# CERTIFICATE

This is to certify that this dissertation in **"NON TRAUMATIC GASTROINTESTINAL PERFORATIONS"** is a work done by **Dr.A.ANANDHI** under my guidance during the period 2005 -2008. This has been submitted in partial fulfillment for the award of **M.S. Degree in General Surgery (Branch-1)** by The Tamilnadu Dr.M.G.R. Medical University, Chennai.

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## INTRODUCTION

Perforation peritonitis is the most common surgical emergency in India. 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 [1] and there is paucity of data from India regarding it's etiology, prognostic indicators, morbidity and mortality patterns [2]. Our study was designed to highlight the spectrum of perforation peritonitis as encountered by us at KILPAUK MEDICAL COLLEGE AND HOSPITAL, CHENNAI.

# AIMS OF THE STUDY

- To study the epidemiology, seasonal trends, etiology and clinical presentation.
- To study the incidence in perforation in different part of GIT.
- To study the different management techniques used.
- To study the factors influencing the outcome.
- To study the morbidity and mortality.

## REVIEW OF LITERATURE

Gastro Intestinal perforation is a complete perforation of the wall of the stomach, small intestine or large bowel, resulting in intestinal contents flowing into the abdominal cavity. Perforation of the intestine results in the potential for bacterial contamination of the abdominal cavity (a condition known as peritonitis). Perforation of the stomach can lead to chemical peritonitis due to leaked gastric acid. Perforation any where along the gastro intestinal tract is a surgical emergency.

## RELEVENT ANATOMY

The peritoneal cavity is lined with a single layer of mesothelial cells, connective tissue (including collagen), elastic tissues, macrophages and fat cells. The parietal peritoneum covers the abdominal cavity (i.e.) abdominal wall, diaphragm, pelvis. The visceral peritoneum covers all the intra-abdominal viscera, forming a cavity that is completely enclosed except at the open ends of the fallopian tubes.

The peritoneal cavity is divided by the transverse mesocolon. The greater omentum extends from the transverse mesocolon and from the lower pole of the stomach to line the lower peritoneal cavity.

Abdominal organs such as the pancreas, duodenum, ascending colon and descending colon are located in the anterior retroperitoneal space. The kidney, ureter and adrenal glands are found in the posterior retro peritoneal space. Other abdominal organs, the liver, stomach, gallbladder, spleen, jejunum, ileum, transverse colon, sigmoid colon, caecum and appendix are found with in the peritoneal cavity.

A small amount of fluid sufficient to allow movement of organs is usually present in the peritoneal cavity. This fluid is normally serous (Protein content of <30g/l, <300WBCs/µl). In the presence of infection the fluid becomes an exudate.

## GASTRO DUODENAL PERFORATION

**Gastric Perforation:** Perforation 1 to 2 cms proximal to the pyloric ring.

Duodenal Perforation: Distal to pyloric ring upto duodeno jejunal flexure perforations.

**ETIOLOGY**

- Peptic ulcer
- Drugs like NSAIDS
- Cushing's ulcer
- Curling's ulcer
- Malignancy
- Trauma
- Iatrogenic perforation
- Radiotherapy of cervical cancer and other intra abdominal malignancy. The late complication of which is obstruction and perforation
- Ingestion of caustic substance
- Zollinger Ellison syndrome

**INCIDENCE:**

The incidence of perforated peptic ulcer is approximately 10 to 15 cases per 1,00,000 population per year. About 0.7% of the ulcers are complicated by perforation(3).

It is the first manifestation of disease in about 2% patients with duodenal ulcer.

Overall and despite the wide spread use of gastric antisecretory agents and eradication therapy the incidence of perforated peptic ulcer has changed little.

**AGE:**

Previously more common in middle aged between 30 to 50 years.

Increasing use of NSAID have resulted in an increased incidence in $6^{th}$ and $7^{th}$ decade. But this data suits only the developed countries. In developing countries like India the young adult population is most commonly affected.

**SEX:**

Male: Female ratio is 2:1. Now steady increase in number of females especially older females due to wide spread use of NSAIDS.

**OCCUPATION:**

More Common in lower socioeconomic status

**SEASON:**

More Common in winter season.

**PATHOPHYSIOLOGY:**

Perforation of an ulcer is due to sudden sloughing of base of the ulcer due to impairment of blood supply. Normally, the stomach is relatively free from bacteria & other micro organism. Presence of bacteria in the peritoneal cavity stimulates an influx of acute inflammatory cells. The omentum and viscera tend to localize the site of inflammation producing a phlegmon. (This usually occurs in perforation of large bowel). The resulting hypoxia on that area facilitates the growth of anaerobes and produces impairment of bactericidal activity of granulocytes, which leads to increased phagocytic activity of granulocytes, degradation of cells, hypertonicity of fluids forming the abscess, osmotic effects, shift of more fluid into the abscess area, and enlargement of the abdominal abscess. If untreated bacteremia, paralytic ileus, generalized sepsis, multi organ failure and shock may occur.

The infection is usually mixed with gram negative aerobes & anaerobes in addition to gram positive bacteria Escherichia coli & Bacteroides fragilis are predominant(4). 90% of all the perforated duodenal ulcer occurs in the anterior wall of the duodenal bulb 60% of perforated gastric ulcer are located on the lesser curvature(5).

**ULCEROGENS**

1. Helicobacter pylori infection

2. Use of NSAIDS

3. Excessive alcohol, coffee & cola consumption
4. Old age
5. COPD
6. Major Burns
7. Multi organ failure
8. Immuno suppression
9. Other drugs like cocaine & Amphetamines

**ABOUT H.PYLORI IN DETAIL:**

H.Pylori bacteria are small, microaerophilic, spiral-shaped gram-negative rods. The presence of H.Pylori in the stomach and duodenum is probably the most common bacterial infection in the world. Areas with a high prevalence of H.Pylori infection have a high incidence of duodenal ulcer.

*H pylori* infection is generally regarded as the most important etiologic factor in the development of duodenal ulcer. Most authors regard *H pylori* as the cause of 85-95% of duodenal ulcers. All evidence supports the assertion that *H pylori* is the major cause of duodenal ulcer. However, the risk of developing a duodenal ulcer in an individual infected with *H pylori* is only about 1% per year, and only 10-15% of individuals with *H pylori* infection develop a duodenal ulcer at any point in life.

*H pylori* organisms produce urease. Urease hydrolyzes urea to ammonia and carbon dioxide. Hydroxide ions produced by equilibration of ammonia with water may damage the gastric and duodenal mucosa. *H pylori* produces proteins that may serve as chemotactic factors for neutrophils and monocytes, which act as proinflammatory cells. *H pylori* also affects the gastric and duodenal mucous layer because these organisms produce proteases that degrade the protective mucous layer. *H pylori* does not lead to the development of gastric and duodenal ulcers through alteration of the bacterial flora.

Several noninvasive laboratory tests are available to aid in the diagnosis of *H pylori* infection.

- Urea breath test
- Enzyme-linked immunoassay (ELISA) can detect both immunoglobulin G (IgG) and immunoglobulin A (IgA) antibodies directed against *H pylori*.
- Fecal antigen test

## CLINICAL FEATURES

### Symptoms:

- Severe pain abdomen
- Shoulder pain (Referred pain)
- Dyspepsia
- Vomiting
- Fever
- Haemetemesis / melena in 5% of Patients(6).

### Signs:

- Diffuse abdominal tenderness
- Muscle gaurding
- Silent abdomen
- Pinched & anxious face
- Sunken eyes
- Hollow cheeks
- Rising Pulse Rate
- Low Volume Pulse
- Shallow respiration
- Obliteration of liver dullness

- Increasing distention of abdomen

**CLINICAL COURSE OF PERFORATION: (7)**

It is divided into 3 stages.

(a) Primary stage: (Stage of peritoneal irritation)

This occurs in the first 2hrs of perforation. Also known as peritonitis. Peritoneal irritation is due to gastric juice in the peritoneal cavity (Chemical Peritonitis)

Patient may or may not vomit. Referred pain to the tip of the shoulder. There will be little change in the pulse, temperature and respiration. Tenderness and muscle guarding are constantly present over the right half of abdomen.

b) Secondary Stage: (Stage of peritoneal reaction)

This occurs at 3-6 hrs. This is a stage of peritoneal reaction. Irritant fluid becomes diluted with the peritoneal exudates. The patient feels comfortable. Symptoms are reduced but signs are still present. Muscular rigidity continues to present. Two other features like obliteration of liver dullness and air under the diaphragm in chest X-ray appears.

C) Tertiary Stage: (Stage of diffuse peritonitis )

This occurs after 6hrs.Typical facies Hippocratic  develops.

Rising pulse rate with low volume & tension, persistent vomiting, board like rigidity, increasing distention of the abdomen.

**MORBIDITY AND MORTALITY PREDICTORS**

- Elderly age group
- Delay in seeking medical attention for more than 24 hours.
- Coexisting medical illness
- Chronic ulcer symptoms
- Amount of peritoneal contamination

## CONDITIONS MIMICKING PERITONITIS

**ABDOMINAL CAUSES:**

- Acute gastritis
- Acute Pancreatitis
- Acute cholecystitis, Biliary colic
- Acute intestinal obstruction
- Mesenteric thrombosis
- Toxic mega colon,  crohns disease

- Torsion (eg: Overian cyst, Fibroid, Omentum, Tumor, Diverticulum )
- Rupture (eg: Ectopic pregnancy, Corpus luteal cyst)
- Colonic diverticulitis & abscess formation
- Ruptured aortic aneurysm
- Endometriosis
- Pelvic inflammatory disease

**EXTRA ABDOMINAL CAUSES:**

- Pleurisy
- Pneumonia
- Pulmonary infarct
- Spontaneous Pneumothroax
- Mediastinitis
- Myocardial infarcts
- Strangulated hiatus hernia
- Acute porphyria
- Diabetic Ketoacidosis
- Multiple Sclerosis
- Neurosyphilis

- Sickle cell disease
- Uremia
- Henoch Schonlein purpura
- Heavy metal poisonings
- Herpes zoster infection
- Typhoid fever

## INVESTIGATIONS

I. Blood investigations.

- CBC-leucocytosis due to infection. Elevated Packed cell volume because of shift of intra vascular fluid.
- Blood culture for aerobic and anaerobic organisms.
- Liver function test.
- Renal function test.

II. Imaging Studies

A) Erect radiograph of the chest & abdomen

First investigation when perforated peptic ulcer diagnosed clinically.

Findings suggestive of perforation include the following:

- Free air trapped in the sub diaphragmatic locations. only 70% of patients with perforation will demonstrates this air under the diaphragm(6).
- Visible falciform ligament. The ligament may appear as an oblique structure from the right upper quadrant towards 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 Hydro pneumoperitoneum or pyopneumoperitoneum on erect film.

**OTHER CAUSES OF PNEUMOPERITONEUM**

- Gas producing bacterial infection
- Iatrogenic (surgery, peritoneal dialysis, drainage catheters)
- Pneumothorax with pleuroperitoneal fistula
- Gynecological (e.g., abortion, iatrogenic perforation of uterus or vagina, culdocentesis, tubal patency test)

**CAUSES OF PSEUDOPNEUMOPERITONEUM**

- Chiliaditi syndrome
- Subdiaphragmatic fat
- Curvilinear pulmonary collapse
- Omental fat
- Subphrenic abscess
- Subpulmonary pneumothorax
- Intramural gas on pneumatosis intestinalis

**(B) Contrast Radiography**

In doubtful cases gastrograffin is used to differentiate sealed from unsealed perforation.

