"A COMPARATIVE STUDY BETWEEN DELAYED ABSORBABLE SUTURE POLYDIOXANONE (PDS) AND NONABSORBABLE SUTURE POLYPROPYLENE SUTURE IN LAPAROTOMY WOUND CLOSURE"

By

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DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SURGERY

IN

GENERAL SURGERY

Under the guidance of

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ABSTRACT

Background and objectives:

Abdominal wound closure is one of the common operation for a general surgeon. Post-operative wound pain, wound infection, wound dehiscence, suture sinus formation, palpable knots and incisional hernia are the parameters which are to be studied. Prevention of complications is important to reduce post-operative morbidity and mortality. The search for the optimal laparotomy technique has gone on for more than 100 years and will continue.

Research hypothesis:

The objective of this study was to compare polydioxanone(PDS) and polypropylene(prolene) suture material for abdominal fascial closure regarding morbidity in terms of post operative wound complications.

Material:

Polydioxanone(PDS) versus polypropylene(prolene) suture material.

Methods:

All patients admitted in department of surgery, R.L.JALAPPA HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR who undergo laparotomy operations, with midline abdominal incisions. This include emergency and elective procedures. The study period will be between JAN 2013 to JAN 2014.

By taking into account, the variables like suture sinus formation and wound granuloma as estimated in previous studies sample size of 120 cases will be allotted in each group, total of 240 patients.

predesigned proforma will be used to collect this information for individual case. Data were collected based on postoperative wound complications including postoperative wound pain, wound infection, wound dehiscence, suture sinus formation, palpable knots and incisional hernia. The patient will be followed up at 2weeks, 4 weeks and once in 3 months upto 1 year.

Results:

People around 25-30 years age group formed the maximum number of this study. Male to female ratio was 4:1. The incidence of wound pain was among all the patients (out of 100 cases) in polypropylene group, whereas 20%(out of 100 cases) of patients in polydioxanone group. The incidence of wound infection was 24%(out of 100 cases) in polypropylene and 2%(out of 100 cases) in polydioxanone group. There were 4 cases(out of 100 cases) of wound dehiscence in polypropylene group and none in polydioxanone group. The incidence of suture sinus formation was 9%(out of 100 cases) in polypropylene group compared to 2%(out of 100 cases) in polydioxanone group. The incidence of palpable knots was 23%(out of 100 cases) compared to polydioxanone group in which no cases were reported. There were 2 cases (out of 100 cases) of incisional hernia in polyproylene group compared to polydiaxanone group in which no cases were reported.

Since the follow up period was limited for one year, the cases which are collected after the study period of JAN 2014 are followed up for the period of 3 to 6 months.

Since the palpable knots and incisional hernia are the complications which are expected to occur at the end of one year. These parameters are not studied in a small group of patients. This is the limitation of our study.

Conclusion

Based on the observations made in this study, it has been concluded that continuous mass closure technique using no.1 Polydiaxanone (PDS) for closure of midline laparotomy incision is superior to no.1 Polypropylene (Prolene) suture material in preventing the wound complications like post-operative wound dehiscence, wound pain, wound infection, suture sinus formation, palpable knots and incisional hernia.

Keywords:

Abdominal wound dehiscence; Burst abdomen; Emergency laparotomy; Midline incisions; Suture technique; Suture sinus; Abdominal closure; Wound infection.

LIST OF ABBREVATIONS

DC	:	Differential count
ESR	:	Erythrocyte sedimentation rate
Hb	:	Hemoglobin
TC	:	Total Count
HBsAg	:	Hepatitis -B surface antigen
HIV	:	Human Immunodeficiency virus
IP No.	:	Inpatient number
RR	:	Relative risk
SL:WL	:	Suture length : wound length
W/V	:	Weight / Volume
LB	:	Large Bowel
SB	:	Small Bowel
PDS	:	Polydiaxanone
PPL	:	Polypropylene
EMERG	:	Emergency
ELE	:	Elective
WI	:	Wound Infection
BA	:	Burst Abdomen

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INTRODUCTION

Wound healing following abdominal closure is a complex and dynamic process with changing wound environment and changing health status of the individual.[1] Among several factors which affects the wound closure. A careful selection of proper suture material is one of the important factor. The ideal suture material with the perspective of fast and comfortable recovery is yet to be ascertained.

Surgeon always follow a primary wound closure because wound heals by primary intention with a minimal time period without gaping and minimal scarring.

Absorbable sutures are required for a wound that heals quickly and needs temporary support. Polydioxanone(PDS) a monofilament synthetic absorbable suture, represents a significant advance in suturing options. With its absorbability and extended wound support for upto 6 weeks, it is well suited for many types of soft tissue approximation. With the combinational property of retaining strength for considerable period and absorbability it is of significant value in laparotomy wound closure and has minimal post operative complications.

Non absorbable sutures are required for the conditions where longer wound support is needed. Polypropylene is a synthetic nonabsorbable suture material. It has a property of non adherent to tissue and so a good suture material, but known to cause palpable knots, wound pain , wound sepsis, wound dehiscence, suture sinus formation, stitch granuloma and incisional hernia.[2]

However, studies comparing these two suture materials are scarce in a rural tertiary care hospital like Kolar. Thus the need to undertake this study to compare the efficacy of these two suture materials for abdominal fascial closure.

OBJECTIVES OF STUDY

1. To study the efficacy of delayed absorbable polydioxanone suture.
2. To study the efficacy of non absorbable polypropylene suture.
3. To compare the efficacy of polydiaxonone and polypropylene in laparotomy wound closure.

REVIEW OF LITERATURE

In abdominal surgeries technique of incisions is of paramount importance. In the same way wound closure method is equally important.[3] The history of sutures begins more than 2000 years ago with first records of eyed needles. Around A.D. 175 Galen, a physician to the Roman gladiators, experimented with catgut. Initially only natural materials were used. These included flex, hemp, horse and human hair, pig bristles, weeds, grasses, and the mouth parts of pincher ants. The Indian plastic surgeon, Sushrutha (AD 380-450) described suture material made from flex, hemp, and hair.[4]

In the 1800's and early 1900's silk, cotton, and catgut were extensively used. In 1869 Lister introduced the practices of impregnating catgut with chromic acid and sterilizing suture material. In the early part of this century, Halsted promoted the advantages of silk over catgut, and silk soon became the most common suture material in surgical practice[5]

In the 1940's synthetic materials such as nylon and dacron initially developed for other purposes, were used for suturing wounds. In the 1960's Frazza and Schmitt started the search for synthetic absorbable sutures. This led to the development of polyglycolic acid, polyglactin 910 and polydioxanone[4,5]

Natural materials are more traditional and still are used in suturing today. Synthetic suture materials are less reactive in that the resultant inflammatory reaction around the suture material is minimal.

The use of plain and chromic catgut had lead into high chances of wall dehiscence in past. Goligher recommends not to use catgut for fascial closure unless the retention sutures are used[6]

Suture also plays an important role in healing of tissue. So proper choice of suture should be done to oppose the tissue. Suture should also help to keep the tissues approximated until it achieves adequate strength.

The absorbable suture material like polyglycolic acid and polygalactin 910 have a property of maintaining tensile strength for adequate period of time.

Non absorbable suture materials like nylon and polypropylene are used for fascia closure. The nylon which is monofilament material, looses 16% of tensile strength by 70 days and 20% by 200 days. Studies conducted by Irvin DJ et al and Corman ML, Veidenheimer MC et al showed wound dehiscence rates of 10% and 6% when Dexon, vicryl or prolene were used respectively for fascial layer closure.[7,8]

Silk and cotton are good suture materials, but promotes infection. Stainless steel also potentiates infection. Wire sutures have a property of breakage and form a nidus for infection. Polypropylene, nylon and non absorbable suture material have lowest incidence of infection in case of contaminated wounds.

In this aspect , polyglycolic acid have minimal chances of causing suture sinus formation.

With these observations it could be derived that experience and personal preference often dictate the choice of suture material by the individual surgeon. Factors such as handling, tensile strength, knottingcharacters, tissue reactivity, visibility and capillarity and absorption characters all play a part in surgeon's final choice.

However an ideal suture material is yet to be found which satisfy all the above said criteria. So surgeons should make a correct choice of suture material according to area of incisions and type of tissue. Also the following factors to be considered like potential for infection, existing infection and even anticipitated post-op complications and rate of healing.

The following principles are followed in any type of suturing,

1. Sutures are to be kept in only till the wound heals and attain a maximum tensile strength.
2. The tissues that heals slowly such as skin, tendons and fascia should be closed with non-absorbable sutures.
3. The tissues such as stomach, intestine and bladder which heals rapidly should be closed with absorbable sutures.
4. Biliary and urinary tract are more prone for stones formation, hence absorbable sutures are to be used.
5. Foreign bodies which are prone for contamination can convert an wound into active infection , therefore
    - Multifilament sutures are avoided because bacteria can lodge within and can lead to infection.
    - Monofilament sutures are used when we suspect burst abdomen.
    - In infective condition, monofilament is the better choice as a suture material.
6. Cosmetic measures which are to be stressed while suturing are
    - Avoid prolonged apposition of wound surfaces
    - Avoid using irritants
    - Use nylon or polypropylene which are small and inert monofilaments.
7 Size of suture material is important because the suture material acts as a foreign body. Hence the suture material should be smaller in size and minimal number of knots is said to be ideal, because too many knots makes the foreign body larger.
    - Retention sutures should be placed whenever we suspect dehiscence.
    - Suturing handling and knotting is also important, therefore
    - When we are using synthetic material such a nylon, prolene and polyester, a

triple throw should be applied.

We should have knowledge of

- Healing characteristics of the tissues to be approximated
- Suture materials, its physical and biological properties.
- Wound and its condition.
- Patients and his/her course in hospital

Various suture material.:

Sutures	Absorbable	Non-absorbable
Monofilament	Catgut	Polyamide
	o plain	Polypropylene
	o chromic	Polyester
	Collagen	
	o plain	
	o chromic	
	PDS	
Multifilament	Polyglycolic acid	Silk
	Polygalactin 910	Linen
		Polyester braided
		Polyamide
		Stainless steel

An ideal suture material should have following properties[9]

- Adequate tensile strength.
- Tissue reaction in-situ should be minimal.
- Handling should be easy.
- Knotting security should be good.

Absorbable suture material:

The ways in which the absorption takes place is by

- Enzymatic digestion eg: catgut and collagen
- Hydrolysis eg All synthetic material (local temperature and PH affects this process)

Advantages of absorbable suture materials:

o Ideal sutures in urinary and biliary system procedures

o Ideal in gastrointestinal anastomosis

o Also in female genital tract(infective conditions)

Advantages of non-absorbable sutures:

They have a property of high tensile strength, which causes them to stay for longer time even after the wound healing process is completed.

So it is used when we suspects that wound healing is slow and chances of infection is more.

Disadvantages:

It stays as a foreign body in-situ. Hence it can lead to formation of post operative site infection, suture sinus formation ,palpable knots and wound dehiscence.

Tensile strength:

The knowledge of tensile strength is important to select an ideal suture which has good tensile strength. A wound will attain tensile strength in a slow process i.e 20% of normal tensile strength is attained by 20 days, 40% at 40 days and about 60% by about 100 days and 70% of the strength by one year, but never attains the normal strength.

Knotting properties:

Another important property of an ideal suture is knotting technique.

Knotting properties of suture is said to be excellent, when it is secured with two knots. A suture can be cut short in case of good knotting properties to avoid foreign body reactions in-situ. eg: polyfilament and braided suture materials like cotton and silk

Monofilament suture material have got poor knotting properties, so to improve this first knot should be surgeons knot followed by atleast two knots.

At present, there are different types of suture materials with different qualities in terms of their handling, knot security and strength depending on purposes.

The ideal suture technique appears to be a mass closure using continuous running suture technique with an adequate, suture length to wound length ratio of atleast 4:1 in situ[10].

With respect to abdominal fascia closure controversy exists regarding the use of absorbable v/s non absorbable suture materials. Non absorbable suture material are commonly associated with scar pain, wound infection, wound granuloma, suture sinus formation and have stirred the interest in the use of absorbable sutures[11.] However resorbable sutures bear an intrinsic loss of tensile strength during the vulnerable post operative period and leading to ventral hernia.[12]

Several trials made to compare between delayed absorbable and non absorbable suture material and other interrupted versus continuous suture techniques. They reported significant higher incidence of wound pain and suture sinus formation where non absorbable suture materials was used.[13]

The suture material is said to be ideal when it stays in-situ and stabilizes the wound during the healing period. Suture should have minimal possibility of causing wound infection, wound granuloma, suture sinus formation, wound dehiscence and incisional hernia. In addition to this it should be easily available and inexpensive[14].

POLYDIAXONONE(PDS):

It is a homopolymer made from paradioxanone to give polydioxanone, a polyester. PDS is manufactured as a monofilamentous suture .It takes approximately 180 days for invivo absorption. It also retains significant tensile strength after 28days, 58% of the original value. Tissue reaction to the suture is minimal. Since it is a monofilament its affinity for microorganisms is negligible[15,16]

POLYPROPYLENE:

It's a synthetic monofilament made from the linear hydrocarbon polymer polypropylene. It remain encapsulated in body tissues and acts a foreign body to cause infection in immediate post operative period and less adherent to tissue in situ [15,16,17].

ANATOMY

EMBRYOLOGY (ANTERIOR ABDOMINAL WALL)[18]

Development of abdominal wall begins very early in the initial stages of embryo formation, but the definite structure of abdominal wall attained only after the separation of umblicus from fetus. Abdominal wall of embryo completes its development during closure of midgut and while body stalk reduces in size. Initially the somatopleura is formed, which is the primitive wall which is composed of ectoderm and mesoderm without muscles's, vessels or nerves. In the sixth week of gestation, this somatopleura is invaded by myotomes into the mesoderm .The mesoderm which looses segmental pattern as grows and it migrates laterally and ventrally in form of a sheet. As this sheet grows, its leading edges differentiates to form right and left rectus abdominus. Ultimately the approximation of the latter will close the body wall.

The splitting of main body of mesodermal sheet will give rise to three layers. They are external layer, middle layer and an internal layer. External layer which give rise to external oblique muscle, middle layer give rise to internal oblique muscle and the inner layer give rise to transverse abdominis. This development completes by middle of seventh week. The recti also approximates in both caudal and cranial ends, which closes by the end of 12th week, except at the area of umblical cord.

SURGICAL ANATOMY OF ANTERIOR ABDOMINAL WALL: [19,20,21,22,23]

A sound knowledge of surgical anatomy of anterior abdominal wall plays a very important role in arriving at decision while planning incision in abdominal wall. Anatomic principles plays important very important role in making incisions in abdominal wall. Our aim is to keep the abdominal wall strong post-operatively

because weak abdominal wall can lead to incisional hernia.

Definition: The abdomen can be defined as the region of the torso, that extends between the diaphragm above to the pelvis inlet below.

Structure of the abdomen wall:

Inferiorly, the abdominal cavity is continuous with the pelvic cavity through pelvic inlet. Anteriorly, the abdominal wall is formed above by the lower part of the the thoracic cage and below by rectus abdominus, external oblique, internal oblique and transversus abdominis muscles and fasciae.

Posterior boundary of the abdominal wall is formed by bony and muscular structures. Five lumbar vertebrae and their intervertebral disc are the midline structures. Laterally by 12th rib and upper part of bony pelvis. Apart from bony stuctures, abdomen is also made up of muscular stuctures like psoas muscles, quadratus lumborum muscles and the aponeurosis of origin of transversus abdominis muscles forms lateral boundary. Iliacus muscles lie in the upper part of bony pelvis. The abdominal walls are lined by fascial envelope and parietal peritoneum.

SURFACE ANATOMY:

For clinical purpose the anterolateral abdominal wall is divided into nine regions by two vertical lines extending from mid-clavicular point to mid-inguinal point bisecting the inguinal ligament and two horizontal lines, the upper one between tips of 9th costal cartilage - transpyloric line and the lower one transtubercular line at the level of two tubercles on the iliac crest about 2" behind the anterior superior iliac spine.

The areas are epigastric, right and left hypochondrium, umbilical, right and left lumbar, hypogastric, right and left iliac fossae. Umbilicus lies midway between xiphisternum and pubic symphysis. Recti form bulging bands on each side of midline. The contour of abdomen is subject to considerable variations.

SUPERFICIAL STRUCTURES:

Abdominal skin is loosely attached to subjacent structures but the skin at umbilicus firmly adherant. The coarse fibrous tissue bundles and arrangement of elastic fibres in the wall produces bodylines of tension in the skin.

Surgical incisions are planned in such a way that it should not cross the lines of tension. Or else it may lead to scars especially when wound get infected.

Deep to skin is subcutaneous tissue which is made up of two named layers , that is fatty Camper's fascia and fibrous fascia of Scarpa's which is contiguous with the fascia lata of thigh. Scarpa's fascia is the one which helps in alignment of skin after suturing.

ABDOMINAL MUSCLES:

Some of the important functions of abdominal wall muscles are protecting the contents, helps in the act of micturition, helps in the act of defecation, assist in the ventilation and vomiting. Uncommon pressure exerted over the muscles can bring about their rupture as seen in muscular strain or parturition where the recti can tear. Transversus abdominis acts only on abdominal contents, and has no effect on vertebral column.

