A STUDY OF SMALL BOWEL PERFORATIONS

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF SMALL BOWEL PERFORATIONS"** submitted by **Dr. J. J. LANKARAM** to The Tamil Nadu Dr. M.G.R. Medical University, Chennai is in partial fulfillment of the requirement for the award of M.S. degree Branch I (General Surgery) and is a bonafide research work carried out by him under direct supervision and guidance.

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DECLARATION

I, Dr. J. J. Lankaram declare that I carried out this work on **"A STUDY OF SMALL BOWEL PERFORATIONS"** at Department of General Surgery, Government Rajaji Hospital during the period of November 2004 – February 2006. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any university, board either in India or abroad.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M.S. Degree examination in General Surgery.

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A STUDY OF SMALL BOWEL PERFORATIONS

INTRODUCTION

There are various causes for small bowel perforations. But among these, Duodenal ulcer perforations and Typhoid ileal perforations are frequently occurring in developing countries like ours.

Peptic ulcer perforation in duodenum is one of the commonest type of gastro intestinal perforation. There has been a marked decrease in the elective surgery for peptic ulceration following the introduction of medical treatment including H2 receptor antagonists and Proton pump inhibitors . By contrast, complications like perforation & bleeding requiring emergency surgery have remained relatively constant, most of the etiological factors are modifiable like smoking, alcohol, NSAIDS and Helicobacter pylori.

In tropical countries like India, enteric fever perforations are still common. Typhoid fever is endemic in India especially among the poor. Medical management is effective in controlling the typhoid fever before the perforation occurs.

Hence prevention, good treatment and saving the people from the complications not only protects the manpower of our country but also adds to its economic growth.

AIM OF THE STUDY

- ✓ To study the various causes for small bowel perforations.
- ✓ To study the morbidity & mortality of intra abdominal hypertension in small bowel perforations.
- ✓ To analyse the various modalities of treatment offered to the patients.
- ✓ To study the effect of immediate decompression of abdomen in patients with perforative peritonitis.

REVIEW OF EMBRYOLOGY

As a result of the cephalo caudal and lateral folding of the embryo, the endoderm – lined cavity is partially incorporated into the embryo to form the primitive gut.

In the cephalic and caudal parts of the embryo, the primitive gut forms a blind ending tube, the foregut and hindgut respectively. The middle part forms the midgut. At an early stage of development the alimentary canal is represented by tube suspended in the midline of the abdominal cavity by ventral and dorsal mesentery.

The terminal part of the forgut and the cephalic part of the midgut form Duodenal portion of the intestinal tract. As the stomach rotates, the duodenum takes on the form of a c- shaped loop , rotates to the right & finally come to lie retro peritoneally.

Midgut grows so rapidly that the intra- embryonic coelom is too small to accommodate it, so that part of the loop is extended into the extra – embryonic coelom.

Superior mesenteric artery sends off branches forwards to the anterior segment of the midgut loop (pre – arterial segment) and backward to its posterior segment (post arterial segment), the midgut loop and its mesentery still lie in the sagittal plan.

ROTATION OF MIDGUT

FIRST STAGE :

The growth of the right lobe of the liver exerts pressure on the base of the pre –arterial segment so that this segment is pushed down and to the right. This movement forces the post arterial segment upwards and to the left . The first stage of rotation is complete when the midgut loop has rotated through 90° in an anticlockwise direction.

SECOND STAGE

The midgut loop returns to the abdominal cavity from the umbilical cord. The pre- arterial portion returns first, commencing with its proximal portion. The returning small gut enter the abdomen to the right of the superior mesenteric artery, but the space here being too limited the coils first reduced are pushed to the left behind the artery by those following on. By their passage to the left, they displace the dorsal mesentery of the hindgut (which occupies the midline) before them, so that the descending colon comes to occupy the left flank.

The caecum still lies in the umbilical cord on a plane anterior to the small intestine and its artery. The caecum and right half of the colon now

reduce, passing upward and to the right, the colon crossing the pedicle of the small gut at the point of origin of the superior mesenteric artery from the aorta, and the caecum comes to lie under the liver . The subsequent growth elongation of the colon pushes the caecum into the right loin.

THIRD STAGE:

During this stage, the caecum descends further, reaching the right iliac fossa. Certain parts of the gut become fixed to the posterior abdominal wall while these processes occur, the midgut loop rotates 270^{o} counter clockwise.

ANATOMY OF THE SMALL INTESTINE

Small intestine extends from the pyloroduodenal junction to the ileocaecal valve. It is about 6 meters long.

Small intestine consists of

- Duodenum
- Jejunum and
- Ileum

DUODENUM

It is 20 –25 cms long and is the shortest, widest and most sessile part of the small intestine . It has no mesentery and is thus only partially covered by peritoneum . It encircles the head of the pancreas and consists of four parts.

- The superior (first) part, about 5cm long
- The descending (second) part, about 8-10 cm long
- The horizontal (inferior or third) part , about 10 cms long
- The ascending (Fourth part) about 2.5cms long

ARTERIAL SUPPLY

It is from the right gastric, right gastroepiploic, supra duodenal and superior and inferior pancreatico- duodenal arteries.

VENOUS DRAINAGE

Veins end in the splenic, superior mesenteric and portal veins.

NERVE SUPPLY

Are from the coeliac plexus

LYMPHATIC DRAINAGE

Duodenal lymph drains by channels that accompany superior and inferior pancreatico duodenal vessels to coeliac and superior mesenteric nodes.

JEJUNUM AND ILEUM

The small intestine except the duodenum is attached to the posterior abdominal wall by the mesentery, the proximal two fifth being the jejunum, the distal three – fifth , the ileum.

The jejunum is comparatively wider bored and thicker walled and measures about 2.3 to 2.8 meters. It lies largely in the umbilical region.

The ileum is thinner than the jejunum. Its length varies from 3.6 to 4.2meters. It is mainly in the hypogastric and pelvic region.

The fan like mesenteric attachment of the jejunum and ileum to the posterior abdominal wall allows free movement, each coil adapting to changes in form and position.

ARTERIAL SUPPLY

Jejunal and ileal arteries from the left side of the superior mesenteric artery .

These branches form arterial arcades

Jejunum has one or two arterial arcades and has high narrow windows in the intestinal border of the mesentery.

Ileum has larger serious of arterial arcades – three to five and the straight vessels branching off the arcades are shorter.

VENOUS DRAINAGE

The veins follow the arteries

LYMPHATIC DRAINAGE

The lymph vessels form an intricate plexus in mucosa & submucosa and are joined by vessels from lymph spaces at the bases of solitary follicles and drain to larger vessels at the mesenteric aspect of the gut. These drain to superior mesenteric lymph nodes.

NERVE SUPPLY

Innervation is by vagus and thoracic splanchnic nerves through the caeliac ganglia and superior mesenteric plexuses.

ETIOLOGY OF SMALL BOWEL PERFORATIONS[3]

- Duodenal ulcer perforation
- Inflammatory diseases
    - Acute – salmonella typhi enteritis
    - Chronic – Tuberculosis common
- Vascular
    - Ischemic enterocolitis
    - Strangulated hernia
- Neoplastic
    - Rare
    - Seen in lymphomas
- Diverticulitis
    - Meckel's diverticulitis
    - Jejunal diverticulitis
    - Duodenal diverticulitis
- Miscellaneous
    - Radiation enteritis
    - Necrotising enterocolitis
    - Meconium peritonitis

PATHOPHYSIOLOGY

PATHOPHYSIOLOGY OF PEPTIC ULCER

Gastric secretion aids in breakdown of food into smaller particles. About 2 litres of gastric juice is produced everyday.

In the stomach, oxyntic cells secrete Hcl & Intrinsic factor. Chief cells secrete pepsinogen . Mucus cells secrete mucus and G cells secretes gastrin .

Hydrochloric acid is a major etiological factor in acid peptic disease. Acetyl choline, histamine and gastrin stimulate Hcl secretion . Somatostatin inhibits Hcl secretion.

GASTRIC MUCOSAL BARRIER

The gastric mucus layer is essential to the integrity of the gastric mucosa. It is a viscid layer of mucopolysaccharides produced by the mucus-producing cells of the stomach and pyloric glands. Gastric mucus is an important physiological barrier to protect the gastric mucosa from mechanical damage, and also the effects of acid and pepsin. Its considerable buffering capacity is enhanced by the presence of bicarbonate ions within the mucus.

Many factors can lead to the breakdown of this gastric mucosal barrier. Those include bile, NSAIDS, alcohol , trauma and shock.

DUODENAL ULCERATION

INCIDENCE

There have been marked changes in the last two decades in the demography of patients presenting with duodenal ulceration.

First, even before the introduction of H_2 receptor antagonists, the incidence of duodenal ulceration and frequency of elective surgery for the condition were falling. This may relate to the widespread use of gastric antisecretory agents and eradication therapy for patients with dyspepsia .

Second , the peak incidence is now in a much older age group than previously and although it is still more common in men, the difference is less marked . These changes mirror the changes at least in part in the epidemiology of H. Pylori infection.

The incidence of perforation and bleeding duodenal ulcers in young and middle aged patients appear to be falling but in contrast, there is currently a marked increase in the number of elderly suffering these complication. This trend can be explained not only by the H. Pylori cohort effect but also by the increased use of NSAIDS in elderly.

ETIOLOGY OF PERFORATION IN PEPTIC ULCER

a) NSAIDS

They interfere with cyclo oxygenase pathway which leads to production of prostanoids which inturn affects mucosal protection by reducing the effectiveness of mucus bicarbonate barrier.

They are not dependent on duration of usage.

The rate of recurrence of ulcer is very less after discontinuation of drugs.

This can be prevented by additional therapy with prostaglandin analogues.

b) HELICOBACTER PYLORI

It is a small curved gram negative micro aerophilic rod with multiple polar flagellae. Stomach is its normal inhabitant.

One of the characteristics of the organism is its ability to hydrolyse urea, resulting in production of ammonia, a strong alkali. This causes release of gastrin from antral G cells. This is probably responsible for hypergastrinaemia in peptic ulcer patients, which in turn may result in gastric acid hypersecretion.

Infection also leads to the disruption of the gastric mucosal barrier by the enzymes produced by the organism.

Gastric metaplasia is the normal response of the duodenal mucosa to excess acidity, an attempt by the mucosa to resist an injurious stimulus .

Although normal duodenal mucosa cannot be infected with H. Pylori, gastric metaplasia in duodenum is commonly infected.

The incidence of infection within a population increases with age, and infection rates of 80-90% are not unusual.

The possibility of infection is inversely related to socio economic group.

Eradication of H. Pylori reduces ulcer recurrence rate. Hence all patients should be treated by H. pyloric eradication therapy as it speeds up healing and decreases the rate of ulcer disease.

c) CIGARETTE SMOKING

It impairs the healing of ulcer

It promotes recurrence of ulcers and also increases the surgical risks.

d) MISCELLANEOUS

- Alcohol damages the gastric mucosal barrier
- Certain personality traits and psychological stress, or poor tolerance to stress leads to ulcer formation.
- An association has been reported that patients with blood group O have an increased risk of duodenal ulcer.

PATHOLOGY

Most of the peptic ulcers occur in the first part of the duodenum

A chronic ulcer penetrates the mucosa and into the muscle coat leading to fibrosis. The fibrosis causes deformities such as pyloric stenosis.

Sometimes there may be more than one duodenal ulcer. The situation in which there is both anterior and posterior duodenal ulcer is referred to as “kissing ulcer”.

Anteriorly placed ulcers tend to perforate and posterior duodenal ulcer tend to bleed, sometimes by eroding a large vessel such as the gastroduodenal artery.

HISTOPATHOLOGY

MICROSCOPICALLY

Destruction of the muscular coat is observed and the base of the ulcer is covered with granulation tissue, the arteries in this region showing the typical changes of endarteritis obliterans.

STAGES IN ACUTE PERFORATION[7]

1. Stage of peritoneal irritation
2. Stage of peritoneal reaction
3. Stage of diffuse peritonitis

1) STAGE OF PERITONEAL IRRITATION

This is first stage known as peritonism.

Due to leakage of gastric juice into the peritoneal cavity – chemical peritonitis.

Usually lasts for about six hours

There will be previous history of peptic ulcer.

There is acute burning pain over epigastrium. Pain may be referred to tip of right shoulder due to irritation of undersurface of diaphragm.

Pain may gradually gravitate down along the paracolic gutter to the right iliac fossa mimicking acute appendicitis.

