

**Dissertation**

*On*

**NON TRAUMATIC HOLLOW VISCUS PERFORATION OF  
THE GASTRO INTESTINAL TRACT  
A COMPREHENSIVE ANALYSIS**

**Submitted to**

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## **CERTIFICATE**

This is to certify that the dissertation on “**NON TRAUMATIC HOLLOW VISCUS PERFORATION OF THE GASTRO INTESTINAL TRACT – A COMPREHENSIVE ANALYSIS**” is a bonafide work done by **Dr. C. KOLANDASAMY**, post graduate student in MS, Department of General Surgery, Government Stanley Hospital, Chennai - 1, under my guidance and supervision in fulfillment of the regulations of The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Degree Branch I, (General Surgery) during the academic period from July 2003 to September 2006.

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## INTRODUCTION

Nontraumatic gastrointestinal hollow viscus perforations have received far less attention in the recent medical literature than inflammations, tumoral or traumatic lesions of solid abdominal organs. This is perhaps related to the more standardized management of non traumatic hollow viscus perforation with fewer controversies. Nevertheless, the delayed diagnosis of the hollow viscus injury can be the cause of multiple organ failure. Current data reported by Barie et al showed that sepsis and multiple organ failure are present in 73% of such cases, with reported mortalities as high as 30%. For these reasons, emphasis must be placed on early diagnosis and adequate management so as to optimize results.

In the last few years important advances have been made in diagnostic techniques, imaging technology, use of USG and CT as well as the selective use of laparoscopic techniques for both diagnostic and therapeutic purposes.

In this study, we review highlights of the diagnosis and treatment of non traumatic hollow viscus perforation and the principles of management that have evolved through years.

## AIMS OF THE STUDY

This study on Non traumatic hollow viscus perforation of Gastrointestinal tract is aimed to analyse the following aspects.

- 1) To study the incidence of non traumatic hollow viscus perforation in Government Stanley Hospital, Chennai during the period July 2004 to February 2006
- 2) Evaluation of non traumatic hollow viscus perforation with regard to
  - a. Age and sex
  - b. Causes
  - c. Symptoms and signs
  - d. Sensitivity of investigational modalities in the diagnosis
- 3) Analysis of various causes of morbidity and mortality in non traumatic hollow viscus perforation peritonitis and factors influencing them.
- 4) To analyse the different treatment modalities available and their outcome

## **MATERIALS AND METHODS**

### **SAMPLE SIZE**

63 patients of non traumatic gastrointestinal hollow viscus perforation who were admitted in our surgical unit, at Government Stanley Hospital, Chennai during the period from august 2004 to February 2006 were taken up for the study.

The total number of emergency surgeries performed by our unit during that period were 210; out of which 63 patients were identified to have non traumatic gastrointestinal hollow viscus perforation.

### **INCLUSION CRITERIA**

- 1) Patients with abdominal pain and features of peritonitis, generalized or localized.
- 2) Patients with abdominal pain, whose investigations revealed hollow viscus perforation.

### **EXCLUSION CRITERIA**

- 1) Patient with blunt / penetrating injury of abdomen with signs of hollow viscus perforation clinically and radiologically.
- 2) Patients with abdominal pain but with no features of hollow viscus perforation radiologically (or) intra-operatively.
- 3) Patients who sustained inadvertent iatrogenic perforation during laparotomy.
- 4) Patients with perforations of genitourinary tract like urinary bladder rupture, ruptured ectopic pregnancy, etc.

## METHODOLOGY

The patients included in the study were subjected to a thorough history elicitation and physical examination. They were subjected to undergo relevant investigations like, complete haemogram, urine analysis, biochemical renal function tests with electrolytes, coagulation profile including bleeding time and clotting time, serum amylase (subjected to availability), widal test (in selected cases suspected of complicated enteric fever), blood culture (in cases with sepsis), radiological investigations like plain X-ray abdomen supine, with erect / left lateral decubitus position depending on the condition of patient, with X-ray chest erect PA or supine AP also depending on patient status. Patients were also subjected to ultrasonographic evaluation of abdomen.

After clinical assessment and basic investigations, patients were first actively resuscitated after nasogastric aspiration with intravenous fluids, antibiotics and analgesics. Antibiotics most widely used was the preferred combination of ampicillin, gentamycin and metronidazole intravenously covering the broad spectrum of Gram positive cocci, gram negative aerobic bacilli and anaerobic gram negative rods. Later antibiotics were changed in due course of illness depending on the culture and sensitivity report of the inflammatory peritoneal fluid or blood culture.

After stabilizing the patient initially, the other necessary investigations like basic radiological investigations and special radiological investigations were completed as per necessity and patients were taken up for laparotomy under epidural, spinal or general anaesthesia, depending on the suspected site of pathology and the general condition of the patient. In the majority, midline abdominal incisions were used and the abdominal viscerae inspected carefully for pathology. The site of lesion was located and appropriate surgery performed depending on the pathology made out intraoperatively.

Thorough peritoneal toileting was done with normal saline and the peritoneal cavity was drained. Abdomen was closed in layers.

Post-operatively patients were managed with nasogastric aspiration, i.v. fluids, and antibiotics. Daily patients were monitored and assessed for recovery and complications which were identified and treated appropriately. Patients were discharged after full recovery to normalcy and were followed up for a minimum period of 3 months to a maximum of 1 year depending on the type of surgery performed.

A separate proforma for each patient, containing all the relevant particulars were maintained and reviewed for the analysis.

## REVIEW OF LITERATURE

Perforations are described as disruptions or lacerations of the full-thickness of the wall of the hollow viscera.

The gastrointestinal perforations have many causes. Holes in the wall of gastrointestinal viscerae can be created by blunt/penetrating trauma, iatrogenic injury, inflammatory conditions penetrating the serosa or adventitia, extrinsic neoplasms invading gastrointestinal tract or primary neoplasm that penetrate outside the wall of gastrointestinal viscerae. Here the organs studied in detail, are, stomach, duodenum, jejunum, ileum, appendix, colon, rectum and gall bladder. Oesophageal and gall bladder perforations were not encountered during this study period and hence are not discussed here.

### AETIOLOGY OF GASTROINTESTINAL HOLLOW VISCUS PERFORATIONS:

#### 1) Perforations of stomach and duodenum:

Ulcers	Acute	Stress	Systemic illness
			Curlings
			Cushings
			Septicaemia

NSAIDS

Corrosive ingestions

Chronic

Benign ulcer

Malignant ulcer

Zollinger Ellison  
Syndrome

		malignancy	<p>Carcinoma ulcerative type</p> <p>Leiomyosarcoma</p> <p>Lymphoma following chemotherapy</p>
Iatrogenic	Endoscopic	Procedures	<p>Polypectomy</p> <p>Biopsy of ulcer</p> <p>Cautery of AV malformations</p> <p>Percutaenous endoscopic gastrostomy</p> <p>ERCP</p> <p>EUS with transduodenal biopsy</p> <p>Endoscopy assisted transgastric jejunal feeding tube placement</p>
Iatrogenic	Post surgical		<p>Anastomotic leak from gastric staple / suture lines</p> <p>Vertical banded gastroplasty for morbid obesity</p> <p>Splenectomy</p> <p>Harvest of right gastroepiploic artery for CABG</p> <p>Duodenal stump leakage after gastrectomy</p> <p>Lateral duodenotomy for periampullary procedures</p> <p>Biliary tract surgeries like dissecting fibrotic adherent gallbladder, adherent choledochal cyst.</p>

