“A COMPARATIVE STUDY BETWEEN POVIDONE IODINE IN NORMAL SALINE AND NORMAL SALINE FOR PERTONEAL LAVAGE IN PERITONITIS”

BY

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IN

GENERAL SURGERY

Under the guidance of

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LIST OF ABBREVIATIONS

S.No.	*Abbreviation*	*Expansion*
1.	*GIT*	*Gastrointestinal tract*
2.	*e.g*	*Example*
3.	*BCC*	*Bacterial cell count*
4.	*HCL*	*Hydrochloric Acid*
5.	*E.coli*	*Escherichia coli*
6.	*No*	*Number*
7.	*Sl. No.*	*Serial Number*
8.	*CNS*	*Central Nervous System*
9.	*IOPL*	*Intraoperative peritoneal lavage*
10.	*MODS*	*Multi-Organ Dysfunction Syndrome*
11.	*AIDS*	*Acquired Immunodeficiency Syndrome*
12.	*Mm*	*milli meter*
13.	*SMA*	*Superior mesenteric artery*
14.	*ECF*	*Extracellular fluid*
15.	*PMN*	*Polymorph nuclear Leukocytes*
16.	*NSAIDS*	*Non Steroidal Anti Inflammatory Drugs*
17.	*HSV*	*Highly Selective Vagotomy*
18.	*TV+D*	*Truncal Vagotomy + Drainage*
19.	*ERCP*	*Endoscopic Retrograde Cholangio-Pancreaticograph*
20.	*CT*	*Computed Tomography*
21.	*ARDS*	*Acute Respiratory Distress Syndrome*

22.	*PE*	*Pulmonary Embolism*
23	*DVT*	*Deep Vein Thrombosis*
24	*Hb*	*Haemoglobin*
25	*WBC*	*White Blood Cell*
26	*ABG*	*Arterial Blood Gas*
27	*CRP*	*C-Reactive Protein*
28	*US*	*Ultrasound*
29	*PPFA*	*Peri-Portal Free Air*
30	*ADH*	*Antidiuretic hormone*
31	*OPA*	*Open abdomen / Laparostomy*
32	*COLA*	*Covered Laparostomy*
33	*PR*	*Planned re-laparotomy*
34	*STAR*	*Staged Abdominal repair*
35	*SSI*	*Surgical Site Infection*
36	*i.e*	*That is*
37	*NG*	*No Growth*
38	*mm Hg*	*Millimetre of mercury*
39	*dl*	*Decilitre*
40	*min*	*Minute*

BACKGROUND

Peritonitis is defined as the inflammation of the peritoneum that lines the inner wall of the abdomen and abdominal organs. Peritonitis usually occurs secondary to contamination of peritoneal cavity by the gastrointestinal contents, either due hollow viscous perforation or due to bacteria translocation through the wall of ischemic gut. Perforation of any part along the gastrointestinal tract is a life threatening emergency and is associated with high morbidity and mortality. Most common perforations are gastroduodenal perforation followed by small intestinal and appendicular perforations. Colonic perforations are uncommon. In developing countries like India, the mortality is still high due to delay in presentation along with the socio-economic reasons.

Acute generalized peritonitis is considered as a surgical emergency, which is very challenging to manage. Early diagnosis, control of sepsis and management of primary cause is very important. Perforation closure with peritoneal lavage has been the critical step in managing peritonitis, the practise continues even today. In cases of small intestinal perforation, resection and anastomosis can be performed. Peritoneal lavage ensures adequate control of infection and minimizes the risk of post-operative infection, thereby preventing prolonged hospital stay. Most commonly used fluids in peritoneal lavage are warm saline, sterile water, aqueous povidone iodine and saline with antibiotics. Peritoneal lavage reduces the bacterial load, thereby reducing the incidence of wound site infection and sepsis.

OBJECTIVES OF THE STUDY:

1. To study the clinical outcome of patients diagnosed with peritonitis who have received peritoneal lavage with povidone iodine in normal saline.
2. To study the clinical outcome of patients diagnosed with peritonitis who have received peritoneal lavage with normal saline.
3. To compare the clinical outcome between two sets of patients.

MATERIALS AND METHODS

SOURCE OF DATA:

This is a prospective clinical study conducted on 172 consecutive patients who presented to surgical department, R.L. JALAPPA HOSPITAL, TAMAKA, KOLAR with peritonitis secondary to hollow viscus perforation. The study period was from December 2016 to June 2018. Number to be studied: 172-divided as 86 in each group comprising of odd and even serial numbers.

GROUP A: Patients with all odd serial numbers were included in this group and peritoneal lavage with povidone iodine in normal saline was used

GROUP B: Patients with all even serial numbers were included in this group peritoneal lavage with normal saline was used.

INCLUSION CRITERIA

All patients with peritonitis of age >20yrs and <75years

EXCLUSION CRITERIA:

1. Peritonitis secondary to trauma to abdomen.
2. Peritonitis secondary to gynaecological interventions.

3. Peritonitis secondary to malignancy and immuno-compromised state
4. Patients with thyroid disorders

RESULTS:

The clinical outcome in the form reduction in postoperative complications and hospital stay were assessed in 174 patients, in Group A (Peritoneal lavage with povidone iodine in normal saline) and Group B (with peritoneal lavage of normal saline). In Povidone Iodine with Normal Saline group, mean cell count at Pre BCC was 1748.28 ± 124.27 and at Post BCC was 1462.07 ± 169.90. There was significant decrease in Cell count at Post BCC compared to Pre BCC in Povidone Iodine with Normal Saline group. In Normal Saline group, mean cell count at Pre BCC was 1700.00 ± 131.88 and at Post BCC was 1554.17 ± 147.38. There was significant decrease in Cell count at Post BCC compared to Pre BCC in Normal Saline group. In both the groups there was a significant decrease in cell count post BCC .However cell count was significantly lower in Povidone Iodine with Normal Saline group than in Normal saline group.

There was 1 death, where the patient had severe form of peritonitis with massive contamination and delayed presentation to the hospital. This study also revealed that men are commonly affected and pre-pyloric perforation is the commonest site of perforation. Escherichia coli is the most common organism isolated.

CONCLUSION:

In our study peritonitis is more common in men compared to women. The most common age group is in between 51 – 60 years in cases of peritonitis with the mean age of 54 years. Pre-pyloric perforation is the commonest site of perforation.

Escherichia coli is the most common organism isolated in the peritoneal fluid. Povidone iodine in normal saline lavage significantly decreases the bacterial load when compared to normal saline lavage

KEYWORDS: Perforative peritonitis, Povidone Iodine, Normal saline, Postoperative complications and Hospital stay.

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INTRODUCTION

INTRODUCTION

Peritonitis is defined as the inflammation of the peritoneum that lines the inner wall of the abdomen and abdominal organs. Peritonitis usually occurs secondary to contamination of peritoneal cavity by the gastrointestinal contents, either due hollow viscous perforation or due to bacteria translocation through the wall of ischemic gut[1]. Perforation of any part along the gastrointestinal tract is a life threatening emergency and is associated with high morbidity and mortality. Most common perforations are gastroduodenal perforation followed by small intestinal and appendicular perforations. Colonic perforations are uncommon[2]. In developing countries like India, the mortality is still high due to delay in presentation along with the socio-economic reasons.

Acute generalized peritonitis is considered as a surgical emergency, which is very challenging to manage. Early diagnosis, control of sepsis and management of primary cause is very important [3]. In early mediaeval times when a person presented with sudden, severe pain, and frequent vomiting, hardness of the belly, fatal illness as is seen in peritonitis was treated with spoonful of lemon juice morning and night. The early treatment of peritonitis had to be medical since surgery had not progressed to the stage where the abdomen was entered intentionally. The generally accepted treatment for peritonitis was absolute rest, purgatives-especially magnesium sulphate, abstention of food, cold applied to the abdomen and opium very sparingly[4]. Later Mikulicz advocated opening the abdomen at the time of presentation. He also brought out the so-called toilette of the peritoneum using a 2% thymol solution in sponging the soiled intestines and the use of drainage tubes. Tait advocated filling the abdomen with blood warm water and washing all organs repeatedly until the water came off clear. In

older days fluids used in peritoneal lavage were ether, amniotic fluid, 25% glucose ,water ,saline and antibiotics lavage and aspiration. Most commonly used fluids in peritoneal lavage are warm saline, sterile water, aqueous povidone iodine and saline with antibiotics[5]. Peritoneal lavage reduces the bacterial load, thereby reducing the incidence of wound site infection and sepsis. Despite recent advances in surgical treatment, antimicrobial agents and intensive medical care, the mortality rates around 15%-30% remains high.

Saline lavage reduces significantly counts in peritoneal fluid of aerobic and anaerobic bacteria in peritoneal fluid and gives us the idea of amount of debris present in the peritoneal fluid. Povidone iodine is a stable chemical complex of poly vinyl pyrolidone and elemental iodine. It contains 9% to 12% available iodine. It is an effective bactericide and is safe when used as peritoneal lavage solution.

The objective of the present study is to compare the efficacy of povidone iodine in normal saline and normal saline in peritoneal lavage .Peritoneal lavage ensures adequate control of infection and minimizes the risk of post-operative infection, thereby preventing prolonged hospital stay.

Perforation closure with peritoneal lavage has been the critical step in managing peritonitis, the practise continues even today. In cases of small intestinal perforation, resection and anastomosis can be performed[6].

OBJECTIVES

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1. To study the clinical outcome of patients diagnosed with peritonitis who have received peritoneal lavage with povidone iodine in normal saline.

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

The first scientific theory of peritonitis was developed by Hippocrates and the Koic School of Medicine and dates back to 5th century. The clinical description of this disease is depicted as "*The patient looks ill and wasted with nose pointing, sunken temples, deep lay eyes, rimmed and dull. The face expresses fear, tongue furred and the skin shiny. The patient breathes shallow and avoids all movements. The abdominal wall is rigid with guarding of muscles and bowel sounds are absent. The pulse is quick and small. The presence of hard, tender mass in the hypochondrium is a poor prognostic sign, if involving the whole area. The death is imminent if there is presence of mass at the beginning of fever",* reads from the old excerpt, written by Hippocrates, the father of medicine[7].

Earlier peritonitis was termed as idiopathic peritonitis but this term is obsolete now, as it was noted that there was always an underlying cause for the condition. In early medieval times when a patient presented with sudden, severe, painful and deadly illness; conditions like peritonitis, obstruction, appendicitis or perforation , was called as iliac passion which is synonymous with our modern term of acute abdomen[8]. Later peritonitis was given the term iliac passion whereas intestinal obstruction was termed as colonic passion. Before the advent of surgery peritonitis was treated with salts of wormwood with a spoonful of lemon juice given orally during morning and night, absolute rest, purgatives-especially magnesium sulphate, abstention of food, cold objects applied to the abdomen in acute cases and opium was very sparingly used. Despite these multiple modalities of treatment, the prognosis was poor.

Later venesection associated with the use of leeches applied to the abdomen and opium was used in larger doses[4]. Using opium caused ileus, which was one of the

cause of death in peritonitis. After the development of principles of antisepsis by Lister[9] and introduction of anaesthesia by Horace Wells and Thomas G Morton using ether, surgeons were better equipped to enter the abdomen safely, in an effort to identify the source of infection and to eradicate it. Following this, bowel surgeries were done using heated lances and cauterising knife. This caused tissues and blood to heat rapidly to extreme temperatures which lead to coagulation of the tissues. Along with the above methods, mandibles of insects such as the black ants were used to clamp and hold the edges of bowel together. Intestinal repairs like end-to-end anastomosis was done using tracheas of animals. In cases of distal ileal perforation resection and anastomosis was done but it carried high morbidity and mortality due to wound infection, faecal fistula formation, septicaemia and respiratory infection. Hence it was proposed that ileo-transverse anastomosis with closure of distal stump showed a better outcome[10]. Omental patch repairs were used more commonly for perforated gastric ulcers[11]. Subsequently peritoneal lavage was done initially using 2%thymol and later by ether, 5% mercurochrome, 25% of glucose, amniotic fluid, warm saline, bicarbonate soda and peroxide.

But peritoneal lavage was not widely accepted in the past, because of the belief that antiseptic agents used in peritoneal lavage would injure the mesothelial cells. This was contradicted by Mickuliz by using peritoneal lavage with encouraging results[12]. Dehydration was one of the cause of death in peritonitis and hence IV fluids administration along with peritoneal lavage was considered lifesaving.

With progression of time and better understanding of the principles of asepsis, anatomy and physiology of peritoneum, peritoneal lavage had an additional benefit in reducing the infection rate in comparison to the patients who had not undergone peritoneal lavage. Peritoneal lavage along with systemic antibiotics was highly effective in bringing down

the morbidity and mortality. Martin Kirschner[13] summarised that any patient with acute diffuse peritonitis should be operated immediately using a midline laparotomy under anaesthesia unless they are contraindicated. The objective of the surgery was to eliminate the source of the infection with effective peritoneal lavage. Subsequently peritoneal lavage became increasingly used to treat peritonitis. Peritoneal lavage was done using 2%thymol, ether, 5%mercurochrome, 25% of glucose, amniotic fluid, warm saline, bicarbonate soda, peroxide, povidone iodine and antibiotics.

ANATOMY OF PERITONEAL CAVITY

At the end of the third week, the intra embryonic mesoderm starts differentiating into paraxial mesoderm, intermediate mesoderm and lateral plate mesoderm that is involved in forming the body cavity. Clefts appear in the lateral plate mesoderm that ultimately coalesces to split the solid layer into:

(a) The parietal (somatic) layer adjacent to the surface ectoderm in contiguity with the extra embryonic parietal mesoderm layer over the amnion.

(b) The visceral (splanchnic) layer adjacent to endoderm forming the gut tube in contiguity with the visceral layer of extra embryonic mesoderm covering the yolk sac.

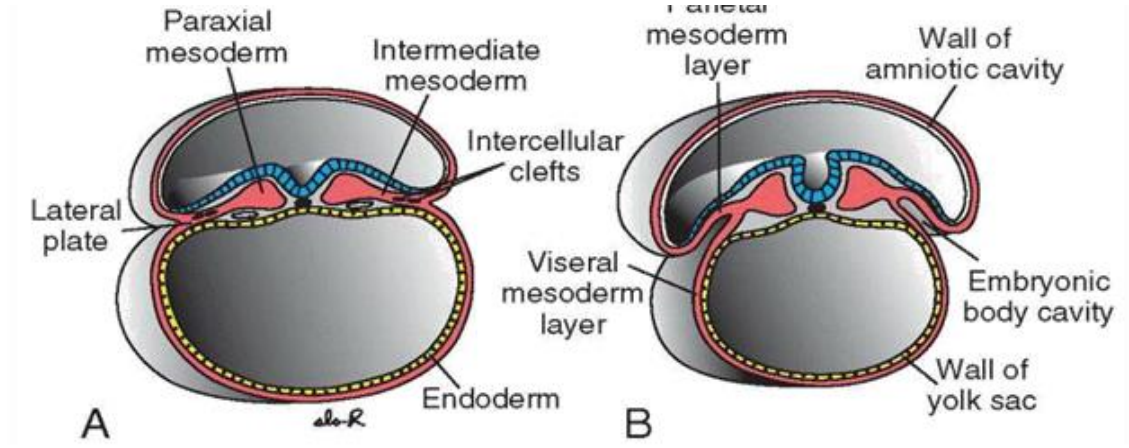
Embryo at 19 days: Intercellular clefts are visible in the lateral plate mesoderm.

Embryology of Peritoneal Cavity:

Embryo at 20 days: The lateral plate is further split into somatic mesoderm and visceral mesoderm layers that line the intraembryonic cavity. Tissue bordering the intraembryonic cavity differentiates into serous membranes.

The primitive body comprises of the space between the two layers of lateral plate mesoderm. Parietal layer cells of lateral plate mesoderm lining the intra embryonic cavity become mesothelial and eventually develop into the parietal layer of the serous membranes lining the outside of the peritoneal, pleural and pericardial cavities. In the same way, lateral plate mesoderm visceral cells form the visceral layer of the serous membranes covering the abdominal organs, lungs and heart[14]

Fig 1:Embryology of peritoneum. A. Embryo transverse section -19 days approximately B. Section through an embryo- 20 days approximately[14].



Formation of the Peritoneal Ligaments and Mesenteries

Peritoneal ligaments are developed from the ventral mesentery and dorsal mesentery. The ventral mesentery in turn is developed from the mesoderm of septum transversum (derivatives of the cervical somites which migrate downward). The ventral mesentery then forms the falciform ligament, the lesser omentum, the coronary ligament and the triangular ligament of the liver.

The dorsal mesentery on the other hand is developed by the fusion of splanchnopleuric mesoderm present on both sides of the embryo. The dorsal mesentery extends from the posterior abdominal wall up to the posterior border of the abdominal parts of the gut. The dorsal mesentery gives rise to the gastro-phrenic ligament, gastro-splenic omentum, spleno-renal ligament, greater omentum, and the mesenteries of the small and large intestines[15].

Formation of Lesser and Greater Peritoneal Sacs

The extensive growth of the right lobe of the liver forces the ventral mesentery

to be pulled to the right and causes rotation of the stomach and duodenum. By this means, the upper right part of the peritoneal cavity becomes incorporated into the lesser sac. The right free border of the ventral mesentery becomes the right border of lesser omentum and the anterior boundary of the entrance into the lesser sac.

The remaining part of the peritoneal cavity, excluded from the lesser sac is called the greater sac and the two sacs are in communication through the epiploic foramen[15].

Formation of the Greater Omentum

The spleen develops from the upper part of the dorsal mesentery and the greater omentum is formed due to the extensively rapid growth of the dorsal mesentery caudal to the spleen. To begin with, the greater omentum extends from the greater curvature of the stomach to posterior abdominal wall above the transverse mesocolon. With continued growth, it reaches inferiorly as an apron like double layer of peritoneum anterior to the transverse colon.

Later, the posterior layer of the omentum fuses with the transverse mesocolon; as a result, the greater omentum attaches to the anterior aspect of the transverse colon. As development proceeds, the omentum becomes laden with fat. The inferior recess of the lesser sac extends inferiorly between the anterior and the posterior layers of the fold of the greater omentum[15].

INTRODUCTION

"I felt as if I had been peering through a newly revealed window, opening upon the once impenetrable gloom enveloping man's earliest endeavors to understand the world he lived in. It was as if I had watched a hand slowly raising the curtain that covered this window, and then suddenly the hand had refused to lift, the curtain further".

- (Description about Peritoneum in Edwin Smith Papyrus Translation by Breasted)

The peritoneum is a large serous membrane lining the abdominal cavity. It comprises an outer layer of fibrous tissue giving strength to the membrane and an inner layer of mesothelial cells which secrete a serous fluid acting as a lubricant to the inner surface thereby allowing free movements of the abdominal viscera without any friction. The peritoneal membrane is invaginated by a few structures dividing it into:

1. An outer layer, the parietal peritoneum.
2. An inner layer, the visceral peritoneum.
3. The folds of peritoneum by which the viscera are suspended and
4. The peritoneal cavity proper.

The parietal portion is richly innervated and when inflamed produces severe pain accurately localized to the affected area whereas the visceral peritoneum is poorly localised as it is not as richly innervated.[16]

PERITONEAL CAVITY

It is a potential space enclosed with in the peritoneum. The free surfaces are lubricated by a thin film of serous fluid secreted by the mesothelial cells. Physiologically there is no space in between the parietal and visceral peritoneum. The peritoneal cavity is broadly divided into two parts. The main greater part is known as greater sac whereas the smaller part which is present posterior to the liver and stomach, is known as lesser sac. The two sacs communicate with each other through epiploic foramen (foramen of winslow). The pockets of peritoneal cavity are enclosed by small folds of peritoneum and peritoneal pockets known as peritoneal fossae (recess), most of which get obliterated. If these persist, they can lead to internal hernia or strangulation.[17,18]

SURGICAL ANATOMY

It is prudent to identify the appropriate surgical anatomy of the abdomen, which is of importance in determining possible sources and routes of spread of infection. The peritoneal cavity is bounded superiorly by the under surface of the diaphragm and inferiorly the floor of the pelvis. In males, the peritoneal cavity is a closed space. In females, the free ends of the fallopian tubes pierce through it. Anteriorly the peritoneal cavity is bounded by the posterior surface of the anterior abdominal musculature. Posteriorly, the peritoneal lining lies just superficial to the retroperitoneal structures which include the aorta, venacava, ureters and kidneys. Collectively, the anterior and posterior peritoneal layers are known as the parietal peritoneum. The visceral peritoneum on the other hand, represents the lining of mesothelial cells which is present on the surface of abdominal viscera. The peritoneum covering the intestine is the serosa of the bowel.[16,18]

The peritoneal cavity is divided into an upper abdominal and lower pelvic portions. The abdominal part is subdivided into upper and lower compartments by the transverse colon. The supracolic space lies between the diaphragm and transverse colon. It comprises the stomach, liver, gall bladder, spleen and first part of the duodenum. The space is further classified into sub- hepatic and sub-phrenic spaces. The infracolic space is divided into right and left infra colic spaces by the mesentery of small intestine. To the right of mesentery of small bowel and transverse colon lies the right infra colic space. It is divided into external and internal paracolic gutters by the ascending colon. To the left of mesentery of small bowel lies the left infra colic space. The descending colon divides this space into external and internal paracolic gutters. The pelvic cavity is sub-divided by the pelvic mesocolon into secondary pouches[16,18]

Intra peritoneal spaces

Four intraperitoneal exist

1. Left anterior intra peritoneal space

2.Left posterior intra peritoneal space

3. Right anterior intra peritoneal space

4. Right posterior intra peritoneal space

1.LEFT ANTERIOR INTRA PERITONEAL SPACE

It is bounded by the diaphragm superiorly and the left triangular ligament and left lobe of liver posteriorly. The lesser omentum, stomach and falciform ligament lie to the right and spleen, gastro-splenic ligament and left dome of diaphragm lie to the left.[16]

2.LEFT POSTERIOR INTRA PERITONEAL SPACE (lesser sac)

Anterior wall is formed by the stomach, lesser omentum along with anterior layers of greater omentum and caudate lobe of liver. Posterior wall is formed by the structures forming the stomach bed (left crus and dome of diaphragm, left suprarenal gland, the left kidney, splenic artery, the pancreas, transverse meso-colon, splenic flexure of the colon) and posterior layers of greater omentum. Lesser sac communicates over the right side with the greater sac through the epiploic foramen and left side is bounded by gastro-phrenic and gastro-splenic and lienorenal ligament.[16]

3. **RIGHT ANTERIOR INTRA PERITONEAL SPACE**

It lies between the right lobe of the liver and the diaphragm. It is bounded posteriorly by anterior layer of coronary ligament and right triangular ligament and to the left by the falciform ligament.[16]

4.**RIGHT POSTERIOR INTRA PERITONEAL SPACE (Rutherford Morison's kidney pouch)**

It lies transversely beneath the right lobe of the liver.

Boundaries:

a. Anteriorly: Inferior surface of the right lobe of the liver and gall bladder.

b. Posteriorly: Supra renal gland, upper pole of right kidney, second part of duodenum, hepatic flexure of the colon, transverse colon, part of the head of the pancreas.

c. Superiorly: Inferior layer of the coronary ligament.

d. Inferiorly: Opens into the general peritoneal cavity. It is the spacious of the four

and the most common site of the subphrenic abscess. Intraperitoneal spaces, showing the circulation of fluid and potential areas for abscess formation Sagittal view of peritoneal attachments[16]

Extra peritoneal spaces

There are three extra peritoneal spaces.

1. Midline extraperitoneal space

2. Right and left extra peritoneal spaces

1. **MIDLINE EXTRAPERITONEAL SPACE** (bare area of liver) This lies between superior and inferior layer of the coronary ligament where liver is in direct contact with the diaphragm. This is the site where amoebic liver abscess is the most prevalent.[17]

2. **RIGHT AND LEFT EXTRA PERITONEAL SPACES** This lies around the left and right supra renal gland and the upper poles of the kidneys. These are the sites for perinephric abscess.

The presence of the transverse mesocolon renders the peritoneal cavity into an upper space and a lower space. The greater omentum which extends from the lower border of the stomach and the transverse mesocolon acts as a curtain which covers the lower peritoneal cavity. Between the rectum and bladder in males is a pouch of peritoneal cavity that extends below the level of the seminal vesicles. In females, the fallopian tubes and the uterus project into the pelvic recess. The Pouch of Douglas which lies just above the posterior fornix of vagina is present between the rectum posteriorly and the body of the uterus anteriorly. The spaces lateral to the rectum and the bladder are known as pararectal and paravesical fossae respectively. The pelvic recess is in continuity with both the left and right paracolic gutters.

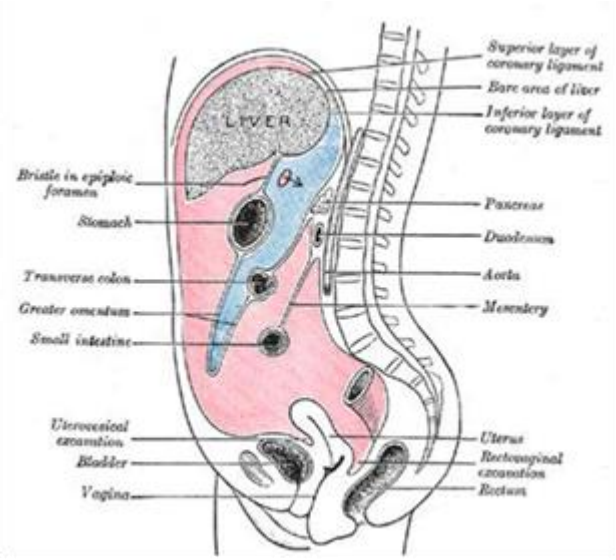
The phrenicocolic ligament which extends from the under surface of the diaphragm to the large intestine partially fills the space between the left perihepatic space and the left paracolic gutter. On the other side, the right paracolic gutter is continues with the right subhepatic space and the right subphrenic space. Morison's pouch is a posterior extension of the right subhepatic sac. It lies above the root of the transverse mesocolon. The falciform ligaments divide the right perihepatic space, into right sub phrenic and right sub hepatic spaces. This probably prevents the flow of pus to the opposite side. Only 5 to 15% of sub phrenic abscess are bilateral. The left sub hepatic space is divided by the lesser omentum into an anterior space and the posterior lesser sac. The lesser sac is the largest recess of the peritoneal cavity. It communicates with the main peritoneal space through the foramen of Winslow. It is surrounded posteriorly by the pancreas and kidneys, anteriorly by the stomach and laterally by the liver and spleen.[18]

The right paracolic gutter serves as the main communication between the superior and inferior peritoneal cavities. Accordingly, any fluid from the right upper peritoneal space tend to gravitate towards the Morison's pouch, then into the right subphrenic space, and finally into the pelvic recess through the right paracolic gutter. Left upper peritoneal space fluid flow is mainly into the left subphrenic space. Flow of fluid inferiorly into the left paracolic gutter is limited by the phrenicocolic ligament. Fluid when introduced into the inferior part of the peritoneal cavity tends to gravitate first in the pelvic recess. Then, it ascends into the right subhepatic space (Morrison's pouch mainly) through the right paracolic gutter and then into the right subphrenic space[21].

On the left side, the phrenicocolic ligament limits the ascent of fluid from the

pelvic space through the left paracolic gutter. Ascent of any fluid from the pelvic cavity into the subphrenic space is due to the hydrostatic pressure difference in the upper and lower parts of the peritoneal cavity secondary to diaphragmatic motion.

Fig 2:SAGITTAL VIEW OF PERITONEAL ATTACHMENTS[19]



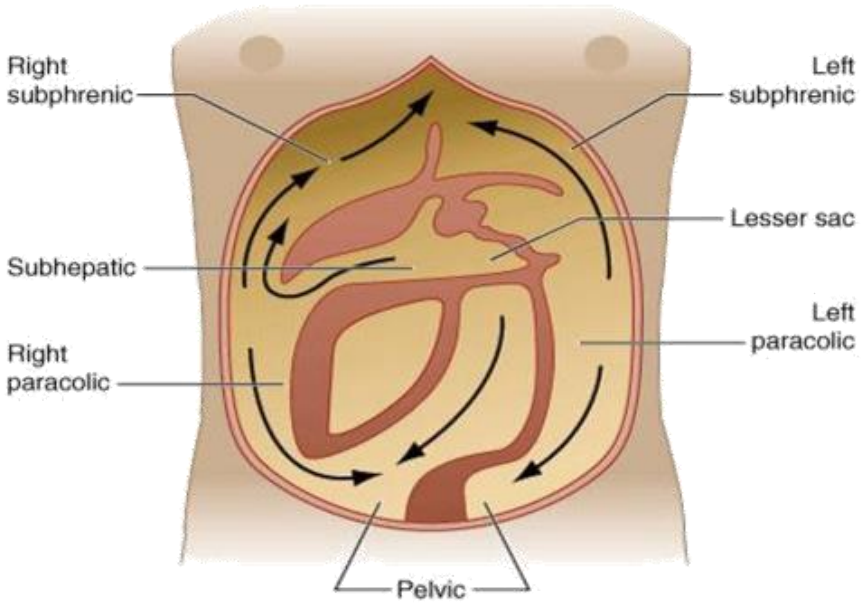


Fig 3:Intraperitoneal spaces, showing the circulation of fluid and potential areas for abscess formation[20]

INNERVATION

Visceral peritoneum is poorly innervated and nerves arise from the visceral structures beneath the lining. Its irritation produces a poorly localized pain. Pain is frequently localized to the dermatomal innervation of the visceral organ[22]. The parietal peritoneum nerve innervation, the somatic afferents, arises from branches of cutaneous nerves in the anterior abdominal wall. The parietal peritoneum produces pain sensation, when stimulated by stretch, light touch and cutting. Pain can be precisely localized. The afferent fibers accompanying the vascular supply furnish sensory innervation to intestines with their associated visceral peritoneum[23]. As a result, irritation in the foregut in turn stimulates the celiac axis afferents, producing pain in the epigastrium. Stimulus in the midgut eg. caecum or appendix results in the activation of afferent nerves which course along the superior mesenteric artery causing pain over the periumbilical region. Hindgut disease stimulates inferior mesenteric artery afferent fibers, causing suprapubic pain.[24]

Pain sensation from the abdomen radiates through autonomic as well as spinal nerves. The phrenic nerve along with the afferent fibres of C3, C4 and C5 dermatomes accompany the phrenic arteries which innervate the under surface of diaphragmatic muscles and the peritoneum. The T5-11 spinal nerves and the phrenic nerve innervate parietal peritoneum. Parietal peritoneal surfaces sharply localize painful stimuli to the site of the stimulus because of spinal nerves innervation. Pain is not well localized with visceral peritoneum. When visceral inflammation irritates the parietal peritoneal surface, localization of pain occurs. The "peritoneal signs" seen in acute abdomen originate in this fashion[23].

