PROSPECTIVE COMPARATIVE STUDY OF COMPLICATIONS OF LAPAROTOMY WOUND IN ELECTIVE AND EMERGENCY SURGERY

BY

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In partial fulfillment

of the requirements for the degree of

M. S.

in

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Under the guidance of

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Date: ***Dr. SOMANI RUSHABHKUMAR C.***

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LIST OF ABBREVATIONS USED

BP	-	Blood Pressure
h/o	-	History of
i.e	-	that is
F	-	Frequency
No	–	Number
PR	-	Pulse Rate
t	-	student’s t
Chi Sq	-	Chi square test
DM	-	diabetes mellitus
ASA	-	American Society of Anesthesiology
SSI	-	Surgical Site Infection

ABSTRACT

Background:

Postoperative wound complications are of common occurrence. The incidence of postoperative wound infections ranging from <2% to 38%. Based on NNIS (National Nosocomial Infections Surveillance)system reports, surgical site infections are the third most frequently reported nosocomial infections among hospitalized patient[1].

Aim is to compare the complications of laparotomy wound in elective and emergency surgery and to study the various determinants affecting it.

Materials And Methods:

It is a prospective randomized study of 72 patients undergoing emergency and elective laparotomy. Postoperative wound complications and various factors effecting it are compared between emergency and elective surgery.

Results:

The study was conducted on 72 patients, aged between 13-80 years, who underwent major elective and emergency laparotomy surgery in Shri B.M. Patil Medical College ,Hospital and Research Centre Bijapur, between the period October 2008 - May 2010.

Among the 72 patients, 5 (14%) out of 36 patients were in elective laparotomy and 9 (25%) out of 36 patients were in emergency laparotomy who developed complications.

Conclusion:

In conclusion, laparotomy wound complications are multifactorial, This study demonstrated no significant increase in incidence of postoperative tissue and wound complications in emergency(25%) and elective surgery(14%) p=0.08. It mainly depends on higher ASA score ,anemia and higher wound class who are more likely to be associated with development of wound complications.

Key words: Wound Complications, SSI, Risk factors.

LIST OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF PHOTOGRAPHS

INTRODUCTION

Postoperative wound complications are of common occurrence. The incidence of postoperative wound infections ranging from <2% to 38%. Based on NNIS (National Nosocomial Infections Surveillance)system reports, surgical site infections are the third most frequently reported nosocomial infections among hospitalized patient[1]

Surgical complications remain a frustrating and difficult aspect of operative treatment of patient regardless of how technically gifted and capable surgeon, will deal with postoperative wound complications.

Despite significant improvement in technology, postoperative vigilance, wound infections continue to occur. In addition to increased morbidity, mortality, loss of work productivity, disruption of family life, stress to employer, society and increased financial burden to health care system in general

Postoperative wound infections range in severity from the most minimal stitch abscess to the extremely virulent infection leading to postoperative wound infections generalized septicaemia, wound dehiscence or death in some cases.

So the study of this aspect of laparotomy wound complications and surgical site infections with a view to identify the factors causing wound infections, microorganisms affecting and its antibiotic sensitivity will not only reduce the post operative morbidity in these patients but also will result in an immense cost benefit, hospital stay to the patient and to the institution

AIMS AND OBJECTIVES

Aim Of The Study :

Comparative study of complications of laparotomy wound in elective and emergency surgery.

Objectives Of The Study :

Comparison of complications of laparotomy wound in elective and emergency surgery and various determinants affecting it.

The following determinants will be taken into consideration in formulating the risk index in our patients :

1. Age
2. ASA score
3. Systemic diseases eg: diabetes mellitus, severe anemia etc
4. Duration of postoperative stay in the hospital wards

REVIEW OF LITERATURE

Definitions

In order to discuss wound infections rationally, certain terms need to be defined. Although wounds that drain creamy pus are definitely infected and those that are closed and heal primarily are definitely not infected, an intermediate category of wounds may be considered "possibly" or "probably" infected. With the infection rate for clean wounds being indeterminate categories can comprise a significant proportion of wounds that are not clearly infected or uninfected and may skew data in any study of infection rates. the term "surgical wound" was internationally replaced with "surgical site" to include infections arising after surgery that were in organ spaces deep to the skin and soft tissue, such as the peritoneum and bone

The identification of SSI involves interpretation of clinical and laboratory findings, and it is crucial that a surveillance program use definitions that are consistent and standardized; otherwise, inaccurate or uninterpretable SSI rates will be computed and reported. The CDC's NNIS system has developed standardized surveillance criteria for defining SSIs. By these criteria, SSIs are classified as being either incisional or organ/space. 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).[1,2]

Centers for Disease Control and Prevention Criteria for Defining a Surgical Site Infection

Superficial Incisional

Infection less than 30 days after surgery

Involves skin and subcutaneous tissue only, *plus* one of the following:

- Purulent drainage
- Diagnosis of superficial surgical site infection by a surgeon
- Symptoms of erythema, pain, local edema

Deep Incisional

Less than 30 days after surgery with no implant and soft tissue involvement

Infection less than 1 year after surgery with an implant; involves deep soft tissues (fascia and muscle), *plus* one of the following:

- Purulent drainage from the deep space but no extension into the organ space
- Abscess found in the deep space on direct or radiologic examination or on reoperation
- Diagnosis of a deep space surgical site infection by the surgeon
- Symptoms of fever, pain, and tenderness leading to dehiscence of the wound or opening by a surgeon

Organ Space

Infection less than 30 days after surgery with no implant

Infection less than 1 year after surgery with an implant and infection; involves any part of the operation opened or manipulated, *plus* one of the following:

- Purulent drainage from a drain placed in the organ space
- Cultured organisms from material aspirated from the organ space
- Abscess found on direct or radiologic examination or during reoperation
- Diagnosis of organ space infection by a surgeon

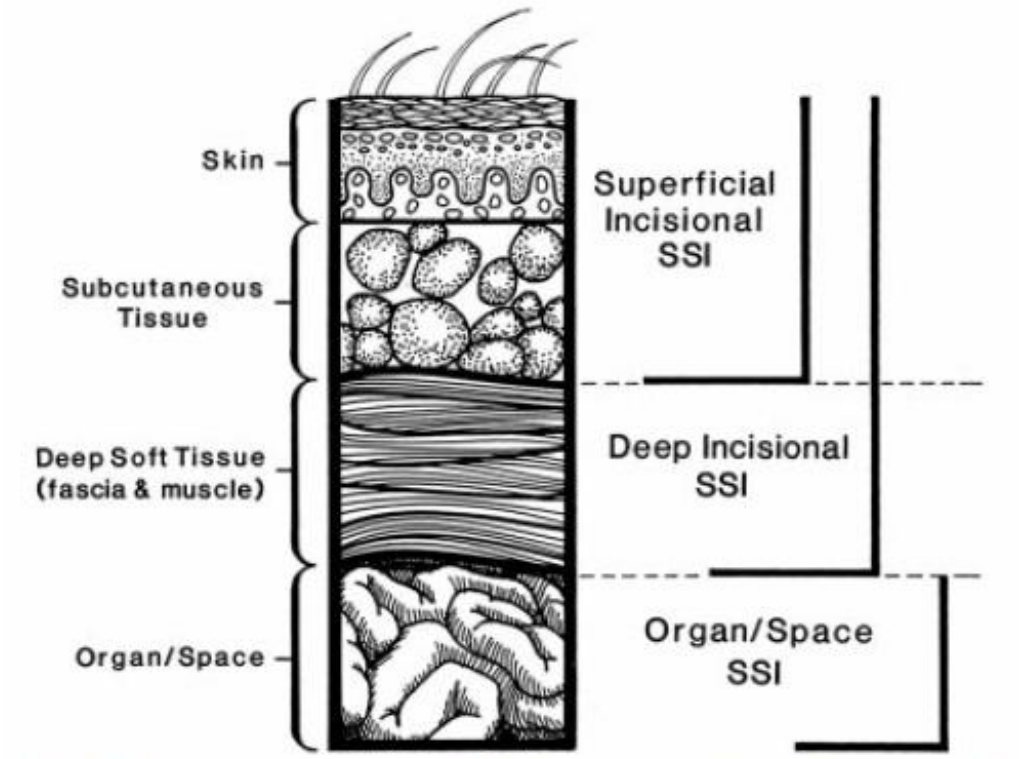


Figure 3: Cross-section of abdominal wall depicting CDC classifications of SSI

Definitions was put forth in 1964 by the National Research Council, Ad Hoc Committee on Trauma, to help predict the probability of wound infection based on the degree of intraoperative bacterial contamination. These definitions, a modified version of which is given, have repeatedly been shown to have a strong association with wound infections.

Table 15-3 -- Classification of Surgical Wounds

CATEGORY	CRITERIA	INFECTION RATE
Clean	No hollow viscus entered	1%-3%
	Primary wound closure	
	No inflammation	
	No breaks in aseptic technique	
	Elective procedure	
Clean-contaminated	Hollow viscus entered but controlled	5%-8%
	No inflammation	
	Primary wound closure	
	Minor break in aseptic technique	
	Mechanical drain used	
	Bowel preparation preoperatively	
Contaminated	Uncontrolled spillage from viscus	20%-25%
	Inflammation apparent	
	Open, traumatic wound	
	Major break in aseptic technique	
Dirty	Untreated, uncontrolled spillage from viscus	30%-40%
	Pus in operative wound	
	Open suppurative wound	
	Severe inflammation	

HISTORICAL REVIEW

Those who cannot remember the past are condemned to repeat it" George totayana. 1992.[3]

Early in the history of man, there was recognition of the interplay between Stinds, infection, and surgical manipulation. In fact, virtually all wounds became infected and infection was associated with high mortality. Wounds were treated by early physicians who were also surgeons. Treatment was based on trial and error and individual physician experience, yet many forms of effective therapy that varied for different cultures were discovered.

The early Egyptians recognized some form of circulation of the blood based on the doctoring of wounds. In addition, some primitive remedies such as the use of pulverized malachite or honey in wounds may have been extremely effective, as noted by the modern day experiments of Manjo. Sushrutha "father of Indian surgery" had also emphasized on the "prevention" of wound infection in his ancient scripts. The Greeks and Romans employed a. variety of remedies that included the use of red wine, poultices of herbs, and other compounds that may have had antibacterial properties. They also were proponents of "laudable pus/' since infection was so common that it was considered the norm after wounding.[3]

There have been two phases of intense revolutionary development in the means employed by surgeons against infections. The first of these two phases was centered on discovery of causes of infections and methods of its prevention. The great names associated with this phase are those of the fathers of bacteriology such as Pasteur, Robert

Koch, and Joseph Lister. Second phase, was that of effective systemic treatment of the same. This phase is associated great names of Domagk and Florey.

The development of bacteriology as a discipline dates from the time of Louis Pasteur (1822-95). He introduced techniques of sterilization that resulted in the development of steam sterilizer, hot air oven and autoclave. He also established the differing growth needs of different bacteria.[4]

Robert Koch(1843-1910) in Germany perfected bacteriological techniques during his studies on the culture and characters of the anthrax bacillus (1876). He introduced staining techniques and methods of obtaining bacteria in pure culture using solid media.[4]

Lord Lister (1827-1912), the Father of Antiseptic surgery revolutionized the science of surgery by introducing the antiseptic, and aseptic surgical techniques in operative and post operative cases. He chanced upon the antiseptic properties of carbolic acid, which had already been strongly recommended by Francois Jules, Lamaire (1860), for treatment of surgical sepsis. Lister first employed carbolic acid dressings, with tremendous success in dealing with compound fractures. He then crystallized his work and presented them in his renowned paper on "The antiseptic principles in practice of surgery", before the British medical association, in Dublin. Lister virtually brought down the mortality of surgery due to infections from 45% to 15%, a tremendous achievement by any standards, present or past

Adolfneubar introduced metal instruments and established the first aseptic hospital in 1883. Halsted, was the first to use rubber gloves (1890) and he advocated

gentleness and fineness in the techniques of surgical operations. Berger, from Paris, in 1897 was probably the first to adopt the use of cap, gown, and facemask as suggested by bacteriologist Flugge.

Willis McDonald was one of the first persons to fix accountability for the development of infection in clean operative wounds on the doctors and nurses. He pointed out that a fine sprays of infective saliva expelled from the mouth during conversation. He further observed that visitors to operations were a constant menace to surgical operations. In their anxiety to see the surgical procedures, ask questions, cough near the table and bring large quantities of microscopic dirt on their shoes to the operating suite. He took cultures of the air in the operating room and demonstrated that the number of visitors present in the operating room influenced the number of colonies on the plate.

In 1926, Meleny demonstrated the necessity of masking adequately the nose as well as the mouth of the surgeon and his team including the anesthetists. Meleny thus proposed that adequate sterilization of suture materials is necessary for effective wound healing and prevention of SSI.

AETIOPATHOLOGY

It is almost axiomatic that injury is followed by inflammation. An understanding of the nature mechanisms, and consequences of inflammation is important to the surgeon i.e surgical procedure results in an inflammatory reaction. Surgeon who understands, the nature and mechanism of this reaction to injury lies within his power the ability to minimize the adverse consequences, and to utilize its reaction to the benefit of the patient

Inflammation resulting from trauma may initially appear to differ from that resulting from bacterial infection or from physical agents such as heat, cold, and radiant energy. This is only apparent, the basic response is the same regardless of the initiating factor. Injury triggers an organized and complex cascade of cellular and biochemical events that result in a healed wound.

Physiology of wound healing[6,7,8,9,10]

The body's ability to replace injured or dead cells and to repair tissues after inflammation is critical to survival. The repair of tissue damage caused by surgical resection wounds and diverse types of chronic injury can be broadly separated into two processes, regeneration and healing.

Regeneration results in restitution of lost tissues; healing may restore original structures but involves collagen deposition and scar formation. Tissues with high proliferation capacity such as haemetopoietic system and the epithelia of the skin and gastrointestinal tract, renew themselves continuously and can regenerate after injury as long as the stem cells of these tissues are not destroyed.

Superficial wounds, such as a cutaneous wound that only damages the epithelium can heal by epithelial regeneration. Incisional and excisional skin wounds that damage the dermis heal through formation of a collagen scar.

Extracellular matrix scaffolds are essential for wound healing because they provide the framework for cell migration and maintain the correct cell polarity for the reassembly of multilayer structures. Furthermore cells in the extracellular matrix such as fibroblasts, macrophages and other cell types are the source of agents that are critical for tissue repair.

