"PROSPECTIVE STUDY OF FACTORS AFFECTING OUTCOME IN GASTROINTESTINAL PERFORATIONS IN RURAL SETUP"

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M. S. GENERAL SURGERY

By

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INTRODUCTION

Gastrointestinal perforation is a hole that develops through the entire wall of the oesophagus stomach, small intestine, large bowel, rectum or gallbladder. Perforation of the intestine leads to leakage of intestinal contents into the abdominal cavity resulting in the inflammation of peritoneum called peritonitis. It is often severe because of faecal contamination and overwhelming sepsis resulting in high morbidity and mortality in colonic perforation. Treatment depends on the underlying cause. The condition is surgical emergency and surgical intervention is nearly always required in form of exploratory laparotomy and closure of perforation with peritoneal wash.

Underlying causes include peptic ulcer, appendicitis, gastrointestinal cancer, diverticulitis, superior mesenteric artery syndrome, trauma and ascariasis. In exceptionally rare cases, any intervention in upper and lower gasterointestinal tract as in case of endoscopy or colonoscopy can lead to respective hollow viscous perforation.Similar event can also be noticed in as seen in sexual exploitation. It may also be due to foreign body such as ingested bone (e.g. a fish bone).

Perforation peritonitis is the most common surgical emergency in India[1]. Despite advances in surgical techniques, antimicrobial therapy and intensive care support, management of peritonitis continues to be highly demanding, difficult and complex. The spectrum of etiology of perforation continues to be different from that of western countries[2] and there is paucity of data from India regarding its etiology, prognostic indicators, and morbidity and mortality patterns[3].

The perforations of proximal gastrointestinal tract were six times as common as perforations of distal gastrointestinal tract as has been noted in earlier studies from India[2], which is in sharp contrast to studies from developed countries like United States, Greece and Japan which revealed that distal gastrointestinal tract perforations were more common[1].

In adults, perforations of peptic ulcer disease were a common cause of morbidity and mortality with acute abdomen until the latter half of the 20th century. The rate has fallen in parallel with the general decline in the prevalence of peptic ulcer disease[4]. Duodenal ulcer perforations are 2-3 times more common than are gastric ulcer perforations[4].

Outcome is improved with early diagnosis and treatment. The factors which increase the risk of morbidity and mortality are advanced age, presence of preexisting underlying disease, malnutrition, the nature of the primary cause of bowel perforation and appearance of complications.

The mainstay of treatment for bowel perforation is surgery. Endoscopic, laparoscopic and laparoscopic-assisted procedures are now being increasingly performed instead of conventional laparotomy[5, 6].

AIMS AND OBJECTIVES OF STUDY:

- To determine the demographic factors in patients presenting with spontaneous gastrointestinal perforations.
- To study the outcome and post operative complications in patients undergoing surgery for gastrointestinal perforations.
- To determine risk factors responsible for gastrointestinal perforations in rural populations.

MATERIALS AND METHODS

Patients aged more than 18 years presenting with spontaneous gastrointestinal perforations to surgical department of R L Jalappa Hospital Research centre. This Prospective study with minimum of 100 patients are considered from December 2008 to August 2010.Related data were collected by predesigned or pretested proforma and data so obtained were compiled and analysed.

A. INCLUSION CRITERIA

Patients aged ≥ 18 years presenting with spontaneous gastrointestinal perforations.

B. EXCLUSION CRITERIA

- Patients aged < 18 years presenting with spontaneous gastrointestinal perforations.
- Peritonitis secondary to penetrating injuries of abdomen like stab injuries, bull gore and gunshot injuries.
- Peritonitis secondary to blunt trauma to the abdomen.

STUDY TOOLS:

A. CLINICAL EXAMINATION :

- Detailed history with regard to the age of the patient with profession, address, habit, type of diet, co-morbid conditions, time of presentation, duration of illness and presence or absence of shock.

- On per abdomen examination – distention, tenderness, guarding, rigidity, obliteration of liver dullness, evidence of free fluid in the peritoneal cavity.

  Per rectal examination – Sphincter tone, roomy rectum and deep seated tenderness.

- Other systemic examinations – cardiovascular system, respiratory system and nervous system examinations will be carried out.

LABORATORY ASSESSMENT :

- Complete blood cell (CBC) count
  - Parameters suggestive of infection (eg, leukocytosis) - Leukocytosis may be absent in elderly patients.
  - Elevated packed blood cell volume - This suggests a shift of intravascular fluid.
  - ESR
  - Blood grouping RH typing
  - Bleeding time and clotting time
  - Blood urea serum creatinine

- Blood glucose
- Liver function test
- Blood culture for aerobic and anaerobic organisms.
- Widal test for enteric fever

- Peritoneal fluid for staining and culture sensitivity :

  Obtained by: 1.Ultra sounded aspiration

  2. During laparotomy

- Urine routine
- ECG
- Liver function and renal function tests - Findings may be within reference ranges (or nearly so) if no preexisting disorder is present.
- Serum amylase : It is not specific for pancreatic disorder but can also be raised in non pancreatic disorder like in bowel perforation, ruptured ectopic, appendicular perforation with or without abcess, salivary gland disorders where false positive results are noted.

IMAGING STUDIES

- Erect X- ray abdomen
- Lateral decubitus X- ray
- Chest X-ray
- USG of abdomen and pelvis

With **Informed Consent** along with fitness for surgery, patients were taken up for laparotomy and peritoneal lavage and closure of perforation, resection anastomosis and colostomy were dealt depending on the cases and post operatively necessary antibiotics, analgesics and necessary care were provided.

Patient were followed up post operatively for three months for any complications like wound infection, wound dehiscence, fistula, burst abdomen, post operative adhesive pain abdomen and advised with respect to occurrence and avoidance of incisional hernia .

STATISTICAL ANALYSIS: Test of significance will be employed wherever needed. Significant value of $P < 0.05$ will be considered as significant.

The data obtained will be compiled and analyzed.

STATUS OF ETHICAL CLEARANCE: Yes, Ethical clearance was obtained from the institutional ethical review committee of Sri Devaraj Urs Medical college.

HISTORICAL REVIEW

Lau and Leow have indicated that perforated peptic ulcer was clinically recognized by 1799, but the first successful surgical management of gastric ulcer was by Ludwig Heusner in Germany in 1892. In 1894, Henry Percy Dean from London was the first surgeon to report successful repair of a perforated duodenal ulcer[7].

Rawlison is credited with the first published report in 1727 of a perforated ulcer which happened to be gastric perforation. The first published report of a perforated duodenal ulcer was by Hambergeri in 1746[8].

Partial gastrectomy, although performed for perforated gastric ulcer as early as 1892, did not become a popular treatment until the 1940s.Definitive surgical management of peptic ulcer has drastically come down due to introduction of proton pump inhibitors and other acid reducing agents. But complications secondary to ulcer are still encountered and needs surgical management. Currently, in patients with gastric perforation, simple closure of perforated ulcers is more commonly performed than is gastric resection[9].

Surgery for the treatment of typhoid perforation began in the late 1800's. After the work of Finney and Cushing, surgery became the standard treatment[10]. First published report concerning surgery of intestinal tuberculosis was that of Hartman and Piteit in 1891[11].

Reginald Fitz published his paper on perforation of appendix associated with peritonitis in 1886; the modern surgical treatment of the diseased appendix thus began[12].

Colonic perforation secondary to tumor occurs in two different settings.Either a transmural tumor perforates itself, or the proximal colon becomes over distended,particularly in the case of competent illeocecal valve.Both conditions may result in diffuse feacal peritonitis with significant morbidity and mortality.In addition, tumor perforation results in spillage of tumor cells and thus has to be considered as stage IV tumor.Surgical management is indicated in every case and requires not only addrerssing the site of colonic perforation but also removing of tumor in a oncologically correct fashion[9].

EMBRYOLOGY

During fourth week the embryo undergoes the process of embryonic folding, transforming the flat endoderm into a gut tube. Initially the gut consists of cranial and caudal blind ending tubes, the foregut and hind gut respectively, which are separated by the future mid gut. The mid gut remains open to the yolk sac, which is later reduced to the slender vitelline duct[13].

At the end of fourth week, almost entire abdominal gut tube, that is from abdominal esophagus up to the superior end of the developing cloaca hangs suspended by the dorsal mesentery. Leaving the region of developing stomach rest of the coelomic cavities in the lateral plate mesoderm coalesce on either side of the germ disc to form a, single continuous peritoneal cavity. The mesentery in the region of the stomach remains attached to the ventral body wall by thick septum transversum which thins to form ventral mesentery by fifth week. Some of the parts of gut tube which are initially suspended by the mesentery, later become fused to the body wall, and are said to secondarily retroperitoneal[13].

STOMACH:

The stomach first becomes apparent in early part of the 4th week as fusiform dilatation of foregut, just distal to the esophagus. Differential growth of dorsal wall of stomach faster than ventral wall results in the formation of greater curvature of stomach where as lesser curvature from the slow growing ventral wall. Further differential expansion of greater curvature results in formation of fundus and cardiac incisures.

The stomach undergoes 90-degree rotation during seventh & eighth weeks around craniocaudal axis so that the greater curvature lies to the left and the lesser curvature leis to the right. Hence left vagus nerve initially innervating the left side of the stomach now innervates the anterior wall, similarly right vagus nerve innervates posterior wall. The stomach also rotates slightly around a ventrodorsal axis so that greater curvature faces caudally and lesser curvature slightly cranially[14].

DUODENUM:

Superior part and upper half of the descending part of duodenum develops from the foregut where as rest of the duodenum develops from proximal part of the mid gut. The duodenal loop is initially surrounded by the mesentery, which fuses with posterior peritoneum making the

duodenum a retroperitoneal viscus. Differential growth and rotation of the stomach makes duodenum to take the shape of 'C'[14].

JEJUNUM AND ILEUM:

Jejunum and most of the ileum are derived from the pre arterial segment of mid gut loop. The terminal portion the ileum is derived from the post arterial segment of mid gut loop, proximal to the caecal bud. Rapid differential growth of ileum produces a primary intestinal loop that herniates into the umbilicus which forms the bases of physiological umbilical hernia[13].

CAECUM AND APPENDIX:

Caecum and appendix are derived from the enlargement of the diverticulum arising from the post arterial segment of the mid gut. Proximal part of the diverticulum grows faster to form caecum and slow growing distal part remains narrow to form appendix.

COLON AND RECTUM:

Entire ascending colon and right two thirds of transverse colon is derived from the post arterial segment of mid gut loop. Left one third of transverse colon and entire descending colon are derived from the hind gut. Rectum is derived dorsal subdivision of the cloaca.

ROTATION OF THE GUT:

During early part of the sixth week, the continuing elongation of the mid gut combined with pressure due to the dramatic growth of liver forces the primary intestinal loop to herniate into the umbilicus[14]. The herniated primary intestinal loop undergoes an initial 90-degree counter clock wise rotation with horizontal orientation of the bowel segments. The pre arterial segment comes to lie on the right side and post arterial segment on the left[13].

The pre arterial segment undergoes rapid growth resulting in series of folds called jejunal –ileal loops. Now these jejunal –ileal loops starts returning to the abdominal cavity. As they do so, the mid gut loop undergoes further 180-degrees anticlockwise rotation. Returned jejunal –ileal loops pass behind the superior mesenteric artery into the left half of abdominal cavity.

Finally post arterial segment of the mid gut loop returns to the abdominal cavity with anticlockwise rotation with the result the transverse colon lies anterior to the superior mesenteric artery and caecum on the right side. In the early stages the caecum lies inferior to the liver. Gradually the differential growth of colon results in iliac position of caecum and development of ascending colon.

FIXATION OF THE GUT:

Before the rotation of the gut all parts of small and large intestine have mesenteries by which they are suspended from the posterior abdominal wall. After completion of rotation of gut the duodenum, ascending colon , descending colon & rectum become secondarily retroperitoneal by the fusion of their mesenteries with posterior abdominal wall[14].

ANATOMY

Eosophagus one centimeter of which lies within the abdominal cavity[15](abdominal eosophagus),stomach, small and large intestine, rectum and part of anal canal forms the gastrointestinal system.

ABDOMINAL PART OF OESOPHAGUS:

The abdominal part of oesophagus measures 1.25 cms and it enters the abdominal cavity through the esophageal opening in the diaphragm at the level of 10^{th} thoracic vertebra. It forms the important site for the portosystemic anastomosis[15].

STOMACH:

Gaster or venter are the synonyms for the stomach[15]. It is muscular bag forming the widest and most distensible part of digestive tract.

Lower end of esophagus end in the stomach, where as stomach continues further into the duodenum. It occupies the epigastric, umbilical and left hypochondriac regions. The capacity of the stomach varies from one ounce at birth, 1 litre at puberty to 1.5 to 2 litres or more in adults.

From surgical view point the gaster is composed of two gastric units or systems, proximal gastric unit and distal gastric unit. Proximal gastric unit contains proximal stomach, distal esophagus & esophageal hiatus of diaphragm. The distal gastric unit includes gastric antrum, pylorus & first part of duodenum[15].

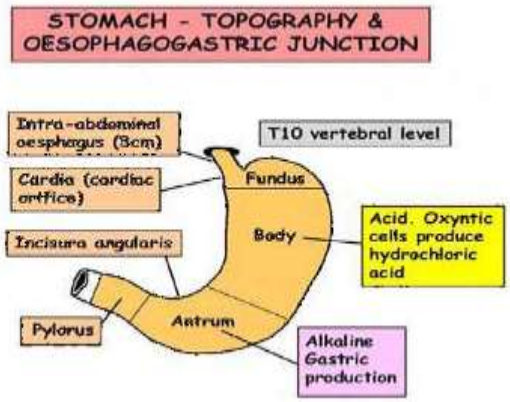


Figure No. 1

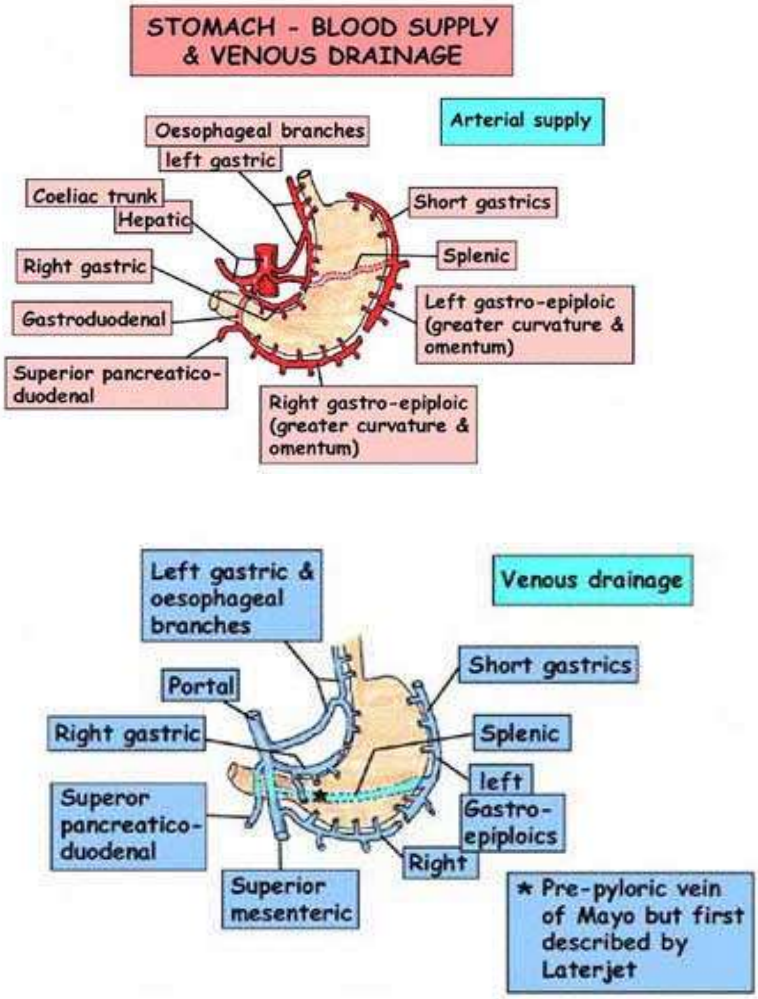


Figure No.2

The stomach has two orifices, two curvatures and two surfaces.

Cardiac orifice is joined by the lower end of the oesophagus situated at the level of 11th thoracic vertebra. There is evidence of physiological sphincter at this site, but cannot be demonstrated anatomically.

Pyloric orifice opens into the duodenum at the level of 1st lumbar vertebrae.

Stomach has two curvatures, lesser curvature and greater curvature.

Lesser curvature is concave and forms the right border of the stomach. It gives attachment to the lesser omentum. A sharp angular depression in the lesser curvature of the stomach at the junction of the body with the pyloric canal is called as incisura angularis or angular notch.

Greater curvature of the stomach is convex and forms the left border of the stomach its gives attachment to the greater omentum, the gastrosplenic ligament & gastrophrenic ligament.

Two surfaces of the stomach are anterior or anterosuperior and posterior or posteroinferior.

Relations of stomach (figure no.5)

Blood Supply: Stomach is supplied by

1. Left gastric artery (branch of celiac trunk)
2. Right gastric artery (branch of common hepatic artery)
3. Right gastroepiploic (branch of gastroduodenal artery)

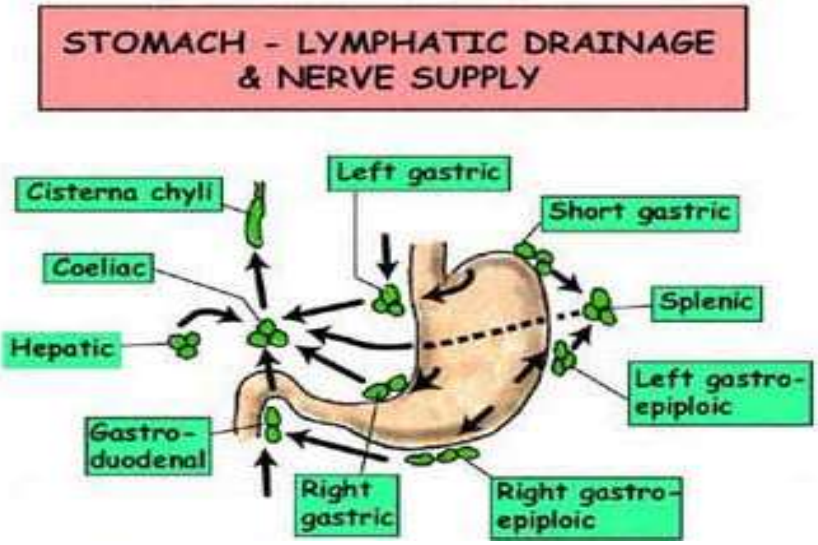


Figure No. 3

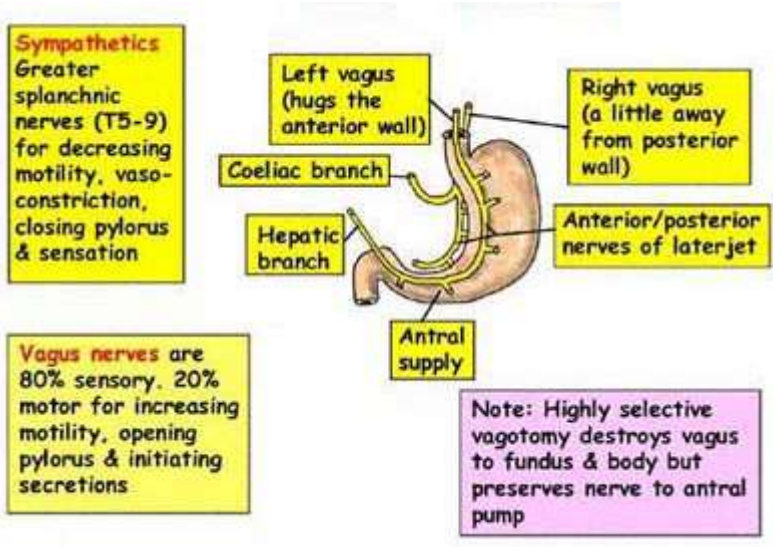


Figure No. 4

4 Left gastroepiploic(branch of splenic artery)

5 Five to seven short gastric arteries (branch of splenic artery)

The veins for all practical purposes follow the arteries and drain into the portal , superior mesenteric & splenic veins.

Lymphatic Drainage:

The lymphatic drainage of stomach is divided into four zones

Zone I: Pancreaticosplenic area, drains into the pancreaticosplenic nodes lying along the splenic artery.

Zone II: Drains into the left gastric nodes lying along the artery of the same.

Zone III: Drains into the right gastroepiploic nodes that lie along the artery of the same.

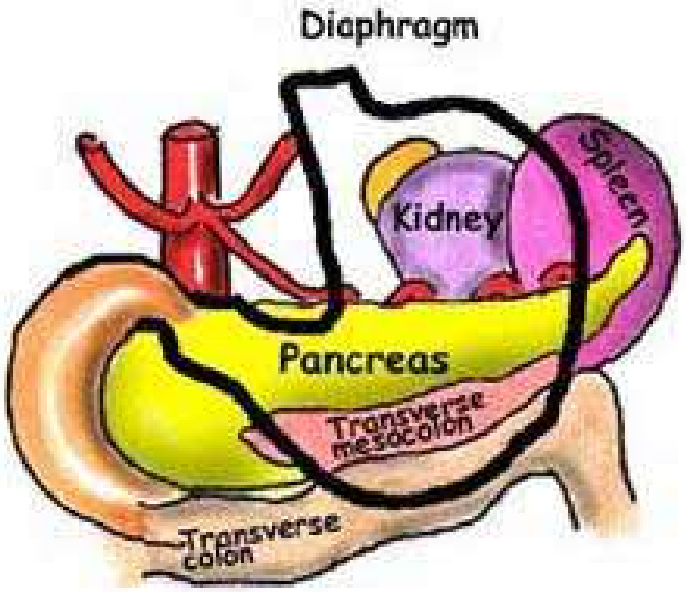
Zone IV: drains into the pyloric, hepatic & left gastric nodes and then all theses nodes passes to the celiac nodes.

Nerve Supply:

The venter is supplied by both **sympathetic** and **parasympathetic** nerves.

Sympathetic nerve supply comes from the 5^{th} to 10^{th} thoracic segments of spinal cord via greater splanchnic nerves, celiac and hepatic plexuses. These nerves are vasomotor, motor to the pyloric sphincter (but inhibitory to the rest of the gastric musculature) and are the chief pathway for pain sensation from the stomach.

STOMACH - RELATIONS



ANTERIOR
Abdominal wall
Left costal margin
Diaphragm
Left lobe of liver

SUPERIOR
Left dome of diaphragm

POSTERIOR
Lesser sac
Pancreas
Transverse mesocolon
Transverse colon
Left kidney/suprarenal gland
Spleen/splenic artery

Figure No. 5

Parasympathetic nerves are derived from the vagi, though esophageal plexus, which forms two main trunks, anterior and posterior. Anterior trunk is mainly derived from the left vagal fibers where as posterior trunk is by right vagal fibers.

Anterior trunk divides in to anterior gastric division (anterior division of Latarjet) & hepatic division supplying the anterior surface of body & fundus, pylorus & pyloric antrum. Posterior trunk divides into posterior gastric division (posterior division of Latarjet) supplying the posterior aspect of body, fundus and pylorus of stomach and celiac division for the celiac plexus. Parasympathetic nerves are motor and secretomotor to the stomach[15].

SMALL INTESTINE:

The small intestine extends from the pylorus to the ileocaecal junction. The small intestine measures 6 meters in length and is divided into fixed retroperitoneal part and mobile peritoneal part.

DUODENUM:

It is Greek word dudekadaktulos, meaning twelve fingers. It is the shortest, widest and most fixed part of the small intestine. It is curved around the head of pancreas in the form of letter 'C'. Duodenum measures 20 to 25 cms in the length and consists of four parts[16]

i. Superior part or first part, measuring about 5.0 cms.
ii. Second or descending part, 7.5 cms
iii. Third or horizontal part, 10 cms
iv. Fourth or ascending part , 2.5 cms

The suspensory muscle or ligament of Treitz is a fibromuscular band arising from the right crus of diaphragm and inserts into the upper surface of duodenojejunal flexure.

Blood Supply:

Duodenum is mainly supplied by the superior and inferior pancreaticoduodenal arteries. Additional blood supply is from right gastric artery, supraduodenal artery of Wilkie which is branch of hepatic artery, retroduodenal branches of gastroduodenal artery & some branches from right gastroepiploic arteries.

Veins from the duodenum drain into the splenic, superior mesenteric and portal veins.

Nerve Supply:

Sympathetic nerves are derived from the T9toT10 spinal segments and parasympathetic nerves from the vagus, through celiac plexus.

JEJUNUM AND ILEUM:

The jejunum and ileum is slung from the posterior abdominal wall by the mesentery of the small intestines and, therefore, is extremely mobile. The mesentery of the small intestine arises from the root of the mesentery which extends from the duodenojejunal flexure to the ileocaecal junction.