Tenderness and muscle guard are constantly present.

There will be little change in the pulse, temperature and respiration.

2) STAGE OF PERITONEAL REACTION

This is second stage.

The irritant fluid becomes diluted with the peritoneal exudates.

The patient feels comfortable.

Muscular rigidity is present.

Obliteration of liver dullness is present.

Rectal examination may elicit tenderness in the recto – vesical or recto uterine pouch.

3) STAGE OF DIFFUSE PERITONITIS

This is third or final stage.

There will be pinched and anxious face, sunken eyes and hollow cheeks, so called facies Hippocratica.

Tachycardia with low in volume & tension, board like rigidity of abdomen, increasing distension of the abdomen are present.

Septicemia and multi system organ failure frequently supervene.

SUBACUTE PERFORATION

An ulcer may perforate and seal rapidly before there is spillage of gastric & duodenal contents into peritoneal cavity.

Sudden onset of acute abdominal pain.

Local tenderness and rigidity are present.

CHRONIC PERFORATION

When an ulcer perforates into an area that is walled of by adhesion or by adjacent viscera like colon, greater omentum or when a gastric ulcer perforates into a omental sac with sealing off the omental foramen, chronic abscess may form.

Features of peritonitis may be less marked.

X ray abdomen may reveal air under diaphragm.

PATHOPHYSIOLOGY OF TYPHOID PERFORATIONS

Typhoid fever is caused by ingestion of salmonella typhi contaminated with water and is a systemic infection. Contamination occurs due to infected stools or urine. They invade the intestinal lymphatics and mesenteric nodes & thus reach the blood stream. Once bacteremia is established, it leads to the development of secondary areas of inflammation in the liver, gall bladder and marrow. After one week, the bacteria are shed into the small bowel and therefore appears in the stool.

The early change is the hyperplasia of the lymph follicles. The peyer's patches become swollen and ulcerated, which can progress to capillary thrombosis and subsequent necrosis. In the second week, necrosis and sloughing occurs and ulceration of the follicles leading on to perforation in the third week of disease.

PATHOLOGY

The organisms cause enlargement of reticulo endothelial and lymphoid tissue throughout the body. Proliferation of the phagocytes swells the lymphatic submucosal nodules of the entire gut mainly peyer's patches of the terminal ileum. These become sharply delineated plateau like elevations upto 8mm in diameter bulging into the intestinal lumen. During second week, the mucosa over the swollen ileal lymphoid tissue is shed, resulting in oval ulcers with their long axis in the direction of bowel flow.

Once passed the peak of the disease, the ulcers heal slowly and lymphatic structures amazingly regenerate without scarring. Histologically there is accumulation of mononuclear phagocytes which form nodular aggregates filled with red cells and nuclear debris.

Perforation is due to the result of rupture of necrotic peyer's patches caused by distension of bowel or by excessive peristalsis. The spleen is enlarged and soft. Microscopically, marked histocytosis and reticulo endothelial proliferation are present. Sometimes spleen may rupture.

The liver shows scattered foci of parenchymal necrosis in which hepatocyte is replaced by phagocytic mononuclear aggregate called as "Typhoid nodule" which can also occur in bone marrow. Gall bladder colonization produces a carrier state often requires cholecystectomy to eliminate bacterial shedding.

CLINICAL FEATURES

The symptoms of head ache, fever, vomiting and abdominal pain and the signs of abdominal tenderness, guarding and rigidity, distension, absent intestinal sounds, presence of free fluid and obliteration of hepatic dullness were considered most important.

The most prominent clinical features were the prolonged debilitating feverishness with diarrhea and on examination, generalized abdominal

tenderness with rebound tenderness were the rule more marked on the right side of abdomen

In typhoid perforations, the classical test like widal and blood culture are little important value as the results are obtained only after few days. Therefore history of fever and physical examination with signs and symptoms suggestive of perforation assume importance.

SITE OF PERFORATION IN TYPHOID FEVER

Since the peyer's patches are more in the terminal ileum, the incidence of typhoid perforation is also more in the terminal ileum. Kim 1975[32] obtained 86% cases of perforation occurred in the last 60cm of ileum of which 72% perforations were within the last 40cm . In kuruvilla series 1978[30] perforations were confined to the last 30 cms of the terminal ileum. Purohit et al[34], noted all the perforations occurring within 40cms from the ileo caecal junction.

PATHOPHYSIOLOGY OF INTESTINAL TUBERCULOSIS

Tuberculosis can affect any part of the GIT from mouth to the anus.

Intestinal Tuberculosis can be of two varieties

1. Ulcerative tuberculosis
2. Hyperplasic tuberculosis

1. ULCERATIVE TUBERCULOSIS

This usually results from swallowing of tubercle bacilli (human type) in sputum in a case of pulmonary tuberculosis

This condition is characterized by multiple ulcers at the terminal ileum

The long axis of the ulcer lies transversely.

The serous coat overlying the ulcer becomes thickened, so perforation is unusual . But perforation occurs in ulcerative tuberculosis than in hyperplastic tuberculosis.

Healing of the ulcers leads to stricture formation. Loss of weight and diarrhea with fecal odour stools containing pus and occult blood are present.

There will be slight tenderness in right iliac fossa.

2. HYPERPLASTIC TUBERCULOSIS

Infection starts in the lymphoid follicles and then spreads to submucous and subserous planes. The intestinal wall becomes thickened with narrowing of its lumen.

There will be early involvement of regional lymph nodes which become matted along with the involved terminal part of ileum and caecum to produce the lump.

Recurrent attacks of abdominal pain with diarrhea and features of blind loop syndrome are present.

There will be a lump in right iliac fossa on examination .

PATHOPHYSIOLOGY OF VASCULAR DISORDERS

ACUTE MESENTERIC ISCHAEMIA[4]

The mortality rate for acute mesenteric infarction varying between 60 and 85% because the condition is not common accounting for only 1-2 % of patients with acute abdominal pain and it is usually diagnosed late.

This result in as many as 40% of patients receiving either no operation or an open and close laparotomy .

PATHOLOGY

Acute arterial ischaemia may arise from an embolus or from formation of in situ thrombus on an underlying stenosis.

It may also occur as a result of low cardiac output state.

Other rare causes include aortic dissection, fibromuscular dysplasia, intimal hyperplasia associated with oral contraceptive pills, arteritis associated with rheumatoid arthritis, systemic lupus erythematosus and polyarteritis nodosa.

CLINICAL FEATURES

There will be acute colicky abdominal pain of abrupt onset in a patient with atrial fibrillation or recent myocardial infarction with no antecedent history of gastrointestinal upset or weight loss.

A more insidious onset suggests mesenteric artery thrombosis

As this is superimposed on chronic occlusive disease there may be a prior history of post prandial abdominal pain (intestinal angina) , weight loss and diarrhea .

History of minor gastrointestinal bleeding is a late symptom.

Abdominal pain may be mild or more classically out of proportion to the physical findings.

Onset of signs of peritoneal irritation or frank peritonitis is usually a late sign and indicative of irreversible bowel ischaemia .

PATHOPHYSIOLOGY OF STRANGULATED HERNIA[1]

A hernia is said to be strangulated when the contents are so constricted as to interfere with their blood supply. Usually the small intestine is involved in the strangulation .

Intestinal obstruction may not be present particularly in case of omentocoele, Richter's hernia and litter's hernia.

There will be no impulse on cough, extremely tense and tender

These are followed by acute intestinal obstruction.

Gangrene may occur as early as 5-6 hours after the onset of first symptoms.

Femoral hernia is more likely to strangulate because of the narrowness of the neck and its rigid surrounds.

PATHOLOGY

Intestine is obstructed & its blood supply impaired. Initially only venous return is impeded, the wall of the intestine becomes congested and bright red with the transudation of serous fluid into the sac. As congestion increases, the wall of the intestine becomes purple in colour. As venous stasis increases, arterial supply becomes more and more impaired.

Blood is extravasated under serosa and is effused into the lumen. The fluid in the sac becomes blood stained and shining serosa dull due to fibrinous ,

sticky exudates . At this stage, the walls of intestine have lost their tone and become friable. Bacterial translocation occurs.

Gangrene appears at rings of constriction than at antimesentric border .

If the strangulation is unrelieved, perforation of the wall of the intestine occurs.

Peritonitis spreads from the sac to peritoneal cavity.

PATHOPHYSIOLOGY OF SMALL BOWEL LYMPHOMAS[4]

The vast majority are Non- hodgkin's lymphoma. They can be B- cell or T- cell lymphoma. They are further subdivided into low grade and high grade.

The commonest types of intestinal Lymphomas are

- MALT lymphoma
- Centrocytic lymphoma
- Mediterranean lymphoma
- Burkitt type lymphoma
- Polymorphic T-cell lymphoma

They are more common in males

The clinical manifestation include malaise , abdominal pain, weight loss, diarrhea and anaemia.

Some are presented with intestinal obstruction or a perforation leading to peritonitis .

In patients with coeliac disease, enteropathy associated lymphoma tend to occur in fifth to seventh decade . The symptoms of celiac disease are abdominal pain, diarrhea & rapid weight loss. Perforation leading to peritonitis is a common presentation in these patients.

PATHOPHYSIOLOGY OF DIVERTICULAR DISEASE[1]

MECKEL'S DIVERTICULITIS

Occur in 2% of patients, usually 2 inches in length and are situated 2 feet from the ileocoecal valve.

It is a congenital diverticulum having all three coats of the bowel.

It represents patent intestinal end of vitello intestinal tract.

Meckel's diverticulitis with or without perforation, may result from obstruction by food residue. The symptoms are those of acute appendicitis .

When the diverticulum perforates , the symptoms may stimulate those of a perforated duodenal ulcer.

DUODENAL & JEJUNAL DIVERTICULA

Duodenal diverticula are of two types .

Primary - mostly occurring in older patients on inner wall of second and third parts & usually do not cause symptoms.

Secondary – Diverticula of the duodenal cap result from long standing duodenal ulceration.

Jejunal diverticulae are usually of variable size & multiple.

Clinically they may

- be symptomless

- give rise to abdominal pain, flatulence and borborygmi
- produce a malabsorption syndrome.
- Present as an acute abdomen with acute inflammation and occasionally rupture.

PATHOPHYSIOLOGY OF MISCELLANEOUS CONDITIONS

RADIATION ENTERITIS[4]

The immediate effect of radiation on the gastrointestinal tract is arrest of cell division in the intestinal crypts. This effect is largely restricted to cells in G1 phase . the mucosa becomes thinner with stunted villi.

The incidence of intestinal radiation induced bowel disease vary from 3 to 25%.

The most common situation is where the pelvis is irradiated usually for rectal or gynaecological cancers.

Anorexia, nausea, vomiting are present during the first few weeks of radiotherapy.

The commonest symptoms referable to chronic bowel damage are vague abdominal discomfort, diarrhea, mild rectal bleeding , and the passage of mucus.

The interval between the tissue of radiation and onset of symptoms varies considerably form 2 months to 2 years.

Intestinal obstruction may be acute or subacute or recurrent.

Occasionally acute presentation with infarction may occur and this carries a high risk of perforation and mortality .

Most of the serious complication tend to occur within 2 years of the initial treatment but may become progressively worse after this time.

NECROTISING ENTEROCOLITIS [1]

This is a common phenomenon among such premature neonates. The risk is inversely proportional to birth weight.

It is associated with hypoxia, hypothermia, hypotension and umbilical artery cannulation.

Ileum, caecum, distal colon and total colon are affected with a complete spectrum from mucosal to transmural necrosis .

The usual presentation in bilious vomiting, abdominal distension, colour change , a lethargy in a high risk neonate . the abdomen is usually soft.

MECONIUM PERITONITIS [1]

It is an aseptic peritonitis that develops late in intrauterine life or during or just after delivery . Meconium is a sterile mixture of epithelial cells, mucin, salts, fats and bile.

Meconium enters peritoneal cavity through an intestinal perforation and in over 50% of cases the perforation is the result of some form of neonatal intestinal obstruction , in the remainder no cause is found. Meconium remain sterile upto 3 hours after birth after wards it leads to acute bacterial peritonitis.

New born baby presents with a tense abdomen who is vomiting and in whom there is failure to discharge meconium.

