Dissertation

On

HOLLOW VISCUS PERFORATION OF GASTRO INTESTINAL TRACT

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CERTIFICATE

This is to certify that **"HOLLOW VISCUS PERFORATION OF GASTRO INTESTINAL TRACT"** is a bonafide work done by **Dr. S. KARTHIKEYAN** Post Graduate Student in MS, Department of General Surgery, Government General Hospital, Chennai - 3 under my guidance and supervision in fulfilment of regulations of The Tamilnadu Dr. M.G.R. Medical University for the award of M.S. Degree Branch II, (General Surgery) during the academic period from March 2003 to February 2006.

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INTRODUCTION

Traumatic and nontraumatic gastrointestinal hollow viscus perforations have received far less attention in the recent medical literatures than inflammations, tumoral or traumatic lesions of solid abdominal organs. This is perhaps related to the more standardized management of 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 ranging 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 hollow viscus perforation and the principles of management that have evolved through years.

HISTORICAL DATA

According to Lister, the earliest case of acute perforation of a peptic ulcer was recognized in 1070. The patient was Lady Henrietta Anne, Duchess of Orleans and daughter of Charles I of England, who was having chronic abdominal pain. She suddenly developed agonizing pain in the abdomen with signs of peritonitis and died after 9 hours. The autopsy revealed perforated gastric ulcer.

Perforation of Appendix was first described by Jeen Fernal in 1554.

Credit of presenting first Duodenal ulcer perforation has gone to Hamburger in 1746.

Heusner pioneered the simple closure technique of perforated Gastric ulcer in 1892.

The first reported successful closure of perforated duodenal ulcer was by Dean in 1894.

Christopher Rawlinson gave accurate description of peritonitis following perforated gastric ulcer in 1927.

In 1929 Cellan Jones developed the technique of using live omental support in closure of duodenal ulcer perforation. Roscoe Graham in 1937 described the technique of closure of duodenal ulcer perforation with a free omental patch.

In 1973 Budhiraja S.N. et al. studied 117 cases of gastrointestinal perforations and found that perforations of peptic ulcer and perforations of appendix were the commonest among them and overall mortality rate in that series was 30% .

AIMS OF THE STUDY

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

1) To study the incidence of Hollow viscus perforation in Government General Hospital, Chennai during the period July 2004 to June 2005.
2) Finding out the incidence of hollow viscus perforation in relation to age group, sex of the patient.
3) Evaluation of relative incidence of various causes of hollow viscus perforation.
4) To analyse the relative incidence of various symptoms and signs of the hollow viscus perforation peritonitis.
5) Evaluation of various investigating modalities to diagnose hollow viscus perforation.
6) Analysis of various causes of morbidity and mortality in hollow viscus perforation peritonitis and factors influencing them.
7) To analyse the different treatment modalities available and their outcome.

MATERIALS AND METHODS

SAMPLE SIZE

74 cases of gastrointestinal hollow viscus perforation who were admitted in our surgical unit, in Government General Hospital, Chennai during the period from July 2004 to June 2005 were taken up for the study.

The total number of emergency surgeries performed by our unit during that period were 214; out of which 74 patients were identified to have 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.
3) Patient with blunt / penetrating injury of abdomen with signs of hollow viscus perforation clinically and radiologically.

PATIENTS EXCLUDED :

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

METHODS

The patients included in the study were subjected to thorough clinical examination of history elicitation and physical examination. They were subjected to following relevant investigations like, complete haemogram, urine routine analysis, biochemical renal function tests with electrolytes, simple coagulation profile of bleeding time and clotting time, serum amylase (subjected to availability), widal test (in selected cases suspected of enteric fever complication), blood culture (in cases with frank sepsis), radiological investigations of 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. In selected cases of blunt injury

abdomen CT abdomen plain and with iv/oral contrast was also taken to supplement the diagnostic armamentarium.

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 majority midline abdominal incisions were used, and the abdominal viscerae inspected carefully for pathology. The site of lesion located and appropriate surgery performed depending on the pathology made out intraoperatively.

Thorough periotoneal toileting done with normal saline and peritoneal cavity 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. Patents 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 of the study.

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 gastrointerstinal 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 perforations were not encountered during this study period and hence are not discussed here.

AETIOLOGYOF 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		Carcinoma ulcerative type
			Leiomyosarcoma
			Lymphoma following chemotherapy
Iatrogenic	Endoscopic	Procedures	Polypectomy
			Biopsy of ulcer
			Cautery of AV malformations

			Percutaenous endoscopic gastrostomy
			ERCP
			EUS with transduodenal biopsy
			Endoscopy assisted transgastric jejunal feeding tube placement
Iatrogenic	Post surgical	Anastomotic leak from gastric staple / suture lines	
		Vertical banded gastroplasty for morbid obesity	
		Splenectomy	
		Harvest of right gastroepiploic artery for CABG	
		Duodenal stump leakage after gastrectomy	
		Lateral duodenotomy for periampullary procedures	
		Biliary tract surgeries like dissecting fibrotic adherent gallbladder, adherent choledochal cyst.	
		Laparoscopic procedures	
			Veres needle for pneumoperitonium
			Fundoplications
			Diaphramatic 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^+ tablets
	Chemotherapy of lymphoma
Diverticular diseases	Meckel's diverticulitis
	Jejunoileal
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	Volvulus
		Malignancy
	Ischaemia	Acute necrotising enterocolitis (pigbell/Darmbrand)
		Radiation enteritis
		Collagen disorders
		Post colonic surgery
		Ischaemic colitis
	Post operative	Anastomotic dehiscence

	Iatrogenic	Embolisation
		Drugs – Steroids
		Ergot alkaloids
		NSAIDS
		Endoscopy
		Barium studies
	Traumatic	Blunt
		Penetrating
	Inflammatory	Crohns disease
		Ulcerative colitis
		Diverticular diseases
	Neoplastic	Carcinoma of large bowel
	Miscellaneous	Stercoral

5. Gallbladder perforations

Acute calculous cholecystitis	
Acute acalculous cholecystitis	Sepsis
	Trauma
	Burns
	Long term Total parenteral nutrition
	Major surgeries like Abdominal aortic aneurysm repair
	CABG
Trauma	Blunt injury
	Penetrating injury
Iatrogenic	

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, filmsy 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-I, PAF, Nitric Oxide , leading to decreased periphral 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 periphral 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 than20 :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 in 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, aperistalytic, > 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 it 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 plan radiograph. The diagnosis often rest 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 atleast 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 demonstrate 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 if 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 β-lactamase inhibitor combination or monobactum + 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. Therfore in a patient presenting with a perforated duodenal ulcer, the presence of significant concomitant heamorrhage should suggest the presence of a "kissing ulcer". In contract 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 cuvature 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 of 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 divercticulum 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 anomally 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 romoval 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 close 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 neocrosis 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 perfringenes. 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 postaortic surgery, therapeutic embolisation and spontaneous ischemic colitis.

ii) Acute and chronic infection

Perforation may complicate acute bacillary dysentry; 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.

