PROSPECTIVE STUDY TO IDENTIFY COMMONEST ORGANISMS AND ANTIBIOTIC SENSITIVITY IN PERITONITIS DUE TO DUODENAL ULCER PERFORATION IN GOVT. RAJAJI HOSPITAL

M.S. DEGREE EXAMINATION

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CERTIFICATE

This is to certify that this dissertation titled **"PROSPECTIVE STUDY TO IDENTIFY COMMONEST ORGANISMS AND ANTIBIOTIC SENSITIVITY IN PERITONITIS DUE TO DUODENAL ULCER PERFORATION IN GOVT. RAJAJI HOSPITAL"** submitted by **Dr.R.TENNISON** to the faculty of General surgery, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS Degree, Branch I, General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from January 2016 to August 2016.

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I , **DR.R.TENNISON** hereby solemnly declare that this dissertation entitled **"PROSPECTIVE STUDY TO IDENTIFY COMMONEST ORGANISMS AND ANTIBIOTIC SENSITIVITY IN PERITONITIS DUE TO DUODENAL ULCER PERFORATION IN GOVT. RAJAJI HOSPITAL"** is a bonafide and genuine research work carried out by me .

This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the regulations for the award of M.S. degree , Branch I, General Surgery.

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CONTENTS

INTRODUCTION:

Perforative peritonitis is one of the most commonly encountered surgical acute abdominal emergencies in surgery casuality. The importance of this study is to reduce the postoperative complications like septicemia, wound infection, burst abdomen, bronchopneumonia, long hospital stay etc., by identifying the inciting organisms and its sensitive antibiotics rather than using empirical antibiotic therapy which is followed at present, in immediate postoperative period.

Among hollow viscus perforations duodenal perforation is the most common site. The organisms setting up the peritonitis features are diverse according to the location of perforation and time interval between disease onset & time of intervention.

Most common organisms isolated in proximal bowel perforation are E.coli, klebsiella, lactobacilli, streptococci, candida . There is an increasing trend in isolating anaerobic organisms as the perforation occurs distally and the time interval between the disease onset and time of intervention increases.

Many research studies reveal that increased postoperative complications are seen in peritonitis patient with polymicrobial isolates. Majority of rural patients present in surgical opd lately. *Also there is a direct relation between interval of disease onset & time of intervention and postoperative morbidity.* In proximal

bowel perforation, proposed antibiotic coverage is against gram positive cocci and gram negative enteric organisms.

Antibiotics commonly sensitive in proximal bowel perforations are cefotaxime, piperacillin + tazobactum, ceftazidime, cefaperazone, ceftriaxone, ciprofloxacin, amikacin etc. Administration of appropriate antibiotics in immediate postoperative period rather than empirical antibiotics, reduces poor postoperative outcome.

AIM :

To study the bacteriological profile in the peritonitis patient due to duodenal ulcer perforation and their sensitive antibiotics by collecting peritoneal fluid and to reduce postoperative morbidity and mortality .

OBJECTIVE OF STUDY :

A. To detect **commonest organisms** in peritonitis due to duodenal ulcer perforation.

B. To find out antibiotic sensitivity pattern in peritonitis due to duodenal ulcer perforation.

C. To see **response of patients after starting antibiotics according to the culture and sensitivity report** in terms postoperative complications like

septicemia, surgical site infection , cost effectiveness of antibiotics and hospital stay.

REVIEW OF LITERATURE:

- ✓ The knowledge of perforation dates back to over 2000 years remote past when *"***Sushrutha***"*, the great Indian surgeon described it as *"Parinamashula"* giving the relation of the food, pain and vomiting.

- ✓ The history of peptic ulcer dates back to 1500 B.C. when peptic ulcer and hemorrhage was noted from **Egyptian Papyri**.

- ✓ The acute pathological condition of abdomen "Hippocratic Facies" which represents the terminal stage of peritonitis was recognized by **Hippocrates.**

- ✓ The symptoms that are caused by peptic ulcer were for the first time described by **Diokles** (350-325 B.C).

- ✓ The first detailed mention of gastric ulcer is done by Italian doctor **Marcello Donati** in the year 1586 and the first patient of perforated gastric ulcer was noted and reported by **Christopher Rawlinson** in

England 1727.

- ✓ DU was 1st mentioned by **Georg Hamberger** in Germany during 1746, and **Jacopo Penada** from Italy, a perforation of duodenal ulcer in 1793 was reported.

- ✓ During 1881, **Theodor Billroth**, Father of Surgical Audit and Father of Abdominal surgery, performed the excision of distal part of the stomach with an anastomoses of the gastric stump to the duodenum (Billroth I Surgery).

- ✓ **Mikulicz** was first to suture a perforated gastric ulcer in 1885.

- ✓ **Bennett** suggested sealing a large perforation with omentum in 1896.

- ✓ **Keetley** of London in 1902 did the first partial gastrectomy for a perforated ulcer.

- ✓ In 1938 **Graham** popularized the simple closure of perforated ulcer.

- ✓ **Wangensteen** in I935 first advocated non operative treatment for duodenal perforation.

- ✓ In 1945, **Taylor** reported a series of 28 consecutive cases of perforated peptic ulcers. Of those, 24 were treated non operatively with intermittent gastric suction with three deaths.

- ✓ **Mathur S.N., Khandelwal R.** (1991): In a study of 43 cases of perforated peptic is a safe procedure in all ulcer patients. Definitive ulcer healing operation may be done in selected cases of perforated chronic duodenal ulcers.

- ✓ **Siu WT et al.** (2004) mentioned and done repair of perforated peptic ulcer by laparoscopic technique as a safe emergency procedure in day today practice for patients with perforated ulcer in pyloro duodenal region

EMBRYOLOGY OF STOMACH AND DUODENUM:

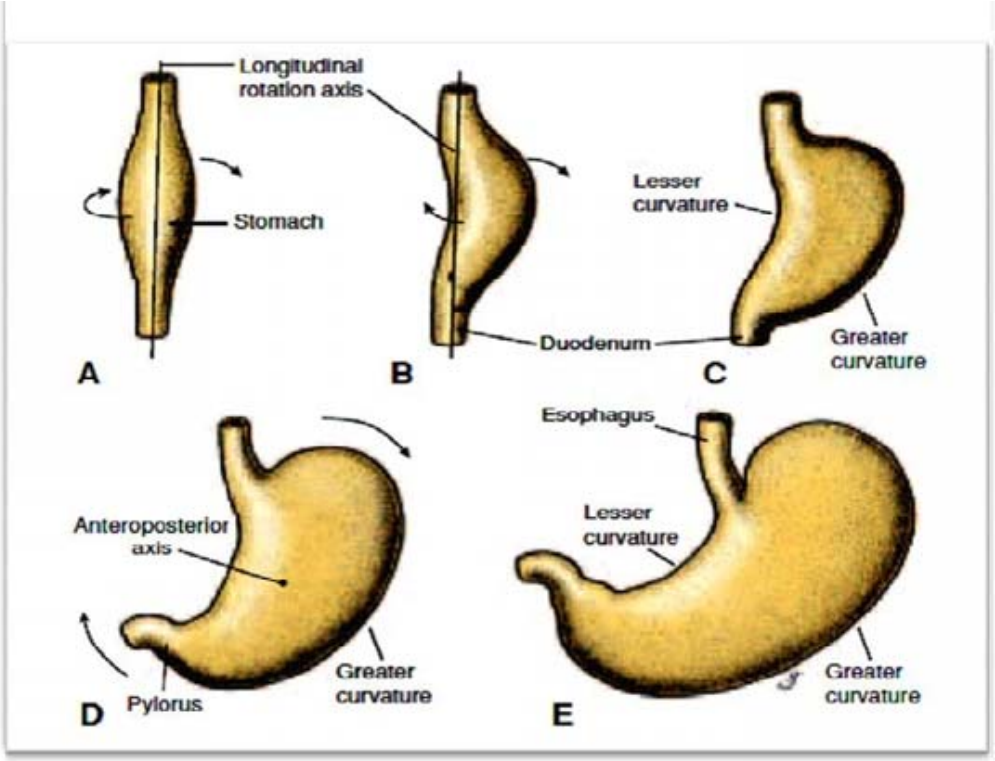
The development of Stomach:

The stomach is formed as a fusiform dilation of the foregut in the 4th week of development. During the following weeks, its morphology and position change greatly as a result of the different rates of development in various regions of its wall and its changes in position of surrounding organs. The stomach has a

rotation 90◦ clockwise around its longitudinal axis, resulting its left side to face anteriorly and its right side to turn posteriorly . During its rotation, original posterior wall of the stomach grows in increased pace than the anterior portion, forming the **greater** and **lesser curvatures**. The cephalic and caudal ends of stomach lie in the midline, but during further growth stomach rotates around an A-P axis, such that the caudal or **pyloric part** turns to the right and upward and the cephalic or **cardiac portion** turns to the left and downward . The stomach thus takes its final position, then its axis running from above left to below right.

Development of Duodenum:

Terminal part of the foregut and cephalic part of the midgut form the duodenum. The junction of both parts is directly distal to the origin of liver bud. As the stomach rotates, the duodenum takes a form of a C-shaped loop and rotates to the right. This rotation, along with rapid growth of the head of the pancreas, duodenum swings from its initial midline position to the left side of abdominal cavity. Since the **foregut** has blood supply by the **celiac artery** and the midgut is supplied by **superior mesenteric artery,** the duodenum has its blood supply by branches of both arteries.



A, B, and C. Rotation of the stomach along its longitudinal axis as seen anteriorly. D and E. Rotation of the stomach around the anteroposterior axis

ANATOMY OF STOMACH:

The stomach is a pouch communicating the abdominal esophagus and the first part of the duodenum. The stomach is a most dilated part of the digestive tube and is J- shaped. The stomach is capable of great dilatation and may shrink into tubular form when empty. The experts in anatomy describes the stomach in terms of several parts, such as the gastroesophageal junction, cardia, fundus, body, antral region, pyloric canal, and sphincter, but also accepts it as a distinct entity, a well-defined organ which is easily visualized, dissected, and demonstrated.

From the surgeons point of view, the stomach is part of two different organ

systems, each with its special pathology and surgical approach. The first part can be called the proximal gastric unit, and contains the proximal stomach, distal esophagus, and the esophageal hiatus of the diaphragm. The second is the distal gastric unit, and contains the gastric antrum and pylorus, together with the first part of the duodenum.

PARTS OF STOMACH:

The stomach contains two orifices, two curvatures, two surfaces and two sphincters.

A. Gastric orifices:

Cardiac orifice starts from junction of the oesophagus and the stomach. It is about 40 cm from the incisor teeth. The pyloric orifice is the communication between the stomach and duodenum, usually noted by a circular pyloric constriction on the surface of the stomach, indicating the pyloric sphincter. It can be identified by the pre-pyloric vein of mayo, which crosses the anterior surface vertically.

B. Gastric curvatures:

Lesser curvature is a concave structure and the right (posterosuperior) border of stomach. The lesser omentum is attached to the lesser curvature. It has the right and left gastric vessels.

Greater curvature is a convex structure and the left border of the stomach. It provides attachment to the greater omentum, have two layers which

are separated by gastroepiploic vessels.

C. Gastric surfaces:

The surfaces of stomach when empty and contracted are almost superior and inferior. With distension, they become anterior and posterior respectively. Hence stomach is described as anterosuperior and posteroinferior surfaces.

D. Sphincters:

The sphincter located in pylorus is a muscular ring formed by marked thickening of the circular layer of the muscular coat. Cardiac sphincter is sometimes described as the esophageal end of the stomach, formed from circular fibers of the gastric wall. The stomach is subdivided into fundus, body and pyloric portion.

• Fundus- the rounded upper part of the stomach, which is situated above the level of the oesophagus junction.

• The body- it is that part which lies between the cardiac notch and the incissura angularis.

• The pylorus- The pylorus is formed between junction of the stomach and the duodenum.

GASTRIC WALL STRUCTURE:

The wall of the stomach is formed by serous, muscular, submucous and mucous layers.

A. Serosa or visceral peritoneum covers the entire surface except at a) along line of attachment of greater and lesser omentum, b) small area at posteroinferior surface, close to cardiac orifice.

B. Muscularis layer is present beneath the serous layer consists of three layers of muscle fibers, the longitudinal, the circular and oblique. The muscle layers of the stomach have its role in gastric motility.

C. Submucous layer have loose areolar tissue and blood vessels, between the mucous and muscle layer. So it has its name and called as "vascular layer" (arterial and venous) of the wall the stomach.