## Laparoscopic procedures

Veres needle for  
pneumoperitonium

Funduplications

Diaphragmatic hernia repair

Paraoesophageal hernia  
repair

Heller's myotomy

Pyloromyotomy

Cholecystectomy

CBD exploration

Miscellaneous Strangulated paraesophageal hernia

Volvulus stomach

Foreign body ingestion

Oesophageal intubation with gastric overpressure

Trauma – Blunt injury, penetrating injury

## 2. Small intestinal perforations:

Infective

Bacterial

Typhoid

Tuberculosis

Clostridial infections

Campylobacter

Viral

CMV

Fungal

Actinomycosis,

Candidiasis

Inflammatory	Idiopathic Crohn's disease Behcet's syndrome Necrotising enterocolitis Coeliac disease Vasculitis Radiation enteritis SLE
Mechanical	Adhesions Obstructed hernias Volvulus
Traumatic	Blunt injury Penetrating injury
Neoplastic	Angiocentric lymphoma Adeno Carcinoma Ulcers in gastrinoma (ZES) Melanoma
Parasitic	Amoebiasis Ascariasis
Drugs	Steroids NSAIDS Slow releasing K <sup>+</sup> tablets Chemotherapy of lymphoma
Diverticular diseases	Meckel's diverticulitis

Jejunioileal  
Miscellaneous Meconium ileus  
Ingested Foreign bodies

### 3. Appendiceal perforations

Acute appendicitis –

Obstruction of lumen due to

- Lymphoid Hyperplasia
- Helminths
- Faecolith
- Bezoars
- Other Foreign bodies

### 4. Colonic perforations

Congenital

- Hirschsprungs disease
- Anorectal malformations
- Colonic reduplication
- Malrotation

Acquired

- Acute infections
  - Acute Bacillary dysentery
  - Acute campylobacter colitis
  - Amoebic colitis
  - CMV colitis
- Chronic infections
  - Tuberculosis
  - Bilharziasis
  - Chaga's disease

Obstruction	<ul style="list-style-type: none"> <li>Volvulus</li> <li>Malignancy</li> </ul>
Ischaemia	<ul style="list-style-type: none"> <li>Acute necrotising enterocolitis (pigbell/Darmbrand)</li> <li>Radiation enteritis</li> <li>Collagen disorders</li> <li>Post colonic surgery</li> <li>Ischaemic colitis</li> </ul>
Post operative	<ul style="list-style-type: none"> <li>Anastomotic dehiscence</li> </ul>
Iatrogenic	<ul style="list-style-type: none"> <li>Embolisation</li> <li>Drugs – Steroids <ul style="list-style-type: none"> <li>Ergot alkaloids</li> <li>NSAIDS</li> </ul> </li> <li>Endoscopy</li> <li>Barium studies</li> </ul>
Traumatic	<ul style="list-style-type: none"> <li>Blunt</li> <li>Penetrating</li> </ul>
Inflammatory	<ul style="list-style-type: none"> <li>Crohns disease</li> <li>Ulcerative colitis</li> <li>Diverticular diseases</li> </ul>
Neoplastic	<ul style="list-style-type: none"> <li>Carcinoma of large bowel</li> </ul>

## **PATHOPHYSIOLOGY OF GASTROINTESTINAL HOLLOW VISCUS PERFORATIONS:**

Bacterial and chemical contamination of the peritoneal cavity following the perforation of gastrointestinal hollow viscus leads to peritonitis, which is referred as secondary peritonitis (infection arising from an intraabdominal source). The pathophysiology of secondary peritonitis are discussed under local response and systemic response.

### **1) Local response to peritoneal infection:**

- An increase in local blood flow and influx of fluid into the infective foci in peritoneal cavity. Histamine and bradykinin are the main mediators of this response. Depending on the extent of peritoneal insult, fluid volumes of 10 L or more may accumulate into peritoneal cavity leading to massive third-space fluid loss which may result in hypovolemic shock. Initially the inflammatory fluid is transudate, which later becomes exudate due to increased vascular permeability resulting in leaking of Igs, complement factors, coagulation factors, autocoids & cytokines.
- Bacterial phagocytosis – The recruitment and accumulation of large number of leucocytes (mainly neutrophils and macrophages) to the site of inflammation is accomplished by changes in local blood flow as well as increased margination and adherence of WBCs to endothelial and mesothelial cells. These are mediated by bradykinin, anaphylotoxins C3a & C5A, platelet activating factor, TNF, IL-1. By 4 to 6 hours following peritoneal insult, significant neutrophil influx had occurred and is peaked at 8 hrs. These inflammatory mediators also stimulate the recruited WBCs to phagocytose and kill the bacteria by release of lysosomal enzymes.

- Fibrin deposition – under normal circumstances, intact mesothelial cells maintain fibrinolytic activity within peritoneal cavity by secretion of tPA. In the setting of mesothelial injury and active inflammation, local fibrinolytic activity is suppressed due to loss of tPA. Moreover, with high fibrinogen concentrations in these situations, fibrin deposition is increased through intrinsic pathway. Fibrin deposition is further enhanced by release of tissue thromboplastin (Factor III) from mesothelial cells which stimulates extrinsic pathway. The objective of fibrin deposition is to isolate and contain the peritoneal contamination and prevent widespread dissemination. These fibrinous adhesions cause the adherence of loops of intestine and omentum to one another and with parietal peritoneum thus creating a physical barrier against widespread peritoneal contamination.
- Abscess formation : is the culmination of the sequestration process described above. Within the adherent mass of viscera, fibrin and bacterial exudate, liquefaction develops due to release of proteolytic enzymes from WBCs and the action of bacterial exoenzymes. The abscess capsule is formed with organized fibrin and adherent adjacent viscera.
- Peritoneal healing: peritoneum heals rapidly after insult/injury. Rate of healing is independent of size of the peritoneal wound. Within 3 days after injury, the wound is covered by connective tissue cells and by day 5, these new cells resemble mesothelial cells. Following resolution of the inflammation, normal fibrinolytic activity returns as mesothelial cell regeneration occurs and fibrinous adhesions are degraded and removed. However in setting of severe peritoneal injury or persistent infection, filmy fibrinous adhesions are transformed to fibrous adhesions by the in growth of fibroblasts, capillaries and collagen deposition.

## 2) Systemic response to peritoneal infection

- Hypovolemia – due to third space fluid loss.
- Hypovolemia leads to decreased cardiac output and compensatory tachycardia. Systemic Hypotension may also be mediated by potent vasodilators like TNF, IL-1, PAF, Nitric Oxide, leading to decreased peripheral vascular resistance.
- Precapillary shunting occurs in pulmonary and splanchnic circulation leading to peripheral hypoxia.
- Decreased urine output occurs due to hypovolemia and decreased renal blood flow with compensatory RAAS activation.
- ‘Warm shock’ sets in with Tachycardia, fever, oliguria, hypotension and warm extremities.
- Abdominal distension create mechanical restriction to diaphragmatic mobility and decreases ventilation, creating atelectasis. Increased pulmonary vascular permeability also leads to pulmonary oedema, increased work of breathing and hyperventilation with worsening of pulmonary oedema and alveolar collapse, severe hypoxaemia resulting in ARDS.
- Tissue metabolism is increased due to high peripheral catecholamines and cortisol. But peripheral hypoxia leads to increased anaerobic glycolysis leading to lactic acid accumulation and metabolic acidosis.
- Following early depletion of glycogen storage, protein catabolism is augmented in skeletal muscles to release branched chain aminoacids for use by myocytes for energy. Other aminoacids are released into circulation for hepatic gluconeogenesis and for production of acute phase proteins in SIRS. Utilisation of free fatty acids as an energy

source is not efficient in early septic period. Thus severe loss in lean body mass occurs rapidly in sepsis.

## **CLINICAL DIAGNOSIS**

The aphorism “The diagnosis of peritonitis is made by clinical evaluation” remains true even today.