Healing is a fibro-proliferative response that "patches" rather than restores a tissue. It is a complex but orderly phenomenon involving a number of processes.[19]

1. Induction of an inflammatory process in response to the initial injury, with removal of damaged and dead tissue
2. Proliferation and migration of parenchymal and connective tissue cells
3. Formation of new blood cells(angiogenesis) and granulation tissue
4. Synthesis of extracellular matrix proteins and collagen deposition
5. Tissue remodeling
6. wound contraction
7. Acquisition of wound strength

Not all of the above mentioned events occur in every repair reaction.

Forms of healing[9]

Surgeons customarily divide types of wound healing into first and second "intention". First intention (primary) healing occurs when tissue is cleanly incised and reapproximated and repair occurs without complication.

Second intention (secondary) healing occurs in open wounds through the formation of granulation tissue. Granulation tissue is the red, granular, moist tissue that appears during healing of the open wounds. Microscopically it contains new collagen, blood vessels, fibroblasts, and inflammatory cells, especially macrophages. Covering of this tissue is then followed by spontaneous regression of the epithelial cells. Most infected wounds and burned tissue heal by the way of second intention.

The nature of repair[9]

In a broader sense, the nature of repair has been depicted schematically.

As this topic is centered on laparotomy wounds and infections, only healing of a surgical incision is described here.

The surgical incision causes death of a limited number of epithelial cells and connective tissue cells as well as disruption of epithelial basement membrane continuity. The narrow incisional space immediately fills with clotted blood containing fibrin and blood cells; dehydration of the surface clot form the well known scab that covers the wound.

Within 24 hours, neutrophils appear at the margins of the incision, moving towards the fibrin clot. The epidermis at its cut edges, thickens as a result of mitotic activity of the basal cells, and within 24 hours to 48 hours, spurs of epithelial cells from the edges both migrate and grow along the cut margins of the dermis, depositing basement membrane components as they move. They fuse in the midline beneath the scab, thus producing a continuous, albeit, thin epithelial layer.

By day 3, the neutrophils have largely been replaced by macrophages. Granulation tissue progressively invades the incision space. Collagen fibers are now present at the margins of the incision, but at first they are vertically oriented and do not bridge the incision. Epithelial proliferation continues and hence the epidermal covering layer is thickened.

By day 5, the incisional space is filled with granulation tissue. Neovascularization is maximal. Collagen fibrils become more abundant and start bridging the incision. The epidermis recovers its thickness, and differentiation of surface cells yields a mature epidermal architecture with surface keratinization.

During the second week, there is continued accumulation of collagen and proliferation of fibroblasts. The leukocytic infiltrate, edema, and increased vascularity have largely disappeared. At this time, the long process of blanching begins, accomplished by the increased accumulation of collagen within the incisional scar and by regression of vascular channels.

By the end of first month, the scar comprises a cellular connective tissue devoid of inflammatory infiltrate, covered now by intact epidermis, the dermal appendages that have been destroyed by the line of incision are permanently lost. The tensile strength of the wound increases thereafter, but it may take months for the wounded area to attain its maximal strength.

When there is more extensive loss of cells and tissue, as occurs in infarction, inflammatory ulceration, abscess formation and surface wounds creating large defects, the reparative process is more complicated. The common denominator in all these situations is a large tissue defect that must be filled. Regeneration of parenchymal cells cannot completely reconstitute the original architecture. Abundant granulation tissue grows in from the margin to complete the repair. This form of healing is referred to as secondary union or healing by second intention. Of the many differences between primary and secondary forms of healing, the most salient is the phenomenon of wound contraction, that is significant feature of healing by secondary intention.

Mechanisms of wound healing[10]

Wound healing, as we have seen is a complex phenomenon involving a number of processes, including induction of an acute inflammatory process by wounding, regeneration of parenchymal cells, migration and proliferation of both parenchymal and connective tissue cells, synthesis of extra-cellular matrix proteins, remodeling of connective tissue and parenchymal components, and collagenization and acquisition of wound strength.

Cutaneous wound healing is generally divided into three phases:

1. Inflammation (early and late)
2. Granulation tissue formation and re-epithelialization
3. Wound contraction, extracellular matrix deposition and remodeling.

Table 3: Growth factors and cytokines affecting various steps in wound healing

Monocyte chemotaxis	PDGF, FGF, TGF-beta
Fibroblast migration	PDGF, EGF, FGF, TGF-beta, TNF, IL-1
Fibroblast proliferation	PDGF, EGF, FGF, TNF
Angiogenesis	VEGF, Angiogenesis, FGF
Collagen synthesis	TGF-beta, PDGF
Collagen secretion	PDGF, FGF, EGF, TNF, (TGF-beta inhibits)

PDGF- platelet derived growth factor, EGF- epidermal growth factor, FGF- fibroblast growth factor, TNF- tumour necrosis factor

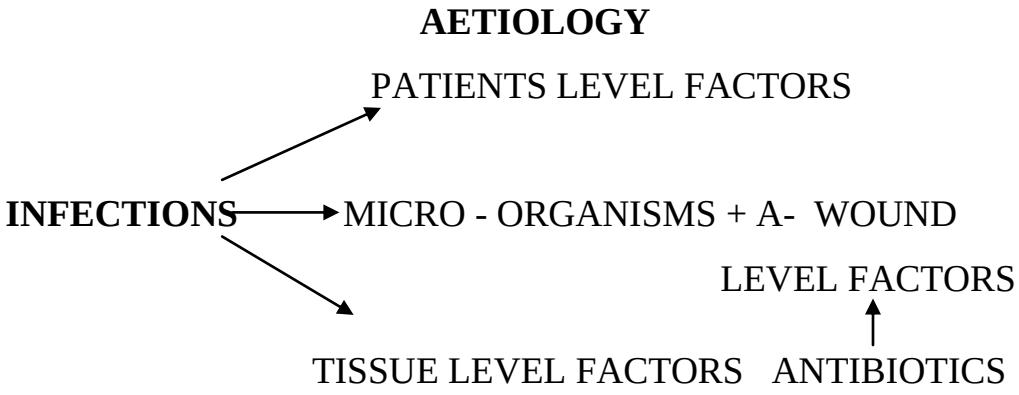
Impaired healing occurs due to many reasons and a wise surgeon recognizes them and attempts a remedy before he wields his scalpel so as to reduce the rate of surgical site infections and help proper wound healing. Of the many causes incriminated in defective wound healing, tissue hypoxia resulting from cardiopulmonary diseases, peripheral vascular diseases, and malnutrition and in chronic inflammatory disorders is a major cause. A prior search into these problems is a must before surgery is undertaken.

The repair process is influenced by many factors including,[10]

1. The tissue environment and the extent of the tissue damage
2. The intensity and duration of the stimulus

3. Conditions that inhibit repair, such as the presence of foreign bodies or inadequate blood supply

4. Various diseases that inhibit repair (diabetes in particular) and treatment with steroids.



Microorganisms Encountered In Wounds[11] :

Although the microbial flora of infected wounds frequently is so varied, a group designated as **organisms most frequently isolated from laparotomy wounds** would include the following;

- Staphylococcus aureus.
- Streptococcus pyogenes
- Coliform bacilli (from the lower half of body)
- Bacteroides species and other anaerobic non sporing gram - negative and gram - positive rods.
- Proteus species
- Pseudomonas species

- Clsotridium species

- Anaerobic cocci peptococcus, Peplo streptococcus

- Enterococci

Since anaerobic microorganisms are the predominant micro flora of humans and are constantly present in the intestinal tract, upper respiratory tract, and genitourinary tract, it is not unexpected to find them invading both usual and unusual anatomical sites, giving rise to severe and often fatal infections. This is particularly true when the host defenses, either naturally or artificially, have been so altered as to permit an overgrowth of these organisms.

A wide variety of aerobic and anaerobic species of bacteria may be present, either singly or in combination, in infection of wounds and other soft tissues. The commonest pyogenic bacteria are S. pyogenes, pneumococcus and colifrom bacilli such as Escherichia coli. Proteus species and Pseudomonas aeruginosa. Anaerobic organisms, particularly Clostridium perfingens and other Clostridia, Bacteroides species and anaerobic cocci, may be important in infections of wounds, especially abdominal wounds, soiled deep or lacerated wounds and wherever devitalized tissues provide suitably anaerobic conditions.[11]

In many cases there is a mixed infection with more than one bacterial species, and in some of these cases a pathogenic synergy may be evident with two or more species acting in concert to cause by either alone. Mixed infection with Gram-positive cocci and coliform bacilli are not uncommon and polymicrobial infections with anaerobes such as bacteroides and fusiforms or fuso-spirochaetal associations are well recognized.[11]

Special associations of certain pathogens with particulate conditions should be borne in mind e.g., Many postoperative abdominal or pelvic wounds have coliform bacilli associated with a moderate exudate during the early healing stage, the infection being often superficial and resolving without specific therapy. But a combination of coliforms with bacteroides may cause a more severe synergic infection calling for prompt antibacterial therapy.

Pathogenic micro-organisms are, logically the major determinates of postoperative sepsis. The micro-organisms involved may be endogenous or exogenous in origin. The former are regarded as normal flora at another site in the body. The latter are the target of cross- infection control measures .

PATIENT CHARACTERISTICS	OPERATION CHARACTERISTICS
Age	Duration of surgical scrub
Nutritional status	Skin antisepsis
Diabetes	Pre-operative shaving
Smoking	Pre-operative skin preparation
Obesity	Duration of operation
Co-existent infections at a remote body site	Antimicrobial prophylaxis
Colonization with micro-organisms	Operating room ventilation
Altered immune response	Inadequate sterilization of instruments
Length of pre-operative stay	Foreign material in surgical site
	Surgical drains
	Poor surgical technique

Patient Level Factors Affecting The Incidence Of Wound Infections :

Following is a consideration of factors thought to affect the susceptibility of any wound to infection at the whole patient level; further, these have been divided into two categories; endogenous and exogenous. Endogenous refers to unique attributes of the patient which either may (e.g., obesity, malnutrition) or may not (e.g., age) be alterable prior to surgery. Exogenous refers to characteristics of the operative experience not unique to any patient which can frequently be influenced by the surgeon (e.g., length of operation).

Endogenous Factors

Age:

Extremes of age have long been thought to influence the likelihood of wound infections, perhaps owing to decreased immunocompetence.[13] Even in clean contaminated procedures, age has been associated with an increased infection rate. Age, obviously, is an immutable patient characteristic and even if it is a risk factor for wound infection, it appears to be at most a modest one,with patients more than 66years old being six times more likely to develop infection than are patients 1 to 14 years old

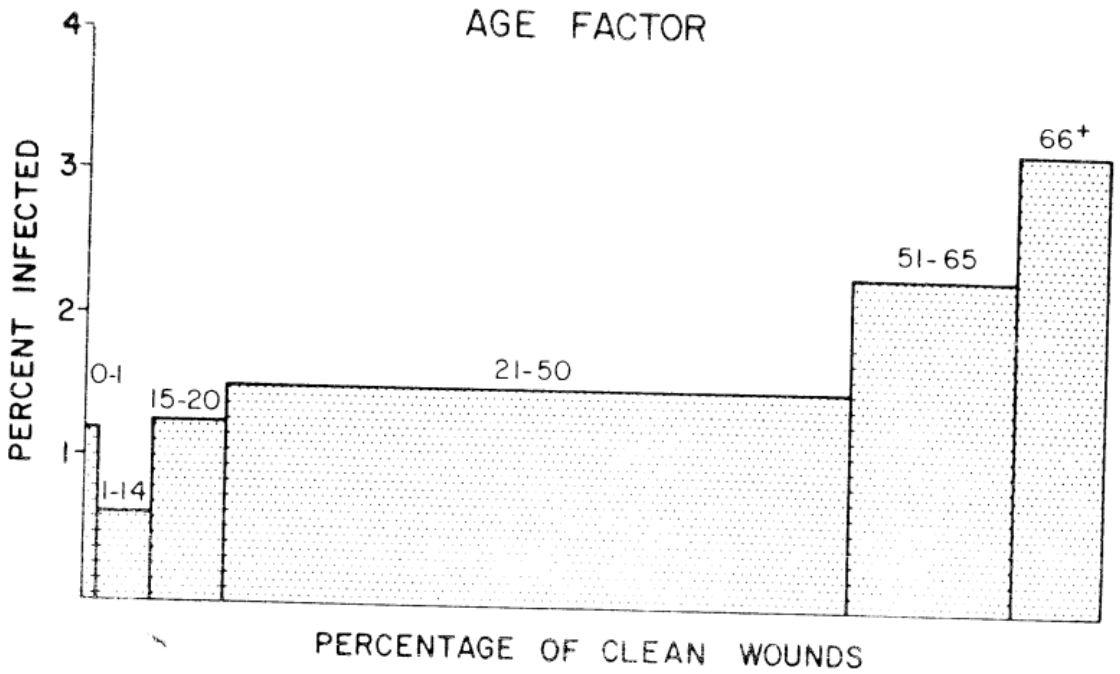


Figure 5. Older patients are more likely to develop infection of clean wounds than are younger patients.

Pre-existing Illness:

It has been logically assumed that wound infection are more common among patients with multiple pre-existing diseases, although how to quantify this factor of generalized illness is unclear. Whether or not newer, more comprehensive measures of patient physiologic status, such as Acute Physiologic Assessment and Chronic Health evaluation (APACHE) II or III, will give more precise prediction of risk remains to be seen

Diabetes mellitus[14]

Hyperglycemia has several deleterious effects upon host immune function, most notably impaired function of neutrophils and mononuclear phagocytes. Hyperglycemia may also be a marker of the catabolism and insulin resistance associated with surgical stress response, and that exogenous insulin administration may ameliorate the catabolic state.

Poor control of blood glucose during surgery and in perioperetive period increases the risk of infection,and worsens outcome from sepsis. Tight control of blood glucose by the anesthesiologist during surgery decreases the risk.Modarate Hyperglycemia(>200mg/dl)at any time on the first postoperative day increased the risk of SSI fourfold after noncardiac surgery.In a large randomised trial of critically ill postoperative patient, exogenous insulin administration to keep blood glucose concentrations<110mg/dl was associated with a 40% decrease of mortality, fewer nosocomial infections, and less organ dysfunction. Metaanalysis of the approximately 35

existing trials indicate that the risk of postoperative infection decreases significantly by tight glucose control, regardless of whether or not the patient had diabetes mellitus.

Obesity

Although intuitively a risk factor, obesity has not consistently been found to be related to wound infections. It is not clear whether this effect was independent of other SSI; diseases also associated with wound infections, such as diabetes mellitus. But it has been found that obesity to be associated with sternal or mediastinal wound infection, independent of other risk factors. Obesity, therefore, may be only weakly associated with wound infections.

