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Incidence Of Surgical Site Infection In Open Mesh Hernioplasty Using Prophylactic Antibiotic

Yash Pandey*, V.V Kanase, Ria Rai, Anish Pandey, Piyush Prajapati, Hrishikesh Deka, Sudhanshu Tripathi, and Shakuntala Prajapati

Department of Gen Surgery, Krishna Institute of Medical Sciences And Deemed University. Karad, Maharashtra, India.

ABSTRACT

Prophylactic antibiotic has been established as a significant preventive mesure in reducing the incidence SSI in open mesh hernioplasty. prophylactic antibiotics may inhibit the adherence of bacteria to the prosthesis and subsequently their growth rates.[11] This study endeavours to find whether antibiotic prophylaxis improves outcome in a hospital setup of a developing country, whether it is feasible in such a set up and lastly whether it is cost-effective. Objectives of the study is to study proportion of early postoperative infection after single dose and multiple dose antibiotic prophylaxis in mesh hernioplasty, and to compare proportion of early post-operative infection in mesh hernioplasty single dose of prophylactic antibiotic and multiple dose post-operative antibiotics. This study was conducted in general surgery department of KIMS, Karad from Oct 2014 to July 2016. Sample size was 100 , equally divided into 2 groups A and B. Patients of either age and gender undergoing mesh hernioplasty surgery and available for complete follow-up were included in the study.Exclusion crieteria: diabetes mellitus, any infective focus in the body, poor quality of the skin at the incision site, allergy to cephalosporin and history of use of antibiotics within last 7 days were excluded from the study. Patients in group A were given only single dose of 1 gram Ceftriaxone + sulbactum 500mg intravenously within 30 minutes of the initial operative incision. This group did not receive postoperative antibiotics and were followed up with regular sterile dressings. Patients in group B were given routine postoperative antibiotics according to usual established protocol in the hospital and were followed up with regular sterile dressings.. Wound was examined on 3rd, 8th, 15th and 28th postoperative. The parameters like age,weight,type of hernia,mean Hb level,pre operative bath,electrocautry use,SSI was compared and was statistically insignificant.The advantage of pre-antibiotic usage could be established over post-antibiotic usage in this study as the results states that infection rate in case group who received single dose of Ceftriaxone 1g with Sulbactum 500 mg was less in comparison to the patients who received routine post-operative antibiotics though p-value in this study was insignificant. Clinical results seem to justify the use of single dose antibiotic prophylaxis to patients undergoing open mesh hernioplasty.

Keywords: surgicial site infection, mesh hernioplasty,prophylactic antibiotic

Corresponding author

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INTRODUCTION

Surgical site infections (SSIs) remain a major clinical problem in terms of morbidity, mortality, time spent in hospital and overall direct and indirect costs.[1–3] Despite progress in their prevention, SSIs remain one of the most common adverse events in hospitals, accounting for 11 % to 26 % of all healthcare-associated infections [4]. Surgical patients can develop several post-operative infections; wound infections, are common causes of post-operative morbidity and prolonged hospitalization. SSIs increase the total hospital bill by an additional 10–20%[5] lead to 80,000 deaths and are associated with an annual treatment cost of two billion US dollars.[6] S. aureus SSIs can be life-threatening, being associated with a mortality rate of 5 %, more than 2 extra weeks of time spent in hospital and around an extra cost of 50,000 US dollars.[7]

The description of Lichtenstein tension free mesh repair introduced a new era in groin hernia repair.[8] It is one of the most common procedures performed by general surgeons. Inguinal hernia repair is the most commonly performed operation in the United States, owing to a significant lifetime incidence and variety of successful treatment modalities. It offers many advantages, such as simplicity, effectiveness, minimal pain, early return to work, low recurrence rates and a high patient satisfaction. It is currently considered as the preferred method for the plastic reconstruction of inguinal region. Inguinal hernia repair is one of the most common procedures performed by general surgeons. Even though hernia is classified as a clean surgery, the reported incidence of wound infection varies from 0% to 9%.[9]

The risk of wound infection increases after introduction of prosthetic material in the body, which is attributed to the detrimental effect of the prosthesis on the host defense mechanism.[10] The fear of infection of the prosthetic mesh raised the question of the potential role of antibiotic prophylaxis. It has been shown that administration of prophylactic antibiotics may inhibit the adherence of bacteria to the prosthesis and subsequently their growth rates.[11]

In earlier studies, the first randomized control trial on the role of antibiotic prophylaxis in mesh repair of inguinal hernia was done in 2001 by Yerdel et al., who advocated the use of prophylactic antibiotics.[12] However, subsequent trials have produced varied results. A Cochrane meta-analysis on this topic in 2004 concluded that antibiotic prophylaxis in mesh repair of inguinal hernias can neither be recommended nor discarded.[13]

Postoperative wound infections have an enormous impact on patients' quality of life and contribute substantially to the cost of patient care. It continues to be a major source of morbidity and a disconcerting source of mortality in surgical patients.

The potential consequences for patients range from increased pain and care of an open wound to sepsis and even death. Approximately 1 million patients have such wound infections each year in the United States, extending the average hospital stay by one week and increasing the cost of hospitalization by 20 percent.[14] This translates to an additional $1.5 billion in health care costs annually.[15]

The impact of such a sepsis doubles in our scenario because not only the cost of hospitalization is to be beared by the patient but also he has to lose his livelihood for the period of hospitalization.

Infection is encountered by all surgeons: by the nature of their craft, they invariably impair the first line of host defenses the cutaneous or mucosal barrier. The entrance of microbes into host tissues is the initial requirement for infection. The occurrence of wound infection requires a local inoculum sufficient to overcome host defenses and establish growth. The process is complex and depends on the interaction of various host, local tissue and microbial virulence factors. Measures intended to prevent wound infection typically attempt to modify the host and local tissue factors and include, for example, preoperative optimization of comorbid illness, control of the operative environment, proper cleansing of the skin and use of aseptic surgical technique. Preventing microbial penetration, reducing the microbial inoculum, and treating established infection have been important developments in field of surgery. Antibiotic prophylaxis is only one relatively minor effort among numerous preventive measures, but the efficacy and impact of antimicrobial prophylaxis has clearly been demonstrated to be significant.[16,17,18] Explicit laboratory studies were confirmed by multiple clinical trials that showed systemic antibiotics to be highly effective when they were used just before, an operation.

This study endeavours to find whether antibiotic prophylaxis improves outcome in a hospital setup of a developing country, whether it is feasible in such a set up and lastly whether it is cost-effective.

AIMS AND OBJECTIVES

- To study proportion of early postoperative infection after single dose antibiotic prophylaxis in mesh hernioplasty.
- To study proportion of early post-operative infection after multiple dose post-operative antibiotics in mesh hernioplasty.
- To compare proportion of early post-operative infection in mesh hernioplasty single dose of prophylactic antibiotic and multiple dose post-operative antibiotics.

HISTORICAL ASPECTS AND REVIEW OF LITERATURE

For most of surgical history, death from infection was common, although it was not until the end of the nineteenth century that the bacterial cause of surgical infection was appreciated.

Before antiseptic practices were instituted, mortality rates for amputations in times of war between 1745 and 1865 were between 25 and 90 percent. Mortality rates for amputation in civilian practice during the same period ranged from 5 to 50 percent.

The introduction of anesthesia by Long in 1842 and by Morton in 1846 increased the scope of surgery by permitting operations on body cavities and allowing surgeons to operate more slowly and deliberately, so that death from blood loss was diminished. Infection remained a great problem, however. Many surgeons realized that a more favorable prognosis was associated with an infection that developed “laudable pus” rather than a more serious infection that was not associated with purulence. The practical source of the belief in “laudable pus” is likely based on the fact that only living patients produced pus. Major surgery was almost invariably followed by infectious complications, typified by erysipelas, rapidly progressive soft tissue infections (streptococcal or mixed synergistic infections) and tetanus. Associated mortality was high. Surgeons did not yet understand the cause of infection.

Attention to surgical wounds is exemplified historically by attending to gunshot wounds with a creechle of worms, rose oiland moss from the skull of a mummy collected at full moon (certain references indicate that this boiling concoction was incomplete without the addition of fresh puppies). So soldiers, already horribly damaged, were then scalded with boiling oil. Ambroise Pare introduced the use of egg yolk, rose oil and turpentine (not boiled) as a less irritating emollient and was regarded as progressive. Ambroise Pare substituted egg yolk, oil of roses, and turpentine for boiling oil after a twist of fate where all the boiling oil ran out at a medical post in the battlefield.[19]

He then used the ancient turpentine remedy and discovered that it was far more efficient at healing the wounds than the boiling oil.

In the 1600s, wound infection was so common that redness, warmth and purulence were thought to be desirable features of wound healing.

Sir John Hunter (1728-1793) in his book 'Lectures on the Principles of Surgery' has made following comment about treatment of infected wounds.

“Many wounds ought to be allowed to scab in which this process is now prevented; and this arises, I believe, from the conceit of surgeons who think themselves possessed of powers superior to nature and therefore have introduced the practice of making sores of all wounds. The mode of assisting the cure of wounds by permitting a scab to form is likewise applicable, in some cases, to that species of accident where the parts have notonly been lacerated but deprived of life this practice is the very best for burns and scalds.”[20]

Joseph Lister (1827-1912) made one of the great contributions to surgery by demonstrating that antisepsis could prevent infection and hence compound fractures did not have to be treated by amputation.[21]

In March 1865 he began placing pure carbolic acid into wounds. Later he gradually reduced the concentration to 10, 5, and 2.5 percent.

In 1867 he published his initial series of papers on antisepsis, reporting among other things, that compound fractures healed without infection when the wounds were treated withcarbolic acid.

Wound antisepsis was not new with Lister. More than 20 articles appeared in British medical publications between 1859 and 1865 describing antiseptic treatment of wounds. Numerous agents had been placed in wounds since ancient times in an attempt to foster healing and prevent death turpentine, pitch and tar, balsams and balms, myrrh and frankincense, honey, alcohol, glycerin, mercuric chloride, silver nitrate, iodine, hypochlorites, creosote, ferric chloride, zinc chloride, and carbolic acid.

In 1871 Lister began to use a carbolic acid spray to reduce contamination of the operating room atmosphere, a practice he abandoned in 1887. The “antiseptic principle” or “Listerian method” emphasized antiseptic treatment of wounds after the operation. Although initially resisted by many surgeons (more by British and American surgeons than by European surgeons), they were gradually adopted. The introduction of carbolic acid spray (used on the entire operating room, patient and surgeons) by Lister in 1867 led to a dramatic reduction in infection rates to less than 10 percent. Nevertheless, the “antiseptic principle” was not widely accepted. Lister's results, however, fostered a context more accepting of Pasteur's theory of putrefaction that purulence was caused by microorganisms.

In 1877, Pasteur also demonstrated the phenomenon of antibiosis by proving that growth of anthrax bacilli in urine was inhibited by airborne bacteria.

In the mid-1800s, Semmelweis documented efficacy of handwashing in reducing puerperal sepsis which was introduced by himself in his ward.

Holmes further popularized the practice.

Despite this the widespread practice of handwashing for the surgical team was not established until the early 20th century. Surgeons washed their hands after, but seldom before, operations. When asked what was new in surgery in 1882, Ernst Bergmann said, “Today we wash our hands before an operation.” Gloves were not worn routinely until the early part of the twentieth century. Only gradually and with much opposition was aseptic surgery adopted. Sterilization of instruments, first by chemicals and then by steam, came into practice in the 1880s and 1890s. Hand washing and the wearing of masks, caps, gowns, and gloves were also introduced about this time. After the adoption of handwashing and the use of sterile gloves, gowns and supplies (autoclave), infection rates for clean procedures approached modern rates. However, infection rates for procedures of the gastrointestinal tract remained high as a result of the endogenous origin of the bacteria.

William Stewgrt Halsted (1852-1922) introduced rubber gloves for his scrub nurse (and future Mrs. Halsted), Caroline Hampton, because the corrosive sublimate used to sterilize instruments, mercuric chloride, irritated her skin. One of Halsted's students, Joseph Bloodgood, introduced their routine use by the entire operating team.

The introduction of antibiotics was a major step in the treatment of infections.

In 1935, Domagk demonstrated the therapeutic effect of Prontosil, a sulfonamide dye, in pyogenic infection.

In 1928, Alexander Fleming first reported the discovery of penicillin but it was not used clinically.

Chain and Howard Florey (1941) made the first clinical use of penicillin.

In 1944, Waksman and colleagues undertook a systematic search of Actinomycetes as a source of antibiotics and discovered streptomycin.

All three groups of scientists Domagk, Fleming-Chain- Florey and Waksman received Nobel prize for their discoveries. Penicillin was then rapidly introduced into general clinical medicine and was followed by streptomycin and numerous other antibiotics. It was hoped that antibiotics would eliminate the risk of infection as a surgical complication and would enable established infection to be cured easily, but this has not been the case.

Wound infection and other postoperative infections continue to be a problem even though antibiotics have reduced their risk.

The widespread use of antibiotics has even led to the emergence of strains of antibiotic-resistant bacteria.

In 1943 just four years after drug companies began mass- production of penicillin, microbes began appearing that could resist it.

The first microbe to battle penicillin was Staphylococcus aureus. This bacterium is often a harmless passenger in the human body, but it can cause illness, such as pneumonia or toxic shock syndrome, when it overgrows or produces a toxin.

In 1967, another type of penicillin-resistant pneumonia, caused by Streptococcus pneumoniae and called pneumococcus, surfaced in a remote village in Papua New Guinea. At about the same time, American military personnel in Southeast Asia were acquiring penicillin-resistant gonorrhea from prostitutes.

In 1976, when the soldiers came home from Vietnam war, they brought the new strain of gonorrhea with them, and physicians had to find new drugs to treat it.

In 1983, a hospital-acquired intestinal infection caused by the bacterium Enterococcus faecium joined the list of bugs that outwit penicillin.

Drug resistance is an especially difficult problem for hospitals harboring critically ill patients who are less able to fight off infections without the help of antibiotics. Heavy use of antibiotics in these patients selects for changes in bacteria that bring about drug resistance. Unfortunately, this worsens the problem by producing bacteria with greater ability to survive even in the presence of our strongest antibiotics. These even stronger drug-resistant bacteria continue to prey on vulnerable hospital patients. Thus the vicious cycle continues.

The nature of postoperative infections has also changed because of the many patients (debilitated, elderly, cancer patients) being operated who have compromised host defenses or who are given drugs that inhibit host defenses (cancer chemotherapy agents, immunosuppressants to prevent organ transplant rejection).

It was also hoped that antibiotics would cure most infections even without operation. While the introduction of antibiotic therapy was a giant step in the treatment of nonsurgical infections, it had a much smaller impact in the treatment of surgical infections. It was found that although antibiotic therapy was a monumental advance in the treatment of infections, for patients with surgical infection it constitutes only a part of the treatment. Surgical infections generally require an operative procedure (or radiology-assisted percutaneous drainage) for a successful outcome. In future, continued improvement in the treatment outcome of surgical infection is more likely to stem from such factors as earlier and better means of diagnosis, improved patient care, and therapy directed against bacterial products or host responses than from improvements in antimicrobial therapy.

Following the introduction of antibiotics, early clinical trials in the 1950s reported either no benefit or a higher infection rate with antibiotic prophylaxis. Moreover, the emergence of resistant strains was attributed, in part, to such use of antibiotics. Although a small number of authors supported the use of prophylactic antibiotics for “dirty” or contaminated cases, most did not recommend their use in cleaner cases.

