A Dissertation on

GASTROINTESTINAL PERFORATIONS CLINICAL STUDY & MANAGEMENT

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GASTRO - INTESTINAL PERFORATIONS CLINICAL STUDY AND MANAGEMENT

DISSERTATION FOR BRANCH –I
M.S(GENERAL SURGERY) DEGREE EXAMINATION

MARCH – 2007

Department of Surgery,
Tirunelveli Medical College,
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CERTIFICATE

I hereby certify that this dissertation entitled "Gastro - intestinal perforations - clinical study and management" is a bonafide work conducted by Dr. M. Karuppasamy under my full supervision and guidance and submitted in partial fulfillment of the requirements for the award of the degree of Master of Surgery – Branch 1 (General Surgery) Eamination, March – 2007 Under the Tamilnadu Dr.M.G.R., Medical University, Chennai.

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Tirunelveli
Date:

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INTRODUCTION

Gastro Intestinal Tract Perforations represent one of the most common acute abdominal emergencies in the surgical field and is still a dreaded condition having a high morbidity and or mortality. Differences in the clinical presentation of Gastro Intestinal tract perforations vary from the typical severe acute abdominal pain at one end, to subtle or no symptoms in the hospitalized patients for unrelated illness at the other end1. The various atypical presentations that mimic other abdominal conditions throw a real challenge over the diagnosis to the emergency surgeon.

A careful clinical history, methodical clinical examination and radiological study plays a major role in the early diagnosis of this acute abdominal emergency. There are multiple factors that influence the prognosis and outcome of the patient. Preoperative resuscitation, appropriate administration of broad-spectrum antibiotics and good postoperative care are the mainstay in the management of Gastro Intestinal Perforations. The operative management depends upon the cause of perforations. Surgeons must continually reassess standard method of treatment and be receptive to new ideas.

AIM OF STUDY

1. To study the presentation of various Gastrointestinal perforations admitted in the General Surgical Department of Tirunelveli Medical College Hospital.
2. To analyse the etiology & clinical features of Gastrointestinal perforations .
3. To compare the reliability of physical findings versus radiological signs in cases of Gastrointestinal perforations.
4. To study various types of managements of gastrointestinal perforations and merits & demerits of them.
5. To study the mortality, morbidity in various groups followed in the management of these cases ,with ref to their manifestation.

Materials and Methods

This study was conducted in the Department of General Surgery, Tirunelveli Medical College Hospital, Tirunelveli for a period of 21 months from November 2004 to August 2006.

127 cases of gastro intestinal perforations were studied during the period.

The diagnosis was established by the Duty surgeon provisionally based on the clinical presentation. Definitive diagnosis established at the time of operation. As pre operative evaluation following investigation done

- ♣ Relevant biochemical tests
- ♣ Blood grouping typing
- ♣ X-ray chest , Abdomen
- ♣ USG (Ultra Sonogram)
- ♣ E.C.G
- ♣ Abdominal paracentesis whenever warranted

Peroperative finding:

Operative details included the

- ♣ Site of the perforation
- ♣ Size of the perforation
- ♣ nature and quantity of peritoneal fluid & soiling
- ♣ the gross appearance of the bowel bearing the perforation
- ♣ the nature of surgical procedure performed
- ♣ Tissue biopsies for histologic confirmation were taken in appropriate cases

Post Operatively:

Morbidity was analysed in terms of associated complications following surgery and duration of hospital stay.

Following details were observed from the clinical course and recorded in case records.

- Patients name, age, sex, inpatient number (pt identity).
- Clinical features and abdominal findings
- Delay in hours between symptoms and surgery
- Operative findings
- Procedures done
- Post operative complications
- Duration of hospital stay

All case included in this study were observed / assisted /operated by the presentor.

Inclusion criteria:

All cases admitted with signs of peritonitis included irrespective of etiology.

Exclusion Criteria:

- ♣ cases of Oesophageal rupture
- ♣ cases of perforations of hepatobiliary system
- ♣ Cases of iatrogenic perforation during laparotomy
- ♣ Cases of delayed presentation with shock and septicemia whose general condition did not warrant any operative management even after all resucitative measures.

PROFORMA

CLINICAL STUDY AND MANAGEMENT OF GASTRO-INTESTINAL PERFORATIONS

l. CASE NO:

2.NAME: AGE: SEX:

ADDRESS:

OCCUPATION:

DATE OF ADMISSION: DATE OF OPERATION:

DATE OF DISCHARGE / EXPIRE:

3.COMPLAINTS:

(a) Pain : 1) Duration:
2) Site:
3) Nature:
4) Radiation:

(b) Vomitting:l) Duration:
2) Frequency:
3) Amount:
4)Vomitus: Bilious/Faecal /Blood/Otherwise

(c) Fever: 1) Duration:
2)Type:remittent/continous/intermittent

(d) Distension of abdomen:

(e) Change of bowel habits:

(f) Other complaints (if any):

4. PAST HISTORY:
(a) Pain abdomen:
(b) Haematesis/ Malaena:
(c) Previous operation:
(d) Drug history:
(e) Fever:
(f) Other complaints(if any):

5. PERSONAL HISTORY:
(a) Habits: Smoker/Alcholic:
(b) Diet: Mixed/Vegetarian:
(c) Appetite: Good / Impaired:
(d) Bowel and Bladder:

6. FAMILY HISTORY:

7. GENERAL PHYSICAL EXAMINATION:

(a) Vital Signs: Pulse: B.P:

Temperature:

Respiration:

(b) Built and Nutrition:

(c) State of Hydration:

(d) Anaemia / Jaundice / Pedal oedema / Lymphadenopathy:

8. EXAMINATION OF THE ABDOMEN:

(a) Inspection: 1) Shape:

2) Umbilicus:

3) Movement with respiration:

(b) Palpation: 1) Site of tenderness:

2) Guarding and rigidity:

3) Any mass:

(c) Percussion: 1) Liver dullness:

2) Evidence of fluid in the peritoneal cavity:

(d) Auscultation: .

9. EXAMINATION OF OTHER SYSTEM:

(a) Cardio Vascular System:

(b) Respiratory System

(c) Central Nervous System

(d) Locomotor System:

10. PER RECTAL EXAMINATION:

11. CLINICAL DIAGNOSIS:

12. INVESTIGATIONS:

BT: CT:'

Albumin: Deposits

Routine: 1) Blood: Hb: Blood Group:

TC: DC: BT: CT:

2) Urine: Sugar:

Special: (a) X-Ray Abdomen- Erect- Supine:

1) Air under the diaphragm: Present / absent

(b) Widal test:

13. PRE OPERATIVE DIAGNOSIS:

14. PRE OPERATIVE TREATMENT:

15. OPERATIVE DETAILS:

    1) Incision:

    2) Exudate: Colour Bloody / Bilious / Prulent

       Amount:

    3) Site, Size and Number of perforations:

    4) Other findings:

    5) Operative Procedures:

16. POST OPERATIVE DIAGNOSIS:

17. POST OPERATIVE TREATMENT:

18. POST OPERATIVE FOLLOW-UP:

    (a) Date of ryle's tube removed:

    (b) Date of sutures remopved:

    (c) Immediate post operative complaints:

    (d) Result: Cured / Relieved / Expired / Otherwise:

19. CONSERVATIVE TREATMENT:

    (a) Parenteral fluid (Nature and quality)

    (b) Antibiotics:

        l)Type:

        2)Dose:

        3)Duration:

    (c) Ryle's tube aspiration:

        1)Continous / Intermittent:

        2)Duration and frequency:

        3)Amount:

        4)Colour: Greenish / Bloody / Otherwise:

    (d) Anticholinergics (Dose and duration):

    (e) Analgesics:

    (f) T.P.R. Chart:

    (g) Flank Drain: Unilateral / Bilateral:

    (h) Time of appearance of bowel sounds:

    (i) Time of start of oral feeds (Quality & Quantity):

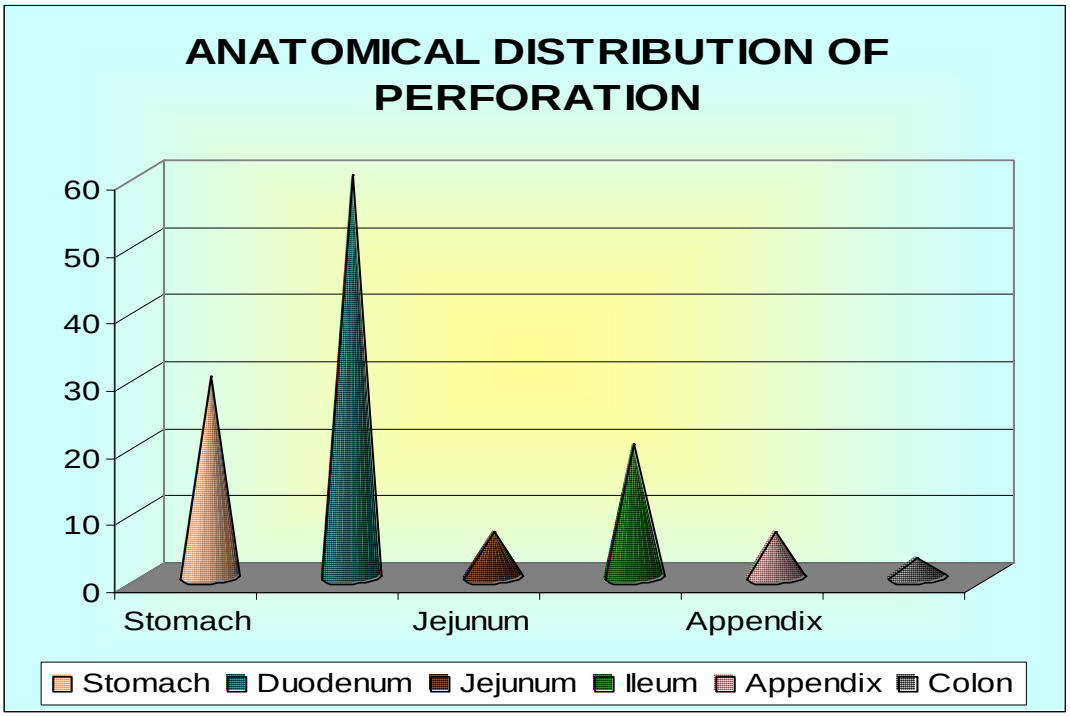
20. FOLLOW-UP:

RESULTS

One Hundred Twenty Seven cases of Gastro Intestinal perforations were studied. Majority of the cases of perforations were Duodenal Perforations.

ANATOMICAL DISTRIBUTION OF PERFORATION

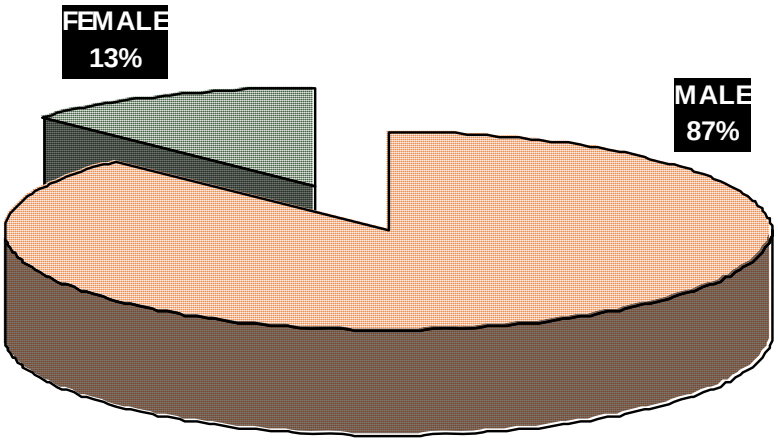
Site	No. of Case	Percentage
Stomach	30	23.62
Duodenum	60	47.24
Jejunum	7	5.51
Ileum	20	15.74
Appendix	7	5.51
Colon	3	2.36
Total	127	100



GI PERFORATION (Gross)

SEX	NO.OF.CASES	PERCENTAGE
MALE	111	87.4
FEMALE	16	12.6
TOTAL	127	100

NO.OF.CASES



Remarks : Male Commonly affected

GASTRIC PERFORATIONS

Gastric perforations were found in 30 cases of the entire study group.

Table 9: Age incidence of gastric perforations

S.No	Age	No.Of cases	Percentage
1.	12 – 29	5	
2.	30 – 39	4	
3.	40 – 49	7	
4.	50 – 59	8	41.66
5.	> 60	6	16.66
	Total	30	100

27 male cases and 3 female cases with a male : female ratio 9 : 1 are studied.

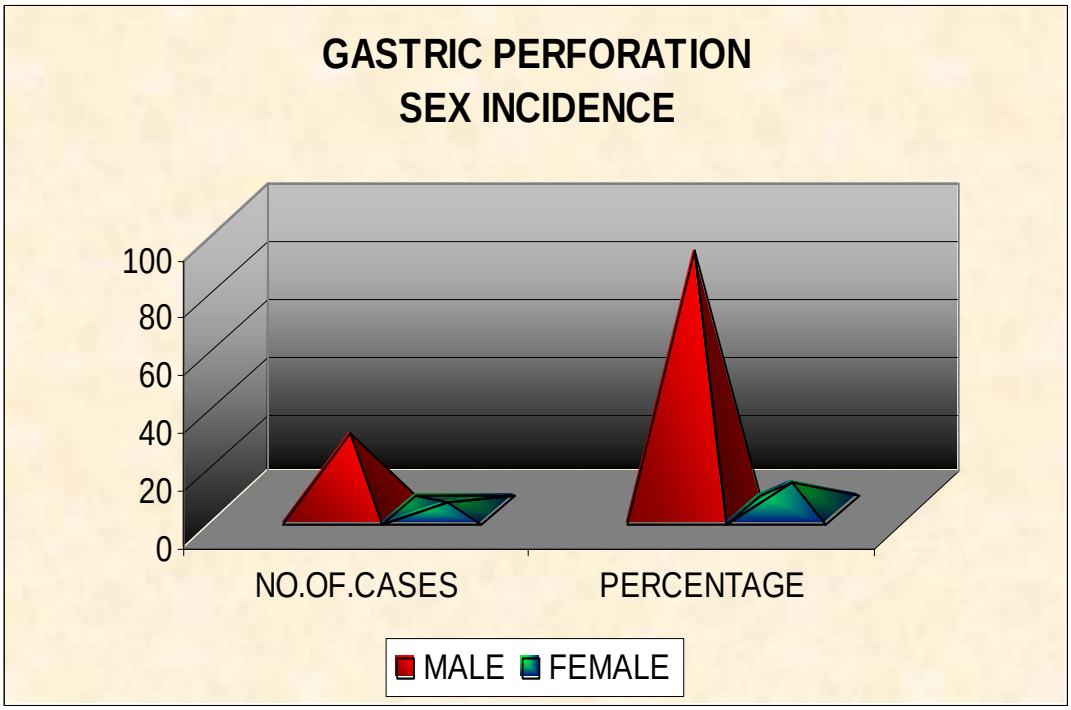
Table 10: Sex Distribution in Gastric Perforation