MANAGEMENT

MANAGEMENT OF PEPTIC ULCER PERFORATION

INVESTIGATIONS

- Increased total count leukocytosis with immature forms
- Increased haematocrit due to fluid loss
- Urine analysis shows increased urinary specific gravity.
- Serum amalyse may be elevated
- X ray abdomen erect view including diaphragm or x ray chest PA view shows gas under diaphragm in about 70% of cases .
- CT scan is useful in a typical cases with doubtful clinical features, which may show localized perforation of the duodenum with leakage in the area of gall bladder and right flank, without gross free air

TREATMENT [8]

- Adequate resuscitation with crystalloids and colloids is important
- Surgical intervention

Operative treatment for perforated duodenal ulcer may be divided into simple closure versus definitive surgery. Simple closure is appropriate for patients with major underlying illness, patients with ongoing shock and perforation lasting for more than 24 hours.

Definitive operative treatment for perforated duodenal ulcers provides the benefit of perforation closure, freedom from continued ulcer symptoms and added protection from recurrence.

Definitive ulcer surgery can be recommended in patients with evidence of chronic ulcer disease who has been treated for H. pylori, patients who lack a major medical illness or patients with short duration of perforations , who are free of significant preoperative shock.

Parietal cell vagotomy combined with omental patching of the perforation is generally the preferred definitive therapy for perforated duodenal ulcer.

In the presence of chronic pyloroduodenal scarring associated with perforated duodenal ulcer, the performance of parietal cell vagotomy with omental patching should be avoided because it may not allow for unimpeded gastric empting. In this setting, truncal vagotomy combined with pyloroplasty with incorporation of perforated ulcer into closure is a better alternative.

Definitive ulcer operation[8] in addition to closing the perforation is indicated in

- Perforated gastric ulcer
- Combined gastric & duodenal ulcer one of which has perforated
- Perforation with preexisting chronic ulcer symptoms
- Coexistent obstruction & perforation

- Coexistent haemorrhage and perforation
- Previous operation for perforated duodenal ulcer
- Young patients <35 years who have perforated duodenal ulcer
- H- pyloric treated or known negative patients who have perforation.

GRAHAM'S PATCH TECHNIQUE

Here simple closure with a live omental patch is used using absorbable suture material.

CELEN JONES TECHNIQUE

Using absorbable suture materials, full thickness sutures are placed on either side of perforation and a strand of omentum is drawn under the arch & sutures tied.

CLOSURE OF PERFORATION BY OMENTAL IMPLANTATION

Perforation is repaired by drawing and implanting a portion of omentum into the perforated site. Omentum gets firmly adherent and undergoes inflammation , necrotic changes , granulation, reduction in size & fibrosis. There is no luminal obstruction

This is used in perforation with friable edges or if the sutures placed at the first operation have given way.

FREE OMENTAL PLUG

A free omental graft of suitable dimensions is cut, rolled & fashioned into the shape of mushroom & fixed to perforated site. The omental plug can also be tied to nasogastric tube using catgut. The edges of omental plug are further tucked to the intestinal wall. The Naso gastric tube is removed after 7 days, by which time the catgut suture attaching into the omentum has dissolved away.

LAPAROSCOPIC SUTURE REPAIR[8]

Suturing is carried out as same as in the open procedure using a patch omentum . This is followed by peritoneal lavage with normal saline. Definitive procedure is not generally recommended, it causes significant technical problem became of bleeding & tissue edema.

Laparoscopic fibrin glue repair using oxidized cellulose sealed with fibrin glue have been described, but they may not provide optimal results.

CONSERVATIVE MANAGEMENT[8]

Non operative therapy is often indicated for patients who have a perforation of longer than 24 hours duration , for patients whose systemic disease or current state of deterioration militates against operative treatment.

Non operative therapy includes naso gastric suction, antibiotics and fluid resuscitation and decompression of abdomen by bilateral flank drainage .

The non operative approach can be used only if a water soluble contrast radiological study confirms that the ulcer crater is sealed by failure of any contrast medium to leak from the duodenum. Of extreme importance in this method of managing perforated duodenal ulcer is close, accurate reassessment of the patients general condition and abdominal findings every 2 - 3 hours .

MANAGEMENT OF TYPHOID PERFORATION

INVESTIGATION

The diagnosis can be confirmed by isolation of the organism from the blood during first week. Identification of the antibodies by widal test is done during second week of illness. Stool and urine culture will become positive only after third week of illness. Typhoid bacilli can also be grown from bile & marrow of patients suffering from typhoid fever.

SIGNS AND SYMPTOMS

In making diagnosis , great emphasis has been given to clinical signs & symptoms. The symptoms of headache, fever, vomiting, abdominal pain, and the signs of abdominal tenderness, guarding and rigidity, distension, absent bowel sound, pressure of free fluid and obliteration of liver dullness were considered most important.

In typhoid perforation , the classical test like widal & blood culture are of little immediate value as the results are obtained only after few days. Therefore history of fever and physical examination with signs & symptoms suggestive of perforation assume importance.

WIDAL TEST

After an attack of typhoid fever, antibodies appear as early as fifth day H- antigen Ig G and O- antigen Ig M agglutinating antibodies . The antibody

levels rise gradually, reaches the maximum in second to third week. These H and O agglutinins can be estimated by widal test. The rising titre of agglutinins against S- typhi H & O Ag more than 1: 200 dilutions in patient's serum is considered as positive.

WHITE CELL COUNT

In general leucocytosis is a feature of peritonitis but typhoid fever is associated with leucopenia. Hence in the enteric fever perforation there is either high normal or leucopenia is seen

BLOOD CULTURE

Most of the blood cultures in typhoid fever are positive only in the first week of illness moreover many cases of fever are treated with antibiotics , But we come across typhoid perforation most commonly in second or third week . At that time blood cultures are less sensitive when compared to the first week.

RADIOLOGY

Abdominal erect view x rays are taken up to find out pneumpeuitoreum in enteric fever perforation cases. The incidence of pneumoperitoneum is varying from one series to other. The percentage of pneumoperitoneum various from 45- 95% cases .

HISTOPATHOLOGICAL EXAMINATION

The specimens of the edges of the ulcer for histopathological examination showed the appearance compatible with typhoid fever. Histological study reveals areas of necrosis containing plasma cells, lymphocytes macrophages containing abundant cytoplasm with bacteria and red cells termed as typhoid cells and monocytes .

TREATMENT

In general typhoid perforation is a surgical emergency and the treatment should be prompt and energetic. The role of conservative treatment is limited. The surgical procedure depends upon the general health of the patients and the extent of the ileum involved.

Patient was put on naso gastric aspiration while intravenous fluids and drugs were administered parenterally. Electrolytes and fluid balance are meticulously maintained. The appropriate operative procedure was decided at the time of laparotomy and depended upon the general condition of the patients and state of the ileum.

SIMPLE CLOSURE OF PERFORATION

The standard surgical management consists of simple closure of perforation. Laparotomy and bowel loop bearing perforation is sought out.

The perforation is closed with atraumatic needle in 2 layers . After thorough peritoneal lavage, peritoneal cavity is mopped & drain kept.

Chauhan et al[29] treated 138 cases of typhoid perforation surgically. In these patients, the principal operation was closure of the perforation and peritoneal drainage. The overall mortality of surgical treatment was 58.7%. Solitary perforations were treated by simple closure in several patients of Kuruvilla et al (1978)[30].

WEDGE EXCISION OF AFFECTED SEGMENT

- easier and quicker than resection
- Safer than simple closure with lower mortality rate
- Provides a healthy area for closure as the perforated segment is friable.
- Following wedge excision, singh et al[35] series showed 25% of mortality when compared to their previous mortality of 60% with simple closure.

SIMPLE CLOSURE WITH ILEOTRANSVERSE ANASTAMOSIS

- It allows perforation to heal quickly
- Earlier passing of flatus in post operative period .
- No chance of reperforation
- Reduces the changes of typhoid state and severity of peritonitis

- It has drawback of increasing the magnitude of surgical procedure in toxic patient with severe peritonitis

RESECTION OF THE MOST OF THE AFFECTED SEGMENT WITH AN END TO END ANASTAMOSIS

When there are multiple perforations or multiple ulcers or where ileal segment looked unhealthy or where perforation was accompanied by hemorrhage , resection becomes the operation of choice .

Resection prevents reperforation . It also prevents further perforation of nearby ulcer.

ILEOSTOMY

It is done in the moribund as well as in the most critically ill patients.

In Kim series 1975[32] , 10 patients were treated by the above method with a mortality of 10%. In Kuruvilla et al 1978[30] , because of poor general condition, ileostomy was done. His study showed 50% mortality.

COMPLICATION OF SURGERY

EARLY COMPLICATIONS : Toxemia, respiratory infections, paralytic ileus, Thrombo phlebitis, Transfusion reaction, uraemia, Meningism, shock, reperforation.

LATE COMPLICATIONS: Wound infection, Burst abdomen, faecal fistula, decubitus ulcer, osteomyelitis, Incisional hernia.

MANAGEMENT OF INTESTINAL TUBERCULOSIS[1,10]

A barium meal and follow – through or small bowel enema will show the absence of filling of the lower ileum , caecum and most of the ascending colon as a result of narrowing and hypermotility of the ulcerated segment.

TREATMENT

A course of chemotherapy - anti tuberculous drug is started .

Laparotomy is done when there is perforation or intestinal obstruction in ulcerative tuberculosis.

Ieocoecal resection is to be done in hyperplastic tuberculosis

SURGICAL PROCEDURE FOR INTESTINAL TUBERCULOSIS

For Acute bowel perforation

- Resection and anastamosis is the treatment
- Simple closure can be done but there is high incidence of reperforation and fistula formation.

For Intestinal obstruction due to Ileocaecal mass

- Right hemicolectomy is the standard of treatment
- Limited resection and ileo ascending colon anastamosis can also be carried out.

For Intestinal obstruction due to multiple strictures involving long segment of bowel.

- Resection and anastamosis

For Intestinal obstruction due to strictures placed apart

- Stricturoplasty.

MANAGEMENT OF ACUTE MESENTERIC ISCHAEMIA[4]

- There will be markedly elevated white cell count.
- Raised serum phosphate and raised serum amalyse are present.
- Angiography is diagnostic
- Superior mesenteric artery thrombosis is seen as a tapering of the origin of the vessel whilst embolic occlusion shows an abrupt blockage often at a branching point.

TREATMENT

The key to successful management is a high index of suspicion.

There is a massive fluid loss as a consequence of mesenteric ischaemia.

The adequate replacement of this is dramatic and often under estimated fluid loss is of private importance.

Embolectomy should be carried out by mainly arteriotomy over superior mesenteric artery. Any non viable bowel is resected.

Despite revascularisation , the mortality rate is reported between 20 and 70%.

Acute mesenteric ischarmia secondary to thrombosis is treated by short aorto superior mesenteric artery bypass, preferably with saphenous vein.

An important aspect of the surgical management of patients with acute mesenteric ischaemia is “second look’ laparotomy within 24 hours of the first procedure.

MANAGEMENT OF STRANGULATED HERNIA

- Diagnosis is mainly based on the clinical examination
- Treatment is by emergency operation.
- Vigorous resuscitation and antibiotics are essential
- During operation, the sac was delivered out and sac is incised near the fundus and toxic fluid was let out. Then neck of the sac was widened. Gangrenous segment of bowel are excised by localized resection. Then repair of the hernia is done.

DIVERTICULAR DISEASE MANAGEMENT

MECKEL'S DIVERTICULITIS

Meckel's diverticulum is very difficult to demonstrate by contrast radiology. Small bowel enema would be most accurate investigation.

In cases of repeated gastro intestinal haemorrhage of unknown cause where a meckel's diverticulum is suspected , technetium – 99 m scanning can be done to detect meckel's diverticulum.

TREATMENT [1]

If it is narrow based then excision of Meckel's diverticulum was done.

If it is broad based, wedge resection of ileal segment is done.

Where there is induration of base of diverticulum extending into the adjacent ileum , it is advisable to resect a short segment of ileum containing the diverticulum, restoring continuity with an end – to end anastamosis.

In jejunal diverticulitis associated with major malabsorbtion problems due to connective tissue disorders, resection of the affected segment with end – end anastamosis can be effective .

MANAGEMENT OF SMALL BOWEL LYMPHOMAS

In acute cases of perforation, diagnosis is made on clinical grounds & laparotomy.

Small bowel contrast enema, CT Scan will be helpful in other cases.

TREATMENT

In complicated cases of small bowel lymphoma with perforation, laparotomy and resection of the involved segment is done.

Further treatment consists of combination chemotherapy with drug regimens CHOP, CMOPP or radiotherapy.

In uncomplicated lymphoma, surgery followed by chemotherapy or radiotherapy in used for stage I and II disease.

