“A CLINICAL STUDY ON PERFORATION PERITONITIS”

A DISSERTATION SUBMITTED TO THE TAMILNADU

DR MGR MEDICAL UNIVERSITY

CHENNAI

In partial fulfillment of the requirement for the degree of

M.S. (GENERAL SURGERY)

BRANCH – I

Register No: 221711354

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

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DR. G. KAMALIN VIJI M.S
Associate Professor,
Department of General Surgery,
Tirunelveli Medical College,
Tirunelveli.

Date:

Place:

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This is to certify that the dissertation entitled **"A CLINICAL STUDY ON PERFORATION PERITONITIS"** is a bonafide research work submitted by **Dr. A. COLLINS EPHREME NOBLE,** Postgraduate student in Department of General Surgery, Tirunelveli Medical College and Hospital, Tirunelveli, under the guidance of **Dr. G. KAMALIN VIJI M.S.,** Associate Professor, Department of General Surgery, Tirunelveli Medical College & Hospital, in partial fulfillment of the requirement for M.S. Degree (Branch - I) in General Surgery.

PROF. Dr.D.ALEX ARTHUR EDWARDS, M.S.,
Professor and HOD of General Surgery
Tirunelveli Medical College,
Tirunelveli

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DR. S.M. KANNAN M. S, M.Ch (Uro)
DEAN
Tirunelveli Medical College
Tirunelveli

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I hereby declare that the dissertation titled **"A CLINICAL STUDY ON PERFORATION PERITONITIS"** is a bonafide and genuine research work carried out by me at Tirunelveli Medical College hospital, Tirunelveli under the guidance of **Dr G. KAMALIN VIJI M.S.,** Associate Professor, Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

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Dr. A. COLLINS EPHREME NOBLE
Postgraduate Student,
Register No: 221711354
M.S.General Surgery,
Department of General Surgery,
Tirunelveli Medical College,
Tirunelveli.

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INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572733-EXT, 91-462-2572844, 91-462-2579785, 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

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THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of The Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
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13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
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State of Tamilnadu, South India

Dr.J.SureshDurai, MD
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

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This is to certify that this dissertation work titled **"A CLINICAL STUDY ON PERFORATION PERITONITIS"** of the candidate **Dr. A. COLLINS EPHREME NOBLE** with registration Number **221711354** for the award of **M.S.** Degree in the branch of **GENERAL SURGERY.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **16 Percentage** of plagiarism in the dissertation.

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Instances where selected sources appear:

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CONTENTS

INTRODUCTION

Peritonitis that occurs due to hollow visceral perforation, is a term frequently encountered in surgical practice. It is defined as the inflammation of the serosal membrane which lines the abdominal cavity and the organs contained within it. Introduction of an infection through a bowel perforation into the otherwise sterile peritoneal ambiance & introduction of a chemical irritant like gastric acid from a perforated ulcer are the common causes of peritonitis. The various modes of presentation of cases might mislead the diagnosis of its origin. The spectrum of causes of perforation in tropical countries is still different from its western counterpart. Contrary to the western countries where lower gastro-intestinal tract perforations preponderate, the majority of cases in India occur due to upper gastro intestinal tract perforations.

This disease has been known to man kind since the days of Hippocrates. Hippocrates described Hippocrates facies in 400 BC. Earlier in the year 1727, Rawlenson was the first to clearly describe the signs and symptoms of gastric ulcer and peritonitis.

Peritonitis that occurs secondary to perforation of the gastro intestinal tract, a common occurrence in our country, is associated with high morbidity and mortality rates and it requires emergency surgical intervention. Crisp was the first to make a clinical description of perforated peptic ulcer in 1843.

The two important risk factors for perforation are

- Smoking and
- Usage of non-steroidal anti inflammatory drugs.

Diagnosis is usually made clinically and confirmation is made radiographically by the presence of pneumoperitoneum. The investigations should be done such that it gives a definitive diagnosis in a short span of time. The treatment of peritonitis has switched to an operative approach instead of the conservative approach owing to the increasing research and development done in the field of surgery and intensive care facilities. Sir Cuthbert Wallace puts it “it is better, to check than being waiting”. Early surgery has got a lot of advantages over the late surgery in case of peritonitis.

In case of patients diagnosed to have a spontaneously sealed perforation proved by water soluble contrast gastro- duodenogram, non-operative management is successful. Operative management consists of age-old practice of omental patch closure, but this can also be done by laparoscopic method.

Ileal perforation is a common surgical emergency in the tropical countries. Reports show that it constitutes the 5th commonest cause of abdominal emergencies due to high incidence of enteric fever and tuberculosis in our countries. The mortality rate from ileal perforations continues to be high in developing countries, in spite of improvement in critical care and timely surgical intervention. In today's world of advanced anaesthesia and enormous improvement of resuscitative measures, every patient with ileal perforation should be recommended for surgery

In 1889, McBurney was the first to describe appendicitis also known as "perityphilitis".

If untreated, appendicitis will progress to local peritonitis with formation of appendicular mass, gangrene of appendix, perforation and generalised peritonitis.

Surgical exploration along with embolectomy is mandatory in acute mesenteric ischemia if there is presence of peritoneal signs. In the absence of the peritoneal signs, embolectomy is the standard of care. In non-occlusive mesenteric ischaemia, infusion of intraluminal vasodilator is done. Colonic perforations which carries high mortality risk is mainly due to diverticular perforation but perforations due to neoplasm, ischaemia are also seen.

Now-a-days, operative management of peritonitis includes simple closure of the perforation with a thorough peritoneal lavage and also resection and anastomosis if required especially in small bowel perforation. Many surgeons do not prefer ostomies.

Resection of the pathologic part with diversion procedure like Hartmann's procedure is alto considered in colon cancer with gross contamination of the peritoneum.

AIMS AND OBJECTIVES

1. To estimate the frequency of peritonitis secondary to hollow viscus perforation in relation to age, etiology, Site of perforation, Symptoms and signs,
2. To estimate the correlation between clinical sign and radiological investigation
3. To find out the outcome of disease

MATERIALS AND METHODS

This study has been conducted in Department of General Surgery Tirunelveli Government Medical College. Based on the analysis of 100 cases of hollow viscous perforation admitted to Tirunelveli Medical College Hospital, Tirunelveli, fulfilling the criteria were selected for the study.

Inclusion Criteria:

- Clinical /Radiologically proven cases of perforation peritonitis
- Age> 13 yrs, irrespective of sex.

Exclusion criteria:

- Perforation peritonitis due to penetrating trauma
- Primary peritonitis,
- Post op peritonitis.

All patients were subjected to Biochemical investigations, Chest X Ray, Abdominal X Ray erect view, USG abdomen and pelvis.

After confirming the diagnosis Emergency Laparotomy and drainage was done. Depending on the site of perforation, appropriate treatment protocol was adopted.

REVIEW OF LITERATURE

Anatomy

The peritoneum is the most complex & the largest serous membrane in the body. It forms a closed sac in males. But in females it is open at the lateral end of the uterine tubes. It forms a closed sac (i.e. coelom) antero-laterally by lining the interior surfaces of the abdominal wall, posteriorly by forming the boundary to the retro peritoneum, anteriorly by covering the extra peritoneal structures in the pelvis, and superiorly by covering the undersurface of the diaphragm.

This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. Hence creating the peritoneal cavity, a potential space between the two layers.

The peritoneum is formed by a single layer of flattened mesothelial cells lying on a layer of loose connective tissue which inturn contains a rich network of vascular and lymphatic capillary channels, nerve endings, and immune – component cells, particularly lymphocytes and macrophages. Mesothelial cells may transform into fibroblasts. These fibroblasts play an important role in the formation of peritoneal adhesions after surgery or inflammation of the peritoneum

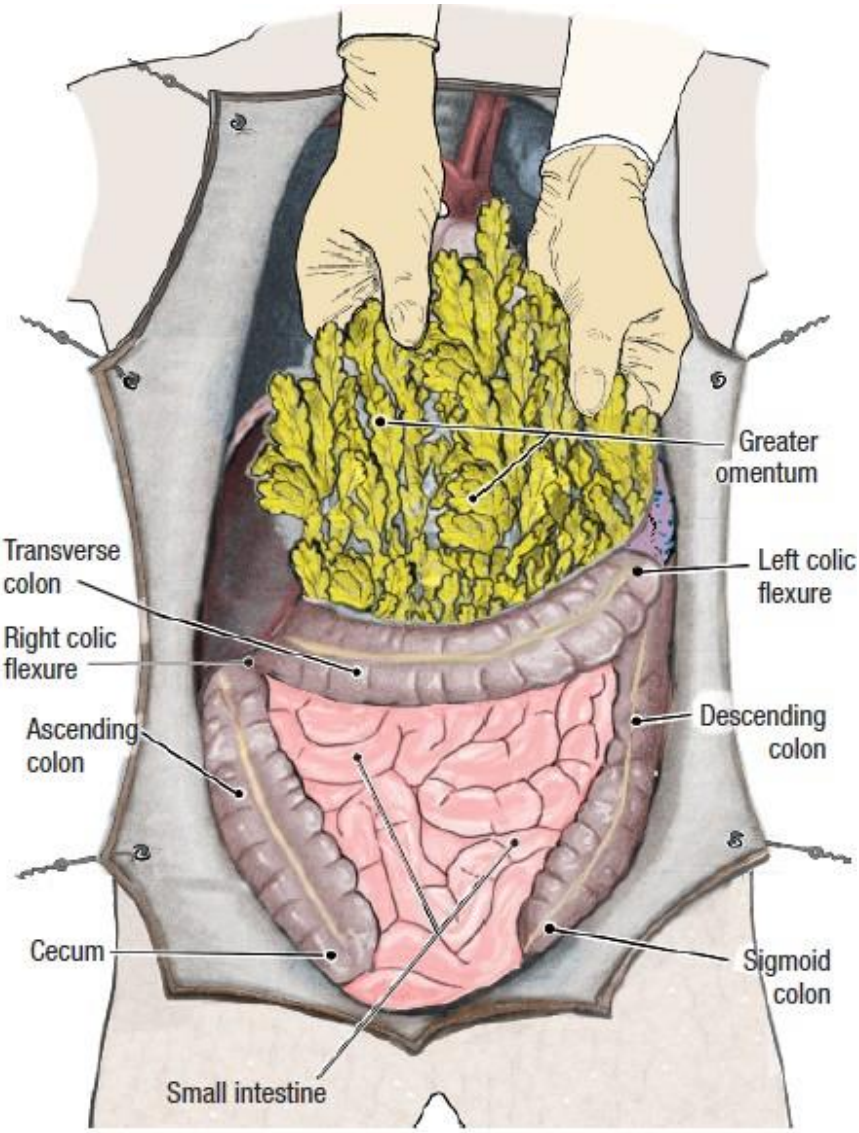


Figure1 :Reflect the greater omentum superiorly to expose the small intestine and large intestine

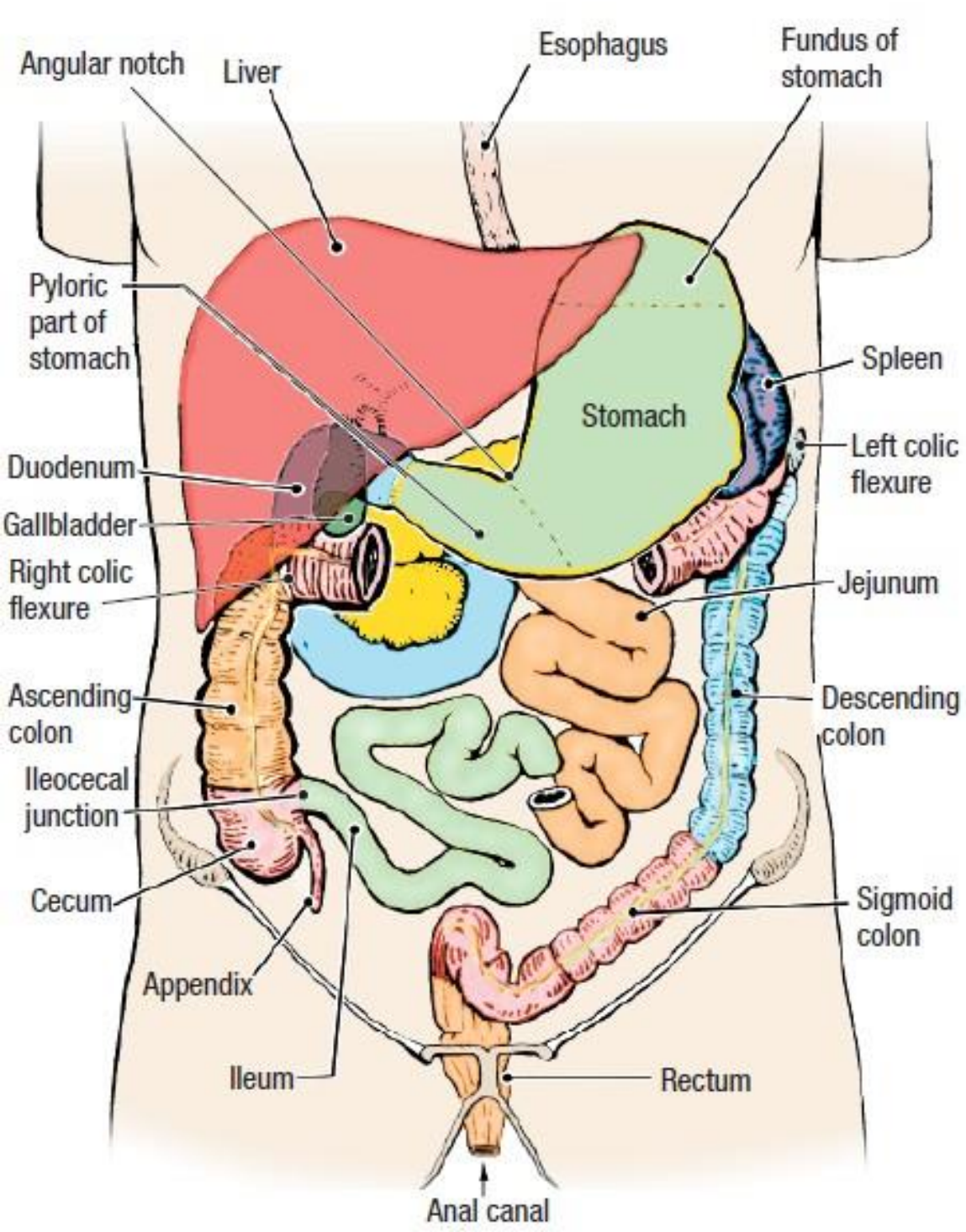


Figure2: General arrangement of abdominal viscera

Peritoneal Cavity

This is the potential space between the parietal and visceral layers of peritoneum. Under normal circumstances, the peritoneal cavity never contains gas although in inflammatory condition of the viscera, the amount of fluid may be increased. This consists of:

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

Greater omentum

The greater omentum, the largest peritoneal fold, is a vascular apron that hangs down from the greater curvature of the stomach, overlying coils of intestine. Though strictly speaking it is four layers fused together, it consists of two closely applied layers of peritoneum enclosing blood vessels and lymphatics making it is the most vascular part of the peritoneum. It is also known as the 'policeman' of the abdomen, since it can move to a site of infection and adhere to it & bring protective leucocytes to the area of pathology and 'wall off' the inflammatory region. It has a continual attachment from abdominal oesophagus to duodenum, along the greater curvature of stomach. It is attached

to the posterior abdominal wall along the origin of the small intestinal mesentry and anterior to the head and body of the ppancreas. Gastrocolic omentum is the part of the greater omentum immediately below the stomach overlies and fuses with the transverse mesocolon and tranverse colon. During surgical mobilization of the transverse colon,the plane between the transverse mesocolon and greater omentum can be entered opposite to taenia omentalis.