D. Mucosa consists of thick soft, velvety layer with smooth surface. During the contracted state it turns into numerous folds or rugae, which for the most part have longitudinal in direction and best marked towards pyloric end and in the greater curvature. Mucosa gets obliterated when the stomach is distended. It is formed by epithelium, lamina propria and muscularis mucosae.

The epithelial layer of mucosal surface has irregular gastric pits (fovelae), polygonal or slit like funnel shaped depressions, base of each pit has long tubular gastric glands.

Gastric glands- Comprises of cardiac glands, main/principle glands and pyloric glands.

1. The glands in cardiac are limited in a small area near the cardiac orifice. Mucous secreting cells are many while parietal and zymogenic cells are few.
2. The principal gastric glands are found in the body and fundus of the stomach.

Five distinct cell types are present.

a. The chief (peptic or zymogenic) cells- present at the basal part of the gland.

b. The parietal (oxyntic) cells- present abundantly on the sidewalls and near the duct of the gland.

c. Mucous (Neck) cells- these are disseminated between other types of cells and are numerous around neck region of the gland.

d. The stem cells are present in the isthmus region of the gland and bases of gastric pits.

e. Enteroendocrine cells- these are situated mainly in the deeper parts of gland. They include G-cells secreting gastrin, D cells (Somatostatin) and EC cells.

3. Pyloric glands secrete the enteric hormone gastrin.

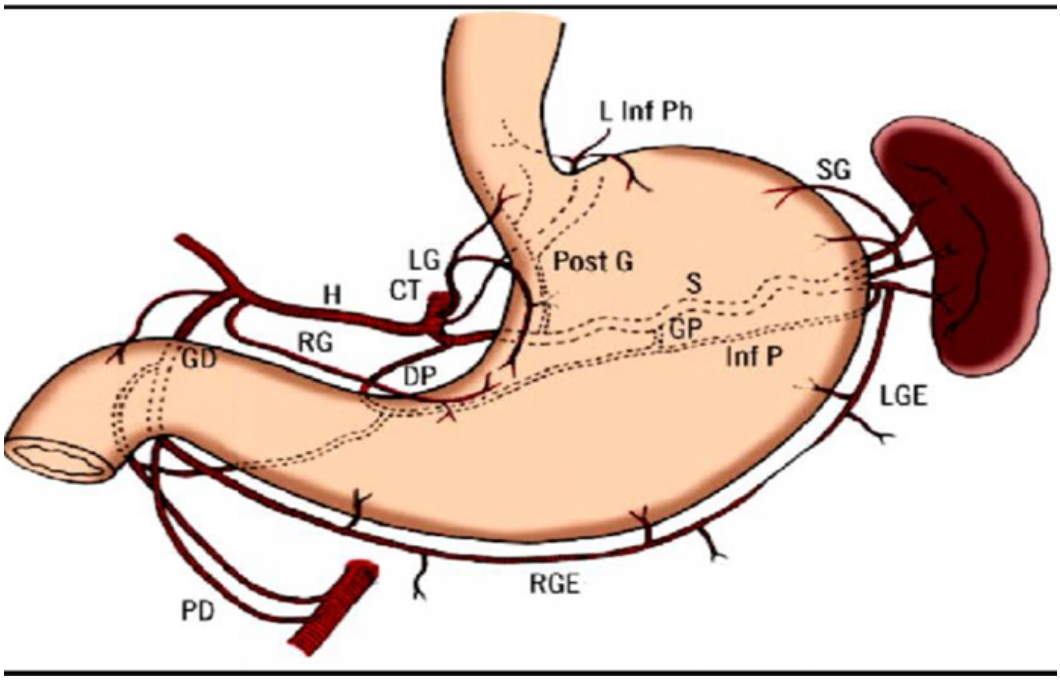
The Exocrine and Endocrine Cells of the Stomach and Their Secretory Products

	Cells	Secretory Products
Exocrine	Mucous	Mucus
	Oxyntic	Acid
	Chief	Pepsin
Endocrine	G	Gastrin
	D	Somatostatin
	A*	Glucagon
	EC	Serotonin plus various peptides
	ECL	Unknown
	P	Unknown
	X	Unknown

VASCULAR SUPPLY OF STOMACH:

Arterial supply- majority of blood supply to the stomach is by the celiac artery. The four main arteries: the right and the left gastric arteries in the lesser curvature and the right and left gastro epiploic arteries along the greater curvature. Celiac artery branches into left gastric artery, hepatic artery and splenic artery. The right gastric artery has its origin from the hepatic artery. The left gastro epiploic artery has its origin from the splenic artery and the right gastro epiploic has its origin from the gastro duodenal artery.

Venous drainage- Veins run parallel to arteries accordingly and drain to hepatic portal system. Right and left gastric veins drain into portal vein. Left gastro epiploic veins and short gastric veins drain into the splenic vein. Right gastroepiploic vein drains into superior mesenteric vein.



LYMPHATICS- The lymphatic vessels of the stomach arise in its submucous and subperitoneal layers, and divide into four main sets.

- Zone I (inferior gastric) drains to subpyloric and omental nodes
- Zone II (splenic) drains to pancreaticosplenic nodes
- Zone III (superior gastric) drains to the superior gastric nodes
- Zone IV (hepatic) drains to suprapyloric nodes

NERVE SUPPLY OF STOMACH:

Extrinsic nerve supply of the stomach-

Parasympathetic componant is by vagus nerve.

Sympathetic supply is by the greater splanchnic nerves

(T5-T9) and celiac ganglia.

The intrinsic or enteric nervous system of the stomach contains neurons in Meissner's and Auerbach's autonomic plexuses.

ANATOMY OF DUODENUM:

The duodenum is a C-shaped, first and shortest (about 10 inches) part forms the most fixed part of small intestine. It extends from the pylorus to the duodenojejunal junction, forming C-shaped curve, which is occupied by the head of pancreas.

Parts of duodenum- the duodenum is divided into four parts.

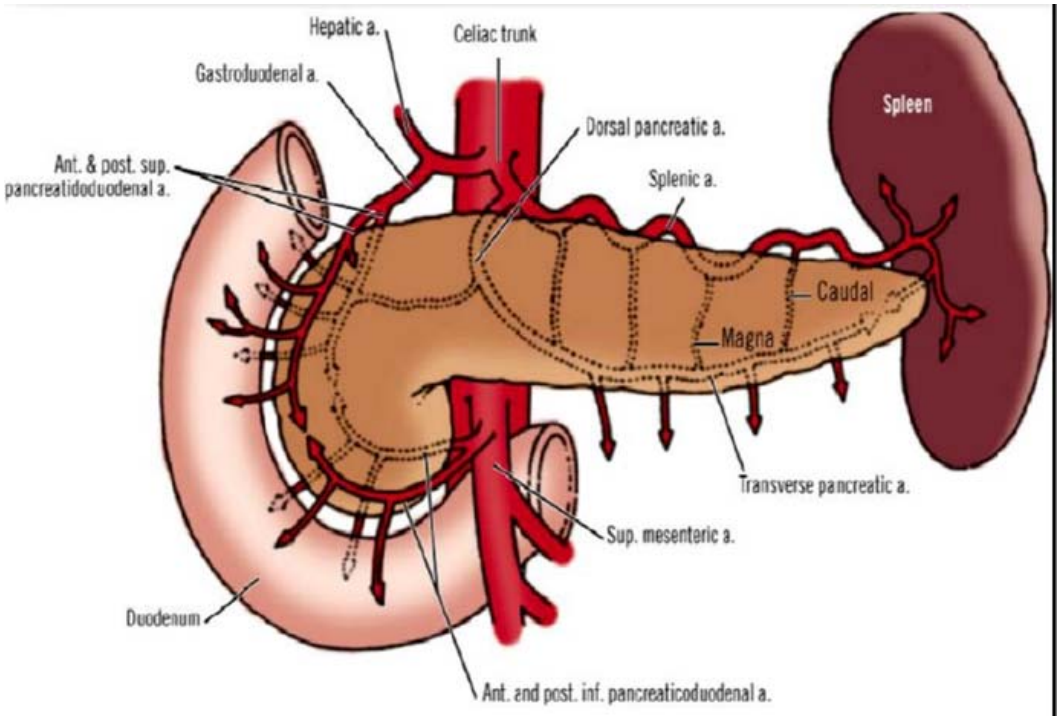
1. 1st/Superior part (2 inches) - First inch is covered with peritoneum and can be moved with stomach. Second inch is covered with peritoneum only above and in front.

2. 2nd/Descending part (3 inches) - The pancreatic duct and bile duct unite to form a short dilated tube called hepatopancreatic ampulla, which opens into the major duodenal papilla. The accessory pancreatic duct if present gets open 2 cms proximal to major duodenal papilla as minor duodenal papilla.

3. 3rd/Horizontal part (3 inches) - It runs horizontally and is crossed by the root of the mesentery.

4. 4th/Ascending part (2 inches) - The duodenojejunal flexure is usually retroperitoneal. It is fixed and held in position by Ligament of Treitz.

Blood supply- The superior half is supplied by superior pancreatoduodenal artery which takes banch from gastroduodenal artery. The inferio half is supplied by inferior pancreatoduodenal artery arises from superior mesenteric artery. Veins drain to splenic, superior mesenteric and portal veins.



Lymphatics drain upward via pancreatoduodenal nodes to gastroduodenal nodes and to coeliac nodes and downward via pancreatoduodenal nodes to superior mesenteric nodes.

Nerve supply is from sympathetic and parasympathetic (vagus) which is from celiac and the superior mesenteric plexus.

Anatomy of Peritoneum and Peritoneal Cavity:

The anatomic relationship within the abdomen plays significant role in identifying possible source and routes of spread of infection. The peritoneal cavity has its boundaries from the inferior surface of the diaphragm to floor of the pelvis. The peritoneal cavity appears like a closed space in males. In

counterparts, cavity is perforated by the distal aspects of the fallopian tubes. The anterior aspect of peritoneum reflects onto posterior aspect of the anterior abdominal muscle. This lining in posterior aspect lies superficial to retro peritoneal viscera, including the inferior venacava, aorta, ureters and kidneys. The anterior and posterior layers, collectively called as the parietal peritoneum. The visceral peritoneum contains the mesothelial cells that are reflected over the visceral surfaces.

