A CLINICAL STUDY OF PERFORATIVE PERITONITIS

Dissertation submitted to

The TamilNadu Dr.M.G.R. Medical University, Chennai.

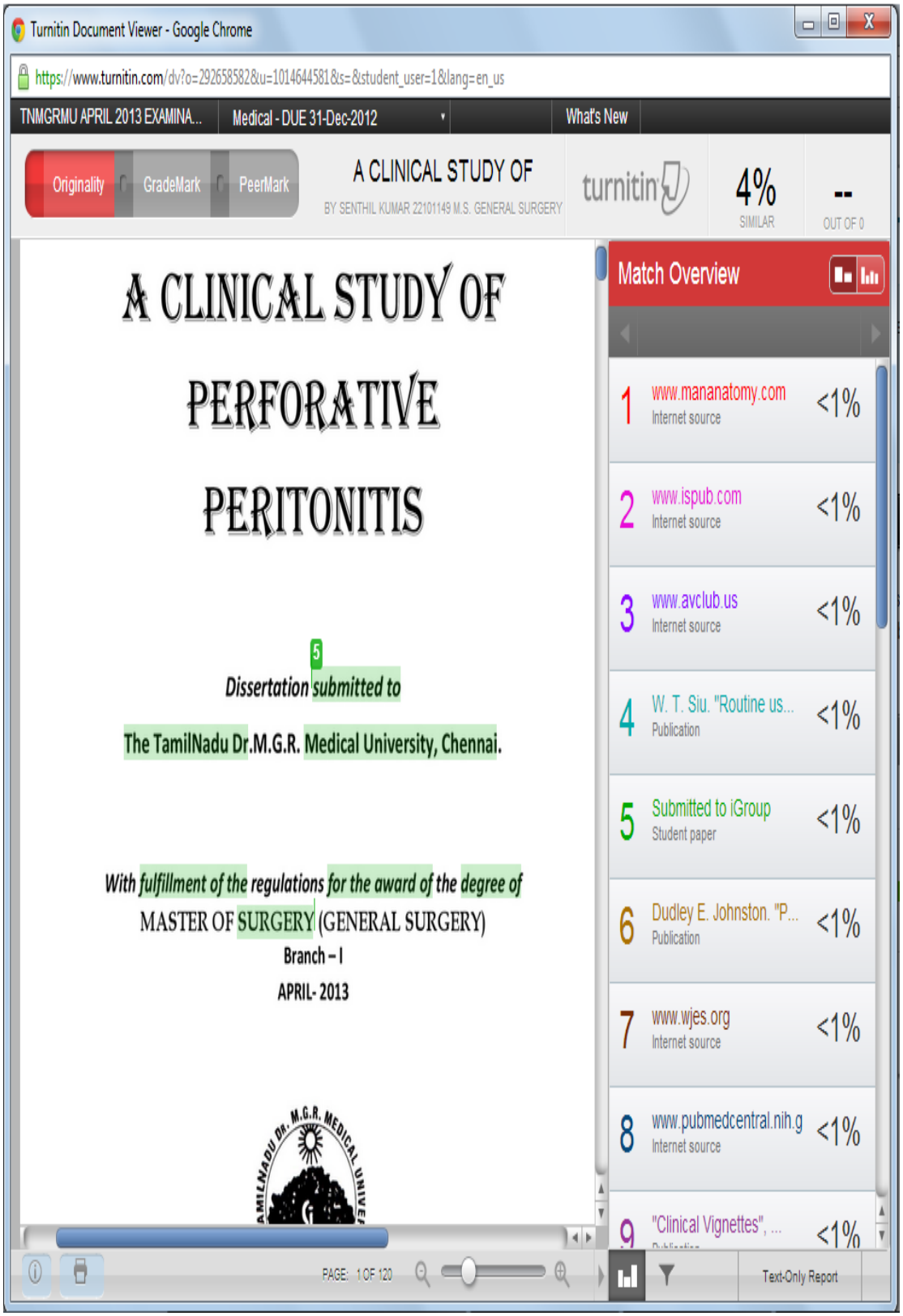
With fulfillment of the regulations for the award of the degree of

MASTER OF SURGERY (GENERAL SURGERY)

Branch – I

APRIL- 2013

DEPARTMENT OF GENERAL SURGERY
MADURAI MEDICAL COLLEGE
MADURAI – 625 020.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A CLINICAL STUDY OF PERFORATIVE PERITONITIS Dissertation submitted to The TamilNadu Dr.M.G.R. Medical University, Chennai. With fulfillment of the regulations for the award of the degree of MASTER OF SURGERY (GENERAL SURGERY) Branch – I APRIL- 2013 DEPARTMENT OF GENERAL SURGERY MADURAI MEDICAL COLLEGE MADURAI – 620 020. CERTIFICATE This is to certify that the Dissertation titled "A CLINICAL STUDY OF PERFORATIVE PERITONITIS" of Dr.K.SENTHIL KUMAR is done in partial fulfillment of the requirements of M.S. Branch I General Surgery Degree Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL 2013. The period of study is from NOV 2011 to OCT 2012. Prof. D....

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I hereby declare that this dissertation entitled **"A CLINICAL STUDY OF PERFORATIVE PERITONITIS"** is a bonafide and genuine research work carried out by me in the Department of General Surgery under the guidance of **Prof. M.SEKARAN. M.S.,** This is submitted to **The Tamilnadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MS degree (Branch I) General Surgery course on April 2013.

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ACKNOWLEDGEMENT

I thank the DEAN of Madurai Medical College and Govt Rajaji Hospital, **Prof. N. MOHAN. M.S.,** for permitting me to conduct this study in the Department of General Surgery of the Government Rajaji Medical College and Hospital, Madurai.

I have absolutely no words to express my deep sense of gratitude and sincere thanks to my guide **Prof. M.SEKARAN. M.S.,** and my Unit Chief, for his constant encouragement, help, invaluable guidance, moral support and blessings throughout my Post Graduation course. I thank him for helping me in selecting and preparing this dissertation.

I pay my humble respect and owe and equally great debt of gratitude to **Prof. SOUNDARARAJAN. D. M.S.**, Professor and Head, Department of Surgery for his immense and constant help in the progression of the dissertation work.

I am greatly indebted and thankful to my Assistant Professors **Dr. M. MANIKANDAN, M.S., M.R.C.S., F.I.A.G.E.S., Dr. J. ANTO FERNANDO, M.S.,** and **Dr.G. SARAVANAKUMAR, D.A., M.S.,F.P.S.,** who have put in countless hours in guiding me in many aspects of this study and also in honing my surgical skills.

My heart felt gratitude to the Professors and Assistant Professors of all other units.

My deep sense of gratitude and indebtedness to my loving parents **Mrs.SUGUNA** and **Mr. N.KRISHNAN** for their blessings, silent encouragement and uncompromising support.

Last but not the least I am thankful to all my **patients** without whom this study could not have been completed.

CONTENTS

INTRODUCTION

Peritonitis is one of the most common surgical emergency encountered in day to day practice.

Peritonitis is the inflammation of serous membrane called peritoneum that lines the peritoneal cavity either in response to injury or infection. It may be localized or generalized infection.

Peritonitis is organized into three types based upon the source and nature of microbial contamination.

Primary peritonitis or **spontaneous bacterial peritonitis** is an infection or inflammation of the peritoneal cavity without any hollow viscous perforation, usually from extra-peritoneal source due to ascites

Secondary peritonitis follows an intraperitoneal source usually following perforation of a hollow viscous organ in gasro intestinal tract.

Tertiary peritonitis develops following inadequate treatment or failure of treatment of secondary peritonitis.

Peritonitis due to perforation of hollow viscous (secondary peritonitis) is the most common type of Peritonitis .

Despite advances in diagnosis, surgical technique, antimicrobial therapy and intensive care support, secondary peritonitis remains a potentially fatal surgical emergency which requires institution of timely treatment for the best possible outcome, if not it may result in severe bacterial Peritonitis leading to deadly complication ranging from toxaemia to circulatory collapse and death.

OBJECTIVES OF OUR STUDY

- To study the relative incidence of peritonitis secondary to hollow viscus perforation in relation to
  - Age
  - Sex
  - Anatomical location
  - Symptoms and signs
- To analyse various etiology of hollow viscous perforation leading to perforative peritonitis.
- To analyse various management modalities and its out come in our Hospital.
- To Review literature on various types of peritonitis.

STUDY CRITERIA

INCLUSION CRITERIA

- Patient with past history of peptic ulcer disease with features of peritonitis.
- Patient who presented with features of peritonitis either radiologically or clinically
- Patient who presented with features of peritonitis in whom peritoneal fluid culture was positive.
- Patient with traumatic (blunt or penetrating) injury of the abdomen with the signs of hollow viscous perforation.

EXCLUSION CRITERIA

- Patient with post operative peritonitis.
- Patients with peritonitis due to entero cutaneous fistula
- Patient with iatrogenic perforation during laparotomy or scopy.
- Patient with oesophageal perforation, and peritonitis due to pancreatic pathology.
- Perforative peritonitis in pediatric age group.

REVIEW OF LITERATURE

History[4]

1st documented case of perforated peptic ulcer is of a man who died in year 167 B.C, whose body was extensively analysed by the Archaeologist in 1975, and the cause of death was found to be diffuse Peritonitis.

MURATO First described the duodenal ulcers at autopsy in the year 1688.

In the year 1767, NOLLESTRON reported the first successful repair of traumatic gastric injury. Aristotle was first to describe intestinal injury as a consequence of abdominal trauma. He is credited in saying "A slight blow will cause rupture of the intestine without injury to the skin."

BAILLE in the year 1799, described a patient who died from perforated duodenal ulcer, just distal to the pyloric ring.

Perforated peptic ulcers were treated non operatively until late 1800.

CRISP in the year 1843, published a paper in which he described the symptomatology of perforated peptic ulcer.

1st successful operation of Gastic ulcer perforation was done in 1892 by LUDWIG'S HUESNER WUPPERTAL, of Germany and in the year 1894 by HENRY PERCY DEAN of London.

Historically, the surgical management of the perforated duodenal ulcer is highlighted by MICKULIZ (1887), reported the first suture plicate of a perforated ulcer,. VON HAVERED (1919) suggested that surgery is the definitive management,

First description of simple closure of perforated Duodenal ulcer was, in the year 1937 by GRAHAM.

SEELEY (1956) suggested the conservative approach of continuous nasogastric suction.

Laparoscopic techniques for perforated duodenal ulcer is no exception. It was introduced by NATHANSON in the year 1990.

The incidence of peptic ulcer and it complications was declining since 1960 after the introduction of Anti Secretory drugs(H_2 Blocker's and proton pump inhibitors), anti H.Pylori regimen.

ANATOMY

Structure of the Peritoneum[1]

Peritoneum is the largest serious membrane, lining the Abdominal cavity. In male it forms a closed sac, but in females, it is open at the lateral ends of Fallopian tube[1].

Peritoneal Cavity

The peritoneal cavity is a potential space formed between two layers of peritoneum.

- Outer layer called Parietal Peritoneum which line the abdominal wall and it is loosely attached to it.
- Inner layer called visceral peritoneum which suspends the abdominal viscera with in the peritoneal cavity.

It never contains gas in normal circumstances although the amount of fluid may be increased in inflammatory condition of the viscera.

This consists of:

The greater sac or general peritoneal cavity.

The lesser sac or the small omental bursa which is a diverticulum of the peritoneal cavity behind the stomach and adjoining structures. It opens into the greater sac through a slit like aperture the epiploic foramen.

Derivatives of Peritoneum[1] :

Omentum :

There are two commonly described omental folds, greater and lesser omentum.

- Lesser omentum[1] is a double layer fold of peritoneum extending from lesser gastric curvature and commencement of duodenum to liver. The right free margin known as gastroduodenal ligament contains hepatic artery, portal vein, bile duct, lymph nodes and lymphatics and hepatic plexus of nerves. It forms anterior wall of lesser sac and allows fluid collection and abscess formation in lesser sac during any pathologic process especially of pancreas.
- Greater omentum[1] is largest peritoneal fold. It is folded on itself to form four layered sheath. It descends from greater curvature and first part of duodenum, downwards up to symphysis pubis, folds on itself to ascend and adhere to peritoneum on superior surface of transverse colon and mesocolon. This line of fusion is known as Avascular plane of Toldt. Its left border is continuous with gastrosplenic ligament and right border continuous on duodenum. The greater omentum is thin and cribriform, and always contain some adipose tissue, the quantity of which is increased in obese individuals. Between 2 layers of anterior fold, it contains right and left gastroepiploic vessels forming an anastomotic arc which is to be preserved during gastric mobilization. Tiny white opaque oval

or irregular shaped spots or bodies (milky spot) may be described within thin membranes of omentum in children and lean adults. Their number decreases with age. Their size increases with pathological conditions in abdomen. Microscopically it contains glomus like pattern of vascular structure, cellular population of fibroblasts, lymphocytes, plasma cells, fat cells and specialized mesothelial cells.

Mesentery:

These are two layered folds of peritoneum connecting parts of the intestines to the posterior abdominal wall

The Peritoneal cavity is divided into interconnected compartments or spaces by 11 ligaments and mesenteries.

The ligaments which are folds of peritoneum include[2]

1) Coronary ligaments
2) Gastro hepatic ligament
3) Hepato duodenal ligament
4) Falciform ligament
5) Gastrocolic ligament
6) Duodenocolic ligament
7) Gastrosplenic ligament
8) Splenorenal ligament
9) Phrenicocolic ligament
10) Transverse Mesocolon,
11) Small bowel Mesocolon

Peritoneal Compartments:

The peritoneum by virtue if its attachments to the posterior abdominal wall and to various viscera, divides the peritoneal cavity into compartments called

- Supracolic
- Infracolic and
- Pelvic

The Supracolic compartment is subdivided into four compartments

- Right upper or right subphernic (sub diaphragmatic) compartment
- Right lower or hepatorenal pouch (of Morrison)
- Left upper or left Subphrenic (subdiaphargmatic) compartment
- Left lower or left subhepatic compartment.

The infracolic compartment has two parts – Right (upper) and Left (lower).

The dividing line between the supracolic and infracolic compartments is the attachment of the transverse mesocolon to the posterior abdominal wall.

When lying supine, the hepatorenal pouch is the lowest part of the peritoneal cavity (with the exception of the pelvis), and hence is an area where intraperitoneal fluid is likely to accumulate.

Nerve Supply:

Parietal peritoneum is supplied segmentally by the spinal nerves that innervate the overlying muscles. Thus the diaphragmatic peritoneum is supplied centrally by phrenic nerve (C4) and peripherally by intercostal nerves. The remainder of the parietal peritoneum is supplied segmentally by intercostal and lumbar nerves.

The visceral peritoneum has no afferent supply and pain form diseased viscera is due to muscle spasm, tension on mesenteric folds or involvement of the parietal peritoneum.

All these ligaments, mesenteries, and peritoneal spaces, direct the circulation of fluid in the peritoneal cavity thus predicting the route of spread of infection and malignant disease. For example, Perforation of Duodenum from peptic ulcer, result in the development of abscess in sub hepatic space, Right paracolic gutter and the pelvis.

SURGICAL PHYSIOLOGY[2]

Peritoneal cavity is the largest cavity in the body. The surface area of its lining membrane is $1.8m^2$ to $2m^2$. It is a bidirectional semi permeable[2] membrane, which is composed of single layer of mesothelial[3] cell resting on a thin layer of fibro elastic tissue. The mesothelium usually forms a continuous layers, but is may be fenestrated. Subepithelial connective tissue may also contain, macro phages lymphocytes, adipocytes. Mesothelial cells may transform in to fibroblast, which may play an important role in the formation of peritoneal adhesion after surgery or inflammation.

Peritoneal cavity contains a small amount of sterile serous fluid of around 50 – 100 ml which is a transudate. This fluid lubricates the two layers of peritoneum and allows the mobile viscera to glide, freely on the abdominal wall and each other with in the limits dictated my their attachment.

It contains water electrolytes and solutes derived from interstitial fluid in the adjacent tissues and from plasma in local blood vessels. It normally contains few cells, including desquamated, epithelium, peritoneal macrophages, mast cells, fibroblasts, lymphocytes and leukocytes.

Macrophages freely migrate between the peritoneal cavity and the surrounding connective tissue.

Lymphocytes play a major role in cellular and Humoral defence mechanism with in the peritoneal cavity.

The peritoneal fluid is finally absorbed into lymphatic circulation via peritoneal surface and through subdiaphragmatic lymphatics by the virtue of negative intra thoracic pressure during inspiration.

The peritoneum and peritoneal cavity responds to infection or injury in the following ways[2] :

1) Bacteria are rapidly removed from the peritoneal cavity through the sub diaphragmatic stomata and lymphatics.
2) Peritoneal macrophages releases proinflammatory mediators that promotes leukocyte migration.
3) Degranulation of peritoneal mast cells, releases histamine and other vasoactive products causing local vasodilatation and the extravasation of protein rich fluid containing complements and immunoglobulin.

Functions of peritoneum[4]

Peritoneum serves various functions:

- Pain perception - parietal peritoneum is richly supplied with nerves and when irritated causes pain.
- Visceral lubrication — the peritoneal fluid secreted in peritoneal cavity lubricates the cavity and allows easy movement of peristalsis.

- Fluid and particulate absorption- peritoneum has large capacity to absorb fluid, this ability is used during peritoneal dialysis in the treatment of renal failure.
- Inflammatory and immune responses.
- Fibrinolytic activity - Normally mesothelial cells are rich source of plasminogen activators. That is why blood shed in peritoneal cavity does not clot.

Functions of Omenum:

It was earlier thought that the omentum possesses inherent motility that allows it to seek out and contain the intra – abdominal infection. Recent studies has shown, that, though the omentum is found at the site of intraabdominal pathology, it has no spontaneous or amoeboid activity and the displacement of the omentum is a consequence of intestinal peristalsis, diaphragmatic excursion and postural changes. In case of an intra-abdominal focus of inflammation the overlying omental macrophages are activated and mobilised. Fibrinous adhesions develop between the inflammatory focus and the omentum, resulting in tethering of the omentum to the site, thereby isolating the focus of inflammation. Therefore the omentum apparently seems to move towards a site of inflammation within the peritoneal cavity and is hence called the ‘policeman of the abdominal cavity’. Having moved to the site of inflammation the omentum along with surrounding loops of paralysed

bowel forms a cavity plastered by fibrinous adhesion, within which a pathological site is confined and walled off from the general peritoneal cavity and the infection is localised. In addition the rich lymphatic supply of the omentum helps in clearing infection, and its vascularity promotes angiogenesis in compromised tissue.

PATHO PHYSIOLOGY[3]

Peritonitis[3] is an inflammatory or suppurative response of Peritoneum and peritoneal cavity (Mesothelial or vascular endothelial injury). It can result from, loss of mucosal integrity of Gastro intestinal organs or Genito urinary system or it may also be due to other inflammatory or infective conditions.

The inflammatory response may be

- Generalized response or
- Localized response depending upon the severity of the inciting agent and the immunity of individual host.

Localized Peritonitis[3,4] :

Localized peritonitis occurs when the contamination is well contained. It is characterized by the release of vasoactive amines, serotonin, bacterial endotoxins, which results in increased capillary permeability and vasodilatation.

As a result fibrin deposition occurs around the site of injury and within the peritoneum. The fluid which is initially transudate sooner becomes exudate with the influx of protein, neutrophils, macrophages, immunoglobulins.

Intact mesothelial cells are bound to have fibrinolytic activity by the ability to secrete tissue plasminogen activator which looses its

property once it got injured resulting in excess fibrin deposition by the activation of intrinsic coagulation path way which further restricts the spread of Bacteremia by the formation of fibrinous adhesion.

Generalized response[3,4]

The inciting injury or agent is severe and not contained by host resulting in varied systemic manifestation involving CVS, RS, renal systems.