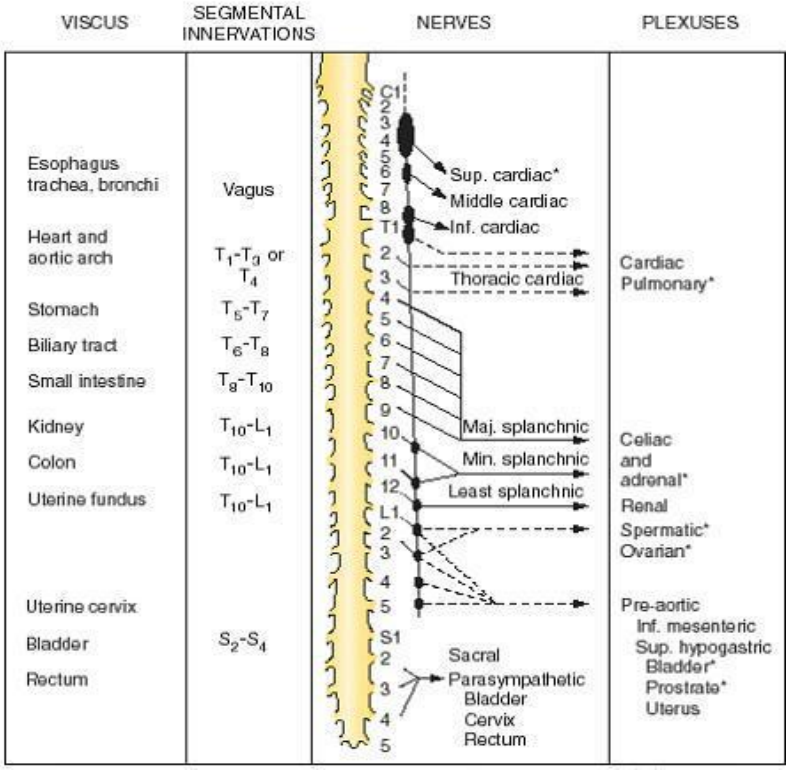
Sudden sharp, well-localized pain is mediated by the peripheral nerves

whereas poorly localized pain of gradual onset, prolonged and dull in nature is characteristic of sensory afferents supplying the intraabdominal cavity. The gastrointestinal tract, pain is not transmitted from vagus nerve. The location of pain is more important leading us to a correct diagnosis because the pain has three basic forms like visceral, somatic and referred pain. Visceral pain is characteristically dull and poorly localized, commonly in the epigastric, periumbilical or suprapubic regions, and usually it is not well-lateralized. It may be associated with sweating, nausea or even restlessness. On the other hand, parietal and somatic pains are known to be more intense and localized.

Table 1 :Dermatome Origin of the Innervation of Intra-abdominal Structures[23]

Organ / Structure	Dermatome Innervation
Esophagus	Oesophageal plexus
Stomach	T5-12
Small Intestine	T8-10
Colon	T10-L2
Liver	T6-8
Gallbladder	T6-8
Uterus	T10-L1
Kidney	T10-L1
Bladder	S2-4
Diaphragm	C4-8

Fig 4 :Visceral sensory innervation.[23]



STOMACH

The stomach (venticulous or preferably gaster)[25] is the most distensible and widest part of the alimentary canal. It is situated between the oesophagus and the small intestine. It is a muscular bag with a mean capacity varying from about 1 ounce at birth, increasing to 1 L at puberty and about 1.5 to 2 L in adults. It occupies the epigastric, umbilical and left hypogastric regions. From surgical point of view, the stomach is composed of two gastric units. One is the proximal gastric unit consisting of the proximal stomach, distal oesophagus and oesophageal hiatus of diaphragm and other, the distal gastric unit comprising the antrum, pylorus and first part of duodenum.[26]

The stomach has 2 openings – **the cardiac orifice** (opening from the oesophagus into the stomach) and the **pyloric orifice** (opening into the duodenum)

It has two curvatures – the lesser and the greater curvature and two surfaces - **Antero-superior surface** and **Postero-inferior surface**.

- **Blood Supply**:

1. Left gastric artery (from the coeliac artery)
2. The right gastric and right gastro-epiploic artery (from the common hepatic artery).
3. The left gastro-epiploic and short gastric (from the splenic) artery.[26]

Venous drainage:

Gastric vein drains into the submucosal veins. Larger veins accompany main arteries to drain into the splenic and superior mesenteric veins.

Lymphatic drainage:

Stomach is drained by four groups

1 .Left gastric arterial nodal group- draining into the lesser curvature of the stomach

2. Short gastric and left gastroepiploic groups- drains left side of greater curvature of stomach

3. Right gastroepipolic groups- drains the right side of greater curvature of stomach.

4. Pyloric groups- drains the pyloric part of stomach.

- **Nerve supply**

Sympathetic supply: is mainly from the coeliac plexus and few rami from the hepatic and left phrenic nerves. The sympathetic nerves are from T_5 to t_{10} segments of spinal cord. The sympathetic nerve supply is vasomotor to the gastric blood vessels and provides the main pathway for gastric pain fibres.

Parasympathetic supply: is from the vagi. It has both secretory and motor effects.

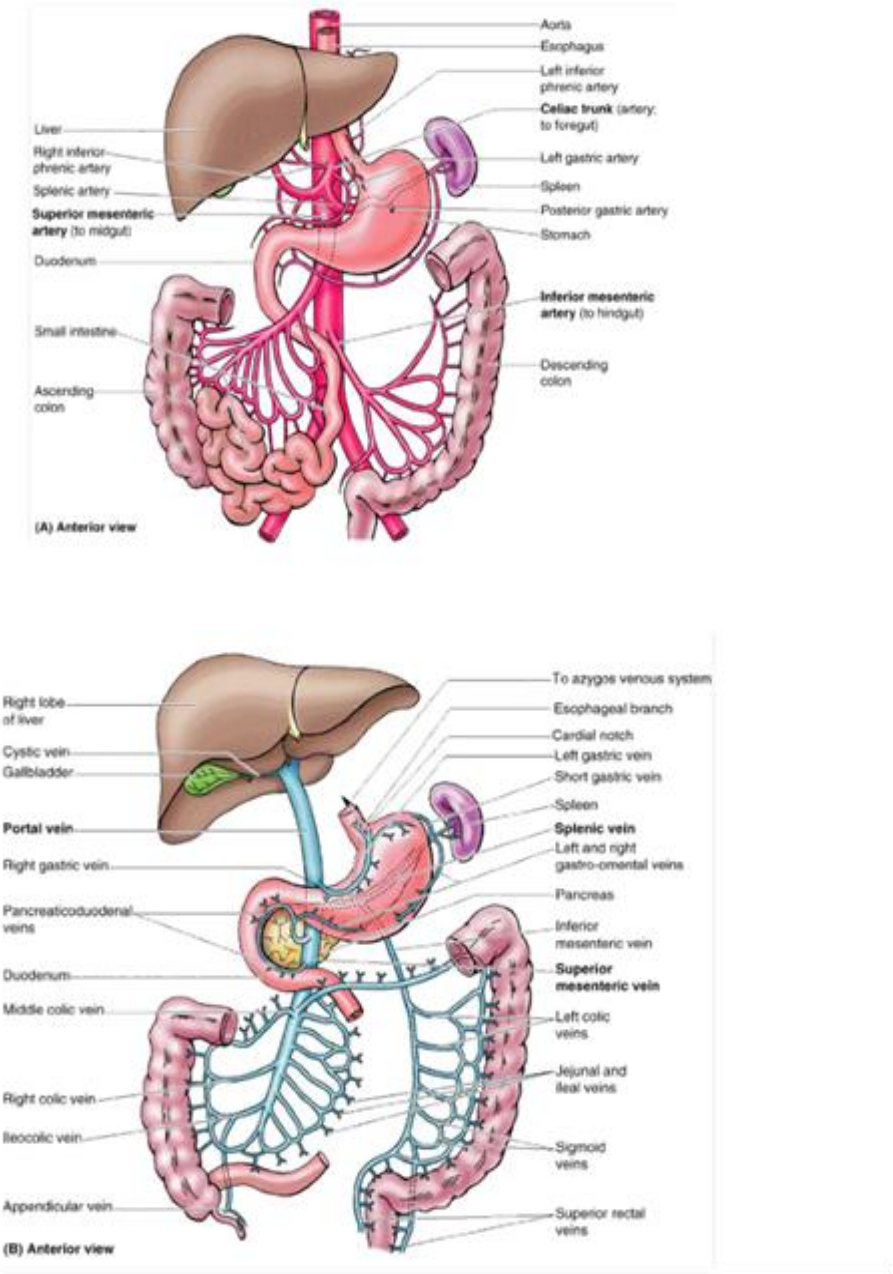


Fig 5 Arterial supply and venous drainage[27]

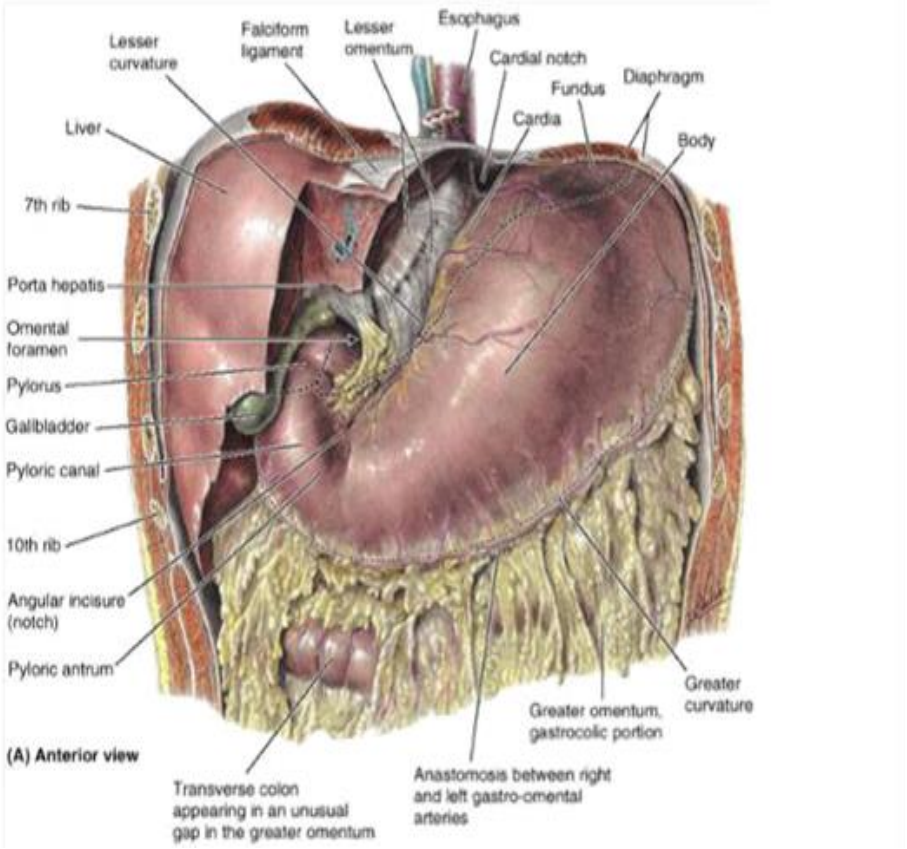


Fig. 6 Stomach with greater and lesser omentum[27]

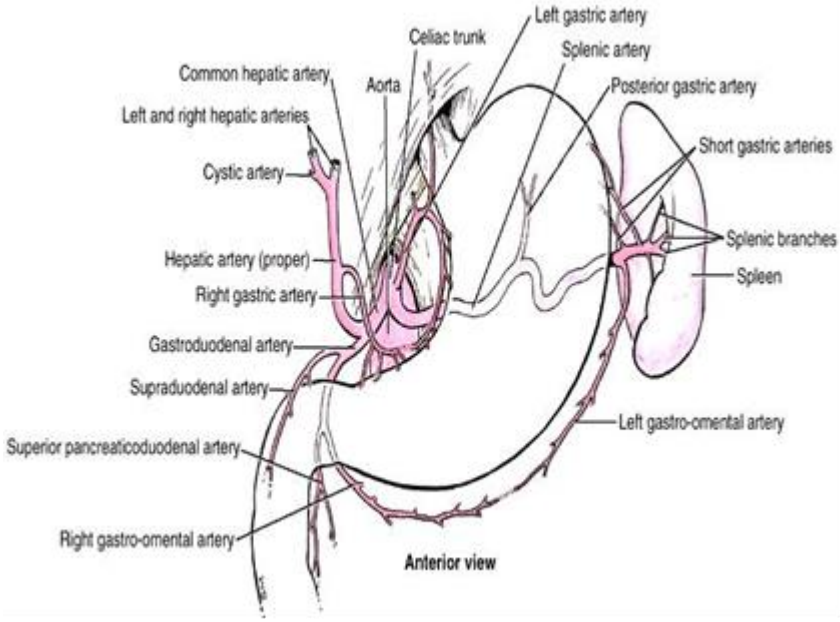


Fig.7.Arterial Supply to Foregut: Esophagus, Stomach, Liver, GB, Pancreas and Spleen[27]

- **THE SMALL INTESTINE**

It extends from the pylorus to the ileo-caecal valve. It measures about 6 meters in length and consisting of duodenum, jejunum and ileum.

- **THE DUODENUM**: (dudekadaktulos – greek word meaning twelve fingers). It is 20 to 25 cms long and is the shortest, widest and most sessile part of the small intestine. It lacks mesentery, and is partially covered by peritoneum. It consists of four parts:

  1. The first (superior) part, about 5 cm long.
  2. The second part (descending) 8 to 10 cm long.
  3. The third (horizontal) part about 10 cm long.
  4. The fourth (ascending) about 2.5 cm long.[27]

- **Arterial supply**: right gastric, right gastro-epiploic, supraduodenal and superior and inferior pancreatico-duodenal arteries.
- **Venous drainage**: Veins end in the splenic, superior mesenteric and portal veins.
- **Nerve supply**: Are from the coeliac plexus.

- **THE JEJUNUM AND ILEUM**

The small intestine except the duodenum is attached to the posterior abdominal wall. The jejunum is proximal and is two fifth , the ileum being distal is three fifth. The jejunum lies largely in the umbilical region. It is comparatively thick walled and measures about 2.3 to 2.8 meters. The ileum is thinner than the jejunum. It is present in the hypogastric and pelvic region. The length varies from 3.6 to 4.2 meters.

- **Blood Supply:**

  Arterial supply: Jejunal and ileal arteries stem from the superior mesenteric artery.

  Venous drainage: The veins follow the arteries.

  Lymphatic drainage: The lymph vessels (lacteals) form an intricate plexus in mucosa and submucosa, and are joined by vessels from lymph spaces present inferiorly in solitary follicles. They drain to larger vessels at the mesenteric aspect of the gut, eventually terminating in pre-aortic lymph nodes.[27]

  Nerve supply: Vagus and thoracic splanchnic nerve innervate through superior mesenteric plexus and celiac ganglion.

- **THE LARGE INTESTINE**

It extends from the ileocecal junction to anus and is about 1.5 meters long. The parts being caecum, ascending colon, transverse colon, descending colon, sigmoid colon, rectum and anus. On the surface there are longitudinal bands of muscle fibres called taenia coli, each about 5mm wide which start from the base of appendix and extend from caecum to rectum. They are termed as taeniae libera, taeniae mesocolica and taeniae omentalis. Along the sides of the taeniae there are tags of peritoneum filled with fat, called epiploic appendages (appendices epiploicae). Haustra are the sacculations seen characteristically in large intestine.

- **THE CAECUM:**

It lies in the right iliac fossa and ia about 6cms long, blind cul-de-sac. It lies behind the greater omentum. There is frequently a peritoneal recess behind the caecum called the retrocaecal recess. The caecum is supplied by the ileocaecal artery. The veins on the other hand drain into the superior mesenteric vein.

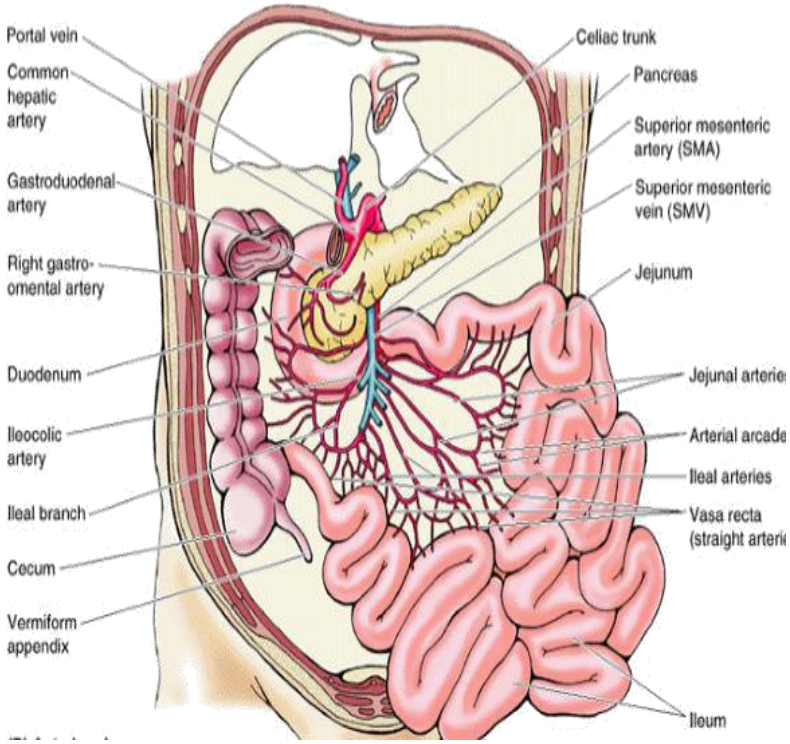


Fig. 8. Arterial supply and venous drainage of the small intestine.

Supply of SMA- jejunoileal, ascending colon and major part of transvers colon

THE VERMIFORM APPENDIX:

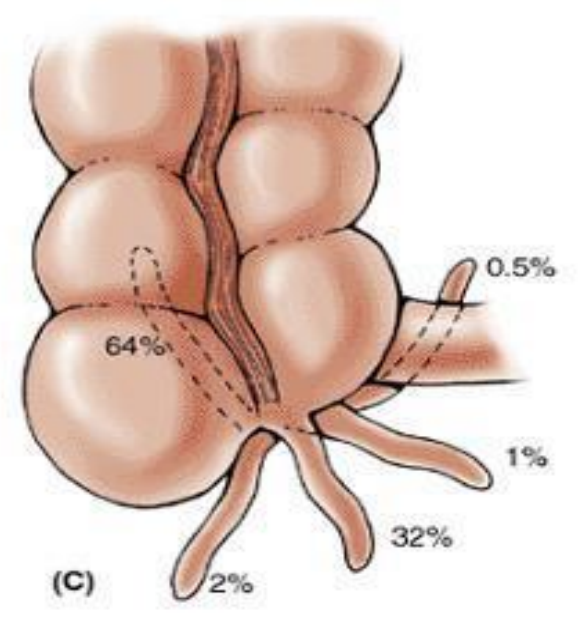


Fig.9. The approximate incidences of various locations of the appendix

It is narrow vermian (worm-shaped) tube, which arises from the posterio-medial caecal wall, measuring 2 cm or less below the terminal part of the ileum. It measures approximately 9 cm long

Different positions of appendix are:

(1) Retro caecal and retrocolic

(2) Pelvic

(3) Pre-ileal

(4) Post ileal

(5) Sub-caecal

(6) Para-caecal.

But the position of the base of the appendix usually remains constant corresponding to the McBurney's point.

The three taeniae coli converge at the base of appendix, merging into its longitudinal muscle. The meso-appendix connects the appendix to the lower part of the ileal mesentery.[26]

- **THE ASCENDING COLON**

It is about 15 cm in length and is covered by peritoneum devoid posteriorly, which is connected to iliac fascia, ileo-lumbar ligament, quadratum lumborum and perirenal fascia. **The right colic flexure**: It is at the junction of the ascending colon and transverse colon. Its posterior surface is not covered by peritoneum and is in direct contact with renal fascia.

- **THE TRANSVERSE COLON**

It is about 50 cms long and is present in the lower umbilical or upper hypogastric region. The posterior surface at its right end is devoid of peritoneum where is attached by areolar tissue to front of the descending part of the duodenum and the head of the pancreas, but from latter to the left colic flexure it is almost completely inverted by peritoneum connecting it to the anterior border of the body of the pancreas by the transverse meso-colon.

The left colic flexure: It is the junction of the transverse colon and descending colon.

DESCENDING COLON:

It is about 25 cm in length extending from the left colic flexure to sigmoid colon and vertically up to the iliac crest, and then inclines medially on the iliacus and psoas major to reach the pelvic brim, where it is continuous with sigmoid colon.

THE SIGMOID COLON (PELVIC COLON)

It is about 40 cm in length and normally lies in the lesser pelvis. It is closely surrounded by peritoneum, forming a mesentery, the sigmoid meso-colon.

THE RECTUM

The sigmoid colon continues as rectum at level of the third sacral vertebra and below with anus and about 2-3 cms in front of and slightly below the coccygeal tip. Sacculations, appendices epiploicae and taeniae are absent in the rectum. The peritoneum is related only to the upper 2/3, covering its front and sides above and covering only in front at lower 1/3 from which reflects on to the bladder in males to form rectovesical pouch and on the posterior vaginal wall in females forming the

recto-uterine pouch. The lower 1/3 is related to urinary bladder, the terminal part of ureters, the seminal vesicles, the deferent ducts and the prostate. The lower 1/3 of rectum is corresponds to the lower part of the vaginal[26]

THE ANAL CANAL

The anal canal is the end part of the large intestine. It measures about 4cm long, situated below the level of pelvic diaphragm which lies in the perineum (anal triangle) between the right and left ischio-rectal fossa extending from the anorectal junction to the anus. It is surrounded by sphincters which keep the lumen closed in antero-posterior slit.

HISTOLOGY OF GASTRO-INTESTINAL TRACT

From inside out, the alimentary canal wall has four layers. The innermost layer is the mucous membrane formed by the lining epithelium, lamina propria and the muscularis mucosa consisting of the smooth muscle. Beneath the mucosal membrane, is a layer of loose areolar tissue, the submucosa. Muscularis externa surrounds the submucosa and the muscularis externa is covered by advential or serous layer.[28]

THE LINING EPITHELIUM AND MUCOSA: -the gut is lined by columnar epithelium except in oesophagus and lower anal canal. In the former two places it is stratified squamous epithelium and serves as a barrier for protective function. The lamina propria comprises collection of lymphoid tissue – gut associated lymphatic tissue (GALT), numerous blood and lymphatic capillaries that take part in the immune mechanism and in transportation of luminal contents away from the lumen of the intestines.

SUBMUCOSA: - This is a loose areolar tissue layer consisting of plexuses of blood vessels as well as lymphatics. An autonomic nerve plexus called Meissner's plexus in this layer controls the activity of the glands and smooth muscle.

MUSCULARIS EXTERNA: - It comprises of 2 layers of smooth muscles - the outer longitudinal muscle layer, and the inner circular muscle layer. The connective tissue between the two muscle layer contains an autonomic nerve plexus called the Myenteric plexus or Auerbach's plexus. This plexus controls the activity of smooth

muscles.

SEROUS OR ADVENTITIAL LAYER: - It consists of mesothelium and loose connective tissue layer. Numerous neurovascular bundles present with in the adventitia

PHYSIOLOGY OF PERITONEUM

Peritoneum is a highly permeable membrane. It is a mesothelial lined cavity. These cells secrete serous fluid into the abdominal cavity, approximately 100 ml of fluid that contain less than 300 cells/mm^3, 40% - macrophages, 50% - leukocytes, 10% eosinophils and mast cells, protein < 3 g /dl. The peritoneal fluid acts as a lubricant and helps to facilitate peristalsis. As for its bactericidal property, it also plays a major role in the immune mechanism of the body by activation of complement cascade.

Transfer of substances across the peritoneal membrane is rapid bi-directional and is largely because of the greater surface area. This property is exploited in peritoneal dialysis and also used for the administration of fluid, electrolytes, antibiotics and even blood.

The direction and rate of fluid movement are mainly determined by the opposing effects of hydrostatic pressure and serum oncotic pressure in the portal venous system and lymphatics. The rate of diffusion is determined by the concentration gradient concentration gradient between blood and peritoneal fluid, where it occurs via blood capillaries and to a lesser extent, the lymphatic channels.[29]

The lymphatic system is responsible for the removal of non-irritating particles

and colloids into the systemic circulation. Absorption into lymph takes place mainly from the diaphragmatic surface which is supported by the diaphragmatic propulsive action.

Peritoneal fluid is a part of extra cellular fluid which is actively collected in the peritoneal cavity hence is vulnerable for excessive collection of fluid. Factors contributing for this include - (1) transudation of fluid rich in protein through the liver surface to abdominal cavity when the pressure in the liver sinusoids rises to 5 to 10 mmHg above the normal, (2) Positive gradient between capillary pressure in visceral peritoneum and elsewhere in body caused by the resistance of portal blood flow through the liver[30,31]

PHYSIOLOGY OF THE GASTRO-INTESTINAL SYSTEM

The major function consists of storage, digestion and absorption and propagation of food. Some of these mechanisms depend upon intrinsic properties of the intestinal smooth muscle. Visceral reflexes or the actions of the gastro-intestinal hormones also play a major role

- **STOMACH**: It is a major reservoir for storage of food, mixing of food with gastric acid.

Gastric secretion: Gastric juice around 2500 ml daily is secreted by cells of the gastric-glands. The main contents of the juice are dilute hydrochloric acid and pepsinogen.

In normal individuals the gastric mucosa is protected by the adverse effects of concentrated hydrochloric acid partially because of mucus present in the gastric juice.

The gastric mucosa also secretes bicarbonate which mixes with the mucus and this bicarbonate mucus forms as unstirred layer containing pH of about 7.0. This unstirred layer plus the surface membrane of the mucosa cells and the tight junctions between them constitute the mucosal carbonate barrier that protects the mucosa cells from damage by gastric acid. Prostaglandins stimulate mucus secretion and aspirin and NSAIDS inhibit prostaglandin synthesis.[32]

Gastric motility and emptying

The peristaltic waves are most marked in distal half of stomach, when well-developed they occur at a rate of 3mm/min. Peristaltic contraction occurs when food enters the stomach which mixes quirt it into the duodenum at a steady rate.

Regulation of gastric secretion and motility

The following phases of gastric juice secretion occurs –

A. **Cephalic phase:** It is mediated by vagal activity from both psychic arousal and reflex from antral stimulation.
B. **Gastric phase:** these are primarily local reflex responses and responses to gastrin. Gastrin stimulates the parietal cell to form HCL. When the intragastric pH falls below 3, the release of gastrin is inhibited and secretion of HCL stops.
C. **Intestinal phase:** Secretin inhibits gastric juice secretion. Duodenal acidification provokes secretin release.

Regulation of gastric motility and emptying

The gastric emptying rate depends on the type of food ingested. Carbohydrate rich food is emptied earlier from the stomach than protein or fat rich food. Distension of the duodenum, products of protein metabolism and hydrogen ions present in the duodenal mucosa initiate the enterogastric reflex - a neural mediated decrease in

gastric motility. With a normal mixed diet the stomach empties in about three and half hours and with a rich fatty diet about four and half hours.[31]

- **SMALL INTESTINE**: Absorption is the major function of small intestine. Intestinal contents are diversified with the secretion from the mucosal cells, pancreatic juice and bile.

Intestinal motility

Small intestine mixes and churns the intestinal contents and forms chime which is propelled towards the large intestine. Movements occur in two forms - segmentation contractions and peristaltic waves. Myenteric nerve plexus play a major role in this. The contraction of the small intestines is co-ordinated by the small bowel slow wave. The frequency of slow wave drops from about 12/min in the jejunum to about 9/min in the ileum.

Brunners gland in the duodenum secretes a thick alkaline mucus that probably helps to protect the duodenal mucosa from the gastric acid. Substantial secretion of HCO_3 also occurs. Vagal stimulation increases the secretion of Brunner's glands.

- **COLON:** The major function of colon is absorption of water, Na^+ and other nutrients.

Motility and secretion

Segmentation contractions and peristaltic waves contribute to the colonic motility. Mass action contraction, i.e. simultaneous contraction of the smooth muscle over large confluent area moving the material from one portion of colon to another also occur.

The circular smooth muscle propogate colonic motions in a slow wave

fashion. The frequency of this wave increases along the colon from about 2/min at the ileo-caecal valve to 6/min at the sigmoid.[31]

Transit time in the small intestine and colon

The first part of a test meal reaches the caecum in about 4 hours and the entire undigested portion enters the colon in about 8 to 9 hours on the average. The first remnant of the meal reaches the hepatic flexure in 6 hours, splenic flexure in 9 hours and pelvic colon in 12 hours.

PERITONITIS

Peritonitis is a life-threatening condition that is often associated with bacteraemia and sepsis. The peritoneal cavity is divided into compartments which is lined with a serous membrane that can serve as a conduit for fluids. This property is exploited in peritoneal dialysis. A small amount of serous fluid is normally present in the peritoneal space, with a protein content (consisting mainly of albumin) of <30 g/L and <300 white blood cells (WBCs, generally mononuclear cells) per microliter. In bacterial infections, early influx of polymorphonuclear leukocytes (PMNs) and a prolonged subsequent phase of mononuclear cell migration occur[33].

BACTERIOLOGY

The normal flora of the intestinal tract plays a role in the production of peritonitis. These microorganisms are virtually non-pathogenic in health, in contrary they are hugely virulent in perforation peritonitis.

Factors favouring localization of peritonitis.[34]

a. Anatomical:

Transverse colon and transverse mesocolon deters the spread of peritoneal cavity from supracolic to infracolic compartment. When supracolic compartment overflows, as is often the case when a peptic ulcer perforates, it does over the colon into the infracolic compartment through the right paracolic gutter and hence to the pelvis.

b. Pathological :

The clinical outcome is determined by the adhesions formed around the affected organ. Inflamed peritoneum loses its glistening appearance and becomes

reddened and velvety. Flakes of fibrin appear and cause loops of intestine to become adherent to one another and to the parities. There is an outpouring of serous inflammatory exudates which are rich in leukocytes and plasma proteins which soon becomes turbid, if localization occurs , the turbid fluid becomes frank pus. Aperistalis in affected bowel which helps to prevent distribution of the infection. The greater omentum by enveloping and adherent to inflamed structures often forms a substantial barrier for spread of infection.[35]

Factors favouring diffuse generalized peritonitis

- Speed of peritoneal contamination is prime factor. If an inflamed appendix or hollow viscous perforates before localization has taken place, there will be an efflux of contents into the peritoneal cavity.
- Initiation of peristalsis by the ingestion of food, or even water, hinders localization. Violent peristalsis occasioned by the administration of a purgative or an enema may cause the widespread distribution of an infection that would otherwise have remained localized.
- The virulence of the pathogen may be so great as to render the localization of infection difficult or impossible.
- Smaller size of omentum in young children makes them vulnerable for infection.
- Disruption of localized collections may occur with injudicious and rough handling, e.g. appendicular mass or pericolic abscess.
- Immunodeficiency may result from drugs (e.g. steroids), disease (e.g. AIDS) or old age[35].

The microbiological organisms commonly isolated in peritonitis are:

1. **Intestinal bacteria**

a. Aerobic bacteria

E-coli (commonest) / Streptococci / Enterococci / Staphylococci / Klebsiella / Pseudomonas / Candida

b. Anaerobic bacteria

Bacteroides / Eubacteria / Clostridium / Pepto Streptococci / Fusobacterium

2. Non intestinal bacteria

Gonococcus / β-haemolytic streptococcus / Pneumococcus / Mycobacterium

Spread of bacterial invasion can occur either by an hollow viscus perforation or through a penetrating wound of the abdomen, through iatrogenic injury from insertion of drains, dialysis tubes, surgical trauma and foreign body or by direct extension of infection from an adjacent inflamed organ like appendix or gall bladder (Appendicitis, cholecystitis etc.)**.**

Peritonitis initiates a sequential cascade of events involving the peritoneal membrane, the bowel, the body fluid compartments, which further leads to secondary endocrine, cardiac, respiratory, renal and metabolic responses and MODS.[33]

AETIOPATHOGENESIS [17]

Contamination of the peritoneal cavity with microbiological organism is designated as peritonitis or intra-abdominal infection, and is classified according to etiology.

Primary microbial peritonitis

It occurs when micro- organisms invade the normally sterile boundaries of peritoneal cavity through haematogenous spread from the distant source of infection

or direct inoculation. This process is more common among patients who have existing long term ascites and in whom being treated with peritoneal dialysis for renal failure. These infections invariably are mono-microbial and rarely require surgical intervention.

Cultures will typically demonstrate the presence of gram-positive organisms in patients receiving peritoneal dialysis. In patients without this risk factor organisms can include E. coli, K. pneumoniae, pneumococci, and others.

Secondary microbial peritonitis

It occurs following contamination of the peritoneal cavity due to perforation or severe inflammation and infection of an intra-abdominal organ. Examples include appendicitis, perforation of any portion of the GI tract, or diverticulitis. Management requires an emergency laparotomy, source control to resect or repair the diseased organ; debridement of the necrotic, infected tissue and debris; antimicrobial agents administration directed against aerobes and anaerobes.

The response rate to effective source control and use of appropriate antibiotics has remained approximately 70 to 90%[17].

