"ONE YEAR RANDOMIZED CONTROLLED TRIAL TO COMPARE THE EFFECTIVENESS OF TRICLOSAN COATED PDS PLUS VERSUS UNCOATED PDS SUTURES IN PREVENTION OF SURGICAL SITE INFECTIONS AFTER MONOLAYER CLOSURE IN OPEN ABDOMINAL SURGERIES"

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ENDORSEMENT

This is to certify that the dissertation entitled **"ONE YEAR RANDOMIZED CONTROLLED TRIAL TO COMPARE THE EFFECTIVENESS OF TRICLOSAN COATED PDS PLUS VERSUS UNCOATED PDS SUTURES IN PREVENTION OF SURGICAL SITE INFECTIONS AFTER MONOLAYER CLOSURE IN OPEN ABDOMINAL SURGERIES"** is a bonafide research work done by **CANDIDATE REG NO. BH0113011**.

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LIST OF ABBREVIATIONS USED

/min	-	Per minute
AD	-	Anno Domini
BC	-	Before Christ
BP	-	Blood pressure
CDC	-	Centers for Disease Control
CFU	-	Colony-forming units
CI	-	Confidence interval
E. coli	-	*Escherichia coli*
e.g.,	-	For example,
ENR	-	Enoyl-acyl carrier protein reductase
ENT	-	Ear Nose Throat
EPS	-	Extracellular polymeric material
ESBL	-	Extended-spectrum beta-lactamase
etc.	-	Etcetera
FDA (US)	-	Food and Drug Administration (United States)
FDA	-	Food and Drug Administration
gm	-	Grams
HAI	-	Healthcare-associated infection
HIV	-	Human immunodeficiency virus
i.e.,	-	That is,
IL	-	Interleukin
Inj.	-	Injection
IV	-	Intravenous
K. pneumoniae	-	*Klebsiella pneumoniae*

LD50	-	Lethal dose 50
LTI	-	Left thumb impression
m	-	Meters
mg/kg	-	Milligram per kilogram
mL	-	Milliliter
mm Hg	-	Milimeters of mercury
MRSA	-	Methicillin-resistant *Staphylococcus aureus*
MRSE	-	*Methicillin resistant Staphylococcus epidermidis*
n	-	Total number
NHSN	-	National Health Care Safety Network
NNIS	-	National Nosocomial Infections Surveillance
NOEL	-	No observed effect level
NSAIDs	-	Non steroidal anti-inflammatory drugs
NTCS	-	Non triclosan coated suture
OR	-	Odd's ratio
p	-	Probability
PDS	-	Polydioxanone suture
PHMB	-	Polyhexamethylene biguanide
PR	-	Pulse rate
PRISMA	-	Preferred Reporting Items for Systematic reviews and
RCTs	-	Randomized controlled trials
RR	-	Respiratory rate
S. epidermidis	-	*Staphylococcus epidermidis*
SA	-	*Staphylococcus aureus*
SD	-	Standard deviation

SLR	-	Systematic Literature Review
SRC	-	Suture-related complications
SSI	-	Surgical site infection
TC	-	Total count
TCS	-	Triclosan coated suture
Temp	-	Temperature
TNF	-	Tumor necrosis factor
U.S.	-	United States
vs.	-	Versus
WHO	-	World Health Organization
µg/m	-	Microgram per meter

ABSTRACT

Background and objectives

Surgical site infections remain one of the most frequent complications in open abdominal surgery. This study compared the effectiveness of Triclosan coated PDS Plus and non coated PDS II after monolayer abdominal fascia closure in preventing SSI in open abdominal surgeries.

Methodology

The randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum for the period of one year from January 2014 to December 2014 were studied. A total of 60 patients undergoing open abdominal surgeries (Clean contaminated) were enrolled. These patients were divided into two groups of 30 each based on suture material used to close the abdominal fascia in monolayer as Group A (PDS II sutures) and Group B (PDS plus sutures).

Results

Most of the patients were males in group A and B (63.33%) with males to female ratio of 1.72:1 (p=1.000). Nearly half of the study population (50%) was aged ≤ 30 years in group A compared to 33.33% in group B (p=0.262). The mean age in group A and group B was comparable (39.90±11.67 vs 41.67±16.08 years; p=0.687). Other pre-intervention characteristics including fever, vomiting, tenderness, vitals and type of surgery were comparable (p>0.050). On post operative day two, discharge was noted in 19 (63.33%) patients in group A and culture was positive in 9 (81.82%) of the patients with commonest organism

being Escherichia coli and pseudomonas aeruginosa (3 patients each [27.27%]). In group B, discharged was noted in 21 (70%) patients of which culture was positive in 7 (77.78%) patients with pseudomonas aeruginosa and commonest organism (22.22%). However these findings were comparable in group A and B ($p>0.050$). Similar trend was noted on day six as well as day ten. The other wound characteristics including pain, local tenderness, redness, raised local temperature, fever were comparable in group A and group B on day two as well as day six and ten ($p=0.050$). The surgical site infections were present in 43.33% of the patients in group A compared to 30% of the patients in group B ($p=0.284$).

Conclusion and interpretation

Abdominal fascia closure with Triclosan coated PDS Plus sutures is as effective as non-coated PDS sutures after monolayer closure in clean contaminated open abdominal surgeries in the prevention of surgical site infections.

Keywords

Surgical site infections; Sutures; Triclosan;

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INTRODUCTION

Post operative wound infection is considered as surgeon's nightmare.[1] Surgical site infections (SSIs) are infections of tissues, organs, or spaces exposed by surgeons during performance of an invasive procedure and remain one of the most frequent complications in open abdominal surgery. This complication while seemingly infrequent adds to the morbidity and delays wound healing.[2]

It was not until late 1860s, after Joseph lister introduced the principles of antisepsis, that post operative infectious morbidity decreased substantially.[3] In 1992, the US Centers for Disease Control (CDC) revised its definition of 'wound infection creating the definition 'Surgical Site Infection' (SSI) The Centers for Disease Control and Prevention (CDC)[4] has classified Surgical site infections (SSI) into superficial or deep, or organ/space SSIs occuring within 30 days of surgery.

An estimated 2,342 million surgeries are performed annually resulting in more than 7 million complications worldwide.[5] According to the CDC healthcare-associated infection (HAI) prevalence survey, there were an estimated 157,500 surgical site infections associated with inpatient surgeries in 2011.[6] NHSN data for 2006-2008 (16,147 SSIs following 849,659 operative procedures) showed an overall SSI rate of 1.9%.[7] The incidence of SSIs with regard to abdominal surgical sites and operating conditions is between 1.5 to 3.7% for clean wounds, 3 to 4% for clean-contaminated wounds, 8.5% for contaminated wounds and as high as 28 to 40% for dirty-infected wounds. In India, various studies have reported high SSI incidence rates.[8]

Surgical site infection is a dangerous condition and a heavy burden on the patient and social health system. These infections lengthen hospital stay for an average up to seven days. 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.[9]

SSIs are associated with a twofold increased relative risk of in-hospital mortality[10] and over one third of postoperative deaths in patients with SSIs are attributable to the infection.[11] Furthermore, several studies have shown an increase in the length of hospital stay between 6–24 days.[12] The resulting direct costs have to be added to the indirect costs resulting in substantial expenses to the health care system and the society.[13]

The causes of SSIs are multifactorial, but, crucially, SSIs are preventable.[14] Patient-related factors such as comorbidities (e.g. Diabetes mellitus) or lifestyle habits (e.g. Smoking) have to be taken into account but are difficult to change once an intervention is needed Therefore further efforts on the surgeon's side are required to reduce the frequency of SSI.[15]

A wide variety of aerobic and anaerobic species of bacteria may be present, either single or in combination. The most frequent pathogens causing postoperative SSIs following abdominal surgery are endogenous pathogens from the skin or gastrointestinal tract.[3]

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. Surgical site infections are also related to suture.[9] 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.[16]

One important factor in the development of SSI is bacterial colonization of suture material, especially on braided sutures and around suture knots.[17] Microorganisms colonize the suture as it is passed through human tissue in the surgical wound, which then forms a "biofilm" that confers immunity from antimicrobial treatment and the immune system.[18] Once a biofilm is established, it is difficult to remove the organism and this potentiates the risk of developing an SSI. Hence, the idea of using sutures with antibacterial properties was a logical extension to minimize this risk.

Triclosan (2,2,4'-trichloro-2'-hydroxyphenyl ether) is synthetic broad-spectrum anti-microbial agent present in the market for more than 40 years, mainly in the personal care or consumer products. Currently, triclosan is found in variety of skin care or personal care products such as hand soaps, shower gels, mouth washes, deodorant soaps, toothpastes, etc. Use of triclosan in health care industry started in 1972, in surgical scrubs. It has also been used in other medical products such as hand scrubs, skin antiseptics, ointments, impregnated/coated catheters and sutures.[19]

However, there is scarcity of the literature on the role of Triclosan coated PDS plus suture in reducing surgical site infections in settings of open abdominal surgeries. Hence the present study was planned to compare the effectiveness of

Triclosan coated PDS Plus for abdominal fascia closure in preventing SSI compared to non-coated PDS sutures after monolayer closure in open abdominal surgeries.

OBJECTIVES

The objective of this study was to compare the effectiveness of Triclosan coated PDS Plus for abdominal fascia closure in preventing SSI compared to non-coated PDS sutures after monolayer closure in open abdominal surgeries.

REVIEW OF LITERATURE

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.[20,21]

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."[22]

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.[23]

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.[24]

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).[24]

Before the mid-19th century, surgical patients commonly developed postoperative “irritative fever,” followed by purulent drainage from their incisions, overwhelming sepsis, and often death.[3]

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 were responsible for death in the postoperative period.[25]

Lister placed carbolic acid into open fractures to sterilize the wound and to prevent sepsis and hence the need for amputation.[26] Lister’s work radically changed surgery from an activity associated with infection and death to a discipline that could eliminate suffering and prolong life.[3]

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.[27] 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.[26]

Alexander Fleming (microbiologist, London, 1881-1955) performed many of his bacteriological studies during World War I and is credited with the discovery of penicillin.[26]

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.[26]

Until the middle of the 19th century, surgery was largely feared because of the likelihood that life-threatening wound infections known as ‘hospital gangrene’ would result. In those times the majority of surgical incision sites became infected and mortality rates of 70–80% were not unusual for patients with deep or extensive infections.[28]

Preventing and treating wound infections must have been difficult before the causes of infection were understood. The recognition by Robert Koch in 1876 that infectious agents caused infectious diseases stimulated early microbiologists to

isolate and characterize many pathogens and the most common causes of wound infection were known before the end of the 19th century. In conflict zones, where traumatic injuries frequently occur, preventing and treating wound infection has always been important. Military surgeons during the American Civil War used tincture of iodine in the treatment of contaminated traumatic wounds. The development of the concept of aseptic surgery with the liberal use of carbolic acid by Joseph Lister dramatically reduced surgical infection rates in civilian operating theatres and generally promoted the use of antiseptics.[28]

During World War I the importance of debridement and the need for delayed closure of traumatic wounds were established. In the beginning of the 20th century, Paul Ehrlich promoted the idea of selectively inhibiting the pathogens that caused infections, but it was the discovery and development of antibiotics that revolutionized the management of infection. Sulphonamides and penicillin were first used to control wound infection during World War II.[28]

By 1969 the availability and diversity of antibiotics prompted the US Surgeon General to suggest that, '. . . The time has come to close the book on infectious diseases'. However, the emergence and wide dissemination of antibiotic-resistant microbial strains has proved this statement to be premature. Despite knowledge of the natural reservoirs of infectious agents and their transmission routes to susceptible patients, surgical patients experience wound infections.[28]

Definition

WHO defines SSI clinically as: "a purulent discharge around the wound or the insertion site of the drain, or spreading cellulitis from the wound". Infections of

the surgical wound (whether above or below the aponeurosis), and deep infections of organs or organ spaces are identified separately.[4,29-30]

The US Centre of Disease Control and Prevention's (CDC) National Nosocomial Infections Surveillance (NNIS) system classifies SSIs as being either incisional or organ/space, occurred within 30 days after the operation. Incisional SSIs are further divided into those involving only skin and subcutaneous tissue (superficial incisional SSI) and those involving deeper soft tissues of the incision (deep incisional SSI). Organ/space SSIs involve any part of the anatomy (e.g. organ or space) other than incised body wall layers that was opened or manipulated during an operation.

The CDC published definitions for nosocomial infection in 1988, which included surgical wound infections. These were used by NNIS to monitor nosocomial infections, although definitions for SSIs were modified in 1992[4] to indicate three wound locations: superficial incisional SSIs, deep incisional SSIs and organ space SSIs. Similar definitions have been used elsewhere, but there is no universally accepted classification system, which makes comparison between hospitals difficult.

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 Centers 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.[4,29,30]

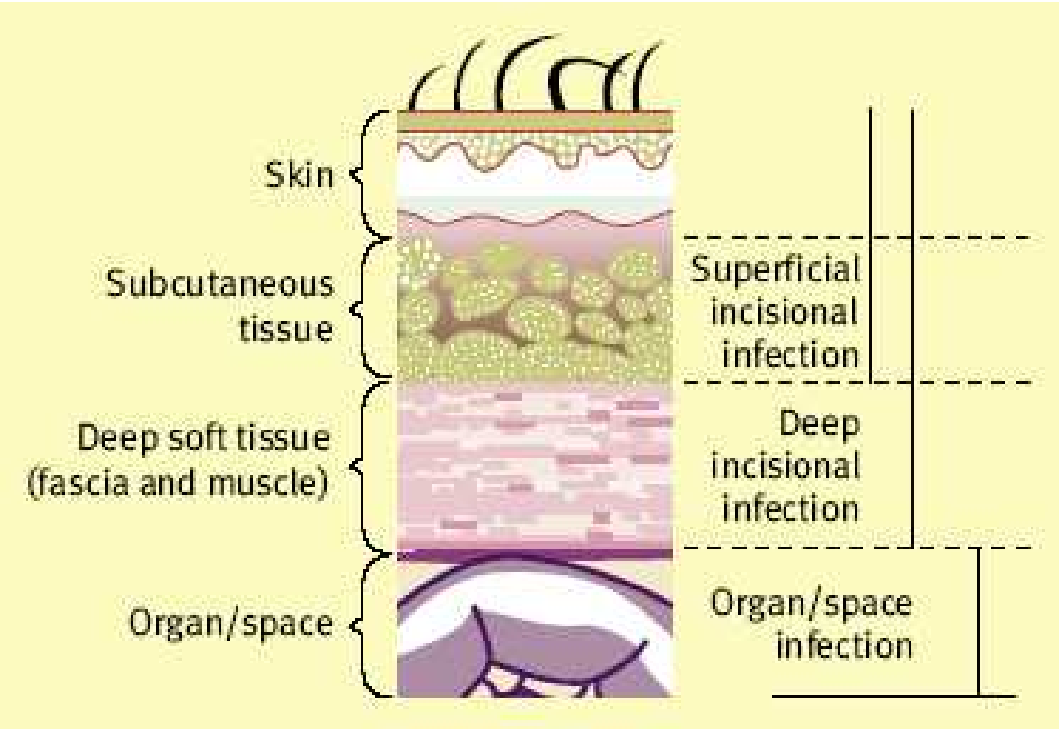


Figure 1. Schematic representation of the anatomical classification of surgical site infections[4]

Classification of surgical site infection[4]

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 1 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

Worldwide

The global data suggests the SSI incidence rate varies from 0.5 to 20% depending upon the type of operation and underlying patient status.[3,31,32] Surgical site infections account for over 20% of all healthcare-associated infections in surgical patients. Approximately 2–5% of surgical patients worldwide have developed an SSI.[33]

In 2010, an estimated 16 million operative procedures were performed in acute care hospitals in the United States.[34] A recent prevalence study found that SSIs were the most common healthcare-associated infection, accounting for 31% of all HAIs among hospitalized patients.[35] The CDC healthcare-associated infection (HAI) prevalence survey found that there were an estimated 157,500 surgical site infections associated with inpatient surgeries in 2011.[6] NHSN data for 2006-2008 (16,147 SSIs following 849,659 operative procedures) showed an overall SSI rate of 1.9%.[7]

India

The rate of SSI varies greatly worldwide and from hospital to hospital. In India, the surgical site infection rate reports by different workers have differed considerably. The rate of SSI in India varies from 2.5% to 41.9% as per different studies.[36-43]

A study[40] 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.[40,41,44,45]

A recent study by Shetty NH, et al.[38] from Mysore, Karnataka reported incidence of SSI as 21.66%.

