“A RANDOMIZED CONTROLLED TRIAL TO COMPARE EFFICACY OF VICRYL PLUS VERSUS VICRYL IN REDUCING ABDOMINAL SURGICAL SITE INFECTION” – A ONE YEAR STUDY

REG NO. BH0110003

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
in
GENERAL SURGERY

DEPARTMENT OF SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

APRIL - 2013

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ENDORSEMENT

This is to certify that the dissertation entitled **"A RANDOMIZED CONTROLLED TRIAL TO COMPARE EFFICACY OF VICRYL PLUS VERSUS VICRYL IN REDUCING ABDOMINAL SURGICAL SITE INFECTION" – A ONE YEAR STUDY** is a bonafide research work done by **THE CANDIDATE REG. NO. BH0110003**.

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LIST OF ABBREVIATIONS USED

^{0}C	- Degree centigrade
AD	- Anno Domini
AIDS	- Acquired immunodeficiency syndrome
BC	- Before Christ
CDC	- Centre For Disease Control And Prevention
CFU	- Colony Forming Unit
Cms	- Centimeter
E.coli	- Escherichia coli
EGF	- Epidermal Growth Factor
EPS	- Extracellular polymeric material
FDA	- Food and Drug Administration
FGF	- Fibroblast Growth Factor
HIV	- Human immunodeficiency virus
IGF	- Insulin Like Growth Factor
IL	- Interleukin
Min	- Minute
MRSA	- Methicillin Resistant Staphylococcus Aureus
NINSS	- Nosocomial Infection National Surveillance Service
NNIS	- National Nosocomial Infections Surveillance
PDGF	- Platelet Derived Growth Factor
PHMB	- Poly Hexamethylene Biguanide
S.aureus	- Staphylococcus aureus
S.epidermidis	- Staphylococcus epidermidis
SIRS	- Systemic Inflammatory Response Syndrome
SSI	- Surgical Site Infection
TGF	- Transforming Growth Factor
TNF	- Tumor Necrosis Factor
VISA	- Vancomycin Intermediate Staphylococcus Aureus

ABSTRACT

Background and objectives

Recently a new antimicrobial suture Polyglactin 910 coated with tricosan (Vicryl Plus) has been introduced. It is a broad spectrum antiseptic that has been widely used in humans for over 30 years. This study was aimed to evaluate the efficacy of new antibacterial suture (Vicryl Plus) compared with a traditional suture (Vicryl) in reducing abdominal surgical site infection.

Methodology

The present one year randomized controlled trial was conducted in Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum between January 2011 to December 2011. A total of 100 patients undergoing abdominal surgeries were included in the study. These patients were randomized into two groups based on computer generated blocked random numbers that is group A (Vicryl plus) and group B (Vicryl).

Results

In this series of 100 patients, 67 were males and 33 were female. Most of the patients (32%) were aged between 46 to 50 years in each group and in group A the same proportion of patients had age between 31 to 45 years. The mean age in Group A was 47.34 ± 15.00 and in group B mean age was 49.08 ± 16.71. Among more than half (52% vs 54%) of patients the duration of surgery was between 61 to 120 minutes in both the groups. 70% of patients had contaminated wound compared to 80% in group B. The demographic characteristics and surgical parameters such as type of wound, surgical time, were comparable in

both the groups ($p>0.05$). In patients with group A, significantly lower incidence of abdominal surgical site infection was observed when compared to group B in all the wound assessments that is day three, five, seven and fifteen ($p<0.05$). The mean length of hospital stay in group A was 14.00 ± 4.94 days compared to 15.26 ± 4.60 days ($p>0.05$).

Conclusion and interpretation

Overall, the study showed that, the use of new antibacterial suture (Vicryl Plus) showed better efficacy in reducing abdominal surgical site infections when compared to traditional suture (Vicryl).

Keywords

Surgical site infections; Suture material; Vicryl plus; Vicryl.

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Chapter 1

Introduction

INTRODUCTION

Surgical Site Infections (SSIs) are infections of tissues, organs, or spaces exposed by surgeons during performance of an invasive procedure.[1] Postoperative SSIs remain a major source of illness and a less frequent cause of death in the surgical patient.[2] These infections number approximately 500,000 per year, among an estimated 27 million surgical procedures,[3] and account for approximately one quarter of the estimated two million nosocomial infections in the United States each year,[4] being a developed nation.

Surgical site infection is a dangerous condition, a heavy burden on the patient and social health system. Such infections lengthen bed stay from an average of seven days and results in higher costs.[5] The incidence of infection varies from surgeon to surgeon, from hospital to hospital, from one surgical procedure to another, and most importantly from one patient to another.[6]

Surgical site infections are among the most common hospital acquired infections comprising 14 to 16% of inpatient infections.[7] Potential sources of infection are the patient (especially contamination by alimentary tract bacteria), hospital environment, food, other patients, staff, infected surgical instruments, dressings, and even drugs and injections.[8] Surgical site infections are also related to suture.[9] The surgical infection contributes to financial morbidity. Despite of strict aseptic technique, antibiotic coverage (when indicated), and an adequate surgical technique, the infection rate remains high.[10]

A suture is a biomaterial device, natural or synthetic, used to approximate tissues together following separation by surgery or trauma. It can also be used to

denote the method used for mechanical wound closure. Although there are other methods for mechanical wound closure such as staples, tape and adhesives, sutures are the most widely used materials in wound closure.

The goals of wound closure include obliteration of dead space; Even distribution of tension along deep suture lines; Maintenance of tensile strength across the wound until tissue tensile strength is adequate and approximation of the epithelial portion of the closure.

Recently a new antimicrobial suture Polyglactin 910 coated with tricosan (Vicryl Plus) has been introduced. Tricosan is a broad spectrum antiseptic that has been widely used in humans for over 30 years.[10] There was no evidence of carcinogenic potential, genotoxicity, and skin sensitization potential in 2 years animal model studies.[11] *In vitro* study, showed efficacy against *S. aureus, S. epidermidis*, MRSA, MRSE, Vancomycin resistant *Enterococcus fecalis*, *Psedomonas aeruginosa*, *Corybavterium spp.* and *E. coli*.[11]

In view of the above context the present study was undertaken to evaluate the efficacy of new antibacterial suture (Vicryl Plus) compared with a traditional suture (Vicryl) in reducing abdominal surgical site infection.

Chapter 2

Objectives

OBJECTIVES

Objective of the present study was to evaluate the efficacy of new antibacterial suture (Vicryl Plus) compared with a traditional suture (Vicryl) in reducing abdominal surgical site infection.

Chapter 3

Review of Literature

REVIEW OF LITERATURE

Surgical Site Infections continues to be a major source of morbidity following operative procedures. The NNIS report for 1986-1996 described an SSI rate of 2.6% for all operations at the reporting hospitals. It seems likely that overall SSI rates are likely to be greater than reported. All surgical wounds are contaminated by bacteria, but only a minority actually demonstrate clinical infection. The SSI are the biological summation of several factors: the inoculum of bacteria introduced into the wound during the procedure, the unique virulence of contaminants, the microenvironment of each wound, and the integrity of the patients host defense mechanisms. Although an SSI rate of zero may not be achievable, continued progress in understanding the biology of infection at the surgical site and consistent applications of proven methods of prevention will allow to further reduce the frequency, cost, and morbidity associated with SSI.[12]

Historical perspective

The ancient Egyptians were the first civilization to have trained clinicians to treat physical aliments. Medical papyri, such as the Edwin Smith papyrus (circa 1600 BC) and the Ebers papyrus (circa 1534 BC), provided detailed information of management of disease, including wound management with the application of various potions and grease to assist healing.[13,14]

Galen (Roman gladiatorial surgeon, 130-200 AD) was first to recognize that pus from wounds inflicted by the gladiators heralded healing. The link between pus formation and healing was emphasized so strongly that foreign material was introduced into wounds to promote pus formation-suppuration. The

concept of wound healing remained a mystery, as highlighted by the famous saying by Ambroise Paré (French military surgeon, 1510-1590), "I dressed the wound. God healed it."[15]

Koch (Professor of Hygiene and Microbiology, Berlin, 1843-1910) first recognized the cause of infective foci as secondary to microbial growth in his 19th century postulates. Semmelweis (Austrian obstetrician, 1818-1865) demonstrated a 5-fold reduction in puerperal sepsis by hand washing between performing postmortem examinations and entering the delivery room. Joseph Lister (Professor of Surgery, London, 1827-1912) and Louis Pasteur (French bacteriologist, 1822-1895) revolutionized the entire concept of wound infection.[16]

Antisepsis derived from the Greek ‘‘against putrefaction’’ and its use in modern medicine is most frequently linked to the work of Lister. It refers to the use of solutions for disinfection.[17]

In the late eighteenth and early nineteenth centuries operative outcomes were poor. Wounds were allowed to heal by secondary intention and morbidity and mortality were associated largely with surgical-site infections (SSIs). The first use of an antiseptic skin agent in surgery is credited to the English surgeon Joseph Lister (1827- 1912).[17]

Before the mid-19th century, surgical patients commonly developed postoperative “irritative fever,” followed by purulent drainage from their incisions, overwhelming sepsis, and often death.[18]

Prior to the mid-19th century, limb amputation was associated with an alarming 50% postoperative mortality from sepsis. Following Louis Pasteur's discovery that tissue decay was caused by microscopic organisms, Lister theorized that the spread of these microbes through surgical wounds was responsible for death in the postoperative period.[19]

Lister placed carbolic acid into open fractures to sterilize the wound and to prevent sepsis and hence the need for amputation.[20] Listers work radically changed surgery from an activity associated with infection and death to a discipline that could eliminate suffering and prolong life.[18]

As early as 1882, Labarraque, a French pharmacist, demonstrated that solutions containing chlorides of lime or soda could eradicate foul odour associated with human corpses and that such solutions could be used as disinfectants and antiseptics.[21] As late as the 19th century, aseptic surgery was not routine practice. Sterilization of instruments began in the 1880s as did the wearing of gowns, masks, and gloves. Halsted (Professor of Surgery, Johns Hopkins University, United States, 1852-1922) introduced rubber gloves to his scrub nurse (and future wife) because she was developing skin irritation from the chemicals used to disinfect instruments. The routine use of gloves was introduced by Halsted's student J. Bloodgood.[20]

Alexander Fleming (microbiologist, London, 1881-1955) performed many of his bacteriological studies during World War I and is credited with the discovery of penicillin.[20]

Penicillin first was used clinically in 1940 by Howard Florey. With the use of antibiotics, a new era in the management of wound infections commenced. Unfortunately, eradication of the infective plague affecting surgical wounds has not ended because of the insurgence of antibiotic-resistant bacterial strains and the nature of more adventurous surgical intervention in immunocompromised patients and in implant surgery.[20]

Definition of Surgical Site Infections[22]

Surgical Site Infection is a difficult term to define accurately because it has a wide spectrum of possible clinical features. Surgical-site infection (SSI) is defined by the Centres for Disease Control and Prevention (CDC) as a proliferation of pathogenic micro-organisms which develops in an incision site either within the skin and subcutaneous fat (superficial), musculo-fascial layers (deep), or in an organ or cavity, if opened during surgery.

Since the skin is normally colonized by bacterial flora, an SSI cannot be diagnosed by the microbiological evidence alone but in conjunction with clinical signs which include: redness, heat, pain and swelling, separation of the suture line (dehiscence), or the presence of an abscess in the deeper tissues. Patients may mount a systemic inflammatory response syndrome (SIRS) with an elevated white cell count, body temperature $<35^0$C or $>38^0$C, pulse rate >100/min, respiratory rate >20/min, or in severe cases develop signs of sepsis, with an attendant increase in morbidity and mortality.

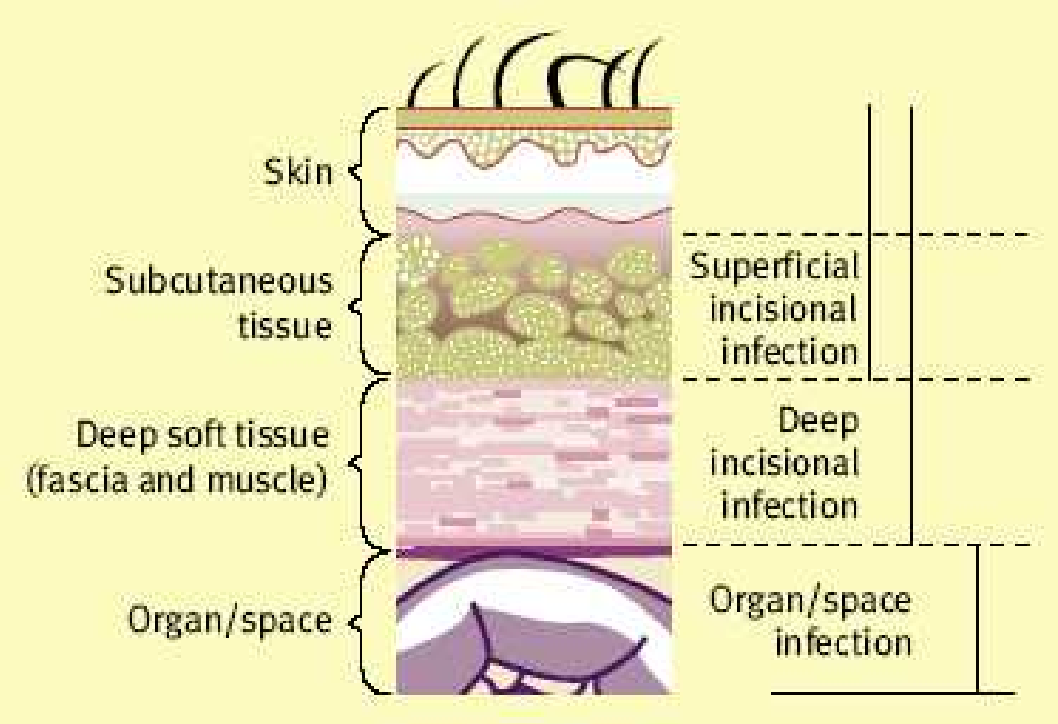


Figure 1. Schematic representation of the anatomical classification of surgical site infections[23]

Classification of surgical site infection[23]

I. Superficial Incisional SSI

Infection involves only skin and subcutaneous tissues of incision.

- Occurs within 30 days after the operation;
- Involves only the skin or subcutaneous tissue; and
- At least 1 of the following:
    - Purulent drainage (culture documentation not required)
    - Organisms isolated from fluid/tissue of superficial incision
    - At least 1 sign of inflammation (eg, pain or tenderness, induration, erythema, local warmth of the wound)
    - Wound is deliberately opened by the surgeon
    - Surgeon or attending physician declares the wound infected.

A wound is not considered a superficial site infection if a stitch abscess is present, the infection is at an episiotomy or circumcision site or a burn wound, or the SSI extends into the fascia or muscle.

II. Deep Incisional SSI

Infection involves deep tissues such as fascial and muscle layers. This also includes infection involving both superficial and deep incision sites.

- Occurs within 30 days of operation or within 1 year if an implant is present;
- Involves deep soft tissues (fascia and/or muscle) of the incision; and
- At least one of the following:
    - Purulent drainage from the deep incision but without organ/space involvement

- Fascial dehiscence or fascia is deliberately separated by the surgeon due to signs of inflammation
- Deep abscess is identified by direct examination or during reoperation, by histopathology, or by radiologic examination
- Surgeon or attending physician declares that deep incisional infection is present.

III. Organ/Space SSI

Infection involves any part of the anatomy in organs and spaces other than the incision, which was opened or manipulated during operation.