S.No	Sex	No. Of Cases
1.	Male	27
2.	Female	3
	Total	30

GASTRIC PERFORATION

SEX INCIDENCE

SEX	NO.OF.CASES	PERCENTAGE
MALE	27	90
FEMALE	3	10
TOTAL	30	100



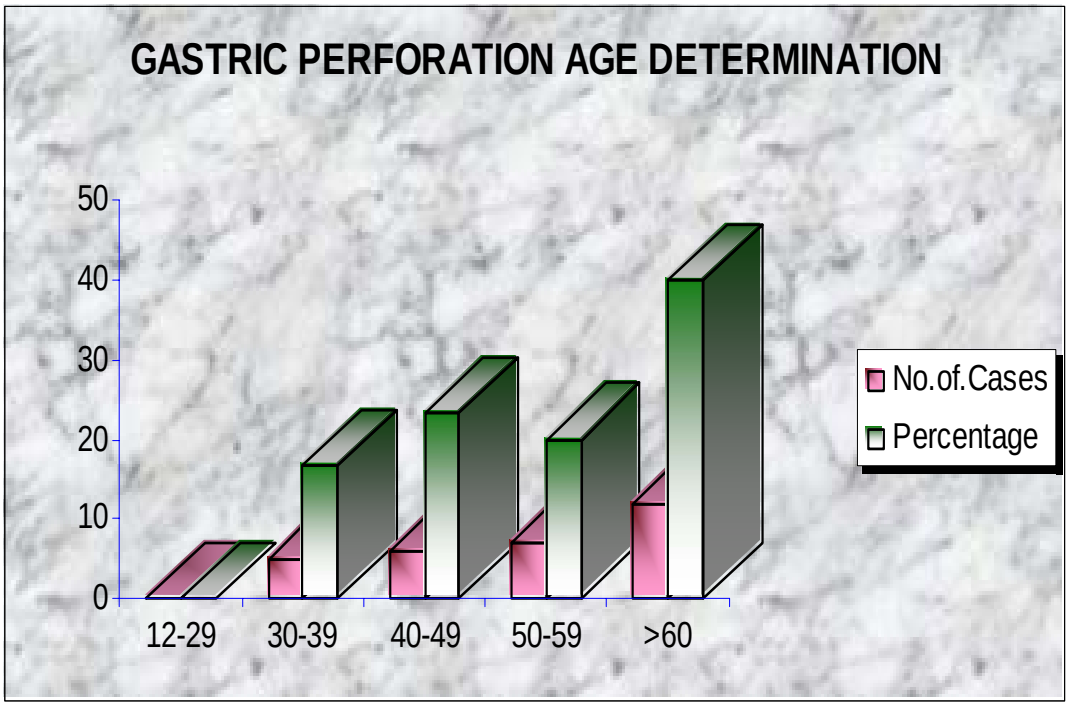
Remarks : Male commonly affected

Male : Female ratio = 9:1

GASTRIC PERFORATION

AGE DISTRIBUTION

Age	No.of.Cases	Percentage
12-29	0	0
30-39	5	16.6
40-49	7	23.33
50-59	6	20
>60	12	40.07
	30	100



Remarks: More common in 40-60 years

Gastric perforation occurred more often in the fifth decade of life.

- ♣ Most cases smokers and 15 cases were alcoholic.
- ♣ 3 cases were malignant ulcer perforation
- ♣ 2 cases were due to trauma (Blunt injury).
- ♣ 1 case during treatment for oleander poisoning developed perforation

  ?Drug induced
- ♣ In 1 case as encountered in the intensive cardiac care unit admitted for myocardial infarction. - ?Drug induced

Plain upright X-ray of the abdomen showed air under the diaphragm in all cases.

- ♣ Stab injury was the cause of lacerated injury over the anterior wall of stomach in one patient.
- ♣ In most cases perforation closed in 2 layers using 2.0 vicryl and 3.0 silk.
- ♣ One case due to giant perforation closed with jejunal patch.

DUODENAL PERFORATION

During the study period a total of 60 cases of Duodenal ulcer perforation were admitted among 127 cases of perforation making a percentage of **47**.

Duodenal ulcer perforation had a preponderance in males. Only 1 case of perforation in female was noted during the study period.

Majority of the perforations were in the age group of 30-50 vrs. The lowest age was 21 yrs and the oldest was 70 yrs.

On analysis of the symptoms retrospectively majority of the patients complained of diffuse abdominal pain. 80% of the patients had diffuse guarding and rigidity. In 70% of the cases the liver dullness was obliterated.

Plain X-Ray Abdomen was the main investigation that was done. 90% of the cases showed free air under diaphragm. 3 cases, which didn't show air initially.

Blood urea, creatinine & electrolytes were taken for all the cases.

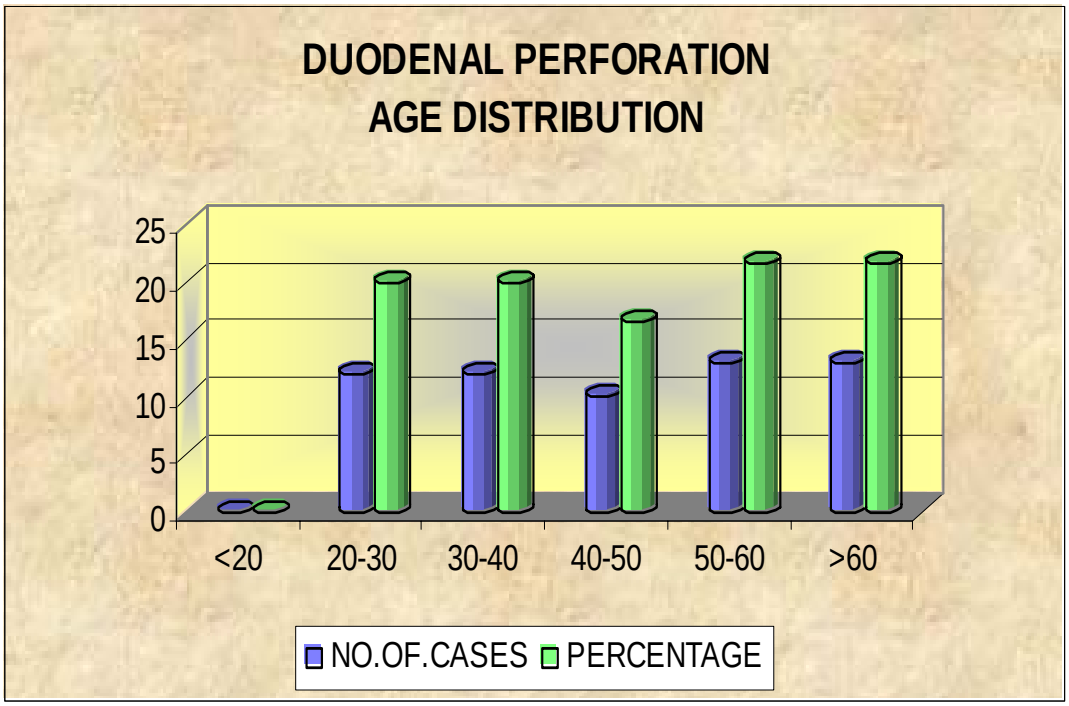
Peritoneal fluid cell count & bacteria were analysed. 90% of cases.

The most common bacteria cultured from the peritoneal fluid was E.Coli.

DUODENAL PERFORATION

AGE DISTRIBUTION

AGE	NO.OF.CASES	PERCENTAGE
<20	0	0
20-30	12	20
30-40	12	20
40-50	10	16.67
50-60	13	21.67
>60	13	21.67
TOTAL	60	100



OPERATIVE MANAGEMENT

Simple closure of perforation

Laparotomy was done by upper mid line or by upper Rt. paramedian incision. Midline incision was preferred in most cases.

Size of duodenal perforation	No. of Cases
< 0.5 cm	45
0.5 – 1 cm	10
> 1.0 Cm	5

Peritoneal cavity was toileted with normal saline taking care of the Rt & Lt sub- phrenic spaces, the pelvic cavity and the Rt & Lt paracolic gutters. 3 stay stitches using 2 ' O ' vicryl / 2 'O' catgut was taken. A live omental patch was placed over the perforation. The upper & lower stitches were tied first and the center one was tied last. This was the procedure that was done in more than 90% cases.

The entire peritoneal cavity was mopped up. The abdomen was closed in single layer with flank drains.

In all the cases perforation site found to be first part of duodenum. anterior wall.

One case found to be postero lateral aspect of first part of duodenum.

4 cases were managed with bilateral flank drainage .

Bilateral Flank Drainage

This was done in patients who were not fit to undergo laparotomy . After preparing the case, skin of flanks were infiltrated with local anaesthetic agent and incision made with a knife, muscles were split and peritoneum opened with a sinus forceps. A corrugated drain or Malecot's catheter was introduced through the opening and fixed.

4 cases of perforation were managed with bilateral flank drain. The mortality was high. One of them survived

About 30% of the operated patients developed complications ranging from minor ones like stitch abscess to major ones like septicaemia and renal failure.

Most of the wound sepsis were treated with antibiotics and dressings with debridement. Wound was resutured after healthy granulation.

Pus collection (sub diaphragmatic & pelvic abscesses) occurred in 16% of the patients. Sub - Diaphragmatic collection was drained with needle aspiration under sonographic guidance .Pelvic abscesses were drained through rectum.

There were 2 cases of duodenal fistulae which closed after 2 wks of conservative management.

The mortality was about 24% the main cause was septicaemia and renal failure.

Follow – Up:-

After discharge patients were advised to continue Anti H.pylori treatment for 3 weeks after which they were on H-2 receptor antagonists or proton pump inhibitors for another 4 weeks. Patients were advised endoscopy after 6 months. 0nly 5 - 10% of the patients came after 6 months.

SMALL BOWEL PERFORATIONS

Ileal perforation during the study period 20 cases of ileal perforations were encountered.

In 20 cases of ileal perforation. Only 3 cases are found to be Widal positive found to be enteric perforations.

Enteric Perforations:-

During the study period only 3 cases of enteric perforations were admitted in the surgical wards. All ileal perforation were investgated for enteric fever pathology.

The incidence of enteric perforations showed a decreased trend.

On analysis of the symptoms 60% of patients had a history of fever equivocal of Typhoid.

Diagnosis:-

Plain X-Ray and revealed multiple fluid levels due to ileus in 35% of the patients.

Air under diaphragm was present only in 60% of cases.

80% of the cases enteric perforations were suspected preoperatively.

20% of cases suspected to be Duodenal ulcer perforation, appendicular perforation turned out to be Ileal Perforation

In All cases closure was done in two layers with 2 or 1-0 vicryl and 2-0 silk.Peritoneal toileting was done. Closure was done with a flank drain.

one patient underwent appendectomy with peritoneal toileting in conjunction with closure of perforation.

Two cases required resection and anastomosis and one case required ileo-transverse anastomosis.

Jejunal Perforations:-

Out of the total of seven cases included in the study, three were due to traumatic perforation (two-stab; one due to blunt injury).

Two cases were due to adhesive obstruction and two cases had multiple areas of bowel involvement.

5 cases were treated by perforation closure in two layers using 2.0 vicryl and 3.0 silk and 2 cases underwent resection anastomosis.

COLONIC PERFORATION:-

Among 3 cases of colon perforations (2 blunt injury and 1 stab injury). 1 patient had associated splenic injury and mesentric tear was treated by splenectomy and left hemi colectomy respectively.

2nd case had ascending colon perforation for which exteriorization loop colostomy was done. Later date colostomy closure was done.

3rd patient with perforation near the splenic flexure injury who underwent perforation closure..

APPENDICULAR PERFORATION

7 cases of appendicular perforations were encountered during the study. 5 cases were males and 2 cases were females.

All the cases presented with R.I.F. pain, vomiting and fever.

Symptoms	
RIF Pain	+
vomiting	+
Fever	+
Signs	
RIF tenderness	+
Guarding – Localished to RIF	5
Diffuse	2
Distension	+
Investigation	
Air under diaphragm	Nil

Plain X-ray abdomen revealed localized ileus in RIF. USG showed evidence of dilated bowel loops in RIF with free fluid.

Management :-

After correcting the fluid and electrolyte imbalance, under cover of antibiotics abdomen was opened by Lanz\Mc Burney's\RPM-incisions. Pus in the peritoneal cavity was sucked out. Most common site of the perforation was the tip of the appendix. In all cases regular appendectomy were done. Thorough toileting was done and wound closed with a drain.

Post-operative Period

Patients were given oral fluid on an average in the 3rd day. Minor complications like wound infection to major ones like faecal fistula occurred. One case of faecal fistula following appendectomy was managed conservatively.

REVIEW OF LITERATURE

HISTORY

The history of disease is atleast as old as the history of mankind. The acute pathological conditions of the abdomen e.g., facies hippocratica which represents terminal stages of peritonitis have been recognized since hippocrates (400 BC).

Rawlinson is credited with the first published report, in 1727, of a perforated ulcer, which happened to be gastric. The first published report of perforated duodenal ulcer was by Hambergeri in 1746. Hensner sutured a perforated gastric ulcer in the patient's home, the case being reported some months later by kriege in 1892, the first report of a successful operation for perforated duodenal ulcer was by Dean in 1894.

Hermon Taylor in 1957 and Donovan in 1979 strongly advocated non-operative management for perforated duodenal ulcer. Me Gee and Sawyers showed in 1987 the clear superiority of resection over simple closure in case of perforated gastric ulcer.

TRAUMATIC PERFORATIONS

In 1767, Nolleston Fils reported the first successful repair of gastric injury. Picacastelli is credited with describing the first case of blunt gastric rupture in 1922.

Aristotle was the first to describe intestinal injury as a consequence of blunt abdominal trauma. He is credited as saying, "a slight blow will cause rupture of the intestines without injury of the skin" the first report of intestinal injury from penetrating trauma is attributed to Hippocrates.

SURGICAL ANATOMY

The peritoneal cavity is lined with a single layer of mesothelial cells. The parietal peritoneum covers the abdominal cavity (i.e., abdominal wall, diaphragm, pelvis), the visceral peritoneum covers all the intra abdominal viscerae, forming a cavity that is completely enclosed except at the open ends of the fallopian tubes.

A small amount of fluid sufficient to allow movements of organs is usually present inthe peritoneal cavity. The fluid is normally serous (protein content <30 g/l, < 300 WBCs/|il). In the presence of infection, the amount of this fluid increases, the protein contents climbs to more than 30 g/l and the WBC count increases to more than 500 WBCS/µI, in other words, the fluid becomes an exudate.