Crohns disease

Despite the recognition of fulminating colitis and toxic Megacolon in Crohns 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 perse 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/mm3. 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 appendicectomy reserved only for cases in which appendix is easily accessible. Otherwise interval appendicectomy 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 appendicectomy with peritoneal lavage and drainage.

PERFORATION OF GALL BLADDER AND ITS MANAGEMENT

Perforation of gallbladder occurs in upto 10% of cases of acute cholecystitis. Perforation is a sequelae of ischaemia and gangrene of the gall bladder wall and occurs most commonly in the gall bladder fundus. The perforation is most frequently (50%) contained within the subhepatic space by the omentum, duodenum, liver and hepatic flexure of colon, and a localized abscess form. Less commonly, the gallbladder perforates into and adjacent viscus (duodenum or colon) resulting in a cholecystoenteric fistula and gall stone ileus. Rarely the gall bladder perforates freely into the peritoneal cavity leading to generalized peritonitis.

Acute acalculous cholecystitis often has a more fulminant course than acute calculous cholecystitis and frequently progresses to gangrene, empyema and perforation.

With gallbladder perforation the abdominal tenderness, fever, WBC count are more pronounced or higher than in uncomplicated acute cholecystitis. Localised right upper quadrant pain and tenderness, which becomes diffuse and generalized should raise the suspicion of free gallbladder perforation.

Management

Intravenous fluids, antibiotics and emergency cholecystectomy are the treatment of choice in patients with gallbladder perforation. In most patients, cholecystectomy can be performed. Occasionally, the severe inflammatory process obscures the structures in the triangle of calot precluding safe dissection or ligation of cystic duct. In these patients partial cholecystectomy, cauterization of the remaining gallbladder mucosa and drainage avoids injury to common bile duct.

OBSERVATIONS OF THE STUDY

TABLE 1

Incidence of Hollow viscus perforation in Govt. General Hospital, Chennai

Total No. of emergency surgeries performed	214
Total No. of Hollow viscus perforations encountered	74

35% of emergency surgeries performed in our unit were for the treatment of hollow visceral perforation peritonitis.

TABLE 2

Incidence of Hollow viscus perforation in relation to age group ,sex of the patient

Age Group	No.of Patients
*12-20	15
21-30	14
31-40	26
41-50	12
51-60	6
> 60 years	1

* Only the patients aged ≥ 12 years are admitted in GGH.

Sex	No. of patients
Male	60
Female	14

M : F ratio = 4.3 : 1

TABLE 3

Evaluation of relative incidence of Hollow viscus perforation to the anatomical site of Gastro intestinal tract involved.

	No.of Cases	Percentage
Stomach	3	4%
Duodenum	22	30%

Appendix	27	36%
Small intestine	15	20%
Large intestine	5	7%
Gall Bladder	2	3%
Total	**74**	**100%**

TABLE 4

Evaluation of relative incidence of various causes of GIT Hollow viscus perforation

	Aetiology	No. of Cases
Stomach	Peptic ulcers	3
Duodenum	Peptic ulcers	22
Small Bowel	Typhoid ulcers	3
	Adhesive intestinal obstruction leading to perforation	3
	Intestinal stricture leading to perforation due to obstruction	1
	Closed loop obstruction and strangulation of hernia	3
	Traumatic (Blunt injury abdomen)	5
Appendix	Localised peritonitis due to appendiceal rupture	22
	Generalised peritonitis with intestinal obstruction	5
Colon	Iatrogenic sigmoid perforation due to colonoscopy	1
	Sigmoid colon perforation due to volvulus	1
	Transverse colon perforation in obstructed umbilical hernia	1
	Caecal perforation due to descending colon growth obstruction	1
	Traumatic rectal (Extraperitoneal) tear	1
Gall Bladder	Acute calculous cholecystitits	2
	Total No. of Cases	**74**

TABLE 5

Relative incidence of various symptoms and signs of Hollow viscus perforation peritonitis

Involved organ	Abdominal pain	Anorexia	Nausea	Vomiting	Fever	Dehydration	Tachycardia	Hypotension	Oliguria	Localised peritonitis	Generaied peritonitis	Obliteration of liver dullness	Total no.of cases
Stomach	3	-	-	3	2	2	3	2	2	1	2	3	3
Duodenum	22	-	4	18	10	20	22	17	16	5	17	20	22
Small bowel	15	3	1	9	9	12	15	6	6	9	6	-	15

Appendix	27	20	12	15	19	10	19	3	3	22	5	-	27
Colon	5	-	-	2	1	4	5	3	3	2	2	2	5
GB	2	-	-	2	2	1	2	-	-	-	2	-	2

TABLE 6

Relative incidence of Radiological signs in plain radiographs of abdomen and chest in Hollow viscus perforation peritonitis

Organ involved	Air under diaphragm chest Xray	Riglers double wall sign supine abdomen X-ray	Ground glass apperance of generalized peritonitis Erect abdomen	Localised ileus erect abdomen	Poor quality
Stomach	3	1	2	-	1
Duodenum	21	2	10	-	6
Small bowel	8	-	6	3	6
Appendix	-	-	4	19	7
Colon	2	1	-	2	2
GB	-	-	-	-	1

TABLE 7

Incidence of various post operative complications in patients of Hollow Viscus perforation peritonitis

Organs involved	Total no of cases	Burst abdomen	Wound infection	Residual intraabdominal abscess		Respiratory complications	EC Fistula	Renal failure	Septicaemia
				Pelvic	Subphrenic				
Stomach	3	-	1	-	1	2	-	2	2
Duodenum	22	-	7	1	-	15	2	8	6
Small bowel	15	-	10	-	-	8	-	2	2
Appendix	27	-	13	1	-	5	2	-	-
Colon	5	-	3	-	-	2	-	1	1
GB	2	-	1	-	-	-	-	-	-
Total	74	-	35	2	1	32	4	13	11

TABLE 8

Incidence of mortality in Hollow viscus Perforation

Organ involved	No.of cases	No.of death	Case fatality rate	Disease specific mortality rate
Stomach & Duodenum	25	7	28%	64%
Small bowel	15	2	13.3%	18%
Appendix	27	1	3.7%	9%
Colon	5	1	20%	9%
GB	2	-	-	-
Total	**74**	**11**	**-**	**100%**