However, 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.[46]

Mortality/Morbidity

Moreover, SSI contributes to surgery-related mortality, despite occurring frequently in superficial incisions. It has been reported that more than one-third of postoperative deaths worldwide are related to SSI.[33]

Kirkland et al calculated a relative risk of death of 2.2 attributable to SSIs, compared to matched surgical patients without infection.[10]

While advances have been made in infection control practices, including improved operating room ventilation, sterilization methods, barriers, surgical technique, and availability of antimicrobial prophylaxis, SSIs remain a substantial cause of morbidity, prolonged hospitalization, and death. SSI is associated with a mortality rate of 3%, and 75% of SSI-associated deaths are directly attributable to the SSI.[47]

Pathogenesis of SSIs

Microbial contamination of the surgical site is a necessary precursor of SSI. The risk of SSI can be conceptualized according to the following relationship:[3]

Risk of surgical site infection = (Dose of bacterial contamination x virulence) / Resistance of the host patient

Quantitatively, it has been shown that if a surgical site is contaminated with >105 microorganisms per gram of tissue, the risk of SSI is markedly increased. However, the dose of contaminating microorganisms required to produce infection may be much lower when foreign material is present at the site (i.e., 100 staphylococci per gram of tissue introduced on silk sutures).[3]

Microorganisms may contain or produce toxins and other substances that increase their ability to invade a host, produce damage within the host, or survive on or in host tissue. For example, many gram-negative bacteria produce endotoxin, which stimulates cytokine production. In turn, cytokines can trigger the systemic inflammatory response syndrome that sometimes leads to multiple system organ failure. One of the most common causes of multiple system organ failure in modern surgical care is intraabdominal infection. Some bacterial surface components,

notably polysaccharide capsules, inhibit phagocytosis, a critical and early host defense response to microbial contamination. Certain strains of clostridia and streptococci produce potent exotoxins that disrupt cell membranes or alter cellular metabolism.[3,48]

A variety of microorganisms, including gram-positive bacteria such as coagulase negative staphylococci, produce glycocalyx and an associated component called "slime," which physically shields bacteria from phagocytes or inhibits the binding or penetration of antimicrobial agents. Although these and other virulence factors are well defined, their mechanistic relationship to SSI development has not been fully determined.[3]

For most SSIs, the source of pathogens is the endogenous flora of the patient's skin, mucous membranes, or hollow viscera. When mucous membranes or skin is incised, the exposed tissues are at risk for contamination with endogenous flora. These organisms are usually aerobic gram-positive cocci (e.g., staphylococci), but may include fecal flora (e.g., anaerobic bacteria and gram negative aerobes) when incisions are made near the perineum or groin. When a gastrointestinal organ is opened during an operation and is the source of pathogens, gram negative bacilli (e.g., *E. coli),* gram-positive organisms (e.g., enterococci), and sometimes anaerobes (e.g., *Bacillus fragilis)* are the typical SSI isolates.[3]

Seeding of the operative site from a distant focus of infection can be another source of SSI pathogens, particularly in patients who have a prosthesis or other implant placed during the operation. Such devices provide a nidus for attachment of the organism. Exogenous sources of SSI pathogens include surgical personnel

(especially members of the surgical team), the operating room environment (including air), and all tools, instruments, and materials brought to the sterile field during an operation. Exogenous flora is primarily aerobes, especially gram-positive organisms (e.g., staphylococci and streptococci). Fungi from endogenous and exogenous sources rarely cause SSIs, and their pathogenesis is not well understood.[3]

Inflammatory Response

With the creation of the surgical incision through the skin and in to subcutaneous tissues, critical initiators of the inflammatory response are activated.

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 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.[3,49]

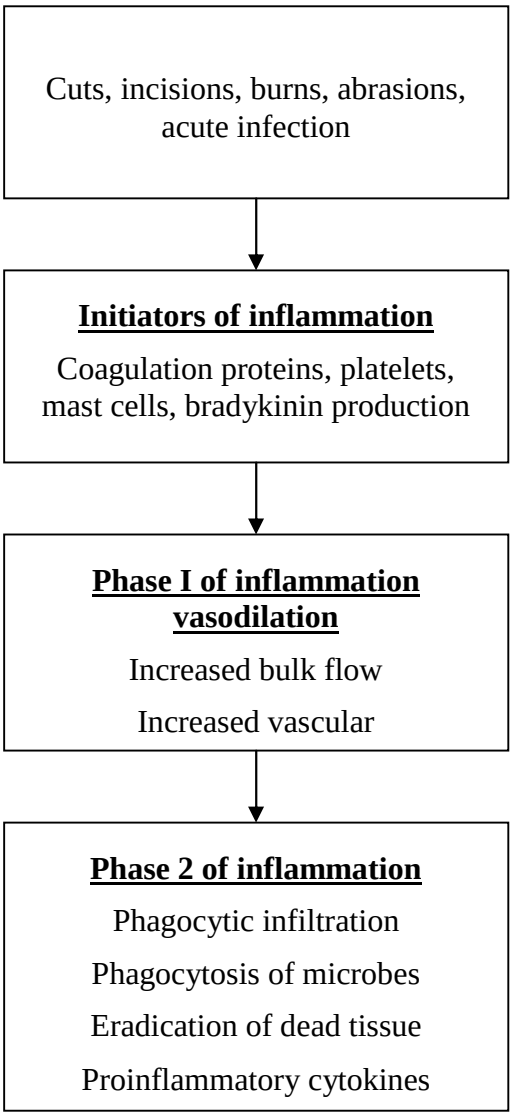


Figure 2. The consequences of inflammation[49]

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.[3,49]

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.[3,49]

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.[3,49]

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.[3,49]

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.[3,49]

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. *Rubor* reflects local vasodilation. *Calor* is the warmth of the vasodilated tissues resulting in increased heat conduction. *Tumor* reflects the presence of edema fluid. *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.[3,49]

Risk factors for the development of SSI

The term *risk factor* has a particular meaning in epidemiology and, in the context of SSI pathophysiology and prevention, strictly refers to a variable that has a significant, independent association with the development of SSI after a specific operation. Risk factors are identified by multivariate analyses in epidemiologic studies. Unfortunately, the term risk factor often is used in the surgical literature in a broad sense to include patient or operation features which, although associated with SSI development in univariate analysis, are not necessarily independent predictors. 80 The literature cited in the sections that follow includes risk factors identified by both univariate and multivariate analyses.[3]

The characteristics are useful in two ways: (1) they allow stratification of operations, making surveillance data more comprehensible; and, (2) knowledge of risk factors before certain operations may allow for targeted prevention measures. For example, if it is known that a patient has a remote site infection, the surgical team may reduce SSI risk by scheduling an operation after the infection has resolved.[3]

An SSI prevention measure can be defined as an action or set of actions intentionally taken to reduce the risk of an SSI. Many such techniques are directed at reducing opportunities for microbial contamination of the patient's tissues or sterile

surgical instruments; others are adjunctive, such as using antimicrobial prophylaxis or avoiding unnecessary traumatic tissue dissection.[3]

Optimum application of SSI prevention measures requires that a variety of patient and operation characteristics be carefully considered.[3]

The risk factors for the development of SSI include;[3]

Patient

- Age
- Nutritional status
- Diabetes
- Smoking
- Obesity
- Coexistent infections at a remote body site
- Colonization with microorganisms
- Altered immune response
- Length of preoperative stay

Operation

- Duration of surgical scrub
- Skin antisepsis
- Preoperative shaving
- Preoperative skin prep
- Duration of operation
- Antimicrobial prophylaxis
- Operating room ventilation

- Inadequate sterilization of instruments
- Foreign material in the surgical site
- Surgical drains
- Surgical technique
    - Poor hemostasis
    - Failure to obliterate dead space
    - Tissue trauma

Surgical Wound Classification and Subsequent Risk of Infection[50.51]

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

Incidence of SSIs with regard to abdominal surgical sites and operating conditions[9]

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%
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%

Prevention of SSI

Utilizing known infection prophylaxis measures is best surgical practice and much more preferable than dealing with the morbidity of surgical-site infection. The majority of the published literature reports on the use of prophylactic antibiotics in open surgery.

Preoperative Measures

Preoperative interventions that may reduce SSI include preoperative clipping rather than shaving hair at the operative site. Proper techniques for patient skin preparation, surgical team member hand/forearm antisepsis, draping of the site, and gowning/gloving.

Preoperative prophylactic antibiotics should be utilized according to the classification of the surgical wound. Prophylactic antibiotics are unnecessary in clean surgical procedures.[50]

For clean-contaminated procedures antibiotics should be utilized. Several trials have demonstrated that no difference exists in SSI rates when preoperative antibiotics are given, making their use unnecessary.[50-54]

Intraoperative Measures

Several intraoperative technical considerations have been studied in efforts to reduce surgical-site infection. Surgical drains are occasionally utilized when there is concern for possible fluid accumulation, such as bile or blood at the operative site. In theory, abdominal fluid collections may harbor bacterial growth; however, the routine use of drains in uncomplicated procedures increases infection rate.[55]

Skin adhesives are an attractive option for closure in clean contaminated surgical procedures due to their speed and ease of use. They are equivalent to suture closure in terms of superficial surgical-site infection rates.[56,57] Therefore, the use of either suture or skin adhesives for skin closure is appropriate.

Plastic wound protectors are commonly used. Their use does not necessarily decrease the incidence of surgical-site infection.[58] However, wound protectors do facilitate exposure and may protect against the development of surgical site infection. There is currently not enough literature to support routine use of these devices simply to reduce the risk of surgical-site infections.

Without larger randomized trials demonstrating superiority of these various techniques in reducing infection rates, the decision to utilize these intraoperative measures usually depends on surgeon preference, experience, and training, and operative time, cost, and equipment availability.

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.[17] Sutures are a contributory factor in infection; in fact, 66% of SSIs are related to the incision.[52]

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.[53]

In fact, it is postulated that in the presence of sutures, only 100 colony-forming units (CFU)/mg are necessary to produce infection.[59] 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.[60]

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.[54]

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.[55]

A study,[18] 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[16] 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.[55]

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[55,56]

Antimicrobial Sutures

The antimicrobial suture is interesting. Howel,[61] 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.[57] 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.[55]

Recently, the only substance being used for impregnation in suture is Triclosan. Triclosan 5-chloro-2 (2, 4-dichlorophenoxyphenol) is a broad-spectrum antimicrobial agent developed over 40 years ago.[58]

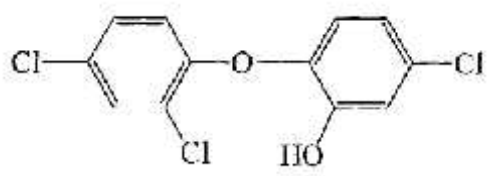


Figure 3. Chemical structure of Triclosan

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.[62]

In 1989, triclosan was approved for use in cosmetics, which can be used up to 0.3% by the European Community Cosmetic Directive.[63]

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.[64]

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).[64]

Safety review

Acute toxicity

Acute toxicity was evaluated through single dose exposure in animal models through variety of routes of administration. LD50 value of 2000 to 5000 mg/kg is considered to be non-toxic.[19]

Subchronic toxicity

Subchronic toxicity has been evaluated for NOEL (no observed effect level) in approximately 90 day oral administration studies in various animal models. The safety factor of 100-1000 fold is considered as safe for many active ingredients.[19]

Chronic toxicity

Chronic toxicity studies with long duration exposure to oral administration of triclosan have shown favorable response. Studies were conducted in rat and hamster model. The NOEL (rats – 52 mg/kg for male, 67 mg/kg for females, hamster - 75 mg/kg) values were within the acceptable limit.[19]

Carcinogenicity

No evidence of carcinogenic changes observed in chronic toxicity studies, showing non carcinogenic property of triclosan at NOEL values and with the acceptable safety factor.[19]

Other toxicity studies

Various other studies have shown no adverse effects of triclosan on pregnancy and reproductive potential. Triclosan is also proved as not potential to develop teratogenicity, mutagenicity and genotoxicities at various NOEL values observed in animal toxicity studies.[65]

Assessment of safety

Barbolt TA has evaluated the gradual exposure of triclosan (related to triclosan dissipation profile for each suture) and worst case scenario ‘immediate

exposure'. The toxicity due to triclosan coated suture are considered to be low due low exposure levels, rapid metabolism and excretion, and lack of accumulation over time.[65]

Considering the dissipation profile of triclosan for each suture type and worst case scenario of 5 m of a 2-0 suture with 472 µg triclosan/m (270 for Europe and India) for vicryl Plus and 2360 µg/m for PDS plus and monocryl plus, the maximal single-day exposure to triclosan was calculated to be 0.03, 0.09 and 0.08 mg/kg body weight.[65,66] The safety margin would be in the range of 160 to 2500, good enough to consider triclosan safe.[19]

Thus, Barbolt TA highlighted the extensive clinical experience with triclosan coated suture, availability of favorable toxicity study data and non-carcinogenic potential, precludes the need to conduct genotoxicity studies and other toxicity studies.[65]

Mechanism of action and antibacterial profile

Triclosan exhibits its bactericidal property by inhibiting FabI gene which encodes the enoyl-acyl carrier protein reductase enzyme (ENR). It is essential in fatty acid biosynthesis, which is inhibited by triclosan. Triclosan disrupts the cell membrane causing cell contents to leak.[65,66] Triclosan has bactericidal activity against most of the microorganisms primarily responsible for SSI.[19]

Microorganisms susceptible to triclosan coated suture using zone of inhibition studies

- *Staphylococcus aureus*
- *Methicillin resistant Staphylococcus aureus (MRSA)*
- *Staphylococcus epidermidis*
- *Methicillin resistant Staphylococcus epidermidis (MRSE)*
- *Eeschirichia coli*
- *Klebsiella pneumoniae*

Some of the bacteria such as Pseudomonas aeruginosa, Acinitobacter requires high concentration of triclosan for the bactericidal effect. Leaper D et al.[66] pointed out that due to multi-drug efflux pumps that remove triclosan from cell also distinct versions of the ENR, Pseudomonas aeruginosa shows innate resistance to triclosan.