- Occurs within 30 days of operation or within 1 year if an implant is present;
- Involves anatomic structures not opened or manipulated during the operation; and
- At least 1 of the following:
  - Purulent drainage from a drain placed by a stab wound into the organ/space
  - Organisms isolated from organ/space by aseptic culturing technique
  - Identification of abscess in the organ/space by direct examination, during reoperation, or by histopathologic or radiologic examination
  - Diagnosis of organ/space SSI by surgeon or attending physician.

Epidemiology

Frequency

I. International

Internationally, the frequency of SSI is difficult to monitor because criteria for diagnosis might not be standardized. A survey sponsored by the World Health Organization demonstrated a prevalence of nosocomial infections varying from 3-21%, with wound infections accounting for 5-34% of the total.[24] The 2002 survey report by the Nosocomial Infection National Surveillance Service (NINSS), which covers the period between October 1997 and September 2001, indicates that the incidence of hospital acquired infection related to surgical wounds in the United Kingdom is as high as 10% and costs the National Health Service in the United Kingdom approximately 1 billion pounds (1.8 billion dollars) annually.

II. United States

Surgical site infections (SSIs) are not an extinct entity; they account for 14-16% of the estimated two million nosocomial infections affecting hospitalized patients in the United States.[25]

III. Indian scenario

The surgical site infection rate reports by different workers have differed considerably. A study[26] done in India reported overall infection rate as 8.95% and

number of studies carried out in India indicate an overall infection rate of 4.04 to 30% for clean surgeries and 10.06 to 45% for clean-contaminated surgeries.[27,28,29]

Collated data on the incidence of wound infections probably underestimate true incidence because most wound infections occur when the patient is discharged, and these infections may be treated in the community without hospital notification.[24]

Incidence of SSIs with regard to abdominal surgical sites and operating conditions[5] are as shown in the table number 1.

IV. Mortality/Morbidity

Surgical site infections are associated not only with increased morbidity but also with mortality. Seventy-seven percent of the deaths of surgical patients were related to surgical wound infection.[18] Kirkland et al calculated a relative risk of death of 2.2 attributable to SSIs, compared to matched surgical patients without infection.[30]

Pathogenesis of SSIs[31]

- *The Human Inflammatory Response*

With the creation of the surgical incision through the skin and in to subcutaneous tissues, 5 critical initiators of the human inflammatory response are activated (Figure 2).

Incidence of SSIs with regard to abdominal surgical sites and operating conditions[5]

Clean wounds	1.5 to 3.7%
Clean -contaminated wounds	3 to 4%
Contaminated wounds	8.5%
Dirty	
Infected wounds	28 to 40%
Laparoscopy	10%
Umbilical hernia	2 to 5%
Cancer of the colon	
Without taking antimicrobial drugs	30 to 60%
With antibiotic and proper intestine wash	10%
Colostomy -	above 50%
Colon perforation	20%
Stomach cancer and surgery	20%
Hernia	50%
Adult appendectomy	10 to 20%
Children's appendicitis	2 to 5%
Aged appendicitis and in pregnant women	10 to 50%
AIDS victims	above 50%
Liver abscess	20%
Hydatid cyst	2 to 5%
Acute and chronic cholecystectomy without stones	10%
Acute septic cholangitis	10 to 20%
Laparoscopic cholecystectomy	2 to 5%
Splenectomy	2 to 5%

Coagulation proteins and platelets are initially activated as part of the human hemostatic mechanism, but they also herald the onset of inflammation. Mast cells and complement proteins are activated, and bradykinin is produced from its ubiquitous protein precursors. The net effect of these 5 factors is vasodilation and increased local blood flow at the site of the surgical incision. While bulk flow is increased, flow velocity is reduced in preparation for margination of phagocytes.

The simultaneous occurrence of increased vascular permeability and local vasodilation facilitates the formation of edema fluid, resulting in increased space between endothelial cells. The increased vascular permeability provides phagocytic access to the injured soft tissue, while edema provides aqueous conduits for the navigation of these phagocytes through the normally condensed extracellular tissues. Activation products from the 5 initiator events described above result in the production of nonspecific chemoattractant signals, while mast cells produce specific chemokine signals that "draw" specific neutrophil, monocyte, and other leukocyte populations into the area of the surgical site.

The important point of this discussion about inflammation is that tissue injury from the incision initiates the mobilization of phagocytes into the wound before bacterial contamination actually occurs from the procedure itself. This mobilization of the innate host defenses before significant intraoperative contamination occurs undoubtedly gives the patient an advantage against infection as an outcome.

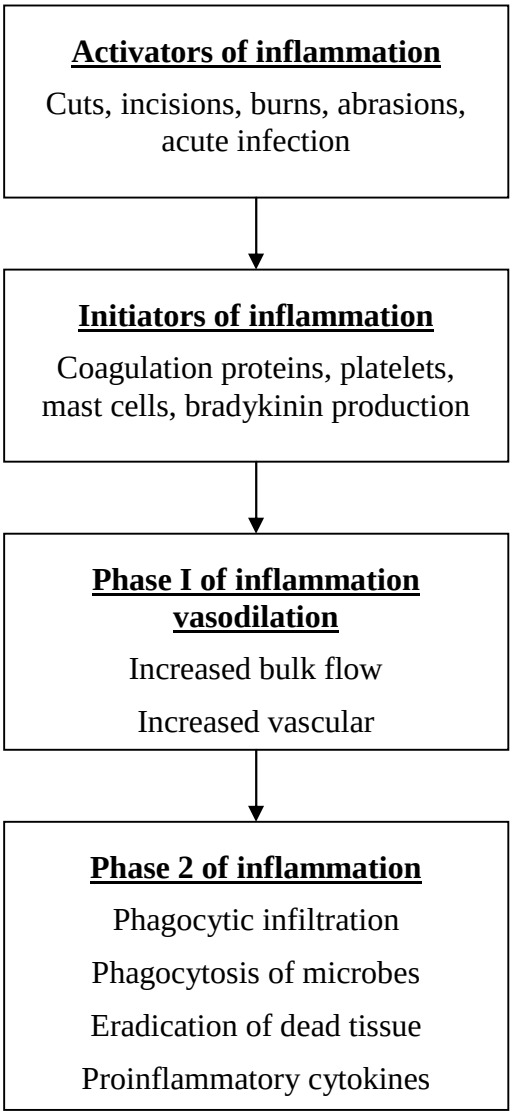


Figure 2. The consequences of inflammation[31]

The abundant release of chemoattractant signals, products of tissue injury, orchestrates the movement of phagocytes into the wound. Chemoattractant signaling proteins bind to local vascular endothelial cells and upregulate selectin proteins on the endothelial surface of these cells, which results in neutrophil "rolling" on the endothelial surface within the post-capillary venule. Further interaction between neutrophil and endothelial cell adhesion proteins anchor the neutrophil to the surface of the endothelial cell, and the chemoattractant gradient

then acts as a biological "beacon" to direct neutrophil movement toward the site of injury. Neutrophil presence at the surgical site allows systematic ingestion and digestion of any microbial contaminants from the operation.

By about 24 hours after creation of the surgical wound, monocytes enter the surgical site and initiate 1 of 2 different scenarios. When microbial contamination has been minimal and the early arriving neutrophils have been able to adequately control the bacteria that are present, then monocytes produce local chemical signals to regulate the wound-healing process. Myofibrocytes migrate into the fibrin matrix of the wound, and collagen deposition displaces its fibrin latticework. However, if microbial contamination and proliferation overwhelm the initial neutrophil infiltration, the monocyte assumes the role of a proinflammatory cell with the release of potent cytokines.

Tumor necrosis factor (TNF)-alpha is produced and released by the monocytes and serves numerous functions; notably, it becomes a potent paracrine signal to upregulate vigorous neutrophil activity within the wound. TNF-alpha-stimulated neutrophils consume microbes, and lysosomal vacuoles may release reactive oxygen intermediates and acid hydrolases into the extracellular space from its lysosomal vacuoles. The extracellular release of reactive oxygen intermediates and the acid hydrolases results in lipid peroxidation of the local environment, with further tissue injury and further activation of the initiator signals. In this way, the entire inflammatory response is further intensified. Interleukin (IL)-1, IL-6, and other proinflammatory signals are released by the activated monocyte and serve as endocrine signals responsible for fever, stimulation of acute phase reactants, and other responses.

The net effect of vigorous neutrophilic stimulation, tissue autolysis, and sustained stimulation of inflammatory initiation is the creation of a wound space that is a host-pathogen battlefield. Ultimately, the wound space is filled with necrotic tissue, neutrophils, bacteria, and proteinaceous fluid that together constitute pus. The viable tissues around the infected wound typically exhibit the classic signs of inflammation. Wound *rubor* reflects local vasodilation. *Calor* is the warmth of the vasodilated tissues resulting in increased heat conduction. *Tumor* reflects the presence of edema fluid about the wound. *Dolor* occurs from stimulation of nerve nociceptors by the numerous products of the inflammatory cascade and tissue injury. The discharge of pus from the wound interface via the incision completes the natural history of SSI.

Pathophysiology of wound healing[20]

Wound healing is a continuum of complex interrelated biological processes at the molecular level. Healing is divided into the following phases for descriptive purposes: inflammatory phase, proliferative phase, and maturation phase.

The inflammatory phase commences as soon as tissue integrity is disrupted by injury; this begins the coagulation cascade to limit bleeding. Platelets are the first of the cellular components that aggregate at the wound, and, as a result of their degranulation (platelet reaction), they release several cytokines (or paracrine growth factors). These cytokines include platelet derived growth factor (PDGF), insulinlike growth factor-1 (IGF-1), epidermal growth factor (EGF), and fibroblast growth factor (FGF). Serotonin is also released, which,

together with histamine (released by mast cells), induces a reversible opening of the junctions between the endothelial cells, allowing the passage of neutrophils and monocytes (which become macrophages) to the site of injury.

This large cellular movement to the injury site is induced by cytokines secreted by the platelets (chemotaxis) and by further chemotactic cytokines secreted by the macrophages themselves once at the site of injury. These include transforming growth factor alpha (TGF-alpha) and transforming growth factor beta (TGF-beta). Consequently, an inflammatory exudate that contains red blood cells, neutrophils, macrophages, and plasma proteins, including coagulation cascade proteins and fibrin strands, fills the wound in a matter of hours. Macrophages not only scavenge but they also are central to the wound healing process because of their cytokine secretion.

The proliferative phase begins as the cells that migrate to the site of injury, such as fibroblasts, epithelial cells, and vascular endothelial cells, start to proliferate and the cellularity of the wound increases. The cytokines involved in this phase include FGFs, particularly FGF-2, which stimulates angiogenesis and epithelial cell and fibroblast proliferation. The marginal basal cells at the edge of the wound migrate across the wound, and, within 48 hours, the entire wound is epithelialized. In the depth of the wound, the number of inflammatory cells decreases with the increase in stromal cells, such as fibroblasts and endothelial cells, which, in turn, continue to secrete cytokines. Cellular proliferation continues with the formation of extracellular matrix proteins, including collagen and new capillaries (angiogenesis). This process is variable in length and may last several weeks.

In the maturation phase, the dominant feature is collagen. The dense bundle of fibers, characteristic of collagen, is the predominant constituent of the scar. Wound contraction occurs to some degree in primary closed wounds but is a pronounced feature in wounds left to close by secondary intention. The cells responsible for wound contraction are called myofibroblasts, which resemble fibroblasts but have cytoplasmic actin filaments responsible for contraction.

The wound continuously undergoes remodeling and try to achieve a state similar to that prior to injury. The wound has 70-80% of its original tensile strength at 3-4 months postoperative.

Causes

All surgical wounds are contaminated by microbes, but in most cases, infection does not develop because innate host defenses are quite efficient in the elimination of contaminants. A complex interplay between host, microbial, and surgical factors ultimately determines the prevention or establishment of a wound infection.[32]

Factors that affect surgical wound healing

Microbiology

Microbial factors that influence the establishment of a wound infection are the bacterial inoculum, virulence, and the effect of the microenvironment. When these microbial factors are conducive, impaired host defenses set the stage for enacting the chain of events that produce wound infection.[32]

Most SSIs are contaminated by the patient's own endogenous flora, which are present on the skin, mucous membranes, or hollow viscera. The traditional microbial concentration quoted as being highly associated with SSIs is that of bacterial counts higher than 10,000 organisms per gram of tissue (or in the case of burned sites, organisms per cm^2 of wound).[32]

Organisms associated with SSIs vary with type of procedure and anatomic location of the operation. The usual pathogens on skin and mucosal surfaces are gram-positive cocci (notably staphylococci, streptococci) account for most exogenous flora involved in SSIs. The most common group of bacteria responsible for SSIs are *Staphylococcus aureus*. The emergence of resistant strains has considerably increased the burden of morbidity and mortality associated with wound infections. However, gram-negative aerobes and anaerobic bacteria contaminate skin in the groin/perineal areas. The contaminating pathogens in gastrointestinal surgery are the multitude of intrinsic bowel flora, which include gram-negative bacilli (eg, *Escherichia coli*) and gram-positive microbes, including enterococci and anaerobic organisms. Sources of such pathogens include surgical/hospital personnel and intraoperative circumstances, including surgical instruments, articles brought into the operative field, and the operating room air.[32]

Methicillin resistant *Staphylococcus aureus* (MRSA) is proving to be the scourge of modern day surgery. Like other strains of *S aureus,* MRSA can colonize the skin and body of an individual without causing sickness, and, in this way, it can be passed on to other individuals unknowingly. Problems arise in the treatment of overt infections with MRSA because antibiotic choice is very

limited. MRSA infections appear to be increasing in frequency and are displaying resistance to a wider range of antibiotics.[32]

Of particular concern are the vancomycin intermediate *Staphylococcus aureus* (VISA) strains of MRSA. These strains are beginning to develop resistance to vancomycin, which is currently the most effective antibiotic against MRSA.[32]

Pathogens Commonly Associated with Wound Infections and Frequency of Occurrence[33]

Pathogen	Frequency (%)
Staphylococcus aureus	20
Coagulase-negative staphylococci	14
Enterococci	12
Escherichia coli	8
Pseudomonas aeruginosa	8
Enterobacter species	7
Proteus mirabilis	3
Klebsiella pneumoniae	3
Other streptococci	3
Candida albicans	3
Group D streptococci	2
Other gram-positive aerobes	2
Bacteroides fragilis	2

In clean surgical procedures, in which the gastrointestinal, gynaecologic, and respiratory tracts have not been entered, *Staphylococcus aureus* from the exogenous environment or the patient's skin flora is the usual cause of infection. In other categories of surgical procedures, including clean-contaminated, contaminated, and dirty, the polymicrobial aerobic and anaerobic flora closely resembling the normal endogenous microflora of the surgically resected organ are the most frequently isolated pathogens.[34]

Others risk factors

Patient Factors[35]

Obesity increases risk substantially when the subcutaneous abdominal fat layer exceeds 3 cm (1.5 inches). The risk is increased by the need for a larger incision, decreased circulation to the fat tissue or the technical difficulty of operating through a large fat layer.

Infection at another site may increase the risk of spreading infection through the bloodstream.

Immunocompromised patients (those with HIV/AIDS, those with chronic corticosteroid use such as occurs with asthma and heavy smokers or users of other tobacco products) are at significantly greater risk of SSIs.

Malnutrition may or may not be a contributing factor. Unfortunately, most studies have not been conducted in developing countries where severe malnutrition is more common.