(**C) USG of the Abdomen**

Localised gas collection related to bowel perforation may be detectable particularly if it is associated with other sonographic abnormalities (e.g., Thickened bowel). The site of bowel perforation can the detected by sonography (e.g., gastric Vs duodenal perforation, perforated appendicitis Vs perforated diverticulitis). Rapid evaluation of other organs possible .

**(D) CT scan of the Abdomen**

Provides differential morphological information not obtainable with plain radiography or ultrasonography.

It shows inflammatory changes in the pericolonic soft tissues and focal abscess due to diverticulitis (may mimic perforated colonic carcinoma).

It provides definitive radiographic evidence of perforated meckel's diverticulitis.

**(D) Diagnostic peritoneal tap**

May be useful in determining the presence of intra abdominal blood, fluid and pus

**(F) Peritoneal lavage**

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 >10 IU/L has been shown to be a sensitive and reliable test in detecting occult small bowel injuries.

**(G) Fine- Catheter peritoneal cytology**

This procedure involves insertion of 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 polymorpho nuclear cells.

A value >50% suggests a significant underlying inflammatory process. However it does not provide the clue regarding the exact cause of inflammation.

## MANAGEMENT

Divided into operative and non operative management

### SURGICAL THERAPY

The goal 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 & promote bacterial growth.

**PREOPERATIVE MANAGEMENT**

- Correct any fluid or electrolyte imbalance
- Central venous pressure (CVP) monitoring is essential in critically ill or elderly patients in whom cardiac impairment may be exacerbated by large fluid loss.
- Administer systemic antibiotics (e.g., Ampicillin, gentamycin, metronidazole) making a best estimation regarding the likely organisms.
- Urinary catheterization is done to assess the urinary output & fluid replacement.
- Administration of analgesics such as morphine in small intravenous doses, preferably as a continuous infusion.
- Nasogastric tube is inserted & aspiration done.
- Consent for surgery and anesthesia should be obtained.

## OPERATIVE MANAGEMENT

This can be divided into

1. simple closure with omental patch.
2. Simple closure with definitive procedure for ulcer.

## DEFINITIVE PROCEDURE FOR DUODENAL ULCER PERFORATION

1. T.V. with suitable drainage procedure
2. H.S.V
3. Taylor procedure (Anterior seromyotomy with posterior vagotomy)

## DEFINITIVE PROCEDURE FOR G.U.P:

1. Resection of ulcer & closure
2. Partial gastrectomy with billroth I anastamosis

## INDICATIONS FOR DEFINITIVE ULCER SURGERY(5)

- Thermodynamically stable young patients.
- Perforation for less then 24 hrs.
- Peritoneal contamination must not be extensive.
- No obvious co-morbidity.

- Patients with long history of peptic ulcer.
- Perforation of an ulcer during antisecretary agent.
- Previous history of ulcer complications like obstruction, hemorrhage or perforation.
- Gastric ulcer with coexisting duodenal ulcer disease.

**CONTRAINDICATIONS**

- Associated co morbidity.
- Delayed presentation for > 24 hrs.
- Gross abdominal contamination with food.

**POST OPERATIVE MANAGEMENT**

- Intravenous replacement therapy.
- Continuous nasogastric drainage until drainage becomes minimal.
- Continuous antibiotic administration.
- Analgesics, such as morphine, should be given continuously or in small doses at frequent intervals.
- H. pylori eradication therapy should be instituted.
- $H_2$ receptor antagonists / PPI should be given for 6-8 wks.

## POST OPERATIVE COMPLICATIONS

1. Wound infection

Wound infection rate correlates with the bacterial load in the bowel. So this complication occurs more often with colonic perforation.

The judicious use of prophylactic antibiotics has been demonstrated to reduce the incidence of wound infection in contaminated wounds.

2. Wound failure

Wound failure is partial or total disruption of any or all layer of the operative wound

This may occur early (wound dehiscence) or late (incisional hernia). Factors associated with wound failure are malnutrition, sepsis, uremia, diabetes mellitus, corticosteroid therapy, obesity, heavy coughing, haematoma.

3. Localised abdominal abscess.

4. Multi organ failure & septic shock:

Septicemia is defined as proliferation of bacteria in the blood stream resulting in systemic manifestations such as rigors, fever,

hypothermia (in gram negative septicemia with endotoxemia), leukocytosis or leukopenia ( in profound septicemia), tachycardia and circulatory collapse.

- Septic shock is associated with a combination of the following
- Loss of vasomotor tone.
- Increased capillary permeability.
- Myocardial depression.
- Consumption of WBCs & Platelets.
- Dissemination of powerful vasoactive substances such as histamine, seratonine and prostaglandins resulting in capillary permeability.
- Complement activation and damage of capillary endothelium.
- Gram negative infections are associated with a much worse prognosis that gram positive infections possibly because of associated endotoxemia.

5. Renal failure.

6. Gastrointestinal mucosal haemorrhage.

7. Mechanical intestinal obstruction- Most often caused by postoperative adhesion

8. Postoperative delirium- predisposed by

- Advanced age
- Drug dependency
- Dementia
- Metabolic abnormalities
- Infection
- Previous H/o delirium
- Hypoxia
- Intra operative / Postoperative hypo tension

9. Respiratory complications.

10. Deep vein thrombosis & pulmonary embolism.

**NON OPERATIVE MANAGEMENT** (5)

**Indications:**

- If anesthesia is contra indicated due to multiple medical problem.
- Stable patients with no signs of peritonitis.

- Gastrograffin swallow reveals no free leak into the peritoneal cavity.

**Relative contra indications:**

- Chronic ulcer history
- Steroids
- Gastric ulcer
- Peritonitis
- Diagnostic uncertainty
- Perforation while on adequate medical therapy

Management includes:

- Nasogastric suction should be maintained.
- Intravenous omeprezole or pantaprazole should be given.
- Careful attention to fluid requirement.
- Consider early institution of total parenteral nutrition.
- If fever and leukocytosis persist over several days a CT scan of the abdomen should be obtained and any loculated fluid collection drained percutaneously. If the clinical picture does not

improve, surgery will be necessary to accomplish adequate drainage.

## GASTRO DUODENAL PERFORATIONS OTHER THAN PEPTIC ULCER DISEASE

### 1. CURLING ULCER:

Curling first described this ulcer in patients with burns in 1842. Curling ulcer can occur both in stomach and duodenum, due to over activity of gastric glands and can be prevented by the use of H2 blockers. Perforation of ulcers in burns patients usually has fatal outcome. Perforation in burns patients tends to appear during convalescence and it is a different entity from acute stress ulcers.

### 2. CUSHINGS ULCER:

First described by Harvey Cushing in 1932. Ulcers that arises in the esophagus, stomach, duodenum usually after neuro surgical illness.

### 3. TRAUMATIC PERFORATION OF STOMACH & DUODENUM:

These may be due to blunt or penetrating injuries or as a procedural complication from upper G.I endoscope & ERCP. Penetrating injuries are more common cause of injury to stomach than blunt injury. Immediate surgical intervention is needed either with simple closure or gastric resection & anastomosis. In blunt injury, fixed part of the

intestine like duodenum is affected more than the stomach e.g., seat belt injuries.

In retroperitoneal rupture pain in the epigastrium & back, vomiting, epigastric and flank tenderness are present. Duodenum should be kocharised and rent should be closed.

In intraperitoneal rupture, there may be severe abdominal pain. Simple closure with proximal diversion is ideal.

## SMALL INTESTINAL PERFORATION

### ETIOLOGY

| | | |
|---|---|---|
| 1) | Infection | Salmonella typhi Mycobacterium tuberculosis Ascariasis |
| 2) | Inflammatory | Idiopathic Inflammatory bowel disease Necrotising enterocolitis Ischaemic enteritis Radiation enteritis Appendicular perforation |
| 3) | Traumatic | |
| 4) | Diverticulitis | Meckels diverticulitis Jejunal diverticulitis Ileal diverticulitis |

Zollinger Ellison syndrome

5) Malignancy

6) Drug induced

## SOME IMPORTANT ETIOLOGICAL FACTORS IN DETAIL

### TYPHOID ULCER PERFORATION

Typhoid fever is caused by salmonalla typhi which was described by William Jenner in 1850(8). It colonises several organs such as liver, spleen, bones and small intestine. The terminal ileum, the region of the Peyer's patches is the commonest site for intestinal infection with the formation of longitudinal ulcer on the anti mesenteric border situated within 45cm of the ileocaecal value in the majority of patients.

Clinical feature: Gradual rise in temperature over about 5 days. Rash over the trunk, splenomegaly, diarrhea and rectal bleeding. If untreated septicemia, coma & death may over in the third week. When perforation of the terminal ileum occurs the patient will develop severe lower abdominal pain and will have signs of peritonitis on examination. The case fatality rate in typhoid is 18-24% (9).

In the first week payer's patches becomes hyperemic and hyperplastic. Necrosis occurs in 2$^{nd}$ week, ulceration in the 3$^{rd}$ week followed by healing or perforation in the 4$^{th}$ week. 5% of the typhoid

ulcers will perforate . It is solitary in 85% of cases. Boyd (10) first reported colonic perforation in 1976 on postmortem findings in 6 patients who died of salmonella poisoning. It is postulated that colonic involvement is due to direct bacterial invasion, while ileal lesions are due to enterotoxins produced from parasites, macrophages that caused hyperplasia, necrosis and ulceration (11)(12).

Investigation: Blood culture is positive in the first week, widal test is positive in the 2nd wk, stool culture becomes positive in the 3rd week.

Management : After resuscitating the patient, he should be taken up for surgery. Two layer closure of the perforation after trimming the edges in a single perforation (or) resection & anastomosing the ends can be done in multiple perforations.

**TUBERCULOUS ULCER PERFORATION**

It mostly affects the ileo caecal region (13). The complications of intestinal TB include intestinal obstruction, intestinal perforation, fistula and bleeding (14). Free perforation in the intestinal TB usually occurs in the terminal ileum (13) and it can occur in patients during ATT (15). In 90% of the cases, perforation is solitary, but multiple perforations occur in 10-40% of the patients (16) and are associated with poor prognosis therefore immediate operative intervention is needed (17).

Four macroscopic forms:

- Hyperplastic
- Ulcerative
- Fibrotic
- Ulcero fibrotic

**Diagnosis:**

- Mantoux test- May be negative
- Culture of mycobacterium from gastric washing, faeces, peritoneal fluid & tissue biopsies from enlarged peripheral lymph nodes.
- Plain X-ray of abdomen – May show extensive calcification.
- Barium studies- May shows altered motility & stenotic areas.
- Laparoscopy - Peritoneal biopsy and sampling of the ascitic fluid.

Treatment : Surgical treatment is indicated for intestinal obstruction and perforation. Resection of the perforated segment and end to end anastomosis.

**ASCARIASIS** (18)

The responsible parasite is ascaris lumbricoides. Infestation occurs directly from person to person by oral route. Adult worm develop in the jejunal lumen.

Clinical features: Most of the patients are asymptomatic but obstruction of the gastrointestinal tract occur commonly leading to vomiting, colicky abdominal pain, fever and palpable mass due to worm bolus are other feature. It is diagnosed by ova in the faecal sample.