Fortunately, studies by Burke in the early 1960s revealed the critical flaw in previous investigations and clinical failures. Burke administered a single dose of penicillin systemically at various times before and

after the inoculation of penicillin-sensitive *Staphylococcusaureus* in the dermis of guinea pigs. Administration of antibiotic either shortly before or after the inoculation of organisms resulted in lesions histologically identical to lesions induced by intradermal inoculation with killed organisms. Delaying the administration of antibiotic by as little as three hours resulted in lesions identical to those in animals not receiving antibiotics. The critical dependence of prophylactic efficacy on timing of administration was soundly established and subsequently shown to depend on the presence of peak antibiotic levels in the tissue at a time when the local concentration of microorganisms would otherwise be high. Subsequent investigation has focused on the delineation of specific procedures, prophylactic regimens and the optimization of efficacy.

ANTIBIOTIC PROPHYLAXIS

Antibiotic prophylaxis is strategy of administering antibiotics to a patient before the evidence of an infection with an intention to prevent infection in that individual.

An appropriate prophylactic antibiotic should

1. Be effective against microorganisms anticipated to cause infection;
2. Achieve adequate local tissue levels;
3. Cause minimal side effects;
4. Be relatively inexpensive,
5. Not be likely to select virulent organisms.

The agent selected should manifest sustained antibiotic activity in the surgical wound.[38] Declining wound antibiotic activity is an indication to re-administer the agent or to seek a drug that produces more sustained activity levels.[22]

Studies recommend that adequate tissue levels of antibiotic should be ensured throughout the duration of the procedure.

The duration of an adequate tissue level of the antibiotic need not exceed the operative period. The duration of administration is extended only in special circumstances, such as gross contamination secondary to a ruptured viscus or severetrauma. The available data provide no evidence for the efficacy of extending coverage to 24 to 48 hours in such contexts.[23]

Although a single dose of antibiotic is acceptable, mechanical cleansing and adherence to guidelines for open management of wounds created more than 12 hours before treatment are the essential elements of prophylaxis.

Various studies have clearly demonstrated a reduction in the risk of infection by administering prophylactic antibiotics to patients undergoing hernia procedures, albeit reduction of an intrinsically low risk.[16,17,24,25] In general, prophylaxis is considered optional. For hernia repairs entailing the insertion of mesh, prophylaxis is considered desirable since the morbidity of infected mesh in the groin is substantial. However, no prospective trials demonstrate the effectiveness or necessity of this practice.

For practical reasons, antibiotic administration should be as easy as possible, and the use of a single dose is often recommended.[26,27]

Another rationale for pre-operative antibiotics is that present knowledge does not justify use of toxic antimicrobials in all patients.[28]

PATHOLOGY OF SURGICAL SITE INFECTIONS

For many years wounds have been classified into four categories according to the theoretical number of bacteria that contaminate wounds:

1. Clean,
2. Clean-contaminated,

3. Contaminated,
4. Dirty.

Wound infection rates in large series are approximately 1.5 to 3.9 percent for clean wounds, 3.0 to 4.0 percent for clean- contaminated wounds, and approximately 8.5 percent for contaminated wounds. Dirty wounds generally are left open, but wound infection rates for dirty wounds of 28 and 40 percent have been reported.

Wound infections encompass infections of the wound that occur above the fascia (superficial wound infection) and those that occur below the fascia (deep wound infection). Some authors have proposed more inclusive terms, e.g., "surgical field" or "surgical site infection," that would include all operative sites potentially exposed to bacteria. These more inclusive terms would include superficial and deep wound infections and infections that do not occur in direct proximity to the surgical incision (e.g., postoperative intraabdominal abscess).

Criteria for defining a surgical site infection (SSI)[29]

A: Incisional SSI:

I. Superficial:

Infection occurs within 30 days after the operation.

Infection involves only skin or subcutaneous tissue of the incision.

And at least one of the following:

1. Purulent drainage, with or without laboratory confirmation.
2. Organisms isolated from an aseptically obtained culture or fluid or tissue.
3. At least one of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat and superficial incision is deliberately opened by surgeon, unless incision is culture negative.

Do not report the following conditions as SSI:

1. Stitch abscess (minimal inflammation and discharge confined to the points of suture penetration).
2. Infected burn wound.
3. Incisional SSI that extends into the fascial and muscle layers (see deep incisional SSI).

II. Deep:

Infection within 30 days after the operation if no implant or within 1 year if implant is in place and infection is related to the operation.

Infection involves deep soft tissues (e.g. fascial and muscle layers) of the incision and at least one of the following:

1. Purulent drainage from the deep incision but not from the organ/space component of the surgical site.
2. A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (> 38°C), localized pain, or tenderness, if site is culture-ndgative.
3. Diagnosis of a deep incisional SSI by a surgeon or attending physician.

Note:

1. Report infection that involves both superficial and deep incision sites as deep incisional SSI.
2. Report an organ/space SSI that drains through the incision as a deep incisional SSI.

B. Organ/Space SSI:

Infection occurs within 30 days after the operation if no implant or within 1 year if implant is in place and the infection is related to the operation and Infection involving any part of the anatomy (e.g., organs or spaces) other than the incision, opened or manipulated during an operation and at least one of the following:

1. Purulent drainage from a drain that is placed through a stab wound into the organ/ space.
2. Organisms isolated from an aseptically-obtained culture of fluid or tissue in the organ/ space
3. An abscess or other evidence of infection involving the organ/ space that is found on direct examination, during reoperation, or by histopathologic or radiological examination.
4. Diagnosis of an organ/ space SSI by a surgeon or attending physician.

Determinants of Infection[30]

The development of surgical infection depends on several factors: (1) microbial pathogenicity and number, (2) host defenses, (3) the local environment, and (4) surgical technique (for postoperative infection).

Microbial Pathogenicity

Distribution of pathogens isolated' from pus Sir Ganga Ram Hospital (January 2000-June 2004)[29]

Pathogen	Percentage of Isolates
Eschcerichia coli	24
Staphylococcus aureus	23
Coagulase-negative *staphylococci*	17
Enterococcus spp.	10
Pseudomonas aeruginosa	9
Klebsiella pneumoniae	8
Enterobacter spp.	5
Candida spp.	4

Pathogens representating less than 2% of isolates are excluded.

The ability of a microbe to cause infection is a balance between host defenses and microbial pathogenicity. Some microbes that have virtually no ability to cause infection in the normal host can cause lethal infection in an individual with compromised host defenses.

Many bacteria (S. pneumoniae, Klebsiellapneumoniae, Streptococcus pyogenes, Staphylococcus aureus, Salmonellatyphi) and fungi (Histoplasmacapsulatum, Candida albicans, Cryptococcus neoformans) have thick capsules that make them resistant to phagocytosis. Other microbes (Mycobacterium tuberculosis, Aspergillusflavus, and Toxoplasma gondii) resist intracellular killing after they have been phagocytosed whenlysosomes that contain enzymes that digest microbes do not fuse with the phagosome. Other microbes successfully resist digestion by lysosomal enzymes.

Some bacteria can elaborate toxins, many of which are enzymes that injure or kill cells or promote spread within tissues. Exotoxins play an important role in the pathogenicity of Clostridium species, Staph, aureus, and Strep, pyogenes. Other bacteria (Clostridium tetani, Clostridium botulinum) elaborate neurotoxins that alter normal neural transmission.

Endotoxins are lipopolysaccharide-protein complexes that are normal constituents of the cell wall of gram-negative bacteria. These molecules activate many biological pathways, including the complement and coagulation systems, and cause release of cytokines and other biologic mediators from macrophages, release of hormones, and alteration in metabolism.

Host Defenses

Local host defenses are important in preventing microbial penetration into the tissues. Systemic host defenses are needed to rid the tissues of microbes once penetration has occurred.

Local Host Defenses

Tissues are protected from microbial invasion by a layer of epithelium. The epithelium of the skin is multilayered, and the superficial layers are keratinized. The epithelium also is multilayered in the nasopharynx, oral cavity, esophagus, and genitourinary tract. At other sites (the tracheobronchial tree,gastrointestinal tract, and eye)' a single layer of epithelium protects the underlying tissues. Each site also provides a local environment that is not conducive to microbial attachment and growth. Among these local environmental features may be lack of moisture (skin), the flushing action of tears and urine, cilia (trachea, bronchi), peristalsis, mucus, pH (gastrointestinal tract), and local immunity(IgA).

Systemic Host Defenses

A complex system of defense mechanisms exists throughout the body that can inactivate and kill microbial agents. These host defenses consist of phagocytic cells, the immune system, and other molecular cascades such as the complement system, the coagulation system, and the kinin system. Phagocytic cells that can ingest and kill microbes include polymorphonuclear leukocytes (PMNs) and tissue macrophages (monocytes in the blood). Through a complex set of interactions of microbes with complement and other activation molecules, PMNs adhere to vascular endothelium, migrate across the endothelium and move in the direction of the microbes (chemotaxis), attach to the microbes (which may involve immunoglobulins or other opsonins), and phagocytose the microbes. Finally, lysosomes containing a variety of enzymes fuse with the phagosome, and the microbe is rapidly digested. The initiation of this process and its attendant chemical, cellular, and physiologic changes result in inflammation.

Macrophages are phagocytic cells found throughout the body tissues: in liver (Kupffer cells), spleen, lymphoid tissue, lung (alveolar microphages), brain (glial cells), connective tissue(histiocytes), and pleura and peritoneum. Macrophages can also move toward microbes in response to chemotactic agents and phagocytose and kill them. In addition, macrophages are important in initiating the immune response and can elaborate cytokines, tissue necrosis factor, interferon, and other biologically active molecules. Humoral and cellular immunity are important systemic host defense mechanisms for many microbial agents. The complement system, clotting system, kinin system, leukotrienes, cytokines, and other biologically active molecules are also activated by microbial agents and play an important role in host defenses.

Host defenses are altered in malnourished individuals, trauma patients, postoperative patients, burn patients, patients with malignant neoplasms, and patients receiving drugs such as cancer chemotherapeutic agents, immunosuppressive agents to prevent transplant rejection, or steroids or other agents that have immunosuppressive effects.

Local Environmental Factors

Local factors may permit an infection to occur in a person with minimal microbial contamination and with otherwise adequate host defenses. These environmental factors inhibit systemic host defenses from being fully effective. A traumatic wound that normally would heal without infection has a greatly increased

likelihood of becoming infected if the trauma has resulted in devitalization of tissue or if foreign bodies have been deposited in the wound. Phagocytic cells do not function effectively in the presence of devitalized tissue or foreign bodies. A suture can reduce the number of Staph, aureus required to produce a subcutaneous infection by a factor of 100,000. Fluid collections and edema also increase the likelihood of infection because they inhibit phagocytosis.

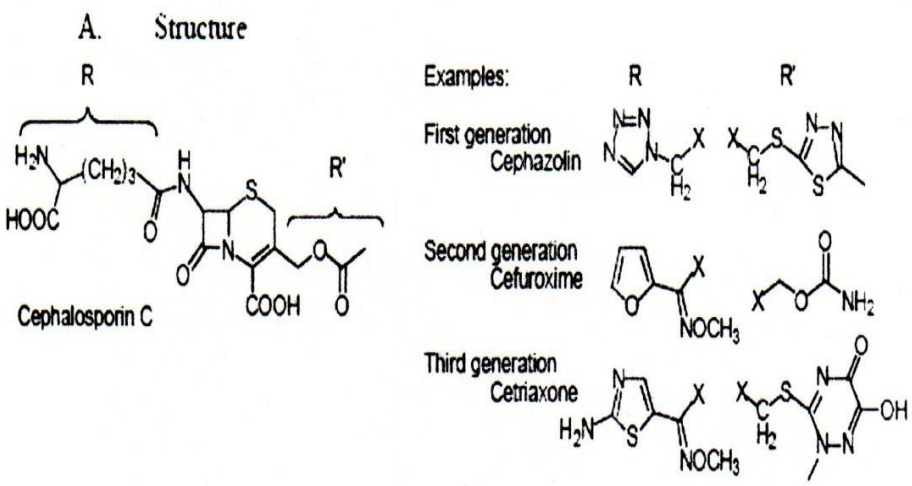
Peripheral vascular disease contributes to soft tissue infection by preventing blood “and the systemic host defenses that it contains (phagocytic cells, immune globulins, and other mediators) from reaching the site of microbial contamination. Shock also decreases the amount of blood that reaches these sites.

These environmental factors can prevent phagocytic cells from functioning efficiently by lowering tissue oxygen tension (PO 2). The lowered PO_2 inhibits function of phagocytic cells and promotes the growth of anaerobes.

Surgical Technique

Surgical technique is an important determinant of postoperative wound infection and other postoperative infections. Surgeons can decrease the likelihood of postoperative infection by handling tissues gently; removing devitalized tissues, blood, and other substances that promote the growth of microbes; using drains appropriately (and avoiding inappropriate use); avoiding excessive cautery; and not performing intestinal anastomoses under tension or when there is any question of inadequate blood supply.

CEFTRIAXONE



Ceftriaxone

A third generation cephalosporine, with a broad spectrum of activity and a longer'half time. Its pharmacokinetics and dynamics are favorable as a prophylactic antibiotic.

Pharmacokinetics of Ceftriaxone

The most important aspects of its pharmacokinetics include a long half-life, excellent tissue penetration and saturable (dose-dependent) serum protein binding of the drug.[31]

Absorption

Average plasma concentrations of Ceftriaxone following a single 30-minute intravenous (IV) infusion of 1 gm dose in healthy subjects was 53 mcg/ml and was achieved at 6 hrs. Ceftriaxone was rapidly absorbed after IM administration with mean peak times ranging from 1.3 to 1.9 hrs. Steady-state plasma concentrations

were apparent after the third dose of both dosage regimens, with trough plasma concentrations of 24±6 and 39±8 pg/ml (mean±SD) after the 0.5 and 1 g q12 h regimens, respectively.[32]

The area under the plasma concentration-time curve after intramuscular administration is equivalent to that after intravenous administration of an equivalent dose, indicating 100% bioavailability of IM Ceftriaxone.

Pharmacokinetic parameters for Ceftriaxone 1 g every 24 hours were as follows:[33]

- Volume of distribution 0.12 ± 0.02 L/kg,
- Half-life 7.5 ± 0.6 hours
- Protein binding 90-97%

Distribution

Following intravenous administration, Ceftriaxone diffuses rapidly into the interstitial fluid, sustaining bactericidal concentrations against susceptible organisms for 24 hours. Ceftriaxone concentrations well above the MICs of most pathogens are detectable for more than 24 hours in over 60 tissues or body fluids including lung, heart, biliary tract/liver, tonsil, middle ear and nasal mucosa, bone as well as cerebrospinal, pleural, prostatic and synovial fluids.

Protein binding

Ceftriaxone is reversiblv bound to albumin and the binding decreases with the increase in concentration. Protein binding is 90- 97%.[33]

Penetration into particular tissues

Ceftriaxone penetrates the inflamed meninges. It also crosses the placental barrier and is secreted in the breast milk at low concentrations.

Elimination

Total plasma clearance is 0.58 to 1.45 L/hour. Renal clearance is 0.32 to 0.73 L/hour.