Chemotherapy alone is used for more advanced disease.

MANAGEMENT OF MISCELLANEOUS CONDITIONS

MANAGEMENT OF PERFORATION OF RADIATION ENTERITIS

Emergency surgery is required for infarction with perforation or acute intestinal obstruction which does not resolve on conservative treatment .

The radionecrosed bowel is ideally excised with primary anastamosis or exteriorization of the bowel ends in the presence of ischaemia and sepsis .

MANAGEMENT OF NECROTISING ENTEROCOLITIS [1]

Abdominal radio graphs may show pneumatosis intestinalis or free intraperitoneal air .

Management consists of aggressive resuscitation with intravenous feeding. The optimal time for surgery is not in the acute phase as the baby can with stand the pressure of necrosis than an adult but not the stress of laparotomy.

At laparotomy, excision of all necrotic bowel with primary anastamosis is usual . The overall mortality is 25% with 10-30% of neonates developing a colonic stricture .

MANAGEMENT OF MECONIUM PERITONITIS[1]

Free air in the peritoneal cavity, an abundant quantity of abdominal fluid, fluid levels, calcification are characteristic findings, all of which are unlikely to be present in every case. Meconium peritonitis has been diagnosed by radiography of the fetus in uteus before birth.

TREATMENT

The prognosis is poor

The greatest chance of survival is in those patients who have an intestinal perforation but not intestinal obstruction in which case closure of the perforation and drainage of the peritoneal cavity are performed expeditiously.

Intestinal lavage can prevent reformation of meconium bolus obstruction & supplements of pancreatic exocrine enzymes are often necessary throughout life.

INTRA ABDOMINAL HYPERTENSION AND THE ABDOMINAL COMPARTMENT SYNDROME

Increased intra abdominal pressure (IAP)[43] occurs in a variety of clinical situations such as accumulation of ascites, bowel distension from the ileus or mechanical obstruction and reduction into peritoneal cavity of large chronic hernia contents lost their domain.

The factors that contribute intra abdominal hypertension after trauma are

- Accumulation of blood & clot
- Bowel edema or congestion from injury to mesenteric vessels or excessive crystalloid resuscitation.
- Closure of swollen & non compliant abdominal wall under abdominal wall under tension.

Abdominal compartment syndrome[43] is a late manifestation of uncontrolled intra abdominal hypertension . Abdominal compartment syndrome is characterized by tensely distended abdomen, elevated intra abdominal & peak airway pressures, inadequate ventilation with hypoxia & hypercarbia , distrubed renal function and improvement of these features after abdominal decompression.

IAP can be monitored by Direct or indirect methods

DIRECT METHOD

It is measurement of IAP by attaching the catheter placed into peritoneal cavity to a saline manometer on a pressure transducers.

Abdominal pressure measurement during laparoscopy is an example of direct method .

INDIRECT METHOD

Here pressure is measured through other accessible abdominal organ which reflect IAP.

IVC PRESSURE

It is by placing the catheter in groin and then into IVC. Complications are infections & thrombosis.

INTRAGASTRIC PRESSRUE

It is measured by water manometery through nasogastric tube or a gastrostomy tube

BLADDER PRESSURE

Kron & Colleagues[41] first described the technique of using bladder pressure as a mean of assessing intra abdominal pressure. Urinary bladder is an extraperitoneal , intra abdominal structure with a very complaint wall. Changes in IAP are parallel to intra luminal bladder pressure. It can be monitored continuously or intermittently.

INTERMITTENT

It is more popular. Here 50 ml of saline is installed into bladder through foleys catheter . The tubing of collecting bag is clamped. The needle is inserted into the tube proximal to clamp and attached to manometer . Bladder pressure measured in water is the height at which the level of the saline column stabilizes with pubic symphysis as the zero point .

INTRA ABDOMINAL HYPERTENSION

Significant organ dysfunction occurs when the intra abdominal pressure is above 10 mm Hg.

Burch and associates[38] classified elevated IAP

Grade I - 10 –15 cms of H_2O

Grade II - 15-25 cms of H_2O

Grade III- 25 - 35 cms of H_2O

Grade IV- >35 cms of H_2O

ADVERSE PHYSIOLOGICAL EFFECTS

When IAP is above 14cm of H_2O , patient developed decreased venous return and cardiac output. Splanchnic hypertension & hypoperfusion occurs when IAP is 20.4 cm of H_2O. Hence whole body O_2 consumption , pH & arterial Po2 are decreased. Diminished venous return is the major cause,

increased after load is the another cause. They show marked elevation of CVP, mean pulmonary artery pressure and pulmonary capillary wedge pressure.

RENAL EFFECTS

Anuria occurs when IAP rises. Decreased cardiac output is one cause. shunting of blood away form the renal cortex into medulla, diminution of renal blood flow , direct compression of kidneys or renal veins, diminished renal arterial flow and a rise in renal vascular resistance and presence of high levels of ADH are other possible causes of renal malfunction.

ABDOMINAL WALL

IAH reduces tissue blood flow. So wound infection with fascial dehiscence are common

SPLANCHNIC FLOW

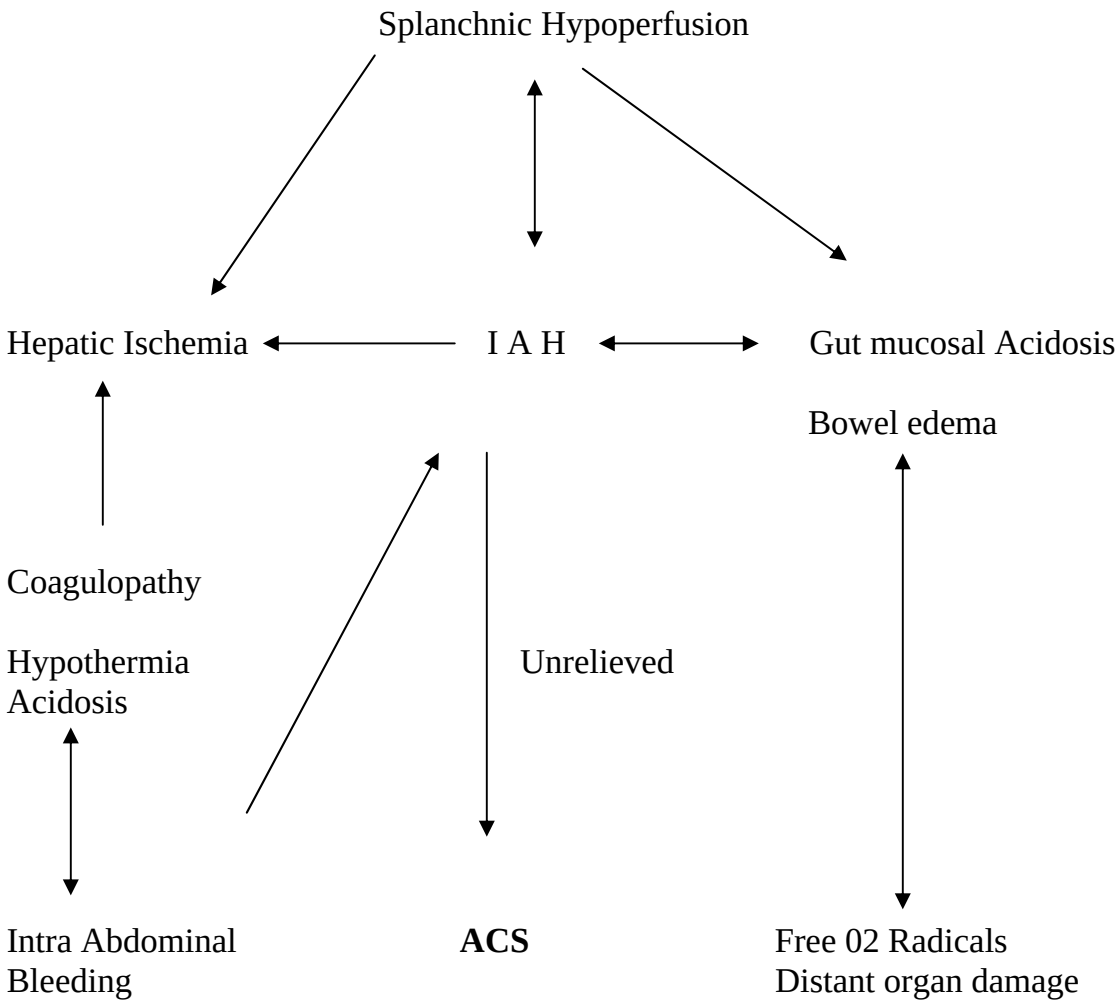
Reduction of blood flow to all abdominal organs except adrenal glands occur when IAP rises. IAH causes significant intestinal ischaemia followed by reperforation injury after abdominal decompression.

INTRA CRANIAL PRESSURE

IAH causes elevated intra cranial pressure and decreased cerebral perfusion pressure. Due to elevated IAP , CVP raised. Venous drainage from the cerebral venous outflow is decreased . The diminished cardiac output due to IAP also causes neuronal damage due to decreased CPP.

THE VICIOUS CIRCLE OF EVENTS CREATED BY IAH

ON SPLANCHNIC CIRCULATION



ADVERSE HEMODYNAMIC CONSEQUENCES OF RELIEF OF IAH.[40]

Sudden and severe hypotension may occur during or immediately following surgical decompression of IAH. It is due to sudden decrease in systemic vascular resistance, either from resumption of flow to a constricted splanchnic bed or the so-called reperfusion syndrome descriped by Morris et al[40]

REPERFUSION SYNDROME

Reperfusion syndrome occurs when the IAH is suddenly relieved.

- An abrupt increase in the true tidal volume delivered to the patient. It will result in metabolic alkalosis.
- Washout of bye products of anaerobic metabolism from below the diaphragm. Potassium and other products of anaerobic metabolism are delivered to the heart, patients develop cardiac asystole following reperfusion.
- To prevent this, 2 litres of solution composed of 1 litre of 45% normal saline with 50 gm mannitol and 40 mEQ of sodium bicarbonate are prepared and are infused immediately before and continued during decompression.

- Complete cardiac resuscitation team should be available at the bed side during decompression.

Each individual member of the team is primarily responsible for a single task or aspect of the patient's care. The team consists of the following people.

- ✓ Two surgeons to conduct the operation
- ✓ An experienced physician to manage the ventilator
- ✓ A person whose sole responsibility is infusion of fluid and blood products
- ✓ Two runners/ circulators and
- ✓ One scribe

ENDORGAN INVOLVEMENT

Three systems are commonly involved in IAH

1. CVS
2. Renal
3. Pulmonary

1. CARDIVASCULAR EFFECTS

Obstruction of blood flow through inferior vena cava , result in decreased cardiac output and leads to hypotension. Splanchnic blood flow also decreased.

2. RENAL SYSTEM

Oliguria occurs due to

- Inadequate renal arterial perfusion from poor cardiac output
- Direct compression of kidney
- Obstruction of renal vascular flow
- Obstruction of ureteral outflow

3. PULMONARY EFFECTS

It may present as intractable hypercarbia and extremely poor compliance. Patients have steadily increasing peak inspiratory pressure. These are due to compression of lung parenchyma and progressive upward displacement of diaphragm.

MATERIALS AND METHODS

This study has been based on the analysis of small bowel perforations 66 cases admitted in the IV surgical unit, Government Rajaji Hospital, Madurai Medical college during a period from November 2004 to February 2006.

In all patients admitted age, sex, associated illness were noted. Time interval between admission and time of intervention was noted.

Investigations including blood urea, blood sugar, serum creatinine, serum electrolytes, chest x ray , blood grouping , x ray abdomen & further investigation as required were done.

All the patients presented were resuscitated with crystalloids and after achieving hemodynamic stability these patients were subjected to emergency laparotomy, when perforation was proved clinically.

The intra abdominal pressure was measured indirectly by measuring the bladder pressure. The bladder was catheterized with foley's catheter. The urine drainage tube was clamped and one needle inserted proximally. It was connected to the three way adapter one to saline stand and other to the manometer. The manometer is filled with saline. About 50ml of saline was infused into the bladder. Then the bladder through catheter and manometer were connected. The manometer reflects the bladder pressure. The bladder pressure is directly correlated to the intra abdominal pressure.

For the patients who presented with extreme degree of shock , an initial flank drainage was done in an attempt to let out toxic fluid and once general condition was improved perforation closure was done.

