"A LONGITUDNAL STUDY OF ASSOCIATION BETWEEN BODY MASS INDEX AND SUPERFICIAL SURGICAL SITE INFECTION IN INCISIONAL HERNIA PATIENTS AT TERTIARY CARE HOSPITAL"

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This is to certify that the dissertation entitled **"A LONGITUDNAL STUDY OF ASSOCIATION BETWEEN BODY MASS INDEX AND SUPERFICIAL SURGICAL SITE INFECTION IN INCISIONAL HERNIA PATIENTS AT TERTIARY CARE HOSPITAL "** is a bonafide research work done by **REG. NO. BH0111007**

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ABSTRACT

Introduction

Surgical site infection is one of the most common postoperative complications following abdominal surgeries. Obese patients have tissue hypoperfusion (subcutaneous adipose tissue), which may predispose to SSI through a greater risk of ischemia/necrosis and suboptimal neutrophil oxidative killing. The growing prevalence of obesity and the increasing number of operations performed on obese patients have a substantial impact on the outcome of open incisional hernia repair**.**

However, data regarding the role of obesity among patients with SSI who underwent incisional hernia are scanty and to date very few studies have been conducted. Hence the present study was an undertaken to find association between body mass index and superficial surgical site infection in incisional hernia patients.

Objective

To find association between body mass index and superficial surgical site infection in incisional hernia patients.

Methodology

All patients diagnosed with incisional hernia during the study period were studied in Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital and MRC, Belgaum during the year 2012 were included in the study. A thorough clinical examination was conducted. Height and weight was recorded

and body mass index was calculated Patients were inspected on day 3, day 5 and day 7, day 15 & day 30 following surgery and signs of surgical site infection if any, were recorded. Outcomes were tested for significance.

Results

Out of 60 patients 5 patients developed superficial surgical infection, 4 patients belonged to normal weight group and one patient in obese group.

Conclusion

There is no significant association between body mass index and superficial surgical site infection (p= 0.241) in incisional hernia patients in our study. Identification of surgical site infection and its frequency in obese patients is critical to approach its reduction. Further studies are required to conclude better association between body mass index and superficial surgical site infection in obese patients. Although this study has not shown significant association between BMI and superficial SSI it is necessary to take extra precautions in obese / overweight patients undergoing hernia surgery.

Keywords

Incisinal hernia; Body mass index; Superficial surgical site infection;

LIST OF ABBREVIATIONS USED

AIDS	-	*Acquired immunodefiency syndrome*
BMI	-	Body mass index
CDC	-	Centres for Disease Control
CDLC	-	Continuous double-loop closure
cm	-	Centimeter
eg.	-	For example
EGF	-	Epidermal growth factor
FGF	-	Fibroblast growth factor
HIV	-	*Human immunodeficiency virus*
i.e.	-	That is
ICU	-	Intensive care unit
IGF	-	Insulinlike growth factor
IH	-	Incisional Hernia
Kg	-	Kilogram
LOS	-	Length of stay
LRYGBP	-	Laparoscopic Roux-en-Y gastric bypass
m	-	Meter
MMP	-	Metalloproteinases
MOP	-	Morbidly obese patients
MRSA	-	Methicillin resistant *Staphylococcus aureus*
n	-	Total number
NINSS	-	Nosocomial Infection National Surveillance Service
PDGF	-	Platelet derived growth factor
PDS	-	Polydioxanone-s

SBO	-	Small bowel obstruction
SIRS	-	Systemic inflammatory response syndrome
SSI	-	Surgical site infections
TGF	-	Transforming growth factor
VISA	-	vancomycin intermediate *Staphylococcus aureus*

CONTENTS

SL. NO.	TOPIC	PAGE NO.
1	INTRODUCTION	1
2	OBJECTIVES	3
3	REVIEW OF LITERATURE	4
4	METHODOLOGY	42
5	RESULTS	47
6	DISCUSSION	60
7	CONCLUSION	66
8	SUMMARY	67
9	BIBLIOGRAPHY	68
10	ANNEXURES	
	ANNEXURE I – CONSENT FORM	82
	ANNEXURE II – PROFORMA	88
	ANNEXURE III – PHOTOGRAPHS	91
	ANNEXURE IV – MASTER CHART	95

LIST OF TABLES

TABLE. NO.	DESCRIPTION	PAGE NO.
1	Sex distribution	48
2	Age distribution	49
3	Body mass index	50
4	Assessment of wound on Day three	51
5	Assessment of wound on Day five	52
6	Assessment of wound on Day seven	53
7	Assessment of wound on day fifteen	54
8	Assessment of wound on day thirty	55
9	Wound culture	56
10	Organism	57
11	Antibiotic sensitivity	58
12	Association of Body mass index with surgical site infections	59

LIST OF FIGURES/GRAPHS

GRAPH NO.	DESCRIPTION	PAGE NO.
1	Sex distribution	48
2	Age distribution	49
3	Body mass index	50
4	Assessment of wound on Day three	51
5	Assessment of wound on Day five	52
6	Assessment of wound on Day seven	53
7	Assessment of wound on day fifteen	54
8	Assessment of wound on day thirty	55
9	Wound culture	56
10	Organism	57
11	Antibiotic sensitivity	58
12	Association of Body mass index with surgical site infections	59

LIST OF PHOTOGRAPHS

PHOTO NO.	DESCRIPTION	PAGE NO.
1	surgical site infection post incisional hernia surgery	91

INTRODUCTION

A hernia, as defined by Astley Cooper, is a protrusion of any viscus from its proper cavity. The protruded parts are generally contained in a sac-like structure, formed by the membrane with which the cavity is naturally lined.[1] Since that time, many different types of abdominal wall hernias have been identified, along with a larger number of associated eponyms. Hernias are brought to the attention of an physician either during a routine physical examination for other medical complaints or when the patient has developed a complication associated with the hernia.

An incisional hernia is any abdominal wall gap with or without a bulge in the area of a previous operative scar [1]. It is a common long-term complication following abdominal surgery. This condition is iatrogenic and occurs in 2-10% of all abdominal operations.It is secondary to breakdown of the fascial closure of the surgery. Even after repair, recurrence rates approach 20-45%.[2] The risk factors for the development of incisional hernia include obesity, diabetes, emergency surgery, postoperative wound dehiscence, smoking and postoperative wound infection.[3,4]

Incisional hernia can be repaired by open or by laparoscopic approach and prosthetic meshes are nowadays implanted in most procedures. The use of laparoscopy for the treatment of incisional hernia was first reported in 1993 by LeBlanc and Booth.[5] The introduction of prosthetic mesh revolutionised the treatment of hernia's[6] The risks of repairing an incisional hernia include seroma formation, wound infection, injury to intra-abdominal structures and recurrence.

Surgical site infections (SSI) are the third most common hospital-acquired infection and account for 14% to 16% of all such infections.[7] The definition of

surgical site infections (SSIs) according to the criteria developed by the Centers for Disease Control and Prevention include every SSI up to 30 days after the operation.[8] Infections are categorized as incisional (superficial or deep) infections or organ–space infections. Superficial SSIs involve only skin and subcutaneous tissue and exclude stitch abscesses. Deep SSIs involve deeper soft tissues at the site of incision. Organ–space SSIs are defined as infections in any organ or space.

In incisional hernia repair the incidence of SSI is low. In a meta-analysis of 8 randomized controlled trials Forbes et al. showed a significant reduced risk of surgical site infections in laparoscopic incisional hernia repair compared to open surgery.[9] The extensive tissue dissection which is associated with the open approach explains the significant higher infection rate in open surgery. Mostly SSIs in laparoscopic surgery are superficial and can be treated conservatively. Mesh removal due to an surgical site infection is very rare.[10]

Obese patients have tissue hypoperfusion (subcutaneous adipose tissue), which may predispose to SSI through a greater risk of ischemia/necrosis and suboptimal neutrophil oxidative killing.[11] The growing prevalence of obesity and the increasing number of operations performed on obese patients have a substantial impact on the outcome of open incisional hernia repair**.**[11]

However, data regarding the role of obesity among patients with SSI who underwent incisional hernia are scanty and to date very few studies have been conducted. Hence the present study was an undertaken to find association between body mass index and superficial surgical site infection in incisional hernia patients.

OBJECTIVES

The objective of the present study was to find association between body mass index and superficial surgical site infection in incisional hernia patients.

REVIEW OF LITERATURE

Incisional hernia

The term “Incisional Hernia” (IH) describes a condition, which occurs from months to years after laparotomy. The same is true for a recurrence of an IH. Factors such as a combination of weakness of the abdominal wall, imperfect formation of connective tissue, conditions increasing the intraabdominal pressure, etc. may lead to weakening and eventual dehiscence of the abdominal wall with the temporary or permanent eventration of parts of the intestine.

Surgical anatomy[12-17]

The anterior abdominal wall extends from the costal margins and xiphoid process superiorly to the iliac crests, pubis and pubic symphysis inferiorly. The groin is that portion of the anterior abdominal wall below the level of the anterior superior iliac spines and has been the subject of great interest to surgeons and anatomists for centuries. A three dimensional mental conception of structural relations must be acquired if one is to understand the groin anatomy and the fundamental features of a groin hernial repair. Anterior abdominal wall tissues form the inguinal canal that connects the abdominal cavity to the scrotum in men, or the labia majora in women and also form the umbilicus; both of these sites being of clinical importance.

The groin or inguinal region, is most often defined as a transitional area in which the thigh and abdomen are joined.

Soft tissue of the anterior abdominal wall

Superficial fascia

The superficial fascia of the abdominal wall consists mostly of a single layer that contains variable amount of fat. It lies between the skin and muscles of anterior abdominal wall. In the lower part, the fascia differentiates into superficial and deep layers between which lie superficial vessels and nerves and, in the groin region, superficial inguinal lymph nodes.

a) Superficial layer (Camper's fascia) being thick, areolar in nature and contains variable amount of fat and is often greatly thickened in obese individuals. Inferiorly, it lies superficial to inguinal ligament and is continuous with superficial fascia of thigh, and the outer layer of fascia covering the perineum, penis and scrotum. In this region, it is generally thin with very little adipose tissue and in the scrotum contains smooth muscle fibres, which form the dartos muscle. In females, it continues from the suprapubic skin of the abdomen into the labia majora and perineum.

b) Deep membranous layer (Scarpa's fascia) This layer contains more elastic fibres and is loosely connected by areolar tissue to the aponeurosis of external oblique, but in the midline it is intimately adherent to linea alba and public symphysis. In male, it extends to form superficial ligament of the penis and continues medially and inferiorly over penis and scrotum where it becomes continuous with membranous layer of the superficial fascia of the perineum.

Transversalis fascia

A thin layer of connective tissue lying between the inner surface of transverse abdominis and extraperitoneal fat. In the inguinal region, it is thick and dense, and augmented by the aponeurosis of transverses abdominis muscle. Medial to the femoral vessels it is thin and fused to pubis behind conjoint tendon. Some fibres spread laterally towards the anterior superior iliac spine, some fibres run medially behind rectus abdominis, and some descend to pubis behind conjoint tendon, forming deep crural arch. The curved fibres of this arch thicken the inferomedial part of the rim of the deep inguinal ring. The spermatic cord in male, or the round ligament of uterus in female, pass through the transversalis fascia at the deep ring. The transversalis fascia is prolonged onto these structures as the internal spermatic fascia surrounding the testes and blends with areolar tissue on the parietal layer of tunica vaginalis.

Superficial vessels

The anterior abdominal wall receives its blood supply from paired superior epigastric artery (terminal branch of internal thoracic artery), and inferior epigastric artery (from the external iliac artery posterior to inguinal ligament) running vertically through the tissues, and from paired posterior intercostal, subcostal and lumbar vessels running obliquely around the anterolateral aspects of the abdomen.

Other vessels namely are the superficial circumflex iliac and external pudendal vessels arising from femoral artery. All the arteries are accompanied by their respective veins and form tributaries to the femoral vein.

Lymphatic drainage

The lymphatic vessels of the anterior abdominal wall lie both superficial and deep to the deep fascia. Superficial lymphatics from the infra-umbilical skin run with the superficial epigastric vessels and vessels from lumbar and gluteal regions run with the superficial circumflex iliac vessels and drain ultimately into the superficial inguinal nodes.

Deep lymphatic vessels accompany the deep arteries. The vessels from the posterior portion of the abdominal wall pass with the lumbar arteries to drain into lateral aortic and retro-aortic nodes. Vessels from upper abdominal wall run with superior epigastric vessels to the parasternal nodes. Vessels of the lower abdominal wall drain into circumflex iliac, inferior epigastric and external iliac nodes.

Innervation

The 7^{th} to 12^{th} lower thoracic ventral rami continue anteriorly from the intercostals spaces into the abdominal wall. The rectus muscle and external oblique are both supplied by lower intercostal and subcostal nerves (T7 – T12), and the internal oblique and transverses adominis by those same nerves with the addition of iliohypogastric and ilio inguinal nerves (L1). The 11th nerve supplies the skin below the umbilicus. The ilio-inguinal nerve accompanies the spermatic cord through the external inguinal ring. It goes on to supply the medial thigh proximal to the inguinal ligament, the root of the penis and upper anterior scrotum. In the female, the nerve exits the external ring to supply the mons pubis and labium majus. Iliohypogastric nerve has some fibres in common with subcostal and ilioinguinal nerve.

The genitofemoral nerve emerges onto the anterior surface of psoas major muscle and its genital branch exits the pelvis via the deep inguinal ring and courses

with the spermatic cord, supplying the cremaster muscle. The femoral branches of the genitofemoral nerves (L1, L2) pass under or pierce the inguinal ligament, travel across the thigh lateral to the saphenous hiatus, and then travel a short distance in the femoral sheath to supply the skin over the femoral sheath.