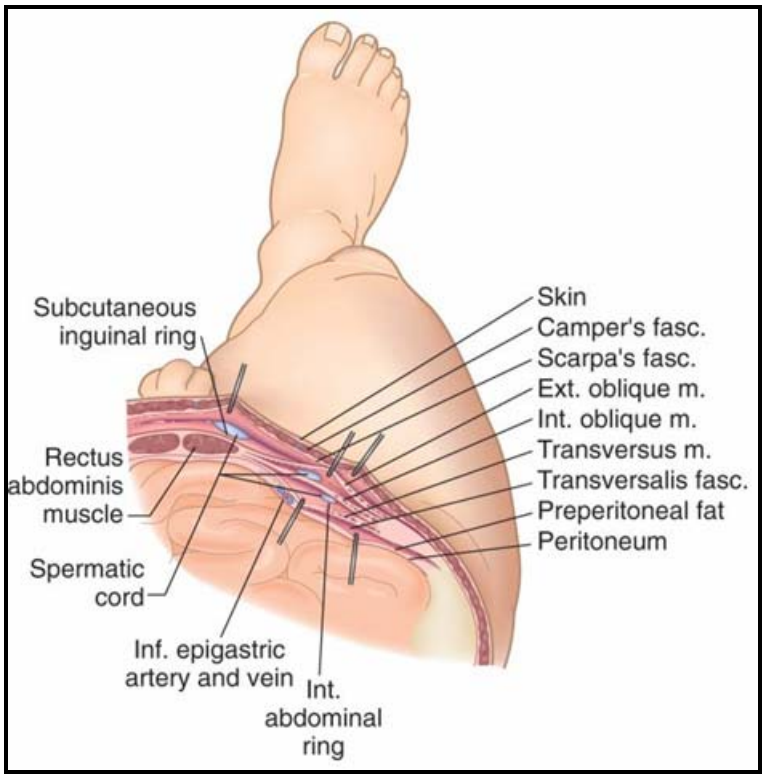


Fig-1 There are nine layers to the abdominal wall: skin, subcutaneous tissue, superficial fascia, external oblique muscle, internal oblique muscle, transversus abdominis muscle, transversalis fascia, pre-peritoneal adipose and areolar tissue, and peritoneum.

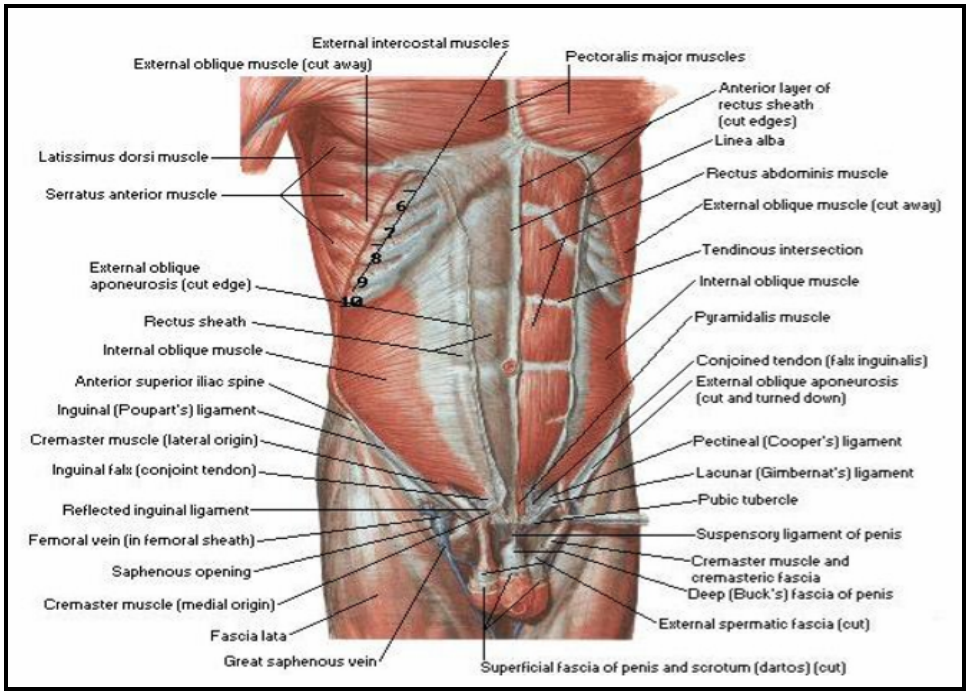


Fig 2 : Abdominal wall and its layers:

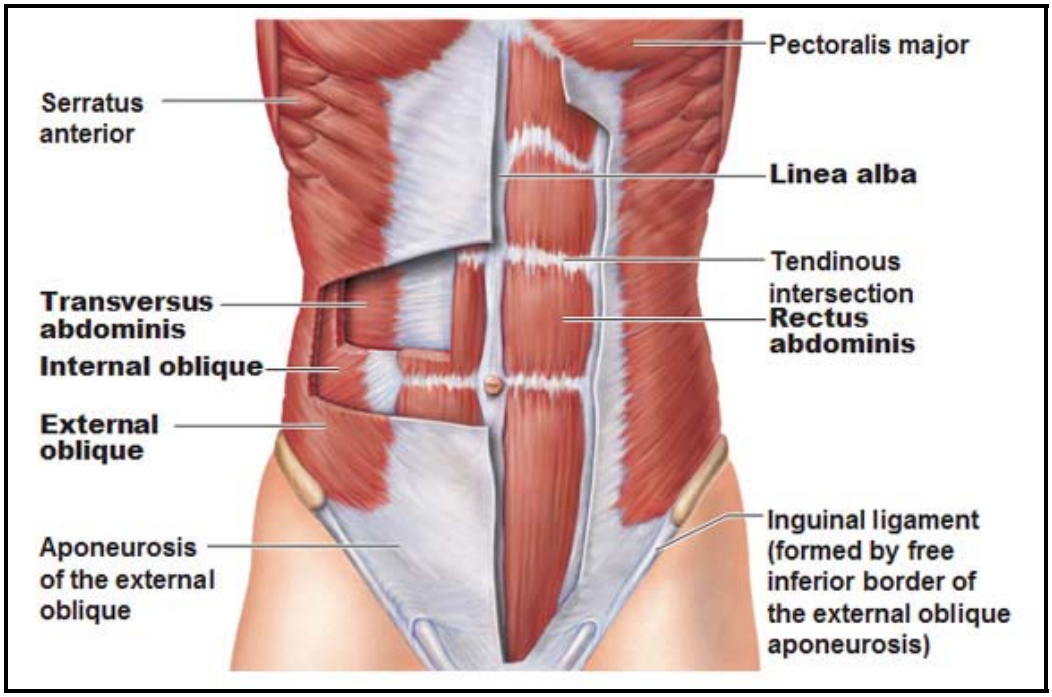


Fig 3: Muscles forming abdominal wall showed in cut sections: rectus abdominus, external oblique, internal oblique and transverse abdominus.

OBLIQUS EXTERNUS ABDOMINIS:

This is the largest and most superficial of the three flat muscles. It arises by 8 slips from external surfaces and inferior borders of lower 8 ribs, which interdigitate with slips of origin of serratus, pass vertically downwards and are inserted into anterior half of the outer lip of iliac crest. Other fibres end in an aponeurosis. None of the fleshy fibres of the muscle extend downwards beyond a line drawn from anterior superior iliac spine to the umbilicus.

In the median plane, the aponeurosis ends in linea alba which is a tendinous raphe extending from xiphoid process to symphysis pubis. The margin of this aponeurosis extending from pubic tubercle to anterior superior iliac spine is thick and folded backwards forming inguinal ligament, below which it is continuous with fascia lata.

Nerve supply:- Ventral rami of lower six thoracic nerves.

OBLIQUS INTERNUS ABDOMINIS:

It is thinner and less bulky, lies under cover of external oblique. Arises by fleshy fibres from lateral 2/3rd of grooved upper surface of inguinal ligament, anterior 2/3rd of intermediate line of the ventral segment of iliac crest and from thoraco lumbar fascia.

They are inserted into the inferior borders of the lower 3 or 4 ribs. Fibres arising from inguinal ligament, run downwards and medially becoming tendinous, are inserted conjointly with the aponeurosis of the transverses abdominis into the crest of pubis-pecten pubis forming conjoint tendon.

Rest of the fibres diverge and end in an aponeurosis. The aponeurosis splits at the lateral border of rectus into two lamellae ensheathing the muscle and reunite at the linea alba.

Anterior layer of this blends with aponeurosis of external oblique and posterior layer blends with aponeurosis of transversus abdominis.

Nerve supply:- Ventral rami of lower six thoracic and first lumbar nerves.

TRANSVERSUS ABDOMINIS:

It arises by fleshy fibres from lateral 1/3rd of the inguinal ligament, anterior 2/3rd of inner lip of ventral segment of iliac crest, thoraco lumbar fascia and inner surfaces of lower costal cartilages.

It ends in an aponeurosis and are inserted together with aponeurosis of lower lamellae of internal oblique into linea alba in the median plane.

Lower fibres form conjoint tendon with arched fibres of internal oblique.

Nerve supply:- Ventral rami of lower 6 thoracic and first lumbar nerves.

PYRAMIDALIS:

Triangular muscle placed at the lower part of abdomen in front of rectus within the sheath of the muscle, arises by tendinous fibres from the front of the pubis ascends upwards , ends in a pointed extremity and is inserted into lina alba midway between umbilicus and pubis. It is a tensor of linea alba.

Nerve supply: Subcostal, T12.

RECTUS ABDOMINIS MUSCLE AND RECTUS SHEATH :

Recti are long flat muscles, broader above than below extending along the whole length of abdomen and is separated from its counter part by linea alba.

It arises by two tendons, lateral larger one arises from pubic crest and medial one interlaces across the midline with the opposite one, it is inserted as three slips into cartilages of 5th, 6th and 7th ribs.

It has three tendinous intersections, upper one opposite the free end of the xiphoid process, 2nd one at the level of umbilicus and the 3rd one midway between these two.

They are intimately adherent to the anterior lamina of the sheath of the muscle. These recti are enclosed in a sheath formed by the aponeurosis of oblique and transversus.

At the lateral margin of the rectus, the aponeurosis of internal oblique divides into two lamellae, one passes infront of the rectus, blending with aponeurosis of external oblique, the other passes behind the recti and blend with aponeurosis of transversus and these joining again in the medial border of the rectus reaching linea alba.

This arrangement exists from costal margin to a variable level, usually midway between umbilicus and symphysis pubis where posterior wall of the sheath ends in a curved margin - Arcuate line, concavity of which is directed downwards. Below the level of this, all the three muscles pass infront of the rectus. Hence the recti are separated from peritoneum only by transversalis fascia.

The lateral margin of the recti are marked on the surface of the anterior abdominal wall by a curved groove termed linea semilunaris, which extends from the tip of the 9th costal cartilage to pubic tubercle.

Nerve supply: Ventral rami of lower 6 or 7 thoracic nerves.

LINEA ALBA

It is a tendinous raphe placed between medial borders of recti formed by interplacement of fibres of aponeurosis of oblique and transversus stretching between the xiphoid process and symphysis pubis. It is narrow below and broader above.

Its supraumbilical portion can usually be recognized as a shallow groove. Its lower end has double attachment-superficial fibres passing infront of the medial heads of the recti to the front of symphysis pubis while its deeper fibres form a triangular lamella attached behind the recti to the posterior surface of the pubic crest called adminiculum linea albae.

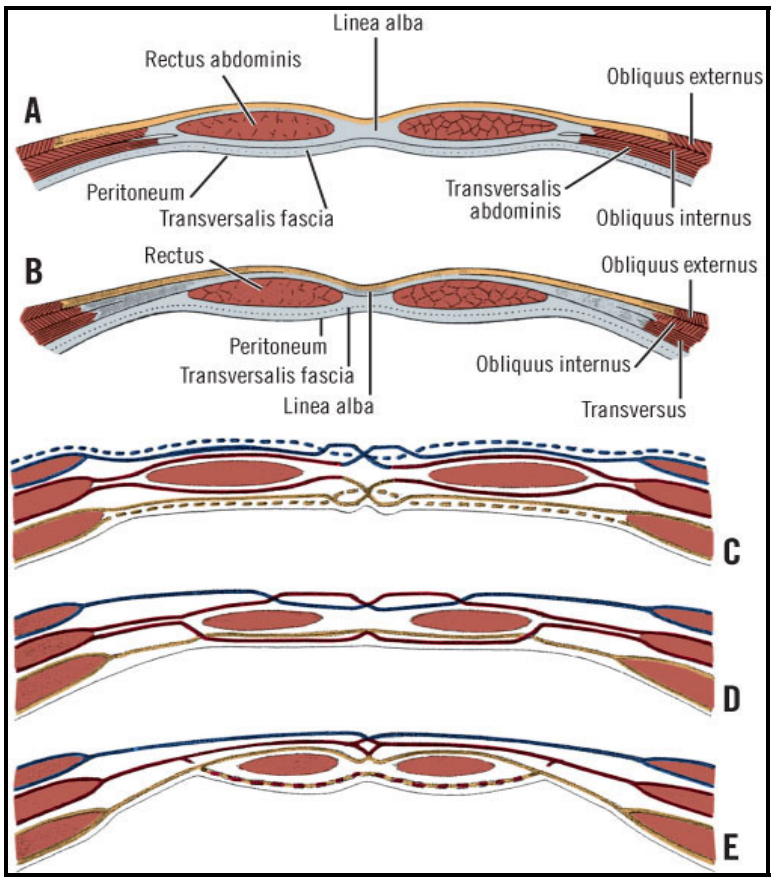


Fig 4 : Abdominal wall in coronal section: showing formation of rectus abdominus and rectal sheath

TRANSVERSALIS FASCIA:

It's the thin fascia which is defined in between inner surface of transversus and extraperitoneal fat. The same fascia continues with iliac and pelvic fascia

In the inguinal region it receives fibres from aponeurosis of transversus. Above it blends with the fascia covering undersurface of the diaphragm.

Behind it is lost on the surface of thoracolumbar fascia with which it blends. Below it has following attachments - posteriorly to the whole length of iliac crest between the origins of transversus and iliacus between anterior superior iliac spine and femoral vessels and to the posterior margin of inguinal ligament which continuous with iliac fascia.

It is fixed to the pectin pubis medial to the femorals, it descends infront of femoral vessels to form the anterior wall of femoral sheath. In well nourished patients, the peritoneum may be separated from the transversalis fascia with ease. It extends into the scrotum as internal spermatic fascia.

EXTRA PERITONEAL TISSUE:

It's the fibroareolar tissue, lying in between transversalis fascia and peritoneum. It is scanty in the anterior abdominal wall, except in the pubic region and above iliac crest.

BLOOD SUPPLY OF THE ANTERIOR ABDOMINAL WALL

Arterial supply of abdominal wall achieved by branches from various arteries and their free anastomosis. Arteries which are present above umbilicus are superficial epigastric artery, musculophrenic artery, lower intercostal arteries. Arteries which are present below umbilicus are femoral artery, superficial epigastric artery, superficial

circumflex iliac artery and superficial external pudental artery. These arteries forms anastomosis and supply abdominal wall.

Venous drainage of abdominal wall :

Above umbilicus, drain into internal mammary, intercostals and long thoracic vein. Veins below the umbilicus drain into femoral vein.

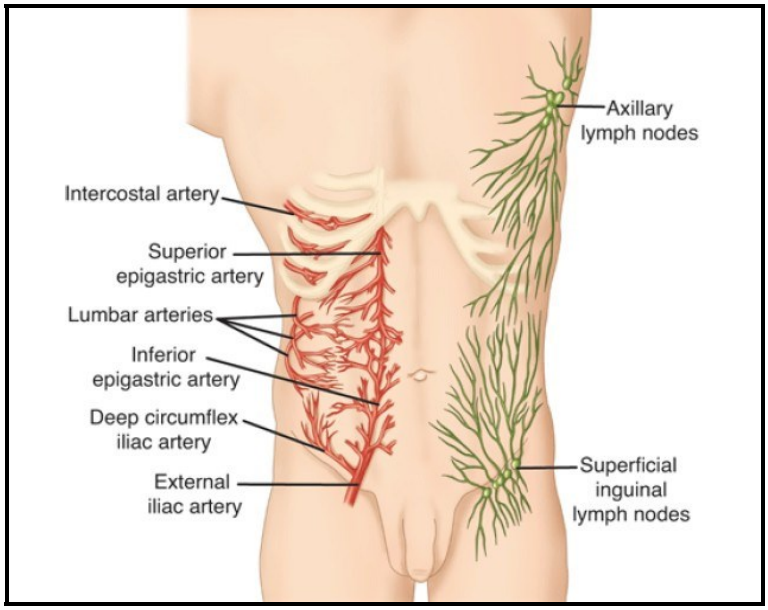


Fig 5: Blood Supply and lymphatic drainage of the Anterior Abdominal Wall

LYMPHATIC DRAINAGE OF ANTERIOR ABDOMINAL WALL:

Lymphatics from the superficial fascia and skin above the umbilicus drain to axillary nodes, those from below the umbilicus drain to superficial inguinal nodes.

Lymphatics from the abdominal wall muscles, the extraperitoneal connective tissue and parietal peritoneum drain along the arteries to nodes associated with parent vessels (lateral aortic, parasternal, external iliac and inguinal lymph nodes).

THE PERITONEUM:

The peritoneum is the serous membrane which is structured and complexly arranged in body.

Peritoneum in males is a closed sac, where as in females it's a opened cavity since the fallopian tubes open into peritoneal cavity.

In both sexes, the part which lines abdominal wall (parietes) is called parietal portion of peritoneum. The one which is reflected over viscera constitutes the visceral portion of peritoneum.

The free surface of the membrane is smooth covered with a layer of flattened mesothelium and kept moist by a small quantity of serous fluid.

Hence the viscera can glide on the wall of the cavity or on one another with a minimum friction and complete freedom within the limits dictated by their attachments to the wall of abdominal cavity or to other structures such as vascular structures.

The peritoneal cavity is of course a coelom, a discontinuity in the mesoderm with its own special surface epithelium which maintains the surface.

Loss of these epithelium entails adherence of the underlying tissues and a consequent interference with function which may be serious and even lethal.

A considerable amount of areolar connective tissue intervenes between the parietal peritoneum and the abdominal walls with the fascial lining of which it blends. It is known as extra peritoneal tissue.

It varies in quantity and contains varying amounts of fat in different regions. This tissue loosely connects the parietal peritoneum to the abdominal and pelvic walls in general and so allows the peritoneum to be relatively easily stripped off these walls.

It is dense in the undersurface of the diaphragm and behind linea alba, so that

parietal peritoneum is more firmly adherent to these parts.

It is especially loosely arranged in some places to allow for alterations in the size of certain organs e.g. in the front part of the pelvis and lower part of anterior abdominal wall where it allows the urinary bladder to distend in an upward direction behind the anterior abdominal wall from which it strips off the peritoneum as it ascends.