Bacterial resistance

Although various studies have shown resistance to triclosan, but these studies basically are the laboratory studies. However, resistance to triclosan has not been demonstrated in various clinical studies or from epidemiological data. Hence, there is no clinical evidence for triclosan resistance.[19]

Zone of inhibition and related studies

Using zone of inhibition studies, antibacterial sutures shown to inhibit bacterial colonization of S. aureus (SA), MRSA, S. epidermidis, MRSE, E. coli and K. pneumonia.[19]

Sengupta M. et al.[67] have conducted in-vitro zone of inhibition studies for triclosan coated and uncoated suture against the bacteria isolated from SSI. In addition to above bacteria, other bacteria such as Acinetobacter, Coagulase negative staphylococcus, Proteus and Pseudomonas. Zone of inhibition was observed in all bacterial plate except for Pseudomonas and Acinetobacter.

Edmiston CE et al.[68] has showed substantial reduction in both gram-positive and gram-negative bacterial adherence to triclosan coated polyglactin 910 (braided) suture compared with non triclosan coated suture, in an in vitro microbiologic model Standardized cultures (2.0 log10 colony forming units/mL and 5.0 log10 colony forming units/mL of three clinical strains, MRSA, S. epidermidis and Escherichia coli.

These results were similar to earlier zone of inhibition study conducted for triclosan coated polyglactin 910 sutures.[69]

In another *in vitro* study, triclosan coated polidioxanone sutures found to be effective against S. aureus, MRSA, S. epidermidis, MRSE, K. pneumoniae, and E. coli. Additionally, antibacterial activity was lasted for 17-23 days till the suture dissolved. In animal models, it was found that TCS inhibited *in vivo* colonization of bacteria compared with the non-coated suture (99.9% reduction in S. aureus and a 90% reduction in E. coli).[70] The same author group also published in vivo antibacterial efficacy of triclosan coated poliglecaprone 25 suture.[71]

Triclosan coated suture - clinical studies published

Since the introduction of triclosan coated sutures, many clinical studies have been published in varied therapeutic area. It includes systematic reviews and meta-analyses, Randomized controlled Clinical Trials (RCTs), and other clinical trials such as cohort studies, case controlled studies and case series. The evidences are published in various therapeutic segment e.g. general surgeries, urosurgery, breast surgery, gynecological procedures, oncology surgery, cardiac and vascular surgery, orthopedic surgery, ENT surgery, etc.[19]

A Systematic Literature Review (SLR) by Daoud FC et al.[72] was conducted in 2014. The main objective of the was to assess the robustness of study results by applying more stringent statistical tests compared to first meta-analysis, to determine the efficacy of TCS in reduction of risk of SSI. The secondary objectives were to assess potential bias or confounding factors which could invalidate the triclosan effect in the pooled RCTs. RCTs included were selected on criteria used to assess quality of study and publication bias. A rigorous 13 step analytical strategies were used to meet the objectives of SLR. The data from 15 RCTs totaling 4000 patients (TCS=2323) and NTCS=2477) were analyzed. Use of TCS was associated with a decrease in SSIs in selected patient populations (RR=0.67; P=0.00053), means 33% reduction in risk of developing SSIs. TCS showed highly statistical significant results in lowering risk of SSI. TCS was effective in clean (P=0.001), clean-contaminated (P=0.010), and contaminated incisions (P=0.026). SLR result was robust to the removal of three RCTs. SLR showed highly statistical significant results favoring TCS in reduction of risk of SSI and robustness of clinical results - relative risk independent of confounding factors.

A meta-analysis by Edmiston CE et al.[73] (2013) was conducted in response to recently published systematic reviews and meta-analysis which have suggested about no benefits of anti-microbial coated suture in reducing the Surgical Site Infections (SSI). Authors have highlighted poor selection of available RCT and low patient numbers for these meta-analyses. The primary endpoint of the systematic review was to determine the ratio of patients who developed an SSI in two comparative groups: closure with TCS versus NTCS sutures. Total 13 RCTs were selected, totaling 3568 patients (TCS=1654) and NTCS=1914). Stringent criteria was applied for selection of RCTs such as protocol with defined objective, accurate SSI definition, specified patient population, proper randomization procedure, study design which enables unbiased comparison between two groups, lost to follow up patients <10%, ethical conduct of study, etc. Publication bias (fixed - assuming same patient population and random effect - assuming clinically heterogeneous patient population), heterogeneity and sensitivity analysis was considered to check the robustness of the model used. Use of TCS was associated with a decrease in SSIs in selected patient populations (fixed effect: RR = 0.734; P = 0.005; random-effect: RR = 0.693; P = 0.011), means 27-31% reduction in risk of developing SSIs. No publication bias was detected (Egger intercept test: P = 0.145).

Wang ZX et al.[74] (2013) included a total 17 RCTs for the meta-analysis, covering 3720 patients (TCS=1726) and NTCS=1994). The meta-analysis was performed in adherence to the guidelines outlined in the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement. All 17 studies selected were assessed independently by 2 investigators. Risk of bias and methodological quality of included studies were assessed using the Cochrane

Collaboration tool for assessing risk of bias. Publication bias also was assessed by using Funnel plots. Results favored TCS with a pooled RR of 0.70 (P <0.001) without statistical heterogeneity (P for Q test = 0.129, *I*2=29 per cent), means TCS provided significant advantage in reducing the rate of SSI by 30%. Subgroup analysis indicates statistical significant results of reduction in SSI by using TCS in adult patients, abdominal surgery and clean or clean contaminated wounds. The advantage of TCS over conventional sutures was consistent regardless of length of follow-up. The qualities of the included studies were acceptable with moderate risk of bias and no evidence for significant publication bias was noted.

In another prospective comparative study[75] in transverse laparotomy for hepatobiliary resections (n=839), TCS showed significant reduction in SSI compared to NTCS arm (4.3% vs. 9.2%, P = 0.05). In spinal surgery, TCS found to be effective in reduction of wound infection (0.5% vs. 3.9%, p=0.020).

Wide range of published evidences is available for toxicity profile of triclosan, antibacterial profile and clinical effectiveness of TCS. Hence, it assures use of TCS in minimizing the risk of SSI. This effort is an attempt to draw attention to the continuous publication around TCS and reemphasize the efficacy and safety parameter as in these published clinical evidences.

Galal I. et al.[76] in 2011 performed a prospective, randomized, double-blinded, controlled multicenter study aimed to compare triclosan-coated polyglactin 910 sutures with polyglactin 910 sutures for the reduction of surgical site infections. They concluded that, use of the triclosan-coated polyglactin 910 antimicrobial suture

lead to reduction of surgical site infection and has an impact on saving health care resources.

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.[77]

The antimicrobial spectrum and speed of activity of triclosan are well documented both as an active ingredient and in a wide array of formulations.[62]

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 difficile*, *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 at both concentrations with a high level of activity on enveloped viruses such as Herpes simplex virus, HIV-l, and Influenza.[62]

As previous mentioned above, FDA (US) has approved polyglactin 910 sutures coated with triclosan for commercial used since 2002.[63]

The first report[78] 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.

A large cohort,[53] 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. Using a PDS loop suture for abdominal wall closure, 10.8% of patients with wound infections was detected. The number of patients with wound infections decreased in TP2 using Vicryl plus for abdominal wall closure to 4.9% ($p<0.001$) 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 (2011), Justinger et al.[53] 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.[79] 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.

In 2009 was a prospective, randomized, controlled, double blind, comparative, a single center study[80] which was 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. Surgeons and assistants were blinded to suture type as similarity in appearance made the two products indistinguishable. Baseline patient characteristics were not different between both groups. 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.

In 2011,[81] 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.

The study[81] 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.

According to an RCT done in Switzerland, When a PDS loop suture for abdominal wall closure was used, 42 (11.3%) patients with wound infections were detected. The number of patients with wound infections decreased significantly to 31 when the PDS plus for abdominal wall closure was used (6.4%, $P < .05$).[82]

According to a Prospective study done in Paris, study used individual predictions of SRC and showed that using TC-coated suture may prevent SRC. This was particularly significant in high-risk patients.[83]

According to an RCT done at Sweden, primary endpoint occurred in 23 patients (12.5%) with triclosan-coated sutures and in 38 patients (20.0%) in the group without triclosan (P = 0.0497, risk ratio 0.63, (95% confidence interval 0.39–1.00).[84]

METHODOLOGY

The present study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Study design

The study design was one year randomized controlled trial.

Study period and duration

This study was done for the period of one year from January 2014 to December 2014.

Place

This study was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belgaum.

Source of Data

Patients undergoing open abdominal surgeries (Clean contaminated) in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were included.

Sample size

A total of 60 patients divided into two groups of 30 each undergoing open abdominal surgeries (Clean contaminated) were studied.

Sampling procedure

Based on the previous literature, considering P_1 as 11.3% and the P_2 as 6.4% and the sample size calculated was 305 per group. However, as it was not feasible in the current setting applying the thumb rule, the sample size was taken as 60.

Selection criteria

Inclusion

- All patients undergoing abdominal surgeries (Clean contaminated surgeries).

Exclusion

Patients with:

- Diabetes mellitus or immunodeficiency
- Received systemic antibiotics within 2 weeks of proposed surgery
- Contaminated and dirty cases.

Ethical clearance

The study was approved by the Ethical and Research Committee of Ethics Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed consent

Patients admitted in the wards of Department of General Surgery at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum undergoing clean contaminated open abdominal surgeries were evaluated based on selection criteria. The selected patients were briefed about the nature of the study and a written informed consent was obtained (Annexure I).

Data collection

Demographic data like sex and age were collected along with relevant history. A thorough clinical examination was done and the findings were recorded on predesigned and pretested proforma (Annexure II).

Investigations

The present study did not required specific investigations. However, routine investigations required for the surgical fitness including complete blood count, blood urea, serum creatinine, chest X-ray and echocardiography were done.

Randomization

Based on the wound closure technique, patients were randomized into to two groups of 30 each by Opaque Envelope Method as group A (primary fascial closure with PDS II sutures) and Group B (primary fascial closure with PDS plus sutures).

Procedure

Pre operative

In both the groups, shaving of the abdomen from nipple to mid-thigh prior to surgery was done. On the operation table the abdomen was cleaned with povidone iodine and spirit. Injection ciprofloxacin 100 mL IV and Inj. metronidazole 100 ml IV were given prior to skin incision. All the patients had standard analgesic and antibiotics protocol.

Surgical technique

Patients in both the groups underwent open abdominal surgeries using similar instruments and accepted general principles of surgery.

Closure technique

The closure of wound was done in monolayer.

Group A

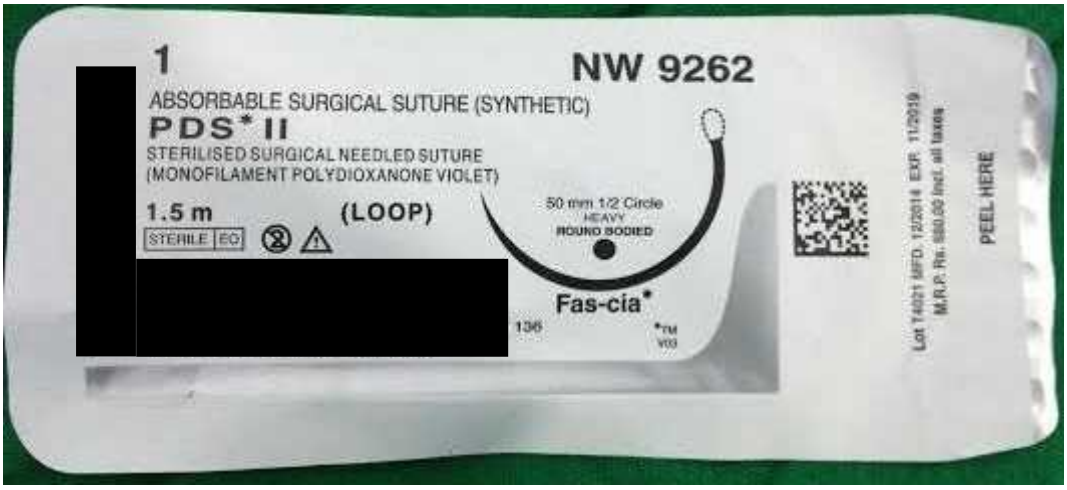
Patients in this group underwent primary fascial closure with PDS II sutures.

Group B

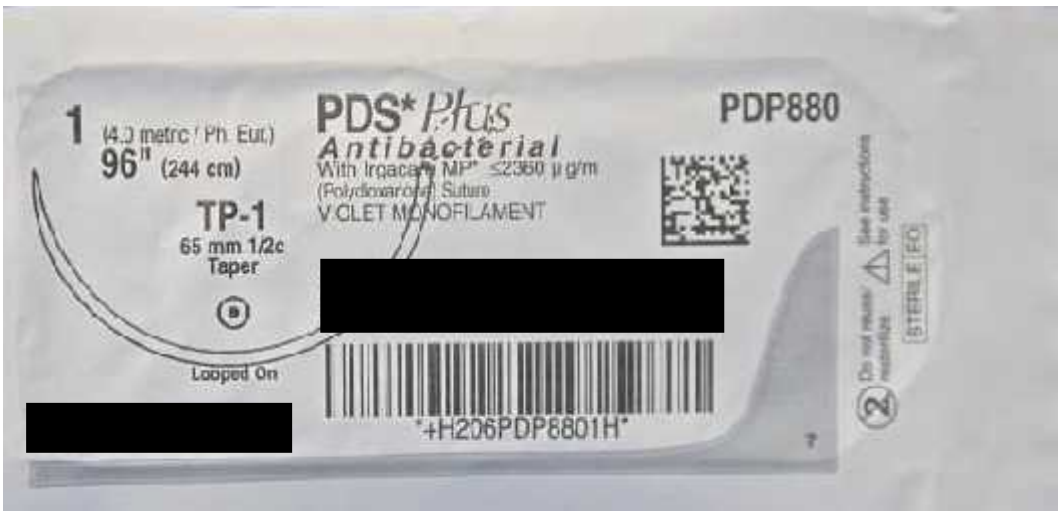
Patients in this group underwent primary fascial closure with PDS plus sutures.

Post operative

The patients were postoperatively medicated with Inj. Ciprofloxacin 100 mL IV twice daily and Inj. metronidazole 100 mL thrice daily and if indicated and were changed to higher antibiotics accordingly.



