL PROPHYLAXIS is administration of antibiotics to prevent the spread of infection after invasive diagnostic-therapeutic procedures.

HISTORICAL ASPECT:

Soon after the invention of the first antimicrobial agents, penicillin and sulfonamides, it has become evident that administration of antibiotic could reduce the infection rate in many surgical procedures. Penicillin alone shown to reduce the infection rate to 10% in abdominal surgery, compared to a control group rate of 25%. Earlier there was no criteria existed for the choice of antimicrobial agent, route of administration, dosage, dosage timing. In the era of increased antibiotic use, simultaneously the problem of antibiotic resistance also emerged and many conditions with multi-drug resistance arose. In order to overcome this pitfall, the Joint Commission on Accreditation of Hospitals in 1977,

imposed a standard that antibiotic review must be performed meticulously on all its participating hospitals and it is the duty of medical staff. This forced many hospitals to review the antibiotic prescribing and usage patterns of their medical staff. Antibiotic prophylaxis has since become an important and integral part of surgical field.

40 years ago the original surgical antibiotic prophylaxis experiments were performed in pigs. The results emphasised that the most effective period for prophylaxis begins the moment when bacteria gain access to the tissues and is over in three hours. Since then many studies in animal models and in humans undergoing surgery has been on process. This has resulted the principles of antibiotic prophylaxis becoming an accepted part of surgical practice.

CHOICE OF IDEAL ANTIBIOTIC PROPHYLAXIS:

- The prophylactic antibiotic should cover the most common pathogen implicated in the causation of Surgical Site infection, although it is impossible for one single agent to cover all the possible pathogens.
- The choice of antibiotic depends on the primary site of the surgical procedure.

- It should be different from those already chosen for therapeutic usage in the same anatomical location. This is done to prevent the emergence of resistance to the therapeutic drugs.
- It should not be accompanied by gross changes in the treatment policy of a particular disease/ procedure because such change may curb the benefits of prophylaxis.
- It should also focus on the type of organisms contaminating particular parts of the body. For example, during head and neck colorectal, gynaecological and procedures, anaerobic organism coverage is a must and should be borne in mind while selecting the antibiotic.
- It should have a narrow therapeutic range in order to reduce the emergence of multi drug resistant organisms
- Prior drug allergic history should be noted and the antibiotic should be chosen accordingly.

ROUTE OF ADMINISTRATION OF ANTIBIOTIC:

Prophylactic antibiotics are usually given intravenously as a bolus which ensures adequate tissue concentration during surgical procedure

Intramuscular antibiotics are less commonly used. They are to be given along with premedication to achieve peak levels during surgery.

Oral or rectal antibiotics need to be given even earlier to ensure adequate tissue concentrations during surgery.

The absorption rate after IM/ oral routes varies grossly between individuals. Topical antibiotics are not recommended, with the exceptions of ophthalmic or burns surgery.

DISTRIBUTION OF ANTIBIOTICS:

An antimicrobial agent whose tissue concentration is more than minimum inhibitory concentration of antibiotic is used to treat the localized infection.

Tissue Penetration depends on protein binding and lipid solubility of the particular drug. Distribution is mainly with Blood, Urine, bile and intestinal tissues and fluids.

TIMING OF ADMINISTRATION OF ANTIBIOTIC:

Most effective during "DECISIVE PERIOD"

The timing of antibiotic administration was divided into four periods:

S.No	Period	Timing
1	Early Period	2 to 24 hours before the incision
2	Preoperative Period	2 hours before the incision

3	Perioperative Period	within 3hours after the incision
4	Postoperative Period	more than 3 hours but less than 24 hours after the incision

The goal of antimicrobial prophylaxis and importance of its timing is to achieve serum and tissue drug levels that peak in immediate perioperative period.

The ideal timing of antibiotic administration is half an hour before incision, when the patient is stabilized after anesthesia induction.

The drug effect should also last beyond the duration of surgical procedure intended.

The timing of antibiotic dosing is particularly important for most beta-lactam group of antibiotics which have relatively short half-lives.

Vancomycin is given as a slow infusion over one hour. So the prophylactic dose must be started earlier so that the infusion completes just before induction of anaesthesia.

Metronidazole suppositories are commonly used in bowel surgery and should be given 2-4 hours prior to the surgical procedure.

DURATION OF ANTIBIOTIC PROPHYLAXIS:

The critical period for development of SSI is 4 hours from bacterial entrance into the wound. The antibiotic coverage should ensure full protection during this critical period. The drug concentration and peak levels should be optimum during this period. The effect of drug should be maximum during this period and also should last for several hours after wound closure.

A single dose half an hour before the surgery is considered sufficient. Additional doses should be considered if the procedure last longer than the double antibiotic half life ($T_{1/2}$).

Re-administration of antibiotics should be considered for every three hours if surgical procedure is prolonged.

ANTIBIOTIC DOSAGE:

Based on body mass index, body weight, adjusted weight of the patient.

Obese patients required double dosing for reaching appropriate tissue concentration.

Additional doses after surgery has no proven prophylactic benefits.

ADDITIONAL DOSES AFTER THE END OF THE OPERATION:

The administration of additional doses after the end of surgery does not provide any proven additional prophylactic benefit.

RE-DOSE FOR LONG SURGERIES:

An antibiotic dose should be adjusted appropriately (redoses) based on the half life.

RISK OF ANTIBIOTIC PROPHYLAXIS:

- Even judicious use of antibiotics in perioperative prophylaxis increases clostridium difficile colitis.
- Bacterial resistance.
- Drug allergy.

QUALITIES AN APPROPRIATE PROPHYLACTIC ANTIBIOTIC THERAPY:

1. The organism should be sensitive to the antibiotic.
2. Antibiotic dosage should ensure adequate peak concentration and tissue penetration.
3. The Antibiotic should have contact with the organism.
4. Dosage frequency and administration is based on the half life and the route of elimination.
5. Bactericidal antibiotic must be chosen when appropriate.

6. Synergistic therapy is recommended when condition demands.
7. Antibiotics with antagonistic actions must be avoided in combination.
8. Antibiotic with narrow spectrum of action should to be used.
9. Adverse effects to be borne in mind and equated with benefits
10. Antibiotics to be administered for adequate duration and dosage to ensure complete eradication of pathogen and to cut down chances of emergence of resistance.

PROPHYLACTIC AGENTS:

The ideal antibiotic should be safer and efficient.

PENICILLINS:

Old group of beta-lactams. Extracted from the penicillium notatum. Using modern biochemical techniques along with molecular techniques, manipulation on the original nucleus has been achieved and a number of alteration and enhancements to bacterial sensitivity been achieved.

BETA - LACTAM ANTIBIOTICS :

Largest class of antibiotic with unique four member beta-lactam ring including penicillin, cephalosporins, the monobactams and the thiocyanins

CARBAPENEMS:

- Meropenem
- Ertapenem
- Imepenem

Sensitive to beta lactamases, having a broad spectrum Gram positive as well as anaerobic activity.

IMIDAZOLES:

- Metronidazole

More sensitive against anaerobic bacteria

Other agents used include Aminoglycosides, Tetracyclins & Quinolones

CEPHALOSPORINS:

It's the drug of choice for surgical prophylaxis over decades since they fulfil most of the criteria of an ideal prophylactic antibiotic

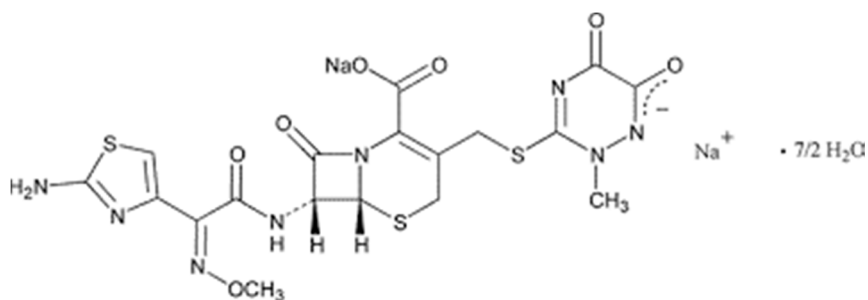
S.No	Generation	Drugs	Sensitivity
1	First	Cefazolin, Cephalexin, Cefadroxil	More active against gram positive organisms
2	Second	Cefuroxime, Cefoxitin, Cefaclor, Cefuroxime Axetil, Cefprozil	More active against gram negative than gram positive organisms and also anaerobes
3	Third	Cefotaxime, Ceftizoxime, Ceftriaxone, Ceftazidime, Cefoperazone, Cefixime, Cefpodoxime Proxetil, Cefdinir, Ceftibuten, Ceftamet Pivoxil	Beta lactamase resistant aerobic gram negative bacteria, anaerobes
4	Fourth	Cefpirome, Cefipime	Both gram positive and negative due to its broader activity
5	Fifth	Ceftaroline, Ceftabiprole	MRSA, Gram positive bacteria

CEFTRIAXONE- CHEMICAL PROPERTIES:

Ceftriaxone is a sterile, semi synthetic, broad-spectrum cephalosporin antibiotic used as intravenous or intramuscular injection.

Ceftriaxone sodium is (6R,7R)-7-[2-(2Amino-4-thiazolyl)glyoxylamido]-8-oxo-3-[[1,2,5,6-tetrahydro-2-methyl-5,6-dioxo-astriazin-3-yl)thio]methyl]-5-thia-1-azabicyclo[4.2.0]oct-2-ene-2-carboxylic acid, 7²-(Z)(O-methyloxime), disodium salt, sesquaterhydrate.

The chemical formula of Ceftriaxone sodium is **C₁₈H₁₆N₈Na₂O₇S₃•3.5H₂O**. It has a calculated molecular weight of 661.59 and the following structural formula.



CHARACTERISTICS

Ceftriaxone sodium is a white to yellowish-orange crystalline powder which is readily soluble in water, sparingly soluble in methanol and very slightly soluble in ethanol. The pH of a 1% aqueous solution is approximately 6.7. The color of Ceftriaxone solutions ranges from light

yellow to amber, depending on the length of storage, concentration and diluents used. Ceftriaxone sodium contains approximately 83 mg (3.6 mEq) of sodium per gram of Ceftriaxone activity.

PHARMACOKINETICS:

ABSORPTION: Ceftriaxone can be administered intravenously and intramuscularly, and the drug is completely absorbed. It is not available orally.

DISTRIBUTION: Ceftriaxone penetrates tissues and body fluids well, including CSF to treat central nervous system infections. The average volume of distribution in adults is 5.8–13.5 liters.

METABOLISM: 33–67% of Ceftriaxone is renally excreted as unchanged drug, but no dose adjustments are required in renal impairment with dosages up to 2 grams per day. The rest is excreted in the bile as inactive compounds from hepatic and gut flora metabolism.

ELIMINATION: The average elimination half-life in healthy adults is 5.8–8.7 hours. In people with renal impairment, the average elimination half-life increases to 11.4–15.7 hours.

MECHANISM OF ACTION:

- It has stronger affinity to Penicillin binding proteins.
- Selectively and irreversibly inhibits bacterial cell wall synthesis by binding to transpeptidases, also called transamidases (Penicillin binding proteins)
- **Bactericidal in action.**

ADVERSE EFFECTS:

Although generally well tolerated, the most common adverse reactions associated with Ceftriaxone are changes in white blood cell counts, local reactions at site of administration, rash, and diarrhea.

INCIDENCE OF ADVERSE EFFECTS GREATER THAN 1%:

- Eosinophilia (6%)
Thrombocytosis (5.1%)
- Elevations in liver enzymes (3.1–3.3%) Diarrhea (2.7%)
- Leukopenia (2.1%) Elevation
in BUN (1.2%)
- Local reactions—pain, tenderness, irritation (1%)
- Rash (1.7%)

Ceftriaxone may precipitate in bile, causing biliary sludge, biliary pseudolithiasis, and gallstones, especially in children.

Hypoprothrombinaemia and bleeding are specific side effects. Haemolysis is reported. It has also been reported to cause post renal failure in children. Like other antibiotics, Ceftriaxone use can result in Clostridium difficile-associated diarrhea ranging from mild diarrhea to fatal colitis.

SUSCEPTIBLE ORGANISMS :

AEROBIC GRAM – NEGATIVE MICROORGANISMS:

Acinetobacter calcoaceticus, Enterobacter aerogenes, Enterobacter cloacae, Escherichia coli, Haemophilus influenzae (including ampicillin-resistant and beta-lactamase producing strains), H. parainfluenzae, Klebsiella oxytoca, Klebsiella pneumoniae, Moraxella catarrhalis (including beta-lactamase producing strains), Morganella morganii, Neisseria gonorrhoeae (including penicillinase- and nonpenicillinase-producing strains), N. meningitidis, Proteus mirabilis, Proteus vulgaris, Serratia marcescens. Ceftriaxone is also active against many strains of Pseudomonas aeruginosa.

Many strains of the above organisms that are resistant to multiple antibiotics, e.g., penicillins, cephalosporins, and aminoglycosides, are susceptible to Ceftriaxone.