### **Symptoms**

Abdominal pain – almost universally the predominant presenting symptom. The area of onset depends on the area of pathology involved. Pain of fully established peritonitis is constant, burning and aggravated by movement. Extent of pain depends on the area of parietal peritoneum that is inflamed. The pain starts typically at the site of local peritoneal inflammation and later becomes more diffuse as more of the peritoneal surface is involved.

Anorexia

Nausea and possible vomiting

Thirst and Oliguria

### **Signs:**

Fever

Diaphoresis

Tachycardia

Hypotension

Warm extremities

If severe shock is present the patient exhibits hypotension, hypothermia and cold, clammy extremities.

Eliciting of tenderness may best be accomplished by percussion followed by palpation.

In the setting of generalized peritonitis, abdominal tenderness is diffuse but is often maximal in the region where the peritonitis originated.

Bowel sounds are markedly diminished or absent

Abdominal distension due to paralytic ileus

Abdominal wall rigidity

These physical findings may be concealed or obscured in patients administered with analgesics or corticosteroids or patient who are unconscious due to head injury, toxic or metabolic encephalopathy or spinal injury, or in post-operative patients and in patients with lax abdominal wall like multiparous women. In these situations diagnosis usually depend on other diagnostic modalities.

## **LABORATORY INVESTIGATIONS**

### **Complete Blood Count**

Leukocytosis with a left shift (immature neutrophils)

### **Renal function tests**

May show pre-renal azotaemia in late stages of hypovolemic shock.

BUN/Se creatinine ratio	Greater than 20 :1
Urine osmolality	Greater than 500 mosm/L
Urinary sodium	Less than 20 meq/L

### **Electrolyte imbalance**

In late stages of shock

Usually features of metabolic acidosis with high anion gap indicating lactic acidosis.

Usually needs ABG monitoring.

## **Serum Amylase**

Can be raised upto three time the normal in these settings.

## **BT/CT**

Coagulation profile can be severely altered due to disseminated intravascular coagulation in late septic shock with elevated BT & CT. Can be confirmed by measuring serum FDP like d –dimers.

## **Blood culture**

Done in patients with features of frank sepsis.

## **Peritoneal fluid culture**

Results depend on the organ of perforation.

Esophagus : Gram positive cocci + anaerobes

Stomach + Duodenum : usually sterile ; only few lactobacilli

Intestines : proceeding in aboral direction, intestinal flora increases in quantity, diversity and number of anaerobes. Usually polymicrobial with Escherichia coli + Bacteroides fragilis being the commonest combination.

Candida may be found in hospital acquired secondary peritonitis from upper GI perforations.

## **Blood WIDAL**

Done in cases of suspected enteric fever perforations of small bowel.

Following facts must be taken into account while interpretation of WIDAL test is done.

- a) Usually antibodies against O & H antigens appear by the end of first week and they gradually increase till fourth week.
- b) Titre of > 1:100 for O and > 1:200 for H antibodies is usually considered positive.
- c) But results of a single test should be interpreted with caution. Demonstration of a rising titre of antibodies in two or more samples is more meaningful than a single test.
- d) Confirmation with culture of salmonella typhi in blood, stool, urine (or) bone marrow usually helps in proving the aetiological diagnosis.

## **RADIOLOGY IN DIAGNOSIS OF HOLLOW VISCUS PERFORATION**

### **PLAIN RADIOGRAPH**

Plain radiographs are an essential part of investigation of patients presenting with an acute abdomen. Interpretation of these radiographs may present a formidable challenge to the surgeon. When the radiological diagnosis is specific or supports the clinical diagnosis, surgery is often indicated. If immediate surgery is indicated on clinical findings, negative or equivocal radiology should be ignored.

A supine abdomen and an erect chest can be regarded as the basic standard radiographs. A horizontal ray abdominal radiograph, either erect or left lateral decubitus is frequently taken to add information like free intra-abdominal air, air-fluid levels, etc.

The bladder should be emptied before the supine radiograph is taken and the film should include the area from diaphragm to the hernial orifices.

A chest radiograph is essential because chest diseases like pneumonia, pulmonary infarction, aortic dissection or myocardial infarction may mimic an acute abdomen. In addition erect chest radiograph is superior to erect abdominal view for demonstration of free intra-abdominal gas, and it is essential therefore that this film includes

diaphragmatic area. It is also helpful to have a chest radiograph as a baseline because chest complications and subphrenic abscesses are frequent post operative complications in patients with an acute abdomen.

### **Pneumoperitoneum in plain radiographs:**

The presence of free, intra-abdominal gas almost always indicates perforation of a hollow viscus. The commonest cause is perforation of a peptic ulcer.

70% of perforated peptic ulcers will demonstrate free gas, a phenomenon which is almost never seen in case of a perforated appendix or gallbladder.

As little as 1 ml of free gas can be demonstrated radiographically, in either an erect chest or a left lateral decubitus abdominal radiograph, an erect chest film being superior to abdominal radiographs.

Patient should remain in position for 5-10 minutes before the horizontal ray radiograph is taken to ensure that any free gas present has had time to rise to the highest position.

In erect radiograph small amount of gas are easily detectable under the right hemidiaphragm, but on left side it may be difficult to distinguish free gas from gastric fundal gas and colonic gas. A left lateral decubitus radiograph will almost always resolve the problem by demonstrating gas between liver and the abdominal wall.

### **Signs of a pneumoperitoneum on the supine radiograph:**

In 56% of patients with a pneumoperitoneum, the gas may be detectable on the supine radiograph.

#### **i) Right upper quadrant gas**

Almost half the patients with intra-abdominal free gas will have a collection in the right upper quadrant adjacent to the liver, lying mainly in the subhepatic space and the hepatorenal (Morrison's) space and visible as an oval or linear collection of gas.

**ii) Rigler's (double wall) sign**

It is the outlining of a bowel wall by gas within the lumen of the bowel and the free intra peritoneal gas outside the bowel wall.

**iii) Ligament visualization**

Free intraperitoneal air outlines the peritoneal reflections and ligaments leading to visualization of ligamentum teres and falciform ligament, urachus, medial and lateral umbilical ligaments.

**iv) Triangular air**

Air trapped between three adjoining bowel loops or between two bowel loops and the parietal peritoneum.

**v) Cupola's sign** : Air accumulates underneath the central tendon of diaphragm and appears as an arcuate collection of gas with a sharp upper margin and an illdefined lower margin.

**vi) Scrotal air**

In male neonates air may pass into an open saccus vaginalis.

**vii) Football sign**

In neonates with extensive pneumoperitoneum, air collects beneath the entire lateral wall of abdomen, presenting as an oval lucent interphase resembling a football as a whole.

### **Pneumoperitoneum without peritonitis:**

Some patients who present with vague clinical symptoms have unequivocal evidence of pneumoperitoneum in radiographs. However, clinical examination will reveal that there is no evidence of peritonitis or indication for immediate surgery. Some of the causes for such situations are

- i) Silent perforations of a hollow viscus which has sealed by itself (in aged, patient on steroids, unconscious patients).
- ii) Post operative pneumoperitoneum
- iii) Peritoneal dialysis
- iv) Perforated jejunal diverticulosis
- v) Perforated cyst in pneumatosis intestinalis
- vi) Tracking down from a pneumomediastinum
- vii) Stercoral ulceration
- viii) Tubal insufflation tests in females
- ix) Therapeutic embolisation of an intra-abdominal organ

### **Conditions simulating a pneumoperitoneum:**

On first appearances, a number of conditions may be remarkably similar to a pneumoperitoneum and these must be considered in every doubtful case, because an error in interpretation may lead to an unnecessary laparotomy in search of a perforated viscus.