Comparison of Ceftriaxone with Cefepime, Ceftazidime, Imipenem, Piperacillin/Tazobactam[34]

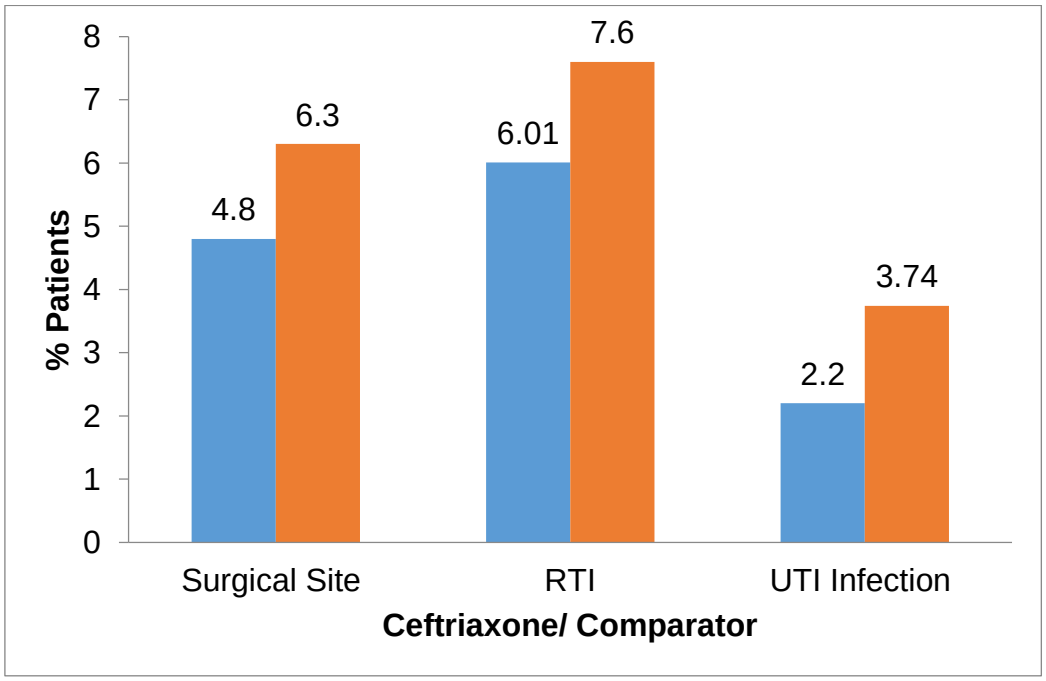
A contemporary collection of 12,295 isolates (2001-2002) consisting of Staphylococcus aureus, coagulase-negative Staphylococci, Streptococcus *pneumoniae,* (3-hemolytic Streptococci and viridans-group streptococci were tested against broad-spectrum R-lactams (Cefepime, Ceftazidime, Ceftriaxone, Imipenem, Piperacillin/ Tazobactam) and comparator agents to determine their continued effectiveness for empiric antimicrobial therapy. The findings confirm that the newer cephalosporins i.e. Ceftriaxone and Cefepime among broad-spectrum R-lactam agents have a spectrum of activity that remains comprehensive for the commonly isolated Gram-positive pathogens.

Ceftriaxone has a spectrum of activity that remains comprehensive for the commonly isolated Grampositive pathogens.

Ceftriaxone in surgical prophylaxis[35]

The objective of this, study was to investigate possible differences in prophylaxis with Ceftriaxone compared with other antimicrobial agents for surgical-site infections and remote infections such as RTIs and UTIs. Evaluations were performed on 48 studies, for a total of 17,565 patients. Overall, 406 patients (4.8%) in the Ceftriaxone group and 525 (6.3%) in the comparator group developed a surgical-site infection (log odds ratio [OR] -0.30 [CI -0.50 to -0.13]; $p < 0.0001$). RTIs were observed in 6.01% patients in the Ceftriaxone group and in 7.6% patients in the comparator group, (log OR -0.30 [CI -0.55 to -0.09]; $p = 0.0013$). UTIs were reported for 2.2% of the Ceftriaxone prophylaxis patients compared with 3.74% of the comparator group patients (log OR -0.54 (CI -1.18 to -0.16]; $p < 0.0001$). Overall, in clean surgery 5.1 % and 6.2% patients developed a surgical

site infection in the Ceftriaxone and comparator groups, respectively (log OR -0.22 [CI -0.51 to 0.01]; p = 0.0476). Ceftriaxone is statistically superior to other antibiotics in preventing both local and remote postoperative infections. RTIs were prevented for all but 1.57% of patients in the Ceftriaxone group and 2.62% of patients in the comparator group (p = 0.01) in clean surgery and for 9.54% of the Ceftriaxone group versus 11.6% of the comparator group (p = 0.01) in clean- contaminated surgery. While results observed in clean surgery did not show statistically significant superiority of Ceftriaxone in preventing UTI insurgence (log OR -0.21 [CI 0.0-0.65]; p = 0.7702), this was clearly shown in the clean-contaminated surgery. In fact, 4.47% of patients in the Ceftriaxone group versus 7.52% of patients in the comparator group developed a UTI (log OR -0.56 [CI -1.25 to -0.16]; p < 0.0001). Adverse events were observed in a similar proportion in the Ceftriaxone prophylaxis and the comparator groups (0.35% and 0.23%, respectively). Duration of prophylaxis did not influence outcome of infection. The metaanalysis showed that Ceftriaxone is statistically superior to other antibiotics in preventing both local and remote postoperative infections and respiratory infections.



Ceftriaxone in penicillin-allergic patients[36]

Recent analysis of clinical data and a clearer understanding of the role of chemical structure in the development of cross- reactivity indicate that the increased risk of an allergic reaction to certain cephalosporins in penicillin allergic patients is smaller than previously postulated. A significant increase in allergic reactions to Cephalothin, Cephaloridine, Cephalexin, Cefazolin and Cefamandole was observed in penicillin-allergic patients; no increase was observed with Cefprozil, Cefuroxime, Ceftazidime orCeftriaxone. Clinical challenges, skin testing and monoclonal antibody studies point to the paramount importance of similarities in side chain structure to predict crossallergy between cephalosporins and penicillins. First-generation cephalosporins have a modest cross-allergy with penicillins, but cross-allergy is negligible with 2nd- and 3rd-generation cephalosporins. Particular emphasis is placed on the role of chemical structure in determining the risk of cross reactivity between specific agents. No increase in allergic reactions was observed with Ceftriaxone in penicillin-allergic patients.

SULBACTAM

Sulbactam is a beta lactamase inhibitor. It does not have significant antibacterial activity, rather it binds and inactivates ß- lactamases (the family of enzymes hydrolyzes the cyclic amide bond of ß-lactam ring, which result in loss of bactericidal activity), thereby protecting antibiotics that are normally the substrates of these enzymes. Sulbactam is used with penicillins and other ß-lactam antibiotics to protect them from enzymatic inactivation.

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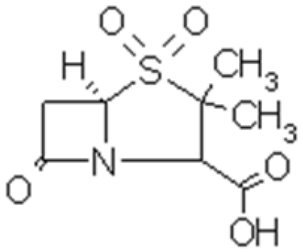
Category:

- 6 Anti-infective drugs
- 6.2 Antibiotics, systemic
- 6.2.1 Beta-lactam drugs
- 6.2.1.1 Penicillins

Primary Characteristics



Sulbactum Trihydrate, Sulbactum Trihydrateare the derivatives of Sulbactam Sulbactum Trihydrate, Sulbactum Trihydrate are the derivatives of Sulbactam It is of Synthetic origin and belongs to Penicillanic acid sulphone. It belongs to Antibacterial pharmacological group.The Molecular Weight of Sulbactam is 233.20.

Pharmacokinetics

Plasma protien binding is 38%. and metabolism is reported 15-25% via liver. Renal Excretion accounts for 30% and plasma half life is 1-2 hr.

Indications

Sulbactam is primarily indicated in conditions like Beta lactamase resistant pseudomonas, Gynecological infections, Intra-abdominal infections, Methicillin resistant staphylococci, Skin infections.

Contraindications

Sulbactam is contraindicated in conditions like Hypersensitivity.

Drug Interactions

No data regarding the interactions of Sulbactam was found.

Interference in Pathology

False +ve Test for Urinary Aminoacids

Side Effects

The severe or irreversible adverse effects of Sulbactam, which give rise to further complications include Exfoliative dermatitis, Bone marrow depression.

Sulbactam produces potentially life-threatening effects which include Anaphylaxis. which are responsible for the discontinuation of Sulbactam therapy.

The signs and symptoms that are produced after the acute overdosage of Sulbactam include Seizures.

The symptomatic adverse reactions produced by Sulbactam are more or less tolerable and if they become severe, they can be treated symptomatically, these include Headache, Fatigue, Abdominal pain, Dysuria, Malaise, Glossitis, Urinary RetentionX, Epistaxis, Urinary retention.

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Single Ingredient

Inj: 1 g, 500 mg,

Multi ingredient

Inj: 1 g, 1 gm, 0.5 gm, 100 mg, 125 mg, 250 mg, 375 mg, 500 mg, 750 mg, 3000 mg,
Inj-IV: 250 mg, 500 mg,
Susp: 125 mg/5ml, 250 mg/5ml,
Tabs: 125 mg, 250 mg, 500 mg,

Dosage

Sulbactam's dosage details are as follows:

Dose	Single Dose	Frequency	Route	Instructions
Adult Dosage				
250 mg	250 (250)	6 hourly	PO/IV/IM	In combination with Amoxycillin 250mg
500 mg	500 (500)	6 hourly	PO/IV/IM	In combination with Ampicillin 1g.
Paedriatic Dosage (20 Kg.)				
16.66 mg/kg	17 (16.66)	8 hourly	Intramuscular	For 5---14 days
16.66 mg/kg	17 (16.66)	8 hourly	Intravenous	For 5--14 days
1.25 to 2.5 mg/kg	1.9 (1.875)	12 hourly	Oral	
Neonatal Dosage (3 Kg.)				
25 mg/kg	25 (25)	12 hourly	Intavenous	For 5---14 days
25 mg/kg	25 (25)	12 hourly	Intramuscular	For 5----14 days

High Risk Groups

Drug should not be given to Paediatrics, Pregnant Mothers, and Neonates.

If prescribing authority justifies the benefits of the drug against the possible damages he/she should reevaluate them and consult the reference material and previous studies.

Warning / Precautions

Sulbactam should be used with caution in patients with illnesses or any allergy especially to penicillin or other antibiotics. In diabetic patients, this can affect the results of clinitest tablets. This may interfere with oral contraceptives, if using oral contraceptives discuss with doctor alternate birth control methods to use, while using this medication. It should be used only if clearly needed during pregnancy or lactation.

Storage Conditions

Inj
Store Below 40°C. Protect from Sunlight.

Bencini PI et al[37] in 1994 did a study on preoperative antibiotic prophylaxis in flexural surgery of difficult contamination-prone areas of the skin. Total of 527 patients were surgically treated for skin neoplasms. The four prophylactic programmes, to which the patients were randomly allocated were as follows: A) No prophylaxis. B) Intramuscular cephazolin, 1 gm every 12 hour beginning 48 hour prior to surgery and continuing for 48 hour after surgery. C) Intramuscular cephazolin, 1 gm every 12 hour beginning 2 hour before surgery and continuing 24 hour after surgery. D)Intramuscular cephazolin, 1 gm single dose 2 hour before surgery.The rate of postoperative infections in group 'A' was 12%, group 'B' 4.6%, group 'C' 0.77%, group 'D' 2.96%. The study confirmed the utility of antibiotic prophylaxis in prevention of postoperative infections and demonstrates that brief regimens are superior to more prolonged regimens. In particular, a single dose antibiotic schedule significantly reduces the infection rate, is cheaper, and is better tolerated by the patients.

Chalkiadakis GE et al[38] in 1995 studied the pharmacokinetics of preincisional injection of 2 gm ceftriaxone in 20 patients who have undergone abdominal surgery, with determination of serum, wound tissue, and wound fluid antibiotic concentrations. Plasma concentrations exceeded the minimal inhibitory concentrations of most aerobic gram-positive and gramnegative organisms with the exception of Pseudomonas aeruginosa, Acinetobacter species, and Streptococcus faecalis for 24 hours . No local or general complications arose in any of the patients. The authors concluded that that preincisional administration of ceftriaxone for prophylaxis is very effective.

AgrawalM et al[39] in 1997 did a study on different single dose antibiotics that were used preoperatively and the post-operative infection if present were compared with different drugs. Agents used were cefotaxime , ceftriaxone , ceftizoxime and cefoperazone. Patients in whom ceftriaxone was used had lower rate of wound infection as compared to that with cefotaxime , ceftizoxime and cefoperazone . Thus, for clean and clean contaminated major elective surgeries the incidence of wound infection can be decreased by the judicious use of proper prophylactic antibiotics to well below 2 percent.

Yousuf M and Hussain M conducted a study on 100 patients by two surgeons and at various sites in the city, with variable operating theatre facilities to avoid the chance of inducing bias as much as possible. In this study 4 out of 8 patients developed surgical site infection and required extended antibiotic treatment. Thus Single dose prophylactic antibiotic therapy was satisfactory in our surgical environment. The authors summarised that this practice would be efficient, cost effective and prevent the emergence of nosocomial infections.[40]

Sanabria A et al[41] in 2007 in a meta-analysis assessed the effectiveness of antibiotic prophylaxis in mesh hernioplasty. Meta-analysis was intended to measure the benefits of antibiotic prophylaxis on surgical site infection rate in adult patients scheduled for mesh inguinal hernioplasty. Six randomized clinical trials were found. Quality was assessed using Cochrane Collaboration criteria. A total of 2507 patients were analyzed. Surgical site infection frequency was 1.38% in the antibiotic group versus 2.89% in the control group (odds ratio = 0.48; 95% confidence interval, 0.27–0.85). There was no statistical heterogeneity. Sensitivity analysis by quality did not show differences in overall results. The authors in the present meta-analysis observed that Antibiotic prophylaxis use in patients submitted to mesh inguinal hernioplasty decreased the rate of surgical site infection by almost 50%.

Surahio AR et al[42] in 2010 did a prospective study to determine the effectiveness of preoperative antibiotic prophylaxis in reduction of postoperative wound infection in clean and clean contaminated procedures and compared the cost of antibiotic prophylaxis in both groups. The authors summarised that Infection is a great problem in surgery and is encountered by all surgeons by nature of their craft; they invariably impair the first line of host defence. Bacteria may enter the wound during or after the operation and may be of endogenous or exogenous origin. Total 400 patients were divided into 2 groups of 200 patients each: Group-A received single dose antibiotic prophylaxis, and Group-B received 3 doses of antibiotic therapy. Only clean and clean contaminated procedures were included and results were compared. In Group A, clean procedures (Group-A1) were 110, and clean contaminated (Group-A2) were 90 patients. In clean procedure, rate of infection was 5 out of 110 (4.54%) and in clean contaminated procedures it was 3 out of 90 (3.33%). In Group B, in clean procedures (Group-B1), rate of infection was 7 out of 90 (7.77%), while in clean contaminated procedures (Group-B2) it was 9 out of 110 (8.18%) patients. Over all wound infection rate after single dose antibiotic prophylaxis was 4% in both procedures and 8% after 3- dose antibiotic therapyThe

authors concluded that Single dose antibiotic prophylaxis is as effective as 3-dose therapy in clean and clean contaminated procedures to prevent wound infection and is cost-effective

Saskia-Javi Y et al[43]in 2013 in aopen label randomized clinical trialdid a study to determine the necessity of prophylactic antibiotics in the hope of setting new procedural standards in elective hernia procedures thus reducing cost and bacteria resistance andaimed to determine incidence differences of post operative infection in patients who underwent tension-free hernioplasty and received prophylactic antibiotics compared to those who received placebo. From 54 subjects 3 (5.6%) of them were found to have a slight erythema around the operation wound, on the 7th,14th,21th, and 28th day no signs of erythema were found. From the three subjects two (7.4%) were from the placebo group and one (3.7%) from the antibiotic group. All clinical assessment of post operative wound was made using Southampton Wound Assessment Scale, where erythema is a grade 1C, all subjects healed primarily. The authors concluded that an Open Label Randomized Clinical Trial comparing SSI in post tension-free hernioplasty patients who were given prophylactic antibiotics and placebo. No significant difference were found.