The transverse colon and the drape of greater omentum divide the abdomen horizontally into supracolic and infracolic compartments. Therefore the symptoms and signs of peritonitis may be localized to upper lower halves of the abdomen for sometime.

The forward convexity of the lumbar spine provides two marked lateral gutters and only a shallow anterior communication between them across the midline. Consequently, liquid spreads by movement largely around the periphery of the abdomen and not a great deal across the midline, hence the initial laterally of many peritoneal processes.

The right subhepatic space (Morison's pouch) is open only to the right, where it communicates with the right paracolic gutter. Liquid from perforated duodenal ulcer or seepage from the gallbladder region passes to the right and then both upwards to reach the right subphrenic space and downwards to tha
right iliac fossa.

Paracolic effusions reach the general peritoneal cavity across the sigmoid flexure. Pelvic effusions pass up both the paracolic gutters and there after to the subphrenic spaces and to the general peritoneal cavity.

A left sided origin above the transverse colon results in left paracolic and left subphrenic spread.

CLASSIFICATION AND ETIOPATHOGENESIS

CLASSIFICATION

It is broadly classified into

A) Non-traumatic, and

B) Traumatic

A) NON-TRAUMATIC PERFORATIONS: Can be classified into those affecting:

I) STOMACH AND DUODENUM:

1) Diseases:

a) Peptic ulcer

b) Chronic gastric ulcer

c) Acute erosive gastritis

2) Neoplasia:

a) Carcinoma stomach

b) Leiomyosarcoma stomach

3) Misc:

a) Volvulus stomach

b) Corrosive gastritis

c) Mallory Weiss syndrome

II) SMALL INTESTINE (Excluding duodenum) AND APPENDIX:

i) Inflammatory diseases:

Tuberculosis , Salmonella enteritis.

Necrotising enterocolitis (also called as staphylococcal enterocolitis, clostridium deficile enterocolitis)

- ❖ Acute apnendicitis (Gangrenous variety)
- ❖ Nonspecific enteritis

ii) Neoplastic: Rare, seen in malignant tumors - mainly leiomyosarcoma

iii) Vascular : Ischaemic enterocolitis

iv) Misc:

a) Meconium peritonitis

b) Parasitic peritonitis due to perforation by round worms

c) Diverticulitis

d) Radiation enteritis

e) Strangulated Hernia

4) Large Colon:

i) Inflammatory diseases

Chronic: - Crohn's disease

-Ulcerative colitis

Acute: - Acute amoebic dysentery

ii) Neoplasia: Rarely malignant tumors cause perforation.

iii) Vascular: Ischaemic colitis

iv) Misc:

a) Volvulus

b) Megacolon

c) Radiation enterocolitis

d) Diverticular disease

e) Strangulated Hernia

B) TRAUMATIC PERFORATIONS:

The causes of the traumatic perforations are:

1) Blunt injury

2) Penetrating injury:

    a) Fire-arm wounds

    b) Stab injuries

3) Sharp foreign bodies

4) Iatrogenic injuries

5) Injuries due to corrosive acids and alkalies

GASTRQ DUODENAL PERFORATIONS

AETIOLOGY

- Complications of peptic ulcer disease.
- Drug induced perforation
- Traumatic perforation
- latrogenic perforation
- Cushing ulcer perforation
- Curling's ulcer perforation
- Zollinger Ellison syndrome
- Malignant perforation: 10% of the perforations in the stomach are malignant.

BACKGROUND

Peptic ulcer disease of the stomach and duodenum has undergone dramatic evolution of over the past 40 years. Overall morbidity, hospitalization and operations for peptic ulcer disease has decreased , thanks to the widespread use of gastric antisecretory agents and H.pylori eradiation.

There has been a relative increase in the incidence of peptic ulcer disease in the elderly, resulting in increased morbidity and hospitalization in that age group, the elderly female has been the most profoundly affected largely because of use of NSAIDs in this population.

PEPTIC ULCER PERFORATION

Incidence

- The incidence of perforation of peptic ulcer is 7 to 10 cases pei 1,00,000 population per year. now reduced because of PPI & H2 blockers
- 7% of the patients hospitalized for peptic ulcer disease present with preforation
- Perforation was he first manifestations in about 2% of the patients with peptic ulcer diseases prior to PPI & H2 blockers
- Pyloroduodenal perforation occurs 6 to 8 times more often than gastric perforation.

Age

- Peptic ulcer perforations occur more commonly in the middle ages between 30 to 50 years.
- **Now increasing use of NSAIDs have resulted in a shift in the incidence of perforation in the 6th and 7th decade of life.**

Sex

a) The sex distribution of peptic ulcer perforation shows a male : female incidence of 10:1
b) At present there is a steady increase in the number of females of the older age group using NSAIDs.
c) Prepyloric perforations occur more often in young men where a gastric perforation is more common in the elderly women.

Occupation

Peptic ulcer perforations are more common in patients of low socio economic status.

RISK FACTORS

1. Use of NSAID
2. Smoking
3. Increasing patients age
4. Patients on immuno suppressive therapy
5. Chronic obstructive pulmonary disease
6. Major bums
7. Multi organ system failure.

PATHOPHYSIOLOGY

A peptic ulcer is said to have perforated when it extends through the muscle wall and serosa of the gastro intestinal tract thereby establishing communication between the lumen and adjacent space or structure. The perforation occurs as a result of sudden sloughing of the base of the ulcer due to impaired blood supply.

The site of pyloroduodenal perforations is usually the anterior wall and majority of the perforated gastric ulcers are located on the lesser curvature. Posterior perforation of a gastric ulcer may occur into the lesser sac.

Perforation leads to leakage of gastric or duodenal contents into the peritoneal cavity initiating an acute peritonitis. Although it is an initial chemical peritonitis, bacterial peritonitis supervenes over the next few hours.

The presence of bacteria in the peritoneal cavity stimulates an inflow of acute inflammatory cells. The omentum and the viscera tend to localize the site of inflammation. This results in an area of localized hypoxia, which in turn facilitates growth of anaerobes and produce impairment of bactericidal activity of granulocytes. This leads to increased phagocytic activity of granulocyte, degradation of cells, hyper secretion of fluid forming the abscess, osmotic effects, shift of more fluids into the abscess area and enlargement of the
peritoneal exudates causing paralytic ileus.

Absorption of bacterial endotoxins through the inflamed peritoneal surface causes endotoxemia. The combination of fluid and electrolyte imbalance and septicemia results in shock and multi organ failure, which is the cause of, increased mortality in untreated patients of perforative peritonitis.

STAGE OF PERITONEAL IRRITATION

This stage lasts for the first 2 to 3 hours following perforation. The sudden outpouring of caustic gastric juice into the peritoneal cavity producing chemical peritonitis causes the initial symptoms. The patient can recall the exact time of perforation by the abrupt on set of intense abdominal pain. The patient may or may not vomit. Referred pain is felt over the tip of left shoulder in 1/3 to 1/2 of the patients due to irritation under the dome of diaphragm. Initially the patient may be shocked with a tachycardia but there is little change in the temperature. Respiration is shallow and the abdomen does not move with respiration. Tenderness and muscle guarding are constantly present over the right side of the abdomen.

STAGE OF PERITONEAL REACTION

During the secondary stage, the irritant gastric juice is diluted by the peritoneal exudates. The patient feels comfortable due to the buffering action of the fluid secreted. Symptoms are reduced but signs are still present. Muscular rigidity continues to be present. This stage is marked by two other features; obliteration of liver dullness and presence of shifting dullness. Evidence of free air within the abdominal cavity may be seen on a plain upright radiograph of the abdomen and chest in nearly 70% of the cases.

STAGE OF DIFFUSE PERITONITIS

In the tertiary stage, with the establishment of bacterial peritonitis, patient has gone a step further towards the grave. The pinched and anxious face, sunken eyes and hollow cheeks - so called fades hippocratica, with rising pulse rate which is low in volume and tension, persistent vomiting, board like rigidity of the abdomen, increasing the distension of the abdomen all are evident in the terminal stage.

At times the spillage of the luminal contents is more of seepage and if seepage becomes contained in a smaller area, the pain though intensive, is located near the site of perforation and muscular rigidity is limited in extent.In posterior perforation the inflammatory reaction is contained in the lesser sac and symptoms may be obscure.

AGE

The peak incidence of peptic ulcer perforation is between 40 and 60 years. Perforation due to chronic gastric ulcer or carcinoma occur in older age group,

usually after 50 years. Appendicular perforation is rarely encountered before the age of 2 years, reaches peak incidence in the second and third decades. Perforation in crohn's disease is independent of age but in Megacolon the symptoms usually appear within 3 days following birth. Perforation in ulcerative colitis is seen more in the third, fourth and second decades in that order. In diverticulitis it occurs after 40 years and in carcinoma

In addition to the above, most of the gastro-intestinal perforations simulate each other so a careful approach to the patient is absolutely essential.

SEX

The male to female ratio in peptic ulcer perforation varies from 10:1 to 4:1. In carcinoma stomach and chronic gastric ulcer this ratio falls to 3:2. In appendicular perforation it is 1: 5 to 1: 6. Perforation in crohn's disease is independent of sex and age group.

SYMPTOMS

Pain: Pain is the one which makes the patient to seek medical advice immediately. The onset, site, type, radiation and character of pain will give us a clue to the diagnosis of the underlying disorder.

Sudden onset of pain is a feature of all perforations. But diminision of pain is not always a happy symptom. In acute appendicitis it may indicate perforation of an obstructive gangrenous appendix. Constant burning pain is a feature of peritonitis and often seen in perforated peptic ulcer.

Characteristically shifting of the pain is usually seen in acute appendicitis. The pain is initially felt around the umbilicus, but later on shifts to the right iliac fossa with the onset of parital peritonitis; In spreading peritonitis the pain is first complained of at the region of affected organ but it soon spreads over the abdomen. In case of peptic perforation the pain is first felt at the right hypochondrium, but soon it is radiated towards the right paracolic gutter. All this times this condition mimicks acute appendicitis.

In case of peritonitis due to any perforation, pain is slightly relieved if the patient lies still. If he rolls about, the pain become worse. In case of pain due to diaphragmatic irritation either due to inflammatory exudate deep inspiration will aggravate the pain.

Past history of periodic pain is a feature of peptic perforation and crampy lower abdominal pain is a feature of tuberculous enteritis, ulcerative colitis and Crohns disease Vomiting: It may be once or twice during early stage. But it is more or less absent in next stages and may re-appear in the terminal stages with the characteristic vomitus of diffuse peritonitis ie., the vomiting is quiet regurgitation of mouthfuls. But nausea is more often complained of and often pain precedes vomiting.

Initially the vomitus is nothing but gastric contents. In late cases of peritonitis the vomitus becomes dark brown, faecolent being mixed with altered blood.

Fever: Fever certainly helps to rule out or clinch some of the diagnosis provided the patient seeks advice in the early stage. Because often in all late cases of perforation fever will make its appearance as a result of peritonitis.

Evening rise of temperature (mild degree) with night sweats is typical of tuberculosis. High fever is Suggestive of appendicular or Hivertirnlar perforation, The sequence of symptoms viz; pain first, vomiting next and fever last is known as Murphy's syndrome and is a feature of acute appendicitis. In Ascariasis the fever is usually 38° C. Step- ladder type of fever with chills and rigors is a feature of enteric fever. A history of fever of 15 to 30 days prior to onset of pain -abdomen is usually seen in enteric fever. But usually the fever will subnormal at the time of perforation. In each ulcerative colitis the fever is very high and the patient will be toxic.

Distension of Abdomen:

It may be a symptom of patients landed to hospitals in late stages of all types of perforations where in paralytic ileus has already ensued. The distension may be in the upper or lower abdomen in early stages but will be all over the abdomen in late stages. The distension of the abdomen is due to ensuing paralytic ileus and fluid collection in the peritoneal cavity.

Bowel Habbits:

In early stages of perforation there may be history of loose motions because of irritation of rectum by pelvic collections. But absolute constipation is a feature of peritonitis. There may be previous history of diarrhoea in amoebic perforation and blood and putrid stool in mesenteric thrombosis.

Past history of alternate constipation and diarrhoea are the features of tubercular enteritis, carcinoma colon and worm infestations. History of Malaena will give clue to the diagnosis of peptic ulcer perforation or carcinoma stomach

Other complaints:

There may be history of drug (particularly NSAID'S, steroids) or strong acid (sulphuric or hydrochloric acid) ingestion.

There may be history of assault leading to blunt or penetrating injuries or road traffic accident.

PHYSICAL EXAMINATION

General:

The patients may be anemic as in peptic ulcer perforation and Ascariasis and may be cachexic, anaemic and jaundiced in perforation due to malignancy.

The patient remains quiet ,because movements will only increase the pain. Only in the last stage of peritonitis and post-operative peritonitis the patient becomes highly disoriented irritable non-cooperative, which is evidenced by throwing of bed cloths, tossing of the hands and feet, etc, nothing seems to give him comfort.

"Abdominal facies" a peculiar facial expression helps the clinician to discriminate an abdominal from an extra abdominal case. In terminal sage of peritonitis, the typical "facies Hippocratica" can be observed. An anxious look, bright eyes, pinched face and cold sweat on the surface are the features of this type of facies. The facies of dehydration is also typical and consists of sunken eyes, drawn cheeks and dry tongue.

In the early stages (except appendicular, diverticular and enteric perforation) the pulse remains normal in rate mid volume. But with spread of peritonitis the pulse begins to quicken and becomes small in volume. In enteric fever there will be initially bradycardia but with onset of peritonitis it becomes thready and tachycardic.

Baring internal hemorrhage and late cases of peritonitis, the respiration rate may seldom be high. If the temperature becomes high, the respiration are often of proportionately increased, the respiration are often of a peculiar grunting type iiT peptic ulcer perforation.

The temperature may be mild (as in late stages of peritonitis') or high (as in peptic or enteric perforation). May be stains of acid over the mouth, cheek and hand.

LOCAL EXAMINATION

In early stages the abdomen may be normal or slight distension may be seen. In late stages there will be generalized distension, the respiratory movement of may be sluggish or absent because of wide spread irritation of diaphragm.

Tenderness is constant over an inflamed organ e.g., in peptic ulcer perforation in the right hypochondrium, in the appendicular perforation at the Mc Burney's point, in amoebic perforation at the amoebic point and etc. in late stages there may be rebound tenderness also. Board like rigidity is characteristic and the cardinal sign of peptic perforation. In the initial stages of peptic perforation the muscle guarding is a

part of protective mechanism, in case of "appendicular perforation it varies according to position of appendix.

Obliteration of the liver dullness will clinch the diagnosis of perforation provided if we rule out emphysematous chest and interposition of colon between liver and the parietal wall. Initially bowel sounds may be present but in late stages they are conspicuous by their absence.