Length of preoperative hospitalization[1,13,17]

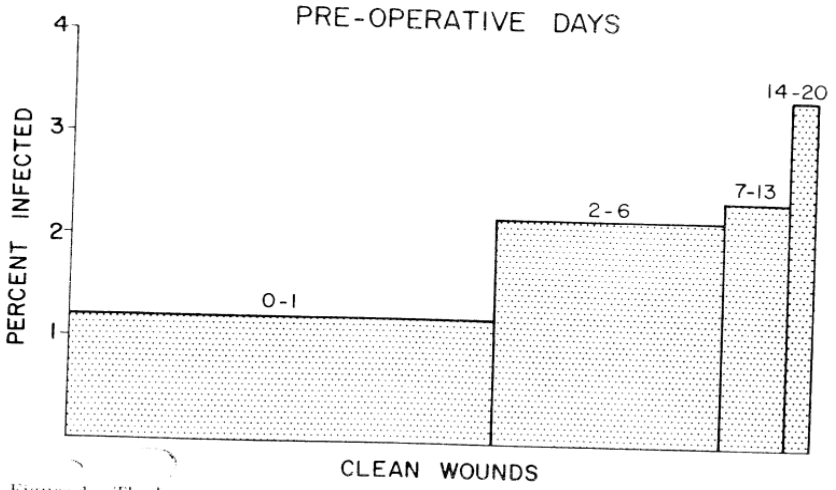


Figure 4. The longer a patient is in the hospital before an operation, the more susceptible he or she is to infection of the wound.

Prolonged preoperative stay is now proven to increase the SSI risk in all patients, independent of age, illness and nature of operation. The theory proposed for this is the colonization of nosocomial bacteria that are more drug resistant than their wild counterparts. Cruse and Ford, in their 10 years study have shown that patients

hospitalized for 0 to 1 day had a clean SSI rate of 1.2%. With a 1 week stay, 2.1% and in those hospitalized for more than two weeks, a 3.4% SSI rate. However, length of preoperative stay is likely a surrogate for severity of the illness and co-morbid conditions that require in-patient workup and/or therapy before the operation. The current recommendation that can be derived at, from the above data is to minimize the duration of preoperative stay, whenever possible, as especially in clean, elective surgeries. P. K. Agarwal in their study shows the infection rate was lowest in patient who were operated upon within seven days of admission while highest in patient who stayed for more than 21 days before operation in the ward.[17]

Malignancy :

The presence of malignancy and its attendant, although poorly understood, alteration in immune status has sometimes been considered a risk factor for wound infection. The presence of malignant disease, especially when widespread and metastatic, is a risk factors in the development of post-operative sepsis. Past malignant disease which had been removed or controlled is probably not a significant factor. The exceptions to these are malignant lyphomas and leukaemias, where the degree of tumor control may be difficult to assess, and patients with these malignancies often remain immunosuppressed.

Remote site Infection :

It has been found an epidemiologic correlation between remote site infection and subsequent surgical wound infection. The greatest risk appeared to be with remote infections involving a medical device, such as an indwelling urinary catheter.

It is unclear whether preoperative treatment of the remote infection successfully reduced the subsequent risk to the wound. Given the current aggressiveness with which distant infections are sought and treated preoperatively, such as by routine urine analysis, it is doubtful whether this question will ever be fully answered. It seems prudent to continue to consider remote site infection a risk factor and to treat it appropriately prior to operation, if possible.

Malnutrition:[1,15]

For some types of operations, severe protein energy malnutrition (PEM), is crudely associated with postoperative nosocomial infections, impaired wound healing dynamics or death. The National academy of sciences, National research council, study on the efficacy of infection control (SENIC) and NNIS schemes for SSI risk stratification do not explicitly incorporate nutritional status as a predictive variable although it may be indirectly represented in the latter too.

It is generally assumed by Clinicians that infections are more abundant, more severe and of longer duration in malnourished, as compared to well-nourished, patient (Btstrian, 1977; Neumann, 1977). This assumption is supported by the observation that

immune functions when tested in its component parts, is depressed by Malnutrition (Bistnan,1977; Murray and Murray, 1979).

In India, however, several studies, as the one represented by the 1985 study of Shukla et al, reported an increased SSI risk in malnourished patients who carried a SSI rate of 17% as compared to the well nourished, who had an overall SSI rate of 8.3%.

Theoretical arguments can be made for a belief that severe preoperative malnutrition should increase the risk of both incisional and organ/space infection. However, an epidemiological association between incisional SSI and malnutrition has been difficult to demonstrate for all surgical sub-specialties.[1]

Cigarette Smoking[1]

The effect of cigarette smoking on wound infection rates has, surprisingly, not been well studied. It is found to be associated with a slightly increased sternal wound infection rate among patients undergoing cardiac surgery. The concept of cigarette smoking as a risk for wound infection should be heeded because it is a potentially alterable behavior in the preoperative period.

ASA Score[14]

As incorporated in the national Nosocomial Infections Surveillance System (NNIS), the most recognized factors are the wound classification,American Society of Anesthesiology Class 3 or higher and prolong operative time.

BOX 2. American Society of Anesthesiology (ASA) physical status score.

ASA 1

A normal healthy patient.

ASA 2

A patient with mild to moderate systemic disturbance that results in no functional limitations. Example : Hypertension, diabetes mellitus, chronic. Morbid obesity, extremes of age.

ASA 3

A patient with severe systemic disturbance that results in functional limitations: Examples : Poorly controlled hypertension, diabetes mellitus with vascular complications, angina pectoris, prior myocardial infraction, pulmonary disease that limits activity.

ASA 4

A patient with a severe systemic disturbance that is life threatening with or without the planned procedure. Example: Congestive heart failure, unstable angina pectoris, advanced pulmonary, renal or hepatic dysfunction.

ASA 5

A morbid patient not expected to survive with to without the operative procedure. Example : Ruptured abdominal aortic aneurysm, pulmonary embolism, head injury with increased intracranial pressure.

ASA 6

Any patient in whom the procedure is an emergency, Example : ASA 4E

Exogenous Factors:

Length of Operation

Risk of wound infection has repeatedly been shown to be proportional to the length of the operative procedure.[13,24] Wound infections with longer procedures, roughly doubling with every hour of the procedures. SEN1C report, also found duration of operation of greater than 2 hours to be the second greatest independent, predictor of risk after multivariate analysis, with a regression coefficient of 1.04. It is unclear from these studies, however, how frequently a prolonged duration of operation was secondary to a case's inherent complexity versus a simpler case taking an unusually long time to complete. This question is partly addressed by Culver et al 1991 modification of the SENIC index. Rather than taking an arbitrary time (e.g., 2 hours) over which an operation was designated prolonged, he considered a procedure lengthy of its length fell above the 75th percentile for other similar procedures. Therefore, an appendectomy was considered prolonged if it lasted more than 1 hour, whereas coronary artery bypass grafting was not prolonged unless it required more than 5 hours. Using this index, operative time was still one of three variables, along with wound class and ASA the independently predicted infection. Prolongation of an operation, whether from an unusually complicated procedure, increased likelihood of normal wound contamination, or lapses in antibiotic coverage, must be considered a significant risk factor for wound infection.

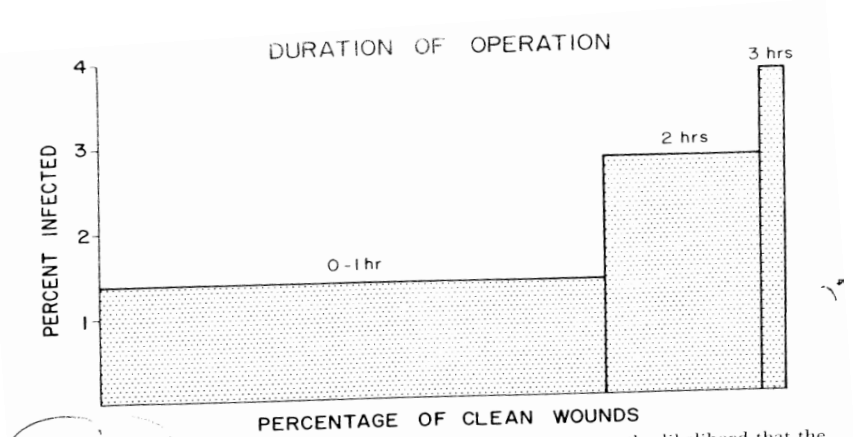


Figure 6. The longer the duration of the operation, the greater the likelihood that the wound will become infected.

Glove Punctures[13,16]

Much attention is given to glove perforations and risks they pose. However, the contribution of glove perforation to infection is over-emphasized. The use of electrical permeability to detect perforations is erroneous (Miller at al. 1972) and may have contributed to the high rates quoted. Recent figures put the perforation rate at around 5 percent (Cruse and Foorde, 1973) and the rate at Flinders Medical Center is similar. In and investigation of glove perforations using inflation and water immersion our recent perforation rate was 6 percent. The infection risk of glove perforation must be considered in conjunction with hand-carriage of Staphylococcus aureus..Peter J E. Cruse et al. in their study found 11.6 percent of gloves were punctured at the end of surgical procedure, not a single wound infection occurred in these patients. Organisms probably escaped from the glove punctures in insufficient numbers to be a serious hazard in a clean wound with adequate local resistance.[13]

Emergency Procedures

Several studies have shown emergency operations to be particularly prone to swound infections. Of the 4465 wounds studied by GikEgea et al 623 were made under emergent situations, and the wound infection rate for these was 5.1%, versus the 3842 elective wounds with an infection rate of 2.9%. Garibaldi et al reported a wound infection odds ratio of 7.6 (95% confidence interval, 3.2 to 18,2) for emergency versus elective operations, but after multivariate analysis, this factor was no longer significant. At this time, emergency operations do not by themselves clearly predispose to wound infection

Time of day

In their initial study of 23,649 wounds, Ruse and Foord found that the clean wound infection rate more than tripled, to 6.8%, for cases done between midnight and 8 A.M. and that the clean contaminated wound infection rate doubled to 18.3% during the same period. These rates do not account for other factors, such as underlying patient illness, and are therefore difficult to interpret.

Month of year

It remains unclear why a consistent rise in wound infection rate appears in the early summer, but Mead et al clearly demonstrated this phenomenon in their study of 8474 wounds over an 18-month period. These findings are similar to those found by Cruse and Foord for both clean and nonclean wounds, with a peak clean infection rate of 2% in July of 1977, the last year of the 10-year study, versus less than 1% for most of the

rest of the year. Condon et al, in a 5-year study of wounds at the Wood Veterans Administration Hospital, also noted peaks in wound infection rates in July Whether this weak risk factor is the result of new surgical staff changes in weather and personal hygiene, or other factors is not known. P. K. Agarwal in their study shows patients operated in winter season(November to March)developed less infection than those operated during summer (April to July) and rainy season (August to October).[17]

Airborne Contamination

With the exception of rare epidemics traced to either air handling or surgical staff airborne contamination of wounds in general surgery appears to play a small role in the pathogenesis of wound infections'. Whyte et al, studied 188 cholecystectomies and found that although the bacterial concentration on drapes distant from the wound and from the drapes close to the wound depended much more on bactibilia or skin flora. In fact, more bacteria were thought to have transferred from the wound and from the drapes close to the wound depended much more on bactibilia or skin flora. In fact, more bacteria were thought to have transferred from the wound to the drapes than vice versa. These results are in contrast to studies of upper-joint procedures, in which 98% of the bacteria found in wounds by Whyte et al were thought to be from the air, and the use of ultra clean operating rooms, age described by Lidwell et al, decreased the rate of joint infection by approximately 25%[1].

Wound Class[2]

The wound classification system proposed by the National Research Council in 1964 continues to be useful 30 years later. In 1980, the 62,939 wound Foothills Hospital project generated a set of wound infection rates for the four wound classes(clean 1.5 % , clean contaminated 7./%, contaminated 15.2%, and dirty 40 %) which other studies continue to use as a reference. More importantly, wound class has been shown to be independently predictive of wound infection in several large studies using multivariate analysis. In the original SENIC study in 1985, Haley et al demonstrated a contaminated or dirty wound to predict infection with a regression coefficient of 1:04. In Culver et al modification of the SENIC risk index published in 1991, wound classification was the only risk factor unchanged from the original index. In Garibaldi el al study of 1552 patients, surgical wound class once again found on stepwise logistic regression analysis to be predictive of wound infection with an odds ratio for contaminated or dirty wounds of 2.7(95% confidence interval 1.9 to 4.6) These findings are more impressive because the finding of bacterial wound contamination, demonstrated by the growth of 30 or more colony-forming units (CPU) of bacteria from a Millipore filter pressed against the wound margin, was also independently predictive of infection in this study.

Wound Contamination

Wound contamination as demonstrated by intraoperative culture, has .been shown to be associated with later wound infection. Whyte et al showed that during cholecystectomy the number and species of bacteria cultured from the bile were predictive of wound contamination by culture and later wound infection. Later, Garibaldi

et al in 1991, found that 30 or more CFU of bacteria cultured from the wound were, by stepwise logistic regression, predictive of wound infection (odds ratio, 3.0;95% confidence interval, 2.0 to 4 6), independent of wound class. In the prospective study of 190 patients undergoing elective colorectal surgery by Claesson and Holmlundu in which all wounds were theoretically classified as clean contaminated, multivariate analysis revealed that 5 or more CFU/ml of bacteria in peritoneal fluid were predictive of wound infection. How and if the routine culture of wounds should be incorporated into normal clinical practice is unclear although any further studies of wound infections ought to include this important

Preoperative Hair Removal[1,18]

Shaving is a ritual which may cause increased infection rates (Seropain and Reynolds 1971). The presence of hair has not been documented as a source of wound infection. If removal of hair is required to achieve adequate visualization or to enhance adhesiveness of dressings then the following should be considered :

1. A depilatory- cream (Seropian and Reynolds, 1971)
2. The use of sterilizable electric clippers
3. If you do decide to shave, use disposable razors
4. Never use a brush because of cross - infection hazards (aerosol shaving - creams are recommended).
5. Hair should be removed as close to the time of surgery as possible so as to reduce infection of traumatized skin.

Adhesive Drapes

The benefits of plastic adhesive wound drapes remain controversial. Cruse and Foord found no benefit to the use of plastic adhesive drapes, with an overall infection : of 1.5% in wounds draped in the standard manner and 2.4% in wounds protected with plastic drapes. In 1985. however, Alexander et al reported on an effective skin preparation consisting of a 1-minute alcohol application followed by the application of iodine-impregnated plastic adhesive drapes. Currently, any benefit to the use of plastic drapes appears to be small.