All the patients were put on preoperative antibiotics. During laparotomy site of perforation, degree of peritoneal contamination were assessed . In all patients bilateral flank drainage was kept irrespective of the degree of contamination .

Post operatively patient was evaluated for the development of complications, if any.

OBSERVATIONS AND RESULTS

66 cases of clinically suspected small bowel perforation were admitted in 2004 to 2006 in our ward. Of these 61 (92%) cases are male, 5 (8%) cases are female

TABLE 1 SEX RATIO

MALE	FEMALE
61(92%)	5(8%)

TABLE 2 AGE – SEX DISTRIBUTION

AGE GROUP	MALE	FEMALE	TOTAL
11-20	6	2	8
21-30	10	1	11
31-40	13	1	14
41-50	22	0	22
51-60	8	0	8
61-70	2	0	2
71-80	0	1	1
Total	61	5	66

Age group of 41-50 years recorded the highest incidence of small bowel perforation in the patients studied with maximum of 22 cases.

More than 50% of the patients (55%) in my study were in the age group 31-50 years.

TABLE 3 ASSOCIATED HISTORY

Associated history	No. of patients
Smoking	45
Alcohol	22
NSAID	16

90% of duodenal ulcer are associated with H.pylori infection.

It is one of the most potent producers of urease. Hence it can be detected by urea breath test and Rapid urease assay. But this facility is not available in our set up and hence it cannot be detected as an association factor in this study.

After H.pylori infection, ingestion of NSAIDS is the most common cause of peptic ulcer disease. NSAIDS are more prone for upper GI bleeding. In this study, NSAIDS form association with perforation in 25% of the patients studied.

Alcohol and smoking increases the acid content in the stomach and impairs ulcer healing thereby resulting in duodenal ulcer and form association with the duodenal ulcer. In this study, Alcohol is associated in 34% of the patients studied and smoking is associated in 69% of patients studied.

TABLE 4 SITE OF PERFORATION

SITE OF PERFORATION	NO OF PATIENTS
Duodenum	44(67%)
Gastric	5 (8%)
Ileum	6 (9%)
Appendix	2
Unknown	6 (9%)
No perforation	3

Among the site of perforations identified intraoperatively 44 cases of duodenal ulcer perforations were present. i.e. about 67%.

The actual small bowel perforations noted in my study after intraoperative findings include 50 cases, about 76% .

MORTALITY

Of these 66 patients, 10 patients died – 15% mortality.

TABLE 5

MORTALITY AFTER	MALE	FEMALE	TOTAL
B/L Flank drainage	3	2	5
Perforation closure	5	-	5

Among them, 6 cases were above 50 years, 8 cases were above 40 years.

The cause of death in 10 cases

6 cases - septicemia

2 cases - septicemia with DM

2 cases - post operative cardiac arrest

Among 10 deaths, intra abdominal pressure were noted.

5 cases - >30cm

3 cases - >27cm

2 cases - >20cm

Among 10 cases of mortality in this study

TABLE 6

SITE OF PERFORATION	NO OF PATIENTS
Unknown	5
Duodenum	4
Gastric	1

There is no mortality in ileal perforation .

CAUSES OF SMALL BOWEL PERFORATION

Among the causes of small bowel perforation in this study

Duodenal ulcer perforation - 44 cases 88%

Typhoid ileal perforation - 4 cases 8%

Tuberculous ileal perforation - 2 cases 4%

Intra abdominal pressure was measured indirectly by means of measuring intravesical pressure

TABLE 7

IAP	TOTAL	MORTALITY	SURVIVED
Grade I 10-15 cms	6	0	6
Grade II 15-25 cms	38	2	36
Grade III 25-35 cms	20	7	13
Grade IV 35 cms	2	1	1

Most of the perforation were presented at Grade II IAP of about 38 cases (58%), among these 36 cases survived.

The perforation with their IAP in Grade III are in the higher incidence of mortality when compared to other cases 7 cases, almost 35%.

Immediately after decompression either by laparotomy or B/L flank drainage, intra abdominal pressure drastically decrease and the patients general

condition was improved. The compression effects of increased intraabdominal pressure were released. There is improvement in respiratory functions, increased splanchnic and renal blood flow thereby increasing renal urine output and decreases the cardiac overload and improves the circulation. Also the septicemic foci was removed thereby improving the general condition of the patients.

Among the 50 cases of proven small bowel perforation in my study, intra abdominal pressure shows

TABLE 8

IAP	TOTAL	MORTALITY	SURVIVED
I	5	0	5
II	31	1	30
III	13	3	10
IV	1	0	1

Among these, 31 cases (about 62%) were presented at Grade II IAP & 44 cases (about 88%) were presented at Grade II and III IAP.

COMPLICATIONS

Among 50 cases of proven small bowel perforation, 14 cases had complications.

TABLE 9

No. of Cases	Complications
7 cases	Wound infection
2 cases	Wound gapping
1 case	B/L pneumonia
1 case	Fecal fistula
1 case	post operative adhesion
1 case	Iatrogenic urethral injury

Of these 13 cases, 7 cases were duodenal ulcer perforation.

All the ileal perforation cases in my study had complications.

- 3 cases has wound infections treated conservatively.
- 2 cases had wound gapping treated with secondary suturing.
- 1 case developed fecal fistula.

TIME INTERVAL BETWEEN ADMISSION AND INTERVENTION

TABLE 10

Among the 49 cases of proven small bowel perforation, Time interval between admission & intervention varies.

TIME INTERVAL BETWEEN ADMISSION & INTERVENTION	NO OF PATIENTS	MORTALITY
<2hrs	2	0
2-4 hrs	6	0
4-6 hrs	17	1
6-8 hrs	15	2
8-10 hrs	5	1
>10 hrs	5	0

About 80% of cases were treated promptly within 8 hours.

90% cases were treated within 10 hours

PERITONEAL FLUID

TABLE 11

The peritoneal fluid, in small bowel perforations were found to be

PERITONEAL FLUID	NO OF PATIENTS	MORTALITY
Bile stained clear	12	0
Bile stained cloudy	20	2
purulent	12	2
Feculent	6	0

All the feculent peritonitis were found to be due to ileal perforation.

TABLE 12

The month of these perforations presented varies

MONTH	NO OF PATIENTS	ILEAL PERFORATIONS
January	4	0
February	8	0
March	5	0
April	0	0
May	1	0
June	3	2
July	1	1
August	4	1
September	2	0
October	3	0
November	10	1
December	9	0

39 cases about 78% occurred between October and march of calendar year.

4 cases 67% of Ileal perforation presented in the midst of calendar year.

TREATMENT - TABLE 13

TREATMENT GIVEN	SURVIVED	MORTALITY	TOTAL NO.
Laparotomy	52	5	57
B/L flank drainage alone	0	5	5
B/L flank drainage followed by surgery	4	0	4

Even though the above table shows an apparent increase in mortality of those patients who have undergone flank drainage, they were having associated factor which increased the mortality independently.

DISCUSSION

Perforation has been found to be a major complication of peptic ulcer disease with a mortality rate ranging form 6 to 31% worldwide. Several factor might contribute to increased mortality in patients with duodenal ulcer perforation. In this study, mortality rate is 15%.

Age of the patient with duodenal ulcer perforation has been increasing in many western series. This is due to complex age cohort phenomenon. But in this study maximal clustering of cases was found in fourth, fifth decades. Stress factor may be an added factor.

Factor associated with increased morality were studied in detail. Older age more than 50 years, presence of shock at the time of presentation, delayed presentation more than 24 hour, presence of co-morbid conditions were found to have significant impact on increasing the mortality rate.

All cases were having anterior perforation. Posterior perforation was not encountered in this study. Awareness of this serious condition however is important because the best chance for survival of patients lies in prompt and thorough surgical exploration, drainage and when appropriate, definitive surgery.

We could not undertake definitive surgery at the time of peptic ulcer closure because majority of the cases was having risk factor which would have

increased the mortality considerably. It is also true that we are not able to compare the efficacy of treatment by different surgical procedures (like omental plugging vs patch) because of the poor matching of confounding factor in the study group.

Simple closure alone was found to be effective in healing of the perforated ulcer with medical therapy preventing recurrence of the disease on follow up.

There was an increased death rate in patients treated with flank drain above, because almost all of them had presence of two or more risk factors which we found to increase the mortality. In this study, there is 100% mortality in patients treated with flank drain alone.

The availability of effective medical treatment for acid peptic disease and the introduction of H. pylori eradication therapy had made definitive surgery unnecessary at the time of perforation closure.

Open surgery still remains the choice of treatment in majority of centres. Laparoscopic technique though allows for perforation closure, does not allow sufficient lavage of all quadrants to remove all sequelae of peritonitis. Minimal access surgery may be useful for early cases. In this study, all have been treated by open surgery.

Perforated peptic ulcer is likely to continue to be a surgical challenge despite the medical treatment. In this study, 69% of patients were found to be

smoker. 25% of patients were taken NSAIDS prior to the symptoms and 34% of patients were alcoholic.

In this study, there were 6 cases of ileal perforation.

All of the ileal perforation cases were managed with primary closure in 2 layers and all of them have been survived.

All the ileal perforations were presented with feculant peritonitis and all of them had post operative complication. All the ileal perforation cases had stormy postoperative period.

Among the 6 cases of ileal perforation, 4 cases were typhoid ileal perforations and were treated with primary closure of perforation site, although there are various modalities of treatment.

2 cases of rare tuberculous peritonitis were managed with primary closure and started anti tuberculous drugs post operatively and they impoved on follow up.

In this study, all typhoid ileal perforations were presented in the male. The reason for this male predominance is yet to be identified, although it is possible that men have an increased risk of exposure to typhoid fever.

The important investigations carried out to confirm the diagnosis are plain x-ray abdomen erect view for evidence of pneumo –peritonium, widal test and biopsy of the ileal at the site of perforation.

This study recommend, when general condition of the patients is good, simple closure alone is adequate in uncomplicated solitary perforation. But resection is necessary if there is bleeding or if multiple ulcers with more than one perforation are present. But in this case study, no resection was carried out.

Bilateral flank drainage may be of help in those patients who are too ill to with stand the laparotomy and after improving the patients general condition, laparotomy can be carried out.

All of the ileal ulcers in my study were confined to the last 40 cm of ileum.

Other than wound infection, the most common abdominal complication is wound dehiscence in Forrest's view[31], and in my study next to the wound infection is wound gapping. Wound gapping or dehiscence is the reflection of both high incidence of infection and debility of the patients. Just like wound dehiscence, the development of fecal fistula is catastrophic. This may be the result of re perforation, perforation in another area or the result of suture line break down. In this study, one case had fecal fistula.

When the intra abdominal pressure rises, there is shunting of blood away from renal cortex into medulla, diminution of renal blood flow leading to anuria and decreased cardiac output, these effects are returned back to normal when the abdominal decompression was done.

By early intervention to decompress the abdomen either by bilateral flank drainage or by laparotomy, patient's general condition can be improved by increasing the Cardiac output, relieving septicemic focus from abdominal cavity and increasing urine output.

In this study, 80% of cases were treated prompty with in 8 hours.

CONCLUSION

Among the small bowel perforation studied, Duodenal ulcer perforation forms the majority of cases about 88%.

Typhoid ileal perforation compromise about 8% of all small bowel perforation. i.e. 4 out of 50 cases. Tuberculous small bowel perforation comprise about 4% of all small bowel perforation i.e. 2 out of 50 cases. More than half of the cases were presented in the age group of 31- 50 years about 36 cases out of 66 cases.

Most of the small bowel perforations were presented at Grade II IAP. More than 95% of these were survived.

The patients with their IAP in Grade III & IV are in higher incidence of mortality 8 out of 14 cases, 36% has mortality .

In this study 80% of cases were managed within 8 hours of presentations with a mortality of 4 cases i.e. 8%.

All the ileal perforation cases were presented with feculent peritonitis & all of them have been survived.

High mortality occurs in small bowel perforations when the age is above 50 years i.e. 6 out of 10 cases about 60%.

About 78% of cases occured between October and March of calendar year.

So, by these methods, we will grade the small bowel perforation and assess the prognosis of these patients.