The jejunum is about 2.5m (8ft) long and passes imperceptibly into the ileum, which is about 4m (12ft) long. This part of the small intestine occupies a central position in the abdominal cavity, below the liver and the stomach, and behind the transverse mesocolon, the transverse colon and the greater omentum. The lowest coils of the intestine lie in the pelvic cavity. The jejunum is thicker compared to the ileum. The ileum occupies the hypogastric and pelvic position.

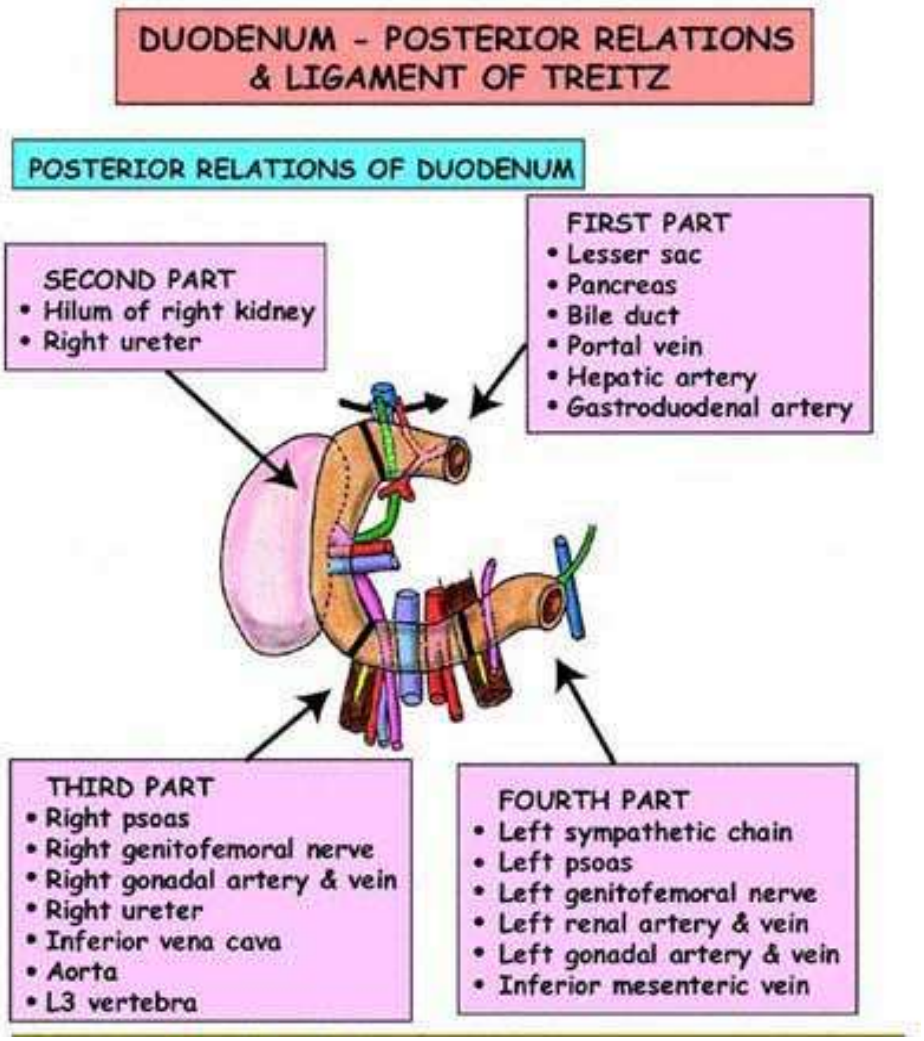

DUODENUM - POSTERIOR RELATIONS & LIGAMENT OF TREITZ
POSTERIOR RELATIONS OF DUODENUM
SECOND PART
• Hilum of right kidney
• Right ureter
FIRST PART
• Lesser sac
• Pancreas
• Bile duct
• Portal vein
• Hepatic artery
• Gastroduodenal artery
THIRD PART
• Right psoas
• Right genitofemoral nerve
• Right gonadal artery & vein
• Right ureter
• Inferior vena cava
• Aorta
• L3 vertebra
FOURTH PART
• Left sympathetic chain
• Left psoas
• Left genitofemoral nerve
• Left renal artery & vein
• Left gonadal artery & vein
• Inferior mesenteric vein

Figure No. 6

Blood supply:

The ileum and jejunum are supplied by the superior mesenteric artery and its intestinal branches. The branches are rather special in that small arcades are formed and from the arcades, the straight vessels, vasa recta arise and supply the intestine. The veins follow the respective arteries.

Lymphatic drainage:

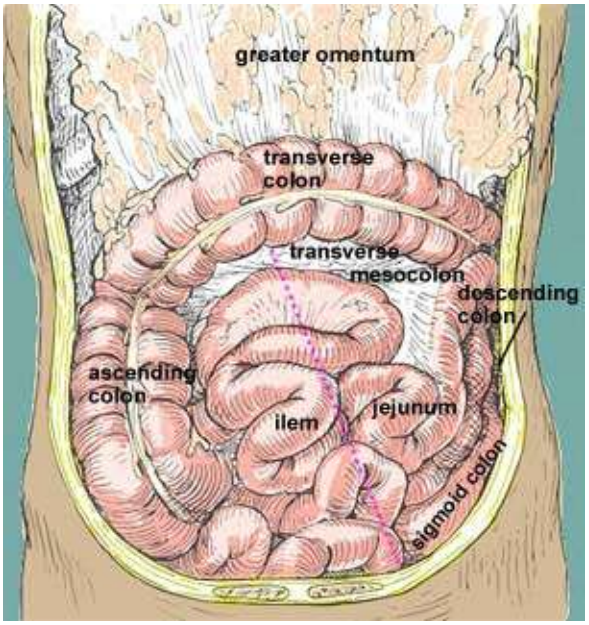
The lacteals in the intestinal villi empty their milk-like fluid into a plexus of lymph vessels in the walls of the jejunum and ileum. These lymph vessels pass between the two layers of mesentery to the mesenteric lymph nodes which drain into the lymph nodes present in front of the aorta at the origin of the superior mesenteric artery.

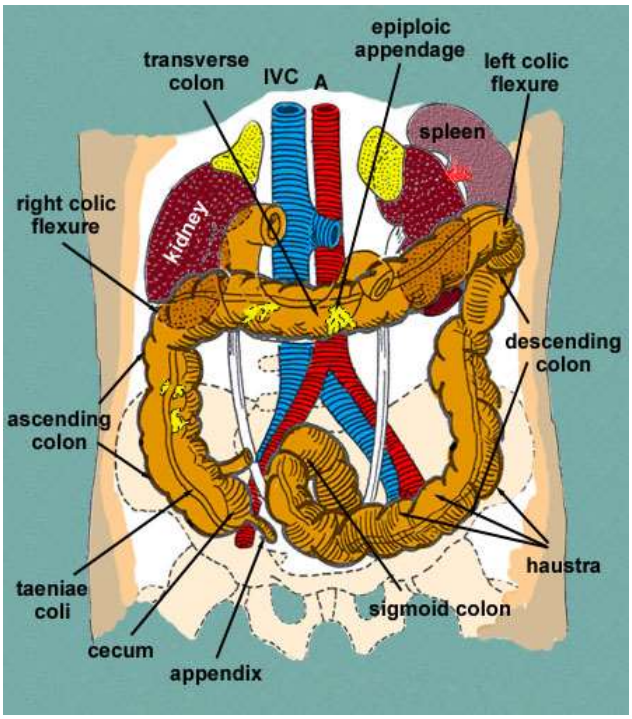
Nerve supply:

The innervation is through the superior mesenteric plexus extensions along the arteries. The sympathetic supply is from the greater splanchnic and lesser splanchnic nerves. The parasympathetic supply is from the posterior vagal trunk via the coeliac plexus.

LARGE INTESTINE:

The large intestine extends from the ileocaecal junction to the anus and is about 1.5m long[15]. On the surface there are bands of longitudinal muscle fibers called taeniae coli, each about 5mm wide. There are three bands called as taeniae coli, they start at the base of the appendix and extend from the caecum to the rectum. They are named as taeniae libera, taeniae mesocolica & taeniae omentalis. Along the sides of the taeniae there are tags of peritoneum filled with fat,


greater omentum
transverse colon
transverse mesocolon
descending colon
ascending colon
ilem
jejunum
sigmoid colon


epiploic appendage
transverse colon
IVC
A
left colic flexure
spleen
right colic flexure
kidney
descending colon
ascending colon
haustra
taeniae coli
sigmoid colon
cecum
appendix

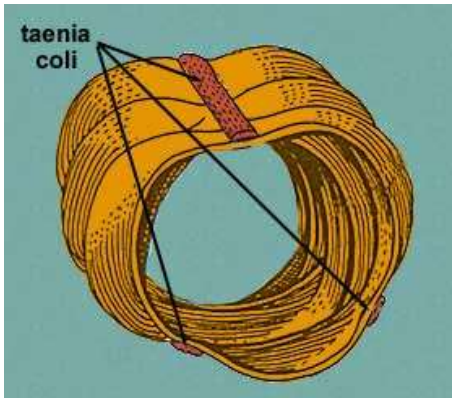

taenia coli

Figure No. 7

called epiploic appendages (or appendices epiploicae). The sacculations, called haustra, are characteristic features of the large intestine, and distinguish it from the rest of the intestinal tract.

The large intestine consists of caecum, ascending colon, transverse colon, descending colon, sigmoid colon, rectum, , anal canal & anus.

CAECUM:

The caecum is about 6cm long and is a blind *cul-de-sac* which lies in the right iliac fossa. It is the part of the colon below the opening of the ileum into the colon. The caecum lies immediately behind the abdominal wall and greater omentum. There is frequently a peritoneal recess behind the caecum called the retrocaecal recess and the appendix is sometimes hiding within this recess and may extend as far superiorly as the liver. The arterial supply of the caecum is derived from the ileocaecal artery. The veins drain into the superior mesenteric vein.

VERMIFORM APPENDIX:

Hanging off the caecum is the vermiform appendix which opens into the caecum about 2cm below the ileocaecal opening. The average length of the appendix is about 10cm and varies from 2 to 20 cms. Although the base of the appendix is fixed, the tip can point in different directions like retrocaecal or retrocolic (65%), pelvic (31%), subcaecal (2.3%) and rarely anterior or posterior to the terminal ileum. It has its own mesentery called the mesoappendix which carries the appendicular artery. Appendicular artery is branch of lower division of ileocolic artery. The appendix drains into the appendicular vein

draining into the superior mesenteric vein. Sympathetic nerve supply is from the T9 to T10 segments and parasympathetic is derived from the vagus nerve. Most of the lymphatic drain into the appendicular node which further drains into the ileocolic nodes.

ASCENDING COLON:

The ascending colon various from 12 to 20 cm in length[15]. It ascends on the right side of the abdominal cavity from the caecum to the right lobe of the liver where it turns to the left at the right colic (hepatic) flexure. It usually has no mesentery and lies retroperitoneally along the right side of the posterior abdominal wall. On the lateral side of the ascending colon, the peritoneum forms a trench or groove called the right paracolic gutter. The depth of this groove depends on how much has the ascending colon contains. . Nerve supply is derived from the coeliac and superior mesenteric ganglia.

TRANSVERSE COLON:

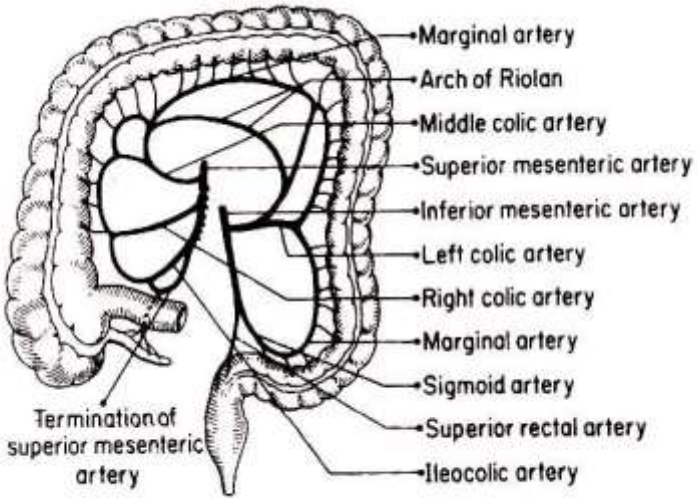
The transverse colon, about 45 cm in length, is the largest and most mobile part of the large intestine. It crosses the abdomen from the right colic flexure to the left colic flexure, where it bends inferiorly to become the descending colon. Between these 2 colic flexures, the transverse colon is freely movable and forms a loop that is directed inferiorly and anteriorly. The transverse colon has a mesentery known as the transverse mesocolon, which is connected to the inferior border of the pancreas and to the greater omentum that covers it anteriorly. Because it is freely movable, the transverse colon is extremely variable in position. Nerve supply is through the both superior and inferior mesenteric plexus.

DESCENDING COLON:

This part of the large intestine measures 22 to 30 cm in length. It descends from the left colic flexure into the left iliac fossa, where it is continuous with the sigmoid colon. The caliber of the descending colon is considerably smaller than that of the ascending colon. It is usually has no mesentery and lies retroperitoneally along the left side of the posterior abdominal wall. Its posterior surface is attached to the posterior abdominal wall like the ascending colon. It receives its sympathetic supply from the lumbar part of the sympathetic trunk and the superior hypogastric plexus by means of plexuses on the branches of the inferior mesenteric artery. The parasympathetic supply is derived from the pelvic splanchnic nerves.

Arterial supply to the colon comes from superior mesenteric artery which are the branches of abdominal aorta, which communicate in water shed area in splenic flexure (artery of Drummond).Right anastomotic branch from inferior mesenteric artery passes through the transverse mesocolon to reach similar branch from middle colic artery to form arch of Riolan. And supplying adjacent colon.With significant degree of anatomic variation,the major vascular stops to the colonic segment consists of the illeocecal and right colic (last branch of superior mesenteric artery), middle colic artery (second branch of superior mesenteric artery), left colic artery (first branch of inferior mesenteric artery).The venous supply peripherally follows the arterial branches but more centrally divides into superior mesenteric vein and inferior mesenteric vein which connect to portal system at two separate levels.

BLOOD SUPPLY AND LYMPHATIC DRAINAGE



Blood supply of the colon showing the marginal artery and the arch of Riolan.

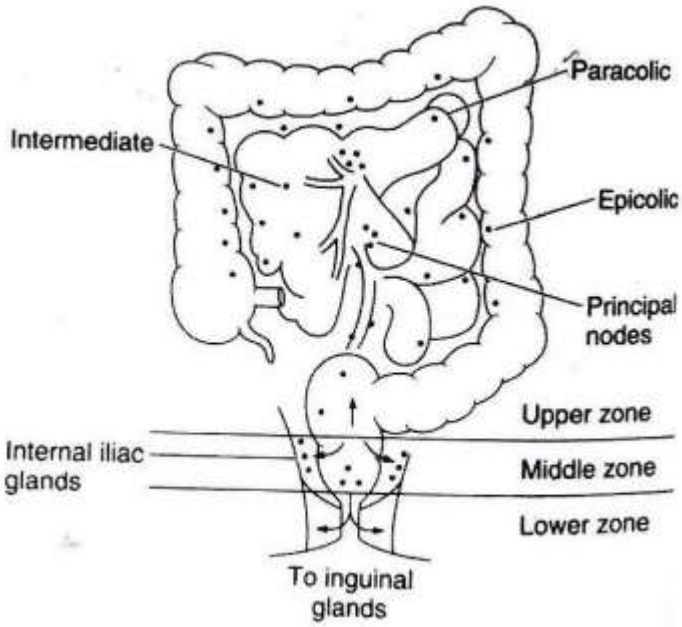


Figure No. 8

Lymphatic drainage starts with lymphatic follicles in the colonic submucosa further passing through colonic muscle layer thus draining to epicolic modes, and continues to the paracolic lymph nodes that follow the blood vessels to the bowel, along the major arteries to the principal lymph nodes at the level of arterial runoff from the aorta.These lymph node groups consist of celiac, the superior mesenteric and the inferior mesenteric group of lymph nodes.

SIGMOID COLON:

The sigmoid colon forms a sinuous, S-shaped loop of variable length (usually 40 cm). This is portion of large intestine between the descending colon and rectum. It extends from the pelvic brim to the 3rd segment of the sacrum, where it joins the rectum. The termination of the taeniae coli indicates the beginning of rectum. It usually has a long mesentery, the sigmoid mesocolon and thus has considerable freedom of movement. The sigmoid colon usually occupies the rectovesical pouch in males and the rectouterine pouch in females. The root of its mesentery has a V-shaped attachment, superiorly along the external iliac vessels and inferiorly from the bifurcation of the common iliac vessels to the anterior aspect of the sacrum. The appendices epiploicae (omental appendages) are very long in the sigmoid colon.

Arterial supply is derived from 2 to 3 sigmoid arteries which are branches of the inferior mesenteric artery. The inferior mesenteric vein drains the blood from the sigmoid colon. Lymph passes to the intermediate colic lymph nodes on the branches of the left colic arteries, and from them to the inferior mesenteric lymph nodes. It receives its sympathetic supply from the lumbar part of the sympathetic trunk and the superior hypogastric plexus

by means of plexuses on the branches of the inferior mesenteric artery. The parasympathetic supply is derived from the pelvic splanchnic nerves.

RECTUM:

This is continuous with the sigmoid colon at the mid piece of the sacrum. It has a length of about 12 cm and descends along the sacro-coccygeal concavity as the sacral flexure. It eventually joins the anal canal at the anorectal junction, 2 to 3 cm in front of the coccygeal tip. The bend at this point is known as the perineal flexure of the rectum. The rectum is covered by peritoneum on its anterior surface and sides in the upper 1/3, anterior surface only in the middle 1/3 and is not covered in the lower 1/3. The lower part of the rectum is dilated as the rectal ampulla

ANAL CANAL:

This is about 4 cm long in adults. The upper half of the anal canal is lined by mucosa, which is plum red due to the internal rectal venous plexus. The lower half is lined with stratified squamous non-keratinising epithelium (continuous with the skin of the anus). In this lower half, there are 6 to 7 anal columns. Each column contains a terminal branch of the superior rectal artery and vein, these being largest at the 3, 7 and 11 o'clock positions[15]. Enlargement of the venous terminal branches and anastomoses give rise to internal haemorrhoids. The lower ends of the columns are linked by anal valves, above each of which is an anal sinus. The anal valves together are known as the pectinate line, which is situated opposite the internal anal sphincter. The anal canal extends below the

pectinate line as the pecten, which is bluish in color. The pecten ends inferiorly at the "white line" of Hilton.

GENERAL HISTOLOGY OF GASTROINTESTINAL TRACT:

The wall of the alimentary canal is formed by 4 distinct layers:

1. Mucosa
2. Submucosa
3. Muscularis externa
4. Serosa or adventitia

MUCOSA:

The mucosa of the gastrointestinal tract has a barrier function, secretory function, and an absorptive function. It is composed of epithelium, lamina propria, and a muscularis mucosa. The epithelium differs throughout the alimentary canal, adapting specifically for one or more of these functions. The lamina propria has glands, lymphatic and fenestrated vascular vessels to receive the absorbed substances and an immunologic barrier consisting gut-associated lymphatic tissue (GALT)[16]. The muscularis mucosa has smooth muscle cells in inner circular and outer longitudinal layers.

There are 4 basic mucosal types in the G.I. tract[17]:

1. Protective - stratified squamous epithelium found in the oral cavity, pharynx, oesophagus and anal canal.
2. Secretory - found in the stomach, where the mucosa consists of long, closely packed tubular glands which may be simple, or branching, depending on the region of the stomach.
3. Absorptive - found in the small intestine, where the mucosa has finger like projections called villi, with intervening short glands called crypts (of Lieberkuhn).
4. Absorptive/Protective - in large intestine, where the mucosa is arranged as closely packed, straight tubular glands with cells specialized for water absorption and mucous-secreting goblet cells for lubrication of the intestine.

SUBMUCOSA:

This is composed of dense connective tissue, containing larger blood vessels, lymphatics, and sensory, parasympathetic, sympathetic and enteric nerves. Ganglion cells of postganglionic parasympathetic neurons and enteric neurons form the submucosal plexus (Meissner's plexus). Glands are present in this region in the oesophagus and duodenum.

MUSCULARIS EXTERNA:

This is generally arranged as 2 thick layers of smooth muscle, with an inner spiral layer of circular fibres and outer, looser spiral layer of longitudinal fibres. Between

these layers lies the myenteric plexus (Auerbach's plexus) of the enteric nervous system. Synchronized rhythmic contractions of these layers, controlled by the enteric nervous system, forms waves of peristalsis, propelling the gut contents distally. The circular muscle layer thickens along several points of the G.I. tract to form sphincters or valves, including pharyngeoesophageal sphincter, pyloric sphincter, ileocaecal valve & internal anal sphincter.

SEROSA AND ADVENTITIA:

The serosa, consisting of mesothelium and loose connective tissue, is the visceral peritoneum of the abdominal cavity, continuous with the mesentery. Those portions of the tract that are retroperitoneal or lie outside the abdominal cavity attach to adjacent structures via loose connective tissue called adventitia.

PHYSIOLOGY

The gastro-intestinal system is essentially a long tube running right through the body, with specialized sections that are capable of digesting material put in at the top end and extracting any useful components from it, then expelling the waste products at the bottom end. The whole system is under hormonal control, with the presence of food in the mouth triggering off a cascade of hormonal actions.

STOMACH:

Each region of the stomach performs different functions; the fundus collects digestive gases, the body secretes pepsinogen and hydrochloric acid, and the pylorus is responsible for mucus, gastrin and pepsinogen secretion.

There are many different gastric glands and they secret many different chemicals. Parietal cells secrete hydrochloric acid; chief cells secrete pepsinogen; goblet cells secrete mucus; argentaffin cells secrete serotonin and histamine; and G cells secrete the hormone gastrin.

The secretion of gastric juices occurs in three phases: cephalic, gastric, and intestinal.

The **cephalic phase** is activated by the smell and taste of food and swallowing. The **gastric phase** is activated by the chemical effects of food and the distension of the stomach.

The **intestinal phase** blocks the effect of the cephalic and gastric phases. Gastric juice also contains an enzyme named pepsin, which digests proteins, hydrochloric acid and mucus.

Hydrochloric acid causes the stomach to maintain a pH of about 2, which helps kill off bacteria that comes into the digestive system via food. The gastric juice is highly acidic with a pH of 1-3. It may cause or compound damage to the stomach wall or its layer of mucus, causing a peptic ulcer.

After receiving the bolus (chewed food) the process of peristalsis is started; mixed and churned with gastric juices the bolus is transformed into a semi-liquid substance called

chyme. Stomach muscles mix up the food with enzymes and acids to make smaller digestible pieces. The pyloric sphincter, a walnut shaped muscular tube at the stomach outlet, keeps chyme in the stomach until it reaches the right consistency to pass into the small intestine. The food leaves the stomach in small squirts rather than all at once.

SMALL INTESTINE:

The small intestine is the site where most of the chemical and mechanical digestion is carried out. Tiny projections called villi line the small intestine which absorbs digested food into the capillaries. Most of the food absorption takes place in the jejunum and the ileum.

The functions of a small intestine are, the digestion of proteins into peptides and amino acids principally occurs in the stomach but some also occurs in the small intestine. Peptides are degraded into amino acids; lipids (fats) are degraded into fatty acids and glycerol; and carbohydrates are degraded into simple sugars.

Coordinated contractions of smooth muscle participate in several ways to facilitate digestion and absorption in the small intestine. Food particles are moved through the small intestine by an alternating wave motion created by muscles in the intestinal wall. The frequency of the slow waves decreases from about 12 per minute in the jejunum to about 9 per minute in the ileum. Two fundamental patterns of motility conducted by the small intestine are propulsion and segmentation contractions. There are three main aspects of intestinal movement. One is of a propulsive nature, including peristaltic and antiperistaltic motions. Pendular movements and rhythmic actions are

related to intestinal absorption. Third, there are control movements which initiate or stop peristalsis by the creation of waves.

COLON:

From the small intestine, the chyme enters the large intestine. With the exception of some volatile fatty acids and vitamins, there is little absorption of nutrients occurring at this level. The large intestine mainly functions to remove water from the intestinal contents, and to concentrate the feed residues for excretion. In order to maximize water removal efficiency, the large intestine causes delay in the rate of passage of intestinal contents. Three patterns of motility are observed the colon Segmentation contractions, Antiperistaltic &Peristaltic contractions, and Mass movements. The frequency of wave of peristalsis in the colon unlike the small intestine is more, from two per minute at ileocaecal junction to six per minute in the sigmoid colon.

BOWEL TRANSIT TIME:

Bowel transit time is simply the amount of time (in hours) it takes for a meal to travel from the mouth, through the digestive tract and for its waste by-products to be eliminated through a bowel movement. It can vary a great deal from person to person due to dietary habits, age, climate, exercise habits, immobility, medications and so on. It is clear however, that a healthy person should have a bowel transit time ranging from 8 to 14 hours. The end results should be two to three loose bowel movements a day.