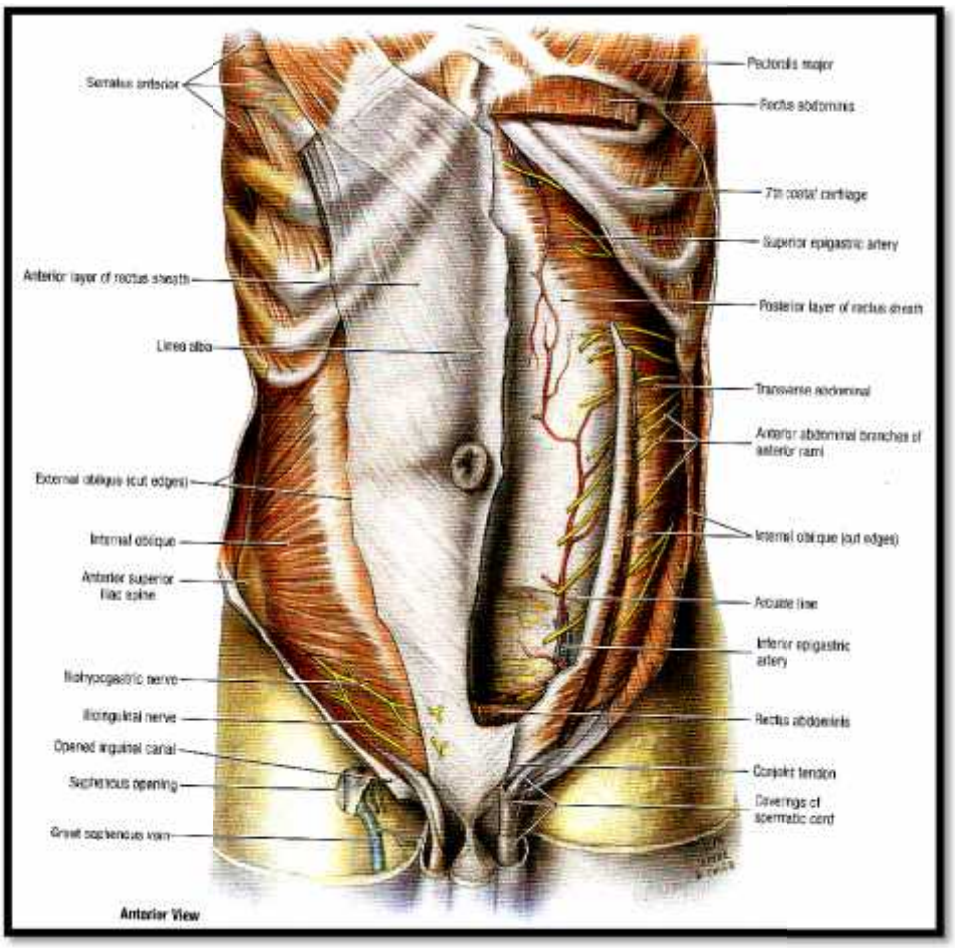


Figure 1. Vessels and nerves of the anterior abdominal wall

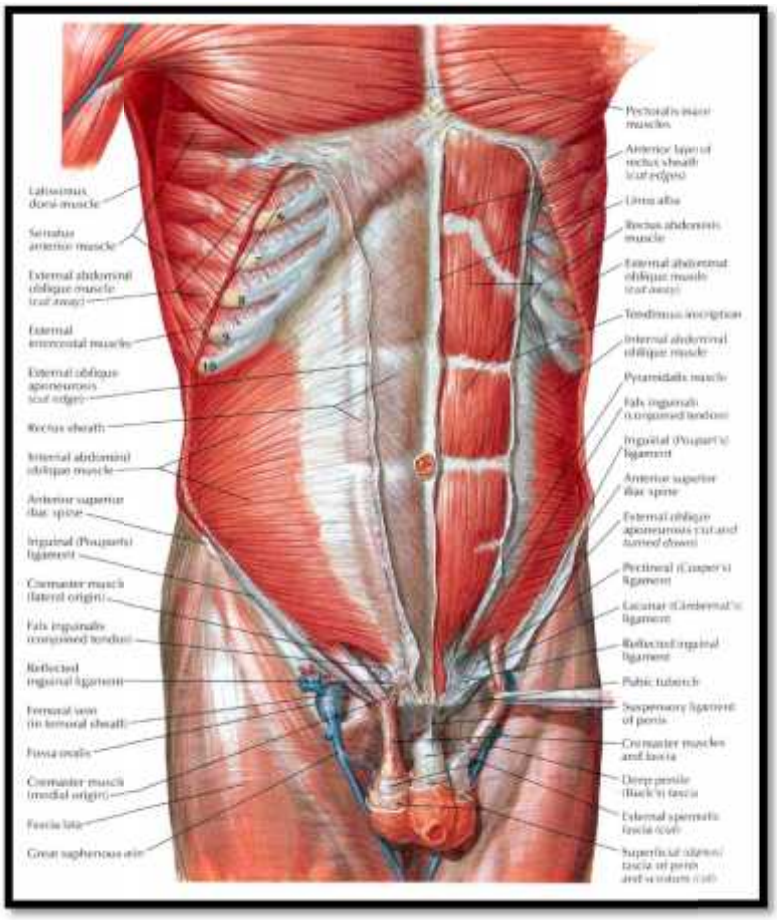


Figure 2.

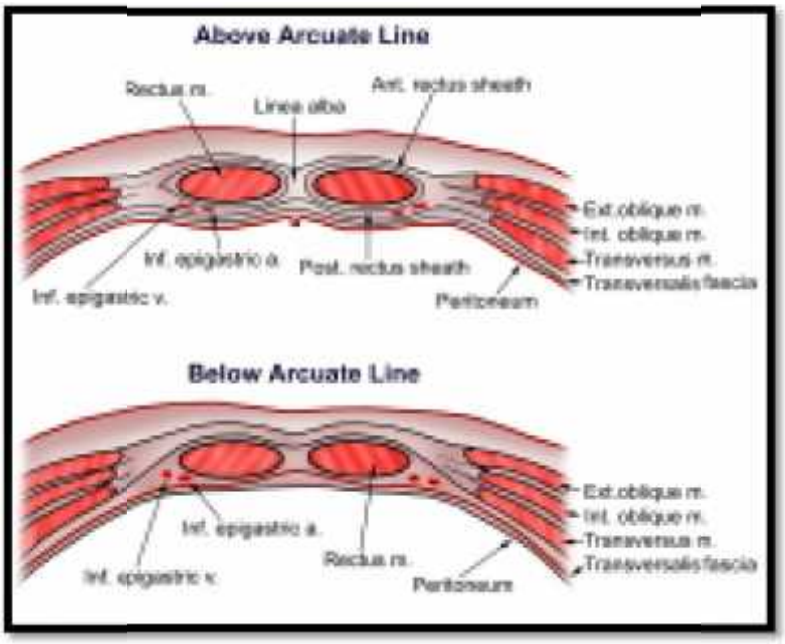


Figure 3.

Epidemiology

Incidence

Abdominal wall hernias are common, classically thought to occur in at least 2% of men[18] while statistics from the USA estimate 15 per 1000 population (1.5%).[19] More than 20 million hernias are estimated to be repaired every year around the world.[20] Every year approximately 700,000 hernia repairs are carried out in the USA,[21] and over 100,000 in the UK.[22,23] In Germany, the number of Incisional Hernia(IH) repairs for the year 2007 was estimated at 41,0003, with roughly 19,0004 Incisional hernia requiring the use of prosthetic material. At the Mesh 2009 meeting in Paris more than 38,000 cases were reported to have required a mesh in France in 2007. In light of the French data, the use of mesh in Germany seems to be underestimated. In major countries, the incidence of the use of meshes varies from 150 to 300 per million inhabitants per year (0.15 to 0.3%).[24]

Etiology

The vast majority of abdominal hernias occur after laparotomy. There is an ongoing discussion concerning which factors contribute to such a situation; is there a 'hernia disease' or simply the coincidence of several conditions or even a physiological reaction to tensile forces stretching the abdominal wall.[25]

Rosch[26] favors the inability to form physiological collagen as an essential cause for the first herniation as well as for the recurrence of such hernias. The weakness of the study is that the controls were probably not under a permanent tensile stress; therefore, the degree of fibroblastic activity in a hernia scar is likely to differ from that in a normal scar. The conclusion of some authors, who use the above

finding as an argument against meshes of biological origin, is therefore not backed up by conclusive evidence.

Post-operative samples of hernia repair with Tutomesh analysed by Klinge, suggest that, in hernias closed with Tutomesh, the collagen I / III ratio reaches almost preoperative levels. Franz[27] assumes this genetic disorder - if it really is a disorder and not a physiological reaction - to be a risk for first herniation after laparotomy; however, he does not consider it to be an essential cause for the recurrence of hernias.

Factors which may influence the risk of re-herniation encompasses a spectrum from the role of the extracellular matrix, collagen disease, surgical wound healing, impaired fibroblast activity and inflammation to impaired signaling pathways and tissue perfusion. Early and occult wound dehiscence seems to be a significant factor as a wound dehiscence of more than 12 mm leads to IH in 94% of cases, while smaller gaps develop IH only in 5% of the cases. There are two factors, which may support Franz's theory. Firstly, even the synthetic meshes obviously need the formation of normal collagen, otherwise the incorporation of mesh and tissue could not be explained. Secondly, no impaired collagen formation has yet been identified as a cause of re-herniation after the previous use of biological meshes. This statement may not apply to resorbable synthetic meshes, for which the resorption time may be too short to allow tension-bearing structures to develop or in patients with a genetic disorder of collagen formation, for instance, Marfan's syndrome.

Classification

There are several classifications for Incisinal Hernia relating to size, degree of contamination, previous surgical interventions, etc. in order to give guidelines to

select the optimal surgical procedure; however, none of them is a generally accepted standard.

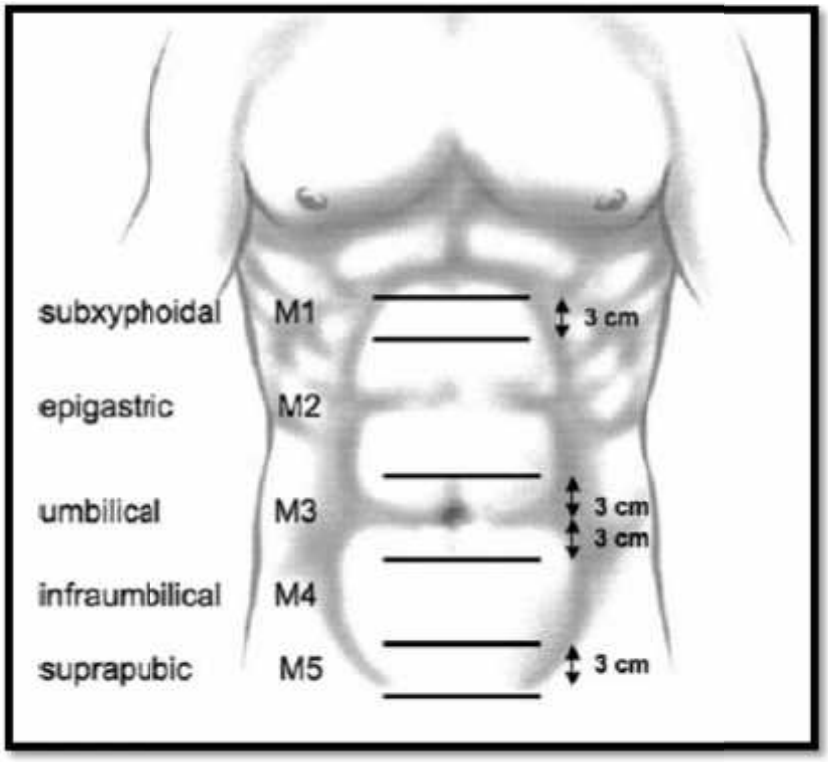


Figure 4.

An easily memorable classification from M1 to M5 going from the xiphoid to pubic bone was proposed. Defines 5 M zones:

(1) M1: subxiphoidal (from the xiphoid till 3 cm caudally)

(2) M2: epigastric (from 3 cm below the xiphoid till 3 cm above the umbilicus)

(3) M3: umbilical (from 3 cm above till 3 cm below the umbilicus)

(4) M4: infraumbilical (from 3 cm below the umbilicus till 3 cm above the pubis)

(5) M5: suprapubic (from pubic bone till 3 cm cranially).

Risk factors

Patient-Related Factors

Mudge and Hughes, among others, showed that more than 50% of the IH occurred during the 1st year postoperatively. Almost 80% of the Incisional Hernia occurred within 3 years in the same series.[28] Another study[29] reported that 52% of the Incisional Hernia appeared within 6 months postoperatively, 68% within 1 year postoperatively, and 79% within 2 years postoperatively. It seems more and more plausible that a malunion occurs between the edges of the incised aponeurosis soon after operation and that the weak fibrous tissue forming that malunion insidiously stretches, until the Incisional Hernia becomes clinically detectable. Hence, as many as 94% of the Incisional Hernia may occur within 30 days after surgery.[28]

Recurrent Incisional Hernia, however, do appear earlier. In one study,[31] 82% of the recurrences became manifest within the 1st year. Langer and Christiansen,[31] among others,[32] found that most recurrences appeared within the first 3 years and that most of them recurred within the 1st year. The most common risk factor of the recurrent Incisional Hernia seems to be the size of the primary Incisional Hernia.[32] Other factors incriminated are obesity, diabetes mellitus, lower midline incision, and wound infection.[32]

Patient-related factors that increase the risk of developing Incisional Hernia include mainly obesity, chronic lung disease, type II diabetes mellitus, age, malnutrition, renal failure, malignancy, and steroid treatment.[28]

Patient-related risk factors for Incisional Hernia

Major factors	**Minor factors**
Chronic lung disease	Age
Obesity	Male gender
Steroids	Post operative ventilation
Type II diabetes mellitus	Renal failure
Malnutrition	Connective tissue disorders
Jaundice	Malignancy
Radiotherapy	Transfusion
Chemotherapy	Anaemia
Oral anticoagulants	

Blood transfusion may predispose to wound failure and Incisional Hernia. This parameter was seldom studied in the literature. Malignant disease is frequently associated with an increased incidence of IH. To our knowledge, this dogma is more theoretical than practical, since many investigators showed no statistically significant result. However, cytotoxic agents, malnutrition, and radiotherapy are thought to be important contributing factors to wound disruption or Incisional Hernia.[28]

Oral anticoagulants, by elevating the incidence of postoperative hematoma and wound infection, seem to be a risk factor for Incisional Hernia.[28]

Jaundice is considered by many to be a risk factor for dehiscence and Incisional Hernia. Experimental evidence suggests that obstructive jaundice decreases the strength of abdominal wound healing and delays fibroplasia and angiogenesis.[28]

Wound-Related Factors

Biochemical Pathogenesis

Despite numerous predisposing factors, including anatomical features (persistence of the peritoneal-vaginal conduit, high insertion point of the transverse arch) and those associated with other diseases (obesity, chronic obstructive pulmonary disease, constipation), the underlying cause of the development of hernias, either primary or incisional, seems to be of a biologic nature. Research aimed at evaluating the role played by biological factors has centered on possible alterations in connective tissue metabolism. An abnormal collagen metabolism has been ascribed an important role in the genesis of Incisional Hernia and the high recurrence rates after surgical hernia repair.[34] This idea is also supported by the fact that diseases such as Marfan and Ehlers-Danlos syndromes, cutis laxa, osteogenesis imperfecta, and congenital hip dislocation have been associated with hernial processes. The expression patterns of certain types of collagen and certain enzyme dysfunctions have also been the subject of several studies. Metalloproteinases (MMP) represent a group of enzymes which degrade and contribute to the turnover of the extracellular matrix, acting on certain types of collagen and elastin. Furthermore, in these patients the expression of MMP-1 and MMP-13 is decreased. The decreased ratio collagen I/III is due to a relative increase of collagen type III which is known to be characterized by thin fibril diameters and lowered mechanical strength. The altered collagen ratio might be the

result of the decreased activity of MMP-1, whereas the absent MMP-13 expression does not seem to modify the scar formation according to some investigators.[34]

A balance between extracellular matrix synthesis and degradation is important for tissue integrity, because remodeling occurs relentlessly. Structural changes or defects in molecules may alter the tissue architecture, resulting in an impairment of the proper assembly of the components and modifying the mechanical properties of the tissue. The pathological hernia process seems then to be related to MMP secretion. For example, fibroblasts from the abdominal wall of young patients with direct hernias show increased active MMP-2 expression.[34] The MMP-2 expression is enhanced in processes such as genitourinary prolapse and aortic aneurysm. Further, it has been reported that patients with this latter pathology have an increased incidence of hernias. This enzyme may be intimately involved in the extracellular matrix degradative process. The persistence of this alteration in the fibroblast cultures appears to indicate a genetic defect or perhaps later transformation as the underlying cause of this pathology, ruling out environmental factors as the main cause.[28]

Peritoneal Closure

The prevention of adherence of small bowel to the abdominal wall is a major argument for proponents of peritoneal closure.[35] However, peritoneal closure causes more adhesions in animals. Moreover, in humans, the incidence of Incisional Hernia has not been reduced by peritoneal closure.[36] Since the holding strengths of sutures placed in the posterior rectus sheath – intimately attached to the peritoneum – alone or in the anterior rectus sheath alone are identical, the holding strength of the closure would be reduced by 50%, if the peritoneum is not closed. However, only 6.1% of the patients have an Incisional Hernia after breast reconstruction with a rectus abdominis

myocutaneous flap with the peritoneum as the only barrier to herniation in the lower abdomen.[28]

Type of Incision

The choice of an incision in abdominal surgery depends on the surgeon's preference, but is also determined by the adequacy of exposure and access provided. It should take into account the anticipated pathology and the contemplated operative procedure, or, on the other hand, be sufficiently versatile in the emergency situation, in order to meet the demands. Patient obesity and previous incisions may also intervene in the decision.