In addition to the above findings there may be demonstrable foci of tuberculosis (in the lungs) or generalized lymphadenopathy (tuberculosis) or enlarged lymph nodes in the left supraclavicular fossa (virchow’s lymph nodes).

Examination tenderness is often elicited in the rectovesical pouch inperforated peptic ulcer and pelvic pendicular perforation. Rectal bulge may be felt in the pelvic collection. May be evidence of fissure, fistula, stricture or abscess in' crohn's disease and growth in carcinoma rectum. Proctoscopy in Ischaemic colitis reveals normal distal segment with blood coming from above.

SPECIAL FEATURES

The clinical features of perforated peptic ulcer can be specially studied under three headings:

a) Stage of peritoneal irritation: Here the patient is pale, anxious, and loath to move. The temperature may be subnormal, but the pulse is raised. The abdomen is held still, moving little or not at all with respiration, the whole abdomen is tender with board like rigidity. It is dull on percussion, sufficient gas may have escaped to reduce

liver dullness in the midaxillary line. It is due to irritation of the peritoneum due to leakage of gastric juice.

b) Period of illusion (After 3 to 6 hours):

duration of pain, or they may notice that the pain radiates into the back or that eating no longer relieves the discomfort.

PERFORATION AND HAEMORRHAGE

The combination of perforation and hemorrhage occurs in either way.

1. Perforation occurring in the course of medical management of hemorrhage.
2. Onset of hemorrhage after a recent perforation.

INVESTIGATIONS

1. IMAGING STUDIES

a) X rays

i) Erect radiographs of the chest and a plain upright radiograph of the abdomen are the most common first line of diagnostic imaging when a perforated peptic ulcer is considered.

As little as 1 ml of free air may be visualized. Free air is present in to 80% of cases. In the upright view, curvilinearJucencies separate the most superior portion of the diaphragm from the liver on the right side and from the stomach and spleen on the left.

ii) On the lateral decubitus view, the free air is usually best seen adjacent to the lateral margin of the liver, but in some patients the iliac portions of the

peritoneum are more superior in location free gas accumulates preferentially over the upper iliac bone.

iii) The supine view may occasionally be the only view ordered and available, especially if pneumoperitoneum is not suspected. Pneumoperitoneum can be detected in a supine view if free gas surrounds a gas-filed bowel loop. In this situation, the inner and outer margins of bowel wall are clearly seen (the Rigler sign).

Some fat may normally outline the serosal surface of bowel loops, but in the presence of pneumoperitoneum the outer surface of the bowel is sharply marginated and more distinct that fav outlined bowel. Small amounts of air rise to the most superior portions of the abdomen and may be seen outlining the anterior margin of the liver an oblique or triangular lucency superimposed over the lower portion of the liver.

A linear lucency overlying the medial mid-liver may represent free air in the fissure for the ligamentum teres.

If large amounts of free air are present, air may outline the falciform liganent anterior to the liver, producing the football sign, a large oval collection of air with a central soft tissue stripe produced by the falciform ligament outlined by surrounding gas. Air under the inferior abdominal wall may outline the umbilical folds the inverted - V sign.

The Rigler sign and air collection overlying the liver are the mot common signs of free air on suphine abdominal view

B) Contrast Radiography

- Contrast radiography using water-soluble diatrizoate meglumine [Gastrograffin] is useful in doubtful cases. In free perforation there is leakage of contrast into the peritoneal cavity.
- Gastrograffin administered contrast is also useful in diagnosis of sealed perforation to plan a conservative management as in the case of
- forme fruste.

Detect free fluid in the peritoneal cavity. The site of bowel preparation can be detected by sonography (e.g.gastric vs duodenl perforation.

Ultra sonograms of the abdomen can also provide rapid evaluation of the liver, spleen, pancreas, kidneys, ovaries, adrenals and uterus, to rule out associated pathology.

D) CT Scans of the Abdomen

This modality can be a valuable investigative tool, providing differential morphologic information no obtainable with plain radiography or ultrasonography.

CT Scans may provide evidence of localized perforation (e.g., perforated duodenal ulcer) with leakage in the area of the gallbladder and
right flank with or without free air being apparent.

2. LAB STUDIES

A) COMPLETE HEMOGRAM:

- Parameters suggestive infection (e.g., leukocytosis),
- Elevated packed blood cell volume suggests a shift of intravascular fluid.

B) WIDAL TEST:

The results of the Widal test should be interpreted taking into account the following.

1) The agglutinin titre will depend on the stage of the disease. Agglutinins usually appear by the end of the first week, so that blood taken earlier may give a negative result.

2) Demonstration of a rise in titre of antibodies, by testing two or more serum samples is more meaningful than a single test. If the first sample

C) ABDOMINAL PARACENTESIS:

Abdominal paracentesis provides useful information in patients with free peritoneal fluid.

Peritoneal lavage. Aspiration of blood, bile or bowel contents is a strong indication for urget laparotomy. On the other hand, infected ascitic fluid may establish a diagnosis in spontaneous bacterial peritonitis or tuberculous peritonitis. Sometimes this paracentesis helps to rule out other conditions simulating gastro-intestinal perforations. E.g., in pancreatitis it is brown colored fluid and in haernorrhagic pancreatitis it is blood stained.

In addition to obtaining red cells and white cell counts, it is important to determine the presence or absence of amylase, bile or bacteria greater than 1,00,000 RBC/cu.m, , 500WBC/cu.mm or detection of bile or bacteria, food fibres or amylase in excess of normal serum values is considered a positive study.

- Blood culture for aerobic and anaerobic organisms.
- Liver function and renal function: Findings may be within reference ranges, when no preexisting disorder is present.

3. OTHERS TESTS

Laparoscopy : Laparoscopy as a diagnostic as well as therapeutic tool in well equiped centers

Laparoscopy improves surgical decision making in patients with acute abdominal pain, particularly when the need for operation is uncertain. and also in laparoscopic closure of perforation in selected cases

DIFFERENTIAL DIAGNOSIS

- Acute appendicitis
- Cholecystitits, biliary colic
- Acute pancreatitits
- Typhoid fever
- Meckel's diverticulum
- Diverticular disease
- Ischemic colitis
- Inflammatory bowel disease

- Colitis
- Acute salphingitis
- Endometriosis
- Pelvic inflammatory disease
- Ovarian torsion

The non-abdominal conditions resembling perforation are

- Myocardial infarction
- Pleurisy
- Spontaneous pneumothroax
- Diabetes mellitus
- Acute porphyria

MANAGEMENT

Divided into conservative

1. Operative management
2. Conservative management

CONSERVATIVE MANAGEMENT

There are several studies advocating non-operative management in selected patients with a successful outcome. The candidates who are tolerating the insult well and in whom perforation seems to have sealed can be managed conservatively. Resuscitation with intravenous fluids naso gastric suction and intravenous antibiotics and H2 blockers resulted in mortality and mobility similar to those of operative management, but hospitalization is prolonged and incidence of subphrenic abscess

is high. If non operative treatment is chosen then the patient will require frequent clinical evaluation, so that operative therapy can be initiated at the first sign of clinical deterioration.

OPERATIVE MANAGEMENT

The goals of operative management are as follows:

- ★ To correct the anatomic problem
- ★ To correct the cause of peritonitis
- ★ To remove any foreign material in the peritoneal cavity that which promote bacterial growth (food, bile, gastric and intestinal secretions

PRE-OPERATIVE DETAILS

RESUSTIATION

The initial priorities are resuscitation and analgesia.

1. Correction of fluid and electrolyte imbalance: Extra cellular fluid losses are replaced by colloids or crystalloids that have an electrolyte composition similar to plasma.
2. Administration of systemic antibiotics and establishing the likely organisms.
3. Judicious use of analgesics
4. Nasogastric suction to empty the stomach and reduce the risk of further vomiting.
5. Urinary catheterization to assess urinary flow and adequacy of fluid replacement.
6. Monitoring of Central venous pressure (CVP) in critically ill and / or

elderly patients, in whom cardiac impairment may be exacerbated by large fluid loss.

OPERATIVE MANAGEMENT

1. Simple closure with dead omental patch.
2. Simple closure with live omental patch.
3. Simple closure with definitive procedure for ulcer.
4. Endoscopic closure of perforated ulcer.
5. Laparoscopic closure of perforated ulcer.
6. Closure with serosal patch

These are the various target oriented operative techniques. All these techniques should be supplemented with thorough peritoneal lavage. Laparoscopic approach holds good in peritoneal lavage permitting irrigation of all comers of the peritoneal cavity.

7. Flank drain and conservative management is a non target oriented technique in patients of poor general conditions.

DEFINITIVE PROCEDURES FOR DUODENAL ULCER PERFORATION

1. Truncal vagotomy with suitable drainage procedure.

Laparoscopic perforation closure, using intracorporeal suturing in a manner identical to open surgery, depending on the experience of surgeon, and complete peritoneal lavage can be done

DEFINITIVE PROCEDURES FOR GASTRIC ULCER PERFORATION

Resection of ulcer and closure

INDICATIONS FOR DEFINITIVE ULCER SURGERY

- ★ Hemodynamically stable patients
- ★ Perforations for less than 24 hours
- ★ No obvious co-morbidity
- ★ Patients with long history of peptic ulcer
- ★ Perforation of an ulcer during antisecretory agent
- ★ Previous ulcer complications.

CONTRAINDICATIONS FOR DEFINITIVE ULCER SURGERY

- ★ Associated medical conditions
- ★ Delay in presentation of more than 24 hours
- ★ Gross abdominal contamination with food.

TREATMENT OF PERFORATED GASTRIC ULCER

But only in selected cases primary definitive operation. A strong case can be made for as primary definitive operation whenever possible in perforated gastric ulcer. Simple closure of a perforated gastric ulcer may be followed by gastric fistula or by post-operative haemorrhage.

Simple closure versus a definitive ulcer operation

"Simple closure of the perforation, together with technical modifications such as the use of an Omental patch, has been the mainstay of surgical treatment of perforation in most centres.

The advantages of this operation are:

a) safety

b) many patients of ulcer perforation may remain symptom free after closure of perforation only, and

c) can be done by even trainee surgeons without direct supervision

The disadvantages are

a) Bleeding from a kissing ulcer

b) Re-perforation due to cutting out of sutures of the friable oedematous tissues.

c) Gastric outlet obstruction after simple closure.

d) Another operation in the future to treat the chronic ulcer and it's complications.

Definitive Surgery

The tendency towards a definitive ulcer operation at the time of perforation came from reports of long terms follow up results after simple closure. Illingworth and colleagues showed that more than half of the patients had a severe relapse of their ulcer disease within 5 years of perforation. A collective review by Jarrett and Donaldson in 1895 cases followed up after simple closure for 1 to 26 years showed

that 2/3rd had subsequent symptoms and that more than l/3rt had subsequent" definitive operation.

Many authors have shown that definitive ulcer operations, including gastrectomy and various forms of vagotomy, can be done with safety at the time of perforation depending on patients general conditions

The indications a definitive ulcer operation at the time of closure of a perforated duodenal ulcer may be classified as definite or relative.

Definite indications are:

a) The presence of a synchronous second ulcer complication.

b) A previous ulcer complications and

c) Perforation of an ulcer during antisecretory treatment.

Relative indications are:

a) Along pre-perforative ulcer history, and

b) A young patient.

Identification of risk factors:

Boey et al (1982) published a clear account from a prospective study of 213 patients with perforated duodenal ulcer and defined three main risk factors:

a) Concurrent medical illness, including cardiorespiratory disease, renal "failure, diabetes mellitus and hepatic pre-coma.

b) Pre-operative shock.

c) Perforation for more than 48 hours (this has subsequently been reduced to 24 hours).

Old age, ulcer history and the extent of peritoneal soiling were not found to be significant factors.

Choice of definitive operation:

If a definitive ulcer operation is deemed an appropriate addition to simple closure of a perforated duodenal ulcer, proximal gastric vagatomy is the procedure of choice. It is difficult to justify other operations, such as gastrectomy or truncal vagotomy with drainage unless technical considerations require their use, because these operations may result in undesirable and unnecessary sequelae for those patients otherwise destined to have no further trouble after perforation.

However, with the advent of H2-recepter antagonists and proton pump inhibitors and H.Pylori eradication therapy an emergency definitive procedure is seldom justifiable even in the presence of a long history. Simple closure of the perforation followed by long term therapy with , ranitidine or omeprazole with documentation of H.Pylori eradication gives good results. Simple closure of a perforated duodenal ulcer in a otherwise healthy patient carries a low mortality rate.

Operation for perforation associated with haemorrhage:

Hemorrhage in association with perforation is usually due to posterior wall "kissing", or penetrating, ulcer. It is often convenient to enlarge the perforation by

converting it into a pyloroplasty with an incision in the long axis of the stomach, so that suture ligation of bleeding point in the posterior wall ulcer can be carried out. The operation may be completed by a truncal vagotomy and transverse closure of the pyloric incision. Any one of the several variations of this technique may be needed to cope with more extensive "saddle" ulcer.

SPECIAL PROBLEMS NECESSITATING SURGERY BY AN EXPERIENCED SURGEON:

Reperforation:

This is particularly dangerous complications and requires partial gastrectomy with gastro-jejunal anastomosis. This duodenal stump may be difficult to close safely and intubation with a large Foley's catheter may be a safer option, thus forming a controlled duodenal fistula rather than risking an uncontrolled fistula. Parentaral feeding and subsequent enteral feeding are important therapeutic measures with in mind it is advisable to feed a fine bore feeding tube into the efferent jejunal loop at the time of the revisional surgery.

Giant duodenal ulcer perforation:

1. Truncal vagatomy and antrectomy with closure of the duodenum with staples or intubating the duodenum.
2. Jejunal patch closure

Perforation of a stomal (gastro-jejunal ulcer):

Treatment of the perforation by closing it with omentum is acceptable,

But revision surgery – excision of perforated segment with fresh anostomosis.

PERFORATIONS OF SMALL BOWEL INCLUDING APPENDIX

The etiological factors for small intestinal perforations are:

INFECTIVE

Bacterial	:	Salmonella typhi, Mycobacterium tuberculosis, Clostridium perfringens, Campylobacter, Yersiniosis.
Fungal	:	Actinomyces
Viral	:	Cytomegalovirus
Parasitic	:	Ascariasis

INFLAMMATORY

Idiopathic, Inflammatory bowel disease, Necrotizing enterocolitis, Ischeamic enteirits, Radiation enteritis.

Traumatic	:	Blunt and penetrating injury
Diverticular disease	:	Meckel's, Jejunal and Heal diverticulitis.
Malignancies	:	Lymphoma, Malignant melanoma.

Drug Induced.