Cardiovascular system

Hypovolemia

As a result of severe third space fluid loss, there is reduction in cardiac output, manifested as Hypotension and tachy cardia.

Renal system

Acute renal failure & Acidosis Electrolyte disturbances

Due to massive 3rd space fluid loss and reduced renal perfusion results in reduced Glomerular filtration, decrease elimination of nitrogenous wastes and H_2 ions, manifesting as ARF and metabolic acidosis, electrolyte disturbances like Hyponatremia and Hyperkalaemia.

Respiratory system

ARDS & Acidosis

Because of reduced respiratory effort of the patient, basal atelectasis as well as tissue hypoxia develops, resulting in Anaerobic glycolysis and acidosis which progresses into ARDS.

BACTERIOLOGY[4]

Disruption of the gastrointestinal tract is the most common cause of peritonitis, although contamination occurs from a variety of other sources including penetrating trauma, secondary contamination from other infected viscera or septicaemia.

The level of perforation in the gastrointestinal tract often determines the type of organism present. Stomach and duodenum are normally sterile owing to presence of gastric acid. Gastric ulcer perforations associated with hypoacidity often lead to infection with gram-positive oral anaerobes, candida species and occasionally gram-negative bacilli. On progressing down the gastrointestinal tract, the flora gradually changes to that of the colon. Both the terminal ileum and colon contain more than 400 different species of bacteria at a concentration of 10^{12} bacteria per gram, with anaerobes outnumbering aerobes by more than 100:1. The most common aerobic pathogen in the colon is Escherichia coli. Other gram-negative facultative organisms include klebsiella, proteus and pseudomonas. The principle gram-negative facultative organism is the Enterococcus. Bacteroides Fragilis is being increasingly recognized as the principle anaerobic pathogen in intra-abdominal Infections. Other significant anaerobic pathogens include Clostridia, Peptostreptococci, Peptococci, Fusobacteria and Velionella**.**

Bacterial peritonitis is polymicrobial containing both aerobic and anaerobic organisms. These organisms gain access to peritoneal cavity via gastrointestinal source or non gastrointestinal source.

Gastro intestinal source[4]

Among the Gastro intestinal source Gram –ve, non sporing aerobes like E.Coli, Klebsiella, Streptococci, Bacteroides, Staphylococcus, is the most common organisms.

Non- Gastrointestinal Source[4]

Chlamydia, Gonocci, Beta-hemolytic, Streptococci, Pneumococci and mycobacterium Tuberculosis. These organisms reach the periotoneal cavity via fallopian tube are responsible for non-gastrointestinal infections of peritoneum.

In immune compromised individuals HIV & Mycobacterium avium intracellulare is responsible for peritonitis.

Primary (spontaneous) Bacterial peritonitis[3] is usually mono microbial and it is due to pure infection of streptococcal, pneumococcal and Hemophillus bacteria

Paths to peritoneal infection[4]:

Bacteria can reach the peritoneum via following routes.

1. Gastrointestinal perforation.
2. Exogenous contamination from drains, trauma, open surgery.
3. Transmural bacterial translocation for eg. Inflammatory bowel disease, appendicitis, ischemic bowel.
4. Female genital tract.
5. Rarely through haematogenous spread eg. Septicaemia.

TYPES OF PERITONITIS

Primary bacterial peritonitis[3]

Mostly seen in patients with ascites due to systemic disorders like cirrhosis, nephrotic syndrome or congestive cardiac failure. It is usually monomicrobial due to aerobic organisms like E.coli and klebsiella pneumonia, streptococcus, staphylococcus.

It is mainly due to bacterial translocation via gut mucosa, but may also be due to haematogenous spread.

Diganosis is usually made by demonstrating >250 neutrophils/ cu.mm of asciticfluid.

Treatment is by 3rd generation cephalosporins.

Tuberculous Peritonitis[3,4]

It may present as acute or chronic form.

Acute tuberculous peritonitis usually present as acute emergency for which laparotomy may be done.

Chronic tuberculous peritonitis usually presents with abdominal pain, fever, weight loss, ascites, night stress.

Origin of infection is via

- Mesenteric adenitis
- TB of ileo ceacal region
- Tuberculous pyosalpinx
- Hematogenous

Both these form have caseating peritoneal nodules. There are 4 varieties of tuberculous peritonitis.

1) Ascitic form
2) Encysted form
3) Fibrous form
4) Purulent form.

These patients are usually managed by ATT drugs (isoniazid, Rifampicin daily for nine months). Surgery is indicated only if obstruction or perforation is suspected.

Granulomatous peritonitis[4]

Certain products such as Talc, starch powder, cellulose fibres from disposable surgical gloves elicit severe granulomatous reaction in peritoneum.

Clinically patients presents with pain abdomen, nausea, vomiting, ileus and other systemic complaints.

Once suspected, it should be treated with cortico steroids (IV methyl prednisolone) or other anti inflammatory agents. Reoperation is usually unnecessary.

Tertiary Peritonitis[8]

It is defined as persistent or recurrent intra abdominal infections following adequate treatment of primary or secondary peritonitis. usually occurs after any abdominal surgeries due to super added infections.

Peritonitis associated with CAPD

Renal failure patients are chronic ambulatory peritoneal dialysis are more prone to develop peritonitis, incidence is about 1 episode every 1 to 3 years.

Patients will have pain abdomen, temperature and cloudy peritoneal dialysate with >100 leukocytes/mm^3 with >50% neutrophils.

Most common etiologic agent is coagulase negative staphylococcus aureus, less commonly due to staphylococcus aureus, gram negative bacilli or fungi.

Usually treated with intra peritoneal instillation of 1^{st} generation cephalosporins.

CAUSE OF SECONDARY PERITONITIS[3]

Secondary bacterial peritonitis is usually contamination of peritoneal cavity due to disruption in integrity of the Gastrointestinal tract (usually due to perforation of hollow viscus), but it may also be due to infection spreading from the genito urinary tract.

Causes of secondary peritonitis :

• It may be either traumatic or non-traumatic

Non-Traumatic:

- In stomach and duodenum:
  - Acid peptic disease: acute peptic ulcer, chronic gastric ulcer, acute erosive astritis.
  - Malignancy: carcinoma stomach
- *In small intestine:*
  - Acute inflammatory disease: typhoid, necrotising enterocolitis, non-specific enteritis.
  - Chronic inflammatory disease: tuberculosis, Crohn's disease.
  - Vascular: ischemic enterocolitis.
  - Miscellaneous: parasitic peritonitis (due to perforation by round worm), strangulated hernia, diverticulitis, radiation enteritis.

❖ In large intestine:

- Acute inflammatory disease: acute appendicitis, acute amoebic dysentery.
- Chronic inflammatory disease: ulcerative colitis, Crohn's disease.
- Malignant perforation.
- Vascular: ischemic colitis.
- Miscellaneous: radiation enterocolitis, diverticular disease.

Traumatic

❖ Blunt injury.

❖ Penetrating injury: fire arm wounds, stab injuries.

❖ Sharp foreign bodies.

❖ Injuries due to corrosive acids and alkalies.

CLINICAL FEATURES

Peritonitis due to perforation of hollow viscus of GIT is the most common type and is called as secondary peritonitis. Intra-abdominal infection initiates a sequence of responses primarily from the peritoneum, the body fluid compartments and the bowel, and secondary endocrine, cardiopulmonary, renal and metabolic responses which form the frame work for clinical signs and symptoms. The aphorism that states "The diagnosis of the peritonitis is made by clinical evaluation", stresses the importance of a thorough knowledge of the clinical signs and symptoms.

Symptoms of Peritonitis[4]

Abdominal pain is almost always the predominant presenting symptoms. The historical characteristics of abdominal pain vary widely with the causative disease process. Initially with stimulation only via the autonomic nervous system the pain may be of a desultory nature, but with stimulation of the parietal peritoneum the classical features set in. the pain is burning, constant, unrelenting and aggravated by movement or motion and is most intense in the region of the most advanced peritoneal inflammation. Increasing intensity points to spreading diffuse peritonitis whereas diminishing intensity suggests localisation. In post operative peritonitis, pain presents as just discomfort. The patient often prefers to remain still recumbent.

Anorexia is almost always presents, **nausea** and possibly **vomiting** may be present. In early stages of peritonitis nausea and vomiting are reflex in origin, later it may be toxic, but in final stages it is caused by paralytic ileus where vomitus changes character from gastric to bilious to finally faeculent.

As a result of the extensive hemodynamic alterations the patient may also complain of thirst and high coloured urine or oliguria secondary to shock. Quite often, the patient presents with **abdominal distention** due to intra peritoneal fluid sequestration and concomitant ileus. Bowels are usually constipated though pelvic peritonitis may cause diarrhoea.

The patient may also complain of **high grade fever** which may or may not be associated with chills and rigors. The patient may complain of palpitations, increase sweating, tiredness and also decreased or absent urine output.

Systemic Signs

At first glance the patient appears anxious, reluctant to move and once he made to lie on the bed he tends to lie still with legs drawn up, the temperature which is initially normal or subnormal tends to rise gradually as peritonitis sets in. Fever associated with rigors signals bacteremia. Fever tends to be spiking ranging between 38^0 C and 41° C in young healthy patients while older or debilitated patients exhibit only a modest febrile response. Secondary to hypovolaemia there is tachycardia. In case

of septic shock in the warm phase the patient may have tachycardia, hypotension and warm pink extremities. In the late cold phase of septicemic shock there is tachycardia, hypothermia, cold and clammy extremities. The tongue is furr coated and dry as a result of dehydration. Respiration is typically rapid because of increased oxygen demand and the need to offset developing acidosis but at the same time it is shallow due to reflex diaphragmatic splinting in order to avoid exacerbation of abdominal distention. Acidotic breathing is seen with time.

Local Signs of Peritonitis :

Inspection

The abdomen is usually distended. There will be a marked diminution of abdominal respiratory movements, and marked retraction of the lower abdomen.

Palpation

Tenderness is present over the entire extent of peritoneum involved in the inflammatory process, and is usually maximal in the region of the organ in which the process originated. In case of salpingitis and pelvic appendicitis rebound tenderness may be better elicited by per vaginal or per rectal examination.

In case of morbid obesity, rigidity may be difficult to elicit.

Fothergill's sign — which is decrease in tenderness on deep palpation with the abdominal muscles contracted helps to confirm rigidity

secondary to diffuse peritonitis rather than due to intramural cause of tenderness and spasm such as rectus sheath hematoma.

Tenderness can also be elicited by direct percussion with less patient discomfort. This method is sometimes more accurate than palpation in defining the point of maximal tenderness and the extent of peritoneal irritation. **Bapat's sign** or the **bed shaking tests** demonstrates the pain on shaking the bed in the presence of peritoneal inflammation. **The cough test** is used to differentiate pleural from peritoneal inflammation as the resulting pain after the cough is localized to the chest in the former and to the abdomen in the later.

Rebound tenderness- pain caused by sudden release of pressure by the deeply palpating examining hand is not a very constant or reliable but is indicative of parietal peritoneal irritation.

Of all the signs, rigidity is the most reliable — classically described as **board like rigidity**. Rigidity can be focal when the inflammatory process is localised or it may be generalised with diffusion. All these physical findings may be concealed or obscured in patients receiving analgesics or corticosteroids or who have altered consciousness due to head injury, toxic or metabolic encephalopathy or in case of spinal injury. In post operative peritonitis, incisional tenderness and operative peritoneal injury may confuse the diagnosis.

Percussion

The abdomen is resonant and tympanic because the intestines are filled with gas. In hollow visceral perforation with release of intraperitoneal gas, there may be **obliteration of liver dullness** , though this sign is not always reliable. In addition the flanks are usually dull.

Auscultation

Intestinal sounds are diminished from the onset. They may even be absent over the site of primary pathology. In well established peritonitis, ileus supervenes and the abdomen is ominously silent except for occasional tinkles.

Per Rectal Examination

Examination is incomplete without a rectal examination — to detect a pelvic collection or mass and a vaginal examination in adult females.

Clinical Presentation at a late stage

This state emerges with a longer duration of peritonitis when intestines have become paralysed, intensely inflammed and inflated with gas. The patient shows signs of peritonitis, toxaemia, ileus and oligemia. The pain becomes exhausting, and vomiting becomes regurgitant, effortless and profuse. The, bounding pulse turns quicker and weaker. The temperature may rise but more frequently falls precipitously. Constipation is absolute with no faeces or flatus.

The hippocratic facies[4] — hollow and sunken eyes that appear inquisitive and bright; drawn, anxious, pale, pinched and blotchy face, cold perspiration covering the brow, heralds stage of despire and lost hope. The lips are cyanotic, the tongue is dry, brown and fissured. The whole body is icy cold and clammy and the abdomen is distended, tympanic, tender and rigid. On auscultation only the transmitted feeble quickened heart beat and laboured breath sounds can be heard.

DIFFERENTIAL DIAGNOSIS

Various types of surgical and non surgical conditions should be differentiated from perforative peritonitis.

Surgical condition

- Acute pancreatitis

  Pain radiating to back is the characteristic feature, Serum amylase may be elevated enormously.

- Acute cholecystitis

  Pain localised to right hypo chondrium with flatulent dyspepsia.

- Acute Intestinal obstruction

  Pain abdomen & distension will be an early feature.

- Acute appendicitis.
- Acute pyelonephritis
- Other types of peritonitis (Tubercular, Primary peritonitis)
- Diverticulitis
- Ruptured aortic aneurysm

In females:

- Acute salphingitis
- Ruptured/ twisted ovarian cyst.
- Ruptured ectopic pregnancy.

Medical condition

- Acute myocardial infarction
- Pericarditis
- Lung pathologies like Basal pneumonia, pleuritis, atelectasis,
- Herpes zoster
- Familial mediterrean fever
- Diabetic acute abdomen in diabetic ketoacidosis.

Pseudopneumoperitoneum:

A number of conditions have been described which simulate free air in the peritoneal cavity i.e. pseudopneumoperitoneum. These are important failure to recognize them may lead to an unnecessary laparotomy in search of a perforated viscus. These are

- ❖ Chilaiditi syndrome: is distended bowel, usually hepatic flexure of the colon, interposed between the liver and the diaphragm.
- ❖ Sub diaphragmatic fat
- ❖ Curvilinear pulmonary collapse.
- ❖ Uneven diaphragm-
- ❖ Distended viscus
- ❖ Subphrenic abscess

INVESTIGATIONS

Haematological

- Hb%, PCV
- Complete Hemogram.

  usually leucocytosis will be present
- Grouping and Rh typing.

Biochemical

Blood – Urea and Glucose.

Serum – Creatinine, electrolytes, amylase

Urine – Albumin, Sugar, deposits.

Microbiological

1) Culture of peritoneal fluid aspirates.
2) Blood widal – in suspected case of Typhoid ileal perforation.

Radiological investigation

Plain x-ray abdomen Erect APview:

A ground glass appearance with obliteration of the flank fat lines and psoas shadow may be seen, inflammatory exudate and edema of the intestinal wall produce widening of space between adjacent loops of bowel. A lateral view may reveal superimposed calcification of the pancreas, indicating pancreatitis.

Free intra peritoneal air is seen secondary to hollow viscous perforation or in cases of established peritonitits associated with gas forming organisms such as E.Coli Air can be made to percolate subdiaphragmatically or between the right edge of the liver and lateral right hemidiaphragm by keeping the patient in the erect or left lateral decubitus positions respectively for about five to ten minutes prior to x-ray exposure. With the upright posture quantities as small as 1-2 cc can be detected. In patients with wide hips the pneumoperitonium can be looked for in the right pelvis in the left lateral decubitus position. Air under the diaphragm is demonstrated in about 60%to 80% of the cases in perforated duodenal ulcer.

Most critically ill patients may not be able to maintain the up right or decubitus position for five to ten minutes. In this case a supine film can be taken and it may show.

1**. Rigler's sign**: Air on both sides of the intestinal wall

2. Air in the right upper quadrant, around the liver.

Late signs include

1. **Falciform ligament sign**: Air outlining the falciform ligament.

2**. Football sign**: Air outlining the peritoneal cavity

3. **Inverted V sign**: Air outlining the umbilical ligaments and are seen in massive pneumoperitoneum. The supine radiograph has an overall sensitivity of 59% for free air.

As Pneumo peritoneum is demonstrated in only 60%–80% of cases, its absence does not rule out perforation.

Plain X-ray chest

To differentiate Medical causes like lobar pneumonia, basal pneumonia.

Free air under diaphragm may also be demonstrated.

Contrast Studies:

X-ray abdomen after injection of 60ml of 50% gastro graffin into the stomach through a nasogastric tube may demonstrate the site of perforation especially in high gut perforations. Barium is not used as it has an adjuvant effect, aggravating peritonitis.

Peritoneal Diagnostic Aspiration

- Bile stained aspirate indicates perforated peptic ulcer or gall bladder.
- Aspiration of frank pus is seen in Bacterial peritonitis.
- Bloody aspirate reveals Hemoperitoneum in blunt or penetrating injuries of the abdomen.

This is not done in all cases, usually done only when diagnosis is doubtful.

Computerised Tomography

CT scanning has been found to be significantly more sensitive than plain films for detecting small amounts of free air. In addition it is a sensitive study for a wide variety of diagnosis including appendicitis, diverticulitis, intestinal ischemia, pancreatitis, intestinal obstruction and perforated viscous which could have precipitated the peritonitis. It is also very sensitive for intra abdominal abscess.

Ultra Sonography

Demonstrates free fluid with in the abdominal cavity with dilated fluid filled bowel loops because of paralytic ileus

Peritoneal biopsy

Peritoneal biopsy is used to make a diagnosis of granulomatous peritonitis. It is not a routinely done investigation. It is done preoperatively when operative findings suggest a possible granulomatous etiology for the peritonitis. The peritoneal biopsy in no way affects the surgical procedure of choice.

MANAGEMENT

The management protocol in perforative peritonitis include

- **Initial Resuscitation**
    - Correction of fluid loss & Electrolyte disturbances
    - Nasogastric decompression
    - Broad spectrum systemic antibiotics.
    - Analgesics.
- **Operative management**

Correction of fluid loss & Electrolyte disturbances[4]

Patients with perforative peritonitis, generally present with features of hypotension because of third space fluid loss, and also they have profound electrolyte disturbances in the form of Hyponatremia, Hypo/Hyperkalemia. Hence their plasma volume must be maintained with adequate and appropriate fluid therapy with crystalloids & colloids. This is achieved my placing central venous catheter and central venous pressure monitoring using (Swan – Ganz Catheter) especially in patients presenting with septic shock, and patients with poor cardio pulmonary and renal reserve.

Nasogastric Decompression[3] :

Nasogastric tube passed into the stomach provides absolute rest to the bowel. And intermittent Nasogastric aspirations done until paralytic

ileus resolves. This also has an additional advantage of preventing the patient from Aspiration of Gastrointestinal contents into lungs.

Systemic Broad spectrum antibiotics :

Most of the perforative peritonitis is Polymicrobial, early administration of systemic (Parenteral) Broad spectrum antibiotics which covers, both aerobic and anaerobic organisms prevents the multiplication of the Bacteria and release of endotoxins. The antibiotics include 2^{nd} (or) 3^{rd} generation cephalosporin, Amoxycillin – Clauvalanic acid and AminoGlycoside. The imidazole group of antibiotics (metronidazole) is effective against anaerobic organisms.

This is continued post operatively until fever settles down and total count returns to normal.

Analgesics

Pain control is absolute necessary in the management of perforative peritonitis in the preoperative as well as postoperative periods. Adequate Analgesia, in the form of Morphine, Pethidine or by other pharmacological agents produces good symptomatic relief, which allows early mobilization of the patient, which prevents deadly postoperative complications like Basal atelectasis, DVT and pulmonary embolism.