In traditional (open) surgery, the midline incision is the simplest, provides adequate exposure to practically all four quadrants, is rapid to open and to close, and is usually bloodless. No muscle fibers need to be divided, and no nerves are injured. The midline incision is the most widely used abdominal incision.[37] However, IH are a major problem with the midline incision. Dehiscence has been rare, if the sutures are placed widely on each side of the incision.[38] Some surgeons place sutures in the linea alba itself rather than beyond the fusion line between the two rectus sheaths in order to avoid muscle tearing. Tera and Aberg[39] have shown that the holding strength of sutures placed widely exceeds that of sutures placed directly through the linea alba, a weak structure prone to spontaneous hernias and diastasis.

The medial paramedian incision provides adequate exposure as well, but with limited trauma to rectus muscle. No nerves are injured, the closure is reputed to be secure, but both opening and closure are more time-consuming as compared with the midline incision. Some investigators failed to establish any difference between

midline incisions and medial paramedian incisions with regard to the occurrence of Incisional Hernia. However, the lateral paramedian incision was shown to be better than midline or medial paramedian incisions in terms of Incisional Hernia risks. To be effective, a paramedian incision must be lateral, not close to the midline in order to avoid ischemia of the linea alba.[28] Cahalane et al.[40] found an incidence of 0.33% of Incisional Hernia at 1 year in 1,203 lateral midline incisions from four different series.

Transverse or oblique incisions have been found to be better than the midline incisions, with less respiratory compromise, less morbidity, and, in particular, less Incisional Hernia. Distracting forces on a vertical incision during activity during the postoperative period are said to be nearly twice as great as those on a transverse incision. Many of the trials showing that Incisional Hernia is more common after midline as opposed to transverse incisions were uncontrolled for disease process. Conditions such as emergency surgery, hemorrhage, trauma, or abdominal sepsis may have had a greater influence on the development of Incisional Hernia than the type of incision used;[28] Ellis et al.,[41] in a prospective randomized trial, found no significant difference in the rates of Incisional Hernia in patients undergoing midline, paramedian, or transverse incisions. Furthermore, the strongest argument against the subcostal incision is that, unless confined exclusively to the rectus muscle, partial denervation of the abdominal wall ensues with permanent muscle weakness.

Wound Infection

Carrel[42] first described impaired wound healing secondary to infectious processes in 1924. He created cutaneous wounds in dogs, and after several days injected turpentine into the flanks to induce abscess formation. Epithelialization and wound contraction were slowed or stopped in the infected animals. In 1974, de Haan

et al.[43] found that infection decreased the bursting strength of both gastric and abdominal wounds. Although the mechanisms governing wound healing impairment by infection are still to be defined, studies on the healing of colonic anastomoses suggest that sepsis inhibits collagen synthesis at the anastomotic site.[28] A similar process affecting abdominal wounds seems likely. Riou et al.[44] identified septicemia as a risk factor for wound dehiscence. Electric cautery seems to lower the threshold of bacterial infection of the laparotomy wound in rats. The coagulation current caused more inflammation, necrosis, and abscesses than the scalpel at all bacterial levels. However, these results were not confirmed by clinical experience in humans.[28]

Wound infection is considered by many as the most important factor contributing to the development of Incisional Hernia. Bucknall et al.[46] found that 48% of the patients who developed Incisional Hernia had wound infection during the postoperative period, the presence of a wound infection conferring a fivefold increase in the rate of Incisional Hernia.[46]

Wound Closure

Many believe that there is no association between the method of wound closure and the incidence of IH. Moynihan[47] stated in 1920 that 'suture material should ideally: (1) achieve its purpose – be sufficient to hold parts together. (2) disappear as soon as its work is accomplished; (3) be free from infection, and (4) be nonirritant...'. These requisites were indeed right and still remain valid nowadays. However, Moynihan concluded: 'The only material which can be made to fulfil these conditions is catgut'.[47] However, catgut is rapidly absorbed, challenging the first item in Moynihan's definition. This illustrates the fact that the perfect suture material does not exist, since each era has had its own 'ideal' suture material. Experimental

research[48] has shown that one year after laparotomy the abdominal fascia retains only 70% of its original tensile strength. Polyglactin (Vicryl) is a polymer of glycolic and lactic acids degradable over 40–60 days, and disappearing at 75 days. Vicryl resists enzymatic digestion, body secretion, as well as infection. Synthetic absorbable sutures such as polyglycolic acid and polyglactin have the advantage of disappearing in time, since they are fully absorbed after 75 days but have no strength left after 30 days. Polydioxanone-s (PDS) is only absorbed after 180 days and retains 70% of its strength after 3 weeks. It is a monofilament suture having the advantage of nylon and polypropylene with a smooth surface which slides easily through the tissues, reducing the risks of tissue necrosis and bacteria adherence.[28]

The importance of the suture length/wound length ratio has been emphasized in many studies. Using a purely mechanical approach to wound closure, Jenkins[49] in 1976 established that the correct closure of a vertical abdominal incision implies a suture length/wound length ratio >4/1.

Measurements of the xiphoid pubic distance before and after closure demonstrate that abdominal distension may lengthen the wound by 30%. The continuous suture can accommodate to this increase in length of the incision by having an adequate reserve suture length in the wound. The continuous suture distributes its tension throughout the wound, limiting the forces on the tissues encircled by the sutures. This technique was experimentally proven to be associated with greater bursting pressures than the simple interrupted sutures or figure-of-8 mattress sutures.[50]

There are many prospective and retrospective studies that have compared various suture materials in abdominal wound closure. However, since the introduction

of synthetic absorbable sutures, the majority of these trials have shown no difference in the overall incidence of wound complications in comparison with various nonabsorbable sutures.

However, PDS seems to be associated with a lower rate of Incisional Hernia. The double-stranded suture type providing nearly twice the initial tensile strength of a single strand of the same diameter may be valuable in high-risk patients (i.e., those having obesity).[28]

Mechanical stress may play an important part in the development of Incisional Hernia. Coughing, abdominal distension, heavy physical exercise, straining during defecation, or vomiting after operation may increase the risk of IH independently of any other associated factors. In a recently published meta-analysis of the trials comparing routine to elective nasogastric decompression after elective laparotomy,[51] the authors concluded that routine decompression results in a significantly increased incidence of pulmonary complications (fever, atelectasis, and pneumonia) and does not decrease the incidence of wound complications (infection and dehiscence).

The continuous double-loop closure (CDLC), using double-stranded sutures, is reputed to withstand raised intra-abdominal pressure, while apposition of the wound edges is maintained.[52] This phenomenon was expected to result in a lowering of wound pain and dehiscence. In a recent study, Niggebrügge et al.[53] compared the CDLC to the more commonly used continuous running suture in patients undergoing midline laparotomy. The CDLC technique was associated with more wound dehiscence and rupture. Although CDLC can resist high intra-abdominal pressures, it seems to decrease the compliance of the abdominal wall, increasing the risk of postoperative pulmonary complications and death.

Incisional Hernia after Laparoscopic Surgery

The incidence of IH after laparoscopic surgery is low, averaging less than 1%.[54] The diameter of the port site seems to be the major factor. Eighty-six percent of such Incisional Hernia develop in port sites of 10 mm or more. There is a tenfold increase in the incidence of extraumbilical hernias, if a 12-mm port is used. Other factors incriminated include long duration of surgery and multiple insertions, large quantities of fluid left in the peritoneal cavity, inadequate evacuation of pneumoperitoneum and unrelaxed abdominal wall at the end of the procedure, and increased abdominal pressure at the end of surgery.[28]

The closure of all port sites of 10 mm or more is indicated, preferentially intracorporeally under direct vision, since Incisional Hernia have been reported after external closure of the aponeurosis.[28]

Optimizing Wound Closure

In 1998, Weiland et al.[55] reviewed the medical world literature of techniques of abdominal wound closure between 1977 and 1997 and found 25 comparative articles of which 23 were randomized. A total of 12,247 patients from nine countries were analyzed. Comparison of continuous and interrupted sutures failed to reach significance.The infection rate was not significantly different in all types of comparisons. The authors concluded that the choice of suture material should be based solely on the rates of IH formation. When continuous closures are chosen, nonabsorbable sutures are most appropriate. If interrupted closures are chosen, absorbable sutures should be favored. Layered closure may increase the risk of infection, hernia, and dehiscence as compared with mass closure. However, although

the authors, in their meta-analysis, used the Stouffer method based on the standard deviation Z score, with a special attention to type II errors, the drawn conclusions are difficult to generalize.

In 2000, Hodgson et al.[56] reviewed all randomized clinical trials conducted in adults and published in English between 1966 and 1998, excluding those comparing two sutures of the same category and with the same technique. Strict methodological barriers for inclusion were set, including mainly the Jadad Quality Score.[57] Incisional Hernia were 32% less frequent with nonabsorbable sutures as compared with absorbable sutures. Although the infection rates were not significantly different, nonabsorbable sutures were associated with an increased rate of cutaneous sinuses and wound pain. The running type of suturing was associated with significantly lower rates of IH. The authors recommended the use of running nonabsorbable sutures as the standard modality of wound closure.

Although some discrepancies may exist with large randomized trials, meta-analysis can be a good tool to resolve clinical controversy. However, the patient populations were heterogeneous without patent data comparing patient-related factors such as obesity, steroid medication, hypoalbuminemia, age, and pulmonary diseases, among others. Additional factors of discrepancies include type of incision, antibiotic prophylaxis, the emergency type of operation, and follow-up period.

Independently of patient-related factors, the type of incision dictated by the type of the operation and the preference of the surgeon, and the type of closure, which may be layered or mass, two major factors may be controlled to some extent: the infection rate and the type of the suture material. Reducing the infection rate seems to be the key factor in reducing IH. Rigorous aseptic technique and limitation of the use

of electric cautery are recommended. The most suitable suture material seems to be PDS which cumulates the short-term benefits of nonabsorbable sutures (tensile force) without accumulating their long-term inconveniences such as sinus formation and pain. The type of closure (interrupted vs. continuous) seems to be secondary, if some rules such as avoiding ischemic suturing (the figure-of-8 type) and reducing early malunion (wide sutures) are implemented. There is no argument for recommending the preventive use of an absorbable (polyglactin) mesh placed on top of a facial closure in high-risk patients.[58]

Pathogenesis

The pathogenesis of hernias is multifactorial. Different etiological factors, such as increased intra-abdominal pressure (in pregnancy, intra-abdominal tumors, chronic obstructive lung disease, ascites, chronic intestinal obstruction, and adiposity), or pathological changes in connective tissue of the abdominal wall, are considered.

Presentation

Symptoms, if present, are disfigurement, discomfort or pain in most of the patients. A few patients may present with features of intestinal obstruction, strangulation or very rarely spontaneous rupture of the hernia contents.

Diagnosis

Clinical examination is done in the standing and supine position and the patient is asked to strain to make the hernia prominent. Edges of the defect are felt and its size is noted. The reducibility is assessed.

History, local examination with inspection and palpation of the hernial opening and auscultation are employed for hernia diagnosis. In case of uncertain clinical findings, sonography is the best means for confirming the hernial opening and content. Radiographic diagnosis of hernias is rarely required.

Management

"Hernia repair remains a challenge to surgeons"-Bilroth stated 150 years ago. Tremendous advances have taken place in the field of hernia surgery recently with the introduction of prosthetic meshes and minimal invasive techniques. Millions of patients are affected each year, presenting most commonly with primary ventral and incisional hernias. When symptomatic these patients present with pain but asymptomatic cases are aesthetically distressing. These concerns, coupled with the risk of complications such as incarceration and strangulation, are the most common reasons patients report to surgeon for repair of hernias. Based on Pascal's principle of hydrostatic forces and the law of Laplace, hernia will continue to enlarge if not repaired. Increased intra-abdominal pressure will exert its greatest force on the thinnest portion of the wall. As the hernia enlarges, the wall thins out at that point, and the diameter increases. This positive feedback loop virtually assures continued progression. Incisional hernias generally do not develop in the immediate postoperative period. Follow up for 3 to 5 years post laparotomy is necessary for the development of incisional hernias.[60]

Mesh repair is an excellent method of repair preferred for patients with large defects of the anterior abdominal wall, especially preferred more than 4 cm, size defect. An excellent method, which has been used, called Rive’s Stoppa technique, where mesh was placed between peritoneum and abdominal wall or rectus muscle and

posterior rectus sheath. The main advantage of pre peritoneal mesh repair are - Less chance of mesh infection and erosion through skin because the graft lies in preperitoneal plane between posterior rectus sheath and peritoneum, avoids adhesions, bowel obstruction, enterocutaneous fistula and erosion of mesh, minimal morbidity and duration of hospital stay is less compared to other techniques. The main disadvantage is more time consuming, extensive preparation of preperitoneal plane and surgical experience.[61]

Complications

Complications of hernia include irreducibility is frequent and partial obstruction, Strangulation, Spontaneous ulceration, rupture. Considering the significant recurrence rate noted after various techniques for incisional hernia repair, the task of repairing this defect can challenge the scientific and artistic talents of the most experienced surgeon. Various types of repair have been described, both anatomical and prosthetic. But the results have been disappointing with a high incidence of recurrence-about upto 50% after an anatomical repair and upto 10% following prosthetic mesh repairs.[62] In general the postoperative complications of incisional hernia include pulmonary atelactasis, bronchitis, pulmonary embolism. postoperative ileus, thrombophlebitis and deep venous thrombosis, where as local complications like wound seroma, haematoma, infection, sinuses and complications of mesh.[61]

Surgical site infection

Surgical Site Infections continues to be a major source of morbidity following operative procedures. The NNIS report for 1986-1996 described an SSI rate of 2.6%

for all operations at the reporting hospitals. It seems likely that overall SSI rates are likely to be greater than reported. All surgical wounds are contaminated by bacteria, but only a minority actually demonstrate clinical infection. The SSI are the biological summation of several factors: the inoculum of bacteria introduced into the wound during the procedure, the unique virulence of contaminants, the microenvironment of each wound, and the integrity of the patients host defense mechanisms. Although an SSI rate of zero may not be achievable, continued progress in understanding the biology of infection at the surgical site and consistent applications of proven methods of prevention will allow to further reduce the frequency, cost, and morbidity associated with SSI.[63]