Complications : Intestinal perforation, intestinal obstruction (common), obstruction of biliary tract, pancreatic duct and the appendix.

Treatment : Mebendazole & albendazole – surgery is indicated in case of obstruction & perforation.

**MECKELS DIVERTICULUM** (18)

Anatomy : It is a remnant of the vitello intestinal duct present in 2% of the population. Arises from the antimesenteric border of the ileum. It has separate blood supply from the omphalo mesenteric artery. Frequently situated 60cm from the ileo caecal valve, 25% of the cases it is situated more proximally in the ileum. 85% of the diverticula are blind ended. The rest have an attachment. Ectopic tissue is found in 70% cases usually gastric mucosa, which is responsible for peptic ulceration secondary to acid and pepsin secretion.

Pathology : Following 3 pathological process occur in meckels diverticulum.

- Inflammation of the diverticulum
- Peptic ulceration of the small bowel
- Intestinal obstruction

Because of this inflammation, gangrene & perforation occurs in the diverticulum. Peptic ulcer occur at the neck or adjacent lleum also responsible for perforation.

Clinical feature : Pain in the central abdominal area. Bleeding from the peptic ulcer site, which may manifest as malena or fresh rectal bleeding, usually in children.

Treatment : Excision of the diverticulum together with a wedge resection of adjacent ileum in case of inflammation & perforation. Although most episodes of perforated diverticulum are confined to the peridiverticular region or pelvis, patients occasionally presents with the signs of generalized peritonitis. Overall mortality is relatively high (20-40%). Largely Because of complications such as septic shock & multi organ failure. .

**GASTRINOMA (ZOLLINGER-ELLISON SYNDROME [ZES])**

First described in 1955, ZES is caused by a tumor of pancreatic islet cells that produces gastrin. It is associated with gastric acid hypersecretion and development of PUD. From 0.1-1% of duodenal ulcers are thought to be secondary to an underlying gastrin-secreting tumor.

**CROHN'S DISEASE**

Free perforation into the peritoneal cavity is rare. Commonest site is ileum. Resection of the perforated segment and anastamosis along with peritoneal lavage is the treatment of choice.

**TUMOURS OF SMALL INTESTINE**

Perforation due to malignancy is rare. It mainly due to western types of lymphoma. The resection of a bowel segment with wide margin should be done, simple closure will result in reperforation.

**TRAUMATIC INJURIES OF SMALL INTESTINE** (18)

Blunt injury cause 15-20% of small bowel injury. Penetrating trauma cause 25-30% of the small bowel injury. Presents with features of peritonitis.

The postulated mechanism are:

- Crushing injury of the bowel between the spine and the blunt object, such as steering wheel or handle bars.
- Deceleration shearing of the small bowel at fixed points such as the ileocaecal valve and around the superior mesenteric artery.
- Closed loop rupture caused by increased intra abdominal pressure.
- At surgery vascular control is the prim importance. Single hole can be closed without debridement.
- If two adjacent holes are found they can be connected across the bridge of bowel and a transverse closure effected so as not to narrow the lumen.
- Large lacerations are debrided & closed.
- Any large segments of bowl that are devascularised or have multiple defects should be resected & re anastomosed.

Complications:

- Intra abdominal abscess
- Anastomotic leakage

- Enterocutaneous fistula
- Intestinal obstruction
- Reperforation
- septicemia

**APPENDICULAR PERFORATION**

Risk factors are:

- Faecolith obstruction (50%)
- Immumosuppresion
- Diabetes mellitus
- Pelvic appendix

Perforation of appendix are common in the extremes of age group (below 5 and above 60). Overall about 20% of all the acute appendicitis patients have perforation at the time of surgery (18).

Appendicectomy with peritoneal lavage is the treatment of choice.

Complication:

- Wound infection
- Intra abdominal abscess

- Ileus
- Venous thrombosis & embolism
- Portal pyaemia
- Right inguinal hernia
- Adhesive intestinal obstruction
- Faecal fistula

## LARGE INTESTINAL PERFORATION

**ETIOLOGY :**

1) Infective - Paratyphoid

   Mycobacterium tuberculosis

   Actinomycosis

   Entamoeba histolytica

2) Inflammatory- Toxic megacolon

3) Diverticular disease

4) Volvulus

5) Trauma

6) Malignancy

**AMOEBIC BOWEL PERFORATION**

Amoebic dysentery is caused by entamoeba histolytica. It is a common cause of diarrhea in warm and humid parts of the world. Amoebic dysentery is complicated by amoebic ulcers, colonic perforation, stricture formation or severe hemorrhages. The most common site of perforation are the caecum and rectosigmoid.

- From localized disease - Segmental colectomy
- For generalized disease -Total colectomy with ileostomy and mucous fistula(Paul-Mickulicz procedure ).

**TOXIC MEGACOLON:**

This is a serious complication of ulcerative colitis which occurs in 2-10% of cases. More common in the transverse colon. It is characterized by

- Abdominal distention
- Absent bowel sounds
- Severe systemic toxicity
- Fever
- Tachycardia
- Leukocytosis

- Marked fluid and electrolyte depletion

Risk of perforation is 50-60% (4).It carries 50% mortality (18) and accounts for 30% of all deaths from ulcerative colitis (18). Spontaneous colonic perforation also occur in ulcerative colitis in the absence of toxic megacolon and in the presence of systemic steroid therapy, a high index of suspicion is required.

Management : Emergency subtotal colectomy with formation of end ileostomy.

**DIVERTICULAR DISEASE PERFORATION**

Diverticular disease of the colon remains asymptomatic in 90% of the patients. 1% of the patients may suffer from localized or generalized peritonitis secondary to perforation(19) which has mortality of about 15%. If gross faecal peritonitis comes >50% mortality rate.

USG and CT scan will confirm the disease formation and rarely a water soluble enema may be required to confirm the leak (20).

Management:

- Primary resection and Hartmann's procedure.

- Primary resection and anastomosis after on table lavage in selected cases.
- Suture of the perforation with drainage with or without proximal diversion.

**PERFORATION IN MALIGNANCIES**

In carcinoma of the large intestine perforation can be due to annular growth causing obstruction and tension gangrene and perforation of caecum due to malignant ulcer. There is a closed loop phenomenon between distal obstruction and proximal ileocaecal valve. The intraluminal pressure raises and the caecum becomes gangrenous and perforate.

Treatment includes primary wide resection with appropriate exteriorization of both the ends.

**TRAUMATIC PERFORATION**(18)

The greatest number of injuries to the colon and rectum are the result of penetrating or perforating trauma. The amount of force required to damage the colon is considerable and, thus, the colon is relatively refractory to blunt injury. Blunt trauma accounts for only 5% of the colonic injuries.

Intra peritoneal injury of the colon and rectum shows clinical signs of diffuse peritonitis which requires peritoneal lavage. The central debate in the operative management of colonic injury is between primary repair of low risk colonic injuries verses repair and proximal colostomy or resection and colostomy.

## PATIENTS AND METHODS

An analysis of 125 patients of perforation peritonitis was done over a period of 27 months (from May2005 to July 2007) at Kilpauk Medical College,CHENNAI

All cases were studied in term of clinical presentation, radiological investigations done, operative findings and postoperative course. Data was colleted from indoor patient records, operation theatre records and outpatient department follow up of cases.

All patients following a clinical diagnosis of perforation peritonitis and adequate resuscitation, underwent exploratory laparotomy in emergency setting. At surgery the source of contamination was sought for and controlled. The peritoneal cavity was irrigated with 5–6 litres of warm normal saline and the decision to insert a drain was left to the discretion of the operating surgeon. Abdomen was closed with continuous, number one non-absorbable suture material. Although all patients received appropriate perioperative broad spectrum antibiotics, the drug regimen was not uniform.

## INCLUSION CRITERIA

All cases found to have peritonitis as a result of perforation of any part of gastrointestinal tract at the time of surgery were included in the study.

## EXCLUSION CRITERIA

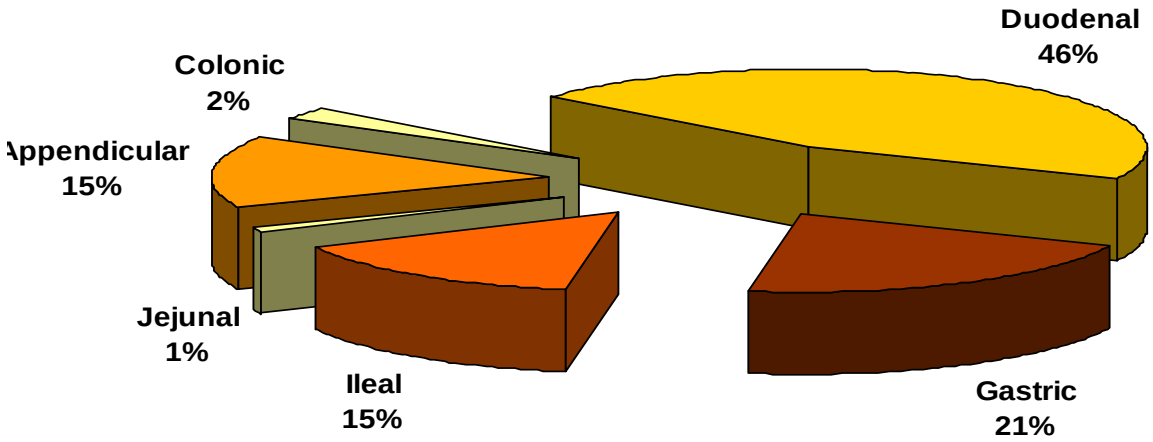
- cases of primary peritonitis
- esophageal rupture or perforation
- perforation of hepatobiliary system
- iatrogenic perforations
- traumatic perforations
- peritonitis due to anastomotic leak

## OBSERVATION

125 cases of perforation were studied. The major cause of perforation was due to acid peptic disease. The most commonest site of perforation is duodenum.