Input – Output Monitoring

This is done by continuous bladder drainage and continuous Ryles tube Aspiration. The insensible water loss is corrected by administering fluid in a phased manner.

Operative management

Surgical control of the infecting organism is the main stay of treatment, if the cause of peritonitis is amenable to surgery as in the case of perforative peritonitis.

The operative procedure is dictated by the site, size and precipitating factors that disturbs the Gastrointestinal Mucosal integrity.

The operative control of sepsis is Primarily directed towards the control of over whelming sepsis.

Main objective for surgery for perforative peritonitis is to resolve all the infected material, to treat the underlying cause and to prevent late complications.

Except in specific situations like localized peritonitis, this is best achieved through midline incision. The primary disease is then treated depending upon the aetiology such as Resection of the perforated viscous (in the case of perforated Appendicitis and perforated Gall bladder). Simple closure of peptic ulcer perforation (Gastric or Duodenal ulcer), Resection anastomosis (ischemic necrosis due to compromised vascularity as in the case of vascular occlusion or

intussusception),resection anastomosis or diversion colostomy (for large bowel disorders)

Reduction of Bacterial load :

This is achieved by identifying the occult pockets of infection, by thorough exploratory laparotomy and removal of infected, contaminated Necrotic Materials.

Peritoneal Lavage[4] :

In patients with diffuse peritonitis, large copious amount 2 to 3 litres of crystalloid solutions (Normal saline) is irrigated into the peritoneal cavity, which removes the bacteria, blood and fibrin clot and dilutes the residual bacteria. Studies have shown that, addition of antibiotics or antiseptics, is useless and it may be even Harmful by causing fibrinous adhesions.

PERITONITS DUE TO INDIVIDUAL ORGAN PERFORATION AND ITS MANAGEMENT.

Peptic Ulcer perforation

Peptic ulcer perforation is one of the most common surgical emergency which requires hospital admission and management. Recently the incidence of peptic ulcer perforations was drastically reduced, because of the introduction of anti-secretory agent.

H. Pylori infection and analgesic (Nonsterioidal antiinflammatory drugs – NSAIDS Abuse) is the most common etiology associated with perforation of peptic ulcer. Other factors which may also be associated with perforative peptic ulcer are smoking, alcohol and certain physiological stress full condition, such as following massive burns major surgeries etc.

Perforation of a peptic ulcer disease can occur either at the level of Gastric or 1st part of Duodenum.

Incidence of Peptic ulcer in males & females is 1 : 1 for Gastric ulcer and 2 : 1 for Duodenal ulcer. Most duodenal ulcer are Benign and malignancy is seen in <5% of cases, unlike Gastric ulcer which is mostly malignant .

Peptic Duodenal ulcer perforation

Peptic Duodenal ulcer usually occurs at the 1st part of duodenum 1–2 cm distal to the pylorus, in more than 90% cases found with equal frequency on anterior and posterior wall. The anterior wall ulcer is the one which usually perforates and posterior one usually bleeds. Duodenal ulcers that usually occurring beyond 1st part is usually due to rare causes such as Zollinger Ellison Syndrome, crohn's disease, malignancy or drug induced ulcers in which cases it is multiple.

Most 80% of patients with perforated duodenal uncer will be previously symptomatic and 20% will have Asymptomatic chronic duodenal ulcer.

Peptic Gastric ulcer perforation

Gastric ulcer perforation is significant that, it carries significant mortality than perforated duodenal ulcer which is about 15 – 20%. Most gastric ulcer perforation are prepyloric. Incidence of malignancy is higher in Gastric ulcer compared to Duodenal ulcer as mentioned earlier.

Peptic Ulcer Perforation:

It can be described in 3 stages:

a) Stage of peritonism[9] : It is due to leak of gastric juice in the peritoneal cavity causing chemical peritonitis lasting for about 6hrs.patient usually gives a previous history of peptic ulcer, sudden pain

over epigastrium. The pain may gradually gravitate down along the paracolic gutter to the right iliac fossa.

Vomiting may or may not be present. On examination, there will be little change in pulse, respiration and temperature. Tenderness and guarding are constantly present over the site of perforation and there will be no peristalisis.

b) Stage of reactionary peritonitis[9] : The irritant fluid becomes diluted with peritoneal exudates. Symptom diminishes and patient feels comfortable.

Rigidity continues to be present. There will be obliteration of liver dullness and shifting dullness. Rectal examination may elicit tenderness in rectovesical or rectovaginal pouch.

c) Stage of diffuse peritonitis[9] : "Facies hippocratica"- pinched and anxious looking face, sunken eyes and hollow cheeks. Tachycardia with low volume pulse, persistent vomiting, board like rigidity of the abdomen, abdomen distension, obstipation, diffuse tympanicity, features of septic shock with onset of multi organ failure.

Surgical Treatment

Peptic Gastric or duodenal ulcer perforation should be surgically intervened as soon as possible because unnecessary delay increases the morbidity and mortality, especially in elderly. Surgical approach is usually through midline incision. After clearing out the peritoneal

exudates thorough laparotomy was done, including, post wall of stomach, following procedures are performed.

Surgery for Perforated Duodenal ulcer

The perforated duodenal ulcer closure was described by Graham. The two principal techniques used in closure are

1) Simple opposition of the perforation

2) Omental patch technique

In cases of large perforation or the scarred, inflexible duodenal wall that makes simple closure difficult two options are available.

a) Conversion of the perforation into a Heineke-Mikulicz pyloroplasty.

b) Serosal patch with proximal Jejunum

Laparoscopic approach [13,14]

Laparoscopic techniques have been applied to virtually all abdominal procedures and perforated duodenal ulcer is no exception. It was introduced by Nathanson in 1990.

Two laparoscopic approaches have been developed

a) Suturing technique

b) Fibrin plug technique

Laparoscopy seems particularly useful for patients without pneumo peritoneum, or for those who present with atypical symptoms and signs. Postoperative pain is reduced , allowing early mobilization and rapid

return to daily activities. Laparoscopy significantly decreases the rate of postoperative chest complications and shortens the duration of hospital stay .

Recommended definite surgical procedure for perforated duodenal ulcer

First choice	Second choice	Third choice
PGV with Closure	TV with Drainage	Simple closure

Conservative management

In few patients with peptic ulcer perforation, on rare occasions conservative management may be beneficial particularly in patients who have concomitant medical illness, perforation of more than 24 hrs, systolic pressure less than 100 mm Hg at the time of admission. These risk factors have definitive bearing on mortality rate. These patients require close monitoring in intensive care unit as they may deteriorate and they may need operative therapy. If abdominal findings do not improve in 12 hours then operation is indicated.

Can be attempted in patients with

- Localized peritonitis
- Haemodynamically stable patient
- Free peritoneal perforation in gastrgraffin study

Surgery for perforated gastric ulcer

The choice of surgical procedure is decided by the clinical condition , and associated co-morbid disease. Surgical option available are

- Simple closure with four quadrant biopsy
- Excision and closure with live omental patch
- Resection (gastrectomy) procedures

Appendicular perforation :

Patients with perforation of the appendix may be very ill and require several hours of fluid resuscitation before safe induction of general anaesthesia. Broad spectrum antibiotics directed against gut aerobes and anaerobes are initiated early in the evaluation and resuscitation phase.

Appendicectomy must be performed in children whether the peritonitis is diffuse or not, since the other course is associated with mortality. But the management of this problem in adults remains controversy. In patients with diffuse peritonitis after perforated appendicitis appendicectomy is the treatment, as the perforation remains a continuing source of peritoneal contamination.

At operation for free perforation, visualization of all peritoneal surfaces is essential. All purulent and feculent material should be

removed and dependent collection of pus should be aspirated, the peritoneal cavity should be repeatedly rinsed with warm saline solution. The placement of multiple prophylactic drains is unwarranted. Not only do these drains fail to improve morbidity and mortality, but there is some evidence to indicate that they may actually increase it.

Typhoid Perforation[7, 31]

Surgery has been generally accepted as the treatment of the choice, but the purpose of it and its choice is still debated. Typhoid ileal perforation has been treated with surgery and antibiotics (Ceftriaxone/ Chloramphenicol/ Tetracycline).

Conservative treatment

It was advocated by Huckstep as lower ileum is paper thin and is liable to perforate at more than one spot and hence repair of the gut is difficult and suturing may result in further tears.

Surgical treatment

This was favoured by Franklin as closure of perforation eliminates contamination and lessens toxaemia. Surgery is necessary as peritonitis is poorly localised and there is no effort by the omentum to seal off the perforation.

The concept of "double attack" on typhoid pathology and anaerobic infection is given importance. This was a study conducted by S. Vaidyanathan[31].

a. Control of typhoid pathology: Ceftriaxone is the drug of choice.

b. Control of anaerobic infection: best method is to provide early and adequate drainage of pus along with antimicrobial agents.

Types of surgical procedure

1. Simple closure of perforation in 2 layers using catgut for inner layer and vicryl for outer layers (as per study by M.A. Noorani[26]).

2. Simple closure and exteriorisation of the sutured loop. This is recommended in established peritonitis when the entire bowel is inflamed and leak of the suture line is to be expected.

Alternate procedures:

Closure of perforation alone as the surgical treatment has been criticised on the ground that leakage is common after the surgery. To avoid this problem, alternate procedures are described which includes:

- Closure of perforation with ileo-transverse colostomy.
- Resection of the affected loop and end-to-end anastomosis.
- Tube ileostomy.

Peritoneal toilet and drainage is done only if no evidence of perforation is found as this reduces the chances of residual abscess formation and wound sepsis.

Small intestinal(ileum) perforations usually occurs in 1-3% of patients with enteric fever. It usually occurs during 3rd or 4th week of illness usually from hyperplasia, ulceration, necrosis of lymphatic aggregates. Payers patches at the anti-mesenteric border.

Tubercular Perforation[36,37]

It is usually associated with strictures. Simple suture of the perforation is adequate after proper drainage of the peritoneal cavity.

Surgical options:

a. Resection of the perforated segment:

This is indicated only if patient is fit and disease segment is short. The affected segment is resected and continuity is restored by end-to-end Anastomosis[37]

b. Simple closure with bypass of stricture:

Bypass of stricture is either by ileo-ileostomy or ileo-transverse colostomy[37]

c. Stricturoplasty:

Perforation usually occurs proximal to the stricture. These strictures may also be seen in the colon. If the perforation site is just proximal to the stricture, perforation site should be included in the stricturoplasty but if the site of perforation is little away from the stricturous site, the perforation is closed in 2 layers and stricturoplasty done for strictures.

Medical management:

Anti-tubercular drugs are given post operatively as a standard treatment usually by 3 drugs Isoniazid, Rifampin, Ethambutol for duration of 9 months.

Use of corticosteroids: it may be given to reduce the degree of cicatrisation that may occur with healing. High index of suspicion is essential for early diagnosis and optimal treatment of the patients with tubercular intestinal perforations.

DIVERTICULAR DISEASE [38]

Small bowel diverticular disease rare . Both acquired and congenital diverticula are frequently asymptomatic and become symptomatic when complicated by infection, perforation, obstruction or haemorrhage.

Duodenal Diverticula

It may be acquired or congenital. Perforation may be secondary to diverticulitis or iatrogenic following ERCP. It commonly occurs in the retroperitoneum over the right kidney and posterior to the head of the pancreas and duodenum.

When a diverticular perforation is suspected, CT scan of the abdomen with oral and intravenous contrast is very accurate in confirming the diagnosis and in defining the extent of inflammatory reaction.

Prophylactic resection of an asymptomatic diverticulum is not recommended.

In the absence of significant retroperitoneal contamination, primary excision of diverticulum with two layer closure is done. In the case of large duodenal defect, serosal patch technique or a Roux-en-Y duodenojejunostomy is preferred. In the presence of a perforation with significant edema and contamination, a **duodenal diverticulization** (e.g. gastrojejunostomy, closure of the pylorus, closure of the perforation, tube duodenostomy, gastrostomy tube and jejunostomy feeding tube)with drainage of the surrounding area.

Jejunal and ileal diverticula

In the presence of diverticulitis or perforation, resection and primary anastomosis is indicated.

Meckel's diverticulum

Resection of the diverticulum is done where there is induration of the base of the diverticulum extending into the adjacent ileum or resection of the segment of ileum bearing the diverticulum with end to end anastomosis.

Perforation of colonic diverticula[39]

In acute perforation, peritonitis soon becomes general and may be purulent,which has a mortality rate of more than 50% and

pneumoperitoneum is usually present; the diagnosis may not be confirmed until emergency laparotomy.

Treatment options are

1. Primary resection and Harmann's procedure.
2. Primary resection and anastomosis after on-table lavage in selected cases.
3. Exteriositation of the affected bowel which is then opened as a colostomy. d. Suture of the perforation with drainage with or without proximal defunction, in selected cases with a small leak and minimal soiling.

Traumatic Perforation[5]

Traumatic injury in the form of blunt or penetrating injury to the abdomen may also produce hollow viscous perforation leading to perforative peritonitis. The incidence due to trauma is on the rise due to modernisation of world

Management of Traumatic Duodenal Injuries[5]

Small duodenal perforations or lacerations usually treated by single layer closure with 3-0 monofilament suture

Extensive injury involving 1st part of duodenum can be repaired by debridement or end-end anastomosis

Injury involving 2nd part of duodenum is treated b Roux-en-Y duodenojejunostomy

Injury to 3rd part of duodenum is treated by Resection with Roux-en-Y duodenojejunostomy

For complex duodenal injuries, pyloric exclusion procedure with gastrojejunostomy is an ideal option

Traumatic Gastric & Small Bowel Perforation.

Because of the Great distensible capacity of stomach it is one of the most commonly involved organ in traumatic injury

Gastric wounds are usually closed by over sewing the defect in single layer suture with or without omental patch

Distal injuries may require partial gastrectomy or antrectomy with Billroth I or II anastomosis

Small bowel injuries involving <1/3rd of circumference requires resection and end-end anastomosis

Management of Traumatic Colonic Injuries[5]

1. Various modalities of traumatic colonic perforations are
2. Primary repair(lateral surface repair) Resection and end-end ileo-colic or colo- colic anastomosis
3. Primary repair with Diversion colostomy
4. End colostomy

Perforation in Ascariasis:

The perforation is usually in the upper reaches of ileum, a site higher than found in most cases of typhoid perforation. In case of localized intra peritoneal abscess with perforation amidst matted bowel, the bowel is devitalized at places. It is not safe to close the perforation. The correct treatment is to resect the whole affected segment of the bowel. For large perforations on the antimesenteric border, it is safe to suture if diagnosed early. If the gut is unhealthy the affected bowel should be resected. If there are multiple perforations, then resection and anastomosis should be done. otherwise the recent perforation can be tackled by a two layer closure, the outer being the non-absorable material as the left over worms in the gut, may burrow the suture line.

Perforation In Amoebic Ulcers

A diagnostic paracentesis in the early phase is beneficial. In cases of fulminant amoebic perforation of colon in intraperitoneal soiling, laparotomy is advised as soon as possible (after stabilising the patient). Exteriorisation and colostomy or subtotal or total colectomy has been advised. Colostomy can be closed 6-8 weeks after the patient has completely recovered from amoebiasis.

COMPLICATIONS OF PERITONITIS[4]

Complications due to perforative peritonitis occurs pre operatively due to peritonitis it self or post-operatively .

It can be systemic ,local, or abdominal complications

Systemic complications[4] :

1. Septic shock
2. Electrolyte disturbance
3. Respiratory complications
    - Basal pneumonia
    - Basal atelectasis
    - ARDS
4. Acute renal failure
5. Vascular complications like DVT ,Pulmonary embolism
6. Multi organ dysfunction syndrome(MODS)
7. Bone marrow suppression

Local complications[3,4]

1. Surgical site infections
2. Sticth abscess
3. Wound dehiscence & burst abdomen

Abdominal complications[4] :

1. Adhesive small bowel obstruction
2. Residual or recurrent intra abdominal or pelvic abscess
3. Paralytic ileus
4. Portal pyemia and liver abscess

PROGNOSIS[3]

The overall mortality rate of generalized peritonitis is about 40% Mortality rates are below 10% in patients with perforated ulcers or appendicitis.

Factors contributing to a high mortality rate include

- The type of primary disease and its duration,
- Associated multiple organ failure before treatment,
- Age of the Patient
- General health of the patient.
- Time interval between the occurrence of perforation and initiation of treatment - there is approximately a five fold increase in the mortality among the patient who received treatment after 24 hours compared to the patients who reached within 6 hours
- Site of perforation - Mortality is more with distal small bowel or colonic perforation.
- Reduced cardiac reserve and renal reserve
- Preoperative hypo albuminemia
- Poor physiologic indices (eg, APACHE II or Mannheim Peritonitis Index)
- These high-risk patients who require intensive treatment to reduce a daunting mortality.

METHODOLOGY

This study is based on the analysis of 141 patients admitted in various emergency wards in Government Rajaji Hospital, Madurai, with features of perforative peritonitis due to hollow viscous perforation of git and genito urinary organs for a period of one year from Nov 2011 to oct 2012. During this period 141 cases were admitted on emergency basis with features suggestive of perforative peritonitis and underwent emergency laparotomy.

After obtaining ethical clearance from the committee, Patient's and their attendants' consent was taken for all the cases in the study. Cases were selected in the age group of above >13 yrs. Both the sexes were included. Both non-traumatic as well as traumatic perforation were included in this study. A pre-tested pro forma was used to collect the relevant information by history, clinical examination of patients, relevant investigations and treatment.

In all the cases, monitoring of the vital signs with pre-operative correction of fluid and electrolyte imbalance were done and broad spectrum antibiotics were started.

The investigations done in the study cases were as follows:

a. **Blood**: routine examination of haemoglobin along with complete haemogram, blood grouping and Rh typing, renal function tests, serum electrolytes, WIDAL (in suspected cases).

b. **Urine**: estimation of sugar, albumin and microscopic deposits

c. **Imaging**: plain X-ray chest and abdomen (erect) to detect free gas under the diaphragm.

d. **Ultrasound abdomen and pelvis** done to see the presence of free fluid in the peritoneal cavity(in selected cases) and to rule out associated injury to solid viscera (in traumatic cases).

d. **Abdominal Paracentesis** : done only in selected cases (for confirmation in cases where diagnosis is doubtful, and X-ray showed no gas under the diaphragm).

e.**Electro cardiogram** and **Echo cardiogram**(in high risk cardiac patients)

Surgical procedure :

After initial resuscitation and evaluation all patients were kept nil by mouth, nasogastric aspiration was done, adequate intra venous fluid was administered and urine output was maintained ,and broad spectrum antibiotics was usually a third generation cephalosporin and aminoglycosides were administered (if renal parameters normal) .

After stabilising the patient and obtaining the anaesthetic fitness all patients with features of perforative peritonitis either radiologically or clinically were subjected for emergency laparotomy.

Laparotomy was done in almost all the cases under general anaesthesia (appendicular perforations were done under spinal anaesthesia). Incision was taken depending upon the suspected site of pathology and when not confirmed, midline incision was taken. Viscera were inspected and site of Perforation of hollow viscous of git was identified. For peptic duodenal ulcer perforation simple closure of the perforation with live omental patch was done. In almost all cases of gastric perforation and ileal perforation, tissue from the edge of the ulcer was taken and sent for histopathological examination. In cases with large bowel perforation either resection anastomosis or diversion colostomy was done . Thorough Peritoneal lavage with 4-5 litres of normal saline was carried out and peritoneal cavity was drained using chest tube drain. Postoperatively patients were put on nasogastric tube with continuous aspiration, intravenous fluids, and appropriate antibiotics. Proton pump inhibitors (Pantoprazole / rabeprazole) were given in cases of peptic ulcer perforation. Vital signs were monitored along with intake-output chart and biochemical parameters. Recovery was observed and complications which occurred were noted and treated accordingly. Patient was discharged after suture removal. Mortality in this study refers to death of the patient in the hospital during same as episode of peritonitis. Regular follow-up of the patients was carried out for a month.