Peritoneal compartments

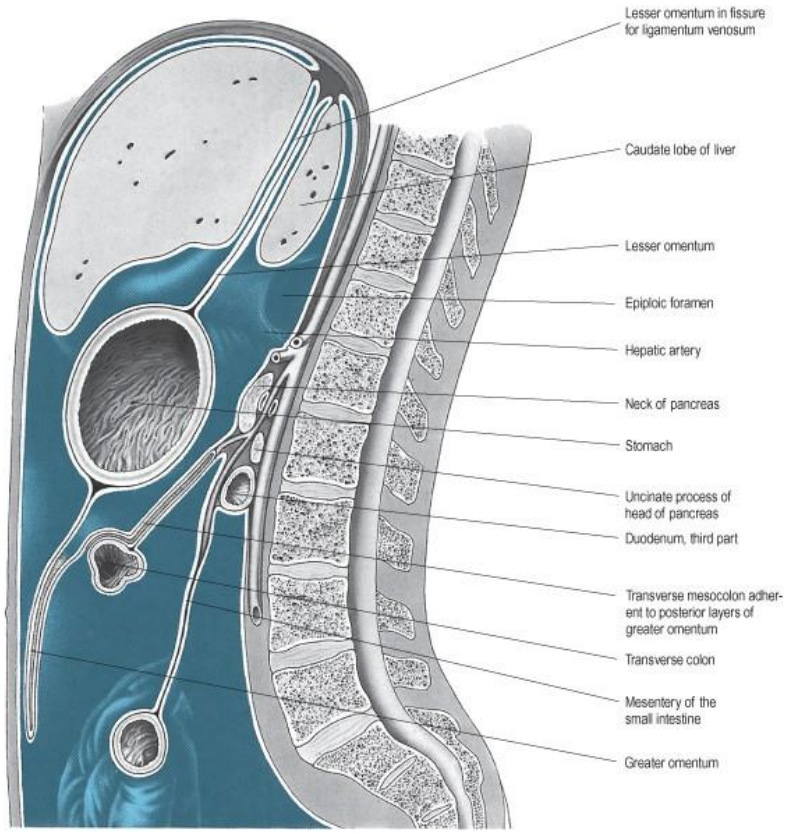


Figure 3 :Saggital section showing arrangement of peritoneum

Lesser omentum

It constitutes the two layers of peritoneum that extend from the liver onto the lesser curvature of stomach and the first inch of duodenum.

The peritoneal cavity can be divided into several spaces for clinical purposes because pathological processes are often contained within these spaces and the diagnosis and treatment might be influenced by their anatomy. By means of its attachments to the posterior abdominal wall and to various viscera, the peritoneum divides the peritoneal cavity into 3 compartments called

1.Supracolic

2.Infracolic and

3.Pelvic

The supra colic compartment:

It lies above the transverse mesocolon between the diaphragm and the transverse colon is sub divided into:

1. Right supra mesocolicspace
2.]Right subphrenic space
3. Right sub hepatic space (hepato renal recess)
4. Lesser sac
5. Left supra meso colic space = Left upper or left sub phrenic(subdiaphragmatic) compartment
6. Left lower or left perihepatic compartment

The infra colic compartment

It has two parts: Right(upper) and Left(lower).

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

The hepatorenal pouch is the lowest part of the peritoneal cavity (with the exception of the pelvis) when lying supine and hence the intraperitoneal fluid is likely to accumulate here. The common site for fluid collection is the left subphrenic space. This particularly occurs after splenectomy.

Nerve supply

Parietal peritoneum is supplied segmentally by the spinal nerves that innervate the overlying muscles and skin. Centrally, the diaphragmatic peritoneum is supplied by phrenic nerve (C4) and peripherally by lower six intercostals nerves and subcostal nerves. The remaining parietal peritoneum is supplied segmentally by intercostals and lumbar nerves. For each area of peritoneum stimulated, the sensation is usually confined to one or two dermatomes and is both lateralized and localised.

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

Discomfort from the following structures is felt in their respective regions:

- Structures derived from the foregut is felt in the region of epigastrium
- From the midgut - in the region of the umbilicus and

- From the hindgut - in the supra-pubic region.

Physiology of the peritoneal cavity

The peritoneum is the largest cavity in the body with the surface area of its lining membrane ($2m^2$ in an adult) nearly equivalent to that of the skin; it is a single layer of mesothelial cells with a basement membrane protected by an underlying layer of highly vascularized connective tissue. An estimation shows that a 1mm increase in the thickness of the peritoneum can result in these sequestration of 1.8 litres of fluid. This is a fact relevant to the massive fluid shifts associated with diffuse peritonitis.

The peritoneal cavity normally contains less than 100 ml of sterile serous fluid secreted from the peritoneal visceral surfaces; the peritoneal cavity is circulated by this liquid. The cephalad movement continues along the paracolic gutter and subhepatic spaces–via diaphragmatic movement due to negative pressure in the subphrenic zone.

Peritoneal fluid is mostly drained by the parietal peritoneal surfaces into the lymphatic circulation, with the remainder absorbed by diaphragmatic lymphatics. The removal of particulate matter, cells and microorganisms depends largely on diaphragm lymphatics.

This diaphragmatic clearance process or "pump" may be affected by many factors.

a. Platelet and talc obstruction of the stomata.
b. Head up position delays appearance of bacteria in the circulation

c. Reducing spontaneous respiration using general anaesthesia.

d. Application of positive end expiratory pressure.

The second clearance mechanism is by phagocytosis by resident peritoneal macrophages.

Local response to peritoneal infection

The inflammatory response that is characterized by hyperemia, the influx of fluid ,recruitment of phagocytic cells and fibrin deposition, occurs within the peritoneal cavity. Any harmful stimulus like endotoxin associated with gram negative bacteria, gram positive bacteria, bacteriodes species, irritants such as gastric juice, bile salts and meconium probably inciting mesothelial cell damage or direct activation of the complement system thus leading to the inflammatory process.

Upon activation, the peritoneal inflammatory cycle consists of changes in blood flow, bacterial phagocytosis enhancement, and fibrin deposition to hold or trap bacteria.

Systemic response to peritoneal infection

The systemic response to peritoneal infection imitates the response of the body to other forms of injury such as trauma or surgery. Hypovolaemia is a key phenomenon in the systemic response and is likely the result of fluid accumulation in the peritoneal cavity. The subsequent intravascular volume change leads to a reduction in venous return and cardiac output. Systemic hypotension also may be the result of the secretion of TNF, IL-1, platelet

activating factor and nitric oxide. Diminished urine flow develops as a result of the effects of increased aldosterone and antidiuretic hormone secretion, reduced cardiac output.

This is the setting that has been dubbed as “warm” septic shock and is characterized by tachycardia, fever, oliguria, hypotension and warm extremities.

Secondary abdominal distension that occurs after collection of fluid within the peritoneal cavity–imposes limitations on diaphragm movement and reduces the volume of ventilation, resulting in potential atelectasis. Accumulation of pulmonary interstitial and alveolar fluid decreases pulmonary adherence and increases breathing activity. Early manifestation is hyperventilation and breathing alkalosis. With the worsening pulmonary edema and alveolar collapse; with the development of extreme hypoxemia, adult respiratory distress syndrome (ARDS) develops.

During the reaction to peritonitis tissue hypoxia, the metabolism of the tissue is significantly altered, leading to anaerobic glycolysis leading to metabolic acidosis.

Classification, Etiopathogenesis and Pathology

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

I.**Primary peritonitis**

A. Spontaneous peritonitis of childhood

B. Spontaneous peritonitis of adult

C. Tuberculosis peritonitis

D. Peritonitis with continuous ambulatory peritoneal dialysis

II . Secondary peritonitis

A.Perforation peritonitis

A. Gastrointestinaltract perforation

B. Peritonitis after translocation of bacteria

C. Pelvic peritonitis

B.Post operative peritonitis

Enterocutaneus fistula

I.Primary peritonitis

Primary peritonitis occurs as a result from bacteria chlamydial, fungal or mycobacterial infection when there is no perforation of the gastro intestinal tract. The infection is a pure infection with Streptococcus, Pneumococcus or Haemophilusbacteria.

II.Secondary peritonitis

It occurs in the setting of the gastrointestinal perforation. These episodes occur majorly as a result of primary lesions of the stomach, duodenum, small intestine, colon and appendix. It is the most common form of peritonitis by far.

III.Tertiary peritonitis

It develops after the treatment of secondary peritonitis and it is a representation of either a failed host inflammatory response or a superinfection.

Peritonitis that occurs secondarily after a hallow viscus perforation is defined as the end result of a disease process of trauma which extends through the muscular and serosal walls of the gastrointestinal tract, thus establishing a communication between the lumen of the viscus and the surrounding body cavity and allows free oozing of the luminal contents into the cavity.

Table No: 1

Causes of perforative peritonitis

Source regions	**Causes**
Stomach	Peptic ulcer perforation Malignancy (e.g.adenocarcinoma, lymphoma, gastrointestinal stromal tumour)
Duodenum	Peptic ulcer perforation
Small bowel	Salmonella enteritis Ischemic bowel, Crohn's disease Meckel diverticulum, intestinal tuberculosis Incarcerated hernia (internal and external) Parasitic peritonitis due to perforation by round worm

	Closed loop obstruction Malignancy (rare)
Large bowel **And appendix**	Ischemic bowel Diverticulitis Malignancy Ulcerative colitis and Crohn's disease Appendicitis Colonic volvulus Amoebic colitis

History;

Till the end of last century, the intra abdominal infections were treated nonoperartively with a mortality rate of 90%. Surgical principles were commenced during the first two decades of this century and have been uniformily applied in the management of peritonitis since 1930. The principles which have remained unchanged are:

(i) Elimination of the source of infection and

(ii) Removal of infected material from the peritoneal cavity.

With the widespread application of these concepts to the treatment of peritonitis, mortality fell to 40-50 % to 18. This fall is due to a better understanding of the disease bacteriology, the availability of effective antibacterial agents against both aerobes and anaerobes seen in peritonitis, and a better understanding of the dysfunction of the organ system in sepsis, and the active multi-system aid of the I.C.U. framework. With the emergence of new issues such as microbial resistance, the declining trend seems to have reached a plateau.

Bacteriology of Peritonitis

The insight gained into the disease's bacterial etiology has led to significant developments in the antimicrobial therapy. While Freidrich and Heyde described most of the bacteriological etiology of peritonitis in the 1920s, most surgeons remained unclear about the important role of anaerobes until the 1970s. The bacteria released into the peritoneal cavity after a hollow viscus perforation induced secondary peritonitis. The two main facts that are of paramount importance in the management of peritonitis are the polymicrobial nature of the disease and the mixed aerobic anaerobic pathogens which occur as the most prevalent offending bacteriological combination. The morbidity and mortality associated with the existence of Enterococcus, which has not been affected by antibiotic treatment, would appear to suggest the pro-inflammatory ole of eneteroccci.

Antibiotic selection

When selecting an antibiotic for the patient with perforative peritonitis, the following consideration should be kept in mind:

1. It should be directed against the well known typical spectrum of aerobic and anaerobic organisms.
2. It should achieve effective concentration in the blood and peritoneal fluid.
3. It should be backed by the results of valid clinical trials.
4. It should be safe and devoid of serious toxicities.

The emerging concepts concerning antibiotic treatment suggests that less in terms of the number of drugs and the duration of treatment is better.

Peptic Ulcer disease

Epidemiology

Peptic ulcer disease remains one of the most prevalent gastrointestinal diseases. Elective admission has decreased dramatically while admissions for complications related to ulcer disease have shown little change. Peptic ulcer disease has decreased in men and increased in women. Although the reason for the decrease in men is unknown, Itmay reflect the decrease in smoking among men. It is speculated that the increase in women with peptic ulcer disease was in past due to an increase in smoking and at present due to an increase in NSAID ingestion. On the other hand there has been a consistent increase in the age of the population affected by perforated peptic ulcer in virtually every study world wide.