AEROBIC GRAM-POSITIVE MICROORGANISMS :

Staphylococcus aureus (including penicillinase - producing strains), Staph. epidermidis, Streptococcus pneumoniae , Strep. pyogenes , Viridans group streptococci , Methicillin - resistant staphylococci are resistant to cephalosporins, including ceftriaxone. Most strains of Group D streptococci and enterococci, e.g., Enterococcus (Streptococcus) faecalis, are resistant.

ANAEROBIC MICROORGANISMS:

- Bacteroides fragilis Clostridium species, Peptostreptococcus species, most strains of Clostridium difficile are resistant.

SUSCEPTIBILITY TESTING:

DILUTION TECHNIQUES: Quantitative methods are used to determine antimicrobial minimal inhibitory concentrations (MICs). These MICs gives the idea of susceptibility of bacteria to antimicrobial agents. The MICs should be determined using a standard procedure such as dilution method either broth or agar. The MIC values should be interpreted according to the following criteria² for aerobic organisms other than Haemophilus spp, Neisseria gonorrhoeae, and Streptococcus spp, including Streptococcus pneumoniae:

Microgram/ml interpretation

≤ 8	(S) Susceptible
16-32	(I) Intermediate
≥ 64	(R) Resistant

DIFFUSION TECHNIQUES: Quantitative methods that require measurement of zone diameters also indicate the susceptibility of bacteria to antimicrobial agents. One such standardized procedure is the use of standardized inoculum concentrations. This procedure uses paper discs impregnated with 30 microgram of ceftriaxone to test the susceptibility of microorganisms to ceftriaxone.

MATERIALS AND METHODS

DESIGN :

Prospective comparative study

COLLECTION OF DATA AND SOURCES:

- This study was conducted in Coimbatore medical college hospital from Jan 2018 to Jan 2019
- Population included for the study –Clean Class 1 cases from all surgical unit
- Results tabulated and appropriate tests significance were worked up

INCLUSION CRITERIA:

- Patients with the age group of 20-80 years
- Clean Class-1 cases were included (e.g.- inguinal hernia- clean case with permanent implant ,hydrocele-clean case without implant)

EXCLUSION CRITERIA:

- History of Hypersensitivity to ceftriaxone
- Patient with co morbid renal, cardiac, hepatic damages
- Patients denied consent for surgery

- Patient on steroid or immune deficiency
- Patients having psychiatric problems

SOUTHAMPTON SCORING SYSTEM:

GRADE

	NORMAL HEALING
1	<p>NORMAL HEALING WITH MILDBRUIISING AND MILD ERYTHEMA</p> <ul style="list-style-type: none"> • 1a Some bruising • 1b Considerable bruising • 1c Mild erythema
2	<p>ERYTHEMA WITH OTHER SIGNS OF INFLAMMATION</p> <ul style="list-style-type: none"> • 2a at one point • 2b around sutures • 2c along the wound • 2d around the wound
3	<p>CLEAR (OR) HEMOSEROUS DISCHARGE</p> <ul style="list-style-type: none"> • 3a At one point (less than 2 centimetres) • 3b Along the wound (more than 2 centimetres) • 3c Large volume • 3d Prolonged (more than 3 days)
4	<p>PUS FORMATION</p> <ul style="list-style-type: none"> • 4a At one point (less than 2 centimetres) • 4b Along the wound (more than 2 centimetres)
5	<p>DEEP, SEVERE WOUND INFECTION WITH OR WITHOUT TISSUE BREAKDOWN,HEMATOMA REQUIRING ASPIRATION</p>

GROUP SELECTION:

Patients under the inclusion criteria were randomized into groups A (study) and B (control) in both category- Clean case with implant (Inguinal hernia) and clean case without implant (hydrocele).

- Group A received inj CEFTRIAXONE 1 gram just before the Skin incision
- Group B received inj. Cefotaxime 1gram iv BD and inj Metronidazole 500mg iv TDS Post operatively for 5 days

PREOPERATIVE PREPARATION:

- Under standard aseptic precautions for other surgery
- All diabetic patients had strict glycemic control

OPERATIVE PROCEDURE:

- All patients were operated in same theatre
- Pre-op preparation of surgical site done according to standard principles
- Lichenstein hernioplasty was done in hernia patients
- Jaboulay's procedure was done in hydrocele patients
- Dressing done postoperatively
- Surgical site inspected after 48 hrs

FOLLOW UP:

The surgical site was inspected daily from 2nd postop day onwards based on the following criteria for SSI.

SSI SURVEILLANCE CRITERIA:

- Evidence of Purulent discharge
- Evidence of erythema, if present noting its extent beyond the wound edges
- A wound which has been left opened and left to heal by secondary intention
- Wound dehiscence

If there was no occurrence of SSI, sutures were removed on 7th Post op day aft discharge. In patients who have SSI, culture and sensitivity test were done and appropriate antibiotics were given. If patient has wound gapping, thorough wound debridement done following which secondary suturing done.

DATA COLLECTION AND ANALYSIS

Following data were collected,

1. Patient demographic profile
2. Clinical type in case of hernia and hydrocele
3. Biochemical parameters
4. ASA grade
5. SSI and its management(additional antibiotic)
6. Hospital stay

STATISTICAL ANALYSIS:

- Analysis of data were done with prime objective to state that if Single dose preoperative CETRIAXONE is therapeutically and cost effectively more beneficial than multiple antibiotics received post operatively in reducing SSI in elective Clean Class 1 surgical cases.
- Differences between groups in distribution of parameters were tested using CHI-SQUARE TEST and P value <0.05 was considered statistically significant.

OBSERVATION AND ANALYSIS

CLASS 1 - CLEAN CASES WITHOUT IMPLANT (HYDROCOELE)

SIDE OF OCCURRENCE AND GROUPS

Graph1.

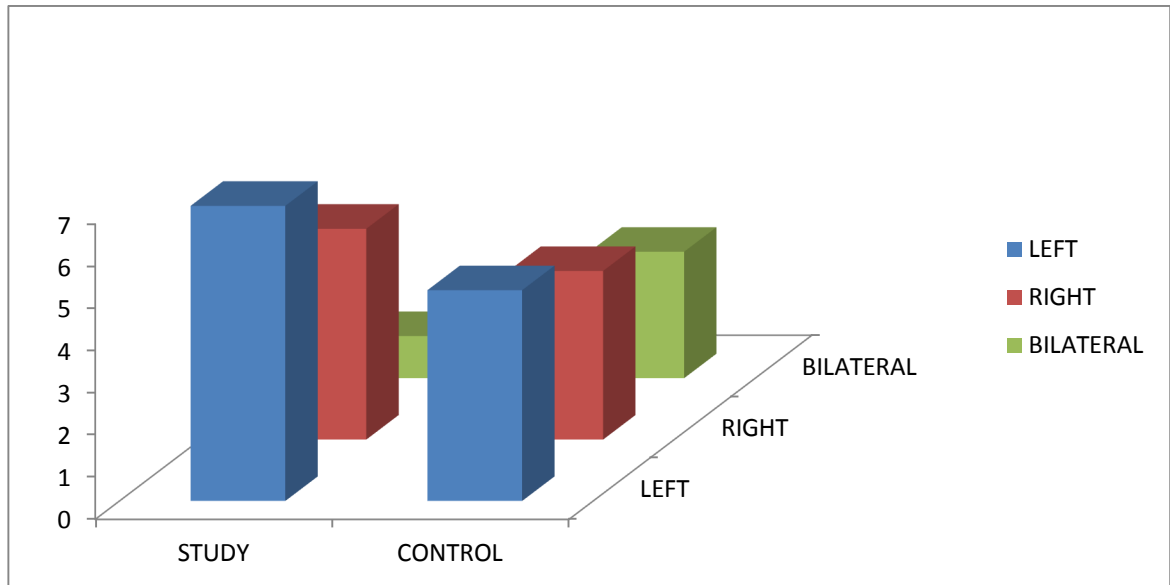


Table 1.

SIDE	STUDY	CONTROL	Chi sq	P value
LEFT	7 (53.8%)	5 (41.67%)	1.046	0.5
RIGHT	5 (38.4%)	4 (33.33%)		
BILATERAL	1 (7.69%)	3 (25.00%)		
TOTAL	13	12		

In our study, the control group had 5 patients (41.67%) with left sided hydrocoele, 4 (33.33%) with right sided hydrocoele and 3 (25.00%) were bilateral. The study group had 7 patients (53.8%) with left sided hydrocoele, 5 (38.4%) with right sided hydrocoele and 1 (7.69%) was bilateral. On comparing the two groups, there is no significant difference in the distribution of laterality among the two groups. (p=0.5)

AGE GROUP PATTERN IN 2 GROUPS

Graph 2.

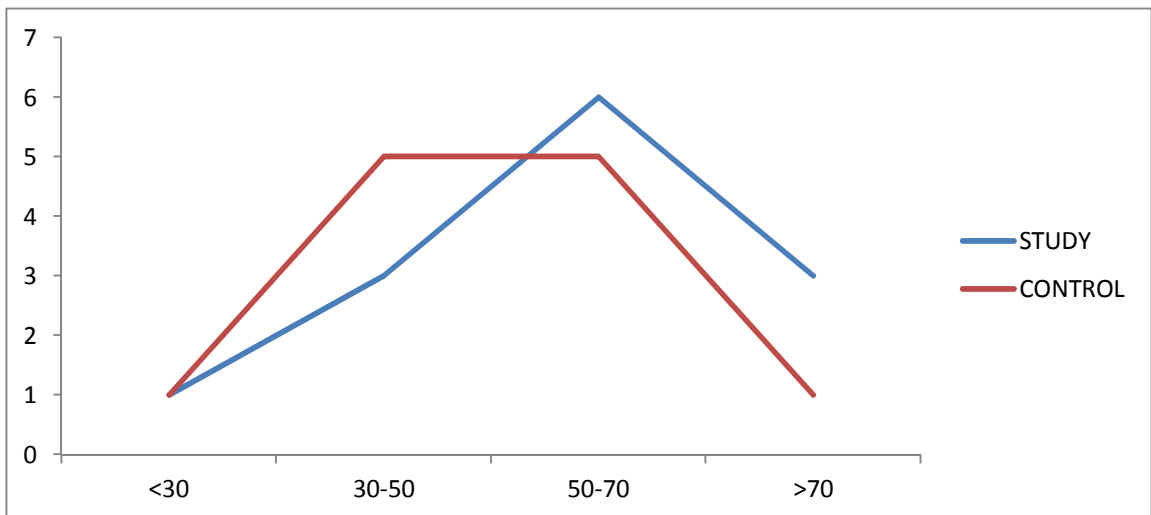


Table 2.

AGE	STUDY	CONTROL	Chi sq	P value
<30	1 (7.69%)	1 (8.33%)	1.56	0.67
30-50	3 (23.07%)	5 (41.66%)		
50-70	6 (46.15%)	5 (41.66%)		
>70	3 (23.07%)	1 (8.33%)		
TOTAL	13	12		

In our study, the most common age group with hydrocoele was 50-70 years. There was almost equal distribution of age groups between control and study groups (p-0.67), difference is statistically non significant) .This shows that age criteria was not a confounding factor in our study and the 2 groups were statistically matched.

AGE & SSI

Graph 3.

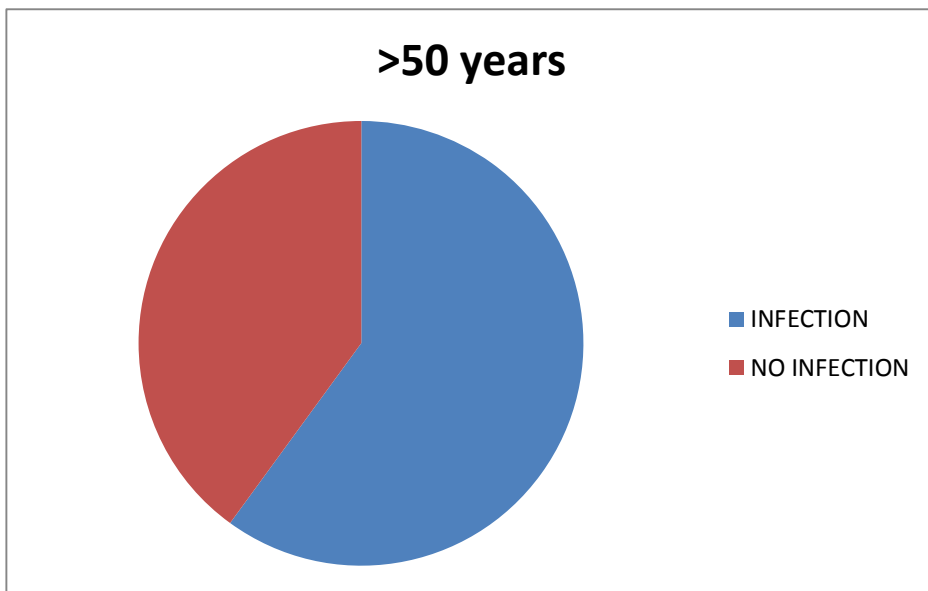
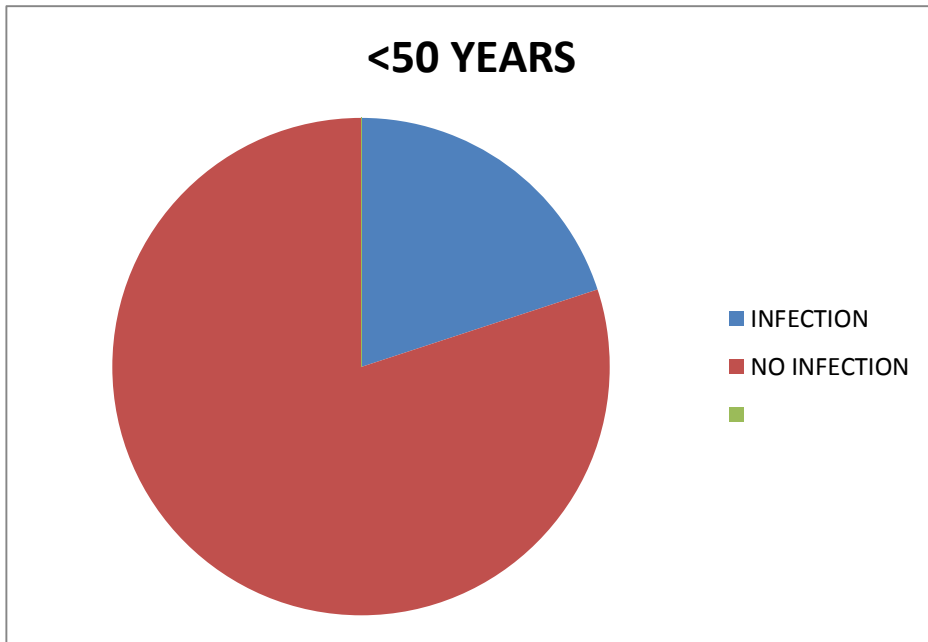


Table 3.