It is usually laden with fat in relation to kidneys. The visceral peritoneum on the other hand is firmly united to the viscera which it covers and cannot be readily stripped off.

Infact the connective tissue layer (tela subserosa) of visceral peritoneum is directly continuous with fibrous tissue stroma of the viscera. Thus from the point of view of pathological condition of an organ, the visceral peritoneum must be considered to be a part of the viscera itself.

The peritoneum consists of a single layer of flattened mesothelial cells which covers a layer of loose connective tissue. In most areas, the mesothelium forms a continuous surface.

Adjacent mesothelial cells are joined by junctional complexes, which probably allow the passage of macrophages to and from underlying connective tissue in the same manner as endothelial cell junctions which allow leucocytes to pass from blood stream.

In other areas, however as in greater omentum the peritoneum may be discontinuous presenting a series of fenestrations which may be visible to unaided eye.

At such points mesothelial surface layers is continuous over trabecular connective tissue which interlace around the margin of fenestrations.

The submesothelial connective tissue carries cells usually found in loose connective tissues. It has been claimed that the mesothelial cells possess macrophages.

They may also transform into fibroblasts and fusion between layer of fibroblasts of mesothelial origin may lead to macroscopic adhesions between the peritoneal surfaces of adjacent structures. If extensive, these may have serious clinical consequences interfering with intestinal motility or even leading to complete intestinal obstruction.

The mesothelium is similar in many respects to the endothelial lining of blood vessels and it forms a dialyzing membrane across which fluids and small molecules of various solutes may pass.

Numerous pinocytotic vesicles are present near pinocytotic cell surfaces, the remaining cytoplasm being relatively poorly provided with organelles indicating a low level of metabolic activity.

Normally small volumes of fluid are transferred across the peritoneal surfaces.

Therapeutically however, considerable volumes of fluid may be administered via intraperitoneal route.

Certain blood borne substances such as urea can be dialysed from the blood stream, when fluid is artificially circulated through peritoneal cavity.

Abdominal incisions and closure [21]:

A surgeon should give importance in choosing an incision, correct methods of making and closing the wounds in abdominal surgery.

When an incision or a closure of wounds is bad due to wrong decisions, it can lead to several complications like haematoma formation, incisional hernia or wound

dehiscence (worst complication of all).

Aim of any surgeon is to choose the incision precisely depending on the surgery he performs. The three essential principles which an incision should follow is that it should have good

- Accessibility
- Extensibilty
- Security

General principles of incisions:

An incision should provide adequate room for the procedure and it should also provide direct access to the anatomy to be examined.

An incision should be amenable to extend further if need arises. But it should not interfere with the function of abdominal wall in the future.

Closure of abdominal wall also plays an important role. It should make the abdominal wall as strong and intact as before.

The another most important principle is to maintain strict aseptic precautions which prevents contamination and infection.

The abdominal wall incisions :

The incisions used to explore abdominal cavity can be classified as

I. Ventral incisions

   A. Ventral midline incision

   B. Ventral paramedian incision

      1. rectus retracting paramedian incision

      2. rectus splitting paramedian incision

Location: supraumblical

Infraumblical

Both

II. *Transverse incisions:*

A. Supraumblical

B. Infraumblical

1. Maylard's incision

2. Pfannensteil incision

III. *Transverse oblique incisions*

Kocher's incision

Inverted V shaped incision

Mc Burney, incision

Rockey Davis incision

Lanz incision

IV. *Abdominothoracic incision*

V. *Alphabetical incision*

S incision of Bevan

J Czerny incision

Γ Bardenbever incision

 Sloan incision

Mixter incision

V *Sprengel incision*

Maingot's incision

Among these incision, except T incision all other incision are rarely used.

Description of incisions:

Midline incision: [23,24]

Midline incision are the most used incision because it can be used to get access both intraperitoneal and retroperitoneum. So universally accepted.

Advantages :

- It can be used for opening and closure quickly
- It is virtually bloodless
- It is easily extensible with a curve around umbilicus.

Midline abdominal incisions can be upper midline and lower midline with the reference to the umbilicus, which differs in anatomy. In upper midline, the incision extends from xiphoid process to immediately above the umbilicus. Abdomen opened in the layers in following order skin, fat, lineaalba, extraperitoneal fat and peritoneum. The peritoneum should be carefully divided just above the umbilicus, so that falciform ligament can be seen and injury can be avoided.

Where as in lower abdomen midline incision, the peritoneum should be opened in uppermost area of incision i.e. just below umbilicus to avoid injury to bladder.

Most important thing is in case of intestinal obstruction and re-operations, care to be taken while opening the peritoneum.

PARAMEDIAN INCISION: [24,25.]

ADVANTAGES :

- Better way of exposure to explore kidney and spleen.
- Since the rectus muscle acts as a buttress when posterior and anterior rectus plane approximated, the closures in these incisions are theoritically more secure.

Site of incision- 2-3 cms lateral to midline

After the incision, skin and subcutaneous fat are divided along the length of wound. This flap is raised medially to expose the underlying fascia. The anterior layer of rectus sheath is incised and rectus muscle to be separated from the sheath. The blood vessels which supplies the segment of must be secured and ligated.

Then the posterior rectus sheath can be easily separated since the rectus is freely mobile over the posterior rectus sheath. The latter and peritoneum are adhered to each other. So while taking incision over the posterior rectus sheath care should be taken to avoid injury to underlying bowel.

The inferior epigastric artery which encounter below umbilicus should be secured and ligated if they come in the line of incision.

The infraumblical paramedian incision is done in the similar pattern. But difference here is, the inferior epigastric artery will be exposed in posterior compartment of rectus sheath. Aponeurosis of transversalis merges with the anterior sheath below the semicircular line of Douglas.

PATHOGENESIS OF WOUND HEALING:[26-28]

Wound repair is the main area of interest of all the specialities of Surgery. So wound repair mechanism is considered as the foundation of surgery.

We surgeons, aim at maximising the human body's capacity to heal disrupted tissue with restoration of form and function.

Wound repair is one of the fundamental processes of life. The knowledge about the cellular and biochemical mechanism of wound healing has been completely understood. All this achievement is the results of studies done by individuals and their dedications in their life time who explored the fundamentals of wound healing. Knowledge about wound healing can be acquired by us by a continuous process of observation of practical aspects in our medical practice comparing the observation with the literature and finally applying the knowledge in getting better outcome.

According to our philosophy, wound healing is merely the function of new tissue and cells . So it is just an extension of morphogenesis. Also it can be termed as an awakening of cellular states that existed at an earlier tissue ontogenetically.

The reformation of tissue which are formed embryologically, by the process of cellular mitosis ,formation of intercellular substances and cellular migration allows the process of wound healing.

Wound healing is a complex phenomenon which involves various processes such as coagulation, inflammation, matrix synthesis and deposition, angiogenesis, fibroplasia, epithelialization, contraction, and remodeling.

Wound is said to be acute, when the healing proceeds in orderly and timely manner and achieves sustained anatomic and functional integrity.

Wound is said to be chronic, when it either does not proceeds in an orderly and timely fashion so ultimately it fails in achieving sustained anatomic and

functional integrity. Wound chronicity can be due to repeated trauma, ischaemia and secondary infection.[28]

HEALING BY PRIMARY INTENTION:

Wounds for example, laparotomy incisions heals by primary intension. Stages by which wound heals by primary intension are inflammatory phase, fibroplasia(proliferative phase), epithelialization and remodeling.

PHASES OF WOUND HEALING:

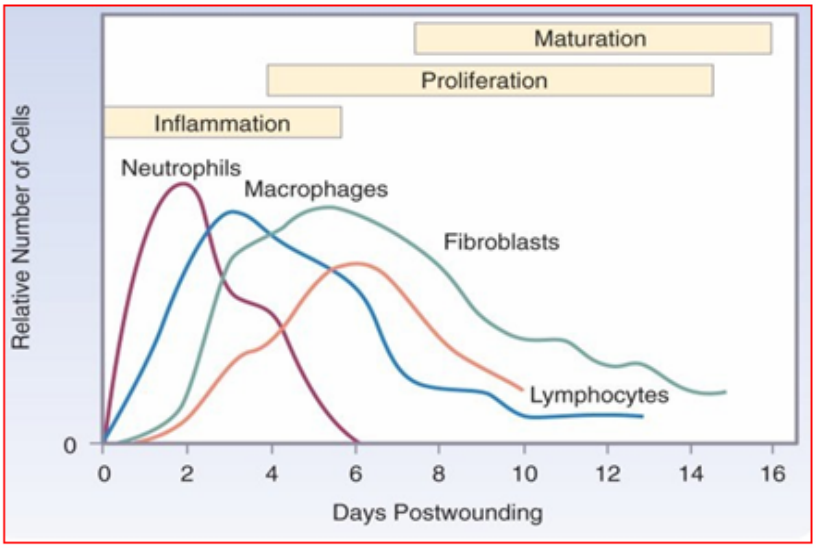


Fig 6. Phases of wound healing

INFLAMMATORY PHASE:

After a wound is formed, immediately there will be a phenomenon of vasoconstriction and followed by vasodilation. Dilated venules leaks serum, plasma, proteins and blood cells. There will be migration of polymorphonuclear cells, RBC and blood into the wound. Agglutination of wound involves formation of coagulum of fibrin which holds the wound edges together. In initial 1 to 2 days, PMN cells are the

predominant cells. PMN cells are replaced by monocytes and lymphocytes which become predominant by 3rd or 4th day .

The monocytes are transformed into macrophages engaged in phagocytosis. As we shall see, the macrophages are the dominant cells in early wound healing, directing the next phase (proliferative phase) and helping to terminate the inflammatory phase.

The main inflammatory phase normally lasts up to 5 days. During the inflammatory phase fibroblasts first appear at 24 to 48 hours showing that collagen synthesis and the laying down of proteoglycans is beginning. Some new blood vessels formation also occurs. During the inflammatory phase, the epidermis begins to thicken and migrate into the wound, eventually crossing the surgical defect. This migration takes place in the midst of the clot that has formed in the surgical defect and is directed in a downward fashion into the wound. In a well-sutured wound, epithelial migration may be complete as early as 24 hours.

PROLIFERATIVE PHASE:

Proliferative phase of wound healing is characterized by intense proliferation of fibroblasts and capillaries in its early phase. First the fibroblasts migration occurs along the fibrin strands which are oriented vertically. Later the capillary endothelium budding occurs in subcutaneous tissue and papillary dermis. The epithelium from each side continues to grow and eventually unites in the upper dermis. Mitosis occurs not within the epidermal tongues moving under the scab but peripherally around the wound. Thickening of epidermis occurs which continues to next phase.

In the later phase of proliferation, strength of wound increased by fibroblasts by laying down collagen. Collagen is laid down vertically at first, then as wound

tension increases, collagen becomes more horizontal. This is because of direction in which migration of fibroblasts and vascular proliferation occurs. It occurs from the papillary dermis above and subcutaneous tissue below.

At this stage of wound healing it will be of poor tensile strength because of lack of orientation and extensive linkage of collagen fibres. Fibroblasts also synthesis and secretes mucopolysacharides. Ultimately wound is filled with granulation tissue and collagen. Duration of this phase of maturation last for 4 days to 2 weeks and it represents a quantitative phase of collagen production. During the time after the scab is shed (usually 1 to 2 weeks), the surface of the wound appears as a depressed furrow. This gradually becomes level at about 3 weeks.

EARLY MATURATION PHASE:

Duration of this phase last for 2 weeks to 3 months. During this phase collagen remodeling occurs along the lines of tensile strength. Gradual gaining of tensile strength of the wound attains because of cross links and covalent bonds between collagen fibres.

LATE MATURATION PHASE:

In the late maturation phase the scar looses vascular network gradually over 6 months to a year and eventually redness of scar fades. As time advances the scar get remodeled itself by becoming white and flat. This is because the collagen which is laid down continuously and leading to hypertrophic scar. Now breakdown of collagen occurs which will be greater than synthesis which leads to flattening.

Other changes which occur in late maturation phase is appearance of elastic fibres (at about 2 months) and in growth of nerve fibres. Collagen which is laid down

is completely different from the normal skin. The strength of sutured abdominal wall depends on integrity of aponeurotic layer but not the muscle and peritoneum which can be easily tored off even with a light force. In the same way, strength does not depends on skin which can be readily stretched.

Once the aponeurotic layer gives way, then there will be progressive enlargement of gaping leading to hernia. So aponeurosis is an important structure which heals by approximately 2 months. Healing curve of wound flattens out. But the attainment of tensile strength by slow process continues even after cessation of healing curve.

Tensile strength of any wounds attains the strength of the normal skin very rarely. At the end of 2 weeks, the values were about 20% normal, at the end of one month 50%, 2 months 60%, and one year up to 90%. Occasionally some wounds showed the tensile strength greater than that of control tissue.

We here come to know that aponeurosis takes prolong period around 2 months to achieve tensile strength, when compared to skin which attain by 14-21 days.

ANATOMY AND FUNCTION OF TISSUE IS IMPORTANT TO UNDERSTAND THE HEALING.

Because of its rich blood supply dermis has capacity to grow rapidly, whereas aponeurotic layer which is relatively avascular has poor capacity to heal, but it helps in supporting viscera.

In conditions causing ischeamia dermis loses its capacity to heal leading to chronic ulcer. Whereas aponeurosis has poor capacity to heal. Usually not subjected to injury. Once injured it will regenerate slowly. Wound remains weaker than parent tissue even at the end of 1 year, in large majority of cases. And this is due to infection of wound in aponeurosis, which leave weakness permanently.

PERITONEAL HEALING: [29,30]

Peritoneal healing is very rapid, which occurs within 3-5 days. And the healed portion of the defect no matter how large the defect will be indistinguishable physically from the normal peritoneum. The defects of peritoneum heals rapidly and covering of which with a polythene shield will retard the restoration of continuity. So this shows that, the restoration is mainly due to covering of defects by desquamated or shed mesothelial cells from visceral or parietal peritoneum.

Some of the other factors which contribute to peritoneal defects closure are centripetal activity and stem cells at the base of wound or defect contributing to remesothelialization process. Ascorbic acid depletion significantly retards the process, suggesting that fibroblasts may be actively involved. Wound contracture is not a problem in peritoneal healing because even large area have a capacity to heal rapidly provided that, the mechanical interference should not retard the normal healing process.

One of the certain way to interfere healing of peritoneum is by inserting foreign materials like sutures, drains, retained cotton from sponges and gauze. Sutures stimulate the formation of bands known to cause intraperitoneal adhesions. Peritoneum has a capacity to regenerate by itself. So this mechanism helps in rapid healing of defects. But in other tissues(skin) healing by regeneration occurs from edges. So suturing a defect in peritoneum specifically when operating on gall bladder(cholecystectomy) or APR (antero-peritoneal resection) in pelvis leads to formation of intraperitoneal adhesions.

FACTORS INFLUENCING WOUND HEALING:[31,32,33]

ADVERSE FACTORS

GENERAL FACTORS	***LOCAL FACTORS***
Age	Tissue tension
Malnutrition	Hematoma formation
Vitamin deficiency	Necrotic tissue
Trace metal deficiency	Local infections
Anaemia	Foreign body
Malignant disease	Poor blood supply due to
	Vascular disease
Uraemia	Trauma
Jaundice	Faulty technique of wound closure
Diabetes	Recurrent trauma
General infections	
Cytotoxic drugs and steroids	Local irradiation

GENERAL FACTORS:

AGE:

The rate of healing generally declines with aging. This is due to reduction of protein turnover, qualitative and quantitative changes in collagen and decreased response to tissue hypoxia.

Elderly patient have more chances of delayed wound healing and even ruptures. This is due to decrease in dermal collagen content and its distorted organization. Also aging shows increase collagenolytic activity.

MALNUTRITION :

Malnutrition with severe form causes failure of wound to heal. While mild malnutrition will lead to delayed healing. Albumin should be <2.0g to affect wound healing. Vit C requirement is essential for collagen maturation. Vitamin A is essential for epithelialisation. Calcium, manganese and other trace elements also play some role. Zinc is a necessary cofactor for RNA polymerase and DNA polymerase, deficiency results in early wound healing delay.

Vitamin K deficiency limits the synthesis of prothrombin and factors VII, IX and X. Hypoxic ischemia can be caused by atherosclerosis, cardiac failure or simple wound tension preventing localized perfusion. Molecular oxygen is critical for the post-translational hydroxylation of prolyl and lysyl residues required for triple-helix formation and crosslinking of collagen fibrils.

URAEMIA:

Urea delays the wound healing by inhibiting the growth of fibroblasts

JAUNDICE :

Jaundice affects the wound healing indirectly by delaying the appearance of fibroblasts and formation of new blood vessels. Jaundiced patient have reduced levels of prolylhydroxylase enzyme which is involved in collagen synthesis ultimately leading to decreased wound strength.

STEROIDS :

Steroids are anti-inflammatory agents, which affect the wound healing by decreasing the inflammatory response which is essential for a wound to heal. So steroids should be used once the inflammatory phase is over.

RADIATION AND CYTOTOXIC DRUGS :

Radiotherapy and cytotoxic agents delays wound healing when administered in either form systemic or local application. These causes cell death by both damaging DNA and disruption of intracellular metabolism. Cytotoxic drugs show their greatest effect on mitotic cells.

MALIGNANCY :

Cachexia and malnutrition associated with malignancy are the main factors for delayed wound healing.

Diabetes :

Diabetes mellitus impairs wound healing. The diabetic patient with associated neuropathy and atherosclerosis is prone to tissue ischemia, repetitive trauma, and infection. Lymphocyte and leukocyte function is impaired and there is increased collagen degradation and decreased collagen deposition.

LOCAL FACTORS:

TENSION :

Tension in the post-operative wound due to various factors like tight closure, due to local edema and haematoma formation is major factor for wound infection. The reason behind this is, the tension inhibits the local blood supply and ultimately leading to wound failure.

INFECTION :

Infection affects by interfering with epithelialization, wound contraction and collagen deposition. Infection affects the collagen deposition by competing with the fibroblasts for oxygen and nutrients, thus ultimately leading to defective collagen and its degradation by increased collagenolytic enzyme activity.