- i) Chilaiditi's syndrome : Interposition of bowel between liver and right hemidiaphragm.
- ii) Subdiaphragmatic fat : Usually distinguished from air by its more lateral situation of its radiolucent line.

- iii) Curvilinear supradiaphragmatic pulmonary collapse.
- iv) Subphrenic abscesses
- v) Diaphragmatic irregularity.

## **RADIOLOGICAL DIAGNOSIS OF ACUTE APPENDICITIS:**

### **Signs of Acute appendicitis on plain abdominal radiograph:**

- i) Appendix calculus (5-60 mm)
- ii) Sentinel loop (dilated atonic ileum containing fluid level)
- iii) Widening and blurring of properitoneal fat line.
- iv) Right lower quadrant haze due to fluid and oedema.
- v) Scoliosis (concave to right)
- vi) Mass indenting the caecum
- vii) Blurring of right psoas shadow
- viii) Gas in appendix (rare)

### **Signs of acute appendicitis on USG:**

Blind ending tubular structure which is non compressible, aperistaltic, > 7mm in diameter

Appendicolith casting acoustic shadow

High echogenicity of surrounding fat

Surrounding fluid in abscess

Oedema of caecal pole

Maximal tenderness over appendix.

### **CT Signs of acute appendicitis:**

Appendix measuring > 6mm diameter

Failure of appendix to fill with oral contrast / air upto its tip.

An appendicolith

Enhancement of appendicular wall with i.v. contrast.

Surrounding inflammatory changes include increased fat attenuation, fluid, inflammatory phlegmon, caecal thickening, abscess, extraluminal gas and lymphadenopathy.

Arrow head sign : Luminal contrast / air in caecum pointing towards the obstructed origin of the appendix (present in 30% cases of appendicitis).

### **Further investigations in cases of suspected hollow viscus perforation:**

Not infrequently, a patient presenting with severe upper abdominal pain has equivocal clinical signs and no free gas is demonstrable on plain radiograph. The diagnosis often rests between an inflammatory condition like acute cholecystitis or pancreatitis and a perforated ulcer. In these cases additional radiological investigations can be used to arrive at a conclusion.

### **Water-soluble contrast study**

Water-soluble contrast (preferably non-ionic) medium about 50ml is given by mouth or injected through a nasogastric tube, with patient lying on his right side. The patient is then examined fluoroscopically or abdominal radiograph repeated after the patient has remained in this position for at least 5 minutes. Perforated ulcers will normally demonstrate evidence of a leak of contrast medium.

### **CT abdomen**

CT is the most sensitive investigation for detection of free peritoneal gas. Small volumes of free peritoneal gas can be seen over the liver and anteriorly in the mid abdomen. Tiny pockets of free gas can also be seen in the peritoneal recesses. CT is the best investigation to diagnose perforation of posterior wall peptic ulcers, which may be evident by small bubbles of air pocket seen trapped near the wall of stomach or duodenum, near the surface of pancreas or in the mesenteries near the duodenal bulb and stomach.

Biliomas, which are the result of perforation in the biliary tree, are best demonstrated by CT, next only to ERCP.

In order not to miss small amount of free gas, the images should be reviewed on 'lung window' settings.

## **MANAGEMENT OF SECONDARY PERITONITIS IN GENERAL**

Secondary peritonitis is the consequence of contamination of peritoneal cavity from an organ within the peritoneal cavity. The majority of these episodes are the result of primary lesions of stomach, duodenum, small intestine, colon, appendix and gallbladder.

The mortality ranges from 10%-40% depending on the disease process and the organ involved. Perforated duodenal ulcer and perforated appendicitis have a mortality rate in range of 0% to 10% whereas those involving small and large intestinal perforations have a mortality rate in range of 20% to 40%.

Once the clinical diagnosis of secondary peritonitis is made rapid institution of both physiologic support and aggressive surgical treatment are imperative .

The primary objectives in the treatment of secondary peritonitis are :

### **i) Resuscitation**

It is an axiom that in all cases of peritonitis, some degree of hypovolemia is present. This is due to the “Third spacing” of extracellular fluid within the peritoneal cavity. The rapidity at which resuscitation is accomplished is dependent upon the degree of hypovolemia and the physiologic status of the patient and also the acuity of the situation. The effectiveness of fluid replacement efforts can be judged by the normalization of pulse rate, blood pressure and mental status. Placement of a urinary drainage catheter is essential since restoration of urine output is a reliable indicator of adequate fluid resuscitation. Invasive peripheral arterial and central venous pressure monitoring catheters should be placed in patients with frank septic shock, advanced age, or in patients with cardiac, pulmonary, renal insufficiency to provide more precised determinations of intravascular volume and cardiac output. Supplemental oxygen may be necessary and in more extreme circumstances endotracheal intubation and mechanical ventilation may be needed to preserve oxygenation. Nasogastric decompression should be done in presence of ileus, to prevent pulmonary aspiration and to reduce abdominal distension and to contain further soiling of peritoneal cavity. Antiacid agents like ranitidine should be administered to prevent stress induced gastric ulceration.

### **ii) Antibiotic therapy**

Antibiotic therapy should be initiated as soon as the clinical diagnosis of peritonitis is obtained, simultaneously with the implementation of resuscitation. The initial selection of antibiotic is empirical. The choice being made depending on

- i) The demonstrated activity of the drug against bacteria presumed to be present upon the level of GIT perforation.
- ii) The bactericidal activity of the antibiotic in the infected tissue.

The microbes generally present in the different parts of gastrointestinal tract have been described previously. It is inferred that presumptive therapy should include coverage for both aerobic gram negative rods and anaerobic organisms. The *E. coli* and *B. fragilis*

combination is the commonest mixed infection found in the peritoneal fluid cultures of secondary peritonitis.

Suggested antimicrobial treatment for the treatment of established secondary bacterial peritonitis are as follows:

Mild to moderate intra-abdominal infection	second or third generation cephalosporin or $\beta$ -lactamase inhibitor combination or monobactam + metronidazole
Severe intra-abdominal infection without renal dysfunction	Carbapenam or Fluoroquinolone +metronidazole or Ampicillin + Aminoglycoside + metronidazole
Severe intra-abdominal infection with renal dysfunction	Carbapenam or Fluoroquinolone +metronidazole

The duration of antibiotic therapy is determined by the clinical circumstances.

When minimal peritoneal soiling are found intra-operatively, then very brief course of antibiotic therapy may be used. One preoperative dose and two subsequent doses post-operatively in a period of 24 hours in sufficient.

In the treatment of established bacterial peritonitis, use of “predetermined” days of treatment should be discouraged. Instead, judgment are made using the clinical indicators like temperature, WBC count, and leucocyte differential count.

### **iii) Surgical management**

Surgical control of the infecting organ is the mainstay of treatment. Operative management primarily should be directed towards the control of the source of contamination. This can be accomplished by closure of perforation, resection of perforated viscus, or exclusion of the affected viscus from the peritoneal cavity. In most instances exploration should be carried out through a midline incision, which affords generous exposure and access to the majority of the peritoneal cavity.

The secondary goal of operative management is to reduce the bacterial inoculum. Standard intraoperative techniques to accomplish this goal include swabbing and debriding fibrin, blood and necrotic material and copious irrigation of the peritoneal cavity. The addition of antibiotic / antiseptic to the irrigant solution has not been shown to decrease the mortality although it may decrease the incidence of wound infections.

### **iv) Continued metabolic support**

Post operative management with intravascular volume correction, electrolyte balance, metabolic support, nutritional support and antibiotic therapy completes the management of secondary peritonitis.