BIBLIOGRAPHY

1. Bailey and love's short practice of surgery – 24th Edition R.C.G. Russel , N.S. Williams , C.J.K. Bolstrode
2. Text book of surgery - Sabiston 17th Edition
3. Principles of Surgery - 8th Edition, Schwartz / Charles Brunicardi
4. Essential surgical practice - 4th Edition. A Cuscieri / J.C. Steele / A.R. Moosa.
5. Farquharson's Text book of operative surgery – 9th Edition, R. F. Rintoul.
6. Maingot's Abdominal operations – 10th Edition , Rodney Maingot.
7. A manual on clinical surgery – 6th Edition: S. Das
8. Surgery Annual – 1991 – Lloyd M. Nyhus .
9. Recent advances in Gastroenterology – 7th Edition , R. R. Pounder
10. Oxford textbook of surgery – 2nd Edition, Peter J. Morris and William C. Wood.
11. Christensen A, Bousfield R , Christensen J. Incidence of perforated and bleeding peptic ulcers before and after the introduction of H2 – receptor antagonist . Ann Surg 1988:207: 4-6.
12. Bliss DW, Stabile BE. The impact of ulcerogenic drugs on surgery for the treatment of peptic ulcer disease . Arch Surg 1991: 126 : 609-612.

13. Lana WL, Leung KL, Kwong KH, Davey IC, Robertson C, Dawson JJ . Chung SC, Li Ak. A randomized study comparing laparoscopic versus open repair of perforated peptic ulcer using suture or sutureless technique . Ann Surg 1996 ;224: 131- 138.
14. Boey J, Wong J, Ong GB . A prospective study of operative risk factors in performed duodenal ulcers . Ann surg 1982: 195: 265- 269.
15. Evans JP, Smith R. Predicting poor outcome in perforated peptic ulcer disease. Aust N Z /Surg 1997; 67 : 792 – 795.
16. Greiser WB, Burner BW, Shamoun JM, Jurkovich GJ , Ferrara JJ . Factors affecting mortality in patients operated upon for complications of peptic ulcer disease. Ann Surg 1989; 55: 7-11.
17. Irvin TT. Mortality and perforated peptic ulcer: a case for risk stratification in elderly patients. Br J Surg 1989; 76:215-218.
18. Suter M. Surgical treatment of perforated peptic ulcer. Is there a need for a change? Acta Chir Belg 1993;93: 83-87.
19. Lee Fy. Leung KL. Lai BS. Ng SS. Dexter S. Lau WY. Predicting mortality and morbidity of patients operated on for perforated peptic ulcers. Arch Surg 2001;139: 30-94.
20. Blomgren LGM. Perforated peptic ulcer: long-term results after simple closure in the elderly. World/Surg 1997;21:412-415.
21. Gray's Anatomy – 39th edition.

22. Last's Anatomy – 9th Edition
23. Hamby L.S. Zweng TN, Strodel WE. Performed gastric and duodenal ulcer: an analysis of prognostic factors. Am Surg 1993;59:319-324.
24. Matsuda M. et al., Laparoscopic omental patch repair for perforated peptic ulcer. Ann Surg 1995;221:336-240.
25. Wakayama T, et al., Risk factors influencing short term results of duodenal perforation. Surg Today 1994;24:681-687.
26. Celen Jones, "A rapid method of treatment of perforated peptic ulcer" Gasroenterology 33:353,1957.
27. Graham R.R. "The treatment of perforated Duodenal ulcer" Sur Gyaence obset. 64: 235, 1937.
28. Archampong E.Q (1976) Typhoid ileal perforations Br. J. Surg Vol 63 (1976) 317-321.
29. M.K. Chouhan and S.K. Pande (Rajasthan) Typhoid Enteric perforation. Br. J. Surg. Vol-69 (1982) 173-175.
30. Kuruvilla M.J.F.R.C.S.F.R.C.S. Role of Resection in Typhoid Perforation Ann. R. Coll. Durg., Eng Vol – 60 (1978).
31. Forrest C. Eggleston and Bannu Santoshi – Typhoid Perforation choice of perforation. Br. J. Surg., Vol – 68 (1981) 341-342.
32. Kim J. P. Oh S.K. & Jarrett E. Management of ileal Perforation due to Typhoid fever. Ann., Surg, 1975; 181: 88-91.

33. Venkataramani Sitaram M.S. (C.M.C.Vellore) Typhoid ileal perforations a Retrospective study. Ann R. Col. Surg., Engl., Nov – 1990 Vol – 72. No.6.
34. Purohit P.G. Surgical Treatment of Typhoid perforation Ind. J. Surg., 40: 227-238, 1978.
35. Egglesten F.C. Santoshi B & Singh C.M. typhoid perforation of the bowel; Experiences in 78 cases. Ann. Surg. 1979; 190: 31-35.
36. Barnes GE, Laine GA, Giam PY, et al: Cardiovascular responses to elevation of intraabdominal hydrostatic pressure. Am J physiol 248 :R208, 1985.
37. Bongard FB, Ryan M, Dubecz S, et al: Adverse consequences of increased intraabdominal pressure on bowel tissue oxygen, J Trauma 39: 519,1995.
38. Burch JM, Moore EE, Moore FA, et al: The abdominal compartment syndrome. Surg clin north Am 76:833, 1996.
39. Cullen DJ, Coyle JP, Teplick R, et al: Cardiovascular, pulmonary, renal effects of massively increased intraabdominal pressure in critically ill patients. Crit care Med 17: 118, 19889.
40. Morris JA Jr, Eddy VA, Blinman TA, et al: The staged celiotomy for trauma. Issues in unpacking and reconstruction, Ann Surg. 217:576, 1993.

41. Kron IL, Harman PK, Nolan SP : The measurement of intraabdominal pressure as a criterion for exploration. Ann Surg 199:28,1984.
42. Ian Aird – A companion of surgical studies – 3rd edition.
43. Rao R. Ivatury, Lawrence, M.Porter, J. Simon: Intraabdominal hypertension and the abdominal compartment syndrome Surg. Clin North Am. 77:783, 1997.

PROFORMA

A STUDY OF SMALL BOWEL PERFORATIONS

Name:	Age:	Sex:
Occupation:	Income:	IP No:
Address:		DOA:
		DOS:
		DOD:

PRESENTING SYMPTOMS:

- Fever
- Time interval between onset of fever and probable time of perforation
- Pain Abdomen
- Vomiting
- Bowel habits
- Urinary symptoms
- Other Symptoms

- H/O Drug intake
- Alcoholic
- Smoker

CLINICAL EXAMINATION:-

- Dehydration
- Pallor

- P/A - Distension

Rigidity / Guarding → Diffuse
Rigidity / Guarding → Localized

Obliteration of liver dullness

Bowel sound

- P/R
- Pulse rate
- BP
- Temperature
- Urinary output
- Time interval between time of admission and surgery

Associated Systemic illness, if any:

INVESTIGATION:

Hb

Blood – Urea

Creatinine

Sugar

Serum electrolytes

Urine

X-ray chest PA view

X-ray Abdomen erect view

other specific tests –

Widal test

Intraabdominal pressure

Pre procedure

Post procedure

OPERATIVE FINDINGS:

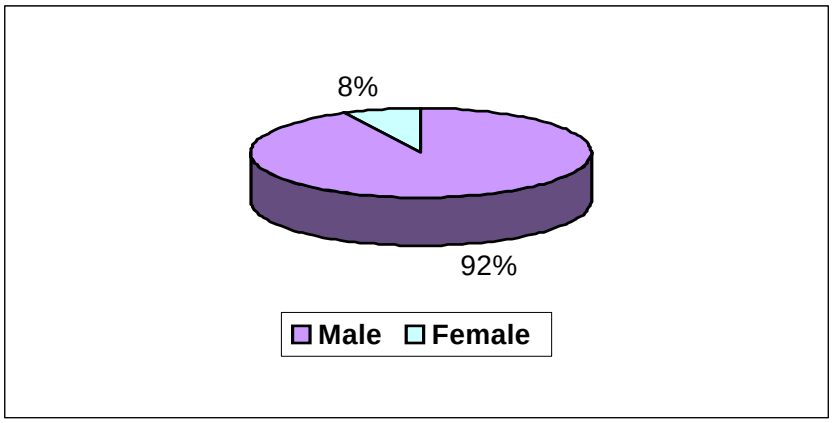
- Amount of Peritoneal fluid
- Site of Perforation
- Size of Perforation
- Other findings

PROCEDURE DONE:

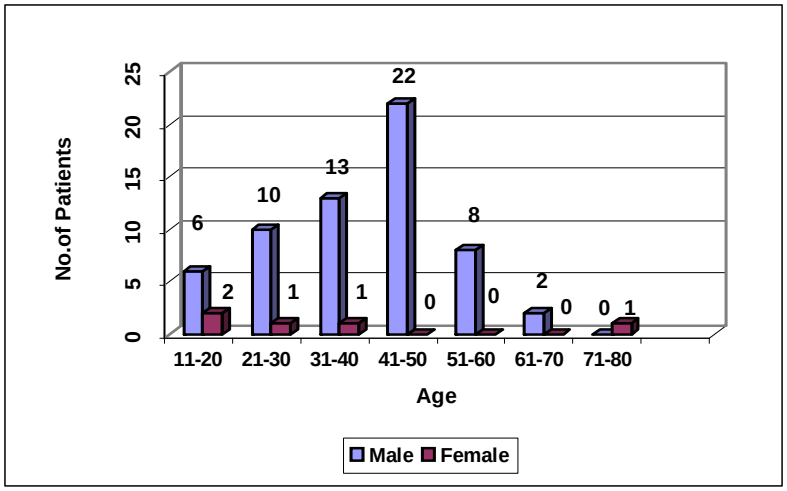
Post Operative Complications, if any:

Follow Up:

SEX RATIO



AGE – SEX DISTRIBUTION



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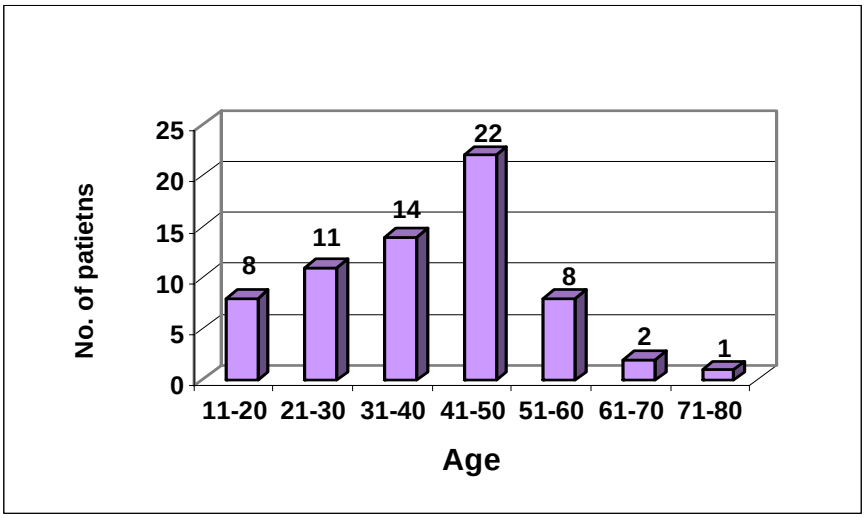
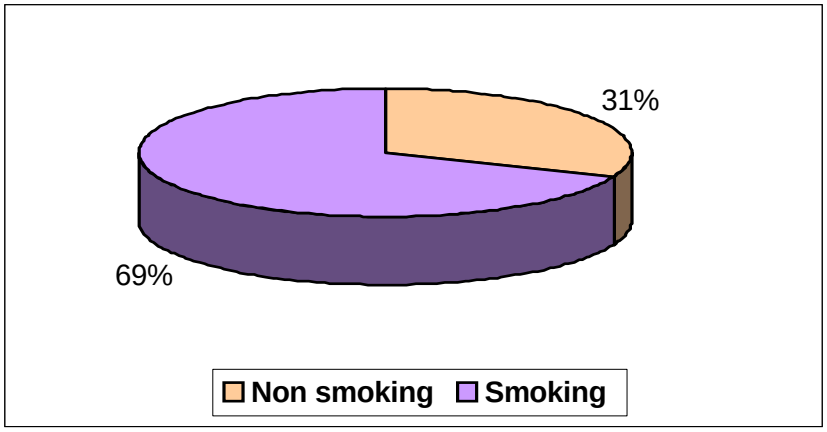
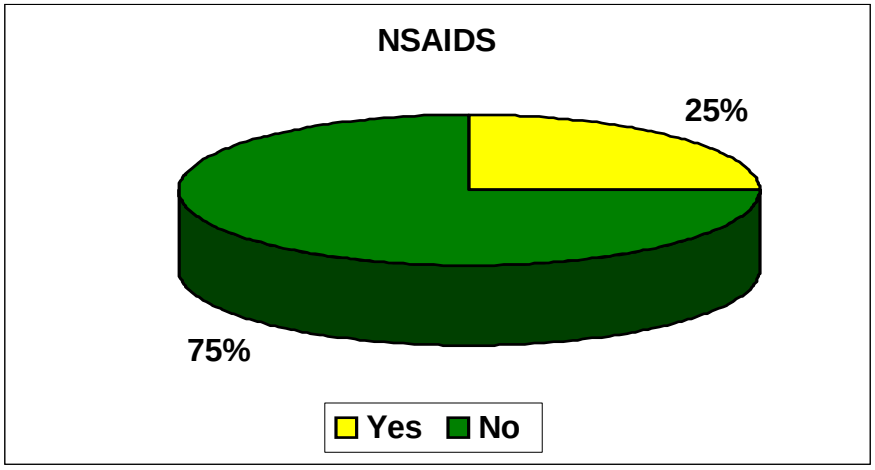
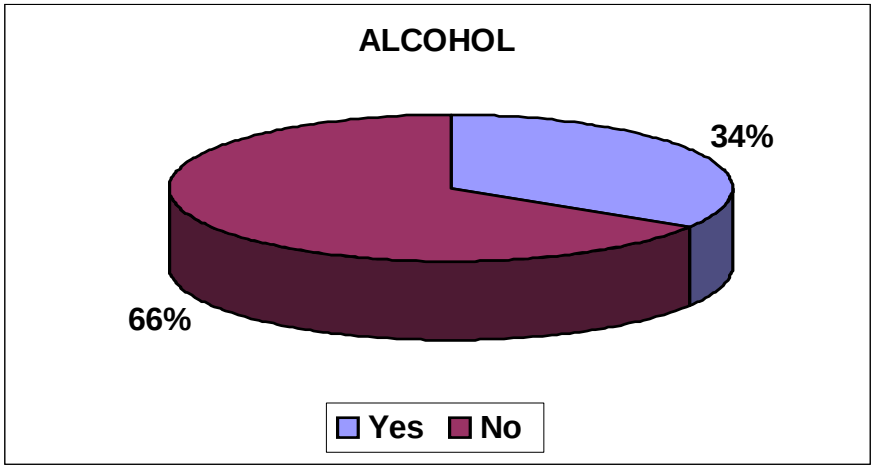