Age, race, socioeconomic status and chronic diseases, such as diabetes and malignancy, are difficult to assess because they are frequently associated with other factors that independently contribute to risk. For example, age over 70 may be accompanied by decreased defense mechanisms, poor nutrition and anemia.[20]

Wound characteristics include nonviable tissue in wound; hematoma; foreign material, including drains and sutures; dead space; poor skin preparation, including shaving; and pre-existent sepsis (local or distant).[20]

Operative characteristics include poor surgical technique; lengthy operation (>2 h); intraoperative contamination, including infected theater staff and instruments and inadequate theater ventilation; prolonged preoperative stay in the hospital; and hypothermia.[20]

The type of procedure is a risk factor. Certain procedures are associated with a higher risk of wound contamination than others. Surgical wounds have been classified as clean, clean-contaminated, contaminated, and dirty-infected as shown in table number 3.

Surgical Wound Classification and Subsequent Risk of Infection[33,36]

Classification	Description	Infective Risk (%)
Clean (Class I)	Uninfected operative wound No acute inflammation Closed primarily Respiratory, gastrointestinal, biliary, and urinary tracts not entered No break in aseptic technique Closed drainage used if necessary	< 2
Clean-contaminated (Class II)	Elective entry into respiratory, biliary, gastrointestinal, urinary tracts and with minimal spillage No evidence of infection or major break in aseptic technique Example: appendectomy	< 10
Contaminated (Class III)	Nonpurulent inflammation present Gross spillage from gastrointestinal tract Penetrating traumatic wounds < 4 hours Major break in aseptic technique	About 20
Dirty-infected (Class IV)	Purulent inflammation present Preoperative perforation of viscera Penetrating traumatic wounds >4 hours	About 40

Role of suture material

The role of suture material in the development of wound infections has been the subject of speculation among surgeons since the 1960s.[37,38] Sutures are a contributory factor in infection; in fact, 66% of SSIs are related to the incision.[39]

Microbial adherence to the surface of suture material has been reported in the surgical literature for many years. The presence of foreign materials in a wound enhances the susceptibility of surrounding tissues to infection. The number of bacteria needed to establish infection can be reduced 10,000-fold by the presence of a silk suture.[40]

In fact, it is postulated that in the presence of sutures, only 100 colony-forming units (CFU)/mg are necessary to produce infection.[11] Various bacteria may contaminate not only the tissue in the surgical wound, but the actual suture material. Once suture material becomes contaminated, local mechanisms of wound decontamination become ineffective.[41,42]

Sutures, that present virtually in all major operative procedures, may create a setting in which low numbers of bacteria proliferate while sequestered from host defenses. Any suture product of natural or synthetic composition and of mono- or multi-filament construction is susceptible to bacterial attachment and colonization. It is also clear that colonization is associated with surgical site infections.[43]

Sutures, like most other implants, have a non-shedding surface to which bacteria can adhere, form biofilms and potentiate SSIs. The adherence of bacteria

to various sutures has been investigated, and variations in adherence-affinity correlated with infection. 'Biofilms' are ubiquitous and form whenever micro-organisms such as bacteria, yeasts, algae, fungi, or protozoa attach to surfaces.[44]

A study,[45] in 1985, reported that, percutaneous sutures approximating skin edges were often colonized from the body surface into the wound track by strains of *S epidermidis* capable of producing an amorphous extracellular matrix (biofilms), protecting the microbial populations from host defense factors.

Another recent study[9] in 2007, showed the presence of biofilms around the bacteria after 60 minutes, and this material appeared adhered to the sutures three hours after contamination. Once attached, free-living bacteria undergo a phenotypic change and, within minutes, deposit 'slime': extracellular polymeric material (EPS) or biofilms matrix. Implants have non-shedding surfaces, which can be colonized by skin or other bacteria during surgery, to form a biofilms.

At least 60% of human infections are believed to involve biofilms and the recognition that biofilms are the dominant mode of microbial growth, and that the majority of bacteria exist in biofilms, is still recent emphasized.[44]

Once established, in the environment or in infections, biofilms bacteria are difficult to treat because, shielded within the matrix, they are less susceptible to antibiotics and antiseptics. This recalcitrance is not reflected by laboratory susceptibility tests and a bacterium shown to be susceptible to antibiotics may be impossible to treat in a biofilms. A reason for the reduced susceptibility of biofilm-embedded organisms, compared with free living bacteria counterparts, and includes: heterogenecity of growth rates; cells being in a stationary

physiological phase, present as recalcitrant 'persister' cells or able to degrade antimicrobials; and reduced rates of penetration of the biofilms by antibiotics. Biofilms can also shield their constituent micro-organisms from the body's immune system. The free-living form of the isolate was susceptible *in vitro* but in biofilms was resistant. Once a biofilm infection is established on an implant, it usually antibiotic treatment and needs removal[11,44,46]

Antimicrobial Sutures

The antimicrobial suture is interesting. Fowler,[47] in 1965, recommended that all suture materials be steeped in a 1/2,000 solution of chlorhexidine before suturing reduces surgical wound infections, although many manufacturers had argued against him.

The actual development of an antibacterial surgical suture has been under consideration since early 1980s.[48] Preventive strategies included prophylactic antibiotics before the biofilm can form, or 'intelligent' surfaces that prevent colonization or have antimicrobial properties. Potential antiseptics for coating surfaces include chlorhexidine, polyhexamethylene biguanide (PHMB), octenidine and triclosan. Compared with antibiotics, which generally have single pharmacological targets, which select for resistance, antiseptics have several or multiple targets and true 'resistance' is rare. Antimicrobial-impregnated implants, which prevent bacterial adhesion and biofilms formation, can avoid long-term, ineffective, systemic antibiotics, reduce the risk of microbial resistance generation and need for implant removal. Ideally, antiseptics should have a rapid potent and broad microbicidal spectrum with long-lasting effects and no risk of

developing antimicrobial resistance. They should be biocompatible with medical products, not impair healing processes and be well tolerated in wounds with no toxicity or systemic absorption.[44]

Recently, the only substance being used for impregnantion in suture is Triclosan. Triclosan 5-chloro-2 (2, 4-dichlorophenoxyphenol) is a broad-spectrum antimicrobial agent developed over 40 years ago.[49]

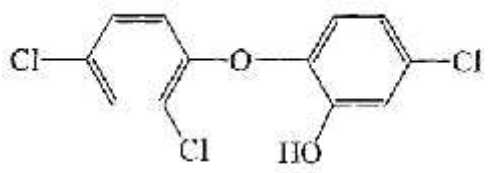


Figure 3. Chemical structure of Triclosan

The chemical structure is as shown in Figure 1. In the United States, triclosan has been used in underarm deodorants and deodorant soaps since 1960s. It was first introduced in the healthcare industry in a surgical scrub at 1% in 1972 and for oral care in toothpaste in Europe in 1985.[50]

In 1989, triclosan was approved for use in cosmetics, which can be used up to 0.3% by the European Community Cosmetic Directive.[51]

Over the last 20 years, the use of triclosan has grown rapidly in personal care products including soap, hand sanitizer, cosmetics, and toothpaste, as well as household products such as odour-fighting socks and germ-resistant sponges, kitchenware, and bedding. A 2001 U.S. study found triclosan in 76% of 395 commercial soaps examined.[52]

At the beginning, the mode of action was supposed to be through nonspecific disruption of the bacterial cell membrane. Newer studies, however, revealed that the target of triclosan is the Fab I gene, which blocks bacterial fatty acid synthesis (particularly the enzyme enoyl-acyl carrier protein reductase).[52]

The combined effect of triclosan with antibiotic, amoxicillin, gentamicin, nitrofurantoin and the fluoroquinolones was superior when considering significant increases in susceptibility. The synergistic effects of triclosan and several antibiotics are consistent with a triclosan-dependent metabolic strain and/or membrane disruptive effect, and offers important insight into the combined use of antimicrobial compounds in clinical practice.[53]

The antimicrobial spectrum and speed of activity of triclosan are well documented both as an active ingredient and in a wide array of formulations.[50]

A comprehensive submission of published, unpublished, and historical data was prepared for the FDA and includes in vitro and in vivo data on triclosan. These references include more than 1000 in vitro tests performed with triclosan formulations on a broad array of microorganisms such as fungi, *Clostridium dificile*, methicillin-resistant *Staphylococcus aureus* (MRSA), and vancomycin-resistant *Enterococcus*. The results indicate the formulations have similar broad-spectrum antiviral activity on adenovirus 2, herpes simplex virus, type 1, HIV-l, influenza A, and rhinovirus 37 at both concentrations with a high level of activity on enveloped viruses such as herpes simplex virus, HIV-l, and influenza.[50]

As previous mentioned above, FDA (US) has approved polyglactin 910 sutures coated with triclosan for commercial used since 2002.[54]

The first report[55] was published in 2005 show prospective, randomized, controlled, open-label, comparative, single-center study was conducted on 147 pediatric patients (age 1-18 years) undergoing various surgical procedures with either polyglactin 910 sutures coated with antibiotic triclosan or polyglactin sutures without triclosan. The endpoints of this study focused on intraoperative handling and wound healing characteristics instead of surgical site infection that the aim of this investigated suture. For intra-operative handlings were favorable and not significantly different for both sutures, although coated polyglactin 910 sutures with triclosan received more "excellent" scores (71% vs. 59%). Wound healing characteristics were comparable for both sutures, except significantly fewer patients with triclosan sutures reported pain on day one compared with patients without triclosan sutures (p=0.01). The overall incidence of adverse events was 18%; none was device related, and there was no difference between treatment groups. This study was sponsored by industry for antimicrobial sutures.

Another retrospective study[52] to evaluate reduction of sternal wound infection was conducted in 2007. A total of 479 patients underwent a cardiac surgical procedure. One hundred and three patients were closed with triclosan-coated suture material, whereas the remaining 376 patients had their incision closed with non-coated sutures. After closing the sternal bone with steel wires, the sternal fascia was closed with interrupted 2-0 Vicryl Plus antibacterial sutures (Vicryl Plus Antibacterial [Ethicon, Sommerville, New Jersey]). Thereafter the subcutaneous tissue was closed with 2-0 Vicryl Plus antibacterial in a continuous fashion. The skin was closed with 3-0 Vicryl Plus antibacterial intra-cutaneous or after the fashion of Donatti in redo cases or patients with diabetes mellitus.

During the study period, 24 patients had superficial (n=10) or deep (n=14) sternal wound infections. All those patients were closed with conventional suture material. In the triclosan group, no wound infection or dehiscence was observed during hospital stay and follow-up visits. This study was also evaluated the cost-effective for this operation.

Although the frequency of sternal infection is reported to be low, between 0.7% and 3.3%, the current Centers for Disease Control (CDC) guidelines recommend the wound to be covered for 24 to 48 hours after surgery. A fibrin scab seals the wound and thereby prevents the admission of bacteria. Several studies have been conducted to evaluate the effectiveness of certain dressings, but unfortunately owing to the lack of empirical evidence, a high variability exists in the type of dressing used.

A prospective, double-blinded, randomized controlled trial[54] evaluate reduction of CSF shunt infection following shunt procedures was conducted in 2008. The study enrolled 61 patients, among whom 84 CSF shunt procedures were performed over 21 months. The shunt infection rate in the study group was 2 (4.3%) of 46 procedures and 8 (21%) of 38 procedures in the control group (p=0.038). There were no statistically significant differences in shunt infection risk factors between the groups (procedure type and time, age < 6 months, weight < 4 kg and recent history of shunt infection). No suture-related adverse events were reported in either group. Wound closure with antimicrobial suture was associated with a favor lower shunt infection risk than placebo suture wound closure in this study but statistic not significant due to small sample size.

A large cohort,[40] evaluated the effect of antibacterial-coated sutures for abdominal closure in 2009. The authors performed 2,088 operations between October 2004 and September 2006 via midline incision and prevent wound infections in different kinds of abdominal surgery, including colorectal, hepatopancreatic, and vascular surgery. In the first time period (October 2004 to September 2005= TP1), a PDS II loop suture was used. In the second time period (October 2005 to September 2006 =TP2), Vicryl plus was used. Using a PDS loop suture for abdominal wall closure in TP1, 10.8% of patients with wound infections were detected. The number of patients with wound infections decreased in TP2 using Vicryl plus for abdominal wall closure to 4.9% ($P < .001$) despite no other changes in protocols of patient care. Other risk factors for the development of site infections were comparable in the two groups. The use of antibiotic-coated loop suture for abdominal wall closure can decrease the number wound infections after abdominal surgery. Although this study was done in a single center in Germany over two different time periods and using two different types of suture material with high volume of sample size. Although these findings of the study are impressive, the design and data analysis appear to be still sub-optimal because of no randomization of the patients, lack of microbial confirmation and multivariate analysis. Additionally, their strategies for the management of contaminated wounds are not shown, which greatly influence the outcomes of such wounds. Despite an increase in the rate of wound infection in the PDS group, the duration of hospital stay was not prolonged in this group, suggesting that complications other than wound infection might occur more frequently in the triclosan coated group. Abdominal wound dehiscence, which is

a deep incisional surgical site infections and a very serious wound complication, appears to be more related to the suture materials used for transfascial mass closure when compared with the association between these suture materials and superficial incisional SSIs. So this study should show whether antibiotic coating of transfascial sutures could decrease the rate of wound dehiscence.[56]

However recently (in 2011), authors[40] extended their study between October 2003 and October 2007 (previous study reported between October 2004 and September 2006) and focused in transverse abdominal incision instead midline incision as previous study.[57] 839 operations were performed using a transverse abdominal incision. In the first time period, a PDSII loop suture was used for abdominal wall closure. In the second time period, we used Vicryl plus. Wound infections after transverse laparotomy. 409 Using a PDSII loop suture for abdominal wall closure in the first time period, 9.2% of the patients developed wound infections. In the second time period, 430 using Vicryl plus, the number of wound infections decreased to 4.3% ($p< 0,005$). Both groups were comparable regarding risk factors despite no other changes in protocols of patient care. The major clinical finding of this study is the superiority of braided Vicryl plus sutures over PDS sutures in relation to wound infections after a two-layered closure of transverse laparotomy in patients undergoing hepatobiliary resections.

Two randomized control trials[10,58] showed no statistical difference in both group. First in trial[58] in 2009 was a prospective, randomized, controlled, double blind, comparative, a single center study which was conducted to to assess the efficacy of an antibacterial suture (polyglactin 910 coated with triclosan) compared to uncoated polyglactin 910 sutures in reducing rates of SSI in patients

undergoing appendectomy. Surgeons and assistants were blinded to suture type as similarity in appearance made the two products indistinguishable. Baseline patient characteristics did not difference between both group. The rate of SSI was not statistically significantly different between the two treatment groups, nor was the complication rate after one year. The authors concluded that polyglactin 910 coated with triclosan was safe in surgical practice, with a comparable outcome to polyglactin 910 but that more study was needed to confirm this.

Second in 2011,[59] which was a prospective study was evaluated the effect of triclosan-coated sutures on surgical wide excision of a head or neck cancer and reconstructive procedures. 241 patients were included in this study, divided into two groups by flip of a coin. The Triclosan group contained 112 patients, whose surgical wounds were closed with Triclosan-coated sutures (Vicryl Plus). The control group included the remaining 129 patients, whose surgical wounds were closed with conventional Vicryl sutures. The results showed cervical wound infection rate was 14.9% (17/112) in the Triclosan group and 14.7% (19/129) in the control group, and these rates were not significantly different. Tumor stage and delayed intra-oral flap healing were independent risk factors for cervical wound infection. In this study, Triclosan-coated Vicryl sutures did not reduce the infection rate of cervical wounds after head or neck cancer surgery. The effectiveness of this suture material in head and neck cancer surgery should be considered with caution. The study showed negative result, which was also stated by another study in 2009.[59]

The study[59] investigated the effect of triclosan on wound healing a double blind prospective pilot study in women undergoing a breast reduction was

performed. Each patient was her own control. After randomization the Triclosan-coated sutures were used either on the left or right side. The contralateral side was used as the control. The incidence of dehiscence was studied. The result showed twenty-six patients were included. In the triclosan breasts there was a wound dehiscence in 16 cases, whereas in the control breasts in seven cases a dehiscence was observed (p=0.023). These results suggest that triclosan-coated sutures should be used with caution. These sutures have already been introduced on to the market without good clinical studies and might have potential adverse effects as shown by these data. The bilateral dehiscence in five cases found that four unilateral dehiscent cases in the triclosan group (p=0.023). The limitation of this study was small sample size but a double blind randomized design in which each patient was their own controls have value because each patient is her own control.