OBSERVATION OF THE STUDY

During our study period of 1 year from, Nov-2011 to Oct 2012, About 141 patients underwent emergency laparotomy for perforative Peritonitis in various wards of our Govt Rajaji Hospital. The observation from our study was given below.

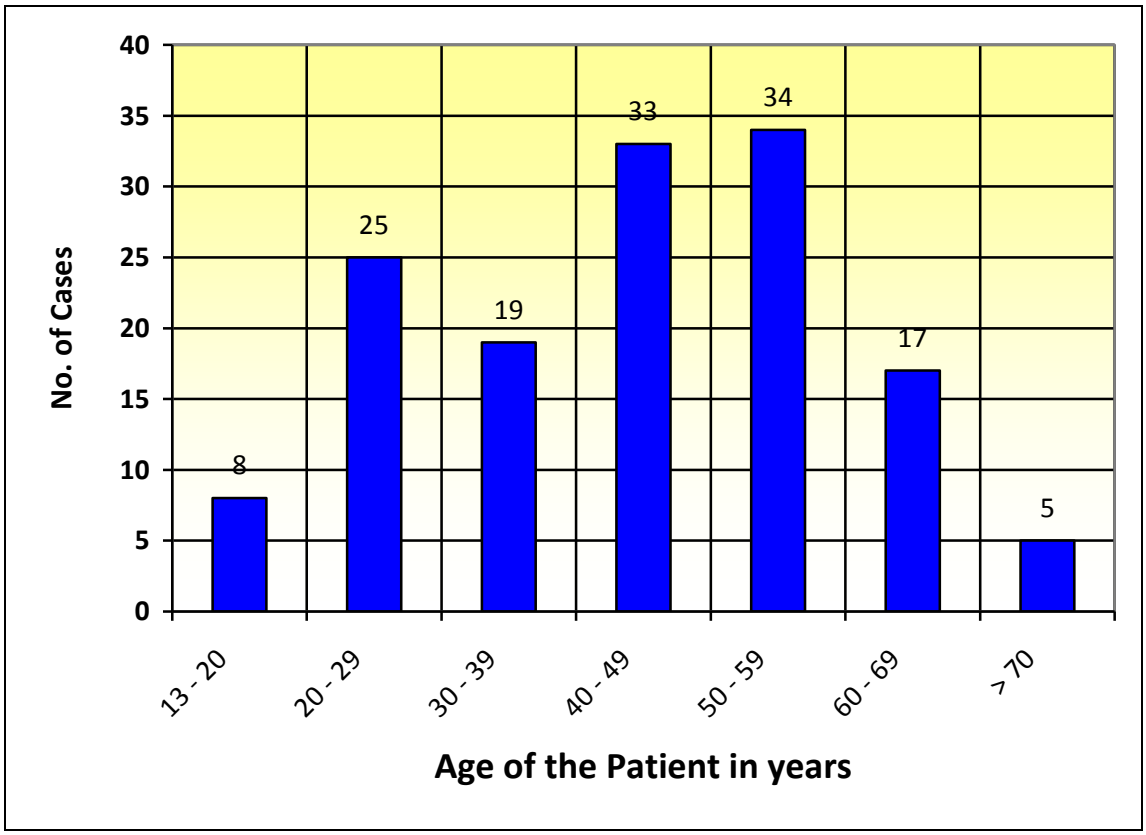
Age Incidence

According to our study, majority of patients, who underwent surgery for emergency laparotomy for perforative peritonitis fall under the age group of 40-59 years which constituted about 47.51%(67 cases) followed by age group of 30 and above .

Table-1: Distribution of Sample by Age

Age in Years	No. of Cases	Percentage
13 to 20	8	5.67%
20 to 29	25	17.73%
30 to 39	19	13.47%
40 to 49	33	23.4%
50 to 59	34	24.11%
60 to 69	17	12.05%
> 70	5	3.54%

Figure-1: Distribution of Sample by Age



Sex Incidence

In our study incidence of perforative peritonitis is higher among male patients which constituted about, 81.56% (115 out of 141 cases) and females about 18.43% (26 out of 141) and the ratio is 4.4 : 1.

Table-2: Distribution of Sample by Sex Incidence

Sex	No. of Cases	Percentage
Males	115	81.56%
Females	26	18.43%

Figure-2: Distribution of Sample by Sex Incidence

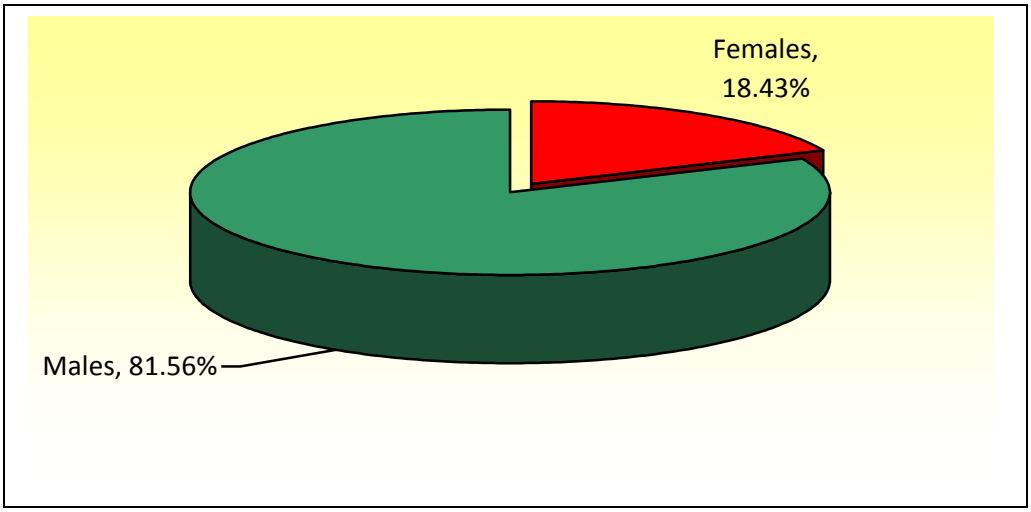


Table-3 : Sex ratio in different studies of Peritonitis

Study	Male : Female Ratio
Illingworth 1971[42]	18 : 1
Rodney Maingot [43]	6 : 1
Our Study	4.4 : 1

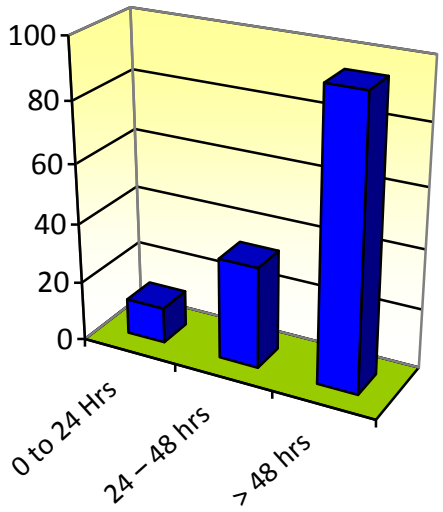
Timing Of Presentation

In our study it is evident that most patients got admitted to the hospital, after a delay of >48 hours from the onset of symptoms and signs of peritonitis, and very few reported in < 24hrs duration. Around 67.37% cases reported to the hospital after a period of > 48 hours from the onset of clinical symptoms of Peritonitis

Table-4: Distribution of Sample by Timing of Presentation

Timing of Presentation	No. of cases	Percentage
0 to 24 Hrs	12	8.51%
24 – 48 Hrs	34	24.11%
> 48 Hrs	95	67.37%

Figure-3: Distribution of Sample by Timing of Presentation



Clinical Features

Classical signs and symptoms of peritonitis were not seen in all cases especially in patients , with early presentation to hospitals and in localised peritonitis.

Abdominal pain and tenderness is present in almost all cases with perforative peritonitis. Most of the cases presented with guarding and rigidity, especially in patients who present late to the hospital

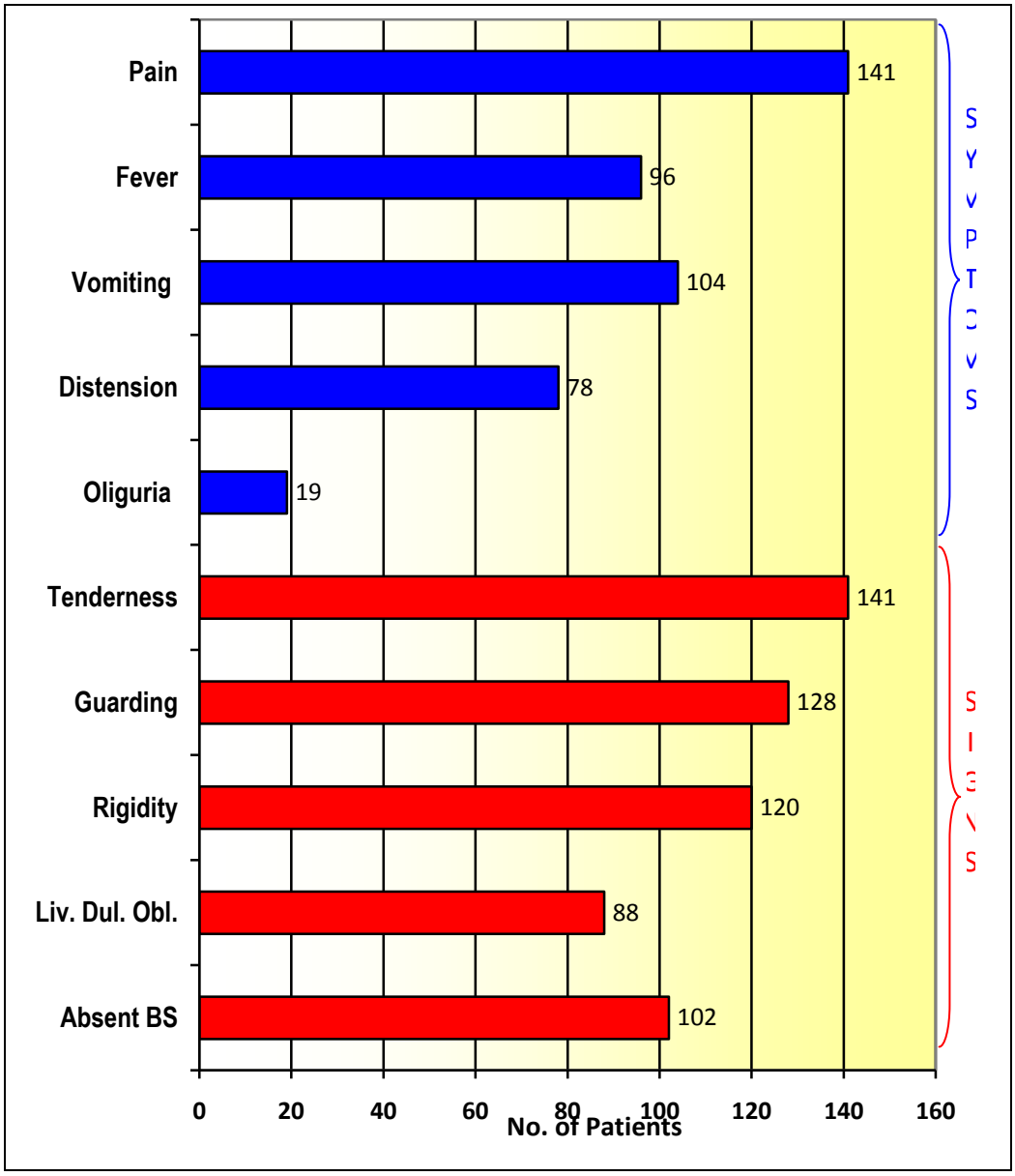
Only few cases 55.31% had abdominal distension. Liver dullness is obliterated in 76.59% and Bowel sounds were absent in 72.34% cases.

Few patients presented with severe dehydration and oliguria(13.47% of cases)

Table-5: Distribution of Sample by Clinical feature

Abdominal Symptoms & Sign	No. of Cases	Percentage
Symptoms		
Pain	141	100%
Fever	96	68.08%
Vomiting	104	73.75%
Distension	78	55.31%
Oliguria	19	13.47%
Signs		
Tenderness	141	100%
Guarding	128	90.78%
Rigidity	120	85.10%
Obliteration of Liver dullness	88	62.41%
Absent Bowel sounds	102	72.34%

Figure-4: Distribution of Sample by Clinical feature



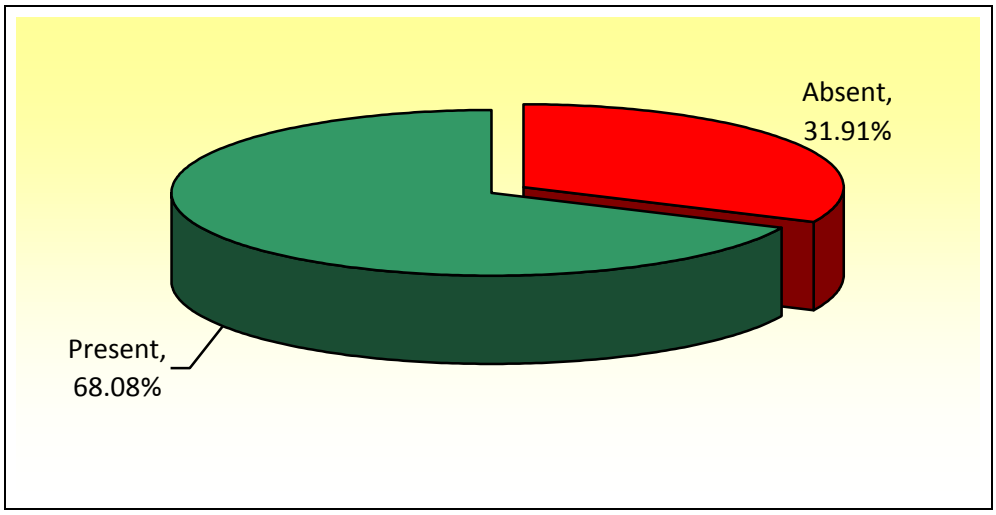
Distribution of Pneumo Peritoneum In X Ray

X ray chest PA view and X ray abdomen erect were taken in all cases with suspected peritonitis. In our study Air under diaphragm were seen in 68.08% of cases.

Tanle-6: Distribution of Sample by Pneumo Peritoneum (In X-Ray)

Pneumo Peritoneum	No. of Cases	Percentage
Present	96	68.08%
Absent	41	31.91%

Figure-5: Distribution of Sample by Pneumo Peritoneum (In X-Ray)



Site of Perforation

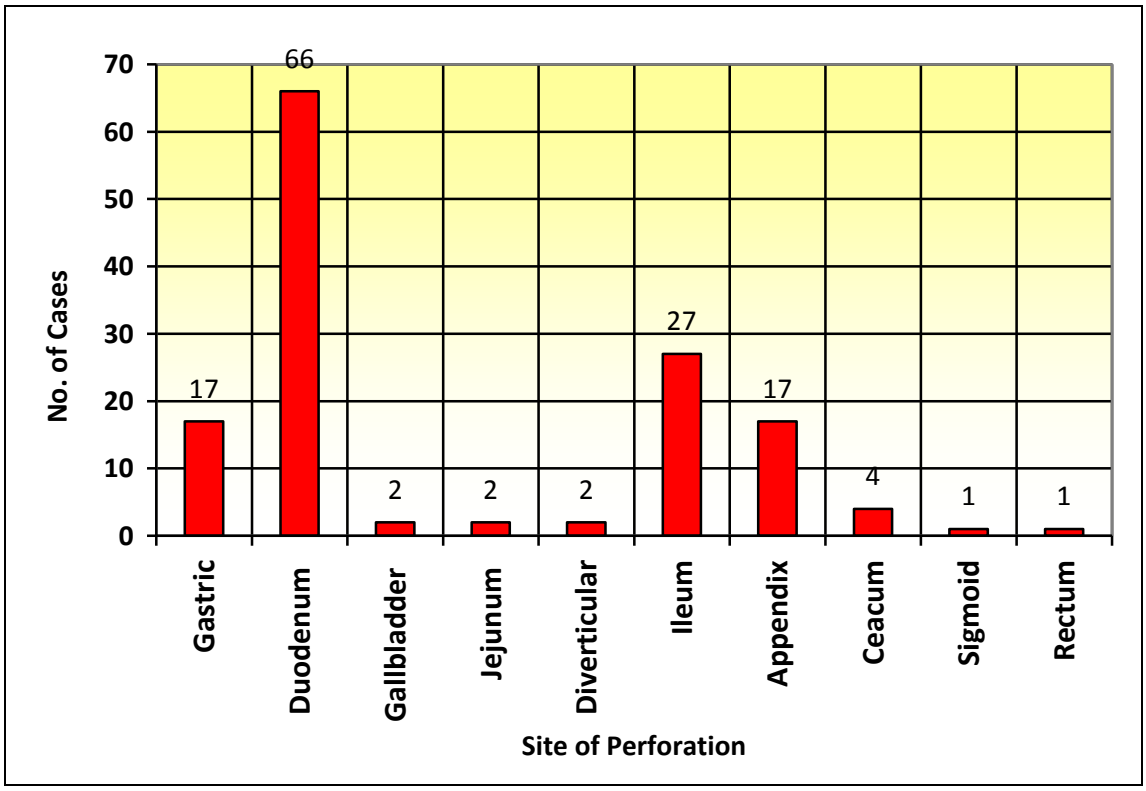
According to our study. Among 141 patients who underwent emergency laparotomy for perforative peritonitis, it was found that Anterior wall of 1st part of duodenum was the most common site, which constituted about 47.51%(67 cases out of 141)

Next most common site for perforation is small bowel(ileum) constituting about 19.14%(27 cases out of 141)

Table-7: Distribution of Sample by site of Perforation

Site	No. of cases	Percentage
Gastric	17	12.05%
Duodenum	66	46.80%
Gallbladder	2	1.41%
Jejunum	2	1.41%
Diverticular	2	1.41%
Ileum	27	19.14%
Appendix	17	12.05%
Ceacum	4	2.83%
Sigmoid	1	0.70%
Rectum	1	0.70%

Figure-6: Distribution of Sample by site of Perforation



Etiology Of Perforation

According to our study, most common etiology that predisposed to perforation of hollow viscous is peptic ulcer, resulting in duodenal ulcer, gastric perforation which constituted about 51.79%(73 cases out of 141).

Next common etiology for perforation of hollow viscous is inflammatory pathology(thphoid, appendicitis, cholecystitis) which altogether constituted about 25.53%(36 out of 141).

We had 1 case of uterine perforation following radiotherapy to ca. cervix. Traumatic etiology for perforative peritonitis was seen in about 8.51% due to both blunt injury and penetrating injury of the abdomen.

We had 1 case of traumatic isolated duodenal laceration involving more than $3/4^{th}$ of circumference for which BERNE's Duodenal diverticulisation procedure was done and patient recovered with minimum postoperative morbidity.