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

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

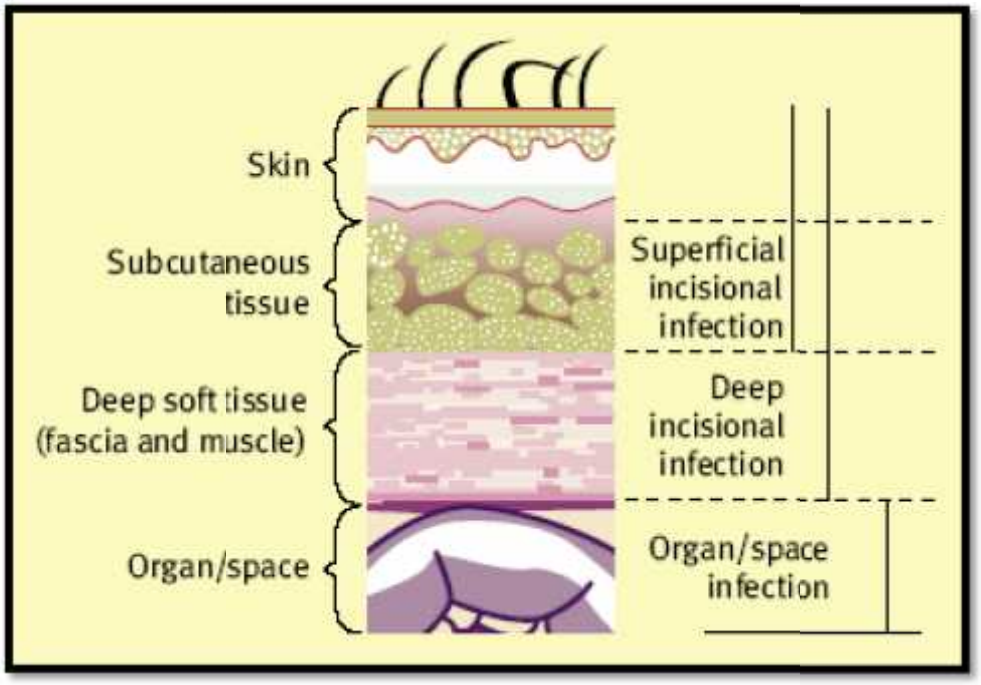


Figure 5. Schematic representation of the anatomical classification of surgical site infections[64]

C. Classification of surgical site infection[64]

I. Superficial Incisional SSI

Infection involves only skin and subcutaneous tissues of incision.

- Occurs within 30 days after the operation;
- Involves only the skin or subcutaneous tissue; and
- At least 1 of the following:
    - Purulent drainage (culture documentation not required)
    - Organisms isolated from fluid/tissue of superficial incision

- At least 1 sign of inflammation (eg, pain or tenderness, induration, erythema, local warmth of the wound)
- Wound is deliberately opened by the surgeon
- Surgeon or attending physician declares the wound infected.

II, Deep Incisional SSI

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

- Occurs within 30 days of operation or within 1 year if an implant is present;
- Involves deep soft tissues (fascia and/or muscle) of the incision; and
- At least 1 of the following:
  - Purulent drainage from the deep incision but without organ/space involvement
  - Fascial dehiscence or fascia is deliberately separated by the surgeon due to signs of inflammation
  - Deep abscess is identified by direct examination or during reoperation, by histopathology, or by radiologic examination
  - Surgeon or attending physician declares that deep incisional infection is present.

III. Organ/Space SSI

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

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

D. Epidemiology

Frequency

International

Internationally, the frequency of SSI is difficult to monitor because criteria for diagnosis might not be standardized. A survey sponsored by the World Health Organization demonstrated a prevalence of nosocomial infections varying from 3-21%, with wound infections accounting for 5-34% of the total.[65] The 2002 survey

report by the Nosocomial Infection National Surveillance Service (NINSS), which covers the period between October 1997 and September 2001, indicates that the incidence of hospital acquired infection related to surgical wounds in the United Kingdom is as high as 10% and costs the National Health Service in the United Kingdom approximately 1 billion pounds (1.8 billion dollars) annually.

United States

Surgical site infections (SSIs) are not an extinct entity; they account for 14-16% of the estimated two million nosocomial infections affecting hospitalized patients in the United States.[66]

Indian scenario

The surgical site infection rate reports by different workers have differed considerably. A study[67] done in India reported overall infection rate as 8.95% and number of studies carried out in India indicate an overall infection rate of 4.04 to 30% for clean surgeries and 10.06 to 45% for clean-contaminated surgeries.[68-70]

Collated data on the incidence of wound infections probably underestimate true incidence because most wound infections occur when the patient is discharged, and these infections may be treated in the community without hospital notification.[65]

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

Mortality/Morbidity

Surgical site infections are associated not only with increased morbidity but also with mortality. Seventy-seven percent of the deaths of surgical patients were

related to surgical wound infection.[71] Kirkland et al calculated a relative risk of death of 2.2 attributable to SSIs, compared to matched surgical patients without infection.[72]

Pathophysiology of wound healing[73]

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

The inflammatory phase commences as soon as tissue integrity is disrupted by injury; this begins the coagulation cascade to limit bleeding. Platelets are the first of the cellular components that aggregate to the wound, and, as a result of their degranulation (platelet reaction), they release several cytokines (or paracrine growth factors). These cytokines include platelet derived growth factor (PDGF), insulinlike growth factor-1 (IGF-1), epidermal growth factor (EGF), and fibroblast growth factor (FGF). Serotonin is also released, which, together with histamine (released by mast cells), induces a reversible opening of the junctions between the endothelial cells, allowing the passage of neutrophils and monocytes (which become macrophages) to the site of injury.

This large cellular movement to the injury site is induced by cytokines secreted by the platelets (chemotaxis) and by further chemotactic cytokines secreted by the macrophages themselves once at the site of injury. These include transforming growth factor alpha (TGF-alpha) and transforming growth factor beta (TGF-beta). Consequently, an inflammatory exudate that contains red blood cells, neutrophils, macrophages, and plasma proteins, including coagulation cascade proteins and fibrin

strands, fills the wound in a matter of hours. Macrophages not only scavenge but they also are central to the wound healing process because of their cytokine secretion.

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

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

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

Causes

All surgical wounds are contaminated by microbes, but in most cases, infection does not develop because innate host defenses are quite efficient in the elimination of contaminants. A complex interplay between host, microbial, and surgical factors ultimately determines the prevention or establishment of a wound infection.[74]

Factors that affect surgical wound healing

Microbiology

Microbial factors that influence the establishment of a wound infection are the bacterial inoculum, virulence, and the effect of the microenvironment. When these microbial factors are conducive, impaired host defenses set the stage for enacting the chain of events that produce wound infection.[74]

Most SSIs are contaminated by the patient's own endogenous flora, which are present on the skin, mucous membranes, or hollow viscera. The traditional microbial concentration quoted as being highly associated with SSIs is that of bacterial counts higher than 10,000 organisms per gram of tissue (or in the case of burned sites, organisms per cm^2 of wound).[74]

Organisms associated with SSIs vary with type of procedure and anatomic location of the operation. The usual pathogens on skin and mucosal surfaces are gram-positive cocci (notably staphylococci, streptococci) account for most exogenous flora involved in SSIs. The most common group of bacteria responsible for SSIs are *Staphylococcus aureus*. The emergence of resistant strains has considerably increased the burden of morbidity and mortality associated with wound infections. However,

gram-negative aerobes and anaerobic bacteria contaminate skin in the groin/perineal areas. The contaminating pathogens in gastrointestinal surgery are the multitude of intrinsic bowel flora, which include gram-negative bacilli (eg, *Escherichia coli*) and gram-positive microbes, including enterococci and anaerobic organisms. Sources of such pathogens include surgical/hospital personnel and intraoperative circumstances, including surgical instruments, articles brought into the operative field, and the operating room air.[74]

Methicillin resistant *Staphylococcus aureus* (MRSA) is proving to be the scourge of modern day surgery. Like other strains of *S aureus,* MRSA can colonize the skin and body of an individual without causing sickness, and, in this way, it can be passed on to other individuals unknowingly. Problems arise in the treatment of overt infections with MRSA because antibiotic choice is very limited. MRSA infections appear to be increasing in frequency and are displaying resistance to a wider range of antibiotics.[74]

Of particular concern are the vancomycin intermediate *Staphylococcus aureus* (VISA) strains of MRSA. These strains are beginning to develop resistance to vancomycin, which is currently the most effective antibiotic against MRSA.[74]

Pathogens Commonly Associated with Wound Infections and Frequency of Occurrence[75]

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

In clean surgical procedures, in which the gastrointestinal, gynaecologic, and respiratory tracts have not been entered, *Staphylococcus aureus* from the exogenous environment or the patient's skin flora is the usual cause of infection. In other categories of surgical procedures, including clean-contaminated, contaminated, and dirty, the polymicrobial aerobic and anaerobic flora closely resembling the normal

endogenous microflora of the surgically resected organ are the most frequently isolated pathogens.[76]

Others risk factors

Patient Factors[77]

Obesity increases risk substantially when the subcutaneous abdominal fat layer exceeds 3 cm (1.5 inches) (Nyström et al 1987). The risk is increased by the need for a larger incision, decreased circulation to the fat tissue or the technical difficulty of operating through a large fat layer.

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

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

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

Age, race, socioeconomic status and chronic diseases, such as diabetes and malignancy, are difficult to assess because they are frequently associated with other factors that independently contribute to risk. For example, age over 70 may be accompanied by decreased defense mechanisms, poor nutrition and anemia.[73]

Wound characteristics include nonviable tissue in wound; hematoma; foreign material, including drains and sutures; dead space; poor skin preparation, including shaving; and pre-existent sepsis (local or distant).[73]

Operative characteristics include poor surgical technique; lengthy operation (>2 h); intraoperative contamination, including infected theater staff and instruments and inadequate theater ventilation; prolonged preoperative stay in the hospital; and hypothermia.[73]

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

BODY MASS INDEX AND SURGICAL SITE INFECTION IN INCISIONAL HERNIA

Morbidly obese patients (MOPs) are predisposed to developing abdominal wall hernias with the potential complication of small bowel obstruction and other morbidity. Hernia prophylaxis has been attempted as a means of decreasing the incisional hernia risk associated with weight loss surgery. The controversy regarding the optimal time and method of repair of abdominal wall hernias in patients undergoing open or laparoscopic gastric bypass is discussed with emphasis placed on either a simultaneous repair or split of the omentum, and of leaving a plug in the hernia defect, to allow time to perform a delayed repair.[78]

In general, it is agreed that obese patients have tissue hypoperfusion (subcutaneous adipose tissue), which may predispose to SSI through a greater risk of

ischemia/ necrosis and suboptimal neutrophil oxidative killing. Proposed pathogenetic mechanisms include a high ratio of tissue mass:capillaries in adipose tissues, larger wound surface areas (hence a larger area to become infected, greater oxygen demand, and a larger dead space with a closed incision and a larger wound fluid volume),and decreased oxygen tension in adipose tissues. Many of these factors may be categorized together as a poor balance between tissue oxygen demand and oxygen supply.

MOPs have a greater chance to develop abdominal wall hernias and their potential complication such as small bowel obstruction. New modern hernia repair approach has increased the opportunities to treat patients in a one-stage procedure in order to reduce the risk of incisional hernias. This has become possible because progress in the surgical approach to hernias with application of minimally invasive surgical techniques. Also, a better understanding of how to apply this to reconstructing abdominal wall defects and the new synthetic meshes with a better understanding of the mechanical properties necessary to secure hernia repair, with newer biomaterials that provide for tissue ingrowth and may be more resistant to infection than traditional meshes, have given a new vista to the treatment of the hernia. Morbidly obese patients are predisposed to developing abdominal wall hernias and these are a common cause of morbidity and mortality.[78]

Overweight is an important issue for incisional hernias and for these reasons many patients have already undergone a hernia repair surgery before the bariatric surgery. Incarceration of such hernias can lead to small bowel obstruction (SBO). In addition, the resultant derangement of the GI anatomy after gastric bypass increases the incidence, and level of difficulty in making a diagnosis of partial SBO. There is no

clear consensus among bariatric surgeons on the optimal time and method of repair of abdominal wall hernias in patients undergoing laparoscopic Roux-en-Y gastric bypass (LRYGBP). The management of primary and incisional abdominal wall hernias continues to evolve from the early days of primary hernia repair. Lastly, there has been a return to primary tissue repairs using components separation technique, augmented with mesh as necessary. This combination of education and new materials such as lightweight meshes has provided the surgeon with a basis of performing a better hernia repair.[78]

Better appreciation of patient characteristics may help to support which type of procedure or mesh use will likely succeed. Risk factors for the development of abdominal wall incisional hernias may include the following: overweight, smoking, age greater than 60 years, wound infection, re-laparotomy, chronic medical conditions (such as cirrhosis or cardiopulmonary disease), and chronic steroid use. The relative ratio and amount of type I collagen may determine patients at risk.[78]

According to some studies, morbid obesity, defined as a body mass index (BMI) exceeding 40, is a major risk factor in the development of incisional hernia, with 20% to 28% of obese patients who undergo abdominal surgery developing an incisional hernia within 12 to 28 months of the initial procedure.[79,80] Increased abdominal pressure, as seen in obesity, particularly in those individuals with large amounts of central adiposity, is associated with a predisposition to umbilical and incisional hernias. In our series, abdominal wall pathology is found in 11% of the patients who underwent to surgery. Also, our series confirms the high recurrence rate among these patients, which amounts to 25%. In fact, Sugerman et al. have stressed that obesity may be a greater risk factor for incisional hernia occurrence than chronic steroid use.[81]

The Southampton system is much simpler than the ASEPSIS system, with wounds being categorized according to any complications and their extent.[81] Both systems, however, have been developed for use following specific types of surgery and this may limit their usefulness.[78]

Southampton scale - by using the worst wound score recorded and information about any treatment instituted either in hospital or the community, wounds were regarded in four categories:[82]

Southampton scoring system

Grade	Appearance
0	Normal healing
I Normal healing with mild bruising or erythema:	
A	Some bruising
B	Considerable bruising
C	Mild erythema
II Erythema plus other signs of inflammation	
A	At one point
B	Around sutures
C	Along wound
D	Around wound
III Clear or haemoserous discharge:	
A	At one point only (<2cm)
B	Along wound (>2cm)
C	Large volume
D	Prolonged (>3 days)
IV Pus:	
A	At one point only (<2cm)
B	Along wound (>2cm)
V Deep or severe wound infection with or without tissue breakdown; haematoma requiring aspiration	

a. normal healing;

b. minor complication;

c. wound infection - wounds graded IV or V, or wounds treated with antibiotics after discharge from hospital, irrespective of the wound grading given to them by the nurse;

d. major haematoma-wound or scrotal haematomas requiring aspiration or evacuation.

METHODOLOGY

The present study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012.

Study design

The study design was a cross-sectional study.

Study period and duration

This one year study was conducted during the period from January 2012 to December 2012.