Wound irrigation

The irrigation of wounds with antibiotic-containing solutions has a long history, starting with the use of topical sulfonamides in wounds in the 1930s . Several later studies appeared to show a benefit to wound irrigation, particularly in clean contaminated or contaminated procedures. For example, in a prospective study of 240 patients undergoing colon operations published in 1072 by Anderson et al the infection rate for patients receiving topical ampicillin was 2.5% versus 18.3% in wounds not receiving intestinal antiseptics, which appeared to be more effective than the topical antibiotics With the introduction of more effective antibiotics for prophylactic use in clean contaminated and contaminated procedures, the added benefit of topical antibiotics is probably minimal in all but the most severely contaminated wounds, in clean wounds, in which the wound infection rate is already low, topical antibiotic irrigation is probably of no benefit, although its low cost and minimal morbidity assure its continued use.

Tissue Level Factors Affecting The Incidence Of Wound Infections :

Tissue Perfusion

Perfusion of a wound is critical to healing for several reasons, the two most important probably being delivery of oxygen and neutrophils, two essential and interrelated elements of normal wound healing. Owing to the inevitable 50 to 100 um of poorly perfused adjacent tissue, the normal wound environment has a Po2 of 5 to 10mm Hg, a Pco2 of 50 to 60 mm Hg, and a pH of 6.5 to 6.9. In vitro studies have demonstrated a decrease in neutrophil killing and response to chemoattractant under these conditions. Further, Knighton et al demonstrated in viva studies a 5 log reduction in wound fluid bacteria counts at 14 days simply by increasing the inspired FiO2 of room air from 20% to 45%. This effect was later noted to be further enhanced by the administration of systemic antibiotics. The deleterious effects of the presence of a wound foreign body are also explained by decreased oxygen tension, as Silver demonstrated in 1978 that the microenvironment immediately adjacent to a foreign body has a Po2 close to 0 mm Hg. It must be noted, however, that clinical experiments to support the use of hyperoxia to aid wound healing have yet to be completed. Meanwhile, it is axiomatic that wounds do not heal in the presence of severe vascular occlusive-disease.

Local immune response

Only in the past 10 years have the tools become available to study the systemic and local immune response at a cell and cell mediator level. The keratinocyte has been shown to be an immunologically active cell able to produce and express a wide spectrum

of immune response mediators, including intracellular adhesion molecule-1 interleukin (1L)-1 tumor necrosis factor-, a 1L-6, 1L.-8. and transforming growth factor-α. The effect of the presence of bacteria on all of these responses is unknown, but it is hoped manipulation of these events will decrease the likelihood of wound sepsis. It is further interesting to note that, although the uninfected fetal wound has been demonstrated to heal by a process closer to regeneration than scar deposition, Frantz et al have shown that the presence of bacteria in fetal wounds induces a more adult-like collagen deposition, fibroplasia, and neovascularization These findings raise the question of the role of bacteria or their products even in normal, uninfected adult wound healing.

COMPLICATION OF LAPAROTOMY WOUND

Seroma[2,20]

A seroma is a collection of liquefied fat, serum, and lymphatic fluid under the incision. The fluid is usually clear, yellow, and somewhat viscous and is found in the subcutaneous (SC) layer of the skin. Seromas represent the most benign complication after an operative procedure and are particularly likely to occur when large skin flaps are developed in the course of the operation,

Presentation and Management

A seroma is usually manifested as a localized and well-circumscribed swelling, pressure or discomfort, and occasional drainage of clear liquid from the immature surgical wound.

Prevention of seroma formation may be achieved by placing suction drains under the skin flaps or in potential dead space created by lymphadenectomy. Premature removal of drains frequently results in large seromas that require aspiration under sterile conditions, followed by placement of a pressure dressing. A seroma that reaccumulates after at least two aspirations is evacuated by opening the incision and packing the wound with saline-moistened gauze to allow healing by secondary intention.

Hematoma[2,20]

A hematoma is an abnormal collection of blood, usually in the SC layer of a recent incision or in a potential space in the abdominal cavity after extirpation of an organ, for example, splenic fossa hematoma after splenectomy or pelvic hematoma after proctectomy. Hematomas are more worrisome than seromas because of the potential for secondary infection. Hematoma formation is related to inadequate hemostasis, depletion of clotting factors, and the presence of coagulopathy. A host of disease processes can contribute to coagulopathy, including myeloproliferative disorders, liver disease, renal failure, sepsis, clotting factor deficiencies, and medications. Medications most commonly associated with coagulopathy are antiplatelet drugs, such as aspirin, clopidogrel bisulfate (Plavix), ticlopidine hydrochloride (Ticlid), eptifibatide (Integrilin), and abciximab (ReoPro), and anticoagulants, such as ultrafractionated heparin, low-molecular-weight heparin (LMWH: enoxaparin [Lovenox], dalteparin sodium [Fragmin], tinzaparin [Innohep]), and warfarin sodium.

Presentation and Management

The clinical manifestations of a hematoma vary with its size and location. A hematoma may appear as an expanding, unsightly swelling or pain in the area of a surgical incision, or both. On physical examination, a hematoma appears as a localized soft swelling with purplish/blue discoloration of the overlying skin. The swelling varies from small to large and may be tender to palpation or associated with drainage of dark red fluid out of the fresh wound.

Hematoma formation is prevented preoperatively by correcting any clotting abnormalities and discontinuing medications that alter coagulation. One must balance the risk of significant bleeding due to uncorrected medication-induced coagulopathy and the risk of thrombosis after discontinuation of therapy. In patients at high risk for thrombosis who are scheduled to undergo an elective major surgical procedure, warfarin must be discontinued 3 days before surgery to allow the international normalized ratio (INR) to be less than 1.5. Then they are given heparin intravenously (IV) or an equivalent dose SC. Those receiving standard heparin can have the medication discontinued 2 to 3 hours before surgery and those receiving LMWH (variable half-life), 12 to 15 hours before surgery. Anticoagulants are then resumed 24 to 48 hours after surgery. Patients taking clopidogrel must have the medication withheld 5 to 6 days before surgery; otherwise, the surgery must be delayed.

Acute Wound Failure (Dehiscence)[2,20]

Acute wound failure (wound dehiscence or a burst abdomen) refers to postoperative separation of the abdominal musculoaponeurotic layers. It is among the most dreaded complications faced by surgeons and of greatest concern because of the risk of evisceration, the need for immediate intervention, and the possibility of repeat dehiscence, surgical wound infection, and incisional hernia formation.

Acute wound failure occurs in approximately 1% to 3% of patients who undergo an abdominal operation. Dehiscence most often develops 7 to 10 days postoperatively but may occur anytime after surgery from 1 to more than 20 days. A multitude of factors may contribute to wound dehiscence

Factors Associated With Wound Dehiscence

Technical error in fascial closure
Emergency surgery
Intra-abdominal infection
Advanced age
Wound infection, hematoma, and seroma
Elevated intra-abdominal pressure
Obesity
Chronic corticosteroid use
Previous wound dehiscence
Malnutrition
Radiation therapy and chemotherapy
Systemic disease (uremia, diabetes mellitus)

Presentation and Management

Acute wound failure may occur without warning and evisceration makes the diagnosis obvious. Sudden, dramatic drainage of a relatively large volume of a clear, salmon-colored fluid precedes dehiscence in a fourth of patients. Probing the wound with a sterile, cotton-tipped applicator or gloved finger may detect the dehiscence.

Prevention of acute wound failure is largely a function of careful attention to technical detail during fascial closure. For very high-risk patients, interrupted closure is often the wisest choice. Alternative methods of closure must be selected when primary

closure is not possible without undue tension. Although retention sutures were used extensively in the past, their use is less common today, with some surgeons opting to use a synthetic prosthesis or tissue graft.

Once dehiscence is diagnosed, treatment depends on the extent of fascial separation and the presence of evisceration or significant intra-abdominal contamination (intestinal leak, peritonitis). A small dehiscence in the proximal aspect of an upper midline incision 10 to 12 days postoperatively can be managed conservatively by packing the wound with saline-moistened gauze and using an abdominal binder. In the event of evisceration, the eviscerated intestines must be covered with a sterile, saline-moistened towel and preparations made to return to the operating room after a very short period of fluid resuscitation. Once in the operating room, thorough exploration of the abdominal cavity is performed to rule out the presence of a septic focus or an anastomotic leak that may have predisposed to the dehiscence. Treatment of the infection is of critical importance before attempting closure. Management of the incision is a function of the condition of the fascia. When technical mistakes are made and the fascia is strong and intact, primary closure is warranted. If the fascia is infected or necrotic, débridement is performed. If after débridement the edges of the fascia cannot be approximated without undue tension, consideration needs to be given to closing the wound with absorbable mesh or the recently developed biologic prostheses (decellularized porcine submucosa and dermis and human cadaveric dermis). Attempts to close the fascia under tension guarantee a repeat dehiscence and possible intra-abdominal hypertension. Definitive

surgical repair to restore the integrity of the abdominal wall will eventually be required if absorbable mesh is used but not if a biologic prosthesis is used.

Absorbable mesh and biologic prostheses protect from evisceration, maintain the abdominal domain, and provide a barrier to prevent bowel desiccation, bacterial invasion, and nonadherent, potentially permanent closure. Autologous skin grafts are used to reconstitute the epithelial barrier, and flaps (local/regional or free) are used to reconstruct the abdominal wall.

For short-term management of a dehisced wound, a wound vacuum system can be used that consists of open-cell foam placed on the tissue, semiocclusive drape to cover the foam and skin of the patient, and suction apparatus. The wound vacuum system provides immediate coverage of the abdominal wound and acts as a dressing that minimizes heat loss and does not require suturing to the fascia. By using negative pressure, the device removes interstitial fluid and thus lessens bowel edema, decreases wound size, reduces bacterial colonization, increases local blood perfusion, and induces the healing response. Successful closure of the fascia can be achieved in 85% of cases of abdominal wound dehiscence.

Surgical Site Infection (Wound Infection)[2]

Presentation and Management

Superficial and deep surgical site infections are accompanied by erythema, tenderness, edema, and occasionally drainage. The wound is often soft or fluctuant at the

site of infection, which is a departure from the firmness of the healing ridge present elsewhere in the wound. The patient may have leukocytosis and a low-grade fever. According to the Joint Commission on Accreditation of Healthcare Organizations, a surgical wound is considered infected if it meets the following criteria:

1. Grossly purulent material drains from the wound
2. The wound spontaneously opens and drains purulent fluid
3. The wound drains fluid that is culture positive or Gram stain positive for bacteria
4. The surgeon notes erythema or drainage and opens the wound after deeming it to be infected

At the time of surgery the operating surgeon plays a major role in reducing or minimizing the presence of postoperative wound infections. The surgeon must be attentive to personal hygiene (hand scrubbing) and that of the entire team.[7] In addition, the surgeon must make certain that the patient undergoes a thorough skin preparation with appropriate antiseptic solutions and is draped in a sterile careful fashion. During the operation, steps that have a positive impact on outcome are followed:

1. Careful handling of tissues
2. Meticulous dissection, hemostasis, and débridement of devitalized tissue
3. Compulsive control of all intraluminal contents

4. Preservation of blood supply of the operated organs
5. Elimination of any foreign body from the wound
6. Maintenance of strict asepsis by the operating team (no holes in gloves, avoidance of the use of contaminated instruments, avoidance of environmental contamination such as debris falling from overhead)
7. Thorough drainage and irrigation of any pockets of purulence in the wound with warm saline
8. Ensuring that the patient is kept in a euthermic state, well monitored, and fluid resuscitated
9. At the end of the case, a judgment with regard to closing the skin or packing the wound

The use of drains remains somewhat controversial in preventing postoperative wound infections. In general, there is virtually no indication for drains in this setting. However, placing closed suction drains in very deep, large wounds and wounds with large wound flaps to prevent the development of a seroma or hematoma is a worthwhile practice.

Once a surgical site infection is suspected or diagnosed, management depends on the depth of the infection. For both superficial and deep surgical site infections, skin staples are removed over the area of the infection, and a cotton-tipped applicator may be easily passed into the wound with efflux of purulent material and pus. The wound is gently explored with the cotton-tipped applicator or a finger to determine whether the fascia or muscle tissue is involved. If the fascia is intact, débridement of any nonviable

tissue is performed, and the wound is irrigated with normal saline solution and packed to its base with saline-moistened gauze to allow healing of the wound from the base anteriorly and prevent premature skin closure. If widespread cellulitis is noted, administration of IV antibiotics must be considered. However, if the fascia has separated or purulent material appears to be coming from deep to the fascia, there is obvious concern about dehiscence or an intra-abdominal abscess that may require drainage or possibly a reoperation.

Wound cultures are controversial. If the wound is small, superficial, and not associated with cellulitis or tissue necrosis, culture may not be necessary. However, if fascial dehiscence and a more complex infection are present, material is sent for culture. A deep surgical site infection associated with grayish, dishwater-colored fluid, as well as frank necrosis of the fascial layer, raises suspicion for the presence of a necrotizing type of infection.

Most postoperative infections are treated with healing by secondary intention (allowing the wound to heal from the base anteriorly, with epithelialization being the final event). In some cases when there is a question about the amount of contamination, delayed primary closure may be considered. In this setting, close observation of the wound for 5 days may be followed by closure of the skin if the wound looks clean and the patient is otherwise doing well.

Recently, wound vacuum systems have been used in large, deep, or moist wounds with generally successful outcomes. Their advantage is a decrease in the nursing time previously required for dressing changes, as well as less pain for the patient.[2]

METHODOLOGY

Source Of Data:

- Patients admitted in B.L.D.E.A's Shri B.M.Patil Medical College Hospital & Research Centre, Bijapur for elective and emergency laparotomy.

Method Of Collection Data:

- Postlaparotomy patients in surgical wards, BLDEA's Shri B.M Patil Medical College Hospital & Research Centre, Bijapur. From October 2008 to May 2010.
- All potential candidates for surgery will be investigated and evaluated as per the required norms which will include; hemoglobin %, blood sugar, blood urea, serum creatinine, urine albumin, urine sugar and microscopy.
- Investigations of other organ system involvement and nutritional status, any other biochemical investigations necessary apart from those investigations necessary to diagnose these cases which necessitates the surgical intervention are also included
- Post-operative observed complications like, seroma, haematoma and wound infection, time of ambulation and duration of hospital stay. Surgical site infections will be classified according to the Center for disease control and prevention (CDC) classification: superficial incisional, deep incisional and organ/space infection. Patient will be followed up for a minimum period of one month following surgery. Final analysis regarding incidence and risk factors will be arrived at from the collected data.