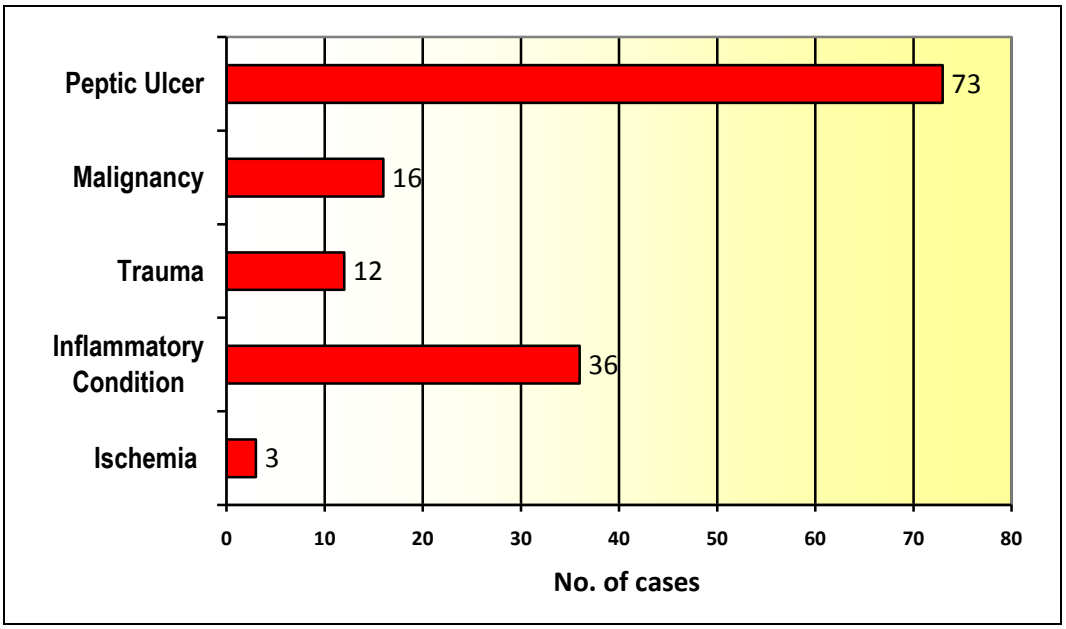
3 cases were due to vascular etiology. Ischaemic necrosis of bowel leading to perforative peritonitis due to SMA thrombosis is seen in two cases 1 case is due to incarcerated ventral Hernia with strangulation.

According to our study, malignant causes for perforation of hollow viscous is seen in around 16 cases constituting around 11.34%.

Table-8: Distribution of Sample by Etiology

Etiology		No. of cases		Percentage
Peptic Ulcer				
	Duodenum	66	73	51.71%
	Gastric	7		
Malignancy				
	Gastric	10	16	11.35%
	Colon	6		
Trauma				
	Duodenum	1	13	8.51%
	Jejunum	2		
	Ileum	9		
	Uterine	1		
Inflammatory Condition				
	Typhoid	6	36	25.53%
	TB	3		
	GB	2		
	Appendix	17		
	Non – Specific inflammatory Condition	8		
Ischemia				
	SMA Thrombosis	2	3	2.12%
	Ventral Hernia with Strangulation	1		

Figure-7: Distribution of Sample by Etiology

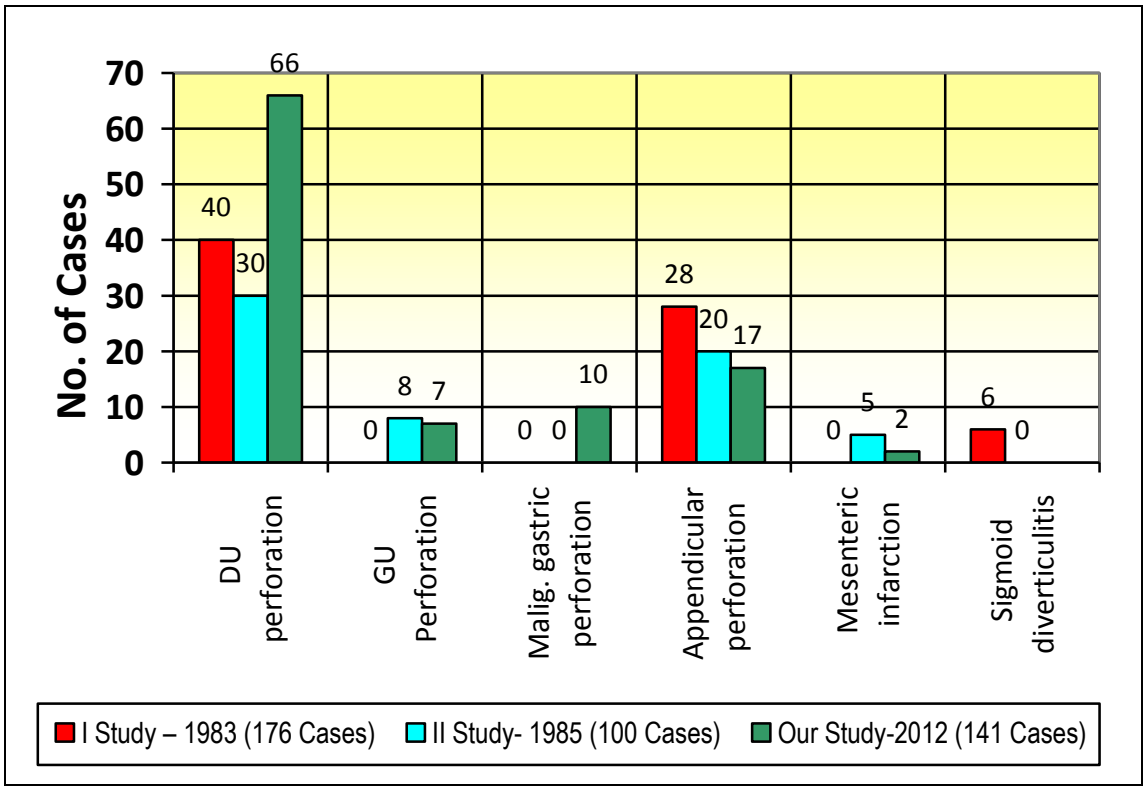


There was a study conducted in 1983by BOHNEN[10] et al based on the statistical data of Royal Victoria hospital regarding the type of perforation, producing peritonitis. Another study was done by R.MCRAWFUND and ELLIS in 1985[41].

Table-9: Comparision of different studies of perforative peritonitis

Site	I Study – 1983 (176 Cases)	II Study- 1985 (100 Cases)	Our Study- 2012 (141 Cases)
DU perforation	40	30	66
GU Perforation	--	8	7
Malig. gastric perforation	--	--	10
Appendicular perforation	28	20	17
Mesenteric infarction	--	5	2
Sigmoid diverticulitis	6	--	--

Figure-8: Comparision of different studies of perforative peritonitis



Complications

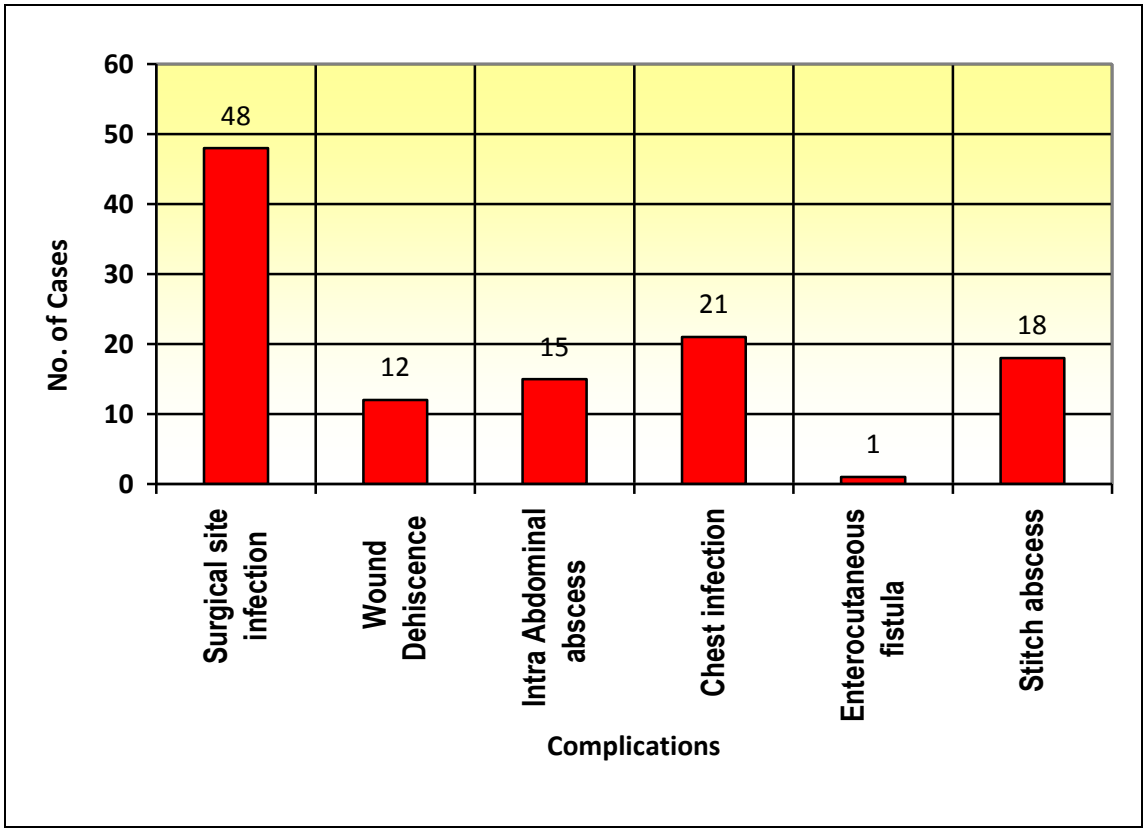
The most complications encountered in our study of perforative peritonitis are Surgical site infection constituting about 34.04% cases, leading onto wound dehiscence, and burst abdomen in about 8.51% cases which required repeat intervention in the form of tension suturing or secondary suturing comprising about 8.51% of cases.

Few cases developed, post operative intra abdominal abscess. One patient who underwent duodenal diverticulisation procedure, for traumatic duodenal laceration, developed enterocutaneous fistula postoperatively whihc closed spontaneously on conservative management.

Table-10: Distribution of Sample by Complications

Post of Complications	No. of cases	Percentage
Surgical site infection	48	34.04%
Wound Dehiscence	12	8.51%
Intra Abdominal abscess	15	10.63%
Chest infection	21	14.89%
Entero cutaneous fistula	1	0.1%
Stitch abscess	18	12.96%

Figure-9: Distribution of Sample by Complications



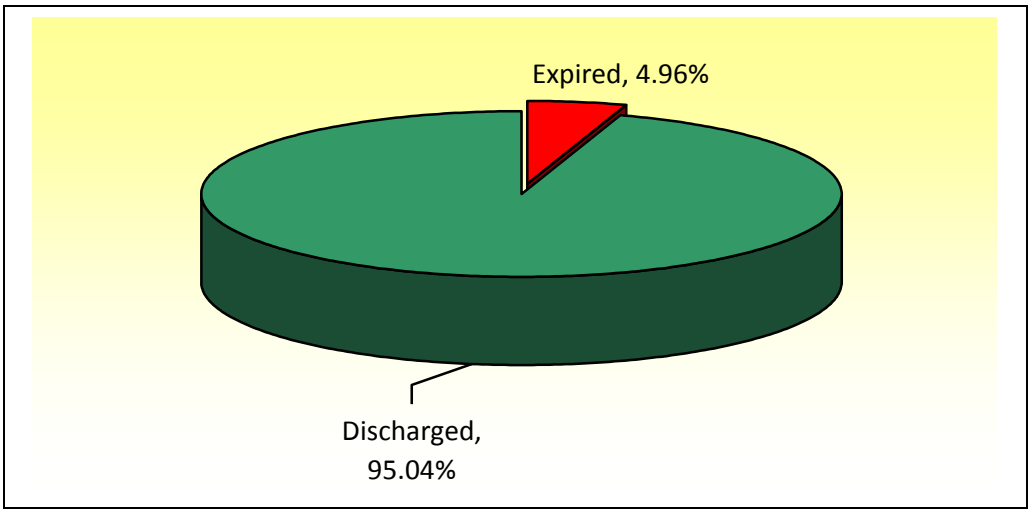
Mortality

Morbidity and Mortality following , surgery in perforative peritonitis, depends up on various factors like Age, Co morbid illness, Time elapse between onset of symptoms and presentation to hospital.

Table-11: Distribution of Sample by Mortality

Outcome	No. of cases	Percentage
Discharged	134	95.04%
Expired	7	4.96%

Figure-10: Distribution of Sample by Mortality



According to our study, mortality is more common in elderly patients, presenting late to the hospital, with features of hypovolemic shock. Most patients succumbed death due to septicaemia, which constituted about 4.96%(7cases).

DISCUSSION

This study was conducted for period of 1 year from November 2011 to October 2012 in Government Rajaji Hospital, Madurai Medical College, Madurai. About 141 patients who underwent emergency surgery for perforative peritonitis were studied and various data's collected from the study were discussed below.

In our study of 141 cases, the most common cause of perforative peritonitis is peptic ulcer perforation constituting 51.77% %. This was followed by perforation due to inflammatory pathology which constituted about 25.53% appendicular (12.05%), and typhoid (4.25%) perforation). Tubercular perforation (4%) and malignant perforation (11.34%) and trauma(8.51%) .

N.D. Swadia [15] and colleagues (1979) found an similar incidence of 59.12% of peptic ulcer perforation, 17% typhoid, 15.65% appendicular and 6.38% traumatic perforation in their analysis of 658 cases

In our study, the incidence of peptic ulcer perforation correlates with the study but variation was seen in incidence of typhoid perforation appendicular perforation. Recently the incidence of typhoid perforation and appendicular perforation has reduced mainly as a result of availability of highly effective antibiotics and early definitive management of acute

appendicitis. The increased incidence of traumatic perforation was mainly due to increasing road-traffic accidents and assaults.

In our study, the commonest site of perforation is anterior wall 1 st part of duodenum followed by ileum, appendix, and gastric . M C Dandapat [16] (1991) and D C M Rao [17] (1984) found that for gastrointestinal perforation the commonest site is duodenum, followed by ileum, stomach and appendix which shows similar results like our study.

In a similar study conducted by Nitin agarwal [11] et al in 2006 showed that most common cause of perforative peritonitis is due to small bowel perforation 41% which differs from our study with regard to site of perforation.

Hence duodenal ulcer perforation due to peptic ulcer was the most common cause of perforative peritonitis. Since most of the patients had history of peptic ulcer disease ,smoking ,alcoholism and analgesic abuse. Site of perforation also shows difference in different geographical location because small bowel perforation is more common in western countries .

Age incidence

Highest number of patients with perforative peritonitis encountered in our study was between 40-59 years . Other studies observed an age trend between 30-39 years (M C Dandapat et al [16] 1991) Rajendra singh jobhta [12] 2006). S N Mathur [19] (1991), D C M Rao [17]

(1984) had reported similar incidences. Appendicular perforation was seen in younger age group in our study, same as the incidence which was observed by Dandapat [16] (1991). Malignant perforation was noted in older age group (Schwartz et al [5]). Traumatic perforation was more common in 4th decade. Similar incidence has been reported by Jen Feng Fang et al [20] (1999) and J P Evans [21] (1973).

Sex Incidence

Regarding sexual predilection our study showed the sex ratio of men to women with all types of perforation irrespective of pathological perforation was 4.5: 1. With male predominance 115cases vs females 26 cases.

M..C. Dandapat [16] reported a sex incidence of 8.4: 1. In peptic ulcer perforation the sex incidence showed remarkable predominance in the ratio of 5:1. Peptic ulcer perforation is predominantly seen in male and it is seen in our study. Similar observation was seen by Illingworth et al [22] (1968) & W T Siu et al [23] (1997).

Clinical Features

Symptoms such as Pain Abdomen, Vomiting, Distension and fever were predominant in our study. Pain abdomen was seen in almost all cases and similar finding has been reported by Kachroo [18] (1984) and J C Baid [24] (1988). In peptic ulcer perforation, most of our patients gave

history of pain in the epigastric region, it has been reported by S N Mathur [19] (1991).

History of fever in the recent past followed by pain abdomen was a diagnostic tool for typhoid perforation clinically. S K Nair [25] (1981) and M A Noorani [26] (1997) have observed similar history. NSADI'S are known to cause peptic ulcer disease and even give rise to complications like perforation, bleeding etc; Mechanism of action being mediated through prostaglandin synthesis blockade. 26 of 73 cases of peptic ulcer perforation revealed the history of NSAIDS injection. W T Siu [23] (1997) found 6 of 33 patients revealed the same. Dehydration was the common cause after gastric perforation and was most consistent physical sign in our patients occurring in about 44% of cases; a feature also observed by S K Nair [25] (1981). Dehydration is mainly due to accumulation of fluid in the peritoneal cavity, intestine and due to vomiting apart from other causes. Tachycardia was commonly seen in cases who presented with intestinal and appendicular perforation (due to shrinkage of circulation fluid volume). In our study, tachycardia was noted in 68% cases. J C Baid noted it in 77% of cases in his study . On examination of abdomen, tenderness was recorded in all the cases, distension in 78 cases, guarding/ rigidity in 120cases, obliteration of liver dullness in 108 cases, absent bowel sounds in 102 cases. Distension was not found in majority of cases of perforative peritonitis especially appendicular perforation as there is

only little spillage and localisation of peritonitis. In most of the study conducted worldwide, tenderness was present in all the cases of gastro-intestinal perforation. In a study conducted by J C Baid and T C Jain [27] (1988)- 54 cases found distension in 46 cases, guarding/ rigidity in 54 cases, obliteration of liver dullness in 28 cases and absent bowel sounds in 29 cases. The study correlates almost with the above mentioned study with regard to signs of perforation.

Investigations

Even though presence of air under the diaphragm is a hallmark of hollow viscous perforation, absence of this does not exclude the possibility of perforation. This sign is visualised only in about 75% of perforation cases. In our study, we found it in 68% of cases. N William and N W Everson [28] (1997) have quotes "in 60-70% of cases the free air under can be detected". M C Dandapat and collegues [16] (1991) notices gas under the diaphragm in 72.35%. Our study correlates well with the above mentioned study**.**

In our study, free fluid was found in almost all the cases in which we did ultrasound. This was confirmed by laparotomy .

Abdominal paracentesis was done in 41 cases where X-ray showed no free air and in all traumatic cases. S P S Rao et al [29] (1997) obtained positive results in 96% of cases of gastro-intestinal perforation.

So, paracentesis should carry out more deligently in all cases of perforation and not only it may help to detect site of perforation and also associated visceral injuries in cases of trauma.

WIDAL test was positive in 5 cases in our study. S K Nair et al [25] (1981) demonstrate positive test in 72.5%. M K Chauhan and S K Pandey[30] (1982) in 70% and S Vaidyanathan et al [31] (1996) in 73.3% of cases.

Treatment

Out of 73 peptic ulcer perforations,66 cases of duodenal ulcer and 7 cases of gastric perforation, none of the cases were taken for definitive surgery. The decision was based upon the operative finding of contamination of the peritoneal cavity. Since most of the patients presented after 48hrs, frank peritonitis was expected and thus definitive surgery was not performed in presence of gross contamination. Thus simple closure of the perforation was performed with graham live omental patch for duodenal and few cases of gastric perforation. Worldwide literature is in agreement with the same. Malignant gastric perforation was managed by partial gastrectomy followed by gastro-jejunostomy if general condition of the patient permitted. M C Dandapat[16] (1991) in his study did the same. Malignant colonic perforation was managed by Hartman's procedure (permanent colostomy) after closure of perforation. Ileal perforation was managed by primary closure and by

resection and anastomosis if perforation is larger involving more than 1/3 of circumferences. For typhoid perforation, after trimming the edges and taking biopsy , simple closure of the perforation was done in 2 cases. 1 case had multiple perforations and thus resection and anastomosis was done. S Vaidyanathan [31] (1986) and M A Noorani et al [26] (1997) have reported the operation of choice as simple closure of perforation in 2 layers. For all the cases of appendicular perforation, appendicectomy was done and most of the literatures suggest the same.