Recognition of the roles that Helicobacter pylori (H.pylori) and non-steroidal anti-inflammatory drugs (NSAIDs) play in peptic ulcer disease has revolutionized the care of patients who suffer from gastro-duodenal ulcer. Recent data suggest that more than 99% of all duodenal ulcers and 96% of all gastric ulcers are associated with either H.pylori infection or NSAID use. Although the indications for surgery have not changed dramatically over the last several decades, i.e. perforation, bleeding, obstruction, the type of operation has changed in the H.pylori era. However, recent studies indicate that vagotomy may not even be

necessary in some situations such as perforation of the duodenum, provided the H. pylori is eradicated.

Location and type of ulcer

Peptic ulcer disease can be divided into gastric and duodenal ulcers. Both types tend occur near mucosal junctions. For example, duodenal ulcers usually occur at the duodenal pyloric junction, whereas gastric ulcers tend to occur at the oxyntic– antral junction, the antral pyloric junction. Duodenal ulcer disease is a disease of multiple etiologies. The only absolute requirements are secretion of acid and pepsin in conjunction with either H.pylori infection or ingestion of NSAIDs.

In comparison gastric ulcer may present in four forms:

Classification of benign gastric ulcers (afterJohnson,1957)

Acute superficial

Single or multiple (erosions)

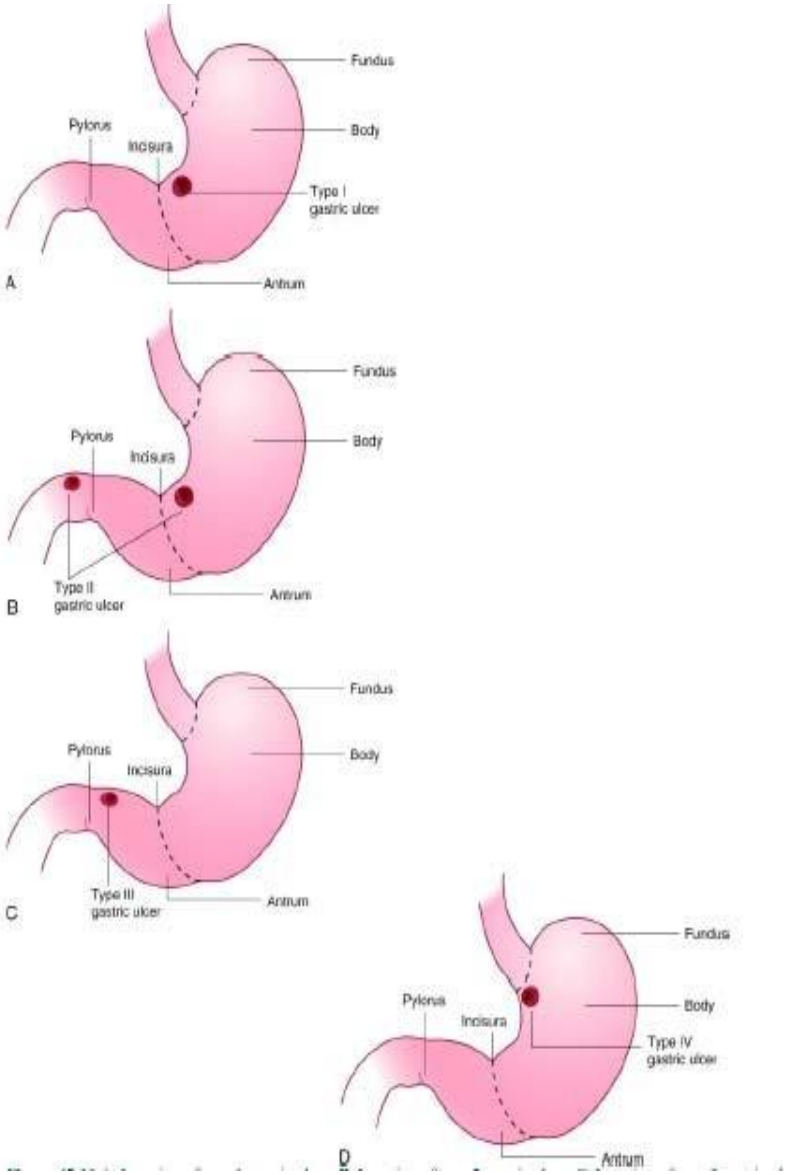
Chronic

TypeI Usually lesser curve

TypeII Combined with duodenal ulcer

TypeIII Prepyloric

TypeIV Proximal stomach <2cm from oesophageal junction


Fundus
Body
Pylorus
Incisura
Type I gastric ulcer
Antrum
A
Fundus
Body
Pylorus
Incisura
Type II gastric ulcer
Antrum
B
Fundus
Body
Pylorus
Incisura
Type III gastric ulcer
Antrum
C
Fundus
Body
Pylorus
Incisura
Type IV gastric ulcer
Antrum
D

A. Location of type1 gastric ulcer. B, Location of type 2 gastric ulcer. C, Location of type3 gastric ulcer. D, Location of type 4 gastric ulcer

Pathogenesis

I. **Helicobacter pylori**

Bircher's first description in 1874 is now known to be associated with 90% of duodenal ulcer and about 75% of gastric ulcer. The first to recognize and isolate the species for which they won the Nobel Prize was Warren and Marshell. The organism is a spiral or helical gram-negative rod with 4 to 6 flagella residing within or below the mucus layers in the gastric form epithelium, which protected it against acid. In its function, it is fastidious.

Proposed methods for Gastrointestinal injury caused by H.pylori is:

1. Development of toxic products to cause injury to the local tissue (especially the Cag A and Vac A cytotoxins).

2. Induction of immune response to the regional mucosal.

3. It hydrolyses urea resulting in the production of ammonia which acts on G cells resulting in increased levels of gastrin resulting in an increase in acid secretion.

The gastric mucosal barrier is spread by the development of a strong mucolytic endopeptidase and by the generation of large amounts of ammonia with an increase in the pH of the epithelial layer. The latter changes the gradient of the

mucosal load, cellular permeability and epithelial activity Na+K+-ATPase leading to back diffusion of H+. It also induces local inflammatory activity. H. Pylori can only exist in gastric epithelium, since only gastric epithelium expresses unique organism-recognized adherence receptors.

II. **Drugs**

After H.pylori infection, ingestion of NSAIDs is the most common cause of peptic ulcer disease. The increased risk of bleeding and ulceration is proportional to the daily dosage of NSAID. In addition, the risk for complications increases with age older than 60 years patients having prior GI event or concurrent use of steroids or anticoagulants. Consequently, the ingestion of NSAIDs remains an important factor in ulcer pathogenesis, especially in relationship to the development of complications and death. NSADs increased the risk of gastro intestinal complications approximately 2 to

10 fold. NSAID ingestion not only causes acute gastro duodenal injury but is also associated with chronic gastro duodenal injury. This risk of mucosal injury and or ulceration is roughly proportional to the anti-inflammatory effect associated with each NSAID. The acute gastroduodenal lesions typically appear within 1-2 weeks of ingestion and range from mucosal hyperaemia to superficial gastric erosions, chronic injury typically occurs after 1 month. Again ulcer risk is dose related. In comparison to H.pylori ulcer frequently found in the duodenum. NSAID induced ulcers are more frequently found in the stomach. H.pylori ulcers

area is nearly associated with chronic active gastritis whereas gastritis is not frequently found with an NSAID induced ulcer.

The increase in perforation in the elderly might largely be due to widespread use of NSAID in this group. There was a significant association between perforation associated with NSAID use and the lack of need for subsequent definitive surgical treatment.

Corticosteroids have similarly been implicated, and the association with perforation appears just as strong.

Stress

Psychological stress has been implicated in the etiology of peptic ulcer. EversinceBeaumont's classic observations, a subsequent study from Belfast failed to show any association between psychological stress and perforation of peptic ulcer.

Duodenal ulcer:

Most occur in first part of the duodenum (generally within few Centimeters of the pyloric Ring). A chronic ulcer penetrates the mucosa and into the muscle coat, leading to fibrosis.

The anterior wall of the duodenum is more affected than the posterior one. The fibrosis causes deformities such as pyloric stenosis.When an ulcer heals, a scar can be observed in the mucosa. The situation in which there in both a posterior and an anterior duodenal ulcer is referred to as'kissing ulcers'. Anteriorly placed ulcers tend to perforate and, in contrast posterior duodenal ulcers tend to

bleed, sometimes by eroding a large vessel such as the gastro-duodenal artery with respect to the giant duodenal ulcer, malignancy in this region is so uncommon. Majority have a single ulcer.

Peptic ulcers are usually round in shape sharply punched out defects in the mucosa that penetrate at least into the submucosa, usually into the muscularis and sometimes more deeply. Most are 2-4cm in diameter; those in the duodenum tend to be smaller. Large chronic ulcers may erode posteriorly into the pancreas and on other occasions, into major vessel such as splenic artery. The mucosal margins of the crater are perpendicular and there is some mild edema of the adjacent mucosa.

Heaping up of these margins is rare in the benign ulcer but is characteristic of malignant lesion. The base of the ulcer is smooth and clear owing to peptic digestion of any exudates. Scaring may involve entire thickness of the stomach; puckering of the surrounding mucosa creates mucosal folds, which radiate from the crater inspoke like fashion. Chronic gastric ulcers are much more common on the lesser curve than on the greater curve.

Ulceration of the small intestine is a lesion of multi factorial origin. In developing countries, spontaneous ileal perforations are reported to be mostly because of foreign bodies, radiotherapy, drugs, Crohn's disease and malignancies. But in developing countries like ours, enteric perforations is still the commonest.

Typhoid enteritis

Typhoid fever remains a significant problem in developing countries. Typhoid enteritis is an acute systemic infection of several weeks duration caused primarily by Salmonella typhosa. The pathologic events of typhoid fever are initiated in the intestinal tract after oral ingestion of the typhoid bacillus. These organisms penetrate the small intestinal mucosa, making their way rapidly to the lymphatics and then systematically hyperplasia of the reticulo-endothelial system, including lymph nodes, liver and spleen is characteristic of typhoid fever. Peyer's patches in the small bowel become hyperplastic and may subsequently ulcerate with complications of hemorrhage or perforation which are the complications requiring surgical intervention. Perforation usually takes place in the 2ndto 3rdweek of illness. It is seen in only 2% of cases.

Pathology

Typhoid ulcer is an oval mucosal defect with the long dimension in the axis of the bowel and usually situated in the terminal ileum. There will be hyperplasia and ulceration of the Peyer's patches of the intestine, mesenteric lymphadenopathy and splenomegaly.

Acute appendicitis

In early19thcentury the appendix was recognized as an organ capable of causing disease. In 1886 Reginald Fitz made a land mark contribution by discussing the appendix as the primary cause of right lower quadrant

inflammation. Acute appendicitis is relatively rare in infants. Appendicitis occurs frequently in very young children and elderly persons. The disease has a maximal incidence in patients in their late teens and 20s, after middle age the risks of developing appendicitis in the future in quite small. There is a slight increased prevalence in males versus females.

Pathophysiology

There is no unifying hypothesis regarding the aetiology of acute appendicitis. The inciting event in most instances of appendicitis is obstruction of the appendicular lumen. Viral infection may initiate the event. This may be due to lymphoid hyperplasia, inspissated stool (a fecolith) or some other foreign body. The incidental finding of a fecolith is a relative indication for prophylactic appendicectomy. Obstruction of the lumen leads to bacterial over growth as well as continued mucus secretion. This causes distension of the lumen, and the intraluminal pressure increases. This may lead to lymphatic obstruction. Resolution may occur at this point venous obstruction occurs then. With bacterial over growth and edema, an acute inflammatory response ensues. The appendix then becomes more edematous and ischemic necrosis of the appendiceal wall subsequently occurs with translocation of bacteria through the ischemic wall. This is gangrenous appendicitis. Without intervention the gangrenous appendix will perforate with spillage of the appendiceal contents into the peritoneal cavity.

The appendix contains an inflammatory response and an omentum or small intestine loops contributing to acute peritonitis and finally a phlegmonic mass or

an appendix abscess. The patient may develop diffuse peritonitis if the body does not wall off the system. Appendix inflammation never reduces, leaving a distended mucus-filled tissue called the appendix's mucocele.

Clinical characteristics

Peritonitis is a inflammation involving either a portion or all of the peritoneal cavity's parietal and visceral surfaces. The perforation's signs and symptoms differ depending on the time that has passed since the rupture occurred. There are three steps which can be identified in the pathological phase.

It is possible to list the symptoms of each phase.

Early (within the first two hours)

- Severe and generalized abdominal pain
- Anxious countenance
- Livido rash appearance
- Cold, sweating face
- Cold extremities
- Subnormal temperature(95°For96°F)
- Pulse low and weak
- Retching or vomiting(slight)
- Shallow respiration
- Pain on top of one or both shoulders

Intermediate (two to twelve hours)

- Cessation of vomiting
- Normal temperature or slightly elevated temperature
- Decreased abdominal pain
- Abdominal wall very rigid, tender
- Tender pelvic peritoneum
- Great pain on movement of the body
- Diminution of liver dullness

Late (after twelve hours)

- Vomiting more frequent but still not profuse.
- Facies of late peritonitis classically described as Hippocratic facies
- Abdomen tender and distended
- Pulse rapid and low
- Hypovolemic shock may be present.
- Temperature usually elevated.

INVESTIGATIONS

Laboratorystudies

Complete blood cell count (CBC) with differential count in patients suspected of having peritoneal infection. Most intraabdominal patients show leukocytosis (> 11,000 cells / mm3) with the differential cell count shifting to the immature forms. Nevertheless, patients who are immunocompromised or patients with certain forms of infection (e.g. typhoid) can show absence of leukocytosis

and may even show leukopenia in patients with a clinical picture troubling of shock, an examination of arterial blood gas and lactate level may provide additional information about the degree of physiological impairment and may help guide recovery.

Analysis of urine is important to exclude diseases of the urinary tract (e.g. pyelonephritis that mimic peritonitis). Nonetheless, in urine and micro hematuria, patients with lower abdominal or pelvic infection also exhibit WBC. The presence of frank pyuria, a large number of red blood cells and bacteria in the sample suggests a urinary cause of symptoms for the patient.