Photograph 1. PDS II suture



Photograph 2. PDS plus suture

Follow up

Patients were followed on postoperative day 2, 6 and 10.

Outcome variables

Patients were evaluated for discharge/pus from the wound, if any on postoperative day 2, 6 and 10. In cases with presence of discharge / pus, samples were collected and sent for culture and sensitivity. Also the wound was inspected for local tenderness, redness and raised local temperature. Further patients were also evaluated for pain and fever. The assessment of pain was subjective.

Surgical site infection

The endpoint of the study was presence or absence of 'Postoperative surgical site infection'. An incisional surgical site infection was considered to be positive if surgical wound drained purulent material or if the surgeon judges it to be infected and opens it. The surgical wound infection was defined according to US Centre for Disease Control and Prevention (CDC) as SSI.[23]

Statistical analysis

The data was tabulated on Microsoft excel spread sheet (Annexure III). The data was analyzed using SPSS version 20.0 Categorical data was expressed as rates, ratios and percentages and continuous data was expressed as mean ± SD. Categorical data was compared using Chi-square test or Fisher's exact test and continuous data was compared using independent sample 't' test. A probability value of ≤ 0.050 at 95% confidence interval was considered as statistically significant.

RESULTS

The present one year randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 60 patients undergoing open abdominal surgeries (Clean contaminated) from January 2014 to December 2014 were studied.

The selected patients divided into two groups of 30 each as below.

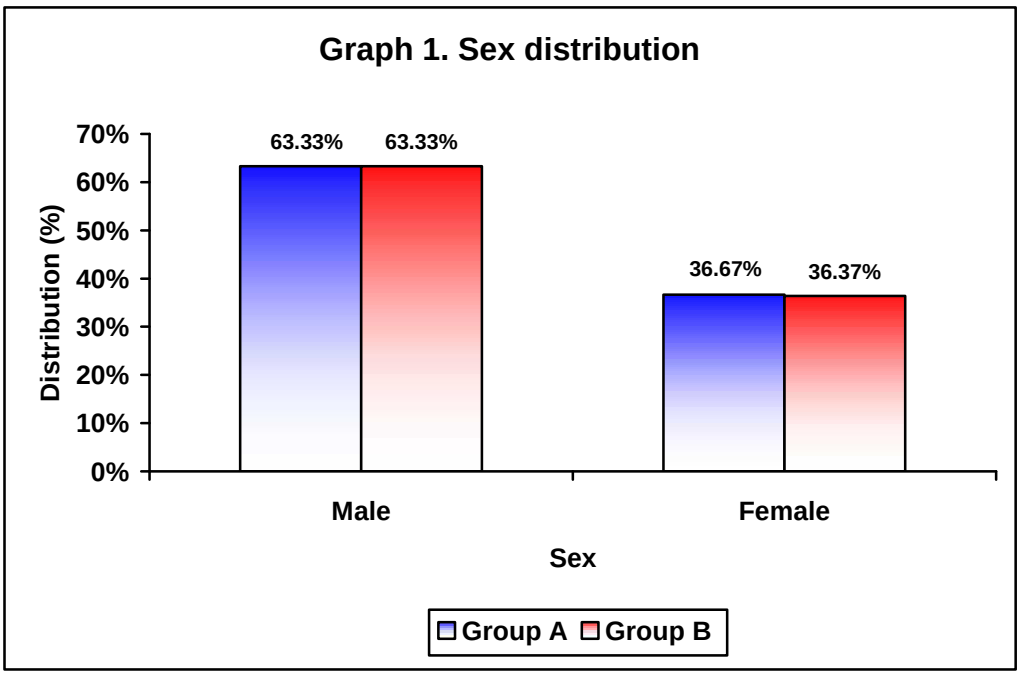
- Group A - Patients in this group underwent primary fascial closure with PDS II sutures.
- Group B - Patients in this group underwent primary fascial closure with PDS plus sutures.

The data obtained was analyzed and the final results and observations were tabulated as below.

Table 1. Sex distribution

Sex	Group A (n=30)		Group B (n=30)	
	Number	**Percentage**	**Number**	**Percentage**
Male	19	63.33	19	63.33
Female	11	36.67	11	36.67
Total	**30**	**100.00**	**30**	**100.00**

p = 1.000

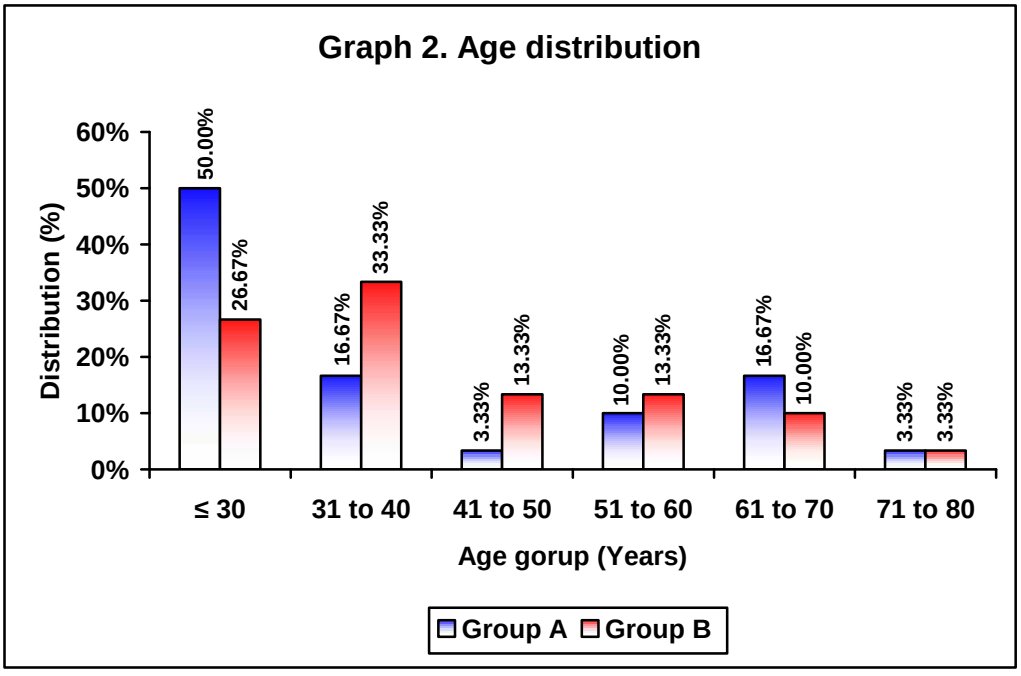


In the present study 63.33% of the patients in group A and group B were males and male to female ratio was 1.72:1 in both the groups.

Table 2. Age distribution

Age group (Years)	Group A (n=30)		Group B (n=30)	
	Number	Percentage	Number	Percentage
≤ 30	15	50.00	8	26.67
31 to 40	5	16.67	10	33.33
41 to 50	1	3.33	4	13.33
51 to 60	3	10.00	4	13.33
61 to 70	5	16.67	3	10.00
71 to 80	1	3.33	1	3.33
Total	**30**	**100.00**	**30**	**100.00**

p = 0.262



In this study 50% of the patients in group A were aged ≤ 30 years while in group B 33.33% of the patients were aged 31 to 40 years. However this difference was statistically not significant (p=0.262).

Table 3. Mean age

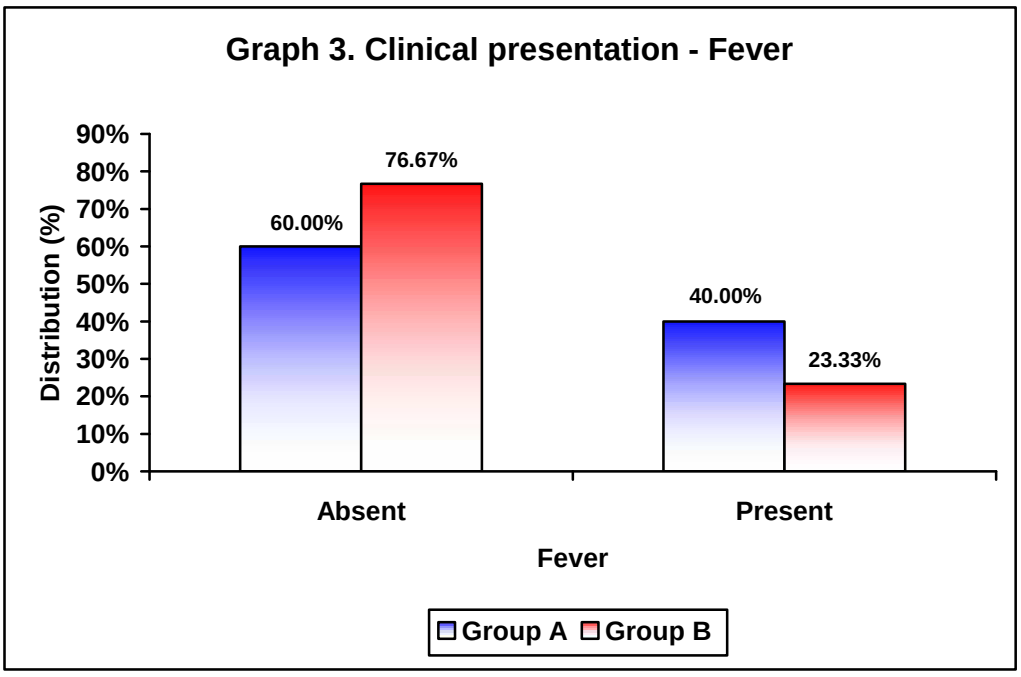
Variables	Group A (n=30)		Group B (n=30)		p value
	Mean	SD	Mean	SD	
Age (Years)	39.90	17.67	41.67	16.08	0.687

In the present study the mean age in group A and group B was comparable (39.90 ± 17.67 years vs 41.67 ± 16.08 years; p=0.687).

Table 4. Clinical presentation - Fever

Fever	Group A (n=30)		Group B (n=30)	
	Number	**Percentage**	**Number**	**Percentage**
Absent	18	60.00	23	76.67
Present	12	40.00	7	23.33
Total	**30**	**100.00**	**30**	**100.00**

p = 0.165

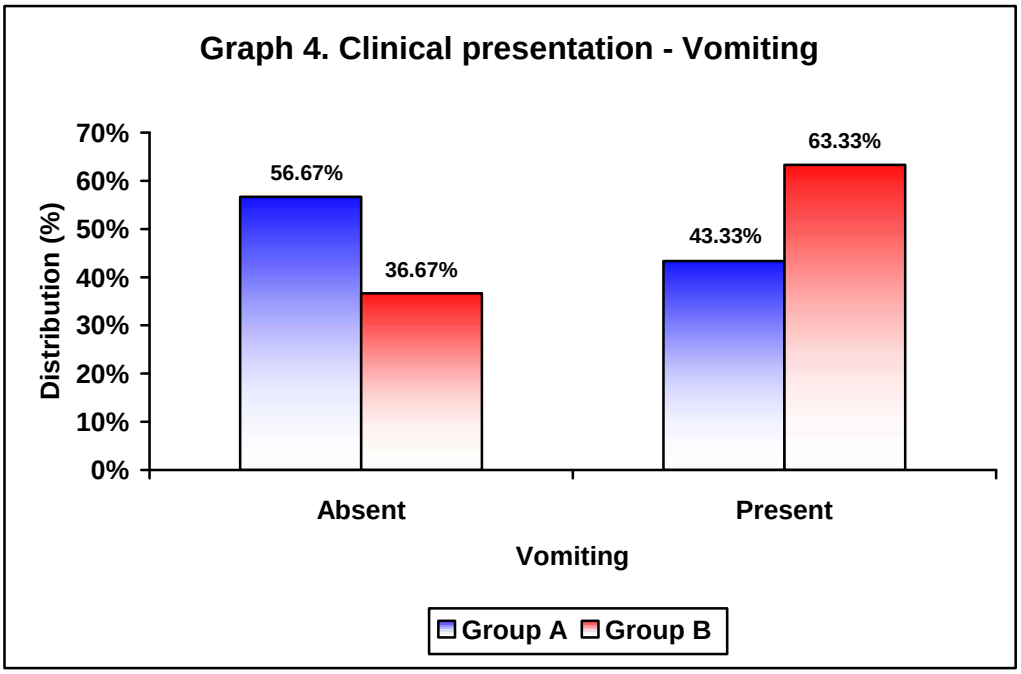


In this study 40% of the patients in group A presented with fever compared to 23.33% in group B. However this difference was statistically not significant (p=0.165).

Table 5. Clinical presentation - Vomiting

Vomiting	Group A (n=30)		Group B (n=30)	
	Number	**Percentage**	**Number**	**Percentage**
Absent	17	56.67	11	36.67
Present	13	43.33	19	63.33
Total	**30**	**100.00**	**30**	**100.00**

p = 0.121

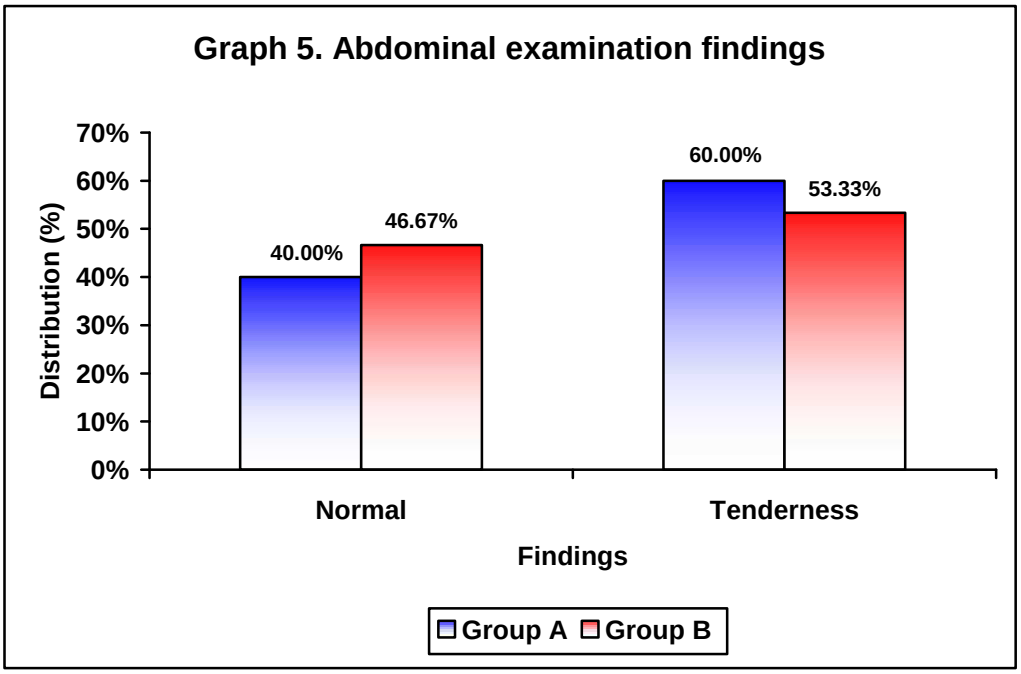


In the present study the features of vomiting at presentation were noted in 43.33% of the patients in group A, compared to 63.33% in group B but the difference was statistically not significant (p=0.121).