## INCIDENCE

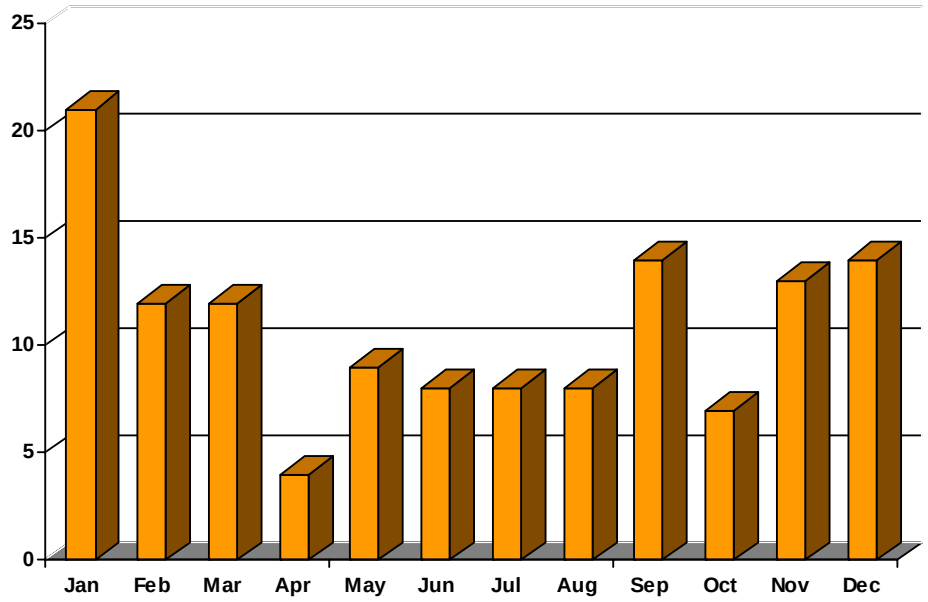
| S.NO | SITE | NO.OF CASES | PERCENTAGE |
|---|---|---|---|
| 1. | Duodenal perf. | 57 | 45.6 |
| 2. | Gastric perf. | 26 | 20.8 |
| 3. | Ileal perf. | 19 | 15.2 |
| 4. | Jejunal perf. | 1 | 0.8 |
| 5. | Appendicular.perf | 19 | 15.2 |
| 6. | Colonic perf. | 3 | 2.4 |
| | TOTAL | 125 | 100 |



## SEASONAL TRENDS

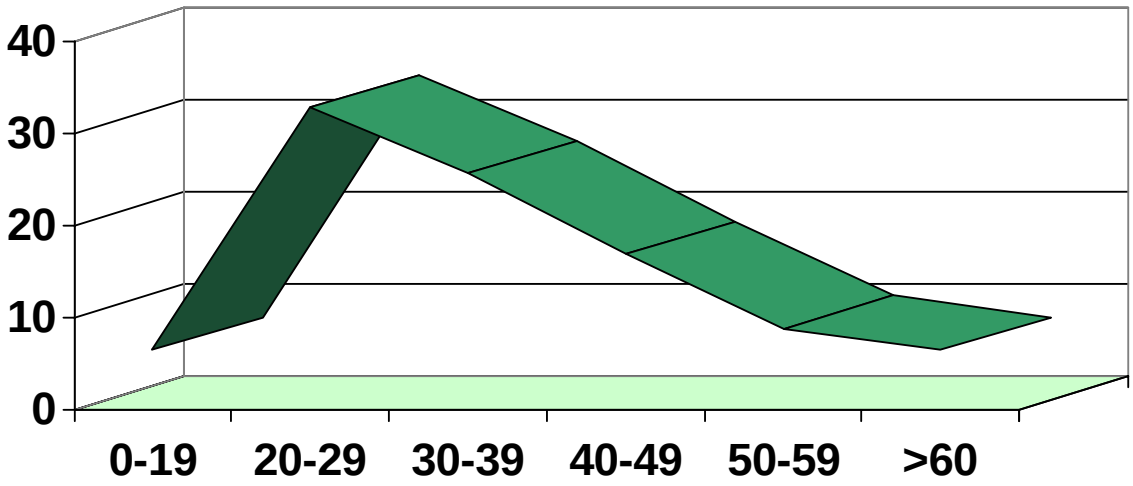
The incidence of cases were more common in winter.

| S.NO. | MONTH | TOTAL CASES |
|---|---|---|
| 1. | JAN | 21 |
| 2. | FEB | 12 |
| 3. | MAR | 12 |
| 4. | APR | 4 |
| 5. | MAY | 9 |
| 6. | JUN | 8 |
| 7. | JUL | 8 |
| 8. | AUG | 8 |
| 9. | SEP | 14 |
| 10. | OCT | 7 |
| 11. | NOV | 13 |
| 12. | DEC | 14 |




**AGE DISTRIBUTION IN GIT PERFORATION**

| S.NO. | AGE | TOTAL CASES | PERCENTAGE |
|---|---|---|---|
| 1. | 0-19 | 8 | 6.4 |
| 2. | 20-29 | 41 | 32.8 |
| 3. | 30-39 | 32 | 25.6 |
| 4. | 40-49 | 21 | 16.8 |
| 5. | 50-59 | 11 | 8.8 |
| 6. | >60 | 8 | 6.4 |




Overall GIT perforation were found to be common in young adults in the age group of 20 to 29 and 30 to 39.

**COMPARITIVE STUDY**

Jhobta et al study-2006(chandigarh) which was published in World J Emerg Surg.2006;1:26 shows the same result which correlates with our study. The mean age of perforation in his study was 36.8

In the same study the percentage of cases in the age group of >50 was 16%.

In our study it was 15.2%

## RADIOLOGICAL SIGN

90 cases showed air under the diaphragm.

## COMPARITIVE STUDY

| | |
|---|---|
| Cusheri –essential surgical practice | 70% |
| Study-2007 | 72% |

## DUODENAL ULCER PERFORATION

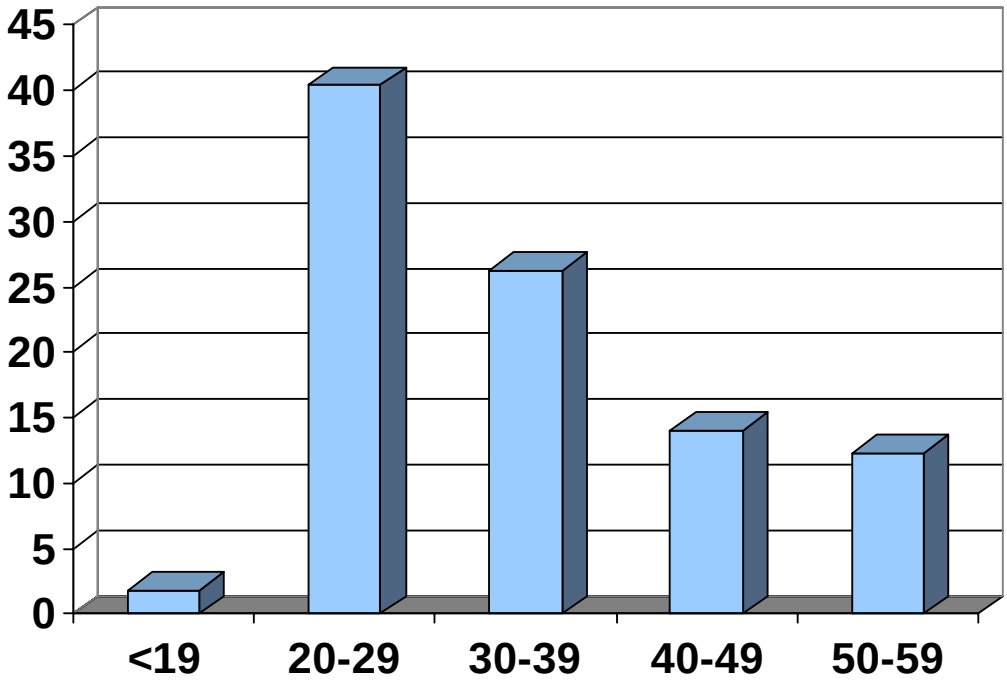
Total number of duodenal ulcer perforation were 57.

## AGE DISTRIBUTION

| S.NO. | AGE | TOTAL CASES | PERCENTGE |
|---|---|---|---|
| 1. | <19 | 1 | 1.8 |
| 2 | 20-29 | 23 | 40.4 |

| | | | |
|---|---|---|---|
| 3 | 30-39 | 15 | 26.3 |
| 4 | 40-49 | 8 | 14.0 |
| 5 | 50-59 | 7 | 12.3 |
| 6 | >60 | 3 | 5.2 |
| | TOTAL | 57 | 100 |

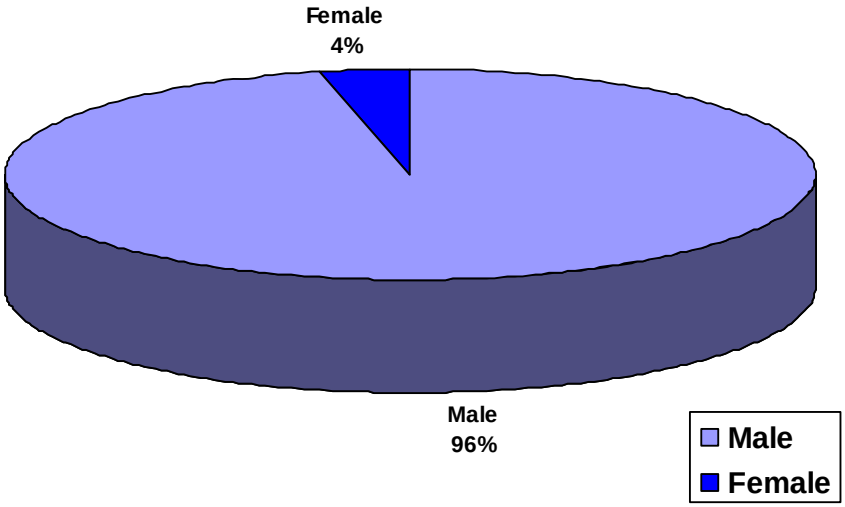
The most common age group affected by duodenal perforation was 20-29 (i.e.)young adults. This is similar to the age incidence of the overall GIT perforation.




SEX DISTRIBUTION IN DUP

Total of two female patients were encountered in our study out of 57 patients of DUP.

Male : female ratio was 28:1




## COMPARITIVE STUDY

| | |
|---|---|
| Jhobta et al –study 2006 | Male patients 83% |
| Study 2007 | 96% |

ASSOCIATION WITH RISK FACTORS

Out of 57 patients, 17 were alcoholic , 13 were smokers and 12 were NSAID users.

## COMPARITIVE STUDY

| STUDY | Association with any one of the risk factors |
|---|---|
| Dr.OP Murty et al (Malaysia) 2005 | 36.07% |
| Study 2007 | 38.6% |

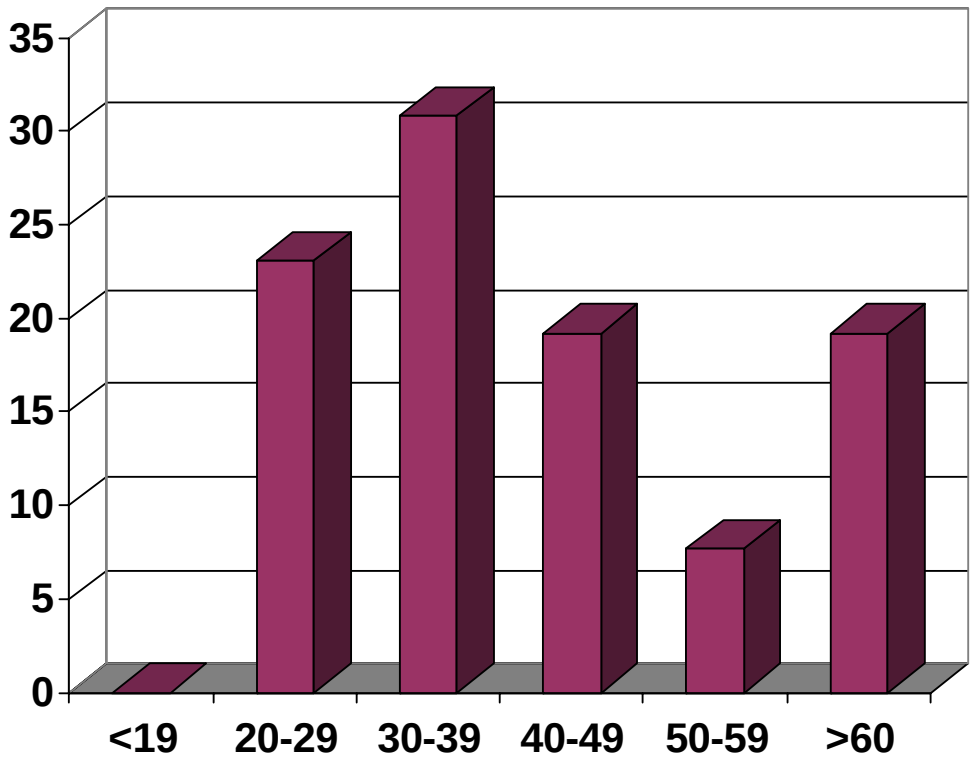
Among the risk factors alcohol consumption was found to predominate in our patients. This may be because of the lower socio economic class, the life style and the education status.