For patients with possible diagnosis of pancreatitis, serum amylase and lipase levels are increased.

Widal test

It is a test for the measurement of H and O agglutins in the patient's sera for typhoid infection. The results are interpreted by the agglutination titre accordingly. The test is taken as positive if titre is greater than 1/100 for O agglutins and 1/200 or more for H agglutins or rise in titre is demonstrated.

Peritoneal fluid

To order to rule out peritoneal disease, a peritoneal fluid should be tested for glucose protein and lactate dehydrogenase, and gram stain, aerobic or anaerobic culture.

In specified acute disease entities (i.e. gastro-duodenal ulcer perforation, appendicitis, and diverticulitis, colon perforation caused by obstruction or

ischemia) routine intraoperative peritoneal fluid cultures is done When pancreatitis or pancreatic leakage is suspected, a peritoneal fluid amylase should be performed; creatinine level when a urinary leak is suspected. It is important to compare peritoneal levels with serum levels. In just 8-10 percent of the time, the antibiotic regimen is based on surgical culture data (Bilik, 1998).

Radiograph:

Free intra-abdominal gas presence almost always suggests hollow viscus perforation. The most common cause is peptic ulcer perforation; diverticulitis and malignant tumors are other causes that are much less common.

Approximately 70% of perforated ulcers can exhibit free gas, a phenomenon that is also observed in perforated appendix cases On a radiograph, as little as 1 milli litre of free gas can be shown, either runs erect chest or left abdominal decubitus. Radiographic techniques are important and the patient should remain in position for 5-10minutes.

The clinical condition of the patient will determine the radiographic technique used. Chest films taken with the patient in an upright position ideal for demonstrating free air because the X-ray beam strikes diaphragms tangentially at their highest point.

A lateral decubitus or a supine radiograph is used in patients are severly ill to be moved. Left lateral decubitus visualization of the abdomen are used in detecting small amount of free air interposed between edge of the liver and the lateral wall of the peritoneal cavity. Care & steps should taken to include the

upper abdomen, because air floats to the highest point of abdomen, which usually is beneath the lower ribs. Films obtained with patient in the right lateral decubitus position are helpful, but gas in stomach or colon mayblur small amounts of the free gas. In erect film, pneumo- peritoneum can be detected in 76% of cases but in left lateral decubitus projection, a pneumoperitoneum can be demonstrated in nearly 90%ofcases.

Signs of a pneumoperitoneum on the supine radiograph

Right upper quadrant gas:

- Perihepatic
- Subhepatic
- Morrison'spouch
- Fissure for the ligamentum teres
- Rigler's or Double wall sign: Ligament visualization Falciform (ligamentum teres)
- Umbilical (inverted'V'sign), medial and lateral
- Urachus Triangular air The cupola sign
- Football or air dome

Pseudo pneumo peritoneum

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

1. Chilaiditi syndrome: is distended bowel, usually hepatic flexure of the colon, interposed between the liver and the diaphragm.
2. Sub diaphragmatic fat
3. Curvilinear pulmonary collapse
4. Uneven diaphragm
5. Distended viscus
6. Subphrenic abscess

Pneumoperitoneum without peritonitis

Occasionally, asymptomatic patients or those with very minimal signs and symptoms are found to have a pneumoperitoneum. Causes of pneumoperitoneum without peritonitis are:

- Silent perforation of a viscus which has sealed itself
- Post operative setting
- Laparoscopy
- Perforated jejunal diverticulosis
- Peritoneal dialysis

Use of contrast media in suspected perforation

Not uncommonly, a patient presenting with severe upper abdominal pain had equivocal clinical signs and no free gas can be on plain radiographs. Water soluble contrast medium (about 50ml) is given orally or injected through a naso-gastric tube, with the patient on his/her right side. After the patient has remained in this position for 5 minutes, the patient can be examined fluoroscopically or the

abdominal radiographs can be repeated. Duodenal ulcers which have perforated but show no free gas will usually demonstrate evidence of a leak of contrast medium. It is used frequently to delineate anatomy and confirm the presence of a contained perforation, usually if non-operative management of the perforated ulcer is considered. Patients with pancreatitis have an oedematous stretched duodenal loop. If the patient's clinical condition is such that there is risk of inhalation and leading to pulmonary oedema ionic water soluble contrast medium should not be given.

Appendicular perforation

A ruptured appendix may lead to the development of a small amount of free intra peritoneal air rarely. The obstructed appendiceal lumen prevents larger collection of gas from entering into the peritoneal cavity except in a ruptured gas containing abscess. It might show a fecolith in the right lower quadrant.

Ultrasound

Ultrasound examination allows quicker screening of patients in suspected patients, for triage of patients who are to undertake more invasive imaging testing. Visualization of a interference echo with a shifting phenomenon is definitive indication of the presence of free air in the abdominal cavity. This interference echo can be defined as the interruption of echo transmission caused due to the space between the parietal peritoneum and the surface of the liver. The free air within the peritoneal cavity shifts by changing the patient's position. Unlike free peritoneal fluid, the localized exudates donot change shape or

location when the patient's position is changed. Subphrenic or subhepatic collections are other findings. And ultrasound can detect ascetic fluid as minimal as10ml. Ultrasound guided paracentesis is yields a fluid aspirate in nearly 100% compared to clinical diagnosis is with a sensitivity of 58% and is also safe

Computed tomography of abdomen

'CT' scan gives more information and the diagnostic test when the differential diagnosis remains wide.

The computed tomography diagnosis of perforation is based on the direct findings of extra luminal air or gastrograffin. Indirect findings are an abscess or Inflammatory mass surrounding an enterolith in the region of appendix or a bowel wall related phlegmon or abscess with fluid in the mesentery or surrounding radio opaque foreign body. Computed tomography is a valuable method in the diagnosis of alimentary tract perforation. The diagnosis can be established rapidly without patient preparation and with a high sensitivity.

Differential diagnosis

There are three conditions, sometimes giving rise to symptoms similar to those of perforated ulcer that either do not call for operation or in which operative interference is possibly contraindicated. They are:

1.Intestinal colic

Diagnosis usually clears on consideration of the patient's history and on careful observation of the condition of the abdominal wall, liver dullness, and the

pelvic peritoneum.The radiation of the pain of biliary colic to the subscapular region and tha to frenal colic to the groin are sufficiently diagnostic. In ureteral stone colic, the abdominal wall is not usually rigid, and the sufferer may throw them selves about .After perforation of an ulcer ,pain increases on movement and prevents movements .The pain of renal colic is nearly always limited to one side.

2.Right sided or bilateral pneumonia or pulmonary infarction

The respiration rate will be greater than one would expect with an early peritonitis without distension .A friction rub may be heard on auscultation of the chest.A postero-anterior and lateral chest X-ray will solve the diagnostic dilemma.

3.Acute pancreatitis

Abdominal rigidity is not so generalized and constant. Cyanosis and slight jaundice are more often seen in pancreatitis,which often occur in obese patients.

4.Intestinal obstruction

In their late stages ,it is difficult to distinguish intestinal obstruction from perforation .The board like rigidity accompanying a perforated ulcer tends to diminish some what as the distension increases. In such cases the history and possibly the character of the vomit may serve to differentiate these conditions.

5.Acute appendicitis

In the second stage perforated ulcer may be and often is misdiagnosed as appendicitis. The intensity of initial collapse and the persistence and maximal

degree of tenderness over the duodenal are a help to differentiate. In appendicitis the abdominal rigidity is seldom as extensive or as marked as in perforated ulcer and the liver dullness is normal.

6.Ruptured ectopic gestation

The main points in diagnosis are features of hemorrhagic shock such as the blanching of the lips, tongue, nails and the absence of true abdominal rigidity, though the abdomen is tender especially in the lower part.

7 .Rupture of an ulcer with formation of localized subphrenic abscess

Due to previous adhesions, slow leakage of the escaping gastric contents does not flood the peritoneal cavity and the symptoms are modified. The pain may be very great but the initial collapse is not so prostrating, and the abdominal signs will soon be localized to the upper segment of the abdomen and lead to the development of a subphrenic abscess containing gas. If such an abscess develops anteriorly,the local signs of intra peritoneal suppuration are very evident, but when the mischief is high up under the diaphragm, the signs and symptoms take longer to develop. Temperature, rigors, leukocytosis and dullness at the base of the lung consequenton pleural effusion or local congestion will diagnose a collection of pus under the diaphragm.

Treatment

Once the clinical diagnosis of peritonitis is made ,rapid institution of both physiologic support and aggressive anti-infective therapy are imperative .[9]In case

of doubt ,early surgical intervention is to be preferred to await and see policy.[59]Primary objectives in the treatment of peritonitis are:

1. Resuscitation
2. Initiation of antibiotic therapy
3. Elimination of the source of bacterial contamination
4. Reduction of the bacterial inoculum
5. Continued metabolic support

Resuscitation

It is an axiom that in all cases of peritonitis ,some degree of hypovolaemiais present. The plasma volume must be restored and the plasma electrolyte concentration corrected. Fluid administered must contain both crystalloids and colloids. The effectiveness of fluid replacement can be judged by the normalization of pulserate, bloodpressure and mental status. Placement of a urinary drainage catheter is essential sincere storation of urine out put is are liable indicator of adequate fluid resuscitation. Placement of central venous line is imperative for monitoring accurate fluid replacement.

Naso gastric decompression using a sump tube should be used in the presence of anileus to prevent pulmonary aspiration and reduce abdominal distension. Supplemental oxygen may be necessary and in more extreme circumstances, endotracheal intubation and mechanical ventilation may be needed to preserve oxygenation.The plasma electrolyte concentrations should be

corrected if the patients recovery is delayed for more than seven to ten days intravenous feeding (hyper- alimentation or total parenteral therapy) is required.

Antibiotic Therapy

Antibiotic therapy should be initiated as soon as a clinical diagnosis of peritonitis is obtained .Administration of antibiotics prevents the multiplication of bacteria and release of endotoxins. The initial selection of antibiotic is emperic. The choice of antibiotics is made with the following considerations:

- (a)The demonstrated activity of the drug against bacteria that are presumed to be present based upon the level of gastrointestinal perforation.
- (b) The bactericidal activity of the antibiotic in the infected tissue.

Presumptive therapy should include coverage for both aerobic gram negative rods and anaerobic organisms. Agents that possess activity against aerobic gram

Negative bacilli include aminoglycoside, second and third generation cephalosporins and either ampicillin or ticarcillin combined with a beta lactam inhibitor (i.e. sulbactam or clavulanicacid).The optimal duration of antibiotic therapy must be individualized and depends on the underlying pathology ,severity of infection ,speed and effectiveness of source control.

Traditionally a 10 days therapy has been recommended, although newer studies suggest that a five- day therapy may be sufficient.

Surgical Management

urgery remains an important therapeutic modality for all cases of peritonitis. Operative management should be directed towards the control of the source of contamination. This can be accomplished by closure of the perforation, resection of the perforated viscus, or exclusion of the affected organs from the peritoneal cavity.

The secondary goal of operative management is to reduce the bacterial innoculum with the intent to prevent recurrent sepsis. Standard intra operative techniques to accomplish these goals includes wabbing and debriding fibrin, blood and necrotic material and copious irrigation of the peritoneal cavity which are generally accepted and practiced maneuvers.

Planned repeated laparotomy for generalized peritonitis is a technique developed to prevent recurrent sepsis by repetitive abdominal exploration to debride necrotic material and drain abscesses.

Perforated pepticulcer

Peptic ulcer perforation has been classified as‘ free perforation’ when duodenal /gastric contents enter in to the peritoneal cavity. It is called ‘contained Perforation when an ulcer produces a full thickness void or free spillage is

avoided by neighboring organs resulting in walling off. The word penetrating ulcer was used to describe pancreatic perforation. It is also a form of perforation that is produced. Perforation is less severe than bleeding, but is more common than obstruction.

Six to eight times more often occurs pyloroduodenal perforation than gastric perforation. Among elderly people, gastric perforation is more normal. Young men are more likely to suffer prepyloric perforation and duodenal perforation. The anterior wall reveals 90 percent of perforated duodenal ulcers. Sixty percent of stomach perforations are less rounded and forty percent are distributed throughout the uterus. A recent review found that 52 perforation patients are on ulcerative agents. Gastrografin or abdominal CT scan may be required to determine the cause of unexplained abdominal pain.60 There are two types of perforation patients: acute perforation patients with a history of ulcer symptoms of less than 3 months or no history, and patients with chronic ulcer perforation with symptoms of more than 3 months.

It is now estimated that acute duodenum perforation occurs in 5-10% of patients. Many of them with ulcer between the age of 40-50 years. 60-70 percent of patients have a history of peptic ulcer disease. All perforation patients should be treated with NSAID therapy. In the case of NSAID patients, after simple closure, the recurrence of ulcer perforation was reported as 7 percent. The chosen

procedure is a simple closure followed by eight weeks–omeprazole therapy. To add definitive surgery at the time of emergency operation is unnecessary

Perforated gastric ulcer occurs in elderly and may be associated with adenocarcinoma. This leads to greater mortality rates than the routine perforated duodenal ulcer. Gasterectomy is the operation of choice as more than 10% of benign looking ulcers may be malignant.

Over 75 years of age, coexisting cardiac or pulmonary disease, perforation of the Cardia or body of the stomach, time gap of more than 12 hours between start of symptoms and operation, and type of operation had a significant influence on hospital mortality. When a patient with peptic ulcer perforation presents to the surgeon, the Surgeons has to make five therapeutic decisions.

- Whether on operation is to be performed or not.
- Whether patient is stable to undergo operation.
- Whether to do an omental patch closure or a definitive surgery.
- Type of definitive surgery to be done.
- Whether availability of new drugs should influence the choice of operation.