Inclusion Criteria *:*

1. All emergency and elective laparotomy patients
2. Age more than 12yrs

Exclusion Criteria *:*

1. Patients with parietal wall hernia.
2. Patients age less than 12 years.
3. Patients with organ space infection
4. Immunocompromised patients
5. Pulmonary and generalized diseases
6. Gynaecological conditions
7. Patients on drugs like corticosteroids,

Research Hypothesis

Postoperative wound complications are more in emergency laparotomy as compared to elective laparotomy.

Sample Size :

Study period from : October 2008 to May 2010.

Incidence rate of 38%[1] in surgical site infection and 95% level of significance with 30% allowable error the calculated sample size is 70.

Statistical formula: $n= 4pq/L^2$

Statistical Analysis:

Following statistical tests will be used to compare the results.

i) Diagrammatic presentation.

ii) Mean ± S D

iii) Z test or chi square test

iv) Regression analysis (if necessary)

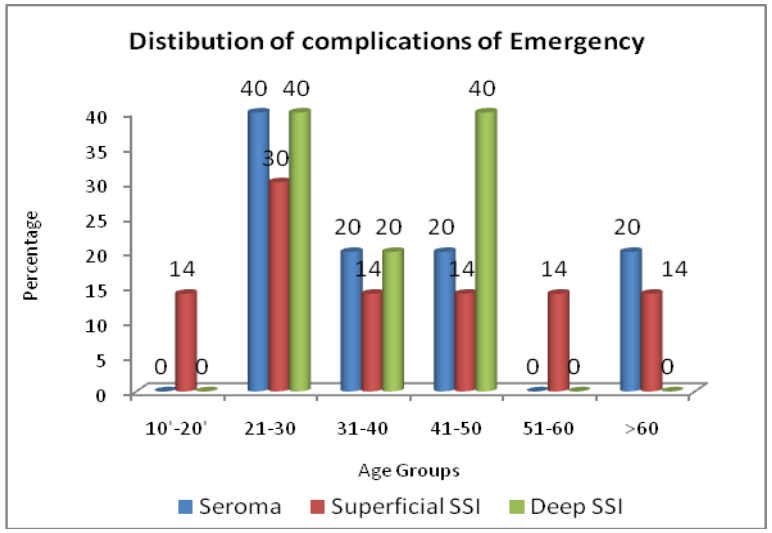
RESULTS

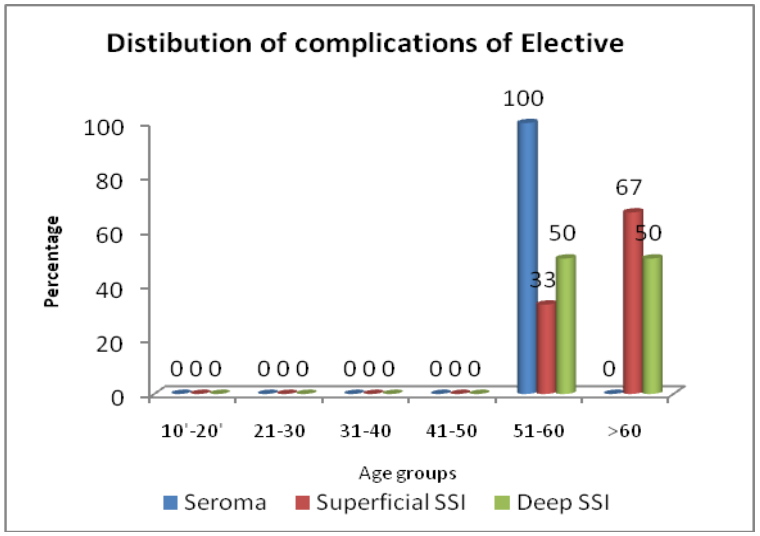
The study was conducted on 72 patients, aged between 13-80 years, who underwent major elective or emergency laparotomy surgery in Sri B.M. Patil Medical College Hospital and Research Centre Bijapur, from October 2008 to May 2010.

Among the 72 patients,5 (36) in elective and 9 (36) in emergency laparotomy patients developed complications.

Distribution of complications in Age

	Emergency									Elective								
	Seroma			Superficial SSI			Deep SSI			Seroma			Superficial SSI			Deep SSI		
Age groups	F	%	P value	F	%	P value	F	%	P value	F	%	P value	F	%	P value	F	%	P value
10'-20'	0	0		1	14		0	0		0	0		0	0		0	0	
21-30	2	40		2	30		2	40		0	0		0	0		0	0	
31-40	1	20		1	14		1	20		0	0		0	0		0	0	
41-50	1	20	>0.05	1	14	>0.05	2	40	>0.05	0	0	>0.05	0	0	>0.05	0	0	>0.05
51-60	0	0		1	14		0	0		1	100		1	33		1	50	
>60	1	20		1	14		0	0		0	0		2	67		1	50	
Total	5	100		7	100		5	100		1	100		3	100		2	100	

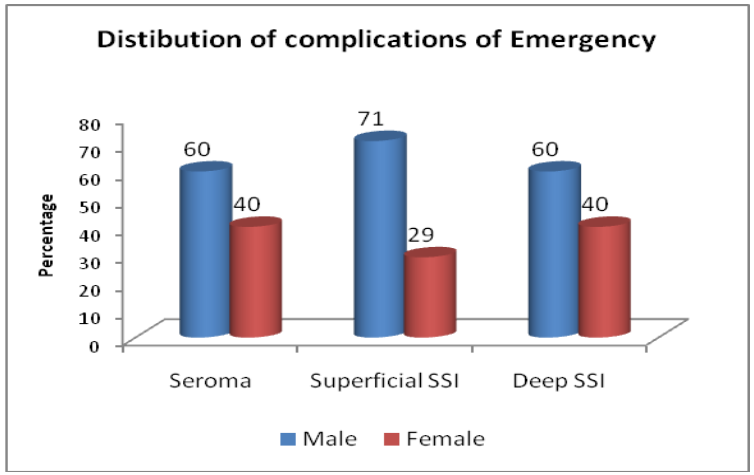


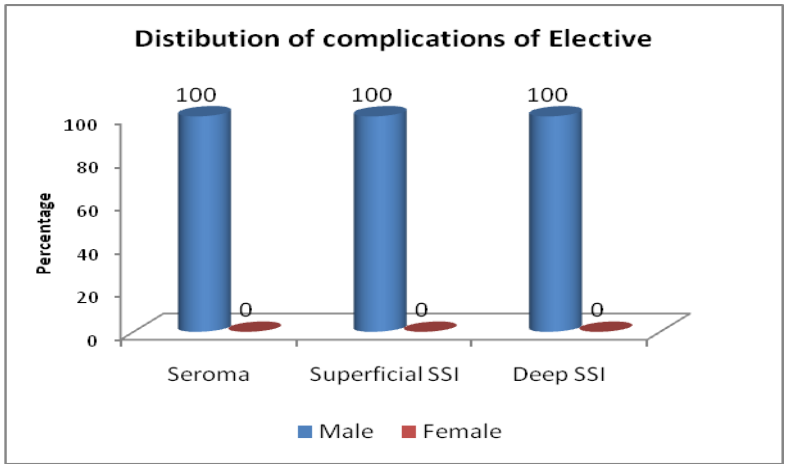


p > 0.05 in emergency and elective surgery.

Distribution of Complications in Sex

	Emergency						Elective					
	Seroma		Superficial SSI		Deep SSI		Seroma		Superficial SSI		Deep SSI	
	F	%	F	%	F	%	F	%	F	%	F	%
Male	3	60	5	71	3	60	1	100	3	100	2	100
Female	2	40	2	29	2	40	0	0	0	0	0	0
Total	**5**	**100**	**7**	**100**	**6**	**100**	**3**	**100**	**3**	**100**	**2**	**100**

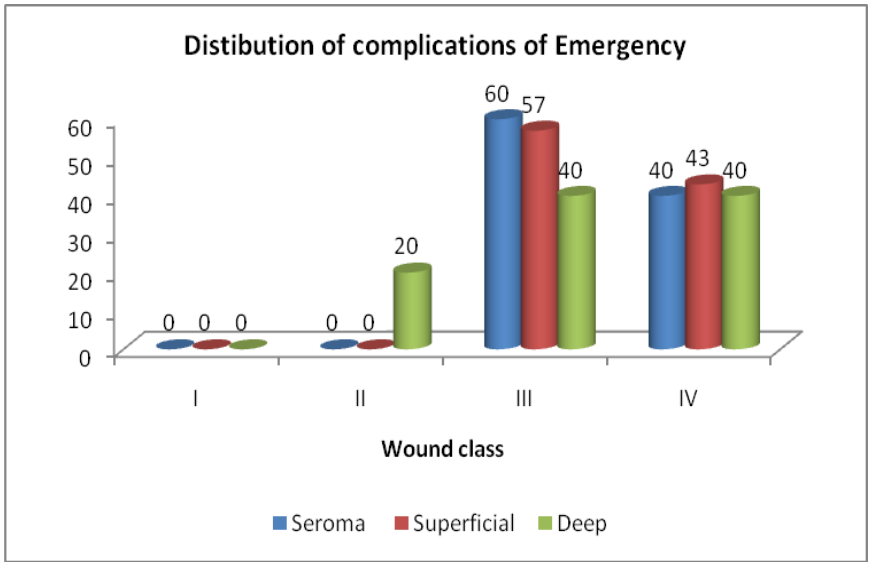


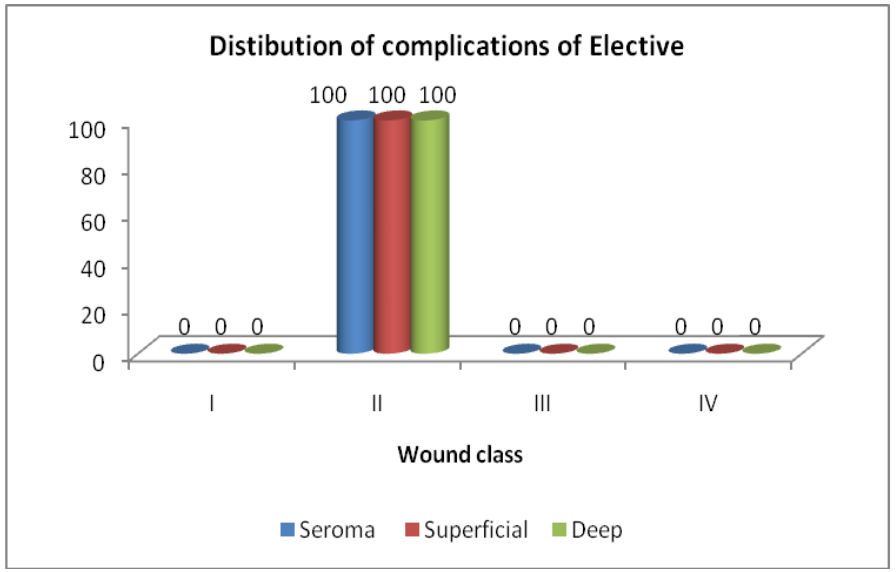


There are no significant difference in development of complication in male and female, in emergency and elective surgery.

Association of complication with Wound Class :

	Emergency									Elective								
CLASS	Seroma			Superficial SSI			Deep SSI			Seroma			Superficial SSI			Deep SSI		
	F	**%**	**p value**	**F**	**%**	**p value**	**F**	**%**	**p value**	F	%	p value	F.	%	p value	F	%	p value
I	**0**	**0**		**0**	**0**		**0**	**0**		0	0		0	0		0	0	
II	**0**	**0**		**0**	**0**	**>0.05**	**1**	**20**		1	100	>0.05	3	100		2	100	
III	**3**	**60**	**>0.05**	**4**	**57**		**2**	**40**	**>0.05**	0	0		0	0	>0.05	0	0	>0.05
IV	**2**	**40**		**3**	**43**		**2**	**40**		0	0		0	0		0	0	
TOTAL	**5**	**100**		**7**	**100**		**5**	**100**		1	100		3	100		2	100	



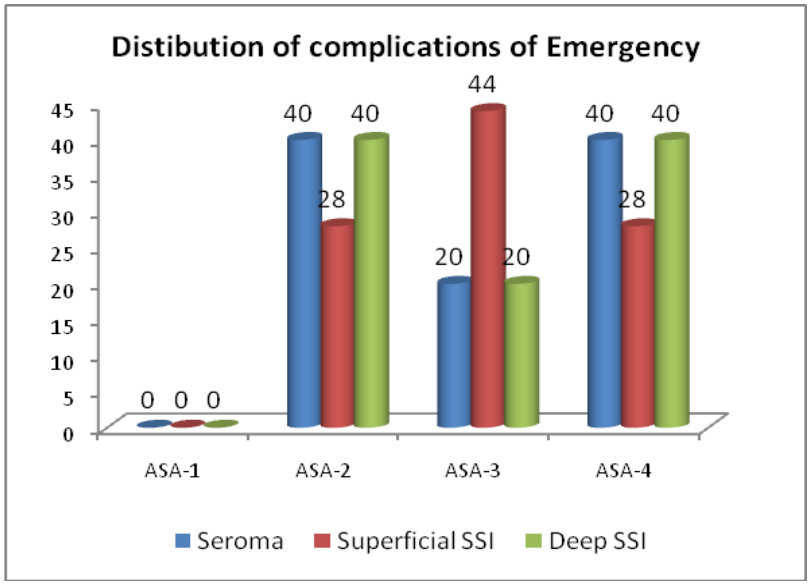


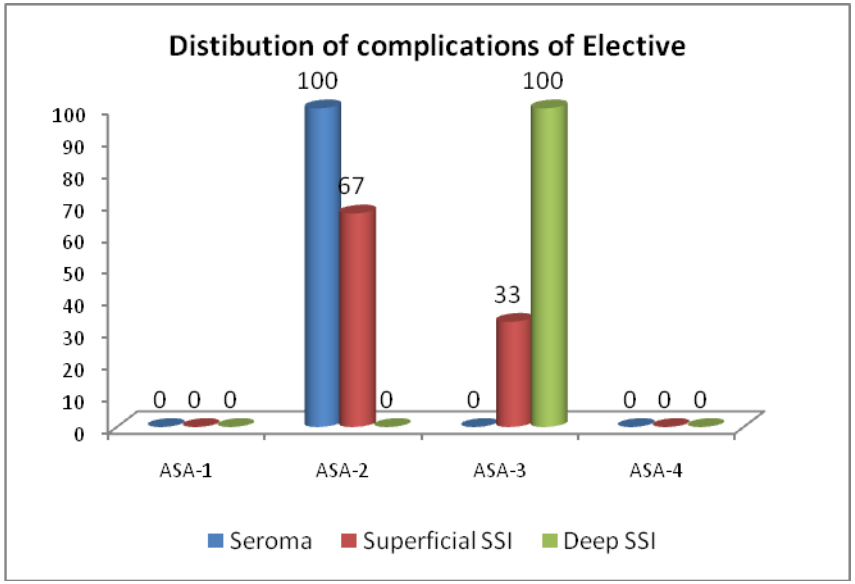
Most of patient were in class III and IV