Table 6. Abdominal examination findings

Findings	Group A (n=30)		Group B (n=30)	
	Number	**Percentage**	**Number**	**Percentage**
Normal	12	40.00	14	46.67
Tenderness	18	60.00	16	53.33
Total	**30**	**100.00**	**30**	**100.00**

p = 0.602



In this study abdominal examination revealed tenderness in 60% of the patients who were in group A and 53.33% in group B (p=0.602).

Table 7. Comparison of vitals

Parameters	Group A (n=55)		Group B (n=55)		p value
	Mean	SD	Mean	SD	
Pulse rate (/Minute)	87.43	12.56	83.67	18.36	0.358
Respiratory rate (/Minute)	13.27	1.78	13.47	1.59	0.648
Systolic BP (mm Hg)	114.00	12.76	112.47	13.57	0.654
Diastolic BP (mm Hg)	72.33	8.17	71.93	9.83	0.865

Table 7 shows comparison of mean pulse rate, respiratory rate, systolic and diastolic blood pressure. These characteristics in group A and B were comparable ($p>0.050$).

Table 8. Comparison of type of surgery

Type of surgery	Group A (n=30)		Group B (n=30)	
	No.	**%**	**No.**	**%**
Small bowel resection and anastomosis	8	26.67	8	26.67
Appendectomy	7	23.33	3	10.00
Splenectomy	3	10.00	5	16.67
Right hemicolectomy	4	13.33	2	6.67
Gastrojejunostomy	0	0.00	3	10.00
Ileostomy	3	10.00	0	0.00
Pancreaticojejunostomy	2	6.67	1	3.33
Feeding jejunostomy	0	0.00	2	6.67
Fundoplication	0	0.00	2	6.67
Diversion colostomy	1	3.33	1	3.33
Abdominoperineal resection	0	0.00	2	6.67
Cystogastrostomy	2	6.67	1	3.33
Total	**30**	**100.00**	**30**	**100.00**

p = 0.172

Table 8 shows the distribution of patients according to the type of surgery. It was observed that the type of surgery was comparable in group A and B (p=0.172)

Table 9. Wound examination findings on day two

Findings	Findings	Group A (n=30)		Group B (n=30)		p value
		No	**%**	**No**	**%**	
Pain	Absent	13	43.33	18	60.00	0.196
	Present	17	56.67	12	40.00	
	Total	**30**	**100.00**	**30**	**100.00**	
Local tenderness	Absent	14	46.67	19	63.33	0.194
	Present	16	53.33	11	36.67	
	Total	**30**	**100.00**	**30**	**100.00**	
Redness	Absent	18	60.00	20	66.67	0.592
	Present	12	40.00	10	33.33	
	Total	**30**	**100.00**	**30**	**100.00**	
Raised local temperature	Absent	18	60.00	20	66.67	0.592
	Present	12	40.00	10	33.33	
	Total	**30**	**100.00**	**30**	**100.00**	
Discharge	Absent	19	63.33	21	70.00	0.584
	Present	11	36.67	9	30.00	
	Total	**30**	**100.00**	**30**	**100.00**	
Fever	Absent	22	73.33	24	80.00	0.542
	Present	8	26.67	6	20.00	
	Total	**30**	**100.00**	**30**	**100.00**	

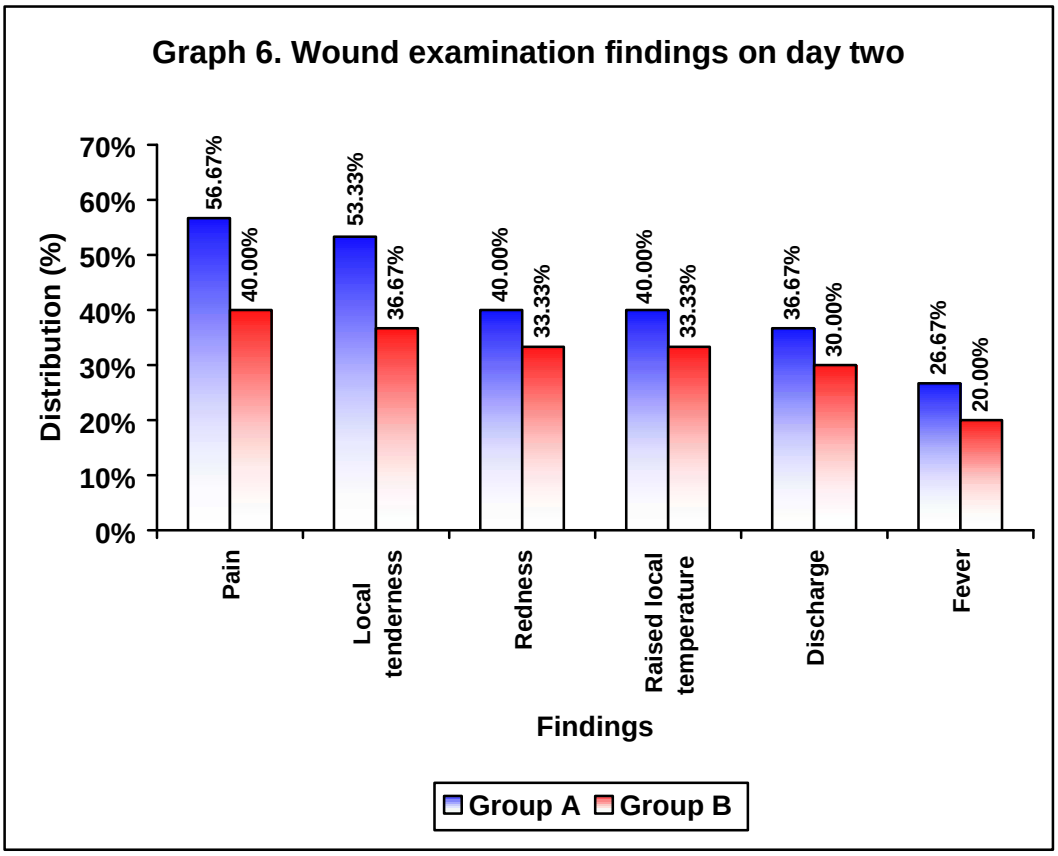


Table 9 and graph 6 shows wound examination findings on day two. It was observed that, pain, local tenderness, redness, raised local temperature, discharge and fever were present in 56.67%, 53.33%, 40%, 40%, 36.67% and 26.67% of the patients in group A, compared to 40%, 36.67%, 33.33%, 33.33%, 30% and 20% in group B respectively However the differences observed in group A and B were statistically not significant ($p>0.050$).

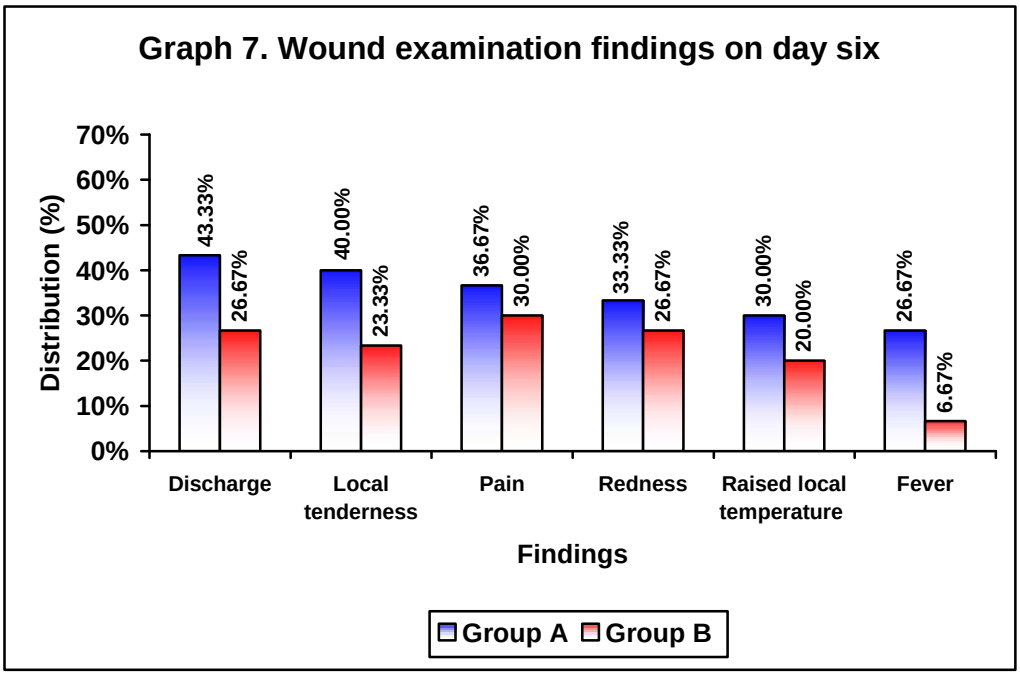
Table 10. Comparison of wound culture and organisms on day two

Findings	Findings	Group A (n=11)		Group B (n=9)		p value
		No	%	No	%	
Culture	Negative	2	18.18	2	22.22	1.000
	Positive	9	81.82	7	77.78	
	Total	**11**	**100.00**	**9**	**100.00**	
Organism	Coagulase-neg staphylococci	0	0.00	1	11.11	0.935
	E. coli	3	27.27	1	11.11	
	Enterobacter sp.	0	0.00	1	11.11	
	Pseduomonas aeruginosa	3	27.27	2	22.22	
	Staphylococcus aureus	2	18.18	1	11.11	
	Staphylococcus epidermidis	1	9.09	1	11.11	
	No growth	2	18.18	2	22.22	
	Total	**11**	**100.00**	**9**	**100.00**	

In the present study, on day two, positive culture was noted in 81.82% of the patients who had discharge/pus in group A compared to 77.78% in group B. However the differences observed in group A and B were statistically not significant (p=1.000). *Pseudomonas aeruginosa* was the commonest organism isolated in 27.27% of the patients in group A and 22.22% in group B (p=0.935).

Table 11. Wound examination findings on day six

Findings	Findings	Group A (n=30)		Group B (n=30)		p value
		No	**%**	**No**	**%**	
Discharge	Absent	17	56.67	22	73.33	0.176
	Present	13	43.33	8	26.67	
	Total	**30**	**100.00**	**30**	**100.00**	
Local tenderness	Absent	18	60.00	23	76.67	0.165
	Present	12	40.00	7	23.33	
	Total	**30**	**100.00**	**30**	**100.00**	
Pain	Absent	19	63.33	21	70.00	0.584
	Present	11	36.67	9	30.00	
	Total	**30**	**100.00**	**30**	**100.00**	
Redness	Absent	20	66.67	22	73.33	0.573
	Present	10	33.33	8	26.67	
	Total	**30**	**100.00**	**30**	**100.00**	
Raised local temperature	Absent	21	70.00	24	80.00	0.371
	Present	9	30.00	6	20.00	
	Total	**30**	**100.00**	**30**	**100.00**	
Fever	Absent	22	73.33	28	93.33	0.038
	Present	8	26.67	2	6.67	
	Total	**30**	**100.00**	**30**	**100.00**	



In this study, on day six, most of the patients had discharge in group A (43.33%) while in group B most of the patients reported pain (30%). However, the wound observations made on day six did not vary significantly in group A and B ($p>0.050$).

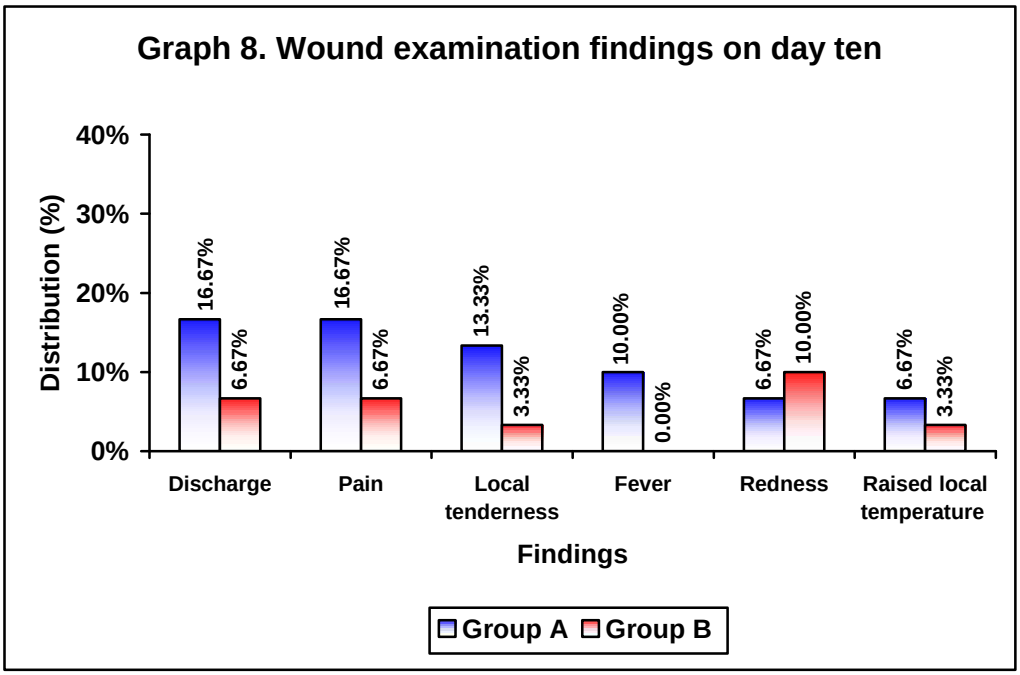
Table 12. Comparison of wound culture and organism on day six

Findings	Findings	Group A (n=13)		Group B (n=8)		p value
		No	%	No	%	
Culture	Negative	7	53.85	3	37.50	0.659
	Positive	6	46.15	5	62.50	
	Total	**13**	**100.00**	**8**	**100.00**	
Organism	*Enterobacter* sp.	2	15.38	0	0.00	0.334
	Pseduomonas aeruginosa	3	23.08	2	25.00	
	Staphylococcus aureus	1	7.69	2	25.00	
	Staphylococcus epidermidis	0	0.00	1	12.50	
	No growth	7	53.85	3	37.50	
	Total	**13**	**100.00**	**8**	**100.00**	

In the present study, on day six, wound culture was positive in 46.15% of the patients who had discharge / pus in group A, compared to 62.50% in group B (p=0.659). *Pseudomonas aeruginosa* was the commonest organism isolated in group A (23.08%) and B (25%) on day six (p=0.334).