## **SURGICAL MANAGEMENT OF GASTROINTESTINAL HOLLOW VISCUS PERFORATIONS IN DETAIL**

### **PERFORATION OF ULCERS OF STOMACH AND DUODENUM AND THEIR SURGICAL TREATMENT**

#### **Incidence**

Incidence of perforated peptic ulcer is approximately 7-10 per 1,00,000 population per year. Perforation occurs in about 7% of patients hospitalised for peptic ulcer disease and is the first manifestation of the disease in about 2% of patients. It is estimated that, after diagnosis of duodenal ulcer, 0.3% of patients perforate annually in first 10 years. In duodenum the aphorism that “anterior ulcers perforate, posterior ones bleed”, is as relevant today as ever. In 5% to 10% of cases a “kissing

ulcer” may be present on the posterior wall of the duodenum opposite the one that perforates anteriorly. Therefore in a patient presenting with a perforated duodenal ulcer, the presence of significant concomitant haemorrhage should suggest the presence of a “kissing ulcer”. In contrast gastric ulcers may perforate freely through either the anterior or posterior wall.

### **Risk factors for perforation**

A strong association has been observed between the use of NSAIDs and perforation of peptic ulcers. A second risk factor is immunosuppression particularly among transplant patients treated with steroids. Other risk factors include increasing age, chronic obstructive lung disease, major burns, multiple organ system failure.

### **Treatment of Duodenal ulcer perforation**

If a duodenal perforation is found it should be closed with full thickness interrupted 2.0 vicryl or silk sutures with a live omental patch as described by Graham. Following ulcer closure, a decision is made whether to add a definitive acid-reductive procedure. Definitive procedure is indicated in following situations,

- a) Combined gastric and duodenal ulcer, one of which has perforated.
- b) Perforation with pre-existing chronic ulcer symptoms.
- c) Co-existing obstruction with perforation
- d) Co-existing haemorrhage with perforation.
- e) Previous operation for perforated duodenal ulcer
- f) Young patient (<35 years) who have perforated duodenal ulcer
- g) H.pylori treated or known negative patient who have perforation.

The current preferred definitive ulcer operation is parietal cell vagotomy. In case of duodenal obstruction Truncal or selective vagotomy with Weinberg Single layer pyloroplasty (closure with Gambee sutures) is the preferred treatment. If technically feasible ulcer should be excised in course of the pyloroplasty.

A definitive ulcer procedure should not be performed if

- a) The patient is unstable
- b) Perforation is > 24 hrs duration
- c) Gross peritoneal contamination.

### **Treatment of Kissing ulcer**

The duodenum is opened through the anterior perforation for suture control of the posterior bleeding ulcer. An acid reductive procedure is mandatory. Failure to recognise and treat a concomitant posterior ulcer may lead to severe haemorrhage requiring reoperation in the early post-operative period, mortality of which is as high as 50%.

### **Treatment of perforated Gastric ulcer**

In perforated gastric ulcer management options include

- a) Simple closure after four quadrant biopsy
- b) Excision and primary closure
- c) Gastric resection.

Factors influencing operative choice include patient age and general condition, location of ulcer, degree of peritoneal contamination, and presence of malignancy on frozen section biopsy.

For ulcers in distal stomach, antrectomy both removes the ulcer and provides definitive therapy. Benign ulcers in unstable or elderly patients can be treated with simple patch closure after biopsy or excision and primary closure.

Ulcers high on lesser curvature should be excised and closed. If excision not possible, biopsy is taken and perforation closed with live omental patch.

### **Conservative treatment of perforated peptic ulcers**

When patients present late (> 24 hrs after perforation). In this group of patients, non-operative management may be considered if

- a) The patient is haemodynamically stable
- b) Generalised peritonitis is absent
- c) Water soluble contrast examination shows no free leak into peritoneal cavity.

Management of these patients include

- a) Nasogastric aspiration and nil per mouth.
- b) Intravenous fluids
- c) Broad spectrum intravenous antibiotics
- d) Intravenous acid suppressors – H2 blockers with close clinical observation.

Surgery is immediately considered if clinical deterioration occurs. These patient are susceptible to development of subphrenic and subhepatic abscesses, which can be managed with percutaneous drainage with USG/CT guidance.

But caution should be exercised in application of this approach to the elderly patients who are less able to tolerate complications of failure of this approach and hence early operation may be preferable in this age group.

## **PERFORATIONS OF SMALL INTESTINE AND THEIR SPECIFIC SURGICAL MANAGEMENT**

### **1) Crohns disease**

It is a chronic, transmural inflammatory disease of the gastrointestinal tract of unknown cause. Crohns disease can involve any part of alimentary tract from mouth to anus but most commonly affects the small intestine and colon. Most common clinical manifestations are abdominal pain, weight loss and diarrhoea. Complications include intestinal obstruction or localised perforation with fistula formation or free perforation with frank peritonitis.

#### **Localised perforation with fistula formation**

Enterocutaneous fistulas should be managed by excising the fistulous tract along with diseased segment of intestine and performing a primary anastomosis. If the fistula forms between two adjacent loops of diseased bowel, both the involved segments should be excised. If the fistula involves an adjacent normal organ, only the diseased segment and the tract to be resected and defect in normal organ closed simply.

#### **Free perforation**

It is a rare complication in crohns disease, when it occurs, the segment of involved bowel resected and when peritoneal contamination is minimal primary anastomosis performed or else enterostomies are performed until intra-abdominal sepsis is controlled and then return for restoration of intestinal continuity. No attempt should be made to resect more bowel than the involved segment, even though grossly evident disease may be apparent in other parts of bowel.

### **2) Typhoid enteritis**

It is an acute systemic infection of several week duration caused by Salmonella typhi or paratyphi. The pathologic event of typhoid fever are initiated in the intestinal tract after oral ingestion of the typhoid bacillus. These organisms penetrate the small bowel mucosa making their way rapidly to the lymphatics and then systemically. Hyperplasia of the reticuloendothelial system including lymphnodes, liver and spleen

is characteristic. Payers patches in small bowel become hyperplastic and may subsequently ulcerate with complications of haemorrhage or perforation.

Intestinal perforation through an ulcerated payers patch occurs in approximately 2% of cases usually in the third or fourth week of the illness. Typically, it is with single perforation in the terminal ileum and simple closure of the perforation is the treatment of choice. With multiple perforations, which occur in about one fourth of the patients, resection with primary anastomosis or exteriorization of the intestinal loops may be required depending upon the degree of peritoneal contamination.

### **3) Diverticular diseases**

#### **Duodenum**

First described by Chomel, a French pathologist, duodenal diverticula are common, second commonest next to colon. It is present in 1-5% of upper GI radiographic studies. Twice more common in women and more in older patients > 40 years age. Two thirds are found in periampullary region (within 2 cms radius of ampulla) and project from medial wall of duodenum.

Perforation of duodenal diverticula is rare. The treatment of a perforated diverticulum may require procedures similar to those in patients with massive trauma-related defects of duodenal wall. The perforated diverticulum should be excised and duodenum closed with a serosal patch from the jejunal loop. If surrounding inflammation is severe, it may be necessary to divert enteric flow with a gastrojejunostomy. Interruption of duodenal continuity proximal to the perforated diverticulum may be accomplished with staplers. Great care is needed if the perforation is near ampulla of Vater.

#### **Jejunum and Ileum**

Incidence much lower, ranging from 0.1% to 1.% noted in upper GI radiographic studies. Jejunal diverticulum are more common and are larger than ileal diverticulum. These are false diverticula, commoner in

older age group, multiple and occurring from mesenteric border of bowel.

Perforation of the diverticulitis is a rare complication. When encountered resection with reanastomosis is the preferred treatment. In diffuse peritonitis, after resection enterostomies are done deferring primary anastomoses.