Chapter 4

Methodology

METHODOLOGY

The present study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2011 to December 2011 on 100 patients undergoing abdominal surgeries by vertical incision.

Study design

The study design was one year randomized controlled trial.

Study period and duration

The present one year study was conducted during the period of 1st January 2011 to 31st December 2011.

Place

This study was carried out at Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, and Jawaharlal Nehru Medical College, Belgaum.

Source of Data

Patients undergoing abdominal surgeries during the study period were included in the study.

Sample size

A total of 100 patients undergoing abdominal surgeries by vertical incision were studied.

Sampling procedure

Based on 80% of the average three years hospital data the sample size was determined as 100 cases undergoing abdominal surgeries.

Selection criteria

Inclusion

- All patients 18 years and above.
- Participants undergoing abdominal surgeries by vertical incision providing informed consent.

Exclusion

- Immuno compromised individuals (diabetics, human immunodeficiency virus (HIV), bleeding disorders, patients on steroid therapy).
- Pregnancy.
- Known hypersensitivity to suture or its components.
- Pre existing surgical site infection.

Ethical clearance

Before the commencement of the study Ethical Clearance was obtained from the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

All the patients fulfilling selection criteria were explained about the purpose of study and a written informed consent was obtained before enrollment (Annexure I).

Method of collection of data

Demographic data such as age and sex were recorded. Patients were interviewed for the history and a thorough physical examination was conducted including vitals and systemic examination. These findings were recorded on a predesigned and pretested proforma (Annexure II).

Randomization

Based on the computer generated blocked random numbers patient were randomized into two groups that is;

- Group A - Vicryl plus
- Group B - Conventional vicryl

Investigations

Routine investigations such as complete blood count, blood urea, serum creatinine and special investigation such as ultrasound/computed tomography of abdomen was done.

Procedure

Pre operative

Shaving of the abdomen from nipple to mid-thigh prior to surgery. On the operation table the abdomen was cleaned with antiseptic and then re cleaning with spirit was done under all aseptic precautions. Injection ciprofloxacin 100 mL IV and Inj. metronidazole 100 ml IV were given prior to skin incision.

Closure technique

In case of laparotomy by vertical incision, elective/emergency the closure of wound was done in layers.

Group A: Vicryl plus was used for the closure of peritoneum and subcutaneous layer.

Group B: Vicryl was used for the closure of peritoneum and subcutaneous layer.

The rectus was closed using PDS/prolene and skin using ethilon/staples.

Post operative

The patients were postoperatively also medicated with Inj. ceftriaxone 1 gm IV twice daily and Inj. metronidazole 100 ml thrice daily and if indicated and were changed to higher antibiotics accordingly.

Wound inspection was done on day three, five, seven and fifteen post operatively. Wound was inspected regularly and in case of infection patients were treated accordingly.

The assessment of wound was done on day three, five, seven and fifteen post operatively based on Southampton wound scoring system.[7]

Statistical analysis

The data obtained was tabulated, categorical data was expressed as rates, ratios and percentages and comparison was done using chi-square test. Continuous data was expressed as mean ± standard deviation and comparison was done using student 't' test. A 'p' value of less than or equal to 0.05 was considered as statistically significant.

Chapter 5

Results

RESULTS

The present one year randomized controlled trial was conducted in Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum between January 2011 to December 2011. A total of 100 patients undergoing abdominal surgeries were included in the study. Further, these patients were randomized into two groups based on computer generated blocked random numbers that is,

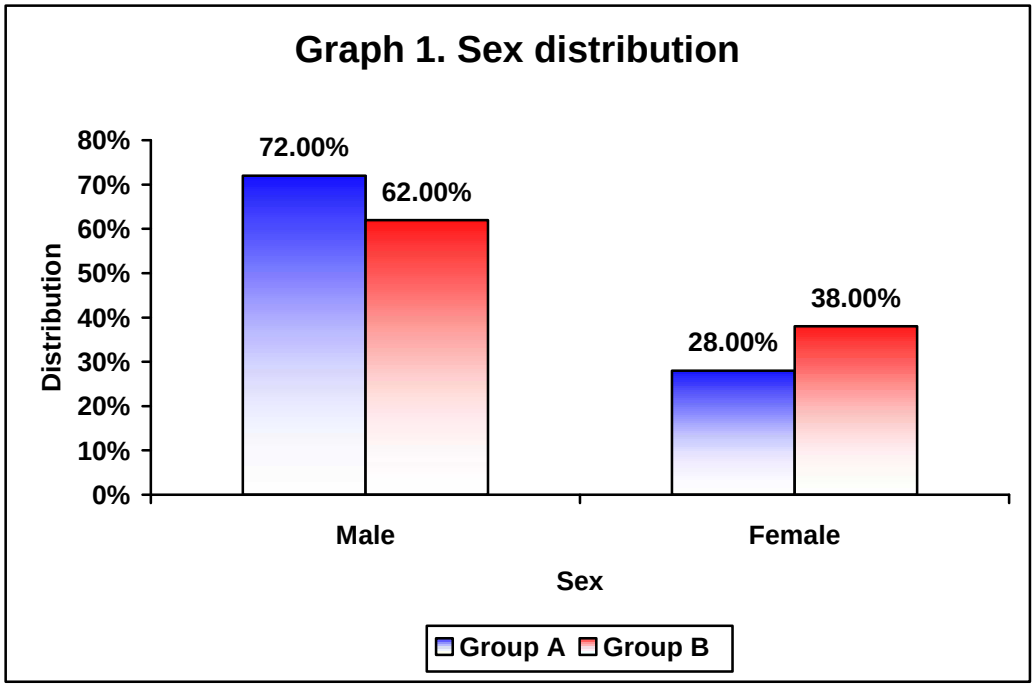
- Group A - Vicryl plus
- Group B - Conventional vicryl

The data obtained was entered into Microsoft Excel Worksheet and analysed. The final results are tabulated as below.

Table 1. Sex distribution

Sex	Group A (n=50)		Group B (n=50)	
	Number	**Percent**	**Number**	**Percent**
Male	36	72.00	31	62.00
Female	14	28.00	19	38.00
Total	**50**	**100.00**	**50**	**100.00**

x^2_1= 1.133 **p = 0.288**

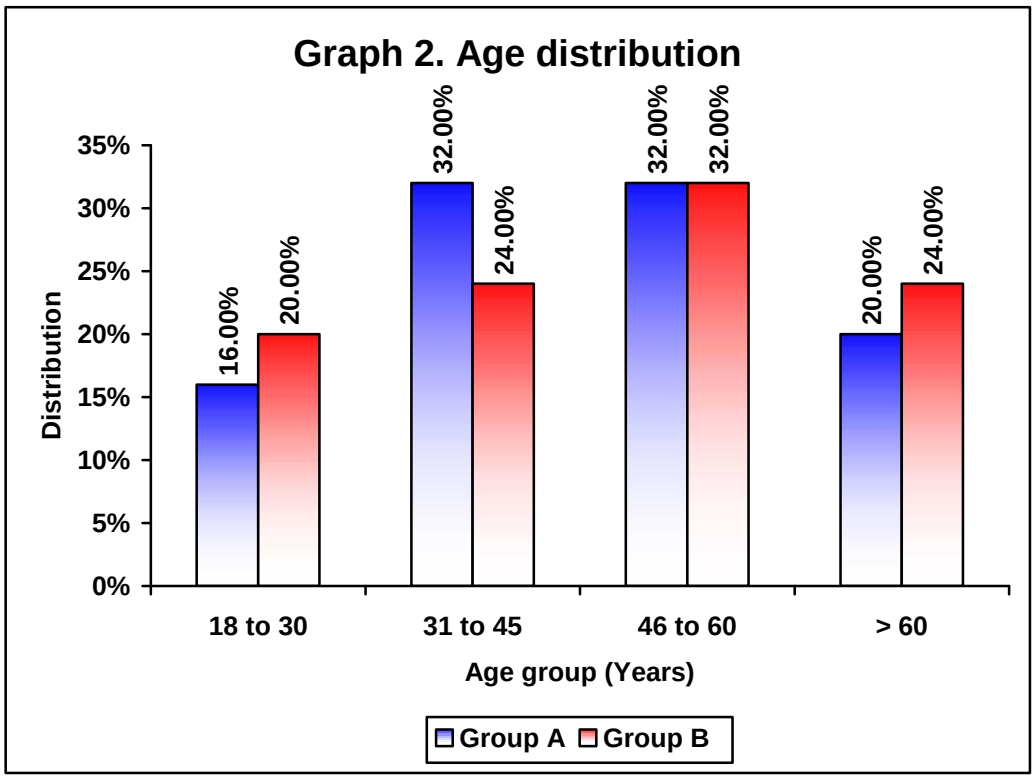


In this series of 100 patients, 67 were males and 33 were female. In the group A, 72% were males and 28% were female. In group B, 62% were males and 38% were females. The comparison between the two groups showed no significant difference in the sex distribution (p=0.849)

Table 2. Age distribution

Age group (Years)	Group A (n=50)		Group B (n=50)	
	Number	**Percent**	**Number**	**Percent**
18 to 30	8	16.00	10	20.00
31 to 45	16	32.00	12	24.00
46 to 60	16	32.00	16	32.00
> 60	10	20.00	12	24.00
Total	**50**	**100.00**	**50**	**100.00**

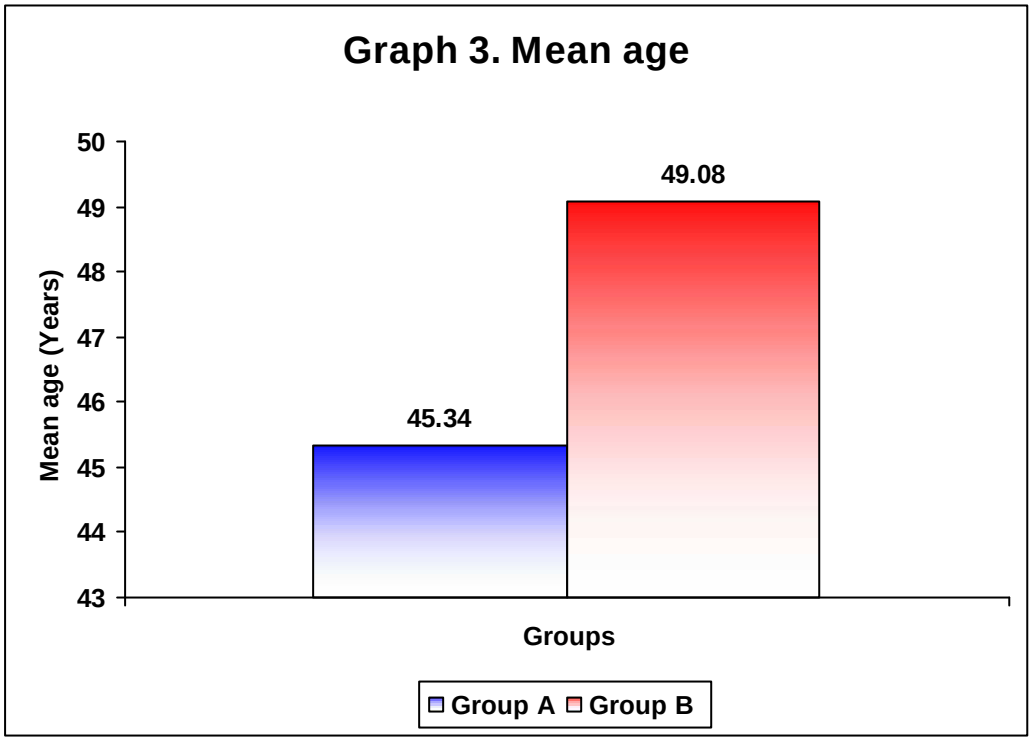
x^2_3= 0.801 **p = 0.849**



In this study, 32% patients were aged between 46 to 50 years in each group and in group A the same proportion of patients had age between 31 to 45 years.

Table 3. Mean age

Variable (Years)	Group A (n=50)	Group B (n=50)
Mean	45.34	49.08
SD	15.00	16.71
Median	48.50	49.50
Minimum	18.00	20.00
Maximum	80.00	82.00
t = 0.853	**DF = 98**	**p = 0.396**

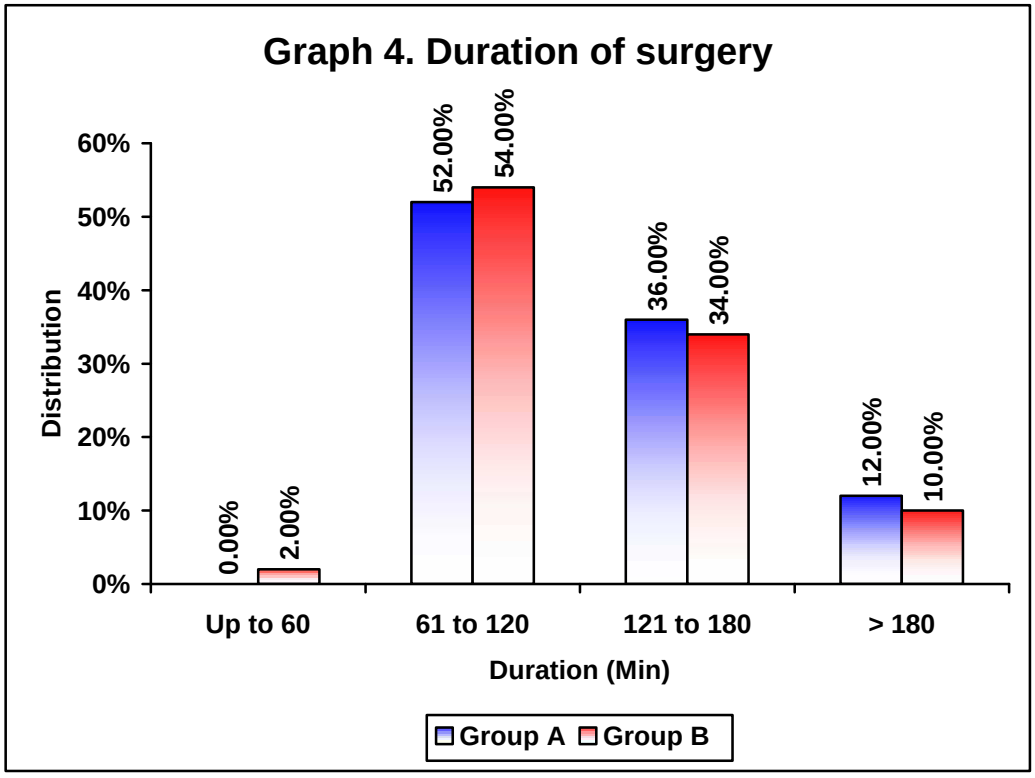


In this study the mean age in Group A was 45.34 ± 15.21 and in group B mean age was 49.08 ± 16.71. However this difference was statistically not significant.