TABLE 9

Different treatment modalities followed for Hollow viscus perforation peritonitis

Stomach and Duodenum	Stomach	Duodenum
Simple closure	-	2
Graham omental patch closure	3	20
Simple or patch closure with definitive surgery	-	-

Small bowel	No. of Cases
Simple closure	2
Simple closure with omental onlay	-
Resection and anastomoses	13
Exteriorisation of small bowel	-

Appendix	No. of Cases
Appendicectomy	18
Appendicular abscess drainage (Extraperitoneal)	4
Laparotomy with abscess drainage with adhesiolysis and appendicectomy	5

Colon	No. of Cases
Resection of perforated segment with primary anastomoses	2
Resection of perforated segment with exteriorization of bowel ends	1
Proximal colostomy alone	1
Simple primary closure	1
Hartmann's procedure	-

Gall Bladder	No. of Cases
Cholecystectomy	2

Total Cases	**74**

DISCUSSION

This study was done on 74 cases of Hollow viscus perforation with secondary peritonitis, admitted in our surgical unit VI of Govt. General Hospital, Chennai, during the 1 year period from July 2004 to June 2005.

During this period of 1 year, 214 emergency surgeries were done in our surgical unit of which hollow viscus perforation, peritonitis accounted for 35% of emergency surgeries performed (Table 1).

Clinical diagnosis was made from history and physical examination and appropriate investigations. The patients were resuscitated and surgeries done depending on the primary cause.

Table 2 summarises the incidence of hollow viscus perforation in different age groups and the sex incidence of the same. Hollow viscus perforation was commonly encountered in the age group of 31-40 years and it was found to be more common in male population with a M : F ratio of 4.3:1.

Table 3 summarises the incidence of hollow viscus perforation in relation to the anatomical region of the gastrointestinal tract involved. Appendix was the commonest site of perforation encountered and accounted for 36% of cases, with duodenum next in frequency accounting for 30% cases of gastrointestinal hollow viscus perforation.

Table 4 illustrates the various aetiologies for the perforation of gastrointestinal hollow viscera encountered in this study. The commonest aetiology for the perforation of GIT hollow viscera was found to be Acute appendicitis, accounting for 36% of total number of perforation cases and perforated peptic (Duodenal) ulcer next in order of frequency accounting for 30% of total number of cases.

The overall rate of perforated appendicitis is 37% in contrast to western standards of 25.8%. This increased rate of perforation in acute appendicitis is probably due to the delayed presentation of the cases to the surgeons, leading to the complication of perforation of the inflammed appendix. Some cases of Appendiceal perforation even presented with features of intestinal obstruction due to gross peritoneal contaminations and adhesions. The commonest age group to have appendiceal perforation is 12-20 years.

Perforation of dudodenal ulcer accounted for 30% of total number of cases of secondary peritonitis. Only 45% (10 out of 22 DU perforation) of these patients had a prior history of peptic ulcer disease and only 18% of DU peforations (4 out of 22 cases) had history of NSAID abuse mainly for generalised myalgia, NSAIDS being purchased over the counter by patients themselves. The ratio of Duodenal ulcer perforation to gastric ulcer perforation in our study is 11:1. No cases of malignant gastric ulcer perforation was encountered and no posterior wall perforations were encountered.

The commonest aetiology for small bowel (Jejunal and Ileal) perforation in our study was perforation secondary to intestinal obstruction (47%) caused by adhesions, small bowel stricture and closed loop obstruction and strangulation of inguinal hernia. Next in order of frequency in aetiology of small bowel perforation was blunt injury abdomen (33% -- 5 of 15 cases) , followed by ileal perforations as a sequelae of enteric fever (20% --3 of 15 cases). 2 out of 3 cases of enteric fever perforations showed positive widal test with increasing titres on two occasions, but

only a single positive titre value was obtained in the last case without an increase in titre on repeat sample.

The commonest cause of blunt injury abdomen was road traffic accident, followed by assault injuries.

Only 4 cases of colonic perforation and 1 case of rectal perforation were encountered in our study period accounting for 7% of gastrointestinal hollow viscus perforations. The aetiology of colonic perforation were transverse colon perforation due to closed loop obstruction of an umbilical hernia, sigmoid volvulus leading to perforation of the loop, iatrogenic sigmoid colon perforation during diagnostic colonoscopy, caecal perforation due to obstruction of descending colon due to malignancy and an extraperitoneal rectal injury due to perineal injury due to train traffic accident.

Only 2 cases of gall bladder perforations were encountered accounting for 3% of all gastrointestinal hollow viscus perforations. Both were due to complication of acute calculous cholecystitis. In one of the cases, who was a known diabetic, even USG did not suggest any evidence of acute cholecystitis, due to distended bowel gas disturbances. In that particular case diagnosis of acute appendicitis was made as patient had severe right iliac fossa tenderness and abdomen was opened through McBurney's incision and intra-operatively found bile staining of intestinal loops and a normal appendix and so laparotomy was done and the gallbladder perforation was then diagnosed intra-operatively. Probably that case could have been an emphysematous cholecystitis as the patient was a known diabetic. But error in diagnosis could have been averted if CT abdomen could have been done, which was not affordable by that patient due to financial constrains.

Table 5 illustrates the frequency of various symptoms and signs of gastrointestinal hollow viscus perforation peritonitis. Abdominal pain was the most predominant symptom and was found in all cases in this study. Tachycardia was the most predominant sign. In stomach and duodenal perforations, vomiting and fever were the other main symptoms and majority of these patients had feature of hypovolemic shock like dehydration, tachycardia, hypotension, cold extremities, oliguria due to established secondary peritonitis due to delay in presentation, necessitating vigorous resuscitation pre-operatively.

Anorexia was an important prominent symptom in appendicular pathology. Incidence of fever increased in appendicular perforation when compared to unruptured appendicitis. Hypovolemia was not present in majority except for those few cases (5 out of 27) which presented with diffuse peritonitis and features of intestinal obstruction.

In colonic perforation, due to severity of faecal contamination, 3 out fo 5 cases presented with hypovalemia needing severe pre-operative resuscitation. The iatrogenic perforation of sigmoid colon by colonoscopy was diagnosed soon after the procedure and hence patient was treated early without any further complication.

Gall bladder perforations presented with abdominal pain, fever, vomiting and generalised peritonitis with tachycardia. But no features of established hypovolemic shock was found.

Table 6 illustrates the frequency of radiological signs elicited in plain radiographs of chest and abdomen of patient with hollow viscus perforations.