### **Meckels diverticulum**

First reported by Hildanus in 1598 and later described in detail by Johann meckel in 1809, meckels diverticulum is the most common congenital anomaly of small intestine, occurring in 2% of population. It is usually located on antimesenteric border of ileum 45 to 60cms proximal to ileocaecal valve and results from incomplete closure of the omphalomesenteric or vitellointestinal duct.

Diverticulitis accounts for 10% to 20% of symptomatic presentations and is more common in adults. Progressive of diverticulitis may lead to perforation and peritonitis. Treatment of a symptomatic meckels diverticulum should be prompt surgical intervention with resection of the diverticulum and transverse closure of the bowel or resection of the segment of ileum bearing the diverticulum and primary anastomoses.

#### **4) Small bowel ulcerations**

Small bowel ulceration are relatively rare and may be attributed to crohns disease, typhoid fever, tuberculosis, lymphoma and ulcer associated with gastrinoma, drug induced ulcers mainly NSAIDs.

Perforation is a complication necessitating operative intervention. The treatment is segmental resection and reanastomosis.

#### **5) Ingested foreign bodies**

Ingested foreign bodies that could lead to subsequent perforation are swallowed accidentally by children/adults or sometimes by mentally deranged. These include glass & metal fragments, pins, needles, toothpicks, fish bones, broken razor blades, etc. For vast majority of

patients, treatment is observation, which can be followed with serial radiographs if swallowed object is radio-opaque.

If abdominal pain, tenderness, fever occurs indicating perforation, immediate laparotomy and surgical removal of the offending object are indicated.

## **6) Radiation Enteritis**

Radiation therapy is commonly used as adjuvant therapy for various abdomino-pelvic cancers. Surrounding normal tissue such as small intestinal epithelium may sustain severe acute and chronic deleterious effects. Serious late complications are unusual if the total radiation dose is  $< 4000$  cGY : Morbidity increases with dosages  $> 5000$  cGY.

Acute effects are self limiting consisting of diarrhoea abdominal pain and malabsorption.

The late effects are due to damage to the submucosal blood vessels with progressive obliterative arteritis, submucosal fibrosis, resulting eventually in thrombosis and vascular insufficiency. This may produce necrosis and perforation of the involved intestine.

Radioprotective drug amifostine is currently used to protect normal cells from radiation injury.

If occurs, perforation of intestine, should be treated with resection and reanastomosis.

## **7) Tumours of small intestine**

Benign tumors of small intestine do not lead to intestinal perforation usually.

It is the malignant tumors which lead to intestinal perforation more commonly. Perforation usually occur in about 10% of lymphomas or sarcomas of small bowel.

Adenocarcinomas account for 50% of small bowel malignancy but perforation is a rare complication here.

Malignant GIST which arise from mesenchymal tissue constitute approximately 20% of small bowel malignancy, more common in jejunum and ileum, usually > 5 cm size. Free perforation may occur as a result of haemorrhagic necrosis in large tumour masses.

Malignant lymphomas may occur primarily or as a part of systemic disease. Primary small bowel lymphomas account for one third of all gastrointestinal lymphomas. More common in ileum. Usually a B cell MALToma variety, GIT lymphoma may also be of T cell variety as in those associated with celiac disease.

Perforation may complicate lymphomas in 25% of patients. The treatment of adenoma and lymphomas of small bowel is wide resection including regional lymphnodes. For GISTs, segmental bowel resection is enough and wide margins and extensive lymphnode dissections are not necessary. Adjuvant radiotherapy and chemotherapy provide best survival rate for lymphomas and not for adenocarcinomas. Adjuvant treatment with imatinib mesylate (inhibitor of CD117 tyrosine kinase) in GISTs are under trial with promising early result.

## **8) Post traumatic injuries of small bowel**

Simple suture of wounds should be carried out whenever possible. Very small perforation can be closed by a single purse-string suture of lembert type. Larger wounds are repaired by two layer of sutures. If edges are ragged or bruised they may be excised and the wound closed transversely in two layers to prevent lumen narrowing. Areas of bruising on the gut wall without perforation should be infolded by lembert sutures. Resection and anastomoses is advisable when there are multiple injuries confined to one segment of gut or when laceration or bruising is extensive or if the blood supply of the gut is destroyed or endangered by associated mesenteric injury.

Drainage of peritoneal cavity must be provided in all cases.

## **COLONIC PERFORATION AND ITS SPECIFIC MANAGEMENT**

## **1. Ischaemia**

In New Guinea, Pig bell causes patchy intestinal gangrene due to necrotizing alpha and beta toxins of *Clostridium perfringens*. A similar form of enteritis necroticans is seen in Germany termed Darmbrand. Acute necrotizing enterocolitis may be ischemic, infective or obstructive. Other ischaemic causes of perforation include post transplantation ischemic necrosis, radiation necrosis, drug induced ischemia, collagen disorders, arteritis, intravascular thrombosis and post-aortic surgery, therapeutic embolisation and spontaneous ischemic colitis.

### **ii) Acute and chronic infection**

Perforation may complicate acute bacillary dysentery; campylobacter colitis, amoebic colitis, CMV and pseudomembranous colitis. TB, bilharziasis and Chaga's disease may occasionally be complicated by perforation.

### **iii) Inflammatory**

#### **Ulcerative colitis**

Toxic megacolon is the most common cause of faecal peritonitis in ulcerative colitis. But perforation can also occur due to stercoral ulcers or steroid therapy. Occasionally perforation can occur from malignancy complicating colitis.

#### ***Crohn's disease***

Despite the recognition of fulminating colitis and toxic megacolon in Crohn's disease, perforation is relatively uncommon. Free perforation is more common in small bowel.

### **iv) Diverticular diseases and stercoral perforation**

Intra abdominal sepsis is a common complication of diverticular disease, which may be localized forming a mass or abscess. Inadequate localization of a pericolic abscess may result in purulent peritonitis.

Stercoral perforation is due to ischaemic necrosis from solid hard faeces, causing an area of ulceration in the wall of the bowel. Clinically it may be difficult to distinguish among these.

**v) Neoplastic**

Perforations complicating malignancy of large bowel may be due to the growth itself having penetrated into the local structures and may therefore be complicated by an abscess. Such cases are nearly always locally advanced tumours.

Alternatively the perforation may be proximal to an obstructing carcinoma, resulting in ischaemic necrosis of caecum, where the ileo caecal valve is competent. Such proximal perforation may be more favourable in terms of curing the underlying malignancy.

But perforation of the tumour per se is more common than the proximal perforation due to distal obstruction.

**vi) Radiation injury**

Intra abdominal sepsis from colonic perforation is less common than injury to small bowel from external beam irradiation. Free perforation of rectum is rare unless endocavity uterine implants have been used. The most common sites of necrosis in large bowel are the caecum and sigmoid since both sites are mobile and more be in close proximity to uterus.

**vii) Obstructions**

Perforations may occur proximal to obstructive lesions other than malignancy like radiation strictures, crohns disease, idiopathic megacolon. Perforation may also complicate caecal or sigmoid volvulus. The incidence of perforation in sigmoid volvulus is only 10%. Much less common than in caecal volvulus. Despite this the mortality is usually over 50% in sigmoid volvulus perforations

### **viii) Trauma and other causes**

Blunt colonic injury is rare, occurs in less than 5% of blunt abdominal injury victims. Diagnosis of such injuries is usually made at the time of laparotomy for other injuries. The most frequent sites involved were the more mobile parts of colon – sigmoid, ascending and transverse colon in decreasing frequency.

Penetrating injury to colon are usually due to gun shot or stab injury. A detailed knowledge of ballistics is not required to treat these patients. Projectiles with great velocity produce extensive injury and multiple perforations due to blast waves.

Stab injuries to the abdomen and flanks without peritoneal signs pose a special situation. This is one of the few circumstances in trauma surgery in which laparoscopy is useful to determine whether there has been perforation into peritoneum. Stab wounds are generally associated with fewer other injuries and less faecal contamination than gunshot wounds.