## GASTRIC PERFORATION

19 cases were gastric perforation out of 125 cases.

## AGE DISTRIBUTION

| AGE | TOTAL CASES | PERCENTAGE |
|---|---|---|
| <19 | O | 0 |
| 20-29 | 6 | 23.1 |
| 30-39 | 8 | 30.8 |
| 40-49 | 5 | 19.2 |
| 50-59 | 2 | 7.7 |
| >60 | 5 | 19.2 |



In our study perforations were common in the age group of 30-39.

Out of 26 cases only one female patient was encountered.

Three perforations were due to gastric malignancy.

7 patients were alcoholic and 10 patients were smokers.

No cases had previous history of perforation or had a bout of alcohol prior to perforation.

Air under the diaphragm was present in 25 patients x-ray out of 26(i.e.) 96.2%

Simple closure with omental patch was done in 24 cases. One malignant ulcer perforation was treated with partial gastrectomy and feeding jejunostomy. That patient died on the $10^{th}$ post operative day. Another malignant perforation was treated with gastrostomy and ileostomy. There were 3 deaths. Out of which 2 were malignant perforation.

The result of biopsy came as adeno carcinoma in two cases and malt lymphoma in one case.

So the major cause of gastric perforation is gastric ulcer and the next common cause is malignancy.

## ILEAL PERFORATION

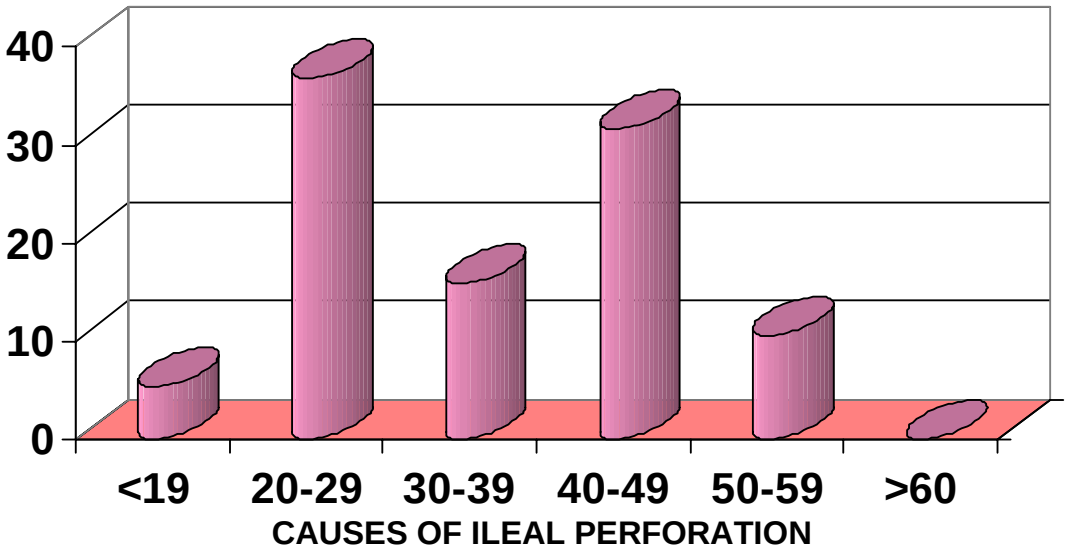
Out of 125 cases 19 were found to have ileal perforation.

AGE DISTRIBUTION

| AGE | TOTAL CASES | PERCENTAGE |
|---|---|---|
| <19 | 1 | 5.3 |
| 20-29 | 7 | 36.8 |
| 30-39 | 3 | 15.8 |
| 40-49 | 6 | 31.6 |
| 50-59 | 2 | 10.5 |
| >60 | 0 | 0 |

The highest age incidence was between 20 – 29.

No female patients with perforation of ileum was encountered.




Out of 19 cases 9 cases were due to enteric fever, 5 cases were due to tuberculosis and 5 cases were due to unknown reason, may be because of nonspecific inflammation.

**COMPARITIVE STUDY**

| CAUSES | Jhobta et al-2006study | Study 2007 |
|---|---|---|

| | | |
|---|---|---|
| Typhoid fever | 45% | 47.4% |
| TB | 22% | 26.3% |

The most common cause of ileal perforation in this study was enteric fever followed by tuberculosis which correlates well with the study of Jhobta et al 2006.

In the total of 19 cases of ileal perforation 12 cases had history of fever for more than two weeks and 9 of them proved to be widal positive.

5 perforated patients were found to be affected by tuberculosis by both intra operative findings like thickened ileal segment around the area of perforation and enlarged and matted lymphnodes and by histo pathological examination which showed the caseating granulomatous inflammation in the resected segment or biopsy.

Out of 19 cases 5 cases found to have multiple ileal perforation.

## COMPARITIVE STUDY

| TYPE OF PERF. | Jhobta et al 2006 | Study 2007 |
|---|---|---|
| SINGLE | 71.7% | 73.7% |
| MULTIPLE | 28.3% | 26.3% |

All patients were taken up for surgery with in 12 hours and resection of the perforated ileal segment with end to end anastomosis was done in 5 cases which were multiple perforations and simple closure by two layers after trimming the edges was done in other cases.

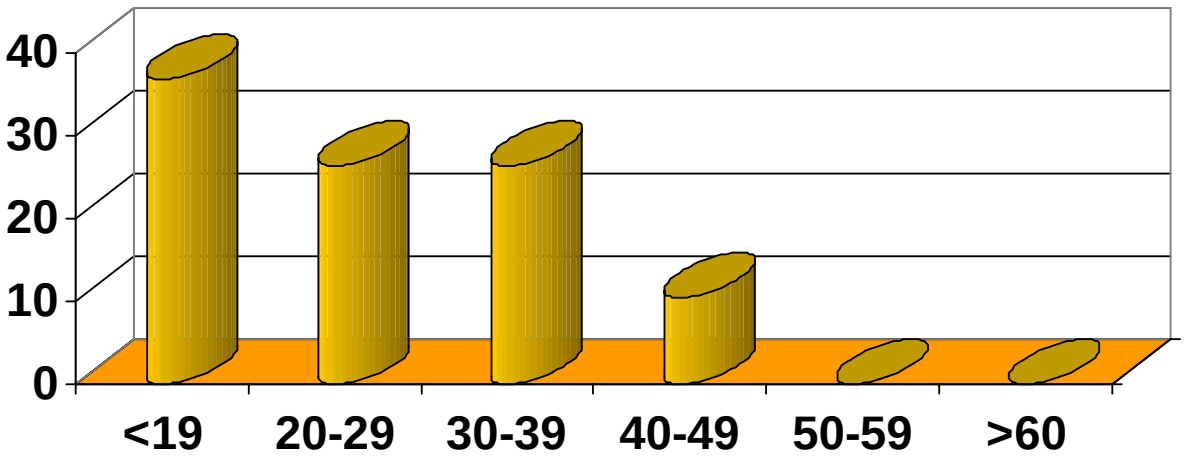
## APPENDICULAR PERFORATION

Total of 19 cases out of 125.

AGE DISTRIBUTION

| AGE | TOTAL CASES | PERCENTAGE |
|---|---|---|
| <19 | 7 | 36.8 |

| | | |
|---|---|---|
| 20-29 | 5 | 26.3 |
| 30-39 | 5 | 26.3 |
| 40-49 | 2 | 10.5 |
| 50-59 | 0 | 0 |
| >60 | 0 | 0 |




More common below the age group of 19.

Male :female ratio was 11:8

The clinical presentation of these patients were mostly fever ,vomiting, abdominal pain and localized guarding with rebound tenderness.

## COMPARITIVE STUDY

| CLINICAL FEATURE | Jhobta et al-2006 study | Study 2007 |
|---|---|---|
| vomiting | 66% | 68.4% |
| fever | 43% | 42% |
| Localized gaurding | 77% | 78.9% |

## MORBIDITY DATA

The postoperative complications (i.e.) morbidity in our study were wound infection, burst abdomen, intra abdominal collections, anastomotic leak and respiratory complications.

In our study post operative complications occurred in 25 patients. Out of which 16 patients had wound infection, 4 patients had burst abdomen, 1 patient had pelvic abscess, 1 patient had anastomotic leak and three patients had respiratory complication.

## COMPARITIVE STUDY

| Post op complication | Jhobta et al-2006 study | Study 2007 |
|---|---|---|
| Wound infection | 25% | 64% |
| Burst abdomen | 9% | 16% |
| Abdom.collection | 9% | 4% |
| Anastomotic leak | 7% | 4% |
| Resp. complication | 28% | 12% |

The unusually high occurrence of wound infection in our patients may be due to poor post operative care, inadequate theatre sterilization ,late presentation of our patients and their poor general hygiene. This shows the necessity of improving the post operative wound care in our hospital and necessity of creating an awareness among our people regarding seeking medical attention early.

## MORTALITY

Out of 125 cases 11 patients were died in the post operative period.

The commonest causes include septicemia, cardiac arrest and respiratory complication.

Number of deaths in DUP were 3, GUP were 3, ileal perforation were 4 and colonic perforation were 1.

## COMPARITIVE STUDIES

Death in gastric perforation.

