"PROSPECTIVE STUDY OF COMPARISON OF USE OF SUBCUTANEOUS DRAINS TO PREVENT WOUND COMPLICATIONS IN EMERGENCY LAPROTOMY PROCEDURES"

IN GOVERNMENT RAJAJI HOSPITAL, MADURAI

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY

BRANCH - I (GENERAL SURGERY)

MAY 2020

REG NO:221711104

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**PROSPECTIVE STUDY OF COMPARISON OF USE OF SUBCUTANEOUS DRAINS TO PREVENT WOUND COMPLICATIONS IN EMERGENCY LAPROTOMY PROCEDURES"** ***IN GOVERNMENT RAJAJI HOSPITAL, MADURAI*** submitted by Dr.R.Giridharan to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. Degree Branch I (General Surgery) is a bonafide research work was carried out by him under my direct supervision & guidance.

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This is to certify that the dissertation entitled PROSPECTIVE STUDY OF **"COMPARISON OF USE OF SUBCUTANEOUS DRAINS TO PREVENT WOUND COMPLICATIONS IN EMERGENCY LAPROTOMY PROCEDURES"*IN GOVERNMENT RAJAJI HOSPITAL, MADURAI*** is a bonafide research work done by **DR.R.GIRIDHARAN.,** Post Graduate Student, Department of General Surgery, MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL, MADURAI, under the guidance and supervision of **Dr.K.SARAVANAN,M.S,** Professor Department of General Surgery, MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL, MADURAI.

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DECLARATION

I, Dr.R.Giridharan declare that, I carried out this work on, **PROSPECTIVE STUDY OF "COMPARISON OF USE OF SUBCUTANEOUS DRAINS TO PREVENT WOUND COMPLICATIONS IN EMERGENCY LAPROTOMY PROCEDURES"** ***IN GOVERNMENT RAJAJI HOSPITAL,MADURAI*** at the Department of General Surgery, Govt. Rajaji Hospital during the period of **February 2019 to September 2019**. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, and diploma to any other University, Board either in India or abroad. This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S. degree examination in General Surgery.

Place: Madurai Dr. R. Giridharan

Date:

ACKNOWLEDGEMENT

At the outset, I wish to thank our Dean **Prof. Dr. VANITHA M.D.,DCH.,** for permitting me to use the facilities of Madurai Medical College and government Rajaji Hospital to conduct this study.

I thank sincerely, Dr.A.M.SYED IBRAHIM, M.S Professor and Head of the Department, Department of General Surgery for his valuable advice and cooperation in completing this study.

My unit chief **Dr. K.SARAVANAN M.S.**, has always guided me, by example and valuable words of advice and has given me his moral support. I will be ever grateful to him. I offer my heartfelt thanks to my unit Assistant Professors **Dr.D.ASHOKACHAKRAVARTY M.S., Dr.MALARVANAN M.S., Dr.RANI M.S.,** for their constant encouragement, timely help and critical suggestions throughout the study and also for making my stay in the unit both informative and pleasurable.

My patients, who form the most integral part of the work, were always kind and cooperative. I pray to God give them courage and strength to endure their illness, hope all of them go into complete remission.

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TABLE OF CONTENTS

INTRODUCTION

The incidence of sudden dehiscence of the abdominal laparotomy wound is a major life threatening disaster of a patient who has undergone an emergency abdominal surgeries and a major psychological stress to the patient as well as the surgeon. The partial or complete postoperative dehiscence of laparotomy wound closure is known as acute wound failure. Acute wound failure is defined as postoperative separation of the abdominal musculoaponeurotic layers, within 1month after operation and requires some form of intervention, usually during the same period hospitalization. Most Wound dehiscence occur between the 5^{th} and 10^{th} postoperative day.

The strength of the sutured abdominal laparotomy depends on the infections and seroma formation at the subcutaneous level. Various clinical trials have shown to reduce the incidence of wound dehiscence. Some studies have done which showed an increased incidence of wound failure due to infections and incisional hernias due to delayed wound closure, and some studies shown no difference in these complications, but no studies have shown an advantage of subcutaneous drain by draining of seroma and infective material like pus. Subcutaneous negative suction drainage will reduce the incidence of SSI and wound dehiscence by draining of seroma which will promote wound healing and the rate of dehiscence has generally been less than 1%, The prevalence of wound failure in Indian scenario is found to range from 10-25% for emergency surgeries.

REVIEW OF LITERATURE

Subcutaneous negative suction drainage will reduce the incidence of surgical site infections and wound dehiscence by draining of seroma which will promote wound healing. Partial or complete separation of an abdominal wound with protrusion (evisceration) of abdominal contents is called as wound failure. Wound dehiscence and incisional hernia are part of the same wound failure process.

A meta analysis on 31 randomized trials showed that ratio of wound dehiscence was reduced to more than half with subcutaneous drain when compared without subcutaneous drain in case of emergency laparotomy.

In emergency surgery the wound is closed after fixing the subcutaneous drain that is *negative suction* drain in order to reduce the seroma formation. Then laparotomy wound is closed in layers. The seroma collections are monitored during post operative periods until the drain becomes less than 30ml for 2-3 consecutive post operative day. Then the drain is removed. Here by collecting the seroma or infective particles like pus in the sealed container by *negative suction*. There fore there would be no collections at or below the subcutaneous plane which will promote the healing.

In India the incidence of abdominal wound failure is still very high and stays above the 15% level because ofnumerousreasons which are described in the following

- Malnutrition
- Lack of tertiary care centre providing emergency surgical management.
- Delayed presentation resulting in high level of contaminations
- More marked systemic inflammatory response syndrome and multi organ failure adversely affecting healing process and collagen synthesis.

ANATOMY

ANTERIOR ABDOMINAL WALL

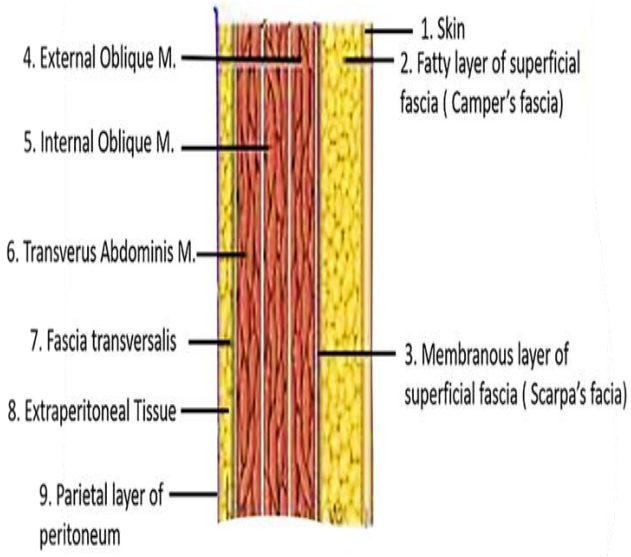
It is formed of the following layers:

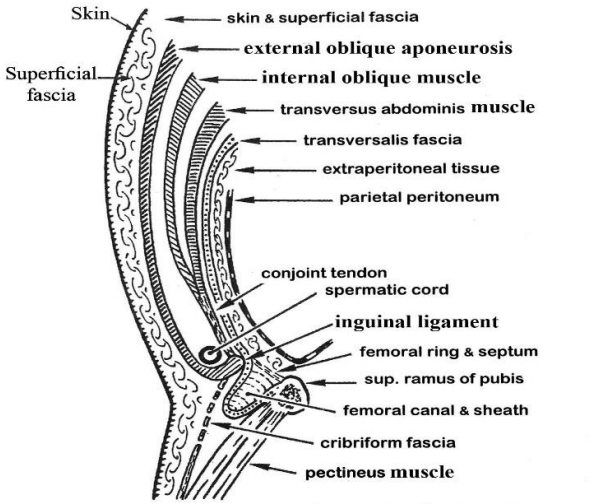
1. Skin
2. Superficial fascia (no deep fascia).
3. Abdominal muscles

4. Fascia transversalis

5.Extraperitoneal fat.

6. Parietal peritoneum.





Layers of the anterior abdominal wall
(Longitudinal section at the level of femoral sheath)

SKIN:

skin is thin and made up of epidermis & dermis. Presents the **umbilicus** which is inverted scar formed by separation of umbilical stump after birth. Umblicus always lies in the lineaalba at different level for different individual. Usually it is located below a point midway between the xiphoid process&symphysis pubis (disc between $L_{3\&4}$).Its level is one of the sites of anastomosis between superior vena cava and inferior vena cavaandporto-systemic anastomosis.

It is umblicated because its posterior surface of the umbilicus is the meeting of falciform ligament , ligamentumteres of liver (obliterated left umbilical vein) , right & left medial umbilical ligaments (obliterated umbilical arteries) & median umbilical ligament (obliterated urachus).Above the level of umbilicus the lymphatic & venous drainage to the axilla while below this level they pass downwards to the inguinal nodes.

Nerve supply:

Skin of anterior abdominal wall is supplied by $T_{7\text{-}12}$&L_1nerves .

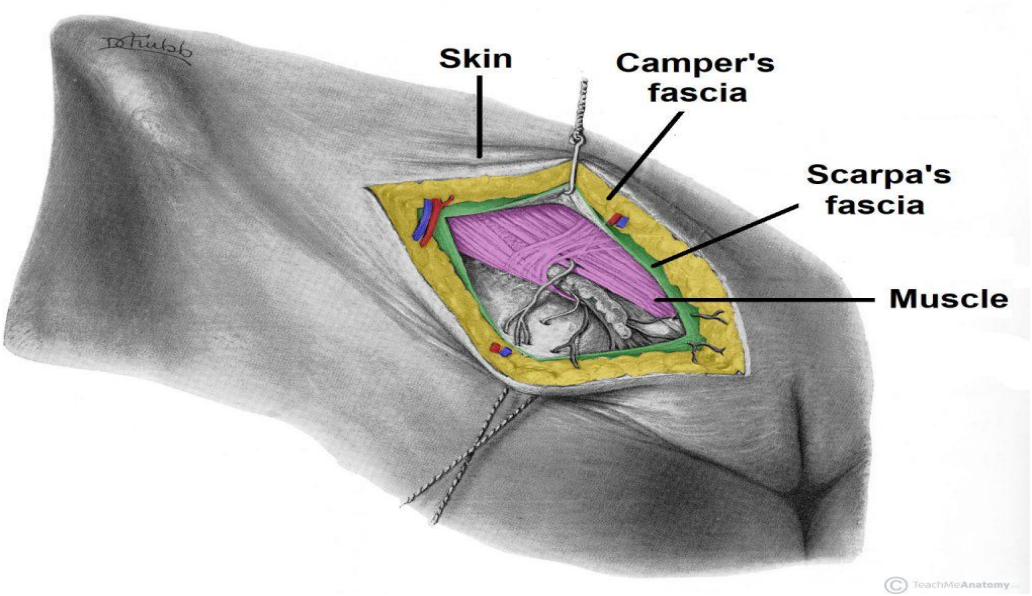
Skin at level of umbilicus is supplied by T_{I0}segment.

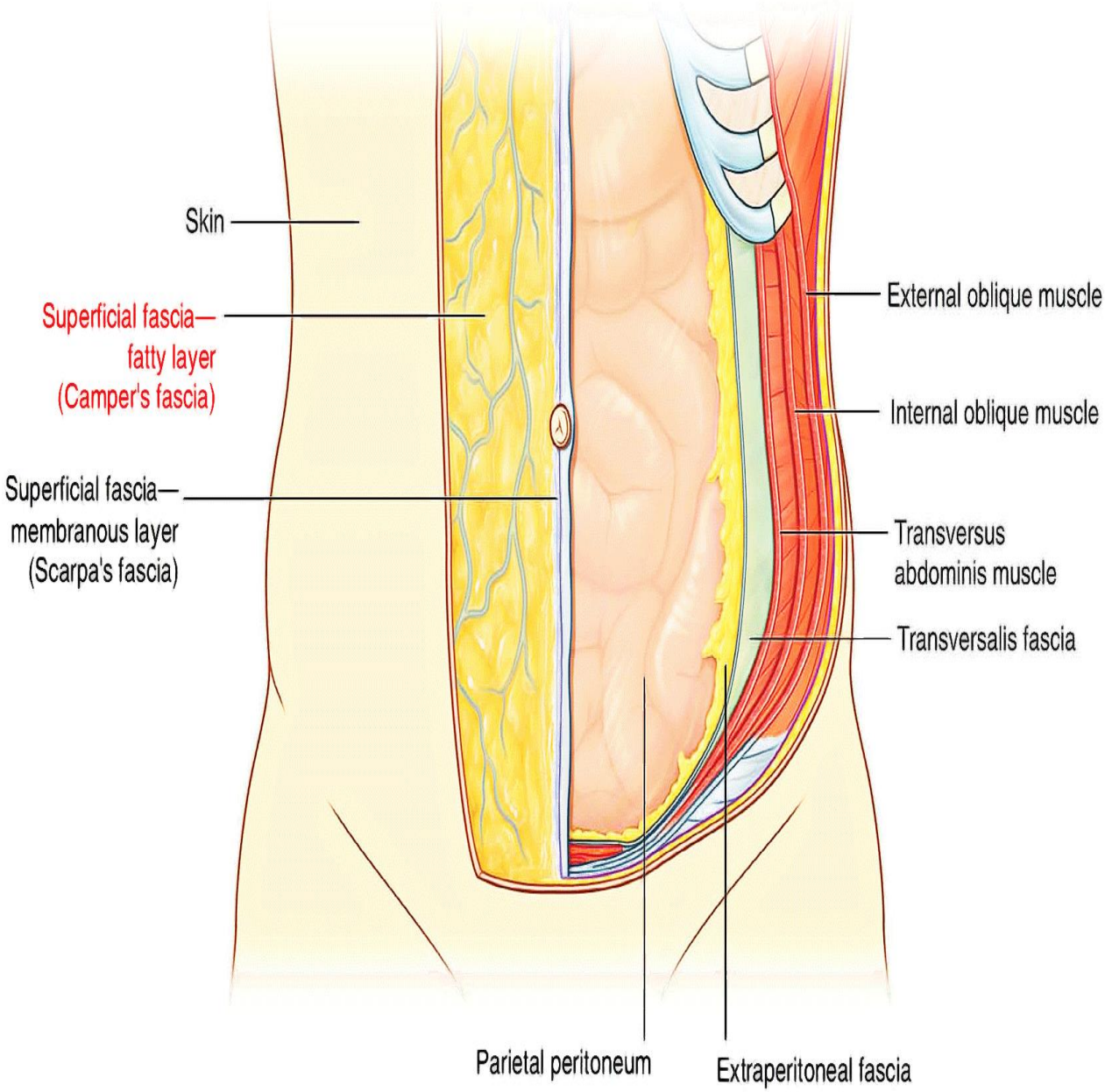
SUPERFICIAL FASCIA

It is differentiated by above the umbilicus and below the umbilicus, into two layers:

a) **Superficial fatty layer:(Camper's fascia)**A major site for accumulation of fat

b) **Deep membranous layer**: **(Scarpa's fascia)** developed below the umbilicus. It is attached to the fascia lata of thigh a 1cm breadth below the inguinal ligament. In the median plane , it envelopes the penis & scrotum then extends backwards into the perineum as Colle's Fascia which is attached to the posterior border of perineal membrane → superficial perineal pouch which contain bulbar urethra → ***in extra-pelvic rupture of male urethra, extravasation of urine into the perineum, scrotum, penis and anterior abdominal wall (between Scarpa's fascia & abdominal muscles).***




Skin
Superficial fascia—
fatty layer
(Camper's fascia)
Superficial fascia—
membranous layer
(Scarpa's fascia)
External oblique muscle
Internal oblique muscle
Transversus
abdominis muscle
Transversalis fascia
Parietal peritoneum
Extraperitoneal fascia

MUSCLES OF ANTERIOR ABDOMINAL WALL

INSERTION:

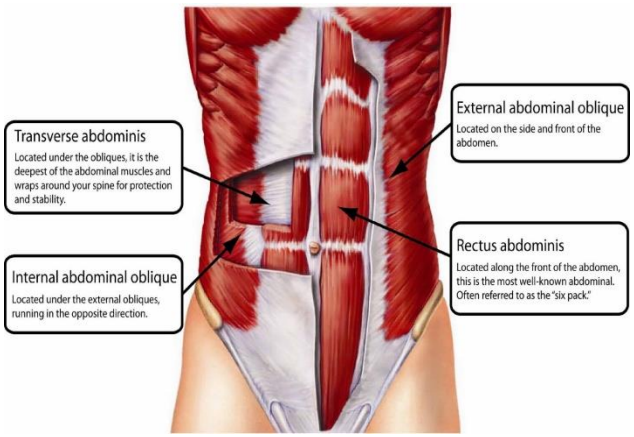
The lateral 3 muscles develop 3 broad aponeuroses towards the median plane to form the rectus sheath, then become inserted in the lineaalba

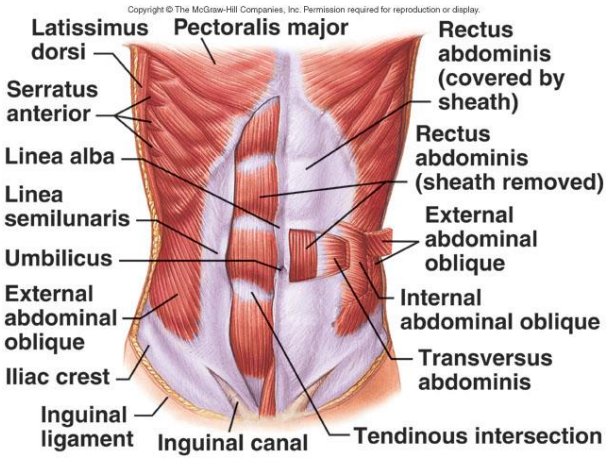
NERVE SUPPLY:

- Lower 5 intercostal,
- subcostal ,
- iliohypogastric
- ilioinguinal nerves .