CAUSES OF FAILURE IN HEALING OF ACUTE WOUNDS:

We, as surgeons, must be more heroic as the tragedy is much our fault.

- J.W. Kennedy, 1934.4

This is perhaps a universal experience of one who performs laparotomy and seems to illustrate the most common cause of wound disruption. Acute wound failure can be attributed to four main causes that is suture break, slipped knot, a loose or excessive stitch interval and suture tearing through the fascia. Most common cause of wound dehiscence is suture tearing through the fascia. This is due to inadequate tissue strength. So patient related factors are also held responsible.

Wound healing can also attributed to method of taking bite that is greater tissue strength can be obtained by taking wider bite of fascia. So tissue tearing is the most common cause of wound dehiscence.

RISK FACTORS:

OPENING THE ABDOMEN

INCISION TYPE:

Wound healing also depends on type of wound incision. Wound dehiscence is noted higher in midline incision compared to transverse incision. This is because midline incision are non-anatomic based incisions that it cuts across the aponeurotic

fibres, but transverse incision cut parallel to fibres. In case of midline incisions, contraction of abdominal wall muscles will rip away the edges. But in transverse incisions edges will be brought closer on contraction. Sutures in midline tear out the tissue more easily than in transverse incision because ripping in former occurs parallel to the fibres.

INCISION LOCATION:

Upper abdominal incision has higher chances of getting dehiscence compared to incision in the lower abdominal incision.

INCISION OPENING:

When midline incisions are made with cautery the wound strength reduces by one third compared to incisions made by scalpel. A coagulation current is short bursts of electrical energy that allow the contacted and surrounding tissues to heat, dehydrate and denature. So current type of cutting is wise to avoid.

SIZE OF TISSUE BITE AND SUTURE LENGTH-TO-WOUND LENGTH RATIO:

(Suture length : wound length) SL:WL should be 4:1 to avoid the wound dehiscence. So surgeon should perform running mass closure with wide bites of fascia(2cm or more) and a short stitch interval to avoid tissue tear and wound dehiscence.

TENSION ON THE SUTURE:

Tension after closure with proper wide bites is also important. Because more the tension, more will be the raise in the interstitial pressure in the centre of incision above the capillary pressure(30-40mm of hg) which hampers the blood supply causing necrosis.

SUTURE:

Catgut is associated with increased incidence of wound dehiscence of 10-12%

Delayed absorbable suture material (polydiaxonone and polyglyconate) are the strongest suture available. Followed by the non absorbable monofilament sutures (nylon and polypropylene). Then comes the braided suture (polyglactin, polyglycolic acid and polyester). The weakest suture material is silk which has dehiscence rate of 7.5%.

A small controversy surrounding suture choice involves absorbable versus non-absorbable suture. Randomized trials have not found a difference in dehiscence rates between absorbable and non-absorbable sutures. It may be wise however to use an absorbable monofilament in the patient who has an excessive number of risk factors for delayed healing as the wound infection rates will become less.[34]

PERITONEAL CLOSURE:

Initially peritoneum closure was thought mandatory because surgeons thought that wound disruption can be due to insinuation of omentum or bowel. Randomized trials comparing one-layer closure (peritoneum not sutured) with two-layer closure (peritoneum closed) in paramedian and midline incisions found no difference in the wound disruption rate. Suturing the peritoneum is not a necessity to prevent wound dehiscence.

MASS VERSUS LAYERED CLOSURE:

Mass closure is enmass closure of all the layers of abdomen which has less chances of wound dehiscence. Mass closure appears to be favored currently because of its safety, efficacy and speed.

INTERRUPTED VERSUS RUNNING CLOSURE:

Running closure has reduced incidence of burst abdomen compared to interrupted closure (1.6% versus 2.0%) in a multicenter randomized trial of 3135 midline incisions closed with polyglycolic acid. Smaller randomized trials comparing running with interrupted closure also reveal no difference in the incidence of wound disruption. Running suture is a reasonable closure technique because of its safety, efficacy and speed.[35]

KNOT SECURITY:

Knot plays a important role in preventing wound dehiscence. So knot should be secured properly by performing more throws. Also double throws are more secure and square knots are more secure than non-square knots. Knots lose some strength after 7 days invivo.

RETENTION SUTURES:

Retention suture are also known as tension sutures. Data from previous studies, both controlled and uncontrolled data still have a conflict. Retention sutures are not recommended today routinely. It is proved that placement of retention sutures is more secure than running a mass closure with wide bites (2- 3 cm).

AGE:

Aged patients (50-55) out number in the incidence for wound dehiscence when compared to young patients. Age cannot be attributed alone as a risk factor, because aged patients will have associated comorbid conditions.

EMERGENCY OPERATION:

Operations which are done on an emergency basis, is also a risk factor for causing wound dehiscence. This is due to hemodynamic instability associated with most of the patients who present to us in emergency department. Other factors causing the wound disruption are infection, malnutrition, uraemia and hypoproteinemia.

OBESITY:

Obesity contributes as a risk factor for wound dehiscence. According to recent case control study report, obesity (defined as greater than 50% above ideal body weight) is considered as a risk factor.

DIABETES:

Diabetes is the major co-morbid condition which retards wound healing and leading to wound dehiscence. In pre insulin era, wounds healing in diabetes patients was very poor. Now the rampant use of insulin has increased the wound healing in diabetic patients and so ultimately there will be less chances of wound dehiscence .

Renal Failure:

Acute renal failure is another important risk factor for wound dehiscence. This is attributed to uraemia resulting from renal failure which increases the risk for burst abdomen.

JAUNDICE:

Hyperbilirubinemia has an inhibitory effect on fibroplasia and angiogenesis. So these patients usually malnourished and wound healing is delayed in such patients.

ANAEMIA:

Hypovolemia and shock,but not acute anemia alone, reduce wound strength.

Chronic anemia secondary to iron deficiency may result in decreased wound strength, but the data are conflicting. Low hemoglobin has been found to be a risk factor in some case control studies, but not in others.

The animal data suggest that iron deficiency anemia associated with severe malnutrition and hemorrhagic anemia with shock may predispose a patient to wound dehiscence.

MALNUTRITION:

PROTEIN DEFICIENCY:

Hypoalbuminemia is one of the malnutrition status may be considered as a causative factor for wound dehiscence. The recommended daily allowance for protein in normal adult men and women is about 1 gm/kg/day. This RDA should be increased to 1.5 to 2.0gm/kg/day with severe sepsis and burns.

VITAMIN C DEFICIENCY:

Ascorbic acid (VIT- C) plays an important role in wound healing process and also in providing wound strength. Routine vitamin C supplementation in malnourished (if not all surgical patients) seems to be useful in making the wound heal faster. The recommended dosage is controversial but ranges from 30 to 75 mg/day.

ZINC DEFICIENCY:

Zinc is another trace essential mineral which is required in wound healing process. It acts as a co-factor for more than 300 enzymes. Wound when it heals sequester zinc. So it leads an impaired wound healing in zinc deficiency states.

Zinc deficiency occur in case of injury due to burns, alchoholism, GI fistula and parenteral nutrition which is deficient in zinc. The recommended allowance of zinc is 12 to 15 mg/day.

POSTOPERATIVE EVALUATION OF INTRA-ABDOMINAL PRESSURE:

Increased intra-abdominal pressure is the pressure which builds up with in abdominal cavity. The reasons accounts for this are postoperative coughing, vomiting and abdominal distension due to various reasons. Several studies shows that increase intra-abdominal pressure acts as an instigator, if not the source of dehiscence. Usually wound will not undergo dehiscence, if properly sutured even in condition of increased intra-abdominal pressure.

WOUND INFECTION:

Wound dehiscence can also occur due to the post operative wound infection. But in present scenario, wound dehiscence will occur even before the wound get infected. So infection cannot be held responsible for dehiscence.

CORTICOSTEROIDS:

Corticosteroids has an affect on wound healing by its anti-inflammatory action. So there will be delayed wound healing and also prevents the wound from gaining the strength. This negative effect can be overcome by adding VIT-A along with corticosteroids. The recommended dose of vitamin A is about 1 mg/day.

ANTINEOPLASTIC AGENTS:

Similarly anti-neoplastic agents has inhibitory effects on wound healing by retarding the fibroplasia and collagen synthesis. So cytotoxic drugs should be withhold until the wound completes acute phase of healing (usually 2 to 3 weeks).

TREATMENT OF WOUND DEHISCENCE:

NON-OPERATIVE TREATMENT:

Wound dehiscence can be managed temporarily by non-operatively by packing with gauze or applying a binder there by protecting viscera. Wound may spontaneously get closed by contraction or it should be closed secondarily by operative procedure. Hernia is the common sequelae to this treatment.

OPERATIVE TREATMENT:

RETENTION SUTURES:-

Operative technique usually followed for wound dehiscence is applying a tension suture (retention suture). Technique of applying are external type (peritoneum including skin) and internal type (all layers except skin). Method usually followed will be placing stitch at an interval of 3 cm or less and a buttress device is placed which is used to avoid suture erosion into the skin. Suture material used will be non-

absorbable monofilament suture. Suture removal can be done after 3 weeks post-operative period or more.

REPAIR WITH PROSTHETIC MATERIALS:

Abdominal wall closure in a case of acute loss of abdominal wall always leads to dehiscence more than 50% of cases. So non-elective mesh closure is recommended in above scenario and even in case of massive visceral edema.

Non-elective mesh closure has some advantages and disadvantages. Advantages is that it avoids an another complicated closure in later date due to tissue tearing. Disadvantages is that chances of faecal fistula, mesh extrusion and hernia can occur upto incidence of 50%.

These complications can be avoided by using PTFE mesh which is an absorbable material used in abdominal wall closure and reconstruction. By using PTFE even in presence of contamination and peritonitis, there will be less bacterial adherence compared to use of polypropylene mesh. Absorbable mesh will have less chances of recurrent dehiscence. Enterocutanous fistula occur even with absorbable mesh but the management is easier when compared to fistula occurring in non-absorbable mesh.

SEQUELAE:

Mortality:

Average mortality rate for abdominal dehiscence from recent reviews is 25%. The dehiscence-associated morality rate does not appear to be declining.

Cardiorespiratory failure is the most common cause of death. Peritonitis is the second most common cause.

Advanced age (not quantified), female sex, and post disruption mechanical ventilation were risk factors for death after dehiscence occurred.

OTHER POSTOPERATIVE COMPLICATIONS:

Other surgical complications after wound disruption repair that have been described in a large series include wound infection (14%), fistula (6%) and intra-abdominal abscess (4%).

MATERIALS AND METHODS

Source of data:

All patients admitted in department of surgery, R.L.Jalappa Hospital and Research Centre, Tamaka, Kolar, who undergo laparotomy operations, with midline abdominal incisions. This include emergency and elective procedures.

The study period will be between JAN 2013 to JAN 2014.

Method of collection of data :

By taking into account, the variables like suture sinus formation and wound granuloma, as estimated in previous studies, sample size of 120 cases will be allotted in each group, a total of 240 patients will be included in the study[36].

These patient are divided into group "A" and group "B" by giving odd and even numbers respectively.

Group A include the patients with odd numbers in whom abdominal incisions are closed with non-absorbable suture material polypropylene.

Group B include the patients with even numbers in whom abdominal incisions are closed with absorbable suture material polydioxonone.

Detailed history of patient, investigations done, nature of operation performed will be noted down in the standard study proforma.

In emergency operations, like peritonitis fluid from peritoneal cavity will be collected for culture and sensitivity. Appropriate antibiotics will be administered after obtaining culture and sensitivity reports.

Wound will be inspected in immediate post operative period for evidence of

infection. Discharge if any will be sent for culture and sensitivity.

Post operative pain will be recorded by using visual analog scale.[37]

Subsequently patient will be followed up regularly at intervals of 2 weeks, 4 weeks and once in 3 months upto 1 year.

During subsequent follow up period wound pain, wound infection, wound dehiscence, suture sinus formation, stitch granuloma and incisional hernia will be inspected and recorded.

Follow up period will be for one year.

Statistical analysis will be done with Mean standard deviation and proportion. Comparison of significance of difference in proportion by "chi-square test" uni-variant analysis by using confident interval and odd's ratio.

Formula $n=[Z1-\alpha\sqrt{2P(1-P)} + Z\ 1-\beta\sqrt{\{\ P1(1-P1) + P2(1-P2)\}}]2\ /\ (\ P1-P2)2$

$Z1-\alpha=1.96$ $Z1-\beta=0.842$ power =80%

$P=P1+\ P2/2$ $\alpha=0.05$

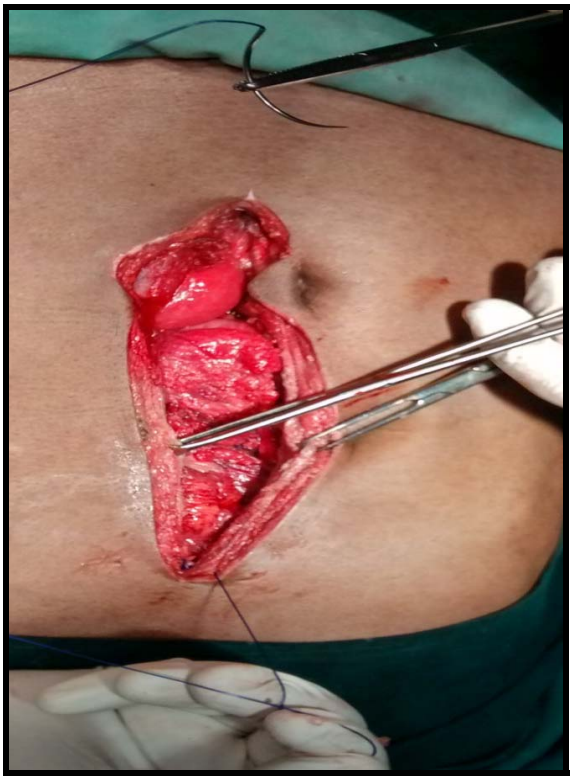
INCLUSION CRITERIA:

- All patients who undergo laparotomy operations by midline abdominal incisions with a continuous type of mass closure.

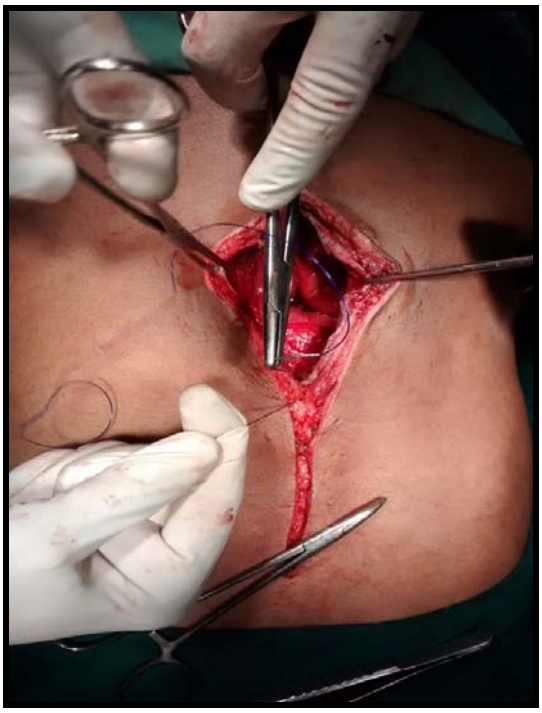
EXCLUSION CRITERIA:

- Patients who have already undergone operations with midline abdominal incisions.
- Patients who require closure of abdominal wall with tension sutures.
- Patients with malignant ascites.

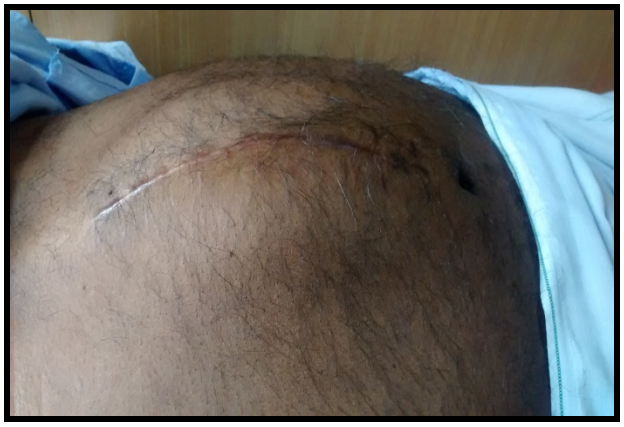
INTRAOPERATIVE AND POST-OPERATIVE FIGURES:



PDS SUTURE MATERIAL IN MIDLINE CLOSURE



POLYPROPYLENE IN MIDLINE CLOSURE.



INCISIONAL HERNIA AND PALPAPABLE KNOTS IN A PATIENT CLOSED THE LAPAROTOMY WOUND WITH POLYPROPYLENE

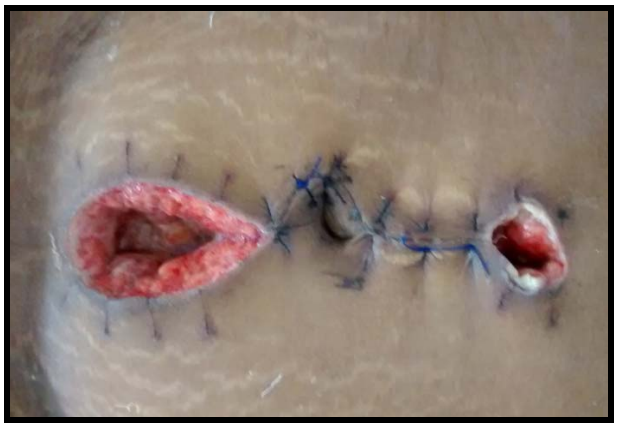


FIGURE:WOUND INFECTION OBSERVED IN CASE OF POLYPROPYLENE SUTURE MATERIAL.