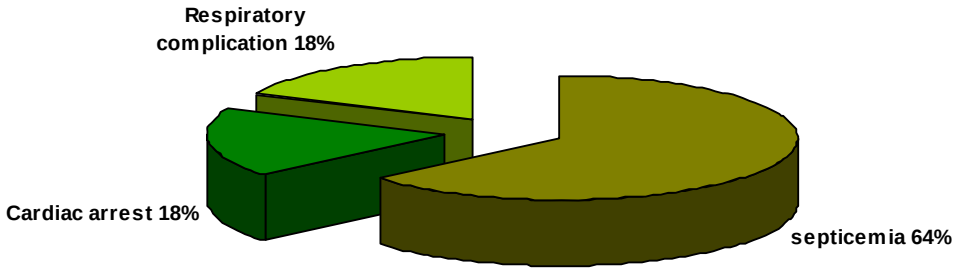
| | |
|---|---|
| Schwartz's manual of surgery | 10 -40% |
| Study 2007 | 11.5% |

Death in Duodenal perforation.

| | |
|---|---|
| Morran&Carter 1983 | 5-10% |
| Study 2007 | 3.5% |

| | **Jhobta et al-2006 study** | **Study 2007** |
|---|---|---|
| Overall mortality | 10% | 8.8% |

| Cause of death | Total patients | Percentage |
|---|---|---|
| septicemia | 7 | 63.3% |
| Cardiac arrest | 2 | 18.2% |
| Resp. complication | 2 | 18.2% |




COMPARITIVE STUDY

The major cause of death was septicemia (63.3%) which correlates with the study of Jhobta et al -2006(59%).

| **STUDY** | **% of death caused by septicemia** |
|---|---|
| Jhobta et al 2006 | 59% |
| Study 2007 | 63.3% |

## MORTALITY CHART

| S.No. | Age/Sex | Pathology | Procedure | Cause of death |
|---|---|---|---|---|
| 1 | 60/M | D.U.P. | PC With OP | septicemia |
| 2 | 52/M | D.U.P | PC with OP | Septicemia |
| 3 | 58/M | D.U.P. | PC with OP | Septicemia |
| 4 | 67/M | Malignant G.P. | Partial gastrectomy with feeding jejunostomy | Resp.complication |
| 5 | 70/M | G.U.P. | PC with OP and biopsy taken | Cardiac arrest |
| 6 | 33/M | G.U.P. | PC with OP | Septicemia |
| 7 | 20/M | Multiple I.P. | Resection anastomosis | Septicemia |

| | | | | |
|---|---|---|---|---|
| 8 | 28/M | I.P. | S.C. in 2 layers | Septicemia |
| 9 | 55/M | I.P. | S.C. in 2 layers | Resp.complication |
| 10 | 40/M | I.P. | S.C. in 2 layers | Septicaemia |
| 11 | 52/M | Sigmoid perf. | Sigmoid loop colostomy | Cardiac arrest |

## DISCUSSION

From the result, I conclude that gastro-intestinal perforation occurs more frequently among men than women. This is believed to be due to the lifestyles and also the risk factors that could contribute to the formation of ulcers and later the perforation of GIT. These factors includes cigarette smoking, consumption of food and beverages containing caffeine, alcohol abuse and physical stress. Men are more prone to these effects as for example they smoke rather than women. GI perforation most commonly affects the young men in the prime of life as compared to the studies in the west(21). Where the mean age is

between 45-60 years. In majority of the cases the presentation to the hospital is late with well established generalized peritonitis with purulent/faecal contamination and varying degree of septicemia. The signs and symptoms are typical and it is possible to make a clinical diagnosis of peritonitis in all patients.

The etiological factors show a wide geographical variations. Khana et al(22) from Varanasi studied 204 consecutive cases of gastro intestinal perforations and found that, over half (108 cases) where due to typhoid. They also had perforations due to duodenal ulcer(58), appendicitis (9), Amoebiasis(8) and Tuberclosis(4). These figures show the importance of infection and infestation in the third world, which is also reflected in the typhoid and tubercular perforation in our study and the study by Jhobta et al (23).

At the other end of the spectrum, Noon et al (24) from Texas studied 430 patients of GIT perforation and found 210 cases to be due to penetrating trauma, 92 due to appendicitis and 68 due to acid peptic disease. This shows the importance of trauma in developed countries.

There were 11 deaths (8.8%) in the immediate post operative days in our study, which is comparable with the other published series (23,25,26,27) despite delay in seeking medical attention . This was

probably because of lower mean age( which is a factor determining mortality) of patients in our study. The main cause of death in the present series of patients was septicemia (63.3%), which was comparable with other similar study (23). Therefore contamination is a crucial consideration in patients with peritonitis and problem of mortality is a problem of infection. So by early surgical intervention we succeed in preventing further contamination by removing the source of infection though the end result will also depends upon the general host resistance and the antibiotic sensitivity of the organisms (28).

The major cause of post operative morbidity were wound infection (64%), burst abdomen (16%). Unacceptably high incidence of burst abdomen in the present series was multi factorial due to delayed presentation, gross contamination of the peritoneal cavity, septicemia and above all the faulty methods of abdominal closure as majority of our patients were operated by inexperienced resident doctors, who are a fleeting population and are still in the learning curve.

To conclude, the spectrum of perforation in India continues to be different from its western counterparts with DUP, GUP, typhoid perforation and TB perforation being the major causes of generalized peritonitis.

# CONCLUSION

- duodenal ulcer perforation was the commonest cause of GIT perforation with male preponderance.
- More common in third decade.
- More common in lower socioeconomic class.
- More common in winter season.
- Smoking and alcohol were aggravating factors.
- Most of the patients presents with pain abdomen, fever and vomiting.
- Simple closure with omental patch was very effective in the management.
- Next to duodenum gastric perforation was more common.
- Gastric perforation was more common in fourth decade.
- Ileal perforation was more common in third decade.
- Commonest causes being typhoid and tuberculosis.
- Single ileal perforation was more common than multiple perforation.
- Closure in two layers was very much effective in small bowel perforation.

- Prognostic determinant in perforation were delay in presentation to the hospital and degree of contamination.
- Conservative management increases the number of hospital stay.
- Incidence of colonic perforation was 2.4% (3 cases) in this study.
- Most common post operative complication was wound infection.
- Deaths were due to septicemia, cardiac arrest and respiratory complication.
- In spite of the recent advances in closing the D.U. perforation by laparoscopy and by other means, still simple closure with omental patch is widely practiced.

# GASTRIC PERFORATION

| | ‹ | Ip No | D.O.A. | P | Vom | Dist | Alc | Nsaids | F | Reg | Bs | Gud | Pre.O.D. | Per.O.D. | D.I.H. | Proc |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 24098 | 12.09.05 | + | - | + | + | + | - | + | - | + | Perf.Peritonitis | Gast. Perf. | 12 | Sc With ( |
| | | 29968 | 08.11.05 | + | - | + | + | + | - | + | - | + | Perf.Peritonitis | Gast. Perf. | 10 | Sc With ( |
| | | 30234 | 14.11.05 | + | - | + | + | + | - | + | - | + | Perf.Peritonitis | Gast. Perf. | 8 | Sc With ( |
| | | 800 | 08.01.06 | + | - | - | + | + | - | + | - | + | Perf.Peritonitis | Gast. Perf. | 24 | Sc With ( |
| | | 2605 | 25.01.06 | + | - | - | - | - | - | + | + | + | Perf.Peritonitis | Gast. Perf. | 10 | Sc With ( |
| | | 2680 | 28.01.06 | + | - | - | - | - | - | - | + | + | Perf.Peritonitis | Gast. Perf. | 8 | Sc With ( |
| | | 19834 | 02.07.06 | + | - | - | - | + | - | + | + | + | Perf.Peritonitis | Gast. Perf. | 6 | Sc With ( |
| | | 27972 | 11.09.06 | + | + | - | + | - | - | + | + | + | Perf.Peritonitis | Gast. Perf. | 10 | Sc With ( |
| | | 34699 | 05.11.06 | + | - | + | + | + | - | - | - | + | Perf.Peritonitis | Gast. Perf. | 12 | Sc With ( |
| | | 39916 | 17.12.06 | + | - | + | - | + | - | - | - | + | Perf.Peritonitis | Gast. Perf. | 12 | Sc With ( |
| | | 40379 | 21.12.06 | + | + | + | + | - | - | + | + | + | Perf.Peritonitis | Gast. Perf. | 24 | Sc With ( |
| | | 40 | 01.01.07 | + | + | + | - | - | + | + | + | - | Subacute Intestinal Perf. | Gast. Perf. | 72 | Sc With ( |
| | | 1024 | 13.01.07 | + | - | + | + | - | - | + | + | + | ?H.V.P. | Gast. Perf. | 6 | Sc With O.P.W |
| | | 1561 | 22.01.07 | + | - | - | - | + | - | - | - | + | Perf.Peritonitis | Gast. Perf. | 8 | Sc With ( |
| | | 1816 | 25.01.07 | + | - | - | + | + | - | + | - | + | Perf.Peritonitis | Sealed Gastric Perfora | 24 | Appedicectomy |
| | | 2023 | 28.01.07 | + | - | - | - | + | - | + | + | + | Perforation Peritonitis With R.I.H. | Gast. Perf. | 8 | P.C. With De Closur |
| | | 2750 | 05.02.07 | + | - | - | - | + | - | + | - | + | Perf.Peritonitis | Malignant Gastric Perforation | 8 | Partial Gastrec Feeding Jejur |
| | | 2803 | 08.02.07 | + | - | + | + | - | + | - | - | + | Perf.Peritonitis | Gast. Perf. | 8 | Sc With ( |
| | | 3091 | 12.02.07 | + | - | + | - | - | + | + | - | + | Perf.Peritonitis | Gast. Perf. | 12 | Sc With ( |
| | | 3755 | 24.02.07 | + | - | + | - | - | - | + | + | + | Perf.Peritonitis | Gast. Perf. | 10 | Sc With ( |
| | | 4318 | 29.02.07 | + | - | + | - | + | - | + | + | + | Perf.Peritonitis | Gast. Perf. | 10 | Sc With ( |
| | | 4926 | 08.03.07 | + | + | - | + | - | - | + | + | + | Perf.Peritonitis | Malignant Gastric Perforation | 14 | Gastrostomy Wi Jejunostc |
| | | 5429 | 15.03.07 | + | - | + | - | - | - | + | - | + | Perf.Peritonitis | Gast. Perf. | 8 | Sc With ( |
| | | 9211 | 05.05.07 | + | - | - | + | - | - | + | Slug | + | Perf.Peritonitis | Malignant Gastric Perforation | 24 | Sc With O.P.&Bi |
| | | 10301 | 17.05.07 | + | - | + | - | + | - | - | - | + | Perf.Peritonitis | Gast. Perf. | 12 | Sc With ( |
| | | 13280 | 22.06.07 | + | - | + | - | + | - | + | - | + | Perf.Peritonitis | Gast. Perf. | 8 | Sc With ( |

# Jejunal Perforation

| | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Murugan | 40 | M | 11543 | 07.05.05 | + | - | + | - | + | + | + | - | + | H.\ |

# Ileal Perforation

| S.No. | Name | Age | Sex | IP.NO. | D.O.A. | Pain | Vom | Dist | Alco | Nsaid | Fever | Regid | Bs | Gud | Pre Op |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Arumugam | 30 | M | 15207 | 14.06.05 | + | - | + | - | - | - | + | + | + | Perf. Pe |
| 2 | Ramesh | 19 | M | 17521 | 04.06.05 | + | - | + | + | - | + | + | - | + | Perf. Pe |
| 3 | Saravanan | 20 | M | 19354 | 25.07.05 | + | - | + | + | - | - | + | - | + | Perf. Pe |

| | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 4 | Ganesan | 28 | M | 23527 | 04.09.05 | + | - | + | - | - | + | + | - | + | Perf. Pe | |
| 5 | Vijay | 30 | M | 23980 | 09.09.05 | + | - | + | - | - | + | + | + | + | Perf. Pe | |
| 6 | Sudhaker | 22 | M | 25236 | 21.09.05 | + | - | - | - | - | + | + | + | + | Perf. Pe | |
| 7 | Kesavan | 23 | M | 33422 | 14.12.05 | + | + | - | + | - | + | + | - | - | Perf. Pe | |
| 8 | Durai | 22 | M | 33751 | 17.12.05 | - | + | + | + | - | - | - | - | + | Perf. Pe | |
| 9 | Mani | 50 | M | 2478 | 24.01.06 | + | - | + | + | + | - | - | + | + | Perf. Pe | |
| 10 | Issac | 55 | M | 35789 | 13.11.06 | + | - | + | - | + | + | + | - | + | Perf. Pe | |
| 11 | Balasundaram | 42 | M | 38858 | 08.12.06 | + | - | + | - | - | + | + | - | + | Perf. Pe | |
| 12 | Kasi | 40 | M | 41120 | 27.12.06 | + | - | + | - | - | + | - | - | + | Perf. Pe | |
| 13 | Kuppan | 40 | M | 40923 | 26.12.06 | + | - | - | - | - | - | + | - | + | Perf. Pe | |
| 14 | Shanker | 40 | M | 41412 | 31.12.06 | + | + | - | - | + | - | + | - | + | Perf. Pe | |
| 15 | Pandiyarajan | 27 | M | 557 | 05.01.07 | + | + | - | + | + | - | + | + | - | Perf. Pe | |
| 16 | Murugesan | 34 | M | 901 | 11.01.07 | + | + | + | + | + | + | + | + | - | Perf. Pe | |
| 17 | Vinoth | 20 | M | 1216 | 17.01.07 | + | - | + | - | - | + | + | Slug | - | Appe<br>Pe | |
| 18 | Mani | 40 | M | 9870 | 12.05.07 | + | - | + | + | - | - | + | - | + | Perf. Pe | |
| 19 | Ganesan | 44 | M | 10611 | 22.05.07 | + | - | + | + | + | - | - | - | + | Perf. Pe | |