Razack A et al[44] in 2015 did a prospective, double blind randomized Trial and assessed the value of single-dose, intravenous, prophylactic antibiotic in the prevention of wound infections during tension free inguinal hernia mesh repair. The overall infection rate was 8.3% (15 out of 180). The incidence of wound infection in antibiotic group was 7.4% and 9.3% in control group. There was no statistically significant difference in the infection rates between the two groups. The authors in the present study observed that aAntibiotics showed a protective effect in preventing SSI after mesh inguinal hernia repair. However significant values cannot be obtained and cost effectiveness of antibiotic prophylaxis needs further evaluation. Therefore routine use is not recommended.

Sganga G, Tascini C, Sozio E et al[45] in 2016 conducted a systematic review of the literature on SSIs, especially MRSA infections, and used the Delphi method to identify risk factors for these resistant infections and focused on the prophylaxis, epidemiology and therapy of methicillin-resistant Staphylococcus aureus surgical site infections and a positioned paper on associated risk factors. Risk factors associated with MRSA SSIs identified by the Delphi method were: patients from long-term care facilities, recent hospitalization (within the preceding 30 days), Charlson score > 5 points, chronic obstructive pulmonary disease and thoracic surgery, antibiotic therapy with beta-lactams (especially cephalosporins and carbapenem) and/or quinolones in the preceding 30 days, age 75 years or older, current duration of hospitalization >16 days, and surgery with prothesis implantation. Protective factors were adequate antibiotic prophylaxis, laparoscopic surgery and the presence of an active, in-hospital surveillance program for the control of infections. MRSA therapy, especially with agents that enable the patient's rapid discharge from hospital is described.

The authors concluded that the prevention, identification and treatment of SSIs, especially those caused by MRSA, should be implemented in surgical units in order to improve clinical and economic outcomes.

MATERIALS AND METHODS

This interventional, quasi-experimental study was conducted in general surgery department of KIMS, Karad from Oct 2014 to July 2016.

Sampling technique was non probability convenience. Data was collected on a prescribed proforma. Sample size was 100 patients, equally divided into two groups A and B. Patients of either age and gender undergoing mesh hernioplasty surgery and available for complete follow-up were included in the study. Patients with any generalized debilitating disease, diabetes mellitus, any infective focus in the body, poor quality of the skin at the incision site, allergy to cephalosporin and history of use of antibiotics within last 7 days were excluded from the study. The protocol was approved by hospital ethical Committee and written informed consent was obtained from all the patients. Ceftriaxone 1gm, with sulbactum a third a generation cephalosporin, was selected for antibiotic prophylaxis because of its broad spectrum, long half life and low toxicity. Patients in group A were given only single dose of 1 gram Ceftriaxone + sulbactum 500mg intravenously within 30 minutes of the initial operative incision. This group did not receive postoperative antibiotics and were followed up with regular sterile dressings.

Patients in group B were given routine postoperative antibiotics according to usual established protocol in the hospital and were followed up with regular sterile dressings.

Each patient was evaluated by an observer unaware of the treatment used. Evaluation was done using the prescribed proforma for postoperative fever developing or persisting 48 hours after the surgery, discharge from the wound and overlying skin inflammation. Wound was examined on 3rd, 8th, 15th and 28th postoperative day. Bacteriological examination was done to confirm the diagnosis if there was any sign or symptom of infection. Wound infection was noted to be whether superficial or deep. It was managed according to culture and sensitivity report. Results were expressed as mean+ standard deviation for continuous variables (e.g. age, duration of stay in hospital and operation time) and number (percentage) for categorical data (e.g. gender, surgical outcome etc). Results were tested by Chi-square test. A p-value of <0.05 was considered as statistically significant. Calculations were done.

STATISTICAL ANALYSIS

Quantitative data is presented with the help of Mean and Standard deviation. Comparison among the study groups is done with the help of unpaired t test as per results of normality test. Qualitative data is presented with the help of frequency and percentage table. Association among the study groups is assessed with the help of Fisher test and student 't' test. 'p' value less than 0.05 is taken as significant**.**

Pearson's chi-squared test

$$\mathrm{X}^2 = \sum_{i=1}^{n} \frac{(O_i - E_i)^2}{E_i}$$

Where,
X2 = Pearson's cumulative test statistic.
Oi = an observed frequency;
Ei = an expected frequency, asserted by the null hypothesis;
n = the number of cells in the table.

Results were graphically represented where deemed necessary.

Appropriate statistical software, including but not restricted to MS Excel, SPSS ver. 20 will be used for statistical analysis. Graphical representation will be done in MS Excel 2010.

Sample size calculation:

Considering a confidence level of 95% and confidence interval of 10 the number of patients in our study to achieve statistical significance is 96. This was calculated by Survey System (http://www.surveysystem.com/sscalc.htm # one). The Survey System ignores the population size when it is "large" or unknown. Population size is only likely to be a factor when you work with a relatively small and known group of people (e.g., the members of an association). Hence a sample size of 100 was considered adequate for our study.

OBSERVATIONS AND RESULTS

Distribution of patients according to Age

Majority of the patients (30%) in Case Group were from the age group of 21-30 years followed by 24% from the age groups of 41-50, 18% from the age group of >60 years & 14% from the age group of 31-40 and 51-60 years. The mean age in Case Group was 44.48 ± 15.72 years.

Majority of the patients (28%) in Control Group were from the age group of 21-30 years followed by 26% from the age group of 41-50, 16% from the age group of 51-60 years and >60 years and 14% from the age group of 31-40 years. The mean age in Control Group was 43.86 ± 14.73 years. The mean age of the patients

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between two groups were comparable and statistically not significant (p>0.05). The student 't' test was applied for the statistical analysis.

Table 1: Distribution of patients according to Age

Age (yrs)	Case Group		Control Group	
	N	%	N	%
21-30	15	30%	14	28%
31-40	7	14%	7	14%
41-50	12	24%	13	26%
51-60	7	14%	8	16%
>60	9	18%	8	16%
Total	50	100%	50	100%
Mean age	44.48 ± 15.72		43.86 ± 14.73	

Std. Error of Diff	95% CI		df	t	p Value
	Lower	Upper			
3.05	-5.43	6.67	98	0.20	p>0.05

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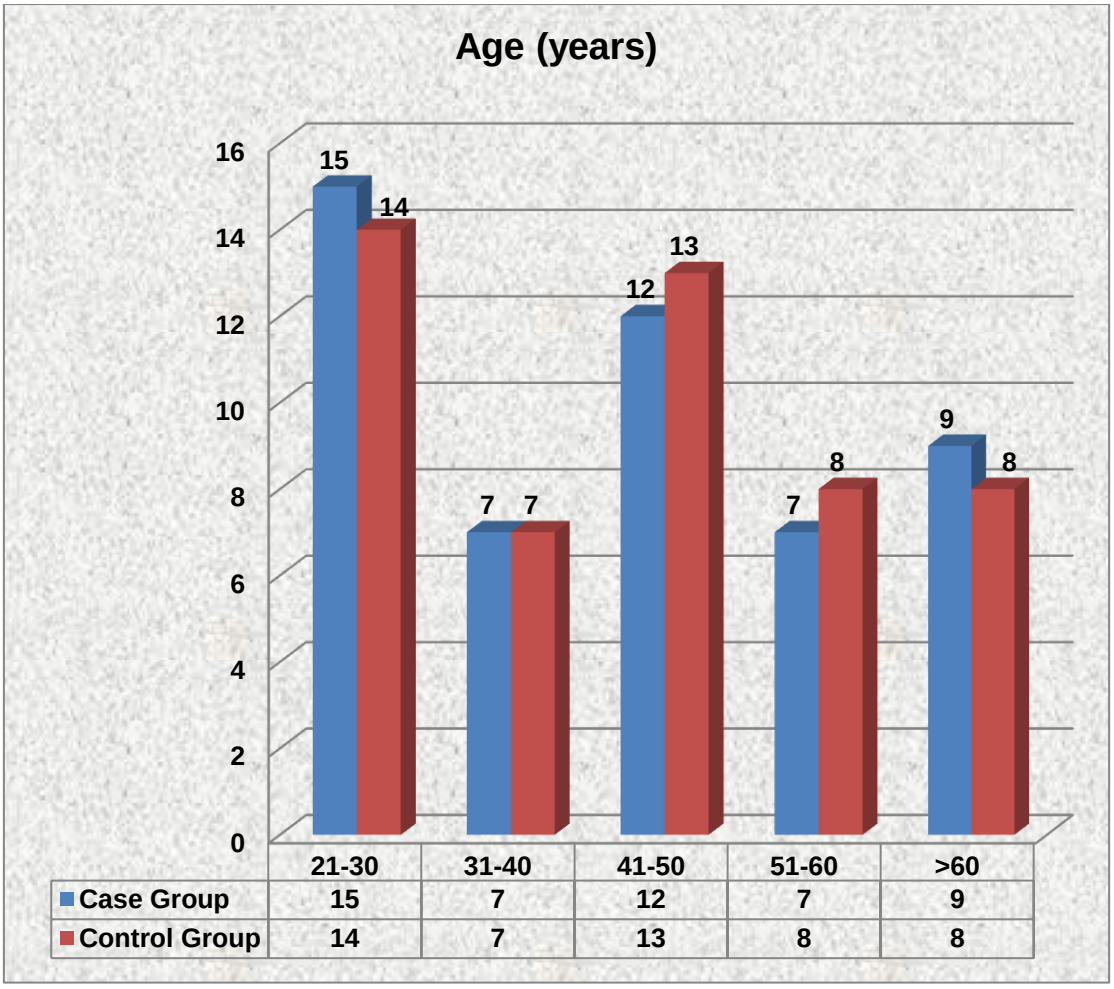

Distribution of patients according to Weight

34% patients in Case Group were from the weight group of 41-50 kgs followed by 32% from the weight group of 51-60 kgs, 30% from the weight group of 61-70 kgs and 4% from the age group of >70 kgs. The mean weight in Case Group was 55.08 ± 7.92kgs.

42% patients in Control Group were from the weight group of 41-50 kgs followed by 32% from the weight group of 51-60 kgs, 16% from the weight group of 61-70 and 10% from the age group of 31-40 kgs. The mean weight in Control Group was 52.02 ± 9.13kgs.

The mean weight of the patients between two groups were comparable and statistically not significant ($p>0.05$). The Student's t-test was applied for the statistical analysis.

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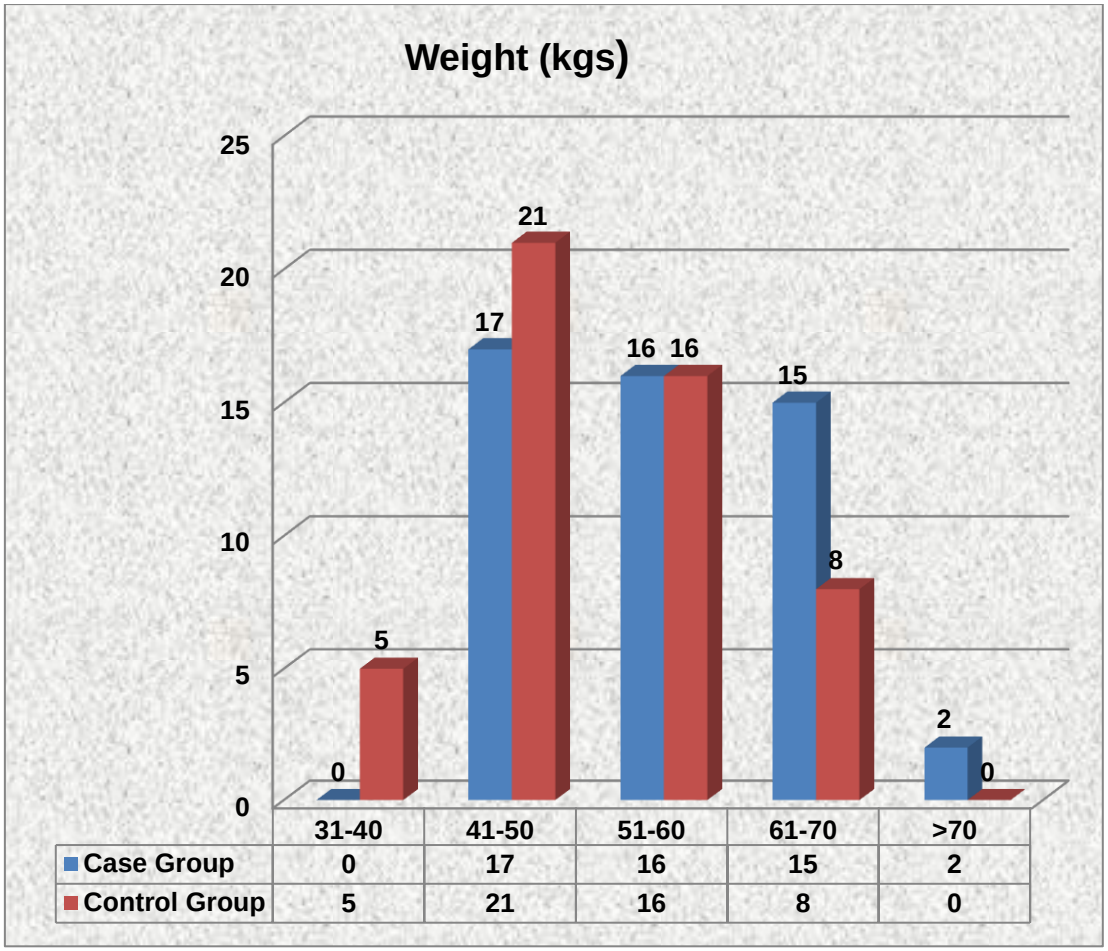

Table 2: Distribution of patients according to Weight

Weight (kgs)	Case Group		Control Group	
	N	%	N	%
31-40	0	-	5	10%
41-50	17	34%	21	42%
51-60	16	32%	16	32%
61-70	15	30%	8	16%
>70	2	4%	0	-
Total	50	100%	50	100%
Mean Weight	55.08 ± 7.92		52.02 ± 9.13	

Std. Error of Diff	95% CI		df	t	p Value
	Lower	Upper			
1.71	-0.33	6.45	98	1.79	p>0.05