Iatrogenic injuries to colon can occur after many procedures like colonoscopy, barium enema, laparoscopy. Perforation from colonoscopy may arise from excessive pressure on colon wall secondary to loop formation especially in a diseased colon. Because of the force involved in such manoeuvres, these injuries are often large tears on antimesenteric border. During colonoscopic polypectomy, perforation may result from snare injury or from cautery injury. These injuries are often small. Perforations during barium enema occur either from trauma from enema tip or rarely due to over distension of colon.

Other reasons for colonic injury / trauma is insertion of foreign bodies, compressed air, etc.

### **Management of the colonic perforation**

If there is necrotic bowel, malignancy, underlying colitis the perforated segment must be removed. In case of ulcerative colitis a total colectomy may be necessary.

In case of perforation due to distal obstruction, the proximal perforation is exteriorized and the obstructive lesion resected either immediately or at a later stage.

It is unwise to attempt an anastomoses in the presence of faecal peritonitis, even if protected by a stoma after on table colonic lavage.

Resection with construction of an end colostomy and either closure of distal bowel or exteriorization as a mucous fistula is still the safest method of treatment.

A policy of peritoneal drainage and proximal diversion by colostomy is not to be recommended as this does not protect against continued faecal contamination from a distal perforation.

In iatrogenic intra-peritoneal endoscopic perforations where there is minimal delay in diagnosis with good mechanical bowel preparation, there may be grounds for conservative treatment. Primary suture of an endoscopic perforation protected by a temporary proximal stoma may be an alternative strategy if there is extensive peritoneal contamination or delay in diagnosis.

Primary suture may be indicated in early laparotomy for stab injuries, where as in gunshot injuries the treatment depends on the extent of other visceral damage, degree of contamination, nature of colonic injury and the time between injury and operation.

## **APPENDICEAL PERFORATION AND ITS MANAGEMENT**

The overall rate of perforated appendicitis is 25.8% and hence immediate appendectomy has long been the recommended treatment of acute appendicitis for the known risk of progression to perforation. Children younger than 5 years of age and patients older than 65 years of age have the highest rate of perforation (45 & 51% respectively). It has been suggested that delays in presentation are responsible for the majority of perforated appendices. There is no accurate way of determining when an appendix will rupture prior to resolution of the inflammatory process.

Appendiceal rupture occurs most frequently distal to the point of luminal obstruction along the antimesenteric border of the appendix. Rupture should be suspected in the presence of fever greater than 39°C (102°F) and a WBC count > 18000/mm<sup>3</sup>. In the majority of cases, rupture is contained and patients display localised rebound tenderness. Generalised peritonitis will be present if the walling off process is ineffective in containing the rupture.

In 2 to 6% cases, an ill-defined mass will be detected on physical examination. This could represent a Phlegmon (matted loops of small bowel adherent to adjacent inflamed appendix) or a periappendiceal abscess.

The ability to distinguish acute, uncomplicated appendicitis from acute appendicitis with perforation on the basis of clinical findings is often difficult but it is important to make the distinction because their treatment differs.

CT Scan is beneficial in this setting.

## **Management**

Phlegmon and small abscesses can be treated conservatively, with intravenous antibiotics.

Well localized abscesses can be managed with percutaneous drainage with USG / CT guidance.

Complex abscesses should be considered for surgical drainage. If operative drainage is required, it should be performed by an extraperitoneal approach with appendectomy reserved only for cases in which appendix is easily accessible. Otherwise interval appendectomy after 6 weeks following the acute event is the classical recommendation, for those patient treated non-operatively or with simple abscess drainage.

Generalised peritonitis needs a laparotomy with drainage of abscess cavities and appendectomy with peritoneal lavage and drainage.

## OBSERVATIONS OF THE STUDY

**TABLE 1**

**Incidence of non traumatic hollow viscus perforation in Govt. Stanley Hospital, Chennai**

Total No. of emergency surgeries performed	210
Total No. of Non traumatic hollow viscus perforations encountered	63 (30%)

30% of emergency surgeries performed in our unit were for the treatment of non traumatic hollow visceral perforation peritonitis.

**TABLE 2**

<b>Sex</b>	<b>No. of patients</b>
Male	50 ( 79.4 %)
Female	13 (20.6 %)

M : F ratio = 3.85 : 1

**TABLE 3**

**Incidence of non traumatic hollow viscus perforation in relation to age group, sex of the patient**

<b>Age Group</b>	<b>No.of Patients</b>	<b>%</b>	<b>Male</b>	<b>%</b>	<b>Female</b>	<b>%</b>
13-20	7	<b>11.1</b>	5	<b>71.4</b>	2	<b>28.6</b>
21-30	15	<b>23.8</b>	12	<b>80</b>	3	<b>20</b>
31-40	16	<b>25.4</b>	9	<b>56.2</b>	7	<b>43.8</b>
41-50	10	<b>15.8</b>	9	<b>90</b>	1	<b>10</b>
51-60	11	<b>17.5</b>	11	<b>100</b>	0	<b>0</b>
> 60 years	4	<b>6.4</b>	4	<b>100</b>	0	<b>0</b>
Total	63	<b>100</b>	50	-	13	-

**TABLE 4**

**Evaluation of relative incidence of non traumatic hollow viscus perforation to the anatomical site of Gastrointestinal tract involved**

	<b>Total No.of Cases</b>	<b>%</b>	<b>Male</b>	<b>%</b>	<b>Female</b>	<b>%</b>
Stomach	4	<b>6.3</b>	4	<b>100</b>	0	<b>0</b>
Duodenum	26	<b>41.3</b>	26	<b>100</b>	0	<b>0</b>
Ileum	6	<b>9.5</b>	4	<b>66.7</b>	2	<b>33.3</b>
Appendix	26	<b>41.3</b>	15	<b>57.7</b>	11	<b>42.3</b>
Colon	1	<b>1.6</b>	1	<b>100</b>	0	<b>0</b>
<b>Total</b>	<b>63</b>	<b>100</b>	<b>50</b>		<b>13</b>	

**TABLE 5**

**Evaluation of relative incidence of various causes of GIT non traumatic hollow viscus perforation**

	<b>Aetiology</b>	<b>No. of Cases</b>	<b>%</b>
Stomach	Peptic ulcers	4	6.3
Duodenum	Peptic ulcers	26	41.3
Ileum	Typhoid ulcers	3	4.7
	Tuberculosis	2	3.2
	Adhesive obstruction	1	1.6
Appendix	Acute appendicitis	26	41.3
Colon	Carcinoma colon	1	1.6
<b>Total</b>		<b>63</b>	<b>100</b>

**TABLE 9**

**Different treatment modalities followed for non traumatic hollow  
viscus perforation peritonitis**

<b>Stomach</b>	<b>No. of Cases</b>	<b>%</b>
Simple closure	0	<b>0</b>
Graham omental patch closure with peritoneal toiletting	4	<b>100</b>
Simple or patch closure with definitive surgery	0	<b>0</b>
<b>Total</b>	<b>4</b>	<b>100</b>
<b>Duodenum</b>		
Simple closure	0	<b>0</b>
Graham omental patch closure with peritoneal toiletting	25	<b>96.2</b>
Patch closure wit GJ	1	<b>3.8</b>
Simple or patch closure with definitive surgery	0	<b>0</b>
<b>Total</b>	<b>26</b>	<b>100</b>
<b>ileum</b>		
Simple closure	0	<b>0</b>
Simple closure with omental onlay	0	<b>0</b>
Resection and anastomoses	6	<b>100</b>
Exteriorisation of small bowel	0	<b>0</b>
<b>Total</b>	<b>6</b>	<b>100</b>
<b>Appendix</b>		
Appendicectomy	18	<b>69.2</b>
Appendicular abscess drainage (Extraperitoneal)	8	<b>30.8</b>
<b>Total</b>	<b>26</b>	<b>100</b>
<b>Colon</b>		
Resection of perforated segment with primary anastomoses	1	<b>100</b>
<b>Total</b>	<b>1</b>	<b>100</b>
<b>Total Cases</b>	<b>63</b>	