Post operative complications

In the present study, the postoperative morbidity was towards higher side because of late presentation to the hospital(>48 hrs), poor build and malnourishment, associated anaemia, hypo protienemia and dehydration at the time of presentation. The most common postoperative complication was wound infection (34.04%)which may be sustained by the fact that surgical incision site gets contaminated and most of the patients are malnourished and anaemic and few cases developed burst abdomen which required further intervention. All this has considerably increased morbidity , mean duration of hospital stay and monetary expense to the patient . Next most common complication encountered by patients was lower respiratory tract infection 14.89%. three patient developed septicaemia and was expired

M C Dandapat [16] (1991) reported wound sepsis in 13.5% of gastrointestinal perforation. Most of the appendicular perforation did not have much complication. This is a result of less contamination and younger age patients who can withstand surgery. Many patients had chest infection as a complication (10%). This may be due to prolonged immobilisation and associated COPD in old patients.

Mortality

Overall mortality in our study was 4.96%, most of which were malignant perforations (gastric malignancy and colonic malignancy). Few cases of perforative peritonitis initially managed with bilateral flank drainage also succumbed to death. In most of the cases the cause of death was diagnosed as septicaemia.

Worldwide literature shows a decrease in mortality of gastro-intestinal perforation. This ranged from 25% in 1940 as reported by DeBakey [32] to 95% as reported by Hartez and michinda et al [33] (1961). This decrease in mortality may be attributed to the use of appropriate antibiotics, adequate resuscitation and advanced surgical techniques.

Recent studies suggest a mortality rate of less than 5% (George L Jordan et al [35] and R A D Booth [34]). Our overall mortality rate of 4% correlates well with other studies.

CONCLUSION

From our "A clinical study of perforative peritonitis " it was concluded that

- Most common age group affected by perforative peritonitis is 40 years and above
- Duodenal ulcer perforation due to peptic ulcer was the most common cause of perforative peritonitis
- The advent of anti secretory drugs (H2 blockers ,proton pump inhibitors) and effective antibiotics against H.Pylori has virtually eliminated the need for elective surgery for peptic ulcer disease. But still perforation due to peptic ulcer disease remains a major cause for emergency laparotomy which carries significant morbidity and mortality.
- Reduction in time elapse between the onset symptoms and signs to presentation to the hospital and prompt resuscitation and early surgical intervention has marked impact on overall prognosis by reducing the morbidity and mortality to the patient
- Diagnosis of perforative peritonitis is purely clinical and minimal investigation is adequate for institution of definitive treatment

- Absence of liver dullness obliteration and pneumo peritoneum on plain radiograph of abdomen and chest does not rule out perforative peritonitis, since both of them were absent in many cases.
- Laparotomy with closure of perforation with live omental patch is enough for peptic duodenal ulcer perforation and biopsy is rarely required since most of them are benign
- Peptic gastric ulcer perforation needed biopsy since most of them are malignant and it is treated by simple closure or with omental patch or resection
- Thorough peritoneal lavage is gold standard in management of perforative peritonitis
- Most important observation from our study is the increased incidence of post operative complication in our patient. mainly surgical site infection, which increases the morbidity and mean duration of hospital stay and monetary expense of the patient which needs to be addressed.
- Patient education about appropriate medical management of peptic ulcer, enteric fever, tuberculosis, & avoiding the precipitating factors such as alcohol, smoking may reduce the incidence of perforative peritonitis.

ANNEXURES

PROFORMA

Name : Age : yrs Sex : M/F

IP No :

Address : Occupation :

Date of admission : Date of surgery :

Date of discharge :

CHIEF COMPLAINTS :

HISTORY OF PRESENTING COMPLAINTS :

a) Pain :

b)Vomiting :

c) Fever :

d)Abdominal distension :

e) Others (if any) :

PAST HISTORY :

a) Pain abdomen :

b) Fever :

c) Drug intake :

d) Others (if any) :

PERSONAL HISTORY :

a) Diet : Veg / Mixed

b) Appetite : Good / Impaired

c) Bowel / Bladder :

d) Habits : Smoker / Alcoholic

e) Drug abuse (if any) :

IN WOMEN- MENSTRUAL HISTORY :

FAMILY HISTORY :

GENERAL PHYSICAL EXAMINATION

VITALS : Pulse rate : / min

BP : mHg

Temp :

RR :

Hydration :

Pallor / Icterus / Cyanosis / Clubbing / Lymphadenopathy / Edema.

EXAMINATION OF THE ABDOMEN:

• INSPECTION :

• PALPATION :

a. Site of tenderness :

b. Guarding / rigidity :

c. Any mass :

d. Others :

• PERCUSSION :

a. Liver dullness :

b. Free fluid :

• AUSCULTATION :

Bowel sounds : Absent / Present.

• EXTERNAL GENITALIA / HERNIAL ORIFICES :

• ANO-RECTAL EXAMINATION :

EXAMINATION OF OTHER SYSTEMS :

a. CVS :

b. RS :

c. CNS :

d. LOCOMOTOR SYSTEM :

PROVISIONAL DIAGNOSIS :

INVESTIGATIONS :

BLOOD : Hb : TC : DC : N L E M B

Blood grouping & Rh typing :

Urea :

Serum Creatine, Electrolytes : Na+ : K+ : Cl- :

Urine : Sugar, Albumin, Microscopy for deposits

X-ray abdomen Erect AP view :

X-ray chest PA view :

USG abdomen & Pelvis :

WIDAL :

PRE OPERATIVE DIAGNOSIS :

PRE OPERATIVE TREATMENT :

SURGERY :

a. Incision :

b. Exudate : Blood/ Bilious / Purulent / Fecal / others.

c. Site and number of perforation :

d. Other findings :

e. Procedure :

POST OPERATIVE DIAGNOSIS :

POST OPERATIVE TREATMENT :

FOLLOW-UP :

a. Date of suture removal :

b. Complications :

c. Results : cured / relieved / expired / others.

REMARKS (IF ANY):

MASTER CHART

Sl. No.	Name	Age	Sex	IPNO.	Symptoms			Signs				T O P	PpX-Ray	SOP	Etiology	Mgt	outcome
					Fever	Vomitting	Abd.Dis.	Guarding	Rigidity	O LIV DU	Bowel Sounds						
1	Velu	60	M	743392	+	–	+	+	+	+	–	>48HRS	present	ILEUM	NS INF	PC	DISCH
2	Suriyarajan	66	M	71990	+	+	–	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
3	Kumaresan	50	M	51580	+	+	+	+	+	–	–	>48HRS	present	GAST	CA STO	PAGJ	DISCH
4	Duraipandi	27	M	84234	+	+	–	+	–	–	–	<24HRS	present	DU	PU	OPC	DISCH
5	Vellai kuppan	37	M	12154	–	+	–	+	–	+	–	<24HRS	present	DU	PU	OPC	DISCH
6	Rajendran	37	M	3337	–	+	–	+	–	+	–	<24HRS	ABS	DU	PU	OPC	DISCH
7	Ranjan	6	M	5105	+	+	–	–	+	–	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
8	Karthick	20	M	6205	+	–	–	–	+	+	–	>48HRS	present	ILEUM	TRAUMA	PC	DISCH
9	Panju	38	M	8580	+	+	–	+	+	–	+	>48HRS	present	GAST	CA STO	PAGJ	DISCH
10	Adhiveerapandi	37	M	8662	–	–	+	+	–	+	–	<24HRS	present	ILEUM	NS INF	PC	DISCH
11	Solaiappan	57	M	8695	–	+	+	+	–	–	–	<24HRS	present	DU	PU	OPC	DISCH
12	Suriyarajan	35	M	12178	–	+	+	+	–	–	–	<24HRS	ABS	DU	PU	OPC	DISCH
13	Rajesh Kumar	17	M	12131	–	–	–	+	–	+	+	<24HRS	present	APP	INF	EMR AP	DISCH
14	Subhashini	48	F	62304	–	+	+	+	–	+	–	<24HRS	present	GAST	PU	OPC	DISCH
15	Savariraj	25	M	25575	+	–	–	+	+	–	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
16	Rahamatualla	52	M	29215	+	–	+	+	+	–	+	>48HRS	present	CEA	CA SIG	HARTM	DISCH
17	Tamil Selvi	38	F	30898	+	–	+	+	+	+	–	>48HRS	ABS	CEA	IC TB	RA	DISCH

18	Lakshmanan	60	M	31012	+	+	+	–	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
19	Vellaidurai	49	M	31000	+	+	–	+	+	+	+	>48HRS	present	DU	PU	OPC	DISCH
20	Krishnamoorthy	75	M	42429	+	+	+	+	+	+	–	>48HRS	present	GAST	CA	PC	DISCH
21	Aaanaisamy	68	M	44201	–	+	–	+	–	–	–	<24HRS	ABS	DU	PU	OPC	DISCH
22	Samsudeen	50	M	48102	+	–	+	+	–	+	–	<24HRS	present	ILEUM	IC TB	RA	DISCH
23	Murugan	25	M	13551	–	–	–	+	+	+	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
24	Alagu	55	M	13480	+	+	+	+	+	+	–	>48HRS	ABS	GAST	CA STO	PC	DISCH
25	Fathima	43	F	17047	+	+	+	+	+	–	+	>48HRS	present	DU	PU	OPC	DISCH
26	Kanagarajan	43	M	15212	–	+	+	+	+	+	–	<24HRS	present	ILEUM	TRAUMA	PC	DISCH
27	Shantha	40	F	12140	+	–	–	+	–	+	+	>48HRS	ABS	APP	INF	EMR AP	DISCH
28	Rajesh	22	M	29422	+	+	+	+	+	+	–	>48HRS	present	ILEUM	NS INF	PC	DISCH
29	Nallu	56	M	25766	+	+	+	+	+	–	+	>48HRS	present	DU	PU	OPC	DISCH
30	Sethu	40	M	25112	+	+	+	+	+	–	–	>48HRS	present	ILEUM	Typhoid	PC	DISCH
31	Mtuthumani	34	M	34384	+	+	+	+	+	–	–	>48HRS	present	ILEUM	NS INF	PC	DISCH
32	Manoharan	59	M	31284	–	+	–	+	+	+	–	<24HRS	ABS	ILEUM	NS INF	PC	DISCH
33	Agnisamy	58	M	31347	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
34	Rahamathullah	58	M	31482	+	+	–	+	+	–	–	>48HRS	present	ILEUM	NS INF	PC	DISCH
35	Mani	65	M	49996	+	+	+	+	+	–	–	>48HRS	present	GAST	CA STO	PAGJ	DISCH
36	Selvam	52	M	49980	+	+	+	+	+	+	–	>48HRS	present	ILEUM	Typhoid	PC	DISCH
37	Palaniyandi	26	M	51772	+	+	–	+	+	+	+	>48HRS	ABS	APP	INF	EMR AP	DISCH
38	Sekar	50	M	51727	+	–	–	+	+	+	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
39	Sevugan	60	M	58059	–	+	–	+	+	–	+	<24HRS	present	DU	PU	OPC	DISCH
40	Raman	45	M	59943	–	+	–	+	–	–	+	<24HRS	ABS	DU	PU	OPC	EXP
41	Selvam	28	M	91841	–	+	+	+	+	+	–	<24HRS	present	DU	PU	OPC	DISCH

42	Vignesh	19	M	63720	+	+	+	+	–	–	–	>48HRS	present	ILEUM	Typhoid	PC	DISCH
43	Saraswathi	59	F	67237	–	+	+	+	+	+	–	<24HRS	present	DU	PU	OPC	DISCH
44	Suredran	57	M	67310	+	+	–	+	+	+	–	>48HRS	present	ILEUM	TRAUMA	RA	DISCH
45	Oondeeran	35	M	67810	+	+	+	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
46	Palaisamy	21	M	72129	–	+	+	+	–	+	–	<24HRS	present	DU	PU	OPC	DISCH
47	Muthu	28	M	73810	+	+	–	+	+	–	+	>48HRS	present	DU	PU	OPC	DISCH
48	Karutha pandiyan	40	M	75355	+	+	+	–	+	+	–	>48HRS	present	GAST	PU	PC	DISCH
49	Sankaran	22	M	72180	–	–	+	+	+	+	+	>48HRS	ABS	APP	INF	EMR AP	DISCH
50	Raman	60	M	78580	–	+	–	+	–	+	–	<24HRS	present	DU	PU	OPC	DISCH
51	Arumugam	80	M	80009	–	+	+	+	+	–	–	<24HRS	present	DU	PU	OPC	DISCH
52	Sankara mahalingam	56	M	81219	+	+	+	+	+	+	+	>48HRS	present	DU	PU	OPC	DISCH
53	Prabhu	19	M	84845	+	+	–	+	+	+	+	>48HRS	present	ILEUM	NS INF	PC	DISCH
54	Sathiyaraju	58	M	86124	–	+	–	+	+	–	+	<24HRS	ABS	DU	PU	OPC	DISCH
55	Chinniah	56	M	86214	+	+	–	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
56	Gurunathan	54	M	80430	–	+	–	–	–	–	+	<24HRS	present	DU	PU	OPC	DISCH
57	Thinakaran	26	M	87530	–	+	+	+	+	–	–	<24HRS	present	ILEUM	NS INF	PC	DISCH
58	Johnpaulraj	55	M	89464	+	+	+	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
59	Saraswathy	70	F	92353	–	–	–	+	–	+	+	<24HRS	present	GB	INF	CHOLES	DISCH
60	Raman	35	M	2459	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
61	Alagappan	58	M	1840	+	+	–	+	+	+	–	>48HRS	ABS	GAST	PU	PC	DISCH
62	Arakiyam	38	M	1869	+	+	+	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
63	Varadarajan	40	M	1899	+	+	–	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
64	Karuppiah	58	M	3336	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH

65	Manthir	40	M	5107	+	+	–	+	+	+	+	>48HRS	present	DU	PU	OPC	DISCH
66	Jauquilin	35	F	5502	+	–	+	–	+	–	+	>48HRS	ABS	ILEUM	IC TB	RA	DISCH
67	Virumandi	45	M	6611	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
68	Rathinakumar	37	F	6630	+	+	–	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
69	Subramani	60	M	6810	–	+	+	+	+	–	–	<24HRS	ABS	GAST	CA STO	PAGJ	DISCH
70	Kannan	46	M	74612	+	–	–	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
71	Raguraman	18	M	76413	+	+	–	+	+	+	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
72	Moorthy	45	M	77990	+	+	+	+	+	+	–	>48HRS	ABS	DU	PU	OPC	DISCH
73	Ramasamy	69	M	79502	–	+	–	+	–	–	+	<24HRS	ABS	JEJ	TRAUMA	PC	DISCH
74	Thangam	35	M	86788	+	–	–	+	+	+	–	>48HRS	present	ILEUM	Typhoid	PC	DISCH
75	Sundaram	45	M	84165	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
76	Raja	26	M	84147	+	–	+	+	+	+	–	>48HRS	present	ILEUM	NS INF	PC	DISCH
77	Dhanuvel	48	M	84270	+	+	+	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
78	Kaliammal	58	F	85871	+	–	–	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
79	Krishnan	48	M	87338	+	+	+	+	+	+	–	>48HRS	ABS	DU	PU	OPC	DISCH
80	Badmini	48	F	87296	+	–	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
81	Kannan	38	M	90542	–	+	+	+	–	+	+	<24HRS	present	DU	PU	OPC	DISCH
82	Thangaraj	60	M	2899	+	+	+	+	+	–	–	>48HRS	ABS	DU	PU	OPC	DISCH
83	Palsamy	55	M	9034	+	+	–	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
84	Ravichandran	47	M	9097	+	+	–	–	+	+	–	>48HRS	ABS	DU	PU	OPC	DISCH
85	Selvam	47	M	12295	+	–	+	+	+	+	–	>48HRS	present	GAST	CA STO	PAGJ	DISCH
86	Kalidas	55	M	12568	+	+	+	+	+	–	–	>48HRS	ABS	DU	PU	OPC	DISCH
87	Vijaya	66	F	12690	–	–	–	+	–	–	+	<24HRS	ABS	UTERUS	POST RT	PC	EXP
88	Thangavel	58	M	35429	–	+	–	+	+	+	–	<24HRS	present	DU	PU	OPC	DISCH

89	Vinothkumar	20	M	37148	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
90	Manmathan	45	M	38456	+	–	+	+	+	–	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
91	Muthulakshmi	30	F	43606	–	+	–	–	+	+	–	<24HRS	present	DU	PU	OPC	DISCH
92	Gopi	23	M	42165	+	–	+	+	+	+	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
93	Chinnappan	41	M	45223	+	–	–	+	+	+	–	>48HRS	present	ILEUM	TRAUMA	RA	DISCH
94	Veeraperumal	50	M	45316	+	+	+	–	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
95	Veelayammal	24	F	45236	–	–	+	+	+	+	–	<24HRS	present	ILEUM	Typhoid	PC	DISCH
96	John Mahimai raj	45	M	56329	+	+	–	–	+	+	–	>48HRS	ABS	DU	PU	OPC	DISCH
97	Murugeswari	40	F	56890	–	–	+	+	+	–	–	<24HRS	present	CEA	CA SIG	RA	EXP
98	Karalin	55	M	57300	+	+	+	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
99	Marimuthu	38	M	69166	–	+	+	+	–	+	+	<24HRS	present	DU	PU	OPC	DISCH
100	Chokkan	62	M	74487	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
101	Suruliyammal	68	F	74541	+	+	–	+	+	+	–	>48HRS	present	GAST	CA STO	PC	DISCH
102	Murugaesan	61	M	1776	+	–	+	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
103	Satheeshkumar	15	M	3466	+	–	–	+	+	+	–	>48HRS	ABS	GAST	PU	OPC	DISCH
104	Kali	55	M	5305	+	–	+	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
105	Yogeshwaran	29	M	8841	–	+	+	–	+	+	+	<24HRS	ABS	APP	INF	EMR AP	DISCH
106	Katchammal	50	F	13948	–	+	–	+	–	+	+	<24HRS	ABS	MECK	INF	RA	DISCH
107	Rani	40	F	13922	+	–	+	+	+	–	+	>48HRS	present	JEJ	TRAUMA	PC	DISCH
108	Santhanam	75	F	13934	+	+	+	+	+	+	+	>48HRS	present	CEA		B/L F DRA	EXP
109	Balan	22	M	17505	–	+	–	+	+	+	+	<24HRS	ABS	DU	PU	OPC	DISCH
110	Panchavarnam	58	F	19248	+	–	+	–	+	+	–	>48HRS	ABS	JEJ DIV	INF	RA	DISCH
111	Senthil kumar	22	F	20741	+	+	+	+	+	–	–	>48HRS	present	APP	INF	EMR AP	DISCH

112	Guruvaiah	51	M	20843	+	+	–	+	+	+	–	>48HRS	present	GAST	CA STO	PC	DISCH
113	Himanson	45	M	20993	+	–	–	+	+	–	–	>48HRS	ABS	GAST	PU	PC	DISCH
114	Sadayandi	47	M	22466	–	+	+	+	+	+	+	<24HRS	present	ILEUM	Typhoid	PC	DISCH
115	Chinnakannan	65	M	22502	+	–	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
116	Kathar Kabir	28	M	31302	+	–	–	+	+	–	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
117	adbdul kadhar	37	M	31368	+	+	+	+	+	+	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
118	Sathish Kumar	25	M	31478	+	+	+	+	+	+	–	>48HRS	present	ILEUM	NS INF	PC	DISCH
119	Murugan	50	M	33447	–	+	+	+	+	+	+	<24HRS	present	GAST	CA STO	PC	DISCH
120	Kavitha	22	F	33568	–	+	+	+	+	+	+	<24HRS	present	ILEUM	NS INF	PC	DISCH
121	Raja	32	M	37198	–	+	–	+	+	+	+	<24HRS	ABS	ILEUM	TRAUMA	PC	DISCH
122	Umar Basha	49	M	44595	+	+	+	+	+	+	–	>48HRS	present	ILEUM	TRAUMA	RA	DISCH
123	Karupayee	40	F	47946	+	+	–	+	+	+	–	>48HRS	present	DU	TRAUMA	BERNE	DISCH
124	Shantha	56	F	53137	+	–	–	+	+	+	–	>48HRS	ABS	RECT	CA	COLOST	DISCH
125	Anbalagan	45	M	53134	–	+	–	+	+	–	+	<24HRS	present	GAST	PU	PY EX PRO	EXP
126	Theda selvam	50	M	54693	–	+	+	+	+	+	+	<24HRS	present	DU	PU	OPC	DISCH
127	Soundharavalli	29	F	54898	+	+	–	+	+	–	+	>48HRS	present	DU	PU	OPC	DISCH
128	Periyakaruppan	60	M	54708	+	–	–	–	+	+	+	>48HRS	present	CEA	CA	RA	DISCH
129	Pothurajan	30	M	56509	–	+	+	+	+	–	+	<24HRS	present	DU	PU	OPC	DISCH
130	Suresh	25	M	56596	+	+	–	+	+	+	–	<24HRS	present	GAST	CA STO	PC	DISCH
131	Malairaja	43	M	55249	+	+	+	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
132	Malai eswari	16	F	58127	+	–	–	+	+	+	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
133	Subramani	40	M	58120	–	+	–	+	+	+	+	<24HRS	present	SIG	CA ANAL	COLOST	EXP
134	Ganesan	28	M	58141	–	+	+	+	+	+	+	<24HRS	present	DU	PU	OPC	DISCH