HOLLOW VISCUS PERFORATION

Perforation peritonitis is the most common surgical emergency in India[1]. Despite advances in surgical techniques, antimicrobial therapy and intensive care support, management of peritonitis continues to be highly demanding, difficult and complex[1].

FREQUENCY

In adults, perforations of peptic ulcer disease were a common cause of morbidity and mortality with acute abdomen until the latter half of the 20th century. The rate has fallen in parallel with the general decline in the prevalence of peptic ulcer disease. Duodenal ulcer perforations are 2-3 times more common than are gastric ulcer perforations. About a third of gastric perforations are due to gastric carcinoma[18].

Approximately 10-15% of patients with acute diverticulitis develop free perforation. Although most episodes of perforated diverticulum are confined to the peridiverticular region or pelvis, patients occasionally present with signs of generalized peritonitis. The overall mortality rate is relatively high (~20-40%), largely because of complications, such as septic shock and multiorgan failure[18].

In elderly patients, acute appendicitis has a mortality rate of 35% and a morbidity rate of 50%. A major contributing factor to morbidity and mortality in these patients is the presence of 1 or more severe medical conditions coexisting with, but predating, the appendicitis[18].

Endoscopy-associated bowel injuries are not a common cause of perforation. For example, perforations related to endoscopic retrograde cholangiopancreatography

(ERCP) occur in about 1% of patients[19].While the overall risk of colonoscopy related perforation is very low,with a much less than 1% incidence[35].

ETIOLOGY

- Ingestion of aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs) and steroids - Intestinal perforation from such causes is particularly observed in elderly patients. Prescribing NSAIDs to patients with diverticular disease carries an increased risk of colonic perforation.
- Presence of a predisposing condition - Predisposing conditions include peptic ulcer disease, acute appendicitis, acute diverticulitis, and inflamed Meckel diverticulum. Indeed, acute appendicitis is still one of the common causes of bowel perforation in elderly patients and is associated with relatively poor outcomes[20].
- Bacterial infections - Bacterial infections (eg, typhoid fever) may be complicated by intestinal perforation in about 5% of patients. Perforation in these patients may unexpectedly occur after their condition has started to improve.
- Inflammatory bowel disease - Bowel perforation may occur in patients with acute ulcerative colitis, and perforation of the terminal ileum may occur in patients with Crohn's disease and tuberculosis.
- Perforation secondary to intestinal ischemia (eg, ischemic colitis)

- Bowel perforation by intra-abdominal malignancy, lymphoma, or metastatic renal carcinoma - Even benign tumors, such as desmoid tumors (eg, those originating from the fibrous tissues of the mesentery), may cause bowel perforation.
- Radiotherapy of cervical carcinoma and other intra-abdominal malignancies - This may be associated with late complications, including bowel obstruction and bowel perforation[21].
- Necrotizing vasculitis - Wegener's granulomatosis affecting the viscera, although uncommon, may cause bowel ulcerations and perforations[22].
- Kidney transplantation - Following kidney transplantation, gastrointestinal perforations may occur as a complication. In these cases, the perforation is usually related to the use of high doses of immunosuppressive medications, a treatment employed in the early postoperative period and in the management of acute rejection.
- Ingestion of caustic substances - Accidental or intentional ingestion of caustic substances may result in acute intestinal perforation and peritonitis. Delayed perforation may occur up to 4 days after acid exposure.

PEPTIC ULCER PERFORATION:

The peptic ulcer perforation is one of the commonest abdominal emergencies after acute appendicitis and acute intestinal obstruction. Perforation of peptic ulcer is being one of the most serious and most overwhelming problems affecting the patients with peptic ulcer who is not on proper treatment for peptic ulcers. There is decline in incidence of peptic ulcers and elective surgery for peptic ulcers, which is attributed to the era of H2 blockers

and proton pump inhibitors, which provides symptomatic relief to patient. But in general the incidences of emergency surgery, hospitalization and mortality for perforated peptic ulcer have remained stable through the last two decades, probably due to increased inadvertent use of NSAIDS, corticosteroids and because of irregular use of H_2 antagonist drugs.

Peptic ulcer is a term used to refer to a group of ulcerative disorders of the gastrointestinal tract, involving principally the most proximal position of duodenum, the stomach, the lower end of the oesophagus, the jejunum after surgical anastamosis to stomach or rarely the ileum adjacent to the Meckel's diverticulum due to ectopic gastric epithelium.

Approximately 98 – 99% of peptic ulcers occur in the first portion of duodenum or in the stomach, in a ratio of about 4%[23]. About 5% of individuals with gastric ulcer develop duodenal ulcers, but 20% of those with duodenal ulcers develop gastric lesions[23].

The pyloric channel, which is 1-2cms in length, is the narrowest portion of the gastric outlet. Because of their gastric acid secretory characteristics and clinical features, pyloric channel are classified with duodenal rather than gastric ulcer. Ulcers in this location often produce symptoms similar to those of duodenal ulcer. In-patients with pyloric channel ulcers, food may accentuate rather than relive ulcer pain.

Peptic ulcers are remitting relapsing lesions, at one time duodenal ulcers were much more common than gastric ulcer, but their incidence and prevalence are now approaching those of gastric ulcers.

Most often diagnosed in middle aged to older adults, but may first become evidence in young adult life. Male to female ratio for duodenal ulcer is about 3:1 and for gastric ulcers around 1.5:2.1. Women are most often affected at or after the menopause[24].

Etiology:

The word "No acid-No ulcer" does not hold good now a days, because peptic ulcer is considered now more an infective disease, caused by Helicobacter Pylori.

In 1983, Warren and Marshall first reported isolation of Helicobacter Pylori from the mucosal biopsy specimen of patient with peptic ulcer diseases[25].

Helicobacter pylori are a small spirally curved, gram negative, microaerophilic rod with multiple pollar flagellae. 80–90% of population are affected with infection of Helicobacter pylori. The incidence of infection within a population increases with age. The possibility of infection is inversely related to socioeconomic group[25].

Pathogenesis:

Helicobacter pylori infection invariably results in chronic gastritis. The clinical result of this infection ranges from asymptomatic gastritis to peptic ulceration and gastric cancer. Helicobacter pylori colonizes in the gastric epithelium causing Type-B gastritis by which it reduces the resistance of gastric mucosa to attack by acid and pepsin resulting in gastric ulcer. Although, Helicobacter pylori normally reside in stomach, it also leads to causation of duodenal ulcers. This can be explained by the fact that antral Helicobacter pylori infection impairs the inhibitory feedback control of acid secretion, thus promoting duodenal ulcerogenesis by increasing duodenal acid load, resulting into duodinitis which leads to local inflammation, mucosal injury and eventually ulcer formation[25].

Irrespective of treatment, peptic ulcer takes one of the courses during the period of its progress like healing, chronicity or complications.

The **complications** of peptic ulcer are:

1. Hemorrhage
2. Perforation
3. Cicatrical contraction
4. Carcinomatous changes.

"Perforation" is the natural termination of an ulcer, which continues to penetrate deeper tissues. Perforation of duodenal ulcer greatly out number gastric ulcer (Illingworth, 1975).

The incidence of perforation approximately 7-10 cases per 1, 00,000 population/year[24]. Perforation occurs in 10-15% of established cases of peptic ulcers and in about 2% of patients perforation is the first manifestation. Anterior ulcer tend to perforate because of the absence of protective viscera and major blood vessels, in contrast to the bleeding ulcers that are usually situated posteriorly in <10% of patients with high death rate[7].

Pathological course:

At the onset of perforation there is sudden spillage of gastric or duodenal contents into the general peritoneal cavity and results in chemical peritonitis. The degree of involvement of the peritoneal cavity by bacteria is always uncertain. It is suggested that at first the visceral contents are sterile and the infective peritonitis in the early case is

unlikely. However, it depends on the general condition of the patient and his resistance to infection.

Perforation of peptic ulcer may be classified as follows[26]:

1. Acute perforation
2. Subacute perforation
3. Chronic perforation
4. Perforation associated with hemorrhage
5. Perforation of intrathoracic gastric ulceration
6. Pseudoperforation

1. Acute Perforation:

The ulcer perforate and the general peritoneal cavity become flooded with gastric and duodenal contents, causing chemical peritonitis.

The clinical features vary according to the stage of perforation. The ulcer perforates and the general peritoneal cavity becomes flooded with gastric and duodenal contents. The clinical course can be divided into three stages, each of variable duration.

a. Primary stage or the stage of peritonism.

b. Secondary stage or the stage of peritoneal reaction.

c. Tertiary stage or the stage of bacterial peritonitis.

a) Primary stage: The clinical course of a perforation is generally unmistakable. At that moment, the patient feels acute agonizing pain in the epigastrium or right

hypochondrium, which usually becomes rapidly generalized. He is plunged into a state of prostration and may be rendered immobile and helpless[24].

b) Secondary stage: Transition of the primary stage to secondary stage takes 3-6 hours, depending on the size and site of perforation and amount of peritoneal soiling. It is during this stage the spontaneous sealing of perforation may occur. If there is gross leakage of gastric contents, the patient may pass onto the stage of septic peritonitis. The length of this stage rarely exceeds 6 hours. During this stage the pain is lessened markedly. There would be general improvement in the patients' condition. For this reason this stage of reaction has sometime been called stage of delusion.

c) Tertiary Stage: This is the stage of diffuse peritonitis, begins about 12 hours after perforation and lasts for about 24 hours until it passes on to final stage of paralytic intestinal obstruction. Pathogenic organism multiplies rapidly. Peritoneal fluid becomes more purulent. The intestines slowly and pragmatically distend with gas and fluid. Intestinal movements diminish and finally disappear with onset of paralytic ileus. Pain is less severe, vomiting frequently; hiccoughs may further depress the patient. Sweating and vomiting and outpouring of fluid into peritoneal cavity, distended paralyzed intestine, dehydration and electrolyte imbalance become more evident. Patient complains of severe thirst, raised temperature, tongue dry and coated, pulse thready, respiration is shallow and rapid. Abdomen distended, guarding still present. On auscultation occasional tinkles heard. The typical Hippocratic facies denotes that end is not too far off. The face is ashen, body cold and calamity. The patient drifts into toxemia, dehydration and circulatory failure. Death usually takes place 4-5 days after Perforation.

2. Subacute Perforation

An ulcer may perforate and the perforation may seal rapidly before there is spillage of gastric and duodenal contents, into general peritoneal cavity. There is sudden onset of acute abdominal pain often more severe to the right upper quadrant. Respiration will be shallow and deep inspiration may be associated with an abrupt catch in the breath.

3. Chronic Perforation:

When an ulcer perforates into an area that is walled off by adhesions or by adjacent viscera such as liver, colon or greater omentum or when gastric ulcer perforates into omental sac, a chronic abscess may develop and will give rise to considerable confusion in diagnosis. As these patients do not present with signs and symptoms of peritonitis, they are seldom diagnosed as having perforated peptic ulcer.

4. Perforation Associated with Haemorrhage:

The association of a perforation with massive haemorrhage is grave but fortunately rare complication. It may present on one of the three ways:

a) Haemorrhage and perforation occurring concomitantly.

b) Haemorrhage following a recently sutured perforation.

c) Perforation occurring during the medical treatment of haemorrhage.

5. PERFORATION OF AN INTRATHORACIC GASTRIC ULCER:

This is a rare variety of perforation. The ulcer is in hiatus hernia, which is fixed in the mediastinum. Unless existence of Hiatus hernia is known it is extremely difficult to make

a correct preoperative diagnosis as the symptoms and signs point to some grave intrathoracic lesions such as coronary thrombosis, acute percarditis and pulmonary embolism.

Rare type of perforated peptic ulcer: A peptic ulcer in a Meckel's diverticulum, in intestinal duplication occasionally perforates. Multiple simultaneous perforation occurs in less than one percent of all cases[26].

APPENDICITIS:

Appendicitis is a common and urgent surgical illness with protean manifestations, generally overlap with other clinical syndromes, and significant morbidity, which increases with diagnostic delay. Obstruction of the appendiceal lumen is the primary cause of appendicitis. An anatomic blind pouch, obstruction of the appendiceal lumen leads to distension of the appendix due to accumulated intraluminal fluid. Ineffective lymphatic and venous drainage allows bacterial invasion of the appendiceal wall and, in advanced cases, perforation and spillage of pus into the peritoneal cavity.

There are four type of appendicitis

1. Acute appendicitis
2. Sub-acute appendicitis
3. Recurrent appendicitis
4. Chronic appendicitis.

Acute appendicitis is the most common surgical condition of the abdomen. Acute appendicitis may occur at all ages, but is most commonly seen in the second and third

decades of life[27]. The sequence given in acute appendicitis is not inevitable. Some episodes of acute appendicitis apparently subside spontaneously before they reach the acute stage. This is called as **sub-acute appendicitis**. If a full-blown appendicitis does not ensue, the appendix may turn into a 'grumbler' precipitating recurrent attacks. This is known as **recurrent appendicitis**. Sometimes pathological examination of the appendix may reveal thickening and scarring suggesting old, healed acute inflammation. This is called as **chronic appendicitis.**

The overall mortality rate of 0.2-0.8% is attributable to complications of the disease rather than to surgical intervention[27]. Mortality rate rises above 20% in patients older than 70 years, primarily because of diagnostic and therapeutic delay[20]. Perforation rate is higher among patients younger than 18 years and patients older than 50 years, possibly because of delays in diagnosis[20]. Appendiceal perforation is associated with a sharp increase in morbidity and mortality rates[27].

Patients presenting late in the course of acute appendicitis are complicated by the development of an inflammatory mass in right iliac fossa. This inflammatory mass is composed of the inflamed appendix, omentum and bowel loops. This is an attempt of the nature to prevent general peritonitis even if rupture of the appendix occurs. Progressive suppurative process in an appendicular mass forms an appendicular abscess walled off by the omentum, inflamed caecum and coils of small intestine. Such abscess may follow rupture of the appendix with the expulsion of small content of the appendix distal to the obstruction.

TYPHOID ENTERITIS:

The pathology of typhoid as a faeco-oral disease caused by Salmonella has been described as far back as 1850 by William Jenner[28]. Salmonella typhi and paratyphi infection (causing typhoid fever), is a serious systemic disease in developing countries and in countries where unhealthy environmental conditions prevail. Typhoid fever is endemic in India. A study in an urban slum showed 1% of children up to 17 years old suffered from typhoid fever every year[28].

Intestinal perforation resulting from complicated typhoid fever is a continuing challenge for the surgeons practicing in an endemic area, because of the high morbidity and mortality rates associated with its operative management. The reasons for these high mortality rates and postoperative complications are, continuing severe peritonitis, septicaemia, malnutrition, fluid, electrolyte derangements and prolongation of perforation and operation time[29]. Terminal ileum is the commonest site of typhoid perforation.

The majority of typhoid fever patients who develop perforation do so within the first 2 weeks of the illness[29]. The mechanism of intestinal perforation in typhoid fever is hyperplasia and necrosis of Peyer's patches of the terminal ileum. The lymphoid aggregates of Peyer's patches extend from the lamina propria to the submucosa, so that in the presence of hyperplasia the distance from the luminal epithelium to the serosa is bridged by lymphoid tissue. Tissue damage in Peyer's patches occurs, resulting in ulceration, bleeding, necrosis, and, in extreme cases, full-thickness perforation. The

process leading to tissue damage is probably multifactorial, involving both bacterial factors and host inflammatory response.

BOWEL NEOPLASM:

Bowel neoplasm causing perforation is rare entity. In general, small bowel perforation secondary to gastrointestinal tumors may occur due to obstructing or invading tumors. Colon carcinoma may present with perforations causing generalized peritonitis or localized peritonitis. Perforation usually occurs at the site of tumor due to its growth and pressure necrosis of walls[30]. Endoluminal hypertension ischemia is usually responsible for perforations in colonic carcinoma but in cases with non-occluded colon, as a consequence of biological problems of immune hyper reactivity of a rejection reaction type leads to perforation[30]. Bacterial and viral overgrowth in obstructed and ischemic bowel may lead to increased intraluminal pressure causing ischemic enteritis and subsequent perforation of dilated small bowel if ileocecal valve is incompetent or malfunctioning.

In small bowel primary tumors like adenocarcinoma, carcinoid, sarcoma and lymphoma are rare causes of perforation. Metastatic tumors to the gastrointestinal tracts may present with perforation, like metastasis from breast carcinoma, subcutaneous angiosarcoma & disseminated rectal carcinoma[30].

TUBERCULAR ENTERITIS:

Tuberculosis can involve any part of the gastrointestinal tract from mouth to anus, the peritoneum and the pancreatobiliary system. It can have a varied presentation, frequently mimicking other common and rare diseases. Both the incidence and the severity of abdominal tuberculosis is expected to increase with increasing incidence of HIV infection in India[31]. Extra-pulmonary forms of tuberculosis which account for 10-15 per cent of all cases may represent up to 50 per cent of patients with AIDS. Tuberculosis of the gastrointestinal tract is the sixth most frequent form of extra-pulmonary site, after lymphatic, genitourinary, bone and joint, miliary and meningeal tuberculosis[32].

The postulated mechanisms by which the tubercule bacilli reach the gastrointestinal tract are: (*i*) hematogenous spread from the primary lung focus in childhood, with later reactivation; (*ii*) ingestion of bacilli in sputum from active pulmonary focus; (*iii*) direct spread from adjacent organs; and (*iv*) and through lymph channels from infected nodes.

The earlier belief that most cases are due to reactivation of quiescent foci is being challenged with a recent study using DNA fingerprinting showing that 40 per cent cases are due to reinfection[31]. In India, the organism isolated from all intestinal lesions has been *Mycobacterium tuberculosis* and not *M.bovis*.

The most common site of involvement is the ileocaecal region, possibly because of the increased physiological stasis, increased rate of fluid and electrolyte absorption, minimal digestive activity and an abundance of lymphoid tissue at this site.

Pathology:

Tuberculous granulomas are initially formed in the mucosa or the Peyer's patches. These granulomas are of variable size and characteristically tend to be confluent, in contrast to those in Crohn's disease. Granulomas are often seen just beneath the ulcer bed, mainly in the submucosal layer. Submucosal edema or widening is inconspicuous. Tubercular ulcers are relatively superficial and usually do not penetrate beyond the muscularis[10]. They may be single or multiple, and the intervening mucosa is usually uninvolved. These ulcers are usually transversely oriented in contrast to Crohn's disease where the ulcers are longitudinal or serpiginous. Cicatrical healing of these circumferential 'girdle ulcers' results in strictures.

Hoon *et al* originally classified the gross morphological appearance of the involved bowel into ulcerative, ulcerohyperplastic and hyperplastic varieties. Tandon and Prakash described the bowel lesions as ulcerative and ulcerohypertrophic types[33].

Ulcerative form has been found more often in malnourished adults, while hypertrophic form is classically found in relatively well nourished adults. The bowel wall is thickened and the serosal surface is studded with nodules of variable size. These ulcerative and stricturous lesions are usually seen in the small intestine. Colonic and ileocaecal lesions are ulcerohypertrophic. The patient often presents with a right iliac fossa lump constituted by the ileocaecal region, mesenteric fat and lymph nodes. The ileocaecal angle is distorted and often obtuse. Both sides of the ileocaecal valve are usually involved leading to incompetence of the valve, another point of distinction from Crohn's disease.

Tuberculosis accounts for 5-9 per cent of all small intestinal perforations in India[34], and is the second commonest cause after typhoid fever. Evidence of tuberculosis on chest X-ray and a history of subacute intestinal obstruction are important clues. Pneumoperitoneum may be detected on radiographs in only half of the cases. Tubercular perforations are usually single and proximal to a stricture. Acute tubercular peritonitis without intestinal perforation is usually an acute presentation of peritoneal disease but may be due to ruptured caseating lymph nodes.

ULCERATIVE COLITIS:

Ulcerative colitis is a form of colitis, a disease of the intestine, specifically the large colon, that includes characteristic ulcers, or open sores, in the colon. The main symptom of active disease is usually constant diarrhea mixed with blood, of gradual onset. As with Crohn's disease, the prevalence of ulcerative colitis is greater among Ashkenazi Jews and decreases progressively in other persons of Jewish descent, non-Jewish Caucasians, Africans, Hispanics, and Asians[35].

Ulcerative colitis is normally continuous from the rectum up the colon. The disease is classified by the extent of involvement, depending on how far up the colon the disease extends, as Proctitis ,Proctosigmoiditis, Left-sided colitis and pancolitis. Toxic megacolon is rare but devastating complication occurring up to 2.5% of patients with ulcerative colitis[36].

Acute perforation is most lethal but infrequent complication of ulcerative colitis. Although the overall incidence of perforation during the first attack is less than 4%, if the

attack is severe, the incidence raises up to the 10%[30]. If the patient has pancolitis the perforation rate can rise up to 15 % [30]. Although free colon perforation occurs much more frequently in the presence of toxic megacolon than in its absence, it is important to remember that toxic megacolon is not a prerequisite for development of perforation.

CROHN'S DISEASE:

Crohn's disease is an idiopathic, chronic, transmural inflammatory process of the bowel that often leads to fibrosis and obstructive symptoms, which can affect any part of the gastrointestinal (GI) tract from the mouth to the anus. The incidence of Crohn's disease in children is much less than that seen in adults. It is more prevalent in Western countries compared to Asia and Africa[37].

In Crohn's disease there is a disordered regulation of mucosal and systemic immune response resulting in the perpetuation of the inflammatory cascade. The transmural inflammation and discontinuous involvement are the characteristic features. Transmural inflammation results in thickening of the bowel wall and narrowing of the lumen. As Crohn disease progresses, it is complicated by obstruction or deep ulceration leading to fistulization by way of the sinus tracts penetrating the serosa, microperforation, abscess formation, adhesions, and malabsorption.

Although any area of the gastrointestinal system may be affected, the most common site of Crohn disease is the ileocecal region, followed by the colon, the small intestine alone, the stomach (rarely), and the mouth. The esophagus is very rarely involved[37].

Spontaneous free perforation is an uncommon event in the natural history of Crohn's disease. Free bowel perforation is one of the indications for emergency surgery in

Crohn's disease[38]. It is generally accepted that 1-3% of patients with Crohn's disease will present with a free perforation initially or eventually in their disease course[38]. It occurs during an acute exacerbation of chronic disease, particularly in the presence of distal obstruction. Free perforation of colon in Crohn's disease is extremely rare.

DIVERTICULAR DISEASE:

Small intestinal diverticulosis refers to the clinical entity characterized by the presence of multiple saclike mucosal herniations through weak points in the intestinal wall. Small intestinal diverticula are far less common than colonic diverticula. Diverticula can be classified as intraluminal or extraluminal. Intraluminal diverticula and Meckel diverticulum are congenital. Extraluminal diverticula may be found in various anatomic locations and are referred to as duodenal, jejunal, ileal, or jejunoileal diverticula. Duodenal diverticula are approximately 5 times more common than jejunoileal diverticula[39]. The incidence at autopsy of duodenal diverticula is 6-22%[39]. Jejunal diverticula are less common, with a reported incidence of less than 0.5% on upper gastrointestinal (GI) radiographs and a 0.3-1.3% autopsy incidence[39].

Meckel diverticulum is the most common congenital abnormality of the small intestine; it is caused by an incomplete obliteration of the omphalomesenteric duct. Although originally described by Fabricius Hildanus in 1598, it is named after Johann Friedrich Meckel, who established its embryonic origin in 1809[40]. The rule of 2's: 2% (of the population) - 2 feet (from the ileocecal valve) - 2 inches (in length) - 2% are symptomatic, there are 2 types of common ectopic tissue (gastric and pancreatic) and males are twice as likely to be affected[40].

The most common complication of Meckel's diverticulum is hemorrhage. Gastrointestinal bleeding is seen in approximately 55% of patients with Meckel's diverticulum[40]. Perforation of Meckel's diverticulum is less common. Perforation of the diverticulum is usually secondary to inflammatory diverticulitis and gangrene, although peptic ulceration also can lead to perforation[40].

Colonic diverticulosis is among the most common diseases in developed Western countries. In the United States, diverticulosis occurs in approximately one third of the population older than age 45 and in up to two thirds of the population older than 85 years, and it also affects a significant proportion of younger adults[30]. In underdeveloped nations in Asia and Africa, diverticulosis occurs in less than 0.2% of the population[30]. This low rate is probably the result of a high-fiber diet.

Colonic diverticulosis in general is an acquired disease, developing as mucosal and submucosal herniations through the circular muscle layer at vulnerable weak points of the colonic wall. Most frequently colonic diverticula are located distally on the sigmoid colon alone (26%) or involving both the sigmoid and descending colon (30%)[30]. However, sometimes they are scattered throughout the colon (16%) or are limited to the caecum and ascending colon (less than 5% of the cases).

Perforated colonic diverticular disease results in considerable mortality and morbidity. Perforation is the most frequent complication of diverticular disease requiring surgical treatment[41]. Because it is preceded by local inflammation, in most of the cases the perforated contents are walled of by adherent pericolic structures leading to a pericolic

abscess. Free perforation occurs rarely and results in diffuse peritonitis, sepsis and shock[41].