Table 4. Duration of surgery

Duration (Min)	Group A (n=50)		Group B (n=50)	
	Number	Percent	Number	Percent
Upto 60	0	0.00	1	2.00
61 to 120	26	52.00	27	54.00
121 to 180	18	36.00	17	34.00
> 180	6	12.00	5	10.00
Total	**50**	**100.00**	**50**	**100.00**

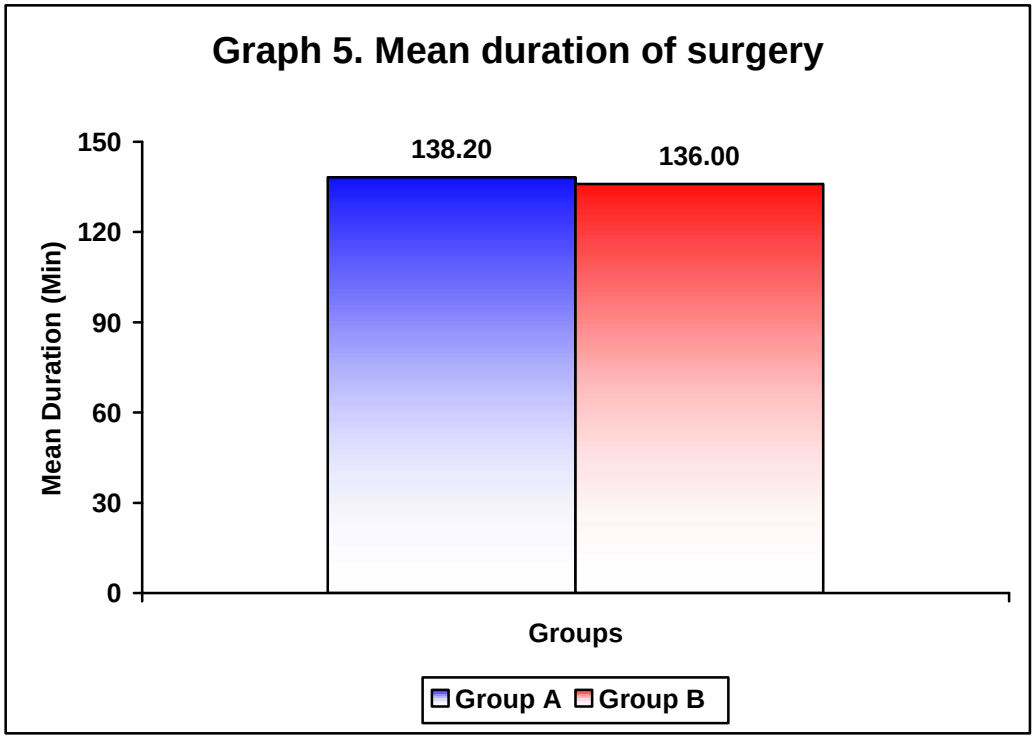
x^2_2= 0.153 **p = 0.925**



In this study among more than half (52% vs 54%) of patients the duration of surgery was between 61 to 120 minutes in both the groups. However the duration of surgery in group A and B were comparable.

Table 5. Mean duration of surgery

Duration (Min)	Group A (n=50)	Group B (n=50)
Mean	138.2	136.60
SD	39.37	44.61
Median	120	120.00
Minimum	90	60.00
Maximum	200	240.00
t=0.190	**DF = 98**	**p=0.849**

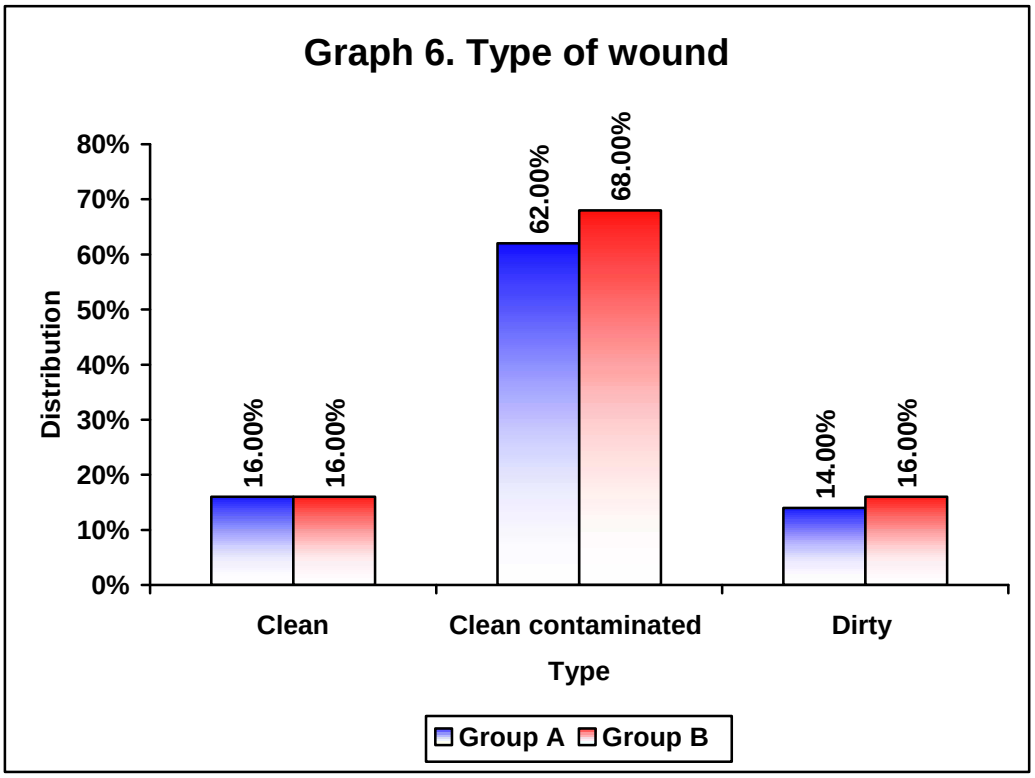


In this study the mean duration of surgery was 138.20 ± 39.37 in group A and 136.00 ± 44.61 minutes. However the mean duration in both the groups were comparable (p=849)

Table 6. Type of wound

Wound	Group A (n=50)		Group B (n=50)	
	Number	**Percent**	**Number**	**Percent**
Clean	8	16.00	8	16.00
Clean contaminated	31	62.00	34	68.00
Dirty	7	14.00	8	16.00
Total	**50**	**100.00**	**50**	**100.00**

x^2_2= 1.100 **p = 0.577**

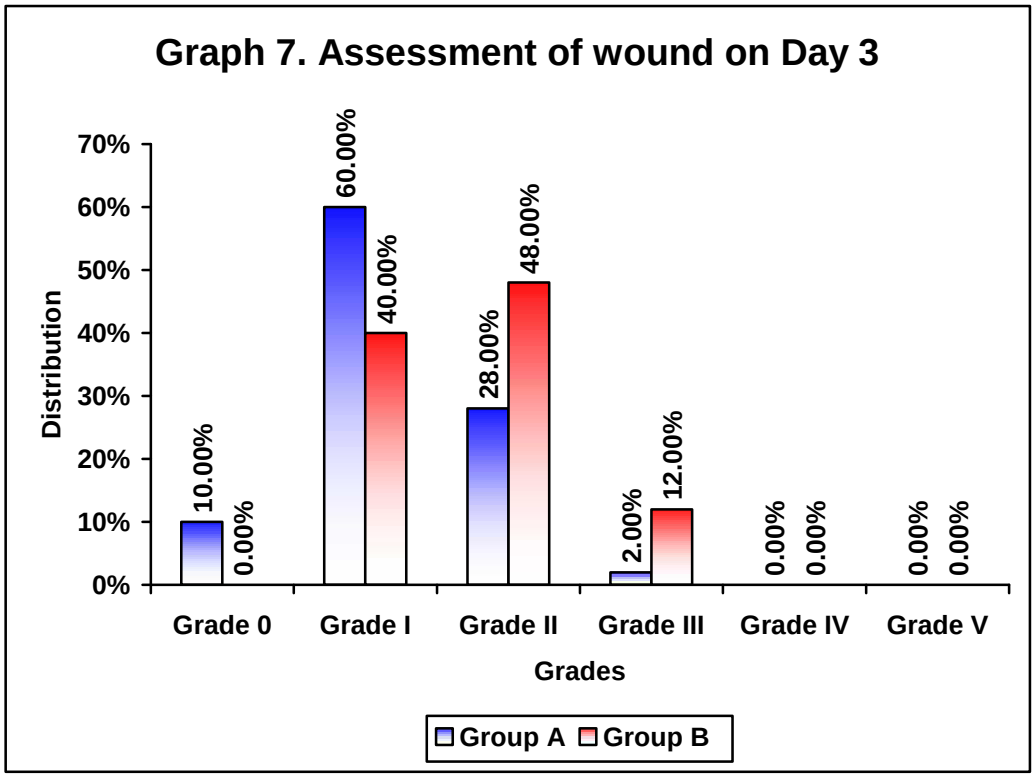


In the present study among the patients with group A 62% of patients had clean contaminated wound compared to 68% in group B. However this difference was statistically not significant (p=0.577).

Table 7. Assessment of wound on Day 3

Grades	Appearance	Group A (n=50)		Group B (n=50)	
		Number	Percent	Number	Percent
0		**5**	**10.00**	**0**	**0.00**
I	A	22	44.00	16	32.00
	B	0	0.00	1	2.00
	C	8	16.00	3	6.00
	Total	**30**	**60.00**	**20**	**40.00**
II	A	11	22.00	14	28.00
	B	1	2.00	5	10.00
	C	1	2.00	3	6.00
	D	1	2.00	2	4.00
	Total	**14**	**28.00**	**24**	**48.00**
III	A	1	2.00	5	10.00
	B	0	0.00	0	0.00
	C	0	0.00	1	2.00
	D	0	0.00	0	0.00
	Total	**1**	**2.00**	**6**	**12.00**
IV	A	0	0.00	0	0.00
	B	0	0.00	0	0.00
	Total	**0**	**0.00**	**0**	**0.00**
V		**0**	**0.00**	**0**	**0.00**
Total		**50**	**100**	**50**	**100**

x^2_3=13.203 **p=0.004**

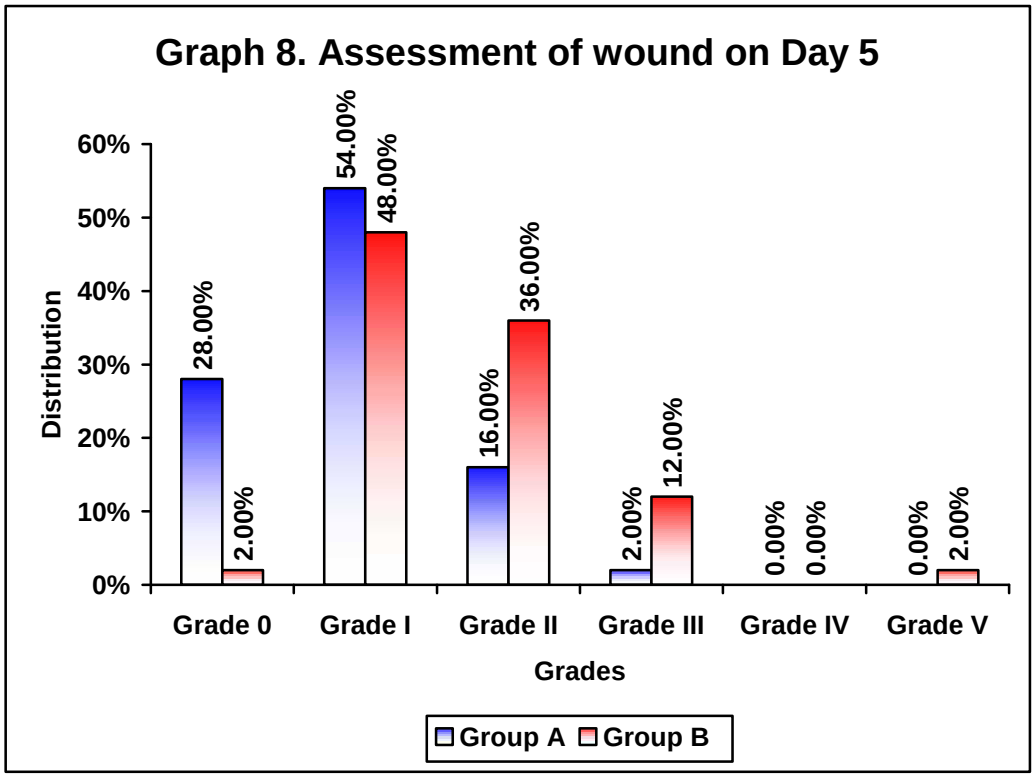


In the present study on post operative day three, the most of the patients (60%) in group A had grade I followed by 28% with grade II and 10% with grade 0 and 2% with grade III whereas, in group B 48% of the patients had grade II, 40% had grade I and 12% had grade III wound assessment grades. None of the patient in group B had grade 0 and this difference was statistically significant (p=0.004).

Table 8. Assessment of wound on Day 5

Grades	Appearance	Group A (n=50)		Group B (n=50)	
		Number	**Percent**	**Number**	**Percent**
0		**14**	**28.00**	**1**	**2.00**
I	A	21	42.00	19	38.00
	B	1	2.00	1	2.00
	C	5	10.00	4	8.00
	Total	**27**	**54.00**	**24**	**48.00**
II	A	6	12.00	15	30.00
	B	1	2.00	1	2.00
	C	0	0.00	2	4.00
	D	1	2.00	0	0.00
	Total	**8**	**16.00**	**18**	**36.00**
III	A	1	2.00	3	6.00
	B	0	0.00	3	6.00
	C	0	0.00	0	0.00
	D	0	0.00	0	0.00
	Total	**1**	**2.00**	**6**	**12.00**
IV	A	0	0.00	0	0.00
	B	0	0.00	0	0.00
	Total	**0**	**0.00**	**0**	**0.00**
V		**0**	**0.00**	**1**	**2.00**
Total		**50**	**100**	**50**	**100**

x^2_3=19.791 **p=0.002**

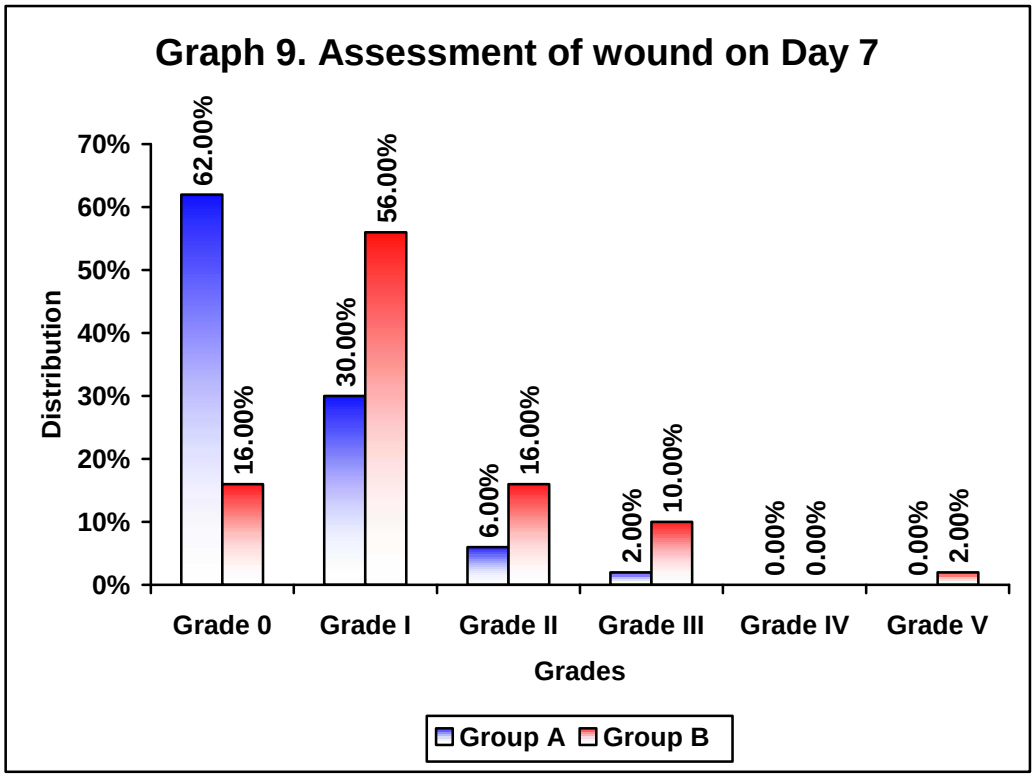


In the present study on post operative day five, more than half (54%) of the patients in group A had grade I followed by 28% with grade 0 and 16% with grade II and 2% with grade III compared to 48% with grade I, 36% with grade II, 12% with grade III and 2% with grade 0 in group B. This difference was statistically significant (p=0.002).