Simple closure vs Definitive procedure

Simple closure was initially suggested in 1894 for patients with gastric ulcer perforation and later popularized in 1937 by Roscoe Graham in perforated duodenal ulcer. Long-term follow-up of these patients with simple closure has significantly influenced operational management over the past 10-15 years. Simple closure would result in 1/3rd of patients being satisfactory. The remaining

2/3 of patients would need acid suppression therapy or definitive surgery. In 52% of these cases, 64 complications occurred (28% had bleeding, 15% had pyloric obstruction, 9% had reperforation), according to Boey and Wong. In this group of patients, reoperation was required by 40%.

Ralph I George followed up 75 patients with simple closure for 5-10 years, 14 of whom were on ulcerative medications; drugs had a recurrence rate of 77%, showing that their ulcer diathesis was sufficiently virulent to require conclusive surgery. Boey and associates compared simple closure and closure with P.G.V. in 78 patients with acute perforation, a recurrence rate of 34% at 36 months.

This group may have a higher reoperation rate due to ethnic and regional variability.

Surgical technique

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

1. Simple opposition of the perforation
2. Omental patch technique

Suture materials such as vicryl, dexon and polydioxonone should be used to do three or sometimes four sutures. The sutures should be at least 1 cm from the edge of the defect through the maximum thickness of the duodenal wall. If the perforation is large or the duodenum is so indurated that it is unlikely to hold

sutures, the omental patch should be used. Sutures are positioned purely to establish apposition, but should not be bound to approximate the edges of the ulcer. It is necessary to bring up adjacent omentum with a vascular pedicle intake. From the superior to the inferior side of the perforation, the sutures are then successively connected to tampon the perforation with the living omental pedicle graft. The downside of stitching the ulcer closed, even if it is technically feasible, is that there is no surface contact with the anterior duodenal serosa in the omental patch applied over such a closure. In cases of large perforation or scarred, rigid duodenal wall making simple closure difficult two options are available.

A. Conversion of the perforation in to a Heineke-Mikuliczpy pyloroplasty.

B Serosal patch with proximal Jejunum

Laparoscopic approach

Laparoscopic technique have been applied to all abdominal procedures and perforated duodenal ulcer is not an exception. It was introduced by Nathansonin1990.

Two laparoscopic approaches have been developed

- Suturing technique
- Fibrin plug technique

Pneumoperitoneum is either open or closed and into the umbilicus, a 10 mm trocar is inserted Exploratory laparoscopy is done in order to confirm the diagnosis and ensure that it is technically feasible to do laparoscopic closure In the right hypochondrium (for grasper), left hypochondrium (for scissors, needle holder) and epigastrium (for suction irrigator), the working ports are then placed.

Using a 5 mm needle holder and a 2-0 absorbable suture mounted on a half-circle tip, the primary closure is carried out. As for the open procedure the omental patch technique is performed. The peritoneum is flushed by saline lavage and aspiration upon closure. It is important to use an intraluminal endoscope to help locate perforation site, to direct repair and pull omentum into perforation.

The procedure of fibrin plug involves supplying fibrinogen and thrombin solution through the separate lumen of a catheter with double lumen. A fibrin plug is formed, which seals the perforation, as the two solution meet at the perforation.

Laparoscopy is particularly useful for patients without pneumo-peritoneum or those with atypical signs and symptoms. Postoperative wound pain is minimal, thus allowing early mobilization and resumption of daily activities is quicker. With laparoscopy, the rate of postoperative chest complications is significantly low.

Perforation associated with hemorrhage:

When perforation of a duodenal ulcer is accompanied by overt gastrointestinal bleeding, a concomitant posterior ulcer should be suspected. Duodenum is opened though the anterior perforation for suture control of the posterior bleeding ulcer. An acid reductive procedure is mandatory – two alternatives being truncal vagotomy or proximal gastric vagotomy.

Definitive operations

Truncal Vagotomy with pyloroplasty

It has been used as definitive operation for perforated duodenal ulcer. It reduces the maximal acid output by approximately 50%.

Advantages

i) The lesion is removed

ii) Pyloric stenosis is avoided.

iii) Length of operation is only slightly prolonged.

The most common pyloroplasty procedure in Heineke M Kulicz pyloroplasty. The transverse closure of gastroduodenostomy is performed using an interrupted one layer closure. Operative mortality of emergency truncal vagotomy with pyloroplasty for perforated ulcers varies from 0-15% in four large series since with recurrence rate of 12-15%. Another alternative drainage procedure is gastro- jejunostomy. Truncal Vagotomy with Hemigastrectomy. The major disadvantages of truncal vagotomy with hemigastrectomy are the only a limited increase in operative time over truncal vagotomy with pyloroplasty, but

there is an 8-10% reduction in recurrent ulceration compared with truncal vagotomy with pyloroplasty. It is the choice of treatment for cases of perforation in pre- pyloric region. The operative mortality rate for resection is negligible in properly selected patients.

Proximal gastric vagotomy

The first clinical experience with this technique in addition to closure of perforation was reported by Johnston and associates in 1973. The cumulative recurrence rate in 60 patients over a period of seven years was

63% - after simple closure,

12% - after truncal vagogomy with drainage and

only 4% - after proximal gastric vagotomy with simple closure.

In case of duodenal scarring, proximal gastric vagotomy must be avoided. As suggested by Jordan, all stable patients with perforated duodenal ulcer in absence of risk factors should undergo proximal gastric vagotomy with closure of perforation.

Postoperative follow up and complications

In certain cases of acute ulcer perforation, perforation may be the end stage e.g. perforation caused by NSAID or ulcerogenic drugs.

The patients should be administered omeprazole for eight weeks. In addition, H pylori therapy is given to reduce the recurrence rate. The rate of reinfection with H. pylori after successful eradication therapy, is usually low in

adults. The calculated reinfection rate per person per year in children was 2.3% only.

Recurrence rate in acute perforation was 43% and in chronic ulcer perforation was 66 to 87% . 52% may develop complications like bleeding, pyloric obstruction and re perforation. The patients with simple closure will need acid suppression agents and eradication of H pylori throughout their life. NSAID, cigarette smoking and alcohol aggravate the disease.

Perforated gastriculcer

Compared to perforated duodenal ulcer, perforated gastric ulcer has a higher mortality rate, in most series, mortality rate of perforated gastric ulcer is 15-20%.

Taking a biopsy from the gastric ulcer or a partial gasterectomy performed is always necessary. Surgical treatment of choices involve simple closure with biopsy excision and closure, and resection. But in case of poor general condition of the patient, a simple omental patch closure along with a biopsy is sufficient. Clinical conditions of juxta pyloric ulcers emulate duodenal ulcersand are treated by truncal vagotomy and pyloroplasty or by truncal vagotomy and resection.

Benign ulcers in unstable or elderly patients may be treated with excision and closure or closure with omental patch. Excision & closure is done for an ulcer high on the lesser curvature. If the possibility of excision is low or nil, the ulcer margin should be biopsied before closure with omental patch.

Perforated stomal ulcer

Stomal ulcers frequently penetrate surrounding structures and occasionally perforate into the transverse colon leading to the formation of gastrojejunal-colic fistula. Perforated stomal ulcers might occur after a simple gastroenterostomy. Resection or reresection of the stomach including the ulcer is the most effective operative operation for patients with perforated marginal ulcers is to and also perform a vagotomy if not done earlier. Revagotomy is a must and careful attention is paid to find out the posterior vagus nerve, which is almost always missed. Patients withgastrojejunal-colic fistula are managed by gastric resection, vagotomy, and partial transverse colectomy. The condition of the patient is taken into consideration &the surgical treatment of a perforated anastomotic ulcer is dictated accordingly and it also depends on the operation originally performed. Simple closure can be done, but recurrence rate is 80%.

Perforation of small intestine

While dealing with a perforation and associated peritonitis that precludes safe primary anastomosis, a proximal stoma and distal mucous fistula are constructed in close proximity to each other. But very close construction might prevent placement of a proper fitting appliance & so it is avoided. When the patient's health returns to normalcy, both stoma and mucous fistula mobilized and an anastomosis is performed outside the peritoneal cavity. The bowel is then replaced in the peritoneal cavity, the fascia is closed, but the skin and subcutaneous tissue are left open.

Typhoid Enteritis

Treatment of typhoid fever and uncomplicated typhoid enteritis is achieved by administering antibiotics. Usage of chloramphenicol, ampicillin, amoxycyllin and trimethoprim-sulfamethaxazole have shown good results. Additionally, short courses of third generation cephalosporins have been used found to be successful in treatment of typhoid fever. Complications that require potential surgical intervention include hemorrhage and perforation. Intestinal perforation through an ulcerated Peyer's patch occurs in approximately 2% of cases. It is a single perforation in the terminal ileum typically. Single layer or two layers of simple transverse closure after excision of the edge of ileal perforation is a wide practise among many markers. Two layer closure of the perforation with or without an omental patch has the highest success rate.

With multiple perforations, which occur in about one-fourth of the patients resection with primary anastomosis or exteriorization of the intestinal loops may be required.

Fecal fistula developing due to reperforation or perforation from another ulcer is an important entity affecting mortality and to avoid this, every effort should be undertaken.

Appendicular perforation

Patients with perforation of the appendix may be severely ill and several hours of fluid resuscitation is a requirement for them before safe induction of general anaesthesia.

Early administration of broad spectrum antibiotics directed against gut aerobes and anaerobes are initiated early in the evaluation and resuscitation phase.

Performing appendicectomy is a must in children whether the peritonitis is diffuse or not, since the other course is associated with mortality. But in adults, the management of this problem is controversial. In patients with diffuse peritonitis after perforative appendicitis appendicectomy is the treatment, as the perforation continues to be a source of peritoneal contamination. Visualization of all peritoneal surfaces is necessary at operation for free perforation. After removal of purulent and feculent material and aspiration of dependent collection of pus, the peritoneal cavity should be repeatedly rinsed with warm saline solution. The placement of multiple prophylactic drains is unjustified because these drains fail to improve morbidity and mortality and there is some evidence indicate that they may actually increase it.

Perforation in ulcerative colitis

Perforation of the colon occurs in about 1 to 3 percent of patients with ulcerative colitis. The likelihood of perforation is highest in the initial attack of colitis ,and the incidence correlates with the severity of the initial attack and---extent of involvement of the colon .Although the overall incidence of perforation

during a first attack is less than 4%, if the attack is severe ,the incidence raises to about 10%,if the patient has pancolitis ,the perforation rate can rise to 15% ,if the pancolitis is associated with a clinically severe attack ,the perforation rate raises tone early 20%. The sigmoid colon is the most common site of perforation; the splenic flexure and transverse colon are next in order .Toxic megacolon precedes perforation in only 1/3rdto 2/3rdof cases ; the remaining patients perforate in the absence of recognized colonic dilatation. Corticosteroid therapy was thought to be casual factor a tone time, but now disproved,but it masks the symptoms and signs of perforation once it occurs.

The diagnosis of perforation is easy to make in an untreated patient-patient has diffuse abdominal pain , tenderness ,rigidity fever and leukocytosis and free air is seen on abdominal radiographs .In hospitalized patients symptoms and signs are blunted by therapeutic agents.

Immediate laparotomy is mandatory in patients with proved or strongly suspected perforation.

Total abdominal colectomy with endileostomy and exteriorization of the distal sigmoid as a mucous fistula is the procedure of choice for free perforation .Inpatients with sealed perforation the multiple blow hole method of Turnballisan option.

Perforation in Crohn’sdisease

All patients must undergo emergency laparotomy .Patients are resuscitated from shock with intravenous fluids and antibiotics .A solitary perforation can be managed by resection or exteriorization. Simple closure with proximal colostomy leaving the perforated segment inside should be avoided .Resection need not be definitive but just enough to excise the perforated segment and leave normal colon for colostomy.A mucous fistula (or Hartmann pouch)completes the procedure.