ASCARIASIS:

Ascariasis is the infestation by the largest intestinal nematode of man, a common problem in the tropics attributed to poor hygienic and low socioeconomic conditions[42]. Common surgical problems caused by Ascaris infestation include small intestinal obstruction, volvulus, intussusception and perforation usually involving the ileum.

Bowel perforation is thought to follow ischemia from pressure by the mass of worms in the ileum. This view was however questioned by Efem who postulated that except in confined spaces like the appendix, Meckel's diverticulum and the biliary tree, the intestine is capable of immense dilatation to accommodate up to 5000 worms without symptoms[42]. Typhoid perforations, non-specific ulcers and anastomotic suture lines are thought to provide exits for the worm.

AMEBIC COLITIS:

Amebic colitis, also known as amebiasis, is a gastrointestinal disorder caused by invasion of the intestine by the protozoan parasite, *Entameba histolytica*. Although primarily a disease found in underdeveloped countries, this condition may exist in patients who have recently traveled outside the United States.

Invasive disease begins with the adherence of *E histolytica* to colonic mucins, epithelial cells, and leukocytes. Adherence of the trophozoite is mediated by a galactose-inhibitable adherence lectin. After adherence, trophozoites invade the colonic epithelium to produce

the ulcerative lesions typical of intestinal amebiasis. Bowel perforation is rare in amebiasis with a frequency of 1-6%, but leads to extremely high mortality of 55-100%[43].

Fulminant amebic colitis, however, is a rare complication of amebic dysentery, which occurs in only 6%- 11% of patients with symptomatic infection. It presents with rapid onset of severe bloody diarrhea, severe abdominal pain, and high fever[43]. The amebic invasion of the colonic wall leads to a fierce superacute course of massive necrosis of wide segments or of the entire colon. The extensive necrosis usually affects all layers of the bowel wall. Toxic megacolon and intestinal perforation are common. The perforations may be microscopic and usually sealed off by the adjacent omentum or bowels. They also may be macroscopic, which results in generalized fecal peritonitis. The mortality rate is greater than 50% if aggressive surgical management is not promptly initiated[43].

CLINICAL FEATURES AND DIGNOSIS

Gastrointestinal perforations forms one of the acute emergencies which may call upon intervention in emergency settings by the general surgical practitioners. Hence careful history, physical findings , proper analysis of clinical data and prompt appropriate management are of prime importance as delay in any of these may adversely affect prognosis.

SYMPTOMS:

Patients with bowel perforation presents with severe abdominal pain, vomiting, nausea, fever, abdominal distension & altered bowel habits.

Abdominal Pain: Sudden severe abdominal pain is a feature of all perforations. Abdominal pain is sole is symptom which makes the patient to seek medical advice in hollow viscus perforations. Depending on the site, type, radiation & character of pain, type of perforation can be guessed.

Sharp , severe , sudden onset epigastric pain that awakens the patient from sleep often suggests perforated peptic ulcer. Patient becomes restless in pain but soon lies still after chemical peritonitis sets in. pain usually radiates along the right paracolic gutter to right iliac fossa.

In appendicitis the classic presentation begins with crampy, intermittent abdominal pain, thought to be due to obstructed appediceal lumen. Initially the pain is either periumbilical or diffuse and difficult to localize. Later the pain classically migrates to right lower quadrant as transmural inflammation of appendix leads to inflammation of the peritoneal

lining of right lower abdomen. Even the character of pain changes from dull and colicky to sharp and constant.

Referred pain is perceived distant from its source and results from convergence of nerve fibers at spinal cord. In the diseases of stomach, duodenum & jejunum (T2 toT8) the pain is felt in the epigastric region. In the lesions of appendix and ileum (T9-T10) the pain is perceived around the umbilicus, where as in case of colon (T11-L2) pain is felt in hypogastrium. Any diaphragmatic irritation gives rise to corresponding shoulder pain.

Previous history of periodic pain is feature of peptic perforation and crampy lower abdominal pain is a feature of tuberculous enteritis, ulcerative colitis and Crohn's disease.

Vomiting: Severe nausea and vomiting are often signs of bowel perforation. Nausea in bowel perforation is so severe that it causes vomiting. In early stages one or two episodes of vomiting is seen, which more or less decreases to reappear in later stages of diffuse peritonitis. Characteristically the vomits of peritonitis is effortless regurgitation of mouthfuls.

In early stages the vomitus may contain only gastric contents; later in stages of peritonitis it may show dark brown feculent material due to mixing of contaminated bowel content and bile but frank blood is seen in case of perforation due to gastric ulcer, duodenal ulcer & gastric neoplasm.

Fever: If the leakage from bowel perforation is not recognized it can cause a severe infection which can result in high degree fever. Later his may proceed to sepsis. Along with fever the patient can suffer from chills and rigors.

Mild degree fever with evening rise of temperature is noted in tuberculosis. High degree fever is noted with perforated appendicitis or diverticulitis with intraperitoneal abscess. Typical step ladder type of fever is noted in the enteric fever.

Distension of abdomen: Patient presenting in late stages of bowel perforation, present with abdominal distension. Abdominal distension is due to combination of paralytic ileus and fluid collection.

Bowel habits: Absolute constipation is a feature of peritonitis. However in early stages patient may present with diarrhea due to irritation of rectum due to pelvic collection. Past history of dysentery may give clue to amoebic colitis. Similarly history of alternating diarrhea and constipation may indicate tubercular enteritis, carcinoma colon or worm infestation. History of malaena may give hint to the diagnosis of peptic ulcer perforation or carcinoma stomach.

CLINICAL EXAMINATION:

General physical examination: Patients with bowel perforation may be pale due to shock. Patients with peptic ulcer disease and ascariasis are anemic whereas those with malignancy will be cachexic and anemic and may be jaundiced. Most of the patients are static with minimal movements, because movements will increase the pain. However in late stages of peritonitis patients will be tossing up in the bed, irritable, disoriented & non cooperative.

Abdominal facies is a peculiar facial expression which helps clinicians to discriminate an abdominal form of extra abdominal cause. In later stages of peritonitis patient will have typical Hippocratic facies with anxious look, bright eyes, pinched face & cold sweat. The

facies of dehydration is also typical and consists of sunken eyes, drawn cheeks and dry tongue.

Pulse rate will be high with small in volume in later stages of peritonitis. In enteric fever there will early bradycardia, but as peritonitis sets in there will be tachycardia. Respiratory rate is usually normal except with high degree fever and intra abdominal abscess; in which cases it will be high. Respiration is manly thoracic without involving the abdomen. Temperature may be mild or high degree as in cases of intraperitoneal abscess & collection.

ABDOMINAL EXAMINATION:

Abdomen is held still and looks straight and rigid. In thin patients it is retracted or scaphoid with rectus muscle thrown into prominence. Respiration is mainly thoracic.

On palpation tenderness is noted over an inflamed organ; for example in the right hypochondrium in peptic ulcer perforation, Mc. Burney's point in appendicular perforation and Manson's point in amoebic perforation. As perforation progresses there will be rebound tenderness with board like rigidity of abdomen. Muscle guarding will be noted as protective mechanism.

Hepatomegaly may be noted secondary to metastasis whereas spleenomegaly in enteric fever. There may be palpable mass in the abdomen or pelvis due to carcinoma or bolus of worms. Mild to moderate free fluid can be noted in the peritoneal cavity. Obliteration is liver dullness may be noted. Initially bowel sounds may be sluggish but later there will be paucity of bowel sounds.

On examination of other systems there may be focus of tuberculosis in lungs or generalized lymphadenopathy in tuberculosis or malignancy or enlarged lymph nodes in the left supraclavicular fossa (Virchow's lymph node).

Digital rectal examination: Tenderness is elicited in rectovesical pouch with rectal bulge secondary to inflammatory collections due to perforated peptic ulcer and in pelvic appendicular perforation. In Crohn's disease fissure, fistula, stricture or abscess may be seen. Irregular hard intraluminal growth is noted in carcinoma rectum.

SPECIFIC FEATURES IN PEPTIC ULCER PERFORATION:

Primary stage or stage of peritonism: Patient feels acute agonizing pain with restlessness but patient soon lies still once chemical peritonitis sets in. patient is pale, anxious with dry tongue. Patient is tachycardic with abdomen held still. Respiration is mainly thoracic with tender rigid abdomen. Rigidity is highest at epigastric & right hypochondriac regions. Painful percussion is elicited with obscuration of liver dullness. On auscultation abdomen is silent.

Secondary stage or stage of reaction: This is the stage of illusion or delusion as patient is relieved due to dilution of irritant fluid by peritoneal exudates. Slight distension of abdomen is noted with absent bowel sounds. Tenderness & rigidity are slightly less marked.

Tertiary stage or the stage of peritonitis: Diffuse peritonitis is sets in. Sufficient fluid is collected in the abdomen to be clinically detected. Tenderness is generalized with muscle guarding. On percussion abdomen is dull with absent bowel sounds with occasional obstructive tinkle.

DIFFERENTIAL DIAGNOSIS:

INFLAMMATORY :

- Peptic ulcer disease
- Gastritis
- Acute pancreatitis
- Cholecystitis, biliary colic
- Acute gastroenteritis
- Colitis
- Pelvic inflammatory disease
- Diverticulitis
- Acute appendicitis
- Inflammatory bowel disease
- Crohn's disease
- Inflammatory bowel disease

PERFORATION :

- Peptic ulcer
- Diverticular
- Typhoid illeal disease

ISCHEAMIC :

- Ischeamic colitis
- Ovarian torsion

OBSTRUCTION:

- Acute and/or subacute intestinal obstruction secondary to any cause

MEDICAL:

- Basal pneumonia
- Diaghramatic pleurisy
- Pre eruptive pain of Herpes zoster
- Acute gastero enteritis
- Colitis

INVESTIGATIONS

Laboratory Studies

- Complete blood cell (CBC) count
    - Parameters suggestive of infection (eg, leukocytosis) - Leukocytosis may be absent in elderly patients.
    - Elevated packed blood cell volume - This suggests a shift of intravascular fluid.
    - ESR
    - Blood grouping Rh typing

- Bleeding time and clotting time
- Blood urea serum creatinine
- Blood glucose
- Liver function test
- Blood culture for aerobic and anaerobic organisms.
- Widal test for enteric fever

- Peritoneal fluid for staining and culture sensitivity :

Obtained by: 1.Ultra sounded aspiration

2. During laprotomy

- ECG
- Liver function and renal function tests - Findings may be within reference ranges (or nearly so) if no preexisting disorder is present.
- Serial serum amylase and its onset of peak helps in the diagnosis of acute pancreatitis and its complications but is not specific as serum lipase. However urinary amylase levels remain elevated for several days after serum level returned to normal. Serum amylase is not specific for pancreatic disorder but can also be raised in non pancreatic disorder like in bowel perforation, ruptured ectopic, appendicular perforation with or without abcess,salivary gland disorders where false positive results are noted.

Imaging Studies

- Erect X- ray abdomen
- Lateral decubitus X- ray
- USG of abdomen and pelvis
- Chest X-ray
- Erect radiographs of the chest are recognized as the most appropriate first-line investigation when a perforated peptic ulcer is considered likely[18]. However, in approximately 30% of patients, no free gas can be identified[18]. Thus, an erect posteroanterior chest radiograph is not sufficiently sensitive to rule out pneumoperitoneum in patients presenting with upper abdominal pain. Thus lateral decubitus X- ray is recommended.
- Erect and lateral decubitus X- ray of the abdomen including chest are the most important steps in the diagnostic evaluation of patients presenting with medical history and/or clinical signs suggestive of bowel perforation. Findings suggestive of perforation include the following:

    - Free air trapped in the subdiaphragmatic locations - If the quantity of free air is great enough, its presence can be visualized on the supine radiograph of the abdomen, allowing clear definition of the inner and outer surface of the wall of the bowel.
    - Visible falciform ligament - The ligament may appear as an oblique structure extending from the right upper quadrant toward the umbilicus,

particularly when large quantities of gas are present on either side of the ligament.

- Air-fluid level - This is indicated by the presence of hydropneumoperitoneum or pyopneumoperitoneum on erect radiographs of the abdomen.
    - Water-soluble radiologic contrast media administered orally or through a nasogastric tube can be used as an adjunct diagnostic tool to detect any intraperitoneal leak.
    - The perforation has sealed at presentation in approximately 50% of patients. For those who favor a nonoperative approach, contrast radiology is routine in the management of these patients.

- Ultrasonograms of the abdomen
    - Localized gas collection related to bowel perforation may be detectable, particularly if it is associated with other ultrasonographic abnormalities (eg, thickened bowel loop).
    - The site of bowel perforation can be detected by ultrasonography (eg, gastric vs duodenal perforation, perforated appendicitis vs perforated diverticulitis).
    - Ultrasonograms of the abdomen can also provide rapid evaluation of the liver, spleen, pancreas, kidneys, ovaries, adrenals, and uterus.
- Computed tomography (CT) scans of the abdomen - This modality can be a valuable investigative tool, providing differential morphologic information not obtainable with plain radiography or ultrasonography.

- CT scans may provide evidence of localized perforation (eg, perforated duodenal ulcer) with leakage in the area of the gallbladder and right flank with or without free air being apparent.
- CT scans may show inflammatory changes in the pericolonic soft tissues and focal abscess due to diverticulitis (may mimic perforated colonic carcinoma).
- CT scans may not provide definitive radiographic evidence of perforated Meckel diverticulitis.

DIAGNOSTIC PROCEDURES

- Peritoneal diagnostic tap may be useful in determining the presence of intra-abdominal blood, fluid, and pus.
- Peritoneal lavage is more valuable in the presence of a history of blunt abdominal trauma.
    - The presence of blood or purulent material or the detection of bacteria on Gram stain suggests the need for early surgical exploration.
    - Alkaline phosphatase concentration in the peritoneal lavage is a helpful and sensitive test that may be used to detect occult blunt intestinal injuries. A concentration greater than 10 IU/L has been shown to be a sensitive and reliable test in the detection of occult small bowel injuries.

- Fine-catheter peritoneal cytology
    - This procedure involves the insertion of a venous cannula into the peritoneal cavity, through which a fine umbilical catheter is inserted while the patient is under local anesthesia.
    - Peritoneal fluid is aspirated, placed on a slide, and stained for examination under a light microscope for percentage of polymorphonuclear cells.
    - A value greater than 50% suggests a significant underlying inflammatory process[18].
    - This test, however, provides no clue as to the exact cause of inflammation.

TREATMENT

The mainstay of treatment for intestinal perforation is surgery. Emergency medical care includes the following steps:

- Establish intravenous access, and initiate crystalloid therapy in patients with clinical signs of dehydration or septicemia.
- Do not give anything by mouth.
- Start intravenous administration of antibiotics to patients with signs of septicemia. Antibiotics should cover aerobic and anaerobic organisms. The goals of antibiotic treatment are to eradicate infection and to minimize related postoperative complications.

However, if symptoms and signs of generalized peritonitis are absent, a non operative policy may be used with antibiotic therapy directed against gram-negative and anaerobic bacteria.

Metronidazole typically used in combination with an aminoglycoside to provide broad gram-negative and anaerobic coverage. Gentamicin is aminoglycoside antibiotic for gram-negative coverage. Used in combination with both an agent against gram-positive organisms and one that covers anaerobes. Cephalosporins like Cefotetan, Cefoxitin & Cefoperazone sodium.

SURGICAL THERAPY

The goals of surgical therapy are as follows:

- To correct the underlying anatomical problem
- To correct the cause of peritonitis
- To remove any foreign material in the peritoneal cavity that might inhibit WBC function and promote bacterial growth (eg, feces, food, bile, gastric or intestinal secretions, blood)

PREOPERATIVE DETAILS

- Correct any fluid or electrolyte imbalance. Replace extracellular fluid losses by administering Hartmann solution or a similar solution that has an electrolyte composition similar to plasma.

- Central venous pressure (CVP) monitoring is essential in critically ill and/or elderly patients, in whom cardiac impairment may be exacerbated by large fluid loss.
- Administer systemic antibiotics (eg, ampicillin, gentamicin, metronidazole), making a best estimation regarding the likely organisms.
- Nasogastric suction is required to empty the stomach and reduce the risk of further vomiting.
- Urinary catheterization is used to assess urinary flow and fluid replacement.
- Administer analgesics, such as morphine, in small intravenous doses, preferably as a continuous infusion.

GASTRODUODENAL PERFORATION:

Conservative Treatment: Wangensteen and Tylor introduced the conservative treatment for gastro-duodenal perforation which is used intermittently throughout the world[30,44]. Donovons and associates recommended non operative treatment in young patients with sealed acute perforated ulcer and in patients with chronic ulcer who had significant medical risk factors.

Non operative management can be considered in patients who do not have generalized peritonitis, hemodynamic instability or free peritoneal perforation on Gastrografin study. Contraindications for conservative surgery are in patients in whom the diagnosis is uncertain or in whom abdominal examination is not reliable. In conservative treatment the patient is observed closely through serial physical and laboratory examinations while being treated with nasogastric suction, intravenous acid secretion suppression &

intravenous broad spectrum antibiotics. If at any time during conservative management the patient deteriorates, an operation is indicated.

SURGICAL TREATMENT:

A**. Simple closure of perforation.**

In perforated duodenal ulcer the most frequently performed operation is simple closure with omental onlay reinforcement or patch. After clearing the peritoneal cavity of purulent and/or bilious fluid, visual inspection and palpation direct the surgeon to site of perforation. Post operative duodenal decompression is done with transpyloric nasogastric sump tube. A retrograde duodenal drain and jejunal feeding tube also can be placed in the proximal jejunum to decompress the jejunum if closure appears tenuous. Post operatively the patients should be treated with anti-secretory medications and antibiotics to eradicate helicobacter pylori.

Proximal vagotomy has been used in conjunction with patching the perforation. However the benefit of proximal gastric vagotomy over closure and antibiotic therapy has not been demonstrated.

Perforated gastric ulcer has higher mortality rate than perforated duodenal ulcer. Most perforated gastric ulcers are prepyloric and are best treated with distal gastric resection. If gastric ulcer is difficult to include in resection, generous biopsies should be taken to exclude malignancy and the ulcer is closed or patched primarily with omentum.

B. **Closure of perforation with definitive surgery.**

This has been advocated as it has been found that patients treated with simple closure have a severe relapse of the disease in >50% of cases in 5 years follow-up[30,44]. It includes,

i. Truncal vagotomy with gastrojejunostomy.

ii. Antrectomy with vagotomy.

iii. Pyloroplasty with vagotomy.

iv. Partial gastrectomy with vagotomy.

v. Highly selective vagotomy.

Laparoscopic Approach:

Recent development in minimal invasive surgery now allows laproscopic approach to the patient with perforated duodenal ulcer[45].

The perforation can be approached using three additional ports. The perforation can be dealt by: - Fibrin glue for minute perforation.

- Simple closure with omental patch and copious irrigation of the abdominal cavity.

- Automatic staples suture can be applied via laproscope (BJS, Dec 1993).

- A proximal gastric vagotomy or Taylor procedure (anterior seromyotomy and

- truncal vagotomy) may be performed.

Laparoscopic closure of perforation offers important advantages:

1. Decreased post operative pain
2. Less abdominal wall complication
3. Better visualization and ability to carry out a thorough peritoneal lavage.
4. Cosmetically better outcome.
5. Lower intra operative and post operative complications.
6. Early return to work.
7. Early mobilization
8. Lower mortality
9. It is as safe and effective as open surgery
10. Patients subjective well being was better after laparoscopic repair of perforated duodenal ulcer.

APPENDICULAR PERFORATION:

Urgent laparotomy is necessary for appendectomy with irrigation and drainage of peritoneal cavity. Exceptions to this are

- Patient is morbid with advanced peritonitis
- Appendicular mass or phlegmon.

Drainage is not generally advised after appendectomy but corrugated drain should be placed down to the appendix base and brought out through the lateral extremity of the wound. Ileocecectomy may be necessary if the inflammation extends to the wall of the caecum. On imaging studies if abscess cavity is demonstrated then, image guided drainage can be performed percutaneously or transrectally. Interval appendectomy can be performed 6 weeks after non operative management.

TYPHOID PERFORATIONS:

The need for aggressive fluid resuscitation and correction of electrolyte derangements and anaemia; together with the choice of a suitable antibiotic combination is crucial to surgical outcome. The antibiotic protocol that has been used over the years included: chloramphenicol, gentamycin, and metronidazole; which are given parenterally at diagnosis and continued for seven days before conversion to oral preparations of chloramphenicol and metronidazole. The rationale is to cover for not only the Salmonella organism but also for anaerobes and gram negative coliforms. The emergence of chloramphenicol resistant, Salmonellae, has led to the use fluoroquinolones (for example, ciprofloxacin), or third generation cephalosporins.

To be certain that the perforation on the ileum is due to typhoid enteritis, a positive blood, stool or urine culture is necessary. However, the yield for blood culture in a patient with typhoid intestinal perforation is low, ranging from 3−34%, in some reports[28]. Higher yields of the *Salmonella* organism is obtained from cultures of the perforation edges, bone marrow, or peritoneal aspirates; but this is often not possible and even when they are done the results do not significantly alter the operative treatment given to the patient.

The classical disposition of the typhoid perforation in the longitudinal axis of the ileum and on the antemesenteric border with an antecedent history of prolonged febrile illness, which do not respond to antimalarials, is enough to make a conclusion as to the etiology of the perforation.

Prompt surgery after adequate resuscitation, is the treatment of choice for typhoid perforation; this has considerably reduced mortality from 30−60% to approximately 6.8% in a recent series[29]. Many surgical techniques have been used, ranging from

- Simple peritoneal drainage under local anesthesia in moribund patients,
- Excision of the edge of the ileal perforation, and simple transverse closure in two layers;
- Segmental ileal resection and primary anastomosis especially in multiple perforations or
- Right hemicolectomy where the caecum is involved.

There are conflicting results of the outcome of these widely practiced techniques. Whereas, better results are reported with simple closure, in many series; others favour segmental ileal resection and anastomosis. Those that favour simple closure argue, that in such very ill patients any prolonged procedure may jeopardize the outcome and that the ileum affected by typhoid fever, take sutures well without cutting through. But any operative technique that is carried out in good time, and allows for a swift clearing of peritoneal contamination by a copious peritoneal lavage is the most likely to give the best outcome.

PERFORATION IN CARCINOMA COLON:

Cancer causing perforation of colonic wall represents a life threatening surgical emergency. Perforation results in diffuse fecal peritonitis with significant mortality and morbidity. In addition, the tumor perforation results in spillage of tumor cells and thus

has to be considered stage IV tumor. The goal of operation is to remove the perforated segment of the bowel. It may be possible to fashion an anastomosis and this is protected by proximal colostomy or ileostomy. The temporary diverting stoma can be closed about ten weeks after emergency operation. An alternative is to resect the perforated segment and exteriorize the proximal and distal limbs or to use Hartmann operation for distal lesions. Prognosis is poor for these patients but the five year survival rate approaches 30 % in patients with no obvious metastasis who are treated by immediate resection of the lesion[30].

INTESTINAL PERFORATION IN TUBERCULOSIS:

Standard Anti tubercular treatment gives a high cure rate for intestinal tuberculosis

1. Simple closure in 2 layers with non-absorbable suture:

It is quicker treatment for those who are critically ill and where tubercular stricture is short. Oval excision of perforation area with transverse closure reinforced by omental patch is done. This is contraindicated in perforations that have involved a segment of a bowel, as there is much granulation tissue and Caseation. These sutures may cut through and blow out of suture line may occur as the stenosis lies distal to perforation.

2. Simple closure with bypass of stricture

Simple closure with bypass of stricture by ileostomy or ileo-transverse colostomy safeguards the closure against a blow out.

3. Resection and end-to-end anastomosis:

If the patient is healthy and perforation involves a short segment, this forms an effective treatment. Perforations usually occur proximal to strictures. If the perforation is close to

the stricture and the stricture is solitary, then excision of perforation and stricture and end-to-end anastomosis is the treatment of choice. If the perforation is away from stricture or strictures are multiple, closure of perforation with by pass is recommended.

PERFORATION IN ULCERATIVE COLITIS AND CROHN'S DISEASE:

Free perforation is an absolute indication for emergent laparotomy with resection of diseased segment and exteriorization of the proximal bowel as an end ileostomy. Primary closure of the perforation should never be attempted, as sutures will not be able to approximate the edges of the perforated, edematous & diseased bowel in a satisfactory and tension –free way. The distal bowel end can be exteriorized as mucous fistula or closed defunctionalized pouch, depending on the degree of peritoneal contamination.

PERFORATION IN DIVERTICULAR DISEASE OF SMALL INTESTINE:

A perforated duodenal diverticulum may cause great deal of concern. When found the perforated diverticulum should be excised & duodenum is closed with serosal patch from jejunal loop. If the inflammation is severe gastro-jejunostomy or preferably duodenojejunostomy to be considered.

In case of jejunal and ileal diverticulum, resection with end to end anastomosis to be done. And the treatment of choice for Meckel's diverticulum is diverticulectomy.

DIVERTICULAR PERFORATION OF COLON:

When either an abscess or diverticulum ruptures into the peritoneal cavity, widespread bacterial contamination ensues with resultant generalized peritonitis. A conservative

approach can be taken with elderly and medically unfit patients with combined use of appropriate antibiotic therapy and regular review.