ACTION :

- Respiration,
- protect viscera,
- keep viscera in position during increase intra-abdominal pressure ,
- flex trunk(rectus) &lateral flex and twist trunk.





MUSCLE	ORIGIN	INSERTION	IMPORTANT FEATURES
External Abdominal Oblique	• Outer Surface of Lower 8 ribs	• Xiphoid process, lineaalba&symphysis pubis • A.S.I.S, outer lip of iliac crest. • Pubic tubercle	1) Its fibers runs downwards, forwards& medially. 2) The Lower part of aponeurosis of ext. oblique form the main part of inguinal lig. 3) The ext. inguinal ring is an opening in the aponeurosis of ext. oblique m. 4) The aponeurosis is prolonged at the ext. ring to form the ext. spermatic fascia. 5) Below a line between A.S.I.S. & umbilicus, the muscle becomes aponeurotic.
Internal Abdominal Oblique	• Lat. 2/3 of upper concave surface of inguinal ligament . • Intermediate part of iliac • crest. Thoracolumbar fascia	• Lower 6 costal cartilages. • Xiphoid process &lineaalba&symphysispubis . • Lower fibers (conjoint tendon) is inserted into pubic crest & medial part of pectineal line.	• The fibers runs upwards, forefwards& medially. • Cremastric m. is derived from int. oblique. It form U shaped loop around the spermatic cord & testis inserted into pubic tubercle. It elevate the testis during coughing, straining & ejaculation. • The lower fibers of int. oblique has a triple relation to the spermatic cord: 1) first ant. to the cord forming the lat. 1/2 of ant. wall of inguinal canal. 2) Arch above the cord forming the roof of inguinal canal. 3) Finally, the conjoint tendon lies behind the cord forming the med. 1/2 of post. wall of inguinal canal.

Transversus abdominis	• Lat 1/3 of the upper surface of inguinal ligament. • Inner lip of iliac crest. • Thoracolumbar fascia. • Inner surface of lower 6 coastal cartilages	• Xiphoid process &lineaalba&symphysispubis . • Lower fibers (conjoint tendon) is inserted into the pubic crest & medial part of pectineal line.	1- The fibres runs transversely. 2- It is lined by transversalis fascia. 3- Conjoint tendon:•It is the fused lower parts of aponeurosis of int. oblique &transversusabdominis near their insertion. • It passes in the med. 1/2 post wall of inguinal canal . • The fibres of trasversusabdominis arches at a higher level than int. oblique (no role in formation of ingunal canal. • The conjoint tendon is supplied by ilioinguinal nerve.
Rectus abdominis	• Pubic crest & front of symphysis pubis	• along a horizontal line into xiphoid process & outer surface of 5,6,7 coastal cartilage.	1- The lat. border of rectus abdominis is called lineasemilunaris. 2- It has 3-4 tendinous intersection (at xiphoid process, umbilicus, midway bet. the above 2 & one below the umbilicus).
Pyramidalis	• Pubic crest & front of symphysis pubis.	• Lower inch of linea alba	It is anatomical landmark to midline in supra-pubic incision.

LINEA ALBA:

It is a strong raphe situated in the middle line of anterior abdominal wall in between the 2 rectus abdominal muscle. It is formed by interdigiting fibers of the 3 aponeuroses of the muscles of anterior abdominal wall (after forming the rectus sheath). It is attached between xiphoid process &symphysis pubis. Above the umbilicus, lineaalba is approximately 1cm wide. Normally, contraction of 2 rectus

muscle will extirpate this wide lineaalba.Below the umbilicus, it is a narrow line which is distinguished by the insertion of pyramidalis muscles. It appears as umbilical scar.

RECTUS SHEATH:

It is a fibrous sheath built by the aponeuroses of the muscles of anterior abdominal wall.

Formation:

1. ***Above the costal margin:***

   Anterior wall: Aponeurosis of external abdominal at an oblique fashion.

   Posterior wall: Is inadequate, the rectus muscle lies on 5, 6, 7 costal cartilages.

2. ***From the costal margin to the midway point between umbilicus and symphysis pubis:***

   Anterior wall: Anterior lamina of internal oblique aponeurosis& External oblique aponeurosis.

   Posterior wall: Posterior lamina of internal oblique aponeurosis& transverses abdominisaponeurosis**.**

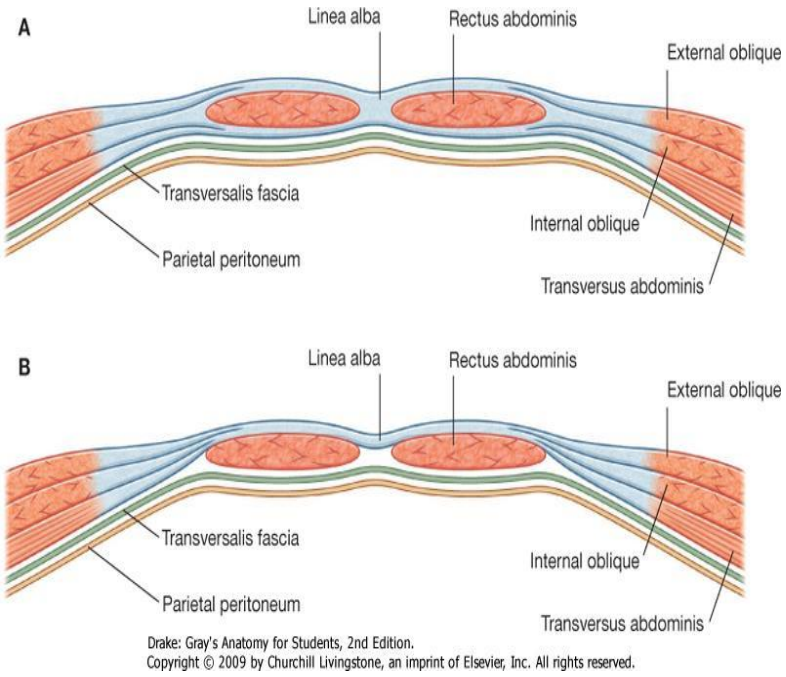
3. ***Below a midway point between umbilicus &symphysis pubis:***

Anterior wall: Aponeurosis of 3 muscles of anterior abdominal wall.

Posterior wall: It is insufficient, the rectus muscle lies on the transversalis fascia. The posterior wall of rectus sheath ends by forming arched border known as ***arcuate line***.

Contents:

- Rectus abdominis
- Pyramidalis
- Superior & inferior epigastric vessels
- lower 5 intercostal & subcostal nerves & vessels which moves through lateral to medial
- Lymph vessels.



FASCIA TRANSVERSALIS

It is a thin fascia which borders the antero-lateral abdominal wall.

- **Above:** It is continual with the fascia of diaphragm.
- **Posterior**: It enters into the formation of perirenal fascia (Zukercandle fascia)
- **Inferior:**

  a) Medially - Attached to pubic crest & medial part pectineal line.

  b) Laterally - Attached to inner lip of iliac crest & lateral half of inguinal ligament.

  c) Between a & b - passed in the thigh to create the anterior wall of femoral sheath.

- **Deep inguinal ring** is a deep opening in the transversalis fascia. At the deep inguinal ring, the fascia transversalis extends around the spermatic cord as internal spermatic fascia

Arteries of Anterior Abdominal Wall

A) Above the umbilicus:

1. Two Terminal branches of the internal thoracic artery:

a) Superior epigastric artery:

It runs down behind the 7th costal cartilage and moves into the rectus sheath to run down at the back of the rectus abdominis muscle upto the mark of the umbilicus and gets anastomosed with inferior epigstric artery.

b) Musculo-phrenic artery:

It runs downwards and sidewards along the costal margin.

2. Lower 5 (7-11) posterior intercostal arteries and the subcostal artery (branches of the descending thoracic aorta)

They run downwards and medially through the ***neuro-vascular plane*** separating the internal oblique and the transverses abdominis to enter the rectus sheath at the back of the rectus abdominis from lateral to medial side.

B) Below the umbilicus:

1. Superficial branches of the femoral artery

a) Superficial epigastric artery.

b) Superficial circumflex iliac artery.

2. Branches of the external iliac artery

a)Inferior epigastric artery:

Originates from external iliac artery just behind the inguinal ligament.It moves upwards and medially, medial to the internalinguinalring,infront of the arcuate line to pass through the rectus sheath behind the rectus abdominis. Finally ends at the level of the umbilicus by anastomosing with superiorepigastric artery.

Branches:

1) ***Cremasteric artery:***

It enters the deep inguinal ring as one of the components of the spermatic cord to supply the cremasteric muscle and terminates by anastomosing with the testicular artery.

2) ***Pubic branch:***

It moves down behind the lacunar ligament and superior pubic ramus to anastomose with the pubic branch of obturator artery. In 20% of cases, the obturator artery is not present and replaced by

abnormal obturator artery, which is a large pubic branch of **inferior epigastric artery**. This artery moves behind the free sharp border of the lacunar ligament and ***is more prone for injury during femoral hernia operation.***

b) Deep circumflex iliac artery:

Originates from **external iliac** artery just at the back of the inguinal ligament and proceeds upwards and laterally behind the inguinal ligament to enter the anterior superior iliac spine. Then **runs** on the inner lip of iliac crest where it pierces the transverses abdominis to enter into the **neurovascular plane**.

Branches:

1) **Muscular branches**
2) **Anastomatic branches** distributes in the anastomosis around anterior superior iliac spine.
3) **Ascending branch:** Anastomose with the lumbar and musculophrenic arteries.

Arterial anastomosis in the anterior abdominal wall:

1) ***Lateral anastomosis:*** between the ascending branch of deep circumflex iliac, lumbar and musclophrenic arteries.

2) ***Medial anastomosis:*** Uniting the superior and inferior epigastric arteries.

Applied anatomy:

This anastomosis is very essential to establish the collateral circulation after obstruction of common or external iliac arteries.

VEINS OF ANTERIOR ABDOMINAL WALL

A) Above the level of the umbilicus:

1) ***Superior epigastric vein:*** Passes in the rectus sheath deep to the rectus muscle to terminate in the internal thoracic vein (a tributary of brachiocephalic vein).
2) ***Lateral thoracic vein:*** Moves in the superficial fascia on the oblique direction of abdomen and thorax to terminate in the axillary vein.

B) Below the level of the umbilicus:

1) ***Inferior epigastric vein:*** enters the rectus sheath deep to the rectus muscle to terminate in the external iliac vein.
2) ***Superficial epigastric and superficial circumflex iliac veins:*** enters the superficial fascia of the lower part of the abdomen to terminate in the long saphenous vein (tributaries of femoral vein).

Venous anastomoses in the anterior abdominal wall:

1. Anastomosis in the middle of the ***superior and inferior epigastric*** veins in the rectus sheath. It joins the superior and inferior venacavae.
2. Anastomosis in the middle of the ***lateral thoracic*** vein and ***superficial epigastric*** veins. This anastomosis forms the ***thoraco-epigastric vein*** which links the superior and inferior venae cavae.

Applied anatomy:

Obstruction of inferior vena cava or iliofemoral veins, conclude in opening of this anastomosis with formation of ***dilated veins crossing the groin***.

3. Anastomosis in-between the systemic veins of the anterior abdominal wall (tributaries of superior and inferior venae cavae) and para-umbilied veins (tributaries of portal vein).
   - Opening of this ***porto-systomic anastomosis*** in portal hypertension ends in formation of **caput medusae**.

LYMPHATIC DRAINAGE OF ANTERIOR ABDOMINAL WALL

A) Superficial lymphatics: (Follow veins)

1) ***Above the umbilicus*** **-** Drains into the pectoral group of axillary lymph nodes.

2) ***Below the umbilicus*** **-** Drains into the superficial inguinal lymph nodes.

B) Deep lymphatics: (Follow arteries)

1) ***Above the umbilicus*** **-** Drains into the parasternal lymph nodes (along internal thoracic artery).

2) ***Below the umbilicus*** - Drains into the external iliac lymph nodes.

3) The ***deep surface of the umbilicus*** is drains into the lymphatics around the ligamentumteres in the falciform ligament, which again drains into the lymph nodes of portahepatis.

NERVES OF ANTERIOR ABDOMINAL WALL

A) Motor supply:

a) **The lower five intercostal and subcostal nerves:**

- Supplies 3 antero-lateral muscles of the abdominal wall.
- Enters through the ***neuro-vascular*** plane of the abdominal wall (inbetween the internal oblique and the transversusabdominus) and then enter the ***rectus sheath*** to runs through the rectus abdominis and the posterior wall of rectus sheath.

- Pierce the rectus abdominis and supplies it and again pierce the anterior wall of rectus sheath to terminate as the anterior cutaneous nerves lateral to the lineaalba.

b) **Iliohypogastric and ilioinguinal nerves**: (branches of the anterior primary ramus of L_1).

- P**ierce the psoas** major muscle to appear from under cover of its **lateral border** and descend laterally on the **quadratuslumborum** muscle at the back of the kidney with the iliohypogastric lying at a higher level than the ilioinguinal nerve.
- Both nerves pierce the transversusabdominis muscle and runs at the front of **neurovascularplane** between the internal oblique and the transversus muscles which supplies both until they reach the anterior superior iliac spine and then they pierce the internal oblique at variable points and moves medially between them and the external oblique as cutaneous nerves.
- The ***iliohypogastric*** nerve pierces the aponeurosis of the external oblique about 2 – 3 cm above the superficial inguinal ring to supply the skin above the symphysis pubis.

- On the other hand, the ***ilioinguinal nerve*** enters the inguinal canal below the spermatic cord and comes out through the superficial inguinal ring to supply the skin of the external genitalia and upper part of medial portion of the thigh. It also supplies the conjoint tendon.