Secondary peritonitis may be classified as

1. **Acute Suppurative Peritonitis**

- eritonitis
- Other forms - Tuberculosis and other granulomatous peritonitis.

2. **Post-Operative Peritonitis**

    - Leaking suture line of anastomosis
    - Leak of simple suture

- Duodenal Blowout
- Other iatrogenic leaks

3. Post traumatic peritonitis

- Peritonitis after blunt abdominal trauma or penetrating abdominal wounds

Non traumatic perforation:

- Peptic ulcer
- Chronic gastric ulcer
- Acute erosive gastritis
- Neoplastic – Carcinoma stomach

Miscellaneous

- Volvulus stomach
- Corrosive gastritis
- Mallory Weiss syndrome

STAGES OF PERITONITIS

Diffuse peritonitis is characterized by three stages clinically

Stage 1: Stage of peritonism: This stage involves irritation of the peritoneum due to leakage of gastric juice into the peritoneal cavity (chemical peritonitis) which usually lasts for about six hours. On examination there might be a slight variation in the pulse, respiration and temperature. Diffuse tenderness with guarding are constantly present at the site of perforation. Great importance should be given to diagnose this condition at this stage as chances of survival of the patient gradually declines with passage of time.[34]

Stage 2: Stage of reaction

The irritant fluid becomes diluted with exudates. The symptoms are relieved

but signs should be looked for peritoneal reaction. Muscular rigidity continues to be present. The other two features are obliteration of liver dullness and shifting dullness. Rectal examination may elicit tenderness in the recto-vesical or rectouterine pouch. Erect x-ray of the abdomen will show air under the diaphragm in 70% of the cases.

Stage 3: Stage of diffuse peritonitis

The pinched and anxious face, sunken eyes and hollow cheeks- the so called Hippocratic facies, with raising pulse rate which is low in volume and tension, persistent vomiting, board like rigidity, increasing abdominal girth all give hint to the diagnosis of this condition and imminent death.[34]

PATHOPHYSIOLOGY

Primary responses in peritonitis

1. **Membrane inflammation**: Hyperaemia followed by transudation occurs in response to the insult. Edema and vascular congestion occur in the sub peritoneal tissues. The inflamed peritoneum alters absorption which in turn leads to transudation of fluid with low protein content from the extracellular interstitial compartment into the abdominal cavity. Diapedesis of polymorphs occurs. During early vascular and transudative phase, toxins and other materials present in the peritoneal fluid, are readily absorbed leading to septicaemia and systemic symptoms. This is followed by exudation of protein rich fluid that contains large amount of fibrin and other plasma proteins which bring about clotting which in turn results agglutination of loops of bowel with other viscera and the peritoneum in the area of inflammation. This response helps to confine the source of peritoneal contamination.[36]

2.Bowel response: Initial transient hyper motility occurs after which the motility becomes depressed and adynamic ileus develops. Distention of bowel occurs due to accumulation of air and fluid inside the lumen. The fluid secretion into the bowel lumen is enhanced and reabsorption is relatively impaired, stimulating sequestration of fluid in the bowel and decreased ECF volume.

3.Hypovolemia: Vascular dilatation and outpouring of exudates from the

extracellular, intracellular and interstitial compartments into the peritoneal space occurs. The loose connective tissue which is present tissue beneath the mesothelium of the visceral mesentery traps extracellular fluid as edema. The atonic dilated bowel also accumulates fluid in its lumen. This translocation of water, electrolytes and protein into a sequestrated "third space" functionally removes this volume temporarily from the body economy[36].

Secondary responses in peritonitis

Endocrine response: The adrenal medullary response is stimulated, with outburst of epinephrine and nor epinephrine which produce systemic vasoconstriction, tachycardia and sweating. Cortical hormone level rises during the first three days following peritoneal injury. Production of aldosterone and ADH are also increased in response to hypovolemia. Dilution hyponatremia occurs. Cardiac response**:** Decrease in extracellular fluid volume and progressive acidosis leads to decreased venous return and cardiac output. The heart rate increases in an attempt to maintain cardiac output. Progressive acidosis causes secondary dysfunction in cardiac contractility and further decrease in cardiac output.

Respiratory response: Abdominal distension, primarily due to adynamic ileus, coupled with restriction of diaphragmatic and intercostal muscles, results in decreased ventilatory volume and basilar atelectasis. Initial phase of peritonitis, increase in the rate of respiration may be noted. Ventilation perfusion imbalance results from continued pulmonary perfusion of under ventilated alveoli, producing increased intrapulmonary shunting of blood and peripheral hypoxemia.

Renal response: Hypovolemia, reduced cardiac output and increased serum levels of antidiuretic hormone and aldosterone all act synergistically on the kidney. The Renal blood flow is decreased which results in decrease in the glomerular filtrate and tubular

urine flow. Reabsorption of both sodium and water is increased.

Metabolic response: The metabolic rate is increased with a corresponding increase in peripheral oxygen demand. Oxygen delivery is diminished. The Poor circulation in the muscle and other peripheral tissues leads to shift to anaerobic metabolism. End products of carbohydrate metabolism accumulate and lactic acidosis develop.

Circulatory insufficiency due to hypovolemia accompanies peritonitis. Cardiac output decreases and compensatory vasoconstriction results in increased peripheral resistance, which in turn maintains essential perfusion to the heart and brain which result in decreased tissue perfusion and oxygenation, persistence of anaerobic glycolysis, progressive increase in lactic acid and other acidic metabolic end products. The decreased renal clearance of these acidic solutes contributes to metabolic acidosis[36]

Sequelae leading to multiorgan failure

Sepsis is a major risk factor in the development of multiorgan failure syndrome (MOFS). MOFS increases with severity and duration of shock. Injury to micro vascular system especially microvascular endothelium, is common to ischaemia reperfusion injury and multiorgan failure syndrome. Toxic neutrophil products like proteases, elastase, collagenase, cathepsin G are bactericidal and during endothelial cell injury which produce free oxygen radicals causing endothelial activation injury directly through both membrane peroxidation and increased neutrophil adherence in chemotaxis. Miles and Burke suggested decisive period for bacterial infection which is the time required for bacterial numbers in fluid or tissue to exceed 105 / mm3 or (per gm. of tissue) and to establish an infection. Steps must be taken to deter infection before bacterial numbers reach these levels[37].

Features of sepsis include temperature >101°F , heart rate >100 / minute, respiratory rate >20 /minute, leukocytosis (WBC > 12,000mm3) (or <4000mm3), manifestation of inadequate organ perfusion/ function, diminished mental status, acidosis (plasma lactate >3.0 mmol /L), urine output < 30ml/ hour or 0.5 ml/kg per hour, hypoxemia (PaO2 <70) and Swan Ganz readings :- Cardiac output <4.0L min per m^2 or PCWP < 8 mm Hg[37].

Criteria for multi organ failure as described by Fry D *et al* are:

- Pulmonary - 5 or more consecutive days of need for ventilator support at an FiO2 of ≥0.4
- Hepatic - Bilirubin >2gm /dl, SGOT / LDH > twice normal.
- Renal Failure - Serum creatinine >2mg/dl
- GIT Failure - Significant upper GI hemorrhage[38]

HOLLOW VISCUS PERFORATION

Perforation peritonitis is the most common surgical emergency in India.

Despite advances in surgical techniques, antimicrobial therapy and intensive care support, the management of peritonitis continues to be highly demanding and complex[39].

Peritonitis is inflammation of the peritoneum and peritoneal cavity and is most commonly due to a localized or generalized infection. The Primary peritonitis occurs as a squeal to infection from bacteria, chlamydia, fungi or mycobacteria in the absence of perforation of the gastrointestinal tract, whereas secondary peritonitis occurs as a result of gastrointestinal perforation. The common causes for the latter include perforated peptic ulcer, acute appendicitis, colonic diverticulitis and pelvic inflammatory disease[40].

Peptic ulcer disease leading to perforations was a common and major cause for morbidity and mortality until the latter half of the 20th century. Recently, both the incidence as well as prevalence of peptic ulcer disease as declined and both are now parallel. Amongst the two, duodenal ulcer perforations are more common (2-3 times) as compared to gastric perforations. Also, it is important to note that about a third of gastric perforations are actually due to gastric carcinomas[41].

The mortality rate when studied in toto shows that there is a relatively high ~20-40% chance of mortality. This is mainly due to complications like septic shock and multi organ failure[41].

PEPTIC ULCER PERFORATION

It is crucial to emphasize on peptic ulcer perforations as it without doubt one of the most frequent surgical emergencies after acute appendicitis and acute intestinal

obstruction. There is a fall in the incidence of peptic ulcer ulcers and the elective surgeries for the same, which is attributed to the era of H2 blockers and proton pump inhibitors. In spite of the reduction in incidence rates, the rates of peptic ulcers going into perforation and requiring emergency surgery, prolonged stay at hospital and chances of mortality in general has remained almost unchanged through the last two decades. This is most likely because of increase in the inadvertent use of NSAIDS, corticosteroids as well as irregular use of H2 antagonists[40].

Perforation of peptic ulcer may be classified as acute perforation, sub-acute perforation, chronic perforation, perforation associated with haemorrhage, perforation of intra thoracic gastric ulceration and pseudo perforation. Acute perforation is further classified into 3 stages

1. Stage of peritonism or primary stage
2. Stage of peritoneal reaction or secondary stage
3. Stage of bacterial peritonitis or tertiary stage.

In the **primary stage,** the patient usually complains of acute agonising pain in the epigastrium or right hypochondrium which is later rapidly becomes generalized. Soon, the patient suffers from a stage of prostration following which he/she may become immobile and helpless.

This is then followed by the **secondary stage,** which is like a transition phase that takes approximately 3-6 hours after the primary stage. The length of this stage depends on the size of the perforation and the amount of peritoneal soiling. In this phase, there are chances of spontaneous sealing of the perforation. But, if there is gross leakage of the gastric contents, patient is likely to progress to the stage of septic peritonitis. The special feature of this phase is that there may be diminution of pain. Thus, this stage can occasionally be referred to as the stage of delusion[42].

Tertiary stage is characterized by diffuse peritonitis. It begins about 12 hours after perforation lasting for about 34 hours until the final paralytic obstruction sets in. this stage is seen by rapid multiplication of the pathogens and peritoneal fluid becomes highly purulent. Intestine is slowly filled with gas and fluid. Intestinal movements diminish and finally there is the onset of paralytic ileus. Patient develops typical Hippocratic facies. The patient is toxic, dehydrated in shock.

Perforation is the second commonest complication of peptic ulcer. Though nonsurgical treatment can be used occasionally in the stable patient without peritonitis in whom radiologic studies document a sealed perforation, surgery is almost always indicated. Ulcer is usually suspected in patients with acute perforation and GI blood loss (either chronic or acute).

Simple patch closure (done in patients with hemodynamic instability and/or exudative peritonitis signifying a perforation >24 hours old), patch closure and high Selective vagotomy, or patch closure and truncal vagotomy+drainage are the options for surgical treatment of perforated duodenal ulcer.. In all other patients, the addition of HSV may be considered because studies have reported a negligible mortality with this approach[43]

In the stable patient without multiple operative risk factors, perforated gastric ulcers are best treated by distal gastric resection. Vagotomy is usually added for type II and III gastric ulcers. Patch closure with biopsy; or local excision and closure; or biopsy, closure, truncal vagotomy, and drainage are alternative operations in the unstable or high-risk patient, or in the patient with a perforation in an inopportune location (e.g., juxtapyloric). All perforated gastric ulcers biopsy should be taken.

Perforated Appendicitis

Appendiceal inflammation may progress to necrosis of the appendiceal wall, followed by perforation. When mere acute appendicitis has progressed to perforation, other symptoms will arise. Patients complain of severe abdominal pain for 2 days or more, commonly localizing to the right lower quadrant if the surrounding intra-abdominal structures have walled the perforation off, or the pain may become diffuse if generalized peritonitis ensues, often with high fevers to 102°F (38.9°C) with rigors. A history of poor dietary intake and dehydration may also be elicited. Most patients with perforation complicating acute appendicitis present with clinical features associated to the inflamed appendix itself or to a localized intraperitoneal abscess

Due to perforation of a retrocecal appendix, abscesses can also form in the retroperitoneum .An intraperitoneal abscess could fistulise to the skin, resulting in an enterocutaneous fistula. Pylephlebitis (septic portal vein thrombosis) presents with high fevers and jaundice and can be confused with cholangitis; it is a dreaded complication of acute appendicitis and carries a high mortality[44].

Small Bowel Perforation

Most common cause of small bowel perforation is iatrogenic injury incurred during GI endoscopy Other etiologies of small bowel perforation include infections (tuberculosis, typhoid), Crohn's disease, ischemia, drugs (e.g., potassium-and NSAID-induced ulcers), radiation-induced injury, Meckel's and acquired diverticula, neoplasms (lymphoma, adenocarcinoma and melanoma).

Among iatrogenic injuries, duodenal perforation during ERCP with endoscopic sphincterotomy is the most common. This complication occurs in 0.3 to 2% of cases. Patients who have undergone Billroth II gastrectomy are at increased

risk of duodenal perforations as well as free jejunal perforations during ERCP.

CT scanning is the most sensitive test for diagnosing duodenal perforations; positive findings include pneumoperitoneum for free perforations, retroperitoneal air, contrast extravasation, and paraduodenal fluid collections. Surgical repair with pyloric exclusion and gastrojejunostomy or tube duodenostomy are required in intraperitoneal duodenal perforations. Jejunal and ileal perforation require surgical repair or segmental resection[16].

Typhoid Enteritis

`Typhoid fever most commonly present in areas with contaminated water supplies and inadequate waste disposal. Most commonly affected are children and young adults. Typhoid enteritis is caused by Salmonella typhi. The pathologic events are initiated in the intestinal tract after oral ingestion of the typhoid bacillus. These organisms penetrate the small bowel mucosa, initially to the lymphatics and then systemically. Hyperplasia of the reticuloendothelial system, including lymph nodes, liver, and spleen occurs. Hyperplastic payer patches in the small bowel may subsequently ulcerate with complications of haemorrhage or perforation.

Typhoid fever diagnosis is made by isolating the organism from blood (90% of the patients during the first week of the illness are positive), bone marrow, and stool cultures. High titters of agglutinins against the O and H antigens are strongly suggestive of typhoid fever.

Complications requiring potential surgical intervention include haemorrhage and perforation. The incidence of haemorrhage was reported to be as high as 20%. Intestinal perforation through an ulcerated peyer's patch occurs in about 2% of cases.

Simple closure of the perforation is the treatment of choice for a single perforation in the terminal ileum .With multiple perforations, which occur in about one fourth of the patients, resection with primary anastomosis or intestinal loops exteriorization may be required[16]

Evaluation of perforation

Clinical features are of sudden onset, followed by a distinct intermediate latent interval, which in turn gives place to the classic signs and symptoms.

Symptoms of early peritonitis

- **Pain**

It is the most important finding in patients with acute abdomen. It varies considerably in intensity. It is as a rule, most intense in that part of the abdominal wall which lies immediately over the spreading edge of the peritoneal inflammation. When the peritoneum inflammation subsides or localizes pain diminishes in severity and becomes limited to one area of the abdomen.

This symptom makes the patient to seek medical assistance. The characteristics of pain like the onset, site, type, radiation aids in the diagnosis.

Sudden onset of pain is feature of all perforation. In acute appendicitis, diminution of pain may indicate perforation of an obstructive gangrenous appendix. Constant burning pain is a feature of peritonitis and often seen in perforated peptic ulcer. Sudden pain due to perforation of peptic ulcer usually takes place in the afternoon

after the meal. Since movement aggravates the pain, patient assumes a still posture. Deep inspiration will aggravate pain due to diaphragmatic irritation[45].

- **Vomiting**

Initially the episodes may be less, but as the peritonitis advances, it becomes persistent. Often pain precedes vomiting. Initially the vomitus consists of gastric contents, later it is bile stained and when the obstruction becomes complete it becomes faeculent. Vomitus may rarely contain frank blood in case of perforation due to gastric ulcer, duodenal ulcer and gastric neoplasm. In early stages of peritonitis vomiting is reflex in origin. Later it is caused by paralytic ileus[45].

- **Fever**

The temperature is often sub-normal, or normal in cases in which onset is sudden.

It tends to rise gradually as true peritonitis supervenes. A rising pulse rate and falling temperature are of the greatest significance. As the disease process advances, the pulse steadily rises and will be bounding. Later it becomes weak and more rapid[45].

- **Distension of the abdomen**: Presents in the later stages where paralytic ileus has already present along with peritoneal fluid collection. The distension may present in the upper or lower abdomen in early stages but will be all over the abdomen in late stages.

Bowel habits: Absolute constipation is a feature of peritonitis. In the early stages, there may be history of loose motions because of irritation of rectum by pelvic collections. Past history of alternate constipation and diarrhoea are the features of tubercular enteritis, carcinoma colon and worm infestation. In ulcerative colitis there will be abrupt explosive severe diarrhoea with bleeding but in crohn's most patients have diarrhoea that is usually not bloody. Peptic ulcer perforation or carcinoma

stomach can be diagnosed by history of melena

- **Other history**: includes history of drugs particularly NSAIDs and steroids or strong acids ingestion, loss of appetite, loss of weight and jaundice.

Signs of early peritonitis

- **Inspection**: The position of the patient in the bed is often characteristic. Patient lies still with the legs drawn up in an effort to relieve tension on abdominal muscles. There is absence or marked diminution of abdominal respiratory movements. Respiration is shallow, rapid and thoraco-abdominal in nature. Patient may look toxic and dehydrated.
- **Palpation**: Marked abdominal tenderness and guarding will be present. Rigidity may be present in later stages. Rebound tenderness can be elicited. It may be localized, in which the peritoneal inflammation has involved only a limited area
- **Percussion**: The abdomen is resonant and tympanic because the intestines are filled with gas. Liver dullness is often obliterated.
- **Auscultation**: Bowel sounds are diminished or absent due to associated ileus[46].
-

INVESTIGATIONS

Blood Studies

A complete blood count showing haemoglobin, haematocrit and white cell counts taken during admission is very important. Serious infection is indicated by a rising or marked leucocytosis, especially with a shift to the left on peripheral blood smear. On the other hand, a low WBC count is indicative of viral infection such as mesenteric adenitis and gastroenteritis. If hypovolemia is suspected, serum electrolytes, blood urea and

creatinine are important. ABG is must in peritonitis, ischaemic bowel, pancreatitis, hypotension, or septicaemia as metabolic acidosis which is often missed, may be the first hint to a serious underlying disease. A raised serum amylase level corroborates a clinical diagnosis of acute pancreatitis. Clotting studies should be done if history is suggestive of a haematological disorder. Recently to asses the severity of infection acute inflammatory markers like C - reactive protein, Interleukins, Ceruloplasmin, and Transferrin are being tested[47,48].

Urine Tests

Dark urine reflects dehydration. Urine ketone bodies may be present in a patient with uncontrolled diabetes mellitus. Routine urine examination can help in assessing any urinary tract infection.

Imaging:

1. Radiography

Erect chest radiograph or erect abdomen radiograph:

The presence of free, intra-abdominal gas indicates perforation of a viscus. Free gas is easily identified on the erect chest radiograph. As minimal as 1 ml of free gas may be identified, on an erect chest or a left lateral decubitus abdominal radiograph. Small amounts of gas are detectable under the right hemi-diaphragm on erect radiographs, but on the left it can be difficult to distinguish free gas from stomach and colonic gas. There are many circumstances when interpretation of an erect chest radiograph is difficult and where radiologist or clinician may be fooled into thinking that there is a perforation (pseudo-pneumoperitoneum). A lateral decubitus radiograph can resolve the problem by demonstrating gas between the liver

and the abdominal wall[49].

Supine radiograph:

It is also important to be able to recognize the signs of pneumoperitoneum on supine radiographs. In many patients, particularly those who are unconscious, have suffered trauma, are old, or are critically ill, perforation may be clinically silent as it is over-shadowed by other serious medical or surgical problems. A supine abdominal radiograph may be the only radiograph that can be obtained in these cases. The gas under diaphgram i.e in right upper quadrant mainly in sub hepatic and hepatorenal fossa (Morrison's pouch) is present in nearly half of the patients. Visualization of both the outer and inner walls of a bowel pneumoloop is known as Rigler'ssign . The bowel loops then take on a _ghost-like' appearance. This sign can be misleading if several loops of bowel lie close together. The falciform or umbilical ligaments may be demonstrated by free gas lying on either side. Air can be made out in the fissure for the ligamentum teres.

Signs of a pneumoperitoneum on supine radiograph:

1. Right upper-quadrant gas

 Perihepatic

 Subhepatic

 Morrison's pouch

 Fissure for ligamentum teres

2. Rigler's[double wall] sign

3. Ligament visualization

 Falciform [ligamentum teres]

 Umbilical[inverted V sign] medial and lateral

4. Urachus

5. Triangular air

6. Foot ball or air dome sign

7. Scrotal air [in children]

Conditions simulating a pneumoperitoneum [pseudo-pneumoperitoneum]

 Intestine between liver and diaphragm- chiladiti‘s syndrome

 Subphrenic abscess

 Curvilinear atelectasis in the lung

 Sub diaphragmatic fat

 Diaphragmatic irregularity

 Cysts in pneumatosis intestinalis

Causes of pneumoperitoneum without peritonitis

(i) Silent perforation of viscus that has sealed itself, in:

 Elderly patients

 Patients on steroids

 Unconscious patients

 Patients being ventilated

 Serious medical conditions

(ii) Post operative

(i) Peritoneal dialysis

(ii) Perforated jejuna diverticulosis

*(iii)*Perforated cyst in pneumatosis intestinalis

(iv) Tracking down from a pneumomediastinum

(v) Stercoral ulceration

(vi) Entry of air through the fallopian tubes

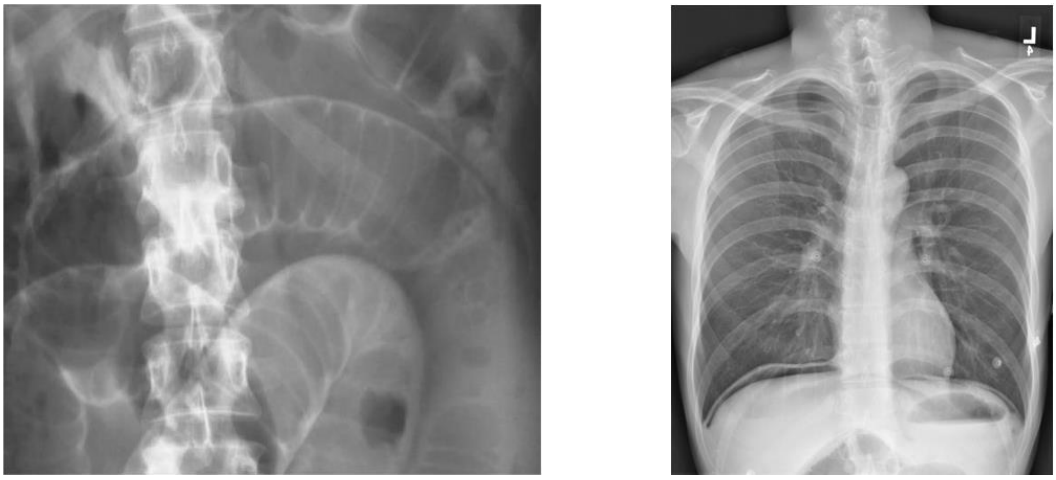


Figure 10 : Radiography: Erect chest radiograph or erect abdomen radiograph

a. Rigler's sign of pneumoperitoneum. The bowel loops have a ‗ghost-like‘appearance due to gas both inside and outside making the wall more apparent.

b. Air under diaphragm

 Ultra sound(US) scanning:

Undoubtedly ultra sound has value in certain situations as pelvic peritonitis in females and localized right upper quadrant peritonism.US plays a role in confirming or excluding specific diagnoses (e.g. subphrenic abscess). The diagnostic accuracy of these modalities has also been affirmed in clinically equivocal cases of acute appendicitis[52].

Computed tomography:

Discontinuity of the bowel wall may indicate the perforation site. Focal wall thickening is a feature of perforation of the alimentary tract which occurs in peptic ulcer disease, trauma, foreign body, iatrogenic event, ischemia, inflammation, appendicitis, diverticulitis and neoplasm. Accurate evaluation of bowel wall thickening can only be performed on the distended bowel loop[50].

Bowel wall thickening:

> 8 mm in stomach and duodenum,

> 3 mm in jejunum and ileum,

> 6 mm of the appendiceal caliber and

> 5 mm in colon and rectum including soft tissue mass

Upright chest films can detect pneumoperitoneum in only 30% of cases but abdominal CT can demonstrate free air in 100% of cases.

CT displays intra and extra-peritoneal free air in amounts too small to be visualized on plain radiography, but it can also recognize the underlying cause and specify the location of the disease. To assess the distribution of free air, the peritoneal cavity is divided into two compartments, the supra-mesocolic compartment and the infra-mesocolic compartment, based on the level of transverse mesocolon. The presence of free air in periportal area in supra-mesocolic compartment was defined as periportal free air (PPFA) and the sign was positive.

The ligamentum teres sign which is free air confined to the intra-hepatic fissure for ligamentum teres can be seen in the perforation of the duodenal bulb or stomach. The falciform ligament sign‖ is that free air or air-fluid level crossing the midline and accentuating the falciform ligament can be seen more in the perforation of the proximal (stomach, duodenum, jejunum, and ileum) GI tract perforation

The probability of perforation is high when free air is noted in periportal area.

The PPFA sign was the most significant finding in distinguishing upper from lower GI tract perforation. When there is free air in the periportal area, it suggests a high probability of perforation in the upper GI tract[51].

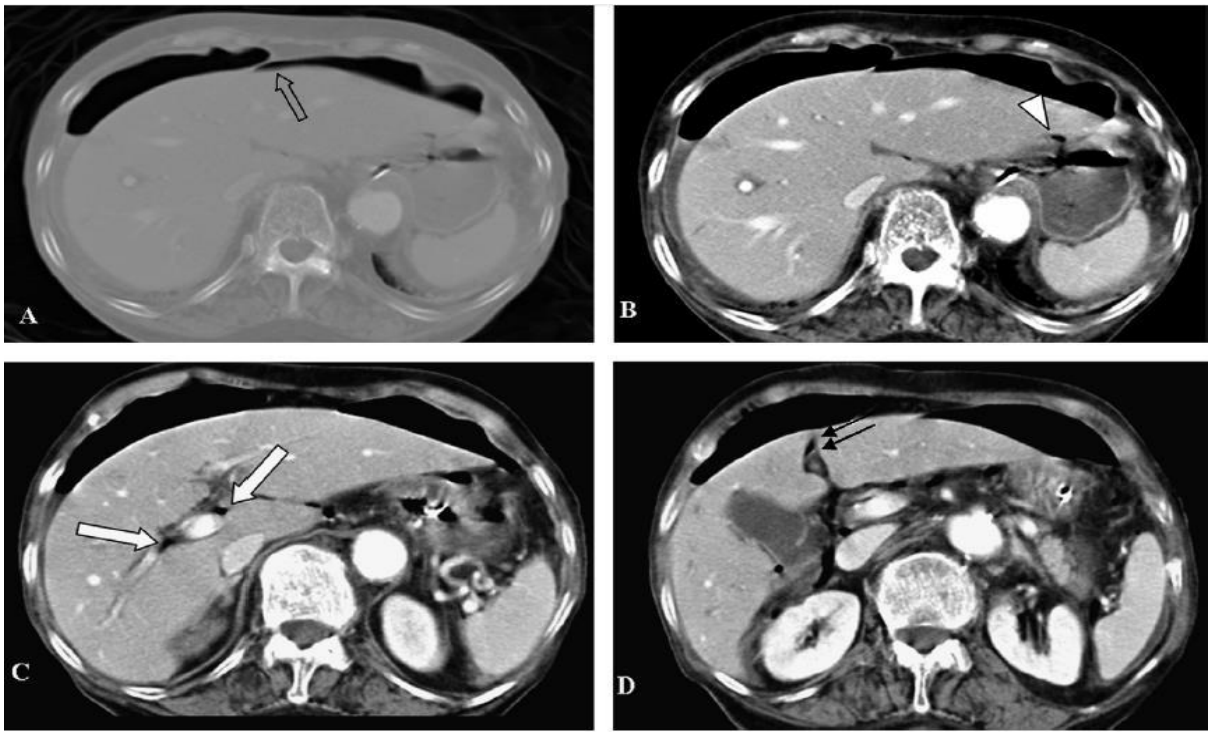


Figure 11 : Computed tomography

(A) Contrast-enhanced abdominal CT scan shows that the falciform ligament sign (open arrow) is well demonstrated on the wide-window setting.

(B) Mural defect in the proximal body of stomach (arrowhead).

(C) CT scan shows the periportal free air sign (arrows).

(D)Free air is noted in the fissure for ligamentum teres

DIFFERENTIAL DIAGNOSIS FOR PERFORATION

These can be divided into

1. Intra-abdominal conditions

2. Intra thoracic diseases and

3. Metabolic or neurologic conditions

Intra-abdominal conditions

- Acute Appendicitis
- Acute pancreatitis
- Acute cholecystitis
- Acute intestinal obstruction
- Mesenteric ischemia / ruptured aneurysm
- Ruptured ectopic gestation
- Perforated diverticulitis and
- Peritonitis following trauma
- Torsion ovary
- Obstructed /inflamed Hernia
- Haemorrhagic ovarian cyst

Intra-thoracic diseases

Myocardial infarction, acute pericarditis

Pneumonia, pleurisy, spontaneous pneumothorax

Rupture of the esophagus due to emetic abuse

Metabolic and neurologic conditions

Acute porphyrias, diabetes, uremia, hyperlipidemia, acute poisoning

Meningitis, multiple sclerosis and neuro-syphilis.

COMPLICATIONS OF PERITONITIS[45]

a) Systemic complications

- Septicemic / endotoxic shock
- Bronchopneumonia / Respiratory failure
- Renal failure
- Bone marrow suppression
- Multisystem failure
- Death

b) Local complications

- Intestinal obstruction
- Paralytic ileus
- Residual or recurrent abscesses - Subphrenic / Paracolic / Pelvic
- Wound infection / Wound dehiscence
- Portal pyaemia[45].

MANAGEMENT OF PATIENT WITH PERITONITIS

Timely surgical intervention is very important in order prevent the bacteria and their adjuvants from invading into the peritoneal cavity.

The management of peritonitis is broadly divided to supportive and surgical line of management.

Principles of Supportive measures:

A. Prevention/Treatment of hypovolemia and shock

B. Elimination bacteria with antibiotics

C. Support of failing organ systems

D. Maintenance of adequate nutrition

Hypovolemia to some degree is inevitable in all cases of peritonitis. This occurs because ECF enters into the peritoneal cavity called as “third spacing” of ECF. The rate of immediate resuscitative care that needs provided depends upon the severity of the condition. In a young, surgically fit patient who has presented at its earliest stages, early surgery is preferred over prolonged resuscitation. In contrast, elderly patients who present late would require resuscitation first, with surgery being on hold until patient is fit following reassessment.

During, fluid replacement vital parameters namely, the mental status, pulse rate, blood pressure and urine output need to be closely monitored. Invasive procedures such as peripheral arterial or central cardiac pressure monitoring catheters are not advocated except in high risk patients. In some cases, positive ventilation with supplementary oxygen may be required.

In order to decrease the abdominal distension as well as prevent aspiration into the lungs, nasogastric decompression is done. These patients may develop stress gastric ulcers for which prophylactic administration of antacids is done[45].

ANTIBIOTIC THERAPY

Empirical therapy should be started as soon as peritonitis is diagnosed, depending on the presumed bacteria present. Of all, E. coli and B. fragilis are the commonest and remain as the main target for therapy.