In patients who are fit for surgery are treated with vigorous resuscitation and antibiotic therapy. The aim of the surgery is to remove the source of sepsis and toilet the abdominal cavity. Resection of the affected colon is performed which has lower morbidity and mortality compared to non resection surgical procedures. Primary anastomosis is performed in emergency setting only if conditions are wholly favorable. If primary anastomosis is not favourable then resection of sigmoid colon with formation of end colostomy and blind rectal stump is performed in the first stage followed by colostomy taken down to reanastamose in second stage.(Hartmann's procedure - it is a classic two stage procedure).

PERFORATION IN ASCARIASIS:

The treatment consists of removal of all the worms up to ileoceacal valve and closure of the perforation. If the bowel is not viable resection and end-to-end anastomosis is done.

COLONIC PERFORATION IN AMOEBIC COLITIS:

Perforation may occur in caecum & recto-sigmoid junction. Usually perforation occurs in confined place where adhesions have previously formed. Local peritonitis develops followed by abscess formation. Laparotomy is performed as soon as patients condition is stabilized by fluids, antibiotics and gastrointestinal decompression. Mortality is very high and can reduced by early surgery. Faecal diversion by proximal colostomy or ileostomy

should be done. Colostomy is closed by 6 to 8 weeks once patient is recovered from amoebiasis and barium enema shows healthy colon. Resection of colon is rarely indicated since amoebic ulcerations heal with amoebicides. In case of retroperitoneal perforation or in those where paracolic leak is well localized it is advised that conservative regime of treatment to be tried, failure to respond fully will require open drainage of a large abscess under anti amoebic cover.

RESULTS AND ANALYSIS

This was hospital based prospective study of factors affecting the outcome in gastrointestinal perforations. Patients aged less than 18 years were excluded from this study. Even patients with history of blunt abdominal trauma were excluded from this study.

This prospective study had 100 patients, in which 81 were male and 19 were female. The age group of patients in this study ranges between 18 to 90 years. Maximum and equal number of patients was noted in three decades, from 31 to 60 years. Least number of patients was noted in the age group of more than 61 years.

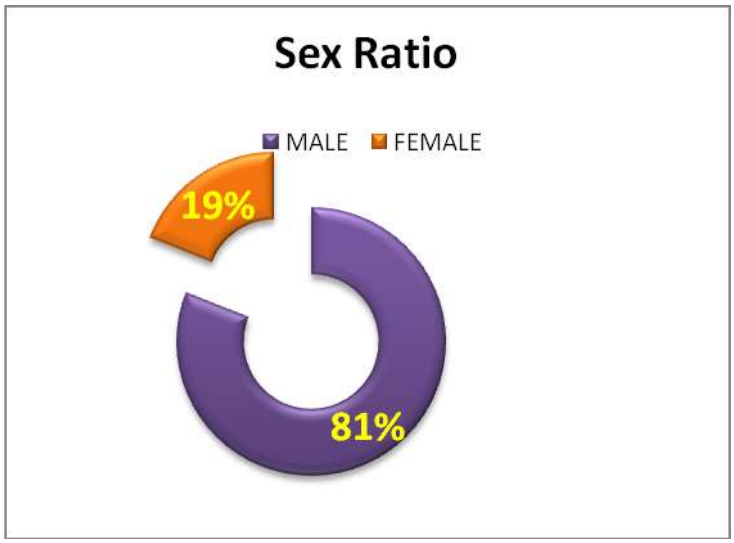
TABLE NO.1 Sex distribution of patients

MALE	**81**
FEMALE	19

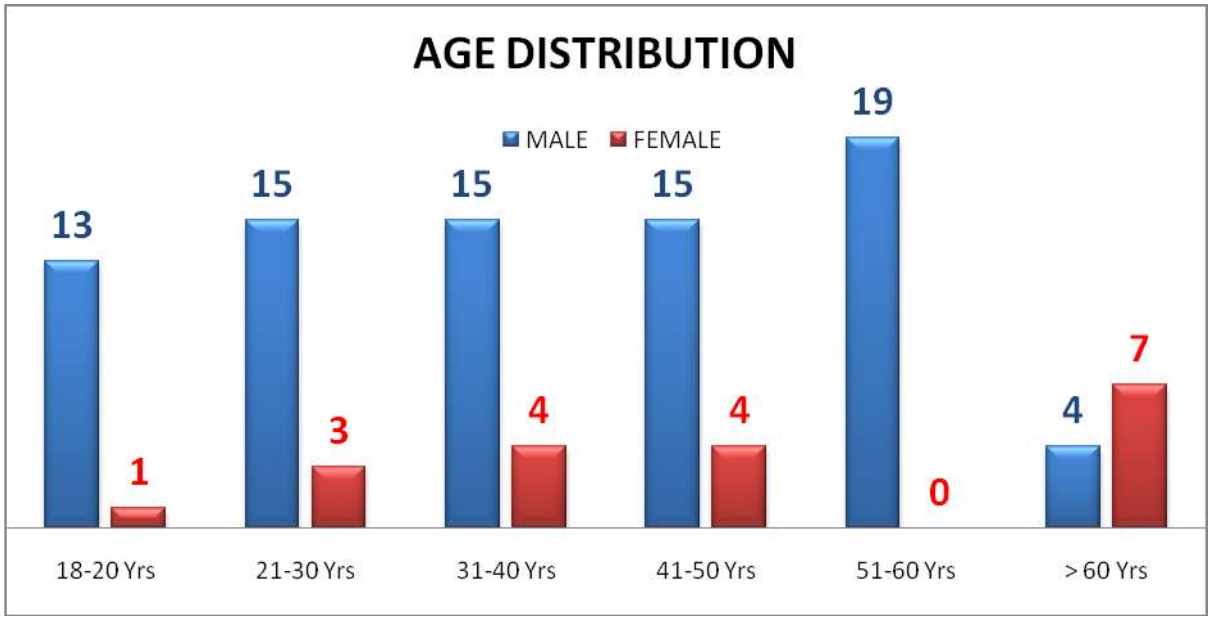
TABLE NO. 2 Age and sex distribution of patients

AGE GROUP	MALE	FEMALE	TOTAL
18-20 Yrs	13	01	14
21-30 Yrs	15	03	18
31-40 Yrs	15	04	19
41-50 Yrs	15	04	19
51-60 Yrs	19	00	19
> 60 Yrs	04	07	11
TOTAL	81	19	100

Graph No. 1 Sex Distribution of Patients



Graph. No 2 Age And Sex Distributions Of Patients



Out of hundred patients, duodenal perforation was most common cause for hollow viscus perforation, followed by appendicular perforations. Jejunal and colonic perforations were least common cause for hollow viscus perforations with a value of 6 each.

Table No. 3 Distribution Of Patients On Type Of Perforations.

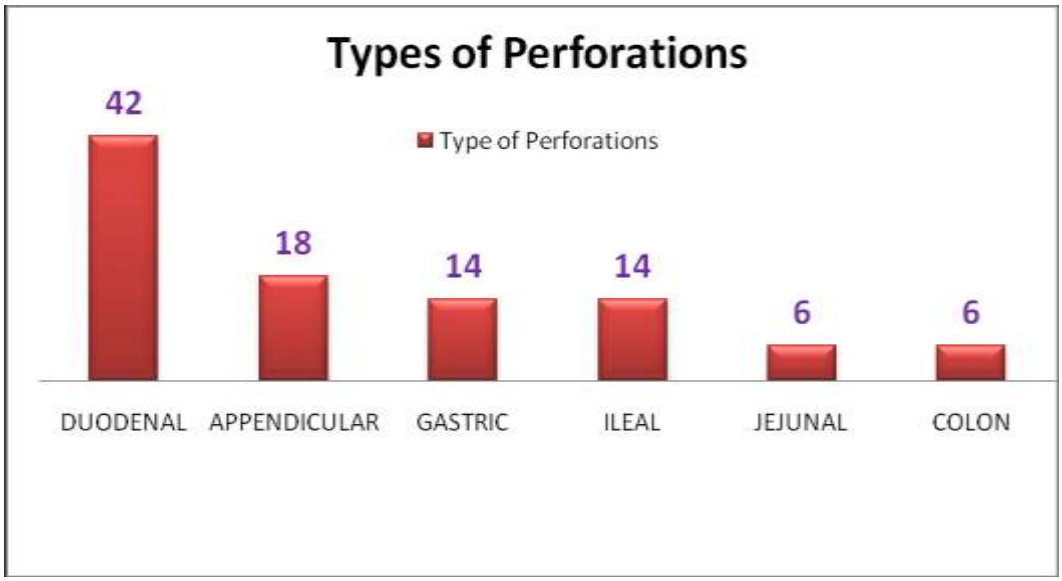
Types of Perforations	Number of patients
DUODENAL	42
APPENDICULAR	18
GASTRIC	14
ILEAL	14
JEJUNAL	06
COLONIC	06

Total mortality in this study was 11 (11 %) with 89 % of successful treatment. Out of 89 patients few had risk factors which made them to have increased morbidity compared to others.

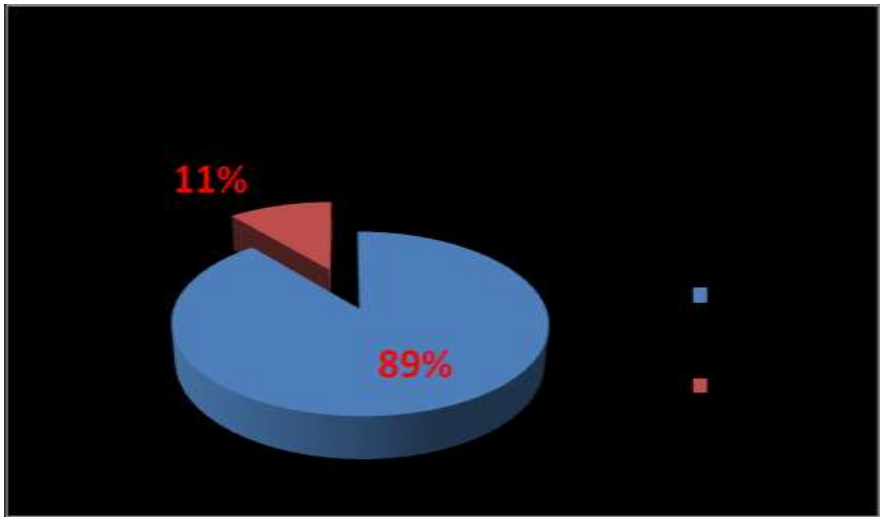
Table no. 4 Distribution of patients on the basis of successful treatment versus mortality.

Successful treatment versus Mortality	Number of patients
SUCCESSFUL TREATMENT	89
DEATHS	11

Graph No. 3 Distribution of Patients on Type Of Perforations



Graph No. 4 Distribution of patients on the basis of successful treatment versus mortality.

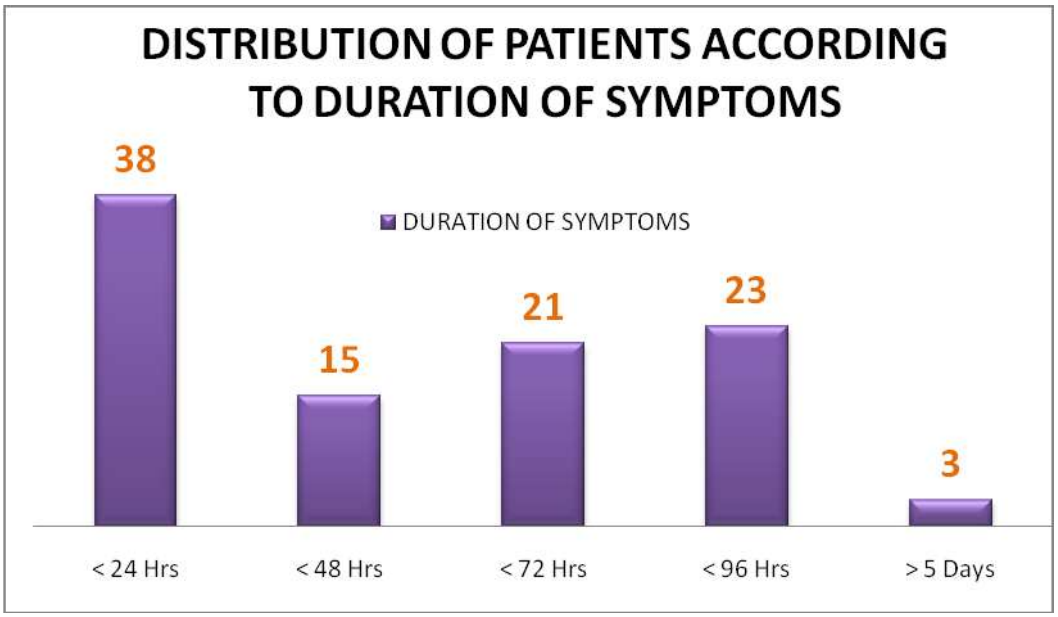


The duration of symptoms was defined as the time span between the initial pain perception due to perforation and the operation. 38 patients presented within 24 hours of pain perception and remaining 62 patients after 24 hours. Three patients presented after 5 days of pain perception.

Table No 5. Distribution of patients according to the duration of symptoms.

DURATION OF SYMPTOMS	NUMBER OF PATIENTS
< 24 HOURS	38
< 48 HOURS	15
< 72 HOURS	21
< 96 HOURS	23
> 5 DAYS	03

Graph no 5. Distribution of Patients According to the Duration Of Symptoms.



Most common post operative complication was post operative pneumonia in 15 patients, whereas most common surgical post operative complication was wound infection in seven patients. The least common surgical post operative complication was entero-cutaneous fistula in two patients.

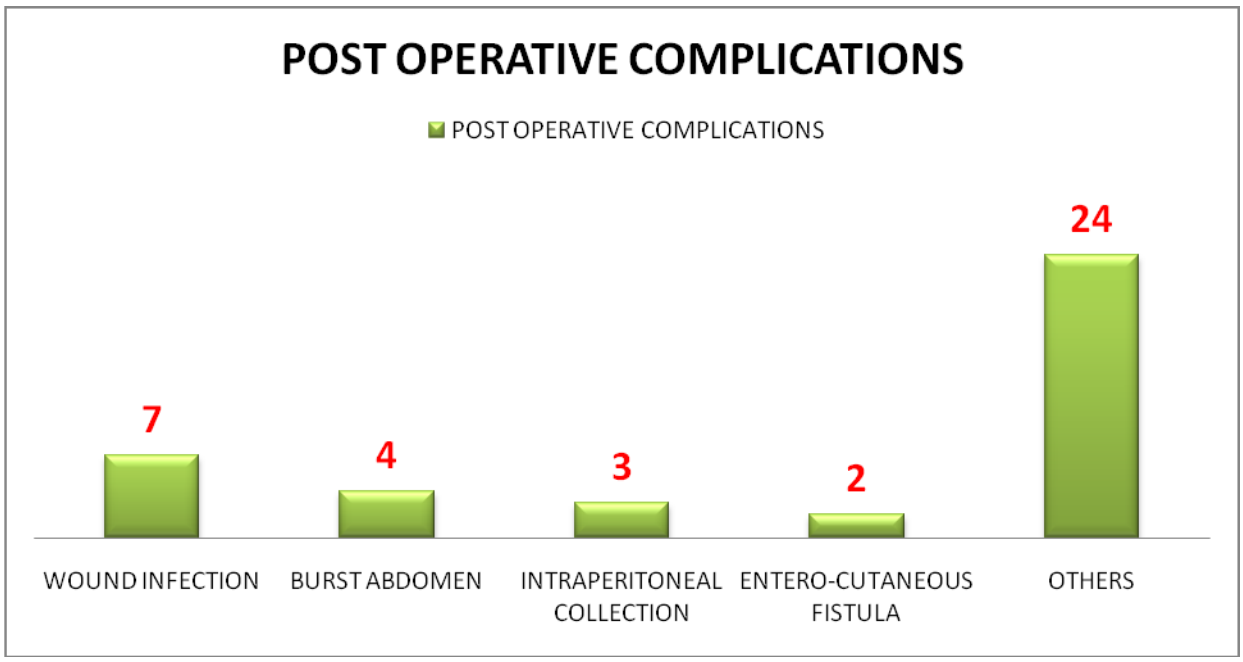
Table No 6. **Distribution of patients according to the post operative complications.**

POST OPERATIVE COMPLICATIONS	NUMBER OF PATIENTS
WOUND INFECTION	07
BURST ABDOMEN	04
INTRAPERITONEAL COLLECTION	03
ENTERO-CUTANEOUS FISTULA	02
OTHERS	24

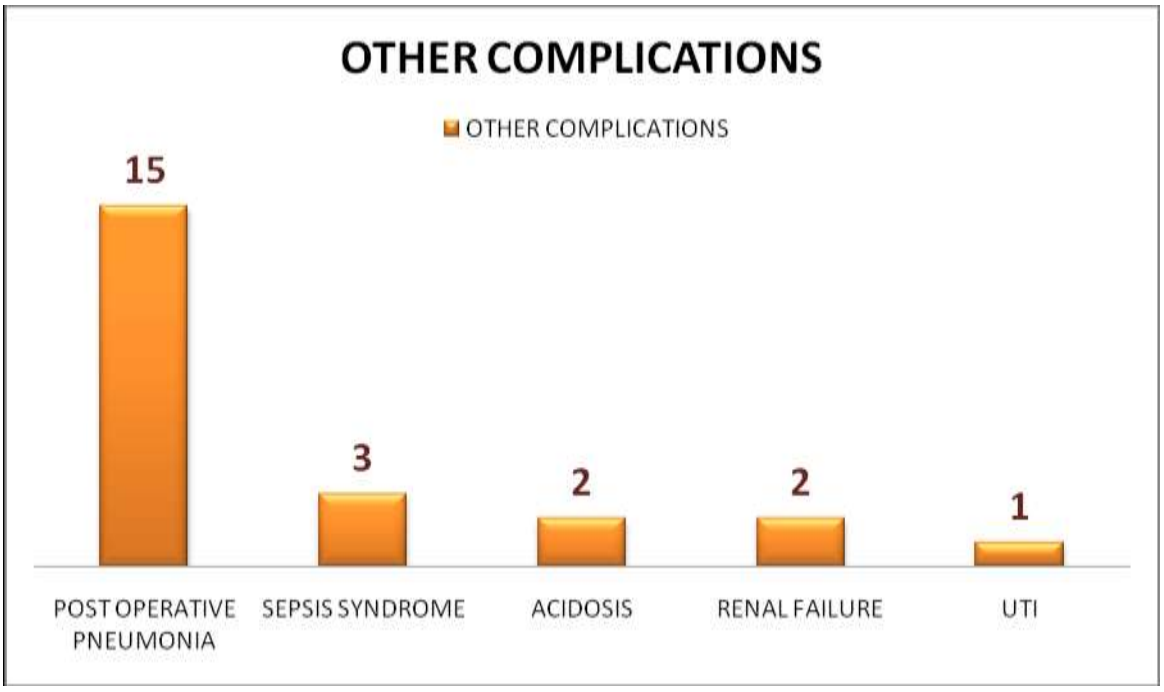
Table No 7. **Distribution of patients according to the non surgical post operative complications.**

OTHER COMPLICATIONS	NUMBER OF PATIENTS
POST OPERATIVE PNEUMONIA	15
SEPSIS SYNDROME	03
ACIDOSIS	02
RENAL FAILURE	02
URINARY TRACT INFECTIONS	01

Graph no 6. Distribution of patients according to the post operative complications.



Graph no 7. Distribution of patients according to the non surgical post operative complications.

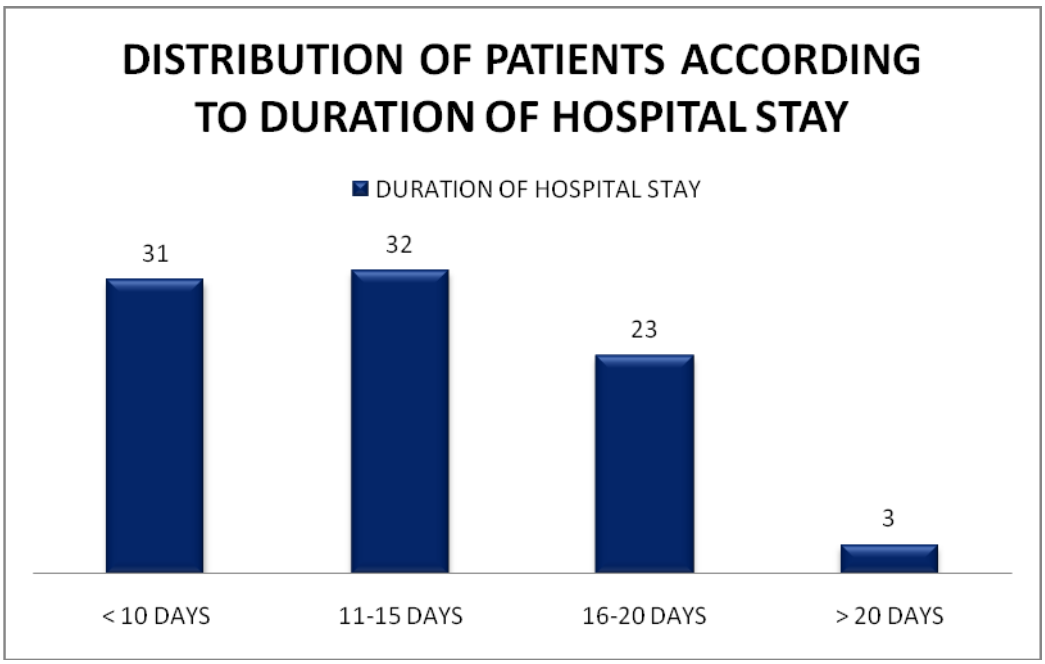


Morbidity of patients with hollow viscus perforation was considered on the basis of number of hospital stay in days. Average hospital stay in patients with no complications or co-morbid conditions was considered ten days. 31 patients were discharged on or before ten days of operation. Rest of the patients was discharged after ten days of operation.

Table no 8. Distribution of patients according to the duration of hospital stay.

DURATION OF HOSPITAL STAY	NUMBER OF DAYS
< 10 DAYS	31
11-15 DAYS	32
16-20 DAYS	23
> 20 DAYS	03

Graph no 8. Distribution of patients according to the duration of hospital stay.


DISTRIBUTION OF PATIENTS ACCORDING TO DURATION OF HOSPITAL STAY
DURATION OF HOSPITAL STAY
31
32
23
3
< 10 DAYS
11-15 DAYS
16-20 DAYS
> 20 DAYS

Average hospital stay in younger age group patients was 11 days whereas average hospital stay in elder age group was 14 days. The study shows average hospital stay in sixth decade was 13 days.

Table no 9. Average hospital stay according to the age group.

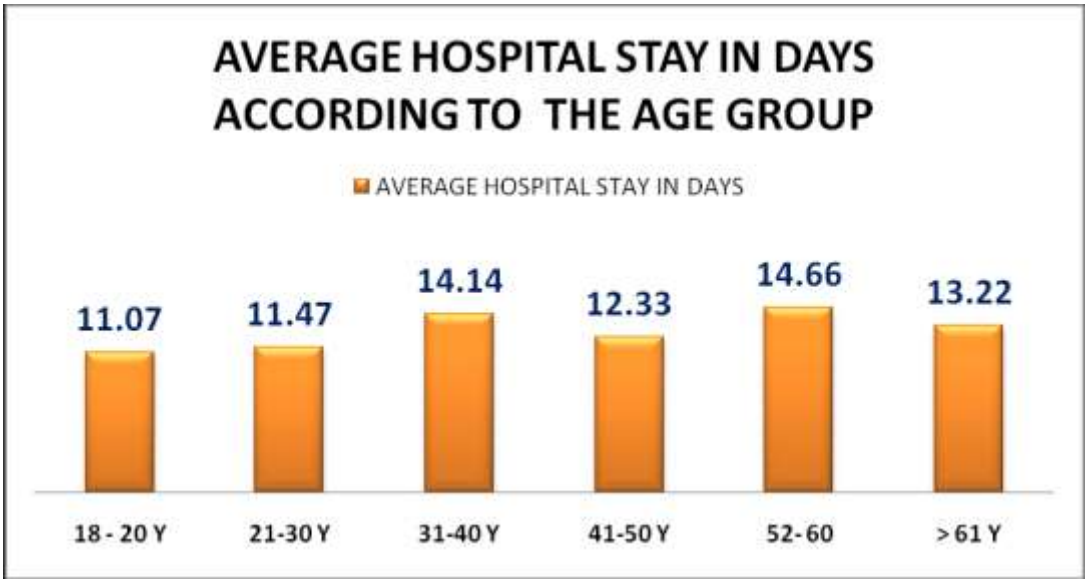
AGE GROUP	NUMBER	AVERAGE HOSPITAL STAY
18 - 20 Yrs	14	11.07 Days
21- 30 Yrs	17	11.47 Days
31-40 Yrs	14	14.14 Days
41- 50 Yrs	15	12.33 Days
51-60 Yrs	20	14.66 Days
> 61 Yrs	09	13.22 Days
TOTAL	89	

Patients with early presentation and operation had lesser morbidity compared to the patients with late presentation. 38 patients presented within 24 hours, in whom one patient died due to complications and remaining 37 patients had average hospital stay of 11 days. 18 patients presented after 4 days with 21.7 % of mortality and average hospital stay of 15 days. The percentage of mortality in patients with duration of symptoms of less than 24 hours is 5.2 % where as with patients with duration of symptoms of more than 5 days is 66.66 %.