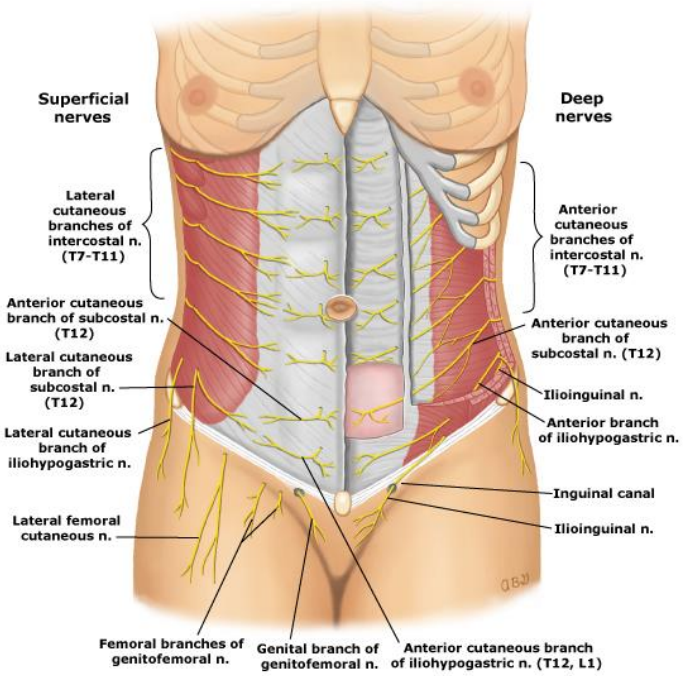
B) Sensory supply:

1. Lateral cutaneous branches of the lower 5 intercostal and subcostal nerves
2. Anterior cutaneous branches of the lower 5 intercostals and subcostal nerves
3. Cutaneous branches of the ***iliohypogastric*** nerve.
4. Cutaneous branches of the ***ilioinguinal***nerve. They supply the lining of the scrotum and the upper part of medial portion of the thigh.

The lower five intercostals, subcostal nerves and the branches of L_1 supply successive and almost horizontal bands of the skin lining the anterior abdominal wall

- Skin at the subcostal angle is innervated by 7^{th} thoracic nerve (T_7).
- Three nerves ($T_{7, 8, 9}$) innervates the region above the umbilicus.
- Skin at the level of the umbilicus is innervated by 10^{th} thoracic nerve (T_{10})
- Three nerves ($T_{11, 12,} L_1$) innervates the region below the umbilicus.

❖ Skin above the symphysis pubis is supplied by the iliohypogastric nerve (L_1).



ABDOMINAL INCISIONS

ESSENTIALS OF GOOD INCISION:

- To attain maximum accessibility.
- Extendible
- Minimal surface of scar after healing
- Minimum damage to the muscle tissue
- Eliminate nerve injury to avoid paralysis of muscles
- Minimal amount of bleeding
- Rapid healing time

TYPES OF ABDOMINAL INCISIONS:

1. The midline incision (through the linea alba):

Incision can be made on the upper or lower, right or left .

- **Advantages:**
  - Provides a good bloodless field.
  - Could be expanded above or below.
- **Disadvantages:**

Healing time is prolonged due to poor supply of blood to the lineaalba.

2. The Paramedian incision:

Incision is made with 2.5 – 4 cm laterally to the midline and runs parallel to it. The anterior rectus sheath is pulled out and opened the rectus muscle is retracted laterally and the posterior wall of rectus sheath along with the parietal peritoneum are again opened in the same plane as the skin incision.

- **Advantages**:
  - Provides a bloodless clear field.
  - Could be expanded.
  - Good healing in a short period as the rectus abdominisfurnish the incision with its arterial supply.

3. Transverse incision:

Incision is made to cut through the lateral abdominal muscles in order to expose some internal organs .eg: ***Pfannenstiel incision*** for the uterus. This can lead to weakness of the abdominal muscles.

4. Right or left subcostal incision:

- It is useful for exposure of the gall bladder on the right side or the spleen on the left side. The skin incision starts at the middle line and extends 1inch below and parallel to the costal margin.
- **Advantage:**

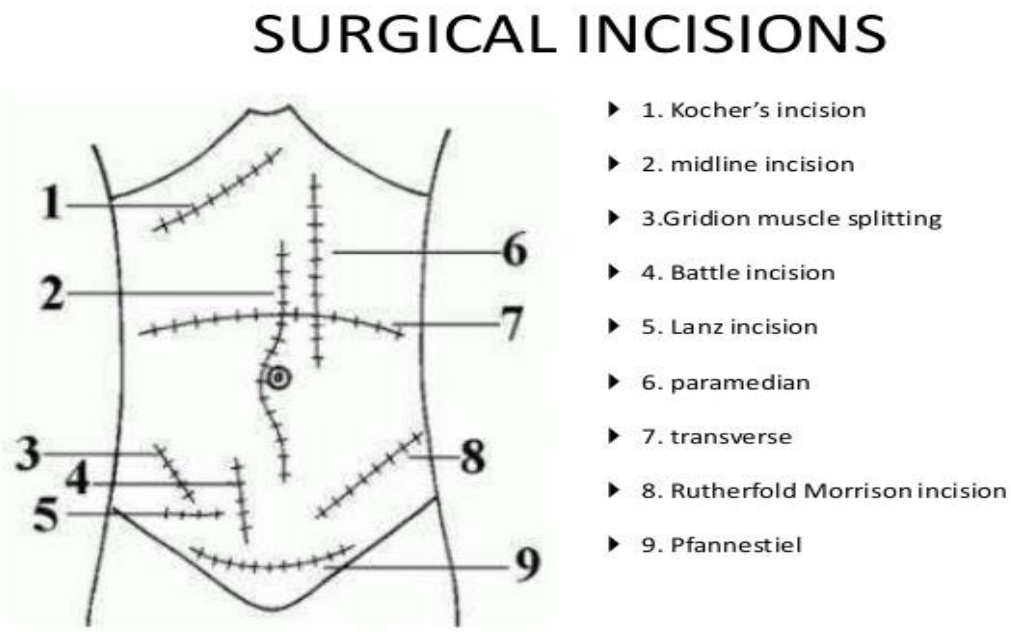
  Gives good exposure.
- **Disadvantage:**

  High incidence of incisional hernia due to muscle cutting incision.

5. McBurney's incision (grid iron):

- Incision is made to expose the vermiform appendix.
- **Method:** An oblique direction incision centered at Mc Burney's point (point which is at the junction of the lateral 1/3rd and medial 2/3rd of a line extends from the umbilicus to the anterior superior iliac spine).Open the external oblique, internal oblique and the transverses abdominiswithout cutting them in the line of their fibers and finally retract them. Then open the fascia transversalis and parietal peritoneum.

- **Advantage:** Muscle splitting incision which doesnot cause any damage to the abdominal muscles.



WOUND CLOSURE IN LAYERS

Closure of the Abdominal Incision

Closing the abdominal wall is a big challenge for all abdominal surgery. It is most important for the surgeons during residency training period. The methods of closure are often based on local traditional method and the preferences of the surgeon are often reluctant to change these methods later on

in his or her career. Abdominal closure is performed in a multiple fashions and there is much abundance of differently tailored studies on this matter.

The goal of wound closure is to repair and restore the functions of the abdominal wall after a surgical procedure. The optimal method should be so technically simple and easier that its results are as good for the hands of the trainee as they are equal to the experienced surgeon. It should leave the patient with a reasonably aesthetic scar and most importantly it should minimize the quantity of wound rupture, incisional hernia, wound infection, and sinus formation.

Closure of the Peritoneum

Traditional surgical dogma says that since all layers of the abdominal wall tissues are opened during an abdominal incision, all layers should be approximated when the incision is closed. Closure of the peritoneum is done in the proposition that normal anatomy will be restored and the risk of infections and wound herniation will be reduced and adhesions will be minimized.

But in current studies it has been found that no difference is seen in the incidence of wound dehiscence or hernia between the nonclosure of peritoneum and the closure of peritoneum respectively.

Corresponding results have been reported found in randomized trials comparing closure versus nonclosure of the peritoneum in open cholecystectomy incisions, lateral paramedian incisions and gynaecological andobstetric incisions. From these studies it is summarized that closure of the peritoneum is not necessarily indicated and not needed. It is accompanied with longer operative time and importantly closure of peritoneum produces postoperative pain and there are some studies suggesting that it may even cause increased formation of adhesions.

Closure of the Fascia

Closure of the abdomen is made in layers. A layered closure technique restores the anterior and posterior aponeurotic sheaths in 2 different layers with the posterior portion of layer generally involving the peritoneum.

Few studies have shown that an increased incidence of wound dehiscence and incisional hernia due to infections and delayed wound closure are reported and some studies show no difference in these complications but none of the studies demonstrate and explains the advantage of the use of subcutaneous drain in preventing the wound healing. Rates of wound sepsis and sinus formation have also been studied in randomized control trials and which do not depend on usage of subcutaneous drain.

It has been declared that a negative suction drain will reduce the cause of infections by draining the seroma. The theoretical advantage of using this negative suction drain is to eliminate the wound complications during postoperative period.

The use of reabsorbable versus non-reabsorbable sutures in closing the fascia has been debated for a longer period. Approximately rates of 17% for scar pain and 8% for suture fistula using permanent suture have been a twitch interest in the use of reabsorbable sutures. Reabsorbable sutures however bear an internal loss of tensile strength during the vulnerable postoperative period and may conclude in an increase in wound disruption and ventral hernia.

The early use of the absorbable catgut suture has been shown to have a high incidence of wound rupture and incisional hernia due to its early degradation.

To overcome these problem synthetic absorbable sutures with delayed degradation were introduced and used to establish the advantages of absorbability with tensile strength compared to nonabsorbable materials.

There are conflicting of interest in the literature about failure of wounds when nonabsorbable and absorbable suture are compared in randomized clinical trials.

The resorbable sutures polyglycolic acid (Dexon), polyglactic acid (Vicryl), polydioxanone (PDS), and polyglyconate (Maxon) have shown to be equally effective as that of nonabsorbable suture with respect to wound dehiscence and incisional hernia. Fewer studies however demonstrate that polydioxanone and polyglactic acid polymer absorbable suture may be associated with an increased frequency of incisional hernia when weighed against nonabsorbable suture.

Another selection of suture is monofilament versus multifilament suture. Multifilament suture is known to provide with a better growth environment for bacteria and is accompined with a higher incidence of wound sepsis when comparing with monofilament suture. Bacteria are enter into the fibers of multifilament suture by capillary action and survive there by escaping phagocytosis. Wound sepsis is a major risk factor for incisional hernia but in spite of these considerations multifilament suture has not been shown to result in a greater incidence of wound failure when compare to monofilament closure. Monofilament catgut suture also attains for special consideration.

It is a reactive material that shows an inflammatory reaction and is accompanied with a higher incidence of wound infection than other monofilament materials.

Our experience and interpretation of the literature is that the optimal surgical method of closing the abdominal wound is a continuous mass closure. This method appears to reduce the incidence of wound rupture and is considerably time consuming, least expensive and decreases the incidence of incisional hernia, wound infection or sinus formation. The choice of suture material is more complex.

We suggest to use a resorbable suture showing delayed degradation such as polydioxanone. Other resorbable materials are appropriate as well, but catgut should not be used. Among nonresorbable sutures monofilament suture is preferred.

Subcutaneous Tissue Closure

With the higher rate of prevalence of obesity in developed countries, treatment of the subcutaneous tissues in abdominal wound closure becomes increasingly important.

The vascular supply to the subcutaneous tissue of the abdominal wall is less, leading its susceptibility to soft-tissue infection. Similarily if this level of the abdominal wall contains an adequate space for promoting accumulation of seroma the risk of infection increases.

Only one prospective randomized trial has been conducted to evaluate the value of suturing the subcutaneous fat. Using a subcostal incision for cholecystectomy, the literature demonstrated no significant differences in complications between closure and nonclosure of the subcutaneous tissues. Wound seepage however was reduced in incisions in which the subcutaneous layer was closed.

Individual trials failed to demonstrate any benefit of suture closure in wounds which is less than 2 cm of subcutaneous tissue but confirm the reduction of wound disruption in wounds with greater than 2 cm of tissue.

We do not regularly close the subcutaneous layer of the wound. On some occasions with obese patients, we will use a series of simple, interrupted, absorbable polyglactic acid (vicryl) sutures to reapproximate the subcutaneous layer. These stitches are inverted to bury the knots within the wound.

Skin Closure

If the surgical site is highly contaminated (class III or class IV wound), the skin should be kept open to heal by secondary intention or by delayed primary skin closure. Many closure techniques for clean (class I) and clean-contaminated (class II) wounds are available for the skin. Methods are

interrupted skin suture, subcuticular suture, surgical skin stapler, surgical tape for skin closure, and skin adhesive glues. Main aim of skin closure are tissue approximation, decreasing wound infection, acceptable cosmetic, and decreasing postoperative pain.

These goals should be achieved with a simple, rapid, and cost-effective method. Four randomized controlled studies have compared skin staples tosubcuticular sutures. In all studies, no difference in the rate of wound infection could be demonstrated. Three of these studies revealed minimal postoperative pain and less postoperative pain killer requirement in wounds closed with subcuticular suture. One of these study also described a high cosmetic result in subcuticular closures over surgical skin stapler; however, these cosmetic difference minimized over time and became less significant by 1 year.

Adhesive tapes are often used to re approximate skin edges in simple lacerations. Following abdominal surgery, adhesive tapes are useful to cover skin incisions closed by subcuticular suture, where they serve to further re approximate skin edges and to dress the wound. The use of adhesive tape without suture material, closure was compared to interrupted silk skin suturing of abdominal wounds in one early trial. There zero difference in the rate of wound infection could be matched. The tapes were relatively more comfortable

and patients preferred them over sutures, but wide scarring occurred more frequently with surgical tapes.

Synthetic glues are attaining popularity in skin closure of surgical wounds. When compared with routine skin-closing technique including sutures, stapler, or adhesive tapes, some cyanoacrylate glues for skin closure have been found to be comparable in effectiveness and safety for repair of lacerations.

They are applied more rapidly and decrease the amount of required wound care by serving as their own dressings. In elective abdominal procedures with small and large (>4 cm) incisions, these glues have been shown in clinical trials to have similar outcomes with respect to wound strength when compared to traditional techniques, although there are conflicting data on wound healing, cosmetic, and postoperative pain. We prefer to close skin with a running, nonbraided, absorbable suture in a subcuticular technique. Adhesive tapes are placed over the closed incision without the use of skin glues.

NORMAL WOUND HEALING

The ideal response to wounding, as seen in lower forms of life, is total regeneration with reconstitution of the original structure. This is lost to a great extent in phylogenetically advanced organisms, leading to repair by scar formation. This in turn has lead to complications like delayed healing, contractures, weak scars, stenosis, adhesions and proliferated scars,etc.

It has always been recognized for long that the efficacy and the speed of repair are influenced by some factors relating to the part as well as to the local milieu. The recent advancements in our knowledge about collagen structure and chemistry as well as the role of the various soluble factors, matrix proteins, angiogenesis, and fibroblastic proliferation in wound healing has induced several studies to modulate and manipulate these factors to optimize healing with minimal yet strong scar. Most of the work has involved healing of skin wounds as this is most easy to control, modulate and study.

PROCESS OF WOUND HEALING

Healing involves **regeneration** and **repair by scar formation.** Regeneration requires two important steps to be fulfilled.

- The tissue should comprise of either labile or stable cells, i.e. be capable of replication e.g. most epithelial cells and some of the other connective

tissues.

- The structural framework should be intact. In all other situations repair with scar formation occurs through the medium of granulation tissue.

HEALING BY 1st INTENTION OR PRIMARY UNION

In a clean surgical wound, with approximation of the edges, the following phases occur in a continuum with overlap.

1. **Acute inflammation**:

Starts soon after wounding and is wellestablished by 24 hours.

This results in

- Vascular response & coagulation cascade activation à formation of clot à primary wound binding as well as fibrin scaffold for later events.
- Platelets and inflammatory cells accumulated and secretion of several Growth factors. By 24 hours inflammation is established and predominantly neutrophilic, to be replaced by macrophages by day3.

2. **Epithelialisation:**

Starts within 24 hours, as mitotic activity in few cells peripheral to the discontinuity, along with migration of proximal cells towards the gap. A continuous monolayer over the clot forms by 48 hours, followed now by increase in number of layers and maturation by 5-7 days. Basement membrane is depositedconcurrently.