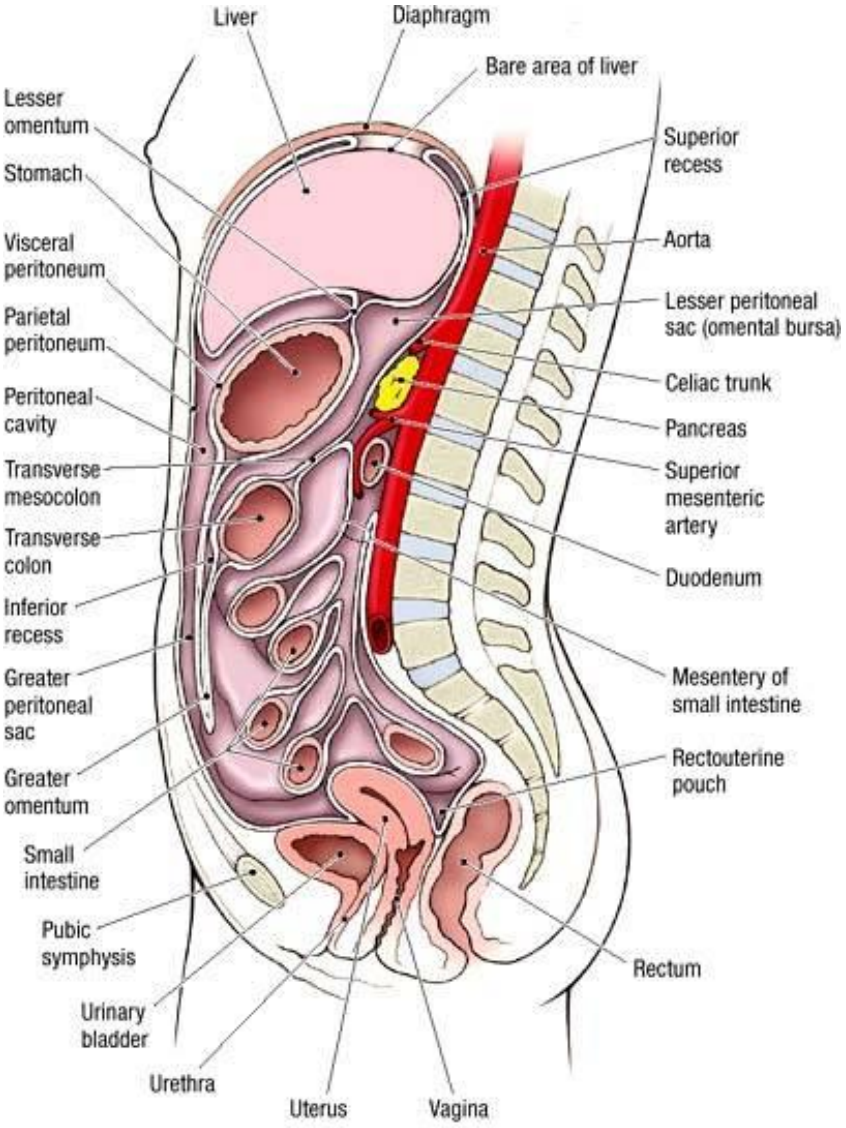
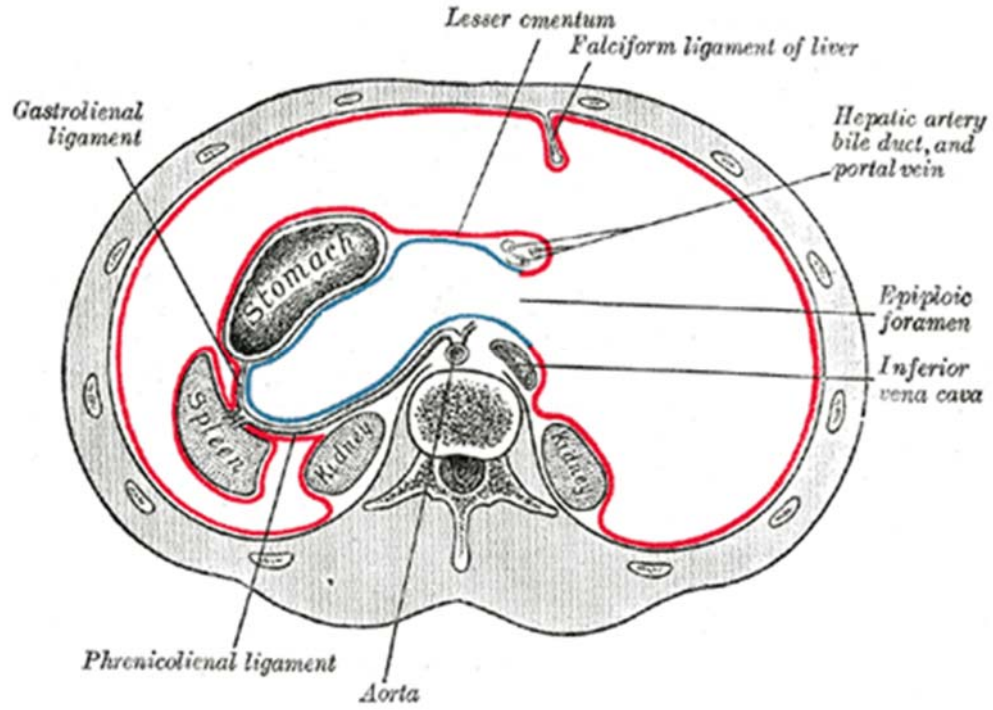


Figure 4.20. Peritoneum and peritoneal cavity, median section.



PERITONEUM AND ITS REFLECTIONS- CORONAL VIEW

The transverse mesocolon transects the peritoneal cavity horizontally into an upper and a lower space. The greater omentum, extending from the The peritoneal reflections and the mesenteric attachments compartmentalize the intraperitoneal space and route, spreading exudates to sites that are often distant from transverse mesocolon and inferior aspect of gastric region.

The peritoneal cavity has many recesses in which exudates may become loculated. The pelvis is the most dependant site in standing posture. Between the rectum and bladder in men is a pouch of peritoneal cavity that extends slightly below the level of the seminal vesicles.

In women, the uterus and fallopian tubes project into the pelvic recess. The pouch of douglas is located between rectum and uterus. The pararectal and paravesical fossa is located on lateral side of bladder and rectal wall. The pelvic recess communicates with both the paracolic gutters. The phrenico colic ligament, attaches the diaphragm and splenic flexure of colon, bridges the junction between the left paracolic gutter and perihepatic space. In contrast, right paracolic gutter communicates with the right sub-hepatic space. A posterior end of the right subhepatic sac, Morrison's pouch, is the site of maximum dependent portion of the supine position of the right paravertebral groove and lies just above the starting of the transverse mesocolon. The horizontal posterior reflection of the serosal surface of the liver onto the diaphragm, (the triangular and coronary ligaments), and the vertical reflection (falciform ligaments)divide the right perihepatic space, into right subphrenic

and subhepatic spaces. The left subphrenic and subhepatic spaces communicates round the smaller left lobe of liver, and it is more superiorly placed left triangular ligament. The falciform ligament lies between right and left subphrenic spaces , which probably prevents pus spread to the opposite side and explains why only about <15% chances of subphrenic abscess being bilateral. The left subhepatic space is divided by the gastro-hepatic omentum into an anterior space and the lesser sac. Abscesses within the perihepatic spaces become localised by the presence of pyogenic membranes.

Abscesses of the left perihepatic space are either in the single left subphrenic space. The lesser sac being the largest recess of all recess of the peritoneal cavity, is well connected to the peritoneal cavity through foramen of Winslow, an opening situated between the edges of the gastro-hepatic omentum and the parietal peritoneum in posterior aspect. The lesser sac is surrounded posteriorly by the pancreas and kidneys, anteriorly by the stomach and laterally by the liver and spleen.

It may also extend to variable extent between the folds of the greater omentum. Due to the limited scope of communication from the lesser sac to major cavity via foramen of Winslow, suppuration in this sac lie between stomach and pancreas but may even spread to the right and lie in front of kidney and below the hepatic region. After intraperitoneal injection of water soluble contrast material, selectively into various intraperitoneal spaces, Autio demonstrated that right paracolic gutter as the main communication between the

upper with lower peritoneal cavities. Fluid introduced up into the right upper peritoneal space usually gravitates towards Morrison's pouch and then into the right subphrenic space and along the right paracolic gutter into the pelvic recess. Flow of the fluid in the left upper peritoneal space always is mainly into the left subphrenic space.

The phrenicocolic ligament limits flow inferiorly to the left paracolic gutter. Fluid introduced up into the lowerperitoneal cavity first gravitates up into pelvic recess and then ascends up, whether in supine or erect position, along the right paracolic gutter into the subhepatic space, especially into Morrison's pouch and into the right subphrenic space. Ascension of fluid from the pelvic space along the left paracolic gutter usually remains minimal and is limited by phrenicocolic ligament. Although gravity would account for the pooling of fluid in the dependent peritoneal recesses, such as the pelvic recess, ascension of fluid from the pelvis to the subphrenic space is probably caused by hydrostatic pressure differences between the upper and lower peritoneal cavities created by diaphragmatic motion.

Normal intestinal and abdominal wall motion would also account for some spread of intraperitoneal fluid. The anterior retro-peritoneal space between the peritoneum and anterior renal fascia contain the ascending and descending colons, duodenum and pancreas. The kidneys and ureters lie within the posterior retro peritoneal (perinephric) space. The renal fascia encloses the kidneys and adrenal superiority and laterally, but not inferiorly,favouring spread

of infection in this space inferior.

Innervation to the peritoneum is much helpful in clinical recognition of peritonitis. The nerve supply of visceral peritoneum is poor. Therefore, it's stretch produces nausea and poorly localized pain. Pain is usually localized to that dermatome distribution of the associated visceral organ. Eg. Irritation of the diaphragmatic peritoneum is felt as pain near the adjacent wall, and irritation of the central portion is felt as a pain referred to shoulder or neck.

The nerve supply of the parietal peritoneum on the other hand is from the somatic afferents, arising from branches of cutaneous nerves of the abdominal wall. The parietal peritoneum is sensitive to stretch and light touch and cutting, especially anterior, pain thus produced is precisely localized by patients and forms the basis for the clinical picture of "peritoneal sign".

Microscopic anatomy

The peritoneal cavity is lined by a serous membrane. The surface area of this membrane approximate that of skin i.e. 1.8 m^2. It is semipermeable membrane .Each of these layers comprises of a layer of flat mesothelial cells, a basement membrane with a layer of loose vascular connective tissue containing collagen bundles, lymphatics and macrophages. The cells contain microvilli 1.5 to 3.00 mm of length, that can greatly improve the surface of the

peritoneum covers the abdominal surface of the diaphragm, the basement membrane disappears and large intercellular gaps stomata become obvious. These measure from to 500 angstroms in diameter which vary according to the contraction of diaphragm.

PHYSIOLOGY:

The various functions of stomach are

1. It begins the process of food breakdown exposing solid meal to proteolytic action of acid and pepsin.

2. It grinds and dilutes the mixture to form a more uniform consistent chyme.

3. The stomach works like storage organ where food particles stays for four hours. The function of stomach is under the control of neurological and hormonal mechanism. Sometimes both mechanisms have interaction to regulate the gastric function. Through hormonal mechanisms, substances like peptides or amines mediates the function stomach by interacting with target cells through 3 mechanisms namely neurocrine, paracrine and endocrine. **Endocrine cell** exerts hormonal function by secreting peptides that reach the target cells and exert their function. Peptides that are released by paracrine cell, by diffusion method through the interstitial space reaches the target cells and neural cells mediates through nerve endings that by diffusion method reaches the synapse of target cells and attaches to the receptors.

Gastric Peptides:

1. G-CELLS produce GASTRIN. G-cells are situated in antral region of stomach. Its secretions are regulated by nature food particals. It exerts negative feedback via acid content in the lumen of stomach. Its also has negative feedback through paracrine hormonal mechanism via somatostatin that acts on G cells present in the antral region.

2. D-CELLS produce Somatostatin. Direct inhibition of acid secreting function of parietal cell is exerted by somatostatin. Also there is indirect inhibition of acid secreting function is exerted by somatostatin inhibiting the gastrin secretion and by down regulating (ECL) "ENTEROCHROMAFFIN LIKE CELLS" is produced by D cells. Acid content in the antral region is the prime stimulating factor in somatostatin secretion. ACETYLCHOLINE which is secreted by vagal stimulation has negative feedback in somatostatin secretion.

3. "Gastrin-Releasing Peptide (GRP)" is rich in nerve endings present is the parts of the stomach which are involved in secretion of acid and in secretion of gastrin. Gastrin-Releasing Peptide have positive feedback in secretion of gastrin and somatostatin by attaching to G and D CELLS respectively.

4. To stimulate histamine parietal cells play an important role. Acetylcholine, Gastrin, and Epinephrine following receptor ligand actions have positive feedback in histamine secretion. Somatostatin has negative feedback over gastrin stimulated histamine secretion by interacting with receptors for

somatostatin present in enterochromaffin like cells. Also it has significant action that takes place in stimulating the positive and negative feedback cascades that regulate acid and histamine secretion.

Other Gastric Secretory Products-

1. The enzymes collectively secreted by parietal , chief and mucous cells is gastric juice .

2. Glands of gastric and duodenal region secrete pepsinogen which acts by breaking down the proteins in the food particles.