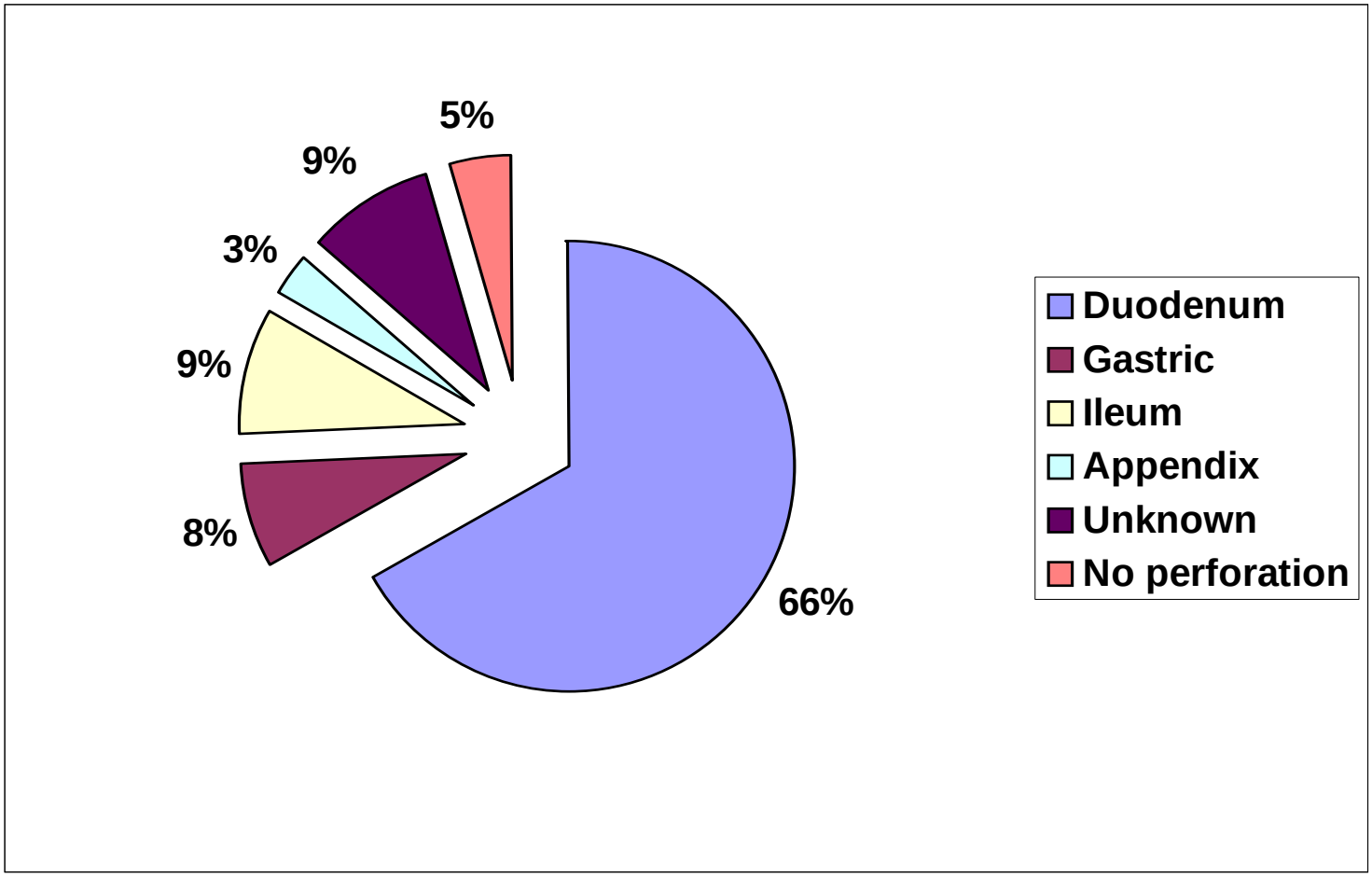
ASSOCIATED HISTORY



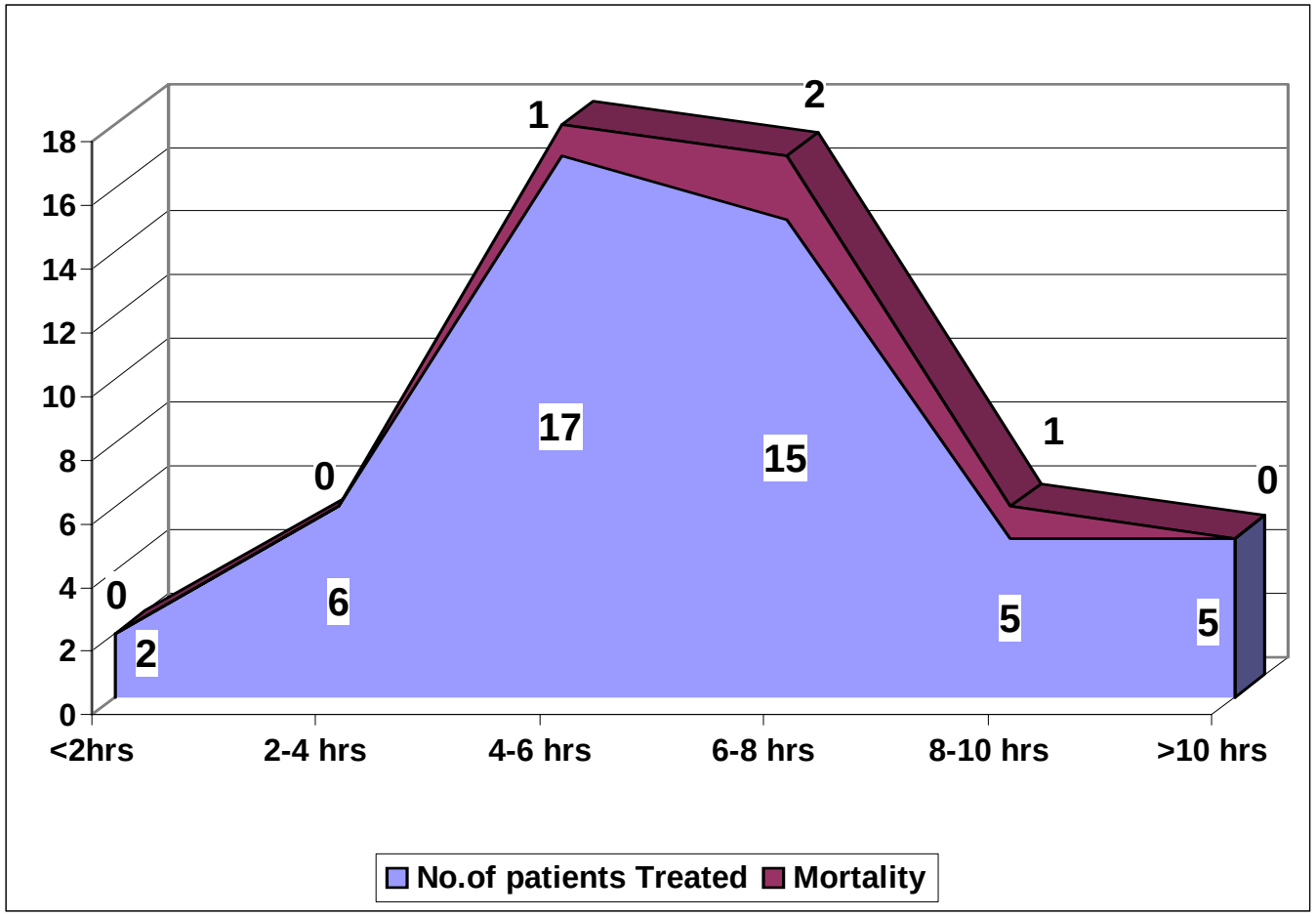




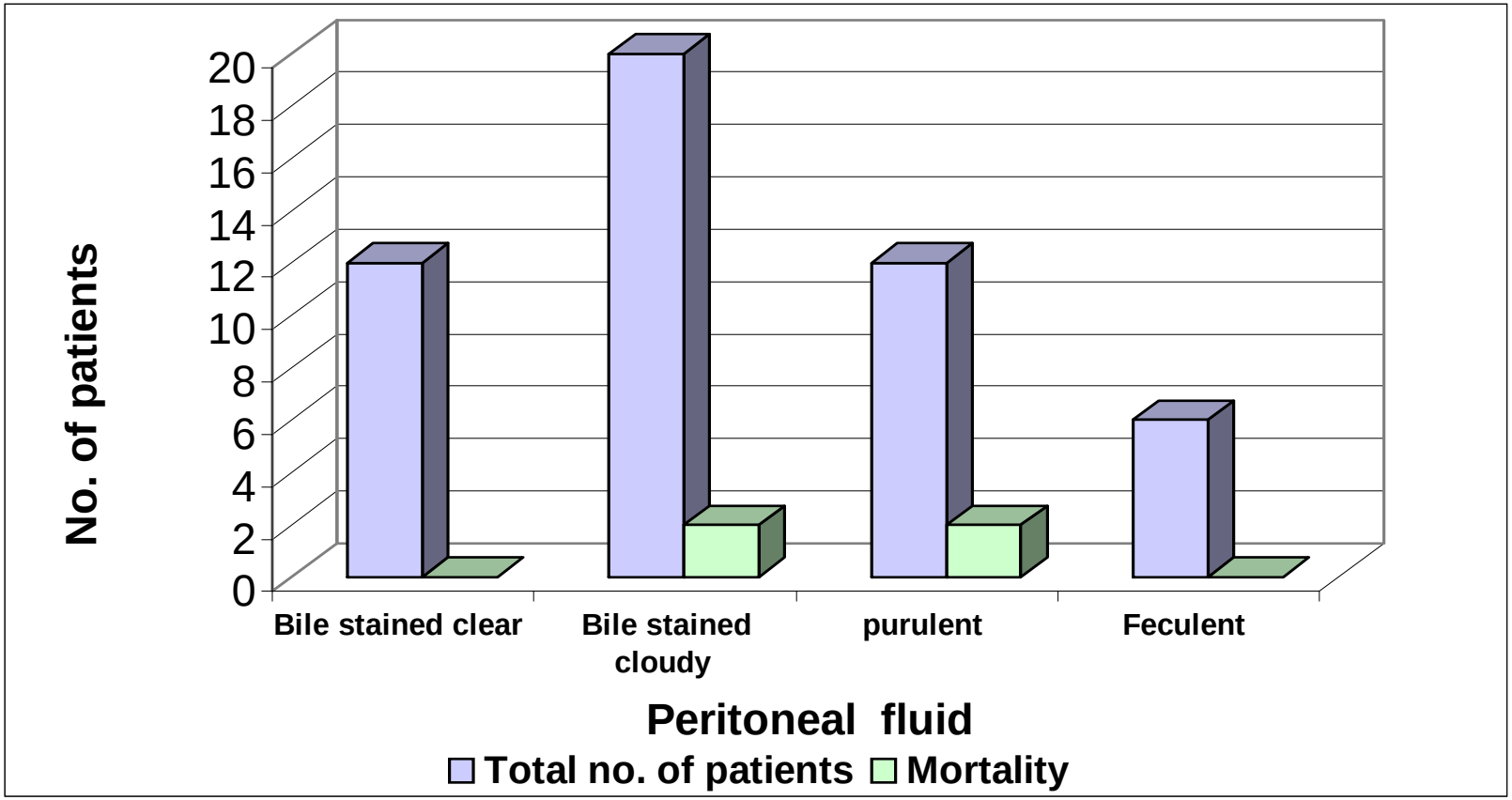
SITE OF PERFORATION



TIME INTERVAL BETWEEN ADMISSION AND INTERVENTION



PERITONEAL FLUID SEEN IN SMALL BOWEL PERFORATIONS



MASTER CHART

Sl. No.	Name	Age	Sex	I.P. No.	Time interval between admissin & intervention	Peritoneal fluid	Site of Perforation	Procedure done	Intraabdominal pressure		Month of admission	Outcome
									Pre Procedure (cm)	Post Procedure (cm)		
1	Alagu	45	M	315957	9 hours	Bile stained cloudy 1200ml	Duodenum I 0.5x0.5cm	Simple closure	29	16	November	Alive
2	Diraviam	55	M	317270	8 hours	Purulent 650ml	Duodenum I 0.5x0.5cm	Simple closure	22	12	November	Alive
3	Subramani	50	M	317246	6 hours	Purulent 350ml	Duodenum I 1x0.5cm	Simple closure	29	14	November	Alive
4	Vengaram	28	M	318462	8 hours	Purulent 800ml	Duodenum I 1x0.5cm	Simple closure	28	13	December	Alive
5	Saradammal	77	F	322905	8 hours	Purulent 350ml	-	B/L flank drainage	34	18	December	Dead
6	Chellamani	18	M	324059	4 hours	Bile stained clear 200ml	Gastric 1x1cm	Simple closure	16	9	December	Alive
7	Nagaraj	22	M	325041	2 hours	Bile stained cloudy 200ml	Duodenum I 1x0.5cm	B/L flank drainage followed by simple closure	24	13	December	Alive
8	Rajeshkannan	22	M	326114	6 hours	Bile stained cloudy 350ml	Duodenum I 1x0.5cm	Simple closure	30	14	January	Alive
9	Palaniappan	40	M	336212	7 hours	Purulent 1250ml	Duodenum I 1x0.5cm	Simple closure	27	13	February	Alive
10	Markandeyan	45	M	336358	2 hours	Purulent 500ml	-	B/L flank drainage	33	18	February	Dead
11	Murugan	36	M	337646	6 hours	Bile stained cloudy 150ml	Duodenum I 0.5x0.5cm	Simple closure	19	10	February	Alive
12	Pandi	45	M	337620	10 hours	Purulent 750ml	Duodenum I 1x1cm	Simple closure	25	12	February	Alive

13	Chinnasamy	45	M	337643	12 hours	Bile stained clear 500ml	Gastric 4x3cm	Partial gasrectomy & Polya cytopericyste - ctomy of hydatid cyst	26	12	February	Alive
14	Chinnraj	51	M	337526	16 hours	Purulent 300ml	Gastric 0.5x0.5 Liver 4x3 ® lobe	Simple closure & drainage	22	9	February	Alive
15	Palani	60	M	342095	13 hours	Purulent 1.5 lit	Duodenum I 0.5x0.5cm	Simple closure & femoral hernia repair	20	12	March	Alive
16	Pandiselvi	14	F	339943	5 hours	Bile stained cloudy 350ml	-	B/L flank drainage	36	22	March	Dead
17	Rafeek	41	M	344480	8 hours	Purulent 1.5 lit	Duodenum I 0.5x0.5cm	Simple closure	24	10	March	Alive
18	Sekkadiyan	37	M	345605	8 hours	Bile stained cloudy 200ml	Duodenum I 0.5x0.5cm	Simple closure	22	12	March	Alive
19	Karuppiah	38	M	344441	5 hours	Bile stained cloudy 600ml	Duodenum I 1x0.5cm	Simple closure	28	15	March	Dead
20	Tamilselvan	45	M	345675	6 hours	Bile stained cloudy 3.5 lit	Gastric 0.5x0.5 cm	Simple closure	22	12	March	Alive
21	Ramamoorthy	57	M	345650	5 hours	Purulent 400ml	Duodenum I 0.5x0.5cm	Simple closure with epigastric hernia repair	19	10	March	Alive
22	Alagarsamy	26	M	356805	6 hours	Bile stained cloudy 800ml	Duodenum I 0.5x0.5cm	Simple closure	24	12	May	Alive
23	Karuppiah	50	M	363826	8 hours	Purulent & feculent 300ml	Ileum 30cm from IC junc. 2x0.5cm	B/L flank drainage follow by simple closure	26	14	June	Alive
24	Kottaisamy	50	M	367314	6 hours	Purulent & feculant 600ml	Ileum 40cm from IC junc. 2x0.5cm	Simple closure	30	15	June	Alive
25	Subramani	40	M	368520	7 hours	Bile stained cloudy 500ml	Duodenum I 0.5x0.5cm	Simple closure	26	13	June	Alive
26	Vigneshwaran	17	M	373395	5 hours	Purulent & feculant 250ml	Ileum 15cm from IC junc. 2x0.cm	Simple closure	28	12	July	Alive

27	Jesudoss	16	M	378148	14 hours	Purulent 350ml	Appendix	Apprendicectomy	28	14	August	Alive
28	Malar	14	F	378197	14 hours	Purulent 500ml	Appendix	Apprendicectomy	25	12	August	Alive
29	Govindasamy	26	M	378203	17 hours	Bile stained cloudy 300ml	Duodeunum I 0.5x0.5cm	Simple closure	22	10	August	Alive
30	Pandiarajan	50	M	374153	7 hours	Bile stained cloudy 600ml	Duodenum I 0.5x0.5cm	Simple closure	26	12	August	Alive