Place

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

Source of Data

All patients diagnosed with incisional hernia during the study period were studied.

Sample size and sampling procedure

All the patients diagnosed with incisional hernia during the study period fulfilling the selection criteria were enrolled in the study.

Selection criteria

Inclusion

- Patients diagnosed with incisional hernia undergoing elective surgery.
- Patients aged between 15 to 60 years.

Exclusion

- Patients undergoing emergency procedures.
- Patients with diabetes mellitus.
- Patients on steroids.
- Patients aged less than 15 years and more than 60 years.
- Patients with anaemia
- Patients with surgeries in which bowel was opened
- HIV positive patients
- Patients with deep and organ space surgical site infection.

Ethical clearance

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

Informed Consent

All the patients fulfilling selection criteria were explained about the purpose and nature of the study and patients willing to participate in the study that is, who consented were enrolled in this study (Annexure I).

Method of collection of data

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

Investigations

Routine investigations such as complete blood count, liver function test, serum creatnine, blood urea nitrogen, serum electrolytes were done and those who were eligible for the study were enrolled.

Body mass index

A thorough clinical examination was conducted. Height and weight was recorded and body mass index was calculated based on formula;

$$\text{Body Mass Index} = \frac{\text{Weight (Kg)}}{\text{Height}^2 \text{ (m)}}$$

Body mass index was classified according to Overweight and obesity by BMI in adult as below.[83]

	BMI (Kg/m^2)	**Risk of co-morbidities**
Underweight	< 18.5	Low (But increased risk of other clinical problems
Normal range	18.5 to 24.9	Normal
Overweight	25.0 to 29.9	Overweight
Obese	30.0 or above	Obese

Procedure

Pre operative

All patients satisfying inclusion criteria were selected for study and prepared for surgery after taking physical fitness. Skin preparation was done immediately before surgery as per cdc guidelines.

Intra operative

All patients underwent pre-peritoneal repair and prolene mesh was placed in preperitoneal space. Drain was kept in subcutaneous plane which was connected to vacuum suction.

Post operative

All patients received antibiotics postoperatively and antibiotics were standardized. Patients were examined daily in postoperative period till the date of discharge. Surgical wound was examined on postoperative day three, five, seven , fifteen and thirty. Patients with surgical site infection were managed with wound care and antibiotics as per the sensitivity of the organism.

Study variables

Assessment of wound

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

The assessment of wound was done on day three, five, seven, fifteen and thirty post operatively based on Southampton wound scoring system.[82]

Culture and sensitivity

In surgical wounds of Southampton wound score of grade iv and v , sample of discharge from surgical site was taken and sent for culture and antibiotic sensitivity.

Statistical analysis

The data obtained was tabulated, categorical data was used to calculate percentage of surgical site infection as per BMI classification.

Percentage was first determine the following values:

- The number of occurrences of a surgical site infection in given BMI group.
- The size of the population of each BMI group.

Percentage was then calculated by dividing the number of occurrences of surgical site infection in given BMI group by the size of the population of each BMI group.

Chi-square with yate's correction(1.38) was done to calculate p value.

RESULTS

The present one year cross-sectional study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012.

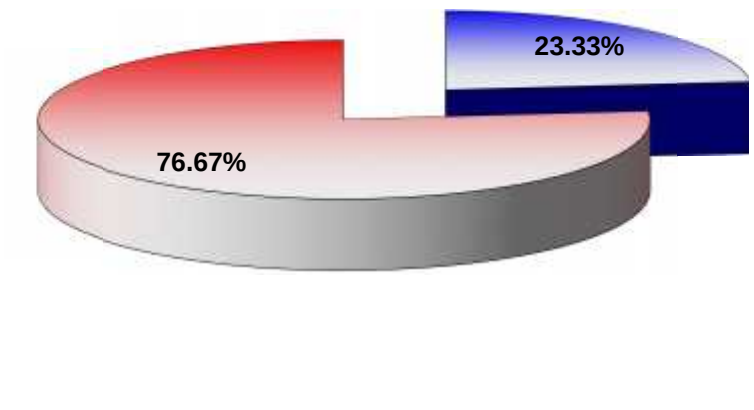
A total of 60 patients diagnosed with incisional hernia during the study period fulfilling the selection criteria were studied.

The data obtained was coded and tabulated on excel spreadsheet. The data was analysed and the final observations and results were tabulated as below.

Table 1. Sex distribution

Sex	Distribution (n=60)	
	Number of patients	Percent
Male	14	23.33
Female	46	76.67
Total	**60**	**100.00**

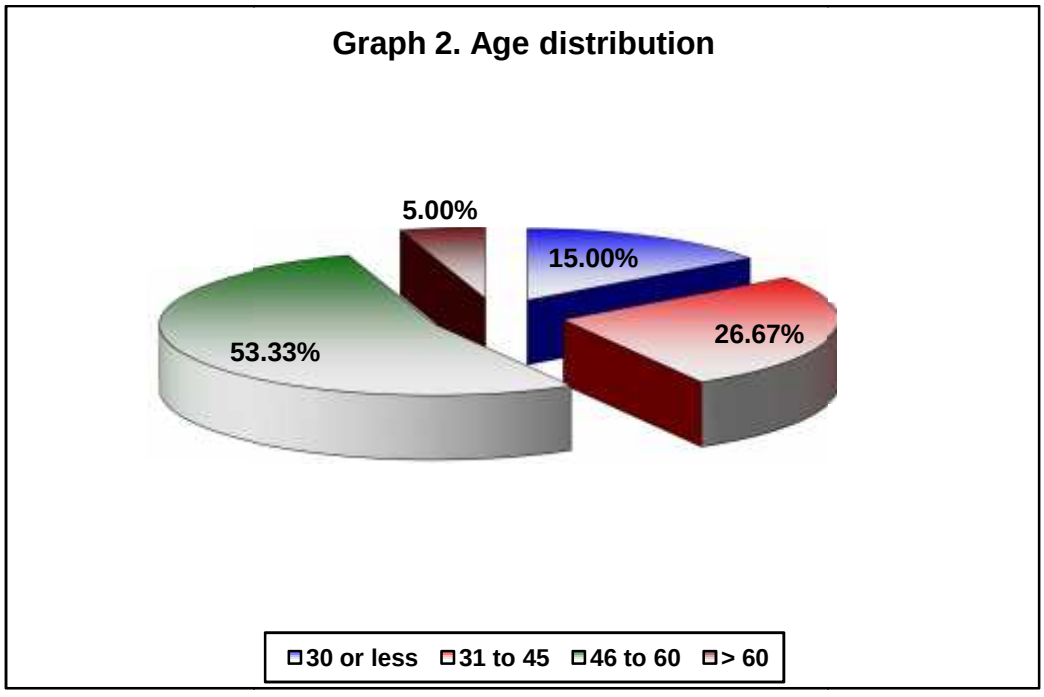
Graph 1. Sex distribution



In the present study majority of the patients were females that is 76.67% and 23.33% were males. The female to male ratio was 3.28:1.

Table 2. Age distribution

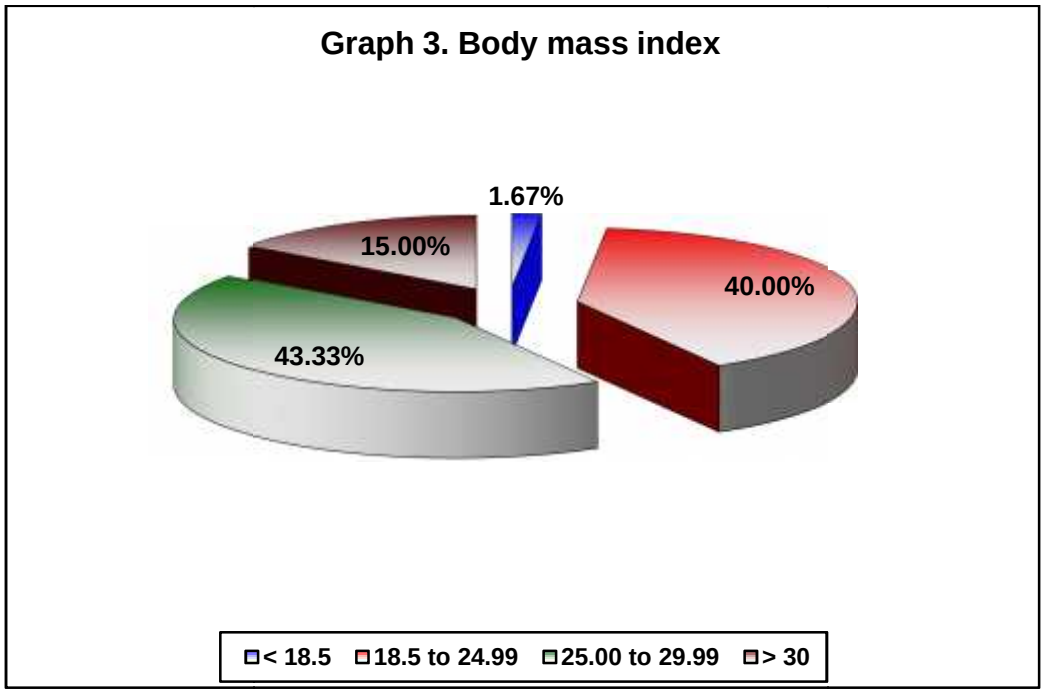
Age group (Years)	Distribution (n=60)	
	Number of patients	Percent
30 or less	9	15.00
31 to 45	16	26.67
46 to 60	32	53.33
> 60	3	5.00
Total	**60**	**100.00**



In this study the commonest age group was 46 to 60 years comprised of 53.33% of the patients followed by 31 to 45 years age group.

Table 3. Body mass index

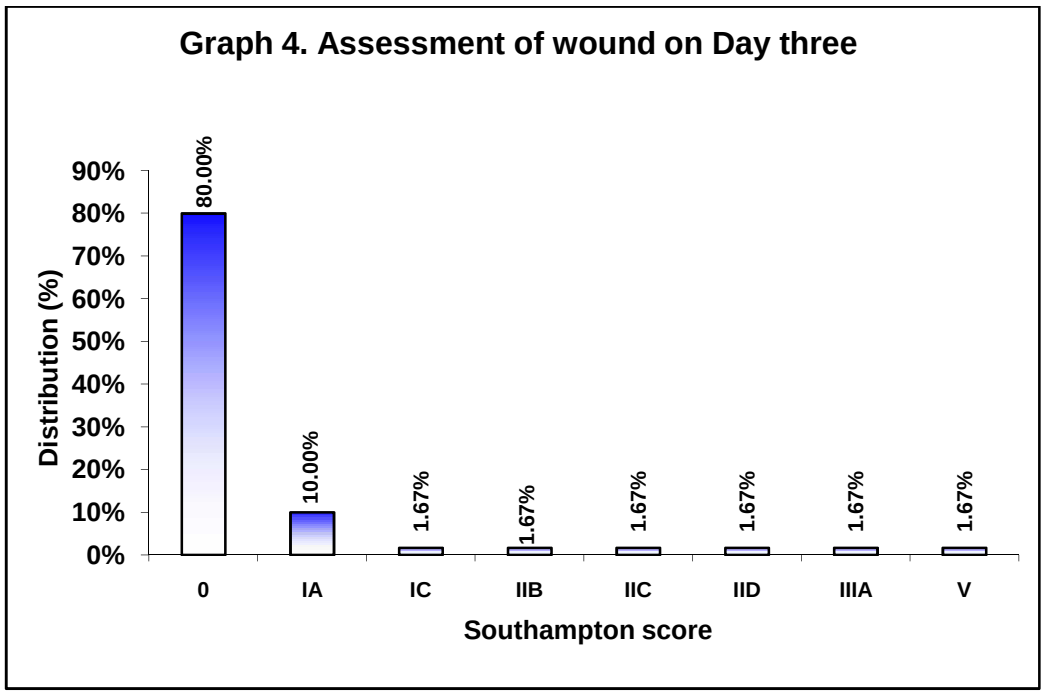
BMI (Kg/m^2)	Distribution (n=60)	
	Number of patients	Percent
< 18.5	1	1.67
18.5 to 24.99	24	40.00
25.00 to 29.99	26	43.33
> 30	9	15.00
Total	**60**	**100.00**



In the present study most of the patients (43.33%) had BMI between 25 to 29.99 Kg/m2.

Table 4. Assessment of wound on Day three

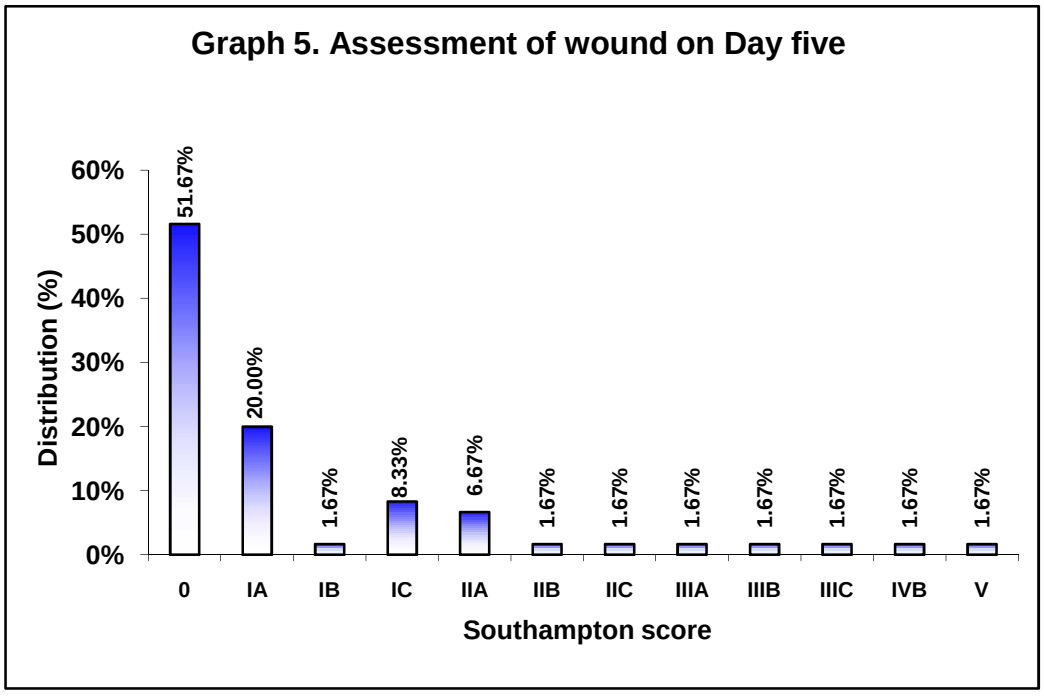
Score	Distribution (n=60)	
	Number of patients	Percent
0	48	80.00
IA	6	10.00
IC	1	1.67
IIB	1	1.67
IIC	1	1.67
IID	1	1.67
IIIA	1	1.67
V	1	1.67
Total	**60**	**100.00**



In the present study on day three 10% of the patients had Grade IA wound score while 1.67% of patients each had Grade IC, IIB, IIC, IID, IIIA and V.