3. **Granulation Tissue Formation:**

Comprising of angiogenesis and fibroblastic proliferation and migration. Proliferation starts by 24 hours. and granulation tissue formation by 3-5 days. **Angiogenesis** occurs by proteolysis ofbasement membrane of existing capillaries, outward migration of endothelium with proliferation proximal to leading edge, maturation into capillary tubes and reformation of basement membrane. The proliferated fibroblasts have contractile filaments and are also called **myofibroblasts.**

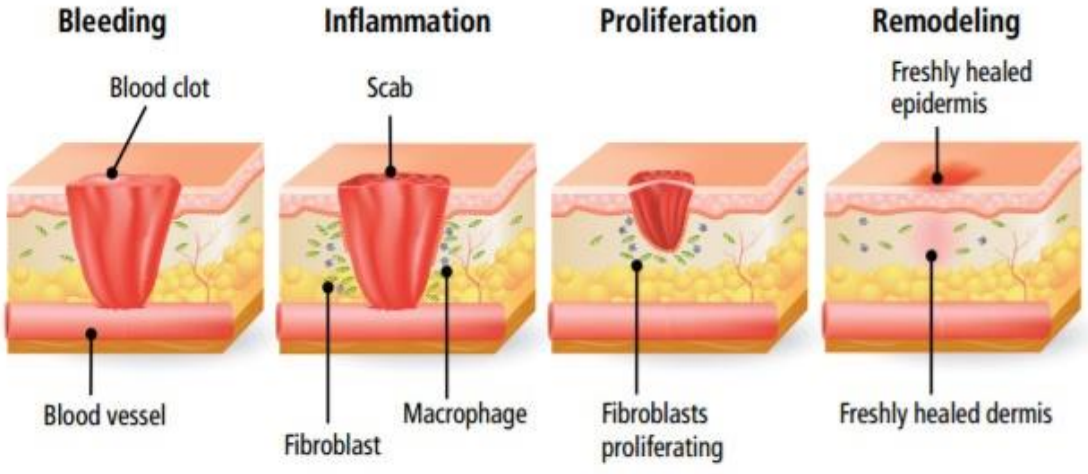
4. **Fibroplasia:**

Collagen deposition in the extracellular matrix is demonstrable by 3-5 days and progressively increases. Fibroblasts synthesise the three chains. These are secreted as triple helix after hydroxylation and glycosylation. They are then spliced in the extracellular matrix followed by linking and fibril formation. Crosslinking of these results in fibre formation.

By one week the wound strength is 10% of normal skin. Continued crosslinking& structural modification increases the strength rapidly till 4 weeks, after which it slows to a plateau by three months at 70% to 80% of normal. Simultaneous devascularisation and subsiding of inflammation causes blanching of thescar.

5. **Remodelling:**

Degradation of collagen occurs simultaneously with synthesis leading to reorientation of fibres across stress lines. Though maximal strength is attained by 3-6 months, remodelling may continue till 6-12 months leading to a flat, mature scar. Degradation is carried out by **metalloproteases,** which are promoted by some of the growth factors, and kept in check by Tissue inhibitors(TIMP).



SECONDARY UNION

This occurs where wound edges are not approximate (eg. Ulcer), resulting in a gap to be filled. Though the steps are the same, it differs from Primary union in the following respects:

- **Epithelialisation**

  occurs over the granulation tissue and hence starts later (by 4-5 days) and takes longer.

- **Granulation tissue**

  is more exuberant (proud flesh) and may need to be trimmed for optimal healing. Resulting scar is larger and takes longer to form.

- **Wound contraction**

  This is decrease in size of gap to be filled and occurs between 3-14 days. It is mediated by the contractile action of myofibroblasts and can reduce the gap by upto 80% thereby decreasing the time taken for healing as well as scarsize.

FACTORS AFFECTING WOUND HEALING

General and local that affect wound healing are:

1) **General:**

Nutritional Deficiency:

Protein deficiency, especially chronic deficiency of methionine and cysteine decreases production and cross linking of collagen.Vitamin C affects the intracellular hydroxylation of procollagenfibres, essential for helix formation .Zinc is essential for activity of metalloproteases.

Age:

Wounds heal better in the young than in the old. This is due to age perse or associated vascular and nutritional status . There is a fall in collagen production as well as cross linking in the elderly.

Steroids:

Glucocorticoids suppress angiogenesis and wound contraction . They also suppress collagen synthesis by attenuating heat shock protein which plays a key role in protein assembly and packaging. DOCA and anabolic steroids promote healing.

a. Within normal ranges of ambient temperature, higher temperature quickens and cold slows down healing.
b. Certain drugs like NSAIDS (Ibuprofen) compromise collagen density and wound strength in experimental animals.
c. Diabetes mellitus*:* Healing is affected mainly due to themicro-angiopathy.The associated neuro-pathy, immune derangement and hyper glycaemia do not play a major role. Neuro-pathy*:* Denervation does not affect healing. However the repeated trauma in an area of sensory loss hampers the healing process.

2) Local:

Tissue O2 is an important factor which gets affected by anaemia, cardio-respiratory problems and smoking or by ischemia, stasis or by vasculitis. Deficient tissue O_2 affects all stages of wound healing. Excessive and continuing inflammation as in the presence of infection or foreign body delays healing and results in poor scar. Radiation exposure inhibits wound contraction and delays healing. UV exposure in contrast has been shown to promote healing .Drying of surface inhibits epithilialization. Mobility (as against fixity to bone) favours healing though movement hampers healing as well as modulates orientation of collagen bundles .Direction of wounding relation to natural folds and creases.Tension across

incision and excessive collagenisation leads to disfiguring large scar / contracture. Dermal / subcutaneous support is important in wounds which have tension across them. Certain anti-bacterialsare cytotoxic to fibroblasts and delay healing. FGF is shown to protect.

SOLUBLE FACTORS IN WOUND HEALING

Several **growth factors** released from damaged tissue as well as native and infiltrating cells are now recognized as important in proliferation and migration of cells as well as in collagen synthesis. Their effects have been demonstrated in experimental and in-vitro situations with a goal towards uncomplicated healing and ideal scar **(Also scarless healing)** Some of the important ones and their actions are as follows:

- **PDGF** Chemotactic to inflammatory cells, migration of endothelium & fibroblasts and mitogenic to fibroblasts.
- **EGF/TGFα** proliferation of epithelial and mesenchymal cells and fibronectin synthesis.
- **FGF** (esp. basic) a Potent angiogenesis a mitogenic to endothelium & fibroblasts and collagen deposition.
- **TGF β** Fibroblast migration and collagen deposition by

differentiation and mitogenic Inhibition.

- **KGF** (rel. to FGF) a Proliferation and differentiation stimulus to epithelium.
- **IGF-1** a Synthesis of proteoglycans, fibroblast and endothelial proliferation and chemotactic to macrophage.

- **IL-1 & TNF** a Fibroblast proliferation, collagen synthesis &degradation.

Wound dehiscence:

Partial or complete disruption of an abdominal laparotomy wound with protrusion (evisceration) of abdominal contents

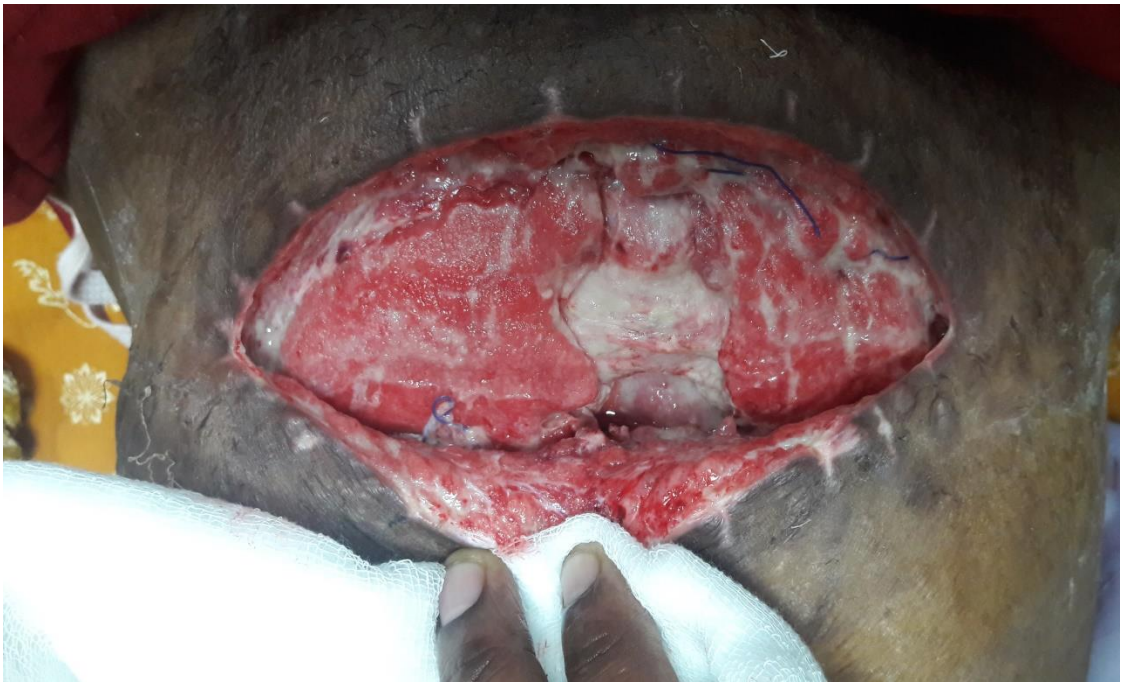
- Wound failure is termed as wound dehiscence and incisional hernia.
- Differentiated by duration and wound healing of overlying skin

PARTIAL

disruption of fascial edges without evisceration with loose fascial sutures. occasionally, fibrin covered intestinal loops

COMPLETE –

full separation of fascia & skin ,intestinal loops and abdominal contents protruded out.



Clinical manifestations

- Are Seen between post operative day 7 – 14
- May develop without warning, following straining or removal of sutures
- May be preceded by a sero-sanguineous discharge

ETIOLOGIC FACTORS OF ABDOMINAL WOUND DEHISCENCE

Etiology of wound dehiscence

Following are the factors responsible for wound dehiscence

- Faulty technique of fascialclosure

- Emergencysurgery
- Intra abdominalinfections
- Malnutrition
- Advancedage
- Chronic steroidsuse
- Wound complications (Hematoma – infection, seroma)
- Previous wounddehiscence
- Increased intra abdominal pressure (coughing, abdominaldistension)
- Radiation therapy and chemotheraphy,Systemic disease (uremia,diabetes mellitus)

Wound Infection:

The most common causative factor in the development of wound dehiscenceis wound infection.Wound infection causes impaired wound healing by interfering with normal healing, resulting in a wound which has less collagen also the collagen is not highly cross linked as in a normally healed wound. This weakness leads to later postoperative abdominal wound dehiscence.

Abdominal Incisions:

The lower incidence of wound dehiscence in the transverse or oblique incision is due to low tension in the suture line in the transverse or oblique incision compared to those over the midline, however this have not been proved clinically. Similarly higher rate of wound dehiscence in incisions in the upper abdomen when compared to that in lower abdomen also has not been proved by clinical studies yet. But clinical studies have proven that wound dehiscence is very low in muscle splitting incision. but the disadvantages of this incision is they a limited access to the abdominal cavity.

Sutures and suture technique:

Absorbable suture materials that lose their 80% of their tensile strength within 14days, thus wound dehiscence are morecommon when they are used for closure.

Multifilament suture materials are associated with more wound infection because bacteria are being enclosed within the interstices of multifilament sutures, where they are protected from phagocytosis.

If a single suture in an interrupted closure is very tight, ischemia will develop in the tissue enclosed. In interrupted closure technique more knots,

more foreign materials will be deposited in the rectus leading to wound infection and sinus formation. The rate of incisional hernia is more if the SL:WL ratio is less than 4. If the stitch length is more than 5cm rate of wound infection is high. Excessive tension placed on the suture reduces local blood flow leading to necrosis of that area and is associated with increased woundinfection.

Age:

Wound dehiscence increases as the age of the patient increases. The reason for poor Wound healing in older patients is not only due to age but also due to the extent of dissection and the potential for intra operative contamination are greater in operations conducted in older patients (i.e., extensive resection for cancer).

Obesity:

Excessive fat in the omentum and the subcutaneous tissue results in increase strain on the wound with all body movements in the early postoperative period. Associated poor muscle tone and lack of muscle mass also are causative factors in the development of wound dehiscence. Surgery in obese patient is associated with an increased potential for postoperative pulmonary complications, wound infection and pulmonary embolus.

Debility and Malnutrition:

Malnourished patients, particularly those who have lost a significant amount of weight over a relatively short period as seen in those patients with malignancy and chronic diseases before operation have poor wound healing and immunity and hence they have high chance of wound dehiscence. This malnutrition can be assessed by levels of serum albumin and other proteins these patients are at higher risk for poor wound healing.

Carbohydrates

Carbohydrates, together with fats, are the primary sources of energy in the body and consequently in the wound healing process. The energy requirements for wound healing consist mainly of the energy required to carry out collagen synthesis in the wound.

Fatty acids

Several unsaturated fatty acids must be supplied in the diet as deficiencies of these lipids cause impairment in wound healing in animals and humans. This impairment is due to the role phospholipids play as constituents of the cellular basement membrane and the participation of prostaglandins in cellular metabolism and inflammation. Total parenteral nutrition (TPN) is the most common cause of essential fatty acid deficiency.

VITAMIN C:

This vitamin plays a very important role in the development of wound dehiscence. vitamin c is needed for the cross linking of the collagen fibers .deficiency of this leads to faulty cross linking leading to poor wound healing and contraction which ultimately leads to wound dehiscence

Branched-chain amino acids

The branched-chain amino acids valine, leucine and isoleucine have been used to treat liver disease and have an additional role in retaining nitrogen in sepsis, trauma, and burns. Branched-chain amino acids support protein synthesis serve as caloric substrates. Despite these useful properties, high supplements of branched- chain amino acids have not proved to be of any significant benefit in improving woundhealing.

Glutamine

Glutamine is the richest amino acid in our human organism. The process of gluconeogenesis involves the shuttling of alanine and glutamine to the liver for conversion to glucose, which is used peripherally as fuel to power certain aspects of wound healing. Glutamine also is an important precursor for the synthesis of nucleotides in cells, including fibroblasts and macrophages. Glutamine is as an energy source for lymphocytes and is essential for

lymphocyte proliferation. Finally, glutamine has a crucial role in stimulating the inflammatory Immune response occurring early in woundhealing.

Postoperative Pulmonary Complication :

Immediately after surgery bucking due to improper anaesthesia technique or vigorous coughing during sedation aggravated due to the underlying lower respiratory tract infection, will produce tear in the suture line. Also postoperative coughing and straining are also etiologic factors for subsequent wound dehiscence.

Steroids:

The use of steroids has negative effect to wound healing. Wounds heal poorly in patients receiving long term steroid therapy ,the reason for this being, the normal inflammatory responses that are necessary to initiate wound healing are lost with chronic steroid use with consequent impaired deposition and polymerization of collagen in the wound.

Chemotherapy:

The early postoperative administration of chemotherapy is associated with impaired wound healing. This is the reason why medical oncologist prefer to give chemotherapy to malignant patient who have been operated after wound have healed.

Ascites:

Patients with cirrhosis and ascites not only have increased abdominal pressure caused by peritoneal fluid, but also often are severely malnourished.

Peritoneal Dialysis:

The patients undergoing continuous ambulatory peritoneal dialysis will have uremia ,obesity, marked anaemia, and chronically elevated intraperitoneal pressure caused by presence of the dialysate ,these all factors will lead to the development of wound dehiscence.

Type of surgery:

Certain types of surgeries have an increased chance of wound dehiscence. These include

- laparotomy for generalized or localized peritonitis,
- perforated peptic ulcer surgeries,
- appendicitis,
- diverticulitis,
- acute pancreatitis,
- intra abdominal malignant diseases,
- chronic inflammatory bowel disease

- Re-surgery through the same incision within the 1st 6month of surgery.

The cause of the wound dehiscence is not due to the surgery but due to the presence of the above mentioned factors.

Type of surgical wounds:

Surgical wounds are classified based on the presumed magnitude of bacterial load at the time of surgery.

Clean wounds (Class I) :

Wounds which don't have infection, only skin microfolora potentially contaminate the wound, and no hollow viscus that contains microbes is entered.

Class ID wounds are similar except that a prosthetic device (mesh) is inserted.