AGE/SSI	<50	>50	Chi sq	P value
INFECTION	2 (20%)	9 (60%)	3.94	0.047
NO INFECTION	8 (80%)	6 (40%)		
TOTAL	10	15		

Among 10 patients of age <50 years, 2 (20%) had SSI. Among 15 patients of age >50 years, 9 (60%) had SSI and 6 (40%) had no infection. On comparison, the occurrence was statistically significant (p-0.047), that is, the occurrence of SSI has a strong correlation with increasing age.

SSI AND GROUPS

Graph 4.

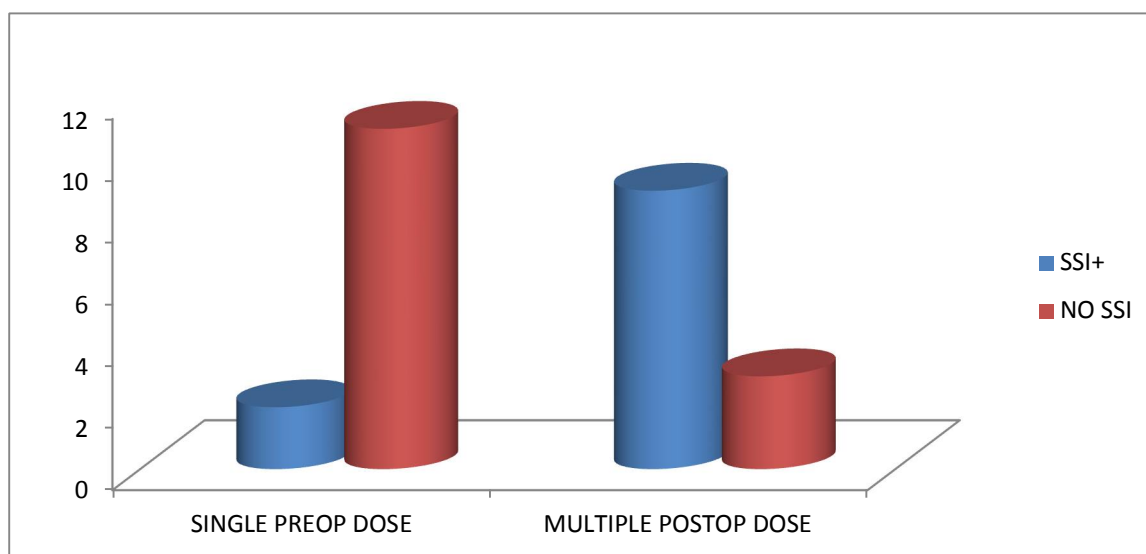


Table 4.

SSI	STUDY (SINGLE DOSING)	CONTROL (MULTIPLE DOSING)	Chi sq	P
INFECTION	2 (15.38%)	9 (75%)	9	0.0027
NO INFECTION	11 (84.61%)	3 (25%)		
TOTAL	13	12		

In our study, the control group had received multiple dosing of antibiotics post operatively. The study group received a single dosing of CEFTRIAXONE preoperatively. On comparing this dosing method and timing with the occurrence of SSI, it was found that the study group had a highly significant reduction ($p=0.0027$) in the occurrence of SSI compared to the control group. This proves the point that a single dose of antibiotic preoperatively is much more effective than multiple postoperative dosing in control of surgical site infection.

CO-MORBIDITY & OCCURRENCE OF SSI

Graph 5.

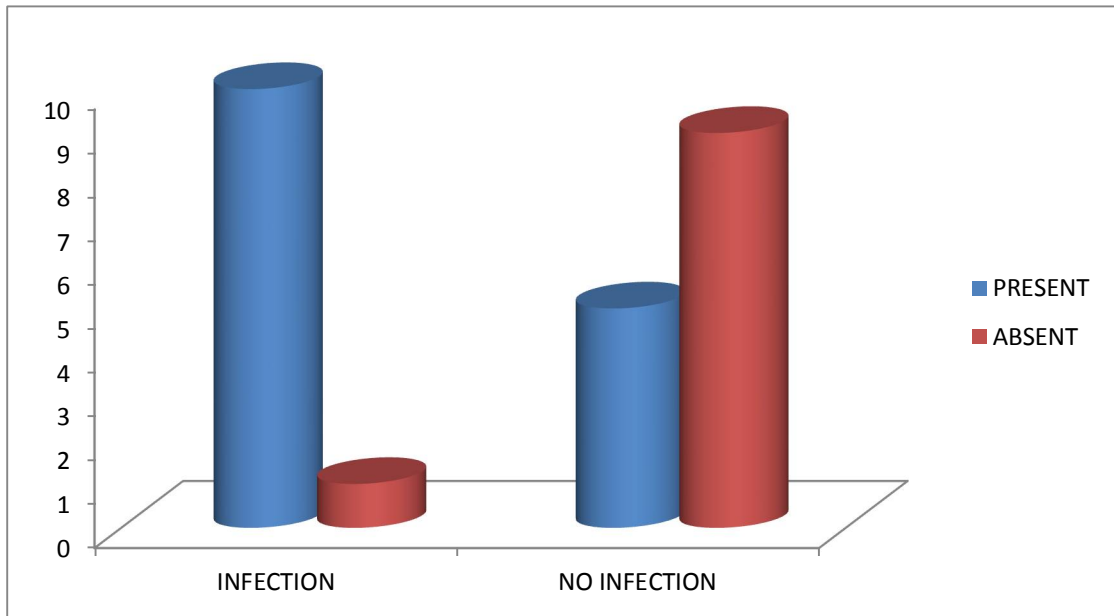


Table 5.

CO-MORBIDITY& SSI	PRESENT	ABSENT	Chi sq	P value
INFECTION	10 (66.67%)	1 (10%)	7.9	0.0049
NO INFECTION	5 (33.33%)	9 (90%)		
TOTAL	15	10		

In our study, out of 25 patients, 15 (60%) had co-morbidities such as diabetes mellitus and hypertension. It was seen that the patients with co-morbidities had a higher chance of occurrence (p-0.0049) of SSI than the other patients, P value is statistically significant.

COMORBIDITY & GROUPS

Graph 6.

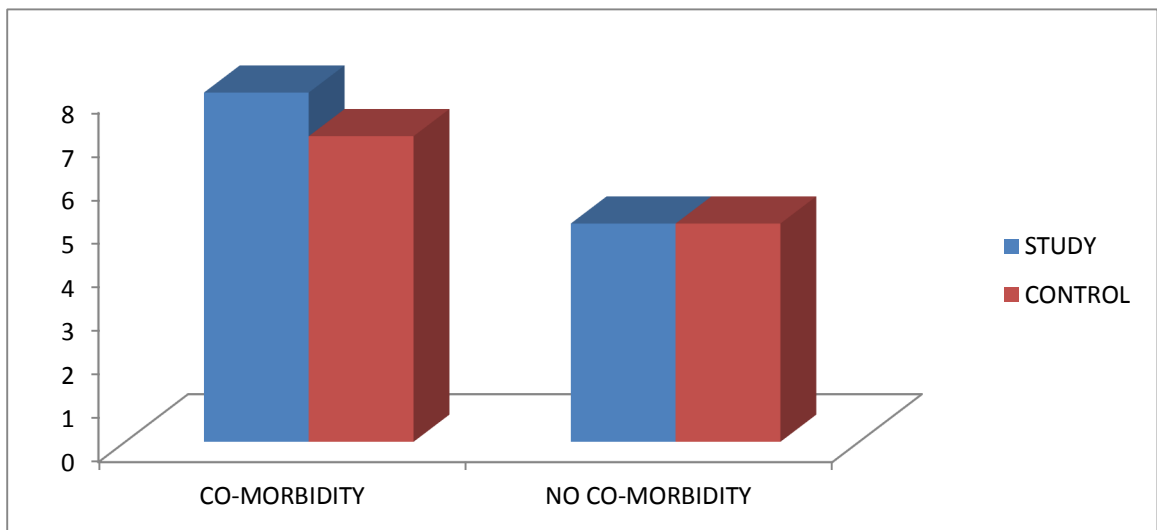


Table 6.

COMORBID & GROUPS	STUDY	CONTROL	Chi sq	P value
CO-MORBID	8 (61.53%)	7 (58.33%)	0.027	0.9
NO CO-MORBIDITY	5 (38.46%)	5 (41.66%)		
TOTAL	13	12		

The 15 patients with co-morbidity were equally distributed between study and control groups. The study group had 8 patients (61.53%) with co-morbidity and the control group had 7 patients (58.33%) with co-morbidity, on comparing the distribution was equal and there was no significant difference (p=0.9) This proves that the co-morbidity factor didn't have an influence on the study result.

DURATION OF HOSPITAL STAY IN THE 2 GROUPS

Graph 7.

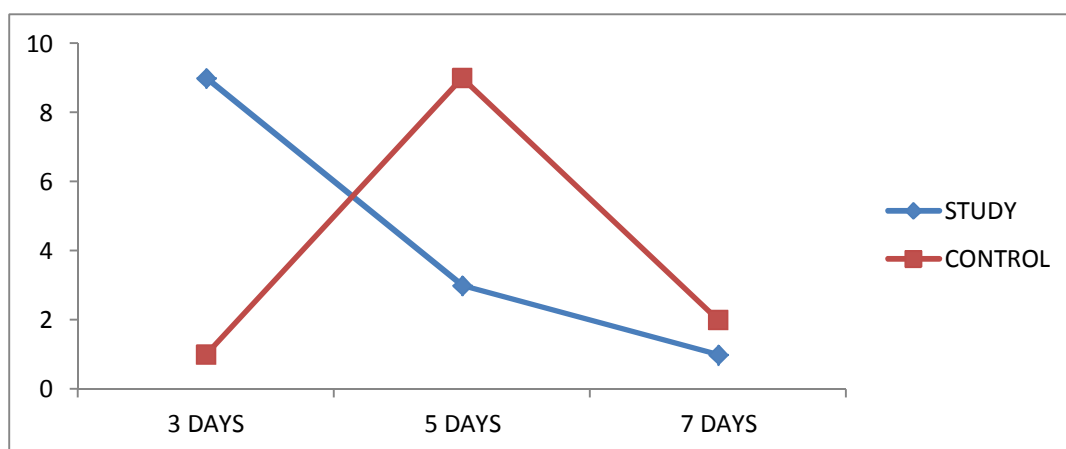


Table 7.

HOSPITAL STAY & GROUPS	STUDY	CONTROL	Chi sq	P value
3 DAYS	9 (69.23%)	1 (8.33%)	9.73	0.008
5 DAYS	3 (23.07%)	9 (75.00%)		
7 DAYS	1 (7.69%)	2 (16.67%)		
TOTAL	13	12		

According to our study, the number of patients who had a long duration of hospital stay was significantly higher (p-0.008) in the control group than the study group. This is because the occurrence of SSI was more in the control group, and so those patients needed extra days of additional antibiotic.

ASA GRADING & SSI

Graph 8.