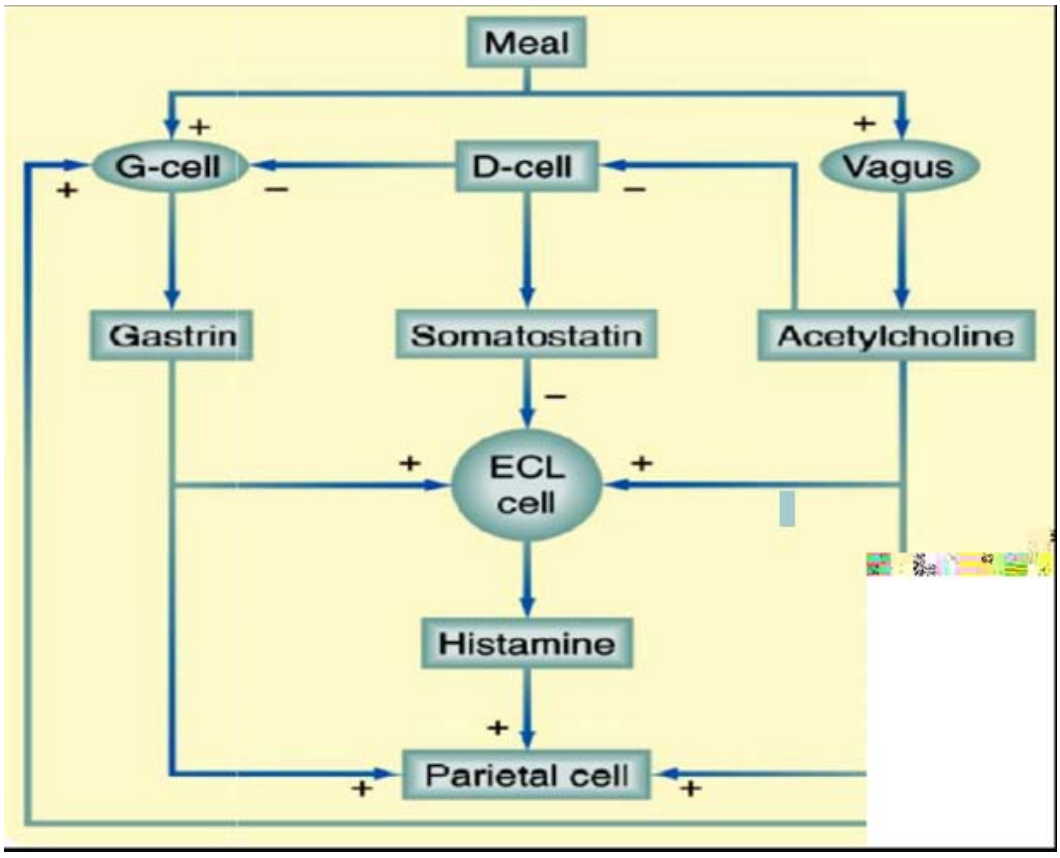
3. Parietal cells in terminal ileum release Intrinsic factor which plays important function in absorbing Vit B12. Pepsinogen - proteolytic proenzymes that are secreted by the glands of the gastroduodenal mucosa.

4. Mucous and HCO3 neutralize acid content in the mucosal surface in gastric surface.

GASTRIC AND DUODENAL ENDOCRINE SECRETIONS:

1. **From the Stomach:** Gastrin is secreted by the 'G' cells of the antrum. Gastrin secretion is inhibited by fall of gastric pH below 3 and by somatostatin.

2. **From the Duodenum:** secretin, Cholecystokinin-pancreozymin (CCK-PZ), enteroglucagon and enterogastrone hormones.



Physiology of Peritoneum

The mesothelial cells lining peritoneum secrete serous fluid into abdominal cavity, contains approximately 100 ml of fluid that contain less than 300 cells / mm3. 40% - macrophages, 50% - leukocytes, 10% - eosinophils and mast cells, protein < 3 g / dl. Function of the peritoneal fluid seem to be lubrication to facilitate peristalsis. It also plays role in defense mechanisms. The initial bactericidal property of peritoneal fluid is due to activation of complement cascade.

The peritoneal membrane is highly permeable. Bi-directional flow of substances between this membrane is rapid due to the large surface area involved potentially large in quantity. This unique property is utilized in

peritoneal dialysis in the treatment of uremia and has also been used for the administration of fluid, electrolytes, antibiotics and even blood. The hydrostatic pressure and the most effective serum oncotic pressure in the portal veins and lymphatics are major factors determing the rate and the direction of fluid movement. The rate of flow of water and the solutes between blood and peritoneal fluid also depends on concentration gradients between these compartments. Water and solute diffuse via blood capillaries and to a lesser extent through the lymphatics. Lymphatics are primarily involved in removal of non-irritating colloids and particles into the blood stream.

Absorption into lymphatics of particulate matter is thought to take place mostly from the diaphragmatic surface and is aided by the pumping action of diaphragmatic motion. After infusion of 5 Cr labeled RBCs into the peritoneal cavity of dogs, Rochlin and Associates reported absorption of about 70% of the labeled cells by 48 to 96 hr. This absorption occurred mostly through the lymphatics.

In human, two thirds of intraperitoneally injected RBC's in anticoagulated blood have been found in the circulation 8 to 12 days after infusion. The quantity of reabsorbed cells was less when no anticoagulant was used, presumably because of trapping of red blood cells intraperitoneal clots. Transport of other particulate matter, such as intraperitoneal bacteria, may be similarly impeded because of trapping in fibrinous intraperitoneal exudates. In addition, there are communications between the peritoneal and pleural cavity

that are independent of the blood stream. Probably as a result of trans diaphragmatic lymphatic transport like in Meig's syndrome.

PEPTIC ULCER DISEASE

Ulcers in Peptic ulcer diseases are local defects in the gastric or duodenal mucosal lining that involves the submucosal layer or even deeper. It is classified as acute ulcer or chronic .They are caused by an imbalance between the mucosal protective mechanisms and acid injury. Gastric ulcers have a high incidence of mortality than DU because of its more incidence in old aged people. Current study groups have reported increased hospitalization and deaths in old aged people. It complicates bleeding and perforation. This is due to NSAIDs abuse especially in old aged people who often involves in such drug abuse and also with increased risk of acquiring H.pylori infection.

Epidemiology:

Peptic ulcers diseases are remitting, relapsing lesions that are most frequently found in middle-aged to older adults, but they are first evident in young adult life. Male: female ratio for duodenal ulcer is about 3:1 and for gastric ulcers around 1.5 to 2:1.

Duodenal ulcers are more often seen in persons with alcoholic cirrhosis, chronic obstructive pulmonary disease, chronic kidney disease, and hyperparathyroidism. With respect to the last two conditions, hypercalcemia,

whatever its cause, stimulates gastrin production and in turn the acid secretion.

Pathophysiology

The pathogenesis of peptic ulceration is multifactorial, but increasingly understood to be a consequence of *H. pylori* infection. Before the recognition of the role of *H. pylori*, ulcer disease was conceived as an imbalance between acid and pepsin secretion and mucosal defense, with the balance shifted toward peptic injury and disease.

A. HELICOBACTER PYLORI INFECTION:

H.pylori is flagellated and urease rich. It has virulence factors and other protective mechanisms that prevent its destruction in the gastric acidic medium. It produces urease enzyme, that turns urea to ammonia and hco3 and produces buffering environment surrounding it and escapes the acidic medium of gastric content. Surface mucosal epithelium gets affected by ammonia produced by urease enyzyme helps the organisms to get resided in the cells of gastric wall.

The proposed mechanisms behind the gastroduodenal injury caused by helicobacter pylori are as follows:

1. It induces local immunity process in the mucosal level.
2. It produces harmful substances that induce and damage the mucosal tissue locally.
3. Further damage is caused by stimulating gastrin secretion that in turn increases the acid secretion which further cause tissue damage

H.pylori infection produces focal environment of alkali nature in the antral region (antral acidity is the important virulent antagonist to antral gastrin secretion). *H. pylori* also cause various cascades of events that ultimately release local mediators of inflammation and cytokines. Ultimately it causes hypergastrinemia and hyperacidity. The increased secretion of gastrin results in parietal cell hyperplasia as encountered in many cases with duodenal ulcer. The acid hypersecretion and the antral gastritis are presumed to result in antral epithelial metaplasia in the first part of duodenum and these duodenal metaplasia allows *H.pylori* get resided in the duodenal mucosal layer and the risk of developing a duodenal ulcer increases 50-fold.

VAC A and CAG A toxins produced by H.pylori incites injury to gastric and duodenal mucosal layers and various other cascades of inflammatory events that releases cytokines like interleukin 8 by infected mucosa, recruitment of inflammatory cells, recruitment and activation of local immune factors, and increased programmed cell death. The final result is a weakening of mucosal defenses. *H. pylori* has important role as an etiologic factor in gastric cancer and lymphoma.

B. ACID SECRETION AND PEPTIC ULCER:

The formation of duodenal ulcers clearly depends on gastric secretion of acid and pepsin. This association is symbolized by the dictum "no acid-no ulcer." *H. pylori* infection is now known to secondarily induce alterations in gastric acid secretion, and a more complete and accurate statement might be "no acid and no

H. pylori-no ulcer."

The increase in acid-secretory capacity in patients with duodenal ulceration has been postulated to be secondary to increased parietal cell mass in the acid- secreting gastric mucosa. Many of the secretory abnormalities are a long-term consequence of *H. pylori* infection.

C.MUCOSAL DEFENCE:

Most cytoprotective agents act via mucosally secreted bicarbonate or on mucosal prostaglandin production. Gastric and duodenal epithelial cells secrete mucus and bicarbonate, creating a pH gradient within the mucus layer. Abnormalities in local bicarbonate secretion could result in exposure of surface epithelial cells to the peptic activity of gastric secretions at low pH. Prostaglandins and synthetic prostaglandin analogues exhibit cytoprotective effects, accelerate ulcer healing, and decrease acid secretion. Decreased mucosal prostaglandin production has also been proposed to contribute to the development of duodenal ulcer.

D. NONSTEROIDAL ANTI-INFLAMMATORY DRUGS:

There is a strong link between NSAIDs drug intake and peptic ulcer disease. Mainly in patients with rheumatoid arthritis and in OA patients who often uses aspirin, there is a risk of annual incidence 10 to 20 percent to develop peptic ulcer disease and its 25 percent in chronic NSAID users to develop peptic ulcer disease. Complications of PUD are

hemorrhage and perforation, more common in patients taking NSAIDs. All patient who are using NSAIDs or aspirin who has one or more of following risk factors should receive acid suppressive drugs

- Age > 60
- H/of peptic disease
- Concurrent use of steroid
- Concurrent anticoagulant intake
- High-dose NSAID or acetylsalicylic acid .

E.SMOKING, STRESS AND OTHER FACTORS:

Various study groups reveal that smokers are twice more prone to develop peptic ulcer disease when compared to non smokers. This is because of hyperacidity and increased duodenogastric reflux. Also reduced secretion of prostaglandin secretion and HCO3 production results in PUD. Both physiological and psychological stresses undoubtedly play a role in the development of peptic ulcer in some patients. During 19th century, scientist called Curling demonstrated duodenitis and ulcer in duodenum in patients met with burns injury. After 10 years again he demonstrated the morphologic picture of ulcer in acute cases of peptic ulcer disease and coined the term "cushing's ulcer". Especially those who are cocaine abuse have peptic ulcerations in juxta-pyloric region which is more prone to get perforate.

Alcoholism is linked to the genesis of peptic ulcer disease inspite of less support in the literature. Irrespective of treatment, peptic ulcer disease takes one of the courses during the period of its progress:-

- Healing
- Chronicity
- Complications.

The **complications** of peptic ulcer are:

1. Perforation
2. Haemorrhage
3. Cicatrical contraction
4. Carcinomatous changes.

PERFORATED PEPTIC ULCER:

"Perforation" is the natural termination of an ulcer, which continues to penetrate into the deeper tissues. Perforations of duodenal ulcer greatly outnumber gastric ulcer.

INCIDENCE AND EPIDEMIOLOGY:

In general, the incidences of emergency surgery, hospital admission, and mortality for perforated peptic ulcer have remained stable through last two decades. In older patients, admission rates for duodenal ulcer perforation increased and gastric ulcer perforation decreased in the last decade. Duodenal perforation currently accounts for approximately 75% of peptic perforation. In

a recent study, a postoperative mortality rate of 19% in perforated peptic ulcer patients increased to 41% among the elderly.

In past days among middle aged persons male female ratio is 2:1. With advent in advanced imaging technique and broad spectrum antibiotics there is increased incidence among old aged patients and there is an increased trend in poor outcome due to severe complications as a result there is decresed immunity in elderly patients. Factors such as *H. pylori* infection, NSAID use, smoking and alcohol consumption are associated with perforated peptic ulcer. Factors such as associated diseases, shock during the time admission, delayed surgery (>24 hours), resectional surgery, and postoperative abdominal and wound infections have been associated with the increased morbidity and mortality in perforated ulcer patients.

For decades, delay in operative treatment has remained a primary determinant of morbidity, mortality, and cost. A study suggests that a positive peritoneal culture positive for fungal organism is common and seems to be a risk factor for poor outcome in patients with a perforated peptic ulcer.

The bacteriological factors are:

Normal bowel flora

Even though anaerobic species make up for majority of normal colonic flora, they contribute very little to clinical intra-abdominal infections. The most commonly bacteria encountered are E.Coli, Klebsiella, Enterobacter and Pseudomonas species.

Level of Perforation:

The prognosis of intra-abdominal sepsis seems varying with the levels of perforation, due to the number and type of micro organisms vary throughout GIT.