TYPHOID ULCER PERFORATION

Typhoid fever remains endemic in tropical and subtropical countries, causative organism are S. typhi, paratyphi A and B, more common following the onset of monsoon, male to female ration is 3:1.Incidence is becoming less because of early diagnosis and good coverage of antibiotics.

In the first week Peyer's patches becomes hyperemic and hyperplastic, necrosis in the second week. Ulceration in the third week, followed by healing or perforation in the fourth week. The perforation is solitary in 85% of cases.

Clinical features include fever, abdominal pain, tenderness, guarding, rigidity, and electrolyte imbalance. Plain X-ray may show air under ' diaphragm or multiple air fluid levels. A low WBC count before perforation, raises after perforation, Positive blood culture in the first week, positive widal test in the second week, positive stool culture in the third week are diagnostic.

TUBERCULOSIS ULCER PERFORATION

Intestinal Tuberculosis .s a rare cause of perforation. Commonest site is ileocaecal region.

MECKEL'S DIVERTICULUM

It is the remnant of vitellointestinal duct, present in 2% of population situated on the antimesentric border of small intestine, 2 feet from ileocaecal
and usually 2 inches long. The presentations include - severe hemorrhage, Intussusception, Meckel's diverticulitis, Chronic peptic ulceration, Intestinal obstruction.

TRAUMATIC INJURIES OF SMALL BOWEL

Penetrating injuries are mere common than blunt injuries. Traumatic ruptures involving mobile parts of small intestine are multiple. In blunt injuries the mechanisms involved

1. Crush injurybetween vertebrae and anterior abdominal wall.
2. Sudden increase in the intra abdominal pressure.
3. Tear at the junction of mobile and fixed portion of bowel due to deceleration.

Clinical features include features of peritoneal irritation and tenderness at the site of injury, diagnostic peritoneal lavage is of great value in detecting intra abdominal injuries, X-ray may reveal pneumoperitoneum. Operative management involves simple two layer closure if tear is small, resection needed when multiple tears within short segment.

IATROGENIC PERFORATIONS OF SMALL BOWEL

Incidents of small bowel perforation in laproscopy _and Trocar suprapubic cystostomy are encountered rarely. ERCP can cause jejunal perforations in patients who have undergone Billroth II gastrectomy.

COMPLICATIONS OF SMALL BOWEL PERFORATIONS

1) Wound infection
2) Intra abdominal abscess
3) Enterocutaneous / Faecal fistula
4) Portal pyemia
5) Adhesive obstruction
6) Reperforation.

PERFORATIONS OF LARGE BOWEL

AETIOLOGY

Infective

Bacterial	:	Paratyphoid B, Mycobacterium tuberculosis.
fungal	:	Actinomyces
Parasitic	:	Entamoeba histolytica

Inflammatory

Ulcerative colitis

Diverticular Disease:

Volvulus : Injury due to compressed air.

Traumatic : Blunt and Penetrating injuries.

latrogenic : Rigid sigmoidoscopy and colonoscopy.

Malignancy :

TRAUMATIC PERFORATION

Traumatic Perforations of colon and rectum are due to penetrating injuries. The force required to damage the colon is considerable and so it is refractory to blunt injury. Blunt trauma accounts for 5% of colonic injuries. Rectal injuries occur in association with pelvic fractures. In intraperiotneal colonic injury present with signs of peritonitis, Diagnostic peritoneal lavage is helpful in diagnosis. In extrapertioneal colonic injury and in rectal injury diagnosis is difficult. Rectal injury should be considered in all patients with penetrating injury to the perineum and accidental high pressure air introduced from below. Management includes early resuscitation prophylactic antibiotics with the surgical options of (a) Primary closure of low risk colonic injuries, (b) Primary closure with proximal colostomy and (c) Resection and proximal colostomy.

COMPLICATIONS OF COLONIC PERFORATION

1. Faecal peritonitis
2. Abscess formation
3. Enterocutaneous fistula,
4. Anastomotic leak.

POST OPERATIVE MANAGEMENT (General)

- Intravenous replacement therapy: The aim of intravenous replacement therapy is to maintain intravascular volume and adequate hydration of the patient that can be monitored by CVP measurement and urinary output.
- Nasogastric drainage: Nasogastric drainage is continued until drainage becomes minimal. At this stage, the nasogastric tube may be removed.
- Antibiotics: the antibiotics commenced preoperatively are continued unless the results of cultures taken at the time of the operation reveal that the causative organisms are resistant to them.
- The goal of antibiotic therapy is to achieve levels of antibiotics at the site of infection that exceed the minimum inhibitory concentrations for the pathogens present.
- In the presence of intra-abdominal infections, gastrointestinal function is often impaired; therefore, oral antibiotics are not efficacious, and intravenous antibiotics are preferred.
- H2 receptor antagonists or proton-pump inhibitors for a period of 6 - 8 weeks
- A full regime of H. pylori eradication therapy to be started at the end of 8 weeks.

COMPLICATIONS

EARLY COMPLICATIONS

- Renal failure and fluid, electrolyte, and pH imbalance.
- Respiratory complications.
- Wound infection:

i. Wound infection rates correlate with the bacterial load in the peritoneal fluid.

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

- Wound failure (partial or total disruption of any or all layers of the operative wound) may occur early (i.e., wound dehiscence)
- The factors are associated with wound failure are malnutrition, sepsis, uremia, diabetes mellitus, corticosteroid therapy, obesity, heavy coughing, hematoma (with or without infection).
- Multiorgan failure and septic shock
- Gram-negative infections are associated with a much worse prognosis than gram-positive infections, possibly because of associated endotoxemia.
- Localized abdominal abscess
- Entero cutaneous, fistula
- Deep vein thrombosis and pulmonary embolism.

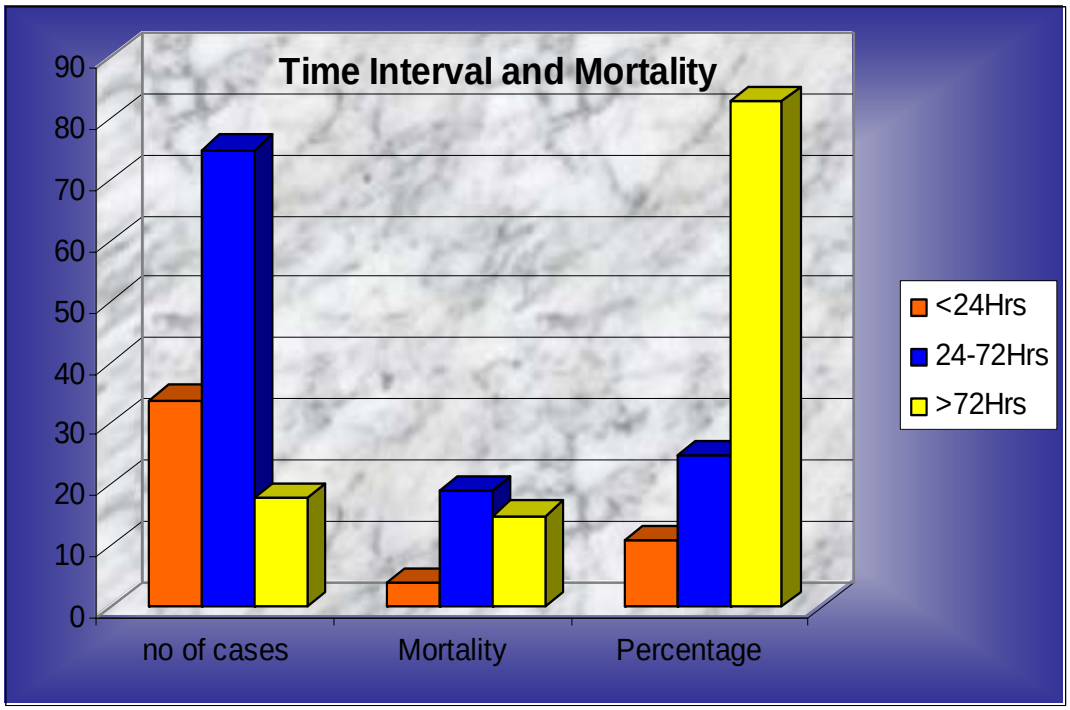
LATE COMPLICATIONS

- Mechanical intestinal obstruction: Mechanical obstruction of the intestine is most often caused by postoperative adhesions.
- Incisional hernia

TIME INTERVAL & MORTALITY

Time Interval in Hrs	No of cases	Mortality	Percentage
<24Hrs	34	4	14.7
24-72Hrs	75	19	25.33
>72Hrs	18	15	83.33

Remarks : Timely intervention crucial factor deciding the prognosis. There is the five fold increase in the mortality among patients without treatment within 24 hours



OBSERVATION

Observation of this study shows Mortality and Morbidity due to peritonitis due to GIT perforation is greatly influenced by timely intervention.

Careful clinical examination along with X ray and USG in all cases will invariably help in arriving at an accurate diagnosis regarding the site of perforation.

Of the 127 cases that were studied the mortality and morbidity of the cases were higher as the ages advanced.

The following factors influence the mortality and morbidity

a. The interval between the occurrence of perforation and initiation of treatment is of prime importance. There is approximately **five fold** increase in the mortality among patients without treatment for 24 hrs compared with that with patients treated within 6 hrs accounted for by true bacterial peritonitis and septicemia which supervene after 12 hrs.

b. Extent of the disease influences prognosis

c. General condition of the patient at the time of presentation.

d. **Age of the patient** - mortality and morbidity higher in older age group

e. **Sex-**Mortality was higher in males than females

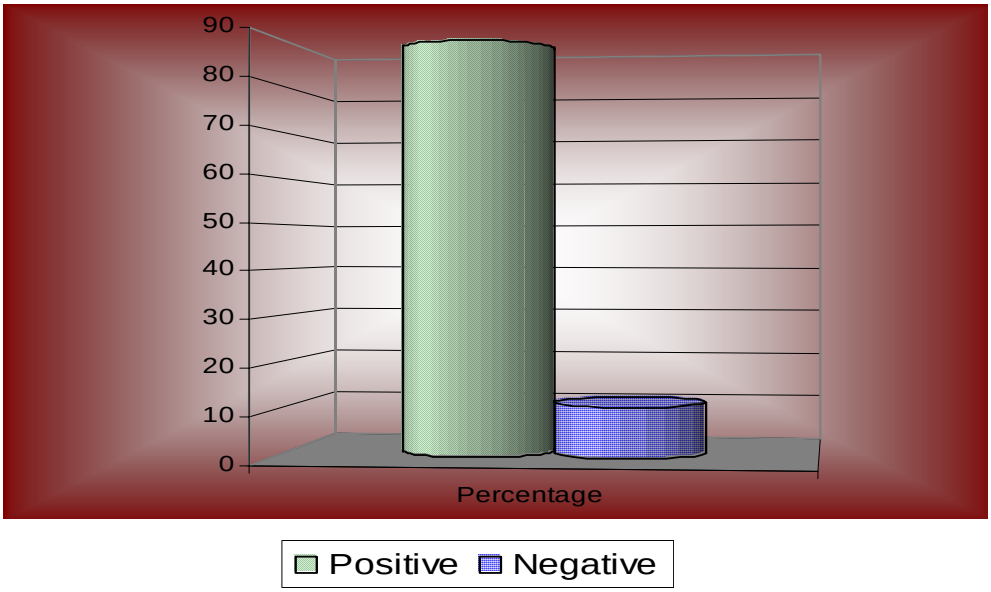
f. Size, site of perforation and type of procedure

PNEUMO PERITONEUM

Pneumo pertoneum	No.of.Cases	Percentage
Positive	117	89
Negative	10	11
Total	127	100

Remarks : High sensitivity in this series

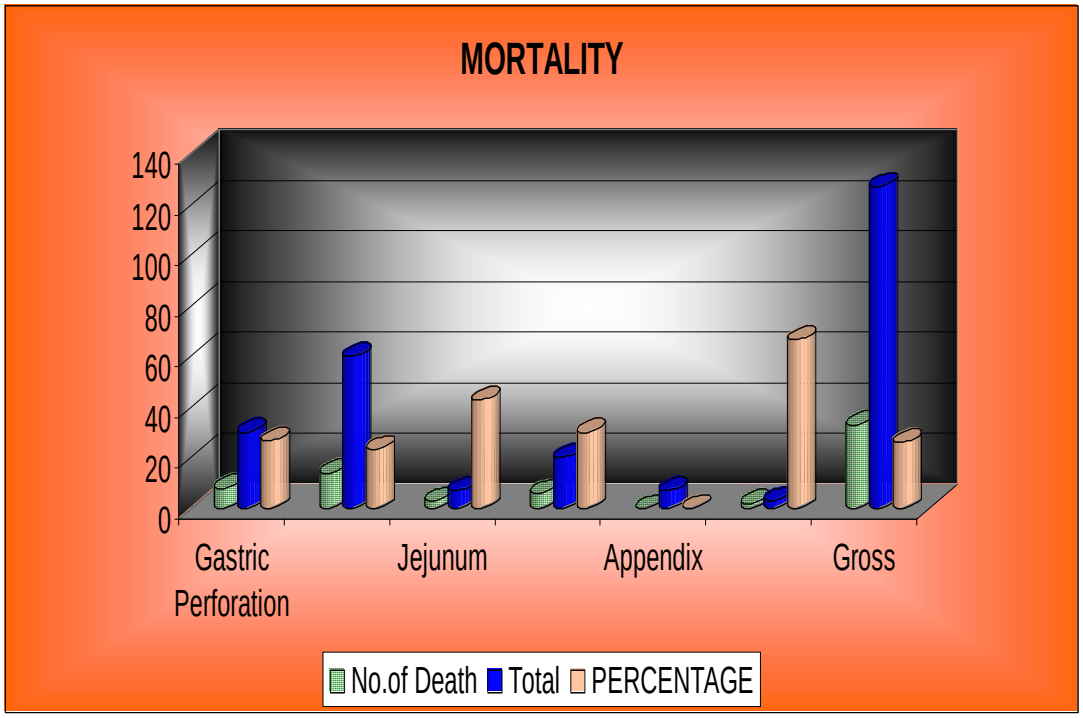
AIR UNDER DIAPHRAGM



MORTALITY (Gross)

	No.of Death	Total	PERCENTAGE
Gastric Perforation	8	30	26.67
Duodenum	14	60	23.33
Jejunum	3	7	42.86
Ileam	6	20	30.00
Appendix	0	7	0.00
Colon	2	3	66.67
Gross	33	127	25.98