135	Azhagar	50	M	368933	+	+	–	+	+	+	–	>48HRS	present	ILEUM	NON SPE	PC	DISCH
136	Lakshmi	45	F	68099	–	+	+	+	+	+	+	<24HRS	present	DU	PU	OPC	DISCH
137	Nehru	46	M	70642	+	+	–	+	+	–	–	>48HRS	ABS	APP	INF	EMR AP	DISCH
138	Periyandi	38	M	74508	+	+	–	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH
139	Pandikumar	18	M	74058	+	+	–	+	+	–	–	>48HRS	present	DU	PU	OPC	DISCH
140	Vellathai	70	F	76662	+	+	+	+	+	+	–	>48HRS	present	ILEUM	VEN HER	RA	EXP
141	Thiagarajan	58	M	76526	+	+	–	+	+	+	–	>48HRS	present	DU	PU	OPC	DISCH

Abd.Dis.	-	Abdominal Distension
APP	-	Appendix
CA	-	Carcinoma
CA ANAL	-	CA Anal canal
CA SIG	-	CA Sigmoid
CA STO	-	CA Stomach
CEA	-	Ceacum
DU	-	Duodenum
GAST	-	Gasteric
GB	-	Gallbladder
IC TB	-	Illeoceacal Tuberculosis
INF	-	Inflammatory
IPNo	-	Inpatient Number
JEJ	-	Jejunum
JEJ DIV	-	Jejunal Diverticulosis
MECK	-	Mackles Diverticulosis
Mgt	-	Management
NS INF	-	Non specific Inflamation
O LIV DU	-	Obliteration of Liver Dullness
PpX-Ray	-	Pneumo Peritoneum in X- Ray
PU	-	Peptic Ulcer
RECT	-	Rectum
SIG	-	Sigmoid
SOP	-	Site of Perforation
TOP	-	Timing of Presentation.
VEN HER	-	Ventral Hernia

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Ref. No. 01104 /E4/3/2012

Govt.Rajaji Hospital,Madurai.20.
Dated: 8.03.2012

Institutional Review Board / Independent Ethics Committee.
Dr. A. Edwin Joe, M.D (FM), BL.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The next Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 23.02.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5.Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6.Dr.M.Gobinath,MS(Gen.Surgery) 097-871-50040	Professor of Surgery Madurai Medical College	Member
7.Dr.S. Dilshadh, MD(O&G)	Professor of OP&Gyn Madurai Medical College	Member
8.Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9.Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 623-B.II.Floor,East II Cross, K.K.Nagar,Madurai.20.	Member
10.Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Senthilkumar. K	PG, M.S (genl surg)	Perforative peritonitis: a clinical study.	Approved

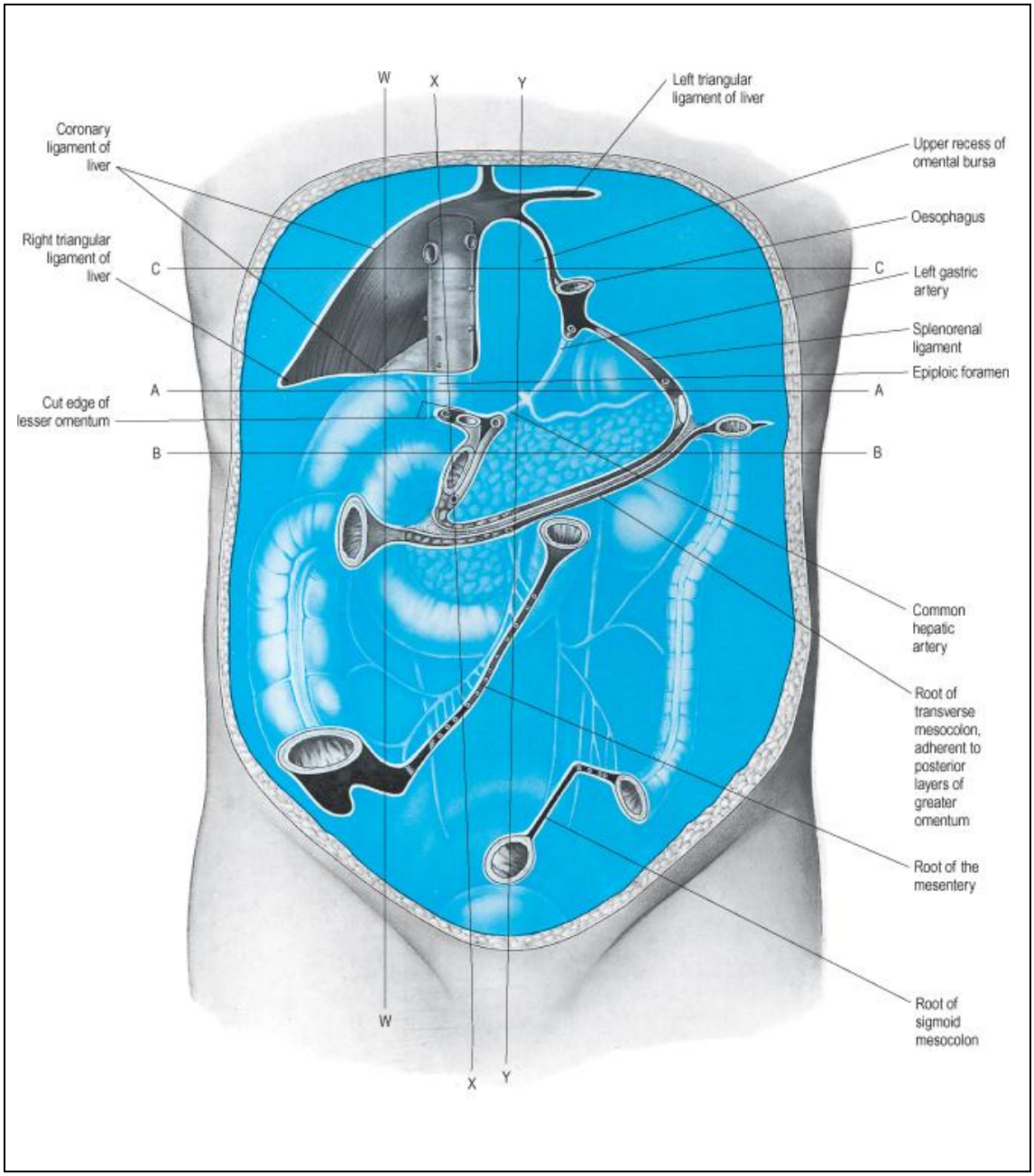
Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance.
She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

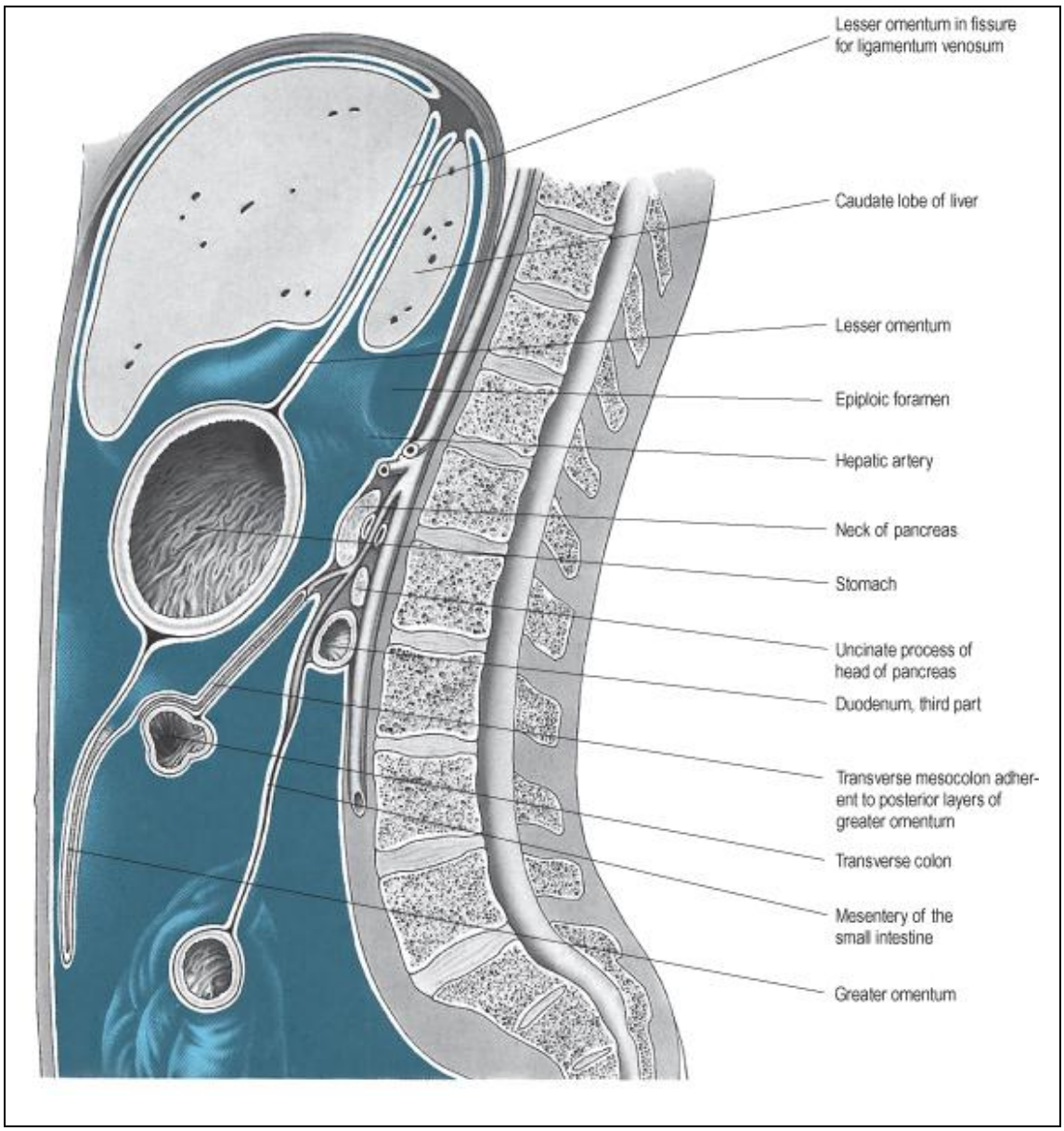
DEAN
13/3/12

To
All the above members and Head of the Departments concerned.
All the Applicants.

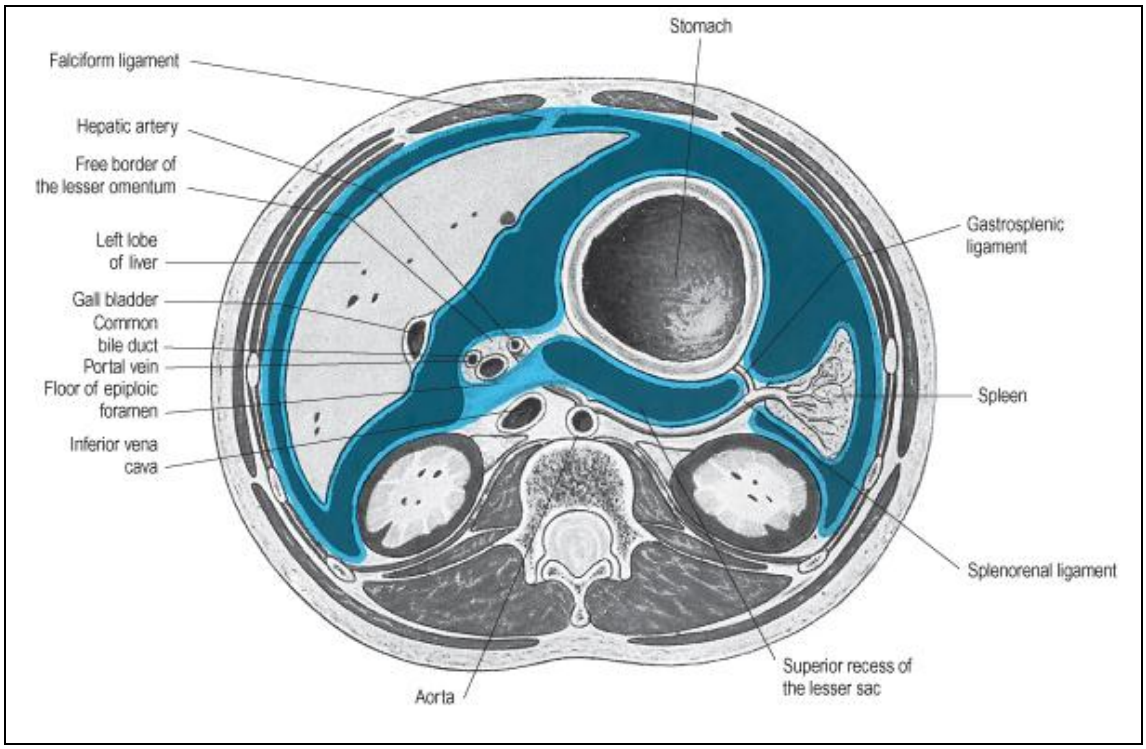
Picture-1: The posterior abdominal wall, showing the lines of peritoneal reflexion, after removal of the liver, spleen, stomach, jejunum, ileum, caecum, transverse colon and sigmoid colon.



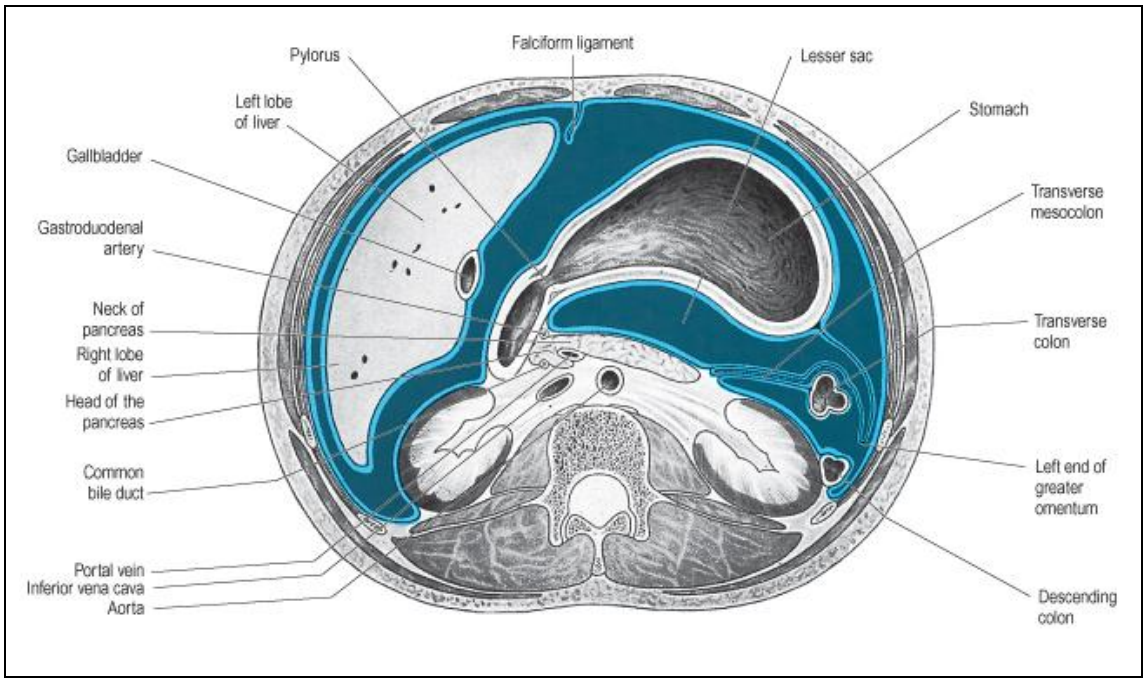
Picture-2: Sagittal section through the abdomen in the median plane (YY of Picture-1).



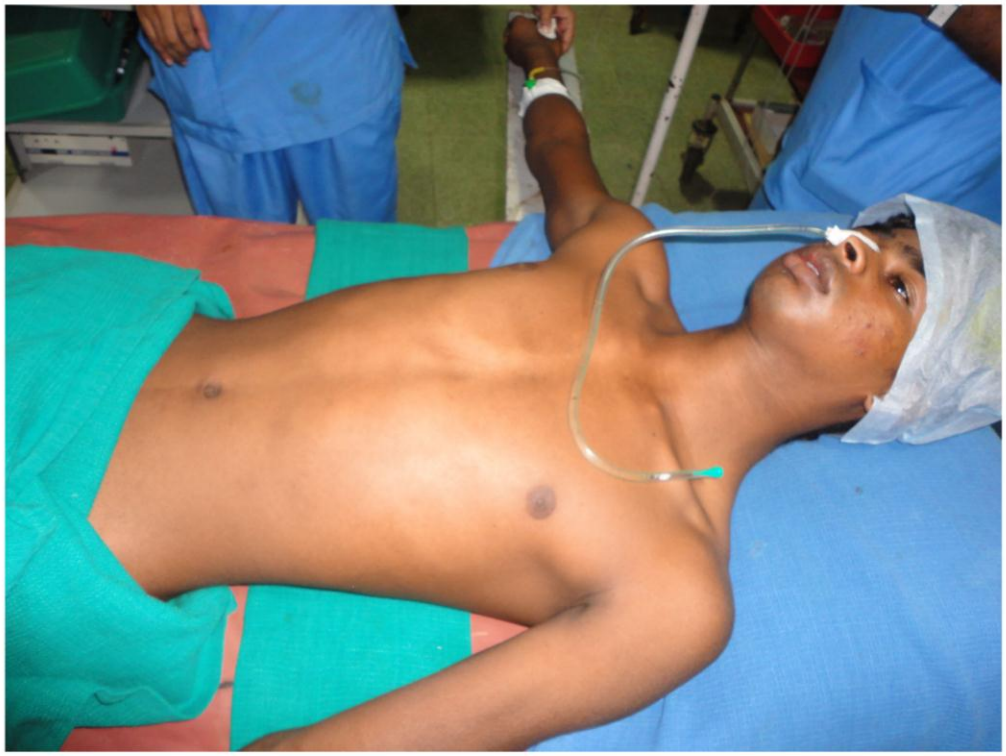
Picture-3: Transverse section through the abdomen. (AA of Picture-1).