All patient were in class II

Association of Complications with ASA

ASA	Emergency												Elective											
	Seroma				Superficial SSI				Deep SSI				Seroma				Superficial SSI				Deep SSI			
	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p	Chi sq.	F	%	p	Chi sq.	F	%	p	Chi sq.
ASA-1	0	0	0.004	13.33	0	0	0.025	9.328	0	0	0.004	13.33	0	0	>.05		0	0	>.05		0	0	0.028	4.813
ASA-2	2	40			2	28			2	40			1	100			2	67			0	0		
ASA-3	1	20			3	44			1	20			0	0			1	33			2	100		
ASA-4	2	40			2	28			2	40			0	0			0	0			0	0		
Total	5	100			7	100			5	100			1	100			5	100			3	100		

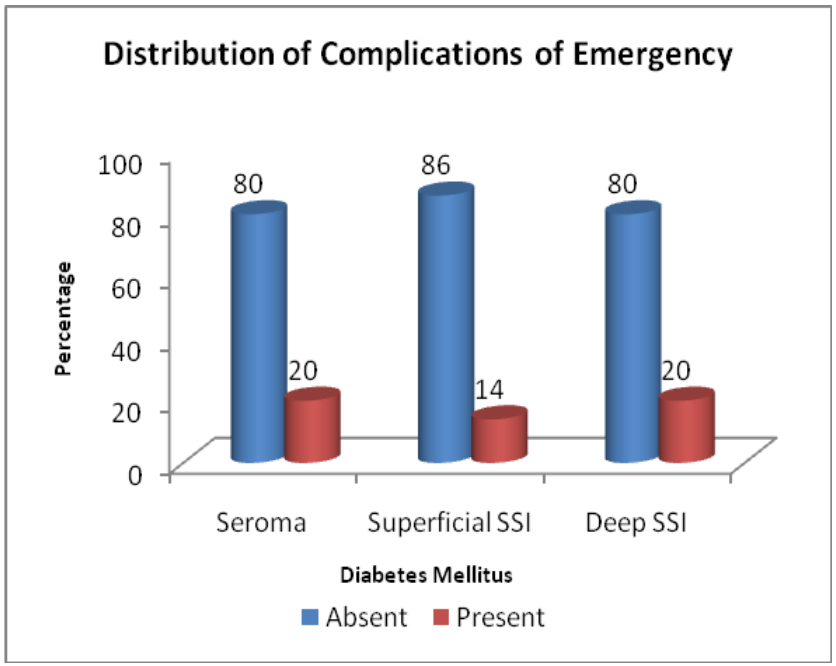




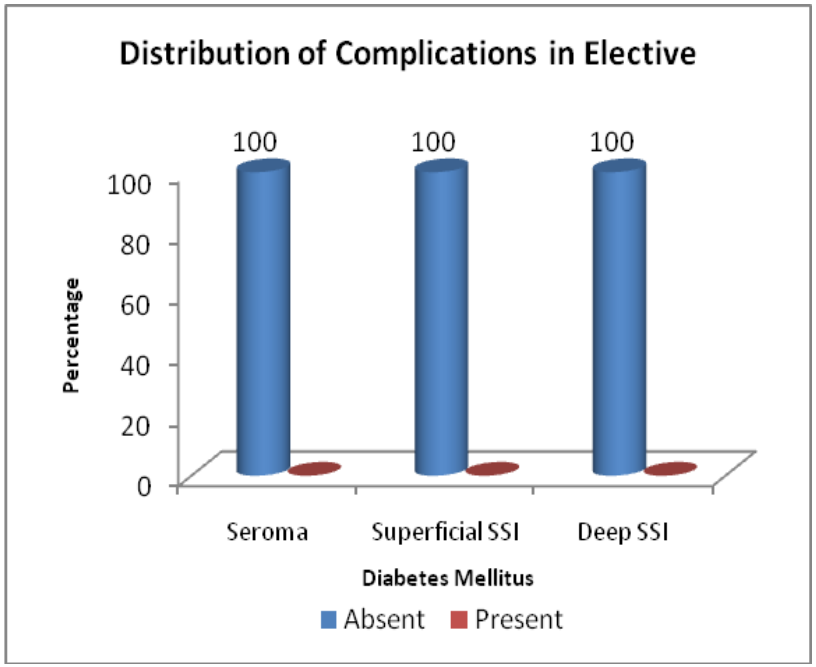
Higher ASA score are significantly associated with development of complication in emergency and elective surgery.

Association of Complications with DM

	Emergency									Elective								
DM	Seroma			Superficial SSI			Deep SSI			Seroma			Superficial SSI			Deep SSI		
	F	%	p value	F	%	p value	F	%	p value	F	%	p value	F.	%	p value	F.	%	p value
Absent	4	80		6	86		4	80		1	100		3	100		2	100	
Present	1	20	>0.05	1	14	>0.05	1	20	>0.05	0	0	>0.05	0	0	>0.05	0	0	>0.05
Total	5	100		7	100		5	100		1	100		3	100		2	100	



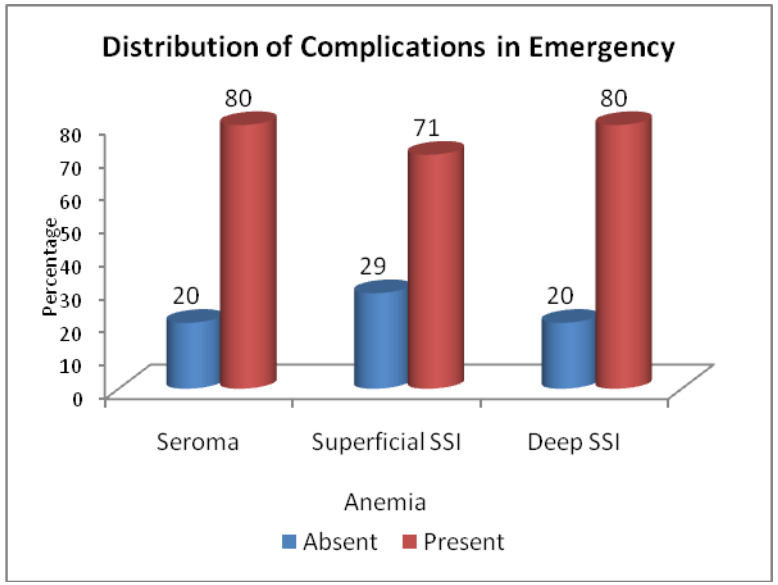
p>0.05

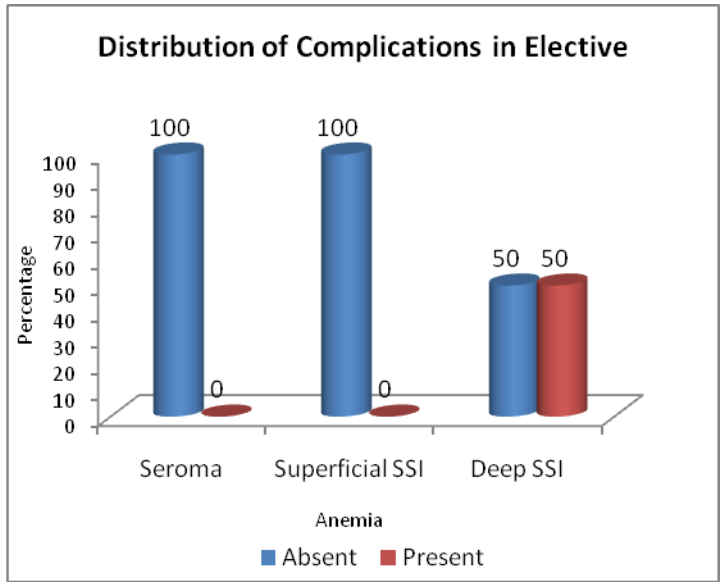


p>0.05

Association of Complications with Anemia

	Emergency												Electve											
	Seroma				Superficial SSI				Deep SSI				Seroma				Superficial SSI				Deep SSI			
	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.
Absent	1	20	0.002	9.368	2	29	0.002	9.990	1	20	0.002	9.368	1	100	>0.05	-	3	100	>0.05	-	1	67	0.00	17.486
Present	4	80			5	71			4	80			0	0			0	0			1	33		
Total	5	100			7	100			5	100			1	100			3	100			2	100		



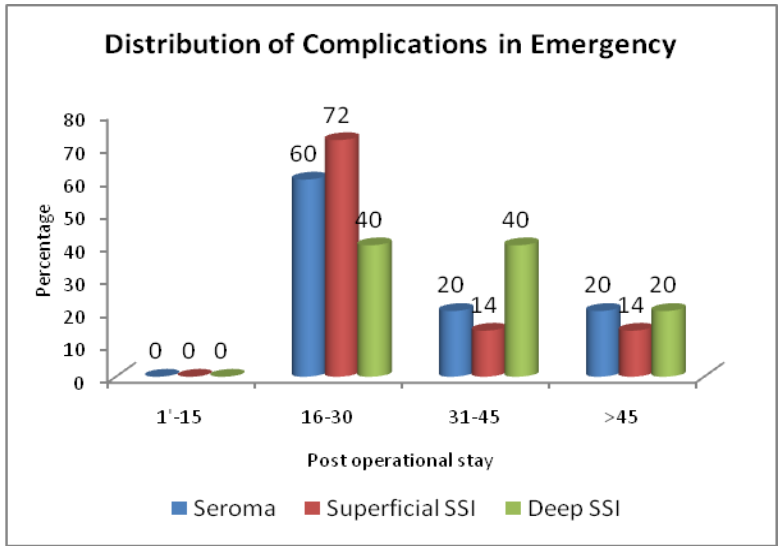


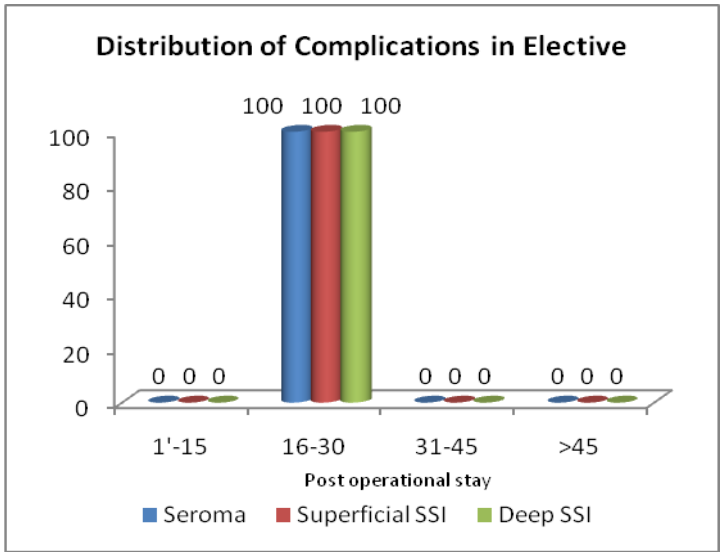
In emergency surgery p value < 0.05

In Elective Deep SSI p value < 0.05

Association of Complications with POS

	Emergency												Elective											
	Seroma				Superficial SSI				Deep SSI				Seroma				Superficial SSI				Deep SSI			
	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.	F	%	p value	Chi sq.
1'-15	1	0			0	0			0	0			0	0			0	0			0	0		
16-30	3	60			5	72			2	40			1	100			3	100			2	100		
31-45	1	20	0.007	12.183	1	14	0.002	14.332	2	40	0.001	16.74	0	0	>0.05	-	0	0	0.006	7.438	0	0	0.028	4.813
>45	1	20			1	14			1	20			0	0			0	0			0	0		
Total	5	100			7	100			5	100			1	100			3	100			2	100		

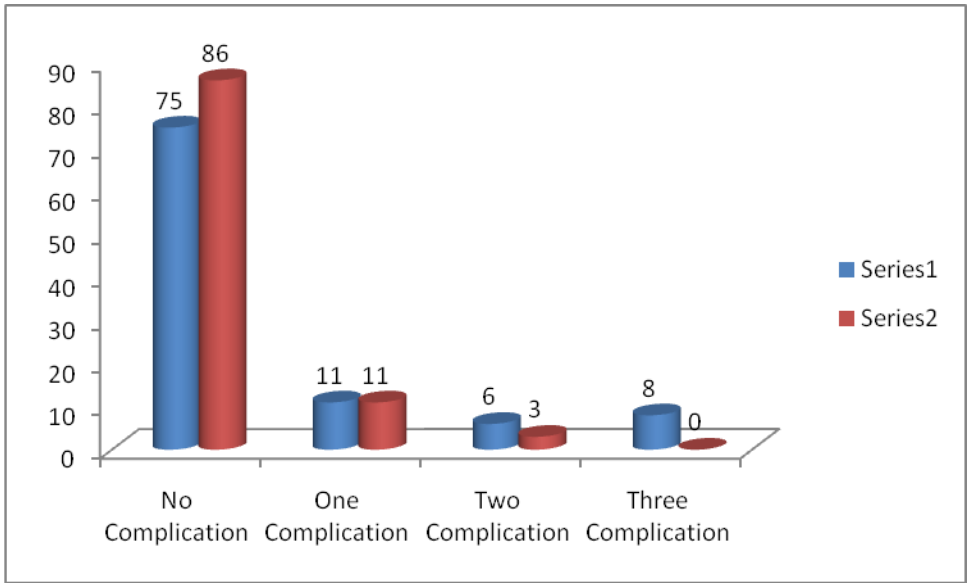




Duration of stay more than 15 days in emergency and as well as elective surgery having more chances of development of complication

Comparison of Complications of laparotomy wound in emergency and elective.