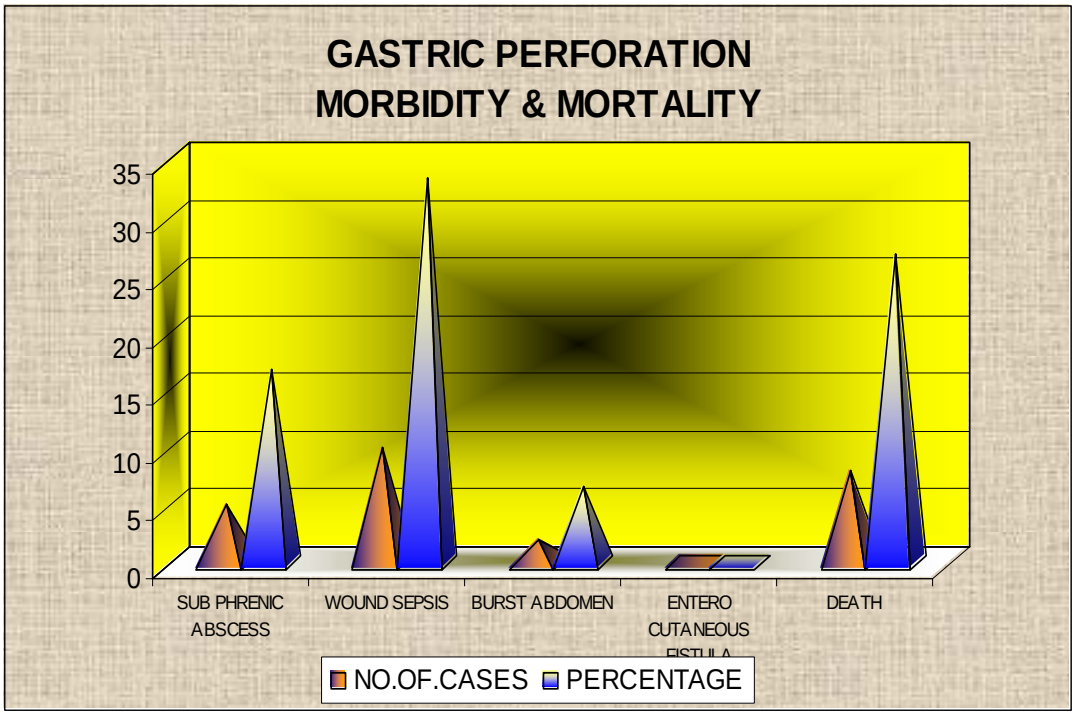
Increased mortality percentage noted in colon and jejunal perforations



GASTRIC PERFORATION

MORBIDITY & MORTALITY

COMPLICATION	NO.OF.CASES	PERCENTAGE
SUB PHRENIC ABSCESS	5	16.67
WOUND SEPSIS	10	33.33
BURST ABDOMEN	2	6.67
ENTERO CUTANEOUS FISTULA	0	0
DEATH	8	26.67



GASTRO DUODENAL PERFORATION

Morbidity and mortality were greatly determined by the time of presentation at the hospital. More the delay more extensive is the peritoneal soiling, more incidence of residual abscesses.

Observation made

(i) There is a drastic decrease in the incidence of complications due to cicatrisation of duodenal ulcer like outlet obstruction. But the incidence of duodenal ulcer perforation has remained the same or increasing. This is mainly due to the increased usage of NSAIDS, Steroids and H.pylori infection

(ii) Evaluation the cases at the time of surgery.

90% of the ulcer were acute (i.e) no evidences of fibrosis or cicatrisation. 10% of the ulcer that perforated showed evidence of fibrosis and cicatrisation at the time of surgery,

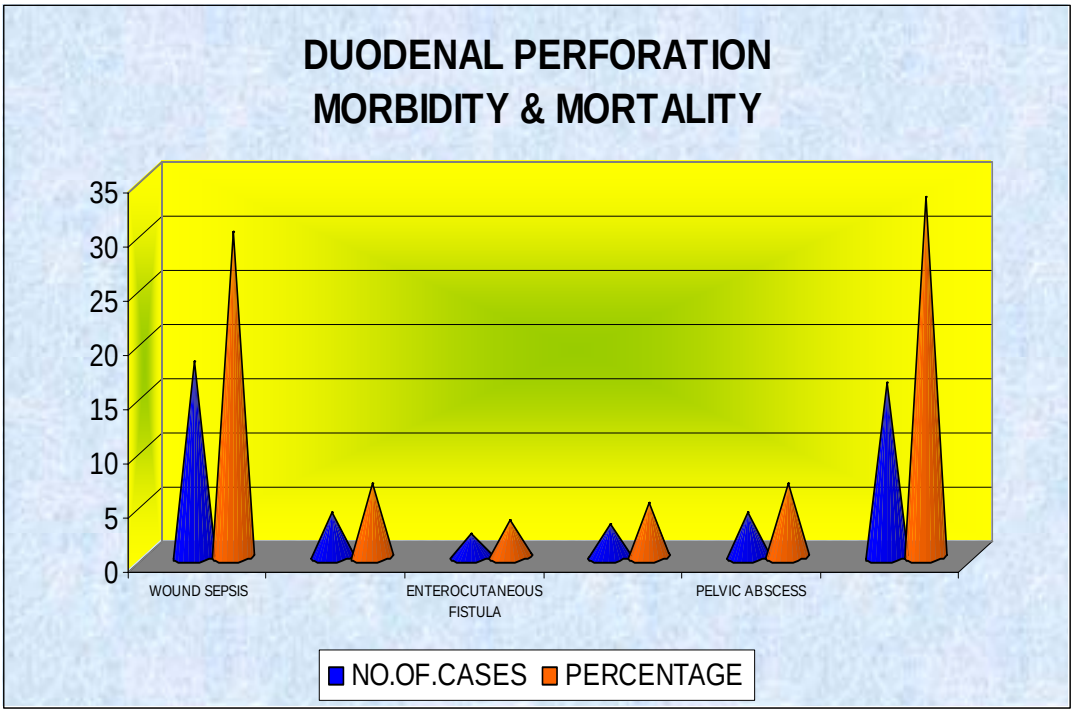
(iii) The incidence of wound infection was lesser in duodenal ulcer perforation closure.

Simple closure of the perforation with the live omental patch was the most efficient method of treating a perforated ulcer.

DUODENAL PERFORATION

MORBIDITY & MORTALITY

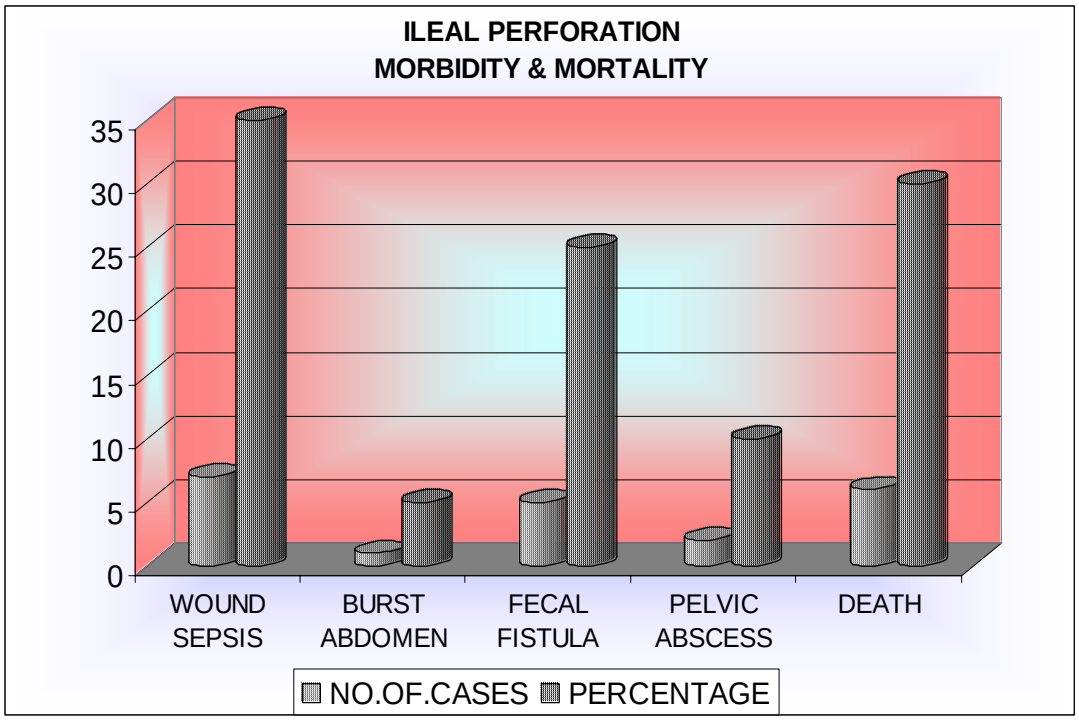
COMPLICATION	NO.OF.CASES	PERCENTAGE
WOUND SEPSIS	18	30
BURST ABDOMEN	4	6.67
ENTEROCUTANEOUS FISTULA	2	3.33
SUBPHRENIC ABSCESS	3	5
PELVIC ABSCESS	4	6.67
DEATH	16	33.33



ILEAL PERFORATION

MORBIDITY & MORTALITY

COMPLICATION	NO.OF.CASES	PERCENTAGE
WOUND SEPSIS	7	35
BURST ABDOMEN	1	5
FECAL FISTULA	5	25
PELVIC ABSCESS	2	10
DEATH	6	30



SMALL BOWEL PERFORATIONS

Incidence of enteric ileal perforations found to be decreasing. Morbidity was higher in small bowel perforation as most of the cases were referred from other hospitals and the time of presentation was delayed leading to extensive peritonitis and soiling.

(i) Most of the perforations were common in the 1st week after the onset of fever when compared to other studies where the perforations were common only in the 3rd week.

(ii) Barring a few cases, all were solitary perforations in the terminal ileum.

(iii) Simple closure of the perforation is more effective.

(iv) Risk of faecal fistula was higher following resection and anastomoses than simple closure. Ileal fistulas fail to respond to conservative management and they mostly required relaparotomy and ileostomy.

(v) Blood Widal was not useful in the post- operative period.

(vi) Ileal perforations were more common than jejunal,

(vii) Morbidity was lesser in cases of traumatic perforation as most of the cases were taken up for surgery immediately when the peritoneal soiling was only minimal. Mortality was mainly due to the associated polytrauma.

(viii) Simple closure of the rent in 2 layers proved effective in all the cases.

CONCLUSION

- Duodenal ulcer perforation was the commonest cause of gastrointestinal perforation with a male preponderance
- More common in the fourth and fifth decade of life
- Smoking and alcohol were the main aggravating factors.
- Perforation was the first manifestation of peptic ulcer disease in a small percentage of patients.
- The role of nonsteroidal anti-inflammatory drugs as the cause of perforation was little in this study group.
- Radiological evidence of pneumoperitoneum could not be established 10% cases.
- Ultrasonogram – useful diagnostic tool to establish free fluid in acute abdomen.
- Simple closure with omental patch with thorough peritoneal toileting was very much effective.
- Definitive ulcer surgery was not warranted in the emergency and treatment with H2 blockers and H. Pylori eradication achieved good control over the disease in the follow up period.

- The prognostic indicators were **early hospitalization**, adequate fluid replacement and absence of co-existing medical illness.

- Gastric perforations were common in the fifth &sixth decade

- The role of biopsy in gastric perforation was established with a case proving positive for malignancy.

- Delayed hospitalization was the major cause of perforation in appendicitis.

- Jejunal perforations were rare and trauma was the single major cause of jejunal perforation.

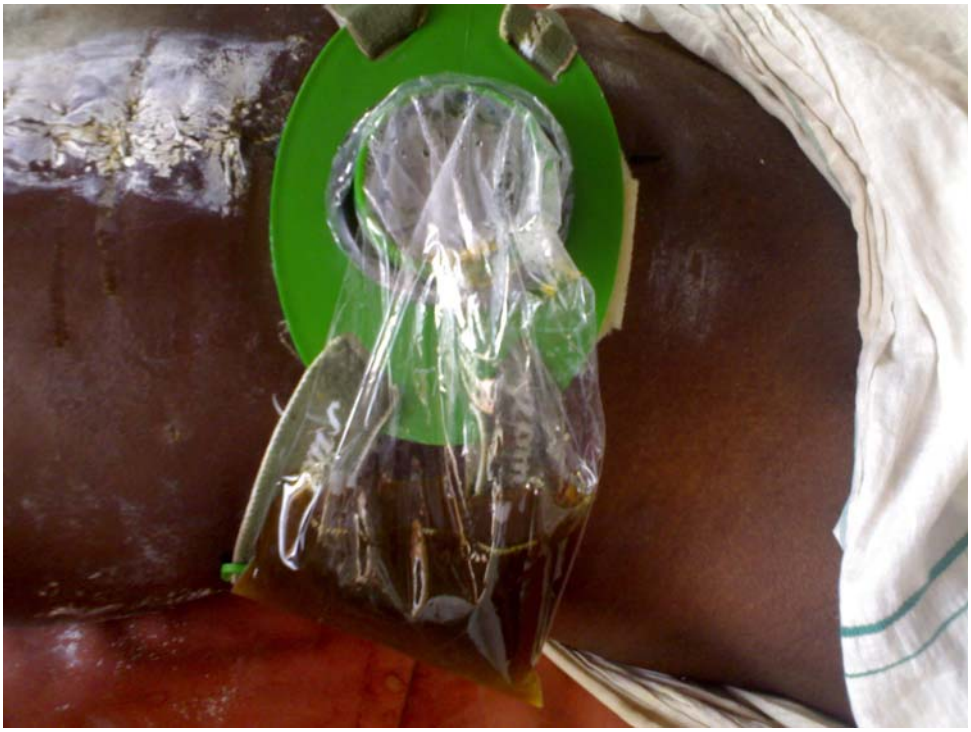
- Closure in two layers was very much effective in small bowel perforations.

- Inspite of recent advances in duodenal perforation - closure by laparoscopy, still simple closure with omental patch is widely practiced in this study group.