Distribution of patients according to ASA Grading

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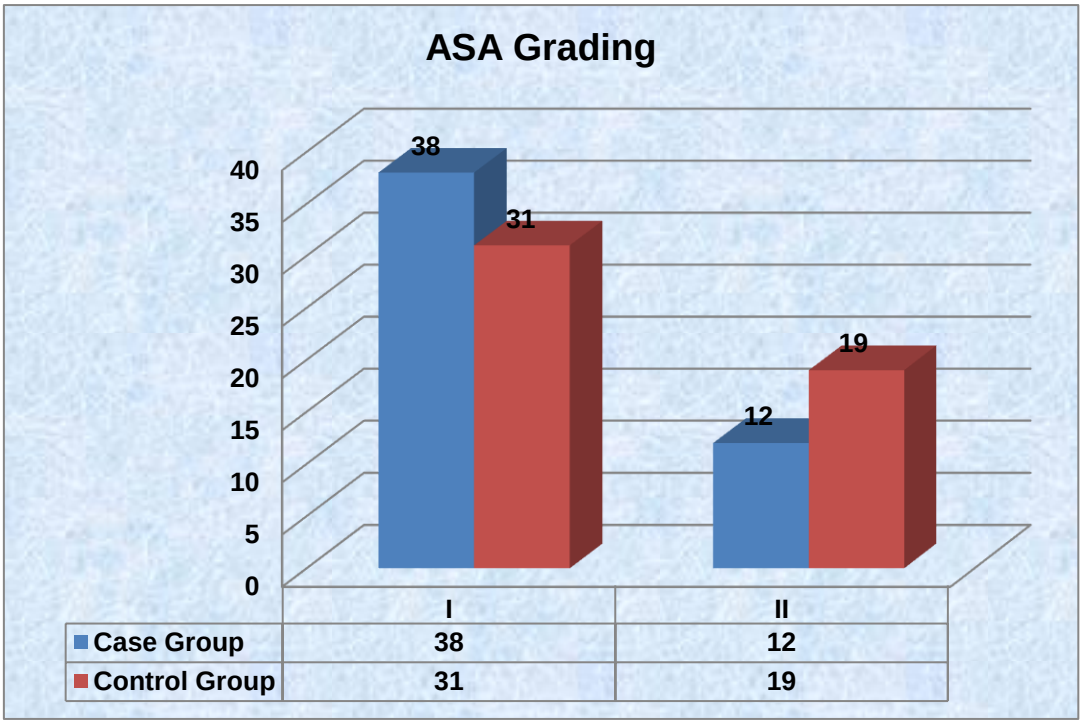
Case Group had 38 patients (76%) with Class I grading and 12 patients (24%) with Class II grading, whereas Control Group had 31 patients (62%) with Class I grading and 19 patients (38%) with Class II grading. The ASA Grading of the patients between two groups were comparable and statistically not significant (p>0.05). The Fisher test was applied for the statistical analysis.

Table 3: Distribution of patients according to ASA Grading

ASA Grading	Case Group		Control Group	
	N	%	N	%
I	38	76%	31	62%
II	12	24%	19	38%
Total	50	100%	50	100%
p Value	p>0.05 (Not Significant)			



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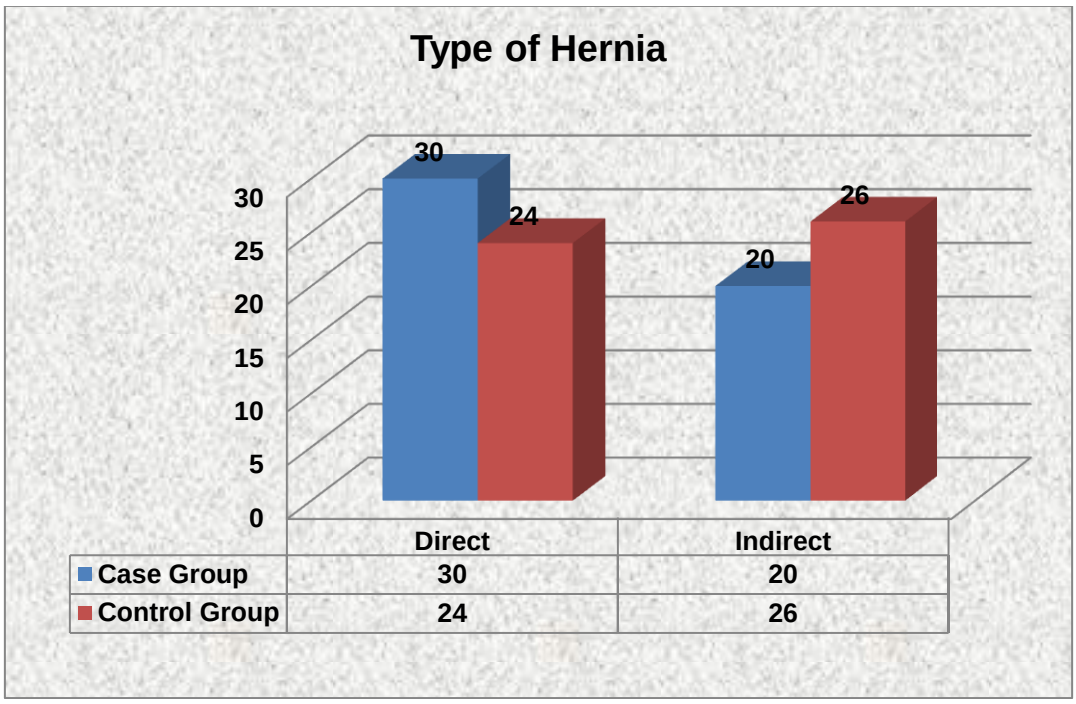
Distribution of patients according to Type of Hernia

Case Group had 30 patients (60%) diagnosed with Direct hernia and 20 patients (20%) diagnosed with Indirect hernia. Control Group had 24 patients (48%) diagnosed with Direct hernia and 26 patients (52%) diagnosed with Indirect hernia. The Type of Hernia of the patients between two groups were comparable and statistically not significant (p>0.05). The Fisher test was applied for the statistical analysis.

Table 4: Distribution of patients according to Type of Hernia

Type of Hernia	Case Group		Control Group	
	N	%	N	%
Direct	30	60%	24	48%
Indirect	20	40%	26	52%
Total	50	100%	50	100%
p Value	p>0.05 (Not Significant)			



Distribution of patients according to Location of Hernia

Hernia was located in the right side in 24 (48%) patients of the Case Group whereas 26 (52%) patients had the hernia located in the left side. Control Group had 32 patients (64%) with the hernia in the right side and 18 (36%) patients with the hernia located in the left side. The Location of Hernia of the patients between two groups were comparable and statistically not significant (p>0.05). The Fisher test was applied for the statistical analysis.

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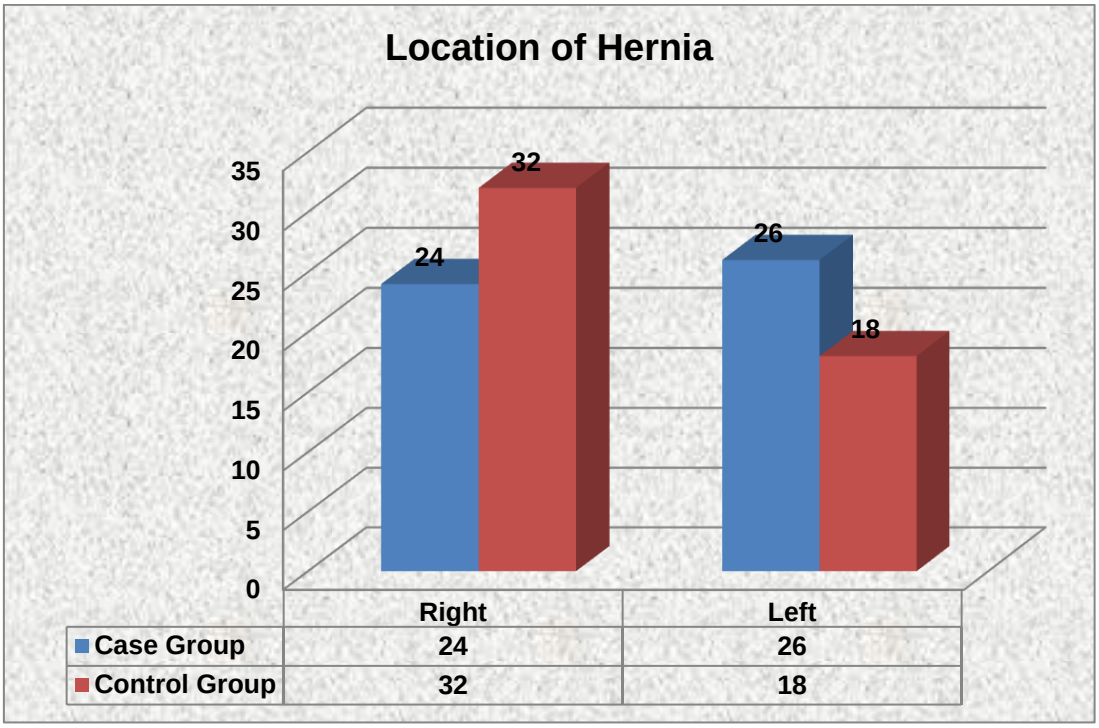
Table 5: Distribution of patients according to Location of Hernia

Location of Hernia	Case Group		Control Group	
	N	%	N	%
Right	24	48%	32	64%
Left	26	52%	18	36%
Total	50	100%	50	100%
p Value	p>0.05 (Not Significant)			



Mean Hemoglobin levels of both groups

Mean Hemoglobin levels in case and control group was 11.56±1.54 and 11.89±1.35 respectively. Mean Hemoglobin levels of both groups were comparable and statistically not significant (p>0.05). The Student t-test was applied for the statistical analysis.

Table 6: Mean Hemoglobin levels of both groups

	Case Group		Control Group	
	Mean	SD	Mean	SD
Mean Hb (gm%)	11.56	1.54	11.89	1.35

Std. Error of Diff	95% CI		df	t	p Value
	Lower	Upper			
0.29	-0.90	0.24	98	1.14	p>0.05

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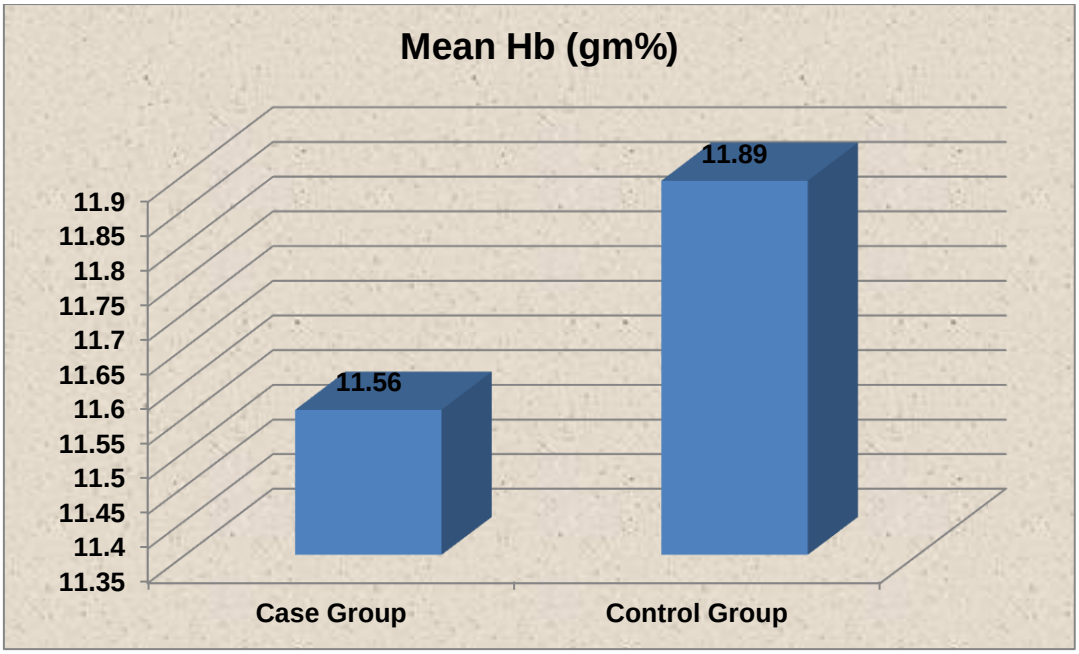

Mean Operative Time of both groups

Mean operative time in case and control group was 39.4±24.4 and 45.2±20.65 respectively. Mean operative time of both groups were comparable and statistically not significant (p>0.05). The Student t-test was applied for the statistical analysis.

Table 7: Mean Operative Time of both groups

	Case Group		Control Group	
	Mean	SD	Mean	SD
Mean Operative Time (mins)	39.40	24.40	45.2	20.65

Std. Error of Diff	95% CI		df	t	p Value
	Lower	Upper			
4.52	-14.77	3.17	98	1.28	p>0.05

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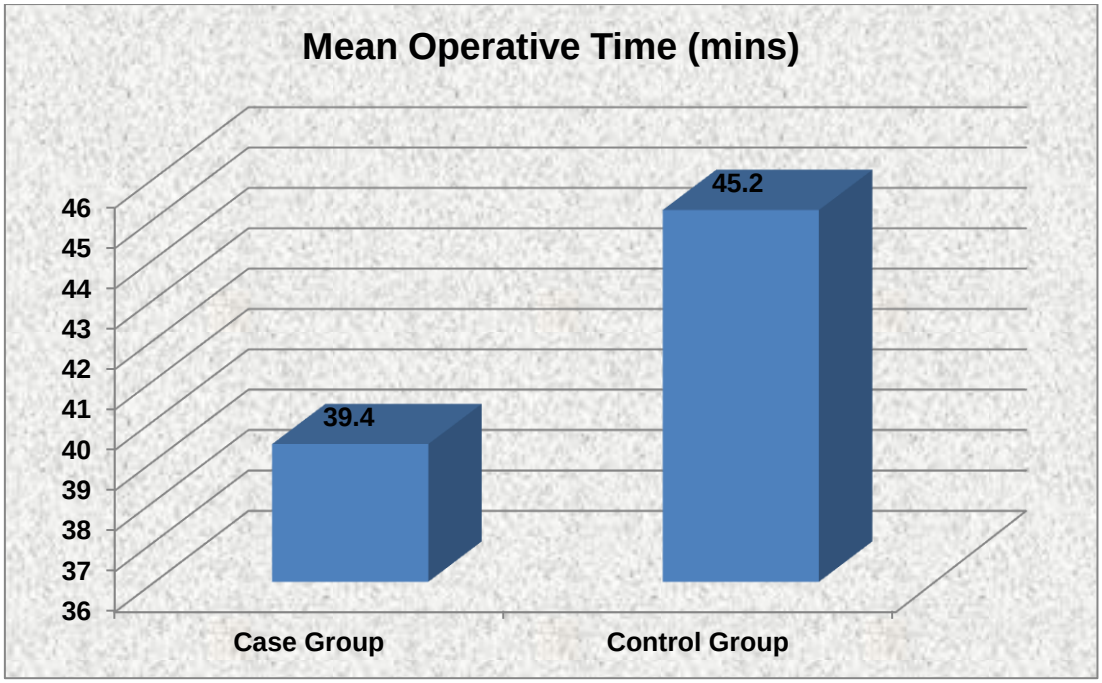
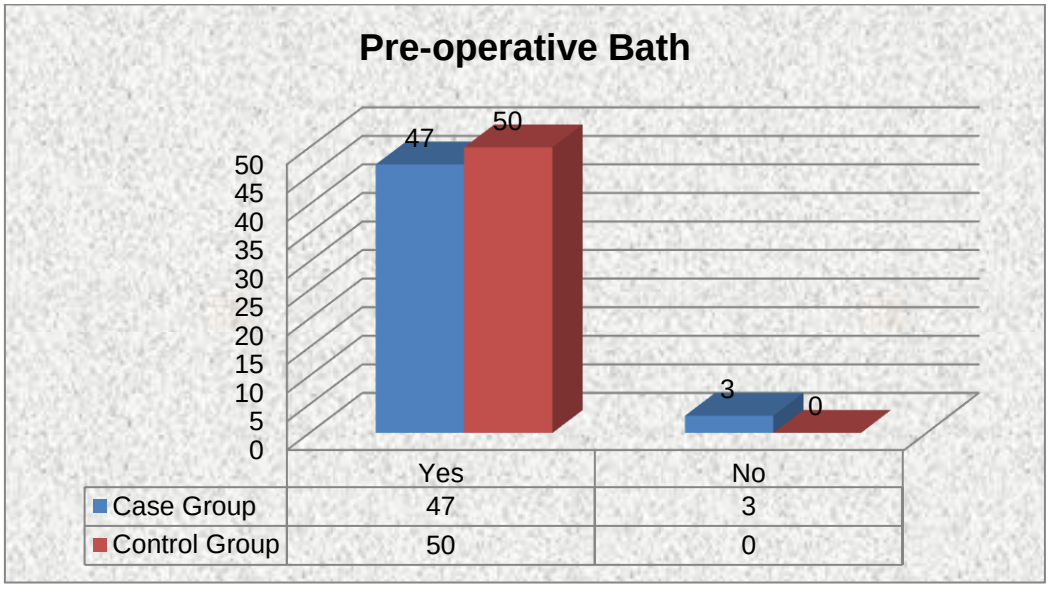


Pre-operative Bath in both groups

Preoperative bath was taken by 94% and 100% patients in the cases and control groups. The pre-operative bath in both groups were comparable and statistically not significant (p>0.05). The Fisher's was applied for the statistical analysis.