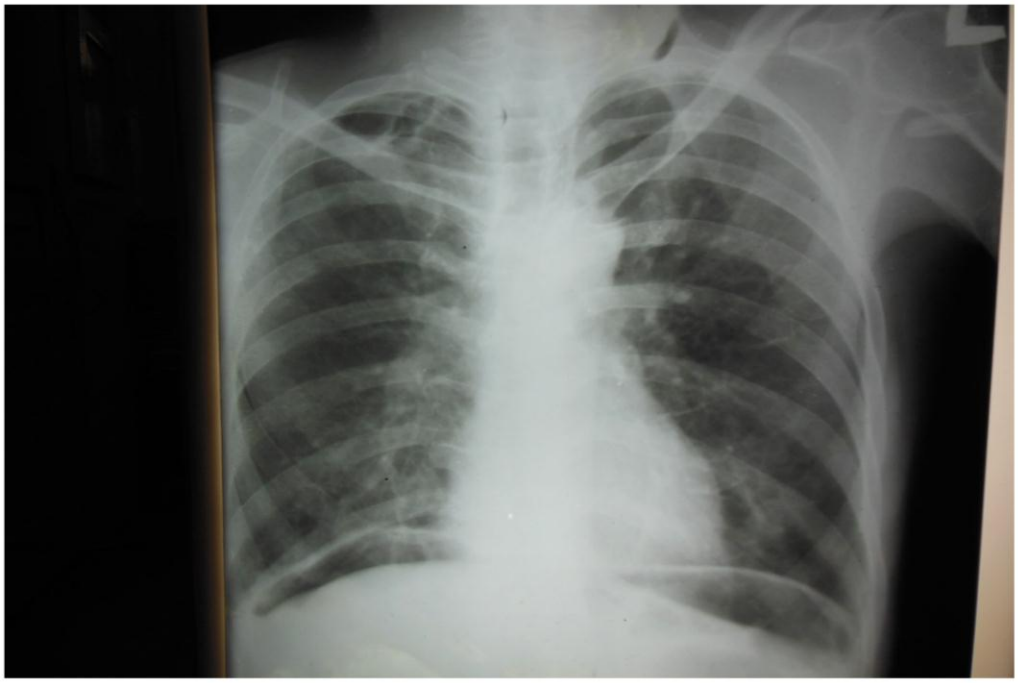
Picture-4: Transverse section through the abdomen. (BB of Picture-1).



1.Patient with perforative peritonitis



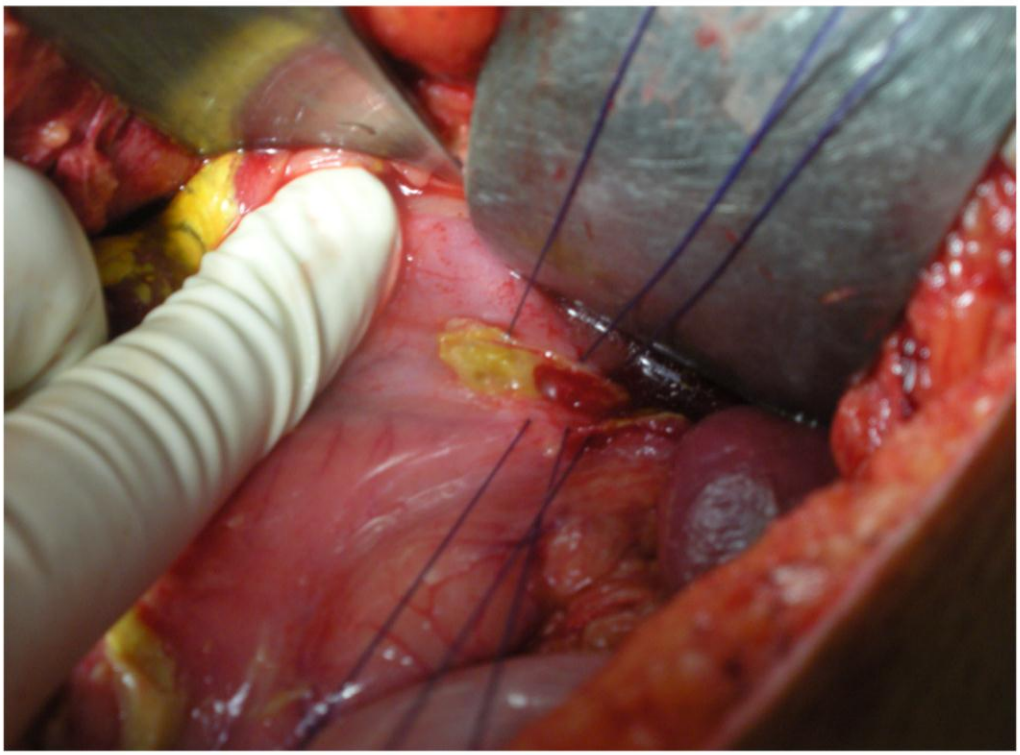
2.X ray showing Air under diapragm



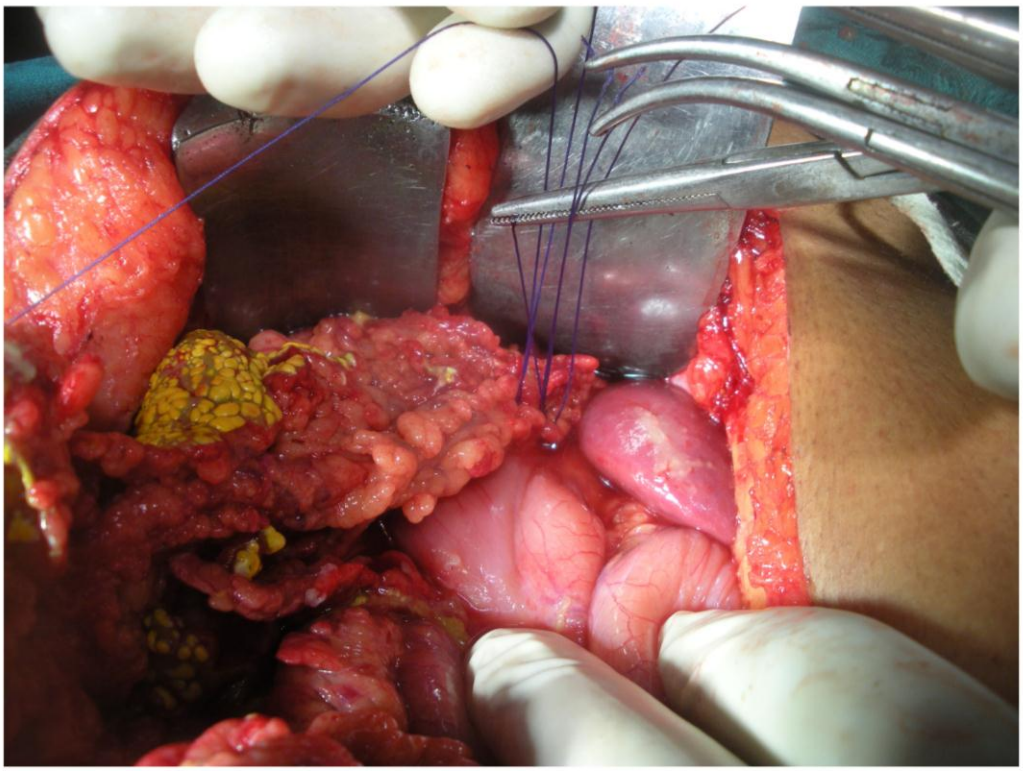
11.Post operative complication-Sub phrenic abscess with purulent discharge

12.EC Fistula in a patient who underwent BERENE'S DUO DIVERTICULISATION

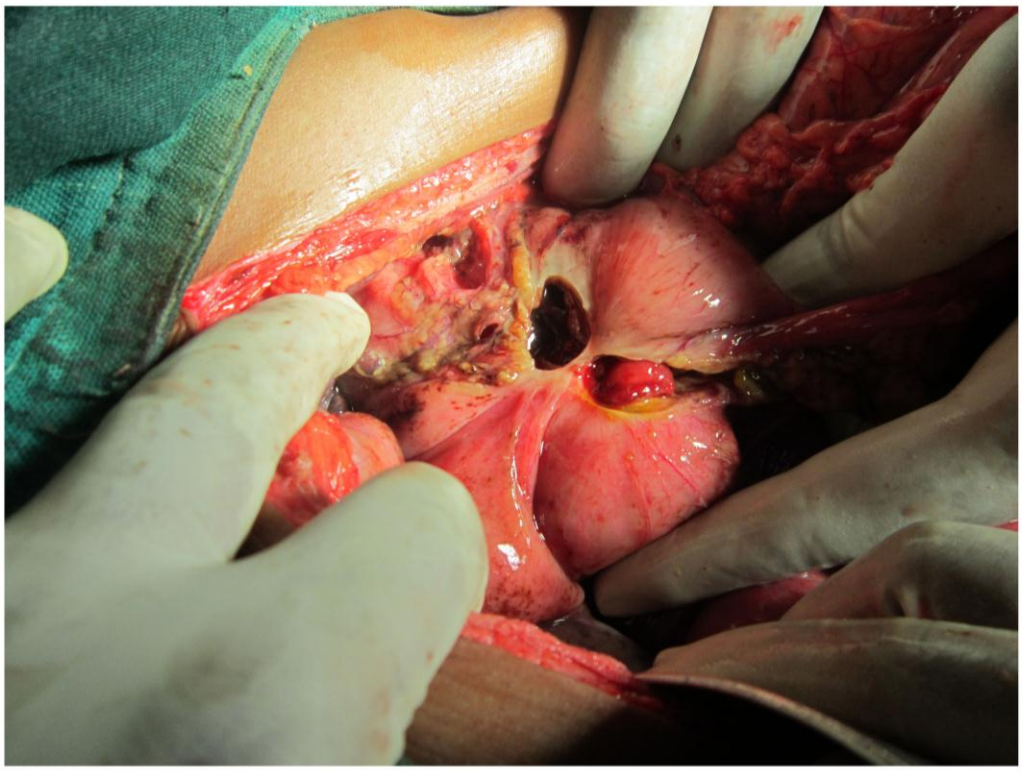
3A. Duodenal perforation closed with live omental patch



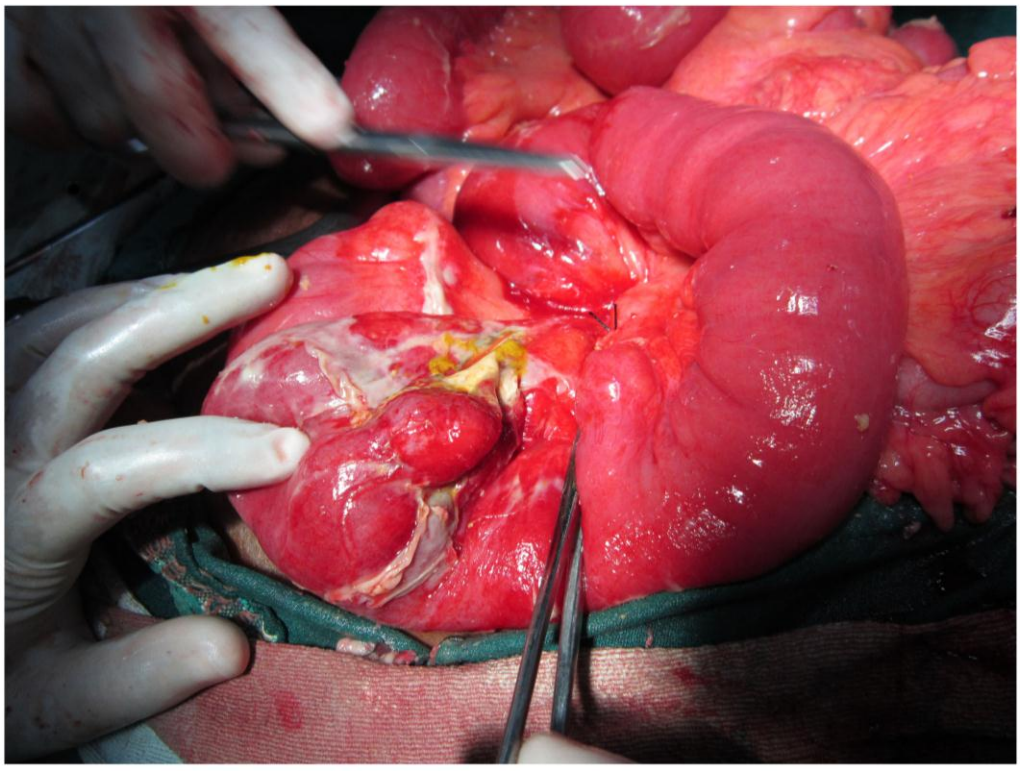
3B. Duodenal perforation closed with live omental patch



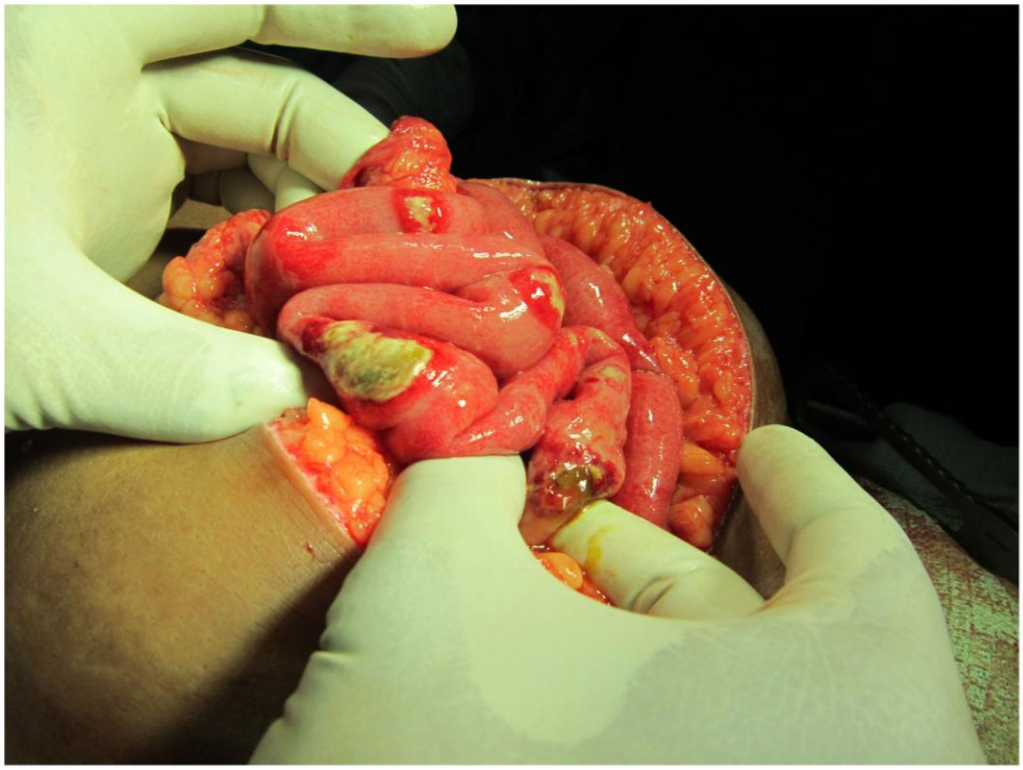
4.Giant Gastric Perforation



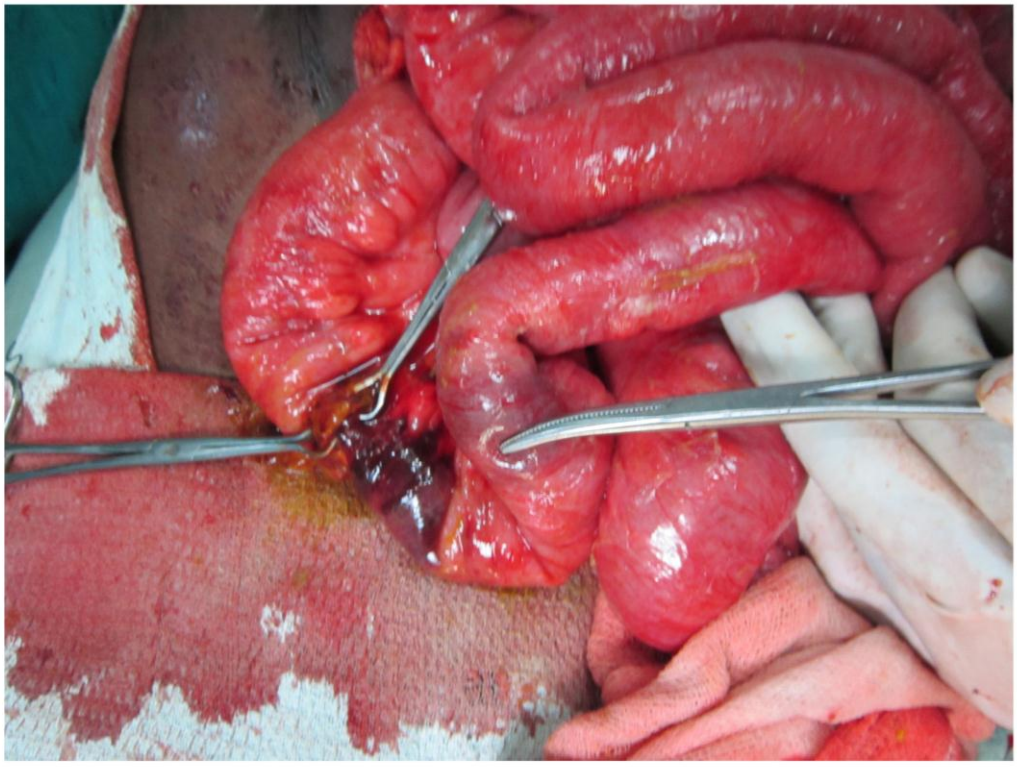
5.Jejunal Diverticulosis with Perforation



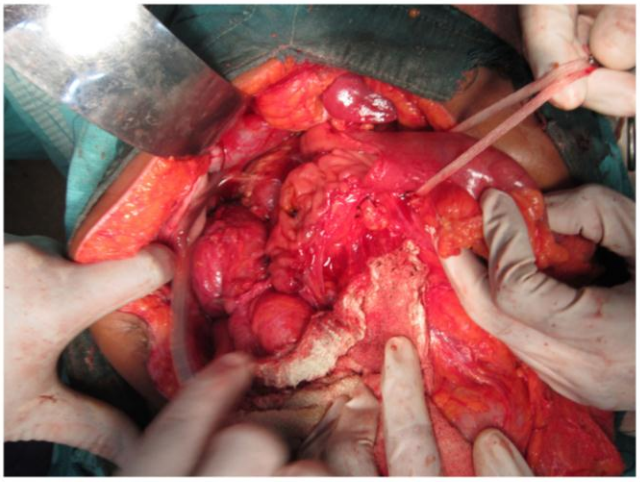
6.Multiple ileal Perforation



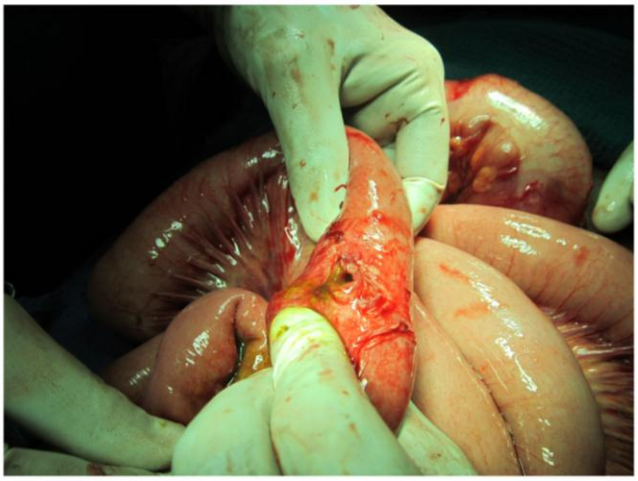
7.SMA Thrombosis with patchy Gangrene and perforation of ileum



8.Traumatic duodenal perrforation



9.Traumatic Jejunal perforation



10.Uterine perforation