Table 2: Antibiotics for intra-abdominal infections

Mild to moderate intra-abdominal infection	**Severe intra-abdominal infection without renal dysfunction**	**Severe Intra-abdominal infection with renal dysfunction**
Second or third generation Cephalosporin Or β-lactamase inhibitor combination or Monobactum + Metronidazole	Carbapenem or Fluoroquinolone + Metronidazole or Aminoglycoside + Metnonidazale +/- Ampicillin	Carbapenem or Fluroquinolone + Metronidazole

OPERATIVE TREATMENT:

Principle 1 (Repair) :

Treat the source of infection

Principle 2 (Purge) :

Evacuate bacterial inoculum, pus (peritoneal 'toilet")

Principle 3 (Decompress):

Treat abdominal compartment syndrome

Principle 4 (Control)

Prevent or treat persistent and recurrent infection or verify both repair and purge

PRINCIPLE 1

The surgeries that are done in order to eliminate the source of infections have a varied spectrum, starting from procedures of simple perforation closure to those involving major resections.

If extensive bowel is gangrenous, exteriorization may be preferred. The perforation may be closed with pedicle or free omental grafts also.

The choice of the procedure, and whether the ends of resected bowel are anastomosed, exteriorized, or simply closed depends on the anatomical area from which the infection originated from, the severity of inflammation occurring within the peritoneal cavity as well as generalized septic process, in addition to the patient's comorbid conditions[55].

PRINCIPLE 2

All infectious peritoneal fluid and pus should be completely removed. Necrotic peritoneal tissue, if present, should be debrided. However, aggressive debridement should be avoided as it may cause excessive blood loss or even bowel injury.

Peritoneal lavage or irrigation must be done thoroughly with adequate pre-warmed normal saline, with or without the addition of antibiotics.

PRINCIPLE 3

It is important to note that when the patient is in the acute stage of peritonitis,

the peritoneum along with its sub mesothelial loose connective tissue has the capacity to absorb as much as 10 liters of inflammatory oedematous fluid.

Hence in most cases, draining the peritoneal fluid will reduce the abdominal compartment pressure. However, coexisting paralytic ileus, visceral and parietal oedema may raise the intraabdominal pressure to an extent resulting in compartment syndrome. The compartment syndrome will worsen when the abdominal wall is closed with tension. Thus, as a step to decrease the rise in intra – abdominal pressure and prevent the development of compartment syndrome, a laparostomy or a staged abdominal repair technique is done.

PRINCIPLE 4

Anticipation by the surgeon regarding the complications that can occur within post-operative period is very crucial as it will result in early diagnosis of any complication and if indicated, re-exploration. A re-laprotomy needs to be done in case the surgeon has any doubt pertaining to the eradication of the septic focus.

The surgical options are classified into:

1. Open abdomen / laparostomy (OPA)
2. Covered laparostomy (COLA)
3. Planned re-laparotomy (PR)
4. Staged abdominal repair (STAR)[53]

The open abdominal techniques (OPA and COLA) avert deleterious effects of increased intraabdominal pressure. These open abdominal techniques mainly consists of laparotomy without any approximation and suturing the abdominal fascia and skin to close it.

Disadvantages :

The major disadvantages that the surgeon may come across in managing these cases is that, exposed intestines are highly prone for perforations as there is no counteraction by the abdominal wall to the intraluminal pressure. Also, it is a very difficult task to have a definitive closure of the abdominal. This is mainly because the fascia retracts. Thus, the abdominal wall becomes weak and patient is at risk of developing a huge incisional hernia.

The main concern is to protect the exposed viscera. So based on the above principle, COLA is done but gap formed between the fascia is closed using a mesh or ethizip. The disadvantage of this method is that, again the patient is prone for raised intra-abdominal pressure and chances of landing with a large incisional hernia.

In order to tackle this situation, the concept of doing a planned re-laparotomy has come into picture. So, by "leaving the abdomen open" following the initial operation, the surgeon will be able to re-explore, irrigate, debride or close fistulas based on the necessity, either in OT or ICU. The time interval for re-exploration usually ranges from12 to 48 hours but may extend longer based on the given situation. The idea is to close the initial laparotomy wound on a temporary basis by using either retention sutures or ethizip.

The current trend is to follow a staged abdominal repair (STAR) which consists of a series of operations done over the abdomen associated with staged re-approximations and to lastly close the abdominal facia by suturing it. Therefore, temporary closure of the abdomen is done and the abdominal fascia is kept under controlled tension. Hence, by using this method the untoward consequences of increased intraabdominal pressure can be prevented[54].

INDICATIONS FOR STAGED ABDOMINAL REPAIR (STAR)

Critical patient's condition (hemodynamic instability) precluding definitive repair

Excessive peritoneal oedema (abdominal compartment syndrome: pulmonary, cardiac, renal, or hepatic dysfunction, and decreased visceral perfusion) can lead to intra-abdominal pressure > 15 mmHg. Thus, this causes undue tension with the abdominal cavity and does not allow closure of the abdomen.

1. Massive abdominal wall loss.
2. Difficulty to control the source of infection.
3. Incomplete debridement of necrotic tissue.
4. Uncertainty of viability of remaining bowel.
5. Uncontrolled bleeding.(the need for "packing")[53]

POST-OPERATIVE COMPLICATIONS[45]

SYSTEMIC COMPLICATIONS :

Acute renal failure

Cardio respiratory failure

Pulmonary oedema

Thrombo- embolic phenomena

Systemic inflammatory response syndrome

Septicaemia

Multi organ dysfunction

LOCAL COMPLICATIONS :

- Subphrenic abscess
- Pelvic abscess
- Leak at site of perforation closure

- Anastamotic leak
- Enterocuteneous fistula
- Wound infection
- Wound failure including burst abdomen
- Intestinal obstruction due to adhesions
- Incisional hernia

MATERIALS & METHODS

MATERIALS AND METHODS

This is a prospective clinical study conducted on 172 consecutive patients who presented to surgical department, R.L. JALAPPA HOSPITAL, TAMAKA, KOLAR with peritonitis secondary to hollow viscus perforation. The study period was from December 2016 to June 2018.

TYPE OF SUBJECTS:

All patients undergoing surgeries for peritonitis admitted under different surgical units.

CHOOSING SUBJECTS:

Number to be studied: 172-divided as 86 in each group comprising of odd and even serial numbers.

GROUP A: Patients with all odd serial numbers were included in this group and peritoneal lavage with povidone iodine in normal saline was used

GROUP B: Patients with all even serial numbers were included in this group peritoneal lavage with normal saline was used

This number was chosen keeping in mind the time restrictions of the study, the feasibility and ease of calculations.

The preoperative preparation of each case consists of correction of shock, electrolyte imbalance, dehydration, gastric aspiration, parenteral broad spectrum antibiotic coverage and tetanus prophylaxis.

Operative details such as date of surgery, hospital number, the site of perforation and degree of peritoneal contamination was noted. At laparotomy a definitive procedure for underlying pathology followed by peritoneal lavage with

either normal saline or normal saline with povidone iodine was done.

2 liters of warm saline and 20 ml of betadine in 2 liters of warm saline is used in the groups to give peritoneal lavage [1%wt/volume]. Abdomen was closed in layers after keeping flank drain.

Samples of peritoneal fluid collected before and after the procedure were labeled and sent immediately for isolation of organism and bacterial colony count.

Semi quantitative bacterial count of the peritoneal fluid collected before wash and the peritoneal fluid collected after wash was performed by plating on blood agar.

Post operative progress was assessed by comparing the development of surgical site infection (SSI), duration of hospital stay, pre wash and post wash bacterial colony count in both the groups.

The treatment to be adopted in each case was decided by the attending surgeon .Post operative fluid and electrolyte balance was maintained by input and output charts and adequacy of replacement was judged mainly on the basis of clinical features.

In most of the cases empirical broad spectrum antibiotics were started pre-operatively. The drainage tubes were removed between 3rd to 5th post operative day and gastric aspiration was discontinued as soon as the patient passed flatus and appearances of normal bowel sounds.

STATISTICAL ANALYSIS

Sample size was estimated based on the difference in proportion of uneventful recovery in group A and group B.

1. Proportion of incidence of complication in NS= 30%(average-overall)=P1

2. Proportion of incidence of complication in PVO=16.25%=P2

3. P (bar)=average of P1 and P2=23%, Q(bar)=77%

4. To detect a difference of 60%reduction in PVP group(18%) compared to NS

5. Group with 80% power(Z beta= 0.842) and at 95% confidence level (Z alpha=1.96)

6. Sample size per group=86

7. 86 in PVP group+86 in normal saline group

FORMULA:

$n = 2*pq\ (Z_a + Z_{(1-b)})^2\ /(P_1 - P_2)^2$

INCLUSION CRITERIA

All patients with peritonitis of age >20yrs and <75years

EXCLUSION CRITERIA:

1. Peritonitis secondary to trauma to abdomen.

2. Peritonitis secondary to gynaecological interventions.

3. Peritonitis secondary to malignancy and immuno-compromised state

4. Patients with thyroid disorders.

Statistical analysis:

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test** was used as test of significance for qualitative data. Continuous data was represented as mean and standard deviation.

Independent t test was used as test of significance to identify the mean difference between two quantitative variables.

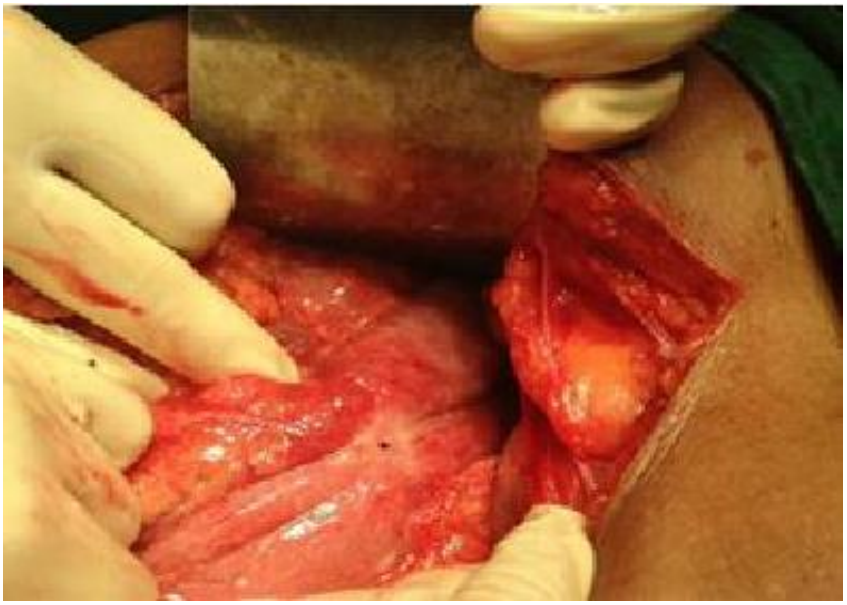
Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

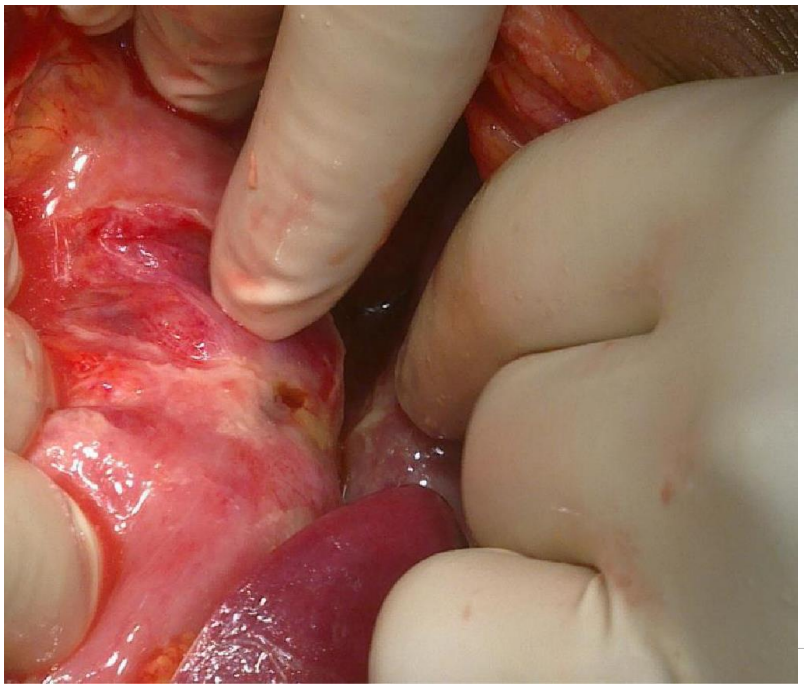
Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

PHOTO GALLERY

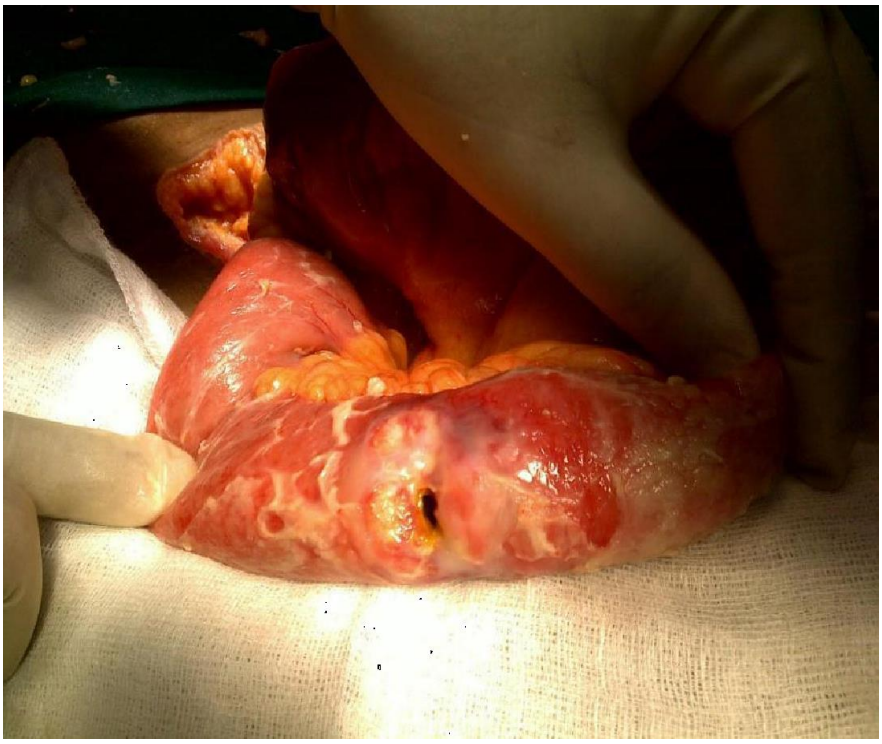
PHOTO GALLERY



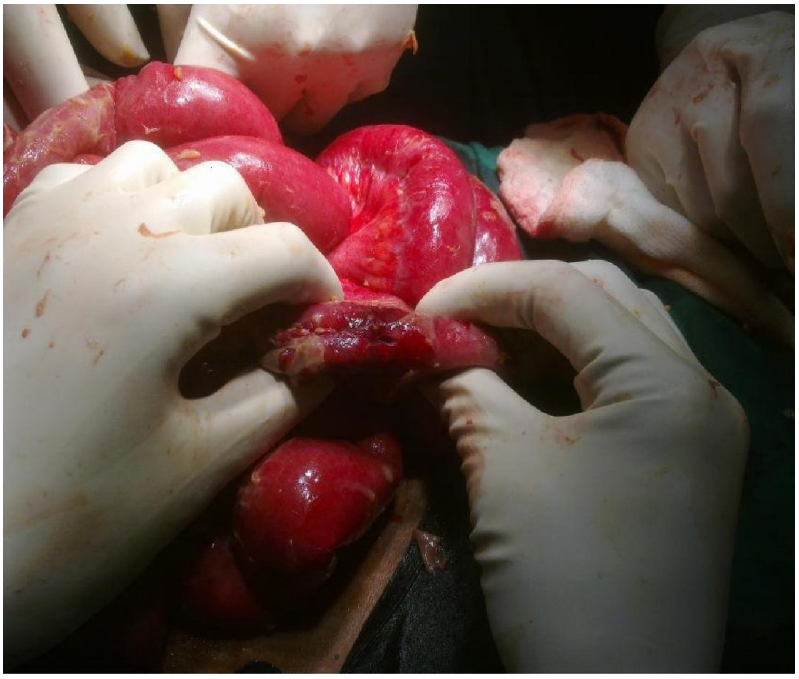
Pre pyloric Perforation



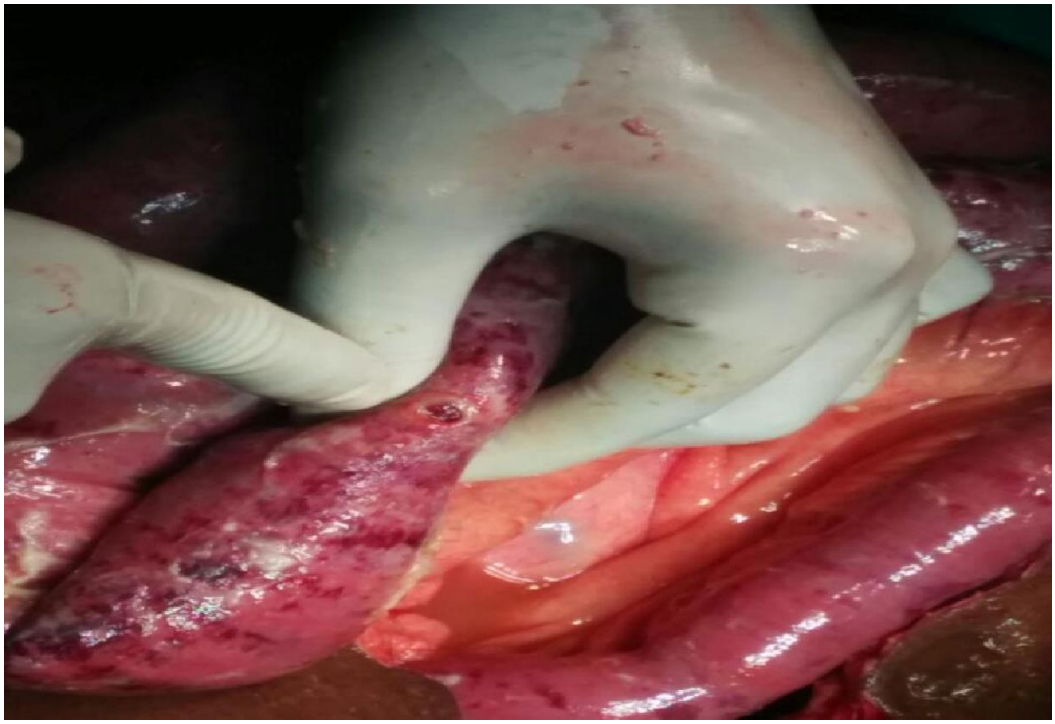
Duodenal perforation



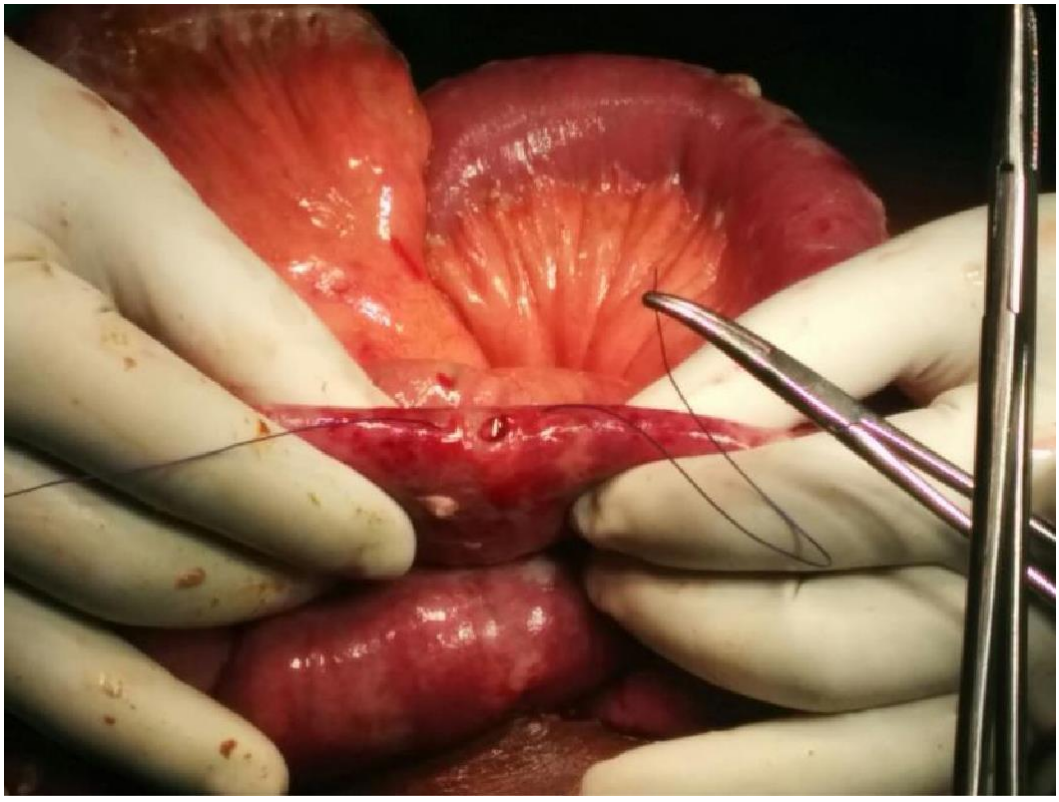
Ileal perforation



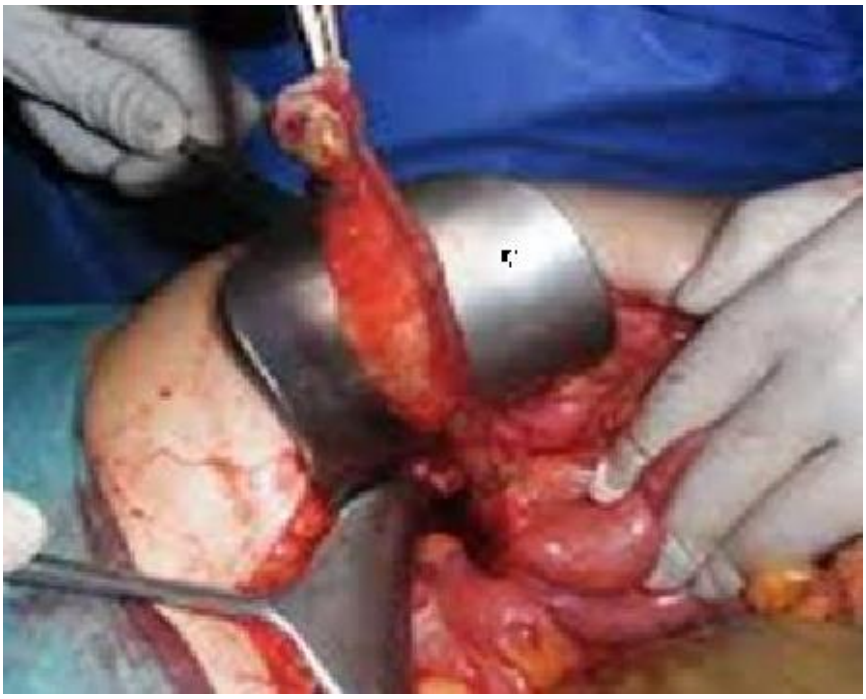
Ileal perforation



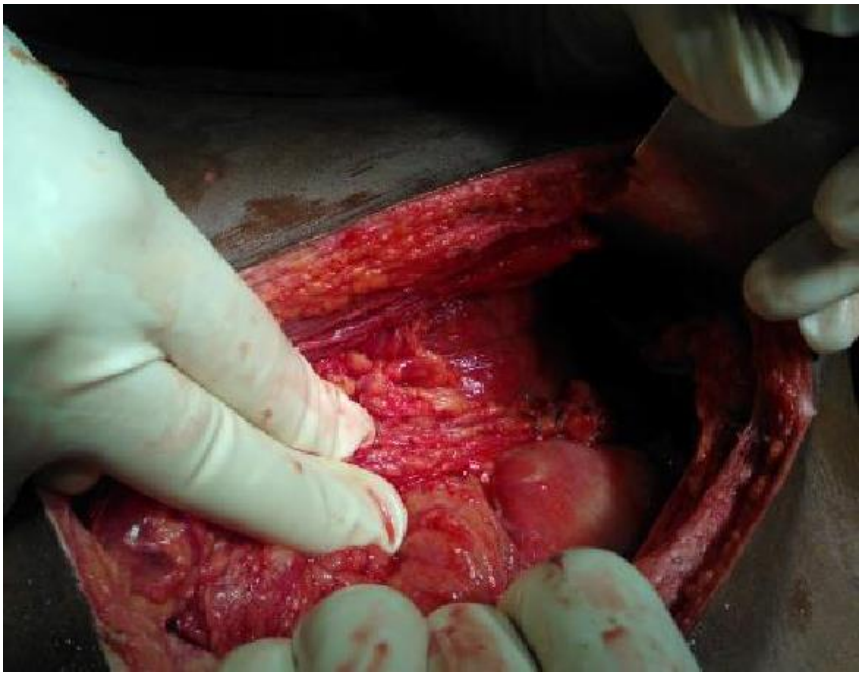
Jejunal perforation



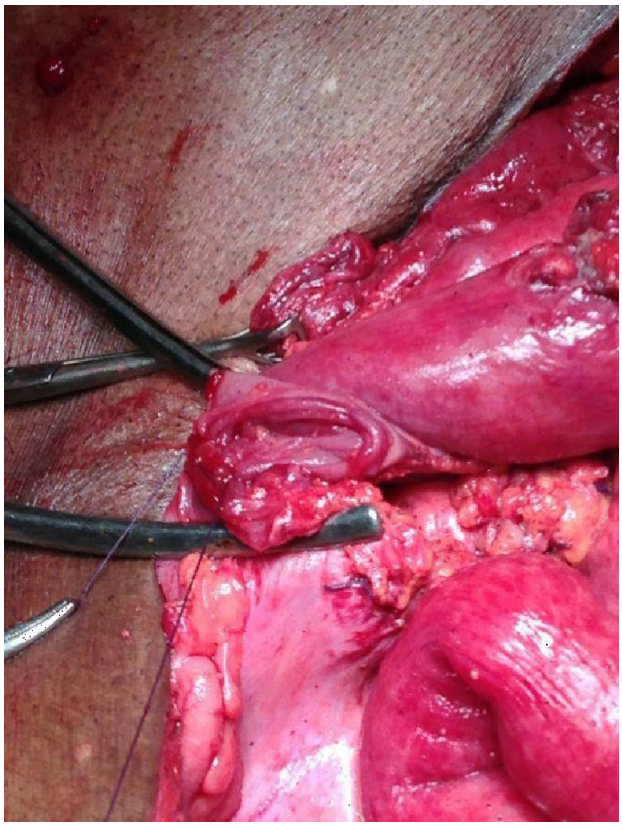
Primary closure of jejunal perforation



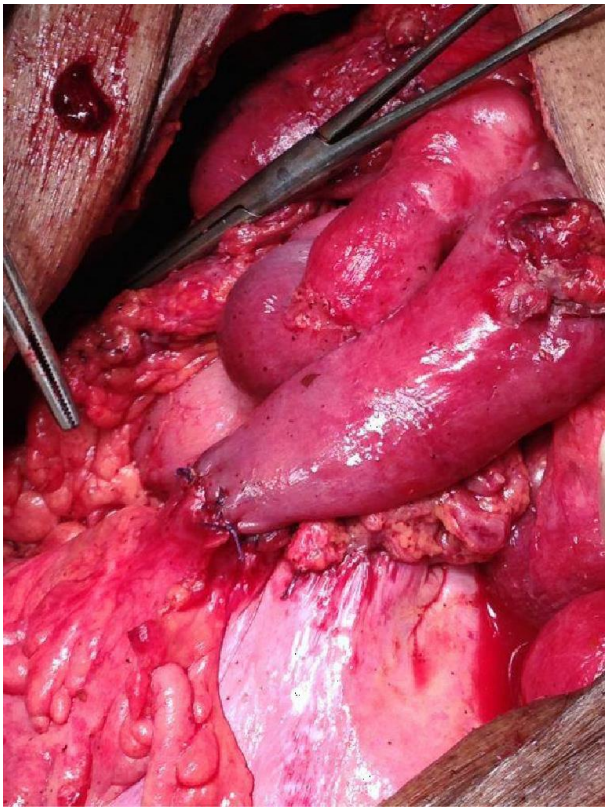
Perforated Appendix



Grahams Omentoplasty repair for duodenal perforation

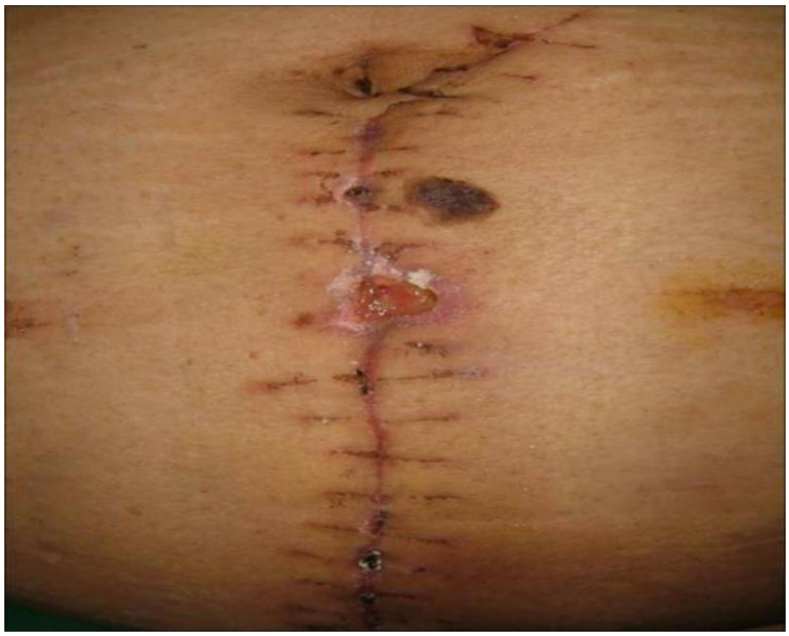


Resection and anastomosis for terminal ileal perforation

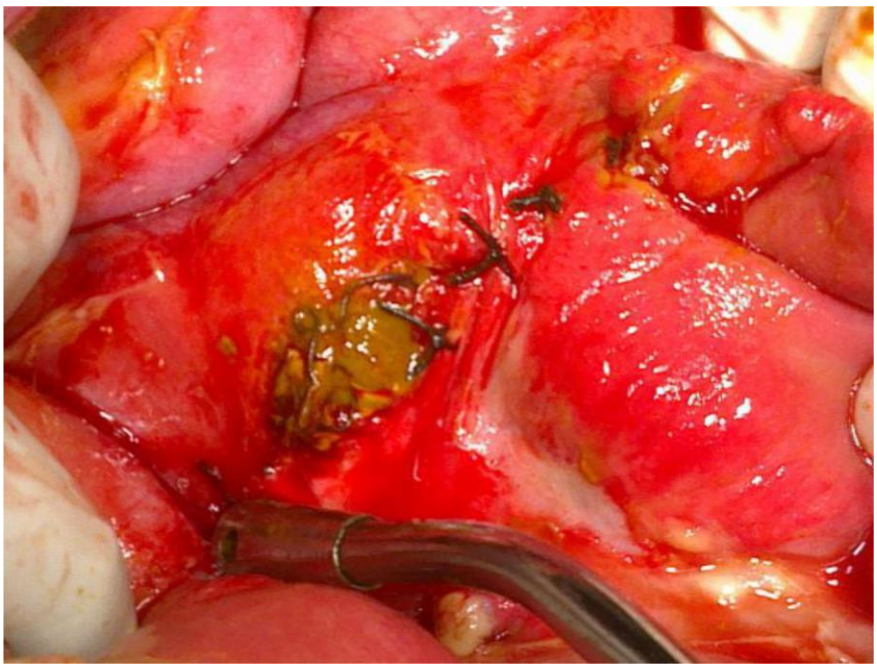


POST OPERATIVE COMPLICATIONS

WOUND INFECTION



Anastomotic leak



RESULTS

OBSERVATION AND RESULTS

Table 3: Age distribution comparison between two groups

		Group			
		Povidone Iodine with Normal Saline		Normal Saline	
		Count	%	Count	%
Age	21 to 30 years	9	10.3%	7	8.0%
	31 to 40 years	4	4.6%	4	4.6%
	41 to 50 years	17	19.5%	11	12.6%
	51 to 60 years	53	60.9%	62	71.3%
	61 to 70 years	4	4.6%	3	3.4%

$\chi 2 = 2.383$, df =4, p =0.666

The most vulnerable age group in this study was between 51 to 60 years Most of the patients, 115/174 cases (66%) ,were in this age group followed by 41-50 years of age which accounted for 28 /174 cases (19%). Patients between age group 41-60 years accounted for 143/174 (82%) . The youngest patient was a 25 years old female with appendicular perforation and the oldest being 69 year old male with duodenal perforation . The mean age at the time of presentation was 54.5 years.