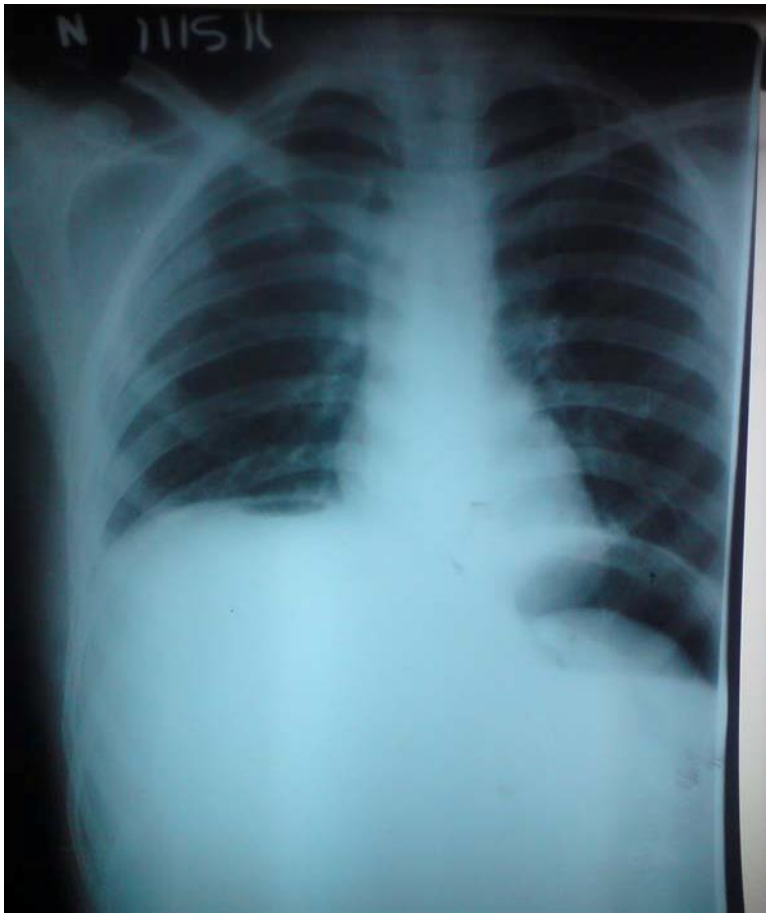


Figure 2: X-ray abdomen gas under diaphragm

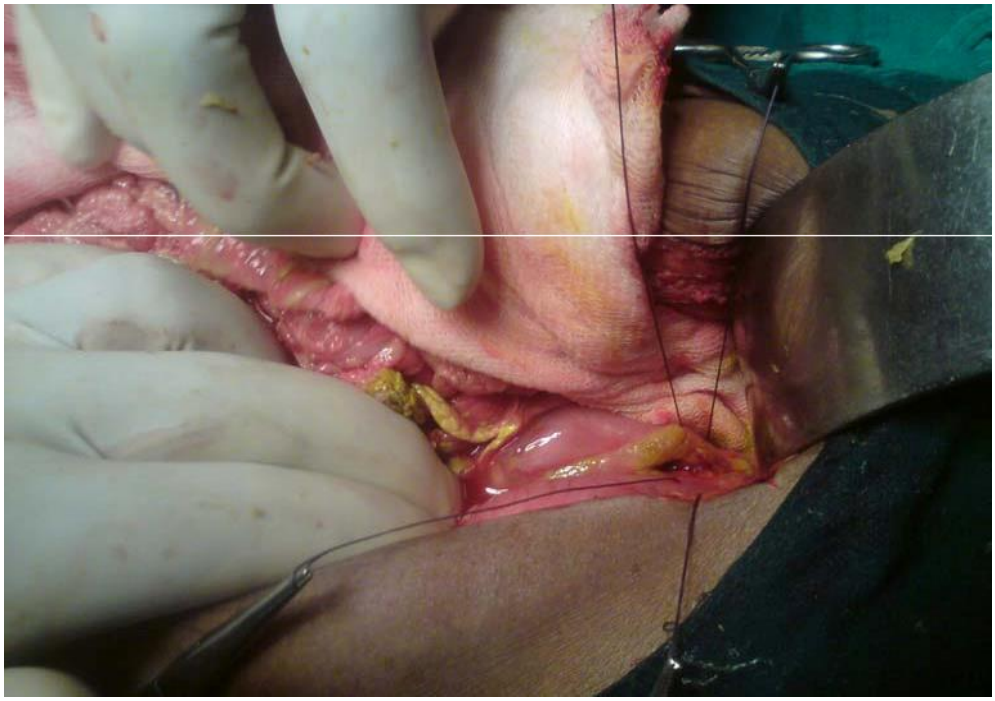


Figure 3:Duodenal ulcerperforation

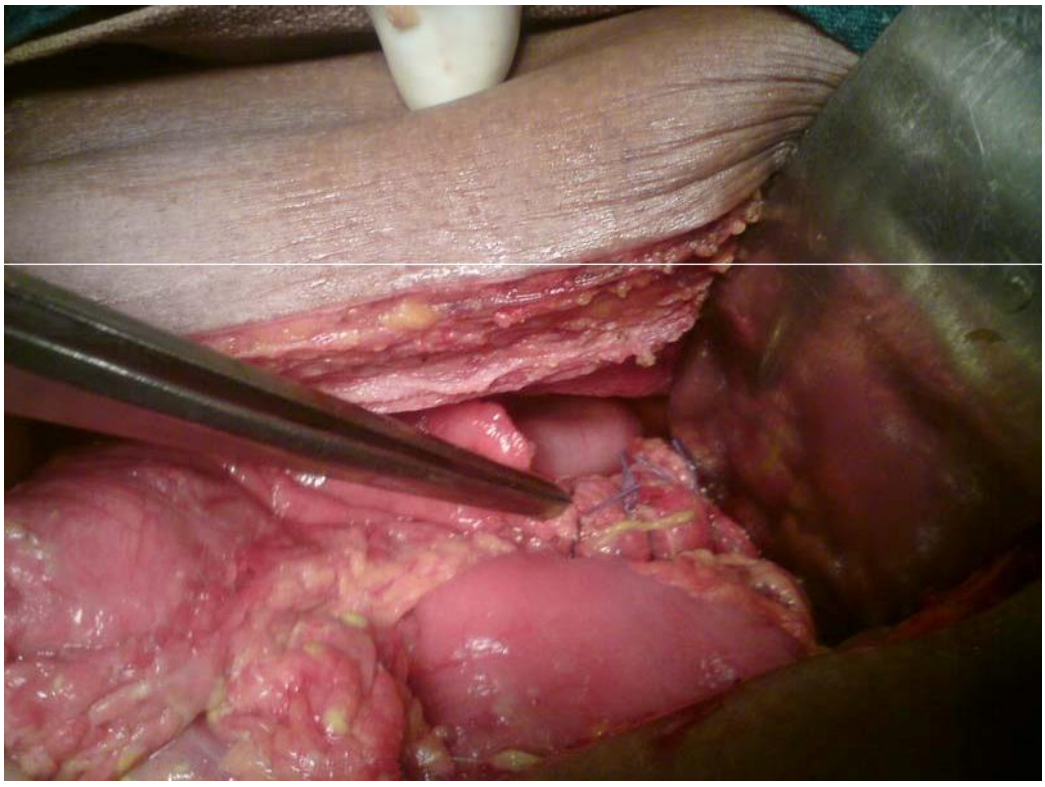


Figure 4 :Duodenal ulcer repair with omental patch closure done with vicry l2-0 with round bodied needle

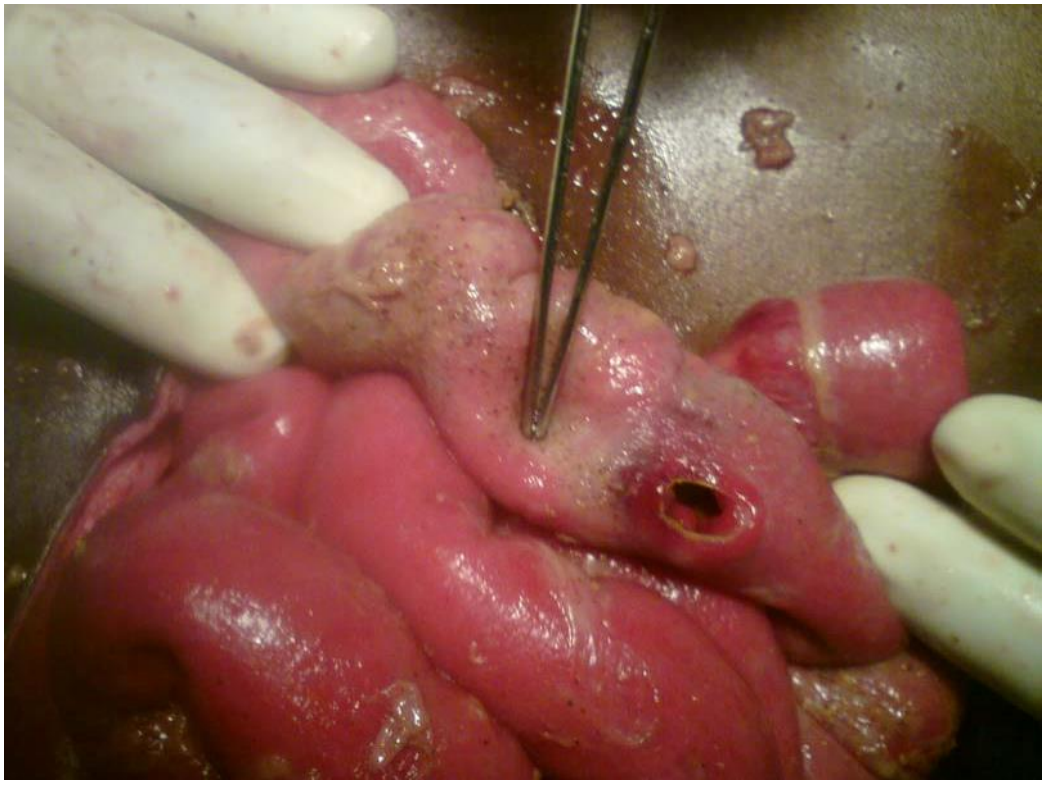


Figure 5:Ileal perforation

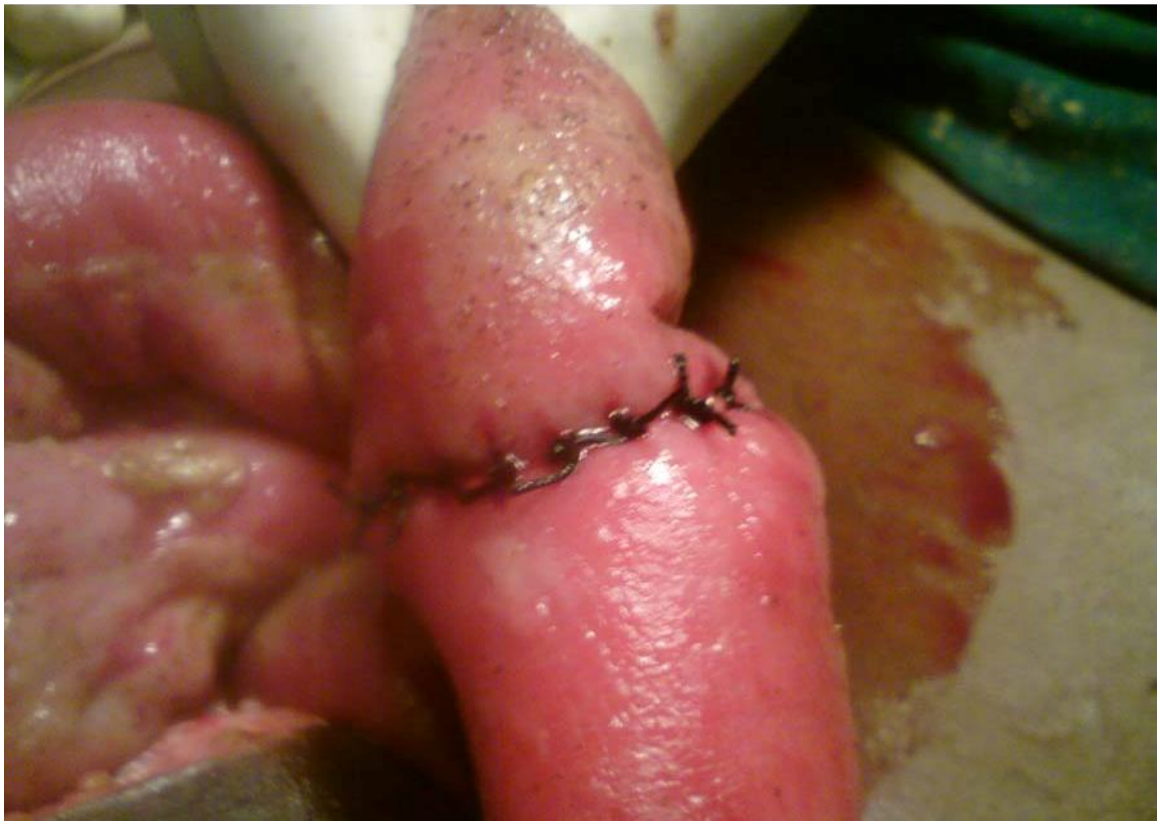


Figure 6:Resection and anastomosis for ileal perforation

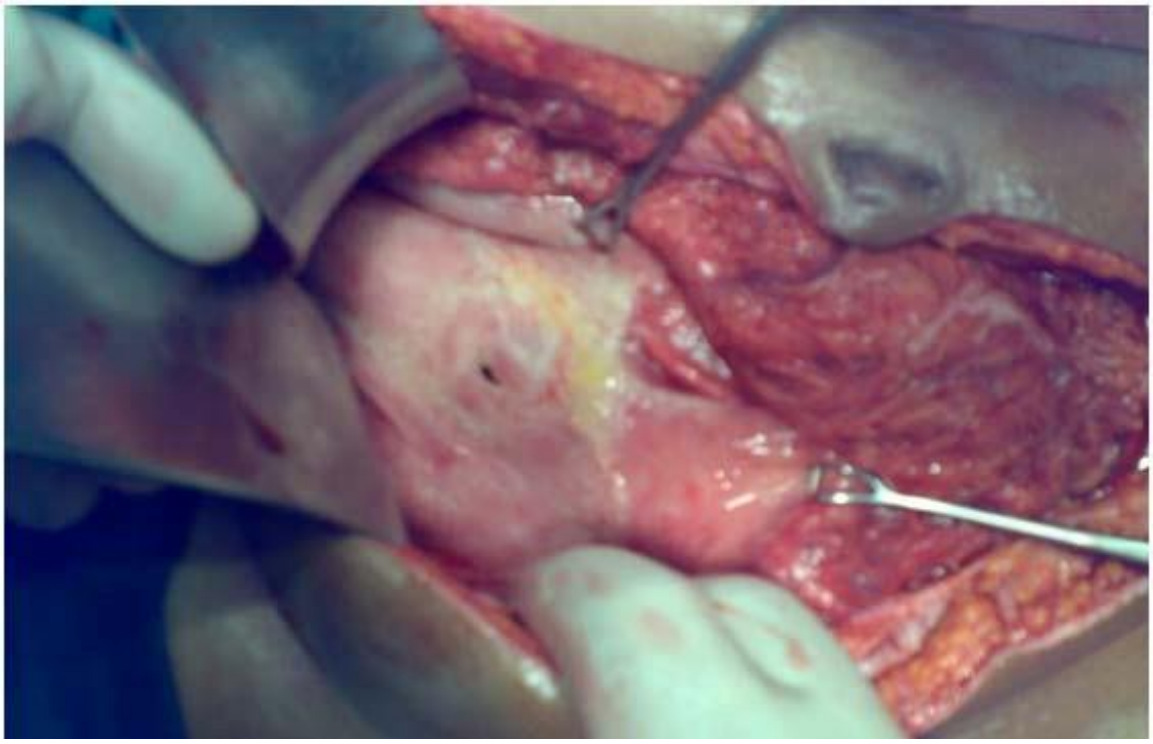


Figure 7:Gastric perforation

RESULTS

100 patients admitted in surgical ward in Tirunelveli Medical College Hospital, Tirunelveli with peritonitis secondary to hollow viscous perforation were studied.