Air under right hemi diaphragm was a feature more prominently elicited in chest Xrays than abdominal radiographs and more commonly elicited in perforations of large hollow viscera like stomach, duodenum and small and large bowel. Appendiceal perforation and Gall bladder perforation did not display this sign in plain radiographs.

Other features elicited in common in abdominal radiographs is ground glass appearance due to fluid in peritoneal cavity in severe peritonitis and localized sentinel small bowel loops in appendicular pathology.

Substantial number of radiographs were of poor quality to be used for interpretation of radiographic signs of hollow viscus perforation.

Table 7 illustrates the frequency of post operative complications in patients with hollow viscus perforations. Wound infection was the commonest complication which occurred in 47% of patients of hollow viscus perforation peritonitis inspite of emperical antibiotics. Respiratory complication of pulmonary collapse and pneumonia occurred in 43% of patients. Serious complications of renal failure and septicemia occurred in 18% and 16% of patients respectively postoperatively. Residual intra-abdominal abscess (pelvic and subphrenic) occurred in 3 patients. Enterocutaneous fistula complicated 4 patients post-operatively, 2 after duodenal ulcer perforation closure and 2 after appendicular abscess drainage. Burst abdomen was not encountered during this study.

In table 8, disease specific mortality rate and case fatality rate estimation shows that perforation of stomach and duodenum due to peptic ulcer disease was the most severe disease of all in causing deaths.

Table 9 shows the different treatment modalities used in our unit for the management of gastrointestinal hollow viscus perforations encountered during this study period.

In peptic ulcer perforations of stomach, the treatment used was ulcer biopsy and closure with omental patch. In duodenal ulcer perforations simple closure with omental patch was the procedure commonly done. Simple closure with 3-0 silk sutures without omental patch was done in 2 cases, one of which ended up in post operative leak and enterocutaneous fistula. Definitive ulcer surgery was not done in any of these cases as there was gross peritoneal contamination due to delayed presentation of patients (> 24 hours) and moreover patients were hemodynamically unstable to undergo a prolonged surgery.

In enteric small bowel perforations with single peforation ileum treatment modality used was simple closure in transverse axis. When it involved ileum at multiple sites then resection of that ileal segment and primary anastomosis was performed. In all traumatic perforations of small bowel, resection and anastomoses were performed, which was also the surgery done for perforations due to intestinal obstructions of various causes.

In acute appendicitis, where pre-oepratively a clear diagnosis of appendicular abscess was made, extraperitoneal drainage of abscess was done, 2 cases of which ended in low output enterocutaneous fistula, which settled with conservative line of treatment. In all other cases with localized peritonitis, appendicectomy done with McBurney's incision and local drainage of pus also done during same procedure. In cases of generalized peritonitis with features of intestinal obstruction midline incision

laparotomy done with adhesiolysis, drainage of pus in the cavities and appendicectomy.

In large bowel perforation due to colonoscopy, immediate early laporotomy was done with primary closure of perforation as the perforation was small and peritoneal soiling was very minimal due to the previous mechanical preparation done for colonoscopy. Covering colostomy was not done in that patient.

In perforation of sigmoid volvulus, resection and primary anastomoses was done.

In the perforation of transverse colon due to closed loop obstruction in umbilical hernia, resection of the gangrenous transverse colon and primary end to end anastomosis was done. Colostomy was not preferred in this setting because there was no soiling in the general peritoneal cavity and the remaining colon did not contain much of faecal matter. So primary anastomosis was performed with on table colonic lavage.

In case of caecal perforation due to left colonic growth obstruction, exteriorisation of bowel ends performed after limited resection of ascending colon with caecum. The left colonic growth was inoperable and hence patient was referred to palliative chemoradiation.

In a case of extraperitoneal rectal injury due to train traffic accident, the patient suffered associated severe crush injury to both lower limbs and extensive perineal injury and hence after immediate resuscitation, guillotine amputation was done at below knee level for both lower limbs and pelvic loop colostomy done for emergency faecal diversion.

In both cases of Gall bladder perforations, cholecystectomy were done. In one of the two cases, which was misdiagnosed as acute appendicitis, abdomen was first opened through Mcburney's incision and later midline laparotomy done, after finding a normal appendix and bile staining of bowel loops. Both patients recovered well after emergency cholecystectomy.

SUMMARY AND CONCLUSION

This study, on gastro intestinal tract hollow viscus perforation was conducted from 74 cases admitted in our General Surgical unit in Govt. General Hospital, Chennai, during 1 year period from July 2004 to June 2005 and the following conclusions were made.

1) Hollow viscus perforations of gastrointestinal tract was more common in males and more common in age group of 31-40 years.

2) Commonest cause of gastrointestinal tract hollow viscus perforation is appendiceal perforation, due to acute appendicitis.

3) Abdominal pain is the commonest presenting symptom and Tachycardia with abdominal tenderness being commonest signs.

4) Plain chest radiograph is more superior to plain abdominal radiographs in demonstrating pneumoperitoneum. Appendiceal perforations do not demonstrate pneumoperiotoneum on plain radiographs.

5) Wound infection is the commonest post operative complication encountered in these patients with secondary peritonitis.

6) Peptic ulcer perforation is the most dangerous among the various causes of gastrointestinal perforations with very high case fatality rate and disease specific morality rate.