CLEAN / CONTAMINATED (CLASS II) :

wound in which a hollow viscus such as the respiratory, alimentary or genitourinary tracts with indigenous bacterial flora is opened under controlled circumstances without significant spillage of contents.

Eg: Elective colorectal cases have classically been included as class II cases,

CONTAMINATED WOUNDS (CLASS III)

wound include open accidental wounds encountered early after injury, those with extensive introduction of bacteria into a normally sterile area of the body due to major breaks in the sterile technique, gross spillage of viscus contents such as from the intestine

eg: perforation.

DIRTY WOUNDS(CLASS IV)

Wound include traumatic wounds in which a significant delay in treatment has occurred and in which necrotic tissue is present, these contain overt infection as evidenced by the presence of purulent material, and those created to access a perforated viscuss accompanied by a high degree of contamination.

Miscellaneous Factors:

There are numerous factors responsible for wound dehiscence. There are etiologic factor cannot be identified in every patient without a wound dehiscence. The role of certain minerals like zinc and manganese in extra cellular fluid has not been established definitively, although they are thought to influence the maintenance of connective tissue integrity. The effect of the

anticoagulants such as warfarin sodium have adverse effect on fibrinogenesisby decreasing the fibrogenesis. Both Warfarin and heparin increase postoperative wound haematoma, modestly increasing the risk of incisional hernia.

PREVENTIVE MEASURES FOR ABDOMINAL WOUND DEHISCENCE

Most of the factors responsible for wound dehiscence cannot be corrected preoperatively or cannot be influenced by the surgeon.

Patient age or over weight cannot of course be influenced, when an emergency laparotomy of a grossly contaminated abdomen is required. However the suture technique which is completely in the hands of the surgeon ,which is one of the most common cause for woundcomplications can be corrected.

Incisions:

The choice of laparotomy incision depends on the time it takes to open and close the abdomen. The access and the visualisation of the required organ must be considered. The rate of wound complications varies with midline, paramedian, lateral paramedian, oblique, transverse and muscle -splitting incisions. When restricted access to the abdomen is sufficient, muscle -splitting incisions is recommended because of the much much lower wound

complications .the reason for this being , the muscles produce a shutter mechanism that tends to close the wound.

Suture Materials:

Monofilament suture materials are associated with a lower rate of wound infection when compared to the multifilament .the reason for this being the bacteria being enclosed within the interstices of multifilament sutures and they get protected from phagocytosis.

Non absorbable suture materials allow support of the wound during the entire healing period and have been used with good results. With slowly absorbable monofilament suture materials that retain an acceptable strength for atleast 6week.

The method of wound closure:

The laparotomy incisions should be closed by a continuous suture technique in single layer. Why this is preferred is, less foreign material and minimal knots are deposited ,this also allows tension to adjust evenly along the suture line in stead of spreading over a single knot. intermittent Self-locking knots should be used for the anchor knot.

In vertical midline incisions, sutures taken should mainly include aponeurotic tissue and be placed at least 1cm from the wound edge. The length

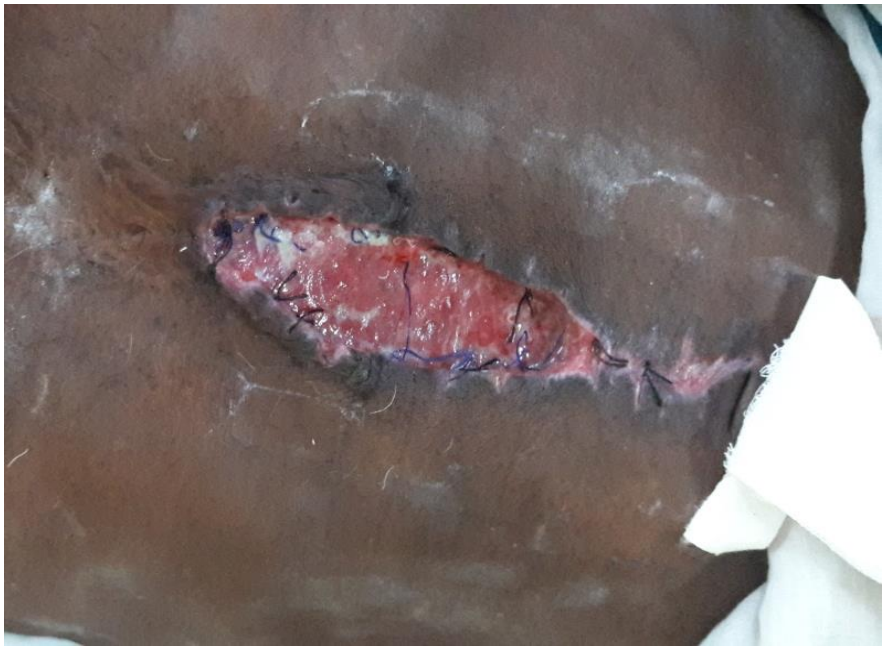
of each suture should be less than 5cm; otherwise it will be associated with an unnecessary high rate of wound infection. Including peritoneum, muscle or subcutaneous fat in the suture is not needed as this doesn't have any advantage infact it has adverse effects on wound healing. The surgeon should be careful not to place excessive tension on the suture.

Wound infection:

In wounds that are expected to have intra- operative contamination, the incidence and subsequent infection is reduced by administration of appropriate antibiotic prophylaxis. The following pre requisite are to be followed

- gentle tissue dissection,
- use of minimal amounts of suture material
- use of minimal amounts of electrocautery,
- avoidance of stoma through the wound,
- irrigation of the wound during closure to remove debris, blood clots and foreign matter,
- meticulous haemostasis reduces the incidence of wounddehiscence

INFECTED WOUND WITH WOUND DEHISCENCE



WOUNDDEHISCENCE WITH BILE LEAK

MANAGEMENT OF WOUND DEHISCENCE:

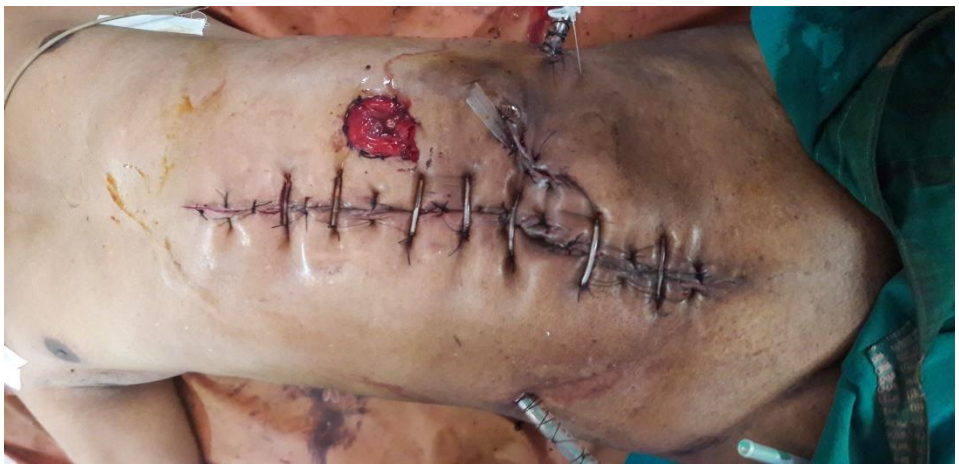
Treatment of wound dehiscence depends on

- extent of fascial separation
- presence of evisceration
- significantintraabdominal contamination.

A small dehiscence in upper aspect of upper midline incision can be managed conservatively. This can be done by with saline-moistened gauze and using an abdominal binder. If evisceration of content is seen then the eviscerated intestines must be covered by sterile, saline-moistened towel then the patient must be shifted to operation theatre after fluid resuscitation. In emergency theatre , through exploration of the abdominal cavity should be done to rule out presence of septic focus or an anastomotic leak that would have predisposed to the dehiscence. Treatment of infection is of critical importance before attemptingclosure.

When technical mistakes are made which had lead to the wound dehiscence and the fascia is strong and intact, then primary closure is can be done. If the fascia is infected or necrotic, debridement is done first,which is then followed by examination about the approximation of the fascia. If after debridement the edges of the fascia cannot be approximated without tension,

then fascia can be closed with absorbable mesh or the recently developed biologic prosthesis



TENSION BAND WIRING

METHODOLOGY

60 patients undergoing emergency laparotomy within the inclusion criteria in General Surgery Department OfGovtRajaji Hospital for a period of 8 months where included in our study after getting proper written informed consent. This patients was followed for 2 week post operative period and were divided into two categories and followed up and findings were collected.A central randomization was performed. The randomization sequence was based on a computer-generated list.

In the control group (Group B), the laparotomy wound is sutured in layers without keeping the subcutaneous drain and were observed and monitored.

In the intervention group (Group A), the laparotomy wound is sutured in layers after keeping the subcutaneous drain and were observed and monitored.

MATERIALS AND METHODS

Patients presenting at the emergency department who meet the inclusion criteria were recruited into the study. After obtaining a detailed history, all patients presenting with acute abdominal pain were isolated in the emergency ward.

DIAGNOSTIC CRITERIA FOR PERITONITIS

Clinically

• Acute pain abdomen, nausea, vomiting

• Fever, Tachycardia

• Guarding, rigidity

• Absent or decreased bowel sounds

On investigations

• Leukocytosis

• X-ray abdomen erect-free air under diaphragm, distended bowel loops.

• USG Abdomen-Free fluid in peritoneal cavity

LAPAROTOMY FINDINGS

Whether pus fluid is present or abdominal cavity is contaminated with bowel contents.

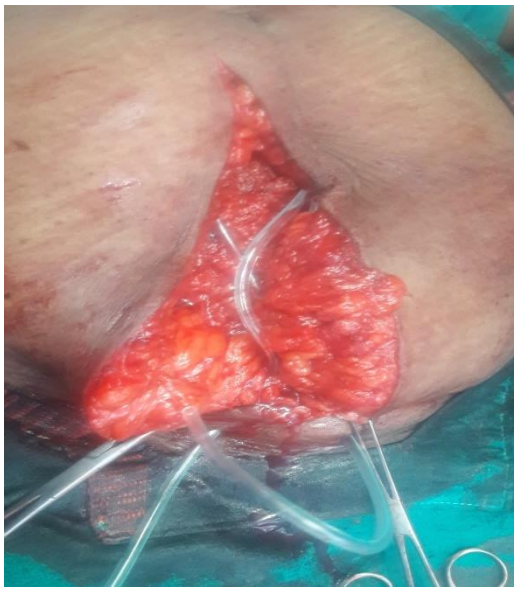
Patients who met the above mentioned diagnostic criteria for peritonitis were included in the study.

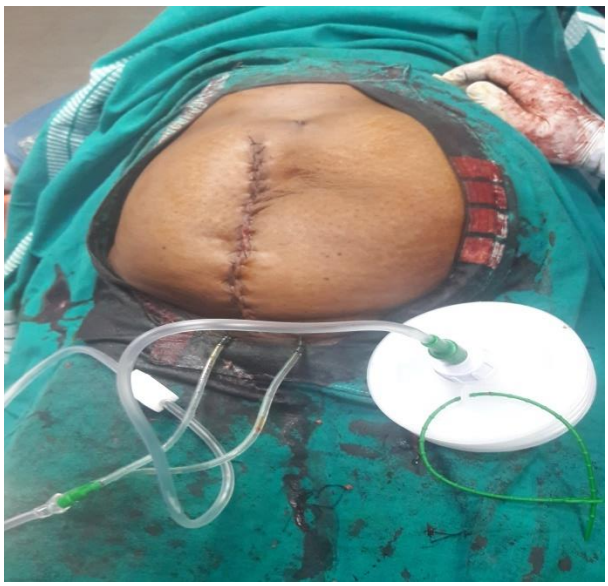
Consent for participation in the study was obtained from the patients after pre-consent counselling. The consent for participation in the study was obtained simultaneously with the consent for surgery.

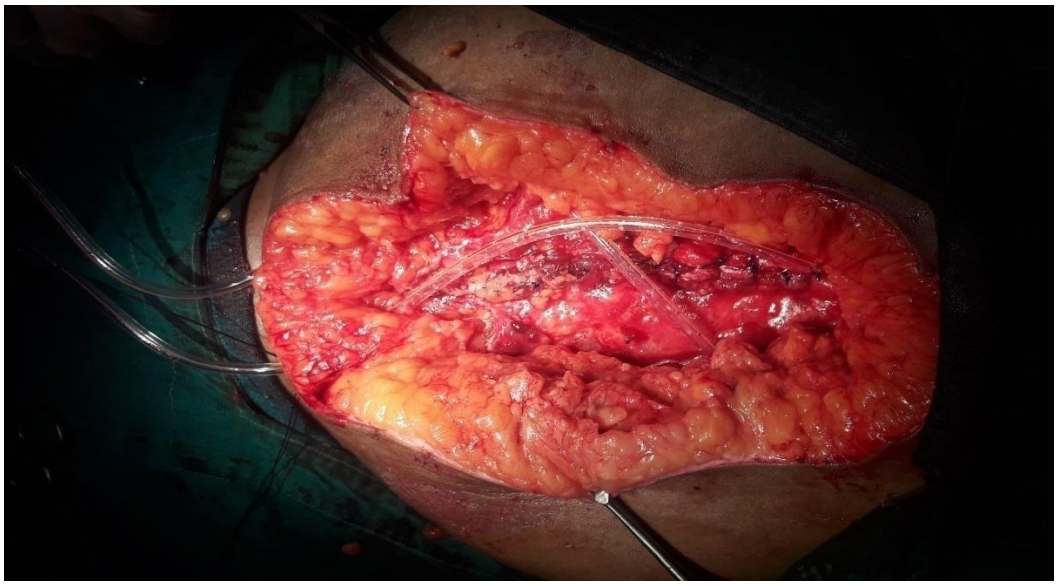
30 cases underwent abdominal wall closure with subcutaneous suction drain and were assigned to Group A. 30 other cases underwent conventional primary skin closure and were assigned to Group B.

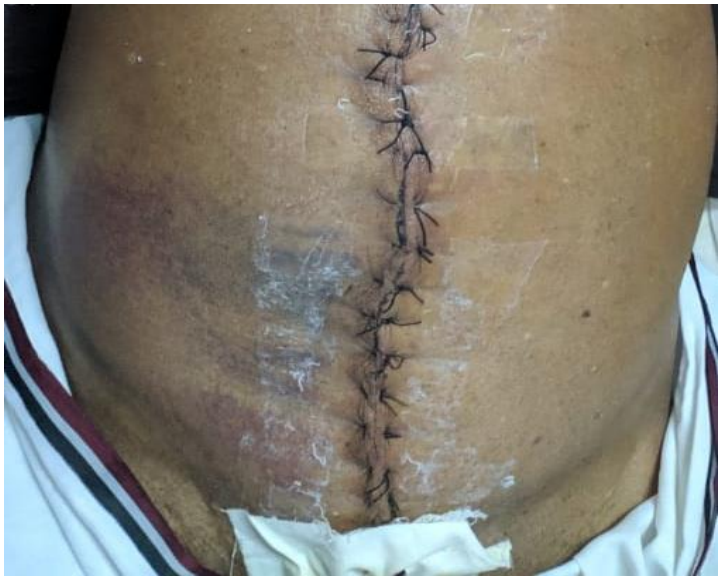
Type of drainage tube-closed wound suction system.

Suction drain (perforated catheter tube) was placed subcutaneously and connected to a container which had the negative suction pressure capacity.