# APPENDICULAR PERFORATION

| S.No. | Name | Age | Sex | IP.NO | D.O.A. | P | Vom | Dis | Alc | NSAID | Fe | Rig | BS | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Neela | 17 | F | 12161 | 13.05.05 | + | + | + | - | - | + | + | + | |
| 2 | Mani | 37 | M | 15401 | 16.06.05 | + | - | + | - | - | - | + | + | |
| 3 | Hem Kumar | 15 | M | 190040 | 22.07.05 | + | + | + | - | - | + | + | + | |
| 4 | Kalaam | 20 | M | 19429 | 26.07.05 | + | + | + | - | - | - | + | + | |
| 5 | Elumalai | 20 | M | 19475 | 26.07.05 | + | + | + | - | - | + | - | + | |
| 6 | Palani | 25 | M | 19823 | 29.07.05 | + | + | + | - | - | + | + | + | |
| 7 | Gomathi | 19 | F | 21987 | 14.08.05 | + | - | + | - | + | + | + | + | |
| 8 | Priya | 14 | F | 22734 | 27.08.05 | + | + | + | - | - | - | + | + | |
| 9 | Lakshmi | 38 | F | 24237 | 15.09.05 | + | + | + | - | - | + | - | + | |
| 10 | Meena | 18 | F | 25449 | 23.09.05 | + | + | + | - | - | - | + | + | |
| 11 | Panchali | 40 | F | 25593 | 24.09.05 | + | + | + | - | - | - | + | + | |
| 12 | Amudha | 17 | F | 28298 | 21.10.05 | + | - | + | - | - | + | - | + | |
| 13 | Shanmuganathan | 28 | M | 28668 | 24.10.05 | + | - | + | - | - | - | + | + | |
| 14 | Govindaraj | 34 | M | 36321 | 17.11.06 | + | + | + | - | - | + | + | + | |
| 15 | Kuppan | 44 | M | 40717 | 27.12.06 | + | - | + | - | - | - | + | + | |
| 16 | Kumar | 30 | M | 2458 | 01.02.07 | + | + | + | - | - | - | + | - | |
| 17 | Ramesh | 18 | M | 4005 | 25.02.07 | + | + | + | - | - | - | - | + | |
| 18 | Sathiyavani | 23 | F | 5316 | 13.03.07 | + | - | + | - | - | - | + | + | |
| 19 | Karunagaran | 35 | M | 7460 | 11.04.07 | + | + | + | - | - | - | + | + | |

# COLONIC PERFORATION

| S.No. | Name | Age | Sex | IP.NO | D.O.A. | P | Vom | Dis | Alc | NSAID | Fe | Rig | BS | GUD | Pre.O.D. | Per.O.D. | D.I.H. | procedure | P.O.C. | N.O.H.S. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Damu | 45 | M | 16582 | 28.06.05 | + | - | + | - | + | + | + | - | + | Perf.peritonitis | Mult.caecal perf. | 12 | Rt.hemicolectomy with ileostomy | Basal pneumonia | 12 |
| 2 | Venkatesan | 25 | M | 271176 | 10.10.05 | + | - | + | - | - | + | + | - | + | Perf.peritonitis | Sigmoid perf. | 24 | Sigmoid loop colostomy | Nil | 14 |
| 3 | Palani | 52 | M | 45000 | 02.03.07 | + | - | + | - | - | + | + | - | + | Perf.peritonitis | Sigmoid perf. | 36 | Sigmoid loop colostomy | Died the next day | 1 |

# GASTRIC ULCER-ENDOSCOPIC VIEW

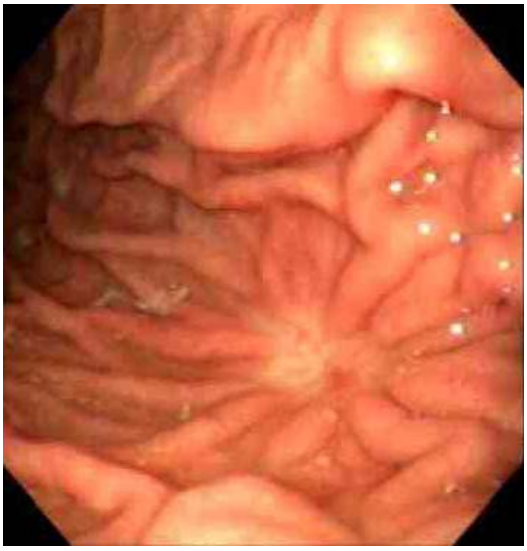


**Fig No.1**

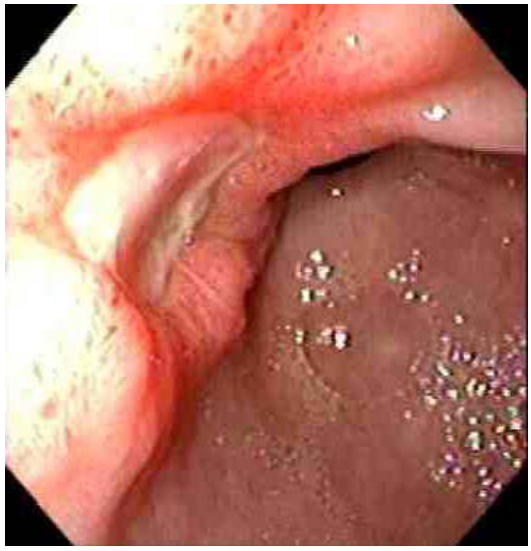


**Fig No.2**

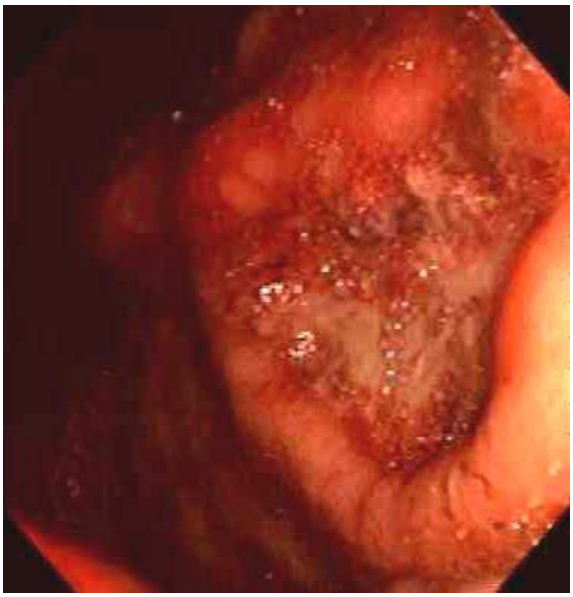
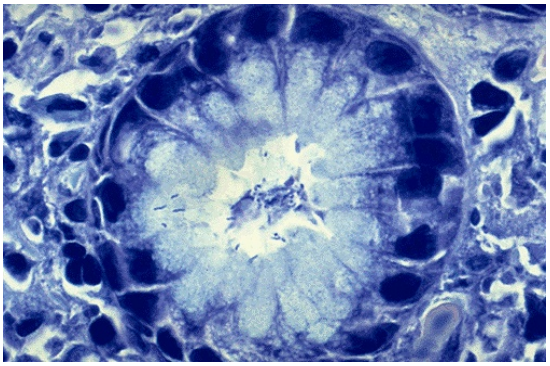
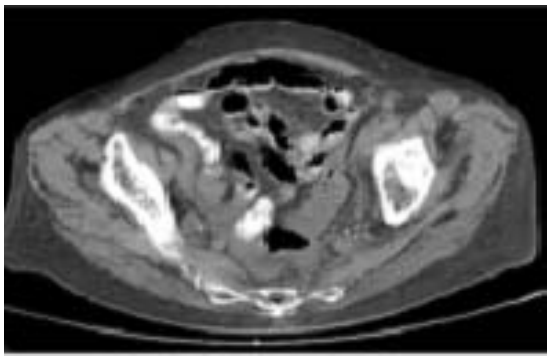