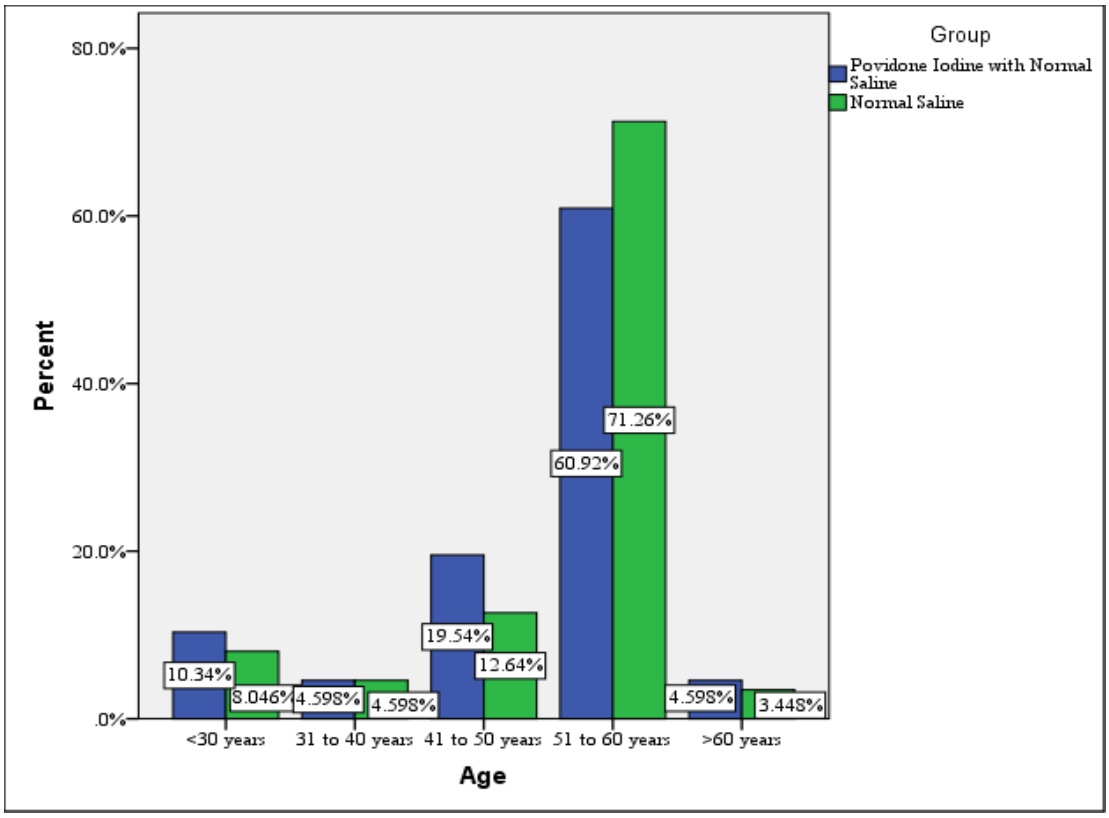


Figure 1: Bar diagram showing Age distribution comparison between two groups

Table 4: Gender distribution comparison between two groups

		Group			
		Povidone Iodine with Normal Saline		Normal Saline	
		Count	%	Count	%
Gender	Female	25	28.7%	29	33.3%
	Male	62	71.3%	58	66.7%

$\chi 2 = 0.430$, df =1, p =0.512

The number of males in the study was 120 which accounted for 69%of the cases and the number of females was 54 who formed 31% of the cases

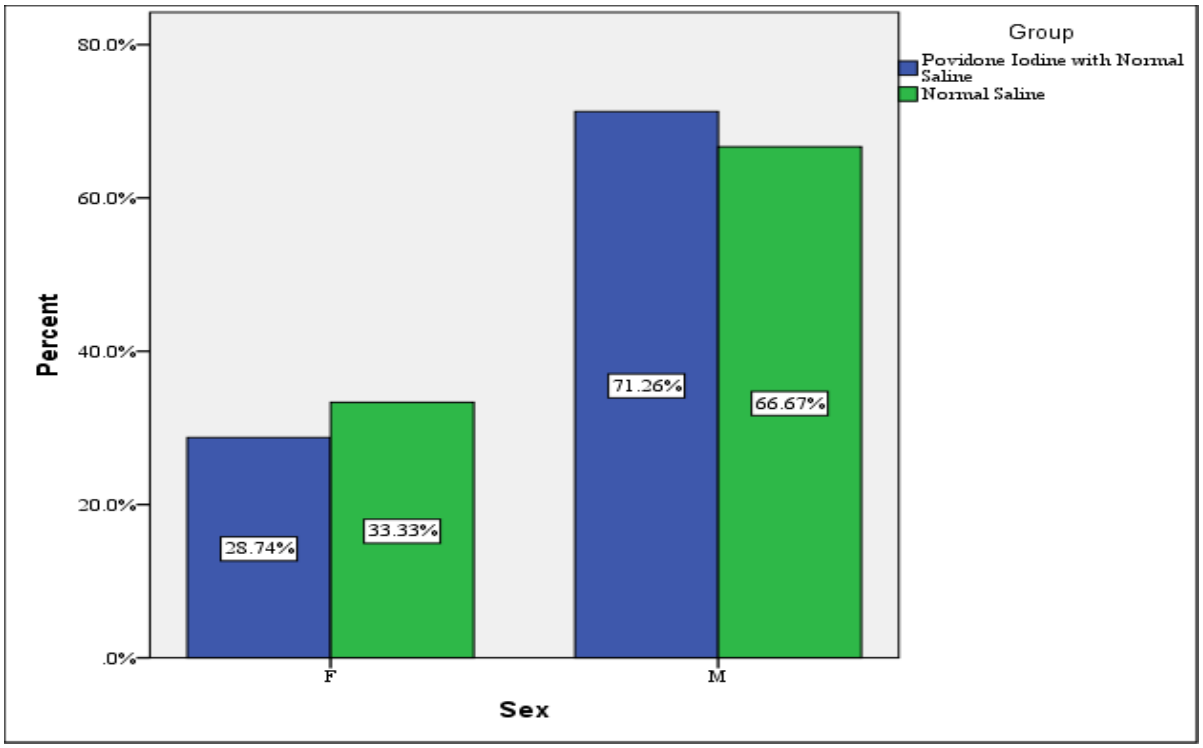


Figure 2: Bar diagram showing Gender distribution comparison between two groups

Table 5: Type of perforation comparison between two groups

		Group			
		Povidone Iodine with Normal Saline		Normal Saline	
		Count	%	Count	%
	Prepyloric Perforation	25	28.7%	27	31.0%
	Duodenal Perforation	24	27.6%	21	24.1%
	Appendicular Perforation	19	21.8%	15	17.2%
	Ileal Perforation	9	10.3%	18	20.7%
	Jejunal Perforation	10	11.5%	5	5.7%
	Colonic Perforation	0	0.0%	1	1.1%

$\chi 2 = 6.414$, df =5, p =0.268

The commonest cause for peritonitis in the study was perforation. Among the various sites of gastrointestinal perforation, prepyloric perforation was the commonest accounting for 52 /174 (29.8%).This was followed by duodenal perforation patients45/174(25%), appendicular perforation 34/174(19%), ileal perforation 27/174 (15%) and jejunal perforation 15/174 (8%) .There was only one case of colonic perforation in the present study

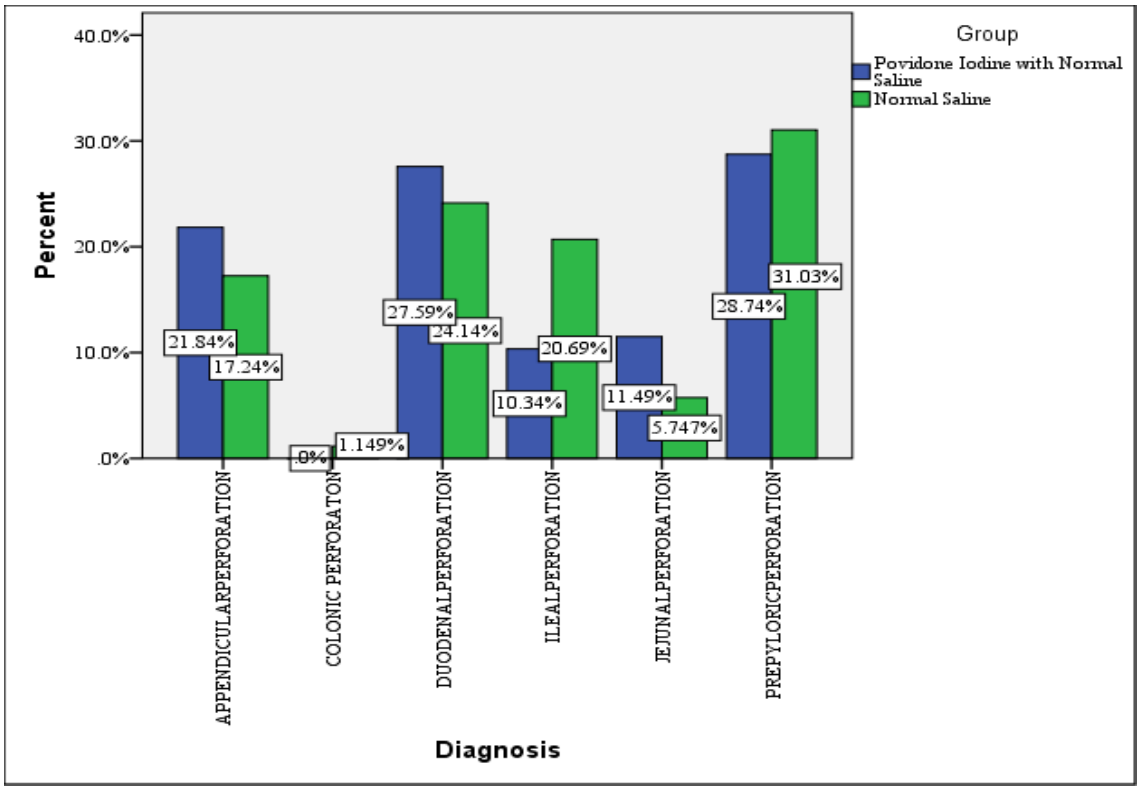


Figure 3: Bar diagram showing Type of perforation comparison between two groups

Table 6: Comparison of number of days of hospital stay between two groups

		Hospital Stay (Days)		P value
		Mean	SD	
Group	Povidone Iodine with Normal Saline	11.05	2.59	0.930
	Normal Saline	11.01	2.54	

The duration of hospital stay ranged from 5 to 29 days and mean duration of hospital stay was 11 days . The minimal duration of stay of a patient was 5 days in case of appendicular perforation and the maximum number of days patient stayed in the hospital was 29 days for a case of ileal perforation .In patients in group A the duration of stay in the hospital ranged from 6 to 25 days with mean duration of 11 days and in group B the duration of stay in the hospital ranged from 5to 29 days with mean duration of 11 days.

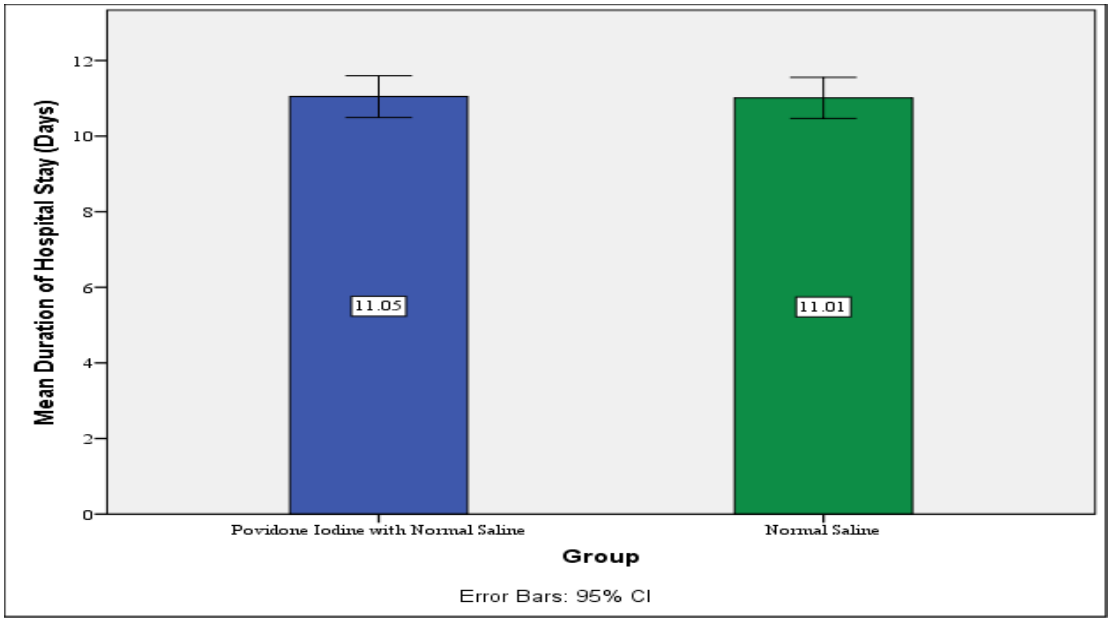


Figure 4: Bar diagram showing Comparison of number of days of hospital stay between two groups

Table 7: Growth comparison between two groups at Pre and Post BCC

		Group				P value
		Povidone Iodine with Normal Saline		Normal Saline		
		Count	%	Count	%	
Pre BCC	No Growth	58	66.7%	63	72.4%	0.410
	Growth Present	29	33.3%	24	27.6%	
Post BCC	No Growth	58	66.7%	63	72.4%	0.410
	Growth Present	29	33.3%	24	27.6%	

The peritoneal fluid was sent for culture sensitivity in all the patients and 53 patients showed positive culture and in 121 patients there was no growth no growth.

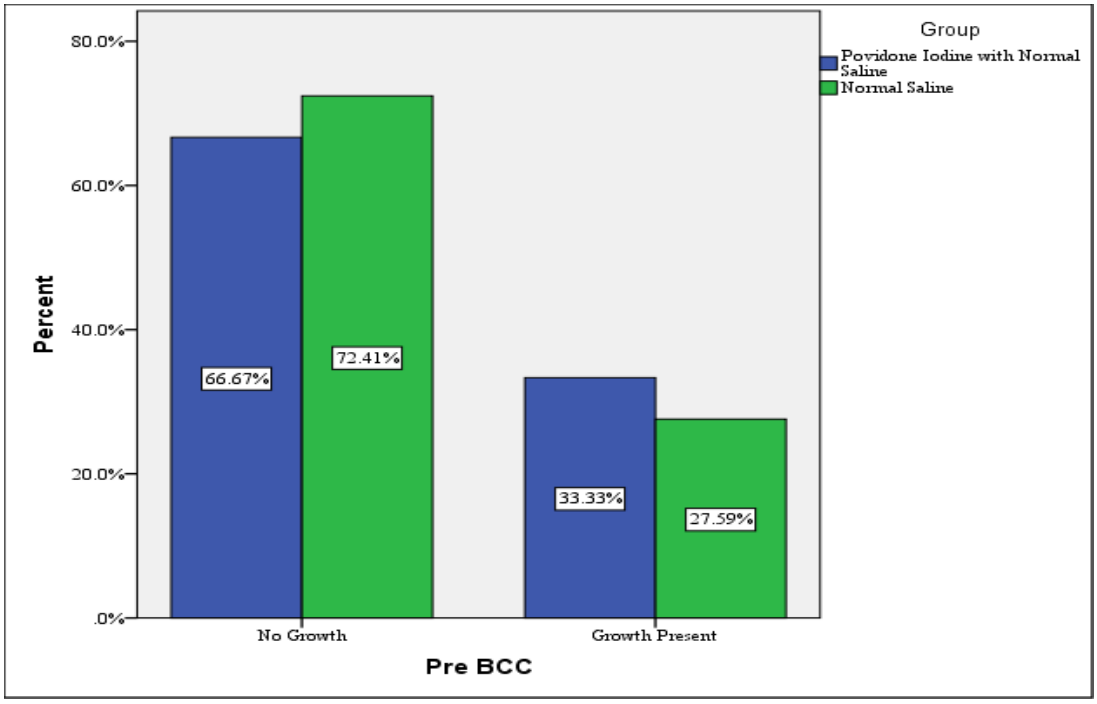


Figure 5: Bar diagram showing Growth comparison between two groups at Pre BCC

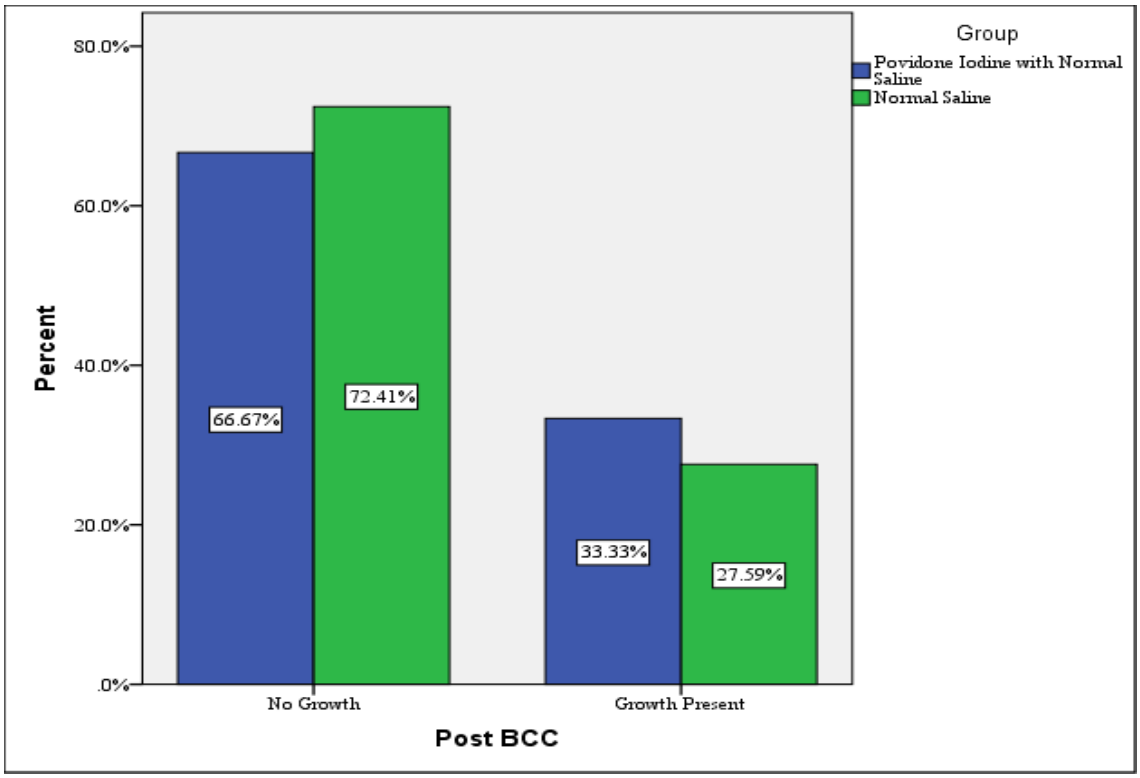


Figure 6: Bar diagram showing Growth comparison between two groups at Post BCC

Table 8: Mean Cell count comparison between two groups

	Group						P value b/w two groups
	Povidone Iodine with Normal Saline			Normal Saline			
	Mean	SD	P value with in group	Mean	SD	P value with in group	
Pre BCC	1748.28	124.27		1700.00	131.88		0.177
Post BCC	1462.07	169.90	<0.001*	1554.17	147.38	<0.001*	0.042*

. In Povidone Iodine with Normal Saline group, mean cell count Pre BCC was 1748.28 ± 124.27 and Post BCC was 1462.07 ± 169.90. There was A significant decrease in cell count Post BCC compared to Pre BCC in Povidone Iodine with Normal Saline group.

In the Normal Saline group, mean cell count Pre BCC was 1700.00 ± 131.88 and Post BCC was 1554.17 ± 147.38. There was significant decrease in cell count Post BCC compared to Pre BCC in Normal Saline group.

In both the groups there was a significant decrease in cell count post BCC .However cell count was significantly lower in Povidone Iodine with Normal Saline group than compared to Normal saline group.

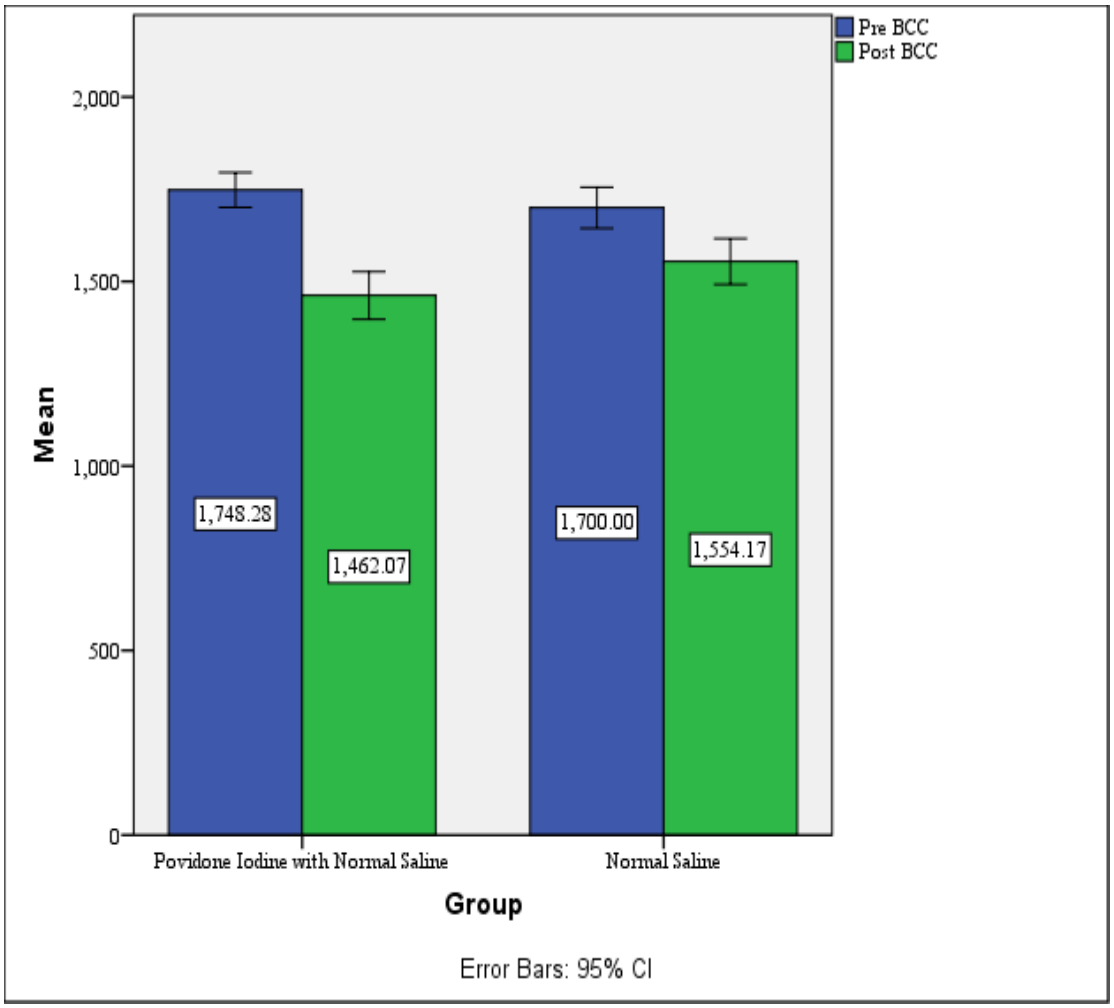


Figure7: Bar diagram showing Mean Cell count comparison between two groups

Table 9: Organism isolated comparison between two groups

		Group			
		Povidone Iodine with Normal Saline		Normal Saline	
		Count	%	Count	%
Organism Isolated	E.Coli	19	65.5%	12	50.0%
	Enterococci	10	34.5%	12	50.0%

$\chi 2 = 1.302$, df =1, p =0.254

The most common organism identified in the study was E.Coli followed by Enterococcus. In this study 53(30%) patients had positive culture, out of which 31(58%) patients were positive for E.coli .In group A, E.coli was isolated in 65% and Enterococcus 34.5% where as in group B the distribution was E.coli (50%) and Enterococcus 50%

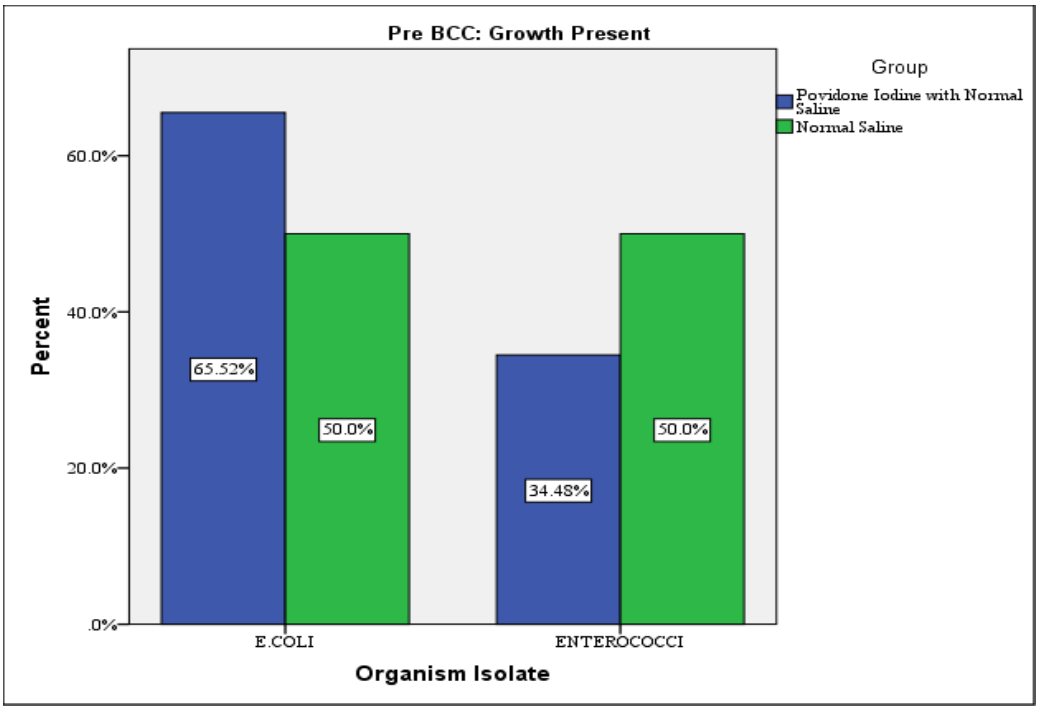


Figure 8: Bar diagram showing comparison of organism isolated between two groups

Table 10: Outcome at Follow up comparison between two groups

		Group			
		Povidone Iodine with Normal Saline		Normal Saline	
		Count	%	Count	%
Follow up	Death	0	0.0%	1	1.1%
	Recovered	87	100.0%	86	98.9%

$\chi 2 = 1.006$, df =1, p =0.316

There was one case of mortality in the present study. All the patients with peritoneal lavage with povidone iodine in normal saline group recovered from the illness and in the normal saline group one patient succumbed to death

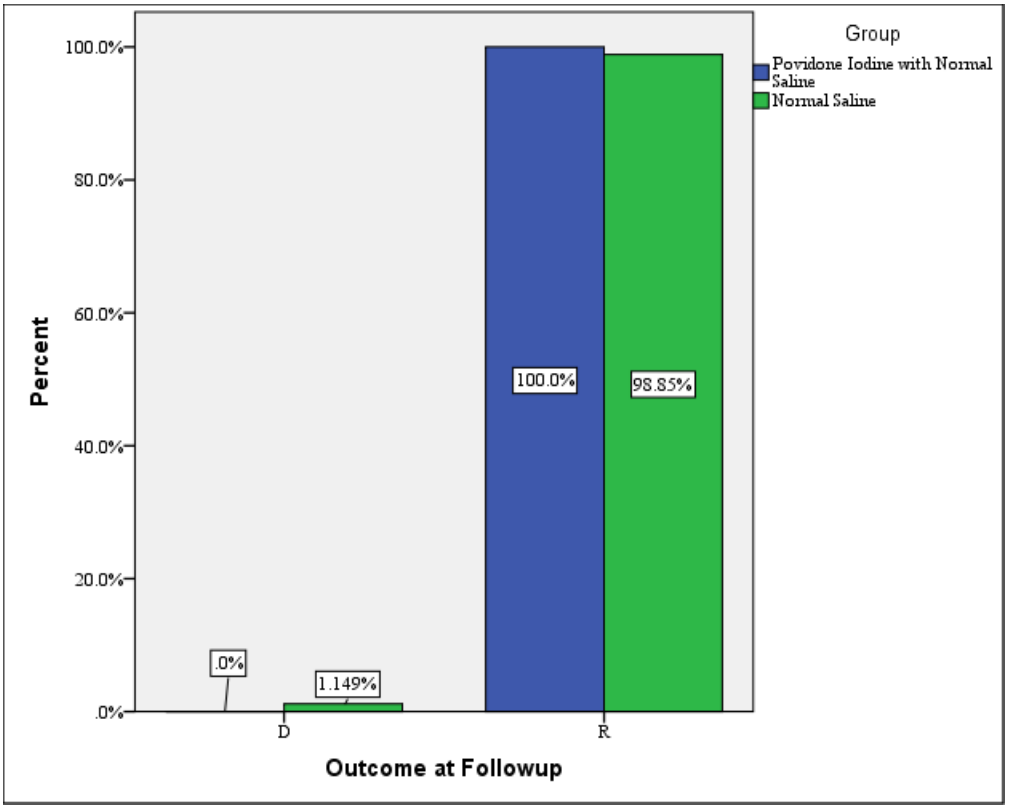


Figure 9: Bar diagram showing Outcome at Followup comparison between two groups

Table 11: comparison of characteristics between two groups in prepyloric perforation

		Group				P value
		Povidone Iodine with Normal Saline		Normal Saline		
		Count	%	Count	%	
Organism Isolate	Nil	17	68.0%	22	81.5%	0.503
	E.Coli	4	16.0%	3	11.1%	
	Enterococci	4	16.0%	2	7.4%	
Wound Infection	Absent	17	68.0%	21	77.8%	0.427
	Present	8	32.0%	6	22.2%	
Outcome at Followup	Recovered	25	100.0%	27	100.0%	-

Fifty two patients had prepyloric perforation and among them in thirteen patients culture was positive. E.coli was the common organism (53%) followed by enterococcus (46%). There was significant decrease in post BCC in both the groups Surgical site infection was noted in 8 (32%)patients in group A and 6 (27%) patients in group B. Three patients developed respiratory infection and two patients had post operative ileus.