Table 13. Wound examination findings on day ten

Variables	Findings	Group A (n=30)		Group B (n=30)		p value
		No	**%**	**No**	**%**	
Discharge	Absent	25	83.33	28	93.33	0.212
	Present	5	16.67	2	6.67	
	Total	**30**	**100.00**	**30**	**100.00**	
Pain	Absent	25	83.33	28	93.33	0.212
	Present	5	16.67	2	6.67	
	Total	**30**	**100.00**	**30**	**100.00**	
Local tenderness	Absent	26	86.67	29	96.67	0.177
	Present	4	13.33	1	3.33	
	Total	**30**	**100.00**	**30**	**100.00**	
Fever	Absent	27	90.00	30	100.00	0.119
	Present	3	10.00	0	0.00	
	Total	**30**	**100.00**	**30**	**100.00**	
Redness	Absent	28	93.33	27	90.00	0.500
	Present	2	6.67	3	10.00	
	Total	**30**	**100.00**	**30**	**100.00**	
Raised local	Absent	28	93.33	29	96.67	0.500
temperature	Present	2	6.67	1	3.33	
	Total	**30**	**100.00**	**30**	**100.00**	



In the present study wound examination on day ten revealed discharge in 16.67% of the patients in group A, and redness in 10% of the patients in group B. However, the wound characteristics on day ten were comparable in group A and B ($p>0.050$).

Table 14. Comparison of wound culture and organism on day ten

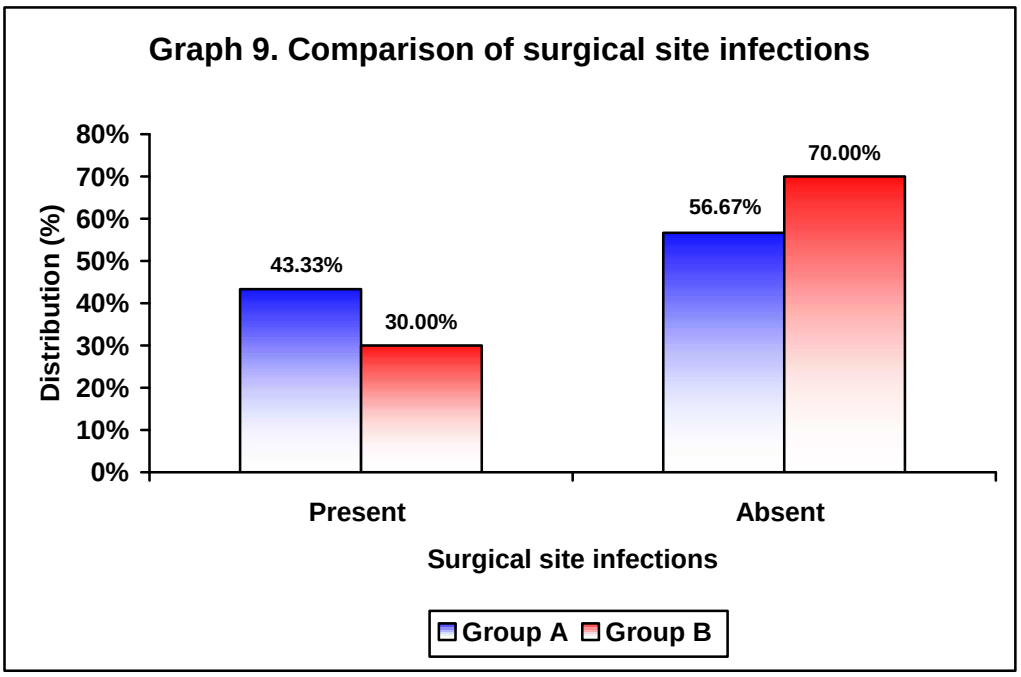
Findings	Findings	Group A (n=5)		Group B (n=2)		p value
		No	%	No	%	
Culture	Negative	3	60.00	1	50.00	1.000
	Positive	2	40.00	1	50.00	
	Total	**5**	**100.00**	**2**	**100.00**	
Organism	MRSA	1	20.00	1	50.00	0.671
	Pseduomonas aeruginosa	1	20.00	0	0.00	
	No growth	3	60.00	1	50.00	
	Total	**5**	**100.00**	**2**	**100.00**	

In patients with presence of discharge / pus on day ten, wound culture was positive in 40% of the patients among the patients of group A, compared to 50% in group B. Wound culture revealed 20% of the patients with MRSA in group A compared to 50% in group B (p=0.671).

Table 15. Comparison of surgical site infections

Surgical site infections	Group A (n=30)		Group B (n=30)	
	No.	%	No.	%
Present	13	43.33	9	30.00
Absent	17	56.67	21	70.00
Total	**30**	**100.00**	**30**	**100.00**

p = 0.284



In the present study, the surgical site infections were present in 43.33% of the patients in group A and 30% of the patients in group B. However this difference was statistically not significant (p=0.284)

DISCUSSION

Surgical site infection plays a pivotal role for prolonged treatment and further complications, increased health care costs as well as reduced quality of life after open abdominal surgery. They are believed to increase the risk of dying 2-11 fold,[85] with 77% of these deaths attributed directly to the infection.[86]

In the majority of surgical patients SSI was the consequence of almost all interventions until the late 19th century. “Irritative fever” was followed by purulent drainage from the incision and later as sepsis and oftentimes death. When Joseph Lister, in the late 1860s, introduced the principles of antisepsis postoperative infectious morbidity substantially decreased.[86] Ever since there have been substantial and successful efforts to further reduce the number of affected patients and severity of infections with various means: hemostasis, conservation of adequate blood supply, hypothermia prevention, atraumatic tissue handling, and infection control practices such as improved operating room ventilation, sterilization methods, and the use of antimicrobial prophylaxis.[87]

However, SSI remain one of the most frequent complications after any type of surgery, ranging as high as 26% depending on types of intervention and definitions of wound infection. This can possibly be attributed in part to the emergence of antibiotic-resistant micro-organisms, larger numbers of elderly surgical patients or those with a variety of chronic and immunocompromising conditions, and greater use of prosthetic implants and organ transplantation.[88]

Antimicrobial coated sutures have been amongst the suggestions to further reduce SSI incidence and there is a small number of studies evaluating triclosan coatings in surgery. Still the evaluation of new interventions in surgery remains a major challenge. Given the evidence of two recent single center historically controlled trials with a substantial reduction of more than 50% of wound infections by a triclosan coated suture in abdominal surgery some surgeons might question the demand for further trials. But treatment effectiveness is best evaluated in randomized controlled trials (RCT). The random allocation to one of the treatment groups is the only method to ensure that an observed effect can actually be attributed to the effectiveness of the investigated procedure and not known or unknown extraneous factors. Only RCTs generate data leading to the practice of evidence based medicine on a high level.[89] Hence this study was undertaken to compare the effectiveness of Triclosan coated PDS Plus for abdominal fascia closure in preventing SSI compared to non-coated PDS sutures after monolayer closure in open abdominal surgeries.

This present study was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2014 to December 2014. A total of 60 patients undergoing open abdominal surgeries (Clean contaminated) were divided into two groups of 30 each as Group A (primary fascial closure with PDS II sutures) and Group B (primary fascial closure with PDS plus sutures). Patients were monitored during the post operative period for the development of SSIs.

In the present study slight male preponderance was noted as male to female ratio in group A and group was 1.72:1 with 63.33% of the patients each in both the

groups. However the sex distribution pattern in group A and Group B were comparable (p=0.262). A prospective randomized controlled trial by Justinger et al.[82] to evaluate the role of PDS II and PDS plus reported similar sex distribution pattern.

In this study nearly half of the study population (50%) in group A was aged ≤ 30 years whereas 33.33% of the patients in group B were aged 31 to 40 years. The mean age in group A was noted as 39.90 ± 11.67 years and in group B it was 41.67 ± 16.08 years. However this difference was statistically not significant suggesting that age distribution the study population in group A and B was almost equally (p>0.050). However the mean age observed in the present study was low compared to a study by Justinger et al.[82]

In this study the clinical presentation including fever (p=0.165), vomiting (p=0.121) were comparable in patients with group A and group B. On systemic examination, the mean pulse rate, respiratory rate, systolic and diastolic blood pressure in group A and group B were comparable (p>0.050). Abdominal examination revealed tenderness in most of the patients which was also comparable in group A and group B (p=0.602). Furthermore small bowel resection was the commonest surgery which was performed in 26.67% of the patients each in group A and group B (p=0.172).

These findings suggest that, demographic, clinical and surgical characteristics of the patient in group A and group B did differ significantly (p>0.050) ruling out the possible bias in the results.

In the present study wound examination of day two revealed pain in 56.67%, local tenderness in 53.33%, redness and raised local temperature in 40% each, discharge in 36.67% and fever in 26.67% of the patients in group A. The frequency of these features was slightly low in patients with group B that is, pain was present in 40%, local tenderness in 36.67%, redness and raised local temperature in 33.33% each, discharge in 30% and fever in 20%. However the difference observed between group A and group B pertaining to the feature of pain, local tenderness, redness, raised local temperature, discharge and fever was statistically not significant (p=0.050).

During the examination on day six, 43.33% of the patients in group A had discharge as compared to 26.67% of the patients in group B (p=0.176). In group B, most of the patients that is, 30% had pain compared to 36.67% in group A (p=0.584). On day ten, discharge was present in 16.67% of the patients in group A compared to 6.67% in group B (p=0.212). In group B, redness was noted in 10% of the patients compared to 6.67% of the patients in group B (p=0.500).

These findings suggest that, group B where patients underwent primary fascial closure with PDS plus triclosan coated sutures resulted in lower rates of pain, local tenderness, redness, raised local temperature, discharge and fever compared to primary fascial closure with PDS II sutures but as the differences observed were statistically not significant (p>0.050).

In the present study among the patients with discharge / pus, rate of positive wound culture was 81.82% among the patients in group A on day two. This rate of positive wound culture on day six reduced to 46.15% and 40% on day ten. Among

the patients with group B, the rate of positive wound culture was 77.78% on day two which reduced to 62.50% on day six and 50%. Further, pseudomonas aeruginosa was the commonest isolate in group A and group B on day two as well as day six while MRSA was the commonest organism isolated on day ten. However, the rate of positive wound culture and organism profile in group A and group B were comparable ($p>0.050$). These findings suggest that, PDS plus triclosan coated sutures and PDS II sutures offer similar antimicrobial properties.

In the present study, frequency of surgical site infections was high (43.33%) in patients with group A compared to group B (30%) but the difference was statistically not significant ($p=0.284$). These findings suggest that PDS plus triclosan coated sutures have positive role in reduction of surgical site infection but not significant compared to PDS II sutures.

Wide range of published studies is available for toxicity profile of triclosan, antibacterial profile and clinical effectiveness of TCS. However, in contrast to our findings they have reported statistical significant results favoring TCS in reduction of risk of SSIs.

Daoud FC et al.[72] in 2014 analyzed the data from 15 RCTs totaling 4000 patients (TCS=2323) and NTCS=2477). Use of TCS was associated with a decrease in SSIs in selected patient populations (RR=0.67; P=0.00053), means 33% reduction in risk of developing SSIs. TCS showed highly statistical significant results in lowering risk of SSI. TCS was effective in clean (P=0.001), clean-contaminated (P=0.010), and contaminated incisions (P=0.026). SLR result was robust to the removal of three RCTs. SLR showed highly statistical significant results favoring

TCS in reduction of risk of SSI and robustness of clinical results - relative risk independent of confounding factors.

Edmiston CE et al.[73] in 2013 conducted a meta-analysis in response to recently published systematic reviews and meta-analysis which have suggested about no benefits of anti-microbial coated suture in reducing the Surgical Site Infections (SSI). Authors have highlighted poor selection of available RCT and low patient numbers for these meta-analyses. The primary endpoint of the systematic review was to determine the ratio of patients who developed an SSI in two comparative groups: closure with TCS versus NTCS sutures. Total 13 RCTs were selected, totaling 3568 patients (TCS=1654) and NTCS=1914). Use of TCS was associated with a decrease in SSIs in selected patient populations (fixed effect: RR = 0.734; P = 0.005; random-effect: RR = 0.693; P = 0.011), means 27-31% reduction in risk of developing SSIs.

Wang ZX et al.[74] in 2013 performed meta-analysis with 17 RCTs covering 3720 patients (TCS=1726) and NTCS=1994). Results favored TCS with a pooled RR of 0.70 (P <0.001) without statistical heterogeneity (P for Q test = 0.129, I2=29 per cent), means TCS provided significant advantage in reducing the rate of SSI by 30%. Subgroup analysis indicates statistical significant results of reduction in SSI by using TCS in adult patients, abdominal surgery and clean or clean contaminated wounds. The advantage of TCS over conventional sutures was consistent regardless of length of follow-up.

Justinger C et al.[53] has published large retrospective study of 2088 patients in mid laparotomy. The results showed the decrease in number of SSIs (TCS: 4.9%,

NTCS: 10.8%, P <0.001) for abdominal wall closure. However, this analysis of the use of triclosan coated sutures in laparotomy previously done by Justinger et al,[35] has limitations few limitations. In the sequential design that was employed over a period of 2 years, per definition internal validity cannot be assumed with certainty. It is not at all unlikely that over this relatively long period of time other factors in the patients' treatment might have changed and remained unrecorded but may have contributed to the reduction in SSI. Furthermore, with a control of PDS II® sutures in history the intervention group received Vicryl plus® sutures, a material different in structure (monofil versus braided) and resorption (210 versus 70 days). Braided and non braided sutures as well as rapidly absorbable and slowly absorbable ones appear to differ in bacterial adherence and interrupted rapid absorbable sutures increase the risk for development of an incisional hernia substantially according to the INLINE systematic review.[82]

In another prospective comparative study in transverse laparotomy for hepatobiliary resections (n=839), TCS showed significant reduction in SSI compared to NTCS arm (4.3% vs. 9.2%, P = 0.05).[79]

In spinal surgery, TCS found to be effective in reduction of wound infection (0.5% vs. 3.9%, p=0.020).[75]

A recent paper on gastric cancer surgery via midline laparotomy also showed the reduction of SSI cases in abdominal wall closure.[90]

Other prospective studies in digestive tract surgery, breast cancer surgeries,[91] abdominal surgeries,[92] and cardiac surgeries (sternal site infections),[93] TCS was found to be effective in minimizing the risk of development of SSI post-surgery.

Even though these study results suggest the use of triclosan-coated sutures in order to lower the risk for SSIs, two important aspects of triclosan use in health care products should be discussed. First, it has been shown that there is a risk of antimicrobial resistance to triclosan, including its use in topical products (e.g. cosmetics) where resistance to populations of S. aureus has been reported. This must be regarded as a major drawback in the use of triclosan as resistance to antibacterial substances represents a growing problem in modern medicine. On the other hand, if the use of conventional antibiotics in patients with SSI can be reduced by 40% with triclosan-coated sutures, this may balance or outweigh the disadvantages. The second issue is the long degradation time of triclosan and the potential risk for bioaccumulation in the environment.[82]

The findings of the present study showed comparable outcomes among the patients with primary fascial closure using PDS plus triclosan coated sutures as well as PDS II sutures in terms of SSIs, antimicrobial properties as well as other characteristics. This can be explained by the several fact. Firstly the present study was performed on patients undergoing clean contaminated open abdominal surgeries only while the latter studies have proved the efficacy of PDS plus triclosan coated sutures in different settings as a study by Justinger et al.[53] included only patients with laparotomy. Secondly the sample size of the present study was small (n=30 each) while other studies included large sample size.