**TABLE 10****Mortality rates in non traumatic hollow viscus perforation**

<b>Organ involved</b>	<b>No. of cases</b>	<b>No. of deaths</b>	<b>Case fatality rate</b>
Stomach	4	0	0
Duodenum	26	2	7.7
Ileum	6	1	16.7
Appendix	26	0	0
Colon	1	0	0
<b>Total</b>	<b>63</b>	<b>3</b>	<b>4.8</b>

## DISCUSSION

The present study was undertaken at the Government Stanley Medical College and Hospital, Chennai which is a tertiary referral centre for all elective and emergency surgeries. During the study period between August 2004 and February 2006 a total of 210 emergency surgeries were performed in our unit. A total of 63 patients were operated upon for non traumatic hollow viscus perforation during the above mentioned period. A systematic analysis of the presentation, management and outcome were performed and the results tabulated.

Table 1 shows the incidence of non traumatic hollow viscus perforation in Govt. Stanley Hospital, Chennai. 30% of emergency surgeries performed in our unit were for the treatment of non traumatic hollow visceral perforation peritonitis.

Table 2 shows the sex distribution of the study population. Males constituted 79.4% of the study group with a sex ratio of 3.85 : 1. This trend of increased incidence among males was noted irrespective of the age group or the site of perforation.

Table 3 shows the data on non traumatic hollow viscus perforation in relation to age group and gender of the patient. The least and highest age of non traumatic hollow viscus perforation operated upon during the study period was 13 yrs and 70 years respectively. Among males the incidence of non traumatic hollow viscus perforation predominated in the age group between 21 and 40 years consisting 49.2% of the total. A steady decline in the number of cases was noted above the age of 40 years. Among females the incidence was highest in the age group between 31 and 40 years. Moreover the incidence was distinctly uncommon below 20 yrs and above 40 yrs.

Table 4 evaluates the relative incidence of non traumatic hollow viscus perforation to the anatomical site of Gastro intestinal tract involved. The most common sites of perforation were the duodenum and the appendix, each consisting of 41.3% of the study group. Stomach (6.3%), ileum ( 9.5%) and the colon (1.6%) were other sites of perforation encountered. No cases of non traumatic hollow viscus perforation involving the esophagus, gall bladder and the jejunum were encountered in the present study. Among females, appendicular

perforation was the commonest etiology closely followed by ileal perforation. However no cases of duodenal, gastric or colonic perforation were noted among females.

Table 5 shows the relative incidence of various etiologies for non traumatic hollow viscus perforation. Peptic ulcers were the only cause identified for non traumatic hollow viscus perforation in the stomach and the duodenum and similarly appendicitis was the only etiological factor in perforations of the appendix. Perforations in the ileum were due to typhoid ulcers (4.7%), tuberculosis (3.2%) and adhesive obstruction (1.6%). The only case of colonic perforation was caused by malignancy of the colon.

Table 6 shows the symptomatology and signs of non traumatic hollow viscus perforation peritonitis. Abdominal pain was the clinical presentation in all patients irrespective of the site of perforation and age of presentation. The clinical features of anorexia, nausea and vomiting were inconsistent and varied widely in all patients and correlated poorly to the site of perforation. All patients with appendicular, colonic perforations and most patients with ileal perforations (83.3%) had fever at presentation. The presence of dehydration correlated well with the presence of generalized peritonitis which was more frequent in perforations of the stomach, duodenum, ileum and colon. Perforations of the appendix tended to manifest as localized peritonitis with dehydration, hypotension and oliguria being less common in this subgroup. Obliteration of liver dullness was noted in all cases of gastric and colonic perforations and was distinctly absent in appendicular perforations.

Table 7 shows the distribution of radiological signs in plain radiographs of abdomen and chest in non traumatic hollow viscus perforation peritonitis. On radiographic evaluation of the abdomen, air under the diaphragm was noted in all cases of stomach and colonic perforations and most cases of duodenal perforations (92.3%). However, this was not a reliable finding in perforations of the ileum (66.7%) or the appendix (0%). The observations were similar with regard to the presence of diffuse ground glass appearance on plain X ray abdomen erect view. Evidence of localized ileus was most common in patients with perforations of the appendix (73.1%).

Table 8 shows the various post operative complications in patients of non traumatic hollow viscus perforation peritonitis. Wound infection was the most common complication that was encountered post operatively (55.6%). It occurred most frequently following an appendicular perforation (65.4%), followed by duodenal (57.7%), ileal (33.3%) and gastric perforation (25%). The patient with colonic perforation did not develop post operative wound infection.

Renal failure was noted in 36.5% of patients most frequently occurring in patients with colonic (100%) and duodenal (65.4%) perforations.

Burst abdomen and enterocutaneous fistula complicated the post operative recovery of duodenal ulcer perforation in one patient (7.7%) each. Perforations at other sites did not lead to either of the two complications.

Intra abdominal abscess developed in 25% of patients with gastric perforation and 23.1% of patients with duodenal perforation, the most frequent site of abscess formation being sub phrenic.

The incidence of respiratory complications were variable being highest in colonic perforation (100%), followed by duodenal perforations (65.4%).

Septicaemia occurred most frequently in duodenal perforations (57.7%) followed by ileal perforations (33.3%). Perforations of the stomach, appendix or the colon were not complicated by septicaemia.

Table 9 shows the treatment modalities followed for non traumatic hollow viscus perforation peritonitis. All patients of peptic ulcer perforations of the stomach and 96.2% of perforations of the duodenum were treated by Graham's omental patch closure. None of the patients of gastric or duodenal ulcer perforation were subjected to a definitive anti ulcer surgery, while only one patient with a duodenal ulcer perforation was subjected to a gastrojejunostomy. Resection and anastomosis was done in all patients with ileal and colonic perforation irrespective of the cause. Only 69.2% of patients with appendicular perforation were subjected to appendicectomy while the remaining 30.8% were treated by extraperitoneal drainage due to the concomitant presence of appendicular abscess.

Table 10 shows the mortality rates in non traumatic hollow viscus perforation. The overall case fatality rate due to non traumatic hollow viscus perforation was 4.8%. The case fatality rate was highest at 16.7% for ileal perforation followed by 7.7% for duodenal perforation. All patients of gastric, appendicular and colonic perforations survived without sequelae following surgery.

## SUMMARY AND CONCLUSION

This study, on non traumatic gastro intestinal tract hollow viscus perforation was conducted on 63 patients admitted to our General Surgical unit at Govt. Stanley Hospital, Chennai, during the period from August 2004 to February 2006 and the following conclusions were made

- 1) Non traumatic hollow viscus perforations of the gastrointestinal tract were more common among males and in the age group between 21 and 40 years.
- 2) The most common causes of gastrointestinal tract non traumatic hollow viscus perforation were duodenal perforation due to peptic ulcer disease and appendicular perforation due to acute appendicitis.
- 3) Abdominal pain was the most common presenting symptom and the incidence of other clinical symptoms and signs were variable. Presence of generalized peritonitis correlated well with the degree of dehydration.
- 4) Wound infection was the most common post operative complication encountered in these patients with secondary peritonitis.
- 5) The case fatality rate was highest for ileal perforation followed by duodenal perforation. The mortality due to perforations in the other sites of the GI tract was uncommon.

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