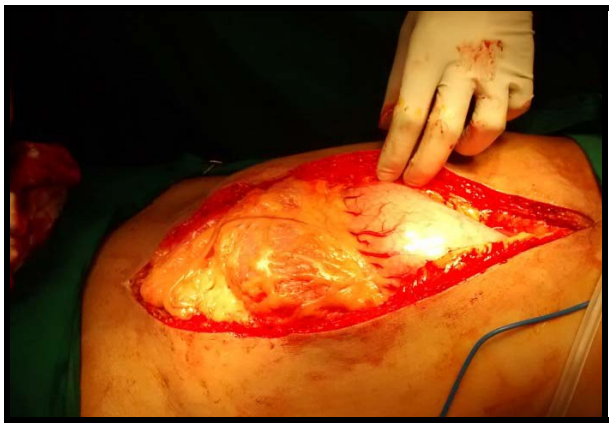


FIGURE: SHOWING ABDOMINAL WOUND DEHISCENCE IN A PATIENT WITH POLYPROPYLENE SUTURE MATERIAL.

RESULTS

Study design: A Comparative observational clinical study

Table 1: Gender distribution of patients studied

Gender	Prolene	PDS	Total
Female	21	19	17.2
Male	79	81	82.8
Total	**100**	**100**	**100**

Gender distribution is statistically similar in two groups with 0.799

Graph 1: Gender distribution

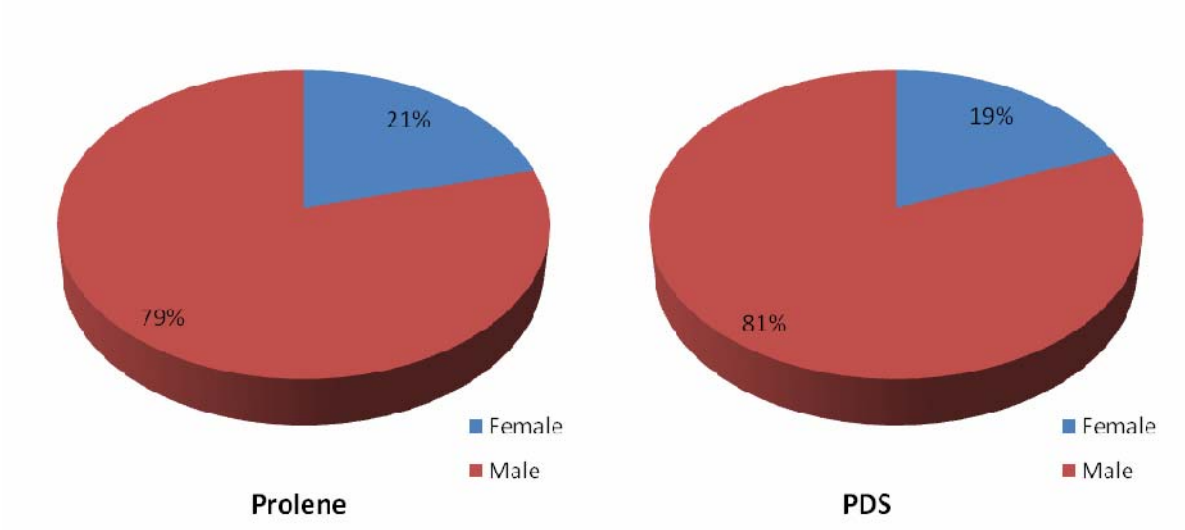


Table 2: Age distribution of patients studied

Age in years	PDS	Prolene	Total
<10	0	1	1
11-20	11	11	22
21-30	22	19	41
31-40	17	22	39
41-50	20	16	36
51-60	12	18	30
61-70	12	8	20
>70	6	5	11
Total	**100**	**100**	**200**
Mean ± SD	**43.10±18.2**	**41.77±17.29**	**42.44±17.75**

Age distribution is statistically similar in two groups with P=0.506

Graph 2: Age distribution of patients studied

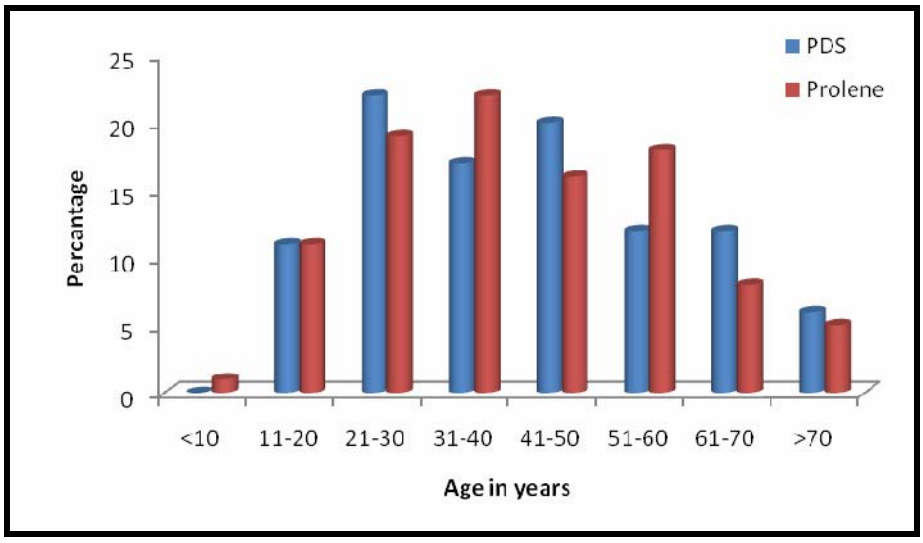


Table 3: Emergency Elective

Emergency elective	PDS	Prolene	Total
Elective	7	11	18
Emergency	93	89	182
Total	**100**	**100**	**200**

Distribution emergency/Elective is statistically similar with P=1.02

Graph 3: Emergency Elective

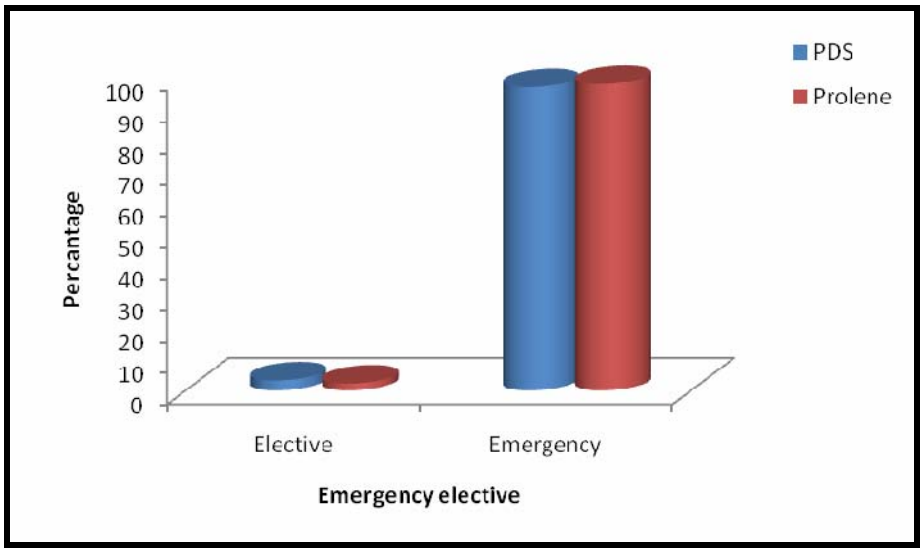


Table 4: Clinical diagnosis

Clinical diagnosis	PDS	Prolene	Total
PERF	56	61	117
AIO	15	16	31
BLUNT	12	6	18
PENETRATING	4	3	7
ABSCESS	2	3	5
CA.STOMACH	3	1	4
CA Colon	1	2	3
T SPLENOMEGALY	1	1	2
INTESSUCEPTION	0	2	2
R.V.F	1	0	1
PID	1	0	1
SAIO	1	0	1
R TUMOUR	1	0	1
CH.PANCREATITIS	1	0	1
A.PHLEGMON	0	1	1
SAIO	0	1	1
STAB INJURY	0	1	1
CHRONIC ULCER	0	1	1

Graph 4: Clinical diagnosis

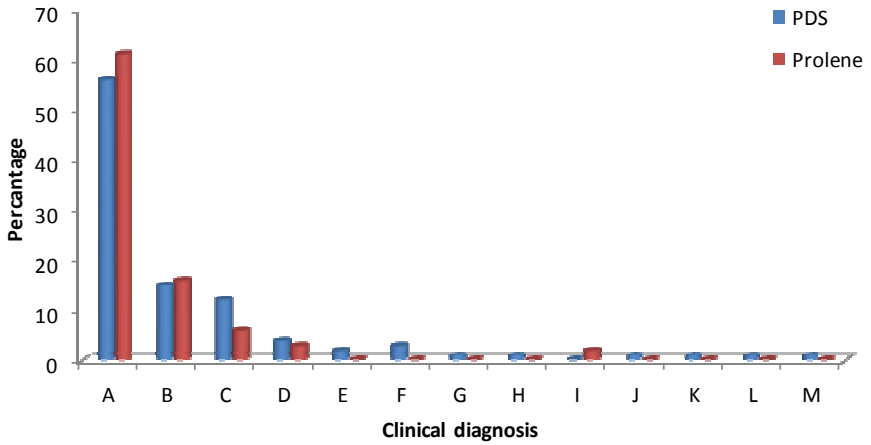


Table 5: Investigations Peritoneal Aspirate

Investigations Peritoneal Aspirate	PDS	Prolene	Total	P value
No growth	84(84%)	81(81%)	165(82.5%)	0.26
E.coli	9(9%)	18(18%)	27(13.5%)	0.182
Klebsiella	1(1%)	0(0%)	1(0.005%)	1.000
N flora	6(6%)	1(1%)	7(3.5%)	1.012
Total	**100(100%)**	**100(100%)**	**200(100%)**	**-**

Graph 5: Investigations Peritoneal Aspirate

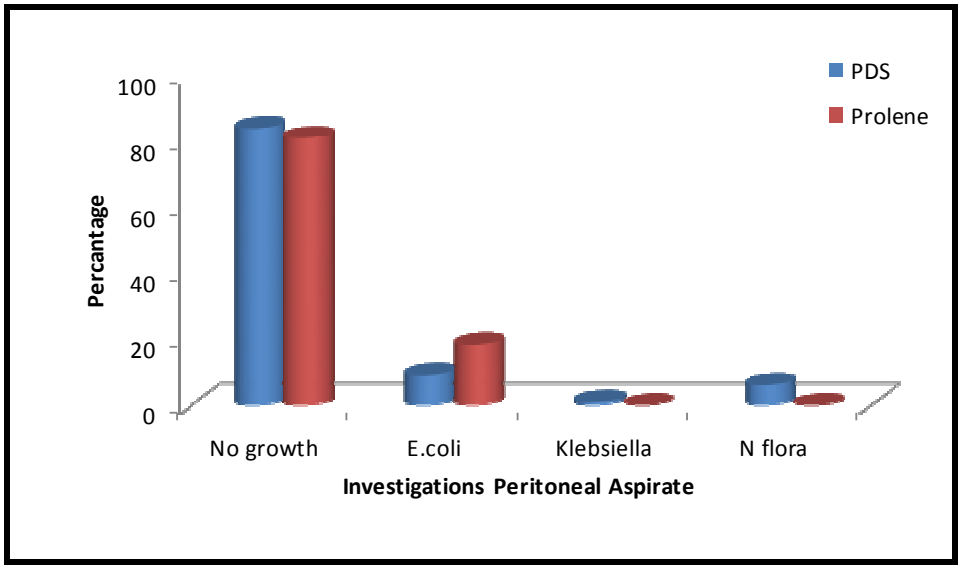
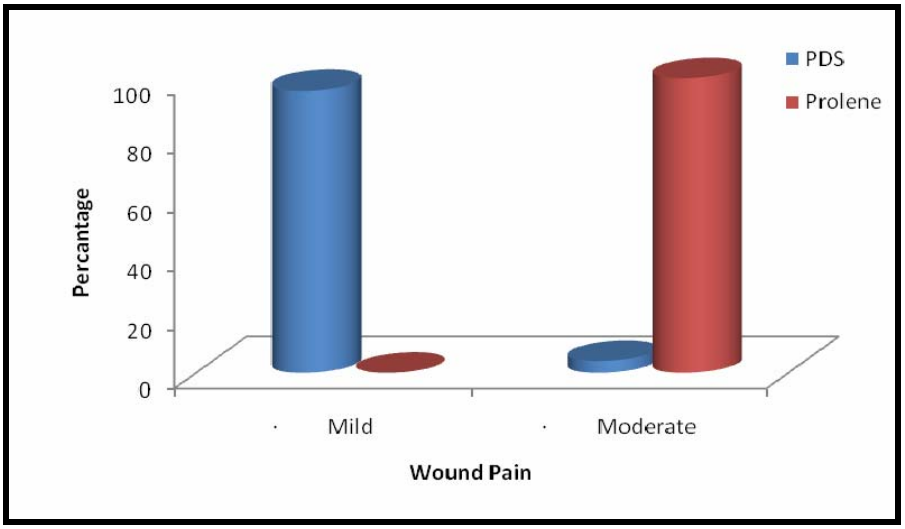
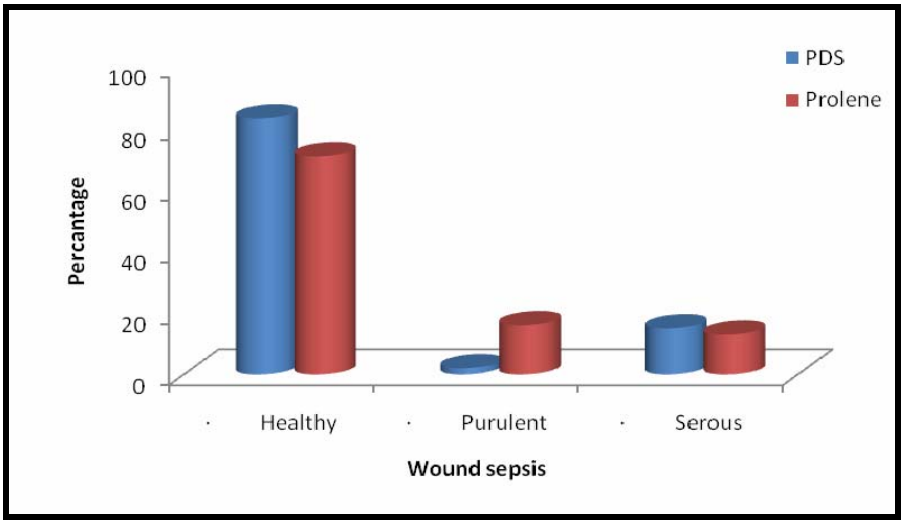


Table 6: Immediate Post Op

Immediate Post Op	PDS (n=100)	Prolene (n=100)	Total (n=200)	P value
Wound Pain				
Mild	96	0	96	**<0.001****
Moderate	4	100	104	
Wound sepsis				
Healthy	83	71	154	**<0.001****
Purulent	2	16	18	
Serous	15	13	28	
Wound Discharge c/s				
Not sent	97	76	173	**<0.001****
Gram +	0	7	7	**0.012***
Klebsiella	1	0	1	**1.000**
MSSA	1	5	6	**0.111**
Pseudomonas	0	2	2	**0.489**
Skin flora	0	6	6	**0.013***
No growth	0	3	3	**0.372**
Streptococci	0	1	1	**0.489**
Acenetobacter	1	0	1	**1.000**

Graph 6: Immediate Post Op





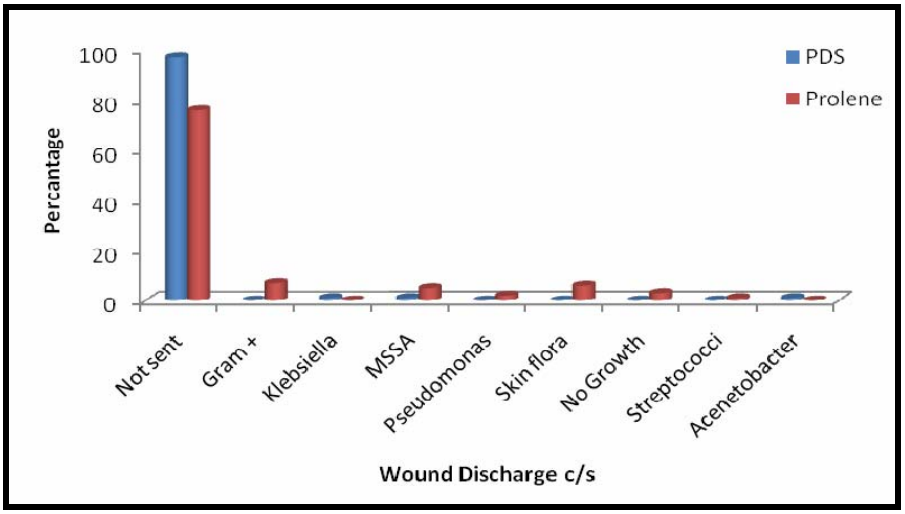


Table 7: Affect of type of operation(emergency/elective) on wound infection:

Type of operation	Wound infection (purulent discharge)	
	PDS	Polypropylene
Emergency	1	8
Elective	1	6
Total	2	14

Graph 7: Affect of type of operation (emergency/elective) on wound infection:

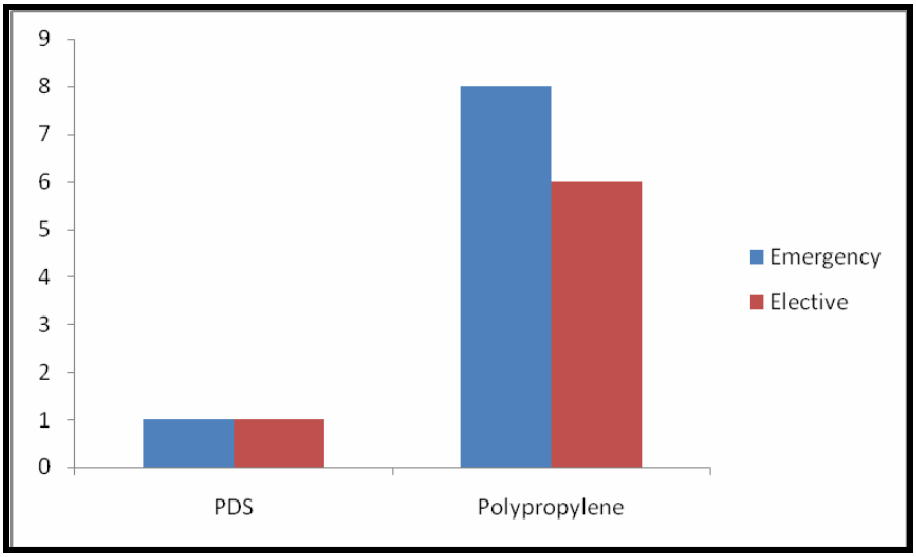
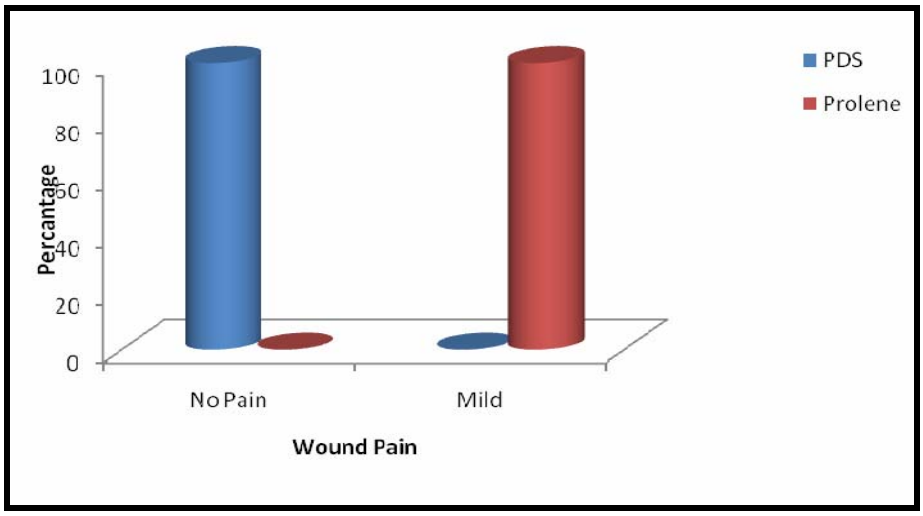
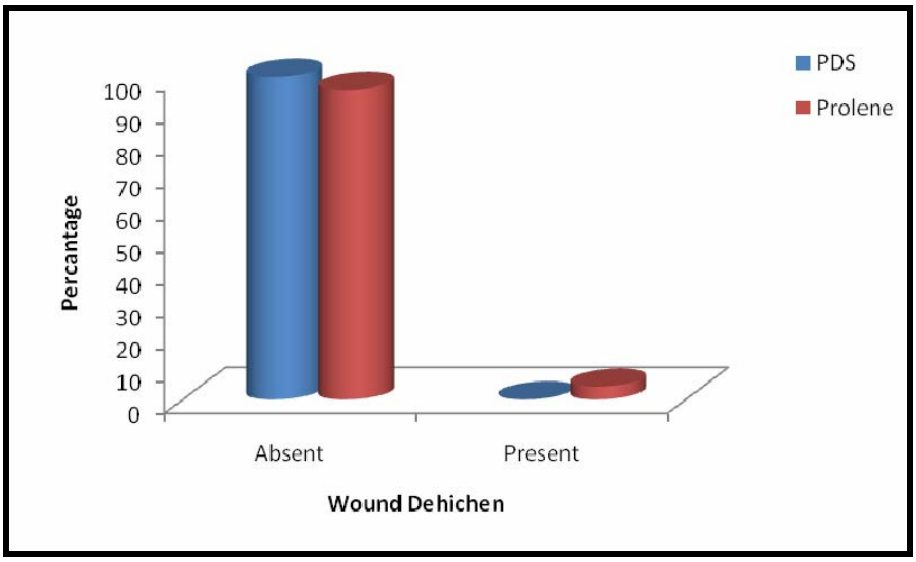


Table 8: Late Post Op findings

Late Post Op	PDS	Prolene	Total	P value
Wound Pain				
No Pain	100(100%)	0(0%)	100 (100%)	**<0.001****
Mild	0(0%)	100(100%)	100 (100%)	
Wound Dehiscence				
Absent	100(100%)	96(96%)	196 (98%)	**0.052**
Present	0(0%)	4(4%)	4(2%)	
Suture Sinus Formation				
Absent	98(98%)	91(91%)	189(94.5%)	**0.091**
Present	2 (2%)	9 (9%)	11 (11%)	

Graph 8: Late Post Op findings




PDS
Prolene
100
90
80
70
60
50
40
30
20
10
0
Percantage
Absent
Present
Wound Dehichen

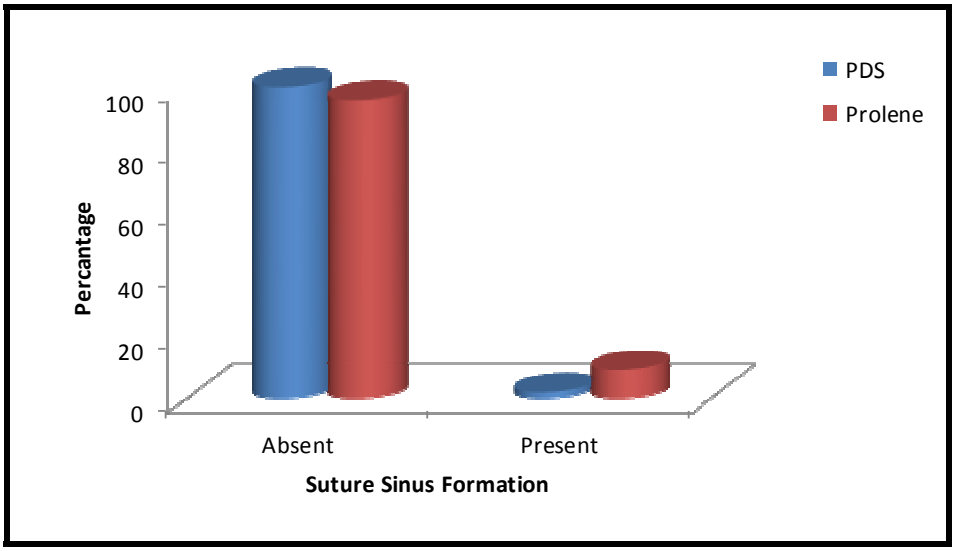

PDS
Prolene
100
80
60
40
20
0
Percantage
Absent
Present
Suture Sinus Formation

Table 9: Palpable Knots

Palpable Knots	PDS	Prolene	Total
Absent	100(100%)	77(77%)	177(88.5%)
Present	0(0%)	23 (23%)	23(11.5%)
Total	**100(100%)**	**100(100%)**	**200(100%)**

Palpable knots is significantly more in Prolene with P<0.001**

Graph 9: Palpable Knots

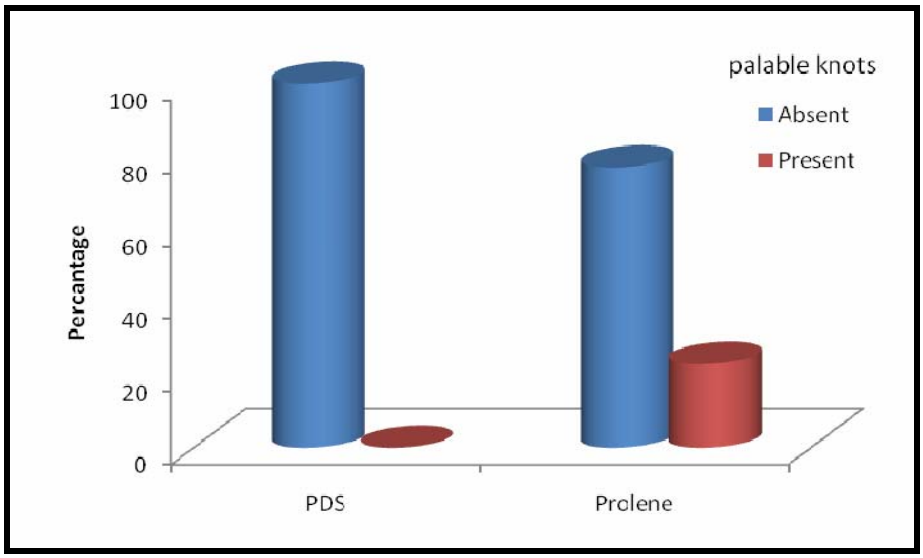
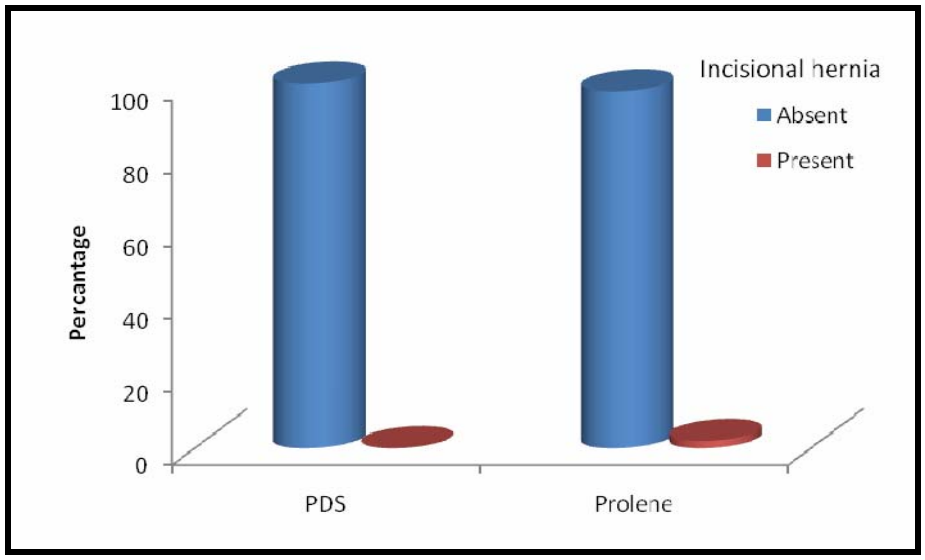


Table 10: Incisional hernia

Incisional hernia	PDS	Prolene	Total
Absent	100(100%)	98(98%)	198(99%)
Present	0(0%)	2(2%)	2(1%)
Total	**100(100%)**	**100(100%)**	**200(100%)**

Incidence of Incisional hernia is not statistically significant with P=0.232

Graph 10: Incisional hernia



DISCUSSION

Lord Moynihan has said *"Never judge a surgeon unless you have seen him close the wound".*

"I dressed the wound and God healed them" is the famous saying of Ambrose Parre (1510-1590) it could mean, "I suture the wounds and God heals them". All surgeons wish that the wounds which he closes should heal without any complications.

Abdominal surgeries are the most commonly done procedures. There by incision and closure(suturing) of abdominal wall is one of the commonest exercises in surgery. There are number of techniques of closure of abdominal wall with its own advantages and disadvantages.[35]

Regarding the healing of abdominal wound however meticulously closed, the healing takes place en-mass, even when closures done in layered technique because of formation of dense fibrous block of tissue. This is evident from the scar of previous surgeries when it is opened.

While suturing, bites should be taken at a minimum distance of 1 cm from wound edge and the interval between two sutures should be 1 cm. The material we take for suturing should be four times the wound length.[38,39,40,41]

There are many factors which delay the wound healing both systemic and local factors. Inspite of improved surgical skills, the morbidity associated with abdominal wound is still high. So surgeons give maximum importance for the wound closure and care.[15,42,43]. Systemic factors include obesity, jaundice, diabetes, malnutrition, protein deficiency, elderly patients, patients on steroids and immunosupressants. Local factors which delay wound healing after laparotomy are wound infection, hematoma, foreign body reaction. All these impose stress on the

freshly sutured abdominal wound. [15]

200 patients undergoing laparotomy were included in this study. 100 cases are grouped under group A, in whom abdominal wound were closed en-mass with polypropylene and 100 cases are grouped under group B, in whom abdominal wound were closed with polydioxanone.

In both, closure of abdominal wound, done in a continuous en-mass. Polydiaxanone sutures are strong, delayed absorbable, retain their strength after implantation, are inert, cause minimal tissue reaction and technically has a better handling during the closure. The only disadvantage is their slipping quality in handling and in tying. This can be overcome by using minimum five knots. [44]

Wound complications:-

1. Wound pain
2. Wound infection
3. Wound dehiscence
4. Suture sinus formation
5. Palpable knots
6. Incisional hernia

Wound pain:

Wound pain is the subjective feeling of pain in the postoperative wound site. Wound pain is graded according to the visual analogue scoring. Wound pain depends on the suture material. If suture material stays for a prolonged period without getting absorbed, it itself is a factor for wound pain. Because it causes irritation and causes pain.

Polypropylene which is a nonabsorbable suture material is more irritant to the

tissue and causes moderate wound pain.

Polydiaxanone which is a delayed absorbable suture material which is less irritant to the tissue causes mild wound pain.

In our study sample involving 200 cases 100 cases were studied under group A, we observed that all the patients(out of 100) experienced moderate degree of wound pain in the immediate post-operative period. Even in the delayed post-operative period all the patients continued to experience mild degree of wound pain and required analgesics for a prolonged period. In group B involving 100 patients pain was of mild degree in 96 % of cases and moderate in 4% of cases in the immediate post-operative period and none of the patients experienced wound pain in the delayed post-operative period hence required analgesics for a lesser duration. In both the groups, the patients were treated with same group of analgesics.

In a similar study conducted wound pain was observed among 9 out of 34 patients in the polypropylene group and 1 out of 30 in the polydioxonone group. As observed in this study polydioxonone has lesser incidence of wound pain as compared to polypropylene which was a similar finding in our study as well.[45]

Wound Infection:

Wound infection is said to be present, when there is seropurulent discharge from the post-operative wound site. Superficial wound infection is the infection confining to superficial layers of the abdomen like skin and subcutaneous tissue. Deep wound infection is the infection confined to deeper layers of abdomen, Linea alba and the peritoneum. In both cases, finding of seropurulent discharge from a stitch or from the incision, with signs of inflammation, with or without constitutional symptoms are present.[38, 40,47]

In our study we observed wound infection(purulent discharge) among 16 patients (out of 100) in group A (16%) and among 2 patients(out of 100) in group B(2%). Also group A patients had a maximum infection rate even in emergency laparotomy as compared to group B.

In a study conducted it showed wound infection occurred amongst 6 cases out of 143 in the polypropylene group and 5 among 141 patients had wound infection in the polydiaxonone group. [45]

In comparison to this study our study showed that polydiaxonone sutures had a lesser incidence of wound infection.

From above discussion it is evident that chances of wound infection in both emergency and elective operations is observed to be higher in Polypropylene (PPL) suture material compared to Polydiaxanone (PDS) and thus Polydiaxanone suture is considered preferable in emergency & elective surgery.

Wound dehiscence:

Wound dehiscence is also termed as burst abdomen, which is one of the serious complications in immediate post-operative period. It can occur in either sex at any age. It follows any of the abdominal operations.

It can be complete or partial. Partial wound dehiscence is said when skin and peritoneum are intact with disruptions of all layers of abdominal wall, whereas complete wound dehiscence is said when all layers of abdominal wall are disrupted which exposes the viscera.[40] Burst abdomen can occur any day during 7-10th post-operative day. It is clinically diagnosed, when a pink colour discharge from the suture line can be seen. when clinically inspected, all layers of abdominal wall may give way all of a sudden and may or may not lead to the evisceration.

In the first four days of wound healing, wound will not be having any intrinsic strength. So proper suturing technique should be followed and a proper selection of suture material should be done. So an ideal suture material is one which obeys the properties like providing adequate tensile strength, approximate tissue in such a way that wound healing takes place under optimum conditions and remains secure in presence of local and systemic factors[46]

Most common cause of wound dehiscence is the intraperitoneal sepsis. The incidence of wound dehiscence has been reported to vary from 0.2% to 10% and mortality associated with dehiscence is considerably high at 10% to 44%. Indian authors have reported burst abdomen to occur in 10% to 30% of emergency cases.

In group A of our study sample, in whom mid-line mass closure was done with polypropylene, the incidence of wound dehiscence was 4%.Whereas in group B in whom mid-line mass closure done with polydiaxonone, no cases were reported with wound dehiscence.

In a prospective study conducted, wound dehiscence was noted in 7.8% patients among 30 belonging to polydiaxonone group and none of the patients had this complication among 34 patients of polypropylene group.[47]

In another prospective study involving 284 patients wound dehiscence was not noted in either groups. [45]

In comparison to above mentioned study, we observed that Polydiaxanone suture material has lesser incidence of wound dehiscence in the post-operative period when compared to polypropylene suture material.

Incisional hernia:

The factors associated with incisional hernia are old age, male sex, obesity, bowel surgery, type of suture, chest infection, abdomen distension and most

important is wound infection.

The mass closure is one which include all layers of abdominal wall except skin and subcutaneous fat. Mid-line mass closure should be done by taking wide bites of the rectus sheath atleast 1 cm from the edge of the incision. The suturing done by taking long bites has a high risk of surgical site infection and incisional hernia. Long stitches also increases the risk of infection because they increase the amount of necrotic tissue in the wound.

In an experimental study[48] long stitches were found to compress or cut through soft tissue included in the stitch, thereby increasing the amount of necrotic tissue. The risk of incisional hernia may be increased with the use of a long stitch length because the stitch slackens which allows wound edges to separate.

Thus, new data indicate that an SL to WL ratio of atleast 4 should be achieved with small tissue bites placed at close intervals rather than with large tissue bites placed at greater intervals[46,48]. Drains, when used, are inserted through a stab wound away from the incision.

The patients were reviewed at one, three, six, and 12 months, the presence of any infection or wound herniation was carefully recorded. Healing of abdominal wall fascia requires 120 days for attaining tensile strength. As PDS is a delayed absorbable suture material, it loose its tensile strength and get absorbed in-situ after a time period of 180days. So in our study, PDS suture material has got property of delayed absorption which is required in the abdominal wall fascia healing. PDS stays in-situ and gives tensile strength till the fascia heals with good tensile strength.