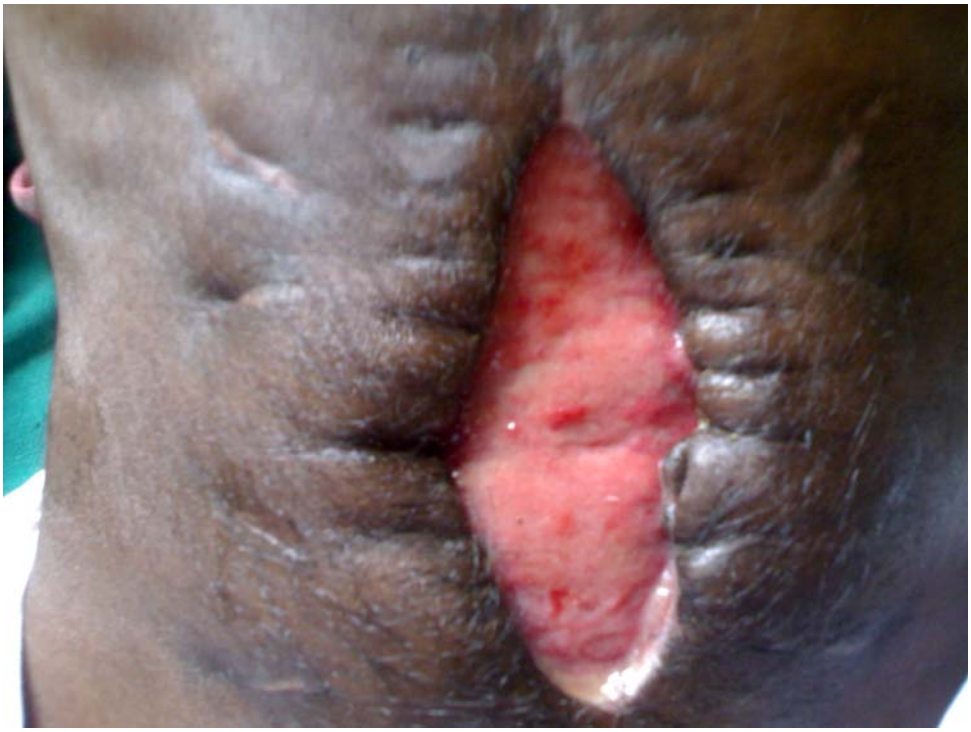
- The most common post-operative complication was wound infection.

- Deaths were due to septicemia , renal failure or cardiac arrest.

- The actual mortality was higher than the mortality in the study group since cases of delayed presentation with shock and septicemia did not warrant anesthesia and were excluded from the study group.