Table 8: Pre-operative Bath in both groups

Pre-operative Bath	Case Group		Control Group	
	N	%	N	5
Yes	47	94%	50	100%
No	3	6%	0	-
Total	50	100%	50	100%



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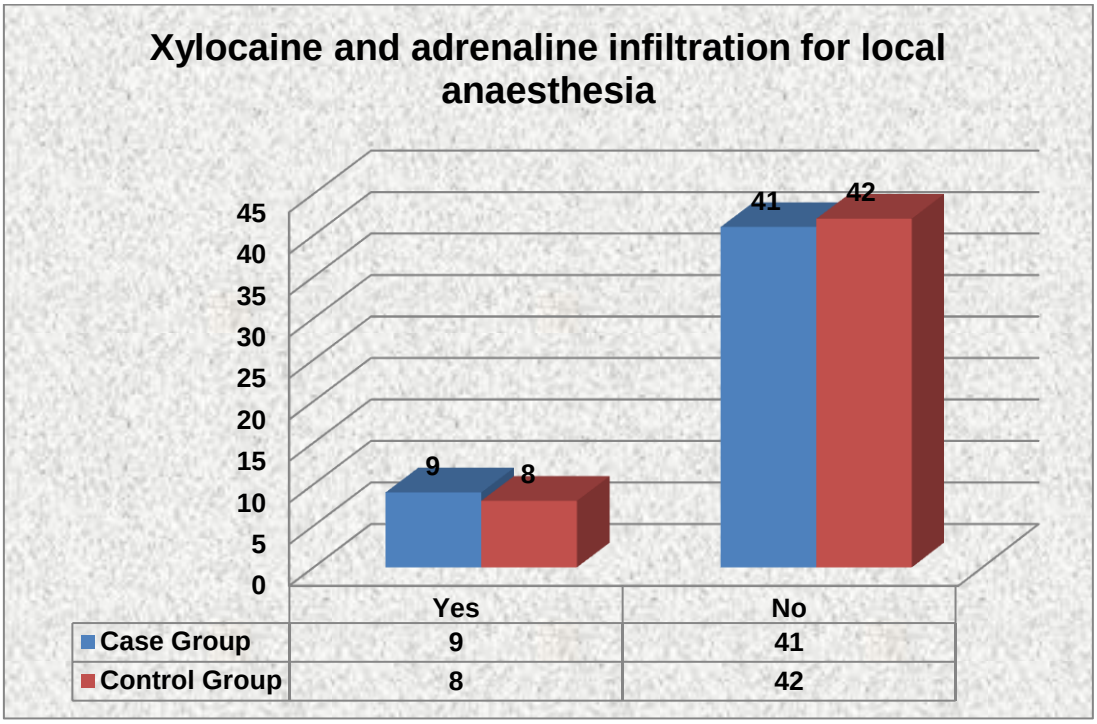
Xylocaine and adrenaline infiltration for local anaesthesia in both groups

Xylocaine and adrenaline infiltration for local anaesthesia was used in 9 (18%) and 8 (16%) patients in case and control groups respectively. The difference was statistically not significant (p>0.05). The Fisher's was applied for the statistical analysis.

Table 9: Xylocaine and adrenaline infiltration for local anaesthesia in both groups

Xylocaine and adrenaline infiltration for local anaesthesia	Case Group		Control Group	
	N	%	N	5
Yes	9	18%	8	16%
No	41	82%	42	84%
Total	50	100%	50	100%



Intra-Operative use of electrocautery in both groups

Electrocautery was used in 26 (52%) and 36 (72%) patients in case and control groups respectively. The difference was statistically not significant (p>0.05). The Fisher's was applied for the statistical analysis.

Table 9: Intra-Operative use of electrocautery in both groups

Intra-Operative use of electrocautery	Case Group		Control Group	
	N	%	N	5
Yes	26	52%	36	72%
No	24	48%	14	28%
Total	50	100%	50	100%

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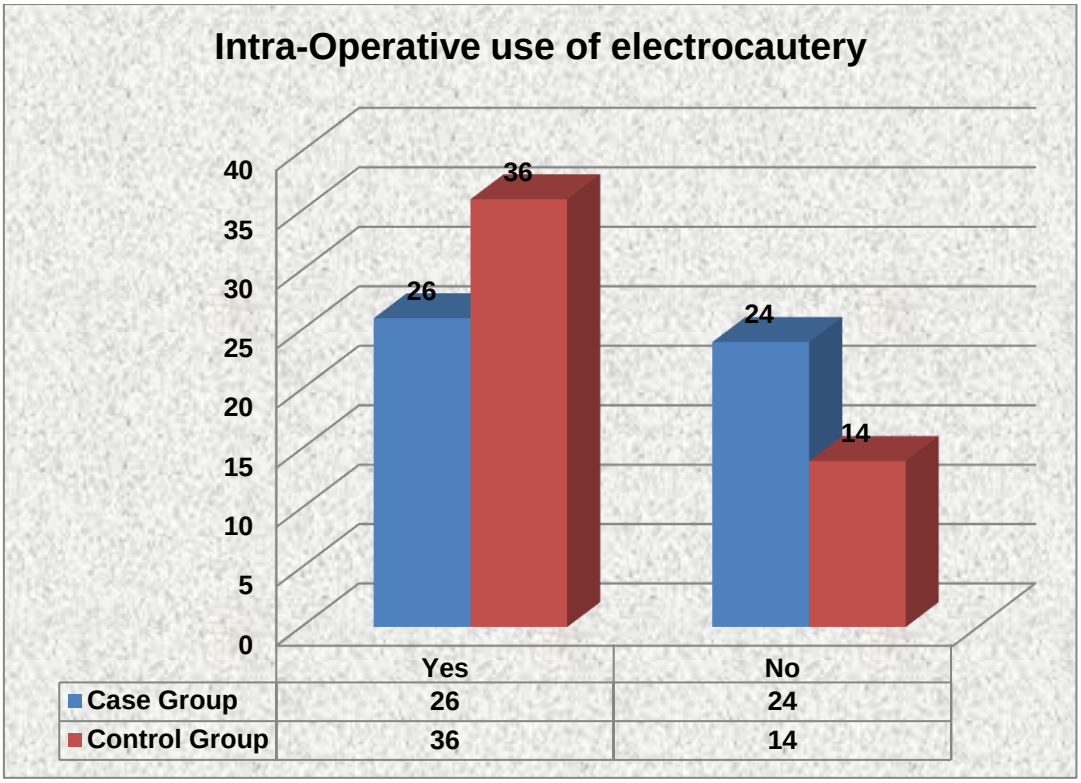

Surgical Site infection in both groups

Surgical Site infection was present in 3 (6%) and 4 (8%) patients in case and control groups respectively. The difference was statistically not significant (p>0.05). The Fisher's was applied for the statistical analysis.

Table 9: Surgical Site infection in both groups

Surgical Site infection	Case Group		Control Group	
	N	%	N	5
Yes	3	6%	4	8%
No	47	94%	46	92%
Total	50	100%	50	100%

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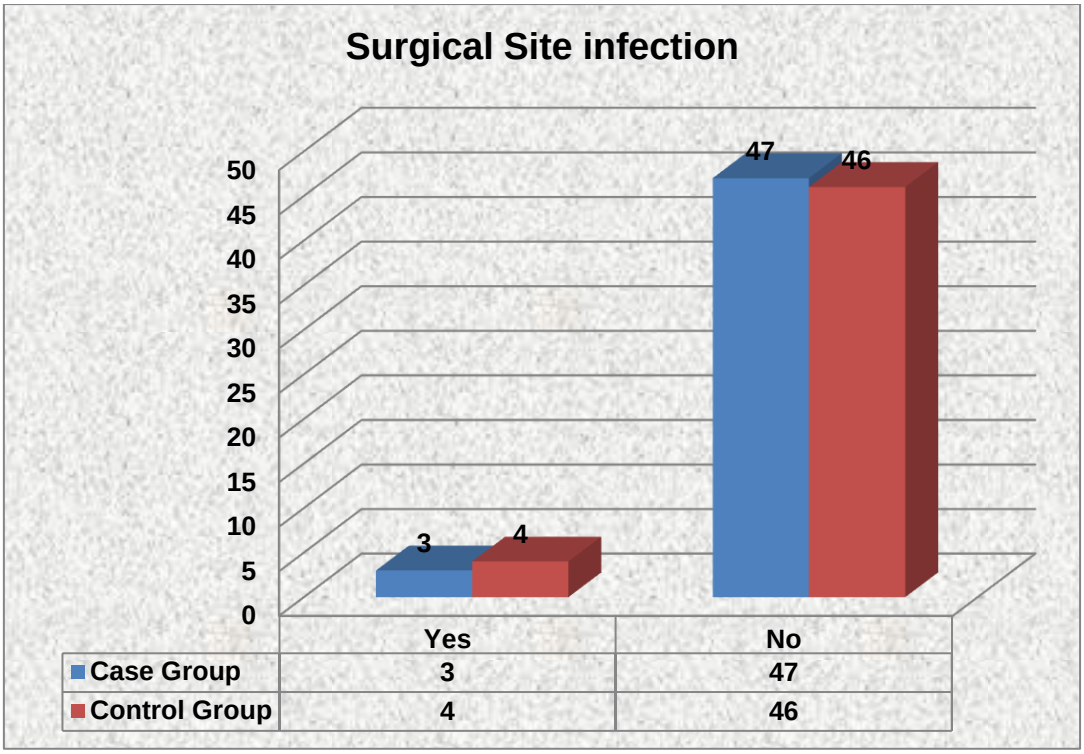

DISCUSSION

Hernia repair is considered as one of the so-called 'clean' operations which may not require antibiotic coverage. Many surgeons, however, continue to give antibiotics empirically, as prophylaxis. This practice was more widely used after the establishment of the tension-free mesh repair technique as the method of choice for hernia repair, because of the fear of infection of the introduced foreign body. It has been shown that administration of prophylactic antibiotics may inhibit the adherence of bacteria to the prosthesis and subsequently their growth rates[11]. Surgical site infections (SSIs) remain a major clinical problem in terms of morbidity, mortality, time spent in hospital and overall direct and indirect costs[1,2,3]. Despite progress in their prevention, SSIs remain one of the most common adverse events in hospitals, accounting for 11% to 26 % of all healthcare-associated infections[4].

This study was done to evaluate incidence of surgical site infection in Open Mesh Hernioplasty using Prophylactic Antibiotic. The prophylactic antibiotic chosen was Ceftriaxone 1g with Sulbactum 500 mg in 20ml considering its broad spectrum, long t1/2 and good post antibiotic effect. Woodfield JC et al[46] had found ceftriaxone to be a versatile choice in antibiotic prophylaxis. They had underlined its particular effectiveness against Staphylococcus Aureus, long half time and no active metabolites. AgrawalM et al[39] did a study on different single dose antibiotics that were used preoperatively and the post-operative infection if present were compared with different drugs. Agents used were cefotaxime, ceftriaxone, ceftizoxime and cefoperazone. Patients in whom ceftriaxone was used had lower rate of wound infection as compared to that with cefotaxime ,ceftizoxime and cefoperazone.

100 patients were divided into two groups as Case and Control groups. Most of the patients in Case and Control Group (30% and 28% respectively) were from the age group of 21-30 years. The mean age in Case and Control Group was 44.48 ± 15.72 and 43.86 ± 14.73 years respectively and was comparable. The mean weight and ASA grading of the patients between two groups were also comparable and statistically not significant ($p>0.05$).

Case Group had 30 patients (60%) diagnosed with Direct hernia and 20 patients (20%) diagnosed with Indirect hernia. Control Group had 24 patients (48%) diagnosed with Direct hernia and 26 patients (52%) diagnosed with Indirect hernia. Also hernia was located in the right side in 24 (48%) patients of the Case Group whereas 26 (52%) patients had the hernia located in the left side. Control Group had 32 patients (64%) with the hernia in the right side and 18 (36%) patients with the hernia located in the left side.

The two groups were compared on the basis of age, weight, ASA grading. Also other factors which directly or indirectly affect the occurrence of surgical site infections were also considered to minimize and eliminate various confounding factors that would affect the results of the trial. The two groups were found comparable on the basis of vital parameters and the inference was incurred after p value that was calculated for each parameter was found to be more than 0.05.

Groups were also compared on the basis of haemoglobin levels and the difference in the two groups was found to be statistically insignificant. It is well accepted that the operative time required does predispose the surgical incision to infection. Reyet al[31] have reported that the development of surgical site infection was significantly associated with the duration of surgery. Increase in operative time not only increases the risk of infection but also increases the need for repeating the dose of prophylactic antibiotic. For longer procedures, re-administration of the drug is indicated at intervals of one or two times the half-life of the drug (using the same dose).[16,47]

In our study the difference in the operative time in both the groups was statistically insignificant and the mean operative time in both the groups did not require use of repeat dose of ceftriaxone and Sulbactum. In conclusion single dose of pre-operative Ceftriaxone is sufficient to cover the incision till the end of surgery with respect to operative time.

The patients were administered I.V. injection Ceftriaxone 1g with Sulbactum 500 mg in 20ml 30 mins before surgery for effective results. This is in agreement to the study of effective as Chalkiadakis GE et al[38].