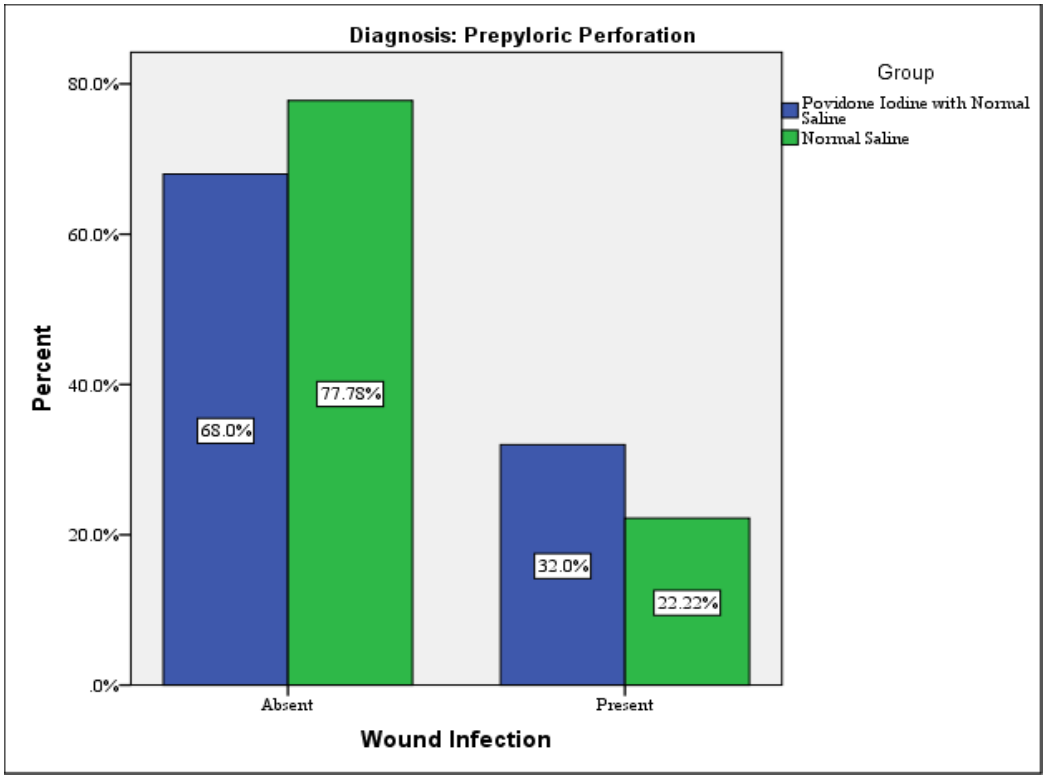


Figure10: Bar diagram showing Wound infection comparison between two groups in Prepyloric perforation

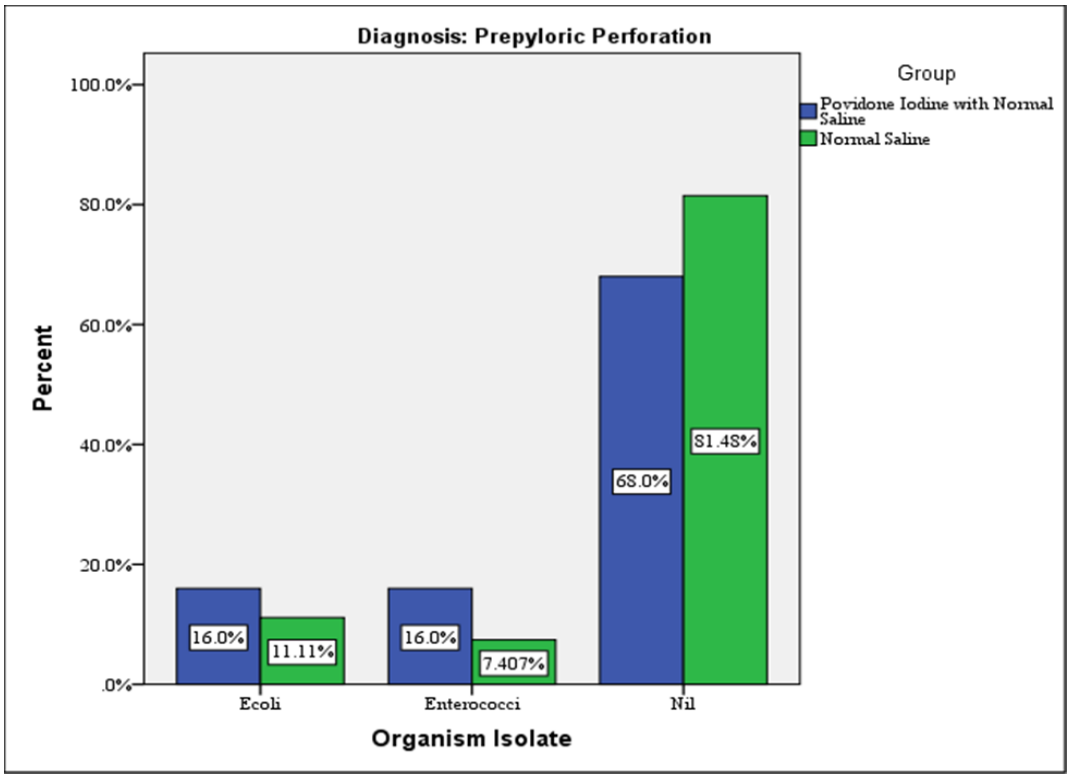


Figure11: Bar diagram showing Organism isolated comparison between two groups in Prepyloric perforation

Table 12: Comparison of characteristics between two groups in duodenal perforation

		Group				P value
		Povidone Iodine with Normal Saline		Normal Saline		
		Count	%	Count	%	
Organism Isolate	Nil	17	70.8%	16	76.2%	0.06
	E.Coli	7	29.2%	2	9.5%	
	Enterococci	0	0.0%	3	14.3%	
Wound Infection	Absent	12	50.0%	12	57.1%	0.632
	Present	12	50.0%	9	42.9%	
Outcome at Followup	Recovered	24	100.0%	21	100.0%	-

Among 174 patients, fourty five patients had duodeneal perforation . Twelve patients(26.6%) were culture positive. E.coli was the commonest isolate in 9patients (75%) followed by enterococcus in 3patinets (25%). There was significant decrease in post BCC in both the groups Surgical site infection was encountered in 12 patients (57%) in group A and 9 patients (42%) in group B. Four patients developed respiratory infection, two patients had post operative ileus ,1patient had burst abdomen and one patient developed fecal fistula.

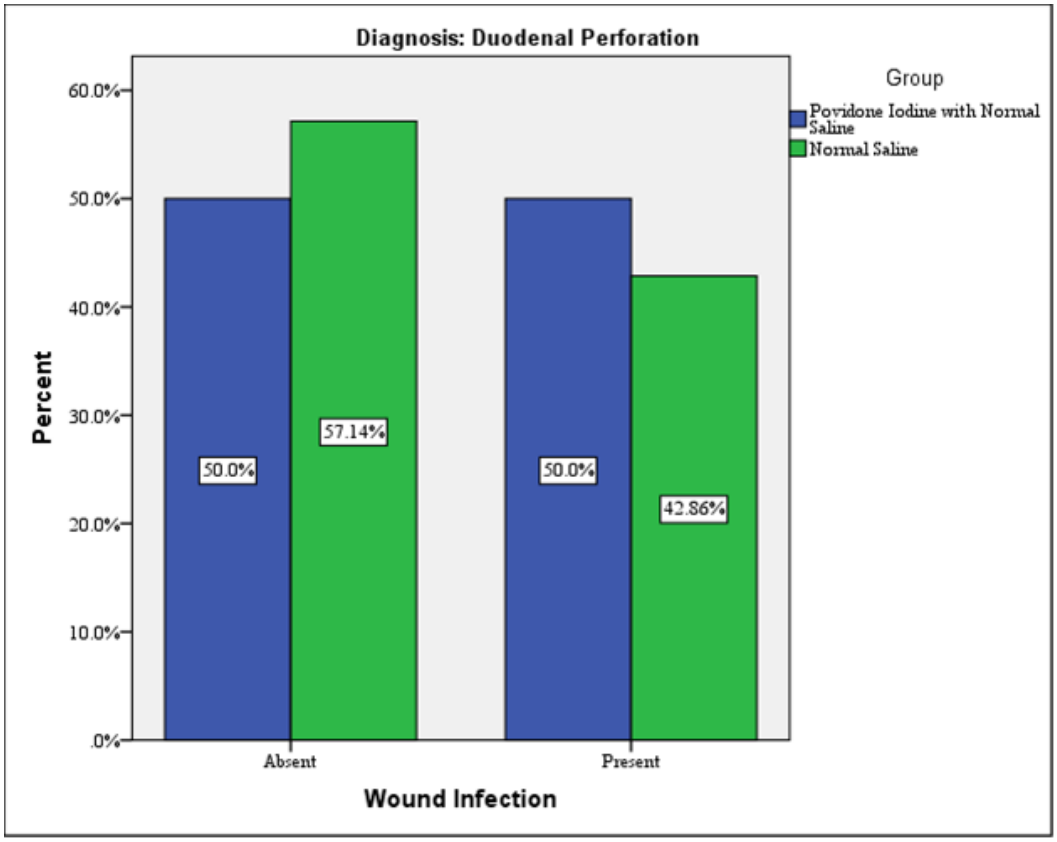


Figure 12: Bar diagram showing Wound infection comparison between two groups in Duodenal perforation

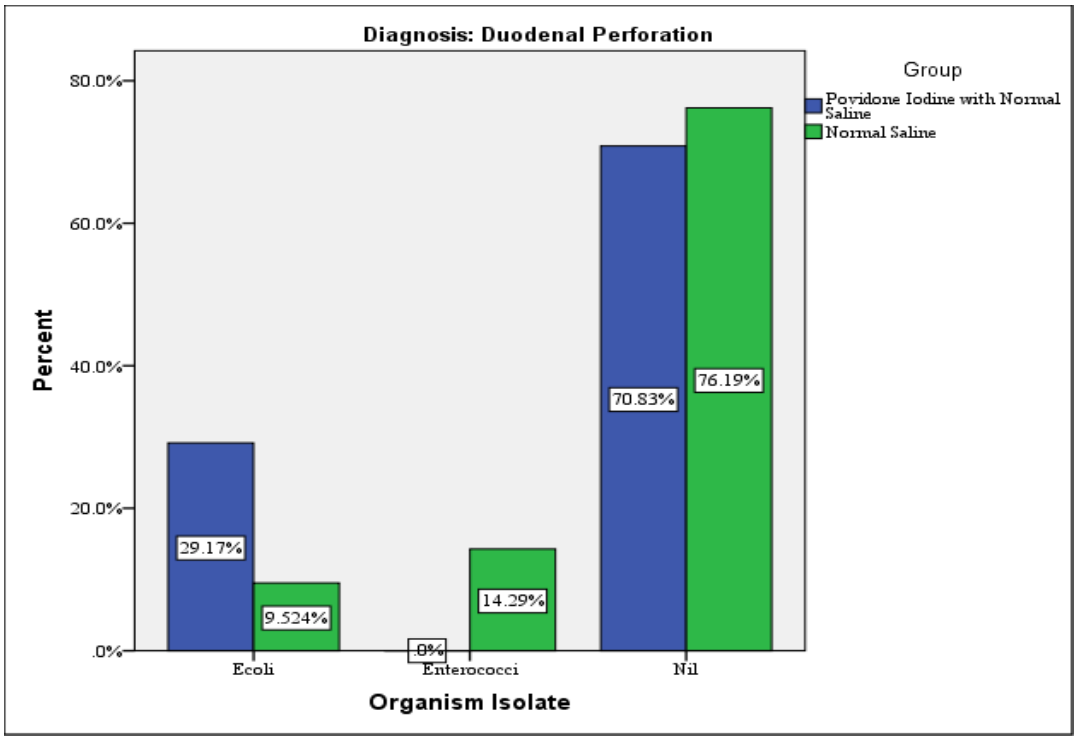


Figure 13: Bar diagram showing Organism isolated comparison between two groups in Duodenal perforation

Table13: Comparison of characteristics between two groups in appendicular perforation

CHARACTERISTICS		Group				P value
		Povidone Iodine with Normal Saline		Normal Saline		
		Count	%	Count	%	
Organism Isolate	Nil	12	63.2%	11	73.3%	0.800
	E.Coli	4	21.1%	2	13.3%	
	Enterococci	3	15.8%	2	13.3%	
Wound Infection	Absent	10	52.6%	8	53.3%	0.968
	Present	9	47.4%	7	46.7%	
Outcome at Followup	Recovered	19	100.0%	15	100.0%	-

Thirty four patients had appendicular perforation and the culture was positive in 11 patients(32%). The most common organism isolated was E.coli(54%) followed by enterococci(45%)There was significant decrease in post BCC in both the groups.In group A Surgical site infection was encountered in 9 patients (26.4%) and in group B 7 patients (20.5%).

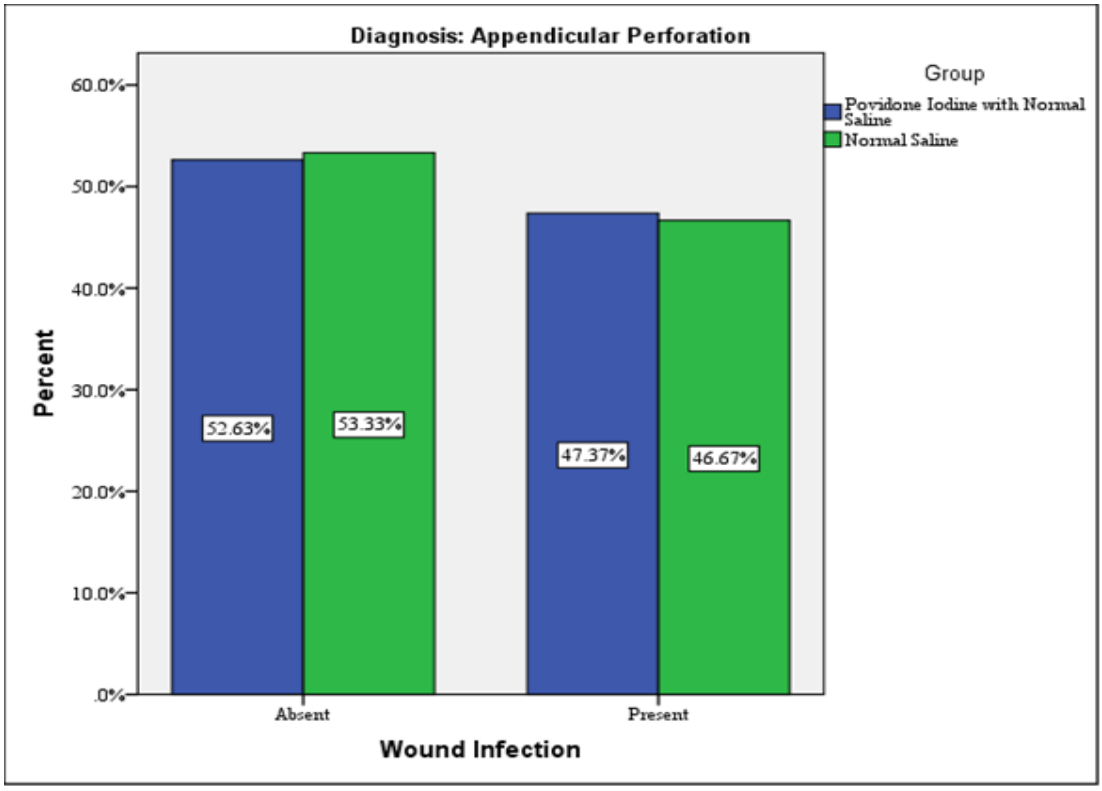


Figure14: Bar diagram showing Wound infection comparison between two groups in appendicular perforation

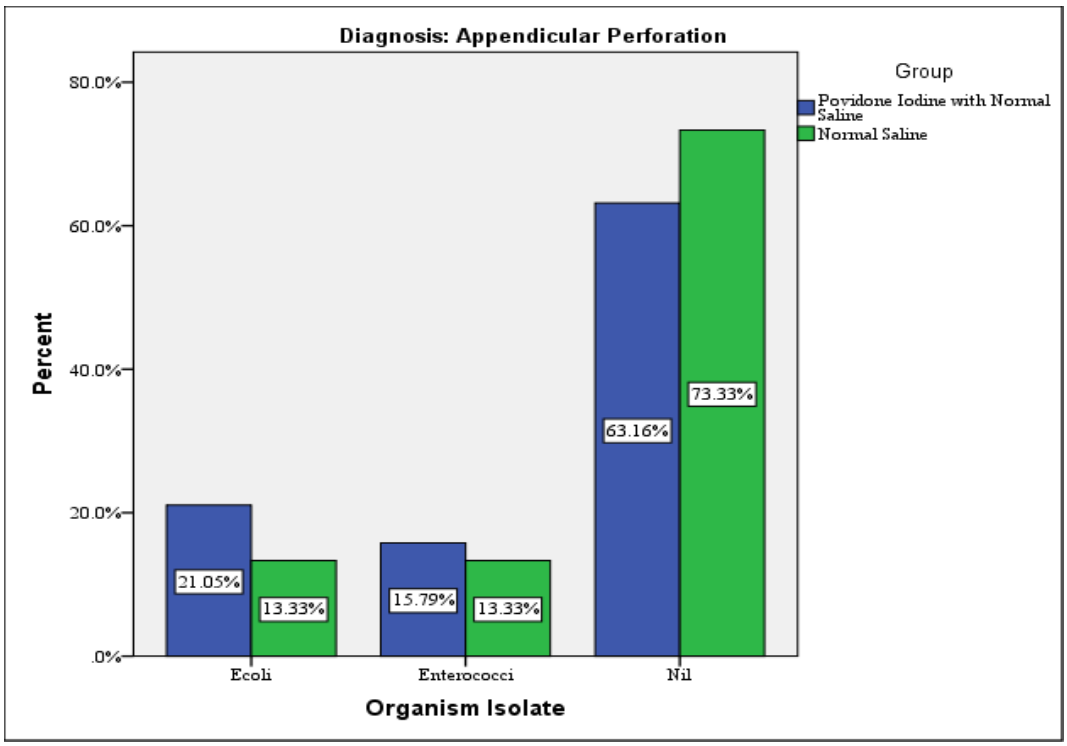


Figure15: Bar diagram showing Organism isolated comparison between two groups in appendicular perforation

Table 15: comparison of characteristics between two groups in Ileal perforation

		Group				P value
		Povidone Iodine with Normal Saline		Normal Saline		
		Count	%	Count	%	
Organism Isolate	Nil	6	66.7%	11	61.1%	0.404
	E.Coli	3	33.3%	4	22.2%	
	Enterococci	0	0.0%	3	16.7%	
Wound Infection	Absent	7	77.8%	12	66.7%	0.551
	Present	2	22.2%	6	33.3%	
Outcome at Follow up	Death	0	0.0%	1	5.6%	0.471
	Recovered	9	100.0%	17	94.4%	

Twenty seven patients had ileal perforation among which 10 patients (37%) was culture positive. The most common isolate was E.coli (70%) followed by enterococci(30%). There was significant decrease in post BCC in both the groups In group A 2 patients (22%) and in group B 6 patients (33%) had surgical site infection. One patient succumbed to death. An elderly male presented to ER in decompensated sepsis due to peritonitis, requiring ventilator support and intensive care. Patient could not tolerate further support and succumbed to death, due to renal failure and septicaemia. Two patients developed respiratory infection, one patient had post operative ileus and three patients developed faecal fistula.

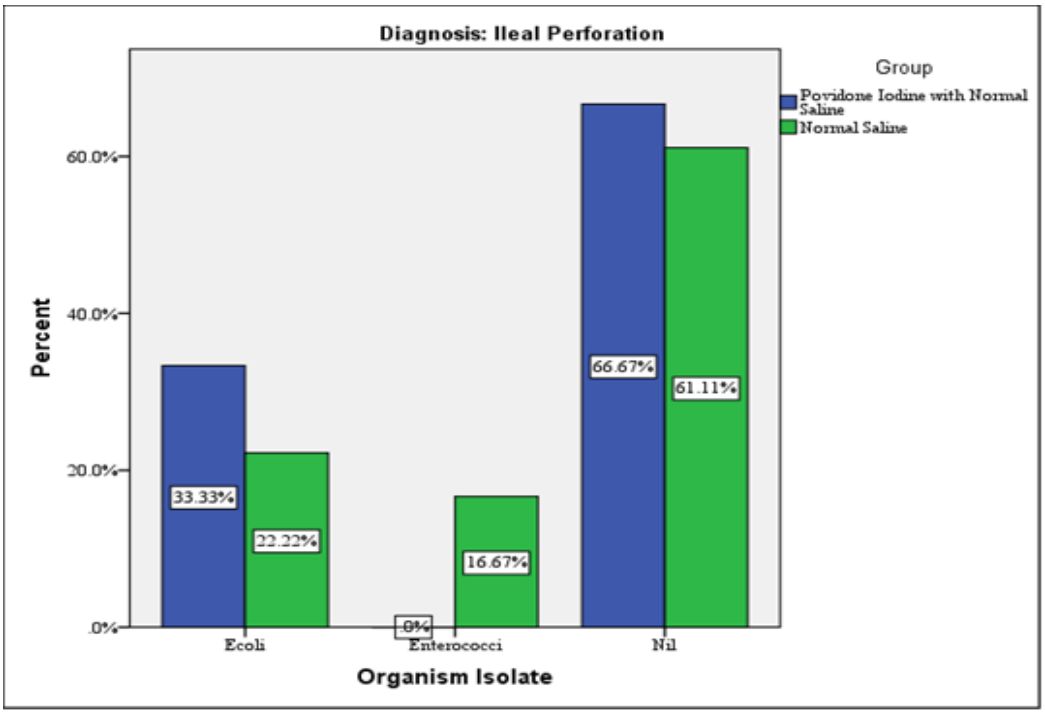


Figure16: Bar diagram showing Wound infection comparison between two groups in Ileal perforation

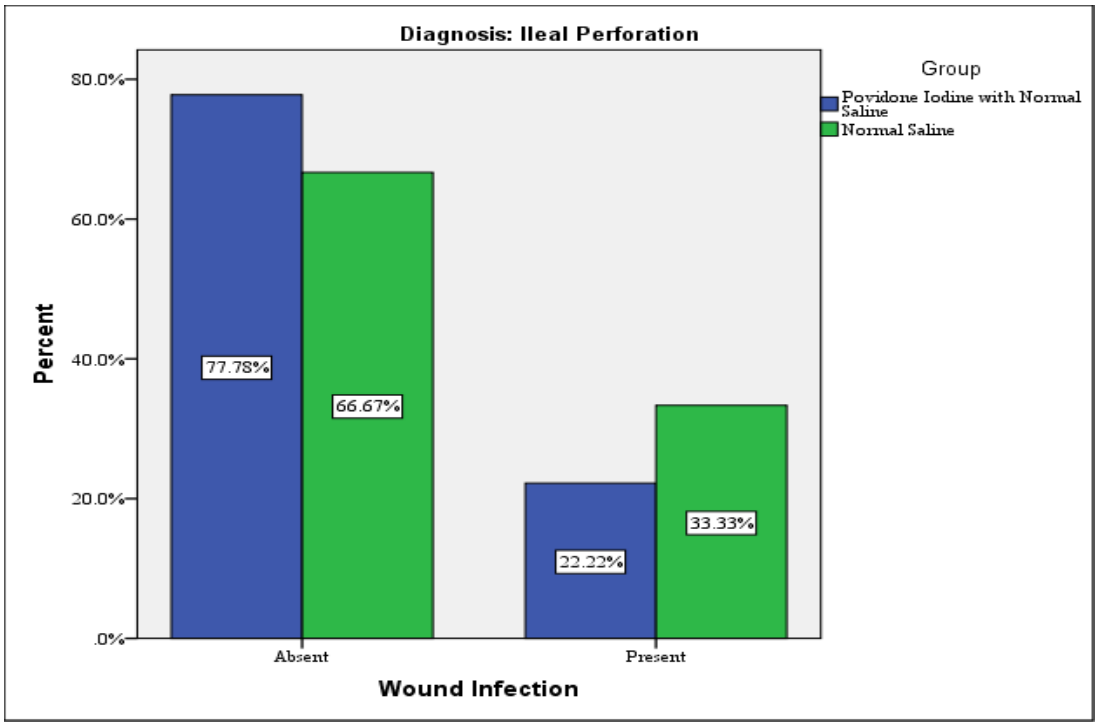


Figure17: Bar diagram showing Organism isolated comparison between two groups in Ileal perforation

Table 15: comparison of characteristics between two groups in Jejunal perforation

		Group				P value
		Povidone Iodine with Normal Saline		Normal Saline		
		Count	%	Count	%	
Organism Isolate	Nil	6	60.0%	3	60.0%	0.829
	E.Coli	1	10.0%	1	20.0%	
	Enterococci	3	30.0%	1	20.0%	
Wound Infection	Absent	6	60.0%	5	100.0%	0.099
	Present	4	40.0%	0	0.0%	
Outcome at Follow up	Recovered	10	100.0%	5	100.0%	-

Fifteen patients had jejunal perforation, culture was positive in 6 patients (40%)..Enterococci was the most common organism isolated followed by E.coli . There was significant decrease in post BCC in both the groups .Surgical site infection was noted in 4 patients in group A (40%)and none of the patients in group B. One patients developed respiratory infection, two patients had post operative ileus ,two patient developed faecal fistula.

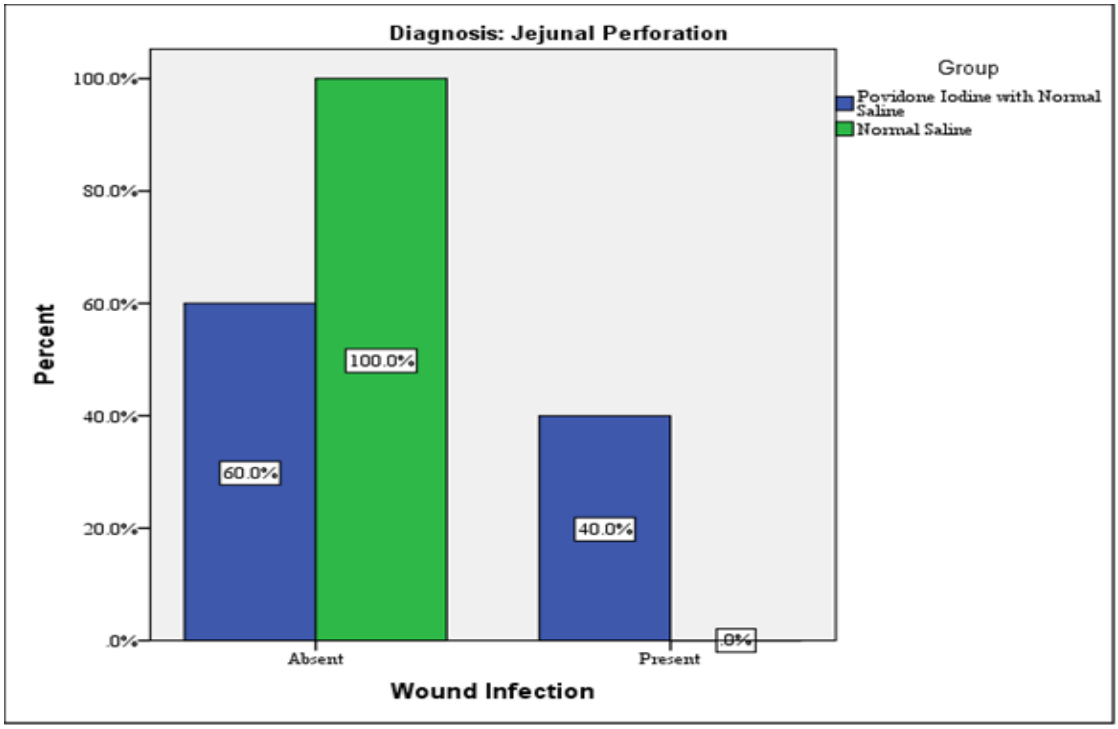


Figure 18: Bar diagram showing Wound infection comparison between two groups in Jejunal perforation

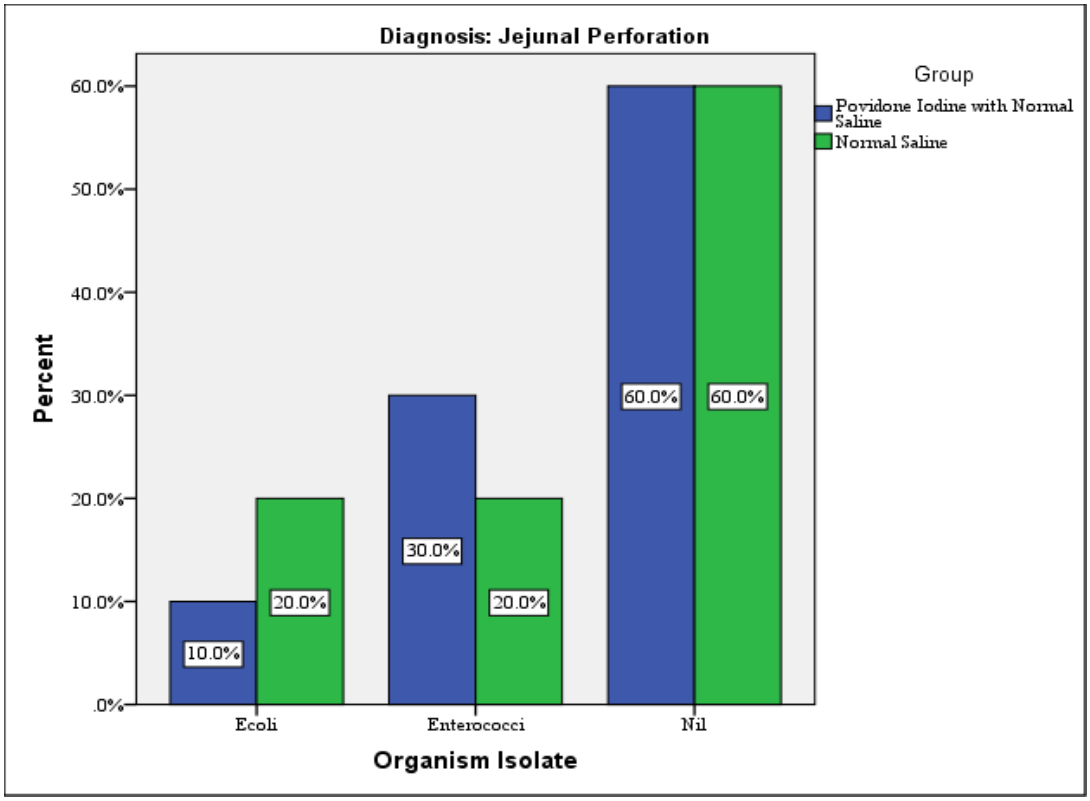


Figure 19: Bar diagram showing Organism isolated comparison between two groups in Jejunal perforation

DISCUSSION

DISCUSSION

A total of 174 patients who presented with features of peritonitis secondary to hollow viscus perforation to R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar, from December 2016 to June 2018 were randomized into two groups and studied.

In Group A, patients with all odd serial numbers were included and received peritoneal lavage with povidone iodine in normal saline and in Group B patients with all even serial numbers were taken and received peritoneal lavage with normal saline.

This clinical study was intended to determine the postoperative progress by comparing the development of surgical site infection, duration of hospital stay, pre wash and post wash bacterial colony count in both the groups

1. AGE :

The majority of the patients were in the age group 51 to 60 years 115 (66%). The next common age group was between 41-50 years accounting for 28 (19%) patients. Together they accounted for 143(82%) patients. The youngest patient was a 25 year old female with appendicular perforation and the oldest patient was a 69 year old male with duodenal perforation. The mean age group in our study was of 54.5 years and pre-pyloric and duodenal perforations were commonly seen in this age group. Among the age groups encountered in our study, perforations at the pre-pyloric region was very commonly seen, followed by duodenal, ileal and colonic in order of decreased frequency. This predilection of peritonitis to commonly affect elderly age groups is due to the chronic ill habits like smoking, alcohol consumption, chronic infection with H Pylori, and faulty dietary habits. A study by Ohmann[58] et al also

showed the vulnerable age group to be 50-69 years and these findings are consistent to ours.

TABLE 16 : COMPARISON OF PREDOMINANT AGE GROUP IN PERITONITIS.