Table No 02 :Distribution of sample by age

Age group (years)	Frequency	Percent
<19	7	7
21-29	15	15
30-39	14	14
40-49	19	19
>50	45	45

In this study most of the patients with hollow viscous perforation were above the age of 50 years followed by the age group of 40-49 years group .The youngest patient in this study was 16 years who was having ileal perforation and the oldest patients are 65years ,2 in number ,one patient with duodenal ulcer perforation and the other patient with stomach ulcer.

Table No 03: Distribution of sample by sex

Gender	**Frequency**	**Percent**
Male	84	100
Female	16	16.0
Total	100	100.0

In this study maximum number of patients were found to be males (84%) and the females constituted about 16%

The frequency of anatomical site of perforation is as follows

Table No 04: Anatomical sites of perforation

Anatomical site involved	Frequency	Percent
Stomach	4	4%
Duodenum	54	54%
Jejunum	4	4%
Ileum	8	8%
Appendix	30	30%

The commonest site involved in hollow viscus perforation in this study was duodenal ulcer perforation (54%) followed by appendicular perforation (30%) and ileal perforation (8%).

In this study ,ileal perforation constituted 26% of the patients abdominal pain was present in all cases ,vomiting was present in 8cases, fever in 12 cases ,bowel sounds was present in 3 cases and free fluid was present in 9 cases.

Three cases of ileal perforation with ischemic part were present in this study on examination there was diffuse tenderness with rigidity present in all cases and bowel sounds was absent in all cases the procedure patient went was

resection and anastomosis among the three patients one developed septicaemia and was expired, one was recovered well and the other patient developed enterocutaneous fistula.

Appendicular perforation was present in 30% of patients most of the patients were in the age group 21-29 years of age, and most presented with classical symptoms of abdominal pain, vomiting, and fever rigidity was present in all cases and tenderness was diffuse in one patient and localized to right iliac fossa in other cases.

Two gastric ulcer perforation cases were present in this study .Both the patients were male patients ,one patient having diffuse pain and the other patient having pain confined to epigastric region, no past history of pain was elicited. Guarding and rigidity was present in both cases and liver dullness was also obliterated in both cases.

Table No 05 :Distribution of the site of pain

Site of pain	**Frequency**	**Percent**
Diffuse	66	66.0
Right iliac fossa	10	10.0
Epigastric	20	20
Right hypo chondriac	4	4.0
Total	100	100

Abdominal pain was the presenting symptom in all the cases in this study and the onset was acute in patients who presented 2 days after the onset of symptoms the pain was diffuse.

Table No 06:Distribution of symptoms

Symptoms	**Frequency**	**Percentage**
Vomiting	68	68
Fever	54	54
Abdominal Pain	100	100

Vomiting is present in 68 cases and it is most commonly observed in patient presenting more than 2 days after the onset of symptoms whereas in the appendicular perforation vomiting was present in most of the patient seven from the first symptomatic day in most of the patients with the duodenal ulcer perforation the patient had previous history of abdominal pain suggestive of peptic ulcer disease history of trauma to the abdomen was present in both the cases of jejuna perforation.

Duration of pain

Table No. 7

Time of admission	Frequency	Percentage
<24 hrs	65	65
2 to 3 days	30	30
>3 days	5	5

The most common time of presenration was within 24 hours and they had good prognosis. Patients presenting after 3 days have poor prognosis.

Table No 08: Distribution of signs

Signs	Frequency	Percentage
Distension	55	55
Dehydration	55	55
Guarding and rigidity	70	70
Liver dullness obliteration	65	65
Free fluid	55	55
Absent bowel sounds	50	50

In this study guarding and rigidity was present in 70% of the patients, obliterationofliverdullnesswaspresentin65%ofcases.

Table No 09: Distribution of pneumoperitoneum in X-ray abdomen

Pneumoperitoneum	Frequency	Percent
Present	76	76.0
Absent	24	24.0
Total	100	100.0

Gas under diaphragm was seen in 76 cases (76%) irrespective of the site of perforation which was statistically significant.

Distribution of etiology

Among the causes, NSAIDS constituted the cause of 36% of the cases followed by

smoking that caused 30% of the cases. Alcohol intake was seen in 28% and steroid abuse

was seen in 2%.

Table No : 10

Distribution of types of operation

Type of operation	Frequency	Percent
Live omental patch closure	58	58
appendicectomy	30	30
Resection and anastomosis	12	12
Total	100	100

The most common procedure done was omental patch closure (58%). Appendicectomy was done in 30% of cases. Resection and anastomosis was done in 12% of cases.

Table No:11

Distribution of complication

COMPLICATION	GASTRIC	DUODENAL	JEJUNAL	ILEAL	APPENDIX	TOTAL
Wound infection	2	16	2	4	6	30
Entero cutaneous fistula	0	0	0	1	0	1
Burst abdomen	0	0	0	0	0	0
Paralytic ileus	0	0	0	1	0	1
Pelvic abscess	0	0	0	0	0	0

The most common complication following laparotomy for perforation is wound infection in my study. It was treated with antibiotic and wound wash.

Table No :12

Distribution of sample by outcome

Outcome	**Frequency**	**Percentage**
discharged	96	96
Expired	4	4

In this study the overall mortality rate was 4% irrespective of site and pathology of perforation out of 4 cases expired ,two was of ileal perforation and another two was of gastric perforation.

Table NO :13

Distribution of culture of peritoneal fluid

Culture	Gastric	Duodenal	Jejunal	Ileal	Appendicular	Total
Sterile	4	40	2	2	10	58
E.coli	-	12	2	4	15	33
Pseudomonas	-	-	-	1	1	2
Klebsiella	-	1	-	-	2	3
B.Fragilis	-	1	-	1	1	3
Staphylococci	-	2	-	-	1	3

Most patients who presented within 24 hours had no growth in their peritoneal aspirate culture. The most common organism was found to be E. Coli followed by klebsiella, B. Fragilis and Staphylococci

Table No :14

Distribution of lab investigations

	gastric	duodenal	jejunal	ileal	appendicular	Total
Anaemia	4	18	2	2	4	30
Leucocytosis	0	10	1	2	20	33
Elevated renal parameter	1	4	1	6	0	12
Electrolyte imbalance	2	4	1	4	0	11

Most of the patients with appendicular perforation has leukocytosis. Anemia was seen common in duodenal perforation followed by gastric and appendicular perforation. Elevated renal parameters and electrolyte imbalance was seen in cases which presented after 48 hours.

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DISCUSSION

This study was conducted in Tirunelveli Medical College Hospital. A total of 100patients admitted with perforation peritonitis were studied. The highest number of patients encountered in this series were in the age group 50 years and above followed by the age group of 40-49 years. The mean age group in this study was 38.56 years. This is comparable with the study by Rajender Singh Jhobta in 2010 who studied 504 cases of perforation peritonitis in which the mean age was 36.8 years. In this present study, duodenal ulcer perforation was more common in the age group of above 50 years.

The ratio of men to women with all types of perforation irrespective of site and pathological condition was 5.25:1 in the present study.

Different authors have find variable results with regard to sex ratio. Ramesh C Bharati et al in 2012 reported sex ratio of 5.50:1 in the ireview of 50 cases.

The commonest site involved in this study was duodenal ulcer perforation(54%) followed by appendicular perforation (24%) and ileal perforation (8%)

Rajender Singh Jhobta inn 2006 in his study of 504 cases of perforation peritonitis found duodenum was the commonest site of involvement, followed by appendicitis, gastro intestinal perforation due to blunt trauma abdomen. Typhoid fever and tuberculosis.

In case of peptic ulcer perforations, pain abdomen and vomiting were the predominant symptoms. In the present study, pain abdomen was present in all cases. It was diffuse in 66% and localized to epigastrium in 20% followed by RIF in 10% and right hypochondrium in 4%. Gaurding and rigidity was present in 70 patients. In 65 patients, liver dullness was obliterated. Liver dullness was not obliterated in 35 patients. Probable reasons suggested are sealing of the perforation or lack of gas at the site of perforation or adhesions around the site of perforations. Absence of liver dullness was present in all the cases of ileal perforation and 80 % of appendicular perforation. Nair S K et al . in their study of 50 cases demonstrated absence of liver dullness in 63.63% of cases.

65% of patients who presented within 24 hours of the onset of pain had good prognosis and early recovery. Those who presented late after 3 days mostly had ileal perforation.

Perforated peptic ulcer is becoming common in older patients and associated with a higher incidence of recent consumption of nonsteroidal anti inflammatory drugs (NSAIDS) and smoking. In the present series perforated peptic ulcer constituted 58% of all hollow visceral perforation. The incidence was more common in the age group 50 years and above.

All patients of perforative peritonitis were treated as a surgical emergency. Preoperatively all patients had broad spectrum antibiotic coverage, nasogastric suction and management of fluid and electrolyte imbalance and oxygen supplementation when necessary.Anemic patients required blood transfusion. Post operatively parenteral antibiotics was continued and after that oral antibiotics were given for 5days.

58 cases of duodenal and gastric ulcer perforation under went closure as described by Graham (Omentalpatchclosure).

Resection of ileum with end to end anastomosis was done in 8 cases of gangrenous bowel with perforation .Of the 30 cases of perforative appendicitis open appendicectomy was done in all the cases. The mortality rate in appendicular perforation was zero. Dandapat M Cetal in 2009 reported zero mortality rate in their study of12 cases .In all cases of peritonitis thorough peritoneal lavage was given with 0.9% saline and drains were kept in the pelvis and the site of perforation which were usually removed on the third and fifth post operative day or when the drainage <30ml. Nasogastric tube was usually removed on the second and third post operative day and started orally on fourth day depending on bowel sounds .All patients were started on chest physiotherapy from the first postoperative day.

In the present study, the mortality rate was 4% .Dandapat MC et al in 2009 recorded a mortality rate of 15.8%. Mathikere Lingaiah Ramachandra in 2008 in his study found the mortality rate as14%.[81]

Table No 14:Comparison of the present series with other studies

Author	**Year**	**Mortalityrate(%)**
Hinshaw	1968	15
Sawyer	1977	6.7
Dandapat MC	2009	10.5
Ramesh C Bharti	1996	4
Present series	2019	4

Follow up

Follow up done for all patients. In duodenal ulcer patients strict diet advise was given. After surgery all duodenal ulcer patients were given H.Pylori regimen. 20 patients had recurrence of symptoms and endoscopy was done and they were advised to continue bland diet and H.Pylori regimen for 3 months.

CONCLUSION

- The most common age group affected is 50 years and above.
- Duodenal ulcer perforations were more common in the age group of 50 years and above.
- Most of these patients present with clinical signs of peritonitis 24 hours within the onset of pain.
- Early admission and prompt treatment after diagnosis had good recovery.
- Diagnosis is made clinically and confirmed by presence of pneumo peritoneum on radiological investigation.
- Laparotomy with peritoneal lavage and perforation closure with omental patch closure of the perforation with omental patch (58%) is the commonest operative management for perforated peptic ulcer and the outcome is good.
- E coli is the most common pathogen grown in peritoneal cavity, followed by Klebsiella, B fragilis, Staphylococci.
- Leucocytosis is most commonly found in appendicular perforation followed by duodenal perforation. Anemia is most commonly found in duodenal perforation
- Early admission, prompt treatment and care will prevent the mortality.
- Irrational use of NSAID is the precipitating factor for perforation followed by smoking.
- So health education and life style modification is mandatory in the community to reduce the incidence of perforation peritonitis.

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PROFORMA

1. Case No :
2. Name :
3. Age Sex :
4. Address :
5. I.P. No :
6. Unit / Ward
7. Date of admission
8. Date of Surgery
9. Date of discharge
10. Chief complaints
    i. Pain – Onset, character, location. Duration, radiation, worsening & relieving factors.
    ii. Nausea / vomiting
    iii. Constipation / diarrhea
    iv. Others like pruritus, melena, hematochezia, hematuria
    v. Similar complaints in the past / previous surgeries
11. General physical examination

    Pallor / Icterus

    BP

    PR

12. Examination of abdomen (including external genitalia)
    i. Inspection
    ii. Palpation
    iii. Percussion
    iv. Auscultation
    v. P/R
13. Clinical diagnosis
14. Biochemical investigation

    CBC

    RFT

    LFT

    RBS

    Urine routine
15. Radiological investigation
    i. Chest X-Ray
    ii. Abdominal X-Ray Erect
    iii. Abdominal ultrasonography
    iv. CT Abdomen
16. Surgery done:
17. Post-operative diagnosis