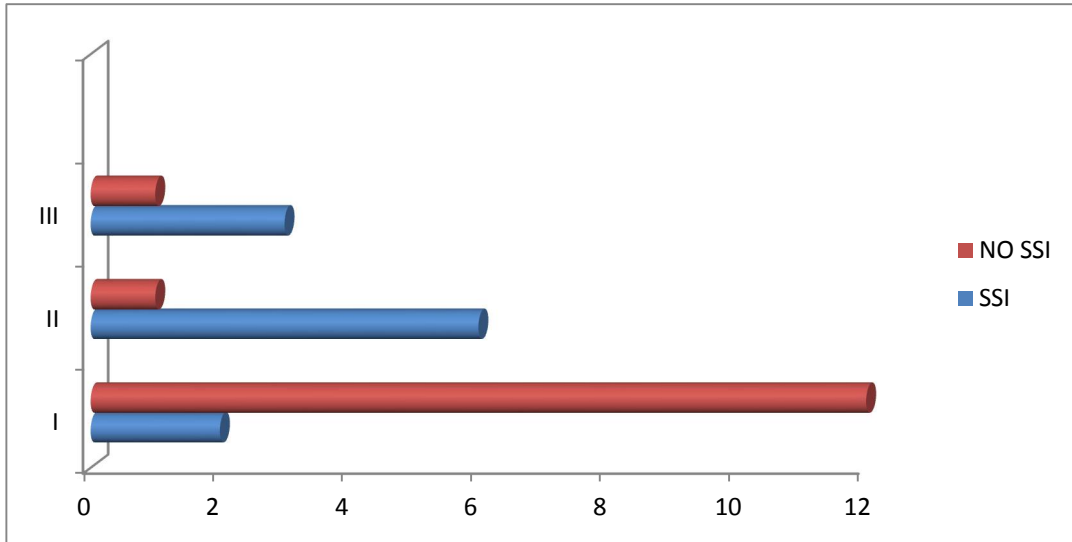


Table 8.

ASA & SSI	I	II	III	Chi sq	P value
INFECTION	2 (14.28%)	6 (85.71%)	3 (75.00%)	11.55	0.003
NO INFECTION	12 (85.71%)	1 (14.29%)	1 (25.00%)		
TOTAL	14	7	4		

In this graph, we compared the occurrence of SSI and ASA grading of anaesthesia the patients belonged to during surgery. Among the 14 patients who belonged ASA grade I, only 2 (14.28%) had SSI and the rest had no infection. In ASA II, 6 (85.71%) had infection and 1 (14.29%) had no infection. In ASA III, 3 (75.00%) had infection and 1 (25.00%) had no infection. The chance of occurrence of SSI is higher with patients of higher ASA grade and is statistically significant ($p=0.003$), the cause being presence of co morbidities and other habitual factors.

ASA GRADE DISTRIBUTION IN 2 GROUPS

Graph 9.

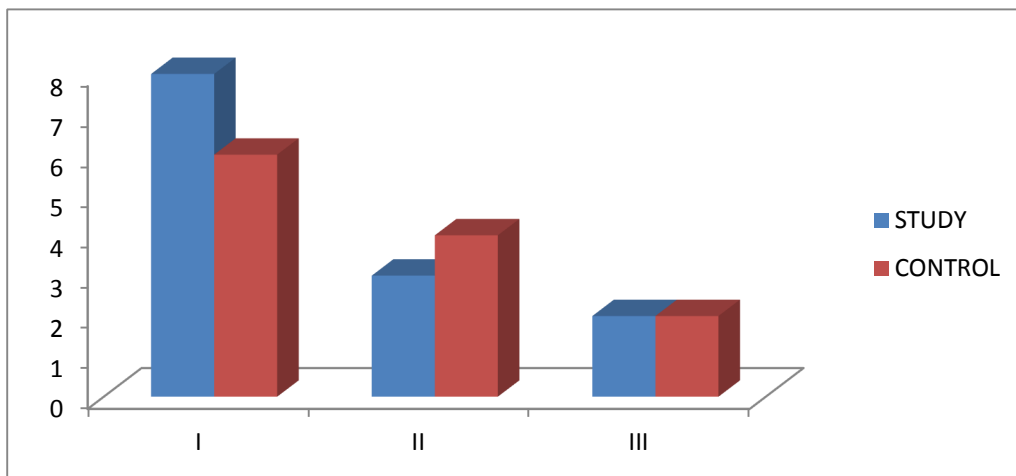


Table 9.

ASA & GROUPS	STUDY	CONTROL	Chi sq	P value
I	8 (61.53%)	6 (50.00%)	0.692	0.7
II	3 (23.07%)	4 (33.33%)		
III	2(15.39%)	2 (16.67%)		
TOTAL	13	12		

This graph shows the distribution of patients with particular ASA grade between our study and control groups. There was no significant difference ($p=0.7$) in the distribution of patients between the 2 groups. This is statistically important because the ASA grading had a linear correlation with the occurrence of SSI and our study results were not affected by it.

MANAGEMENT

Graph 10.

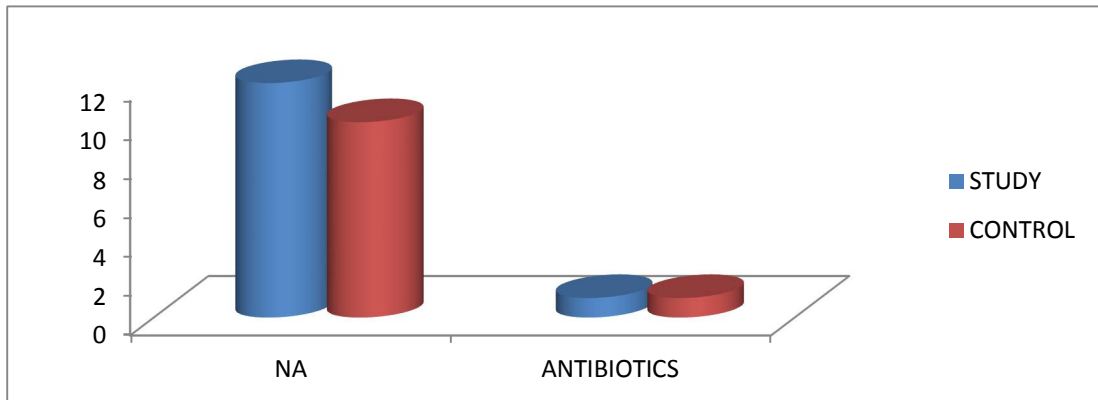


Table 10.

MANAGEMENT	STUDY	CONTROL	Chi sq	P value
NA	12 (92.30%)	10 (83.33%)	0.46	0.5
ADDITIONAL ANTIBIOTIC	1 (7.69%)	2 (16.67%)		
TOTAL	13	12		

Among 2 patients who developed SSI in study group, 1 (7.69%) needed additional antibiotic. 2 (16.67%) of 9 patients in the control group needed additional antibiotic for recovery. On comparison, there is no significant difference (p0.5) in the administration of additional antibiotic between the 2 groups during their hospital stay.

CLASS 1- CLEAN CASES WITH IMPLANT (HERNIA)

SEX DISTRIBUTION IN THE GROUPS

Graph 11.

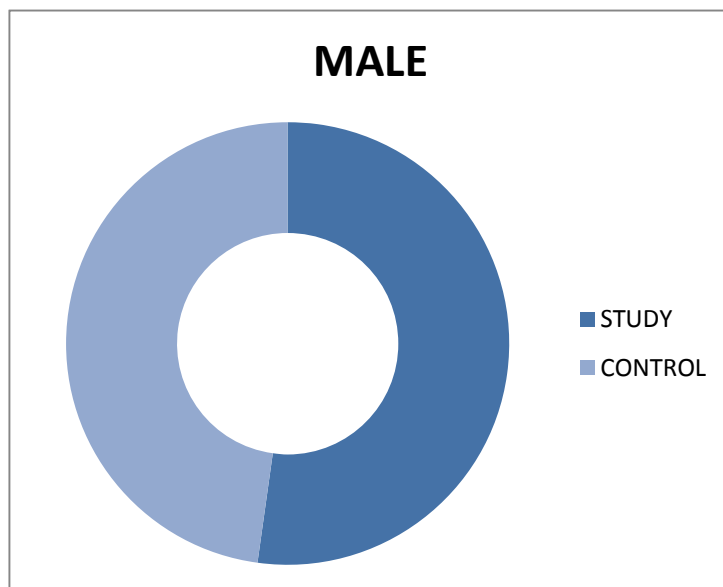
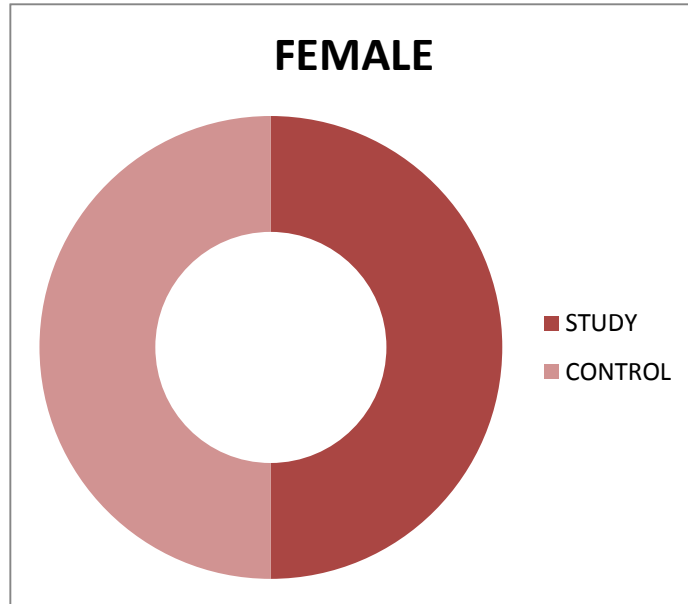


Table 11.

SEX	STUDY	CONTROL	Chi sq	P value
MALE	12 (92.30%)	11 (91.67%)	0.0042	0.95
FEMALE	1 (7.69%)	1 (8.33%)		
TOTAL	13	12		

Our study had 23 males and 2 females with hernia. This is in terms with other studies which show that hernia is common among males than females. Also the distribution of gender between the study and control groups is almost equal (p-0.95, the difference is insignificant)

SIDE OF HERNIA AMONG THE 2 GROUPS

Graph 12.

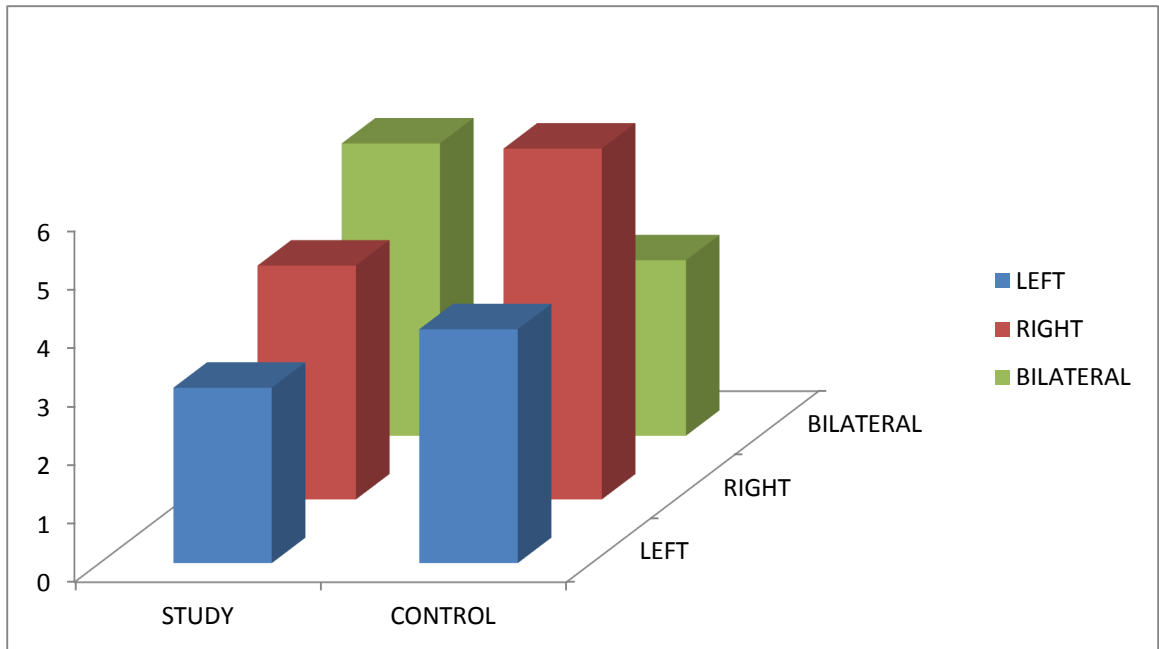


Table 12.

SIDE	CONTROL	STUDY	Chi sq	P value
LEFT	3 (25.00%)	4 (30.77%)	1.05	0.6
RIGHT	4 (33.33%)	6 (46.15%)		
BILATERAL	5 (41.67%)	3 (23.07%)		
TOTAL	12	13		

In our study, the study group had 3 patients (25.00%) with left sided hernia, 4 (33.33%) with right sided hernia and 5 (41.67%) was bilateral. the control group had 4 patients (30.77%) with left sided hernia, 6 (46.15%) with right sided hernia and 3 (23.07%) were bilateral On comparing the two groups, there is no significant difference in the distribution of laterality among the two groups. (p=0.6)

TYPE OF HERNIA AMONG THE GROUPS

Graph 13.