Follow up

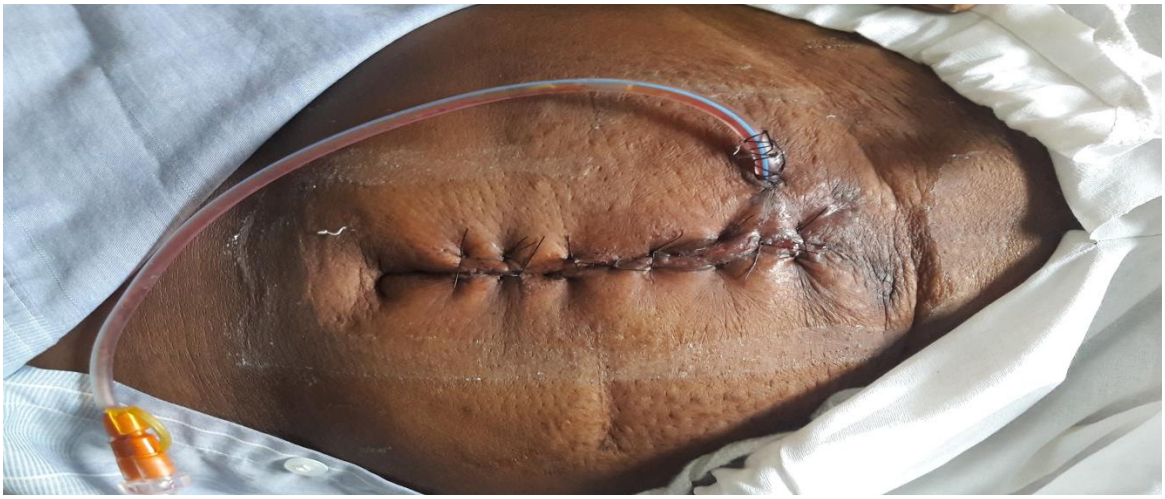
DOS: On table pus c/s was sent. Empirical antibiotic therapy was started

POD 2/3/4 : If wound discharge/sero-purulent discharge in bellow container was present, pus c/s was sent.

POD 3: Antibiotic changed according to on table pus c/s result

POD 4/5/6 : Comparison of on table pus c/s with wound/bellow container discharge pus c/s was done to identify whether infection is due to abdominal cavity infection or hospital acquired cross infection.

The collection in the bellow container was emptied and measured every post-operative day. If the collection in the drain was negligible for two consecutive days and wound apposition was good, the suction drain was removed. Average period of suction drain placement was analyzed.



Wound infection:

superficial incisional SSI was assessed based on the CDC criteria for surgical site infection as follows:

Infection involving only skin or sub-cutaneous tissue of the incision and at least any one of the following:

• Pus drainage with micro organism from the skin incision.

• Micro Organisms isolated from the skin incision site or from the collected fluid.

• At least one signs among the inflammatory response are- pain or tenderness, localized swelling, redness, or heat.

Wound dehiscence was identified as per the definition, i.e. postoperative separation of musculoaponeurotic layers of the abdominal wall. Post-operative follow up was for 30 days. The patients were reviewed at two and four weeks from the date of discharge.

RESULTS

Indications of surgery

The following were the indications for surgery in all peritonitis cases (on table finding - pyoperitoneum / fecal peritonitis) in the order of decreasing frequency:

• PERFORATION(most common)

1. Small bowel perforation: duodenal/ileal, obstruction with pyo peritoneum
2. Gastric-antro pyloric
3. Large bowel perforation: Transverse colon

• INTESTINAL OBSTRUCTION

1. Obstructed incisional hernia
2. Obstructed umblical/supra umblical hernia
3. Volvulus

• Spleen injury with various grading

• Parietal wall abscess with pyo-peritoneum, post appendicectomyfecal peritonitis..

The difference in the indications of the surgery in both the groups was not statistically significant, i.e. the indications were similar in both the groups.

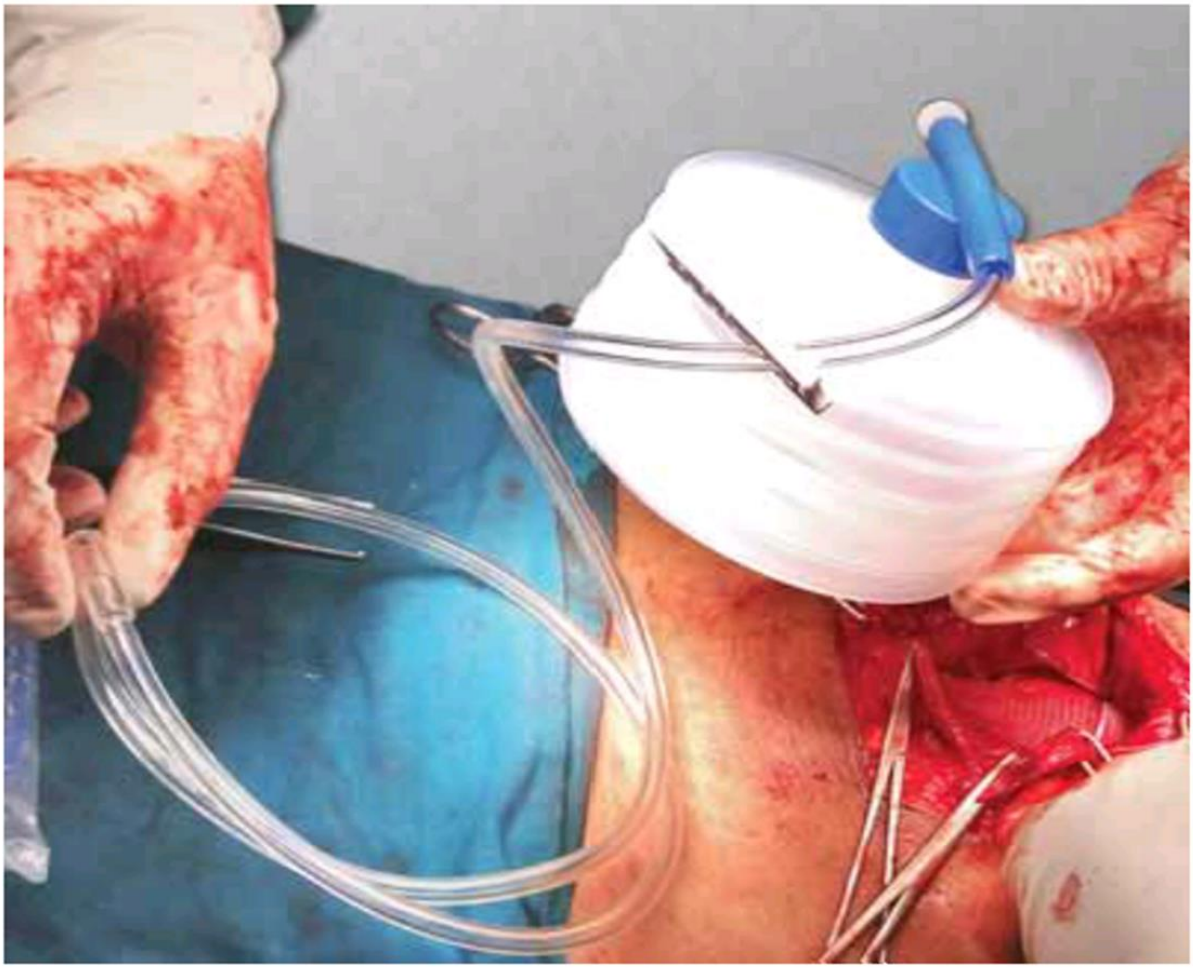
Type of incision

The most common incision performed was midline laparotomy in both the groups, right subcostal were the other incisions performed. There was no statistically significant difference in the type of incisions performed between the two groups.

Two methods

Using long artery forceps is pushed from subcutaneous plane inside out and skin is incised using a scalpel, proper track is created. Drain is held with the forceps and pulled into the subcutaneous plane. Drain is secularly fixed using Romangarter technique

When inbuilt trocar is present it is passed from subcutaneous plane inside out and skin is incised using a scalpel, proper track is created. Drain is held with the forceps and pulled into the subcutaneous plane. Drain is secularly fixed using shoe-lace sutures.



Drain have tendency to become occluded or clogged resulting in retained fluid that can contribute to infections. Thus efforts must be made to maintain and assess patency when they are in use either by irrigating the tube with saline or gently squeezed and released at different points to remove clogging

Pain can be reduced by analgesics

As advantages are more than disadvantages hence this study has more significance

SOURCE OF DATA:

All patients satisfying inclusion criteria admitted in General Surgery Department, Government Rajaji Hospital for a period of 8 months

METHOD OF COLLECTION OF DATA:

All patients undergoing midline laparotomy within the inclusion criteria was followed for 2 week period and were divided into two categories and followed up and findings were collected.

INCLUSION CRITERIA

Patients who gave informed and written consent

Patients undergoing Emergency abdominal surgeries 10cm surgical incision minimum and having 2 of the pre-operative risk factors for wound dehiscence

Poor nutritional status (clinical cachexia or hypoalbuminemia)

Intra – abdominal infection(>3days old)

Use of corticosteroids in last 12months(>10mg/dl prednisolone)

Uremia

Hemodynamic instability (BP <90mm Hg)

Haemoglobin <10mg/dl (due to perioperative blood loss or anemia

Predicted abdominal distension

Chronic pulmonary disease

Clinical jaundice (bilirubin >3mg/dl)

Diabetic mellitus

Age >60yrs

Exclusion criteria

Patients <18yrs

Incisional length >10cm

OBSERVATION AND RESULTS

Statistical analysis

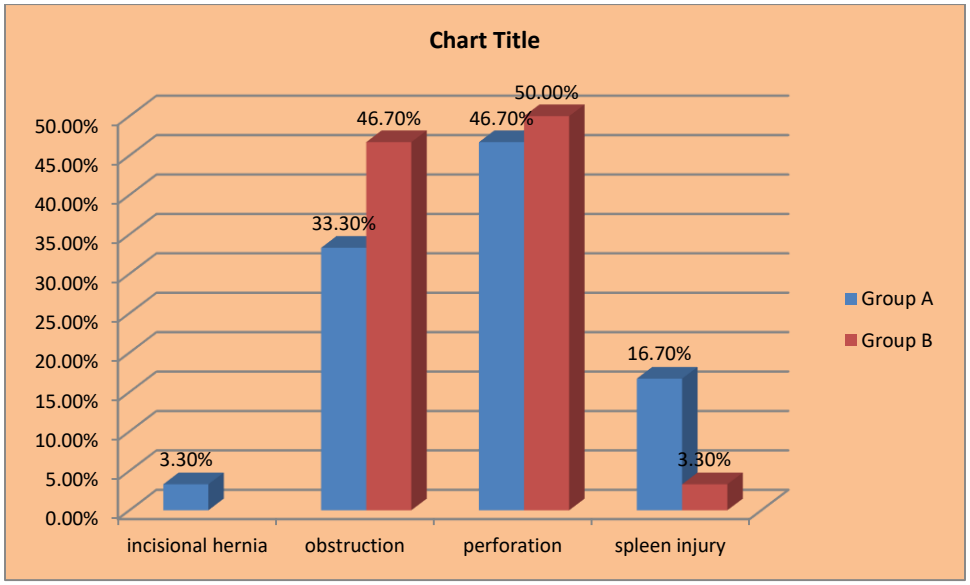
Data were analyzed using SPSS Version 17.0 software version. Descriptive statistics were performed; Categorical data were presented as percentage and frequency. Continuous data was analysed to assess normality using Shapiro wilks test. Based on the distribution of the data, Mann whitney U test was used for intergroup comparison of continuous variables. Categorical data were analysed using Chisquare test or Fisher's exact test. p value <0.05 is considered as statistically significant.

Results

Comparison of study groups based on indications for surgery.

Demography indications of surgery	Group A N(%)		Group B N(%)		p value
Incisional hernia	1	3.30%	0		
Obstruction	10	33.30%	14	46.70%	
Perforation	14	46.70%	15	50.00%	0.224
Spleen injury	5	16.70%	1	3.30%	

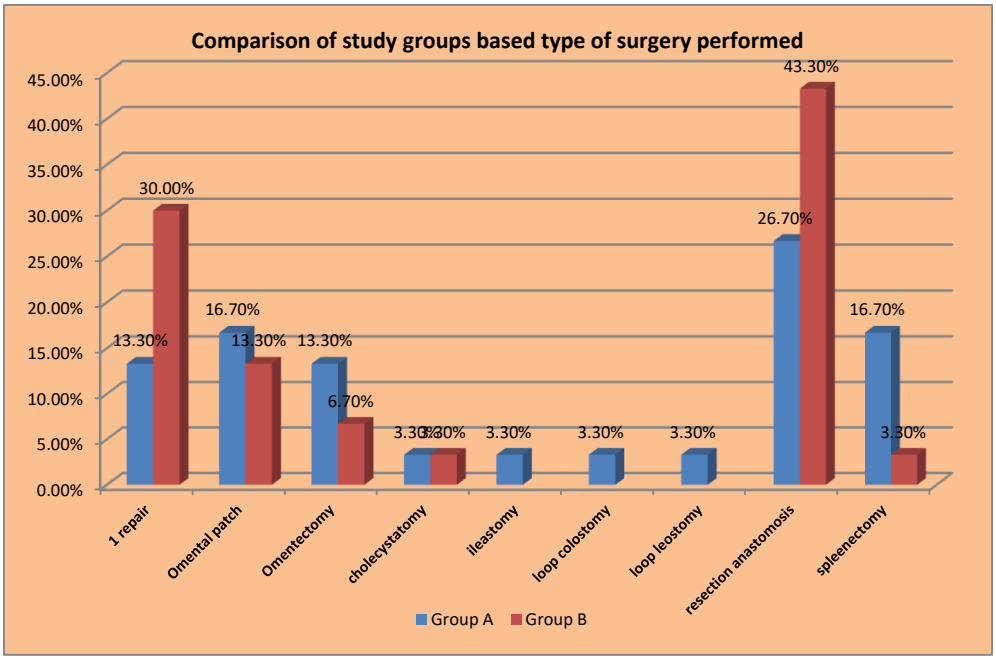
Chisquaretest ; * shows (p<0.05)



Comparison of study groups based type of surgery performed

Type of surgery	Group A (N=30)		Group B (N=30)		p value
Primary repair	4	13.30%	9	30.00%	0.297
Omental patch	5	16.70%	4	13.30%	
Omentectomy	4	13.30%	2	6.70%	
cholecystatomy	1	3.30%	1	3.30%	
ileastomy	1	3.30%	0		
loop colostomy	1	3.30%	0		
loop leostomy	1	3.30%	0		
resection anastomosis	8	26.70%	13	43.30%	
spleenectomy	5	16.70%	1	3.30%	

Chisquaretest ; Not significant

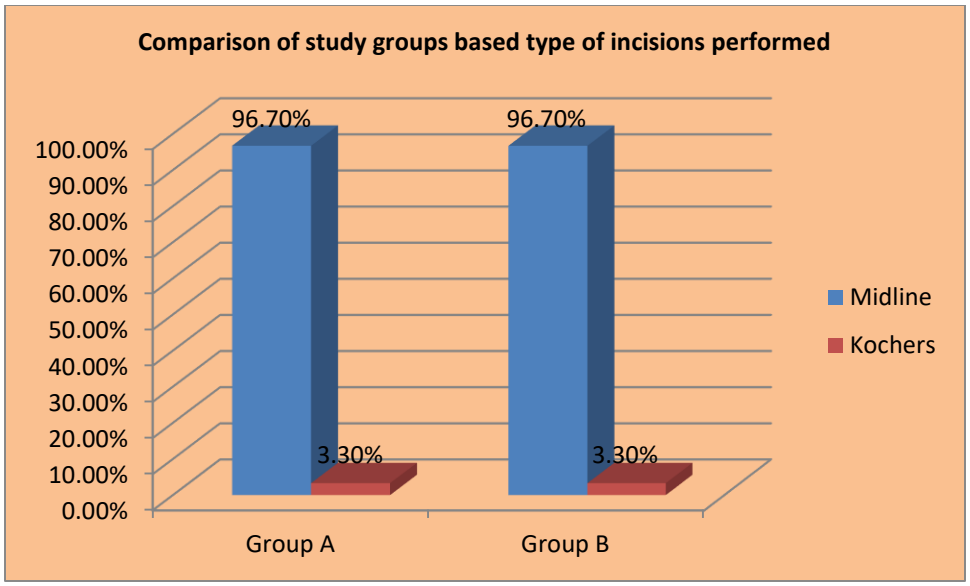


There was no significant difference in between two groups in case selecting and surgery which performed. All the cases were randomized and obtained the statistical analysis.