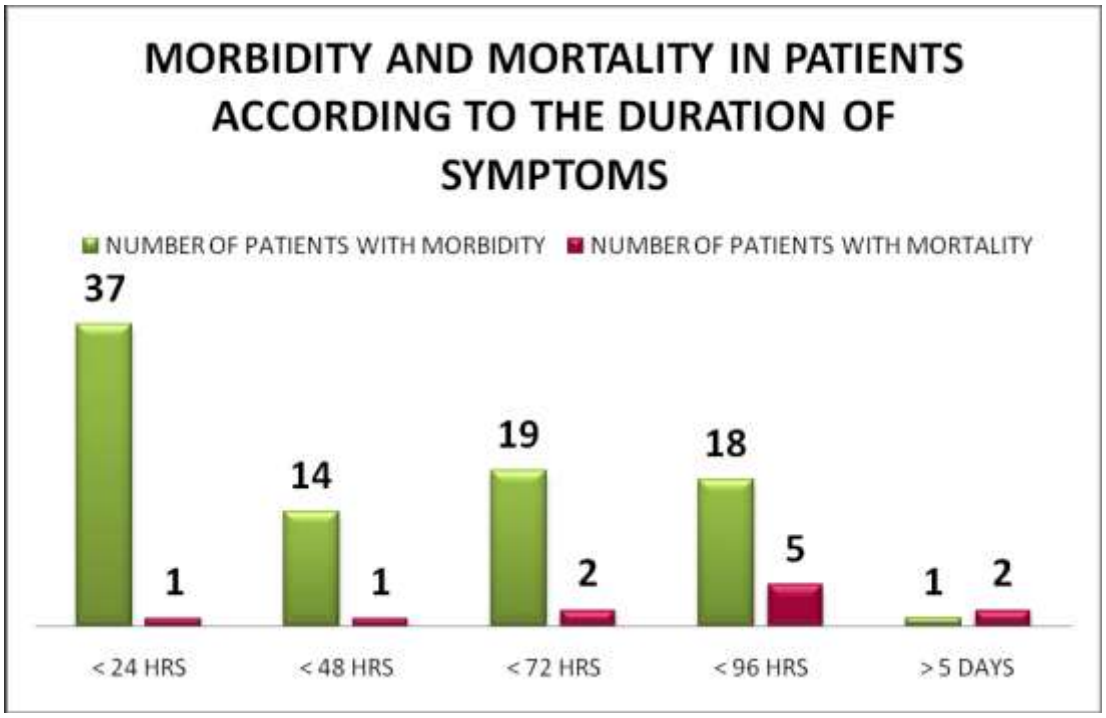
Table no 10. Average hospital stay according to the duration of symptoms.

DURATION OF SYMPTOMS	NUMBER OF PATIENTS	AVERAGE HOSPITAL STAY IN DAYS	NUMBER OF PATIENTS WITH MORTALITY
< 24 Hrs	37	11.16	01
< 48 Hrs	14	13.92	01
< 72 Hrs	19	15.63	02
< 96 HRS	18	15.27	05
> 5 DAYS	1	11	02
TOTAL	89		11

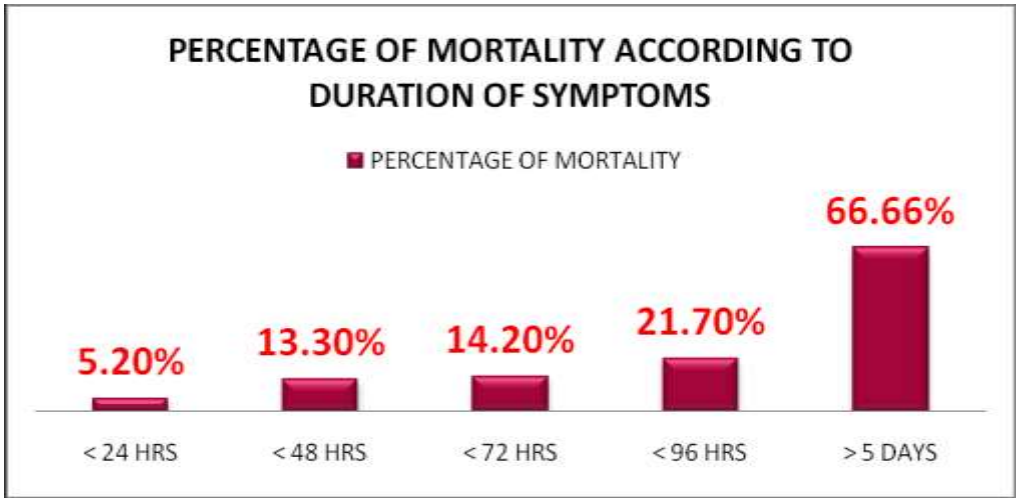
Graph no 9. Average hospital stay in days according to the age group.



Graph no 10. Morbidity and mortality in patients according to the duration of symptoms.



Graph no 11. Percentage of mortality according to duration of symptoms.



In this prospective study increased morbidity was noted in colonic perforations compared to other hollow viscus perforations. Patients with appendicular perforation had least average hospital stay of 10.5 days compared to other type of perforations.

Table no 11. Average hospital stay in days according to type of perforations.

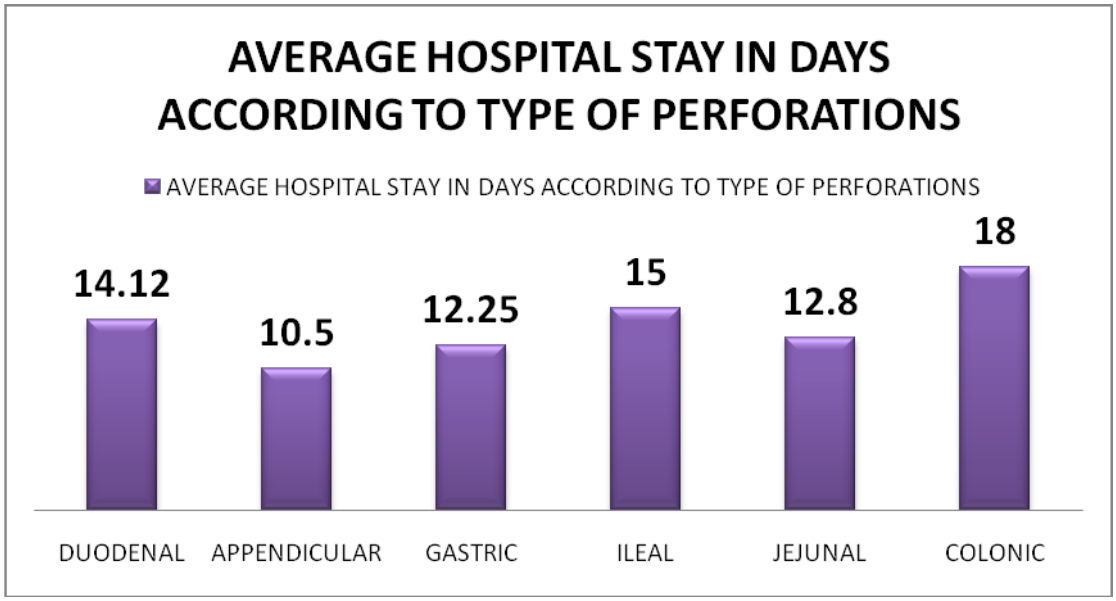
TYPE OF PERFORATIONS	NUMBER OF PATIENTS	AVERAGE HOSPITAL STAY IN DAYS
DUODENAL	40	14.12
APPENDICULAR	18	10.50
GASTRIC	13	12.25
ILEAL	10	15.00
JEJUNAL	05	12.80
COLONIC	03	18.00

Increased mortality is noted in colonic perforations with percentage of 50 % followed by ileal perforation with percentage of 28.57 %. No mortality is noted in appendicular perforations.

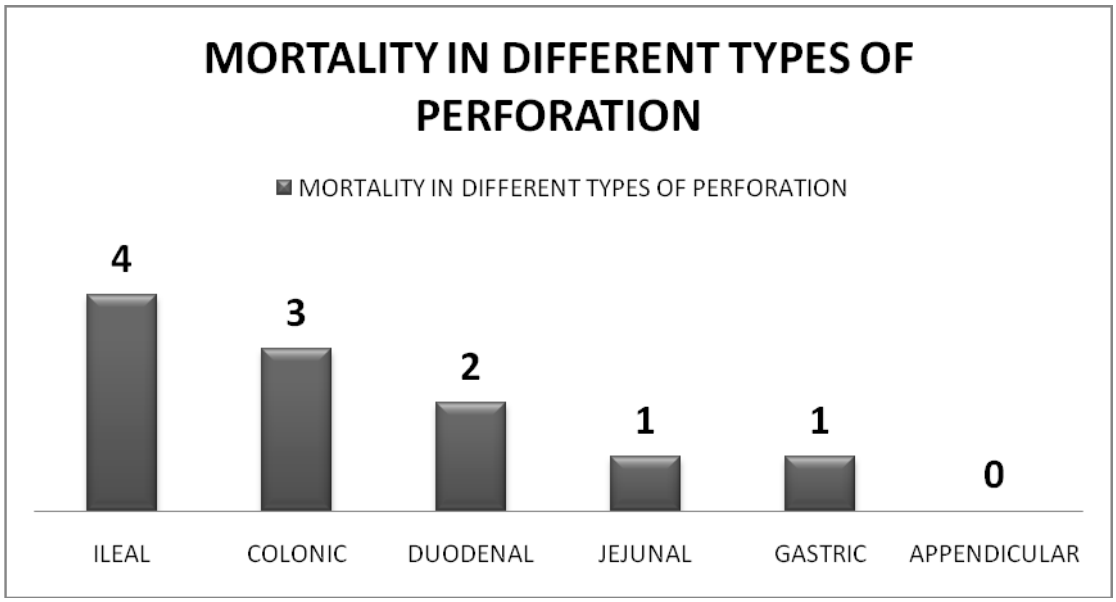
Table no 12. Mortality and Morbidity in different type of perforations.

TYPES OF PERFORATIONS	NUMBER OF PATIENTS	MORTALITY	PERCENTAGE
DUODENAL	42	02	4.7 %
APPENDICULAR	18	00	-
GASTRIC	14	01	07.1 %
ILEAL	14	04	28.57 %
JEJUNAL	06	01	16.6 %
COLONIC	06	03	50.00%
TOTAL	100	11	

Graph no 12. Average hospital stay in days according to type of perforations.



Graph no 13. Mortality in different types of perforations.



Out of hundred patients 15 presented with shock at the time of admission. Out of 15 patients 8 patients died.

Table no 13. Morbidity and mortality in patients presenting with shock

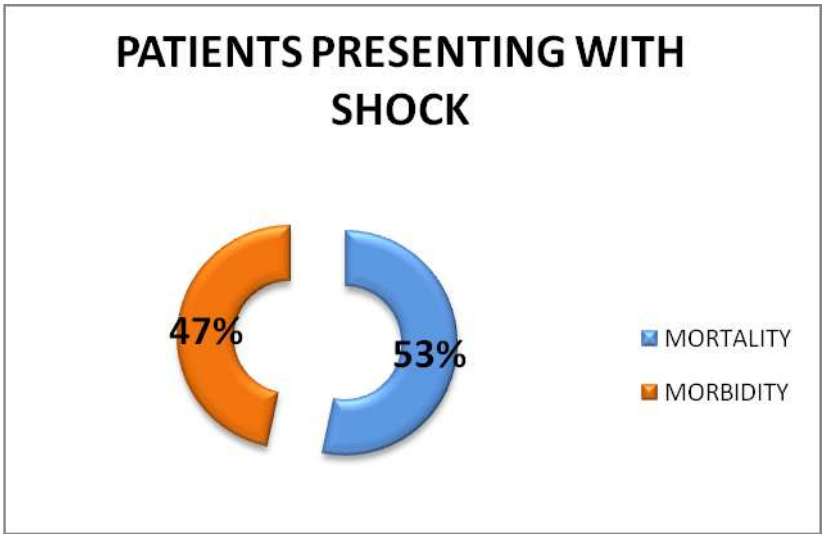
PATIENTS PRESENTING WITH SHOCK	NUMBER OF PATIENTS
MORBIDITY	07
MORTALITY	08
TOTAL	15

Seven patients who survived with shock at the time of admission had longer hospital stay compared to the patients without shock.

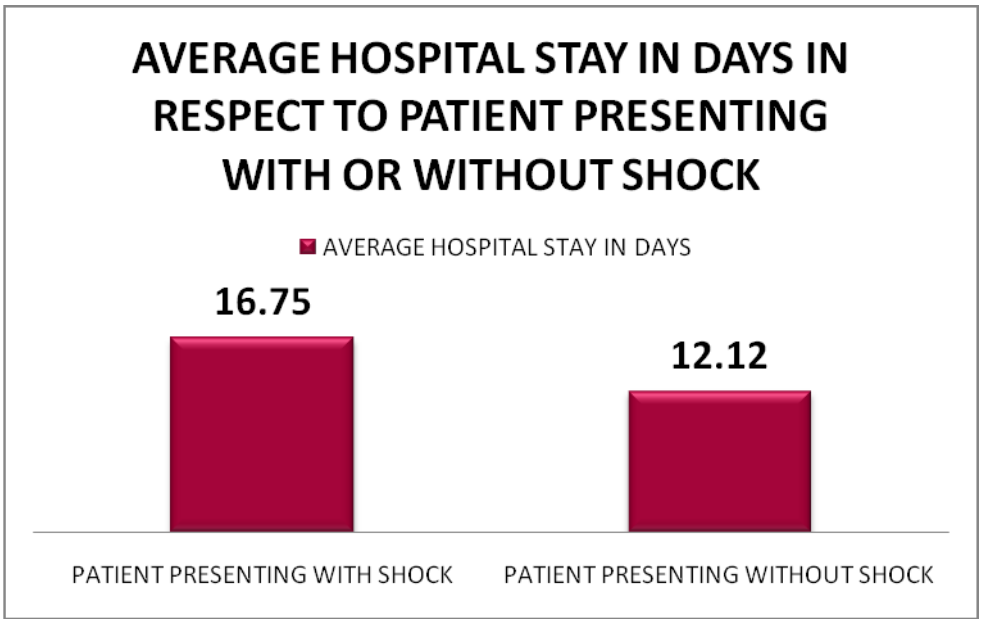
Table no 14. Average hospital stay in patients presenting with shock.

	AVERAGE HOSPITAL STAY IN DAYS
PATIENTS PRESENTING WITH SHOCK	16.75
PATIENT PRESENTING WITHOUT SHOCK	12.12

Graph no 14. Morbidity and mortality in patients presenting with shock



Graph no 15. Average hospital stay in patients presenting with shock.



36 patients had surgical and non surgical post operative complications, in whom eight patients succumb to death.

Table no 15. Morbidity and mortality in patients with post operative complications.

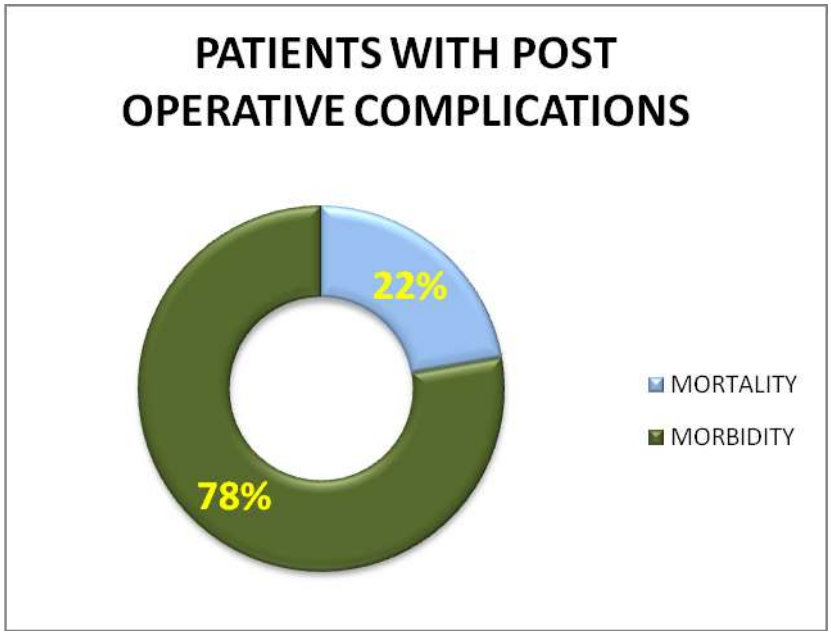
PATIENTS WITH POST OPERATIVE COMPLICATIONS	NUMBER OF PATIENTS
MORBIDITY	28
MORTALITY	08
TOTAL	36

28 patients who had post operative complication had longer average hospital stay compared to the rest of the patients without post operative complications.

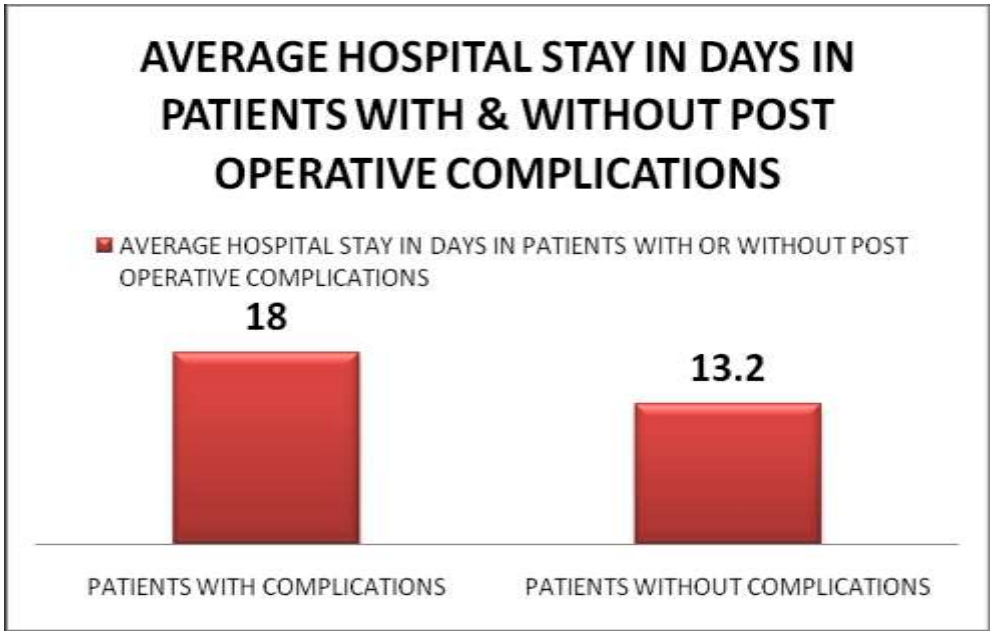
Table no 16. Average hospital stay in patients with or without post operative complications.

	AVERAGE HOSPITAL STAY IN DAYS
PATIENTS WITH POST OPERATIVE COMPLICATIONS	18.00
PATIENTS WITHOUT POST OPERATIVE COMPLICATIONS	13.20

Graph no 16. Morbidity and Mortality in patients with post operative complications.



Graph no 17. Average hospital stay in patients with or without post operative complications.



14 patients who presented with hollow viscus perforations had preexisting co-morbid conditions like asthma, diabetes mellitus and hypertension. Two patients with co-morbid conditions succumb to death.

Table no 17. Morbidity and mortality in patients with co-morbid conditions.

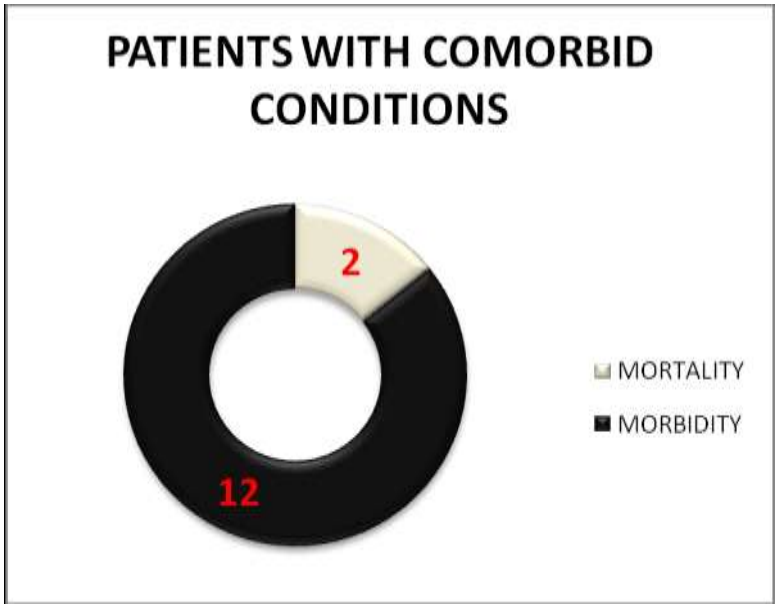
PATIENTS WITH COMORBID CONDITIONS	NUMBER OF PATIENTS
MORBIDITY	12
MORTALITY	2
TOTAL	14

Average hospital stay for patients with co-morbid conditions was much higher with a value of 18.9 days compared to patients with no co-morbid conditions.

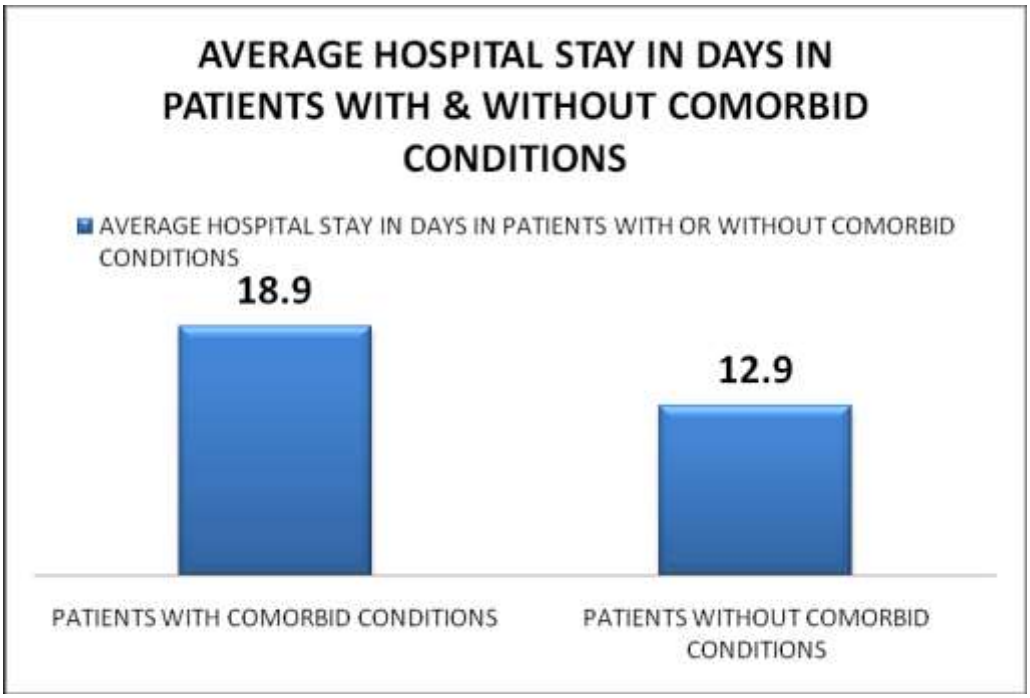
Table no 18. Average hospital stay in patients with or without co-morbid conditions.

	AVERAGE HOSPITAL STAY IN DAYS
PATIENTS WITH COMORBID CONDITIONS	18.90
PATIENTS WITHOUT COMORBID CONDITIONS	12.90

Graph no 18. Mortality and morbidity in patients with co-morbid conditions.



Graph no 19. Average hospital stay in patients with or without co-morbid conditions.