Table 5. Assessment of wound on Day five

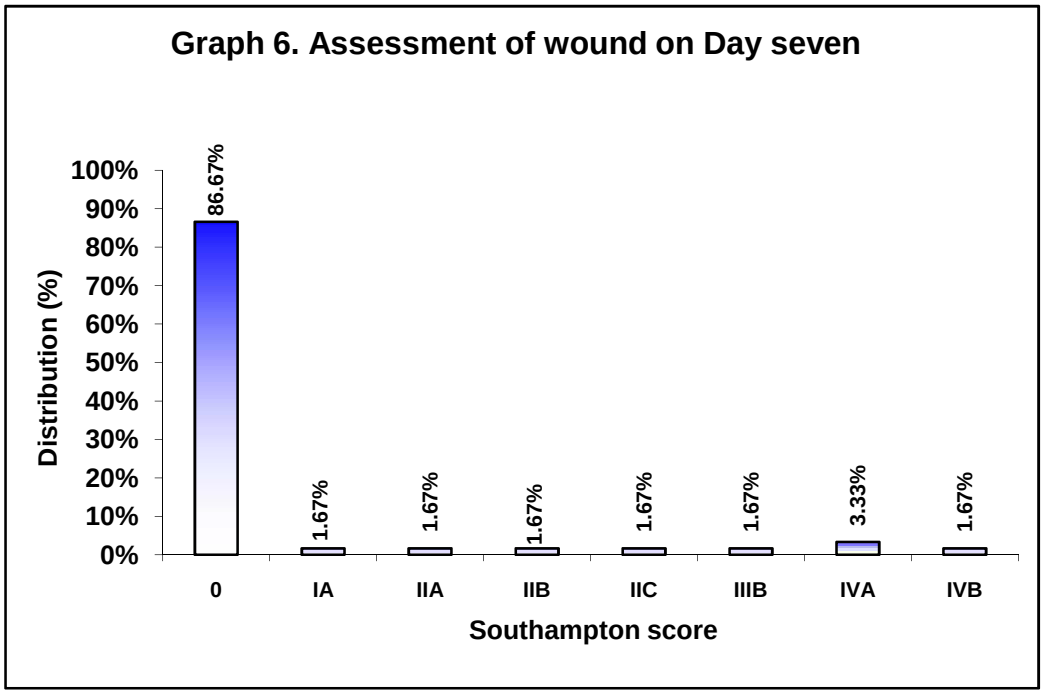
Scores	Distribution (n=60)	
	Number of patients	Percent
0	31	51.67
IA	12	20.00
IB	1	1.67
IC	5	8.33
IIA	4	6.67
IIB	1	1.67
IIC	1	1.67
IIIA	1	1.67
IIIB	1	1.67
IIIC	1	1.67
IVB	1	1.67
V	1	1.67
Total	**60**	**100.00**



In this study on day five 20% of the patients had Grade IA wound score while 8.33% and 6.67% of the patients had Grade IC and IIA respectively.

Table 6. Assessment of wound on Day seven

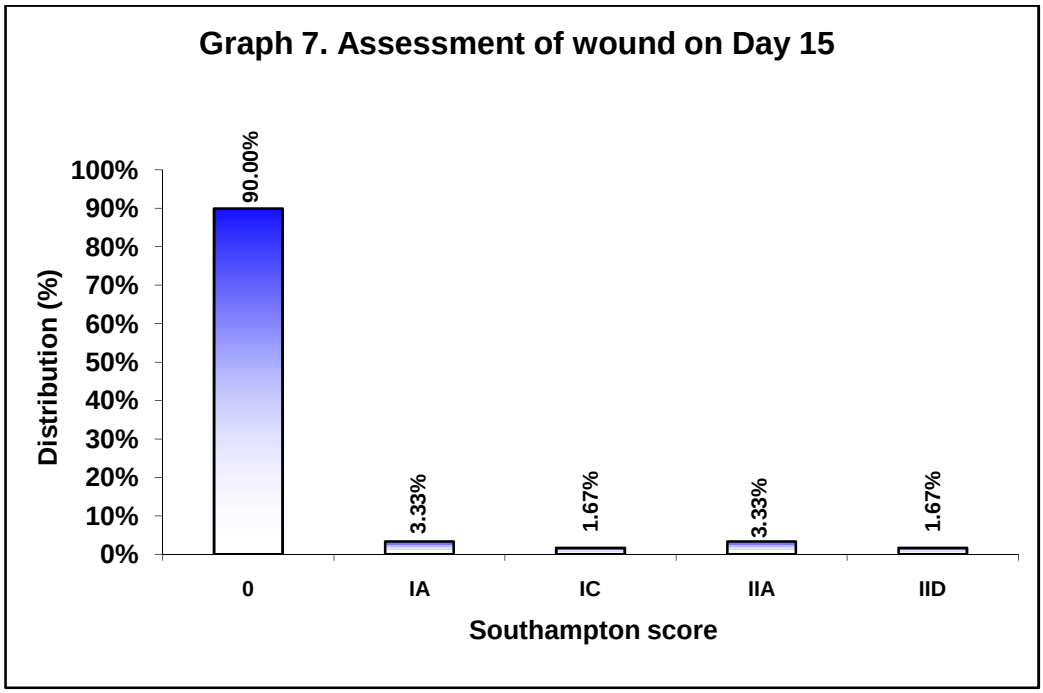
Score	Distribution (n=60)	
	Number of patients	Percent
0	52	86.67
IA	1	1.67
IIA	1	1.67
IIB	1	1.67
IIC	1	1.67
IIIB	1	1.67
IVA	2	3.33
IVB	1	1.67
Total	**60**	**100.00**



In the present study on day seven 3.33% of the patients had Grade IVA wound score while 1.67% of the patients had Grade IA, IIA, IIB, IIC, IIIB, IVA and IVB.

Table 7. Assessment of wound on day 15

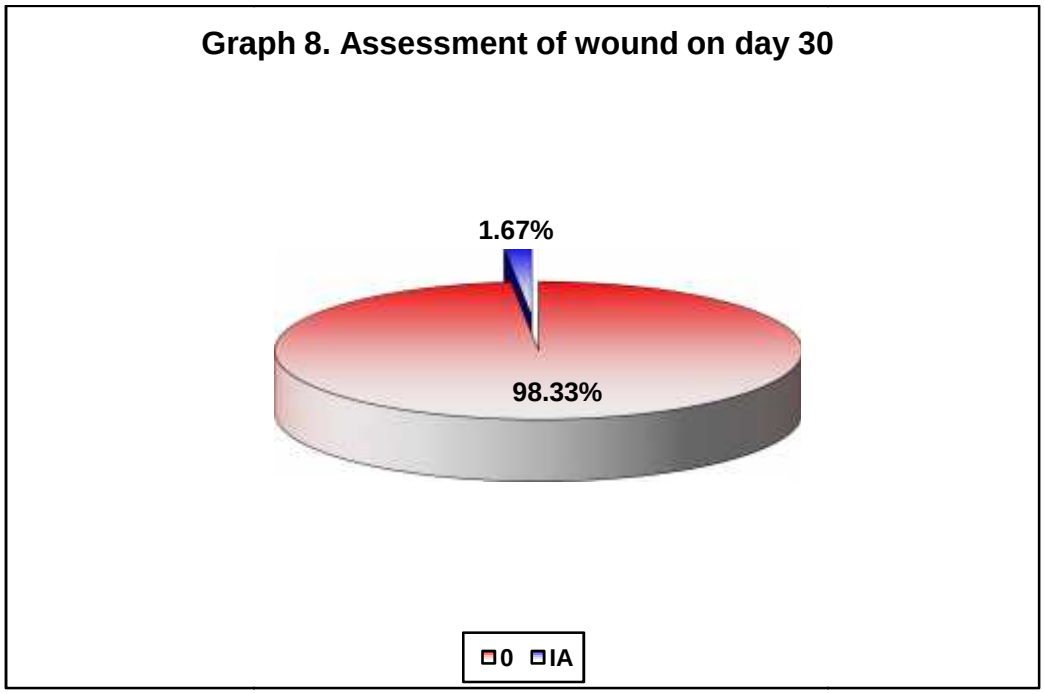
Score	Distribution (n=60)	
	Number of patients	Percent
0	54	90.00
IA	2	3.33
IC	1	1.67
IIA	2	3.33
IID	1	1.67
Total	**60**	**100.00**



In this study on day fifteen 3.33% of the patients each had wound score IA and IIA SSIs while 1.67% of patients each had Grade IC and IID.

Table 8. Assessment of wound on day 30

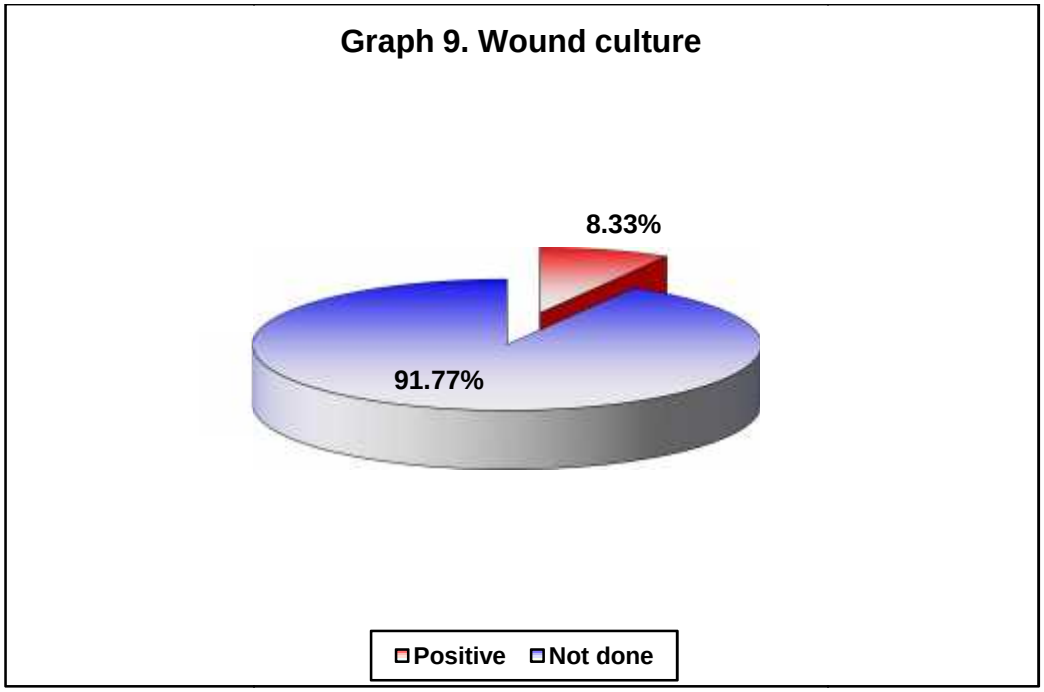
Score	Distribution (n=60)	
	Number of patients	Percent
0	59	98.33
IA	1	1.67
Total	**60**	**100.00**



In this study on day thirty 1.67% of the patients had Grade IA wound score.

Table 9. Wound culture

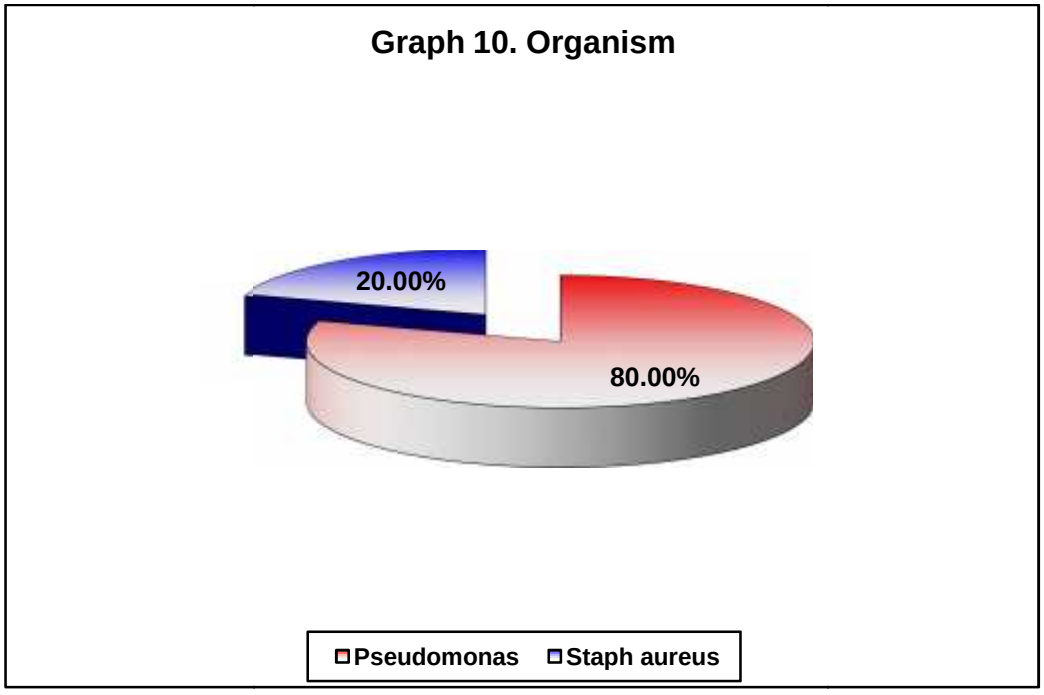
Culture	Distribution (n=60)	
	Number of patients	**Percent**
Positive	5	8.33
Not done	55	91.77
Total	**5**	**100.00**



In this study the wound culture was positive in five patients (8.33%).

Table 10. Organism

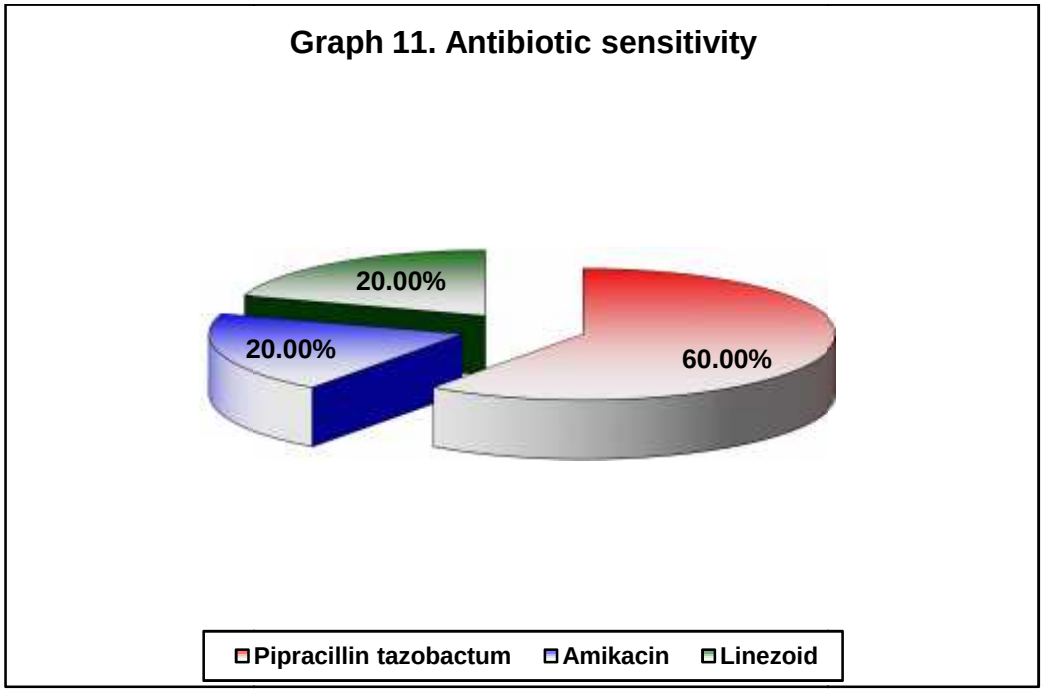
Organism	Distribution (n=5)	
	Number of patients	Percent
Pseudomonas	4	80.00
Staph aureus	1	20.00
Total	**5**	**100.00**



In the present study of the five patients with positive wound culture, pseudomonas were isolated in 80% of the patients while staph aureus was present in 20%.