Comparison of study groups based type of incisions performed

Type of incision	Group A		Group B		P value
Midline	29	96.70%	29	96.70%	1.00
Kochers	1	3.30%	1	3.30%	

Chisquaretest ; Not significant

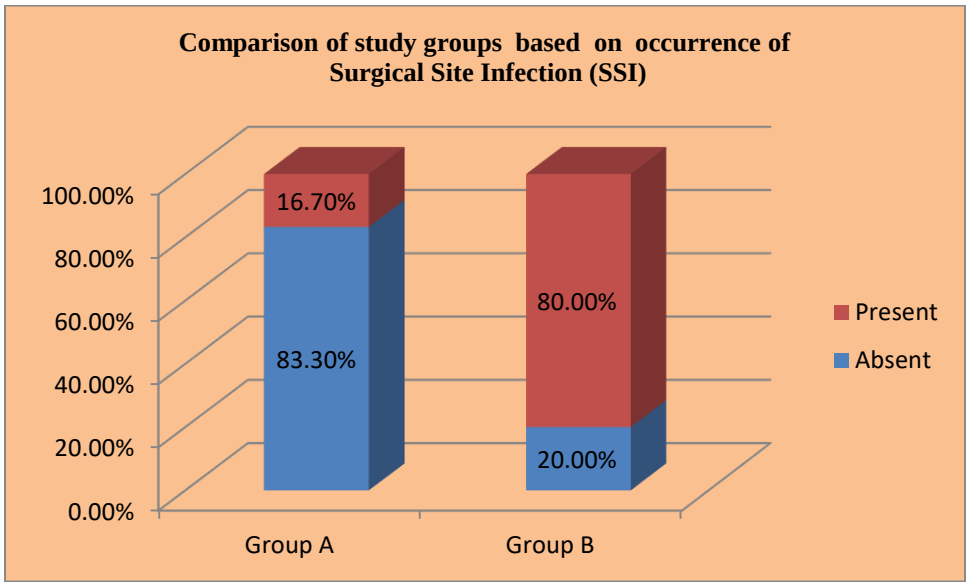


There were 29 Midline laparotomy incisions were performed in each study group A and control group B. only one kochers incision was performed in each group. This statistical analysis showed equality among the groups.

Comparison of study groups based on occurrence of Surgical Site Infection (SSI)

SSI	Group A		Group B		P value
Absent	25	83.3%	6	20%	0.001*
Present	5	16.7%	24	80%	

Fisher's exact test ; shows *($p<0.05$)

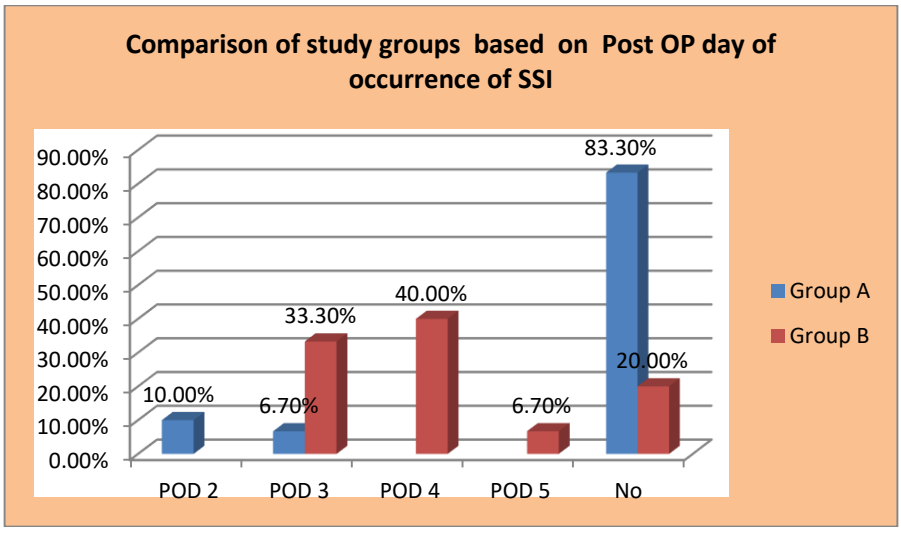


Surgical site infection were observed among group A, out of 30 cases only 5 (16.70%) of them developed subsequent SSI with drain. In case of control group B 24 cases (80.00%) out of 30 cases developed surgical site infection. Significant P value is 0.001.

Comparison of study groups based on Post OP day of occurrence of Surgical Site Infection (SSI)

SSI					
	Group A		Group B		P value
POD 2	3	10.0%	0		
POD 3	2	6.7%	10	33.3%	0.001*
POD 4	0		12	40.0%	
POD 5	0		2	6.7%	
No	25	83.3%	6	20.0%	

Fisher's exact test; shows *(p<0.05)

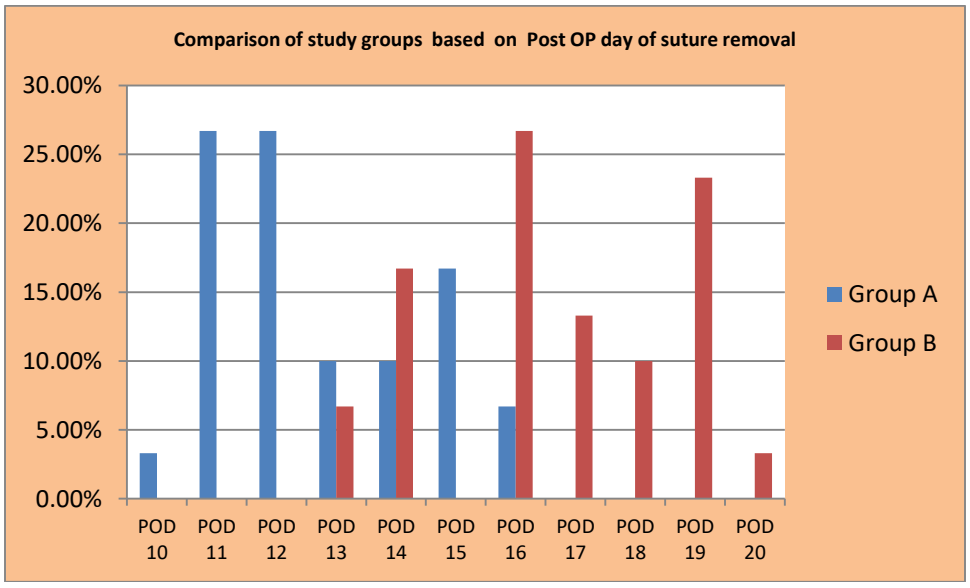


Incidence of SSI in 5 cases (16.70%) in GROUP A are detected 3(10.0%)cases at POD 2 & 2(6.7%) cases at POD 3. In GROUP B 24 cases (80.00%) were developed with SSI, 10 cases (33.3%) at POD 3, 12 cases (40.0%) at POD 4, 2 cases (6.7%) at POD 5. Hence this analysis clearly states that all the cases in GROUP B developed SSI at later post operative days when compared with GROUP A.

Comparison of study groups based on Post OP day of suture removal

	Group A		Group B		P value
POD 10	1	3.3%	0		
POD 11	8	26.7%	0		
POD 12	8	26.7%	0		
POD 13	3	10.0%	2	6.7%	0.001*
POD 14	3	10.0%	5	16.7%	
POD 15	5	16.7%	0		
POD 16	2	6.7%	8	26.7%	
POD 17	0		4	13.3%	
POD 18	0		3	10.0%	
POD 19	0		7	23.3%	
POD 20	0		1	3.3%	

Fisher's exact test; shows *($p<0.05$)

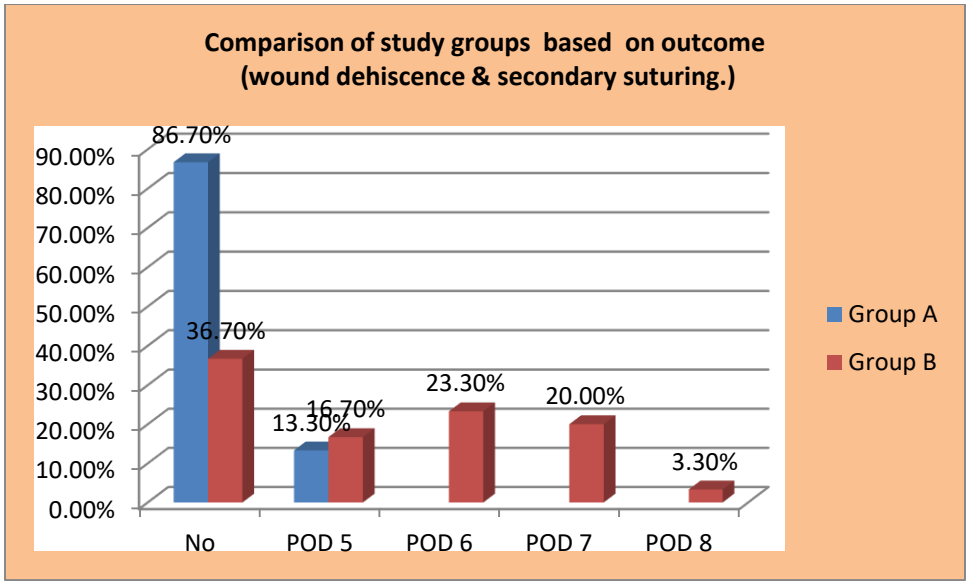


All the cases in GROUP A were suture removed and discharged earlier when compared with GROUP B

Comparison of study groups based on outcome (wound dehiscence & secondary suturing.)

wound dehiscence & secondary suturing.	Group A		Group B		P value
No	26	86.7%	11	36.7%	
POD 5	4	13.3%	5	16.7%	
POD 6	0		7	23.3%	0.001*
POD 7	0		6	20.0%	
POD 8	0		1	3.3%	

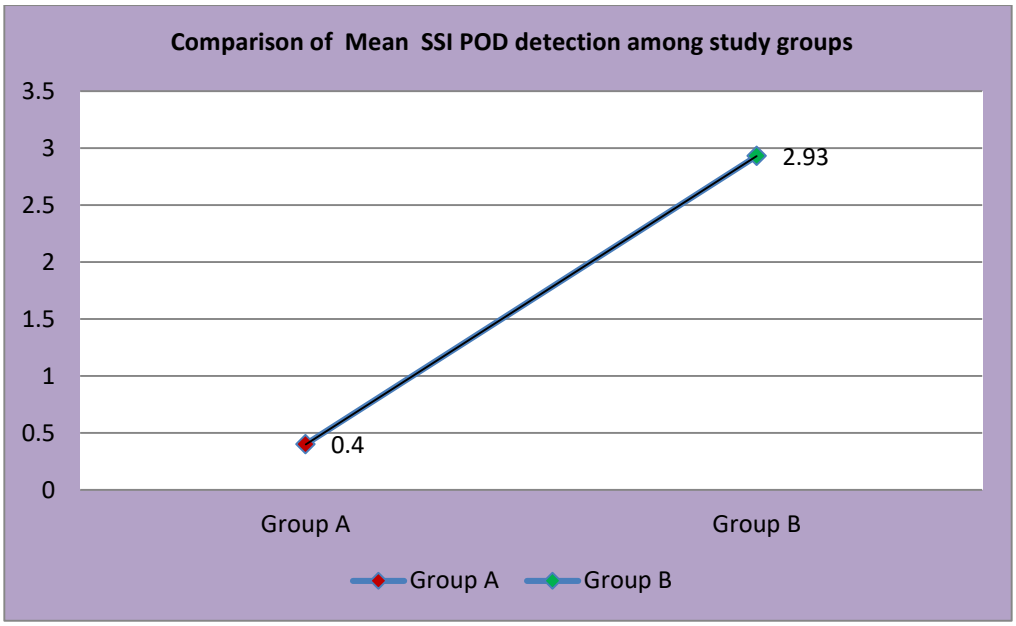
Fisher's exact test; shows *(p<0.05)



Comparison of Mean SSI POD detection among study groups

Groups	N	Mean	Std. Deviation	Std. Error Mean	Median	IQR	
Group A	30	0.4	0.932	0.17	0	0	0.001*
Group B	30	2.93	1.596	0.291	3	1	

Mann whitney U test; shows *($p<0.05$)



SUMMARY

WOUND DEHISCENCE and SURGICAL SITE INFECTIONS are devastating incident that can cause pain, mental distress, infectious complications, and financial burdens for the patient, as well as complications including evisceration and reoperation. Surgeon expertise, type of incision, suturing material, surgical site infection, nutritional status, persistent cough, abdominal distension, leakage of pancreatic enzyme, anaemia, obesity, diabetes, jaundice, old age, emergent operation, particular procedures such as colon surgery, and late wound healing due to malignancy have all been suggested to predispose patients to abdominal wound dehiscence and SSI.

Some of these factors are unavoidable .To lower the incidence of abdominal wound dehiscence, it has been recommended to improve patient nutritional status, prevent possible infection of surgical sites by administration of prophylactic antibiotics, restrict plasma glucose in diabetic patients, provide efficient oxygenation, maintain hemodynamic stability and sufficient tissue perfusion, control hypothermia, apply proper surgical techniques, and reduce tissue tension.

Keeping negative suction drain in the subcutaneous plane and closing the laparotomy wound will essentially evacuate the wound seroma collection during post operative day. This seroma collection will be noticed at the earliest when there is a formation of pus. Therefore wound healing is improved.

In **my study included only patients at a high risk for developing wound dehiscence who would benefit the most with the help of NEGATIVE SUCTION DRAIN.**

CONCLUSION

Surgical site infection is commonly due to abdominal cavity infection rather than nasocomical infection. Subcutaneous suction drainage tube is an effective method of abdominal wall closure in cases of peritonitis when compared to conventional primary skin closure without drain as it significantly reduces the incidence of wound infection, dehiscence, wound secondary suturing and duration of hospital stay in SSI.

Subcutaneous suction drainage tube enables improved rate of recovery and finally decreased morbidity and early rehabilitation. Hence, subcutaneous suction drainage tube should be considered in abdominal wall closure in patients who undergo surgery for emergency laparotomy.

REFRENCES

1.Kirby JP, Mazuski JE. Prevention of surgical site infection SurgClin N. 2009;89:365-89.

2. Edwards PS, Lipp A, Holmes A. Preoperative skin antiseptics for preventing surgical wound infections after clean surgery. Cochrane database.Syst Rev. 2004:CD003949.

3. Homer-Vanniasinkam S. Surgical site and vascular infections: treatment and prophylaxis. Int J Infect Dis. 2007;11(1):17-22

4. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR.Guidelines for prevention of surgical site infection. Infect Control HospEpidemiol. 1999;20:250-78.

5. Baier PK, Glück NC, Baumgartner U, Adam U, Fischer A, Hopt UT. Subcutaneous Redon drains do not reduce the incidence of surgical site infections after laparotomy. A randomized controlled trial on 200 patients.Int J Colorectal Dis. 2010;25:639-43.

6. Hellums EK, Lin MG, Ramsey PS. Prophylactic subcutaneous drainage for prevention of wound complications after cesarean delivery-a metaanalysis. Am J Obstet Gynecol. 2007;197:229- 35.

7. Numata M, Tanabe H, Numata K, Suzuki Y, Tani K, Shiraishi R, et al. The efficacy of subcutaneous penrose drains for the prevention of superficial surgical site infections. Jpn J Gastroenterol Surg. 2010;43:221-8.

8. Watanabe A, Kohnoe S, Shimabukuro R, Yamanaka T, Iso Y, Baba H, et al. Risk factors associated with surgical site infection in upper and lower gastrointestinal surgery. Surgery Today. 2008;38(5):404-12.

9.Sharma A, Deeb AP, Iannuzzi JC, Rickles AS, Monson JRT, Fleming FJ. Tobacco smoking and postoperative outcomes after colorectal surgery. Ann Surg. 2013;258(2):296-300.

10. Sørensen LT. Wound healing and infection in surgery. The clinical impact of smoking and smoking cessation: a systematic review and metaanalysis. Arch Surg. 2012;147(4):373-83.

11. Kwaan MR, Sirany AME, Rothenberger DA, Madoff D. Abdominal wall thickness: is it associated with superficial and deep incisional surgical site infection after colorectal surgery? Surg Infect. 2013;14(4):363-8.

12. van Walraven C, Musselman R. The surgical site infection risk score (SSIRS): a model to predict the risk of surgical site infections. PLoS ONE. 2013;8(6):Article ID e67167.