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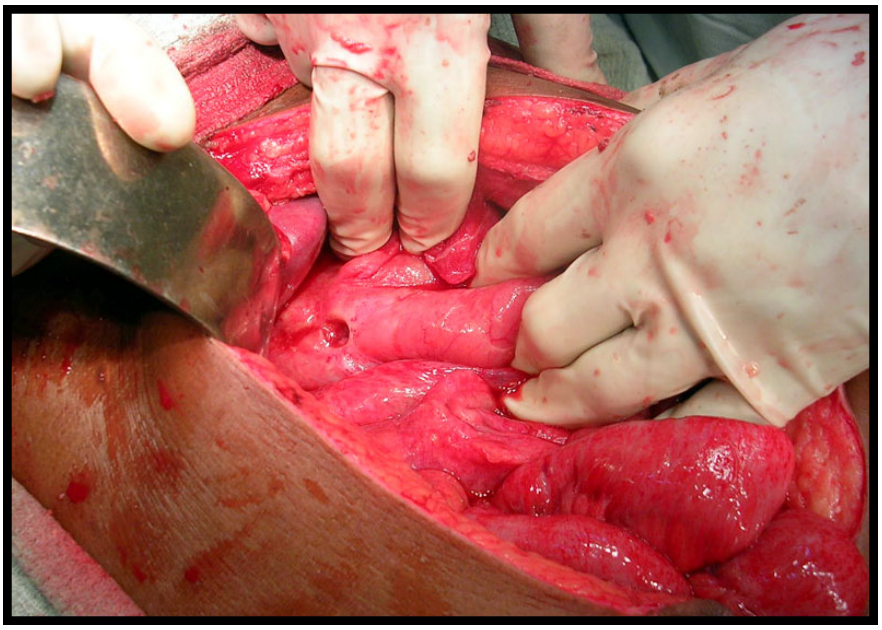
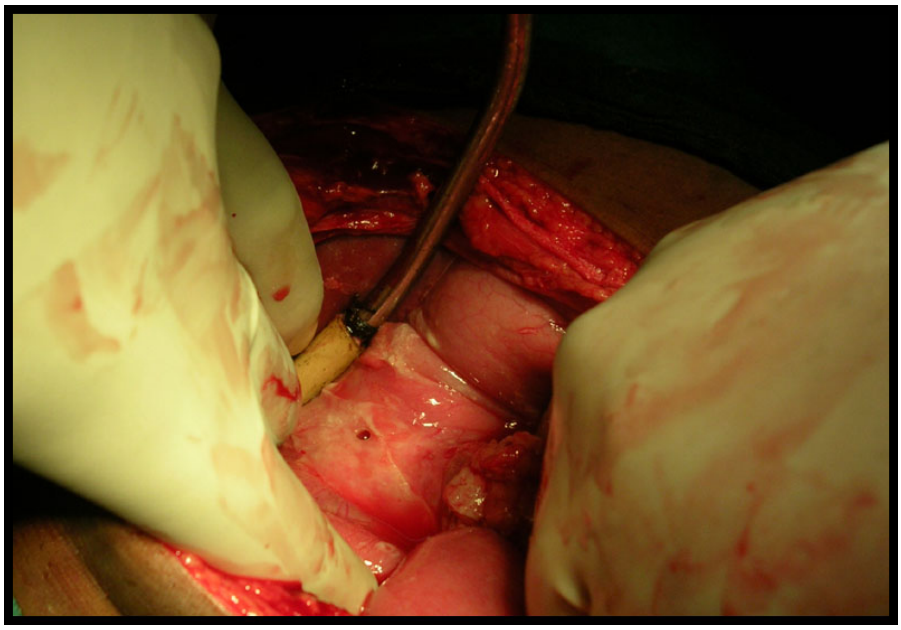


ANTERIOR WALL DUODENAL ULCER PERFORATION



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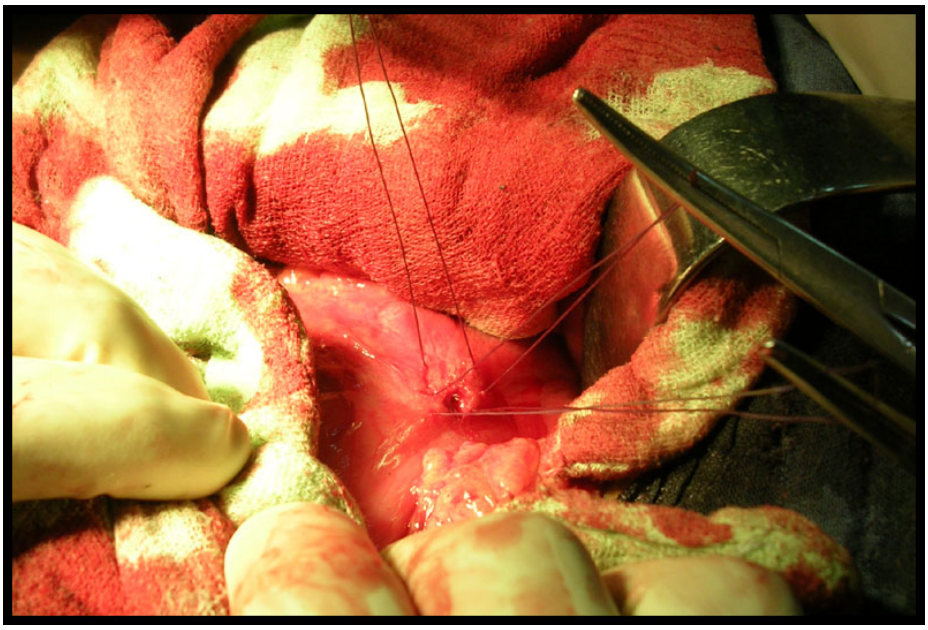
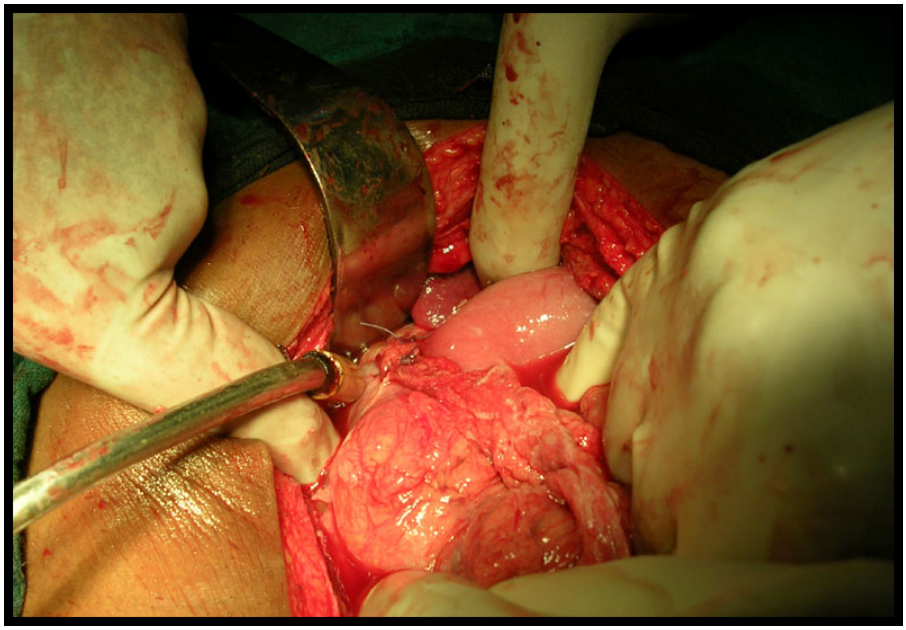