Table 11. Antibiotic sensitivity

Antibiotics	Distribution (n=60)	
	Number of patients	Percent
Pipracillin tazobactum	3	60.00
Amikacin	1	20.00
Linezoid	1	20.00
Total	**5**	**100.00**



In this study the 60% of the patients had organisms sensitive to antibiotics viz piperacillin tazobactum and 20% each were sensitive to Amikacin and Linezoid.

Table 12. Association of Body mass index with surgical site infections

Body mass index (Kg/m2)	Total	Surgical site infection			
		Absent		Present	
		No	%	No	%
< 18.50	1	1	100.00	0	0.00
18.50 to 24.99	24	20	83.33	4	16.67
25.00 to 29.99	26	26	100.00	0	0.00
30.00 or more	9	8	88.89	1	11.11
Total	**60**	**55**	**91.67**	**5**	**8.33**

In this study of the 26 patients with BMI 25 to 29.99 none of the patient (0%) developed surgical site infection. In 24 and 9 patients with BMI 18.5 to 24.99 and ≥ 30 16.67% and 11.11% of the patients had SSIs while one patient who had BMI < 18.5 Kg/m^2 did not develop SSI.

The association between superficial surgical site infection and BMI was not statistically significant calculated by using chi-square test with Yate's correction (=1.38%) with (p= 0.241).

DISCUSSION

Obesity is one of the common medical conditions in the world. The burden of obesity is currently one of the main concerns in health care worldwide; more than a million of deaths per year in the world are attributed to obesity-related co-morbidities, including diabetes mellitus, hypertension, and coronary artery disease. Millions are spent each year on direct treatment of obesity and obesity-related complications.[84,85]

Nosocomial infections are common and have a substantial impact on mortality and healthcare costs. Surgical site infection (SSI) alone accounts for 38% of all nosocomial infections of surgical patients. Such infection is associated with other wound complications (e.g., dehiscence, hernia), a two- to three-fold higher risk of death, and a 60% higher risk of requiring an intensive care unit (ICU) stay. Length of stay (LOS) is increased by 7–12 days, the patient is five times more likely to require readmission, and direct healthcare costs are increased by a nosocomial infection.[86,87]

Incisional hernia remains a frequent complication of abdominal surgery, with a reported incidence of 2% to 20%.[88-92] In the United States, 4 to 5 million laparotomies are performed annually,[93] which means that at least 400,000 to 500,000 incisional hernias can be expected to develop each year. Incisional hernia repair is performed approximately 200,000times per year.[94,95] These data imply that in in general, 4% of patients undergoing a laparotomy will go through additional surgery to repair an incisional hernia. When morbidity is added to the vast numbers and the tremendous costs associated with incisional hernia repair,[96] it becomes clear that the efficacy of incisional hernia repair is of major importance.

The present one year hospital based longitudinal study was done at the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012. A total of 60 patients admitted with clinical diagnosis of incisional hernia were studied.

Our rate of superficial SSI for elective incisional hernia repair(8.3%) is lower than predicted by general review of the literature. Although there is a wide range of frequencies reported from 3% to 30%.[97-104]

Our rate is also greater than that suggested by the 2001 NNIS report,26 which demonstrated median rates of 3.57% for cases with a 0 risk factors to 12.88% for cases with 3 risk factors.

There are a number of potential explanations for these discrepancies. First, our population of patients may be different than those generally reported in the surgical literature.

Finally, 26 (43.33%) of 60 of our patients had a BMI $\geq$ 25, and 9(15%) of 60 had a BMI $\geq$ 30, indicating a fair rate of obesity in this population, which may not be representative of populations such as that of Tang et al,[104] a study from the Far East.

We do, however, believe that this high SSI rate is not entirely explained by these potential population differences. Our data suggest that methodology of complication measurement may also be an important contributing factor.

Our experience also subjectively supports the importance of direct involvement of the primary care team in the diagnosis and calculation of complications such as SSI.

We made the diagnosis of superficial SSI on the basis of Southampton wound scoring system score of grade iv and v. We feel that these potential patient population differences and differences in our SSI detection methodology may explain the discrepancies between our reported and expected rates of incisional SSI.

The objective of this study was to identify association between BMI and superficial surgical site infection and increasing BMI being a risk factors that independently predict development of incisional SSI.

The association between obesity and postoperative infections has been the focus of recent Studies. Choban et al. did a retrospective review of the effect of obesity on nosocomial Infections in 849 surgical patients having 6.8 % of patients developing surgical site infection, Despite a relatively small number of patients with nosocomial infections, obese patients had a significantly higher rate of SSI.[105] in our study 8.3% of patients developed superficial surgical sit infection which had similar prevalance as compared to above mentioned study.

Garibaldi et al.[106] and, more recently Canturk et al.[107] showed a trend toward a higher risk of pneumonia and nosocomial infections in the obese population.In our study none of the patients developed pneumonia and nosocomial infections except superficial surgical site infection.

A more recent larger study by Pessaux et al.,[108] in which they evaluated the risk factors for nosocomial infections in patients undergoing operations, found that obesity was an independent predictor of postoperative infection by multivariate analysis.In our study 4(80%) of patients who developed superficial surgical site infection belonged to BMI between 18.5-24.9 which was normal BMI range and

1(20%) belonged to obese group and we could not conclude obesity as a risk factor for surgical sit infection.

Although one can speculate about the reason for a higher risk of SSI in obese patients, there are few studies offering plausible explanations.

From the existing data, it is clear that there are at least four strategies that should be considered in order to decrease the risk of SSI when operating on obese patients. First, tight perioperative glucose control is key to minimizing episodes of hyperglycemia that are associated with a higher rate of SSI. Second, optimizing tissue oxygen tension through increased perioperative FIO2 and appropriate resuscitation improves the perfusion of tissuesand oxygen radical-mediated defense mechanisms against infection. Third, larger doses of prophylactic antibiotics maximize serum and tissue concentrations, providing a real (and expected) decrease in SSI. Fourth, performing laparoscopic operations whenever feasible certainly decreases the area at risk and has a demonstrated ability to reduce SSI.[109]

Obesity also has been associated with longer operations, which is one of the few independent predictors of SSI that is commonly significant in multiple series as well as in the NNIS data.[110] In our study surgical procedures were performed by senior surgeons having no significant differences in duration of operation.

In general, it is agreed that obese patients have tissue hypoperfusion (subcutaneous adipose tissue), which may predispose to SSI through a greater risk of ischemia/ necrosis and suboptimal neutrophil oxidative killing. Proposed pathogenic mechanisms include a high ratio of tissue mass: capillaries in adipose tissues, larger wound surface areas (hence a larger area to become infected, greater oxygen demand,

and a larger dead space with a closed incision and a larger wound fluid volume),and decreased oxygen tension in adipose tissues. Many of these factors may be categorized together as a poor balance between tissue oxygen demand and oxygen supply.

Another mechanism may be the tissue concentrations of prophylactic antibiotic achieved in obese patients. A number of papers have demonstrated the importance of antibiotic concentrations in serum and tissue during an operative procedure in prevention of SSI.[111-112]

Forse et al. observed a high rate of SSI in patients undergoing gastric bypass and recorded low serum concentrations of antibiotic in these patients.[113]

A recent study from the Memorial Sloan Kettering Cancer Center also identified obesity as a risk factor for SSI development in patients undergoing colorectal resections.[114] However in our study we concluded no significant association between obesity and superficial surgical site infection as the inclusion criteria was elective and clean surgery and surgeries in which bowel was opened or injured were not included.

The growing epidemic of obesity in our country may be responsible for increasing the overall rate of surgical site infection.

None of the patients developed intraoperative hypotension or intraoperative hypothermia, which could add risk to development of surgical site infection and alter the results as it can be theorized that the contribution of poor wound tissue perfusion related to hypotension. Nonetheless, the data suggest the importance in the

maintenance of intraoperative normotension and normothermia in the reduction of SSI, and they identify this variables importance for further investigation.

Our data suggest that the development of incisional SSI was associated with longer hospital stay. Unlike other studies, we could detect no additional length of stay related to incisional SSI. This is probably due the extensive growth and availability of the home health care resources over the last decade as well as the strong institutional pressures regarding early discharge of most surgical patients. These data clearly highlight the transfer of the substantial medical expenditures related to the formation of an incisional SSI from the inpatient to outpatient setting.

There are several important study factors that require further discussion. First, all diagnoses of incisional SSI were recorded prospectively. Therefore, diagnosis was made by direct examination interpretation rather than interpretation.

This greatly reduces the possibility of the misdiagnosis of SSI when compared with a retrospective review, and the possibility of inaccuracies and incompleteness of all variable data points. Second, infection detection was done by southampton scoring system and culture study ,other studies variability in study done by chart review could not be assessed because only 1 surgeon adjudicated all the cases, and discernable differences in surgeons' tendencies to make a diagnosis of SSI has been reported.[116]

Despite these limitations, we believe the present study accurately portrays the rate of superficial SSI in this patient population.

CONCLUSION

There is no significant association between body mass index and superficial surgical site infection (p= 0.241) in incisional hernia patients in our study. Identification of surgical site infection and its frequency in obese patients is critical to approach its reduction. Further studies are required to conclude better association between body mass index and superficial surgical site infection in obese patients. Although this study has not shown significant association between BMI and superficial SSI it is necessary to take extra precautions in obese / overweight patients undergoing hernia surgery.

SUMMARY

Obese patients have tissue hypoperfusion (subcutaneous adipose tissue), which may predispose to SSI through a greater risk of ischemia/necrosis and suboptimal neutrophil oxidative killing. The present study was an undertaken to find association between body mass index and superficial surgical site infection in incisional hernia patients.

The present one year cross-sectional study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2012 to December 2012. A total of 60 patients diagnosed with incisional hernia during the study period fulfilling the selection criteria were studied.

Majority of the patients were females that is 76.67% and 23.33% were males. The female to male ratio was 3.28:1. The commonest age group was 46 to 60 years comprised of 53.33% of the patients followed by 31 to 45 years age group. Most of the patients (43.33%) had BMI between 25 to 29.99 Kg/m2. The wound culture was positive in five patients (8.33%). Of the five patients with positive wound culture, pseudomonas were isolated in 80% of the patients while staph aureus was present in 20%. 60% of the patients had organisms sensitive to antibiotics viz piperacillin tazobactum and 20% each were sensitive to Amikacin and Linezoid. Of the 26 patients with BMI 25 to 29.99 none of the patient (0%) developed surgical site infection. In 24 and 9 patients with BMI 18.5 to 24.99 and ≥ 30 16.67% and 11.11% of the patients had SSIs while one patient who had BMI < 18.5 Kg/m^2 did not develop SSI.

The association between superficial surgical site infection and BMI was not statistically significant (p= 0.241).

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ANNEXURE-I

INFORMED CONSENT

Dear Mr/Mrs/Dr_____, you are kindly requested to participate in a research study tilted "A LONGITUDNAL STUDY OF ASSOCIATION BETWEEN BODY MASS INDEX AND SUPERFICIAL SURGICAL SITE INFECTION IN INCISIONAL HERNIA PATIENTS AT TERTIARY CARE HOSPITAL " conducted by Dr._____, a post graduate student in M.S. General Surgery in Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid out criteria for a study 'subject'/ participant. The important elements of this study have been explained below in a question-answer format to help clear your queries/doubts.

During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your participation in this research is voluntary. Your decision whether or not to participate in the study will not affect your treatment in any form during your hospital stay. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"A LONGITUDNAL STUDY OF ASSOCIATION BETWEEN BODY MASS INDEX AND SUPERFICIAL SURGICAL SITE INFECTION IN INCISIONAL HERNIA PATIENTS AT TERTIARY CARE HOSPITAL"

OBJECTIVE/PURPOSE OF THE STUDY:

To study association between body mass index and superficial surgical site infection in incisional hernia patients.

Why am I being asked to participate in this research?

You are being asked to participate in this research as you fit in all the criteria laid by the author of the study as 'subjects'

Who is being recruited or selected?

Patients being selected are -

i. Patients diagnosed with incisional hernia undergoing elective surgery.

ii. Age group of patients between 15 to 60 years

Who are the investigators? Who is funding the study?

The investigator/author of this study is Dr. _____, a postgraduate student in Dept. of General Surgery, Jawaharlal Nehru Medical College.

The study is self funded by the author the study.

PROCEDURES:

What procedures are involved?

Procedure(s) involved would be a estimation of body mass index

$$BMI = \frac{\text{Weight in kg}}{\text{Height x Height(metre)}}$$

Will they cause pain or discomfort?

It will not cause any pain or discomfort.

Will they cause any temporary or lasting problems to me?

None

How many times I need to undergo the procedure?

Only one estimation of body mass index is required.

RISKS AND BENEFITS:

What are the potential risks and discomforts?

There are no potential risks involved with the procedure.

Are the benefits to taking part in this research?

None

ALTERNATIVES:

What other options are there?

What happens if I decline participation?

You would be excluded from the study and all your details shall be kept confidential.

Will I be told about new information that may affect my decision to participate?

Yes, you would be informed as when their is any information that may affect your desicion to participate in this study.

WITHDRAWING / REMOVAL FROM THE STUDY:

Can I withdraw from the study any time I want?

You would always have the voluntary will to withdraw from participation from the study anytime during the study.

Will I be penalized for that in anyway?

No.

Can I be removed from the study?

Yes, you can be removed from the study anytime as the author wishes to, if at any stage the author concludes that you may not continue to be an appropriate 'subject' for the study.

PRIVACY AND CONFIDENTIALITY:

What about privacy and confidentiality?

All data collected or disclosed by you during the course of participation of study, will be kept fully confidential.

Will my identity be disclosed?

No. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent.

FINANCIAL INCENTIVES FOR PARTICIPATION:

What are the costs for participating in this research? e.g., for services, etc

No additional costs shall be incurred upon you for the purpose of this study.

Will be reimbursed for any of my expenses for participation in this research?

N.A

Will they be available all the time?

Yes.

Will I be given the contact numbers & addresses of these persons?

Yes. If desired by you, contact numbers and addresses will be provided to you separately.

AUTHORIZATION TO PUBLISH RESULTS:

How will the results of the study be used?

The results of the study may be used to publish an article.

CONSENT STATEMENT:

I the undersigned Mr/Mrs/Dr_____ do hereby give consent for my participation in this research study after being explained in-depth about the important elements of this study in own my vernacular language. I give this consent voluntarily in my sound mind knowing very well the risks involved and been given enough time to clear my doubts and other queries to participate as a 'subject' in this study. I do hereby also give consent for publication of this article in any media / journal and have no objections whatsoever.