Chalkiadakis GE et al[38] studied the pharmacokinetics of preincisional injection of 2 gm ceftriaxone in 20 patients who have undergone abdominal surgery, with determination of serum, wound tissue, and wound fluid antibiotic concentrations. Plasma concentrations exceeded the minimal inhibitory concentrations of most aerobic gram-positive and gramnegative organisms with the exception of Pseudomonas aeruginosa, Acinetobacter species, and Streptococcus faecalis for 24 hours. No local or general complications arose in any of the patients. The authors concluded that that preincisional administration of ceftriaxone for prophylaxis is very effective.

The primary cause of infection of surgical wound is the endogenous bacteria harboured by the patients on their skin[48].These bacteria are a potential source of infection and thus practice of pre-operative bath, shaving and pre-operative cleansing have a profound effect on the infection rates in various surgeries. Pre-operative bath was taken by majority of patients in both the case and control groups and the difference was statistically insignificant. Use of xylocaine with adrenaline infiltration at the incision site does cause a transient local ischemia and hence creates a local environment that adversely affects healing. Thus this can be a confounding factor in such studies. But in our study, the two groups were comparable in this aspect.

Intra-operative use of electrocautery creates local ischemic environment and also increases the seroma formation. Both these factors increase the risk for surgical site infection. This factor was specially considered in our study and it was found that both the groups were comparable with respect to use of electrocautery.

In present study, surgical site infection in Case group was 6% and that in Control group was 8%. The infection rate was similar to the studies of Surahio AR et al[42], Razack A et al[44] and Saskia-Javi Y et al[43].

Surahio AR et al[42] did a prospective study to determine the effectiveness of preoperative antibiotic prophylaxis in reduction of postoperative wound infection in clean and clean contaminated procedures and compared the cost of antibiotic prophylaxis in both groups. The authors summarised that Infection is a great problem in surgery and is encountered by all surgeons by nature of their craft; they invariably impair the first line of host defence. Bacteria may enter the wound during or after the operation and may be of endogenous or exogenous origin. Total 400 patients were divided into 2 groups of 200 patients each: Group-A received single dose antibiotic prophylaxis, and Group-B received 3 doses of antibiotic therapy. Only clean and clean contaminated procedures were included and results were compared.

In Group A, clean procedures (Group-A1) were 110, and clean contaminated (Group-A2) were 90 patients. In clean procedure, rate of infection was 5 out of 110 (4.54%) and in clean contaminated procedures

it was 3 out of 90 (3.33%). In Group B, in clean procedures (Group-B1), rate of infection was 7 out of 90 (7.77%), while in clean contaminated procedures (Group-B2) it was 9 out of 110 (8.18%) patients. Over all wound infection rate after single dose antibiotic prophylaxis was 4% in both procedures and 8% after 3- dose antibiotic therapy. The authors concluded that Single dose antibiotic prophylaxis is as effective as 3-dose therapy in clean and clean contaminated procedures to prevent wound infection and is cost-effective.

Razack A et al[44] in 2015 did a prospective, double blind randomized Trial and assessed the value of single-dose, intravenous, prophylactic antibiotic in the prevention of wound infections during tension free inguinal hernia mesh repair. The overall infection rate was 8.3% (15 out of 180). The incidence of wound infection in antibiotic group was 7.4% and 9.3% in control group. There was no statistically significant difference in the infection rates between the two groups. The authors in the present study observed that Antibiotics showed a protective effect in preventing SSI after mesh inguinal hernia repair. However significant values cannot be obtained and cost effectiveness of antibiotic prophylaxis needs further evaluation.

Saskia-Javi Y et al[43] in 2013 in a open label randomized clinical trial did a study to determine the necessity of prophylactic antibiotics in the hope of setting new procedural standards in elective hernia procedures thus reducing cost and bacteria resistance and aimed to determine incidence differences of post-operative infection in patients who underwent tension-free hernioplasty and received prophylactic antibiotics compared to those who received placebo. From 54 subjects 3 (5.6%) of them were found to have a slight erythema around the operation wound, on the 7th, 14th, 21th and 28th day no signs of erythema were found. From the three subjects two (7.4%) were from the placebo group and one (3.7%) from the antibiotic group.

All clinical assessment of post-operative wound was made using Southampton Wound Assessment Scale, where erythema is a grade 1C, all subjects healed primarily. The authors concluded that an Open Label Randomized Clinical Trial comparing SSI in post tension-free hernioplasty patients who were given prophylactic antibiotics and placebo. No significant difference was found.

Sganga G, Tascini C, Sozio E et al[45] conducted a systematic review of the literature on SSIs, especially MRSA infections, and used the Delphi method to identify risk factors for these resistant infections and focused on the prophylaxis, epidemiology and therapy of methicillin-resistant Staphylococcus aureus surgical site infections and a positioned paper on associated risk factors.

Risk factors associated with MRSA SSIs identified by the Delphi method were: patients from long-term care facilities, recent hospitalization (within the preceding 30 days), Charlson score > 5 points, chronic obstructive pulmonary disease and thoracic surgery, antibiotic therapy with beta-lactams (especially cephalosporins and carbapenem) and/or quinolones in the preceding 30 days, age 75 years or older, current duration of hospitalization >16 days, and surgery with prothesis implantation.

Protective factors were adequate antibiotic prophylaxis, laparoscopic surgery and the presence of an active, in-hospital surveillance program for the control of infections. MRSA therapy, especially with agents that enable the patient's rapid discharge from hospital is described. The authors concluded that the prevention, identification and treatment of SSIs, especially those caused by MRSA, should be implemented in surgical units in order to improve clinical and economic outcomes.

The study could not establish a significant difference in outcome between the case and control group. The results in both the groups as assessed by incidence of surgical site infection were equivocal. This is in line with the observations of Cochrane meta- analysis[49] which stated that antibiotic prophylaxis (mesh hernioplasty surgeries) cannot be firmly recommended or discarded. But it also reflected that the restriction of antibiotic usage to preoperative period and omission of antibiotics in post-operative period did not amount to increase the incidence of surgical site infections especially in clean general surgeries.

SUMMARY

This study was done to evaluate incidence of surgical site infection in Open Mesh Hernioplasty using Prophylactic Antibiotic. Based on the observations, the following conclusions were drawn:

1. Majority of the patients in Case and Control Group (30% and 28% respectively) were from the age group of 21-30 years. Age distribution of both the groups was similar.
2. The mean weight and ASA grading of the patients between two groups were also comparable and statistically not significant.
3. Case Group had 30 patients (60%) diagnosed with Direct hernia and 20 patients (20%) diagnosed with Indirect hernia. Control Group had 24 patients (48%) diagnosed with Direct hernia and 26 patients (52%) diagnosed with Indirect hernia.
4. Hernia was located in the right side in 24 (48%) patients of the Case Group whereas 26 (52%) patients had the hernia located in the left side. Control Group had 32 patients (64%) with the hernia in the right side and 18 (36%) patients with the hernia located in the left side.
5. The difference in mean hemoglobin levels was insignificant.
6. Operative time required for procedures in both the groups was comparable.
7. Preoperative bath was taken in majority of patients in Case and Control group.
8. Use of xylocaine and adrenaline and electrocautery did not confound the results.
9. Surgical Site infection was present in 6% and 8% patients in case and control groups respectively
10. The advantage of pre-antibiotic usage could be established over post-antibiotic usage in this study as the results states that infection rate in case group is less as compared to control group though p-value in this study was insignificant.

CONCLUSION

Hernia repair is considered as one of the so-called 'clean' operations which may not require antibiotic coverage. Many surgeons, however, continue to give antibiotics empirically, as prophylaxis. It has been shown that administration of prophylactic antibiotics may inhibit the adherence of bacteria to the prosthesis and subsequently their growth rates.

This study was done to evaluate incidence of surgical site infection in Open Mesh Hernioplasty using single dose of Ceftriaxone 1g with Sulbactum 500 mg. The advantage of pre-antibiotic usage could be established over post-antibiotic usage in this study as the results states that infection rate in case group who received single dose of Ceftriaxone 1g with Sulbactum 500 mg was less in comparison to the patients who received routine post-operative antibiotics though p-value in this study was insignificant. Clinical results seem to justify the use of single dose antibiotic prophylaxis to patients undergoing open mesh hernioplasty.

PROFORMA

CASE NO. REGNO:

NAME:

AGE: SEX:

ADDRESS:

DOA: DOO: DOD:

Socioeconomic status

CLINICAL history
Past History
Personal History

1. H/O alcohol intake
    a. Amount of Alcohol Intake
    b. Duration of Alcohol Intake
2. H/O smoking
    a. No. of cigarettes or bidis per day
    b. Duration of smoking

3. H/O of tobacco chewing

4. H/O chronic drug intake
    a. Name of Drug
    b. Dose of Drug
    c. Duration of Drug intake
    d. Drug Allergy

General examination
Body weight:

- Height:
- BMI:
- Pallor:

Investigations

- Hb (gm%)
- TLC
- Sr. Creatinine (mg%)
- BSL (mg%)
- BUL (mg%)
- Chest X-ray

Culture and sensitivity report of wound swab

Clinical diagnosis:

Final diagnosis:

Group in which included:

Presence of other risk factors:

- Diabetes
- Corticosteroid use Obesity
- Malnutrition
- Massive transfusion
- Multiple (3 or more) preoperative comorbid medical diagnoses
- ASA class 3, 4 or 5

Operative procedure:

ASA score:

Operative time (min)

Preoperative bath

Timing of preoperative shaving:
Any preoperative local cleansing done:
Prophylactic antibiotic administered YES / NO
Timing of administration (min before surgery)
Name and dose of the antibiotic administered
Length of the incision

Injection with epinephrine YES / NO
Electrocautery used YES / NO
Wound drains kept YES / NO
Previous irradiation of site done YES / NO
Post-operative antibiotic used YES / NO
Antibiotic regimen used

Name of antibiotic	Dose	Days	Cost

	Infection present	Infection absent	Type of SSI
Chek dressing (day3)			
8th Day:			
15th Day:			
28th Day:			

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MASTER CHART

CASE GROUP																							
Sr.No	Name	Age	Sex	DOA	DOD	General Examination		Investigation		Cilinical Diagnosis	Final Diagnosis	Operative procedure	ASA Score	Operative Time	Preoperative Bath	Preoperative shaving	Injection xylo + adr	Electroautery	Infections				type of SSI
						wight	Hight	HB	C & S Report										3rd	8th	15th	28th	
1	BGB	70	M	14/03/2015	19/03/2015	65	165	11	NA	RDIH	RDIH	HERNIOPLASTY	1	60	Y	NIGHT BEFORE	N	YES	N	N	N	N	
2	KDB	45	M	27/01/2015	31/01/2015	45	100	10.5	NA	RIIH	RIIH	HERNIOPLASTY	2	20	Y	N	N	N	N	N	N	N	
3	JRB	62	M	08/01/2015	11/01/2015	50	152	12	NA	RDIH	RDIH	HERNIOPLASTY	2	60	Y	NIGHT BEFORE	N	N	N	N	N	N	
4	ANS	43	M	08/01/2015	12/01/2015	42	175	12	NA	LDIH	LDIH	HERNIOPLASTY	1	15	Y	N	N	N	N	N	N	N	
5	YVK	48	M	22/10/2014	08/11/2014	45	190	12	NA	LIIH	LIIH	HERNIOPLASTY	1	30	Y	N	N	N	N	N	N	N	
6	TRM	62	M	22/09/2014	08/10/2014	50	154	9	NA	LDIH	LDIH	HERNIOPLASTY	2	100	Y	NIGHT BEFORE	N	YES	N	N	N	N	
7	YMA	28	M	28/10/2014	02/11/2014	56	152	9	NA	RIIH	RIIH	HERNIOPLASTY	1	15	Y	N	YES	N	N	N	N	N	
8	MRM	50	M	02/02/2015	16/02/2015	50	150	12	NA	RIIH	RIIH	HERNIOPLASTY	2	30	Y	EARLY MORNING	N	YES	N	N	N	N	
9	KDB	28	M	27/01/2015	30/01/2015	50	150	9.8	NA	LDIH	LDIH	HERNIOPLASTY	1	35	Y	NIGHT BEFORE	N	YES	N	N	N	N	
10	MDR	60	M	20/10/2014	26/10/2014	60	180	12.5	NO ORG	LIIH	LIIH	HERNIOPLASTY	1	90	Y	EARLY MORNING	N	YES	N	SUP	N	N	TYPE I
11	NRS	24	M	28/11/2014	03/12/2014	65	170	14.5	NA	RDIH	RDIH	HERNIOPLASTY	1	60	Y	EARLY MORNING	N	YES	N	N	N	N	
12	KPR	26	M	14/11/2014	17/11/2014	55	160	11	NA	LDIH	LDIH	HERNIOPLASTY	1	20	Y	NONE	YES	N	N	N	N	N	
13	SKK	22	M	14/03/2015	19/03/2015	45	145	12	NA	LIIH	LIIH	HERNIOPLASTY	1	20	Y	YES	YES	N	N	N	N	N	
14	KNK	32	M	14/03/2015	18/03/2015	65	160	10	N	LDIH	LDIH	HERNIOPLASTY	1	15	N	N	N	N	N	N	N	N	

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15	PYJ	21	M	18/01/2015	19/01/2015	65	165	11	N	LDIH	LDIH	HERNIOPLASTY	1	15	Y	JUST BEFORE	YES	N	N	N	N	N	
16	SPV	40	M	03/02/2015	08/02/2015	64	168	10	N	RIIH	RIIH	HERNIOPLASTY	1	20	N	N	N	N	PUS	N	N	N	TYPE I
17	DSK	28	M	24/06/2015	27/06/2015	55	165	11	N	RIIH	RIIH	HERNIOPLASTY	1	15	N	N	YES	N	N	N	N	N	
18	SRB	60	M	14/11/2014	19/11/2014	58	165	10	N	RDIH	RDIH	HERNIOPLASTY	2	20	Y	N	N	N	N	N	N	N	
19	PRV	45	M	28/10/2014	03/11/2014	45	160	9	N	LDIH	LDIH	HERNIOPLASTY	1	20	Y	N	N	N	N	N	N	N	
20	DNT	50	M	17/11/2014	23/11/2014	55	165	10	N	LIIH	LIIH	HERNIOPLASTY	1	15	Y	N	N	N	N	N	N	N	
21	SGH	40	M	08/06/2015	09/06/2015	55	170	11	N	LDIH	LDIH	HERNIOPLASTY	1	15	Y	N	N	N	N	N	N	N	
22	MMH	21	M	15/06/2015	16/06/2015	65	150	11	N	LDIH	LDIH	HERNIOPLASTY	1	15	Y	N	N	N	N	N	N	N	
23	BSA	28	M	20/05/2015	22/05/2015	75	175	11	N	LIIH	LIIH	HERNIOPLASTY	1	15	Y	YES	N	N	N	N	N	N	
24	MAA	44	M	11/04/2015	12/04/2015	55	160	11	N	LDIH	LDIH	HERNIOPLASTY	1	20	Y	N	N	N	N	N	N	N	
25	GAB	62	M	24/06/2015	29/06/2015	62	170	11	N	RDIH	RDIH	HERNIOPLASTY	1	45	Y	NIGHT BEFORE	N	Y	N	N	N	N	
26	PVM	50	M	08/06/2015	16/06/2015	47	150	14	N	RDIH	RDIH	HERNIOPLASTY	1	50	Y	NIGHT BEFORE	Y	Y	N	N	N	N	
27	MGB	75	M	22/09/2014	28/09/2015	65	170	12.5	N	RIIH	RIIH	HERNIOPLASTY	2	75	Y	NIGHT BEFORE	N	Y	N	N	N	N	
28	MHP	28	M	27/01/2015	04/02/2015	68	180	15	NO ORG	RDIH	RDIH	HERNIOPLASTY	1	70	Y	NIGHT BEFORE	Y	Y	N	N	N	N	
29	JKS	38	M	14/03/2015	22/03/2015	47	160	14	N	LDIH	LDIH	HERNIOPLASTY	1	60	Y	NIGHT BEFORE	N	Y	N	N	N	N	
30	JSJ	58	M	19/06/2015	26/06/2015	58	150	14	N	LIIH	LIIH	HERNIOPLASTY	1	35	Y	EARLY MORNING	N	Y	N	N	N	N	
31	JSH	24	M	25/06/2015	12/07/2015	65	170	14.5	N	LDIH	LDIH	HERNIOPLASTY	1	60	Y	EARLY MORNING	N	Y	N	N	N	N	
32	KBH	66	M	18/10/2014	08/11/2014	47	155	11	N	LIIH	LIIH	HERNIOPLASTY	2	60	Y	EARLY MORNING	N	Y	N	N	N	N	
33	TSS	46	M	22/09/2014	24/09/2014	54	170	12	N	LDIH	LDIH	HERNIOPLASTY	1	25	Y	N	N	N	N	N	N	N	
34	PBA	42	M	22/10/2014	24/10/2014	67	166	10.5	N	RIIH	RIIH	HERNIOPLASTY	1	15	Y	N	N	N	N	N	N	N	