	Emergency		Elective			
	F	%	F	%	p value	t
No Complication	27	75	31	86	0.083	1.76
One Complication	04	11	04	11		
Two Complication	02	06	01	03		
Three Complication	03	08	00	00		
Total	36	100	36	100		

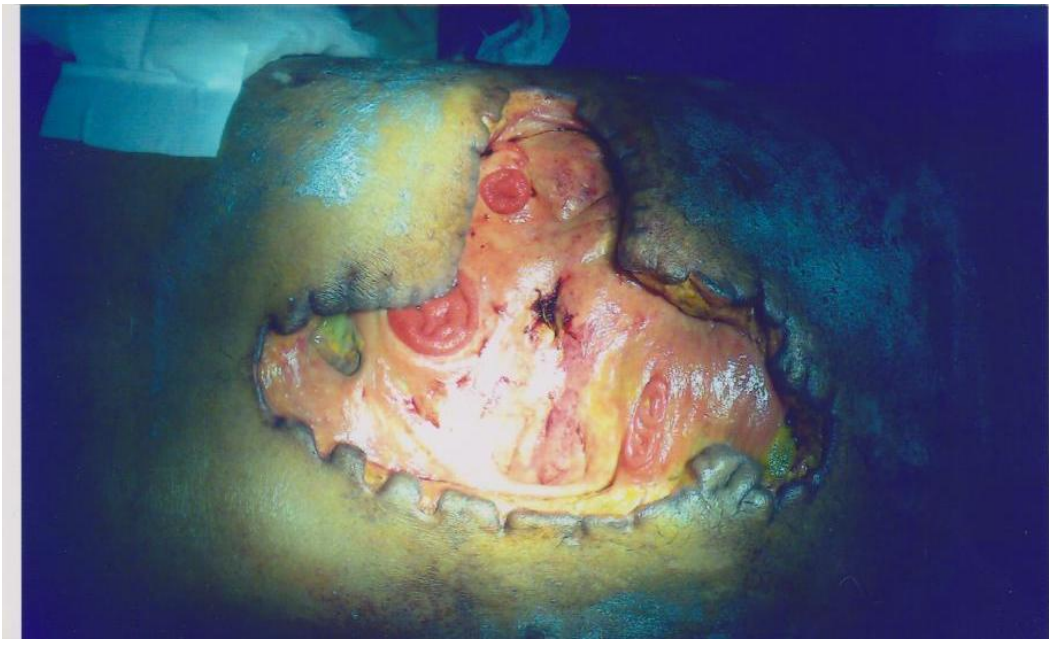


Emergency surgery – 25% complication

Elective surgery – 14% complication

NORMAL WOUND HEALING

SUPERFICIAL INCISIONAL SURGICAL SITE INFECTION



DEEP INCISIONAL SURGICAL SITE INFECTION

DISCUSSION

According to Lars Tue Sorensen MD et al. and Milorad Paunovic in their study demonstrated a significant increase in incidence of postoperative tissue and wound complications in emergency than elective surgery($p<.05$)[27,28]. Cavit COL, Atilla SORAN demonstrate no much difference in postoperative tissue and wound complications in emergency and elective surgery($p>0.05$)[29,] Similarly, in this study there was no significant increase in incidence of postoperative tissue and wound complications in emergency and elective surgery,($p=0.08$).

Common for all tissues subject to surgery is a disruption of the local vascular supply, thrombosis of the vessels, and tissue hypoxia. Once the blood supply is restored, several factors may complicate healing. The most important seems to be the proliferation of bacteria in the wound and tissue, which affects each process involved in healing and increases the risk of wound infection, delayed healing, and dehiscence[27,28]. Pathogenic organisms cause a decrease in TS(tissue strength) and fibroblast concentration, so that tissue destruction occurs[29.]

Whereas factor affecting SSI, according to CDC are extremes of age, poor nutritional status, presence of diabetes, obesity, nicotine or steroid use, a coincident infection or colonization and a dysfunctional immune system[31]. In this study patients with age > 50 years had more complications, but overall it is not significantly associated with wound complications in emergency and elective surgery. ($p>0.05$)

According to Lars Tue Sorensen MD et al. following emergency surgery males were associated with Increased risk[27].Similar finding was found by Milorad Paunovic in his

study[28]. According to Suchitra Joyce B et al females are significantly associated with SSI[32]. In this study there was no significant association between male or female.

Traditional wound classifications are a reasonably effective method to predict the inherent risk of developing an SSI from a specific procedure. For example, although the risk of developing an infection from a clean, Class I surgical procedure is low, the risk progressively increases from a Class I surgery to a Class IV surgery[31.]

The risk for developing an SSI was heightened for patients undergoing Class III or Class IV surgical procedures, as well as for patients with > 3 diagnoses at the time of surgery. Abdominal procedures and surgical procedures that last > 2 hrs were additional risk factors for microbial contamination and resultant surgical site[31.]

Generally, the wound classification method does not take into account the varying intrinsic patient risk factors within any wound class. Patients undergoing surgical procedures may exhibit a number of risk factors that make them more susceptible to infection by an exogenous pathogen than the wound classification might indicate[1,31]. In emergency surgery most of the patients were in class III or class IV category, compared to elective surgery in which all patient were class II category. Thus this is one of the risk factor which is associated with development of more complications in emergency surgery.

As incorporated in national nosocomial infections surveillance system(NNIS),the most recognized factors are the wound classification, American Society of Anesthesiology, class III or higher, and prolonged operative time, where time is longer than the 75 th percentile for each such procedure[14]. In this study as ASA score was high, more chances of associated complications of laparotomy wound was noted. In emergency surgery significant association for seroma (p=0.004)superficial (p=0.025)and deep incisional SSI (0.004) were

found compared to elective Surgery where only deep incisional SSI had significant association of development of complications(p=0.028)

Poor control of blood glucose during surgery and in perioperative period increases the risk of infections ,and worsens the outcome from sepsis. Tight control of blood glucose by the anesthesiologist during surgery decreases the risk. Moderate Hyperglycemia(>200mg/dl)at any time on the first postoperative day increased the risk of SSI fourfold after noncardiac surgery. Metaanalysis of the approximately 35 existing trials indicate that the risk of postoperative infection decreases significantly by tight glucose control, regardless of whether or not the patient had diabetes mellitus[14]. In this study there was no significant association between DM and wound complications(p=>0.05)

Following elective operations, perioperative blood loss was a predictor of postoperative tissue and wound complications in a dose-dependent manner, when adjusting for other risk factors and confounders. This findings confirms previous reports and suggests that hypovolemia and reduction of tissue oxygenation by loss of red blood cells is detrimental to healing and increases the risk of infection and tissue dehiscence[27].In emergency surgery anemia was significantly associated with seroma, superficial and deep incisional SSI (p=<0.05) compared to elective surgery which was significantly associated with only deep incisional SSI.

Infection and disruption of wounds and tissues were associated with a higher risk of reoperation, and a prolonged postoperative admission [27,28]. Similarly, in this study postoperative stay was significantly associated with wound complications p<0.05 (seroma p=0.007,Superficial SSI=0.002, Deep SSI=0.001)

Most SSI are caused by gram positive cocci including Staphylococcus aureus, stapthylococcus epidermidis and enterococcus species which are mostly skin derived as well as Escherichia coli which is from intestinal tract[14]. In this study Ecoli and Staphylococcus aureus being most common in emergency surgery and Coagulase- negative Staphylococcus in elective surgery .

Lars Tue Sorensen MD et al. and Milorad Paunovic in their study found that overall incidence of tissue and wound complications was 6% following elective operation and 16% following emergency operation[27,28], In this study incidence of postoperative tissue and wound complications in elective surgery was 14% and in emergency surgery was 25%. These values are higher compare to other studies , In elective surgery most of patients having malignancy, which itself is a risk factor might be associated with higher value of complications.

The limitation of this study is small sample size, as well as wound complications which are multifactorial and depends on other factors also like obesity, nutritional status mainly hypoprotinemia, immunocompromised state like tuberculosis HIV etc. Still this study found that ASA score ,anemia are significantly associated with wound complications and as wound class is higher there are more chances of development of complications according to NNIS there are the most recognized factors.

CONCLUSION

In conclusion, laparotomy wound complications are multifactorial, It depends on many factors. This study demonstrated no significant increase in incidence of postoperative tissue and wound complications in emergency(25%) and elective(14%) surgery (p=0.08).It mainly depends on higher ASA score ,anemia and higher wound class are more likely to associated with development of wound complications.

Patients with a larger number of predictors are under highest risk. This study provided data for preoperative identification of patients with a high risk of postoperative tissue and wound complications. Further, development of clinical pathways would prove valuable if the absolute risk of each patient could be estimated when planning surgery to specifically optimize the patient's preoperative condition to reduce the risk of complications.

SUMMARY

The study was conducted on 72 patients out of which 36 patient underwent major emergency and 36 patient underwent elective laparotomy surgery at Shri. B. M. Patil Medical College, Hospital and Research centre, between October 2008 to May 2010.

Patients age, ASA score, anemia, DM and postoperative stay are taken into consideration and comparative study of complications of laparotomy wound in elective and emergency surgery is done.

Statistical analysis was done accordingly, p-value less than 0.05 was considered significant. Study demonstrated no significant increase in incidence of postoperative tissue and wound complications in emergency (25%) and elective surgery (14%), (p=0.08). Higher ASA score ,anemia and higher wound class are more likely to be associated with development of wound complications.

BIBLIOGRAPHY

1) Alicia J , Mangram MD , Teresa C, Horan, MPH, CIC et al. Guidelines for prevention of surgical site infection 1999 ; 20 (4) : 247 – 270
2) Merril T, Dafton, MD. In Surgical complications Sabiston text book of Surgery. . 18th edn. Saunders; Elsevier ; 2008 ; 1(15) : 311 – 335
3) Dunn L David, "History repeats itself Connections and Casuality in the study of surgical Infections". Arch surg 1994; 129 : 21 – 22
4) Ananthanarayanan R and C. K. Jayaram Panikar, " Text book of Microbiology". Madras, Onent Longmen – Hydrebad. 2005 ;1-6.
5) Peter J.E Cruse In: History of Surgical Infection Fry surgical Infections 1995; 3 – 9
6) David J. Leaper, In : Surgical infection Bailey and Loves short practice of surgery 25th edn 4 : 32-47.
7) Maria B, Wittle MD, Adrian Barbul. General Principles of wound Healing ; Surgical Clinics of North America June 1997 ; 77: 509 – 526
8) Richard T, Ethridge, Mimi Leong et al. Wound healing, Sabiston text book of surgery . 18th edn. Saunders ; Elsevier ; 2008; 1 :191 – 216
9) Thomas K, Hunt MD. Wound Healing. Current surgical diagnosis and treatment. 12th edn, 2009; 6: 75 – 88
10) Robbins and Cotran "Pathologic Basis of Disease" 7th edn. Saunders; Elsevier 2004 ; 3: 107 - 116
11) Bailry , Robert W and Elvyn G Scott. Diognostic Microbiology, Saint Louis : The C.V. Mosby company 1974; 90 – 97

12) Thomas K, Hunt, Harriet Williams. Wound Healing and Wound Infection. Surgical clinics of North America June 1997 ; 77 : 587 – 606

13) Peter J, Cruse E , Rosemany Fourd M.B. The Epidemology of wound Infection; surgical clinics of North America 1980; 60 (1) : 27 – 40

14) Philip S, Barie, Soumitra R, Eachempati. Surgical site Infections. Surg. Clin N Am 2005 ; (85) :1115 – 1135

15) Lee M, Ellis, Edward M, Copeland III et al. Perioperative Nutritional Support; "Surgical clinics of North America" 1991 June; 71(3) : 493 – 507

16) William D, Hardin, Jr. Ronald L. Nichols "Aseptic Technique in the operating Room Surgical Infections 'Fry' 1991 ; 109 – 117

17) P.K. Agarwal, M Agarwal et al. "Incidence of Post-operative wound Infection at Aligarh 'Indian Journal of Surgery 1984 June – July ; 326 – 333

18) J. Wesley Alexander, Josef E Fischer et al. The Influence of Hair – Removal Methods on wound Infections, Arch surg 1983 March; (118) : 347 – 352

19) M.A. Khan, M.N Ansari et al. Post – operative wound Infection IJS 1983 : 383 – 386

20) Gerard M. Doherty " Post operative complications" Current surgical diagnosis and treatment. 12$^{th\ edn}$ (4) : 21 -23

21) Maria D, Allo, Maureen Tedesco. "Operating Room Management " Operative suite considerations, Infection Control. Surg Clin N Am 2005; 85 : 1291 – 1297

22) Ronald Lee Nichols. Surgical Antibiotic Prophylaxis; Medical clinics of North America 1995 May; 79 (3): 509 – 521

23) James T. Lee Surgical wound Infections : Surveillance for Quality Improvement Donald E Fry: Surgical Infections 1995; (14) : 145 – 159

24) Lilani SP, Jangale N, Chowdhary A et al. Surgical site Infection in clean and clean – contaminated cases Indian Journal of microbiology 2005 ; 23 : 249 – 252

25) Ussiri EV, Mkony CA, Aziz MR et al. Surgical wound Infection in clean contaminated and contaminated Laparotomy wounds of Muhimbili National Hospital, East and central African Journal of surgery 2005; 10(2) : 19 – 23

26) Kamat US, Fereirra A.M.A, Kulkarni M.S et al. A prospective study of surgical site Infections in a teaching hospital in Goa Indian J. Surg 2008; 70 (3): 120 – 124

27) Finn kallehave MD, Peer wille – Jorgensen Lars Tue Sorensen et al. Risk factor for tissue and wound complication in Gastrointestinal surgery Ann Surgery 2005; 241 (4) : 654 – 658

28) Milorad Paunovic. Predictors of postoperative complications in gastrointestinal surgery Acta Medica Medianae 2008;47:15-19

29) Cavit COL, Atilla SORAN and Meltem COL. "Can Postoperative Abdominal wound Dehiscence be Predicted" Tokia J Exp clin Med 1998; 23 (3):123-127

30) John Spiliotis, Konstantinos Tsiveriotis et al. Wound Dehiscence: is still a problem in the 21th century: A retrospective study World Journal of Surgery 2009;4:12

31) Sander Florman, Ronald Lee Nichols et al. Current Approaches for the Prevention of Surgical Site Infections Am. J. Infect. 2007 Dec ; 3(1): 51-61

32) Suchitra Joyce B. and Lakshmidevi N. Surgical Site Infections: Assessing risk factors, outcomes and antimicrobial sensitivity patterns. Africa Journal of microbiology Research 2009; 3(4): 175-179