Stomach - < 10^3 bacteria / mm^2

Proximal small bowel - 10^4 to 10^5 bacterial /cu.mm

Terminal part of ileum - greater than 10^9 bacterial / cu.mm

Colon - 10^{10} to 10^{12} bacterial / mm^3

The type of bacteria also changes. The upper GIT has facultative gram -ve aerobic bacteria.

Virulence factors:

Virulence factors like coagulase and catalase of Staphylococcus aureus polysaccharide capsule of B. fragillis, enhances virulence by impairing polysaccharide capsule of B. fragillis, enhances virulence by impairing opsonization or phagocytosis leading to increased complications like abscesses.

Microbial adherence:

Rapid adherence of some bacteria to peritoneum soon after perforation and their resistance to removal by peritoneal lavage enhances their virulence. Organisms like B. fragillis are strongly adherent to peritoneum and is usually unaffected by extended lavage. So, infections caused by these organisms may not be amenable to lavage and other forms of treatment, after 24 hours when

they become strongly adherent to peritoneum.

Synergism:

Synergestic interactions between anaerobes and endotoxic gram -ve organisms suppress local defence mechanism and facilitate the establishment of severe infection. Aerobic bacteria lower the oxidation reduction potential, favoring the growth of several species of anaerobic bacteria.

PATHOLOGICAL COURSE:

At the onset of perforation there is sudden spillage of gastric or duodenal contents into the general peritoneal cavity and results in chemical peritonitis. Perforation of peptic ulcer may be classified as follows:

1. Acute perforation
2. Subacute perforation
3. Chronic perforation
4. Perforation associated with haemorrhage
5. Perforation of intrathoracic gastric ulceration
6. Pseudoperforation

1. Acute perforation: The ulcer perforates and the general peritoneal cavity becomes flooded with gastric and duodenal contents, causing chemical peritonitis. The clinical features vary according to the stage of perforation. The clinical course can be divided into three stages, each of variable duration.

a. Primary stage or the stage of peritonism: The patient feels acute agonizing pain in the epigastrium or right hypochondrium, which usually becomes generalized. The symptoms are due to the intense irritation of peritoneum by the gastric and duodenal contents. Pulse rate is normal or raised. Respiration is shallow with increased respiration rate. On inspection, the abdomen will be seen to be immobile with no movement with respiration; the muscles are rigid and board like. On auscultation bowel sounds are absent. This stage lasts for 3- 6 hours.

b. Secondary stage or the stage of peritoneal reaction: In this stage the spontaneous sealing of perforation may occur. If there is gross leakage of gastric contents, the patient may pass onto the stage of septic peritonitis. The length of this stage rarely exceeds 6 hours. During this stage the pain is lessened markedly. There would be general improvement in the patients' condition. For this reason this stage of reaction has sometimes been called stage of delusion.

c. Tertiary stage or the stage of bacterial peritonitis: This is the stage of diffuse peritonitis, begins about 12 hours after perforation and lasts for about 24 hours until it passes on to the final stage of paralytic intestinal obstruction. Pathogenic organism multiplies rapidly. Peritoneal fluid becomes more purulent. Intestinal movements diminish and finally disappear with the onset of

paralytic ileus. The patient drifts into toxemia, dehydration and circulatory failure. Death usually takes place 4-5 days after perforation.

2. Sub acute Perforation: An ulcer may perforate and the perforation may seal rapidly before there is spillage of gastric and duodenal contents, into general peritoneal cavity. There is sudden onset of abdominal pain, more severe to the right upper quadrant. On examination, there is local tenderness and rigidity, but rest of the abdomen will be soft to palpate and non-tender. Unusually X-ray film reveals a small amount of gas under the diaphragm. After an hour or two, the pain will usually subside. Rarely tenderness and rigidity may extend and the signs of an acute perforation develop.

3. Chronic Perforation: When an ulcer perforates into an area that is walled off by adhesions or by adjacent viscera such as liver, colon or greater omentum or when gastric ulcer perforates into omental sac, a chronic abscess may develop and give rise to considerable confusion in diagnosis. As these patients do not present with signs and symptoms of peritonitis, they are seldom diagnosed as having perforated peptic ulcer. An X-ray of abdomen may show subphrenic abscess, containing gas, and diaphragm is raised and fixed on the right side. USG of abdomen is the most reliable investigation on diagnosing intraperitoneal abscess.

4. Perforation associated with Hemorrhage:

The association of a perforation with massive haemorrhage is grave but fortunately rare complication. It may present in one of the three ways:

a. Haemorrhage and perforation occurring concomitantly.

b. Haemorrhage following a recently sutured perforation.

c. Perforation occurring during the medical treatment of haemorrhage.

The clinical features are that of perforated peptic ulcer with signs of haemorrhage.

5. Perforation of an Intrathoracic gastric ulcer: This is a rare variety of perforations.

The ulcer is in hiatus hernia,
which is fixed in the
mediastinum. Unless
existence of Hiatus hernia is known it is extremely difficult to make a correct diagnosis.

SEQUENCE OF EVENTS PERITONITIS:

The first peritoneal response to infection occurs within minutes of bacterial challenge. Bacteria and debris are cleared through the diaphragmatic lacunae into the lymphatic system. After a bacterial challenge, positive blood cultures can be demonstrated in less than 12 minutes. Dumon elucidated the

diaphragmatic peritoneum's role in bacterial peritonitis and systemic sepsis. In other studies, they treated the diaphragmatic peritoneum of rats with platelet rich plasma or talc powder before cecal puncture.

By sealing these lymphatic stomata, they substantially decreased their 24 hour mortality and incidence of positive blood culture. Hau have proposed that positive pressure ventilation also decreases bacterial dissemination by decreasing the port size of the diaphragmatic stomata. Associated with the humoral response to peritonitis is the production of antigen antibody complexes. Degranulation of mast cells and release of histamine causes an increase in vascular permeability fluid flux across the peritoneum occurs at rates in excess of 500 cm^3/hr.

Activation of complement leads to production of components C3-a and C5-a, which are powerful chemotactic factors for neutrophils. The initial bactericidal properties of peritoneal fluid are due to activation of the complement cascade. Other cytokines such as interleukin-2 and -8 have been shown to play a key role in recruitment in the cellular defense mechanisms. Within the first 4 to 6 hours, an influx of phagocytic cells into the free peritoneal space occurs that may be associated with a measurable granular cytopenia in the patient’s serum.

The omentum also plays a strategic role in the peritoneal cavity's host defense mechanism. Like the stomata of the diaphragm, the omentum has the ability to absorb foreign particles and bacteria. "Milky spots" in the omentum

have been described, which are aggregates of polymorphonucleocytes, macrophages and lymphocytes. When stimulated, these milk spots increase in number, develop germinal centers and produce antibodies. The mobility of omentum, allows it to migrate to areas of infection and assist in "walling off" the offending organ. The defect in mesothelium (perforation) is repaired by "metastasis" of nearby mesothelial linings. Peritoneal defects can heal everywhere very simultaneously. A large defect heals in the same time as a small defect, usually within 3-5 days. Fibrin formation starts usually after ten days and turns maximal two weeks after peritoneal injury

This explains the significance of difficulty of reoperation 2-4 weeks after an acute insult. With time passage , the fibrous adhesions usually undergoes remodelling and becomes progressively attenuated. In the patients with intra-abdominal soiling complications, like patients with EC fistulas, mortality is usually 20% in operations done between 10 to 120 days, but reduces to 10% before 10 days and after a period of 120 days..

Sequelae leading to multiorgan failure:

Sepsis is the major risk factor in the development of multiorgan failure syndrome. Sequential pulmonary, hepatic, GIT and renal failure may be recognized as early as 12 hours beginning of sepsis in septic shock or as late as 7-10 days. The observation that MOFS increases with severity and duration of shock highlights the importance of vigorous resuscitation and complete restoration of perfusion as rapidly as possible for better prognosis.

Injury to micro vascular especially microvascular endothelium, is a factor common to ischaemia reperfusion injury and multiorgan failure syndrome. Neutrophils are potential mediators of micro vascular injury. These cells produce an assortment of agents.

Toxic neutrophil products

Proteases	Toxic Oxygen Products
Elastase	$OH^{+}O_2$-
Collagenase	-HO2
Cathepsin G	HOC, H_2O_2

These products not only destroy bacteria, they also act in a non-specific fashion producing injury to normal microcirculation. The endothelial cell itself produces injury, when they are exposed to ischaemia, i.e. depression of ATP levels and increased xanthine oxidase. They produce free radical oxygen which causes endothelial activation and injury directly through both membrane peroxidation and increased neutrophil adherence in chemotaxis.

Considering importance of oxidant injury, as the main cause of MOFS, several clinical trials are being conducted to evaluate oxidant scavengers, as a treatment modality to prevent MOFS, NSAIDS that inhibit cyclooxygenase and prostanoid production may reduce pulmonary and myocardial injury in sepsis and ischaemia. Pentoxyphillin is an agent that may

benefit patients with ischemic and septic injury through inhibition of neutrophil adherence. Anti capsule (LPS) antibodies are being tried to prevent gram -ve endotoxin damage.

Decisive Period

After understanding the sequence of perforation sepsis leading to MOFS, Miles and Burke brought a concept of decisive time for bacterial infection. This period refers to time needed for bacterial numbers in peritoneal fluid or tissue to exceed a number greater than 10^5 / mm^3 or (per gm of tissue) and to establish a potent infection. Surgeons must deal with the infection before the bacterial numbers proliferates and reach these levels or remove the foci so that after operation, the residual numbers of bacteria are controlled and kept less than 10^5 / mm^3.

Diagnosis

The respected aphorism that states that **the diagnosis of peritonitis is made by clinical evaluation remains true even today.**

The diagnosis can be divided into

1. Evaluation of perforation
2. Diagnosis of sepsis syndrome.

1. Evaluation of perforation

Clinical feature

Abdominal pain is almost universally the predominant presenting symptom.

The historical characteristics of the abdominal pain can vary tremendously depending upon the ultimate cause. The pain of a fully established peritonitis is constant, burning and greatly aggravated by movement. Pain is usually localized to that dermatome distribution of the associated diseased visceral organ. Visceral peritoneum irritation usually is from the distension of a hollow viscous, causes a dull, poorly localized, and very often periumbilical and often severe crampy pain. Most symptoms result from inflammation of the visceral peritoneum, which receives afferent innervation only from the autonomic nervous system and is relatively insensitive, visceral afferent nerves respond primarily to traction or distension, but less well to pressure. Hence, stimuli are perceived as poorly localized dull discomfort. As inflammation spread from visceral to parietal peritoneum, the somatic pathways of the parietal peritoneum becomes involved, the pain seems to "migrate" from the region of epigastrium or umbilicus, to that involved quadrant or to the entire abdomen, depending on the extent of inflammation. Patient may present with other signs and symptoms like nausea, vomiting, alteration in bowel habits and systemic features like fever, sweating, tachycardia depending on extent of inflammation. They can be conveniently divided into localized peritonitis and diffused (generalized) peritonitis.

1. Localised Peritonitis

Here, the signs and symptoms are intimately related to the origin of the condition. Patient present with abdominal pain and usually there is associated

vomiting. **The important sign is guarding and rigidity of abdominal wall over the area of abdomen, which gets involved, with a very typical positive "release" sign.** The guarding may be severe to produce board like rigidity, (rebound tenderness). It may be associated with increased local temperature and increase in pulse rate, depending upon the inflammation, the features may either subside or progress to diffused peritonitis.

2. Diffuse peritonitis

Diffuse peritonitis presents in different ways depending on the period of infection.

Early :

Abdominal pain is quiet severe and made worse by moving. Patient usually lies still in this case. Tenderness with rigidity and palpation are typically found when the peritonitis affects the abdominal wall. Patients with pelvic peritonitis may not have abdominal wall tenderness and may complain of urinary symptoms. Vomiting may occur. The pulse rises continously, but if the peritoneum is filled with irritant fluid, there is a sudden immediate rise. Temperature changes can be variable and can be even subnormal.

Late : If resolution or localization of generalized type of peritonitis does not follow,the abdomen remains silent and increasingly gets distended.Circulatory failure can ensue,with cold,clammy extremities,sunken eyes,tongue turns dry,pulse can be thread and anxious face(Hippocratic facies).The patient finally lapse into unconsciousness.

Diagnosis of sepsis syndrome

Diagnosis of sepsis and sepsis syndrome hinges on understanding and identifying at an early stage the existence of a generalized inflammatory state.The systemic responses include hyperpyrexia, tachycardia, tachypnoea, decreased urine output, leucocytosis. While the presence of fever, leucocytosis, hypotension and hypermetabolic state are suggestive of sepsis, overwhelmingly may also result in leucopenia, cardiac suppression and shock.