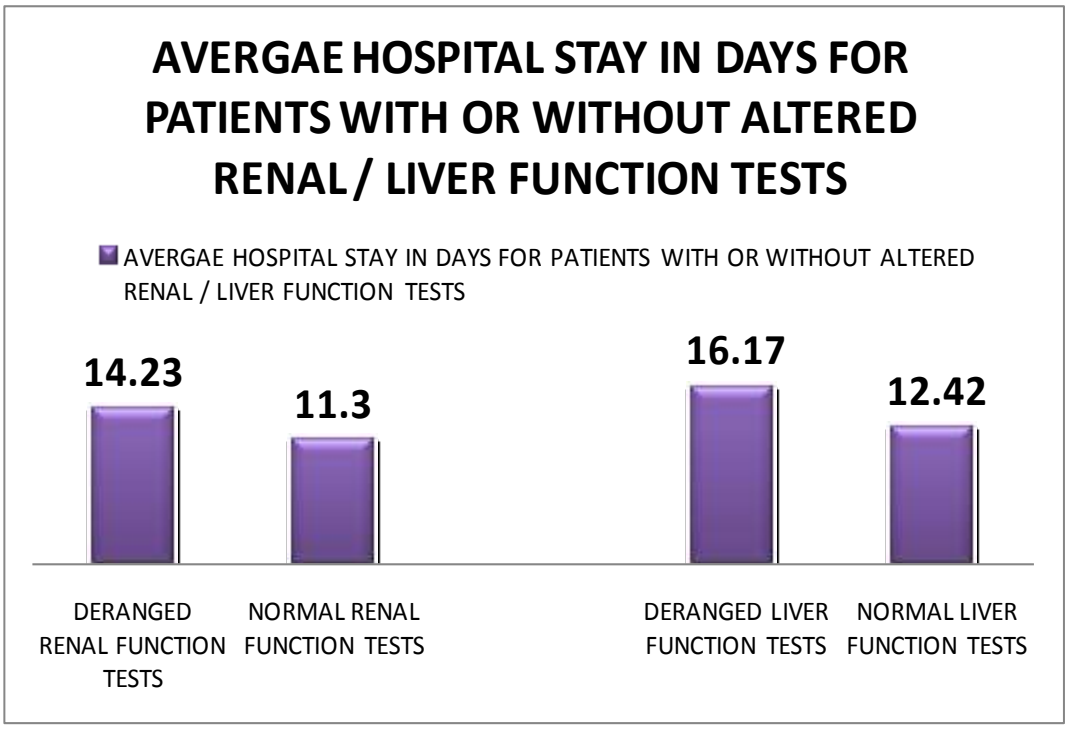
Renal function tests were conducted in all patients with hollow viscus perforations. 31 patients had deranged renal functions on admission, in whom ten patients succumb to death whereas remaining 14 patients had longer average hospital stay.

Liver function tests were conducted in 40 patients with hollow viscus perforation. 24 patients had deranged liver functions on admission in which 7 patients died. Remaining patients with deranged liver function tests had longer average hospital stay compared to the patients with normal liver function

Table no 19. Morbidity & mortality in patients with deranged liver or renal function tests.

	NUMBER OF PATIENTS	AVERAGE HOSPITAL STAY	MORTALITY	PERCENTAGE OF MORTALITY
DERANGED RENAL FUNCTION TESTS	31	14.23	10	32.25 %
DERNAGED LIVER FUNCTION TESTS	24	16.17	7	29.1 %

Graph no 20. Morbidity & mortality in patients with deranged liver or renal function tests.



STATISTICAL ANALYSIS.

All calculations were performed with statistical soft ware. The Chi-square test was used to compare proportions. Probability (*p*) value of less than 0.05 was considered statistically significant.

	N	*P* value
• **Male : Female**	81:19	NS
• **Age**	< 50 : > 50 Yrs	0.04
• **Average hospital stay according to age group**	18 -30 : 31-50 Yrs (31:49)	0.03
• **Average hospital stay according to duration of symptoms**	< 24 Hrs : > 96 Hrs (37 : 18)	0.009
• **Mortality in patients with shock**	08: 100	0.007
• **Mortality in post operative complications**	08 : 100	0.005
• **Mortality in patients with co-morbid conditions**	02 : 100	0.04

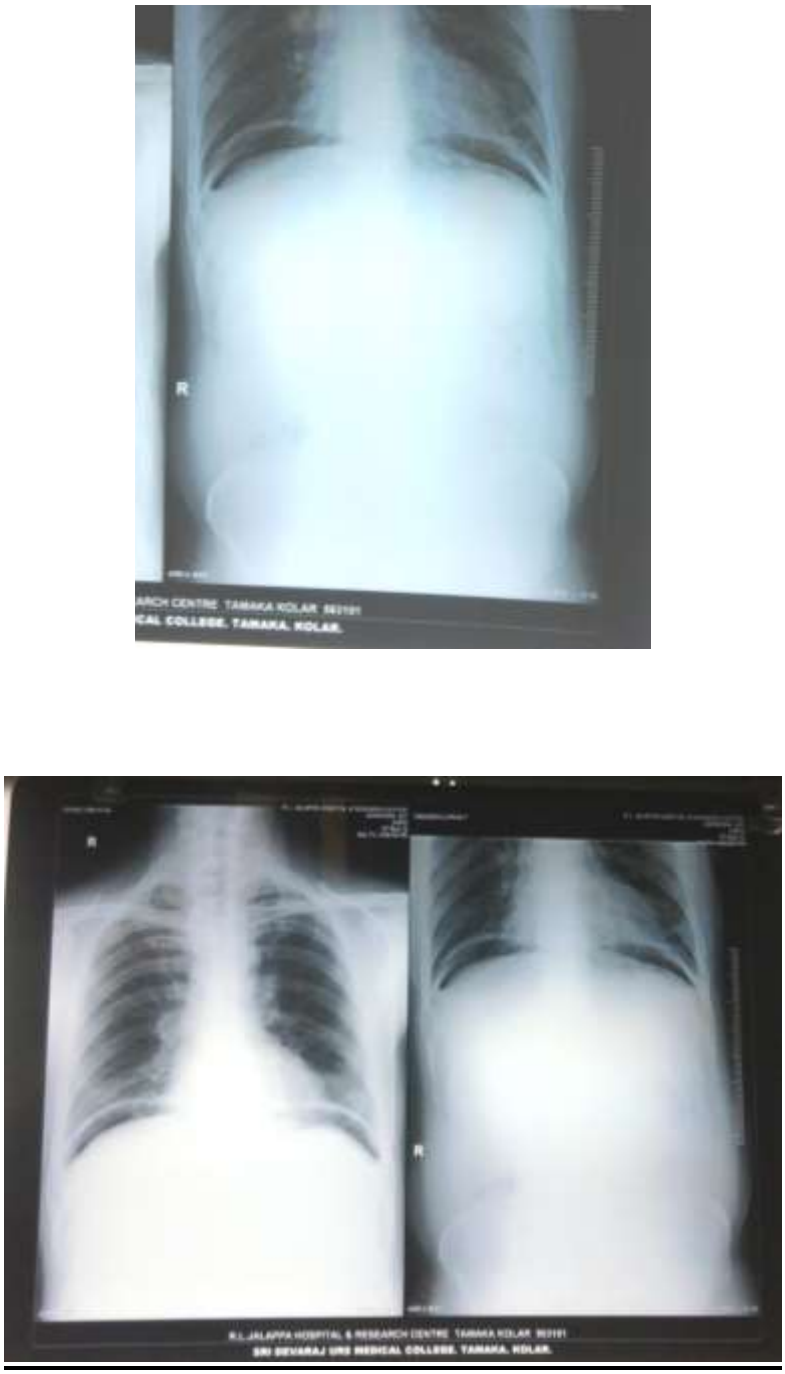


Figure no 9. Erect abdomen and chest radiographs with air under domes of diaphragm.

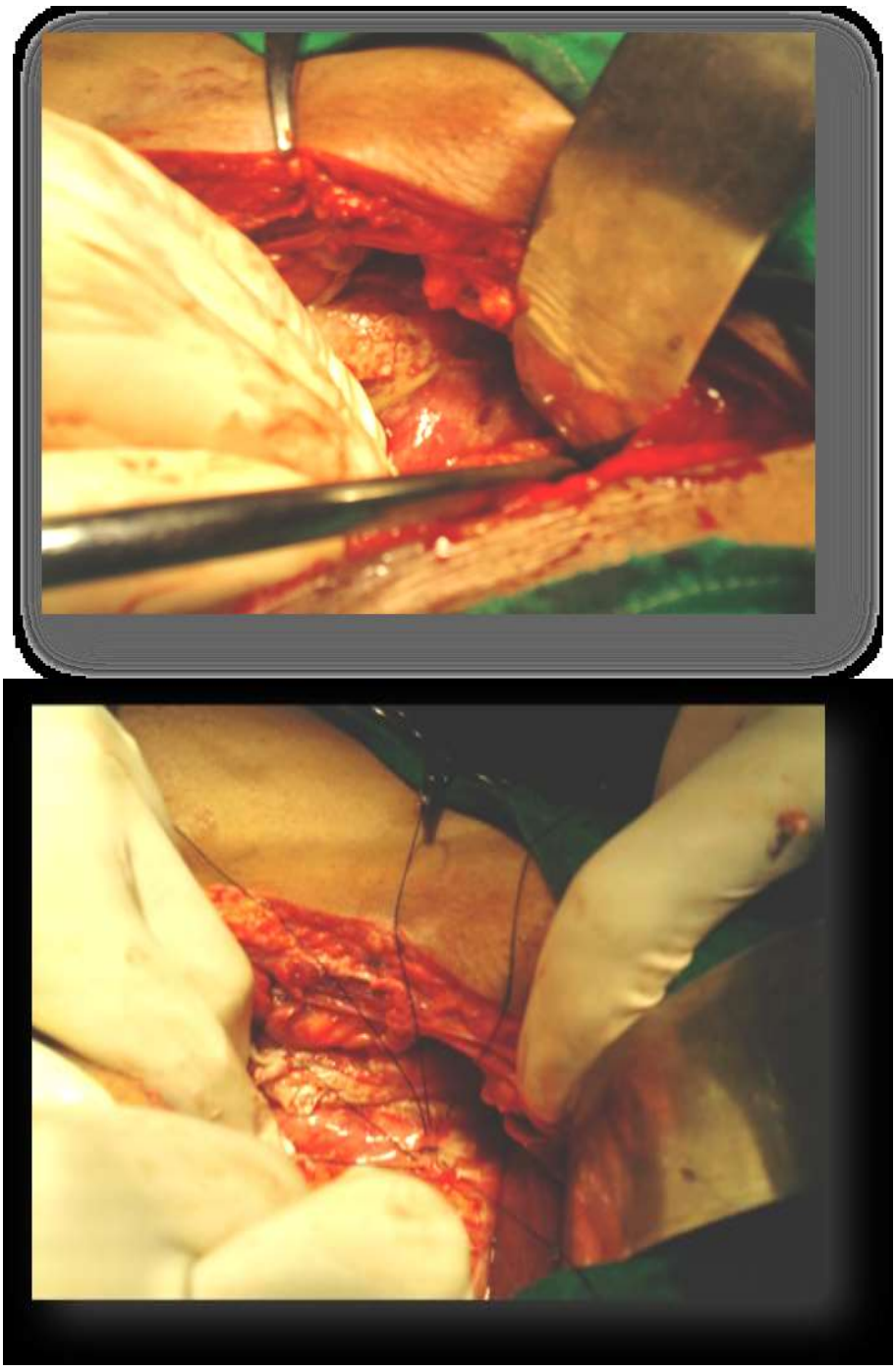


Figure no 10 . Duodenal perforation and primary closure of perforation.

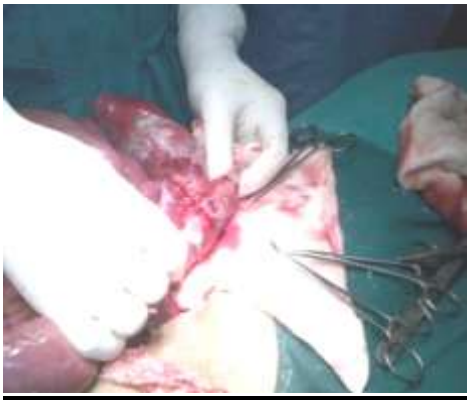


Figure no 11. Terminal ileum perforation and primary closure of perforation.

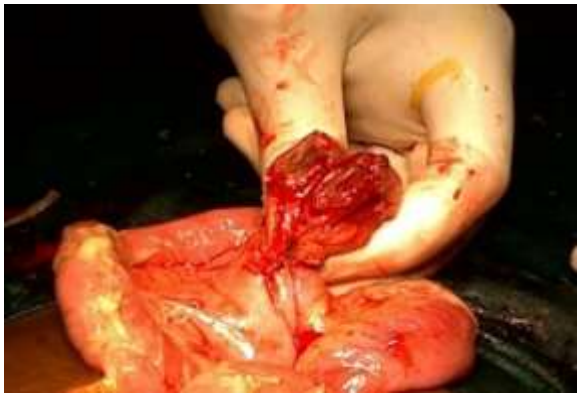


Figure no. 12. Resected Ileal Segment

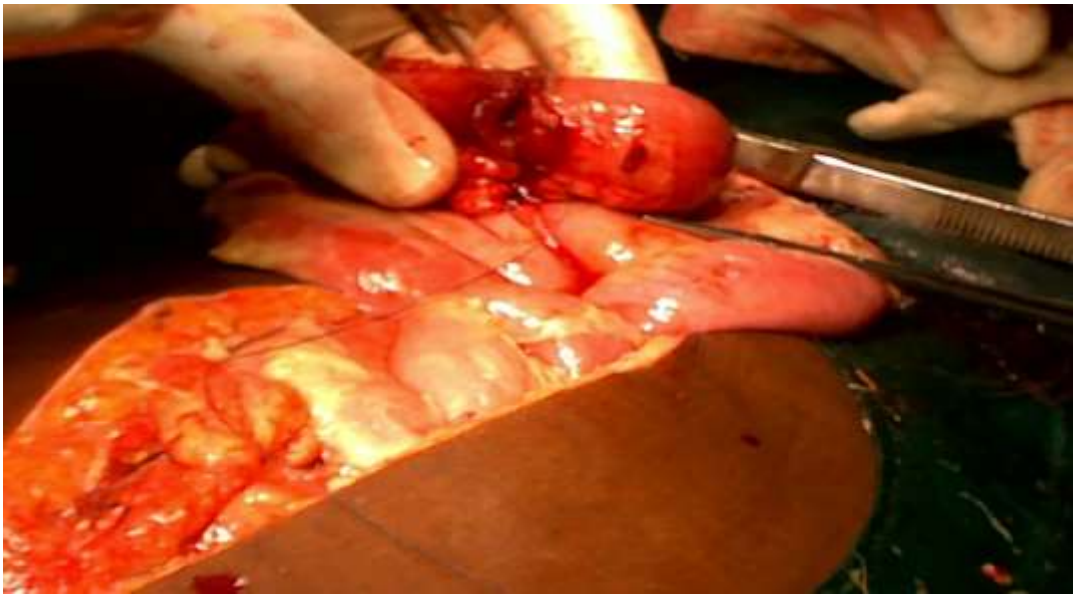


Figure no 13. Resection anastomosis for Ileal Perforation

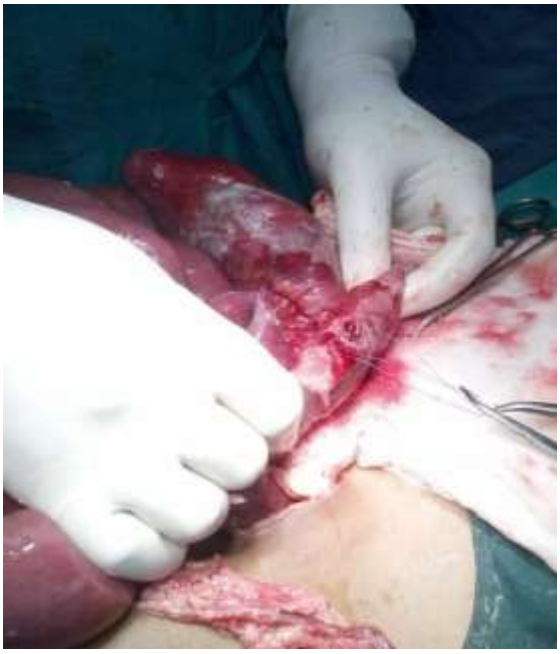


Figure no 14. Ileal perforation with primary closure of perforation.

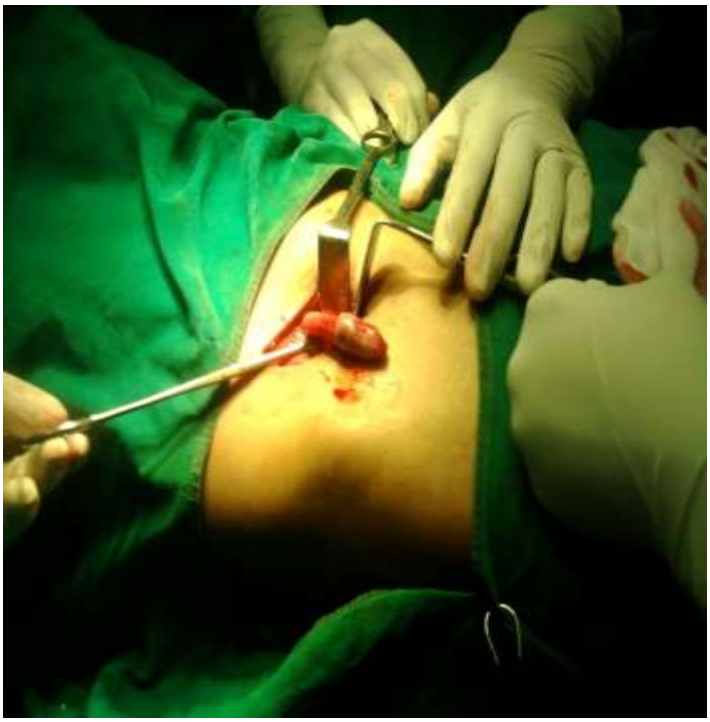


Figure no. 15 Perforated appendix at the tip

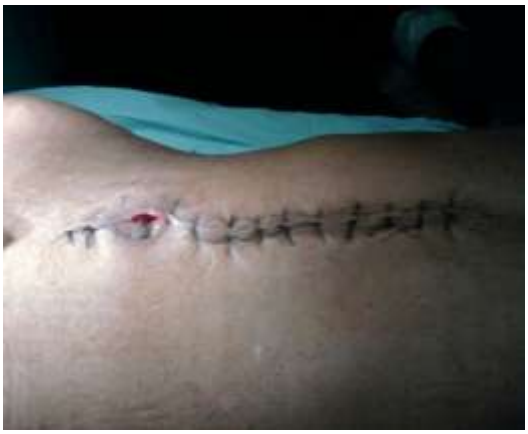


Figure no 16. Wound infection

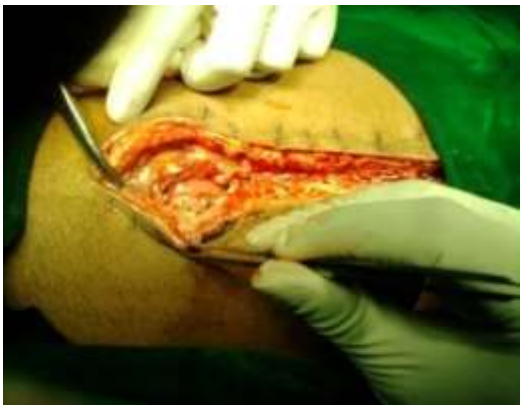


Figure no 17. Burst Abdomen

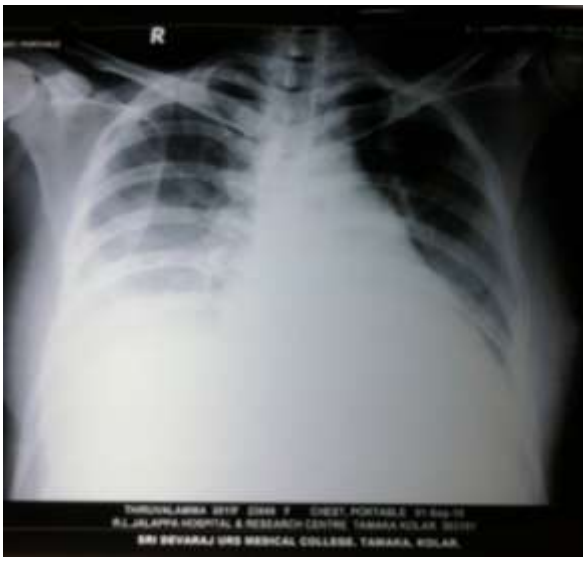


Figure no 18. Post operative pneumonia.

DISCUSSION

Intestinal perforations are common causes of generalized peritonitis. Despite advances in surgical techniques, antimicrobial therapy and intensive care support, management of peritonitis continues to be highly demanding, difficult and complex[1]. The spectrum of etiology of perforation continues to be different from that of western countries and there is paucity of data from India regarding it's etiology, prognostic indicators, morbidity and mortality patterns[3]. This is a hospital based prospective study conducted in and around Kolar at rural setup to determine the factors affecting the outcome in gastrointestinal perforations.

The study includes total number of 100 patients in which 81 were male and 19 were female patients. A similar study conducted at Chandigarh by Rajendrasingh et al shows the males being affected in gastrointestinal perforations is 84 % compared to the 16 % in females[1].

In our study the young & middle age group (70 %) is most commonly affected as compared to the elder age group (30 %). Maximum and equal number of patients with hollow viscus perforations were noted in 2nd to 5th decade. A western study conducted by Suanes et al shows most common age group affected in gastro-intestinal perforations is elder age group[46]. But similar studies conducted in India at Chandigarh shows young population will have major brunt of gastrointestinal perforations as compared to the elderly age group[1].

Duodenal perforations are most common cause of hollow viscus perforations in our study followed by appendicular perforations. Whereas Jejunal and colonic perforations are least common causes. The perforations of proximal gastrointestinal tract is six times as common as perforations of distal gastro-intestinal tract as has been noted in earlier study conducted by Dorairajan L N et al[47]. In sharp contrast to our study, the studies from developed countries like United states, Greece & Japan reveal distal gastrointestinal tract perforations are more common compared to the proximal gastrointestinal tract[1].

89 % of patients had successful treatment with or without morbidity. The overall mortality rate in our study is 11 % with septicemia associated multiorgan dysfunction syndrome (MODS) is most common cause followed by entero-cutaneous fistula and respiratory failure with acidosis. Seven out of 11 died due to multiorgan dysfunction syndrome secondary to irreversible septic shock. Two patients with entero-cutaneous fistula died secondary to sepsis, malnutrition & renal failure. Two patients died due to respiratory failure secondary to post operative pneumonia, one of which had respiratory acidosis with metabolic alkalosis.

Duration of symptoms is defined as time span between initial pain perception due to perforation and the surgery. 38 patients had early presentation compared to 62 patients with late presentation in our study. Study conducted by Rajendrasingh et al shows 47 % of patients had early presentation[1].

In our study 40 patients had post operative complications which resulted in increased morbidity and mortality. Most common post operative complication was post operative pneumonia in 15 patients. The most common surgical post operative complication was wound infection in seven patients. The fatal complication was entero-cutaneous fistula with 100 % mortality. The study conducted by Rajendrasingh et al shows post operative pneumonia as most common post operative complication followed by wound infection[1]. A prospective study conducted in Nigeria on typhoid ileal perforations shows wound infection as most common post operative complication followed by wound dehiscence. The mortality was 75 % in patients with entero-cutaneous fistula[48].

Average hospital stay in patients with no complications or co-morbid conditions was arbitrarily considered as ten days in our study. 31 patients were discharged on or before ten days of surgery. Depending upon the average hospital stay in days morbidity has been calculated for different factors affecting the outcome of gastro-intestinal perforations.

The study shows shorted average hospital stay of 11 days in younger patients compared to the 14 days 14 elder age group. Increased hospital stay in elder age group is because of co-morbid conditions and complications.

Patients with early presentation and early operative treatment had lesser morbidity compared to the patients with late presentation. Average hospital stay in patients presenting after 24 hours of pain perception is 14 to 15 days compared to the patients with early presentation who had average hospital stay of 11 days.

The percentage of mortality is significantly increased as duration of symptoms increased. The mortality in patients with early presentation is 5.2% compared to the patients with duration of symptoms of more than 5 days is 66.66 %. This indicates there is high fatality in patients with late presentation. A study conducted in Turkey by G Bas et al shows that the risk of morbidity and mortality is statistically significant in patients with late presentation (> 24 hours)[49]. Another study conducted in Nigeria by A R K Adesunkanmi shows surgical intervention sharply reduces the mortality from 70-100 % to about 30 %, but also early surgical intervention further improved the prognosis[48]. Similarly even the literature emphasis the early surgical intervention to improve the outcome.

The mortality was higher in colonic perforations with average hospital stay of 18 days followed by ileal perforations with 15 days. Appendicular perforations had least number of average hospital of 10 days. Similarly there is significant mortality in patients with colonic perforations (50 %) and ileal perforations (28.7%). However the mortality in appendicular perforations was nil. A study conducted by Ali Nuhu et al at Azare on typhoid ileal perforations shows 28.3 % mortality[50]. Kriwanek S conducted study in Austria to evaluate the prognostic factors in colonic perforations and showed 17 % mortality in patients[51]. Thomas M Beaver conducted study on colon perforation after lung transplantation and showed mortality of 50 %[52].

15 patients presented with shock at the time of admission in whom eight succumb to death and remaining seven had increased morbidity in terms of longer hospital stay

(16.75 days). Mario Testini et al shows 55.6 % mortality in patients with shock at the time of admission[53]. Similar studies conducted by Boey J and McIntosh JH also show greater mortality in patients with shock at the time of admission[54,55].