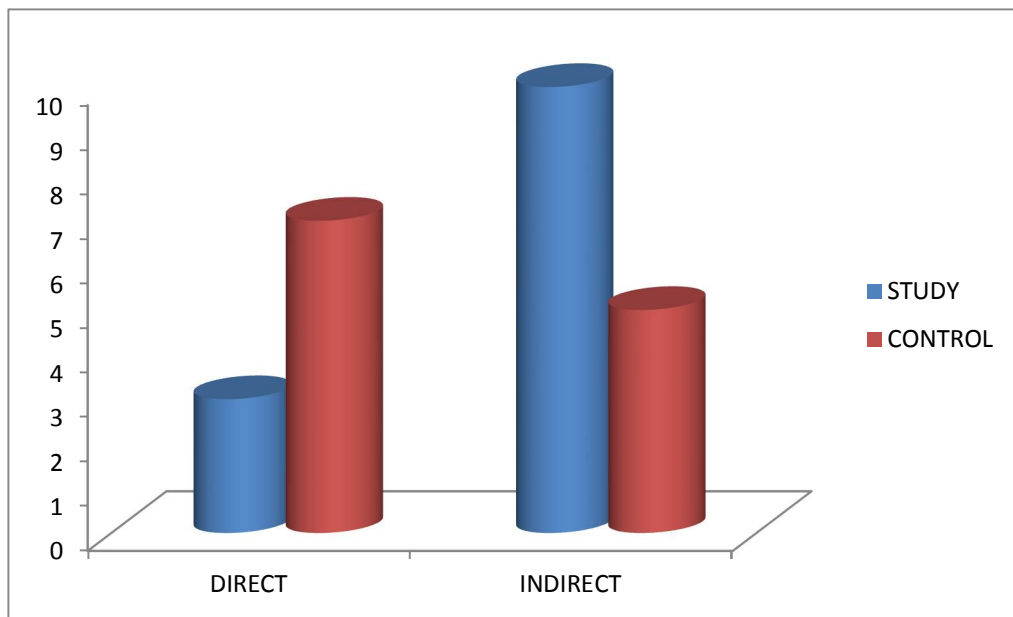


Table 13.

TYPE	STUDY	CONTROL	Chi sq	P value
DIRECT	3 (23.07%)	7 (58.33%)	2.35	0.13
INDIRECT	10 (76.92%)	5 (41.67%)		
TOTAL	13	12		

Our study included both direct as well as indirect hernia. The study group had 3 patients (23.07%) with direct hernia and 10 patients (76.92%) with indirect hernia. The control group had 7 patients (58.33%) with direct hernia and 5 patients (41.67%) with indirect hernia. This difference of distribution is statistically insignificant (0.13)

AGE GROUP PATTERN IN THE 2 GROUPS

Graph 14.

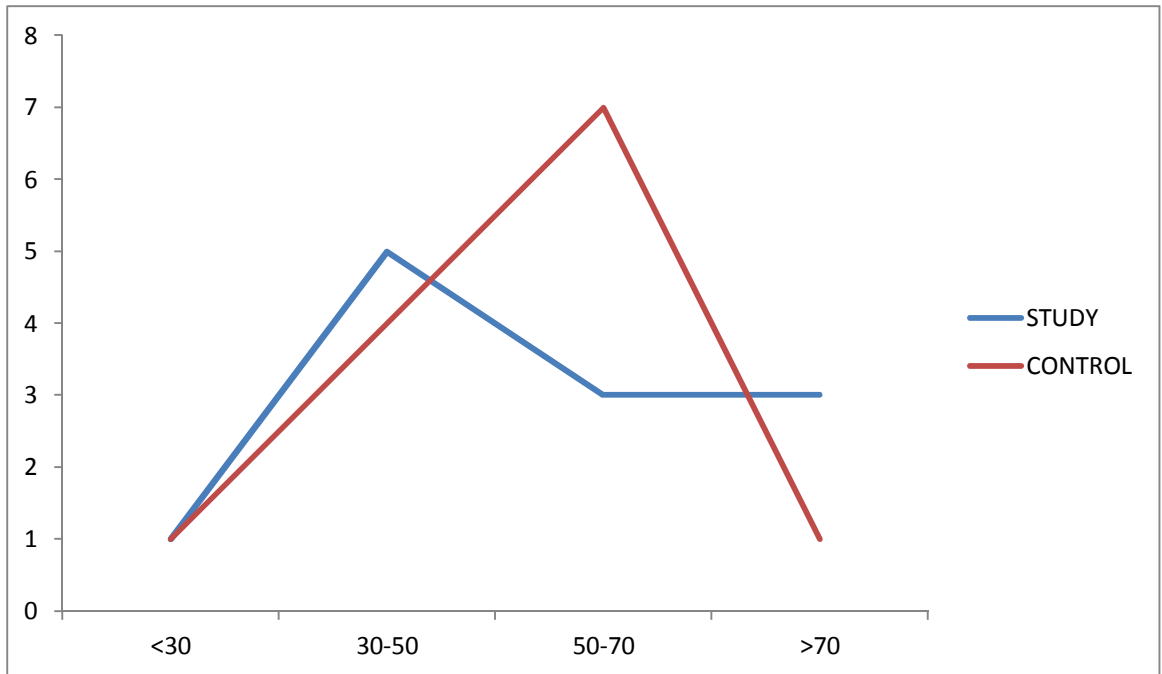


Table 14.

AGE	CONTROL	STUDY	Chi sq	P value
<30	1 (8.33%)	1 (7.69%)	3.612	0.31
30-50	5 (41.67%)	4 (30.77%)		
50-70	3 (25.00%)	7 (53.85%)		
>70	3 (25.00%)	1 (7.69%)		
TOTAL	12	13		

In our study, the most common age group with hernia was 40-60 years. There was almost equal distribution of age groups between control and study groups (p=0.31, difference is statistically non significant). This shows that age criteria was not a confounding factor in our study and the 2 groups were statistically matched.

OCCURRENCE OF SSI IN THE 2 GROUPS:

Graph 15

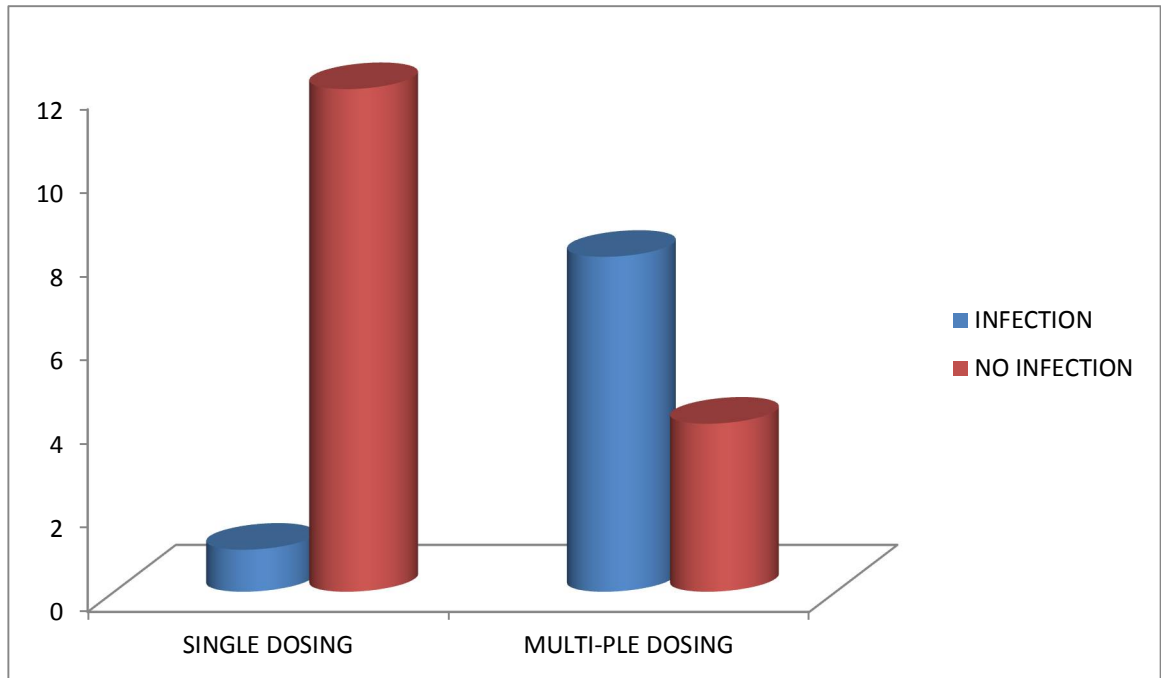


Table 15.

DOSING AND SSI	STUDY (SINGLE DOSING- PREOP)	CONTROL (MULTIPLE DOSING- POSTOP)	Chi sq	P value
INFECTION	1 (7.69%)	8 (66.67%)	9.42	0.002
NO INFECTION	12 (92.31%)	4 (33.33%)		
TOTAL	13	12		

In our study, the control group had received multiple dosing of antibiotics post operatively. The study group received a single dosing of CEFTRIAXONE preoperatively. On comparing this dosing method and timing with the occurrence of SSI, it was found that the study group had a highly significant reduction (p-0.002) in the occurrence of SSI compared to the control group. This proves the point that a single dose of CEFTRIAXONE preoperatively is much more effective than multiple postoperative dosing in control of surgical site infection.

AGE GROUPS AND SSI

Graph 16.

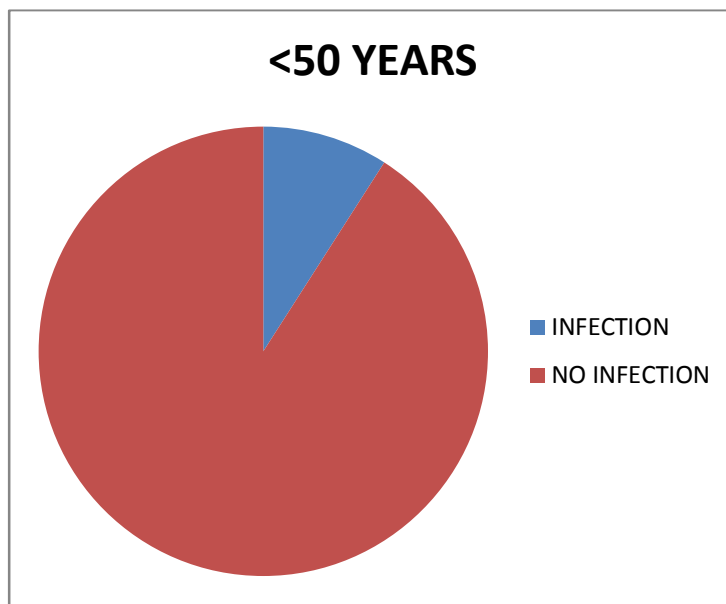
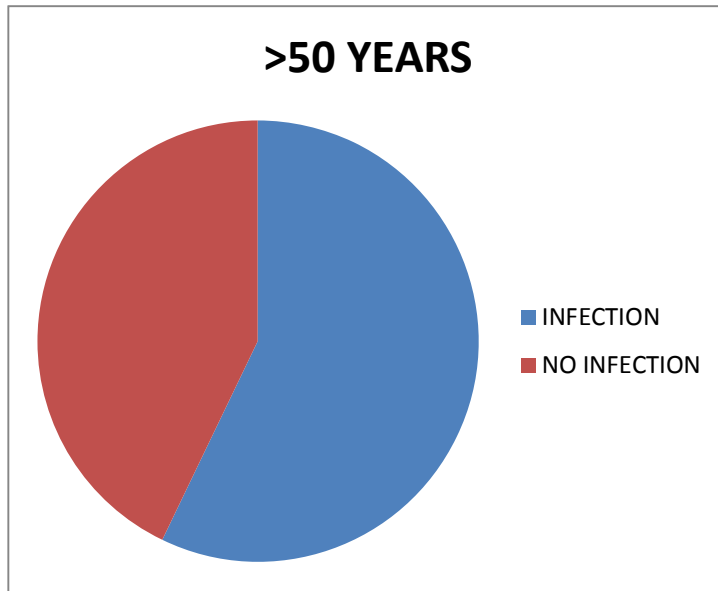


Table 16.

AGE & INFECTION	<50	>50	Chi sq	P value
INFECTION	1 (9.09%)	8 (57.14%)	6.19	0.013
NO INFECTION	10 (90.90%)	6 (42.85%)		
TOTAL	11	14		

Among 11 patients of age <50 years, 1 (9.09%) had SSI and 10 (90.90%) had no infection. Among 14 patients of age >50 years, 8 (57.14%) had SSI and 6 (42.85%) had no infection. On comparison, the occurrence was statistically significant (p-0.013), that is, the occurrence of SSI has a strong correlation with increasing age.

GENDER& INFECTION

Graph 17.