Feature of Sepsis

- Temperature > 101ºF (or <96ºF as is frequently encountered in elderly)
- Heart rate > 100 / minute
- Respiratory rate > 20 / minute
- Leukocytosis (> 12,000 / mm^3 or < 4000 / mm^3)
- Manifestation of inadequate organ perfusion
- Diminished mental status
- Acidosis -plasma lactate.
  >3.0nmol / L
- Urine output < 30 ml / hour or 0.5 ml / kg /hour.
- Hypoxemia (Pa O2 < 70 on room air in the absence of underlying pulmonary disease)

Swan Ganz readings:

CO < 4.0 L min perm^2

(Cardiac output)

SVR < 600 dyne - sec / cm3 per m

PCWP < 8 mm Hg

The most important complication of sepsis is MOFS. There has been efforts to quantitate MOFS using practical bedside information. According to Fry, the criteria for failure were:

- Pulmonary failure in this system was defined as 5 or more consecutive days of need for ventilator support at an FIO_2 of 0.4 or greater.

- Hepatic - Bilirubin > 2 gm / dl, SGOT / LDH > twice normal. The inclusion of enzyme data was designated to exclude transient hyper billirubinemia that might be associated with retroperitoneal hematoma, pelvic fracture or potential icterus from an incompatible unit of blood.

- Renal failure – S. creatinine > 2 mg /dl.

- GIT failure - UGI haemorrhage

The above criteria are mainly for the use of clinical trails an epidemiological studies. Inclinical practice, the above criteria should be correlated with clinical findings as most of these criteria are hypothetical and further

clinicopathological studies are needed to confirm its validity.

CLINICAL FEATURES:

Age and sex: It is common in 30-40 years age group and common in males than females.

History of Present illness:

- **Time of onset:** Very often the patient is able to tell the exact time of onset of perforation, common particularly after an exertion in the evening.
- **Mode of onset:** Sudden in onset, at times the patient may wake up from the sleep, due to onset of pain.
- **Pain:** Pain is intense in the epigastrium then spreads all over the abdomen.
- **Shifting of pain:** The pain shifts to right iliac fossa as the fluid flows along the right paracolic gutter to settle in right iliac fossa, thus mimicking appendicitis.
- **Referred pain:** Pain is referred to the tip of the shoulder.
- **Nausea:** Present in some cases.
- **Vomiting:** Initially reflex vomiting occur due to irritation of nerves in the peritoneum and mesentery. In the later stages the vomiting is due to toxic action at the medullary centre's and causing paralytic ileus. The

vomiting then contains undigested food material and occasionally blood when hemorrhage is present.

- **Bowels:** In the later stage, there may be desire to defecate due to irritation of retrovesical pouch. Malena occurs when the hemorrhage is associated.
- **Micturition:** Oliguria is present if the patient is in shock.

Past History:

In 80% of patients, there is a past history of dyspepsia of variable duration. In the rest of the cases, the perforation may be the early clinical manifestation of a silent peptic ulcer.

Physical Examination:

- **General Appearance:** In the initial stage, the face is pale livid with sweating.
- **Decubitus:** The patient lies in a characteristic posture of supine, rigid and immovable, refusal of any attempt to shift his postures.
- **Pulse:** Initially it is normal, rapid when peritonitis sets in.
- **Respiration:** It becomes rapid and shallow when peritonitis sets in.

- **Temperature:** Initially normal; rises with the onset of peritonitis.

Examination of Abdomen:

Respiratory movements: Thoracic movement predominates over the abdominal movement with respiration. The abdomen does not move with respiration.

Rigidity of abdomen: The abdomen exhibits a board-like rigidity. Rigidity of abdomen is constant, continuous and characteristic board like. It is due to reflex contraction of the abdomen with predominance in the epigastrium and right hypochondrium.

Liver dullness: Obliteration of liver dullness elicited in front and in midaxillary line, is characteristic of this abdominal catastrophe in the second stage.

Free fluid: Free fluid is present in variable degree in many acute abdominal conditions. When internal hemorrhage is excluded, fluid of appreciable amount points out the provisional diagnosis of perforation in acute abdomen.

Rectal examination: There may be fullness in rectovesical or rectovaginal pouch.

INVESTIGATIONS:

1. Plain x-ray: The "gold standard" imaging to diagnose the finding of pneumoperitoneum, which can be seen on an upright anteroposterior

radiograph of the chest or the left lateral decubitus view of the abdomen. If the radiograph is taken with the patient in sitting posture and the patient has been in the upright position for 5 to 10 minutes, as little as 5 mL of free air can be seen under one or the other hemidiaphragm. With the left lateral decubitus position, the patient should be lying on the left side, and the first film should be taken with the patient on the cart in that position so that even a very small amount of air will become visible with, again, 5 to 10 minutes in the indicated position

2. Gastroduodenogram: Some clinics have used X-ray pictures of abdomen following injection of 60ml of 50% gastrograffin through nasogastric tube. The dye escapes through perforation, thus enabling to demonstrate the site and size of perforation, evidence of chronicity, associated gastric ulcer and if any second ulcer present. Use of barium for contrast radiography is contraindicated.

3. Ultrasound Examination: Ultrasonography of abdomen and pelvis was done with a convex multi -frequency probe (3.5-5 MHz) presence of intraperitoneal free fluid and of decreased intestinal peristalsis was considered indirect evidence of perforation. Ultrasound will also demonstrate the free air and occasionally a "fish-eye sign" when the anterior wall of the duodenum is perforated.

4. Computerized Tomographic Examination: Computed tomography (CT) is not often necessary, although it can be used when free air is not detected on conventional films or ultrasound; it is highly accurate in detecting even very small amounts of extra luminal free air.

5. Helicobacter Pylori infection diagnosis:

I..Non invasive -

a) Serology- ELISA.

b) Urea breath test.

II .Invasive -

a) Rapid urease test e.g. Eco, Pyloritek

b) Histology.

c) Culture.

Noninvasive tests do not require endoscopy, whereas invasive tests do.

For decades, delay in operative treatment has remained a primary determinant of morbidity, mortality, and cost. A recent study suggests that a positive peritoneal fungal culture is common and is a significant factor for adverse results in patients with a perforated peptic ulcer.

Some of the scoring systems for predicting the outcome of perforated peptic ulcer disease are

- Boey score ,
- Hacettepe score The Jabalpurscore

- American Society of Anesthesiologists (ASA) score
- Charlson comorbidity index
- Peptic Ulcer Perforation (PULP) score
- Acute Physiology and Chronic Health Evaluation II (APACHE II)
- sepsis score
- Mannheim Peritonitis Index (MPI)
- Simplified Acute Physiology Score II (SAPS II)
- Mortality Probability Models II (MPM II)
- Simplified Acute Physiology Score II (SAPS II)
- Physiological and Operative Severity Score for the enumeration of Mortality and Morbidity physical subscore (POSSUM-phys score)

In some studies, comparison of above mentioned scoring systems for predicting the outcome was done by "ROC-analyses" with reporting on the area under the curve (AUC). Some studies presented specificity and sensitivity, relative risks (RR) and odds ratios (OR), while most studies reported on performance by calculation of the chi square test.

Scoring systems aimed at prediction of outcome in PPU:

BOEY'S SCORE

This is the first scoring system designed to predict mortality in perforated peptic ulcer disease. The original study of Boey depicts when there is delay in surgery after onset of the symptoms more than 48 hours, in state of

shock during emergency opd visit and with comorbid condition, there is high risk for mortality and poor outcome. Same study with delay in surgery for 24 hrs is validated in a cohort from Hong Kong.

HACETTEPE SCORE:

Hacettepe score was designed for PPU patients and contains four factors. One study evaluated 173 cases from parts of Turkey and found the Hacettepe score to be equivalent to the Mannheim Peritonitis Index (MPI), with a sensitivity being calculated as 83% and specificity found to be 94% for predicting mortality. The sensitivity for the MPI in this study was calculated as 75% and the specificity as 96%.

JABALPUR SCORE:

Jabalpur score was on the basis of studying one hundred and forty cases from parts of India and average age of 39 years old. The score consists of 6 factors, based on preoperative evaluation. The morbidity and mortality were predicted on the basis of high value "AUC".

PEPTIC ULCER PERFOERATION SCORE:

In recent days, the "Peptic Ulcer Perforation (PULP) score" has been formulated as a scoring system for perforated peptic ulcer. PULP score is based on a study from parts of Denmark and included some 2668 PPU cases with a median age of 70.9 years, where fifty five cases was female. Based on seven factors with weighted points applied for individual factor, with a maximum of eighteen points being the maximum value. The cutoff value was assigned to be

seven points. The results found to have “positive predictive value” (PPV) of 25% for cases having 0-7 points, and the PPV of 38% in patients having eight or more points. The Peptic Ulcer Perforation score is compared with various scores.

ASA SCORE:

American Society of Anesthesiologists score designed in 1941 and formulated for assessing preoperative status of patients. It is one of the ageold scoring systems. ASA score is often demonstrated along with various patient datas like age, sex, and physiologic factors. This study has no particular role in predicting outcome of the patient.

CHARLSON COMORBIDITY INDEX:

Charlson comorbidity index was designed to stage the comorbid conditions into various risk levels by assigning scores to different diseases . The Charlson index has taken into account nineteen conditions deemed clinically important, and they are assigned with “1 to 6” points. CVD is given 1 point, late stage liver illness 3 points and metastatic cancer and AIDS with six points. It was designed in predicting long-term mortality. Latter researches found to have its usefulness in predicting hospital morbidity and mortality. Some studies used this Charlson comorbidity index in predicting outcome in PPU cases. A significant corelation between a medium or high Charlson score and 30-day mortality was reported, having “odds ratio (OR)” of 4.17 for high score (3 or more points on the Charlson score) and an OR of 3.99 for medium score (1-2

points on the Charlson score). There is nostudies to confirm this scoring system pertaining to PPU.

SEPSIS CRITERIA:

In Sepsis criteria there is less cumbersome to assess preoperatively and the presence of sepsis. To tell there is septicemia there should any two or more parameters which are mentioned here should be positive which includes temp >38 degree celsius or < thirty six degree celsius, RR > 20 per minute or PCO2 < 4.3kPa, HR > 90 /min, leucocytosis > 12.0×109 or < 4.0×109. It is clearly revealed that this scoring system has its usefulnesss in many fields of medical sciences, and also used to predict the outcomes of cohort in PPU.

MANNHEIM PERITONITIS INDEX:

"Mannheim Peritonitis Index (MPI)" comprises of 7 parameters which are closely have its significance in the operative findings. By its name, the design has its importance to patients who are undergoing surgeries for peritonitis. "Mannheim Peritonitis Index (MPI)" was taken account of preoperative and peri-operative conditions, and it was found to have its usefulness in predicting morbidity and not much used so in predicting mortality in PPU cases.

APACHE II SCORE:

"Acute physiology and chronic health evaluation II (APACHE II) score" is one of the commonest score universally and the most used ICU score in United States. APACHE II score comprises 12 various physiological

parameters, age of patient and previous history. It was initially formulated to stratify intensive care unit patients. According to this maximum score is given to extreme ends (high or low), between 0 (36.0°C -38.4°C) and 4 (≥41°C and ≤29.9°C). Initially found to give good prediction for ICU patients and in due course started to use to predict the outcomes in perforated peptic ulcer. A study from the USA revealed nil mortality in PPU patients with less than score 11 points, and a 35% mortality rate in persons with at least 11 points, which indicates this as a useful cut-off. Various studies revealed different results which contradicts the previous studies. Some demerits lie in its complex mathematical calculations and scoring should be done before 24hr. These reasons make concerns regarding its usage. In spite of these, it is used frequently and globally. APACHE II has been shown to predict the outcome well also for PPU patients.

SAPS II SCORE:

"Simplified acute physiology score II (SAPS II)" is developed to predict outcomes of INTENSIVE CARE UNIT PATIENTS, which comprises 17 parameters. Devised in 1980s and a modified score was made in use during 1993. The "Simplified acute physiology score II" system is often practiced to predict the outcomes in ICU PATIENTS in European and scandinavians, and has various resemblance with APACHE II system. These systems are complex, with a number of factors incorporated in the calculations, including physiologic parameters. The SAPS II system predicts mortality and morbidity well, but also

seems more suitable for ICU patients.Nevertheless, this score performed well for outcome prediction of PPU patients.