Table 9. Assessment of wound on Day 7

Grades	Appearance	Group A (n=50)		Group B (n=50)	
		Number	Percent	Number	Percent
0		**31**	**62.00**	**8**	**16.00**
I	A	12	24.00	19	38.00
	B	0	0.00	1	2.00
	C	3	6.00	8	16.00
	Total	**15**	**30.00**	**28**	**56.00**
II	A	2	4.00	6	12.00
	B	1	2.00	0	0.00
	C	0	0.00	2	4.00
	D	0	0.00	0	0.00
	Total	**3**	**6.00**	**8**	**16.00**
III	A	1	2.00	2	4.00
	B	0	0.00	2	4.00
	C	0	0.00	0	0.00
	D	0	0.00	1	2.00
		1	**2.00**	**5**	**10.00**
IV	A	0	0.00	0	0.00
	B	0	0.00	0	0.00
	Total	**0**	**0.00**	**0**	**0.00**
V		**0**	**0.00**	**1**	**2.00**
Total		**50**	**100**	**50**	**100**

x^2_3=23.341 **p <0.001**

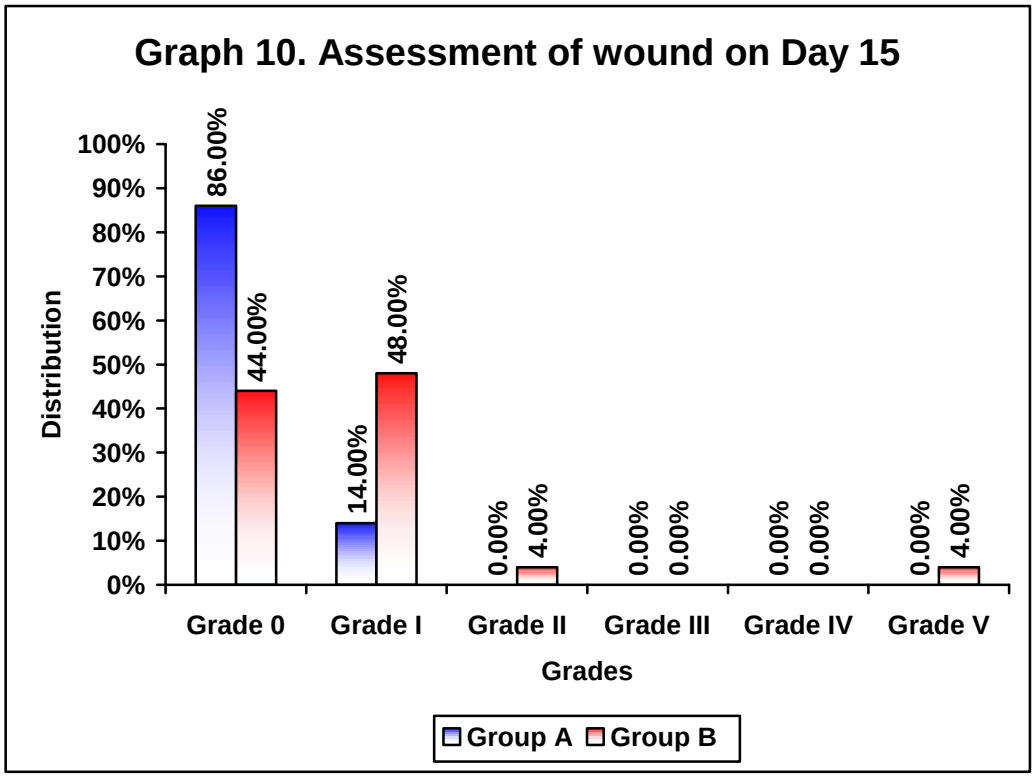


In the present study on post operative day seven, most of the patients in group A (62%) had grade 0 followed by 30% with grade I and 6% with grade II and 2% with grade III. In group B more than half (56%) had grade I followed by 16% each with grade 0 and II and 10% with grade III. This difference was statistically significant ($p<0.001$).

Table 10. Assessment of wound on Day 15

Grades	Appearance	Group A (n=50)		Group B (n=50)	
		Number	**Percent**	**Number**	**Percent**
0		**43**	**86.00**	**22**	**44.00**
I	A	6	12.00	21	42.00
	B	0	0.00	1	2.00
	C	1	2.00	2	4.00
		7	**14.00**	**24**	**48.00**
II	A	0	0.00	2	4.00
	B	0	0.00	0	0.00
	C	0	0.00	0	0.00
	D	0	0.00	0	0.00
		0	**0.00**	**2**	**4.00**
III	A	0	0.00	0	0.00
	B	0	0.00	0	0.00
	C	0	0.00	0	0.00
	D	0	0.00	0	0.00
		0	**0.00**	**0**	**0.00**
IV	A	0	0.00	0	0.00
	B	0	0.00	0	0.00
		0	**0.00**	**0**	**0.00**
V		**0**	**0.00**	**2**	**4.00**
Total		**50**	**100**	**50**	**100**

x^2_2= 20.113 **p =<0.001**

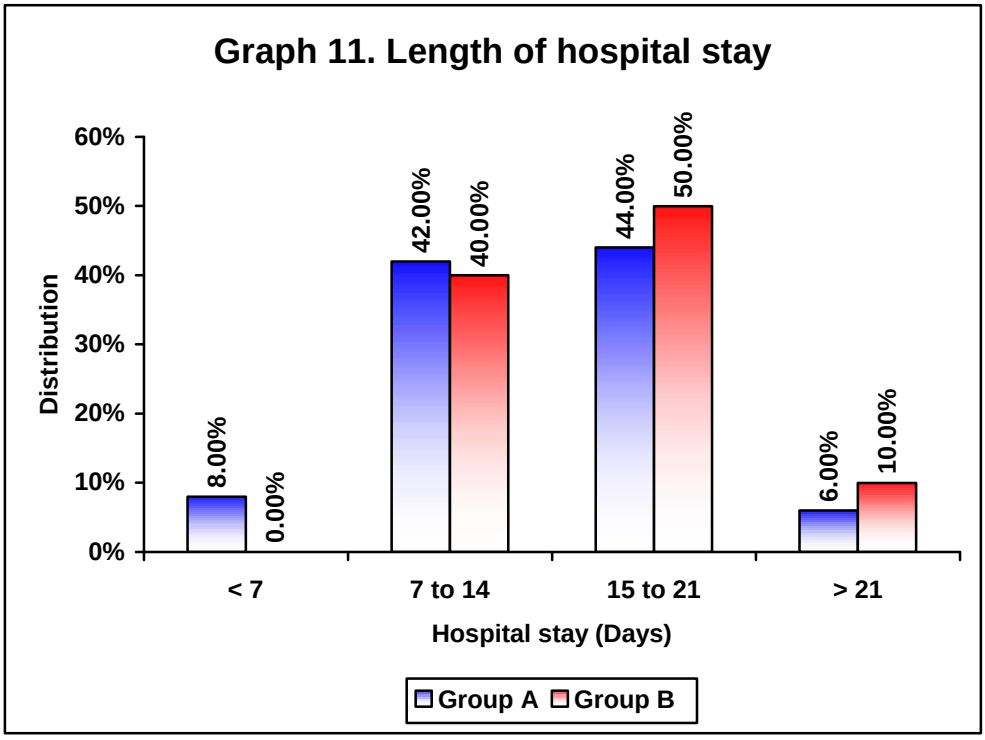


In this study on post operative day 15, most of the patients in group A (86%) had grade 0 followed by 14% with grade I whereas, in group B 44% patients had grade 0 and 48% had grade I. Also, among 4% patients the wound grade was II in group B. This difference was statistically significant ($p<0.001$).

Table 11. Length of hospital stay

Hospital stay (Days)	Group A (n=50)		Group B (n=50)	
	Number	Percent	Number	Percent
< 7	4	8.00	0	0.00
7 to 14	21	42.00	20	40.00
15 to 21	22	44.00	25	50.00
> 21	3	6.00	5	10.00
Total	**50**	**100.00**	**50**	**100.00**

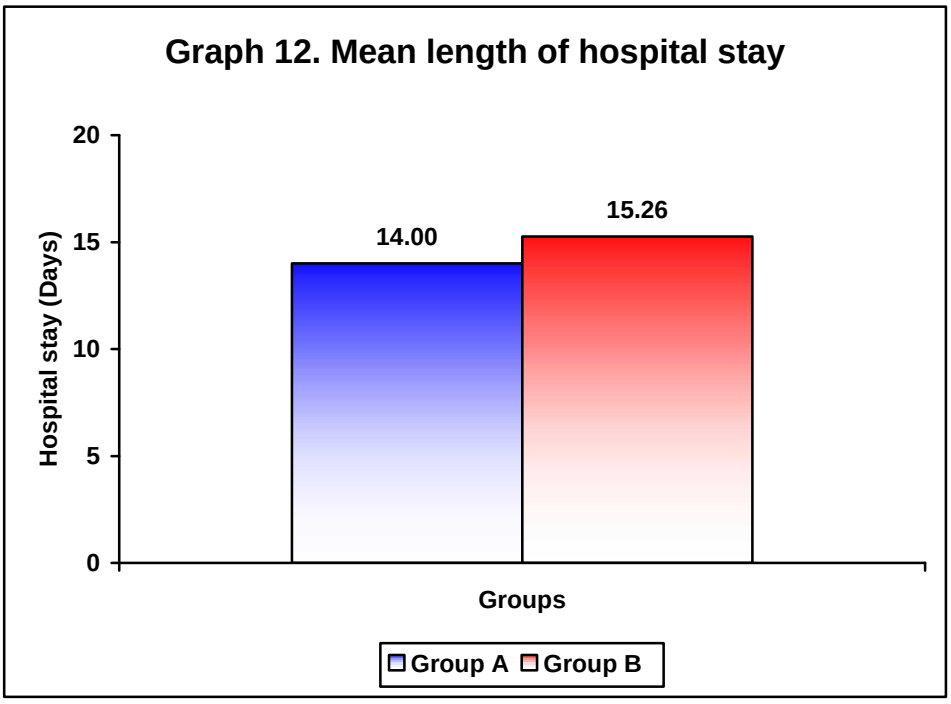
x^2_3= 4.777 **p = 0.194**



In this study most of the patients (44%) in group A had hospital stay between 15 to 21 days compared 50% in group B. However this difference was statistically not significant (p=0.194).

Table 12. Mean length of hospital stay

Duration (Days)	Group A (n=50)	Group B (n=50)
Mean	14.00	15.26
SD	4.94	4.60
Median	14.50	15.00
Minimum	5.00	8.00
Maximum	25.00	25.00
t = 0.826	**DF = 98**	**p = 0.411**



The mean length of hospital stay in group A was 14.38 ± 6.15 days compared to 15.26 ± 4.60 days. However this difference was not significant (p=0.411).

Chapter 6

Discussion

DISCUSSION

Despite the advances made in asepsis, antimicrobial drugs, sterilization and operative techniques, surgical site infections (SSI) continue to be a major problem in all branches of surgery in the hospitals.[60] They have been responsible for the increasing cost, morbidity and mortality related to surgical operations and continue to be a major problem even in hospitals with most modern facilities and standard protocols of preoperative preparation and antibiotic prophylaxis. A major 30%-50% of antimicrobials prescribed in hospital practice are for surgical prophylaxis to prevent post-operative wound infection. A reduction in the infection rate to a minimal level could have significant benefits in terms of both patient comfort and medical resources used.[61]

Most surgical site infections are related to suture. The superficial and deep reached 90% of SSI. Although efficacy against surgical infection: a strict aseptic technique, antibiotic coverage (when indicated), and an adequate surgical technique were applied, Infection rate remains high.[10]

Recently a new antimicrobial suture Polyglactin 910 coated with tricosan (Vicryl Plus) has been introduced. Tricosan is a broad spectrum antiseptic that has been widely used in humans for over 30 years.[10] The present study was undertaken to evaluate the efficacy of new antibacterial suture (Vicryl Plus) compared with a traditional suture (Vicryl) in reducing abdominal surgical site infection.

This one year randomized controlled trial was conducted in Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre,

Belgaum between January 2011 to December 2011 on 100 patients undergoing abdominal surgeries. These patients were randomized into two groups based on computer generated blocked random numbers namely Group A (Vicryl plus) and Group B (Conventional vicryl).

In this study overall, out of 100 patients, 67 were males and 33 were female. In the group A 76% were males and 28% were females. In group A 62% were males and 38% were females. The most common age group in group A and B was 46 to 50 years (32% each) and in group A the same proportion of patients had age between 31 to 45 years. The mean age in Group A was 45.74 ± 15.21 and in group B mean age was 49.08 ± 16.71 years suggesting the demographic characteristics of the study population were comparable in both the groups ($p>0.05$).

In the present study among more than half (52% vs 54%) of patients the duration of surgery was between 61 to 120 minutes in both the groups. The mean duration of surgery was 138.20 ± 39.37 in group A and 136.00 ± 44.61 minutes suggesting the duration of surgery in group A and B were comparable ($p=0.849$).

In present study according to wound classification commonly used, in group A the number of clean cases were 12 whereas in group B it was 8, in clean contaminated grade, maximum no of cases were there, group A had 31 cases whereas in group B had 34 cases. Small no of cases were in dirty grade in which group A had 7 subjects and group B had 8. In both the groups the numbers were comparable suggesting that there was no significant difference in class of wound

in two groups ruling out the possibility of bias which were similar to other study[40] performed in Germany.

In the present study on post operative day three, five, seven and fifteen significantly higher number of patients in group A showed better wound assessment grades. Most of the patients (60%) in group A had grade I followed by 28% with grade II and 10% with grade 0 and 2% with grade III whereas, in group B 48% of the patients had grade II, 40% had grade I and 12% had grade III wound assessment grades. None of the patient in group B had grade 0 (p=0.004).

In this study on post operative day three significantly higher number of patients (60%) in group A had grade I followed by 28% with grade II and 10% with grade 0 and 2% with grade III whereas, in group B 48% of the patients had grade II, 40% had grade I and 12% had grade III wound assessment grades. None of the patient in group B had grade 0 and this difference was statistically significant (p=0.004).

In the present study on post operative Day five, more than half (54%) of the patients in group A had grade I followed by 28% with grade 0 and 16% with grade II and 2% with grade III compared to 48% with grade I, 36% with grade II, 12% with grade III and 2% with grade 0 in group B (p=0.002).

In the present study on post operative Day seven, most of the patients in group A (62%) had grade 0 followed by 30% with grade I and 6% with grade II and 2% with grade III. In group B more than half (56%) had grade I followed by 16% each with grade 0 and II and 10% with grade III. This difference was statistically significant (p<0.001).