DUODENAL ULCER PERFORATION GRAHAM'S PATCH CLOSURE IN PROGRESS



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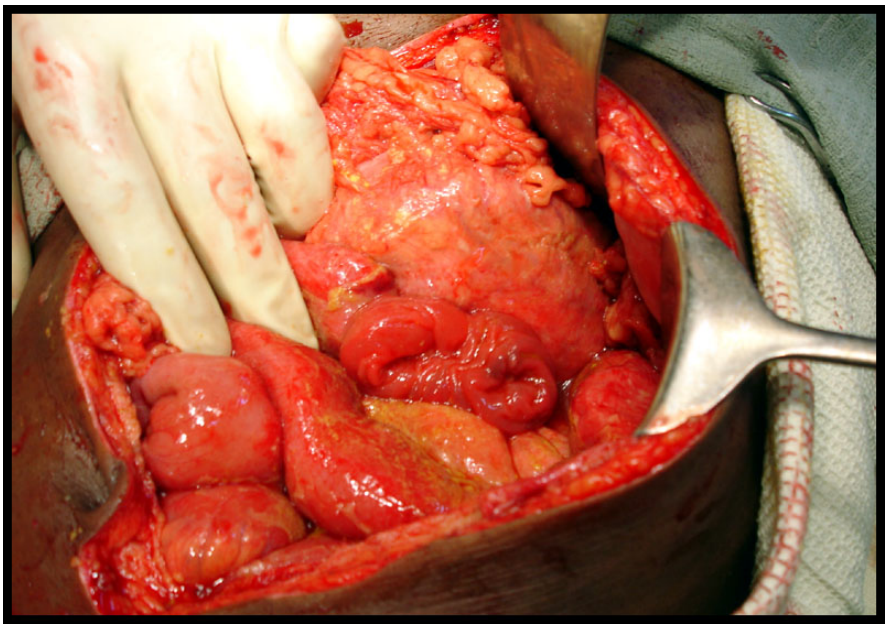
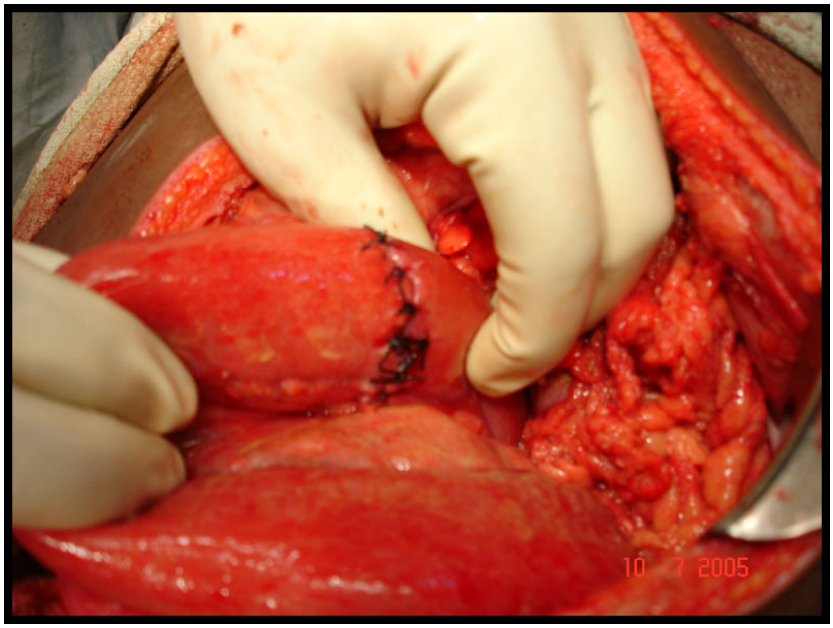
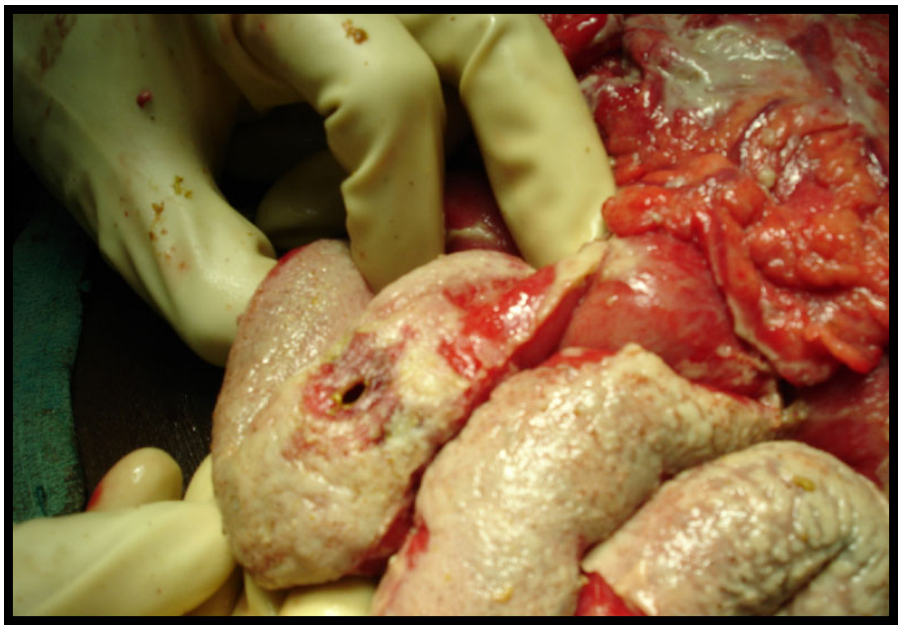