Did you read the contents or were the contents read out to you?

Tick one as appropriate:

- ☐ Read the contents by myself
- ☐ The contents were read out to me in English/Marathi/Kannada/Vernacular language

Were you informed that participation is voluntary?

Tick one as appropriate:

- ☐ Yes
- ☐ No

Did you have adequate time to clarify any doubts about the study or your rights as a study participant?

Tick one as appropriate:

- ☐ Yes
- ☐ No

Signature or left thumb print of participant or legally authorized representative

Participant'

Name_____Signature_____Experimenter's

name_____Signature_____Witness'

name_____Signature_____Guardian's

name_____Signature_____Date_____

(If the participant are Minors (under 18), the parents sign the form, rather than the participants)

ANNEXURE-II

PROFORMA / QUESTIONNAIRE TO BE USED FOR DATA COLLECION

The proposed proforma / questionnaire to be used for data collection for the study tilted "A LONGITUDNAL STUDY OF ASSOCIATION BETWEEN BODY MASS INDEX AND SUPERFICIAL SURGICAL SITE INFECTION IN INCISIONAL HERNIA PATIENTS AT TERTIARY CARE HOSPITAL " is as:

Personal Details:		
	Name:	Age / Sex:
	Address:	
	Educational Status:	Occupation:
	IP No.	Date of Admission:
	Ward:	Date of Discharge:
Chief Complaints: Abdominal swelling		
Past history: i. any past history of Diabetes Mellitus ☐Yes ☐No ii. Drug history of steroid intake ☐Yes ☐No any other significant p/h :		

Personal history:	
Family history (if significant) :	
General examination:	
	Built : Pulse : BP : Respiratory rate : Jaundice: Pallor / Clubbing / Cyanosis / Lymphadenopathy
Systemic Examination:	
	CNS: CVS: RS: P/A:
Clinical Diagnosis:	

Investigations:		
	Serological Investigations	Normal Range
	Body Mass Index	18.5 – 24.9
	Culture and sensitivity report:	

Grades	Appearance		Interval				
			Day 3	**Day 5**	**Day 7**	**Day 15**	**Day 30**
0		**Normal Hearing**	✓		✓	✓	
I		**Normal Hearing with mild bruising or erythema**					
	A	Same bruising		✓			
	B	Considerable bruising					
	C	Mild erythema					
II		**Erythema plus other signs of inflation**					
	A	At one Point					
	B	Around sutures					
	C	Along wound					
	D	Around wound					
III		**Clear or haemoserous discharge**					
	A	At one point only (<2 cm)					
	B	Along wound (>2 cm)					
	C	Large Volume					
	D	Prolonged (>3days)					
IV		**Major Complications**					
	A	At one Point (<2 cm)					
	B	Along wound (>2 cm)					
V		**Deep Sever Wound infection with or without tissue breakdown; haematoma requiring spiration**					

ANNEXURE III – PHOTOGRAPHS

Photograph: surgical site infection post incisional hernia surgery

Serial number	In patient number	Sex	Age (Years)	Date of admission	Date of discharge	General physical examination										Systemic examination				Diagnosis	Blood culture		Wound assessment				
						Pulse	Blood pressure		Respiratory rate	Jaundice	Pallor	Lymphadenopathy	Height (Cms)	Weight (Kgs)	BMI (Kg/m2)	Central nervous system	Cardiovascular system	Respiratory	Per abdomen		Culture	Sensitivity	Day 3	Day 5	Day 7	Day 15	Day 30
							Systolic	Diastolic																			
1	529435	F	60	11/5/2012	19/5/11	68	110	70	16	AB	AB	AB	1.65	70	25.73	N	N	N	N	IH	N	N	IA	IIIA	0	0	0
2	486934	M	57	11/8/2012	20/8/12	90	150	90	18	AB	AB	AB	1.72	92	31.83	N	N	N	N	IH	N	N	0	IA	IIA	IC	0
3	455935	M	51	23/1/12	30/1/12	80	130	76	16	AB	AB	AB	1.7	62	21.453	N	N	N	N	IH	N	N	0	0	0	0	0
4	502522	F	25	21/11/2012	29/11/12	90	110	80	16	AB	AB	AB	1.72	50	16.83	N	N	N	N	IH	N	N	0	IA	0	0	0
5	500932	M	40	10/9/2012	19/9/12	70	120	70	18	AB	AB	AB	1.7	74	25.6	N	N	N	N	IH	N	N	0	IC	0	0	0
6	468725	F	33	19/4/12	26/4/12	68	110	72	18	AB	AB	AB	1.5	68	29.43	N	N	N	N	IH	N	N	0	IIA	IA	0	0
7	530039	F	56	10/5/2012	20/5/12	70	110	80	16	AB	AB	AB	1.6	70	27.34	N	N	N	N	IH	N	N	IIA	IA	0	0	0
8	529417	F	31	2/5/2012	10/5/2012	70	110	80	16	AB	AB	AB	1.62	58	22.05	N	N	N	N	IH	N	N	IA	IA	0	0	0
9	528573	F	50	2/5/2012	10/5/2012	80	120	70	16	AB	AB	AB	1.65	70	25.73	N	N	N	N	IH	N	N	0	IA	0	0	0
10	536378	F	45	22/4/12	30/4/12	90	120	70	18	AB	AB	AB	1.65	66	24.26	N	N	N	N	IH	N	N	0	IC	0	0	0
11	528868	F	55	1/4/2012	10/4/2012	70	120	70	16	AB	AB	AB	1.55	60	25	N	N	N	N	IH	N	N	0	0	0	0	0
12	516304	F	52	8/9/2012	16/9/12	88	110	70	16	AB	AB	AB	1.65	60	22.05	N	N	N	N	IH	N	N	0	IB	0	0	0
13	514659	F	79	7/8/2012	14/8/12	70	110	80	16	AB	AB	AB	1.55	49	20.14	N	N	N	N	IH	N	N	0	IA	0	0	0
14	513287	F	62	30/8/12	7/9/2012	72	130	90	20	AB	AB	AB	1.5	55	24.44	N	N	N	N	IH	N	N	0	IA	0	0	0
15	472868	F	46	13/5/12	20/5/12	70	110	60	18	AB	AB	AB	1.72	58	19.52	N	N	N	N	IH	N	N	IA	0	0	0	0
16	482791	F	52	12/7/2012	21/7/12	68	110	70	18	AB	AB	AB	1.67	66	23.74	N	N	N	N	IH	N	N	IA	IA	0	0	0
17	497244	F	30	8/10/2012	18/10/12	76	110	70	18	AB	AB	AB	1.6	64	25	N	N	N	N	IH	N	N	0	0	0	0	0
18	457605	F	37	4/2/2012	13/2/12	82	130	70	16	AB	AB	AB	1.49	58	26.12	N	N	N	N	IH	N	N	0	0	0	0	0
19	480864	F	52	1/7/2012	7/7/2012	66	130	90	18	AB	AB	AB	1.6	76	29.68	N	N	N	N	IH	N	N	0	0	0	0	0
20	499467	F	52	26/10/12	5/11/2012	90	130	90	16	AB	AB	AB	1.75	84	24.22	N	N	N	N	IH	N	N	0	0	0	0	0
21	489692	F	36	24/8/12	2/9/2012	82	118	90	16	AB	AB	AB	1.55	64	28.44	N	N	N	N	IH	N	N	0	0	0	0	0
22	475292	M	58	12/5/2012	18/5/12	90	130	90	18	AB	AB	AB	1.75	71	23.2	N	N	N	N	IH	PSEUDOMONAS	LINEZOLID	IIIA	IIIC	IIC	IIA	0
23	491567	F	30	6/9/2012	14/9/12	88	110	70	16	AB	AB	AB	1.6	66	24.26	N	N	N	N	IH	S.AUREUS	AMIKACIN	V	V	IVA	IIA	IA
24	463625	F	55	16/3/12	21/3/1280	80	122	76	16	AB	AB	AB	1.62	62	23.57	N	N	N	N	IH	N	N	0	0	0	0	0
25	504912	F	32	10/12/2012	19/12/12	68	130	90	18	AB	AB	AB	1.5	68	30.22	N	N	N	N	IH	N	N	0	0	0	0	0
26	496090	M	44	2/8/2012	10/8/2012	90	118	70	16	AB	AB	AB	1.55	64	28.44	N	N	N	N	IH	N	N	0	0	0	0	0
27	478990	F	45	17/8/12	21/8/12	80	130	80	18	AB	AB	AB	1.54	72	30	N	N	N	N	IH	N	N	0	0	0	0	0
28	500093	F	46	29/10/12	6/11/2012	82	130	90	16	AB	AB	AB	1.64	76	27.94	N	N	N	N	IH	N	N	0	0	0	0	0
29	497488	F	50	10/10/2012	18/10.10	60	110	70	18	AB	AB	AB	1.6	66	25.78	N	N	N	N	IH	N	N	0	IC	0	0	0
30	452931	F	56	11/4/2012	20/4/12	70	110	70	20	AB	AB	AB	1.6	76	29.68	N	N	N	N	IH	N	N	0	IC	0	0	0
31	504905	F	50	6/12/2012	12/6/2012	80	120	90	16	AB	AB	AB	1.7	77	26.64	N	N	N	N	IH	N	N	0	0	0	0	0
32	481526	F	58	14/7/12	22/7/12	70	130	90	18	AB	AB	AB	1.7	74	25.6	N	N	N	N	IH	N	N	0	IIA	IA	0	0
33	485704	F	54	30/7/12	6/8/2012	68	120	70	16	AB	AB	AB	1.6	68	26.56	N	N	N	N	IH	N	N	0	IC	IA	0	0

Serial number	In patient number	Sex	Age (Years)	Date of admission	Date of discharge	General physical examination										Systemic examination				Diagnosis	Blood culture		Wound assessment				
						Pulse	Blood pressure		Respiratory rate	Jaundice	Pallor	Lymphadenopathy	Height (Cms)	Weight (Kgs)	BMI (Kg/m2)	Central nervous system	Cardiovascular system	Respiratory	Per abdomen		Culture	Sensitivity	Day 3	Day 5	Day 7	Day 15	Day 30
							Systolic	Diastolic																			
34	472917	F	48	16/5/12	24/5/12	80	140	80	18	AB	AB	AB	1.77	82	26.11	N	N	N	N	IH	N	N	0	0	0	0	0
35	504886	F	25	8/12/2012	16/12/12	84	116	80	20	AB	AB	AB	1.65	60	22.05	N	N	N	N	IH	N	N	IA	0	0	0	0
36	453151	F	25	14/2/12	20/2/12	76	118	80	18	AB	AB	AB	1.5	68.5	30.44	N	N	N	N	IH	N	N	IIB	IA	0	0	0
37	470847	M	57	3/5/2012	9/5/2012	80	130	90	18	AB	AB	AB	1.7	79	26.29	N	N	N	N	IH	N	N	0	0	0	0	0
38	452531	f	40	2/1/2012	10/1/2012	76	100	70	20	AB	AB	AB	1.625	58	21.96	N	N	N	N	IH	N	N	0	0	0	0	0
39	479852	M	50	27/6/12	5/7/2012	90	130	90	16	AB	AB	AB	1.7	76	26.29	N	N	N	N	IH	N	N	0	IA	0	0	0
40	484365	F	44	9/1/2012	15/1/12	68	114	70	18	AB	AB	AB	1.6	60	23.45	N	N	N	N	IH	N	N	0	0	0	0	0
41	497134	F	60	9/10/2012	15/10/12	82	110	90	16	AB	AB	AB	1.6	68	26.56	N	N	N	N	IH	N	N	0	0	0	0	0
42	492345	F	40	9/11/2012	15/11/12	90	120	90	18	AB	AB	AB	1.65	72	26.47	N	N	N	N	IH	N	N	0	IA	0	0	0
43	460008	M	41	23/2/12	1/3/2012	76	140	80	18	AB	AB	AB	1.5	66.5	29.55	N	N	N	N	IH	N	N	0	IIB	IIB	0	0
44	464424	M	50	23/3/12	29/3/12	84	130	70	20	AB	AB	AB	1.65	70	24.22	N	N	N	N	IH	N	N	0	0	0	0	0
45	479435	M	30	22/6/12	1/7/2012	90	160	100	16	AB	AB	AB	1.7	78	26.98	N	N	N	N	IH	N	N	0	0	0	0	0
46	475668	M	35	1/6/2012	7/6/2012	80	120	90	18	AB	AB	AB	1.77	76	24.2	N	N	N	N	IH	N	N	0	0	0	0	0
47	460796	M	50	25/2/12	18/3/12	82	130	70	16	AB	AB	AB	1.7	70	24.22	N	N	N	N	IH	PSEUDOMONAS	AMIKACIN	IID	IVB	IVB	IA	0
48	473140	F	59	15.5.12	21/5/12	74	130	90	18	AB	AB	AB	1.64	79	29.04	N	N	N	N	IH	N	N	0	0	0	0	0
49	476616	F	28	8/6/2012	16/6/12	70	130	90	18	AB	AB	AB	1.64	66	24.26	N	N	N	N	IH	N	N	0	0	0	0	0
50	482268	M	48	3/7/2012	9/7/2012	90	130	80	18	AB	AB	AB	1.725	76	25.58	N	N	N	N	IH	N	N	0	IA	0	0	0
51	471755	F	58	9/8/2012	19/8/12	68	118	90	16	AB	AB	AB	1.54	68	28.33	N	N	N	N	IH	N	N	0	0	0	0	0
52	472100	F	30	2/5/2012	10/5/2012	76	126	78	16	AB	AB	AB	1.6	62	24.21	N	N	N	N	IH	N	N	0	0	0	0	0
53	463494	F	28	17/3/12	26/3/12	80	120	90	16	AB	AB	AB	1.7	58	20.06	N	N	N	N	IH	PSEUDOMONAS	PIPRACILLIN TAZOBACTUM	IA	IIC	IVA	IID	0
54	461568	F	55	3/6/2012	10/6/2012	90	130	90	16	AB	AB	AB	1.6	62	24.21	N	N	N	N	IH	N	N	0	0	0	0	0
55	542336	F	34	1/8/2012	9/8/2012	68	130	80	16	AB	AB	AB	1.55	68	28.33	N	N	N	N	IH	N	N	0	0	0	0	0
56	544577	F	38	26/7/12	1/8/2012	70	110	70	18	AB	AB	AB	1.6	62	24.21	N	N	N	N	IH	N	N	0	0	0	0	0
57	541029	M	58	18/7/12	2/8/2012	76	130	90	16	AB	AB	AB	1.7	92	31.83	N	N	N	N	IH	PSEUDOMONAS	AMIKACIN	IC	IIIB	IIIB	IA	0
58	539690	F	58	4/7/2012	10/7/2012	76	130	70	16	AB	AB	AB	1.65	80	29.41	N	N	N	N	IH	N	N	0	0	0	0	0
59	530431	F	48	18/5/12	26/5/12	90	120	90	16	AB	AB	AB	1.67	68	24.37	N	N	N	N	IH	N	N	0	IIA	0	0	0
60	535256	F	72	12/6/2012	18/6/12	76	130	80	16	AB	AB	AB	1.55	76	31.66	N	N	N	N	IH	N	N	0	IIA	0	0	0