In this study on post operative Day 15, most of the patients in group A (86%) had grade 0 followed by 14% with grade I whereas, in group B 44% patients had grade 0 and 48% had grade I. Also, among 4% patients the wound grade was II in group B. This difference was statistically significant ($p<0.001$).

In this study most of the patients (44%) in group A had hospital stay between 15 to 21 days compared 50% in group B ($p=0.194$). The mean length of hospital stay in group A was 14.00 ± 4.94 days compared to 15.26 ± 4.60 days. This is mainly because most patients wanted to stay in hospital till sutures were removed which was usually done after post op day 10.

In a study[52] from Vienna, Austria authors compared post op infection rates in sternal wounds closed with vicryl plus with conventional vicryl, results were in conventional vicryl 24 pts had wound infection whereas in vicryl plus group none of patient had wound infection.

A similar study[40] from Germany compared reduction in wound infections by using Vicryl plus Vs PDS for midline laparotomy wounds. In PDS II loop suture for abdominal wall closure, of the 113 patients, 10.8% of midline laparotomy with wound infections were detected. The number of patients with wound infections decreased ($p<0.001$) in group using Vicryl plus for abdominal wall closure to 51 (4.9%) of midline laparotomy, results of this are similar to our study.

In another study[62] from Cairo University Hospital, Egypt, aimed to assess the incidence of surgical site infection using triclosan-coated polyglactin 910 antimicrobial suture (Vicryl Plus) compared with the conventional polyglactin

910 suture (Vicryl). The overall incidence of surgical site infection within the sample occurred in 50 of 450 patients enrolled in the study (11%). In the Vicryl Plus group the incidence of surgical site infection was 17 of 230 patients (7%) and in the control group it was 33 of 220 patients (15%). This difference was statistically significant (p=0.011), which was similar to the present study.

In 2009, a prospective, randomized, controlled, double blind, comparative, single center study[58] conducted to assess the efficacy of an antibacterial suture (polyglactin 910 coated with triclosan) compared to uncoated polyglactin 910 sutures in reducing rates of SSI in patients undergoing appendectomy. The rate of SSI was not statistically significantly different between the two treatment groups, nor was the complication rate after one year. The authors concluded that polyglactin 910 coated with triclosan was safe in surgical practice, with a comparable outcome to polyglactin 910.

A recent prospective study[59] evaluated the effect of triclosan-coated sutures on surgical wide excision of a head or neck cancer and reconstructive procedures. 241 patients were included in this study, divided into two groups. The Triclosan group contained 112 patients, whose surgical wounds were closed with Triclosan-coated sutures (Vicryl Plus). The control group included the remaining 129 patients, whose surgical wounds were closed with conventional Vicryl sutures. The results showed cervical wound infection rate was 14.9% (17/112) in the Triclosan group and 14.7% (19/129) in the control group, and these rates were not significantly different.

Chapter 7

Conclusion

CONCLUSION

Based on the findings of this study it may be concluded that, the use of new antibacterial suture (Vicryl Plus) showed better efficacy in reducing abdominal surgical site infections when compared to traditional suture (Vicryl).

Chapter 8

Summary

SUMMARY

Potential sources of infection are the patient (especially contamination by alimentary tract bacteria), hospital environment, food, other patients, staff, infected surgical instruments, dressings, and even drugs and injections. Surgical site infections are also related to suture. Recently a new antimicrobial suture Polyglactin 910 coated with tricosan (Vicryl Plus) has been introduced. Tricosan is a broad spectrum antiseptic that has been widely used in humans for over 30 years. The present study was undertaken to evaluate the efficacy of new antibacterial suture (Vicryl Plus) compared with a traditional suture (Vicryl) in reducing abdominal surgical site infection.

The present one year randomized controlled trial was conducted in Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum between January 2011 to December 2011. A total of 100 patients undergoing abdominal surgeries were included in the study. These patients were randomized into two groups based on computer generated blocked random numbers that is group A (Vicryl plus) and group B (Conventional vicryl).

In this series of 100 patients, 67 were males and 33 were female. Most of the patients (32%) were aged between 46 to 50 years in each group and in VP group the same proportion of patients had age between 31 to 45 years. The mean age in Group A was 45.74 ± 15.21 and in group B mean age was 49.08 ± 16.71. Among more than half (52% vs 54%) of patients the duration of surgery was between 61 to 120 minutes in both the groups. 70% of patients had clean contaminated wound compared to 80% in group B. The demographic

characteristics and surgical parameters such as type of wound, surgical time, were comparable in both the groups ($p>0.05$). In patients with group A, significantly lower incidence of abdominal surgical site infection was observed when compared to group B all the follow-ups that is day three, five, seven and fifteen ($p<0.05$). The mean length of hospital stay in group A was 14.00 ± 4.94 days compared to 15.26 ± 4.60 days ($p>0.05$).

Overall, the study showed that, the use of new antibacterial suture (Vicryl Plus) showed better efficacy in reducing abdominal surgical site infections when compared to traditional suture (Vicryl).

Chapter 9

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Annexures

Annexure I

ANNEXURE I – CONSENT FORM

Mr / Mrs / Miss _____ we are requesting you to enrol yourself in study entitled, "**A RANDOMIZED CONTROLLED TRIAL TO COMPARE EFFICACY OF VICRYL PLUS VERSUS VICRYL IN REDUCING ABDOMINAL SURGICAL SITE INFECTION – A ONE YEAR STUDY"** is being conducted by Dr. ***** ******, post graduate in Surgery at Jawaharlal Nehru Medical College Belgaum, Karnataka. Under guidance of Dr. ***** ****** Professor, Department of Surgery, Jawaharlal Nehru Medical College, Belgaum, under KLE University, Belgaum.

Respected Sir/Madam, we request you to enrol yourself to participate in our study as you are eligible for participating in this study. During the study you will be asked some questions regarding your present complaints and your are suppose to answer to the best of your knowledge.

Your participation in research is voluntary. If you decide to participate you are free to withdraw at any time.

The purpose of research is to evaluate the efficacy of new antibacterial suture (Vicryl Plus) compared with a traditional suture (Vicryl) in reducing abdominal surgical site infection.

Procedure involved

If you agree to enrol yourself in my study, you will be interviewed regarding your present and past history then you will be clinically examined in detail and investigated accordingly. Computer generated blocked random

numbers will be used to assign the type of surgery to the patients that is, Group A or Group B. If you are in Group-A vicryl plus suture will be used and if you are in Group-B conventional vicryl suture will be used. In case of laparotomy by vertical incision, elective/emergency the closure of wound will be done in layers from inside out that is peritoneum using vicryl plus, rectus using PDS/prolene, subcutaneous layer with vicryl plus and skin using ethilon/staples. The Post-Operative care will be similar for both groups of participants. The wound will be assessed on post operative day three, five and seven and on first follow-up that is day 15.

Benefits and Risks

The benefits of taking part in this research are you will have reduced abdominal surgical site infection. There are no observable risks associated in this study.

Voluntary participation / Withdrawal

Taking part in the study is voluntary. You may choose not to enrol yourself in this study. Your decision will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Alternatives

Even if you decline the participation in the study, you will get the routine line of management.

Privacy and confidentiality

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except: In emergency to protect your rights and welfare and if required by law.

Authorization to Publish Results

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

Financial Incentives for participation

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator.

Compensation

In the event of injury, related to the study, treatment will be made available at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. There is no compensation or payment for such medical treatment by law.

Questions/Contact details

If you have any queries, in future or in case of study related injury or illness, you may contact. Dr. ***** ****** at Department of Surgery, KLES Dr.

Prabhakar Kore Hospital and Medical Research Centre, Belgaum Phone Number **** ******* or on ***** *****.

If you have any queries about your rights as a study subject, you may call Principal and Chairman, J. N. Medical College Institutional Ethical Committee for Human Subjects Research, Ph. **** ****** at J. N. Medical College, Belgaum.

CONSENT TO PARTICIPATE IN A RESEARCH STUDY:

I, Mr./Mrs. _____

voluntarily agree to take part in this study, by signing this consent form I am not giving up my legal rights. I may withdraw at any time. I am signing after having read, or been read to me in the vernacular language including risks and the benefits and having all queries cleared.

Subject Name: _____

Signature of the participant _____ Date _____
Or Left thumb print

Witness name: _____

Signature: _____ Date _____

Investigator's name: _____

Signature: _____ Date _____

Place: _____

Annexures

Annexure II

ANNEXURE II – PROFORMA

STUDY: A RANDOMIZED CONTROLLED TRIAL TO COMPARE EFFICACY OF VICRYL PLUS VERSUS VICRYL IN REDUCING ABDOMINAL SURGICAL SITE INFECTION – A ONE YEAR STUDY

PATIENT DETAILS

Name : IP No :

Sex : Age :

Date of admission : Date of Surgery : Address :

HISTORY

Chief Complaints

EXAMINATION

General Examination

Built and Nourishment: ***Weight:***

Pallor/Icterus/Cyanosis/Clubbing/Edema/Lymphadenopathy

Vitals

Pulse Rate : /Min B.P. : mm Hg

Temperature : ^{0}F R. R. : /Min

Systemic Examination

Respiratory :

CVS :

CNS :

Local examination

Per abdomen :

INVESTIGATIONS

Blood - Routine : Hb: Total count:

Platelet:

Random blood sugar :

Blood urea. :

Sr. Creatinine. :

Bleeding time :

Clotting time :

Urine routine and microscopy

OPERATION DETAILS

Group : Group A / Group B

Date of surgery :

Procedure done :

Anaesthesia :

Duration of the surgery :

Assessment of wound

Grades		Appearance	Interval			
			Day 3	**Day 5**	**Day 7**	**Day 15**
0		**Normal healing**				
I		**Normal healing with mild bruising or erythema**				
	A	Same bruising				
	B	Considerable bruising				
	C	Mild erythema				
II		**Erythema plus other signs of inflammation**				
	A	At one point				
	B	Around sutures				
	C	Along wound				
	D	Around wound				
III		**Clear or haemoserous discharge**				
	A	At one point only (< 2 cm)				
	B	Along wound (> 2 cm)				
	C	Large volume				
	D	Prolonged (> 3days)				
IV		**Major complications**				
	A	At one point (< 2 cms)				
	B	Along wound (> 2 cm)				
V		**Deep severe wound infection with or without tissue breakdown; haematoma requiring aspiration**				

Outcome :

Annexures

Annexure III

ANNEXURE III – PHOTOGRAPHS

Photograph 1. Suture material

Photograph 2. Suture material

Photograph 3. Patient with wound Grade 0

Photograph 4. Patient with wound Grade I

Photograph 5. Patient with wound Grade II

Photograph 6. Patient with wound Grade IV

ANNEXURE IV - MASTER CHART GROUP A

Serial Number	In patient Number	Sex	Age (Years)	Date of admission	History	Diagnosis	Operation Details				Assessment of wound			
							Date of Surgery	Procedure	Type of wound	Duration of surgery (min)	Day 3	Day 5	Day 7	Day 15
1	4E+05	M	33	#########	Abdominal distesion	Hollow viscus perforation	#########	Exploratory Laprotomy With Segmental Resection And Anastomosis	CC	120	2C	1C	0	0
2	4E+05	F	45	#########	Pain abdomen	Hollow viscus perforation	#########	Primary Closure Of Perforation	D	180	1C	1A	0	0
3	4E+05	M	40	#########	Mass per rectum	Complete rectal prolapse	#########	Abdominal Rectopexy	CC	150	1C	1C	0	0
4	4E+05	M	40	#########	Abdominal distension	Gastric outlet obstruction	#########	Gastrojejunostomy	CC	90	1A	1A	0	0
5	4E+05	F	70	#########	Pain abdomen, vomiting	Cholecystitis	#########	Cholecystectomy	CC	120	1A	0	0	0
6	4E+05	M	45	#########	Pain abdomen since morning	Appendicular perforation	#########	Exploratory Laprotomy With Primary Repair	D	150	1C	1A	1A	0
7	4E+05	M	27	#########	Stab injury	Hollow viscus perforation	#########	Exploratory Laprotomy With Resection Of Part Of Ieum And Anastomosis	CC	180	1A	1A	0	0
8	4E+05	M	50	#########	Swelling	Paraumbilical hernia	#########	Repair Of Hernia	C	100	0	0	0	0
9	4E+05	M	40	#########	pain abdomen since morning	Hollow viscus perforation	#########	Exp Laprotomy With Primary Closure With Omental Patch	D	90	1A	0	0	0
10	4E+05	F	28	#########	Splenomegaly	Tropical splenomegaly	#########	Splenectomy	C	150	1A	0	0	0
11	4E+05	M	54	#########	pain abdomen with septic shock	Hollow viscus perforation	#########	Laprotomy With Primary Closure Of Perforation	D	120	2A	1C	1A	1A
12	4E+05	M	61	#########	Mass per abdomen	Pseudocyst pancreas	#########	Cystogastrostomy	CC	100	1A	0	0	0
13	4E+05	M	68	#########	pain abdomen, abdominal distention	Ca colon	#########	Left Hemicolectomy Transverse Colon Sigmoid Colon Anastomosis	CC	150	1C	1A	1A	0
14	4E+05	M	19	#########	pain abdomen, loss of appetite	Chronic pancreatitis	#########	Modified Pusteows Procedure	CC	180	1A	1A	0	0
15	4E+05	F	21	#########	Pain abdomen	Appendicular perforation	#########	Laprotomy With Drainage Of Pelvic Abscess	CC	120	2A	2A	1A	1A
16	4E+05	F	41	#########	Pain abdomen	Adhesions	#########	Exploratory Laprotomy With Drainage Of Pelvic Abscess With Left Oopherectomy	CC	90	2A	2A	1C	1A
17	4E+05	M	24	#########	Pain abdomen, vomiting	Ca colon	#########	Right Hemicolectomy	CC	200	1C	1A	0	0
18	4E+05	M	55	#########	Multiple swab injuries in abdomen	Hollow viscus perforation	#########	Exp Laprotomy With Resection Anastomosis	CC	180	2A	2A	1A	0
19	4E+05	M	49	#########	Mass per abdomen, vomiting	Ca stomach	#########	Partial Gastrectomy With Gastrojejunostomy	CC	150	2A	1A	0	0
20	4E+05	M	52	#########	Pain abdomen, distention, passing malena	Gangrene bowel	#########	Exp Laprotomy With Resection Anastomosis	CC	150	2A	1A	1A	0
21	4E+05	M	52	#########	Pain abdomen, abdominal distention	Hollow viscus perforation	#########	Exp Laprotomy With Primary Closure Of Perforation	D	120	2A	1C	1C	1A
22	4E+05	M	45	#########	Pain abdomen, abdominal distention	Gangrene bowel	#########	Exp Laprotomy With Resection Anastomosis	CC	150	3A	2A	2A	1C
23	4E+05	F	38	#########	Abdominal distention, vomiting	Jejunal stricture	#########	Exp Laprotomy With Resection Anastomosis	CC	120	1A	1A	0	0
24	4E+05	M	18	#########	Pain abdomen, vomiting	Lt paraduodenal hernia	#########	Repair Of Internal Hernia	C	100	1A	0	0	0
25	4E+05	M	50	#########	Pain abdomen, vomiting	Intestinal obstruction	#########	Exp Laprotomy With Adhesionlysis	CC	90	1A	1A	1A	0
26	4E+05	F	55	#########	Pain abdomen	Adhesions	#########	Exp Laprotomy With Multiple Perforation Closure With Resection Anastomosis	CC	200	2D	2D	1C	0
27	4E+05	M	57	#########	Swelling	Paraumbilical hernia	#########	Umbilical Hernia Repiar	C	100	1C	0	0	0
28	4E+05	M	30	#########	Pain abdomen	Hollow viscus perforation	#########	Exp Laprotomy With Primary Repair Of Jejunal Perforation	D	90	2B	1C	0	0
29	4E+05	M	80	#########	Pain abdomen	Colo colic intususception	#########	Exp Laprotomy With Transverse Sigmoid Colon Anastomosis	CC	200	1A	1A	0	0
30	4E+05	M	50	#########	Hematemesis	Portal hypertension bleeding varices	#########	Devascularisation Of Feeding Vessels	C	180	1A	0	0	0
31	4E+05	F	57	#########	Swelling	Incisional hernia	#########	Excision Of Sac With Primary Repair And Meshplasty	C	120	2A	3A	3A	1A
32	4E+05	M	48	#########	Pain abdomen, jaundice	Choledocolithiasis	#########	Exp Laprotomy With Partial Cholecystectomy With Cbd Stenting	CC	180	1C	2B	2B	1A