The observations of the present study were similar to the results of Diener MK et al. in PROUD trial.[94] They evaluated effectiveness of triclosan-coated PDS Plus sutures for abdominal wall closure and reported that the occurrence of SSIs did not differ between the PDS Plus group (87 [14·8%] of 587) and the PDS II group

(96 [16·1%] of 598; OR 0·91, 95% CI 0·66–1·25; p=0·64). Serious adverse events also did not differ between the groups 146 of 583 (25·0%) patients treated with PDS Plus had at least one serious adverse event, compared with 138 of 602 (22·9%) patients treated with PDS II; p=0·39). PROUD trial concluded that, Triclosan-coated PDS Plus did not reduce the occurrence of surgical site infection after elective midline laparotomy.

Overall the results of this study showed triclosan-coated sutures that is PDS plus result in lower rate of SSI but no significant reduction was noted. Hence innovative, multifactorial strategies need to be developed and assessed in future trials to reduce surgical site infections.

CONCLUSION

The present study showed that, Triclosan coated PDS Plus sutures are as effective as PDS II sutures in reducing surgical site infection among the patients undergoing clean contaminated open abdominal surgeries. Further, Triclosan coated PDS Plus for abdominal fascia closure results in lower rate of pain, local tenderness, redness, raised local temperature, discharge and fever compared to primary fascial closure with PDS II sutures but does not reduce significantly. Also the antimicrobial resistance pattern of Triclosan coated PDS Plus sutures is comparable with PDS II sutures. Hence it may be concluded that, abdominal fascia closure using Triclosan coated PDS Plus sutures is as effective as non-coated PDS sutures after monolayer closure in clean contaminated open abdominal surgeries.

SUMMARY

Surgical site infections remain one of the most frequent complications in open abdominal surgery. This study compared the effectiveness of Triclosan coated PDS Plus and non coated PDS II after monolayer abdominal fascia closure in preventing SSI in open abdominal surgeries.

The randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum for the period of one year from January 2014 to December 2014. A total of 60 patients undergoing open abdominal surgeries (Clean contaminated) were enrolled. These patients were divided into two groups of 30 each based on the suture material used to close the abdominal fascia in monolayer as Group A (PDS II sutures) and Group B (PDS plus sutures).

Most of the patients were males in group A and B (63.33%) with males to female ratio of 1.72:1 (p=1.000). Nearly half of the study population (50%) was aged ≤ 30 years in group A compared to 33.33% in group B (p=0.262). The mean age in group A and group B was comparable (39.90±11.67 vs 41.67±16.08 years; p=0.687). Other pre-intervention characteristics including fever, vomiting, tenderness, vitals and type of surgery were comparable (p>0.050). On post operative day two, discharge was noted in 19 (63.33%) patients in group A and culture was positive in 9 (81.82%) of the patients with commonest organism being Escherichia coli and pseudomonas aeruginosa (3 patients each [27.27%]). In group B, discharged was noted in 21 (70%) patients of whom culture was positive in 7 (77.78%) patients with pseudomonas aeruginosa and commonest organism (22.22%). However, these

findings were comparable in group A and B (p>0.050). Similar trend was noted on day six as well as day ten. The other wound characteristics including pain, local tenderness, redness, raised local temperature, fever were comparable in group A and group B on day two as well as day six and ten (p=0.050). The surgical site infections were present in 43.33% of the patients in group A compared to 30% of the patients in group B (p=0.284).

Abdominal fascia closure with Triclosan coated PDS Plus sutures is as effective as non-coated PDS sutures after monolayer closure in clean contaminated open abdominal surgeries in the prevention of surgical site infections.

BIBLIOGRAPHY

1. Ananthakrishnan AN, Kanungo R, Kumar A, Badrinath S. Detection of extended spectrum beta lactamase producers among surgical wound infections and burns patients in JIPMER. Indian J Med Microbiol 2000; 18(4):160-5.

2. Brunicardi CF, Andersen DK. Schwartz's manual of surgery. 9th ed. New York, NY: McGraw-Hill; 2010

3. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. The Hospital Infection Control Practices Advisory Committee Guideline for Prevention of Surgical Site Infection, 1999. Special Report. Infection Control and Hospital Epidemiology 1999;20(4):247-78.

4. Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol 1992;13(10):606-8.

5. Weiser TG, Regenbogen SE, Thompson KD, Haynes AB, Lipsitz SR, Berry WR, et al. An estimation of the global volume of surgery: a modelling strategy based on available data. The Lancet 2008;372:139-44.

6. Magill SS, Edwards JR, Bamberg W, Beldavs ZG, Dumyati G, Kainer MA, et al. Multistate point-prevalence survey of health care-associated infections. New Engl J Med 2014;370(13):1198-208.

7. Mu Y, Edwards JR, Horan TC, Berrios-Torres SI, Fridkin SK. Improving risk-adjusted measures of surgical site infection for the national healthcare safety network. Infection Control Hospital Epidemiology 2011;32(10):970-86.

8. Prasannagupta. A study of comparison of infection rate among various surgical site infection cases in a tertiary care hospital. Int J Biol Med Res 2013;4(1):2905-9.

9. Razavi SM, Ibrahimpoor M, Kashani AS, Jafarian A. Abdominal surgical site infections: incidence and risk factors at an Iranian teaching hospital. BMC Surg 2005;5:2.

10. Kirkland KB, Briggs JP, Trivette SL, Wilkinson WE, Sexton DJ. The impact of surgical-site infections in the 1990s: attributable mortality, excess length of hospitalization, and extra costs. Infect Control Hosp Epidemiol 1999;20:725-30.

11. Astagneau P, Rioux C, Golliot F, Brücker G, INCISO Network Study Group. Morbidity and mortality associated with surgical site infections: results from the 1997–1999 INCISO surveillance. J Hosp Infect 2001;48: 267-74.

12. Merle V, Germain JM, Chamouni P, Daubert H, Froment L, Michot F, et al. Assessment of prolonged hospital stay attributable to surgical site infections using appropriateness evaluation protocol. Am J Infect Control 2000;28:109-15.

13. De Lissovoy G, Fraeman K, Hutchins V, Murphy D, Song D, Vaughn BB. Surgical site infection: incidence and impact on hospital utilization and treatment costs. Am J Infect Control 2009;37:387-97.

14. Chang WK, Srinivasa S, Morton R, Hill AG. Triclosan-Impregnated Sutures to Decrease Surgical Site Infections: Systematic Review and Meta-Analysis of Randomized Trials. Ann Surg 2012;255:854-9.

15. Rodeheaver GT, Kurtz LD, Bellamy WT, Smith SL, Farris H, Edlich RF. Biocidal braided sutures. Arch Surg 1983;118:322-7.

16. Gomez-Alonso A, Garcia-Criado FJ, Parreno-Manchado FC, Garcia-Sanchez JE, Garcia-Sanchez E, Parreno-Manchado A, et al. Study of the efficacy of coated Vicryl plus antibacterial suture (coated polyglactin 910 suture with triclosan) in two animal models of general surgery. J Infect 2007;54:82-8.

17. Katz S, Izhar M, Mirelman D. Bacterial adherence to surgical sutures: a possible factor in suture induced infection. Ann Surg 1981;194:35-41.

18. Gristina AG, Price JL, Hobgood CD, Webb LX, Costerton JW. Bacterial colonization of percutaneous sutures. Surgery 1985;98:12-9.

19. Sewlikar SA, Pillai RS, Mahajan NS, Desai AA. Triclosan coated sutures: an overview of safety and efficacy in reducing risk of surgical site infection. Int Surg J 2015;2(1):1-7.

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

21. Bryan PW. The Papyrus Ebers. London/Washington DC: Government Printing Office; 1883.

22. Cohen IK. A Brief History of Wound Healing. Yardley, Pa: Oxford Clinical Communications Inc; 1998.

23. Lister J. On a new method of treating compound fractures. Lancet 1867;1:326-9,387-9,507-9.

24. Humes DJ, Lobo DN. Antisepsis, asepsis and skin preparation. Surgery 2009;27:10.

25. Hemani ML, Lepor H. Skin preparation for the prevention of surgical site infection: which agent is best? Rev Urol 2009;11(4):190-5.

26. Singhal H. Wound infection – History. Available from: URL: http://emedicine.medscape.com/article/188988-overview Accessed on 24.07.2011.

27. Labarraque AG. The use of the chloride of soda and lime. Translated by Porter J. New Haven, CT: Baldwin and Treadway; 1829.

28. Gottrup F, Apelqvist J, Bjansholt T, Cooper R, Moore Z, Peters EJG, et al. Antimicrobials and non healing wounds: Evidence, controversies and Suggestions. J Wound Care 2013;22(5):S2-88.

29. Garner JS, Jarvis WR, Emori TG, Horan TC, Hughes JM. CDC definitions for nosocomial infections, 1988. Am J Infect Control 1988; 16:128-40.

30. McGeer A, Ashraf MS, Calder J, Crnich CJ, Crossley K, Drinka PJ, et al. Definitions of infection for surveillance in long-term care facilities. Am J Infect Control 1991;19:1-7.

31. Ramasubramanian V, Iyer V, Sewlikar S, Desai A. Epidemiology of healthcare acquired infection – An Indian perspective on surgical site infection and catheter related blood stream infection. Indian J Basic Applied Med Res 2014:3(4):46-63

32. Leaper DJ, van Goor H, Reilly J, Petrosillo N, Geiss HK, Torres AJ, Berger A. Surgical site infection - A European perspective of incidence and economic burden. Int Wound J 2004;1(4):247-73.

33. Fan Y, Wei Z, Wang W, Tan L, Jiang H, Tian L, et al. The incidence and distribution of surgical site infection in mainland China: a meta-analysis of 84 prospective observational studies. Sci Rep 2014;4:67-83.

34. CDC. Data from the National Hospital Discharge Survey. 2010. Available from: http://www.cdc.gov/nchs/data/nhds/4procedures/2010pro_numberpercentage.pdf. Access Date 28.06.2015

35. Magill SS, Hellinger W, Cohen J, Kay R, Bailey C, Boland B, et al., Prevalence of healthcare-associated infections in acute care hospitals in Jacksonville, Florida. Infection Control Hospital Epidemiology 2012;33(3):283-91.

36. Saxena A, Singh MP, Brahmchari S, Banerjee M. Surgical site Infection among postoperative patients of tertiary care centre in Central India - A

prospective study. Asian Journal of Biomedical and Pharmaceutical Sciences 2013;3(17):41-4.

37. Mahesh C B, Shivakumar S, Suresh BS, Chidanand SP, Vishwanath Y. A prospective study of surgical site infections in a teaching hospital. Journal of Clinical and Diagnostic Research 2010 October;4:3114-9.

38. Setty NH, Nagaraja MS, Nagappa DH, Giriyaiah CS, Gowda NR, Laxmipathy Naik RD. A study on Surgical Site Infections (SSI) and associated factors in a government tertiary care teaching hospital in Mysore, Karnataka. Int J Med Public Health 2014;4:171-5

39. Reichman DE, Greenberg JA. Reducing Surgical Site Infections: A Review. Rev Obstet Gynecol 2009;2:212-21.

40. Lilani SP, Jangale N, Chowdhary A, Daver GB. Surgical site infection in clean and clean-contaminated cases. Indian J Med Microbiol 2005;23: 249-52.

41. Anvikar AR, Deshmukh AB, Karyakarte RP, Damle AS, Patwardhan NS, Malik AK, et al. A one year prospective study of 3280 surgical wounds. Indian J Med Microbiol 1999;17:129-32.

42. Patel SM, Patel MH, Patel SD, Soni ST, Kinariwala DM, Vegad MM. Surgical site infections: Incidence and risk factors in a tertiary care hospital, Western India. Natl J Community Med 2012;3:193-6.

43. Ganguly. PS, Khan Y. Malik A. Nosocomial infection and hospital procedures. Indian J. common Med. 2000; 990-1014.

44. Rao AS, Harsha M. Postoperative wound infections. J Indian Med Assoc 1975;64:90-3.

45. Tripathy BS, Roy N. Post-operative wound sepsis. Indian J Surg 1984;47: 285-8.

46. Mayon-White RT, Ducel G, Kereselidze T, Tikomirov E. An international survey of the prevalence of hospital-acquired infection. J Hosp Infect 1988;11(lA):43-8.

47. Awad SS. Adherence to surgical care improvement project measures and post- operative surgical site infections. Surgical Infection (Larchmt) 2012;13(4):234-7.

48. Centre for Disease Control. Procedure associated manuals. 2015. Available from: URL: http://www.cdc.gov/nhsn/PDFs/pscManual /9pscSSIcurrent.pdf Access Date 18.06.2015

49. Fry DE. Surgical Site Infection:Pathogenesis and Prevention. Mexico: University of New Mexico School of Medicine; 2003.

50. National Nosocomial Infections Surveillance (NNIS) System. NNIS report, data summary from October 1986-April 1996, issued May 1996. A report from the NNIS System. Am J Infect Control 1996;24(5):380-8.

51. Cruse PJ, Foord R. The epidemiology of wound infection. A 10-year prospective study of 62,939 wounds. Surg Clin North Am 1980;60(1):27-40.

52. Marco F, Vallez R, Gonzalez P, Ortega L, de la Lama J, Lopez-Duran L. Study of the efficacy of coated Vicryl plus antibacterial suture in an animal model of orthopedic surgery. Surg Infect (Larchmt) 2007;8:359-65.

53. Justinger C, Moussavian MR, Schlueter C, Kopp B, Kollmar O, Schilling MK. Antibacterial [corrected] coating of abdominal closure sutures and wound infection. Surgery 2009;145:330-4.

54. Geiger D, Debus ES, Ziegler UE, Larena-Avellaneda A, Frosch M, Thiede A, et al. Capillary activity of surgical sutures and suture-dependent bacterial transport: a qualitative study. Surg Infect (Larchmt) 2005;6:377-83.

55. Leaper D, McBain AJ, Kramer A, Assadian O, Sanchez JL, Lumio J, et al. Healthcare associated infection:novel strategies and antimicrobial implants to prevent surgical site infection. Ann R Coll Surg Engl 2010; 92:453-8.

56. Ming X, Rothenburger S, Yang D. In vitro antibacterial efficacy of MONOCRYL plus antibacterial suture (Poliglecaprone 25 with triclosan). Surg Infect (Larchmt) 2007;8:201-8.

57. Edmiston CE, Seabrook GR, Goheen MP, Krepel CJ, Johnson CP, Lewis BD, et al. Bacterial adherence to surgical sutures:can antibacterial-coated sutures reduce the risk of microbial contamination? J Am Coll Surg 2006; 203:481-9.

58. Cooney CM. Triclosan comes under scrutiny. Environ Health Perspect 2010;118:A242.

59. Storch ML, Rothenburger SJ, Jacinto G. Experimental efficacy study of coated Vicryl plus antibacterial suture in guinea pigs challenged with Staphylococcus aureus. Surg Infect (Larchmt) 2004;5:281-8.