Study	Predominant age group
Samir Delibegovic et al[56]	21-40 years
Ashis Ahuja et al[57]	21-40 years
C Ohmann et al[57]	50-69years
Our study	51-60 years

GENDER DISTRIBUTION:

The number of males in our study were 120, constituting for 69% of the cases. The number of females were 54 which formed 31% of the cases. The male to female ratio was 2.2 : 1, showing a male preponderance. This could be due to various personal and social factors like habits, diet, socio-economic status, cumulatively making the male gender more prone for peritonitis. Male preponderance was also found in Samir Delibegovicet et al with male to female ratio of 3:1, Ajazahamed Malik et al with 2:1 and in Sharma R, Huttunen et al.

The conditions encountered in this study was perforations at the appendix, pre-pylorus, duodenum, ileum which were commonly seen in males.

TABLE 17: COMPARISON OF MALE TO FEMALE DISTRIBUTION

Studies	M:F ratio
Samir Delibegovicet al[56]	3:1
Ajazahamed Malik et al[59]	2:1
Sharma R, Huttunen et al[60]	2:1
Our study	2.2:1

Gastrointestinal Tract is the most common site of perforations and gastric perforations are still more common (55%) followed by small intestinal perforations (24 %). Pre-pyloric perforation was the commonest accounting for 52 /174 (29.8%).This was followed by 45/174(25%) duodenal perforation patients, 34(19%) appendicular perforation, 27/174 (15%) ileal perforation and 15/174 (8%) jejunal perforation There was only one case of colonic perforation in the present study. Considering the various sites of perforations, in our study, male preponderance was noted in almost all the locations. Majority were the perforations of the stomach in all the age groups followed by bowel perforations. Another well known cause of perforations is malignancy. But this study does not include malignancy induced perforations. These results correlate to various other studies done. .

TABLE 18 : SITE OF PERFORATION IN DIFFERENT STUDY GROUP:

Study	SITE OF PERFORATION		
	Gastroduodenal	Small intestine	Large intestine
1 .AjazAhamed Malik et al[59]	30.6%	5.9%	9.9%
2 .Notash et al	60%	42.5	-
3.RS Jhobta[61]	65.67%	8.27%	3.7%
4.Nithin Agarwal et al[62]	23%	43%	6%
5.Our study	55%	24%	1.1%

Duration of hospital stay

The duration for which patients stayed in hospital varied from 5 to 29 days. Earliest to get discharged was a case of appendicular perforation and patient with ileal perforation stayed for longer duration due to faecal fistula that had developed as a result of post operative complication. .Abdominal contamination was less in pre-pyloric and duodenal perforations compared to ileal, jejunal and colonic perforations. The time duration between patient developing symptoms and seeking intervention also has an influence on the kind of exudate encountered intra-operatively. Patients presenting early were found to have serous exudates with or without bacterial contamination, whereas, patients with delayed presentation were found to have purulent/feculent exudates with increased bacterial counts compared to early presentation.

Mean post operative hospital stay was 11.05 ± 2.59 days in povidone iodine group and 11.01±2.54 days in normal saline group. Patients in whom povidone iodine was used stayed for a comparatively shorter duration in the hospital. Sheeraz Khan et

al (2009) reported reduction in hospital stay by 1.5 days. Vallance et al. (1985) found no improvement in the duration of hospital stay of patients treated with intraperitoneal lavage with chlorhexidine gluconate or povidone iodine when compared with those who received only saline lavage.

Table 19 :Mean duration of hospital stay in different types of perforation

Perforation	**Duration of hospital stay (pValue)**
Prepyloric	0.391
Duodenal	0.739
Appendicular	0.605
Ileal	0.943
Jejunal	0.740

Growth

It was found that irrespective of the kind of lavage patients received, all peritoneal fluids were subjected to culture and sensitivity of the organisms were noted. Out of 174 patients studied fifty three patients showed positive culture. Most common organism to be isolated was E.coli, followed by Enterococci. The duration between the onset of symptoms and presenting to the hospital plays a vital role in the prognosis of the patient. The contamination levels also go hand in hand with the duration of perforation.

Among 174 patients who had peritonitis, one hundred and four patients presented within 6 hours of onset of symptoms. Thirty seven were pre-pyloric, twenty three were duodenal, fourteen were appendicular, nineteen were ileal and eleven were

jejunal perforations. These patients had no bacterial growth and post-operative period was uneventful.

Patient who presented within 12hours of onset of symptoms were sixteen. Prepyloric were three, duodenal were six, appendicular was five and jejunal were two. 12 patients were positive for culture with minimal bacterial count of about 1200-1400cfu/mm. In group A, one patients of pre-pyloric, four of duodenal, two were ileal and two of jejunal perforation had bacterial count of about 1200-1400cfu/ml in pre-lavage which decreased to 1000-1200cfu/ml post lavage. Patients in group B, two of pre-pyloric, two of duodenal and one of appendicular perforation had bacterial count of about 1200-1400cfu/ml in pre-lavage and decreased to 1100-1300cfu/ml.

Patient who presented within 24 hours of onset of symptoms was nineteen. Among them pre-pyloric were four, duodenal were seven, appendicular were three, jejunal were one and ileal were three .Nineteen patient were positive for culture with bacterial count of about1400-1600cfu/ml. In group A, three of pre-pyloric, four of duodenal, two of appendicular and one of jejunal perforation had bacterial count of 1600-1800cfu/ml in pre lavage which reduced to1400-1600cfu/ml in post lavage. In group B, one of pre-pyloric, three of duodenal ,one of appendicular and three of ileal perforations had bacterial count of 1600-1800cfu/ml pre-lavage reduced to1400-1600cfu/ml post lavage.

Patient who presented after 24 hours of onset of symptoms was thirty five. Among them eight were pre-pyloric, nine were duodenal, twelve were appendicular, 1 was jejunal and five were ileal perforation. Thirty five patient were positive for culture with bacterial count of about 1800 -2,000cfu/ml. In group A, five of pre-pyloric, five of duodenal, five of appendicular, one of jejunal and two of ileal had bacterial count of 1800-2,000cfu/ml in pre lavage which reduced to 1600 -1800cfu/ml

in post lavage. In group B three of pre-pyloric, four of duodenal, seven of appendicular and three of ileal perforations had bacterial count of 1800 -2,000cfu/ml pre-lavage reduced to1600 -1800cfu/ml post lavage.

Based on the above mentioned observations, early perforations(0-12hrs)irrespective of the site have a fairly good prognosis, as the contamination anticipated will be low. The microbial colony count was very low in patients with early perforations and majority of the peritoneal fluids subjected to culture yielded no growth. A direct proportion can be seen in between time of presentation to hospital, contamination levels and prognosis. 'Too much is too bad' for the fluid that accumulates in perforation peritonitis. Irritant bile, faeces mixed, purulent are amongst the commonly seen contaminations. Post operative wound healing also depends upon the level of contamination. Patients who were intervened late due to delayed presentation (>12 hrs) had purulent/feculent exudate and the microbial counts was also found to be higher than those intervened early. Lower the perforation in the GIT (jejunal, ileal colonic) more severe contamination and higher the perforation(pre-pyloric and duodenal) less contamination was anticipated. But in our study, though a few patients had perforations distal in the GIT, they recovered fairly well as the time between onset of symptoms and intervention was considerably less, but few patients in pre-pyloric and duodenal presented late to ER as the time between onset of symptoms and intervention was more and encountered gross contamination of the peritoneal cavity with the complications like wound dehiscence, faecal fistula and burst abdomen.

At Pre BCC and Post BCC, in Povidone Iodine group 33.3% had Growth and in Normal saline group 27.6% had growth.

Among patients included in Povidone Iodine group, mean cell count at Pre BCC was

1748.28 ± 124.27 and at Post BCC was 1462.07 ± 169.90. There was significant decrease in Cell count at Post BCC compared to Pre BCC in Povidone Iodine group compared to Normal Saline group.

In Normal Saline group, mean cell count at Pre BCC was 1700.00 ± 131.88 and at Post BCC was 1554.17 ± 147.38. There was significant decrease in Cell count at Post BCC compared to Pre BCC in Normal Saline group.

Table 20 :Comparison of Pre BCC and Post BCC value in different types of perforation

Perforation	PreBCC(P VALUE	Post BCC (PVALUE)
Prepyloric	0.017	0.694
Duodenal	0.366	o.117
Appendicular	0.434	0.464
Ileal	0.564	0.509
Jejunal	0.765	0.024

COMPLICATIONS

A lot of complications after exploratory laparotomy has been studied worldwide. This study focussed more on the development of intra abdominal abscess (pelvic abscess, sub-diaphragmatic abscess and sub hepatic abscess) stitch abscess,burst abdomen, paralytic ileus ,faecal fistula ,intestinal obstruction due to adhesions and incisional hernia.

64 patients showed signs of SSI, 10 patients developed pneumonia, 8 patients had

paralytic ileus and 6 developed faecal fistula. All complications were treated conservatively and the patients recovered well.

SURGICAL SITE INFECTION

Surgical site infection was most common in the age group of 51 -60 years accounting for 45 /115patients .In 174 patients, surgical site infection was developed in 64 patients. Among them 56 patients had superficial surgical site infection and 8 patients had deep surgical site infection. Surgical site infection depends on the site of perforation, time of presentation to the hospital, peritoneal contamination and bacterial count.

Patients with pre-pyloric and duodenal perforation have less chances of contamination with minimal or without bacterial count with minimal chance of surgical site infection if presented to the hospital within 6hrs of presentation. On the other hand if they present to the hospital after 12hr, chance of contamination is more with increase in the bacterial count around 1200-2000cfu/ml, which leads to surgical site infection. Increase in time of presentation leads to increase in bacterial counts which causes deep surgical site infection. Fourteen patients of pre-pyloric perforation had surgical site infection, among them twelve patients who developed surgical site infection presented to the hospital with in and after 24 hrs. Among them three patients developed deep surgical site infection who had presented delayed to the hospital with gross contamination and increased bacterial count around 1800-2000cfu/ml.

In duodenal perforation twenty one patients developed surgical site infection. Among twenty one , sixteen patients presented to the hospital within and after 24 hours .Two patients among them developed deep surgical site infection, as they had gross contamination with a bacterial count around 1800-2000cfu/ml.

Sixteen patients of appendicular perforation ,eight patients of ileal perforation and four patients of jejunal perforation developed surgical site infection .Among them three patients of ileal perforation had deep surgical site infection as eight patients of ileal perforation presented to the hospital within and after 24 hours.

More distal the site of perforation in gastrointestinal tract (GIT), more is the peritoneal contamination and bacterial growth, as the bacterial count increases distally around 1800cfu/ml and chances for the postoperative complications are higher.

More proximal is the site of perforation, less is the degree of peritoneal contamination and bacterial growth with decreased bacterial count around 1200cfu/ml, provided the patient presents earlier to the hospital.

40.2% patients in the povidone iodine group, had wound infection and 33.3% of patients had wound infection in saline group. Incidence of infection was more in duodenal perforations, followed by appendicular, pre-pyloric, ileal, jejunal perforations. Sheeraz Khan et al[63] reported 20% reduction in incidence of wound infection, when superoxide solution was used for IOPL. On contrary, Schein et al[64] did not find any difference in incidence of wound infection when Chloramphenicol was used for IOPL.

Chest infection

Patients in our study were mostly in the age group of 51-60 years. Due to poor respiratory reserves and almost all patients being chronic smokers, developing respiratory infection was inevitable. We observed that though all patients were operated under general anaesthesia, only 10 patients developed respiratory infections. Schein[64] et al reported 16 % of the patients who has old age and delayed presentation had respiratory infection.

Fecal fistula

In our study 6 patients developed faecal fistula, 4 of them belong to normal saline group and 2 to povidone iodine group. 3 fistulas were noted in those who were operated for ileal perforations, followed by 2 jejunal perforations and 1 duodenal. There was no significant difference in the development of faecal fistulas in both the groups. Sheeraz Khan[63] et al (2009) reported 2.5% reduction in the incidence of faecal fistula in the study group, when superoxide solution was used for IOPL. This was not significant statistically.

Mortality

With modern treatment, diffuse peritonitis carries a mortality rate of about 10 percent reflecting the degree and duration of peritoneal contamination, age and fitness of the patient and the nature of the underlying cause.

Patients with delayed presentation has high rate of mortality. In our study 1 patient operated for ileal perforation succumbed to death. The patient was elderly male who was in severe degree of sepsis and had delayed presentation On the contrary

SUMMARY

SUMMARY

We analyzed 174 patients with perforative peritonitis which were confirmed on emergency laparotomy. Most of the patients in our study group were in the age group 51-60 years.

The perforations of proximal gastrointestinal tract (gastroduodenal) constituted about 69.2% of all the perforations. Majority of the patients had peptic ulcer perforation which included both pre-pyloric and duodenal perforations. Site of perforations showed wide variability in different studies. Only few patients with pre-pyloric perforation had developed post-operative complications. Patients were subjected to emergency exploratory laparotomy after adequate resuscitation. These patients were managed with procedure like closure of the perforation /resection of the bowel depending upon the operative findings Lower the perforation in the GIT (jejunal, ileal colonic) more severe contamination and higher the perforation(pre-pyloric and duodenal) less contamination was anticipated but in patients who presented to the hospital within 0-12 hrs, irrespective of the site of perforation, contamination was low compared to those who presented after 12 hours .

The difference in the postoperative hospital stay and complications were studied using povidone iodine in normal saline and normal saline in perforative peritonitis which were proved surgically.

There was a decrease in the post operative hospital stay in povidone iodine with normal saline. The p – value (0.930) was not significant in patients between these groups

The difference in bacterial colony count was studied using povidone iodine with normal saline and normal saline both pre and post peritoneal lavage in cases of

peritonitis which were proved surgically. There was decrease in bacterial colony count with the use of both the agents. At post BCC there was no significant decrease in mean cell count between these two groups Cell count was significantly lower in povidone iodine in normal saline than in normal saline.

There was one mortality in the study .This study also revealed that men are commonly affected and pre-pyloric perforation is the commonest site of perforation. E.coli is the most common organism isolated.

64 patients showed signs of SSI among them 56 had superficial SSI and 8 patients had deep SSI, 10 patients developed pneumonia, 8 patients had paralytic ileus and 6 developed faecal fistula. These complications were treated conservatively.

CONCLUSION

CONCLUSION

This clinical and bacteriological study has demonstrated the following:

1. Commonest cause for peritonitis is perforation.
2. Peritonitis is more common in men compared to women.
3. The common age group is in between 51 – 60 in cases of peritonitis.
4. Pre-pyloric perforation is the commonest site of perforation.
5. E.coli is the commonest organism isolated from the peritoneal contamination.
6. Bacterial /peritoneal contamination increases with time.
7. .Delayed presentation i.e., more than 12-24 hours increases the degree of contamination.
8. Postoperative complications like surgical site infections, pneumonia intra abdominal abscess are more in the patients with distally situated perforation and who had delayed presentation to the health care centre. None of the patients till date with long term follow up developed incisional hernia.
9. Povidone iodine in normal saline lavage significantly decreases the bacterial load when compared to normal saline lavage.
10. As for as clinical outcome is concerned there is no significant differences in both the groups.

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ANNEXURES

ANNEXURE-1

STANDARD PROFORMA

A COMPARATIVE STUDY BETWEEN POVIDONE IODINE IN NORMAL SALINE AND NORMAL SALINE FOR PERITONEAL LAVAGE IN PERITONITIS

Particulars of the patients

Name:

Ward:

Age:

OP No.

I.P. no:

Gender:

Unit:

Occupation:

Address:

Date of Admission:

Date of Surgery:

Date of Discharge:

Complaints

1) **Pain:**

-time of onset

-mode of onset

-site of pain

-migration of pain

-character of pain

-relation to vomiting

-relation to food intake

-aggravating factors

-relieving factors

2) Vomiting:

- onset

-duration

-frequency

-character of onset

-amount

-content

3) Bowels:

-last evacuation

-constipation/normal

-history of passing worms

4) Distension:

-duration

-location

-relation to pain

5) **Fever:**

-duration

-nature: continuous/intermittent/remittent

-relation to pain

-whether associated with chills and rigors

Previous History

- Of similar complaints

-Hematemesis

- Treatment of peptic ulcer

-Ingestion of drugs

Personal History

-Diet

-Appetite

-Smoking

-Alcohol

-Bowel habits

-Menstrual history

Family History

-Peptic ulcer/Diabetes/Hypertension/TB

General Physical Examination

-Appearance

-Attitude

-Build and nourishment

-Level of consciousness

-Dehydration

-Temperature

-Pulse

-Blood pressure

-Respiration

Local Examination(abdomen)

Inspection

-contours of the abdomen

-distension :uniform/upper/lower

-visible peristalsis

-umbilicus

-operation scars

-hernial orifices

-genitalia

Palpation

-temperature

-tenderness :localized/diffuse/rebound

-muscular rigidity: localized/generalized

-mass

-liver

-spleen

-abdominal girth

Percussion

-obliteration of liver dullness

-shifting dullness

Auscultation

-bowel sounds: present/absent

Other Relevant Examinations:

-Per rectal

empty/loaded

bleeding/mass felt

-Per vaginal

Systemic Examination:

-CVS/CNS/RS

Investigations:

Routine investigations: CBC with blood grouping and typing,

BT,

CT ,

RBS,

RFT,

Serum electrolytes,

HIV,

Hbs Ag,

Chest X ray,

widal test,

Urine routine,

ECG,

Erect Xray abdomen.

USG abdomen and pelvis.

Peritoneal fluid for culture and sensitivity

Pre and post bacterial cell count

CT scan abdomen and pelvis as and when required

Pre operative treatment

-Antibiotics(If yes, Drug, dose, frequency , duration)

-Other drugs

-Intravenous fluids

-blood transfusion

-gastric aspiration

Pre medication and Anaesthesia

Operative details

-Type of surgery

-Duration of surgery

-Type of drain

-Type of peritoneal fluid drained

Post operative management

-Iv fluids

-antibiotics

-blood transfusion

-other drugs

-Gastric aspiration

-oral fluids

-removal of drains

Sample for HPE: YES/NO

Histopathological report:

Organism isolated

Antibiotic sensitivity pattern

Post op antibiotics:

-Type, dose, frequency, duration

Complications

Local

-intra abdominal abscess (pelvic abscess, sub-diaphragmatic abscess and subhepatic abscess)

-stitch abscess

-burst abdomen

-paralytic ileus

-faecal fistula

-intestinal obstruction due to adhesions

-incisional hernia

General

-Pulmonary/toxaemia/cardiac/thrombotic/renal/agranulocytosis

Treatment of complications

Follow up in immediate post op period: 3^{rd} , 5^{th} 7^{th} days.

No. of days in hospital

Condition at the time of discharge:

Follow up after 1 month:

ANNEXURE-II

INFORMED CONSENT FORM

IMr./Mrs. have been explained in my own understandable language, that I will be included in a study which is A COMPARTIVE STUDY BETWEEN POVIDONE IODINE IN NORMAL SALINE AND NORMAL SALINE FOR PERITONEAL LAVAGE IN PERITONITIS being conducted in RL JALAPPA HOSPITAL.

I have been explained that my clinical findings, investigations, intraoperative findings, post-operative course, will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the follow up details and possible benefits and adversities due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.I have principal investigator mobile no for enquiries.I in my sound mind give full consent to be added in the part of this study.

Signature of the patient: Name:

Signature of the witness: Name:

Relation to patient:

Date:

Place:

ಭಾಗವಹಿಸಲು ಸಮ್ಮತಿ

ನಾನು, ರುಜುಮಾಡಿರುವ, ಈ ಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲು ಮತ್ುಈ ಒಪ್ಪಿಗೆ ರೂಪದಲ್ಲಿಅಂಶಗಳಂತೆ ನನನ ವೆ್ಯಕ್ತುಕ ಮಾಹಿತಿಯ ಸಂಗರಹಣೆ ಮತ್ುಡಿಸೊ್ಲೋಸರ್ ಅಧಿಕೃತ್ಗೊಳಿಸಲು ಒಪಪಿತು್ೂೀರಿ.

ಈ ಅಧ್ಯಯನದ ಉದೆದೋಶ,ಬಳಸಲಾಗುತ್ುದೆಎಂದು ಕಾಯಯವಿಧಾನಗಳು, ಅಧ್ಯಯನ ಮತ್ುಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗರಹಿಸಿದ ಮತ್ುಬಹಿರಂಗ ನಡೆಯಲ್ಲದೆ ಮಾಹಿತಿಯನುನ ಗೌಪಯ ಪರಕೃತಿಯಲ್ಲಿನನನ ಒಳಗೊಳುವಿಕೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯಗಳನುನ ಮತ್ುಲಾಭಗಳನುನ ಅರ್ಯಮಾಡಿಕೊಂಡಿದೆದೋನೆ.

ನಾನು ವಿವಿಧ್ ಈ ಅಧ್ಯಯನದ ಅಂಶಗಳು ಮತ್ುನನನನ ಪರಶ್ೆನಗಳಿಗೆ ನನನ ತ್ೃಪ್ುಕರಉತ್ುರಗಳನುನಮಾಡಲಾಗಿದೆ ಸಂಬಂಧಿಸಿದ ಪರಶ್ೆನಗಳನುನ ಕೆ್ೂೀಳಲು ಅವಕಾಶ ಹೊಂದಿದದರು.

ನಾನು ಯಾವಪದೆ್ೂೀ ಸಮಯದಲ್ಲಿಈ ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳುವಂತೆ ಮತ್ುಈ ನನನ ಮುಂದಿನ ಆರೆ್ೃಕೆ ಬದಲಾಗುವಪದಿಲಿಉಚಿತ್ ಉಳಿಯಲು ಎಂದು ಅರ್ಯ

ವಿಷಯದ ಹೆಸರು ಮತ್ತುಸಹಿ / ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತ್ತು

ಪೋಷಕ / ಪೋಷಕರು ಹೆಸರು ಮತ್ತುಸಹಿ

ಒಪ್ಪಿಗೆ ಪಡೆದ ವಯಕ್ತುಯಹೆಸರು ಮತ್ತುಸಹಿ

ANNEXURE-3

PATIENT INFORMATION SHEET

"A COMPARATIVE STUDY BETWEEN POVIDONE IODINE IN NORMAL SALINE AND NORMAL SALINE FOR PERITONEAL LAVAGE IN PERITONITIS

Study site RLJALAPPA HOSPITAL, TAMAKA, KOLAR

Objective of the study: To compare the clinical outcome of the patients with peritonitis who have received peritoneal lavage with povidone iodine in normal saline and normal saline

Intra-abdominal infections are among the most difficult infections to diagnose early and treat effectively. Peritonitis resulting from visceral inflammation or perforation is polymicrobial, which contains anaerobic and aerobic nature of bacterial flora. Peritonitis requires draining the abscess, cleaning the peritoneal cavity and eliminating contamination. Peritoneal lavage acts as a mechanical cleanser, which reduces the bacterial growth in the peritoneal cavity, reducing sepsis, surgical site infections, intra-abdominal abscess faecal fistula and promotes rapid recovery. Several potential benefits of the peritoneal lavage have been advanced. Saline lavage reduces significantly counts in peritoneal fluid of aerobic and anaerobic bacteria in peritoneal fluid and gives us the idea of amount of debris present in the peritoneal fluid. Povidone iodine is an effective bactericide

Procedure and Protocol; This is a comparative study 172 patients admitted for peritonitis during the period from December 2016 to June 2018 will be included in the study

After obtaining informed consent, patient will be divided into two groups of 86 using even odd method

Group 1:peritoneal lavage with povidone iodine in normal saline

Group2:peritoneal lavage with normal saline

Reimbursements you will not be given money or gifts to take part in this research

Confidentiality we will not share the identity of the participant, the information we collect from you will be confidential and only researchers involved in this project will have to access to it

Right to refuse or with draw: you don't have to take part in this research if you do not wish to do so and you are free to withdraw at any time. The care you will get will not change if you don't wish to participate

You are required to sign/provide thumb impression only if you voluntarily agree to participate in this study

Principal investigator's details

Dr.spurthi Sanganboina

Post graduate(08861447665)

DEPT. OF GENERAL SURGERY

ರೋಗಿಯ ಮಾಹಿತಿ ಪ್ರತಿ

ಅಧ್ಯಯನ ಶೀರ್ಷಿಕೆ: ಜಠರದ ಸೋಂಕಿನಲ್ಲಿ ಕಂಡುಬರುವ ಪೆರಿಟೋರಿಯಂನ ಮಾರ್ಜನ, ಸಮಾನ್ಯ ಲವಣ ಮತ್ತು ಸಾಮಾನ್ಯ ಲವಣದಲ್ಲಿನ ಕರ್ಣಾಟ ಐಯೋಡಿನ್ ಬಗೆಗಿನ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನ.

ಅಧ್ಯಯನ ಕೇಂದ್ರ: ಆರ್.ಎಲ್. ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ, ಕೋಲಾರ

ಗುರಿ: ಜಠರದ ಸೋಂಕು ಕಂಡು ಬರುವ ರೋಗಿಗಳಲ್ಲಿ ಪೆರಿಟೋರಿಯಂನ ಮಾರ್ಜನ, ಸಮಾನ್ಯ ಲವಣ ಮತ್ತು ಸಾಮಾನ್ಯ ಲವಣದಲ್ಲಿನ ಕರ್ಣಾಟ ಐಯೋಡಿನ್‌ನ ವೈದ್ಯಕೀಯ ಫಲಿತಾಂಶದ ಮೇಲೆ ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನ.

ಅಧ್ಯಯನದ ಉದ್ದೇಶ: ಜಠರದೊಳಗಿನ ಸೋಂಕಿನ ತಪಾಸಣೆ ಮತ್ತು ಪರಿಣಾಮಕಾರಿ ಚಿಕಿತ್ಸೆಯನ್ನು ನೀಡಲು ಬಹು ಕಠಿಣವಾಗಿದ್ದು, ಜಠರದೊಳಗಿನ ಸೋಂಕು ಒಳಾಂಗಗಳ ಉರಿಯೂತ /ಬ್ಯಾಕ್ಟೀರಿಯಾ ಫ್ಲೋರದಲ್ಲಿ ಏರೋಬಿಕ್ ಮತ್ತು ಅನೇರೊಬಿಕ್ ಇದಲ್ಲಿ, ಪಾಲಿಮೈಕ್ರೋಬಿಯಲ್ ರಂಧ್ರ ಉಂಟಾಗಿ ಜಠರದ ಸೋಂಕು ಉಂಟಾಗುತ್ತದೆ. ಜಠರದ ಸೋಂಕಿನಿಂದಾದ ಬಾವನ್ನು ಬರಿದಾಗಿಸಲು, ಜಠರದ ಕುಳಿಯನ್ನು ಸ್ವಚ್ಛಗೊಳಿಸಲು ಮತ್ತು ಕಲ್ಮಶಗಳನ್ನು ತೊಲಗಿಸಲು ಚಿಕಿತ್ಸೆಯ ಅವಶ್ಯಕತೆ ಇರುತ್ತದೆ. ಪೆರಿಟೋರಿಯಂನ ಮಾರ್ಜನ ಯಾಂತ್ರಿಕ ಸ್ವಚ್ಚಗೊಳಿಸುವ ಏಜೆಂಟ್‌ನಂತೆ ಕಾರ್ಯನಿರ್ವಹಿಸುವುದಲ್ಲದೆ, ಜಠರದ ಕುಳಿಯಲ್ಲಿ ಬ್ಯಾಕ್ಟೀರಿಯಾ ಬೆಳವಣಿಗೆಯನ್ನು ಕಡಿಮೆ ಮಾಡಲು, ಸೆಪ್ಸಿಸ್ ಸೋಂಕನ್ನು ಕಡಿಮೆ ಮಾಡಲು, ಸರ್ಜಿಕಲ್ ಸೈಟ್ಸ್ ಸೋಂಕು, ಜಠರದೊಳಗಿನ ಬಾವನ್ನು, ಚರಟದ ನಾಳದ ಸೋಂಕನ್ನು ನಿವಾರಿಸುವುದಲ್ಲದೆ, ಸೋಂಕನ್ನು ಶೀಘ್ರವೇ ನಿವಾರಣೆ ಮಾಡುತ್ತದೆ. ಪೆರಿಟೋರಿಯಂನ ಮಾರ್ಜನದೊಳಗಿನ ಕೆಲ ಚಿಕಿತ್ಸಕ ಬಳಕೆಯು ಅತ್ಯುನ್ನತವಾಗಿರುತ್ತದೆ. ಪೆರಿಟೋರಿಯಂನ ಮಾರ್ಜನ ಪೆರಿಟೋನಿಯಲ್ ದ್ರವದಲ್ಲಿನ ಏರೋಬಿಕ್ ಮತ್ತು ಅನೇರೊಬಿಕ್ ಬ್ಯಾಕ್ಟೀರಿಯಾದ ಗಮನಾರ್ಹ ಎಣಿಕೆಗಳನ್ನು ಕಡಿಮೆ ಮಾಡಿ, ಪೆರಿಟೋನಿಯಲ್ ದ್ರವದಲ್ಲಿನ ಕಸದ ರಾಶಿಯ ಅಂದಾಜನ್ನು ನೀಡುತ್ತದೆ. ಕರ್ಣಾಟ ಐಯೋಡಿನ್ ಒಂದು ಪರಿಣಾಮಾತ್ಮಕ ಬ್ಯಾಕ್ಟೀರಿಯವನ್ನು ಕೊಲ್ಲುವ ಏಜೆಂಟ್ ಆಗಿರುತ್ತದೆ.

ವಿಧಾನ ಮತ್ತು ಶಿಷ್ಟಾಚಾರ: ಇದು ಒಂದು ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನವಾಗಿದ್ದು, ನವೆಂಬರ್ 2016 ರಿಂದ ಜೂನ್ 2018 ರವರೆಗೆ ದಾಖಲಾಗಿರುವ ರೋಗಿಗಳನ್ನು, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ತೊಡಗಿಸಿಕೊಳ್ಳಲಾಗುವುದು.

ಮೊದಲನೇ ಗುಂಪು: ಪೆರಿಟೋರಿಯಂನ ಮಾರ್ಜನವನನ್ನು ಸಾಮಾನ್ಯ ಲವಣದೊಂದಿಗೆ ಪರೀಕ್ಷಿಸುವುದು.

ಎರಡನೇ ಗುಂಪು: ಪೆರಿಟೋರಿಯಂನ ಮಾರ್ಜನವನನ್ನು ಸಾಮಾನ್ಯ ಲವಣ ಮತ್ತು ಸಾಮಾನ್ಯ ಲವಣದಲ್ಲಿನ ಕರ್ಣಾಟ ಐಯೋಡಿನ್‌ನೊಂದಿಗೆ ಪರೀಕ್ಷಿಸುವುದು.

ಮರುಪಾವತಿ: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವರಿಗೆ ಯಾವುದೇ ರೀತಿಯ ನಗದು ಅಥವ ಬಹುಮಾನವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ಗೌಪ್ಯತೆ: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವರ ವಿವರಗಳನ್ನು ಬೇರೆ ವ್ಯಕ್ತಿಗೆ ಹಂಚಿಕೆ ಮಾಡಿಕೊಳ್ಳುವುದಿಲ್ಲ. ನಿಮ್ಮ ಬಳಿ ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯು ಗೌಪ್ಯತೆಯಿಂದ ಇರಿಸಿಕೊಳ್ಳಲಾಗುವುದು. ಮತ್ತು ಈ ಮಾಹಿತಿಯು ಸಂಶೋಧನೆ ನಡೆಸುವವರಿಗೆ ಬಳಸಿಕೊಳ್ಳಲು ಮಾತ್ರ ಹಕ್ಕಿರುತ್ತದೆ.

ತಿರಸ್ಕರಿಸಲು ಅಥವ ಹಿಂಪಡೆಯಲು ಇರುವ ಹಕ್ಕು: ನಿಮಗೆ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಇಚ್ಛಿಸದಿದಲ್ಲಿ, ನೀವು ಈ ಅಧ್ಯಯನದಿಂದ ನಿಮ್ಮ ಮಾಹಿತಿಯನ್ನು ಹಿಂಪಡೆಯಬಹುದು. ನೀವು ಭಾಗವಹಿಸಲು ಇಚ್ಛಿಸದಿದಲ್ಲಿ, ನಿಮ್ಮ ಮೇಲೆ ಇರುವ ವಿಶ್ವಾಸವು ಎಂದಿಗೂ ಬದಲಾವಣೆಯಾಗುವುದಿಲ್ಲ. ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ, ಈ ಅಧ್ಯಯನಕ್ಕೆ ಭಾಗವಹಿಸಲು ಇಚ್ಛಿಸಿದಲ್ಲಿ ಮಾತ್ರ ನೀವು ಸಹಿ / ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತನ್ನು ನೀಡಬೇಕಾಗುವುದು.