ANNEXURE IV - MASTER CHART GROUP A

Serial Number	In patient Number	Sex	Age (Years)	Date of admission	History	Diagnosis	Operation Details				Assessment of wound			
							Date of Surgery	Procedure	Type of wound	Duration of surgery (min)	Day 3	Day 5	Day 7	Day 15
33	4E+05	F	45	#########	Pain abdomen, vomiting	Ca stomach	#########	Anterior Gastrojejunostomy	CC	120	1A	1A	0	0
34	4E+05	M	35	#########	Abdominal distension, Ca rectum	Intestinal obstruction	#########	Exp Laprotomy With Adhesionlysis With Ileostomy	CC	200	1A	2A	2A	0
35	4E+05	M	42	#########	Swelling	Paraumbilical hernia	#########	Excision Of Sac With Primary Repair And Meshplasty	C	90	0	0	0	0
36	4E+05	F	70	#########	Swelling	Incisional hernia	#########	Excision Of Sac With Primary Repair And Meshplasty	C	100	0	0	0	0
37	4E+05	F	65	#########	Pain abdomen, vomiting	Intestinal obstruction	#########	Lap Adhesionlysis, Exp Laprotomy With Resection Anastomosis	CC	180	0	0	0	0
38	4E+05	M	50	#########	Pain abdomen, jaundice	Choledocolithiasis	#########	Cholecystojejunostomy With Feeding Jejunostomy	CC	180	1A	1A	1A	0
39	4E+05	M	67	#########	Swelling	Paraumbilical hernia	#########	Excision Of Sac With Primary Repair And Meshplasty	C	90	1A	1A	0	0
40	4E+05	M	42	#########	Difficulty in swallowing	Ca esophagus	#########	Ivor Lewis Operation	CC	200	1A	1A	0	0
41	4E+05	M	63	#########	Hematemesis	Portal hypertension bleeding varices	#########	Devascularisation Of Feeding Vessels	C	180	1A	1B	0	0
42	4E+05	F	55	#########	Difficulty in swallowing	Ca esophagus	#########	Feeding Jejunostomy	CC	90	1A	1A	0	0
43	4E+05	F	45	#########	Swelling	Incisional hernia	#########	Excision Of Sac With Primary Repair And Meshplasty	C	100	1A	0	0	0
44	4E+05	M	18	#########	Mass per abdomen	ITP	#########	Splenectomy	C	120	0	0	0	0
45	4E+05	M	55	#########	Intestinal obstruction, Ca bladder	Intestinal obstruction	#########	Exp Laprotomy Withresection And Ileotransverse Anastomosis	CC	150	1C	1A	1A	0
46	4E+05	M	71	#########	Pain abdomen	Hollow viscus perforation	#########	Exp Laprotomy With Primary Closure Of Perforation	D	100	2A	1A	1A	0
47	4E+05	M	38	#########	Pain abdomen	Appendicular perforation	#########	Exp Laprotomy With Appendectomy	CC	90	2A	2A	1A	0
48	4E+05	M	59	#########	Pain abdomen	Empyema gall bladder	#########	Appendectomy And Clolecystectomy	CC	200	2A	1A	1A	0
49	4E+05	M	67	#########	Pain abdomen, abdominal distention	Intestinal obstruction	#########	Exp Laprotomy With Adhesionlysis	CC	120	1A	1A	0	0
50	4E+05	F	38	#########	Pain abdomen	Appendicitis, Meckel's diverticulum	#########	Exp Laprotomy With Appendectomy With Excision Of Meckels	CC	180	1A	0	0	0

ANNEXURE IV - MASTER CHART GROUP A

Duration of hospital stay (Days)
10
10
12
10
10
10
20
10
10
15
18
15
18
18
15
22
15
18
10
20
18
15
10
10
12
15
7
7
15
22
10
20

ANNEXURE IV - MASTER CHART GROUP A

Duration of hospital stay (Days)
12
25
5
8
18
18
8
20
20
12
7
18
20
10
8
20
14
10

ANNEXURE IV - MASTER CHART GROUP B

Serial Number	In patient Number	Sex	Age (Years)	Date of admission	History	Diagnosis	Operation Details				Assessment of wound			
							Date of Surgery	Procedure	Type of wound	Duration of surgery (min)	Day 3	Day 5	Day 7	Day 15
1	4E+05	M	29	#########	Pain abdomen	Appendicular perforation	#########	Ileo Transverse Anastomosis	CC	160	2B	3B	3B	1A
2	4E+05	M	60	#########	Pain abdomen	Hollow viscus perforation	#########	Primary Closure Of Perforation	D	120	3A	2A	1A	0
3	4E+05	M	60	#########	Blunt injury abdomen	Hollow viscus perforation	#########	Splenectomy And Left Hemicolectomy	CC	240	2B	3B	4B	5
4	4E+05	M	50	#########	Pain abdomen, vomiting	Ca stomach	#########	Gastrojejunostomy	CC	90	1A	1A	1A	0
5	4E+05	M	40	#########	Pain abdomen	Cholecystitis	#########	Cholecystectomy	CC	90	2A	1A	1A	0
6	4E+05	M	50	#########	Pain abdomen	Hollow viscus perforation	#########	Exploratory Laprotomy With Primary Closure	D	120	2B	2A	1C	1A
7	4E+05	F	45	#########	Pain abdomen	Empyema gall bladder	#########	Open Cholecystectomy	CC	150	1A	1A	0	0
8	4E+05	F	50	#########	Pain abdomen, abdominal distention	Hollow viscus perforation	#########	Exploratory Laprotomy With Perforation Closure With Omental Patch	D	120	2A	1A	1A	1A
9	4E+05	M	65	#########	Abdominal distention, vomiting	Intestinal obstruction	#########	Exp Laprotomy With Adhesionolysis	C	100	1A	1A	0	0
10	4E+05	F	28	#########	Bowel injury	Adhesions	#########	Diagnostic Laprotomy With Adhesionlysis	C	60	1A	1A	1A	0
11	4E+05	M	80	#########	Pain abdomen, vomiting	Ca stomach	#########	Anterior Gastrojejunostomy	CC	90	1A	1A	0	0
12	4E+05	F	45	#########	Pain abdomen, abdominal distention	Ca Head pacreas	#########	Triple Bypass	CC	240	1A	1A	1A	0
13	4E+05	M	65	#########	Pain abdomen, vomiting	Gastric outlet obstruction	#########	Truncal Vagotomy With Posterior Gj With Cholecystectomy	CC	180	1C	1C	1A	0
14	4E+05	M	60	#########	pain abdomen persistant vomiting,	Gastric outlet obstruction	#########	Cholecystectomy With Posterior Gj With Incisional Hernia	CC	240	1A	1A	1A	0
15	4E+05	M	35	#########	Pain abdomen, abdominal distention	Gangrene bowel	#########	exploratory laprotomy with resection & end to end anastomosis with orchidopexy	CC	180	3	2A	2A	1C
16	4E+05	M	70	#########	Vomiting, weight loss	Ca Head pacreas	#########	Gastrojejunostomy	CC	90	1C	1A	1A	0
17	3E+05	M	71	#########	Mass per abdomen	Mesentric cyst	#########	Exploratory Laprotomy And Cyst Excision	C	90	1A	1A	0	0
18	4E+05	F	56	#########	Pain abdomen, abdominal distention	Hollow viscus perforation	#########	Exp Laprotomy With Primary Closure Of Perforation	D	120	3A	2C	2A	1A
19	4E+05	M	23	#########	Pain abdomen, abdominal distention	Intestinal obstruction	#########	Exp Laprotomy With Adhesionolysis	CC	180	2A	2A	1C	1A
20	4E+05	M	46	#########	Pain abdomen, abdominal distention	Hollow viscus perforation	#########	Exp Laprotomy With Primary Closure Of Perforation With Feeding Jejunostomy	D	150	2C	2A	2A	1A
21	4E+05	M	75	#########	Pain abdomen, abdominal distention	Hollow viscus perforation	#########	Exp Laprotomy With Primary Closure Of Perforation	D	100	2A	2A	1C	1A
22	4E+05	M	55	#########	Pain abdomen, abdominal distention	Gangrene bowel	#########	Exp Laprotomy With Resection Anastomosis	CC	180	3A	3A	2C	2A
23	4E+05	M	49	#########	pain abdomen with jaundice	Ca Head pacreas	#########	Exp Laprotomy With Posterior Gastrojejunostomy	CC	150	2A	1C	1A	1A
24	4E+05	M	20	#########	Pain abdomen	Hollow viscus perforation	#########	Exp Laprotomy With Primary Closure Of Perforation	D	90	1A	1A	1A	0
25	4E+05	F	33	#########	Pain abdomen, vomiting	Gastric outlet obstruction	#########	Gastrojejunostomy With Vagotomy	CC	100	2A	1A	1A	0
26	4E+05	M	69	#########	Mass per abdomen, vomiting	Gastric outlet obstruction	#########	Gastrojejunostomy	CC	120	1C	1A	1A	0
27	4E+05	M	48	#########	Abdominal distention, vomiting, malena	Gangrene bowel	#########	Exp Laprotomy With Resection Anastomosis	CC	180	3A	2C	2A	1A
28	4E+05	F	48	#########	Pain abdomen	Hollow viscus perforation	#########	Exp Laprotomy With Resection Anastomosis	CC	120	2A	2A	1C	0
29	4E+05	F	82	#########	Pain abdomen, mass per abdomen	Ca colon	#########	Right Hemicolectomy	CC	180	2C	2A	2A	1A
30	4E+05	M	27	#########	Pain abdomen, blunt injury	Hollow viscus perforation	#########	Exp Laprotomy With Repair Of Multiple Jejunal Perforation	CC	200	3C	3A	2C	1A
31	4E+05	M	65	#########	Blunt injury abdomen	Hollow viscus perforation	#########	Splenectomy	C	120	2B	1C	1B	1B
32	4E+05	F	63	#########	Blunt injury abdomen	Appendicular perforation	#########	Exploratory Laprotomy With Appendectomy	CC	90	2B	2B	1C	0

ANNEXURE IV - MASTER CHART GROUP B

Serial Number	In patient Number	Sex	Age (Years)	Date of admission	History	Diagnosis	Operation Details				Assessment of wound			
							Date of Surgery	Procedure	Type of wound	Duration of surgery (min)	Day 3	Day 5	Day 7	Day 15
33	4E+05	M	38	#########	Blunt injury abdomen	Hollow viscus perforation	#########	Exp Laprotomy With Resection Anastomosis Of Small Bowel And Sigmoid Colostomy	CC	200	2D	3B	5	5
34	4E+05	M	65	#########	Pain abdomen, vomiting	Empyema gall bladder	#########	Exp Laprotomy With Cholecystectomy	CC	180	2D	1C	1A	1A
35	4E+05	M	55	#########	Pain abdomen, vomiting	Gastric outlet obstruction	#########	Exp Laprotomy With Distal Gastrectomy (Bilroth 1)	CC	100	2C	5	3B	1C
36	4E+05	F	28	#########	Rt iliac fossa mass	Rt iliac fossa mass	#########	Exp Laprotomy With Excision Of Rt Iliac Fossa Mass	CC	90	2A	3A	3A	2A
37	4E+05	F	50	#########	Pain abdomen, Carcinoma Ca	Hollow viscus perforation	#########	Exp Laprotomy With Resectio And Ileoileal Anastomosis	CC	120	1A	1A	0	0
38	4E+05	M	42	#########	Mass per abdomen	Splenic abscess	#########	Exploratory Laprotomy With Splenectomy	C	120	1B	1A	1A	0
39	4E+05	F	40	#########	Swelling	Paraumbilical hernia	#########	Excision Of Sac With Primary Repair And Meshplasty	C	90	1A	1A	0	0
40	4E+05	F	25	#########	Pain abdomen	Gangrene bowel	#########	Segemental Resection Of Caecum And Ileoasc Colon Anastomisis	CC	150	1A	2A	3A	1A
41	5E+05	F	50	#########	Blunt injury abdomen	Splenic laceration	#########	Exp Laprotomy With Splenectomy	C	90	1A	0	0	0
42	4E+05	M	22	#########	Pain abdomen	Appendicular perforation	#########	Exp Laprotomy With Appendectomy	CC	120	1A	1A	0	0
43	4E+05	F	45	#########	pain abdomen with jaundice	Choledocolithiasis	#########	Choledocoduodenostomy With Appendectomy	CC	150	1A	2A	1C	1A
44	4E+05	F	65	#########	Swelling	Paraumbilical hernia	#########	Excision Of Sac With Primary Repair And Meshplasty	C	100	1A	1B	1A	1A
45	4E+05	M	27	#########	Cecal perforation, iliostomy	Ileostomy	#########	Ileostomy Closure	CC	100	2A	1A	1A	1A
46	4E+05	F	54	#########	Mass per abdomen	Ca colon	#########	Exp Laprotomy With Resection And Ileotransverse Anastomosis	CC	120	2A	2A	1A	1A
47	4E+05	M	31	#########	Painful swelling, inguinal hernia	Gangrene bowel	#########	Exp Laprotomy With Resection And Ileotransverse Anastomosis	CC	180	2A	2A	1C	1A
48	4E+05	M	35	#########	Abdominal distension, vomiting	Intestinal obstruction	#########	Exp Laprotomy With Resection Anastomosis	CC	150	2A	2A	1A	1A
49	4E+05	F	82	#########	Abdominal distension	Intestinal obstruction	#########	Exp Laprotomy With Loop Colostomy	CC	150	2A	2A	1C	1A
50	4E+05	F	38	#########	Pain abdomen	Hollow viscus perforation	#########	Exp Laparotomy With Closure Of Perforation	D	180	2A	2A	2A	1A

ANNEXURE IV - MASTER CHART GROUP B

Duration of hospital stay (Days)
20
8
25
15
8
15
15
12
18
15
12
20
20
20
24
15
10
15
12
22
12
12
15
10
10
15
20
10
18
20
15
12

ANNEXURE IV - MASTER CHART GROUP B

Duration of hospital stay (Days)
25
20
20
15
13
12
8
12
20
10
15
10
15
18
10
12
22
16

Annexures

Annexure IV

ANNEXURE IV – MASTER CHART

A	- Group A (Vicryl plus)
B	- Group B (Vicryl)
C	- Clean
Ca	- Carcinoma
CC	- Clean Contaminated
D	- Dirty
F	- Female
GA	- General Anaesthesia
GJ	- GastroJejunostomy
ITP	- Idiopathic Thrombocytopenic Purpura
Lap	- Laproscopic
M	- Male
min	- Minutes
Op	- Operative
SMA	- Superior Mesentric Artery