60. Uff CR, Scott AD, Pockley AG, Phillips RK. Influence of soluble suture factors on in vitro macrophage function. Biomaterials 1995;16:355-60.

61. Howell JJ. Chlorhexidine and Suture Materials. Br Med J 1965;1:449-50.

62. Bhargava HN, Leonard PA. Triclosan:applications and safety. Am J Infect Control 1996;24:209-18.

63. Rodricks JV, Swenberg JA, Borzelleca JF, Maronpot RR, Shipp AM. Triclosan:a critical review of the experimental data and development of margins of safety for consumer products. Crit Rev Toxicol 2010;40:422-84.

64. Fleck T, Moidl R, Blacky A, Fleck M, Wolner E, Grabenwoger M, et al. Triclosan-coated sutures for the reduction of sternal wound infections:economic considerations. Ann Thorac Surg 2007;84:232-6.

65. Barbolt TA. Chemistry and safety of triclosan, and its use as an antimicrobial coating on coated vicryl* plus antibacterial suture (coated polyglactin 910 suture with triclosan). Surg Infect (Larchmt). 2002;3(Suppl 1):S45-53

66. Leaper D, Assadian O, Hubner NO, McBain A, Barbolt T, Rothenburger S, et al. Antimicrobial sutures and prevention of surgical site infection: assessment of the safety of the antiseptic triclosan. Int Wound J 2011; 8(6):556-66.

67. SenGupta M, Banerjee D, Sengupta M, Sarkar S, Nag S, Singh M. In vitro efficacy of triclosan coated polyglactin 910 suture against common bacterial pathogen causing surgical site infection. Int J Infect Control 2014;10(2):1-6.

68. Edmiston CE, Seabrook GR, Goheen MP, Krepel CJ, Johnson CP, Lewis BD, et al. Bacterial adherence to surgical sutures: can antibacterialcoated sutures reduce the risk of microbial contamination? J Am Coll Surg 2006; 203(4):481-9.

69. Rothenburger S, Spangler D, Bhende S, Burkley D. In vitro antimicrobial evaluation of coated vicryl plus antibacterial suture (coated polyglactin 910 with triclosan) using zone of inhibition assays. Surg Infect (Larchmt). 2002;3(Suppl 1):S79-87.

70. Ming X, Rothenburger S, Nichols MM. In vivo and in vitro antibacterial efficacy of PDS plus (polidioxanone with triclosan) suture. Surg Infect (Larchmt). 2008;9(4):451-7.

71. Ming X, Nichols M, Rothenburger S. In vivo antibacterial efficacy of MONOCRYL plus antibacterial suture (Poliglecaprone 25 with triclosan). Surg Infect (Larchmt). 2007;8(2):209-14.

72. Daoud FC, Edmiston CE, Leaper D. Metaanalysis of prevention of surgical site infections following incision closure with triclosan-coated sutures: robustness to new evidence. Surg Infect (Larchmt). 2014 Jun;15(3):165-81.

73. Edmiston CE, Daoud FC, Leaper D. Is there an evidence-based argument for embracing an antimicrobial (triclosan)-coated suture technology to reduce

the risk for surgical-site infections? A metaanalysis. Surgery 2014; 155(2):362-3.

74. Wang ZX, Jiang CP, Cao Y, Ding YT. Systematic review and meta-analysis of triclosan-coated sutures for the prevention of surgical-site infection. Br J Surg 2013;100(4):465-73.

75. Ueno M, Saito W, Yamagata M, Imura T, Inoue G, Nakazawa T, et al. Triclosan-coated sutures reduce wound infections after spinal surgery: a retrospective, nonrandomized, clinical study. Spine J 2013;27:pii:S1529.

76. Galal I, El-Hindawy K. Impact of using triclosan-triclosanantibacterial sutures on incidence of surgical site infection. Am J Surg 2011;202(2): 133-8.

77. Wignall GR, Goneau LW, Chew BH, Denstedt JD, Cadieux PA. The effects of triclosan on uropathogen susceptibility to clinically relevant antibiotics. J Endourol 2008;22:2349-56.

78. Ford HR, Jones P, Gaines B, Reblock K, Simpkins DL. Intraoperative handling and wound healing:controlled clinical trial comparing coated VICRYL plus antibacterial suture (coated polyglactin 910 suture with triclosan) with coated VICRYL suture (coated polyglactin 910 suture). Surg Infect (Larchmt) 2005;6:313-21.

79. Justinger C, Schuld J, Sperling J, Kollmar O, Richter S, Schilling MK. et al. Triclosan-coated sutures reduce wound infections after hepatobiliary surgery-a prospective non-randomized clinical pathway driven study. Langenbecks Arch Surg 2011;396(6):845-50.

80. Chen SY, Chen TM, Dai NT, Fu JP, Chang SC, Deng SC, et al. Do antibacterial-coated sutures reduce wound infection in head and neck cancer reconstruction? Eur J Surg Oncol 2011;37:300-4.

81. Deliaert AE, Van den Kerckhove E, Tuinder S, Fieuws S, Sawor JH, Meesters-Caberg MA, et al. The effect of triclosan-coated sutures in wound healing. A double blind randomised prospective pilot study. J Plast Reconstr Aesthet Surg 2009;62:771-3.

82. Justinger C, Slotta JE, Ningel S, Gräber S, Kollmar O, Schilling MK. et al. Surgical site infection after abdominal wall closure with triclosan-impregnated polydioxanone sutures: Results of a randomized clinical pathway facilitated trial. Surgery 2013;154:589-95.

83. Laas E, Poilroux C, B´ezu C, Coutant C, Uzan S, Rouzier R, et al. Antibacterial-Coated Suture in Reducing Surgical Site Infection in Breast Surgery: A Prospective Study. International J Breast Cancer 2012; Article ID 819578, 8 pages.

84. Thimour-Bergström L, Roman-Emanuel C, Scherstén H, Friberg Ö, Gudbjartsson T, Jeppsson A. Triclosan-coated sutures reduce surgical site infection after open vein harvesting in coronary artery bypass grafting patients: a randomized controlled trial. Eur J Cardio-Thoracic Surgery 2013;44:931-8.

85. Engemann JJ, Carmeli Y, Cosgrove SE, Fowler VG, Bronstein MZ, Trivette SL, Briggs JP, Sexton DJ, Kaye KS. Adverse clinical and economic outcomes attributable to methicillin resistance among patients with

Staphylococcus aureus surgical site infection. Clin Infect Dis. 2003;36:592–598.

86. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. Infect Control Hosp Epidemiol. 1999;20:250–278.

87. Anderson DJ, Sexton DJ, Kanafani ZA, Auten G, Kaye KS. Severe surgical site infection in community hospitals: epidemiology, key procedures, and the changing prevalence of methicillin-resistant Staphylococcus aureus. Infect Control Hosp Epidemiol. 2007;28:1047–1053.

88. Hedrick TL, Heckman JA, Smith RL, Sawyer RG, Friel CM, Foley EF. Efficacy of protocol implementation on incidence of wound infection in colorectal operations. J Am Coll Surg. 2007;205:432–438.

89. Heger U, Voss S, Knebel P, Doerr-Harim C, Neudecker J, Schuhmacher C, et al. Prevention of abdominal wound infection (PROUD trial, DRKS00000390): study protocol for a randomized controlled trial. Trials. 2011;12:245.

90. Jung KH, Oh SJ, Choi KK, Kim SM, Choi MG, Lee JH, et al. Effect of triclosan-coated sutures on surgical site infection after gastric cancer surgery via midline laparotomy. Ann Surg Treat Res. 2014 Dec;87(6):311-8.

91. Laas E, Poilroux C, Bézu C, Coutant C, Uzan S, Rouzier R, et al. Antibacterial-coated suture in reducing surgical site infection in breast surgery: a prospective study. Int J Breast Cancer. 2012;2012:819578.

92. Justinger C, Slotta JE, Schilling MK. Incisional hernia after abdominal closure with slowly absorbable versus fast absorbable, antibacterialcoated sutures. Surgery. 2012 Mar;151(3):398-403.

93. Fleck T, Moidl R, Blacky A, Fleck M, Wolner E, Grabenwoger M, et al. Triclosan-coated sutures for the reduction of sternal wound infections: economic considerations. Ann Thorac Surg. 2007;84(1):232-6.

94. Diener MK, Knebel P, Kieser M, Schüler P, Schiergens TS, Atanassov V, et al. Effectiveness of triclosan-coated PDS Plus versus uncoated PDS II sutures for prevention of surgical site infection after abdominal wall closure: the randomised controlled PROUD trial. Lancet 2014;384(9938):142-52.

ANNEXURE I – CONSENT FORM

TITLE OF RESEARCH STUDY: ONE YEAR RANDOMIZED CONTROLLED TRIAL TO COMPARE THE EFFECTIVENESS OF TRICLOSAN COATED PDS PLUS VERSUS UNCOATED PDS SUTURES IN PREVENTION OF SURGICAL SITE INFECTIONS AFTER MONOLAYER CLOSURE IN OPEN ABDOMINAL SURGERIES

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Introduction and purpose

You are requested to participate in a study that is an attempt to find out the effectiveness of PDS sutures versus PDS PLUS sutures in preventing the surgical site infection during open abdominal surgeries. Postoperative surgical site infections (SSI) are one of the most common complications after laparotomy. Many strategies have been developed to reduce this burden such as the introduction of less invasive procedures (e.g. laparoscopic interventions) but still most of the intra-abdominal procedures are performed as open surgeries. SSIs increase morbidity and mortality as well as impose a financial burden worldwide. Apart from the financial cost, other consequences for the patient such as pain, reduced quality of life, time off work, and loss of productivity are difficult to quantify. Today 75% of all SSI are superficial incisional infections whereas the remaining 25% are deep incisional or deep organ

space SSI. The causes of SSIs are multifactorial, but, crucially, SSIs are preventable. PDS Plus is a triclosan coated suture materials which has antimicrobial properties and previous studies have shown some benefits over the uncoated PDS sutures.

In an effort to avoid the above mentioned problems, this study has been undertaken to evaluate the effectiveness of Triclosan coated PDS Plus versus uncoated PDS in preventing the surgical site infection in monolayer abdominal closure in open abdominal surgeries.

Dr. ***** ******, Post Graduate in Department of Surgery, will conduct this study under the direct supervision and guidance of Dr. **** *******, Associate Professor, Department of Surgery, J. N. Medical College, Belgaum.

You need to be eligible, meeting all the selection criteria to participate in this study. You should be willing to provide information about yourself. 60 subjects will be enrolled in this study who will then be randomized in either of 2 groups (details below).

Procedure

If you agree to participate in this study, you will be randomly allotted into a group (A or B) and accordingly receive either PDS or PDS Plus sutures for primary abdominal fascia closure. Post operatively, the wound is inspected on 2nd, 6th and 10th day to look for surgical site infection.

Benefits

Studies have some proven benefits with the use of PDS PLUS sutures over the regularly used PDS sutures.

Risk involved

There is no additional risk compared to the standard method of treatment.

Compensation

Taking part in the study will not affect the cost of treatment i.e. it will be similar to the cost of standard procedure. In the event that you become injured as a result of taking part in this study, treatment will be offered to you or you will be given information about where to receive medical care: but you/your insurance company will be responsible for the costs. However, no reimbursement, compensation or free medical care will be given.

Confidentiality

Every effort will be made to protect the confidentiality of the information you provide. This means that the researchers will not let anyone, not a part of the study, see the information you provide. Only Dr. ***** ******** and Dr. ***** ******* will have access to the information collected. Results of this study may be published but your name will not be revealed.

Voluntary participation / withdrawal

Taking part in this study is voluntary; you may choose not to enroll in this study. Your decision will not change the present or future health care services offered to you at KLES Dr. Prabhakar Hospital, Belgaum. The alternative that you have is to undergo the traditional procedure that is carried out in KLES Hospital.

If you have any queries about the study, you may contact Dr. **** ****** (Mobile No. ***** ******); or Dr. ***** ****** (Mobile No. ***** *****). If you need any further information regarding your rights as a study participant, you may also contact Dr. **** *******, Chairman of Institutional Ethics Committee, JNMC, Belgaum (Mobile No. +***** *******).

CONSENT TO PARTICIPATE IN THE STUDY

I Mr./Ms._____ have been explained about the research study, the need of the study, the intervention, their risks, benefits and alternatives available in my own vernacular language.

I voluntarily agree to participate in this study by signing up this form below. I understand that I may withdraw at any time from this study. I have been given adequate time to clarify my doubts about the study and my rights as a study participant.

My signature / thumb impression below indicates that I have read or information in the consent been read to me including the risks and benefits and have cleared my doubts.

Name of participant:

Signature/LTI:

Name of legally authorized

Signature/LTI:

Representative (if applicable):

Relationship with participant:

Name of witness:

Signature:

Name of investigator:

Signature

Date:

Place:

ANNEXURE II – PROFORMA

TITLE: ONE YEAR RANDOMIZED CONTROLLED TRIAL TO COMPARE THE EFFECTIVENESS OF TRICLOSAN COATED PDS PLUS VERSUS UNCOATED PDS SUTURES IN PREVENTION OF SURGICAL SITE INFECTIONS IN OPEN ABDOMINAL SURGERIES.

In Patient Number:

Name:

Age:

Sex:

Address:

Ward:

Education:

Religion:

Marital Status:

Occupation:

Socio-Economic Status:

History

Details

Associated features and duration

Fever:

Vomiting:

Other:

Previous history of use of Opioids / NSAIDs/steroids:

Past History:

Personal History:

Family History:

General physical examination

Built and Nourishment:

Weight:

Pallor: Icterus: Cyanosis:

Clubbing: Edema Lymphadenopathy:

Vital Signs

PR: /min; BP: mmHg; RR: /min;

Temp:

Systemic examination

Per Abdomen examination:

Respiratory System:

Central Nervous System:

Cardio-Vascular System:

Postoperative wound assessment

Day 2: Day 6: Day 10:

Discharge from surgical site	Present	Absent
Pain:		
Local tenderness:		
Fever (If present, temperature in Fahrenheit):		
Redness:		
Increased local temperature:		
Culture and Sensitivity of the discharge:		
Result of culture and sensitivity, if sent		

ANNEXURE III – KEY TO MASTER CHART

-	-	Absent
+	-	Present
A	-	Absent
BP	-	Blood pressure
BS	-	Bowel sounds
CNS	-	Central nervous system
CVS	-	Cardiovascular system
E. coli	-	*Escherichia coli*
F	-	Female
GA	-	General anaesthesia
IP	-	In patient
M	-	Male
mm Hg	-	Millimeters of mercury
MRSA	-	*Methicillin-resistant Staphylococcus aureus*
N	-	Normal
NSAIDS	-	Non steroidal anti-inflammatory drugs
NT	-	Non tender
P	-	Present
Post op	-	Post operative
S	-	Soft
T	-	Tenderness