ನಿಮಗೆ ಯಾವುದೇ ರೀತಿಯ ಸ್ಪಷ್ಟ ವಿವರಣೆ ಬೇಕಾದಲ್ಲಿ, ನೀವು ಈ ಕೆಳಗೆ ಸಂಬಂಧಪಟ್ಟ ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಗಳನ್ನು ಸಂಪರ್ಕಿಸುವುದು.

ಡಾ॥ ಸ್ಫೂರ್ತಿ ಸಂಗನ್‌ಬೋನಿಯ

ಸ್ನಾತಕೋತರ ಪದವಿಧರರು (ಸಾಮಾನ್ಯ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ)

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 8106674893

ಈ – ಮೇಲ್ ಐಡಿ: ssspurthiyadav@gmail.com

ANNEXURE-4

KEYS TO MASTER CHART

SN	SERIAL NUMBER
A	AGE
IP. No.	IN PATIENT NUMBER
G	GENDER
DOA	DATE OF ADMISSION
DOS	DURATION OF SURGERY
DOD	DATE OF DISCHARGE
DOD1	DATE OF DEATH
PRE BCC	PRE LAVAGE BACTERIAL CELL COUNT
POST BCC	POST LAVAGE BACTERIAL CELL COUNT
O I	ORGANISMS ISOLATED
R	RECOVERED
M	MALE
H S	HOSPITAL STAY IN DAYS
SSI	SURGICAL SITE INFECTION
CFU	COLONY FORMING UNIT

E. COLI	ESCHERICHIA COLI
A	ABSENT
R	RECOVERED
D	DEATH
NS	NORMAL SALINE
PI	POVIDONE IODINE
P	PRESENT
F	FEMALE
ml	Milli litre

NO.	IP.NO.	A	G	DIAGNOSIS	DOA	DOS	DOD/DOD1	HS	LAVAGE	PRE.BCC	POST.BCC	OI	SSI	FOLLOWUP
1	462331	39	M	PREPYLORICPERFORATION	01/01/2017	01/01/2017	08/01/2017	7 DAYS	NS	NO GROWTH	NO GROWTH		A	R
2	462747	48	M	PREPYLORICPERFORATION	04/01/2017	04/01/2017	12/01/2017	8 DAYS	PI	NO GROWTH	NO GROWTH		A	R
3	463108	56	M	PREPYLORICPERFORATION	07/01/2017	07/01/2017	15/01/2017	8 DAYS	NS	NO GROWTH	NO GROWTH		A	R
4	463543	50	M	APPENDICULARPERFORATION	11/01/2017	11/01/2017	31/01/2017	20 DAYS	PI	1800CFU/ML	1700cfu/ml	E.COLI	P	R
5	463897	51	M	PREPYLORICPERFORATION	13/01/2017	13/01/2017	19/01/2017	6 DAYS	NS	NO GROWTH	NO GROWTH		A	R
6	464567	59	M	PREPYLORICPERFORATION	19/01/2017	19/01/2017	26/01/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
7	465086	62	M	ILEALPERFORATION	24/01/2017	24/01/2017	03/02/2017	10 DAYS	NS	1200cfu/ml	1100cfu/ml	ENTEROCOCCI	A	R
8	465596	54	M	ILEALPERFORATION	29/01/2017	29/01/2017	16/02/2017	18 DAYS	PI	1900CFU/ML	1700cfu/ml	E.COLI	P	R
9	465875	55	M	PREPYLORICPERFORATION	02/02/2017	02/02/2017	10/02/2017	8 DAYS	NS	NO GROWTH	NO GROWTH		A	R
10	466534	61	M	ILEALPERFORATION	07/02/2017	07/02/2017	16/02/2017	9 DAYS	PI	1300CFU/ML	1100 CFU/ML	E.COLI	A	R
11	546632	45	M	ILEALPERFORATION	13/02/2017	13/02/2017	22/02/2017	9 DAYS	NS	1600CFU/ML	1400CFU/ML	E.COLI	P	R
12	546696	56	M	PREPYLORICPERFORATION	16/02/2017	16/02/2017	23/02/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
13	568973	56	M	PREPYLORICPERFORATION	22/02/2017	22/02/2017	11/03/2017	17 DAYS	NS	1600CFU/ML	1400CFU/ML	E.COLI	P	R
14	564123	50	M	ILEALPERFORATION	24/02/2017	24/02/2017	03/03/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
15	520013	54	M	PREPYLORICPERFORATION	28/02/2017	28/02/2017	07/03/2017	7 DAYS	NS	NO GROWTH	NO GROWTH		A	R
16	546331	56	M	PREPYLORICPERFORATION	03/03/2017	03/03/2017	10/03/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
17	596321	26	M	APPENDICULARPERFORATION	06/03/2017	06/03/2017	15/03/2017	9 DAYS	NS	1500CFU/ML	1300CFU/ML	E.COLI	P	R
18	596478	62	M	PREPYLORICPERFORATION	12/03/2017	12/03/2017	19/03/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
19	596325	60	M	PREPYLORICPERFORATION	17/03/2017	17/03/2017	24/03/2017	7 DAYS	NS	NO GROWTH	NO GROWTH		A	R
20	582143	49	M	PREPYLORICPERFORATION	22/03/2017	22/03/2017	29/03/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
21	582312	60	M	COLONIC PERFORATON	25/03/2017	25/03/2017	08/03/2017	14 DAYS	NS	1800cfu/ml	1600CFU/ML	ENTEROCOCCI	P	R
22	564211	56	M	PREPYLORICPERFORATION	29/03/2017	29/03/2017	05/03/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
23	546001	54	M	PREPYLORICPERFORATION	01/04/2017	01/04/2017	08/04/2017	7 DAYS	NS	NO GROWTH	NO GROWTH		A	R
24	593211	52	M	PREPYLORICPERFORATION	05/04/2017	05/04/2017	15/04/2017	10 DAYS	PI	1600cfu/ml	1400CFU/ML	E.COLI	P	R
25	564113	58	M	PREPYLORICPERFORATION	17/04/2017	17/04/2017	25/04/2017	8 DAYS	NS	NO GROWTH	NO GROWTH		A	R
26	563221	60	M	ILEALPERFORATION	18/04/2017	18/04/2017	26/04/2017	8 DAYS	PI	NO GROWTH	NO GROWTH		A	R
27	568960	58	M	ILEALPERFORATION	23/04/2017	23/04/2017	07/05/2017	14 DAYS	NS	1600cfu/ml	1500CFU/ML	ENTEROCOCCI	P	R

28	564120	56	M	APPENDICULARPERFORATION	25/04/2017	25/04/2017	31/04/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
29	596324	60	M	ILEALPERFORATION	26/04/2017	26/04/2017	02/05/2017	7 DAYS	NS	NO GROWTH	NO GROWTH		A	R
30	563961	32	M	DUODENALPERFORATION	29/04/2017	29/04/2017	03/05/2017	5 DAYS	PI	NO GROWTH	NO GROWTH		A	R
31	578966	52	M	ILEALPERFORATION	30/04/2017	30/04/2017	13/05/2017	14 DAYS	NS	1800CFU/ML	1600CFU/ML	E.COLI	P	R
32	586411	54	M	JEJUNALPERFORATION	02/05/2017	02/05/2017	08/05/2017	6 DAYS	PI	NO GROWTH	NO GROWTH		A	R
33	569366	50	M	APPENDICULARPERFORATION	05/05/2017	05/05/2017	20/05/2017	15 DAYS	NS	1900CFU/ML	1700cfu/ml	ENTEROCOCCI	P	R
34	547820	52	M	JEJUNALPERFORATION	11/05/2017	11/05/2017	19/05/2017	7 DAYS	PI	NO GROWTH	NO GROWTH		A	R
35	563296	54	M	ILEALPERFORATION	15/05/2017	15/05/2017	29/05/2017	14 DAYS	NS	1600cfu/ml	1500CFU/ML	E.COLI	P	R
36	586933	56	M	ILEALPERFORATION	18/05/2017	18/05/2017	27/05/2017	8 DAYS	PI	NO GROWTH	NO GROWTH		A	R
37	544361	58	F	ILEALPERFORATION	22/05/2017	22/05/2017	31/05/2017	6 DAYS	NS	NO GROWTH	NO GROWTH		A	R
38	547896	62	M	JEJUNALPERFORATION	26/05/2017	26/05/2017	14/06/2017	9 DAYS	PI	NO GROWTH	NO GROWTH		A	R
39	569361	54	M	APPENDICULARPERFORATION	30/05/2017	30/05/2017	16/06/2017	16 DAYS	NS	1600cfu/ml	1500CFU/ML	ENTEROCOCCI	P	R
40	566485	56	M	JEJUNALPERFORATION	02/06/2017	02/06/2017	12/06/2017	10 DAYS	PI	NO GROWTH	NO GROWTH		A	R
41	582369	54	M	ILEALPERFORATION	06/06/2017	06/06/2017	14/06/2017	11 DAYS	NS	NO GROWTH	NO GROWTH		A	R
42	546931	50	M	ILEALPERFORATION	11/06/2017	11/06/2017	02/07/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
43	563973	58	M	JEJUNALPERFORATION	19/06/2017	19/06/2017	28/06/2017	10 DAYS	NS	NO GROWTH	NO GROWTH		A	R
44	546392	60	M	ILEALPERFORATION	22/06/2017	22/06/2017	03/07/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
45	596321	54	M	JEJUNALPERFORATION	26/05/2017	26/05/2017	05/07/2017	10 dAYS	NS	NO GROWTH	NO GROWTH		A	R
46	547121	52	M	JEJUNALPERFORATION	29/06/2017	29/06/2017	13/07/2017	15 DAYS	PI	NO GROWTH	NO GROWTH		P	R
47	569632	54	M	ILEALPERFORATION	31-06-2017	31-06-2017	14/07/2017	14 DAYS	NS	NO GROWTH	NO GROWTH		A	R
48	596321	55	M	JEJUNALPERFORATION	03/07/2017	03/07/2017	15/07/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
49	568962	56	M	APPENDICULARPERFORATION	09/07/2017	09/07/2017	20/07/2017	11 DAYS	NS	1800 cfu/ml	1600 cfu/ml	E.COLI	P	R
50	578146	54	M	APPENDICULARPERFORATION	11/07/2017	11/07/2017	26/07/2017	15 DAYS	PI	1600CFU/ML	1500CFU/ML	ENTEROCOCCI	P	R
51	576632	50	M	PREPYLORICPERFORATION	16/07/2017	16/07/2017	24/07/2017	8 DAYS	NS	NO GROWTH	NO GROWTH		A	R
52	547893	54	M	DUODENALPERFORATION	19/07/2017	19/07/2017	31/07/2017	12DAYS	PI	NO GROWTH	NO GROWTH		A	R
53	546230	56	M	DUODENALPERFORATION	23/07/2017	23/07/2017	05/08/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
54	598756	58	M	JEJUNALPERFORATION	25/07/2017	25/07/2017	07/08/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
55	584196	54	M	DUODENALPERFORATION	30/07/2017	30/07/2017	13/08/2017	13 DAYS	NS	1800 cfu/ml	1600 cfu/ml	E.COLI	P	R
56	594586	52	M	APPENDICULARPERFORATION	02/08/2017	02/08/2017	14/08/2017	12 DAYS	PI	1800 cfu/ml	1600 cfu/ml	E.COLI	P	R
57	532569	50	M	PREPYLORICPERFORATION	06/08/2017	06/08/2017	18/08/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
58	586341	56	M	DUODENALPERFORATION	19/08/2017	19/08/2017	31/08/2017	12 DAYS	PI	1700cfu/Ml	1600CFU/ML	E.COLI	P	R
59	587961	54	M	APPENDICULARPERFORATION	21/08/2017	21/08/2017	02/09/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		P	R

60	536981	60	M	DUODENALPERFORATION	23/08/2017	23/08/2017	09/09/2017	16 DAYS	PI	NO GROWTH	NO GROWTH		p	R
61	581452	58	M	PREPYLORICPERFORATION	25/08/2017	25/08/2017	09/09/2017	14 DAYS	NS	NO GROWTH	NO GROWTH		A	R
62	569354	57	M	DUODENALPERFORATION	29/08/2017	29/08/2017	11/09/2017	13 DAYS	PI	NO GROWTH	NO GROWTH		A	R
63	564789	55	M	ILEALPERFORATION	31/08/2017	31/08/2017	02/09/2017		NS	1900CFU/ML	1700cfu/ml	E.COLI	P	D
64	520146	53	M	APPENDICULARPERFORATION	01/09/2017	01/09/2017	18/09/2017	18 DAYS	PI	1800 cfu/ml	1600 cfu/ml	ENTEROCOCCI	P	R
65	542365	56	M	PREPYLORICPERFORATION	06/09/2017	06/09/2017	16/09/2017	10 DAYS	NS	NO GROWTH	NO GROWTH		A	R
66	526932	54	M	DUODENALPERFORATION	13/09/2017	13/09/2017	27/09/2017	15 DAYS	PI	NO GROWTH	NO GROWTH		P	R
67	578621	54	M	DUODENALPERFORATION	14/09/2017	14/09/2017	04/10/2017	20 DAYS	NS	NO GROWTH	NOGROWTH		P	R
68	587632	25	M	APPENDICULARPERFORATION	18/09/2017	18/09/2017	29/09/2017	11 DAYS	PI	NO GROWTH	NO GROWTH		P	R
69	588756	62	F	DUODENALPERFORATION	24/09/2017	24/09/2017	16/10/2017	22 DAYS	NS	1800CFU/ML	1700cfu/ml	ENTEROCOCCI	P	R
70	563321	60	M	DUODENALPERFORATION	29/09/2017	29/09/2017	11/10/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
71	596370	56	M	DUODENALPERFORATION	30/09/2017	30/09/2017	12/10/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
72	574631	61	F	APPENDICULARPERFORATION	04/10/2017	04/10/2017	16/10/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
73	542310	35	M	DUODENALPERFORATION	10/10/2017	10/10/2017	28/10/2017	18 DAYS	NS	NO GROWTH	NO GROWTH		P	R
74	546935	28	M	APPENDICULARPERFORATION	15/10/2017	15/10/2017	27/10/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
75	549632	54	M	DUODENALPERFORATION	17/10/2017	17/10/2017	29/11/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
76	586635	39	F	ILEALPERFORATION	23/10/2017	23/10/2017	05/12/2017	13 DAYS	PI	NO GROWTH	NO GROWTH		A	R
77	546321	50	F	ILEALPERFORATION	29/10/2017	29/10/2017	23/12/2017	29 DAYS	NS	1800cfu/ml	1700CFU/ML	ENTEROCOCCI	P	R
78	546991	54	F	APPENDICULARPERFORATION	09/11/2017	09/11/2017	25/12/2017	16 DAYS	PI	1800 cfu/ml	1600 cfu/ml	E.COLI	P	R
79	569325	50	F	ILEALPERFORATION	10/11/2017	10/11/2017	23/11/2017	13 DAYS	NS	NO GROWTH	NO GROWTH		A	R
80	563214	54	F	APPENDICULARPERFORATION	12/11/2017	12/11/2017	25/11/2017	13 DAYS	PI	NO GROWTH	NO GROWTH		A	R
81	586412	56	F	ILEALPERFORATION	15/11/2017	15/11/2017	28/11/2017	13 DAYS	NS	NO GROWTH	NO GROWTH		A	R
82	586311	58	M	PREPYLORICPERFORATION	17/11/2017	17/11/2017	04/12/2017	15 DAYS	PI	1700 CFU/ML	1500CFU/ML	E.COLI	P	R
83	569842	56	F	APPENDICULARPERFORATION	20/11/2017	20/11/2017	01/12/2017	11 DAYS	NS	NO GROWTH	NO GROWTH		A	R
84	536987	27	F	APPENDICULARPERFORATION	23/11/2017	23/11/2017	03/12/2017	10 DAYS	PI	NO GROWTH	NO GROWTH		A	R
85	582642	58	F	PREPYLORICPERFORATION	25/11/2017	25/11/2017	07/12/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R

86	548691	52	F	PREPYLORICPERFORATION	28/11/2017	28/11/2017	11/12/2017	13 DAYS	PI	NO GROWTH	NO GROWTH		A	R
87	514763	58	F	APPENDICULARPERFORATION	30/11/2017	30/11/2017	05/12/2017	05 DAYS	NS	NO GROWTH	NO GROWTH		A	R
88	536972	50	F	DUODENALPERFORATION	01/12/2017	01/12/2017	11/12/2017	10 DAYS	PI	NO GROWTH	NO GROWTH		A	R
89	546662	29	F	APPENDICULARPERFORATION	04/12/2017	04/12/2017	16/12/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
90	547893	54	M	PREPYLORICPERFORATION	08/12/2017	08/12/2017	30/12/2017	22 DAYS	PI	1800CFU/ML	1600CFU/ML	ENTEROCOCCI	P	R
91	569998	35	M	PREPYLORICPERFORATION	09/12/2017	09/12/2017	21/12/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
92	548963	54	M	DUODENALPERFORATION	13/12/2017	13/12/2017	24/12/2017	11 DAYS	PI	NO GROWTH	NO GROWTH		A	R
93	584635	56	F	APPENDICULARPERFORATION	16/12/2017	16/12/2017	27/12/2017	11 DAYS	NS	NO GROWTH	NO GROWTH		A	R
94	526982	58	F	DUODENALPERFORATION	18/12/2017	18/12/2017	30/12/2017	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
95	547896	54	F	PREPYLORICPERFORATION	19/12/2017	19/12/2017	31/12/2017	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
96	512478	55	M	DUODENALPERFORATION	21/12/2017	21/12/2017	01/01/2018	11 DAYS	PI	NO GROWTH	NO GROWTH		A	R
97	546932	51	F	PREPYLORICPERFORATION	24/12/2017	24/12/2017	04/01/2018	11 DAYS	NS	NO GROWTH	NO GROWTH		P	R
98	589672	30	F	APPENDICULARPERFORATION	25/12/2017	25/12/2017	06/01/2018	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
99	582631	50	M	DUODENALPERFORATION	27/12/2017	27/12/2017	08/01/2018	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
100	547869	48	F	DUODENALPERFORATION	30/12/2017	30/12/2017	14/01/2018	15 DAYS	PI	NO GROWTH	NO GROWTH		P	R
101	546321	45	F	PREPYLORICPERFORATION	31/12/2017	31/12/2017	11/01/2018	11 DAYS	NS	NO GROWTH	NO GROWTH		A	R
102	541963	25	F	APPENDICULARPERFORATION	01/01/2018	01/01/2018	11/01/2018	10 DAYS	PI	NO GROWTH	NO GROWTH		A	R
103	589647	40	F	DUODENALPERFORATION	02/01/2018	02/01/2018	11/01/2018	09 DAYS	NS	NO GROWTH	NO GROWTH		A	R
104	514789	44	M	PREPYLORICPERFORATION	04/01/2018	04/01/2018	13/01/2018	09 DAYS	PI	NO GROWTH	NO GROWTH		A	R
105	561473	54	M	DUODENALPERFORATION	07/01/2018	07/01/2018	15/01/2018	08 DAYS	NS	NO GROWTH	NO GROWTH		A	R
106	549652	56	F	PREPYLORICPERFORATION	09/01/2018	09/01/2018	31/01/2018	22 DAYS	PI	1800 cfu/ml	1600CFU/ML	ENTEROCOCCI	p	R
107	566652	25	F	APPENDICULARPERFORATION	12/01/2018	12/01/2018	24/01/2018	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
108	547851	55	M	PREPYLORICPERFORATION	15/01/2018	15/01/2018	10/02/2018	24 DAYS	PI	1800CFU/ML	1600CFU/ML	E.COLI	P	R
109	596324	26	F	APPENDICULARPERFORATION	16/01/2018	16/01/2018	28/01/2018	12 DAYS	NS	NO GROWTH	NO GROWTH		A	R
110	589647	56	F	PREPYLORICPERFORATION	18/01/2018	18/01/2018	30/01/2018	12 DAYS	PI	NO GROWTH	NOGROWTH		A	R
111	587496	29	M	APPENDICULARPERFORATION	20/01/2018	20/01/2018	09/02/2018	20 DAYS	NS	1800CFU/ML	1600CFU/ML	E.COLI	P	R
112	521463	30	F	APPENDICULARPERFORATION	21/01/2018	21/01/2018	05/02/2018	15 DAYS	PI	NO GROWTH	NO GROWTH		P	R
113	543982	50	F	PREPYLORICPERFORATION	23/01/2018	23/01/2018	08/02/2018	16 DAYS	NS	NO GROWTH	NO GROWTH		A	R
114	547896	45	F	JEJUNALPERFORATION	26/01/2018	26/01/2018	15/02/2018	20 DAYS	PI	1800CFU/ML	1700cfu/ml	ENTEROCOCCI	P	R

115	523645	54	F	JEJUNALPERFORATION	27/01/2018	27/01/2018	06/02/2018	10 DAYS	NS	1200 cfu/ml	1100cfu/ml	ENTEROCOCCI	A	R
116	589612	54	F	JEJUNALPERFORATION	28/01/2018	28/01/2018	15/02/2018	18 DAYS	PI	1600CFU/ML	1400CFU/ML	ENTEROCOCCI	P	R
117	581472	56	M	PREPYLORICPERFORATION	29/01/2018	29/01/2018	11/02/2018	13 DAYS	NS	NO GROWTH	NO GROWTH		A	R
118	563957	58	M	JEJUNALPERFORATION	31/01/2018	31/01/2018	13/02/2018	13 DAYS	PI	1200cfu/ml	1100cfu/ml	E.COLI	P	R
119	569872	58	F	JEJUNALPERFORATION	03/02/2018	03/02/2018	2/117/2018	14 DAYS	NS	1200cfu/ml	1100cfu/ml	ENTEROCOCCI	A	R
120	547896	52	F	PREPYLORICPERFORATION	05/02/2018	05/02/2018	15/02/2018	10 days	NS	NO GROWTH	NO GROWTH		P	R
121	589678	56	F	JEJUNALPERFORATION	07/02/2018	07/02/2018	19/02/2018	12 days	PI	1200 cfu/ml	1100cfu/ml	E.COLI	P	R
122	524368	28	F	PREPYLORICPERFORATION	14/02/2018	14/02/2018	26/02/2018	12 days	PI	NO GROWTH	NO GROWTH		A	R
123	589641	56	M	DUODENALPERFORATION	15/02/2018	15/02/2018	05/03/2018	18 days	NS	1600 cfu/ml	1400 cfu/ml	ENTEROCOCCI	P	R
124	569123	54	M	DUODENALPERFORATION	16/02/2018	16/02/2018	09/03/2018	21 days	PI	1800CFU/ML	1600CFU/ML	E.COLI	P	R
125	514789	56	F	DUODENALPERFORATION	17/02/2018	17/02/2018	27/02/2018	10 days	NS	NO GROWTH	NO GROWTH		A	R
126	563951	54	F	PREPYLORICPERFORATION	19/02/2018	19/02/2018	28/02/2018	9 days	PI	NO GROWTH	NO GROWTH		A	R
127	546987	58	F	DUODENALPERFORATION	22/02/2018	22/02/2018	14/03/2018	20 days	NS	1700CFU/ML	1600CFU/ML	E.COLI	P	R
128	568231	45	M	DUODENALPERFORATION	24/02/2018	24/02/2018	06/03/2018	10 days	PI	NO GROWTH	NO GROWTH		A	R
129	514785	52	F	DUODENALPERFORATION	26/02/2018	26/02/2018	08/03/2018	10 days	NS	NO GROWTH	NO GROWTH		A	R
130	546392	54	F	PREPYLORICPERFORATION	28/02/2018	28/02/2018	22/03/2018	22 days	PI	1800CFU/ML	1600CFU/ML	ENTEROCOCCI	P	R
131	562311	27	F	APPENDICULARPERFORATION	01/03/2018	01/03/2018	12/03/2018	11 days	NS	NO GROWTH	NO GROWTH		A	R
132	564487	40	F	DUODENALPERFORATION	02/03/2018	02/03/2018	17/03/2018	15 days	PI	NO GROWTH	NO GROWTH		P	R
133	524789	58	M	PREPYLORICPERFORATION	04/03/2018	04/03/2018	27/03/2018	23 DAYS	NS	1800cfu/ml	1600CFU/ML	E.COLI	P	R
134	547123	53	M	DUODENALPERFORATION	06/03/2018	06/03/2018	19/03/2018	13 DAYS	PI	NO GROWTH	NO GROWTH		A	R
135	569741	56	M	DUODENALPERFORATION	07/03/2018	07/03/2018	18/03/2018	11 days	NS	NO GROWTH	NO GROWTH		A	R
136	547896	45	M	PREPYLORICPERFORATION	10/03/2018	10/03/2018	21/03/2018	11 days	PI	NO GROWTH	NO GROWTH		A	R
137	584766	56	M	DUODENALPERFORATION	12/03/2018	12/03/2018	22/03/2018	10 days	NS	NO GROWTH	NO GROWTH		A	R
138	532178	54	F	DUODENALPERFORATION	15/03/2018	25/03/2018	28/03/2018	23 DAYS	PI	1800CFU/ML	1700cfu/ml	E.COLI	P	R
139	569321	56	M	DUODENALPERFORATION	17/03/2018	17/03/2018	27/03/2018	10 days	NS	NO GROWTH	NO GROWTH		A	R
140	561247	48	M	DUODENALPERFORATION	18/03/2018	18/03/2018	03/04/2018	15 days	PI	1400 cfu/ml	1300 cfu/ml	E.COLI	P	R
141	578325	58	M	DUODENALPERFORATION	20/03/2018	20/03/2018	13/04/2018	23 DAYS	NS	1800CFU/ML	1600CFU/ML	ENTEROCOCCI	P	R
142	514789	44	M	DUODENALPERFORATION	21/03/2018	21/03/2018	06/04/2018	15 DAYS	PI	NO GROWTH	NO GROWTH		A	R
143	569871	62	F	PREPYLORICPERFORATION	24/03/2018	24/03/2018	05/04/2018	11 days	NS	NO GROWTH	NO GROWTH		A	R
144	518963	56	M	DUODENALPERFORATION	28/03/2018	28/03/2018	11/04/2018	13 DAYS	PI	NO GROWTH	NO GROWTH		A	R
145	587463	58	M	DUODENALPERFORATION	29/03/2018	29/03/2018	22/04/2018	23 DAYS	NS	1800CFU/ML	1700cfu/ml	E.COLI	P	R
146	589602	27	M	APPENDICULARPERFORATION	30/03/2018	30/03/2018	20/04/2018	20 DAYS	PI	NO GROWTH	NO GROWTH		P	R

147	584632	54	M	DUODENALPERFORATION	01/04/2018	01/04/2018	22/04/2018	20 days	NS	1800CFU/ML	1700cfu/ml	E.COLI	P	R
148	596842	44	F	PREPYLORICPERFORATION	04/04/2018	04/04/2018	14/04/2018	10 days	PI	NO GROWTH	NO GROWTH		A	R
149	578421	59	M	PREPYLORICPERFORATION	05/04/2018	05/04/2018	30/04/2018	25 DAYS	NS	1900cfu/ml	1700CFU/ML	ENTEROCOCCI	P	R
150	546932	60	M	PREPYLORICPERFORATION	07/04/2018	07/04/2018	20/04/2018	13 DAYS	PI	NO GROWTH	NO GROWTH		A	R
151	523641	54	M	PREPYLORICPERFORATION	08/04/2018	08/04/2018	28/04/2018	20 DAYS	NS	1700cfu/ml	1600CFU/ML	ENTEROCOCCI	P	R
152	536452	58	M	PREPYLORICPERFORATION	10/04/2018	10/04/2018	20/04/2018	10 days	PI	NO GROWTH	NO GROWTH		A	R
153	596478	56	F	ILEALPERFORATION	12/04/2018	12/04/2018	05/05/2018	23 days	NS	NO GROWTH	NO GROWTH		A	R
154	569412	54	M	APPENDICULARPERFORATION	21/04/2018	21/04/2018	03/05/2018	13 DAYS	PI	NO GROWTH	NO GROWTH		P	R
155	589615	53	M	ILEALPERFORATION	24/04/2018	24/04/2018	03/05/2018	10 days	NS	NO GROWTH	NO GROWTH		A	R
156	596448	44	M	DUODENALPERFORATION	30/04/2018	30/04/2018	19/05/2018	20 days	PI	1900CFU/ML	1700cfu/ml	E.COLI	P	R
157	546321	54	F	APPENDICULARPERFORATION	04/05/2018	04/05/2018	17/05/2018	13 DAYS	NS	NO GROWTH	NO GROWTH		A	R
158	569874	50	M	DUODENALPERFORATION	09/05/2018	09/05/2018	25/02/2018	16 days	PI	NO GROWTH	NO GROWTH		P	R
159	563245	50	M	ILEALPERFORATION	18/05/2018	18/05/2018	30/05/2018	12 days	NS	NO GROWTH	NO GROWTH		A	R
160	564565	55	M	APPENDICULARPERFORATION	19/05/2018	19/05/2018	07/06/2018	18 DAYS	PI	NO GROWTH	NO GROWTH		P	R
161	550024	45	F	ILEALPERFORATION	22/05/2018	22/05/2018	05/06/2018	13 days	NS	NO GROWTH	NO GROWTH		A	R
162	554632	55	M	ILEALPERFORATION	28/05/2018	28/05/2018	21/06/2018	23 days	PI	1800CFU/ML	1700cfu/ml	E.COLI	P	R
163	512478	59	M	PREPYLORICPERFORATION	03/06/2018	03/06/2018	16/06/2018	13 DAYS	NS	NO GROWTH	NO GROWTH		A	R
164	569325	36	M	APPENDICULARPERFORATION	06/06/2018	06/06/2018	28/06/2018	22 days	PI	1900CFU/ML	1700cfu/ml	ENTEROCOCCI	P	R
165	547821	56	M	PREPYLORICPERFORATION	09/06/2018	09/06/2018	19/06/2018	10 days	NS	NO GROWTH	NO GROWTH		A	R
166	536214	54	F	PREPYLORICPERFORATION	14/06/2018	14/06/2018	04/07/2018	20 days	PI	1900CFU/ML	1700cfu/ml	ENTEROCOCCI	P	R
167	584682	53	M	APPENDICULARPERFORATION	18/06/2018	18/06/2018	02/07/2018	15 DAYS	NS	NO GROWTH	NO GROWTH		A	R
168	541236	54	M	DUODENALPERFORATION	21/06/2018	21/06/2018	06/07/2018	17 DAYS	PI	NO GROWTH	NO GROWTH		P	R
169	540023	28	M	PREPYLORICPERFORATION	24/06/2018	24/06/2018	07/07/2018	14 DAYS	NS	NO GROWTH	NOGROWTH		P	R
170	536998	45	F	APPENDICULARPERFORATION	26/06/2018	26/06/2018	07/07/2018	12 DAYS	PI	NO GROWTH	NO GROWTH		A	R
171	542286	54	F	DUODENALPERFORATION	29/06/2018	29/06/2018	09/07/2018	15 DAYS	NS	NO GROWTH	NO GROWTH		P	R
172	564112	55	M	PREPYLORICPERFORATION	31-06-2018	31-06-2018	20/07/2018	20 DAYS	PI	1800CFU/ML	1600CFU/ML	E.COLI	P	R
173	631854	58	M	ILEALPERFORATION	27/08/2018	27/08/2018	15/09/2018	15 DAYS	NS	NO GROWTH	NO GROWTH		A	R
174	631851	30	M	DUODENALPERFORATION	25/09/2018	25/09/2018	05/10/2018	10 DAYS	PI	NO GROWTH	NO GROWTH		A	R