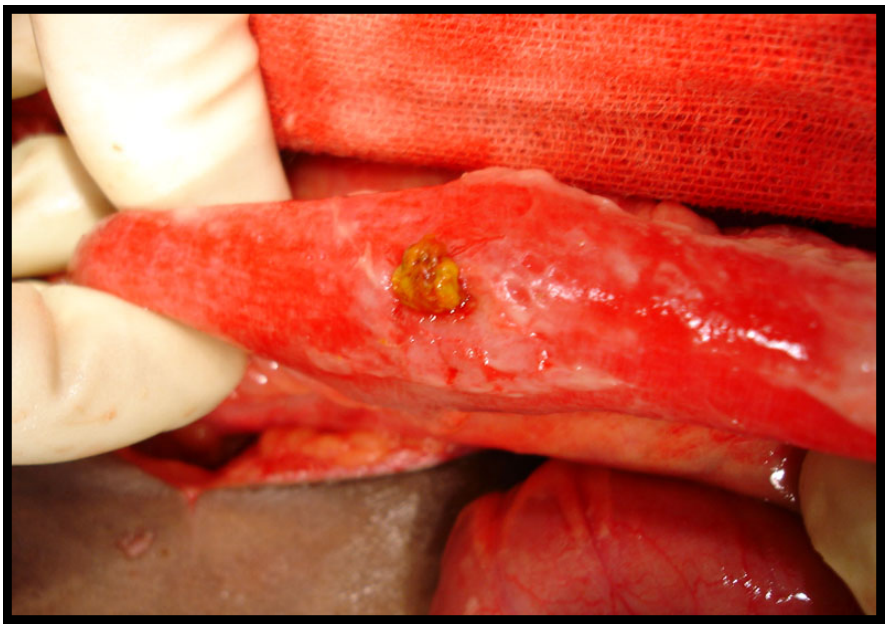
TRAUMATIC JEJUNAL TEAR



RESECTION AND ANASTOMOSIS OF TRAUMATIC JEJUNAL TEAR



ILEAL PERFORATION – ENTERIC FEVER SEQULAE



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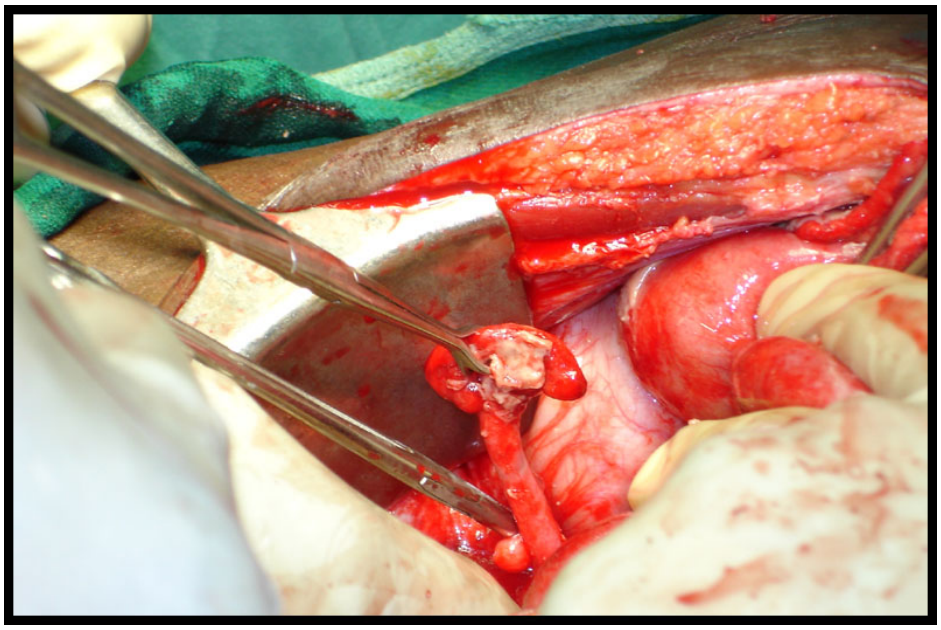
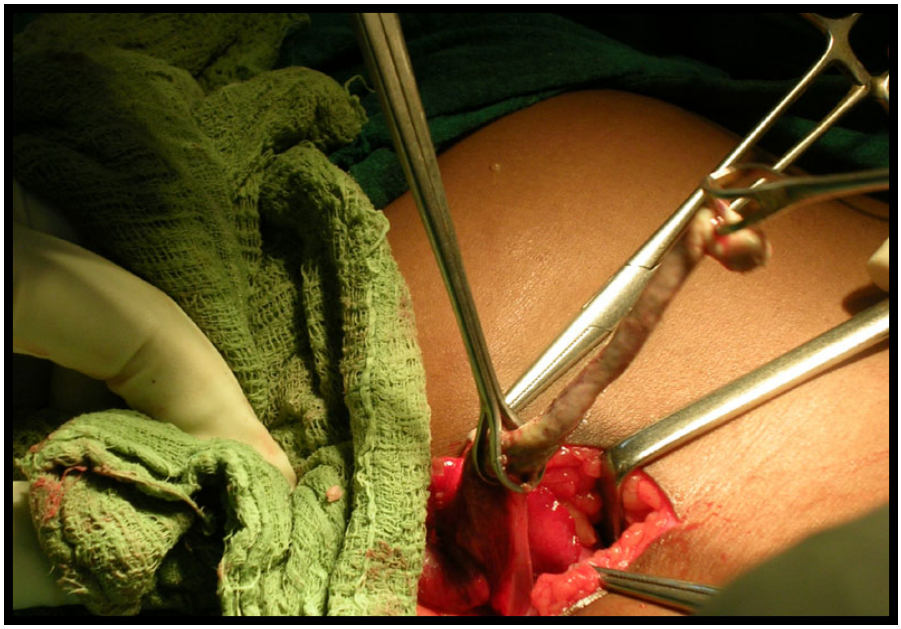
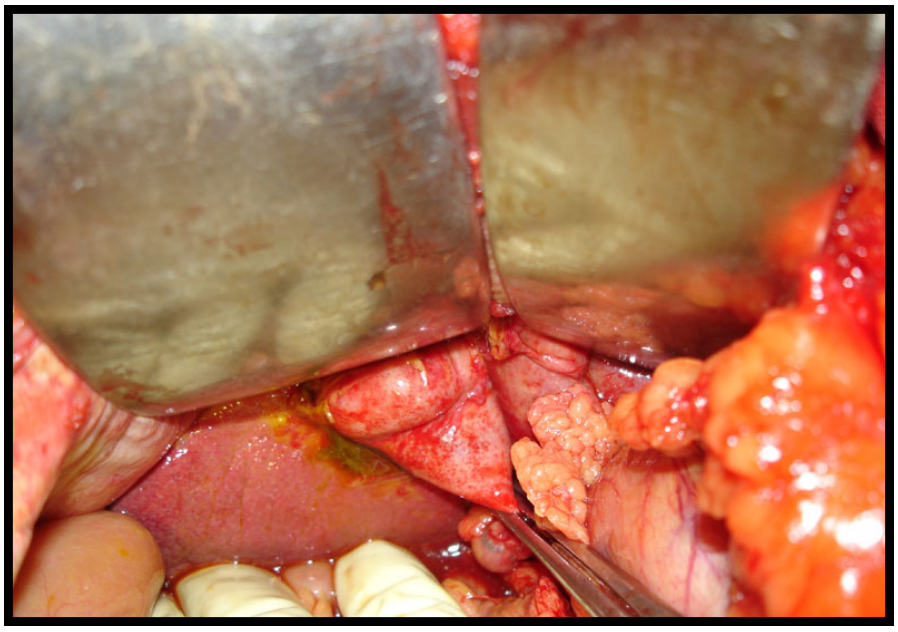