MPM II SCORE:

"Mortality probability models II (MPM II)" was developed in predicting the outcomes in intensive care unit patients. It uses fourteen different parameters pertaining to systemic perfusion. Mortality probability models II predicted mortality in a better manner than SAPS II and APACHE II in some studies. It produces small and skewed in patients belongs to male gender and age in 20s and 30s, which contradicts to current perforated peptic ulcer cohorts. Few study groups in Asians and Africans have produced similar results. Current study groups of Scandinavian and Northern European have reported data with a 1:1 m:f ratio and average age near 70 years old. MPM II is a rather complex system, thus not much useful for a pre-operative evaluations in the clinically presenting patients with perforated peptic ulcer disease.

POSSUM SCORE:

"Physiological and Operative Severity Score for Enumeration of Mortality and Morbidity (POSSUM score)" uses 12 different parameters on the basis of physiological status of the patients and six various parameters pertaining to operative conditions. The parameters are filled in two mathematical equations for risk assessment. The "Physiological and Operative Severity Score for Enumeration of Mortality and Morbidity" score was developed in UK in predicting outcomes of intensive care unit patients. The

POSSUM score seems to be overestimation of the mortality in low risk groups. Therefore (P-POSSUM) was developed to correct this. In predicting the mortality and other outcomes in emergency cases are well estimated by the P-POSSUM score, when compared to the original POSSUM score. The POSSUM-phys score, which is the physiologic sub score, contains the 12 physiologic parameters, which can be assessed preoperatively. Only a study was found that applied POSSUM-phys to PPU patients. In this study 261 PPU patients with a mean age of 67 years were studied and the POSSUM-phys score predicted well both the mortality and morbidity. The POSSUM-phys score, in contrast to the POSSUM score, is preoperatively assessed. In Perforated Peptic Ulcer cases, we have not came across any study groups comparing POSSUM score with various other systems of scoring.

TREATMENT:

Immediate management (Resuscitation):

1. Patient is kept to be nil per oral.
2. Treatment begins with insertion of a nasogastric tube to decompress the stomach and limit additional peritoneal spillage.
3. A Foley's catheter is inserted to monitor urine output and direct resuscitation.
4. The patient is resuscitated aggressively by administration of intravenous crystalloid.

5. Intravenous broad-spectrum antibiotics are also administered.

6. Invasive hemodynamic monitors (e.g., central venous, arterial, and pulmonary artery catheters) are inserted as clinical status and comorbid medical conditions dictate.

7. Associated medical illnesses such as respiratory disease should be treated quickly and effectively so as to minimize complications.

8. Informed consent should be taken.

Operative treatment is generally advocated as the best option, but conservative treatment is an alternative in carefully selected patients.

NON OPERATIVE / CONSERVATIVE TREATMENT: It is considered in

• In the patients who do not have the generalized peritonitis features,

• In patients who are hemodynamic stable,

• In patients whom a water-soluble contrast study has confirmed that the ulcer is sealed with no leakage of contrast into the peritoneal cavity.

The patient can be treated expectantly with nasogastric suction, intravenous fluids, antibiotics, and bed rest. If at any time during conservative management the patient's general condition deteriorates, an operation is indicated. Conservatively managed patients often develop intra-abdominal abscesses, especially in the subhepatic or subdiaphragmatic locations, and these abscesses usually can be managed percutaneously.

In the largest published series of patients with perforated duodenal ulcer who were managed conservatively, patients who were 70 years of age and older were much more likely to require operative therapy and had a higher mortality rate.

OPERATIVE TREATMENT:

In perforated duodenal ulcers, options include-

• Simple closure with an omental onlay reinforcement or patch.

• Simple closure with proximal gastric vagotomy

• A truncal vagotomy and drainage procedure

In perforated gastric ulcers, options include-

•Simple closure after four quadrant biopsy.

• Excision and primary closure.

• Gastric resection.

Simple closure of perforated peptic ulcer has been compared with truncal vagotomy and pyloroplasty and vagotomy and antrectomy in randomized clinical trials prior to the *H. pylori* era. Ulcer recurrence rates were 61% following simple closure compared with 6% following a definitive operation. However, recurrent ulcer disease after peptic ulcer perforation occurs mainly in patients infected with H. pylori. This is supported by several studies that demonstrated low recurrence rates following simple closure of the ulcer with H.pylori eradication. Therefore, patients presenting with perforated duodenal

ulcers should be tested for *H. pylori* and if found to be positive treated with eradication therapy.

One study that followed patients for more than 10 years after truncal vagotomy and pyloroplasty for perforated duodenal ulcer reported a mortality rate of 5.5% with a recurrence rate of 8.8%. Operative mortality rates from perforated duodenal ulcers range from 5% to more than 30% in the elderly. Risk factors that increase mortality include a severe comorbidity, the presence of shock on admission, and a presentation delay of more than 24 hrs.

Perforated Ulcer Patients Requiring Definitive Ulcer Procedure:

1. Large perforations (>2.0cm): require vagotomy, antrectomy, Billroth II reconstruction.
2. Synchronous bleeding and perforation: generally require vagotomy, resection of perforation, pyloroplasty, U-stitch control of posterior bleeding.
3. Chronic ulcer symptoms, *H. pylori* negative: patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty with ulcer excision.
4. NSAID dependence (or noncompliant patient): patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty, ulcer excision.
5. Previous *H. pylori* treatment failure or known *H. pylori* negative patients: patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty, ulcer excision.

6. Previous ulcer complications: patch closure, parietal cell vagotomy, *or* vagotomy, pyloroplasty, ulcer excision.

7. Perforated gastric ulcer (more than 1-2 cm proximal to pyloric vein): antrectomy, with or without vagotomy, Billroth I reconstruction.

8. Previous operation for duodenal ulcer: if previous vagotomy, requires 60%-70% gastric resection, Billroth II anastomosis; if previous adequate gastrectomy, requires truncal vagotomy, possible reresection.

9. Young patients (under 40 years): patch closure, parietal cell vagotomy, *or* vagotomy, excision of ulcer, pyloroplasty.

Contraindications to definitive ulcer surgery at the time of closure of a perforation

1. Serious concurrent medical illness (e.g., myocardial ischemia, previous congestive heart failure, diabetes out of control, chronic obstructive lung disease with chronic respiratory acidosis, and marginal or inadequate renal function)

2. Patients who are in shock or are hemodynamically unstable on presentation.

3. The patient has been perforated for more than 24 hours.

SURGICAL TECHNIQUE:

IN PERFORATED DEODENAL ULCER:

Simple closure with an omental onlay reinforcement or patch:

For patients who have not been treated previously for "Peptic ulcer disease" and who can be administered with proton pump inhibitors and with anti- H.pylori regimen , the preferred treatment is "Simple patch closure" is appropriate.

• "Graham Patch Closure" is so called after the surgeon Roscoe who first described this technique during the year 1938.

• Steps:

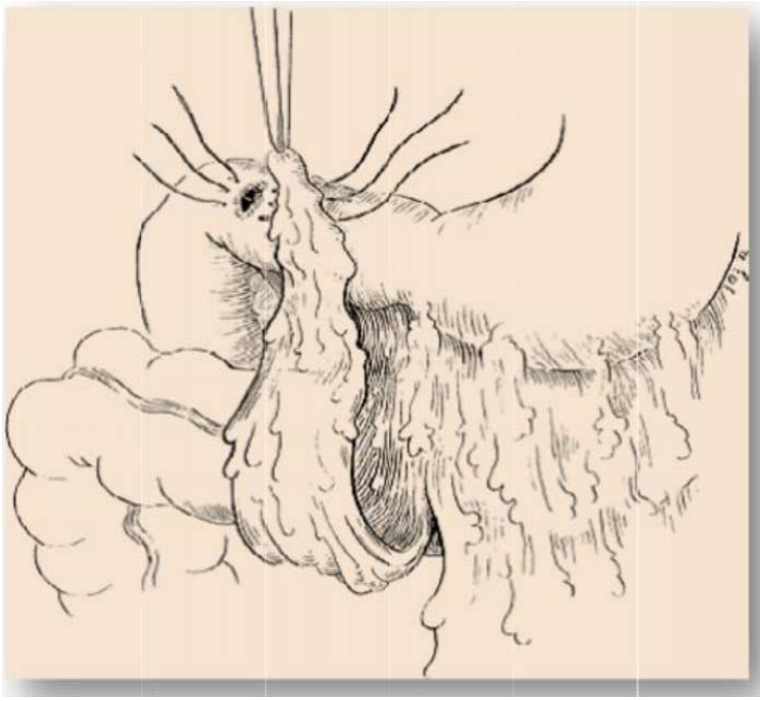
1. Upper midline incision made. Incision deepened. Peritoneal cavity entered
2. After letting out the purulent or bile stained toxic fluid, the perforation site should be searched.
3. The most common site of perforation will be I part of duodenum over the anterior surface.
4. If perforation site could not be found proximal duodenum, exploration in other parts of duodenum, anterior wall of stomach and along the jejunal loops have to be done.
5. Then three through and through stay sutures with non absorbable material is taken intermittently in perforated bowel wall.
6. Before placing the knots place adjacent healthy omentum over the perforated

area within the suture material. Knotting should be in the following sequence, first the sutures in the periphery are tied and lastly the suture in the middle is tied. This facilitates the omentum with the vascular pedicle is snugly approximated without tension so that the vascularity is not jeopardized. .

7. There are some modifications in above techniques where the perforated site is primarily closed with non absorbable suture material and then omentum is patched over the closed perforated site with another sets of intermittent suture materials

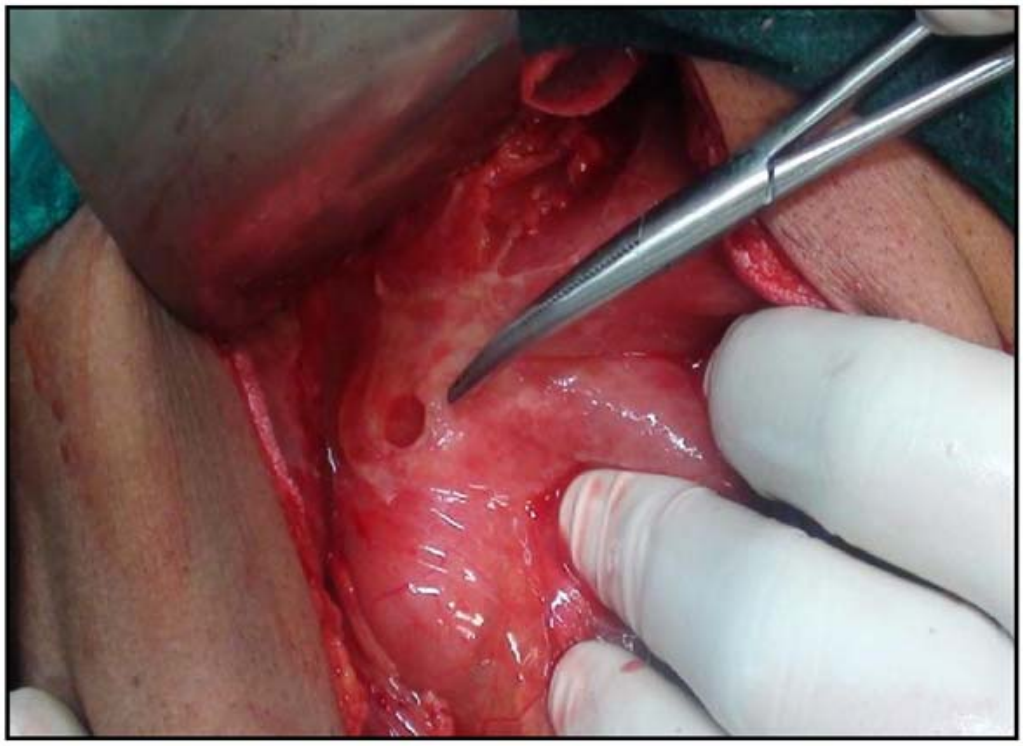
8. At the end before closing the rectus thorough peritoneal irrigation have to be given with normal saline {minimum of 1 to 2L}.