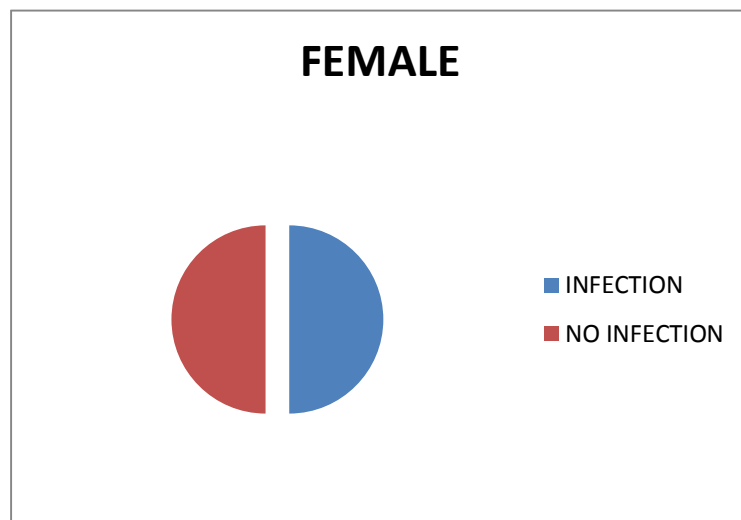
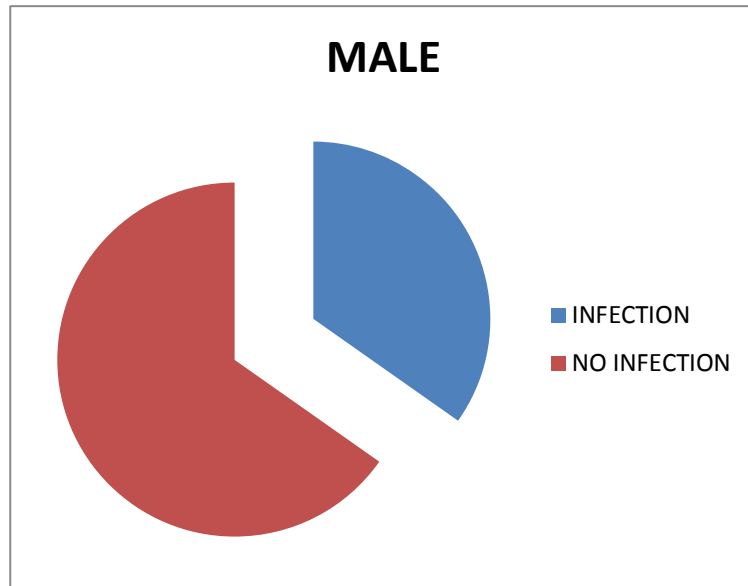


Table 17.

SEX & INFECTION	MALE	FEMALE	Chi sq	P value
INFECTION	8(34.78%)	1 (50.00%)	0.182	0.67
NO INFECTION	15 (65.22%)	1 (50.00%)		
TOTAL	23	2		

The occurrence of SSI had no significant difference (p-0.67) between males and female patients. Gender was not a deciding factor in the occurrence of SSI.

COMORBIDITY& SSI

Graph 18.

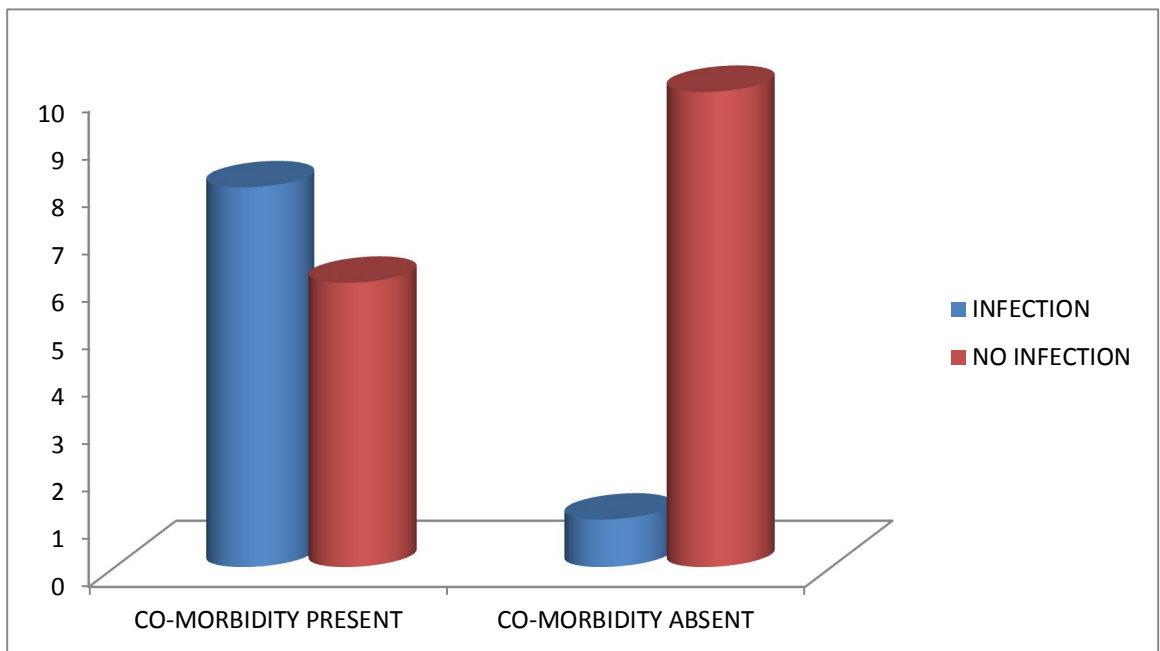


Table 18.

COMORBID & SSI	PRESENT	ABSENT	Chi sq	P value
INFECTION	8 (57.14%)	1 (9.09%)	6.17	0.01
NO INFECTION	6 (42.86%)	10 (90.90%)		
TOTAL	14	11		

In our study, out of 25 patients, 14 had co-morbidities such as diabetes mellitus and hypertension. It was seen that the patients with co-morbidities had a higher chance of occurrence (p-0.01) of SSI than the other patients.

COMORBID & STUDY GROUPS

Graph 19.

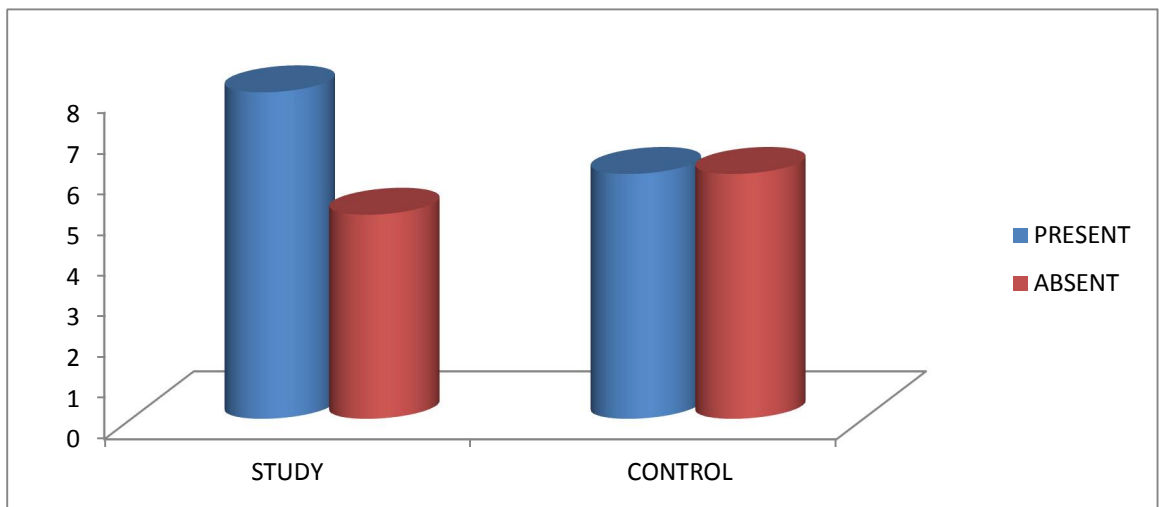


Table 19.

COMORBID & STUDY GROUPS	STUDY	CONTROL	Chi sq	P value
PRESENT	8 (61.54%)	6 (50.00%)	0.34	0.6
ABSENT	5 (38.46%)	6 (50.00%)		
TOTAL	13	12		

The 14 patients with co-morbidity were equally distributed between study and control groups. The study group had 8 patients (61.54%) with co-morbidity and the control group had 6 patients (50.00%) with co-morbidity, on comparing the distribution was equal and there was no significant difference (p=0.9) This proves that the co-morbidity factor didn't have an influence on the study result.

HOSPITAL STAY & GROUPS

Graph 20.

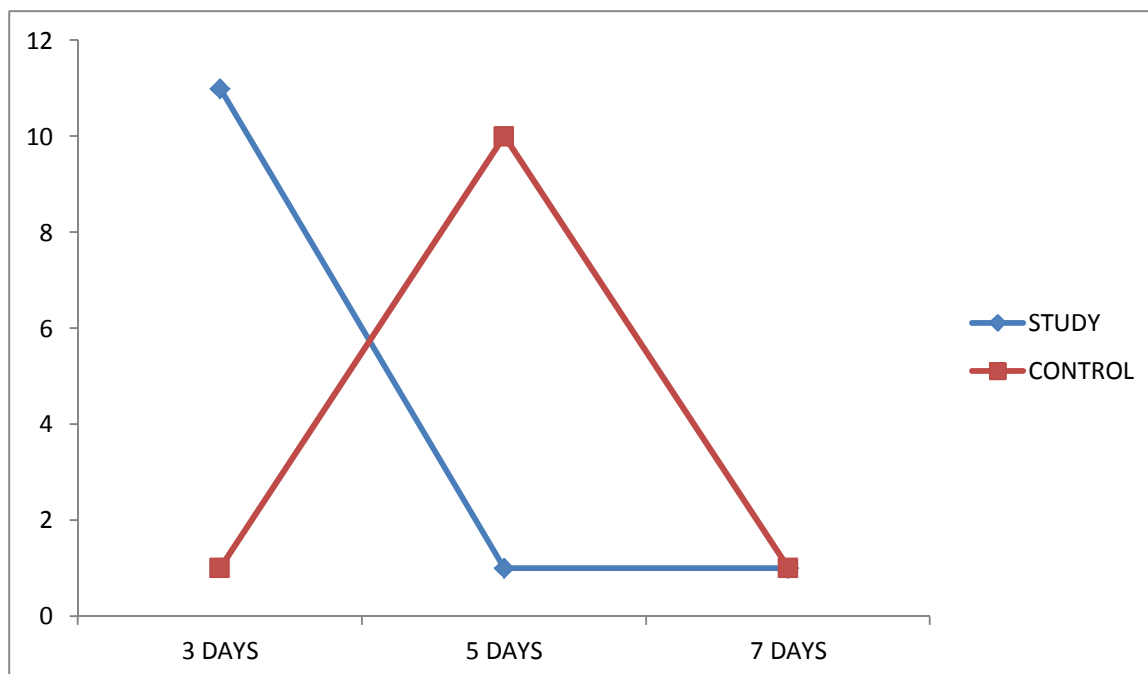


Table 20.

HOSPITAL STAY & GROUPS	STUDY	CONTROL	Chi sq	P value
3 DAYS	11 (84.61%)	1(8.33%)	15.684	0.0004
5 DAYS	1 (7.69%)	10 (83.33%)		
7 DAYS	1 (7.69%)	1 (8.33%)		
TOTAL	13	12		

According to our study, the number of patients who had a long duration of hospital stay was significantly higher ($p=0.0004$) in the control group than the study group. This is because the occurrence of SSI was more in the control group, and so those patients needed extra days of additional antibiotic coverage for treatment.

DURATION OF SURGERY & SSI

Graph 21.

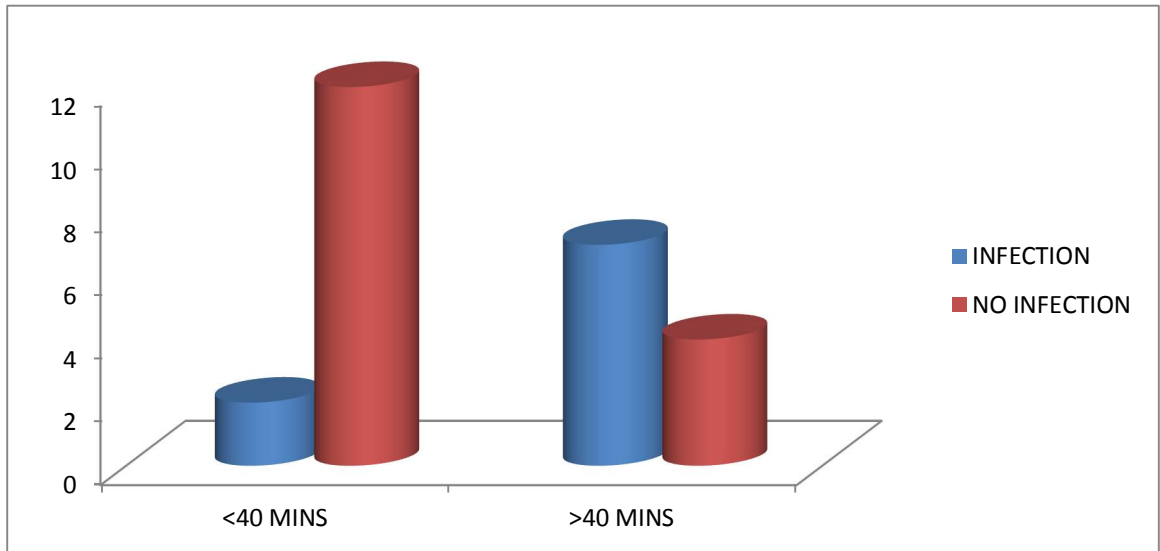


Table 21.