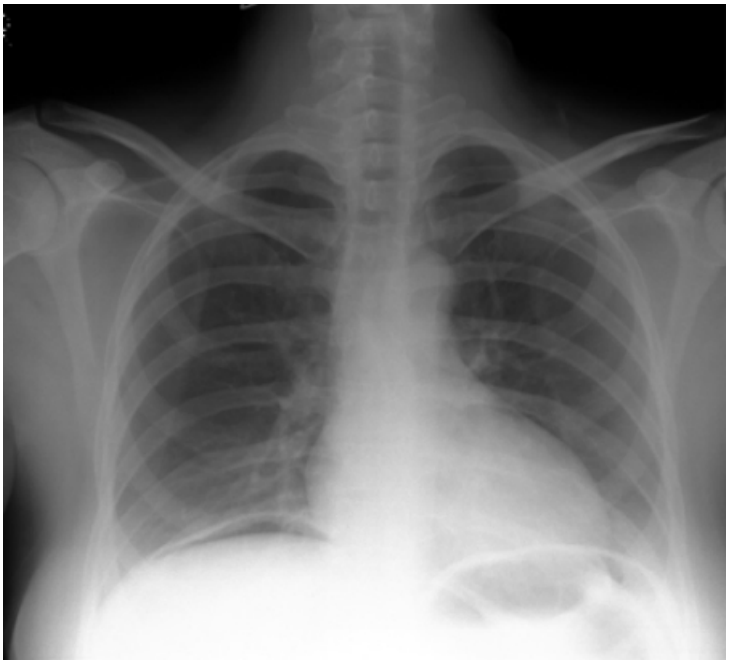
**Fig No.3**



**Methylene blue staining of H.Pylori**
**Fig No.4**

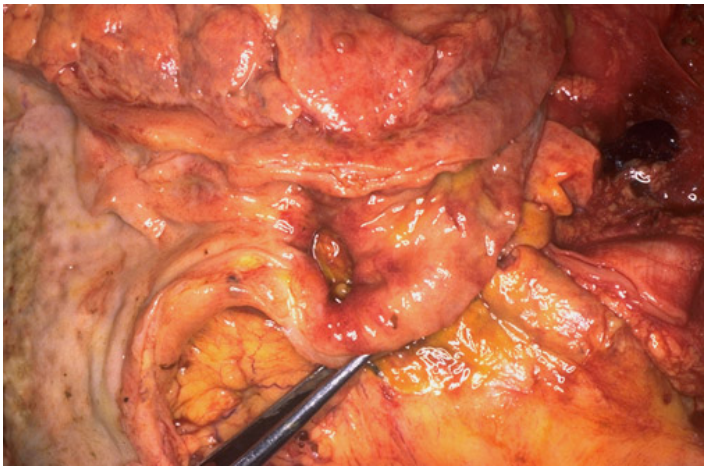


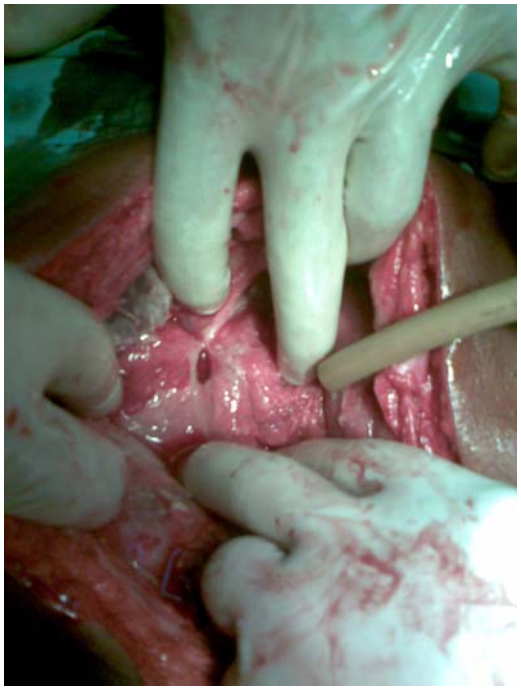
**CT scan of the abdomen showing moderate amount of free air anteriorly and a small linear area of free air outside the sigmoid colon.**
**Fig No.5**



**Free air under the right hemi diaphragm.**
**Fig No.6**

**Duodenal Ulcer Perforation**
**Fig No.7**





**Duodenal Ulcer Perforation**
**Fig No.8**

**Acute Duodenal Ulcer-Endoscopic View**
**Fig No.9**

## SIMPLE CLOSURE WITH OMENTAL PATCH OF DU PERFORATION

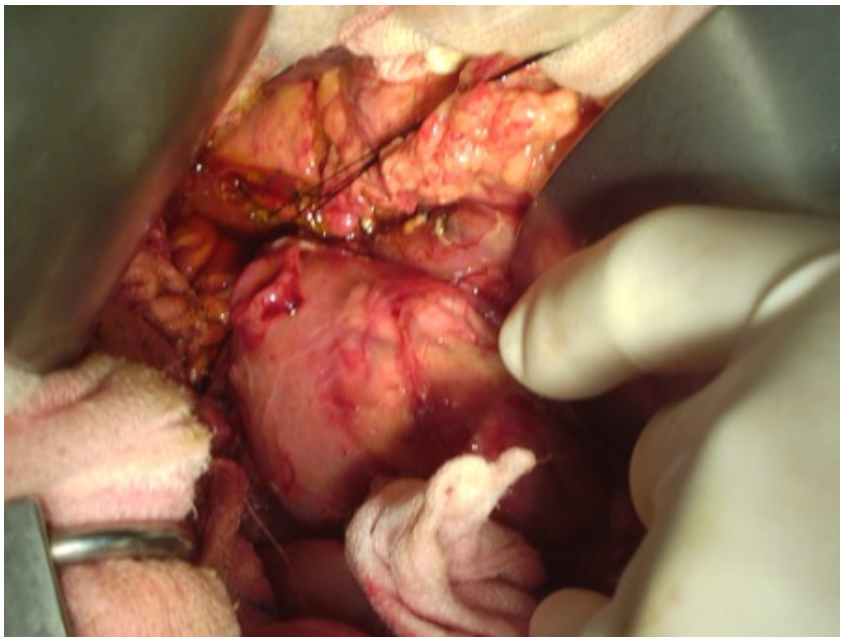


**Fig No.10**

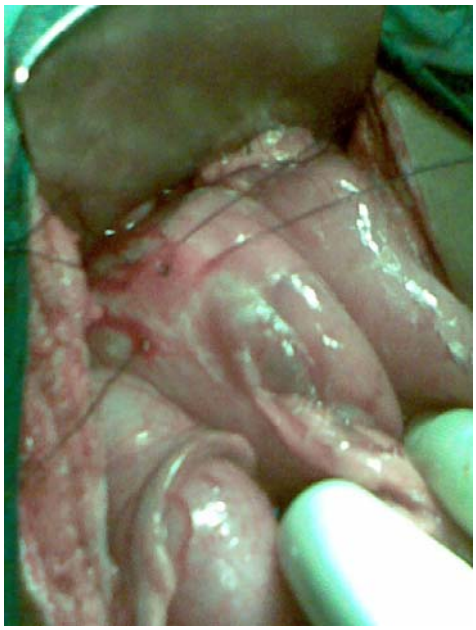


**Fig No.11**

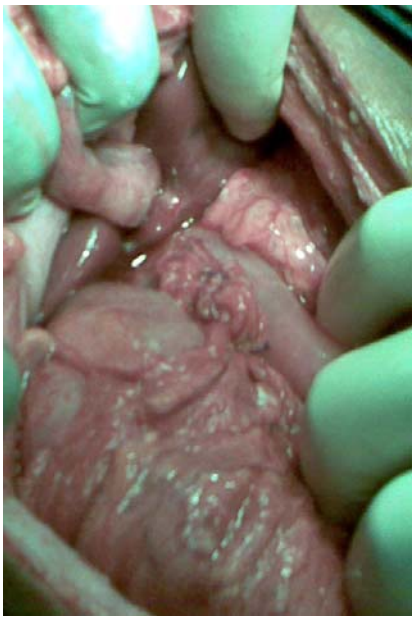
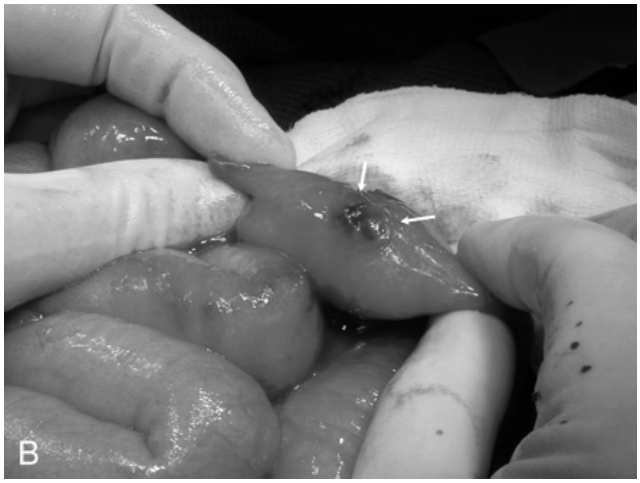


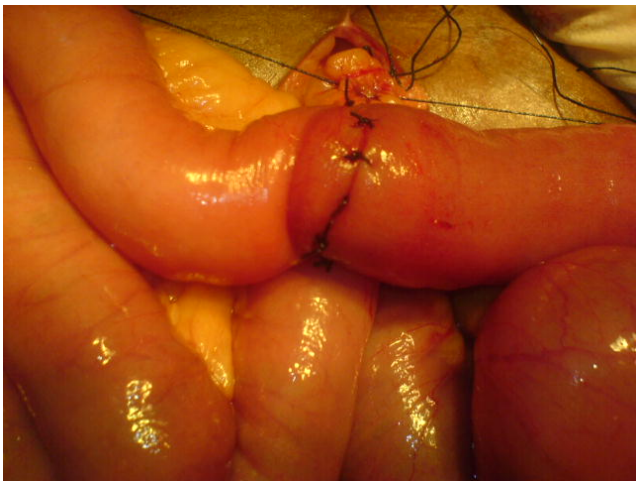
**Fig No.12**


**Perforated Diverticulam**
**Fig No.13**

:




**Small Intestinal Perforation**
**Fig No.14**



**Resection and End-to-End Anastamosis**
**Fig No.15**

## GANGRENOUS SIGMOID COLON WITH PERFORATION

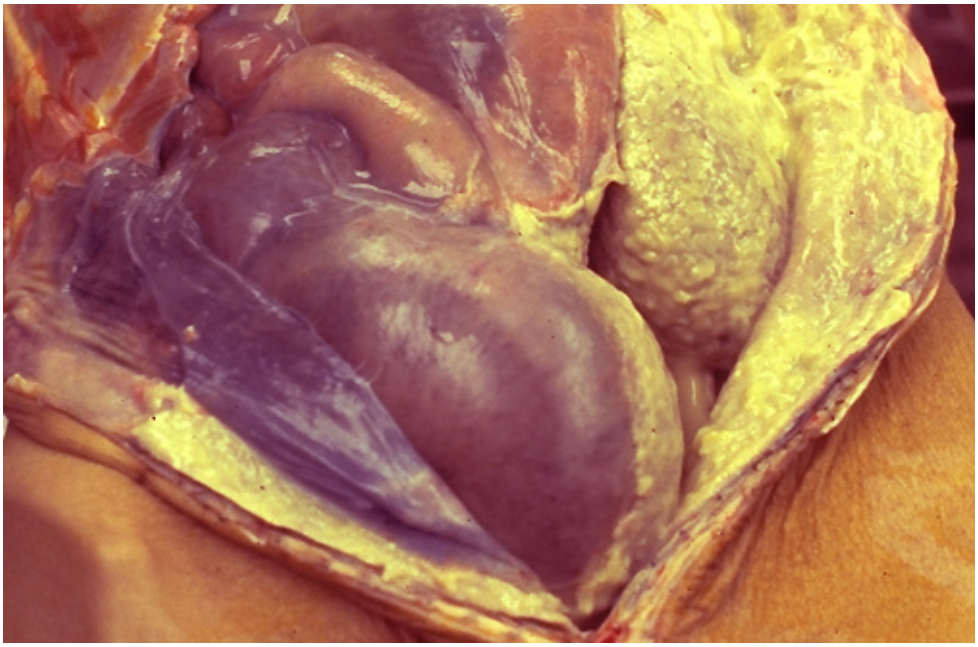


**Fig No.16**

# ABBREVIATIONS

| | | |
|---|---|---|
| BS | - | Bowel Sounds |
| GIT | - | Gastro Intestinal Tract |
| DUP | - | Duodenal Ulcer Perforation |
| GUP | - | Gastric Ulcer Perforation |
| IP | - | Ileal Perforation |
| NSAID | - | Nonsteroidal Anti Inflammatory Agents |
| Pre OP Diag. | - | Pre Operative Diagnosis |
| Per Op Diag. | - | Per Operative Diagnosis |
| Post Op Comp | - | Post Operative Complication |
| Perf. Peritonitis | - | Perforation Peritonitis |
| Sc With Op | - | Simple Closure With Omental Patch |

**ABBREVIATIONS IN MASTER CHART**

| | | |
|---|---|---|
| P | - | Pain |
| VOM | - | Vomiting |
| DIS | - | Distention |
| Alc | - | Alcoholic |
| F | - | Fever |
| Rig | - | Rigidity |
| BS | - | Bowel sound |
| GUD | - | Gas under the diaphragm |
| POD | - | Pre op diagnosis |
| DIH | - | Delay in hours |
| PRO | - | Procedure |
| POC | - | Post op complications |
| NOHS | - | Number of Hospitals stay |