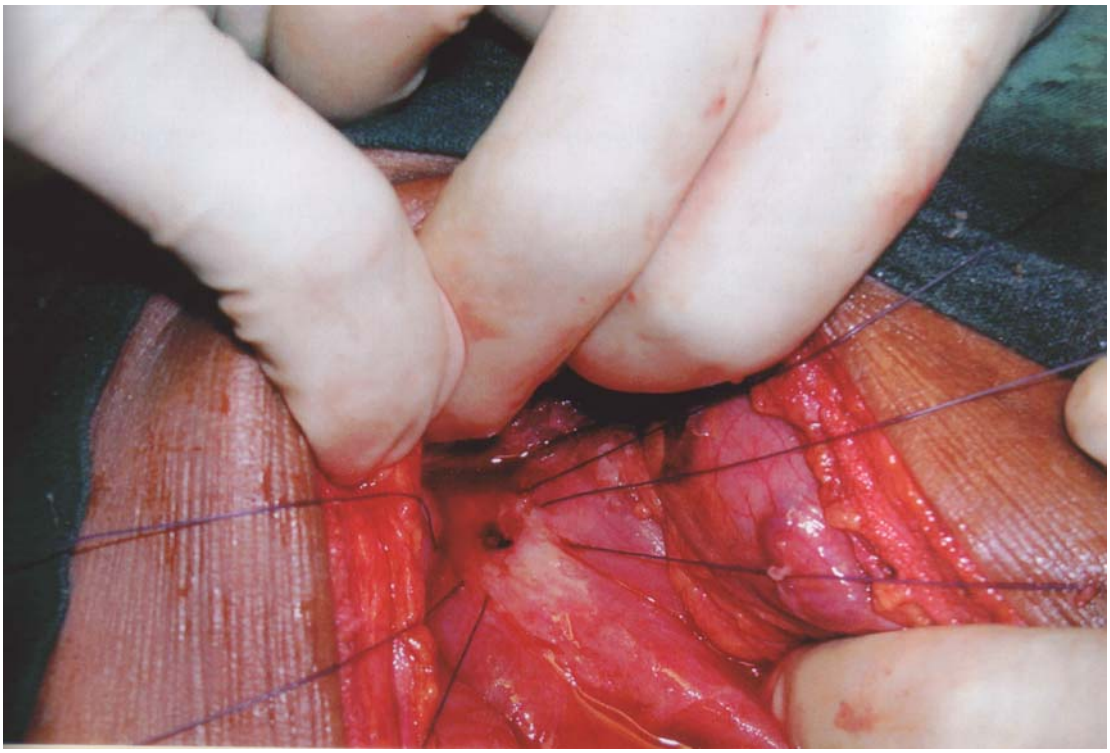


ENTERO CUTANEOUS FISTULA FOLLOWING ILEAL PERFORATION

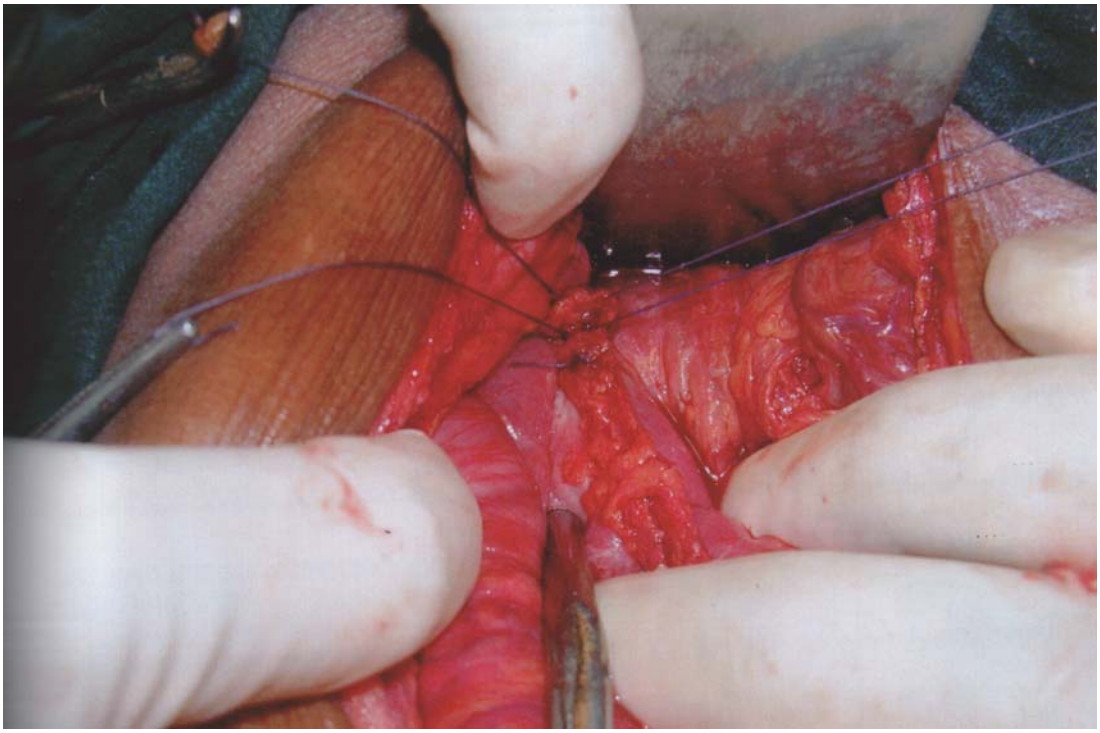


WOUND DEHISENCE

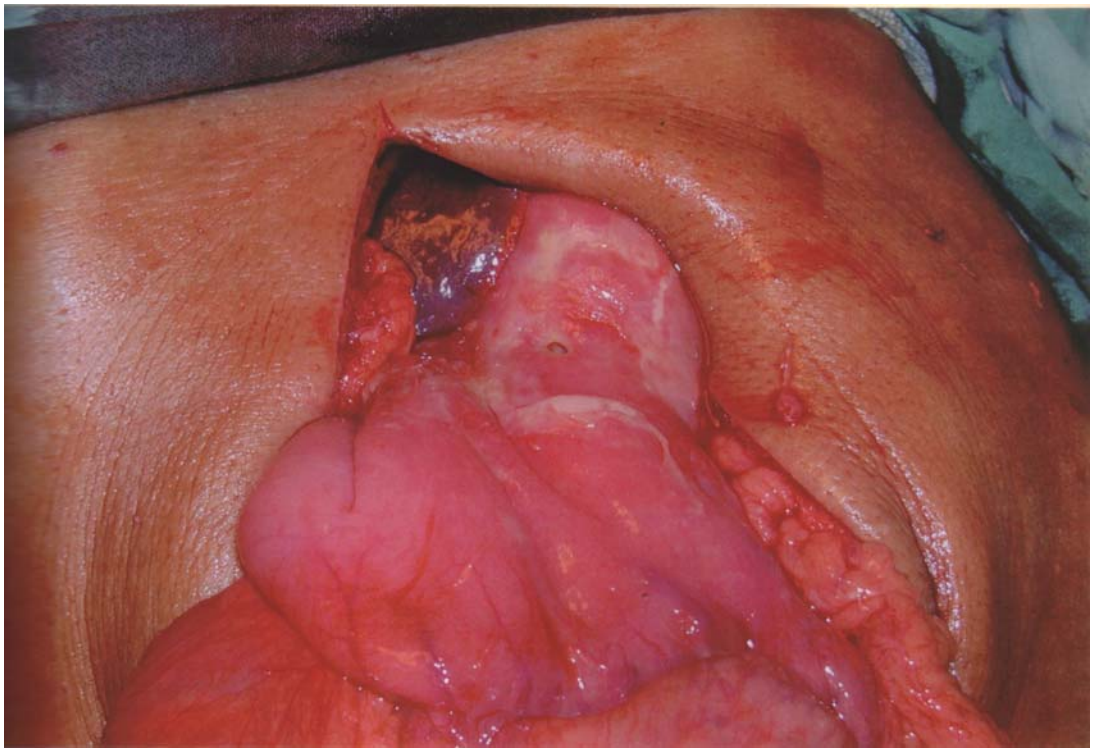
A CASE OF PREFORATIVE PERITONITIS



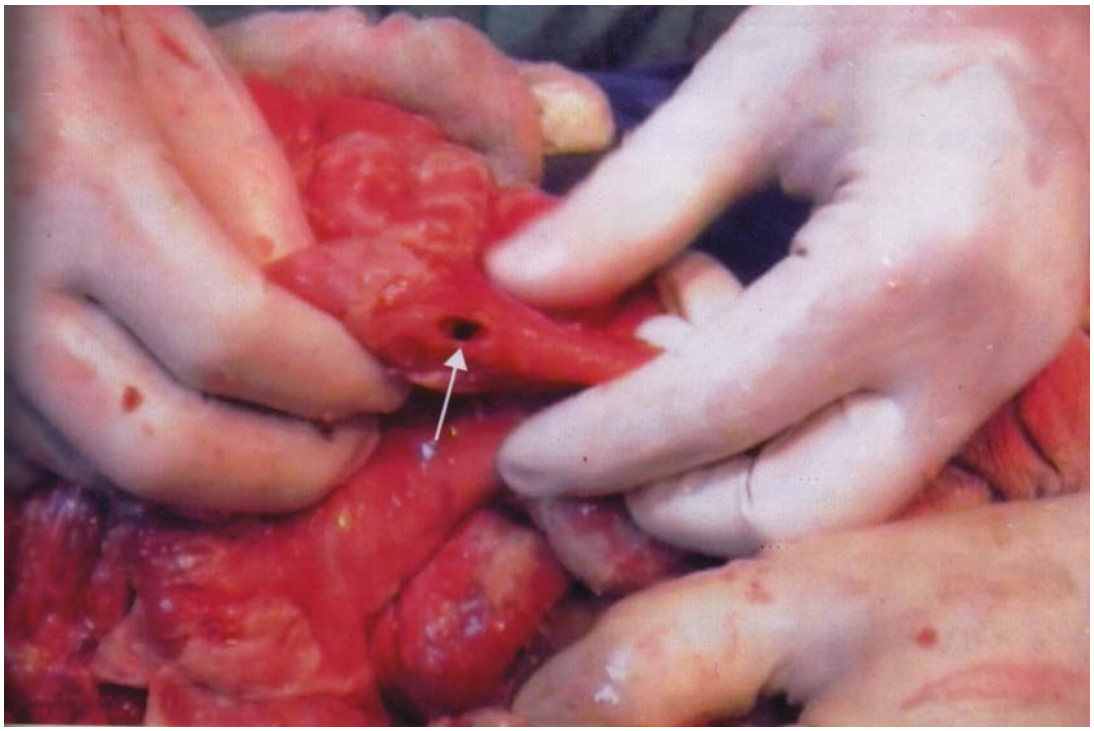
DUODENAL PERFORATION - 3 STICHES OF VIKRYL PLACED



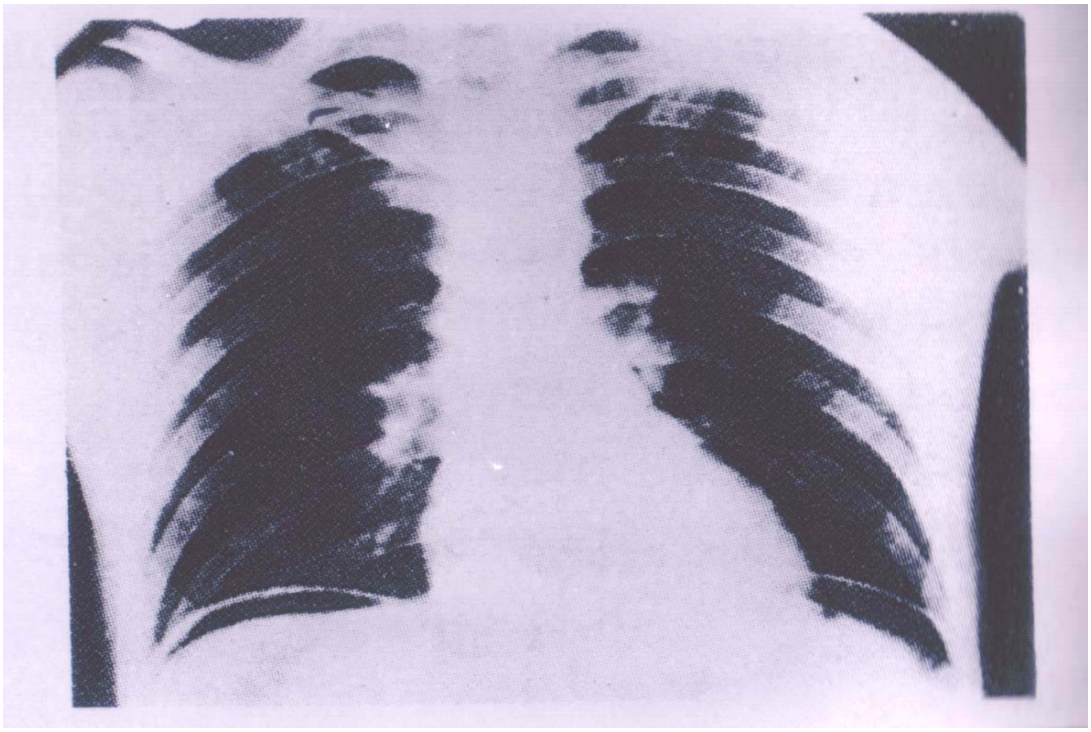
DUODENAL PERFORATION - CLOSURE WITH LIVE OMENTAL PATCH



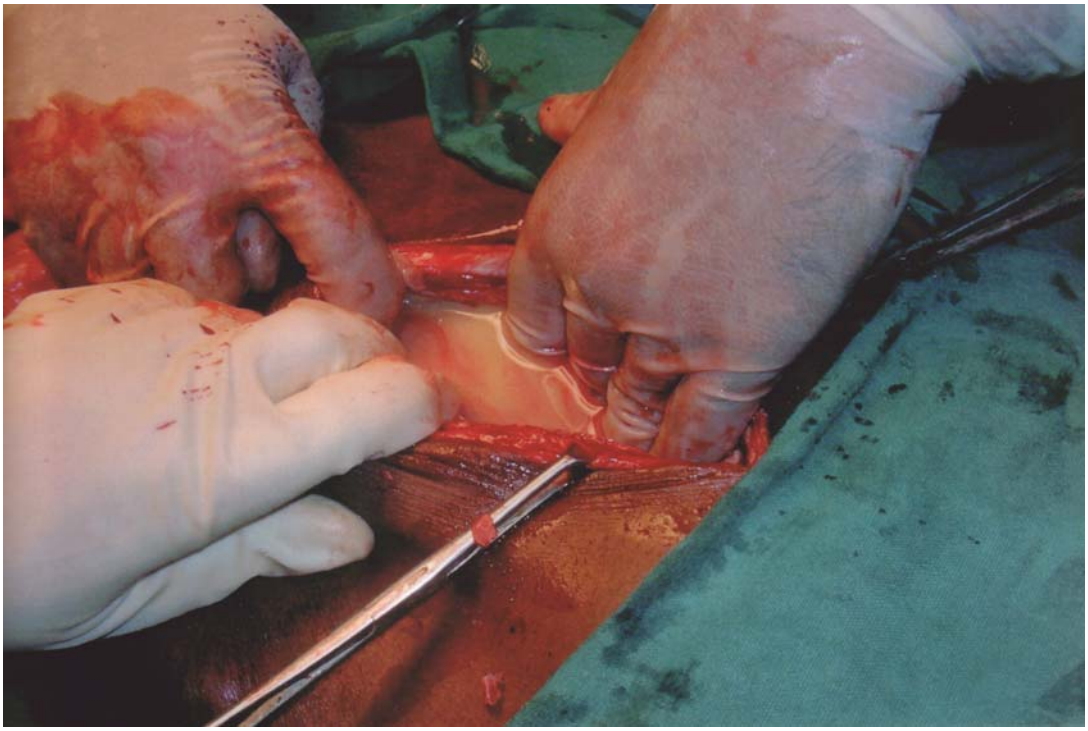
GASTRIC PERFORATION



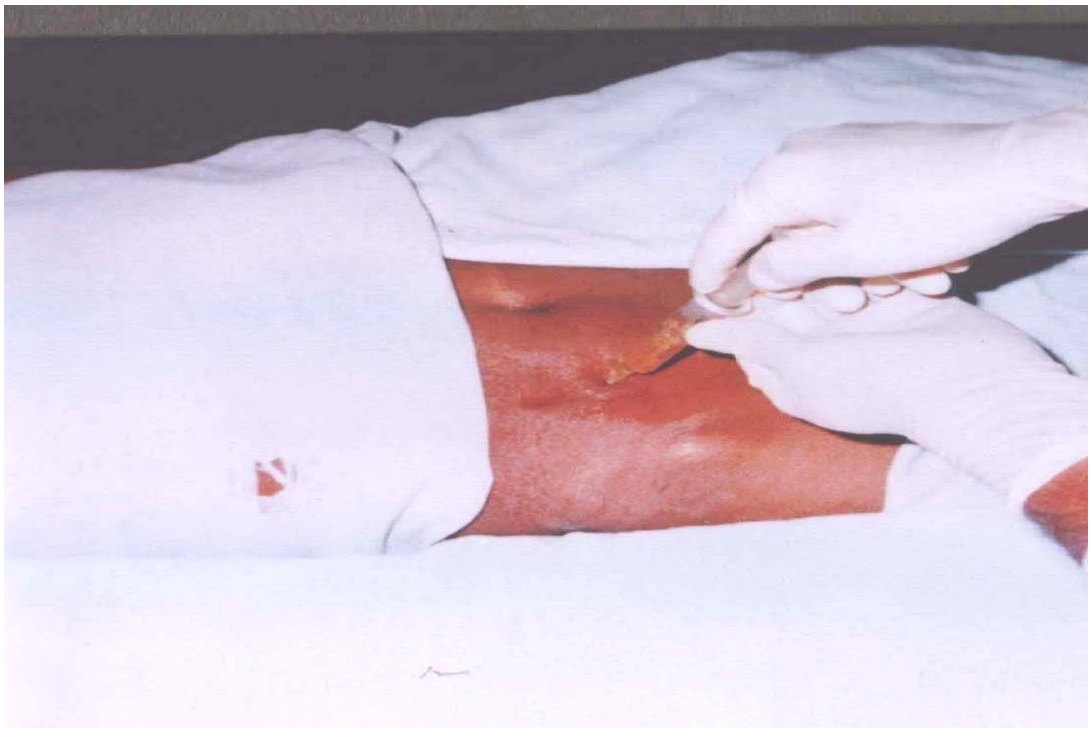
ILEAL PERFORATION



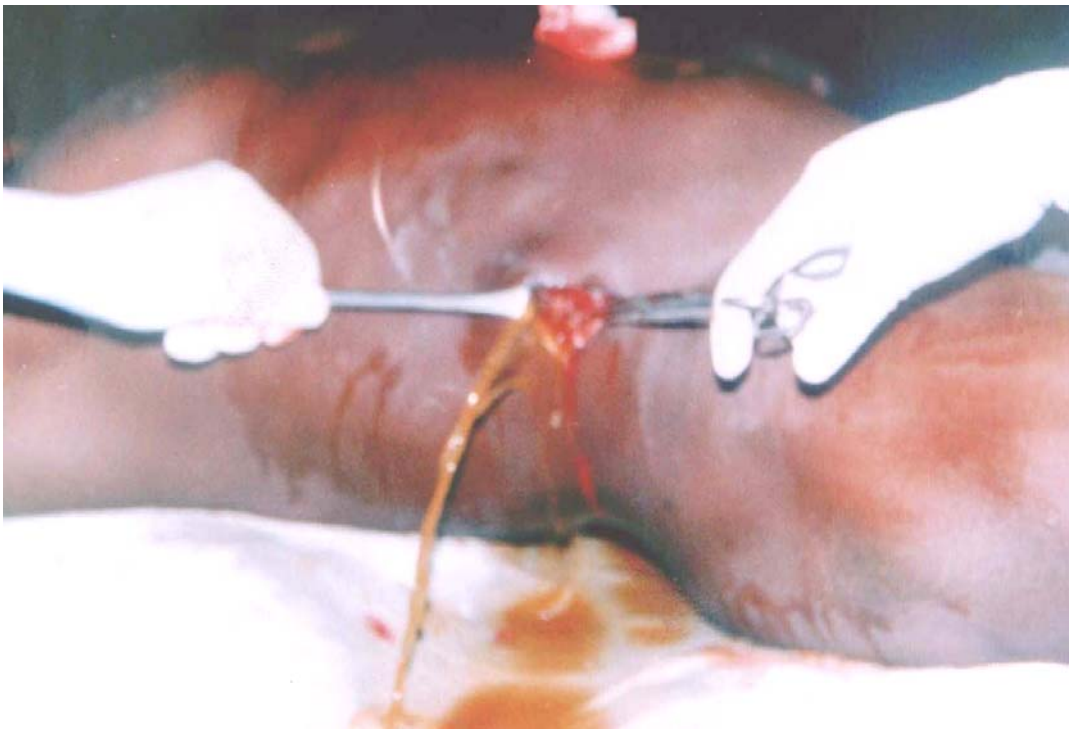
PNEUMO PERITONEUM



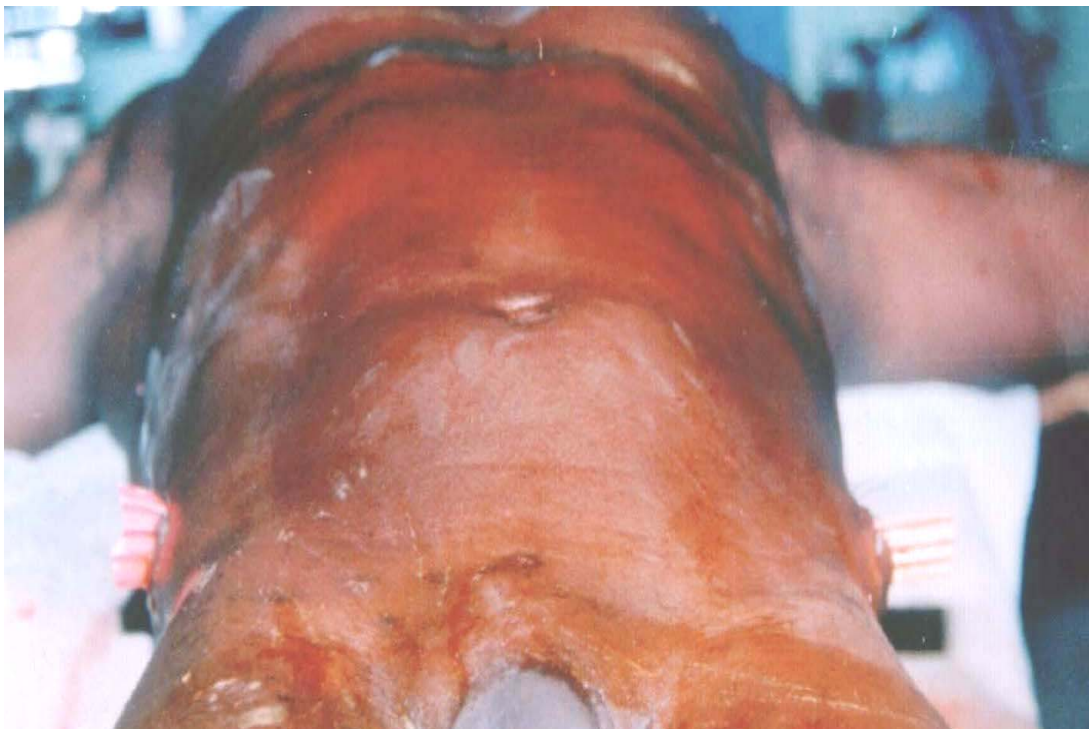
ABDOMINAL CAVITY SHOWING FULL OF PURULANT FLUID



DIAGNOSTIC PERITONEAL ASPIRATION



FLANK DRAINAGE IN PROGRESS



FLANK DRAINAGE COMPLETED

Duodenal Perforations

S.No	Patient Name	Age	Sex	Patients Identity No
31	Pandy	38	M	12048
32	Mariappan	35	M	253214
33	Ramachandran	34	M	254125
34	Saraswasti	26	F	212
35	Ramesh	24	M	1532
36	Kalisamy	40	M	1118
37	Arumugam	44	M	14402
38	Annamalai	24	M	2573
39	Krishnamoorthy	23	M	3991
40	Kadarkarai	65	M	31618
41	Mariappan	40	M	4217
42	Uthaminathan	25	M	4236
43	Palaniyandi	37	M	5123
44	velladurai	47	M	5131
45	Sudalaimuthu	54	M	5974
46	Narayanan	25	M	10797
47	Sudalaimani	35	M	10865
48	Kalathiyan	55	M	13870
49	Raju	31	M	14144
50	Balakrishnan	44	M	14156
51	Shanmugaiah	56	M	12150
52	Soundaram	26	M	20404
53	Chelliah	68	M	20781
54	Palani	60	M	22815
55	Vallivel	42	M	27554
56	Sekar	30	M	27413
57	Kannan	21	M	31049
58	Natarajan	50	M	36315
59	Duraisingh	62	M	36339
60	Sivan	58	M	38904

61	Valliappan	67	M	38921
62	Pathy	54	M	40026
63	Irulandi	50	M	41320
64	Saravanan	26	M	41326
65	Verrapatran	60	M	42479
66	Subramanian	45	M	43260
67	Gopalakrishnan	65	M	463035
68	Velusamy	40	M	46818
69	Krishnasamy	68	M	659
70	Kadermydeen	31	M	1212
71	Mthukutti	65	M	38610
72	Thirumalai	79	M	3236
73	Sivanu	57	M	2980
74	Kamamuthu	55	M	21557
75	Manickam	47	M	25131
76	Palavesam	56	M	9038
77	Ponnudurai	42	M	15430
78	Samidurai	40	M	16521
79	Paramasivam	64	M	16754
80	Ganesan	35	M	16729
81	Karuppasamy	60	M	17769
82	Mahalingam	65	M	19872
83	Natarajan	65	M	20456
84	Nallasivam	35	M	21189
85	Ramakrishnan	60	M	21729
86	Uchimakali	27	M	23726
87	Petchidevar	65	M	27593
88	Paramasivam	70/m	M	29396
89	Ayyappan	30	M	31274
90	Narayanan	52	M	31302

Ileal Perforations

91	Lakshmi	16	F	251721
92	Sabarimuthu	50	M	320
93	Kadalselvam	42	M	436
94	Suresh	23	M	1734
95	Arunkumar	24	M	1736
96	Felica	45	F	256844
97	Panneerselvam	20	M	4670
98	Subramanian	33	M	45012
99	Pappathy	30	F	4975
100	Viyagappan	65	M	38859
101	Mahalingam	15	M	16612
102	Amutha	34	F	33921
103	Petchiammal	35	F	43426
104	Balakrishnan	48	M	44277
105	Subbiah thevar	55	M	1850
106	Vembu	52	M	12316
107	kalpana	23	F	12772
108	Gurusamy	66	M	14243
109	Pattusamy	13	M	14697
110	Mohan	26	M	21560

Jejunal Perforations

111	Natarajan	38	M	12048
112	Jothey	35	F	15216
113	Rakkia	40	M	21432
114	Raja	65	M	14556
115	Vadivel	42	M	2755A
116	Marisweri	45	M	33570
117	chellappa	25	M	261105

Colon Perforations

118	Ayyammal	25	F	261105
119	Thangam	43	F	8146
120	Veerammal	45	F	24490

Appendicular Perforations

121	Ayyappan	38	M	12048
122	Kumar	32	M	27568
123	Velladurai	47	M	51312
124	Jeya	21	F	40512
125	Maharajan	35	M	16782
126	Sankaran	28	M	16659
127	Mahalakshmi	29	F	24612