DURATION OF TREATMENT & SSI	<40 MINS	>40 MINS	Chi sq	P value
INFECTION	2 (14.28%)	7 (63.64%)	6.5	0.01
NO INFECTION	12 (85.72%)	4 (36.36%)		
TOTAL	14	11		

In our study, it was observed that prolonged duration of surgery, i.e. patients who were operated for more than 40 minutes had a higher chance of developing SSI than those who were operated for less than 40 minutes. This difference in occurrence of SSI is statistically significant (p-0.01)

DURATION OF SURGERY & GROUPS

Graph 22.

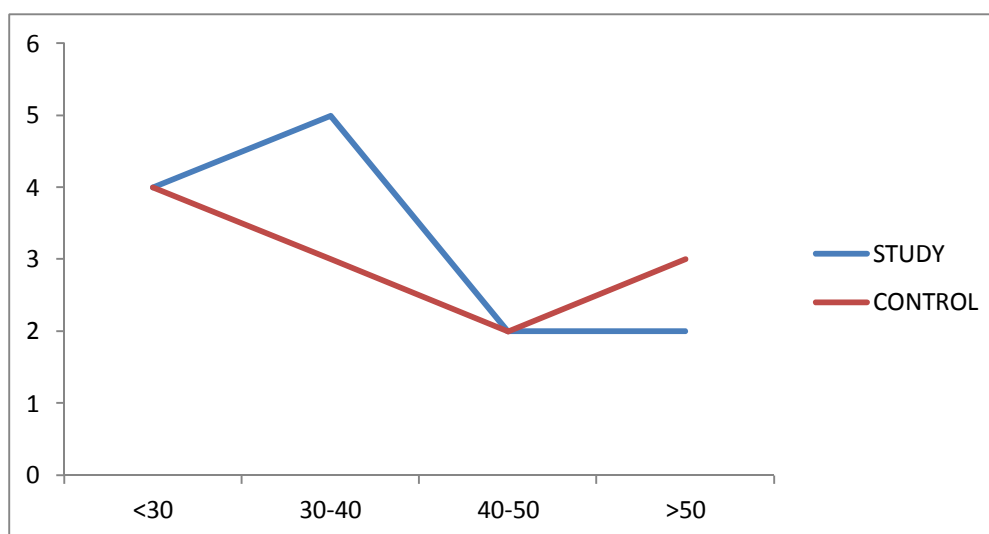


Table 22.

DURATION OF TREATMENT & GROUPS	STUDY	CONTROL	Chi sq	P value
<30	4 (30.77%)	4 (33.33%)	0.659	0.88
30-40	5 (38.46%)	3 (25.00%)		
40-50	2 (15.39%)	2 (16.67%)		
>50	2 (15.39%)	3 (25.00%)		
TOTAL	13	12		

In both the study and control groups there was random and almost equal distribution of patients who were operated for different duration.

(p-0.88)

ASA & SSI

Graph 23.

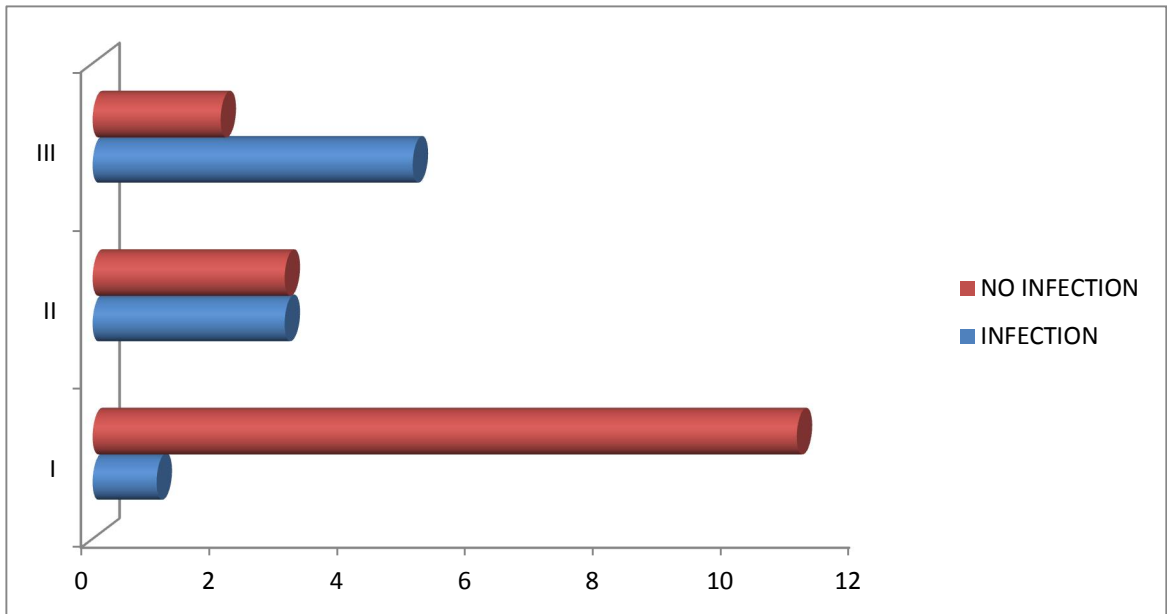


Table 23.

ASA & SSI	I	II	III	Chi sq	P value
INFECTION	1 (8.33%)	3 (50.00%)	5 (71.42%)	8.31	0.01
NO INFECTION	11 (91.67%)	3 (50.00%)	2 (28.57%)		
TOTAL	12	6	7		

In this graph, we compared the occurrence of SSI and ASA grading of anaesthesia the patients belonged to during surgery. Among the 12 patients who belonged ASA grade I, only 1 (8.33%) had SSI and the rest had no infection. In ASA II, 3 (50.00%) had infection and 3 had no infection. In ASA III, 5 (71.42%) had infection and 2 had no infection. This higher chance of occurrence of SSI in higher ASA grades is statistically significant ($p=0.01$), the cause being presence of co morbidities and other habitual factors.

TYPES OF ANESTHESIA & GROUPS

Graph 24.

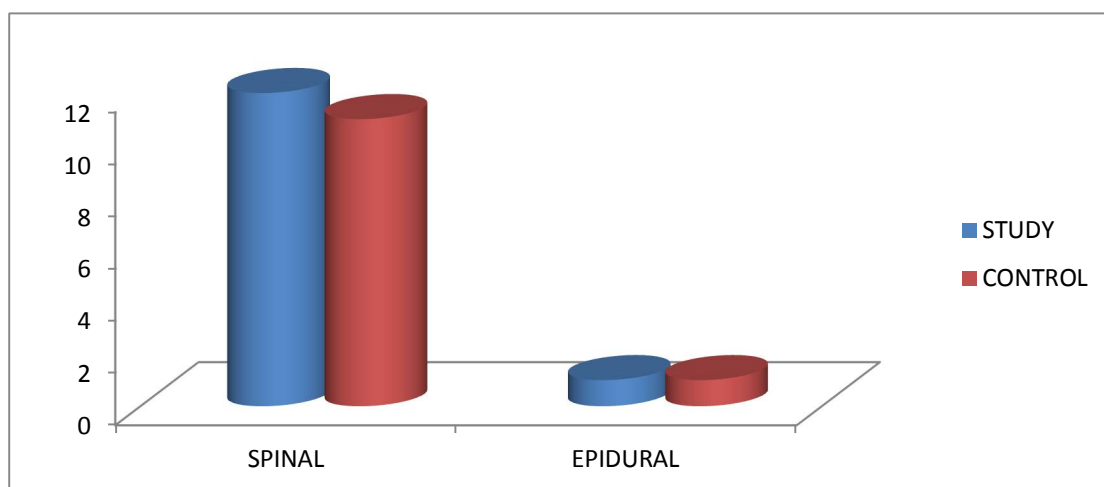


Table 24.

TYPES OF ANESTHESIA & GROUPS	STUDY	CONTROL	Chi sq	P value
SPINAL	12 (92.30%)	11 (91.67%)	2.794	0.09
EPIDURAL	1 (7.70%)	1 (8.33%)		
TOTAL	13	12		

This graph shows the distribution of patients who had spinal/epidural anaesthesia between our study and control groups. There was no significant difference ($p=0.09$) in the distribution of patients based on type of anaesthesia between the 2 groups.

MANAGEMENT

Graph 25.

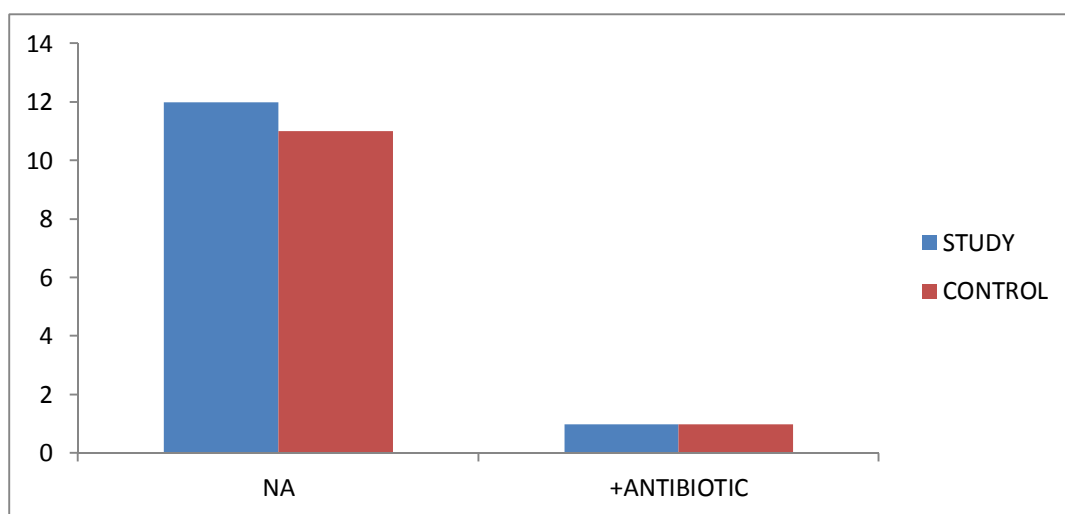


Table 25.

MANAGEMENT	STUDY	CONTROL	Chi sq	P value
NA	12 (92.30%)	11 (91.67%)	2.794	0.09
ADDITIONAL ANTIBIOTIC	1 (7.70%)	1 (8.33%)		
TOTAL	13	12		

Among 8 patients who developed SSI in control group, 1 (7.70%) needed additional antibiotic. The patient who developed SSI in study group also needed additional antibiotic. On comparison, there is no significant difference (p-0.09) in the administration of additional antibiotic as a part of SSI management between the 2 groups during their hospital stay.

CONCLUSION

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

Single dose preoperative CEFTRIAXONE prophylaxis was therapeutically efficient as well as cost effective in comparison with multiple post operative antibiotics usage in CLEAN CLASS 1 elective surgeries for preventing surgical site infection. The study shows that the cost of management of patients who is undergoing CLEAN CLASS 1 surgeries with respect to use of antibiotics can be reduce in Government set up by use of single dose CEFTRIAXONE antibiotic ,thereby reducing the financial burden to the Government.

RECOMMENDATION

Based on my study I would like to recommend single dose CEFTRIAXONE prophylaxis for all clean class I cases, as per the study results there is a 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. 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.
2. Reid MR. Some considerations of the problem of wound healing N Engl J Med 1936 ; 215 : 753.
3. Grey, J.E. and Harding, K.G. (2006) *ABC of Wound Healing*. BMJ Books. Blackwell, Oxford.
4. Williams, J.D. and Taylor, E.W. (2003) *Infection in Surgical Practice*. Arnold, London
5. Dunn DL, Simmons RL. The role of anaerobic bacteria in intra abdominal infections. *Rev Infect Dis*. 1984;6:S139-S146.
6. Bratzler DW, Dellinger EP, Olson KM, et al. Clinical practice guidelines for antimicrobial prophylaxis in surgery. *Am J Health Syst Pharm*. 2013;70:195-283.
7. Belda FJ, Aguilera L, Garcia de la Asuncion J, et al. Supplemental perioperative oxygen and the risk of surgical wound infection: a randomized controlled trial. *JAMA* 2005;294:2035–2042.
8. Keighiey MRB, Burdon DW. Antimicrobial prophylaxis in Surgery. Pitman, London 1979.