13. Soper DE, Bump RC, Hurt WG. Wound infection after abdominal hysterectomy: effect of the depth of subcutaneous tissue. Am J Obstet Gynecol. 1995;173(2):465-71.

14. Fujii T, Tsutsumi S, Matsumoto A. Thickness of subcutaneous fat as a strong risk factor for wound infections in elective colorectal surgery: impact of prediction using preoperative CT. Digest Surg. 2010;27(4):331-5.

15. Cheadle WG. Risk factors for surgical site infection.Surg Infect, 2006;7: S7-11.

16. Diana M, Hübner M, Eisenring MC, Zanetti G, Troillet N, Demartines N. Measures to prevent surgical site infections: what surgeons (should) do. World J Surg. 2011;35(2):280-8.

17. Kosins AM, Scholz T, Cetinkaya M, Evans GRD. Evidence-based value of subcutaneous surgical wound drainage: the largest systematic review and meta-analysis. Plastic Reconst Surg. 2013;132(2):443-50.

18. Bohnen JMA. Use of drains in Abdominal Wall Hernias: Principles and Management, R. Bendavid, Ed, Springer, New York, NY, USA; 2001:328.

19. He XD, Guo ZH, Tian JH, Yang KH, Xie XD. Whether drainage should be used after surgery for breast cancer? A systematic review of randomized controlled trials.MedOncol. 2011;28:S22-30

20. Chelmow D, Rodriguez EJ, Sabatini MM. Suture closure of subcutaneous fat and wound disruption after cesarean delivery: a meta-analysis. Obstetrics Gynecol. 2004;103(5):974-80.

21. Saunders DI, Murray D, Pichel AC, Varley S, Peden CJ. Variations in mortality after emergency laparotomy: the first report of the UK emergency laparotomy network. Br J Anaesth. 2012;109(3):368-75.

22. Imada S, Noura S, Ohue M. Efficacy of subcutaneous penrose drains for surgical site infections in colorectal surgery. World J Gastro Surg. 2013;5(4):110-4.

23. NICE, "Clinical Guideline 74—prevention and treatment of surgical site infection," NICE, October 2008, http://www.nice.org.uk/nicemedia/pdf/CG74NICEguideline.pdf.

24. A. Watanabe, S. Kohnoe, R. Shimabukuro et al., "Risk factors associated with surgical site infection in upper and lower gastrointestinal surgery," Surgery Today, vol. 38, no. 5, pp. 404–412, 2008.

25.HPS, Surveillance of Surgical Site Infection. Annual Report for Procedures Carried out from: January 2003–December 2011,Health Protection Scotland, Glasgow City, UK, 2012, http://www.documents.hps.scot.nhs.uk/hai/sshaip/publications/ssi/ssi-2011.pdf.

26.HealthProtectionAgency, Surveillance of Surgical Site Infections in NHS Hospitals in England, 2010/2011, Health Protection Agency, 2011.

27. R. L. Smith, J. K. Bohl, S.T.McElearney et al., "Wound infection after elective colorectal resection," Annals of Surgery, vol. 239,no. 5, pp. 599–607, 2004.

28.V. Satyanarayana, H. V. Prashanth, B. Bhandare, and A. N.Kavyashree, "Study of surgical site infections in abdominal surgeries," Journal of Clinical and Diagnostic Research, vol. 5, no.5, pp. 935–939, 2011.

29. P. Astagneau, C. Rioux, F. Golliot, and G. Br¨ucker, "Morbidity and mortality associated with surgical site infections: results from the 1997–1999 INCISO surveillance," The Journal of Hospital Infection, vol. 48, no. 4, pp. 267–274, 2001.

30.A. Sharma, D. M. Sharp, L. G. Walker, and J. R. T. Monson,"Predictors of early postoperative quality of life after elective resection for colorectal cancer," Annals of SurgicalOncology, vol.14, no. 12, pp. 3435–3442, 2007.

31. D. E. Reichman and J. A. Greenberg, “Reducing surgical site infections: a review,” Reviews in Obstetrics and Gynecology, vol.2, no. 4, pp. 212–221, 2009.

32. N. Kashimura, S. Kusachi, T. Konishi et al., “Impact of surgical site infection after colorectal surgery on hospital stay and medical expenditure in Japan,” Surgery Today, vol. 42, no. 7, pp.639–645, 2012.

33. R. Coello, A. Charlett, J. Wilson, V. Ward, A. Pearson, and P.Borriello, “Adverse impact of surgical site infections in English hospitals,”The Journal of Hospital Infection, vol. 60, no. 2, pp.93–103, 2005.

34. Department of Health, Under the Knife Report, Department of Health, 2011.6 Surgery Research and Practice

35. R. Plowman,N. Graves, M. A. S. Griffin et al., “The rate and cost of hospital-acquired infections occurring in patients admitted to selected specialties of a district general hospital in England and the national burden imposed,” The Journal of Hospital Infection, vol. 47, no. 3, pp. 198–209, 2001.

36. A. Sharma, A.-P.Deeb, J. C. Iannuzzi, A. S. Rickles, J. R. T.Monson, and F. J. Fleming, “Tobacco smoking and postoperative outcomes after colorectal surgery,” Annals of Surgery, vol. 258, no. 2, pp. 296–300, 2013.

PROFORMA

Name	:	I.P. No	:
Age	:	Unit	:
Sex	:	D.O.A	:
Occupation	:	D.O.D	:
Address	:		
Phone No	:	D.O. Surgery	

CHIEF COMPLAINTS

PAST HISTORY:-

1) History of similar complaints
2) Treatment taken
3) History of Drug intake
4) History suggestive of Hypertension / Diabetes / Tuberculosis / heart disease / jaundice / thyroid disorder.

PERSONAL HISTORY:-

Diet : Vegetarian / Mixed

Habits : Smoking / Alcohol / Tobacco

Bowel habits

Bladder

Sleep

FAMILY HISTORY:-

Relevant / Not

MENSTRUAL HISTORY:-

Amenorrhoea / menorrhagia

Regular / Not

Duration

Associated / Not with pain

L.M.P.

GENERAL PHYSICAL EXAMINATION : -

1. General survey
2. Body build and nourishment
3. Appearance
4. Attitude : Restless / Quiet

5. Dehydration : Mild/ Moderate / Severe / Nil
6. Anaemia / Jaundice / Clubbing Cyanosis / Lymphadenopathy / Pedal oedema.
7. Eye signs
8. Skin Changes
9. Pulse
10. Temperature
11. Respiratory rate
12. Blood pressure

SYSTEMIC EXAMINATION

- Cardiovascular system
- Respiratory System
- Central nervous system
- Genito - urinary system
- Abdomen
- Oral cavity

INVESTIGATIONS:-

1. Blood : Hb%
2. TLC
3. DLC

4. BT
5. CT
6. ESR
7. Blood group and rh type.
8. Urine : Albumin / Sugar / Microscopy
9. Blood : sugar / Urea / creatinine
10. ECG
11. USG abdomen and pelvis
12. CECT Abdomen/pelvis
13. HPE
14. HIV
15. HbsAg
16. Others

DIAGNOSIS

MANAGEMENT

SURGICAL

Pre operative instructions

Type of Anaesthesia

Post - operative instructions

Post - operative period/Post - operative complication management

GROUP - A

S.NO	NAME	AGE	SEX	IP. NO	DIAGNOSIS	SURGERY	INCISION TYPE	SUCTION DRAIN	SS1	SUTURE REMOVAL	WOUND DEHISCENCE
1	Jeyanthi	54	F	36482	Incisional hernia	Omentectomy	1	yes	No	POD 11	No
2	Perumal	60	M	32719	duodenal perforation	Omental patch	1	yes	No	POD 12	NO
3	Saleemkhan	52	M	428661	Intestinal obstruction	resection anastomosis	1	yes	No	POD 14	No
4	Karupaiya	65	M	57448	perforation	Omental patch	1	yes	No	POD 11	No
5	VINOTHKUMAR	19	M	39030	spleen injury	spleenectomy	1	yes	No	POD 10	No
6	SARAVANAKUMAR	35	M	31039	perforation transcolon	loop ileostomy	1	yes	No	POD 13	No
7	PASUPATHY	20	M	4700	blunt spleen injury	spleenectomy	1	yes	No	POD 11	No
8	RAGUNATHAN	40	M	5861	perforation transcolon	1 repair	1	yes	yes POD 2	POD 15	No
9	VELMURUGAN	37	M	7544	sigmoid perforation	resection anastomosis	1	yes	yes POD 2	POD 15	yes
10	VISWANATHAN	20	M	7604	jejunal perforation	1 repair	1	yes	No	POD 12	No
11	RAMALAXMI	50	F	79519	spleen injury	spleenectomy	1	yes	No	POD 12	No
12	RAVICHANDRAN	43	M	13135	jejunal perforation	1 repair	1	yes	No	POD 12	No
13	ISSAKIRAJA	22	M	3849	ileal perforation	1 repair	1	yes	No	POD 11	No
14	UMESHSHANKAR	40	M	1449	prepyloric perforation	Omental patch	1	yes	Yes POD 2	POD 15	yes POD 5
15	VINOTHKUMAR	18	M	3903	spleen injury	spleenectomy	1	yes	No	POD 12	No
16	SELVARAJ	60	M	17016	D1 perforation	Omental patch	1	yes	No	POD 11	No
17	MURUGAESAN	63	M	1039	prepyloric perforation	Omental patch	1	yes	No	POD 13	No
18	GOPAL	35	M	15363	sigmoid perforation	loop colostomy	1	yes	No	POD 11	No
19	KRISHNAVENI	65	F	3173	Intestinal obstruction	resection anastomosis	1	yes	No	POD 13	No
20	PARAMASIVAM	57	M	33396	ileal perforation	ileastomy	1	yes	yes POD 3	POD 16	yes POD 5
21	KAVITHA	42	F	47491	Intestinal obstruction	resection anastomosis	1	yes	No	POD 15	No
22	MUTHUKUMAR	40	M	29825	Intestinal obstruction	Omentectomy	1	yes	yes POD 3	POD 16	yes POD 5
23	SETHU	52	M	37905	Intestinal obstruction	resection anastomosis	1	yes	No	POD 15	No
24	NAGARATHINAM	55	F	38005	G.B. perforation	cholecystatomy	2	yes	No	POD 14	No
25	BOOMA	34	F	17066	spleen injury	spleenectomy	1	yes	No	POD 12	No
26	MUTHU	50	M	27034	Intestinal obstruction	Omentectomy	1	yes	No	POD 11	No
27	INDRA	43	F	4610	Intestinal obstruction	Omentectomy	1	yes	No	POD 12	No
28	SHANTHI	54	F	50448	Intestinal obstruction	resection anastomosis	1	yes	No	POD 11	No
29	MERY	63	F	53060	Intestinal obstruction	resection anastomosis	1	yes	No	POD 14	No
30	GOVINDAMMAL	49	F	59244	Intestinal obstruction	resection anastomosis	1	yes	No	POD 12	No

GROUP - B

S.NO	NAME	AGE	SEX	IP. NO	DIAGNOSIS	SURGERY	INCISION	SUCTION DRAIN	SSI	SUTURE REMOVAL	WOUND DEHISCENCE	SECONDAI
1	PITCHAKUTTY	70	M	3179	ileal perforation	1 repair	1	No	yes POD 3	POD 19	No	No
2	JAYA	42	F	48771	Intestinal obstruction	resection anastomosis	1	No	yes POD 4	POD 20	NO	No
3	RAJAPANDI	19	M	50234	Intestinal obstruction	resection anastomosis	1	No	yes POD 4	POD 19	yes POD 7	yes
4	BABAI	22	M	3582	Intestinal obstruction	resection anastomosis	1	No	yes POD 4	POD 18	No	No
5	KATHIREASAN	50	M	60378	prepyloric perforation	Omental patch	1	No	yes POD 3	POD 19	yes POD 6	yes
6	MUTHUPANDI	38	M	3105	perforation transcolon	resection anastomosis	1	No	yes POD 4	POD 19	yes POD 7	yes
7	CHINNAKARUPU	43	M	6179	jejunal perforation	1 repair	1	No	yes POD 3	POD 17	No	No
8	RAMU	56	M	10653	ileal perforation	1 repair	1	No	No	POD 14	No	No
9	RAMESH	33	M	12813	jejunal perforation	1 repair	1	No	No	POD 13	No	No
10	PANJAVARNAM	56	M	120177	Intestinal obstruction	resection anastomosis	1	No	Yes POD 3	POD 19	yes POD 6	yes
11	MUTHUVELPANDI	35	M	34466	D1 perforation	Omental patch	1	No	Yes POD 4	POD 16	No	No
12	MUNIYASAMY	42	M	39430	G.B. perforation	cholecystatomy	2	No	Yes POD 5	POD 19	yes POD 8	yes
13	INDRA	48	F	4651	Intestinal obstruction	resection anastomosis	1	No	Yes POD 4	POD 19	yes POD 6	yes
14	JOHN BRITTO	48	M	50446	Intestinal obstruction	Omentectomy	1	No	No	POD 14	No	No
15	RAKKU	50	F	33400	Transverse colon perfora	resection anastomosis	1	No	Yes POD 4	POD 18	yes POD 7	yes
16	GOPI	28	M	34760	Intestinal obstruction	resection anastomosis	1	No	No	POD 14	No	No
17	ISSAKI	54	M	31638	ileal perforation	1 repair	1	No	Yes POD 3	POD 16	yes POD 7	yes
18	PUSPARAJ	56	M	36395	ileal perforation	1 repair	1	No	Yes POD 4	POD 16	yes POD 5	yes
19	ADILAKSHMI	60	F	34885	ileal perforation	1 repair	1	No	No	POD 14	No	No
20	SEKAR	42	M	37753	Intestinal obstruction	resection anastomosis	1	No	No	POD 13	No	No
21	PONNAMAL	55	F	21958	Intestinal obstruction	Omentectomy	1	No	Yes POD 3	POD 14	yes POD 5	yes
22	MOHAMMED	50	M	2286	Intestinal obstruction	resection anastomosis	1	No	yes POD 4	POD 17	yes POD 7	yes
23	PANJAVARANAM	48	F	21943	Intestinal obstruction	resection anastomosis	1	No	Yes POD 4	POD 17	yes POD 6	yes
24	BALASUBRAMANI	35	M	25151	ileal perforation	1 repair	2	No	Yes POD 3	POD 16	yes POD 5	yes
25	GOPI	48	M	34760	spleen injury	spleenectomy	1	No	Yes POD 3	POD 17	yes POD 6	yes
26	MADURAIVEERAN	62	M	45362	Transverse colon perfora	1 repair	1	No	Yes POD 4	POD 16	yes POD 7	yes
27	JAYAPRIYA	46	F	48881	Intestinal obstruction	resection anastomosis	1	No	Yes POD 5	POD 16	yes POD 6	yes
28	PANDIAN	60	M	68179	prepyloric perforation	Omental patch	1	No	Yes POD 3	POD 16	yes POD 5	yes
29	THIRUNAVUKARASU	51	M	51676	Intestinal obstruction	resection anastomosis	1	No	Yes POD 3	POD 16	yes POD 5	yes
30	MUNIYANDI	47	M	56921	prepyloric perforation	Omental patch	1	No	Yes POD 4	POD 18	yes POD 6	yes

MADURAI MEDICAL COLLEGE
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ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.R.Giridharan
Course	:	PG in MS., General Surgery
Course of Study	:	2017-2020
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Comparison of use of subcutaneous drains to prevent wound complications in emergency Laprotomy procedures
Ethical Committee as on	:	11.02.2019

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.,(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
Madurai

DEAN
MADURAI MEDICAL COLLEGE,
MADURAI - 625 020

19 FEB 2019

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Urkund Analysis Result

Analysed Document:	giridharan.thesis.docx (D57236072)
Submitted:	18/10/2019 13:18:00
Submitted By:	ragiridharan@gmail.com
Significance:	7 %

Sources included in the report:

https://ijsurgery.com/index.php/isj/article/download/2207/1752
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4486392/
https://www-d0.fnal.gov/trigger/stt/commiss/agilent/16700-97012.pdf
https://www.flashcardmachine.com/abdomen.html

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15