Where as polypropylene has got property of non-absorbability. So it stays permanently in-situ even the wound heals with maximal tensile strength. But polypropylene can act as a local irritant which can lead to local tissue reactions which

leads to infection and subsequently incisional hernia. Because wound which gets infected in the past has more tendency for incisional hernia.

In our study 2 out of 100 patients of group A developed incisional hernia, no such complication was observed in group B. patients who developed hernia were reported to us in subsequent follow up period of 6 months and another at 1 year.

In a prospective study conducted, 6 out of 141 patients developed incisional hernia in the polydioxanone group, while 5 patients developed incisional hernia in polypropylene group (P=0.981).

This finding was found to be statistically insignificant as concluded in the other study as well.

Suture sinus formation:

Suture sinus formation is the wound complications in the delayed post-operative period. The polypropylene suture material which is a non-absorbable suture material stays permanently in-situ, so this leads to irritation to the tissue subsequently it leads to infection and leading to suture sinus. Patient will have pain and pus discharge from the suture sinus and leading to morbidity. Polydioxanone suture material is a delayed absorbable suture material, which gets absorbed in-situ without leading to tissue irritation and suture sinus formation.

In our study group A, 9(9%) patients developed suture sinus formation whereas 2(2%) patients in group B developed suture sinus formation. So this observation is not statistically significant.

In a study conducted 1 patient out of 30 in the polypropylene group developed this complication, and none of the patients in the polydioxonone group developed this complication.[47]

In comparison with the above study polydiaxonone had lesser incidence of suture sinus formation as observed in our study.

Palpable knots:

Palpable knots is the subjective feeling of the sutures underneath skin which is painful and causing the patient difficult in doing routine daily activities. Surgeons perform knotting to safeguard the slipping of sutures.

Polypropylene suture material which is non-absorbable has tendency to stay permanently even after the wound is healed completely. So this property leads to formation of palpable knots in the region where the knots are secured while performing a mass closure of a laparotomy wound. Polydioxanone suture material which is delayed absorbable, stays till the wound achieves the maximum tensile strength, so the wound cannot have the palpable knots in the delayed postoperative period.

In our study, 23(23%) patients reported palpable knots in group A, whereas no patients reported with complaints of palpable knots in group B. So this part of study showed statistical significance ($p<0.001$).

SUMMARY

To study the various effects of suture material on wound healing, this study was done by taking into account patients undergoing midline laparotomy. Wherein 200 cases of continuous enmass closure of midline laparotomy incisions were studied to compare the results of PDS (polydioxanone) and polypropylene suture material. 100 cases were included in group A and 100 cases were included in group B.

Since the time period of follow up was limited upto one year, we here by explain the limitations of the study by collecting 200 cases out of 240 cases and the study is carried out among the 200 cases. The cases which are collected after the period of collection (JAN 2014) had been followed up for a period of only 3 to 6 months and the observations has been noted down. Since the palpable knots and incisional hernia are the complications which are expected to occur at the end of one year This is the limitations of our study.

Both elective and emergency cases were included in study, out of which elective cases were 18 and emergency cases were 182 cases.

The male to female ratio was 4:1. Patients aged 25-30 years formed maximum number of the study group.

The early and late wound complications encountered in both the suture material used were as follows.

The incidence of wound pain was observed in all the patients(out of 100) in both immediate(moderate pain) and delayed(mild pain) post-operative period in the polypropylene suture material compared to polydioxanone where in only 20% of patients(out of 100) had wound pain.

The incidence of wound infection was higher in polypropylene (24% out of 100) compared to PDS(2% out of 100). The use of polydioxanone was better in emergency cases with low infection rate as compared to polypropylene suture

material.

There were 4 cases(out of 100) of wound dehiscence in the present study. All the cases of burst abdomen were noted in patients in whom midline closure done with polypropylene suture material. The factors responsible for burst abdomen may be abdomen distension, malnutrition, anemia and intraperitoneal sepsis. Postoperative wound infection is also a most important factor for wound dehiscence. So the use of Polydioxanone(PDS) suture material was better in the emergency cases with no cases of burst abdomen as compared to prolene suture material with burst abdomen of 4%. The incidence of suture sinus formation was higher in the polypropylene suture material (9% out of 100) compared to the polydioxanone suture material(2% out of 100) in the delayed postoperative period.

The incidence of palpable knots was higher in the polypropylene suture material(23%out of 100) compared to the polydioxanone suture material in which no cases were reported in the follow up of patients in delayed post operative period.

The incidence of incisional hernia was noted in two cases in the polypropylene suture material and the cases were operated on emergency basis. Among two cases, one of the case treated for wound infection. No cases of incisional hernia were reported with polydioxanone suture material.

The overall morbidity from abdominal closure was considerably reduced in the Polydioxanone group. We encountered reduction in wound complications like burst abdomen, wound infection, wound pain, suture sinus formation, palpable knots and incisional hernia. Polydioxanone is a synthetic absorbable material which is a monofilament has a tensile strength 1.7 times greater than Polypropylene (Prolene) which retains approximately 50% of its strength for 6 weeks. It can be employed rewardingly in emergency situations where closure can be carried out safely and rapidly.

CONCLUSION

Based on the observations made in this study, it has been concluded that continuous mass closure technique using no.1 Polydioxanone (PDS) for closure of midline laparotomy incision is superior to no.1 Polypropylene (PPL) suture material in preventing the wound complications like post-operative wound dehiscence, wound pain, wound infection, suture sinus formation, palpable knots and incisional hernia.

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ANNEXURES

ANNEXURE – I

PROFORMA

NAME **DATE OF ADMISSION**

IP NUMBER

AGE

SEX **TIME AND DATE OF OPERATION**

RELIGION

ADDRESS **TIME AND DATE OF DISCHARGE**

CLINICAL DIAGNOSIS:

COMORBIDITIES:

TYPE OF OPERATION: EMERGENCY **ELECTIVE**

GROUP A ***GROUP A***

GROUP B ***GROUP B***

OPERATIVE PROCEDURE:

TYPE OF INCISION:

INTRAOPERATIVE FINDINGS:

INVESTIGATIONS:

1. **PERITONEAL FLUID SAMPLE(FOR CULTURE AND SENSITIVITY):**

2. **WOUND DISCHARGE(FOR CULTURE AND SENSITIVITY):**

GROUP A : INCLUDE LAPAROTOMY WOUND CLOSURE BY USING POLYPROPYLENE

GROUP B : INCLUDE LAPAROTOMY WOUND CLOSURE BY USING POLYDIOXANONE

FOLLOW UP:

IMMEDIATE POSTOPERATIVELY:

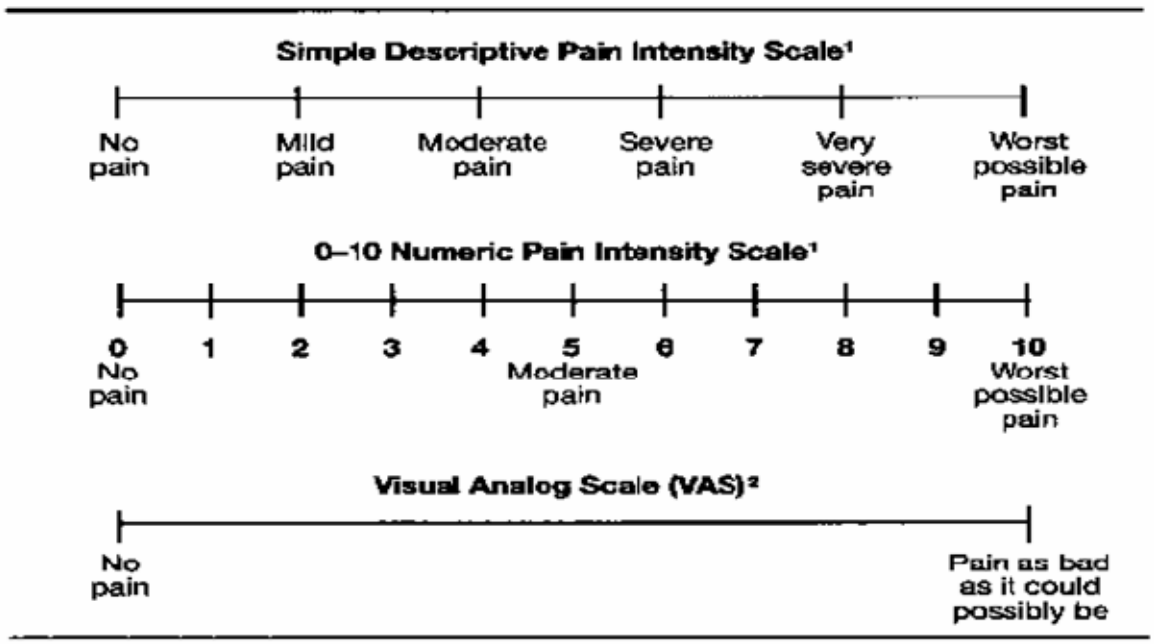
1. WOUND SEPSIS :

TYPE OF DISCHARGE SEROUS SEROSANGUNOUS PURULENT

AMOUNT **SCANTY** **MODERATE** **COPIOUS**

WOUND PAIN:

ACCORDING TO VISUAL ANALOG SCALE:



[1] If used as a graphic rating scale, a 10 cm baseline is recommended.
[2] A 10-cm baseline is recommended for VAS scales.

ANTIBIOTIC COVERAGE GIVEN:

PRE OPERATIVELY:

POST OPERATIVELY:

PAIN MANAGEMENT (ANALGESIC GIVEN):

POSTOPERATIVE PERIOD :

	2 Weeks	4 Weeks	3 Months	6 Months	9 Months	1 Year
1.WOUND PAIN						
2.WOUND DEHISENCE						
3.SUTURE SINUS FORMATION						
4.PALPABLE KNOTS						
5.INCISIONAL HERNIA						

SUMMARY :

KEY TO MASTER CHART

PID	pelvic inflammatory disease
AIO	acute intestinal obstruction
Perf	hollow viscus Perforation
R tumour	Retroperitonealtumour
Pen injury	Penetrating injury abdomen
Blunt	blunt injury abdomen
L. abscess	Liver Abscess
A abscess	Apendicular Abscess
Pelvic Abscess	Pelvic Abscess
A phlegmon	Appendicular Phlegmon
Ano rectal CA	Ano rectal Carcinoma
MSSA	Methicillin Sensitive Staph Aureus
NIL	not present in follow up period
EMERG	Emergency surgeries
Elect	Elective surgeries
P	Present
M	Male
F	Female

MASTER CHART PROLENE GROUP

SL No	Name	OP No	Age	Sex	Date of Admission	Date of Surgery	Emergency Elective	Date of Discharge/Death	Clinical Diagnosis	Investigations Peritonial Aspirate	Immediate Post Op			Late Post Op				Incisional hernia
											Wound Pain	Wound Sepsis	Wound Discharge c/s	Wound Pain	Wound Dehichen	Suture Sinus Formation	Palable Knots	
1	Ra	858303	25	F	7/11/2012	14/11/2012	EMERG	16.11.12	PID	no growth	moderate	purulent	gram +	mild	Nil	Nil	Nil	Nil
2	Vmma	882182	55	F	4/2/2013	9/2/2013	EMERG	09.02.13	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
3	NA	877853	62	F	6/2/2013	10/2/2013	ELECTIVE	17.02.2013	SAIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
4	An	861374	50	M	12/2/2013	12/2/2013	EMERG	12.12.12	perf	e.coli	moderate	healthy	not sent	mild	Nil	Nil	P	Nil
5	Mun	876455	55	F	15/02/2013	15/02/2013	ELECTIVE	25.02.13	R tumour	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
6	Mo	885941	20	M	16/02/2013	16/02/2013	EMERG	28.02.13	pen injury	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
7	Vppa	860105	55	M	19/02/2013	19/02/2013	EMERG	01.12.12	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
8	Lak	865317	45	M	20/02/2013	20/02/2013	EMERG	07.01.13	perf	e.coli	moderate	serous	not sent	mild	Nil	Nil	Nil	Nil
9	Ma	892356	36	M	12/3/2013	12/3/2013	EMERG	10.04.13	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
10	Vi	864277	58	F	13/03/2013	13/03/2013	EMERG	19.12.12	AIO	N flora	moderate	serous	not sent	mild	Nil	Nil	Nil	Nil
11	Na	857261	35	M	10/4/2013	10/4/2013	EMERG	17.11.12	PERF	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
12	Sh	854989	45	M	10/4/2013	1004/2013	EMERG	21.11.12	AIO	no growth	moderate	serous	not sent	mild	P	P	P	P
13	ch	902921	28	F	18/04/2013	18/04/2013	EMERG	16.05.13	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	P	Nil
14	Na	902962	23	M	19/04/2013	19/04/2013	EMERG	18.05.13	L abscess	no growth	moderate	serous	not sent	mild	Nil	Nil	P	Nil
15	Si	905540	40	M	27/04/2013	27/04/2013	EMERG	15.05.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
16	Na	910629	45	M	14/05/2013	14/05/2013	EMERG	15.06.13	perf	e.coli	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
17	Ve	910161	60	F	20/05/2013	20/05/2013	EMERG	21.06.13	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
18	Bi	912629	23	M	22/05/2013	22/05/2013	EMERG	10.06.13	perf	no growth	moderate	serous	not sent	mild	Nil	Nil	Nil	Nil
19	SU	913645	25	M	28/05/2013	28/05/2013	EMERG	25.06.13	PERF	e.coli	moderate	serous	not sent	mild	Nil	Nil	Nil	Nil
20	Sr	916348	35	M	3/6/2013	3/6/2013	EMERG	28.2.12	Perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
21	SO	922159	23	M	24/06/2013	24/06/2013	EMERG	29.06.13	Perf	no growth	moderate	serous	not sent	mild	P	Nil	P	Nil
22	JO	922912	28	M	27/06/2013	27/06/2013	EMERG	08.07.13	perf	no growth	moderate	purulent	MSSA	mild	Nil	Nil	P	Nil
23	Na	924521	75	M	2/7/2013	2/7/2013	EMERG	08.07.13	perf	e.coli	moderate	healthy	not sent	mild	Nil	Nil	P	Nil
24	La	926222	58	M	9/7/2013	9/7/2013	EMERG	20.07.13	perf	e.coli	moderate	purulent	gram +	mild	Nil	Nil	P	Nil
25	Na	928211	35	F	16/07/2013	16/07/2013	EMERG	06.09.13	blunt	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
26	De	885574	75	M	26/07/2013	26/07/2013	EMERG	15.08.13	AIO	no growth	moderate	purulent	skin flora	mild	Nil	Nil	Nil	Nil
27	Sa	931357	60	F	27/07/2013	27/07/2013	EMERG	10.08.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
28	Na	932367	81	M	30/07/2013	30/07/2013	EMERG	19.08.13	perf	no growth	moderate	healthy	skin flora	mild	Nil	Nil	Nil	Nil
29	SY	933668	40	M	4/7/2013	4/7/2013	EMERG	22.08.13	perf	no growth	moderate	healthy	skin flora	mild	Nil	P	P	Nil
30	La	933583	38	F	5/8/2013	5/8/3013	EMERG	22.08.13	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
31	Na	936776	26	M	15/08/2013	15/08/2013	EMERG	31.08.13	perf	no growth	moderate	purulent	seudomona	mild	Nil	Nil	P	Nil
32	Su	938468	50	M	21/08/2013	21/08/2013	EMERG	07.09.13	perf	e.coli	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil

MASTER CHART PROLENE GROUP

33	Ra	941502	23	M	5/9/2013	5/9/2013	EMERG	19.10.13	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
34	Sri	946324	40	M	6/9/2013	6/9/2013	EMERG	19.10.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
35	Ch	945302	50	M	12/9/2013	12/9/2013	EMERG	07.10.13	AIO	no growth	moderate	purulent	skin flora	mild	Nil	Nil	P	P
36	Ak	948904	18	M	18/09/2013	18/09/2013	EMERG	07.10.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
37	Ma	949894	25	M	27/09/2013	27/09/2013	EMERG	29.09.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
38	Sa	950686	20	M	30/09/2013	30/09/2013	EMERG	15.10.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
39	BYR	945945	55	M	1/10/2013	6/10/2013	ELECTIVE	20.10.13	CA stomach	no growth	moderate	serous	not sent	mild	Nil	P	P	Nil
40	Na	953236	40	M	1/10/2013	1/10/2013	EMERG	28.10.13	perf	no growth	moderate	serous	not sent	mild	Nil	Nil	P	Nil
41	Na	953265	35	M	5/10/2013	5/10/2013	EMERG	30.10.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	P	Nil
42	LAK	835733	53	F	8/10/2013	12/10/2013	ELECTIVE	20.10.13	R.V.F	no growth	moderate	purulent	seudomona	mild	Nil	Nil	P	Nil
43	Am	734086	42	M	10/10/2013	10/10/2013	EMERG	26.09.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
44	Re	958392	84	M	12/10/2013	12/10/2013	EMERG	10.11.12	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
45	Ve	945304	60	M	15/10/2013	15/10/2013	EMERG	18.10.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
46	Lak	960439	65	M	20/10/2013	20/10/2013	EMERG	27.11.13	perf	no growth	moderate	purulent	skin flora	mild	Nil	Nil	Nil	Nil
47	Ka	842320	56	M	25/10/2013	25/10/2013	EMERG	25.11.13	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
48	Lo	968448	38	M	30/11/2013	30/11/2013	EMERG	21.12.13	perf	no growth	moderate	healthy	not sent	mild	Nil	P	P	Nil
49	Sa	968485	25	M	1/12/2013	1/12/2013	EMERG	02.01.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	P	Nil
50	Sh	966367	63	F	2/12/2013	2/12/2013	EMERG	07.01.14	BLUNT	no growth	moderate	purulent	gram +	mild	Nil	Nil	Nil	Nil
51	Fu	969484	40	M	5/12/2013	5/12/2013	EMERG	27.12.13	perf	no growth	moderate	healthy	MSSA	mild	Nil	Nil	Nil	Nil
52	Ch	945302	50	M	12/9/2013	12/9/2013	EMERG	7.10.13	AIO	no growth	moderate	healthy	gram +	mild	Nil	Nil	Nil	Nil
53	Ba	934996	42	F	10/12/2013	10/12/2013	EMERG	21.08.13	AIO	no growth	moderate	purulent	MSSA	mild	Nil	Nil	Nil	Nil
54	Lak	973414	30	F	19/12/2013	19/12/2013	EMERG	29.01.14	perf	e.coli	moderate	healthy	no growth	mild	Nil	Nil	Nil	Nil
55	Sr	974698	50	M	23/12/2013	23/12/2013	EMERG	02.01.14	perf	no growth	moderate	healthy	no growth	mild	Nil	Nil	Nil	Nil
56	Sr	977485	27	M	3/12/2013	3/12/2013	EMERG	18.01.14	perf	no growth	moderate	healthy	no growth	mild	Nil	Nil	Nil	Nil
57	Ak	946304	18	M	6/1/2014	6/1/2014	EMERG	24.10.13	perf	no growth	moderate	healthy	not sent	mild	Nil	P	P	Nil
58	Ku	979628	50	M	9/1/2014	9/1/2014	EMERG	12.02.14	perf	no growth	moderate	purulent	MSSA	mild	Nil	Nil	Nil	Nil
59	Su	982481	24	M	20/01/2014	20/01/2014	EMERG	06.02.14	perf	no growth	moderate	healthy	gram +	mild	Nil	Nil	Nil	Nil
60	DEVA	984190	32	F	6/2/2014	10/2/2014	ELECTIVE	18.02.14	ch.pancreatitis	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
61	DODD	984406	70	M	11/2/2014	17/02/2014	ELECTIVE	26.02.14	CA.stomach	no growth	moderate	serous	not sent	mild	Nil	Nil	Nil	Nil
62	GOU	991960	52	F	20/02/2014	24/02/2014	ELECTIVE	02.03.2014	CA caecum	no growth	moderate	healthy	not sent	mild	Nil	P	P	Nil
63	AK	994516	17	M	25/02/2014	25/02/2014	EMERG	21.06.14	perf	e.coli	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
64	sub	981047	60	m	17/01/2014	30/01/2014	ELECTIVE	14.05.14	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	P	Nil
65	KR	1000001	45	M	12/3/2014	12/3/2014	EMERG	25.03.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
66	MU	1002954	26	M	19/03/2014	19/03/2014	EMERG	21.04.14	ABSCESS	e.coli	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
67	MU	997332	35	M	5/3/2014	5/3/2014	EMERG	22.03.14	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
68	MA	892356	36	M	12/3/2014	12/3/2014	EMERG	10.04.14	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
69	MO	978113	60	M	4/1/2014	4/1/2014	EMERG	20.01.14	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
70	AK	946304	18	M	6/1/2014	6/1/2014	EMERG	24.10.13	PERF	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
71	VEN	985404	60	M	28/01/2014	28/01/2014	EMERG	08.02.14	perf	e.coli	moderate	purulent	gram +	mild	Nil	Nil	Nil	Nil
72	VA	992728	25	F	20/02/2014	20/02/2014	EMERG	20.02.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	P	Nil

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73	NA	998528	32	M	7/3/2014	7/3/2014	EMERG	25.03.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
74	SON	1003036	62	M	20/03/2014	20/03/2014	EMERG	03.04.14	perf	no growth	moderate	healthy	not sent	mild	Nil	P	P	Nil
75	HAN	1006532	20	M	29/03/2014	29/03/2014	EMERG	14.04.14	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
76	VE	1006649	56	M	31/03/2014	31/03/2014	EMERG	05.04.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
77	CHA	1007265	40	M	3/4/2014	7/4/2014	ELECTIVE	20.04.14	CA.stomach	no growth	moderate	purulent	gram +	mild	Nil	Nil	P	Nil
78	GA	1000843	11	M	15/04/2014	15/04/2014	EMERG	28.03.14	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
79	VE	1014757	70	M	23/04/2014	23/04/2014	EMERG	21.05.14	perf	e.coli	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
80	BHA	1012951	58	M	25/04/2014	25/04/2014	EMERG	22.04.14	pen injury	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
81	KEM	1785	70	M	1/5/2014	1/5/2014	EMERG	21.05.14	perf	e.coli	moderate	purulent	MSSA	mild	Nil	Nil	Nil	Nil
82	NA	907624	39	M	4/5/2014	4/5/2014	EMERG	18.05.14	BLUNT	no growth	moderate	healthy	not sent	mild	P	Nil	Nil	Nil
83	VE	2979	35	M	8/5/2014	8/5/2014	EMERG	22.05.14	perf	e.coli	moderate	purulent	streptococc	mild	Nil	Nil	Nil	Nil
84	AP	5952	35	M	16/05/2014	16/05/2014	EMERG	29.05.14	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
85	NA	12244	72	F	31/05/2014	31/5/2014	EMERG	14.06.14	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
86	LAX	11725	40	M	31/05/2014	31/05/2014	EMERG	14.07.14	AIO	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
87	CH	108299	65	M	2/5/2014	2/5/2014	EMERG	14.08.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
88	DE	946300	20	M	6/6/2014	16/06/2014	ELECTIVE	02.10.13	T SPLENOMEGALY	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
89	SAN	17149	19	M	13/06/2014	13/06/2014	EMERG	02.10.13	pen injury	no growth	moderate	serous	not sent	mild	Nil	Nil	Nil	Nil
90	NAT	17633	45	M	18/06/2014	18/06/2014	EMERG	21.06.14	AIO	e.coli	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
91	RA	21916	45	M	26/06/2014	26/06/2014	EMERG	21.06.14	perf	no growth	moderate	serous	not sent	mild	Nil	P	Nil	Nil
92	SH	25974	16	M	6/7/2014	6/7/2014	EMERG	21.07.14	perf	e.coli	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
93	RA	26473	23	M	7/7/2014	7/7/2014	EMERG	16.07.14	pen injury	e.coli	moderate	healthy	not sent	mild	P	Nil	Nil	Nil
94	MA	26870	22	M	9/7/2014	9/7/2014	EMERG	21.07.14	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
95	SHI	33642	35	M	27/07/2014	27/07/2014	EMERG	06.08.14	perf	no growth	moderate	serous	not sent	mild	Nil	Nil	Nil	Nil
96	MUN	34987	29	F	30/07/2014	30/07/2014	EMERG	07.08.14	BLUNT	no growth	moderate	healthy	not sent	mild	Nil	P	Nil	Nil
97	VEN	38092	55	M	8/8/2014	8/8/2014	EMERG	09.08.14	perf	e.coli	moderate	purulent	skin flora	mild	Nil	Nil	Nil	Nil
98	SO	38904	45	M	11/8/2014	11/8/2014	EMERG	23.08.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
99	KEE	40914	5	F	16/08/2014	16/08/2014	EMERG	27.08.14	perf	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil
100	PUSH	32776	45	F	19/08/2014	25/08/2014	ELECTIVE	02.09.14	CA.rectum	no growth	moderate	healthy	not sent	mild	Nil	Nil	Nil	Nil

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SL No	Name	OP No	Age	Sex	Date of Admission	Date of Surgery	Emergency Elective	Date of Discharge/Death	Clinical Diagnosis	Investigations Peritonial Aspirate	Immediate Post Op			Late Post Op				Incisional hernia
											Wound Pain	Wound Sepsis	Wound Discharge c/s	Wound Pain	Wound Dehichen	Suture Sinus Formation	Palable Knots	
1	Ra	860731	46	M	2/1/2013	2/1/2013	EMERG	11.12.12	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
2	Na	872835	60	M	2/1/2013	2/1/2013	EMERG	26.01.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
3	YAS	873021	45	F	7/1/2013	12/1/2013	ELECTIVI	20.01.13	SAIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
4	Gu	878863	60	M	10/1/2013	10/1/2013	EMERG	30.03.13	perf	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
5	Sh	868839	40	F	11/1/2013	11/1/2013	EMERG	03.01.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
6	MAS	875232	45	M	21/01/2013	26/01/2013	ELECTIVI	5/2/2013	CA.stomach	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
7	Sh	878724	25	M	23/01/2013	23/01/2013	EMERG	15.02.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
8	Ra	880556	35	F	26/01/2013	26/01/2013	EMERG	09.02.13	A abscess	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
9	Ba	881569	50	M	2/2/2013	2/2/2013	EMERG	25.03.13	Intessuception	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
10	Ba	882299	27	M	5/2/2013	5/2/2013	EMERG	15.02.13	perf	N flora	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
11	Na	886777	50	M	20/02/2013	20/02/2013	EMERG	09.03.13	PERF	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
12	LIN	879629	65	M	1/3/2013	16/03/2013	ELECTIVI	20/03/13	CA sigmoid	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
13	Ve	890070	45	M	2/3/2013	2/3/2013	EMERG	13.03.13	perf	no growth	mild	serous	NOT SENT	no pain	Nil	P	Nil	Nil
14	Ma	891135	40	M	6/3/2013	6/3/2013	EMERG	12.03.13	penetrating	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
15	Mu	895902	35	M	27/03/2013	27/03/2013	EMERG	20.1.12	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
16	As	903010	70	M	18/04/2013	18/04/2013	EMERG	13.2.12	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
17	Ve	877405	24	F	22/04/2013	22/04/2013	EMERG	20.04.13	AIO	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
18	Na	907624	32	M	4/5/3013	4/5/2013	EMERG	18.05.13	blunt	no growth	moderate	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
19	Sh	909409	50	M	10/5/2013	10/5/2013	EMERG	23.05.13	blunt	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
20	Ch	908741	45	M	8/5/2013	8/5/2013	EMERG	01.06.13	perf	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
21	Ve	909043	35	M	9/5/2013	9/5/2013	EMERG	24.01.13	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
22	Na	911366	19	M	17/05/2013	17/05/2013	EMERG	18.05.13	blunt	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
23	Su	912676	25	M	21/05/2013	21/05/2013	EMERG	11.06.13	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
24	An	913911	14	F	25/05/2013	25/05/2013	EMERG	27.06.13	perf	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
25	Sr	918590	38	M	11/6/2013	11/6/2013	EMERG	24.06.13	perf	e.coli	mild	purulent	klebsiella	no pain	nil	Nil	Nil	Nil
26	Pa	918948	18	M	13/06/3013	13/06/3013	EMERG	27.06.13	blunt	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
27	Va	938065	45	M	23/06/2013	23/06/2013	EMERG	07.09.13	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
28	Ve	923853	64	M	30/06/2013	30/06/2013	EMERG	16.07.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
29	Vi	921471	23	M	22/06/2013	22/06/2013	EMERG	22.06.13	stab	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
30	Na	922910	55	M	27/06/2013	27/06/2013	EMERG	09.06.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	P	Nil	Nil
31	Sh	924836	20	M	4/7/2013	4/7/2013	EMERG	11.07.13	BLUNT	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
32	Sr	929794	26	M	26/07/2013	26/07/2013	EMERG	12.08.13	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil

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33	Ma	933254	25	M	2/8/2013	2/8/2013	EMERG	31.10.13	perf	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
34	Ve	936102	60	M	13/08/2013	13/08/2013	EMERG	24.08.13	perf	no growth	mild	purulent	MSSA	no pain	nil	Nil	Nil	Nil
35	Sh	934734	20	M	8/8/2013	8/8/2013	EMERG	08.09.13	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
36	Ch	928495	60	F	19/08/2013	19/08/2013	EMERG	08.09.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
37	Ga	941872	30	M	30/08/2013	30/08/2013	EMERG	09.09.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
38	Kr	942230	56	M	2/9/2013	2/9/2013	EMERG	03.06.13			moderate	healthy	NOT SENT					
39	Ak	946304	18	M	15/09/2013	15/09/2013	EMERG	26.5.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
40	Lak	954470	21	M	13/10/2013	13/10/2013	EMERG	30.5.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
41	Ma	955771	45	F	15/10/2013	15/10/2013	EMERG	31.10.13	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
42	Sr	957883	45	M	25/10/2013	25/10/2013	EMERG	06.11.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
43	Me	961617	30	F	6/11/2013	6/11/2013	EMERG	23.11.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
44	Mu	961711	45	M	8/11/2013	8/11/2013	EMERG	20.11.13	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
45	Mo	962601	23	M	9/11/2013	9/11/2013	EMERG	04.12.13	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
46	Na	892306	86	M	o2/11/2013	o2/11/2013	EMERG	05.12.13	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
47	Ga	964287	15	F	28/11/2013	28/11/2013	EMERG	12.12.13	p abscess	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
48	Sh	966367	63	M	1/12/2013	1/12/2013	EMERG	07.01.14	blunt	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
49	Ra	969473	38	M	4/12/2013	4/12/2013	EMERG	28.12.13	penetrating	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
50	Fu	969484	40	M	5/12/2013	5/12/2013	EMERG	27.12.13	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
51	Th	969691	22	F	5/12/2013	5/12/2013	EMERG	19.12.13	AIO	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
52	Pa	867544	37	F	15/12/2012	15/12/2012	EMERG	14.12.13	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
53	Sr	973381	35	M	19/12/2013	19/12/2013	EMERG	20.01.14	perf	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
54	Ra	974748	50	M	24/12/2013	24/12/2013	EMERG	26.12.13	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
55	Sr	980007	28	M	21/01/2014	21/01/2014	EMERG	05.01.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
56	ch	985424	27	M	29/01/2014	29/01/2014	EMERG	12.02.14	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
57	VE	985404	60	M	29/01/2014	29/01/2014	EMERG	08.02.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
58	MA	489879	45	F	12/2/2014	12/2/2014	EMERG	26.02.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
59	VA	992788	25	M	20/02/2014	20/02/2014	EMERG	06.03.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
60	Na	990764	60	M	14/02/2014	14/02/2014	EMERG	24.02.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
61	NAG	998528	32	M	7/3/2014	7/3/2014	EMERG	25.03.14	PERf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
62	SR	972485	27	M	3/1/2014	3/1/2014	EMERG	23.12.13	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
63	RAJ	980396	30	M	13/01/2014	13/01/2014	EMERG	29.01.14	A abscess	E COLI	moderate	healthy	ACENETOBAC TER	no pain	nil	Nil	Nil	Nil
64	SRI	980007	30	M	21/01/2014	21/01/2014	EMERG	05.03.14	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
65	MUN	982876	70	M	13/02/2014	16/02/2014	ELECTIVI	27.02.14	A.phlegmon	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
66	AK	994516	17	M	25/02/2014	25/02/2014	EMERG	21.06.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
67	PY	883102	67	M	27/02/2014	27/02/2014	ELECTIVI	26.02.14	ANO-REC CA	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
68	PA	998038	45	F	4/3/2014	4/3/2014	EMERG	27.03.14	perf	E COLI	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
69	KR	1000001	40	M	12/3/2014	12/3/2014	EMERG	25.03.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
70	AB	1001619	21	M	15/03/2014	15/03/2014	EMERG	21.03.14	penetrating	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
71	SH	979703	45	M	15/03/2014	15/03/2014	EMERG	20.01.14	PERF	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil

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72	SO	1003036	63	M	20/03/2014	20/03/2014	EMERG	03.04.14	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
73	SR	1003718	70	M	20/13/2014	20/03/2014	EMERG	16.04.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
74	SR	1003770	75	M	21/03/2014	21/03/2104	EMERG	26.03.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
75	LAX	973414	30	M	21/03/2014	21/03/2014	EMERG	29.01.14	PERF	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
76	MUN	1003273	60	M	25/03/2014	30/03/2014	ELECTIVI	09.04.14	CHRONIC ULCER	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
77	RAN	1009813	58	M	7/4/2014	7/4/2014	EMERG	28.04.14	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
78	RAJ	1012899	50	F	20/04/2014	20.04/2014	EMERG	14.05.14	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
79	MAL	914348	45	F	26/04/2014	26/04/2014	EMERG	14.06.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
80	AM	1015254	20	M	3/5/2014	3/5/2014	EMERG	21.05.14	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
81	SO	14894	85	M	7/6/2014	7/6/2014	EMERG	22.07.14	perf	KLEBSIELLA	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
82	NAN	2365	75	M	7/5/2014	7/5/2014	EMERG	20.05.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
83	VEN	11829	50	M	31/05/2014	31/05/2014	EMERG	07.06.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
84	SU	19531	26	M	19/06/2014	19/06/2014	EMERG	03.07.14	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
85	USH	8648	18	F	23/05/2014	23/05/2014	EMERG	05.06.14	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
86	AM	1015254	20	M	4/5/2014	4/5/2014	EMERG	21.03.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
87	NA	5184	40	M	14/05/2014	14/05/2014	EMERG	18.05.14	Intessuception	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
88	NEE	15603	36	F	19/06/2014	27/06/2014	ELECTIVI	18.05.14	SPLENIC HAEMANGIOMA	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
89	SY	10047	65	M	26/05/2014	26/05/2014	EMERG	18.06.14	PERF	E COLI	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
90	RA	25991	65	M	14/07/2014	14/07/2014	EMERG	18.08.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
91	MA	36971	25	M	5/8/2014	5/8/2014	EMERG	18.08.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
92	RA	39777	60	M	14/08/2014	14/08/2014	EMERG	29.08.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
93	SO	14894	85	M	6-Jul	7/8/2014	EMERG	22.07.14	perf	no growth	mild	SEROUS	NOT SENT	no pain	nil	Nil	Nil	Nil
94	SH	29835	46	M	18/07/2014	18/08/2014	EMERG	22.07.14	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
95	SRI	13758	35	M	12/6/2014	12/6/2014	EMERG	27.06.14	perf	E COLI	moderate	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
96	SHI	5394	40	F	4/7/2014	4/7/2014	EMERG	28.07.14	AIO	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
97	MU	36196	55	M	3/8/2014	3/8/2014	EMERG	07.08.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
98	MUN V	1018727	82	M	4/8/2014	4/8/2014	EMERG	20.08.14	perf	no growth	mild	serous	NOT SENT	no pain	nil	Nil	Nil	Nil
99	LAK	41535	62	F	18/08/2014	18/08/2014	EMERG	26.08.14	AIO	e.coli	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil
100	Chi	45517	65	M	27/08/2014	27/08/2014	EMERG	05.09.14	perf	no growth	mild	healthy	NOT SENT	no pain	nil	Nil	Nil	Nil