9. The choice of antibacterial drugs *Med Lett Drugs Ther* 1999; 41 : 95-104.
10. Kaiser A B. Antimicrobial prophylaxis in surgery. *N Engl J Med* 1986; 315 : 1121.
11. Cohen, I.K., 1998. A brief history of wound healing. 1st ed. Yardley Pa: Oxford Clinical Communications Inc.
12. Nosocomial infection rates for interhospital comparison: limitations and possible solutions. A Report from the National Nosocomial Infections Surveillance (NNIS) System. *Infect Control Hosp Epidemiol* 1991;12: 609–621.
13. Forse RA, Karam B, MacLean LD, et al. Antibiotic prophylaxis for surgery in morbidly obese patients. *Surgery* 1989;106:750–756.
14. Turina M, Cheadle WG: Management of established surgical site infections. *Surg Infect (Larchmt)* 7:S33–S41, 2006.
15. Smith BP, Fox N, Fakhro A, et al. “SCIP” ping antibiotic prophylaxis guidelines in trauma: The consequences of noncompliance. *J Trauma Acute Care Surg.* 2012;73(2):452-456.
16. Niederman MS: Appropriate use of antimicrobial agents: Challenges and strategies for improvement. *Crit Care Med* 31:608–616, 2003.
17. Bull Lowburg, Lilly. Methods of disinfection of hands and operative site. *British Journal of Medicine* 1964; 2 : 531.

18. 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.
19. Mohammed Sharif Auran, Dept. of Surgery, Peoples Medical College and Hospital, Nawabshah, *J. of Surg. Pakistan*. Jan-March. 2001 ; 16 (1).
20. Elek, S.D., and P.E. Conen, 1957. The virulence of *Staphylococcus pyogenes* for man: a study of problems with wound infection. *Br. J. Exp. Pathol.* 38, 573-86.
21. Richet, H.M., P.C. Craven, J.M. Brown, B.A. Lasker, C D . Cox, M.M. McNeil , et al., 1991. A cluster of *Rhodococcus (Gordona) bronchialis* sternal wound infections after coronary-artery bypass surgery. *N . Engl. J. Med.* 324,104-9
22. Cunningham M, Bunn F, Handscomb K: Prophylactic antibiotics to prevent surgical site infection after breast cancer surgery. *Cochrane Database Syst Rev* (2):CD005360
23. Leaper, D.J. and Harding, K.G. (1998) *Wounds: Biology and Managemen* Oxford Medical, Oxford.
24. Solomkin JS, Yellin AE, Rotstein OD, et al. Protocol 017 Study Group. Ertapenem versus piperacillin/tazobactam in the treatment of complicated intra abdominal infections: results of a double-blind, randomized comparative phase III trial. *Ann Surg.* 2003;237:235-245.

25. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. *Clin Infect Dis* 2005;41:1373–1406
26. Cruise PJE and Foord R. A five year prospective study of 23,649 surgical wounds' *Archives of surgery* 1913 ;107 :206.
27. Lilani, Jangale N. Department of microbiology, Department of surgery, Grand medical college, Byculla, Mumbai *Indian J Surg* 1997 ;90-3.
28. Sanchez-Manuel FJ, Seco-Gil JL. Antibiotic prophylaxis for hernia repair. *Cochrane Database Syst Rev*. 2004;(4):CD003769.
29. Smith RL, Bohl JK, McElearney ST, Friel CM, Barclay MM, Sawyer RG. Wound infection after elective colorectal resection. *Ann Surg*. 2004;239(5):599–605.
30. Stone HH. Basic principles in the use of prophylactic antibiotics. *J Antimicrob Chemother*. 1984;14(Suppl B):33–7.
31. Ronald AR. Antimicrobial prophylaxis in surgery. *Surgery* 1983; 93(1 Pt 2):172 3. Antimicrobial prophylaxis for surgery. *Med Lett Drugs Ther*. 1985;27(703):105
32. EWMA (2006) *Management of Wound Infection*. EWMA Position Document. Medical Education Partnership, London.

33. Wideman GL, Matthijssen C. Comparative efficacy of cefotaxime and cefazolin as prophylaxis against infections following elective hysterectomy. *Clin Ther* 1982;5 Suppl A:67-73.
34. Woods RK, Dellinger EP. Current guidelines for antibiotic prophylaxis of surgical Wounds. *Am Fam Physician* 1998;57:2731-40
35. Fernandez Arjona M, Herruzo Cabrera R, Gomez-Sancha F, Nieto S, Rey Calero J. Economical saving due to prophylaxis in the prevention of surgical wound infection. *Eur J Epidemiol* 1996;12:455-9.
36. Mu Y, Edwards JR, Horan TC, et al. Improving risk-adjusted measures of surgical site infection for the national healthcare safety network. *Infect Control Hosp Epidemiol*. 2011; 32(10):970-986.

CONSENT FORM

For Inclusion in study “**A COMPARATIVE STUDY OF SINGLE DOSE PREOPERATIVE CEFTRIAZONE AND ROUTINE CONVENTIONAL POSTOPERATIVE PROPHYLAXIS IN ELECTIVE GENERAL SURGICAL CASES**”.

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 anaesthesia 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

(Patient)

Investigator Sign

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

Height

Pallor yes/no

Weight

Icterus yes/no

Body Mass Index

cyanosis yes/no

Clubbing yes/no

Pulse Rate

CVS :

RS :

CNS :

P/A :

INVESTIGATIONS :

BLOOD INVESTIGATIONS :

HB :

TC :

DC :

ESR :

PLATELET COUNT :

SERUM PROTEINS :

HIV :

HBSAG :

IMAGING :

CXR PA VIEW :

AXR ERECT :

USG ABDOMEN :

CT ABDOMEN :

DIAGNOSIS :

CLASS I:

CONTROL/STUDY :

TREATMENT GIVEN :

POST OPERATIVE COMPLICATIONS: Yes/No

ORGANISM GROWN :

SSI : Yes / No

CLASSIFICATION : Superficial / Deep

MANAGEMENT :

FOLLOWUP :

MASTER CHART - CLASS I (CLEAN CASES WITHOUT IMPLANT) STUDY GROUP

S.NO	NAME	AGE/SEX	IP.NO	TYPE OF SURGERY	SSI	TYPE OF SSI	SOUTHAMPTON SCORE	ORGANISM	ADDITIONAL ANTIBIOTIC	DURATION OF POST OP STAY
				HYDROCOELE						
1	DURAISAMY	90/M	57970	B/L	PRESENT	DEEP	3	E COLI	YES	7
2	DAVID	22/M	29817	LEFT	NIL	-	0	-	NA	3
3	VELLAISAMY	37/M	46996	LEFT	NIL	-	0	-	NA	3
4	KARRUPUSAMY	98/M	26128	LEFT	NIL	-	0	-	NA	5
5	PALANIYAPPAN	74/M	48104	RIGHT	PRESENT	SUPERFICIAL	3	STAPH AUREUS	NA	5
6	ARULSELVAM	50/M	65341	RIGHT	NIL	-	0	-	NA	3
7	KUMAR	50/M	10272	LEFT	NIL	-	0	-	NA	3
8	CHINNAIYA	58/M	61387	LEFT	NIL	-	0	-	NA	3
9	KANAGARAJ	64/M	51390	RIGHT	NIL	-	0	-	NA	3
10	BASHA	55/M	82309	LEFT	NIL	-	0	-	NA	5
11	SUNDARRAJ	66/M	43940	LEFT	NIL	-	0	-	NA	3
12	SOUNDARRAJ	54/M	244929	RIGHT	NIL	-	0	-	NA	3
13	ROMAN	60/M	46333	RIGHT	NIL	-	0	-	NA	3

MASTER CHART - CLASS I (CLEAN CASES WITHOUT IMPLANT) CONTROLGROUP

S.NO	NAME	AGE/SEX	IP.NO	TYPE OF SURGERY	SSI	TYPE OF SSI	SOUTHAMPTON SCORE	ORGANISM	ADDITIONAL ANTIBIOTIC	DURATION OF POST OP STAY
				HYDROCOELE						
1	MUTHU	65/M	50853	B/L	PRESENT	SUPERFICIAL	3	STAPH AUREUS	NA	5
2	SUSAIYAN	69/M	49565	B/L	PRESENT	DEEP	3	E COLI	YES	7
3	MUSTAFFA	53/M	43963	B/L	PRESENT	SUPERFICIAL	3	E COLI	NA	5
4	KAVIYARASU	23/M	64327	LEFT	NIL	-	0	-	NA	3
5	JAGENDRAN	75/M	52283	RIGHT	PRESENT	-	3	NO GROWTH	NA	3
6	KRISHNAN	50/M	97607	LEFT	PRESENT	DEEP	3	PSEUDOMONAS	YES	7
7	RAJAN	50/M	52228	RIGHT	PRESENT	SUPERFICIAL	3	E COLI	NA	5
8	DINESH	35/M	41009	LEFT	NIL	-	0	-	NA	3
9	JAYAKUMAR	38/M	28537	LEFT	PRESENT	SUPERFICIAL	3	STAPH AUREUS	NA	5
10	SARAVANAN	52/M	39900	RIGHT	NIL	-	0	-	NA	3
11	ELANGO VAN	62/M	43669	LEFT	PRESENT	-	3	NO GROWTH	NA	3
12	NEELAMEGAM	49/M	239556	RIGHT	PRESENT	-	3	E COLI	NA	5

MASTER CHART - CLASS I (CLEAN CASES WITH IMPLANT) STUDY GROUP

S.NO	NAME	AGE/SEX	IP.NO	TYPE OF SURGERY	SSI	TYPE OF SSI	ORGANISM	SOUTHAMPTON SCORE	ADDITIONAL ANTIBIOTIC	DURATION OF POST OP STAY
				INGUINAL HERNIA						
1	PONNAMAL	70/F	184729	LEFT INDIRECT	NIL	-	-	0	NA	3
2	VELAYUDHAM	48/M	3794	B/L DIRECT	NIL	-	-	0	NA	3
3	PRAMOD	38/M	8863	B/L INDIRECT	NIL	-	-	0	NA	3
4	ARUMUGAM	71/M	16894	B/L DIRECT	NIL	-	-	0	NA	5
5	NATRAJAN	67/M	209405	LEFT DIRECT	PRESENT	DEEP	E COLI	3	YES	7
6	RAMACHANDRAN	35/M	15417	RIGHT INDIRECT	NIL	-	-	0	NA	3
7	IBRAHIM	40/M	174702	RIGHT INDIRECT	NIL	-	-	0	NA	3
8	KATHANNA	55/M	182633	RIGHT INDIRECT	NIL	-	-	0	NA	3
9	NAZAR	21/M	213066	RIGHT INDIRECT	NIL	-	-	0	NA	3
10	THANGAVEL	58/M	216704	RIGHT INDIRECT	NIL	-	-	0	NA	3
11	RAVIKUMAR	58/M	206324	LEFT INDIRECT	NIL	-	-	0	NA	3
12	MURUGESAN	58/M	203964	LEFT INDIRECT	NIL	-	-	0	NA	3
13	SIVANANDHAN	62/M	182376	RIGHT INDIRECT	NIL	-	-	0	NA	3

MASTER CHART - CLASS I (CLEAN CASES WITH IMPLANT) CONTROL GROUP										
S.NO	NAME	AGE/SEX	IP.NO	TYPE OF SURGERY	SSI	TYPE OF SSI	ORGANISM	SOUTHAMPTON SCORE	ADDITIONAL ANTIBIOTIC	DURATION OF POST OP STAY
				INGUINAL HERNIA						
1	YAMUNA	32/F	218258	LEFT INDIRECT	PRESENT	SUPERFICIAL	E COLI	3	NA	5
2	JAYACHANDRAN	25/M	201925	LEFT INDIRECT	NIL	-	-	0	NA	5
3	MANI	77/M	203898	B/L DIRECT	PRESENT	SUPERFICIAL	E COLI	3	NA	5
4	MURUGAN	46/M	216550	LEFT INDIRECT	NIL	-	-	0	NA	5
5	VAIYAPURI	74/M	183933	RIGHT DIRECT	PRESENT	SUPERFICIAL	E COLI	3	NA	5
6	RAMAIYA	75/M	207697	RIGHT DIRECT	PRESENT	SUPERFICIAL	NO GRWOTH	3	NA	5
7	RAVICHANDRAN	62/M	16874	RIGHT DIRECT	PRESENT	SUPERFICIAL	NO GROWTH	3	NA	5
8	PALANISAMY	50/M	17896	RIGHT DIRECT	PRESENT	SUPERFICIAL	KLEBSIELLA	3	YES	5
9	JANARTHANAN	63/M	192540	B/L DIRECT	PRESENT	SUPERFICIAL	NO GROWTH	3	NA	5
10	GURUVAIYA	68/M	198002	B/L DIRECT	PRESENT	DEEP	STAPH AUREUS	3	NA	7
11	SENTHILKUMAR	40/M	12412	B/L INDIRECT	NIL	-	-	0	NA	5
12	SARAVANAKUMAR	34/M	211208	B/L INDIRECT	NIL	-	-	0	NA	3