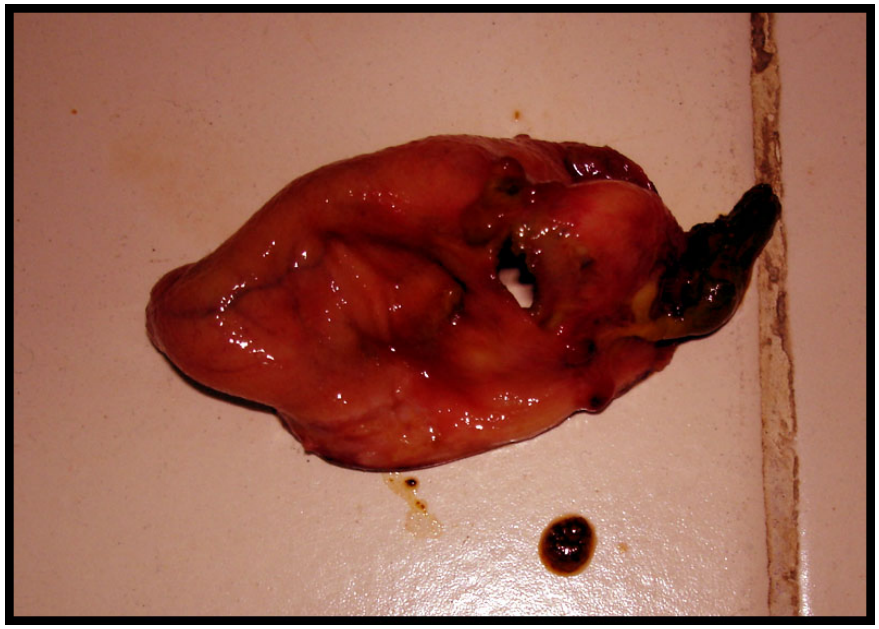
APPENDICEAL PERFORATION – SEQULAE OF ACUTE APPENDICITIS



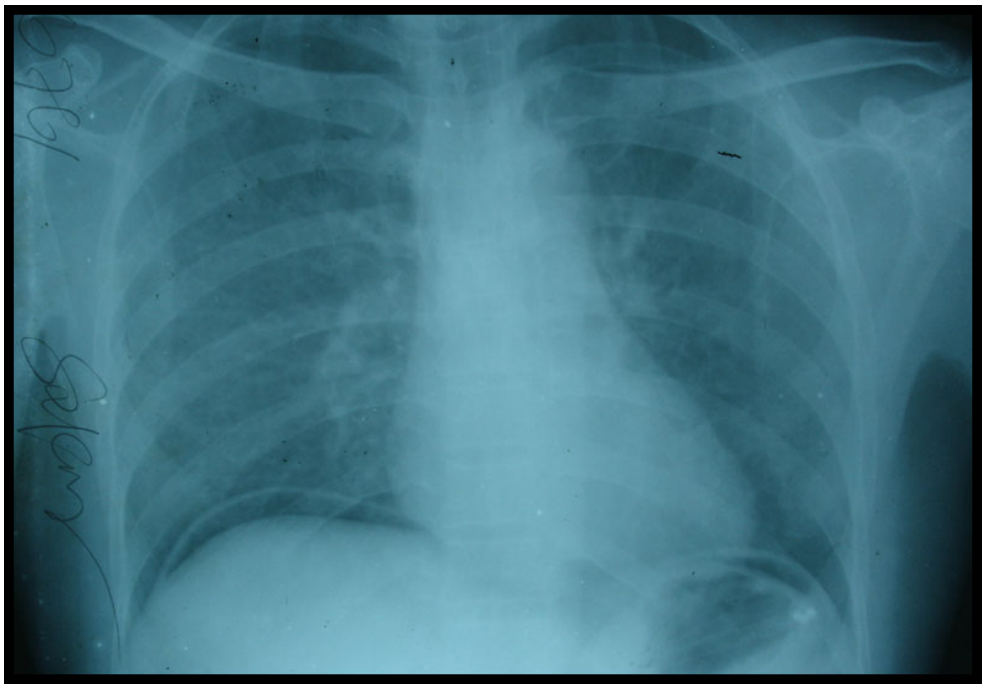
APPENDICEAL PERFORATION – APPENDICECTOMY IN PROGRESS



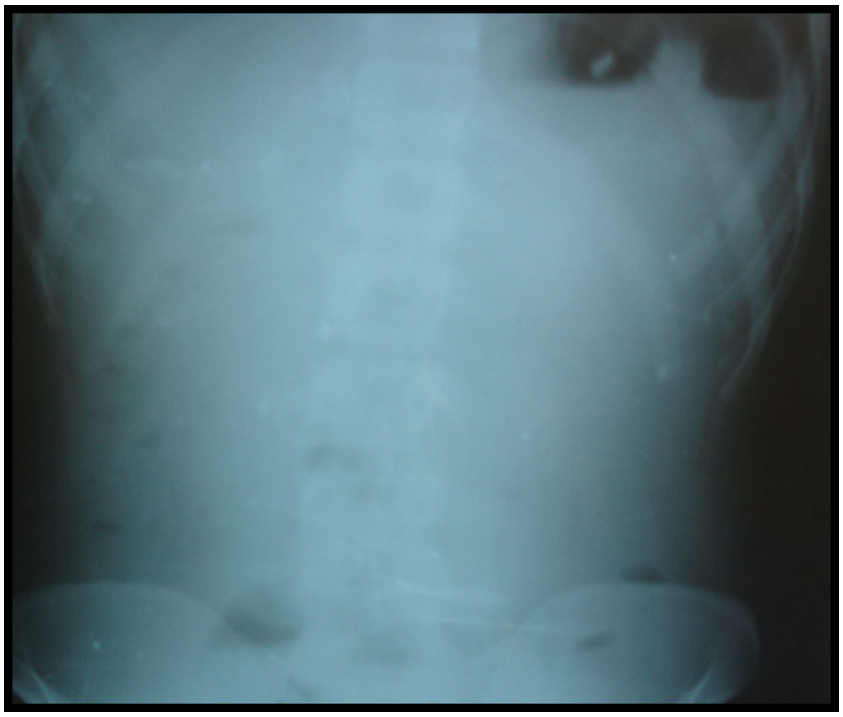
GALLBLADDER PERFORATION - SEQULAE OF ACUTE CALCULUS CHOLECYSTITIS



PERFORATED GALL BLADDER SPECIMEN WITH THE GALLSTONE



CHEST X-RAY WITH AIR UNDER RIGHT HEMI DIAPHRAGM IN DUODENAL ULCER PERFORATION



ERECT ABDOMINAL X-RAY WITH GROUND GLASS APPEARANCE OF GENERALISED PERITONITIS