31	Lokeshmuthupandi	16	M	379458	8 hours	Purulent & feculant 1.5 lit	Ilem 30cm from IC junc. 2 perforations 1x0.5cm	Simple closure	40	15	August	Alive
32	Shanmugam	70	M	379180	10 hours	Purulent 400ml	Duodenum I 1x0.5cm	Simple closure	27	14	August	Dead
33	Subbaiah	52	M	384731	7 hours	Bile stained cloudy 700ml	Duodenum I 0.5x0.5cm	Simple closure	24	11	September	Alive
34	Mariagoundar	50	M	385917	7 hours	Bile stained cloudy 1.2. lit	Duodenum I 0.5x0.5cm	Simple closure	29	12	September	Dead
35	Selvam	25	M	378189	9 hours	Bile stained cloudy 800ml	Duodenum I 0.5x0.5cm	Simple closure	14	14	October	Alive
36	Arumugam	50	M	398235	6 hours	Bile stained clear 600ml	Duodenum I 0.5x0.5cm	Simple closure	10	9	October	Alive
37	Somasundaram	55	M	398243	7 hours	Purulent 1.5 lit	Duodenum I 0.5x0.5cm	Simple closure	22	13	October	Dead
38	Karthik Raja	17	M	398812	12 hours	Feculant 1 lit	Ilem 20cm from IC junc.1x0.5cm	Simple closure	18	12	November	Alive
39	Rajeswari	22	F	399196	12 hours	Purulent 1.8 lit	Sealed perforation	Drainage only	19	12	November	Alive
40	Anthonimuthu	35	M	399367	3 hours 45 min	Purulent 1.5 lit	Duodenum I 2x1cm	Simple closure	17	13	November	Alive
41	Singaperumal	45	M	400709	4 hours 45 min	Bile stained clear 500ml	Duodenum I 0.5x0.5cm	Simple closure	17	13	November	Alive
42	Tamilagan	43	M	400683	4 hours	Bile stained clear 200ml	Duodenum I 0.5x0.5cm	Simple closure	16	8	November	Alive
43	Muthiah	45	M	401912	4 hours	Bile stained cloudy 800ml	Duodenum I 0.5x0.5cm	Simple closure	22	14	November	Alive

44	Meiappan	50	M	400677	9 hour 30 min	Bile stained cloudy 3 lit	-	B/L flank drainage	20	9	November	Dead
45	Arumugam	35	M	403004	5 hours	Bile stained cloudy 900ml	Duodenum I 0.5x0.5cm	Simple closure	19	10	November	Alive
46	Srirengam	42	M	403200	4 hours	-	-	Adhesive obstruction release	24	16	November	Alive
47	Perumal	45	M	403465	6 hour 30 min	Bile stained cloudy 800ml	Duodenum I 0.5x0.5cm	Simple closure	19	9	November	Alive
48	Sangusekarandi	28	M	403104	5 hours	Bile stained clear 1.5 lit	Gastric 1x0.5cm	Simple closure	32	16	November	Dead
49	Ayothiraman	25	M	404240	7 hours	-	-	Adhesiolysis	11	9	December	Alive
50	Karuppiah	50	M	407784	5 hours	Bile stained cloudy 900ml	Duodenum I 0.5x0.5cm	Simple closure	20	13	December	Alive
51	Alagappan	65	M	407771	3 hours	Purulent 100ml	-	B/L flank drainage	28	18	December	Dead
52	Ramalingam	40	M	409038	5 hours	Bile stained clear 500ml	Duodenum I 0.5x0.5cm	Simple closure	16	8	December	Alive
53	Ganesh	60	M	409041	7 hours	Bile stained clear 300ml	Duodenum I 0.5x0.5cm	Simple closure	18	9	December	Alive
54	Karthik	18	M	409055	3 hours	Bile stained clear 200ml	Duodenum I 0.5x0.5cm	Simple closure	17	9	December	Alive
55	Murugan	35	M	410317	10 hours	-	-	obstructed hernia release	9	8	December	Alive
56	Andisamy	42	M	410330	4 hours 30 min	Bile stained clear 200ml	Duodenum I 0.5x0.5cm	Simple closure	16	9	December	Alive
57	Karuppasamy	45	M	411632	9 hours	Biel stained cloudy 1.5 lit	Duodenum I 0.6cm	Pyloroplasty	18	12	December	Alive
58	Julie	32	F	411686	11 hours	Bile stained cloudy 3.5 lit	Duodenum I 1x0.5cm	Simple closure	25	14	December	Alive
59	Sagayaraj	28	M	413993	4 hours	Bile stained clear 250ml	Duodenum I 0.5x0.5cm	Simple closure	18	8	January	Alive
60	Mahalingam	21	M	417376	5 hours	Bile stained cloudy 1.5lit	Duodenum I 0.5x0.5cm	B/L flank drainage followed by simple closure	24	14	January	Alive
61	Kannan	40	M	418637	2 hours	Purulent 1lit	Duodenum I 3x2cm	B/L flank drainage followed by duodenal fistula with AGJ	14	10	January	Alive

62	Karuthan	35	M	420011	3 hours	Bile stained clear 1lit	Duodenum I 0.75x0.75cm	Simple closure	12	9	February	Alive
63	Mayandi	42	M	420010	4 hours 30 min	Bile stained clear 500ml	Duodenum I 1x0.5cm	Simple closure	18	12	February	Alive
64	Patchaimuthu	34	M	421350	7 hours	Bile stained cloudy 500ml	Duodenum I 0.5x0.5cm	Simple closure	18	10	February	Alive
65	Bose	37	M	421351	13 hours	Bile stained clear 500ml	Duodenum I 0.5x0.5cm	Simple closure	19	12	February	Alive
66	Muthu	68	M	425643	6 hours	Purulent & feculant 200ml	Ileum 40cm from IC junc. 1x0.5cm	Simple closure	12	8	February	Alive