Out of 100 patients 36 patients suffered from post operative complications with mortality rate of 22.22 %. Rest of the patients had longer hospital stay of 16.75 days. Mario Testini et al show 37.5 % mortality in patients with post operative complications[53].

14 patients had preexisting co-morbid conditions at the time of admission like, asthma, diabetes mellitus and hypertension. The mortality rate in patients with co-morbid conditions was 14.28 %. The survivors had longer average hospital stay of 19 days compared to the patients with no co-morbid conditions. A study conducted in Italy by Mario Testini shows 7.6 % mortality rate in patients with co-morbid conditions[53]. However the study conducted by G Bas on perforated peptic ulcer shows no importance of co-morbid conditions on morbidity and mortality of patients[49].

The indented hypothesis of study of factors affecting the outcome of gastrointestinal perforations and their impact on morbidity and mortality of patients is determined. The factors like advanced age group, delayed presentation, type of perforation, shock on admission, post operative complications and associated co-morbid conditions will bear major brunt on morbidity and mortality of patients with hollow viscus perforations and this fact is revalidated in this prospective study.

SUMMARY AND CONCLUSION

In conclusion the factors like advanced age group, delayed presentation, type of perforation, general condition at the time of admission, associated co-morbid conditions and post operative complications are associated with increased morbidity and mortality in patients with hollow viscus perforations.

Most common type of hollow viscus perforation was duodenal perforation followed by appendicular perforation.

Colonic perforation had highest mortality followed by ileal perforation.

Chest infection was most common post operative complication whereas wound infection was the most common surgical complication, least being entero-cutaneous fistula with highest mortality thus should be handled carefully with appropriate prevention and further management accordingly.

Causes of death in patients presented with peritonitis secondary to hollow viscus perforation could be as, multiorgan dysfunction syndrome secondary to irreversible septic shock, entero-cutaneous fistula with secondary sepsis, malnutrition & renal failure and respiratory failure secondary to post operative pneumonia.

Morbidity and mortality could be reduced by avoiding delay in diagnosis and treatment, especially in older age group, by instituting proper treatment of coexisting medical illness & shock and thorough and proper treatment of post operative complications.

Not only the surgical interventions will improve the outcome of the patients but also much emphasis is given to the timely intervention with appropriate treatment in patients presented with peritonitis secondary to hollow viscus perforation.

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ANNEXURE

PROFORMA

Name : IP. NO:

Age / Sex : Unit :

Religion / Occupation : Address:

DOA & DOD : DOS :

Clinical diagnosis:

Operative diagnosis:

Chief complaints with Duration:

HISTORY OF PRESENTING ILLNESS:

1. Pain Duration:

   Time of onset:

   Site of pain / radiation / referred pain:

   Related to posture:

   Aggravating and relieving factors:

2. Vomiting Duration, character, contents, frequency & quality.

   Related to pain.

3. Fever

4. Distension of abdomen

5. Haematemesis/Melaena

6. Last food intake.

7. History of drug intake.

8. Shock.

PAST HISTORY: H/o fever, peptic ulcer disease.

PERSONAL HISTORY:

Diet :	Appetite :
Weight Loss :	Sleep :
Bladder :	Bowel :
Habits :	

FAMILY HISTORY:

GENERAL EXAMINATION:

Attitude	Appearance	Nutritional status
Facies	Anemia	Jaundice
Dehydration	Cyanosis	Edema
Clubbing	Lymphadenopathy.	

Vital Data

Temperature :	Pulse :
Respiratory rate :	Blood pressure :

LOCAL EXAMINATION:

Inspection:

Contour of abdomen	Movements with respiration	Umbilicus
Skin over abdomen	Hernia orifices	Flanks.

Palpation :

Tenderness	Guarding & rigidity
Distension	Organomegaly.

Auscultation:

Bowel sounds.

Per rectal and per vaginal examination.

OTHER SYSTEMIC EXAMINATION :

Respiratory system.

cardiovascular system.

Nervous system.

LABORATORY INVESTIGATIONS:

Complete hemogram:

ESR, Blood grouping, Rh typing, Bleeding time and clotting time

Prothrombin time and activated partial thromboplastin time

Renal function tests Liver function tests

Blood glucose Blood culture sensitivity

Widal test for enteric fever

Urine analysis:

Peritoneal fluid for staining and culture sensitivity.

Chest radiograph Ultrasound abdomen ECG

Provisional diagnosis

Treatment:

Operative treatment:

Post operative period:

Condition at the time of discharge:

Advice :

Sl.No	Name	Age / Sex	DOA	IP.NO	Presenting Complaints			Duration	Past history						General Condition			Abdominal examination findings					Investigations		
					Pain abd	Vomiting	Fever		Pain abd	Fever	TB	Co-morbid	H/O Drug i	Habit of alc	Dehydrtaio	Tachycardi	Shock	Tendernes	Guarding	Rigidity	Free fluid	Bowel sou	Abdominal	Electrolyte	RFT
1	Rajesh	20 Y / M	20.06.2008	432545	Present	Present	Present	> 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Absent	Absent	Present	Normal	Normal
2	Reshma	25 Y / F	28.06.2008	434045	Present	Present	Present	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Absent	Absent	Present	Normal	Normal
3	Imran	19 Y / M	29.06.2008	434253	Present	Present	Present	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Absent	Absent	Present	Normal	Normal
4	Imthiaz	31 Y / M	01.07.2008	432848	Present	Present	Absent	> 72 Hrs	Present	Nil	Nil	Nil	Nil	Nil	Nil	Absent	Nil	Present	Absent	Absent	Absent	Present	Present	Normal	-
5	VenkataR	45 Y / M	11.07.2008	437490	Present	Absent	Absent	> 96 Hrs	Present	Nil	Nil	Nil	Present	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Deranged
6	Devaraj	42 Y / M	12.07.2008	437530	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
7	Pandurang	63 Y / M	20.09.2008	452126	Present	Absent	Absent	<24 hrs	Present	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
8	Vitthal	53 Y / M	21.09.2008	452198	Present	Absent	Absent	<24 hrs	Present	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
9	Govindapp	40 Y / M	11.10.2008	456828	Present	Present	Absent	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
10	Najma Beg	21 Y / F	05.11.2008	462262	Present	Present	Present	> 96 Hrs	Present	Present	Present	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
11	Babu	58 Y / M	02.12.2008	468452	Present	Present	Absent	< 24 Hrs	Present	Nil	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
12	Naseemba	40 Y / F	24.12.2008	476572	Present	Present	Present	> 72 Hrs	Present	Present	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
13	Chinnaswa	45 Y / M	30.12.2008	478910	Present	Present	Absent	> 72 Hrs	Present	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
14	Maqbul	27 Y / M	20.01.2009	479190	Present	Present	Absent	> 48 Hrs	Nil	Nil	Nil	DM	Nil	Present	Nil	Present	Present	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
15	Nagamma	38 Y/ F	21.01.2009	479597	Present	Present	Present	< 24 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
16	Ramesh	39 Y / M	30.01.2009	481638	Present	Present	Absent	> 48 Hrs	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
17	Yamanaw	70 Y / F	03.02.2009	482576	Present	Present	Present	> 72 Hrs	Nil	Nil	Present	Nil	NII	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
18	Pyarezan	60 Y / M	07.02.2009	482976	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	DM,HTN	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	-	Normal
19	Venkatesh	45 Y / M	15.03.2009	491276	Present	Present	Absent	< 24 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Normal	Deranged
20	Basama	35 Y / F	27.03.2009	493821	Present	Present	Present	> 48 Hrs	Present	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Normal	Normal
21	Ramachan	32 Y/M	01.04.2009	495144	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
22	Ramachad	42 Y / M	02.05.2009	495144	Present	Present	Present	> 72 Hrs	Present	Present	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Normal
23	Srinivas	29 Y / M	08.06.2009	511745	Present	Present	Present	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Absent	Absent	Present	Normal	Normal
24	Venkatesh	60 Y / M	10.07.2009	520191	Present	Present	Present	> 48 Hrs	Nil	Nil	Nil	DM	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
25	Nagesh	20 Y / M	11.07.2009	520729	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Absent	Absent	Present	Normal	Normal
26	Sangames	25 Y / M	13.07.2009	520929	Present	Present	Present	> 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
27	Kantamma	65 Y/ F	15.07.2009	521099	Present	Present	Absent	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
28	Mamata	40 Y / F	17.07.2009	521609	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	NII	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
29	Munivenkte	50 Y / M	18.07.2009	522590	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	DM,HTN	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Absent	Absent	Absent	Deranged	Deranged
30	Dhanushre	18 Y / F	24.07.2009	524157	Present	Present	Absent	> 96 Hrs	Present	Present	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Absent	Absent	Present	Deranged	Normal
31	Muniyappa	52 Y / M	24.07.2009	524060	Present	Absent	Present	> 72 Hrs	Nil	Nil	Nil	Asthama	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
32	Venkatesh	45 Y / M	26.07.2009	524502	Present	Present	Absent	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
33	Krishna	18 Y / M	01.08.2009	526184	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
34	Govindapp	30 Y/ M	4.08.2009	526900	Present	Absent	Present	> 96 Hrs	Nil	Nil	Nil	HIV	Nil	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
35	Ravikrishn	22 Y / M	06.08.2009	526999	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
36	Manjunath	18 Y / M	12.08.2009	529217	Present	Absent	Absent	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
37	Munikrishn	40 Y / M	13.08.2009	529484	Present	Absent	Present	> 2 Weeks	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
38	Vishwanath	27 Y / M	14.08.2009	529534	Present	Absent	Absent	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
39	Gowrappa	55 Y / M	16.08.2009	530085	Present	Absent	Present	> 96 Hrs	Nil	Nil	Nil	HTN	NSAID	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
40	Nagaraj	40 Y / M	17.08.2009	530409	Present	Absent	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
41	Gowramm	70 Y / F	20.08.2009	530992	Present	Present	Present	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
42	Yellappa	35 Y / M	20.08.2009	531016	Present	Absent	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Absent	Deranged	Normal
43	Subramani	40 Y / M	21.08.2009	531297	Present	Present	Absent	> 72 Hrs	Nil	Nil	Nil	Nil	NSAID	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Normal
44	Venkatesh	48 Y / M	24.08.2009	532149	Present	Absent	Present	<24 hrs	Nil	Nil	Nil	Nil	NSAID	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
45	Gnana She	45 Y / M	27.08.2009	532786	Present	Absent	Absent	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
46	Syed amee	65 Y / M	28.08.2009	533106	Present	Absent	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Deranged	Normal
47	Doddachin	50 Y / M	28.08.2009	533109	Present	Present	Absent	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
48	Basawaraj	19 Y / M	30.08.2009	533661	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
49	Krishnappa	45 Y / M	30.08.2009	533660	Present	Present	Present	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
50	Mumtaz	45 Y / F	31.08.2009	533753	Present	Present	Absent	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	-	Deranged
51	Rudraiah	30 Y / M	31.08.2009	533763	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
52	Manjula	37 Y / F	05.09.2009	535237	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Normal
53	Mennaksh	65 Y / F	07.09.2009	535792	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
54	Bashasab	70 Y / M	21.09.2009	539510	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	N	Present	Nil	Present	Present	Present	Present	Present	Present	Absent	Present	Normal	Deranged
55	Arun	19 Y / M	22.09.2009	539540	Present	Absent	Present	<24 hrs	Nil	Nil	Nil	Asthama	Present	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Normal	Normal
56	Gopalappa	55 Y / M	27.09.2009	541112	Present	Present	Present	< 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
57	Bharathkur	19 Y / M	1.10.2009	541471	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
58	Erappa	60 Y / M	2.10.2009	542869	Present	Present	Present	< 48 Hrs	Nil	Nil	Nil	DM	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
59	Venkatesh	55 Y / M	03.10.2009	542540	Present	Present	Absent	> 96 Hrs	Nil	Nil	Nil	DM, HTN	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
60	Manjunath	27 Y / M	6.10.2009	543212	Present	Present	Absent	< 48 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Nil	Nil	Present	Present	Present	Absent	Absent	Present	Normal	Normal
61	Rajashetty	45 Y / M	18.10.2009	548596	Present	Present	Present	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
62	Venkatesh	40 Y/ m	26.10.2009	548416	Present	Absent	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
63	Chikkaram	65 Y / M	27.10.2009	460225	Present	Present	Absent	> 72 Hrs	Nil	Nil	Nil	HTN	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
64	Rahamath	58 Y / M	29.10.2009	549089	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Absent	Deranged	Deranged
65	Ameeljan	60 Y / M	03.11.2009	550281	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Asthama		Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Absent	Normal	Deranged
66	Ameettan	60 Y / M	03.11.2009	550281	Present	Present	Absent	> 72 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Present	Present	Normal	Normal
67	Narasimha	55 Y / M	07.11.2009	551388	Present	Present	Present	< 48 Hrs	Nil	Nil	Nil	HTN	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged

68	Raghu	20 Y / M	12.11.2009	552481	Present	Present	Absent	< 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
69	Venkataran	55 Y / M	13.11.2009	552723	Present	Present	Present	< 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
70	Suresh	28 Y / M	13.11.2009	552733	Present	Present	Present	< 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
71	Jayaramap	60 Y / M	20.11.2009	554563	Present	Present	Absent	<24 hrs	Present	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
72	Venkatarav	60 Y / M	30.11.2009	557415	Present	Present	Absent	> 72 Hrs	Nil	Nil	Nil	COPD	Nil	Present	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Normal	Deranged
73	Munivenka	49 Y / M	04.12.2009	558654	Present	Present	Absent	< 48 Hrs	Present	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
74	Munivenka	40 Y / M	04.12.2009	558695	Present	Present	Absent	> 96 Hrs	Present	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
75	Venkatalak	90 Y / F	13.12.2009	560701	Present	Absent	Absent	<24 hrs	Nil	Nil	Nil	HTN, Astha	Present	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
76	Raghamma	62 Y / F	17.12.2009	561787	Present	Present	Present	1 Week	Nil	Nil	Present	Nil	NII	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
77	Venkataran	36 Y / M	17.12.2009	561778	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
78	Rajamma	66 Y / F	27.12.2009	561989	Present	Present	Present	1 Week	Nil	Nil	Present	Nil	NII	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
79	Subramani	55 Y / M	08.01.2010	567154	Present	Present	Absent	< 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Absent	Absent	Normal	Normal
80	Nagesh	19 Y / M	08.01.2010	567243	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
81	Venkatarav	65 Y/ M	09.01.2010	567453	Present	Absent	Absent	<24 hrs	Nil	Present	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Normal
82	Rathnamm	45 Y / F	11.01.2010	568794	Present	Present	Absent	> 96 Hrs	Present	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Present	Present	Absent	Present	Normal	Deranged
83	Irappa	51 Y / M	18.01.2010	569535	Present	Absent	Absent	<24 hrs	Present	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Present	Present	Deranged	Normal
84	Ashwathap	29 Y / M	25.01.2010	571168	Present	Absent	Present	<24 hrs	Present	Nil	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
85	Gundappa	28 Y / M	28.01.2010	567243	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
86	Ramangou	56 Y / M	29.11.2009	554563	Present	Present	Absent	> 96 Hrs	Present	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	-
87	Venkatesh	22 Y / M	31.01.2010	567243	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	-
88	Mujidpasha	18 Y/M	04.02.2010	573488	Present	Present	Present	> 72 Hrs	Present	Nil	Nil	Nil	Nil	Nil	Present	Nil	Nil	Present	Present	Present	Present	Present	Absent	Normal	Normal
89	Ramesh K	45 Y / M	07.02.2010	574277	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
90	Somashek	23 Y/M	18.03.2010	583615	Present	Absent	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
91	Mahendra	19 Y/M	08.04.2010	588579	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Normal
92	Narasimhr	24 Y / M	04.05.2010	595334	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Normal
93	Ramprasa	38 Y/M	09.05.2010	596761	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged
94	Ramlakshr	44 Y/F	10.05.2010	597210	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
95	Nagaraj	18 Y/M	09.06.2010	605117	Present	Absent	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
96	Eranna	28 Y / M	10.06.2010	605229	Present	Present	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Deranged	Normal
97	Sagarappa	53 Y/M	07.07.2010	612464	Present	Absent	Absent	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
98	Anadamma	21 Y / F	13.07.2010	614326	Present	Present	Present	<24 hrs	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Normal
99	Baichappa	50 Y/M	22.07.2010	616959	Present	Present	Present	> 96 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Present	Present	Present	Present	Present	Absent	Present	Deranged	Deranged
100	Nagesh	32 Y/M	23.07.2010	617077	Present	Present	Present	< 48 Hrs	Nil	Nil	Nil	Nil	Nil	Nil	Present	Present	Nil	Present	Present	Present	Present	Absent	Present	Normal	Deranged

						Operative findings			Post operative complications						Results		
LFT	Total coun	Hb	Widal	Abdominal US		Perforation	Definitive s	Intraperitor	Burst abdo	Wound infe	Entero-cut	Others	Mortality	Recovred	Second Su	Duration of
				Free Fluid	Mass											
Normal	Leucocytos	Normal	Negative	Minimal	Present	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
Normal	Leucocytos	Normal	Negative	Minimal	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	9 Days
Normal	Leucocytos	Normal	Negative	Minimal	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	9 Days
Normal	Normal	Anemic	Negative	Minimal	Present	Caecum	Right hemicol	Nil	Nil	Present	Nil	Nil	Nil	Successful	Nil	20 Days
Deranged	Normal	Anemic	Negative	Minimal	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	15 Days
Normal	Normal	Normal	Negative	Minimal	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	09 Days
Normal	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	07 Days
Normal	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	07 Days
Normal	Leucocytos	Normal	Negative	Moderate	Nil	Ileal	Exp lap with r	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	20 days
Deranged	Leucocytos	Anemic	Negative	Moderate	Nil	Ileal	Exp lap with r	Nil	Nil	Present	Nil	Nil	Nil	Successful	Nil	22 Days
Normal	Normal	Anemic	-	Moderate	Nil	Caecum	Exp lap wih rig	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	18 Days
Deranged	Normal	Anemic	Negative	Moderate	Present	Rctosigmo	Exp lap with tr	nil	nil	Nil	nil	Nil	Death	Death	Death	Death
Deranged	Normal	Anemic	-	Minimal	Nil	Gastric	Exp lap with s	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
Deranged	Leucocytos	Anemic	Negative	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Ketoacidos	Nil	Successful	Nil	22 Days
Normal	Normal	Normal	Negative	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
Deranged	Normal	Anemic	Negative	Moderate	Nil	Jejunal	Exp lap jejuna	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	11 Days
Deranged	Leukocytos	Anemic	Negative	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Present	Nil	Nil	Death	Nil	Death
Normal	Leucocytos	Anemic	Negative	Nil	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	13 Days
-	Normal	Normal	-	Minimal	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	11 Days
Normal	Normal	Normal	Negative	Moderate	Nil	Colon	Exp lap with r	Nil	Nil	Nil	Nil	Acidosis	Death	Death	Death	Death
-	Normal	Normal	Negative	Moderate	Nil	Ileal	Loop ileostom	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	17 Days
-	Normal	Normal	Negative	Minimal	Nil	Ileal	Exp lap resctn	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	15 Days
-	Normal	Normal	Negative	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
Normal	Leukocytos	Normal	Positive	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Present	Nil	Nil	Nil	Successful	Nil	19 Days
-	Normal	Normal	Negative	Moderate	Nil	Duodenal	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	07 Days
Normal	Leukocytos	Normal	Positive	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Present	Nil	Nil	Nil	Successful	Nil	17 Days
-	Leucocytos	Anemic	Negative	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	17 Days
Deranged	Leucopenia	Normal	Negative	Moderate	Nil	Duodenal	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Death	Death	Nil	Death
Deranged	Normal	Anemic	Negative	Minimal	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	17 Days
Normal	Leucocytos	Normal	Negative	Moderate	Nil	Caecum	Exp lap with il	Nil	Nil	Nil	Nil	Sepsis syn	Nil	Successful	Nil	15 Days
-	Normal	Anemic	Negative	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Present	Nil	Nil	Nil	Successful	Nil	15 Days
-	Normal	Normal	Negative	Gross	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	17 Days
-	Leucocytos	Anemic	Negative	Minimal	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
-	Leucopenia	Normal	Negative	Minimal	Nil	Ileal	Exp lap with Ile	Nil	Nil	Nil	Nil	Chest infec	Death	Death	Nil	Death
-	Leucocytos	Anemic	Negative	Minimal	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	14 Days
-	Normal	Anemic	Positive	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
Normal	Normal	Normal	Positive	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
-	Normal	Anemic	Positive	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
-	Leucocytos	Normal	Negative	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Death	Death	Nil	Death
Deranged	Leucocytos	Normal	Negative	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Death	Death	Nil	Death
-	Leucocytos	Normal	Positive	Nil	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Renal failu	Nil	Successful	Nil	20 Days
-	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
-	Normal	Normal	Negative	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
-	Normal	Normal	Positive	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
-	Normal	Normal	Negative	Minimal		Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
-	Normal	Normal	Negative	Nil	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	12 Days
Normal	Normal	Normal	Negative	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Death	Death	Nil	Death
Normal	Leucocytos	Anemic	Negative	Moderate	Nil	Jejunal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	12 Days
Normal	Normal	Normal	Negative	Minimal	Nil	Jejunal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
-	Leukocytos	Normal	Negative	Minimal	Nil	Jejunal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Death	Nil	Death
Normal	Leucocytos	Anemic	Negative	Moderate	Nil	Jejunal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
-	Leucocytos	Anemic	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
-	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	15 Days
Deranged	Normal	Normal	Positive	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	13 Days
Deranged			Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Burst abdo	Nil	Nil	Chest infec	Nil	Successful	Secondary	40 Days
Deranged	Leucocytos	Normal	Negative	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Sepsis syn	Nil	Successful	Nil	18 Days
Normal	Leucocytos	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
Deranged	Leukocytos	Normal	Negative	Moderate	Nil	Duodenal	Exp lap with c	Nil	Burst abdo	Nil	Nil	Nil	Nil	Successful	Secondary	18 Days
Normal	Normal	Anemic	Negative	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	16 Days
Deranged	Normal	Normal	Negative	Minimal	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	09 Days
Normal	Normal	Normal	Negative	Minimal	Nil	Colon	Right hemicol	Nil	Nil	Present	Nil	Nil	Nil	Successful	Secondary	16 Days
Deranged	Normal	Normal	Positive	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	17 Days
-	Normal	Anemic	Negative	Minimal	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	17 Days
-	Normal	Normal	Negative	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
Deranged			Negative	Minimal	Minimal	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Renal failu	Nil	Successful	Nil	17 Days
Normal	Normal	Normal	Negative	Minimal	Nil	Duodenal	Exp lap with c	Nil	Burst abdo	Nil	Nil	Nil	Nil	Successful	Secondary	17 Days
Normal	Leucocytos	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	15 Days

Normal	Leucocytos	Normal	Negative	Moderate	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	09 Days
Deranged	Leucocytos	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
Deranged	Leucocytos	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
-	Normal	Anemic	Negative	Nil	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
Deranged	Leucocytos	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	18 Days
Normal	-	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
Normal	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Present	Nil	Nil	Nil	Successful	Nil	20 Days
Normal	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	15 Days
Deranged	Leukocytos	Anemic	Negative	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Death	Nil	Death
Normal	Normal	Normal	Positive	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	20 Days
Deranged	Leukocytos	Anemic	Negative	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Present	Nil	Nil	Death	Nil	Death
-	Leucocytos	Normal	Negative	Moderate	Present	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	13 Days
Normal	Normal	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
-	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	UTI	Nil	Successful	Nil	14 Days
Deranged	Leucocytos	Normal	Negative	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Death	Nil	Death
Normal	Neutrohilia	Normal	_	Minimal	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
Normal	Normal	Normal	_	Moderate	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
Normal	Normal	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
-	Normal	Normal	Negative	Nil	Nil	Gastric	Exp lap with c	Nil	Burst abdo	Nil	Nil	Nil	Nil	Successful	Secondary	19 Days
-	Normal	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
-	Leucocytos	Normal	-	Minimal	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	SIRS	Nil	Successful	Nil	15 Days
-	Leucocytos	Normal	-	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	08 Days
-	Normal	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	20 Days
-	Leucocytos	Normal	Negative	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	11 Days
-	Normal	Normal	-	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	09 Days
Deranged	Leucocytos	Normal	-	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	07 Days
Normal	Normal	Normal	-	Nil	Nil	Jejunal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
-	Normal	Normal	-	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
-	Normal	Normal	-	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	09 Days
Normal	Normal	Normal	-	Nil	Nil	Gastric	Exp lap with c	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	10 Days
Normal	Leucocytos	Normal	Negative	Moderate	Nil	Appendicu	Exp lap with a	Nil	Nil	Nil	Nil	Nil	Nil	Successful	Nil	09 Days
-	Leucocytos	Normal	Negative	Nil	Nil	Duodenal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	13 Days
Normal	Normal	Anemic	Negative	Moderate	Nil	Ileal	Exp lap with c	Nil	Nil	Nil	Nil	Chest infec	Nil	Successful	Nil	18 Days