PROFORMA

SCHEME OF CASE – TAKING

Case No :

NAME: **I.P. No:**

AGE: **SEX:**

UNIT: **RELIGION:**

OCCUPATION: **ADDRESS:**

D.O.A: **D.O.D:**

CLINICAL DIAGNOSIS:

OPERATIVE DIAGNOSIS:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING COMPLAINTS:

1. PAIN

- Duration
- Time of onset
- Site of pain
- Radiation of pain
- Relation to posture
- Aggravating / relieving factors

2. VOMITING

Duration Character

Contents Colour

Frequency and quality

3. H/O FEVER:

4. DISTENTION OF ABDOMEN:

5. HAEMATEMESIS/MELAENA:

6. LAST FOOD INTAKE:

PAST HISTORY

H/O Pain abdomen

PERSONAL HISTORY

Habits

Appetite

Micturition

Bowels

Diet

FAMILY HISTORY

GENERAL EXAMINATION

Attitude - Quite/Rolling	Nutritional Status
Appearance	Anemia
Jaundice	Dehydration

VITAL DATA:

Temperature	Pulse
B.P	Respiratory Rate

LOCAL EXAMINATION

INSPECTION

Contour of Abdomen	Movement on Respiration
Umbilicus	Visible peristalsis
Skin over abdomen	Hernial Orifices

PALPATION

Tenderness	Rigidity and Guarding
Distension	

PERCUSSION

Shifting dullness	Obliteration of liver dullness

AUSCULTATION

Bowel Sounds

PER RECTAL EXAMINATION

PER VAGINAL EXAMINATION

SYSTEMIC EXAMINATION

RESPIRATORY SYSTEM

CARDIOVASCULAR SYSTEM

CENTRAL NERVOUS SYSTEM

ASA Score

INVESTIGATONS

1. Blood ;- Hb%
2. TC
3. DC N L E M B
4. BT CT
5. Blood Grouping
6. Blood Urea
7. Blood glucose
8. Serum Creatinine
9. Urine Albumin
   Sugar
   Microscopy
10. ECG
11. HIV/HbsAg
12. X-ray Abdomen (erect),
    Chest X-ray
13. U.S.G Abdomen and pelvis
14. Others (if any)

PROVISIONAL DIAGNOSIS:

INTRA-OPERATIVE EVALUATION

1) Surgical procedure

2) Prophylactic antibiotics

3) Type of anaesthesia

POST-OPERATIVE EVALUATION OF SURGICAL SITE

1. Wound evaluation

    a. Normal wound healing

    b. Superficial incisional SSI

    c. Deep incisional SSI

2. Wound discharge

    a. Serous

    b. Serosanguinous

    c. Purulent

    d. Other

3. Culture and sensitivity of the discharge

4. Treatment given

5. Duration of post -operative stay

6. Condition at time of discharge

7. Follow-up

8. Comments

DATE

PLACE **SIGNATURE OF THE ATTENDING DOCTOR**

SAMPLE INFORMED CONSENT FORM

TITLE OF THE PROJECT	:	PROSPECTIVE COMPARATIVE STUDY OF COMPLICATIONS OF LAPAROTOMY WOUND IN ELECTIVE AND EMERGENCY SURGERY.
GUIDE	:	Dr. BALASAHEB BHIMRAO METAN (PROFFESOR OF SURGERY)
	:	
P.G. STUDENT	:	Dr. SOMANI RUSHABHKUMAR C

PURPOSE OF RESEARCH:

I have been informed that this study is conducted to know post operative wound complications associated with laparotomy. I have also been given free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study will help to know the wound complications in postlaparotomy patients.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime

Dr. Somani Rushabhkumar C. at the department of surgery who will be available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. Somani Rushabhkumar C. may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr. Somani Rushabhkumar C. Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Somani Rushabhkumar C. has explained to me the purpose of research, the study procedure, that I will undergo and the possible discomforts as well as benefits that I may experience in my own language. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to signature) Date

KEY TO MASTER CHART

IO	:	Intestinal obstruction
FP	:	Fecal contamination
Pro	:	Procedure
SSI	:	Surgical site infection
POS	:	Post operative stay
Cul	:	Culture
MI	:	Mesentric ischemia
HVP	:	Holo viscus perforation
GB	:	Gangrenous bowel
RA	:	Resection anastomosis
PACA	:	Periampullary carcinoma
GIST	:	Gastrointestinal stromal tumors
GOO	:	Gastric outlet obstruction
CA S	:	Carcinoma of Stomach
TV	:	Truncal vagotomy
GJ	:	Gastrojejunostomy
CHD	:	Choledochoduodenostomy
CHJ	:	Choledochojejunostomy
CA P	:	Carcinoma Pancreas
THE	:	Transhiatial oesophagectomy
C	:	Cholecystitis
Ch	:	Cholecystectomy

S O : Salphingo oopherectomy

HC : Hemicolectomy

CA C : Carcinoma of Caecum

CA R : Carcinoma of Rectum

APR : Abdomino perineal resection

W : Whipples procedure

CA E : Carcinoma Esophagus

CBDS : CBD stricture

PS P : Pseudocyst of Pancreas

CG : Cystogastrostomy

APP : Appendicectomy

SPL : Splenic laceration

SP : Splenectomy

CL-P : Closure of perforation

Ad : Adhesiolysis

I : Ileostomy

L-L : Liver laceration

He-P : Hepatorrhaphy

G-I : Gangrenous Ileum

S-V : Sigmoid Valvulus

Staph : Staphylococcus

Coag-ve cocei : Coagulase- negative Staphylococcus

MASTER CHART – EMERGENCY LAPARATOMY

Sl No	Name / IP No	IP NO	Age	sex	Dignosis	Pro	Class	Serome	SSI		ASA	DM/Anemia		POS	CUL
									Sup	Deap		DM	Anemia		
1	SHIVAPPA	13650/08	66	M	HVP	CL-P	III	A	A	A	3	A	A	12	-
2	AMOGA	14578/08	40	M	HVP	RA	III	A	A	A	3	A	A	12	-
3	PRAKASH	15964/08	47	M	HVP	CL-P	III	A	A	A	2	A	A	11	-
4	ASHOK TELI	16047/08	24	M	SP-L	SP	III	A	A	A	2	A	A	14	-
5	DATTA KOLI	16050/08	25	M	L-L	He-p	III	A	A	A	2	A	A	12	-
6	SHANTAMMA	164/09	65	F	G-I	RA	IV	A	P	A	2	P	P	20	Ecoli
7	LACHAPPA	372/09	55	M	HVP	CL-P	III	A	A	A	3	A	A	16	-
8	DUNDAPPA	379/09	50	M	HVP	CL-P	III	A	A	A	2	A	A	10	-
9	LAXMIBAI	920/09	44	F	HVP	CL-P	III	A	A	A	2	A	P	15	-
10	SOMANNA	1131/09	45	M	MI-G-I	RA	IV	P	P	P	2	A	P	49	Ecoli /staph
11	DUNDAPPA	1134/09	65	M	HVP	CL-P	III	A	A	A	3	A	P	16	-
12	GURAPPA	1155/09	60	M	HVP	CL-P	III	A	A	A	2	A	A	12	-
13	GURAPPA	1193/09	24	M	MI	RA	III	P	P	A	4	A	P	15	staph
14	MALAKANNA	2548/09	45	M	HVP	App	IV	A	A	A	2	A	A	10	-
15	SUJATA	3058/09	16	F	HVP	App	III	A	A	A	2	A	A	13	-
16	SIDDAMMA	3662/09	52	F	S-V	RA	III	A	A	A	2	A	A	13	-
17	BASAGONDAPPA	3728/09	55	M	SPL	SP	III	A	A	A	3	A	A	11	-
18	PREMKUMAR	3946/09	13	M	HVP	CL-P	III	A	A	A	1	A	A	13	-
19	M.A.PATIL	4976/09	40	M	HVP	App	III	A	A	A	2	A	A	9	-
20	RAJU WALIKAR	9109/09	28	M	SPL	SP	III	A	A	A	3	A	P	16	-
21	RAMESH	10211/09	21	M	IO	Ad	III	A	A	A	2	A	A	15	-
22	RENUKA	10452/09	22	F	IO-FP	I	IV	A	A	P	2	A	P	45	Ecoli
23	NAGAPPA	12541/09	46	M	AC-C	Ch	II	A	A	P	3	P	A	32	staph

Sl No	Name / IP No	IP NO	Age	sex	Dignosis	Pro	Class	Serome	SSI		ASA	DM/Anemia		POS	CUL
									Sup	Deap		DM	Anemia		
24	MANAPPA	14264/09	55	M	AC-G-C	Ch	III	A	A	A	3	P	A	12	-
25	MATARBA	16395/09	60	M	HVP	App	III	A	A	A	3	A	A	14	-
26	KRISHNA	17219/09	30	M	SPL	SP	III	A	A	A	2	A	A	11	-
27	MAYAWWA	17225/09	35	F	IO	Ad	II	A	A	A	2	A	A	14	-
28	BASAPPA	17248/09	55	M	HVP	CL-P	III	A	A	A	2	A	A	14	-
29	GURUNATH	18765/09	18	M	HVP	HVP	III	A	A	A	3	A	A	13	-
30	MADVAMANI	18860/09	53	M	G-I	RA	IV	A	P	A	3	A	P	22	Ecoli
31	SHARANAMMA	18898/09	32	F	HVP	CL-P	III	P	P	P	4	A	P	20	Ecoli /staph
32	KADAYYA	1916/10	18	M	HVP	CL-P	III	A	P	A	3	A	A	23	Sterile
33	CHANDRAVVA	4444/10	34	F	IO	Ad	II	A	A	A	3	A	A	13	-
34	HANIF	13101/10	25	M	HVP	CL-P	III	P	P	A	3	A	A	35	Sterile
35	NAGANNA	14847/10	55	M	IO	Ad	II	A	A	A	2	A	A	13	-
36	KULASUMA	15169/10	35	F	IO	I	IV	A	A	A	3	A	P	17	-

MASTER CHART – ELECTIVE LAPARATOMY

Sl No	Name	IP NO	Age	sex	Dignosis	Pro	Class	Serome	SSI		ASA	DM/Anemia		POS	CUL
									Sup	Deap		DM	Anemia		
1	BASAPPA	14508/08	70	M	CA R	APR	II	A	A	A	2	A	A	17	-
2	BHIMRAYA	14543/08	56	M	C	Ch	II	P	P	A	2	A	A	22	Staph
3	JINNAPPA	1771/09	70	M	CA S	GJ	II	A	A	A	2	A	A	15	-
4	NEELAMMA	2880/09	32	F	CAC	R HC	II	A	A	A	2	A	A	16	-
5	PEERAMMA	3611/09	55	F	PA CA	W	II	A	A	A	2	A	A	13	-
6	CHANDRAMMA	3812/09	65	M	CA S	GJ	II	A	A	A	2	A	A	14	-
7	GURUSIDDAMMA	5280/09	46	F	CA E	THE	II	A	A	A	2	A	A	11	-
8	IRABASAYYA	7579/09	45	M	CA S	GJ JJ	II	A	A	A	3	A	A	16	-
9	SHANTGOUDA	7779/09	64	M	CA E	THE	II	A	A	A	3	A	A	18	-
10	NAJEMA	8962/09	58	F	CA C	RA	II	A	A	A	2	P	A	17	-
11	NEELAMMA	9033/09	32	F	CA S	GJ	II	A	A	A	2	A	A	12	-
12	CHANDRAWWA	9194/09	65	M	CA E	THE	II	A	P	A	2	A	A	24	Coag-ve cocei
13	DURADUNDAYYA	10094/09	76	M	CBDS	CHD	II	A	A	A	2	A	A	11	-
14	GURAPADEPPA	10135/09	67	M	CA S	GJ	II	A	A	P	3	A	P	21	Coag-ve cocei
15	JAKIR	10262/09	36	M	PS P	CG	II	A	A	A	3	A	A	12	-
16	MUKTUMSAB	11193/09	35	M	PA CA	GJ	II	A	A	A	2	A	A	12	-
17	DHULAPPA	12589/09	50	M	GIST	BL-2	II	A	A	A	3	A	A	13	-
18	RAMAGONDAPPA	14289/09	51	M	GOO	TV+ GJ	II	A	A	A	2	A	A	10	-
19	SHIVAPPA	15675/09	56	M	CA S	GJ	II	A	A	A	3	A	A	8	-
20	MAHANAND	15983/09	35	M	CA CH	GJ	II	A	A	A	2	A	A	11	-
21	YAMANAPPA	16054/09	44	M	GOO	TV+ GJ	II	A	A	A	2	A	A	11	-
22	DHANESHING	19410/09	35	M	CA CH	CHD	II	A	A	A	3	A	A	11	-
23	SHARANAPPA	3752/10	25	M	D CA	RA	II	A	A	A	2	A	A	10	-

Sl No	Name	IP NO	Age	sex	Dignosis	Pro	Class	Serome	SSI		ASA	DM/Anemia		POS	CUL
									Sup	Deap		DM	Anemia		
24	MALAKANNA	4898/10	64	M	PA CA	CHJ+ GJ	II	A	P	A	3	A	A	27	Sterile
25	SANGAPPA	4819/10	55	M	CA CH	W	II	A	A	A	2	A	A	12	-
26	BHEEMAWA	7939/10	73	F	CA P	GJ	II	A	A	A	2	A	A	15	-
27	SHIVAPPA	10588/10	38	M	GOO	PG+ GJ	II	A	A	A	2	A	A	13	-
28	YAMANAPPA	11484/10	56	M	CA E	THE	II	A	A	A	2	A	A	18	-
29	VEERUPAKSHAPPA	11770/10		M	CA R	EC	II	A	A	A	2	A	A	14	-
30	RUDRAGOUDA	15783/10	60	M	C	Ch	II	A	A	P	3	A	A	29	Sterile
31	SHAVANIRAWWA	15997/10	35	F	TO	R S O	II	A	A	A	2	A	A	12	-
32	RAMESH	16688/10	34	M	PS P	CG	II	A	A	A	2	A	A	12	-
33	RAJMAL	16943/10	58	M	CA C	R H C	II	A	A	A	2	A	A	12	-
34	GURASIDDAPPA	17496/10	78	M	SMA	G J	II	A	A	A	3	A	A	15	-
35	KALLAPPAGOUD	18644/10	70	M	C	Ch	II	A	A	A	3	A	A	9	-
36	SHALU	21579/10	58	f	C	Ch	II	A	A	A	2	P	A	6	-