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35	GSS	40	M	28/11/2014	02/12/2014	42	165	9.6	N	RIIH	RIIH	HERNIOPLASTY	2	25	Y	EARLY MORNING	N	Y	N	N	N	N	
36	NHV	56	M	02/12/2014	18/12/2014	45	154	11	N	RDIH	RDIH	HERNIOPLASTY	1	45	Y	EARLY MORNING	N	Y	N	N	N	N	
37	MML	55	M	04/02/2015	11/02/2015	56	154	10.5	N	RDIH	RDIH	HERNIOPLASTY	2	60	Y	NIGHT BEFORE	N	Y	N	N	N	N	
38	TAB	40	M	15/03/2015	22/03/2015	48	162	12.5	N	RDIH	RDIH	HERNIOPLASTY	1	20	Y	NIGHT BEFORE	N	N	N	N	N	N	
39	JBK	28	M	24/06/2015	10/07/2015	46	152	10.5	N	LDIH	LDIH	HERNIOPLASTY	2	30	Y	NIGHT BEFORE	Y	Y	N	N	N	N	
40	GAM	29	M	06/04/2015	13/04/2015	54	152	10.5	N	LIIH	LIIH	HERNIOPLASTY	1	75	Y	NIGHT BEFORE	N	Y	N	N	N	N	
41	CAK	29	M	18/05/2015	22/05/2015	60	180	12.5	NO ORG	LDIH	LDIH	HERNIOPLASTY	1	90	Y	EARLY MORNING	N	YES	N	SUP	N	N	TYPE I
42	SBP	75	M	17/06/2015	23/06/2015	65	170	12.5	N	RIIH	RIIH	HERNIOPLASTY	2	75	Y	NIGHT BEFORE	N	Y	N	N	N	N	
43	RDR	43	M	29/04/2015	01/05/2015	62	175	12	NA	RIIH	RIIH	HERNIOPLASTY	1	15	Y	N	N	N	N	N	N	N	
44	JNA	70	M	10/04/2015	17/04/2015	65	165	11	NA	RDIH	RDIH	HERNIOPLASTY	1	60	Y	NIGHT BEFORE	N	Y	N	N	N	N	
45	PSA	40	M	10/07/2015	14/07/2015	56	175	12	NA	RDIH	RDIH	HERNIOPLASTY	1	65	Y	NIGHT BEFORE	N	YES	N	N	N	N	
46	SOA	70	M	05/07/2015	12/07/2015	55	155	10	N	RDIH	RDIH	HERNIOPLASTY	2	35	Y	NIGHT BEFORE	YES	Y	N	N	N	N	
47	KMS	58	M	14/03/2015	21/07/2015	42	150	14	N	LDIH	LDIH	HERNIOPLASTY	1	35	Y	EARLY MORNING	N	Y	N	N	N	N	
48	SKH	48	M	18/01/2015	20/01/2015	75	170	12	NA	LDIH	LDIH	HERNIOPLASTY	1	30	Y	N	N	N	N	N	N	N	
49	SYA	53	M	08/01/2015	10/01/2015	52	175	12	NA	LIIH	LIIH	HERNIOPLASTY	1	15	Y	N	N	N	N	N	N	N	
50	SPS	24	M	01/11/2014	17/11/2014	65	170	14.5	N	RIIH	RIIH	HERNIOPLASTY	1	60	Y	EARLY MORNING	N	Y	N	N	N	N	

CONTROL GROUP																		
Sr.No	Name	Age	Sex	DOA	DOD	General Examination	Investigation	Clinical Diagnosis	Final Diagnosis	Operative procedure	ASA Score	Operative Time	Preoperative shaving	Xylo + Electrod	Operative Antibio	Days	Infcetions	type of SSI

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						wight	Hight	HB	C & S Report													3rd	8th	15th	28th	
1	TRN	51	M	22/09/2014	28/09/2014	65	182	11	NA	LIIH	LIIH	HERNIOPLASTY	1	115	Y	EARLY MORNING	N	Y	Y	7	N	N	N	N	
2	KHT	54	M	14/03/2015	18/03/2015	30	145	11.5	NA	RIIH	RIIH	HERNIOPLASTY	2	35	Y	NONE	N	Y	Y	5	N	N	N	N	
3	KAB	57	M	10/03/2015	15/03/2015	35	147	10.5	NA	RDIH	RDIH	HERNIOPLASTY	1	35	Y	NONE	N	Y	Y	7	N	N	N	N	
4	TBM	45	M	02/08/2015	10/08/2015	46	140	12.8	NA	LDIH	LDIH	HERNIOPLASTY	1	35	Y	NONE	N	N	Y	4	PUS	N	N	N	TYPEI
5	DHB	49	M	18/04/2015	23/04/2015	40	135	10	NOORG	RIIH	RIIH	HERNIOPLASTY	1	25	Y	NONE	N	Y	Y	5	N	N	N	N	
6	CRR	60	M	08/05/2015	15/05/2015	54	166	12	NA	LIIH	LIIH	HERNIOPLASTY	2	55	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
7	PCV	69	M	08/01/2015	15/01/2015	55	170	10.7	NOORG	RIIH	RIIH	HERNIOPLASTY	1	40	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
8	GAM	59	M	10/01/2015	16/01/2015	50	165	12.5	NOORG	RDIH	RDIH	HERNIOPLASTY	1	35	Y	NIGHT BEFORE	N	Y	Y	5	FEVER	N	N	N	TYPE I
9	CNK	40	M	14/03/2015	18/03/2015	56	175	12	N	RDIH	RDIH	HERNIOPLASTY	1	65	Y	NIGHT BEFORE	N	Y	Y	4	N	N	N	N	
10	SBP	30	M	16/03/2015	22/03/2015	65	175	11	N	LDIH	LDIH	HERNIOPLASTY	2	60	Y	EARLY MORNING	N	Y	Y	5	N	N	N	N	
11	BDR	26	M	18/11/2014	25/11/2014	65	180	13.5	NOORG	RIIH	RIIH	HERNIOPLASTY	1	60	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
12	JNA	34	M	02/12/2014	09/12/2014	70	170	12	N	LIIH	LIIH	HERNIOPL	1	65	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	

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												ASTY												
13	PSA	63	M	19/07/2015	27/07/2015	55	165	11	N	RIIH	RIIH	HERNIOPLASTY	2	45	Y	NIGHT BEFORE	N	N	Y	5	N	N	N	N	
14	PSS	67	M	04/07/2015	14/07/2015	65	165	10	N	RIIH	RIIH	HERNIOPLASTY	2	50	Y	NIGHT BEFORE	Y	Y	Y	5	N	N	N	N	
15	PBD	68	M	16/03/2015	20/03/2015	62	160	13.5	STAPH	RDIH	RDIH	HERNIOPLASTY	2	55	Y	EARLY MORNING	N	Y	Y	5	N	PUS	SUP	N	TYPE I
16	GSJ	49	M	19/03/2015	24/03/2015	47	165	12.5	NOORG	LDIH	LDIH	HERNIOPLASTY	2	75	Y	NIGHT BEFORE	Y	Y	Y	5	N	N	N	N	
17	NHV	24	M	17/07/2015	22/07/2015	46	150	16	N	RIIH	RIIH	HERNIOPLASTY	1	60	Y	NIGHT BEFORE	Y	Y	Y	5	N	N	N	N	
18	MHL	70	M	05/07/2015	12/07/2015	55	155	10	N	LIIH	LIIH	HERNIOPLASTY	2	40	Y	NIGHT BEFORE	Y	Y	Y	5	N	N	N	N	
19	TAB	29	M	10/07/2015	17/07/2015	46	146	12.5	N	RIIH	RIIH	HERNIOPLASTY	2	35	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
20	JBK	65	M	23/01/2015	01/02/2015	45	135	14	N	RDIH	RDIH	HERNIOPLASTY	1	65	Y	EARLY MORNING	Y	Y	Y	5	N	N	N	N	
21	SGM	29	M	30/01/2015	08/02/2015	70	180	10	N	LDIH	LDIH	HERNIOPLASTY	2	60	Y	EARLY MORNING	N	Y	Y	5	N	N	N	N	
22	MNN	40	M	24/11/2014	28/11/2014	56	175	12	N	RDIH	RDIH	HERNIOPLASTY	1	65	Y	NIGHT BEFORE	N	Y	Y	4	N	N	N	N	
23	BSA	54	M	21/03/2015	24/03/2015	36	128	10.5	N	LDIH	LDIH	HERNIOPLASTY	1	15	Y	NONE	N	N	Y	2	N	N	N	N	
24	MAA	52	M	14/03/2015	20/03/2015	50	145	15	N	RIIH	RIIH	HERNIOPLASTY	1	20	Y	NIGHT BEFORE	N	N	Y	5	N	N	N	N	
25	GER	59	M	27/10/2014	30/10/2014	52	152	15.5	N	LIIH	LIIH	HERNIOPLASTY	1	70	Y	NIGHT BEFORE	N	Y	Y	2	N	N	N	N	

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26	PVH	28	M	20/10/2014	29/10/2014	60	160	11	N	RIIH	RIIH	HERNIOPLASTY	1	60	Y	NIGHT BEFORE	N	Y	Y	6	N	N	N	N	
27	MGB	65	M	24/06/2016	28/06/2016	54	165	12	N	RDIH	RDIH	HERNIOPLASTY	2	65	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
28	MMP	25	M	14/07/2015	24/07/2015	52	180	12	N	RDIH	RDIH	HERNIOPLASTY	2	45	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
29	JKH	23	M	23/07/2015	01/08/2015	50	165	14	N	LDIH	LDIH	HERNIOPLASTY	1	20	Y	EARLY MORNING	Y	Y	Y	5	N	N	N	N	
30	JST	35	M	08/06/2015	11/06/2015	60	180	12	N	RIIH	RIIH	HERNIOPLASTY	2	45	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
31	JSH	42	M	15/06/2015	21/06/2015	52	165	12	N	LIIH	LIIH	HERNIOPLASTY	2	50	Y	NIGHT BEFORE	N	Y	Y	8	N	N	N	N	
32	KBM	43	M	14/11/2014	16/11/2014	45	160	12	N	RIIH	RIIH	HERNIOPLASTY	1	15	Y	N	N	N	Y	2	N	N	N	N	
33	PKB	23	M	06/07/2015	12/07/2015	54	165	12.5	N	RDIH	RDIH	HERNIOPLASTY	2	60	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
34	PNT	22	M	15/06/2015	21/06/2015	42	150	11	N	RDIH	RDIH	HERNIOPLASTY	1	60	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
35	KSM	65	M	25/06/2015	02/07/2015	42	150	11	N	RDIH	RDIH	HERNIOPLASTY	1	45	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
36	NAA	27	M	20/08/2015	23/08/2015	44	152	12	N	LDIH	LDIH	HERNIOPLASTY	1	20	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
37	BLB	49	M	24/03/2015	02/04/2015	44	165	11	N	RDIH	RDIH	HERNIOPLASTY	1	45	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
38	MJG	48	M	19/01/2015	26/01/2015	42	154	11	N	LDIH	LDIH	HERNIOPLASTY	1	45	Y	NIGHT BEFORE	N	N	Y	5	N	N	GAPE	N	TYPE I
39	KAA	37	M	10/07	18/07/	43	150	12	N	RII	RII	HERN	2	5	Y	EARLY	N	Y	Y	5	N	N	N	N	

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				/2015	2015					H	H	IOPL ASTY		0		MORNING								
40	JYA	42	M	12/06 /2015	17/06/ 2015	50	168	10.5	N	LII H	LII H	HERN IOPL ASTY	1	15	Y	N	N	N	Y	2	N	N	N	N	
41	JRB	48	M	06/07 /2015	10/07/ 2015	38	142	12	N	RII H	RII H	HERN IOPL ASTY	1	25	Y	N	N	N	Y	2	N	N	N	N	
42	AGS	39	M	31/08 /2015	07/09/ 2015	58	152	10.5	N	RDI H	RD IH	HERN IOPL ASTY	1	65	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
43	GLM	46	M	16/03 /2015	21/03/ 2015	47	165	12.5	NO ORG	LII H	LII H	HERN IOPL ASTY	2	60	Y	NIGHT BEFORE	Y	Y	Y	5	N	N	N	N	
44	PSM	46	M	24/06 /2015	27/06/ 2015	54	165	12	N	RII H	RII H	HERN IOPL ASTY	1	25	Y		N	N	Y	5	N	N	N	N	
45	PCM	28	M	14/11 /2014	16/11/ 2014	48	150	12	N	RDI H	RD IH	HERN IOPL ASTY	1	30	Y	N	N	N	Y	2	N	N	N	N	
46	PYJ	38	M	12/11 /2014	20/11/ 2014	54	165	12	N	RDI H	RD IH	HERN IOPL ASTY	2	65	Y	NIGHT BEFORE	N	Y	Y	5	N	N	N	N	
47	KDB	21	M	06/04 /2015	09/04/ 2015	65	150	11	N	LDI H	LDI H	HERN IOPL ASTY	1	15	Y	N	N	N	Y	5	N	N	N	N	
48	MR M	22	M	08/08 /2015	10/08/ 2015	45	145	12	N	RII H	RII H	HERN IOPL ASTY	1	20	Y	YES	Y	N	Y	5	N	N	N	N	
49	NSP	43	M	16/03 /2015	19/03/ 2015	48	160	12	N	LII H	LII H	HERN IOPL ASTY	1	15	Y	N	N	N	Y	2	N	N	N	N	
50	PRH	45	M	08/07 /2015	11/07/ 2015	50	165	10.5	N	RII H	RII H	HERN IOPL ASTY	2	20	Y	N	N	N	Y	2	N	N	N	N	

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