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

S NO	NAME	AGE(YEARS)	SEX	IP NO	DOPdays	risk factos	symptoms			SIGNS						LAB INVESTIGATIONS		CULTURE	X AY aud	CLINICAL DIAGNOSIS	MANAGEMENT	OUTCOME
							abd	vo	fe	sot	dehy	g&r	old	ff	bs	ane	leuko					
1	Rajkumar	15	m	56743	>3	nil	p	a	a	rif	a	a	p	a	a	a	a	ec	a	ap	aptmy	dis
2	Balamurugan	51	m	90764	<24	nsaid	p	p	p	epi	a	p	p	p	p	p	p	s	p	dp	ome	dis
3	kalimuthu	26	m	25096	<24	a	p	p	p	epi	a	p	p	a	a	a	a	s	p	dp	ome	dis
4	Madasamy	44	m	12907	2_3	nsaid	p	p	p	epi	p	p	p	p	p	a	a	ec	p	dp	ome	dis
5	Serini vasagam	61	m	90648	2_3	s	p	p	p	epi	p	a	p	p	p	a	a	b fra	p	dp	ome	dis
6	Arun kumar	45	m	90422	<24	a	p	p	p	epi	p	a	p	p	p	a	a	s	p	dp	ome	dis
7	Sankar	52	m	21590	>3	nsaid	p	a	a	epi	p	p	p	p	p	a	a	ec	p	dp	ome	dis
8	Maiaspal	21	m	95425	<24	s	p	p	p	rif	p	p	p	a	a	a	p	s	a	ap	aptmy	dis
9	pushpam	62	f	90033	<24	S/A	p	p	p	epi	p	p	p	p	p	a	p	s	p	dp	ome	expied
10	Sornam	33	m	93210	2_3	a	p	p	p	epi	a	p	p	p	p	a	a	ec	p	dp	ome	dis
11	Ganesh	71	m	45209	<24	nsaid	p	p	p	epi	p	p	a	a	a	a	a	s	p	dp	ome	dis
12	Saravanan	27	m	7890	2_3	s	p	p	p	rif	p	a	a	a	a	a	p	ec	a	ap	aptmy	dis
13	jeyavel	34	m	24112	<24	s	p	p	p	epi	p	p	p	p	p	a	p	s	p	dp	ome	dis
14	dinesh	45	m	24245	2_3	a	p	p	p	dif	a	a	p	a	a	a	a	pseu	p	ip	r&a	dis
15	sakthi	76	f	90075	<24	c	p	p	p	epi	p	p	a	a	p	a	a	s	p	dp	ome	dis
16	Arun kumar	21	m	64952	2_3	a	p	p	p	rif	p	p	a	a	a	a	p	ec	p	ap	aptmy	dis
17	pandiyan	53	m	96259	<24	s	p	p	p	epi	p	p	p	p	p	a	a	s	p	dp	ome	dis
18	muniyandi	45	m	52112	<24	s	p	p	p	epi	a	p	p	p	pp	a	a	s	p	dp	ome	dis
19	vinoth	65	m	64214	>3	s	p	a	a	epi	p	p	p	p	p	a	a	sta	p	dp	ome	dis
20	thangarasu	21	m	35795	<24	a	p	p	p	rif	p	p	p	a	a	a	p	s	a	ap	aptmy	dis
21	Rajkumar	47	m	62699	2_3	s	p	p	p	epi	p	p	a	p	p	a	a	s	p	dp	ome	dis
22	muniyandi	54	m	679000	<24	nsaid	p	p	p	epi	p	p	a	p	p	a	p	s	p	dp	ome	dis
23	thangarasu	16	m	90909	<24	a	p	a	a	rif	p	p	p	a	a	a	a	ec	a	ap	aptmy	dis
24	guru	57	m	44212	2_3	a	p	p	p	rif	a	p	p	a	a	a	a	ec	a	ap	aptmy	dis
25	Balamurugan	33	m	43637	<24	a	p	a	a	epi	a	p	p	a	a	a	p	s	p	dp	ome	dis
26	guru	36	m	46294	2_3	a	p	p	p	dif	p	p	p	p	a	a	a	e c	p	ip	r&a	expied
27	muniyandi	67	m	90490	<24	nsaid	p	a	a	epi	p	p	a	p	p	p	a	s	p	gp	ome	dis
28	thangarasu	54	m	65992	<24	s	p	a	a	rif	p	p	a	p	a	a	p	s	p	ap	aptmy	dis
29	Rajkumar	63	m	79374	2_3	nsaid	p	p	p	epi	p	p	p	p	p	a	p	sta	p	dp	ome	dis
30	kalyan	23	m	57972	<24	nsaid	p	a	a	rif	p	p	p	p	a	a	a	s	p	ap	aptmy	dis
31	guru	62	f	64792	<24	nsaid	p	a	a	epi	a	p	p	p	p	a	p	s	p	dp	ome	dis
32	vinoth	66	m	79595	<24	a	p	a	a	dif	p	p	p	a	a	a	a	b fra	p	ip	r&a	dis
33	thangarasu	32	m	72324	>3	a	p	a	a	rif	a	p	p	a	a	a	p	s	p	ap	aptmy	dis
34	Rajkumar	61	m	46342	<24	nsaid	p	a	a	epi	p	p	p	p	p	a	a	s	p	dp	ome	dis
35	guru	56	f	45662	<24	nsaid	p	a	a	rif	p	p	p	a	a	a	p	ec	p	ap	aptmy	dis
36	dinesh	22	m	45627	<24	s	p	a	a	rif	p	p	a	a	aa	a	p	s	p	ap	aptmy	dis
37	muniyandi	13	m	24252	2_3	s	p	a	a	rif	p	p	a	a	a	a	p	ec	p	ap	aptmy	dis
38	Ganesh	45	m	74722	<24	s	p	p	p	dif	p	p	p	p	a	a	a	ec	p	jp	r&a	dis
39	Rajkumar	55	m	72412	2_3	s	p	p	p	epi	a	p	p	p	p	a	a	kleb	p	dp	ome	dis
40	kalyan	53	m	54620	<24	nsaid	p	p	p	epi	a	p	p	p	p	a	p	s	p	dp	ome	dis
41	Balamurugan	57	m	24131	<24	c	p	a	a	epi	p	p	p	p	a	p	a	s	p	gp	ome	dis
42	thangarasu	23	m	23142	<24	s	p	a	a	rif	p	p	p	a	a	a	a	s	p	ap	aptmy	dis
43	kala	35	f	79050	2_3	nsaid	p	a	a	dif	p	p	p	p	p	a	a	ec	p	jp	r&a	dis
44	muniyandi	53	m	43522	<24	nsaid	p	a	a	dif	a	p	p	pp	a	a	a	s	p	dp	ome	dis
45	dinesh	45	m	45262	<24	s	p	a	a	dif	p	p	p	p	p	a	a	s	p	dp	ome	dis
46	vinothini	58	f	23425	<24	nsaid	p	a	a	dif	p	p	p	p	p	p	p	ec	p	ip	r&a	dis
47	guru	42	m	23242	2_3	a	p	a	a	rif	p	p	p	a	a	a	p	ec	p	ap	aptmy	dis

S NO	NAME	AGE(YEARS)	SEX	IP NO	DOPdays	risk factos	symptoms			SIGNS						LAB INVESTIGATIONS		CULTURE	X AY aud	CLINICAL DIAGNOSIS	MANAGEMENT	OUTCOME
							abd	vo	fe	sot	dehy	g&r	old	ff	bs	ane	leuko					
48	kalimuthu	44	m	32424	<24	nsaid	p	p	p	epi	p	p	a	p	p	a	p	s	p	dp	ome	dis
49	thangarasu	33	m	56565	<24	a	p	p	p	rif	p	p	a	a	a	a	a	ec	p	ap	aptmy	dis
50	vinoth	59	m	92423	<24	nsaid	p	p	p	epi	a	p	p	p	p	p	a	s	p	dp	ome	dis
51	kalyan	42	m	98467	<24	a	p	p	p	epi	p	p	p	p	p	a	a	s	p	dp	ome	dis
52	Balamurugan	53	m	93242	<24	nsaid	p	p	p	epi	p	p	p	p	p	a	a	s	p	dp	ome	dis
53	Rajkumar	55	m	45323	2_3	nil	p	p	p	epi	p	p	p	p	p	p	a	s	p	gp	ome	dis
54	vinoth	59	m	23901	<24	nsaid	p	p	p	epi	p	p	p	p	p	p	a	s	p	dp	ome	dis
55	guru	24	m	32724	<24	nil	p	a	a	rif	p	p	p	a	a	p	p	s	a	ap	aptmy	dis
56	thangarasu	33	m	39024	2_3	nil	p	p	p	rif	p	p	p	a	a	a	p	e c	p	ap	aptmy	dis
57	anitha	55	f	56290	2_3	nsaid	p	p	p	epi	a	p	p	p	p	p	a	s	p	gp	ome	dis
58	surya	13	m	43427	<24	s	p	a	a	rif	p	p	p	a	a	a	p	s	a	ap	aptmy	dis
59	kumar	67	m	90453	<24	a	p	a	a	epi	p	p	a	p	p	a	a	s	p	dp	ome	dis
60	kalyani	55	f	45239	>3	nsaid	p	p	p	dif	p	p	a	a	a	p	a	e c	a	ip	r&a	dis
61	Balamurugan	34	m	37922	2_3	s	p	p	p	epi	p	p	p	p	p	a	a	e c	a	ap	aptmy	dis
62	savitha	45	f	23780	<24	nsaid	p	p	p	dif	p	p	p	p	p	p	a	s	p	dp	ome	dis
63	muniyandi	43	m	21739	<24	s	p	p	p	epi	a	p	p	p	p	a	a	s	p	dp	ome	dis
64	rahini	67	f	44221	<24	a	p	a	a	epi	a	p	p	p	p	a	a	s	p	dp	ome	dis
65	vinoth	39	m	67932	<24	s	p	a	a	dif	p	p	a	p	p	p	a	s	a	jp	r&a	dis
66	Balamurugan	49	m	45454	2_3	s	p	p	p	epi	p	p	a	p	p	a	a	ec	p	dp	ome	dis
67	kalyan	14	m	65645	<24	a	p	a	a	rif	p	p	p	a	a	a	a	ec	a	ap	aptmy	dis
68	Ganesh	68	m	90676	<24	nsaid	p	a	a	dif	p	p	p	p	p	p	a	s	p	dp	ome	dis
69	thangarasu	67	m	14094	<24	a	p	a	a	dif	p	p	p	p	p	a	a	s	p	dp	ome	dis
70	lisa	69	f	22119	<24	a	p	a	a	epi	p	p	p	p	p	p	a	s	p	dp	ome	dis
71	venkatesh	26	m	29202	<24	a	p	a	a	rif	a	p	a	a	a	a	p	e c	a	ap	aptmy	dis
72	fathima	63	f	32901	2_3	nsaid	p	a	a	rif	a	p	a	p	a	p	p	ec	a	ap	aptmy	dis
73	uma	56	f	23290	<24	nsaid	p	a	a	dif	p	p	p	a	a	a	p	e c	a	ip	r&a	dis
74	guru	27	m	54638	<24	a	p	p	p	rif	p	p	p	p	p	a	a	ec	p	ap	aptmy	dis
75	natham	55	m	43231	<24	nsaid	p	p	p	dif	a	p	p	p	p	p	p	s	p	jp	r&a	expied
76	Rajkumar	44	m	73290	<24	nsaid	p	p	p	epi	p	p	p	p	p	p	a	s	p	dp	ome	dis
77	Maiaspal	54	m	32901	<24	nsaid	p	p	p	epi	p	p	p	p	p	p	a	s	p	dp	ome	dis
78	govindammal	42	f	32172	<24	a	p	a	a	epi	a	p	p	p	a	p	a	s	p	dp	ome	dis
79	annakili	46	f	23190	2_3	nsaid	p	p	p	epi	p	p	p	p	p	a	p	ec	p	dp	ome	dis
80	mairisa peer	27	f	23456	2_3	s	p	p	p	rif	a	p	p	p	p	a	a	pseu	a	ap	aptmy	dis
81	kala	58	m	69042	<24	nsaid	p	a	a	dif	p	p	p	p	p	a	a	s	a	ip	r&a	dis
82	kavitha	37	m	42125	<24	nsaid	p	a	a	rif	p	p	p	p	p	p	a	s	p	dp	ome	dis
83	venkatesh	58	m	4321	<24	s	p	a	a	rif	a	p	p	p	p	p	a	s	p	dp	ome	dis
84	dinesh	29	m	43319	<24	s	p	a	a	epi	p	p	p	p	a	p	p	ec	p	ap	aptmy	dis
85	muniyandi	42	m	49032	<24	s	p	a	a	epi	p	p	a	p	p	p	a	s	a	dp	ome	dis
86	kumar	52	m	23549	<24	nsaid	p	a	a	dif	p	p	a	p	p	a	a	s	a	ip	r&a	expied
87	hariharan	28	m	32490	2_3	a	p	p	p	epi	a	p	p	a	p	p	p	kleb	a	ap	aptmy	dis
88	rahul	53	m	52137	<24	nsaid	p	a	a	dif	p	p	p	p	p	p	a	s	p	dp	ome	dis
89	venkatesh	59	m	27821	<24	s	p	a	a	dif	a	p	p	p	p	p	a	s	p	dp	ome	dis
90	dinesh	55	m	23939	<24	s	p	a	a	dif	p	p	p	p	p	p	a	s	p	dp	ome	dis
91	mahesh	13	m	43429	2_3	nsaid	p	p	p	rif	p	p	p	a	a	a	p	sta	p	ap	aptmy	dis
92	vinoth	51	m	32924	<24	nsaid	p	a	a	dif	a	p	p	p	p	a	a	s	p	dp	ome	dis
93	surya	21	m	24229	2_3	s	p	p	p	epi	a	p	p	p	a	a	p	b fra	a	ap	aptmy	dis
94	Balamurugan	44	m	49239	<24	nsaid	p	a	a	epi	a	p	p	p	p	p	a	s	p	dp	ome	dis

S NO	NAME	AGE(YEARS)	SEX	IP NO	DOPdays	risk factos	symptoms			SIGNS						LAB INVESTIGATIONS		CULTURE	X AY aud	CLINICAL DIAGNOSIS	MANAGEMENT	OUTCOME
							abd	vo	fe	sot	dehy	g&r	old	ff	bs	ane	leuko					
95	venkatesh	59	m	90900	<24	s	p	a	a	epi	p	p	p	p	p	p	a	ec	p	dp	ome	dis
96	pandiyan	32	m	84999	<24	s	p	a	a	epi	p	p	p	p	p	a	a	ec	p	dp	ome	dis
97	kumar	43	m	52190	2_3	s	p	p	p	epi	p	p	p	p	p	p	a	ec	p	dp	ome	dis
98	kalyan	67	m	23690	<24	a	p	a	a	epi	p	p	a	a	p	a	a	ec	a	dp	ome	dis
99	surya	39	m	56712	<24	nsaid	p	a	a	rif	a	p	a	a	p	a	p	kleb	a	ap	aptmy	dis
100	muniyandi	31	m	45423	2_3	s	p	p	p	epi	a	p	p	p	p	a	a	ec	p	dp	ome	dis