9. Then abdomen is closed in layers by usual manner.

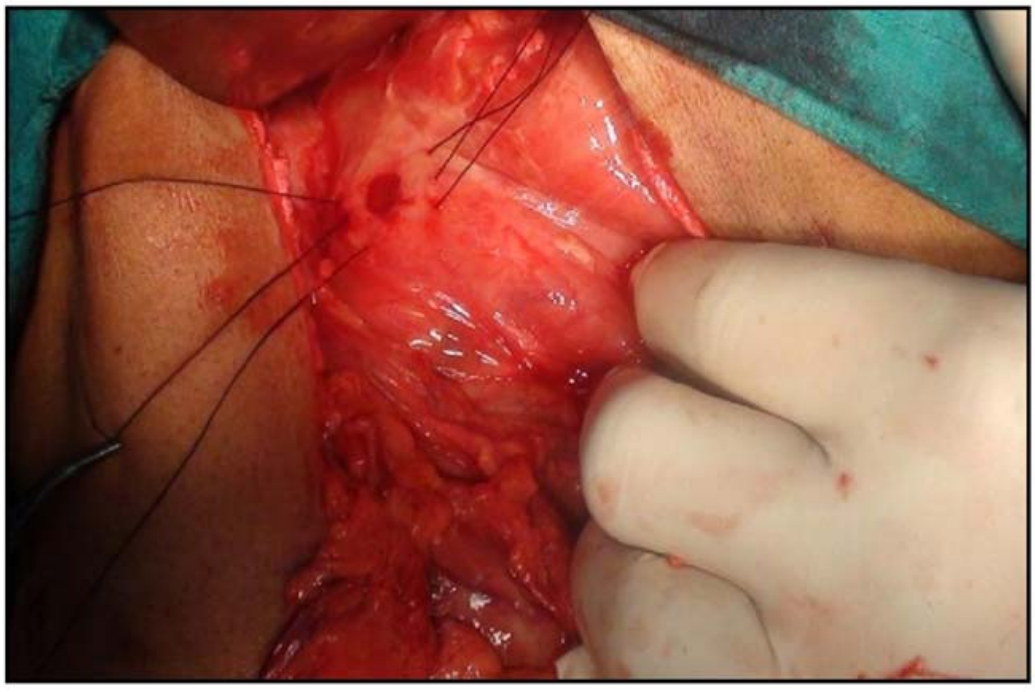


After placing three or four sutures, a vascularized tongue of omentum is mobilized and brought superiorly to close the defect. It is not necessary to push the end of the omentum into the defect like an obturator, but rather use the omentum as an external patch.

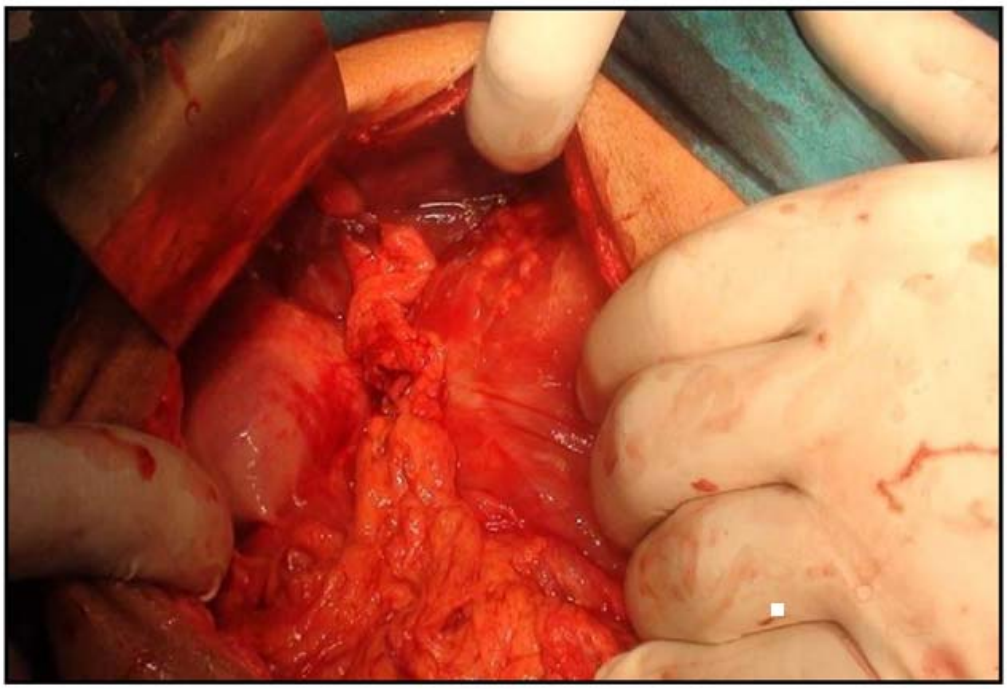
When the sutures are tied loosely enough so that the blood supply to the omentum is not compromised, the seal is complete, even with larger perforations.



Showing Duodenal ulcer in the first part of the duodenum



Showing Duodenal ulcer in the first part of the duodenum



Showing closed duodenal perforation with Graham's Omental patch

2. Simple closure with proximal gastric vagotomy:

- Proximal gastric vagotomy is done in some cases additional with patch closure of perforated ulcer
- This is done to reduce the acid secretion by selectively denervating the vagal branches to parietal cells and preserving the smooth muscle which plays an important role as motor pump.
- Hence there is no need for drainage surgeries. In patients with perforated duodenal ulcers, proximal gastric vagotomy

3. A truncal vagotomy and drainage procedure

- Traditionally the definitive surgery peptic ulcer disease is "Truncal vagotomy and pyloroplasty or antrectomy".
- Above procedure is usually done in cases who need continuous usage of " non-selective NSAIDs ", those had h/o failure of adequate medical treatment and cases where there is a doubtful compliance for anti- H.pylori regimen
- Option between " Pyloroplasty and Gastroenterostomy " is decided according to the nature of conditions present in the pyloroduodenal region including whether the perforated site can be included in pyloroplasty.
- Sometimes above technique cause significant stenosis that nessacitates closure of perforated site and gastroenterostomy. In case of scarred duodenal ulcer causing gastric outlet obstruction requires gastroenterostomy.

In PERFORATED GASTRIC ULCER:

Options include-

• Simple closure after four quadrant biopsy.

• Excision and primary closure.

• Gastric resection.

There is different school of thoughts regarding “partial gastrectomy” or “simple patch closure” in patients with Type I and Type IV. In patients with increased risk factors like old age, comorbidities, unstable vitals, gross peritoneal contaminations with gastric contents,it is preferred to proceed with “Partial Gastrectomy” .

• In patients with “Perforated Type I gastric ulcer” , it is advisable to proceed with “Partial Gastrectomy” provided the patient’s vitals are stable and without any significant comorbid illness.

• For High Type IV Gastric ulcer, which requires extensive resection sometimes compels “Total Gastrectomy” in a patients with associated severe comorbid illness, it is preferred to take Biopsy from abnormal mucosal surface or along the edges and patch closure. Performing 4 quadrant biopsies in ulcer surface is must in case where the ulcer is not excised. Among “simple suturing and closure of ulcer” and “patch closure”, patch closure has favorable outcome than the other technique previously mentioned.

LAPAROSCOPIC REPAIR PERFORATED PEPTIC ULCER:

Laparoscopic procedures are gaining its position in treating perforated peptic ulcer for patients with stable vitals and with mild severity of septicemia.

• The idea and methods in laparoscopic repair is as similar as open procedure. In open procedure greater omentum is used for patch closure whereas in laparoscopic repair falcifom ligament is laid over the perforated site and sutured. Falciform

ligament patch closure is easier procedure than omental closure through laparoscopic technique.

•In circumstances like naturally sealed omental closure, peritoneal irrigation alone is preferable. Laparoscopic technique gives good visual and manipulating access during irrigation of peritoneum especially to sub-phrenic and pelvic cavity.

• Efficacy, morbidity, mortality is equivalent to open repair. Also operative time, days of hospital stay, usage of analgesics and recovery period are comparably better when compared with open repair.

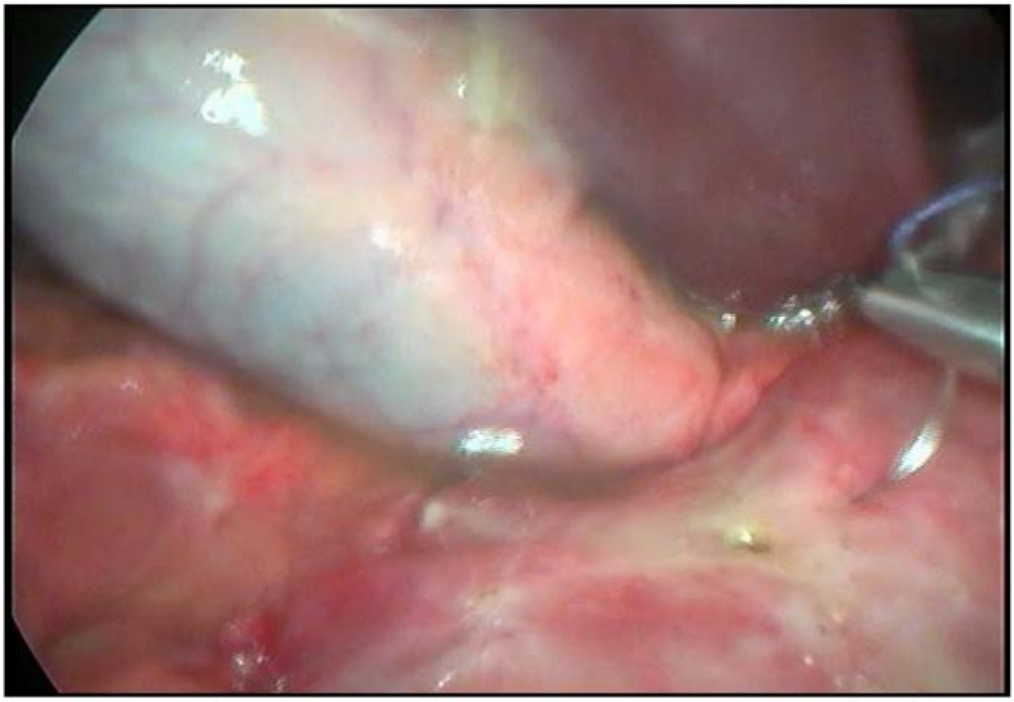
The outcome of patients with a perforated ulcer depends on the following:

1. Delay from initial evaluation to treatment: recent data suggest increasing delay until surgical treatment.

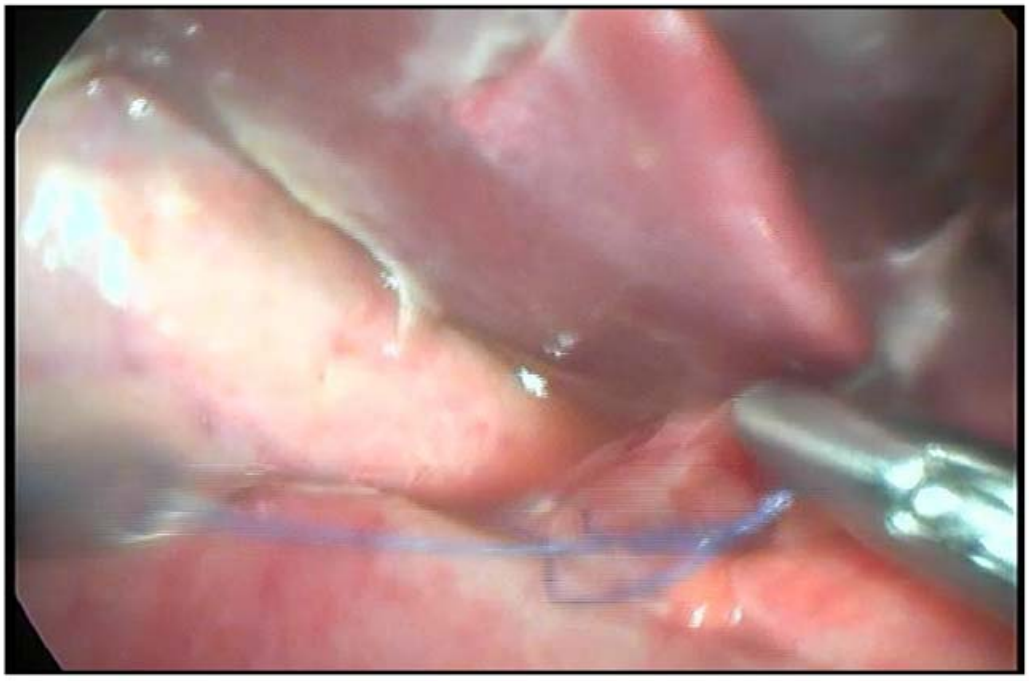
2. Site of perforation: gastric perforations are associated with a poorer prognosis.

3. Patient's age: elderly patients, who often have associated comorbid conditions, have a worse outcome.

4. Presence of hypotension at initial evaluation (systolic blood pressure <100 mm Hg).



Showing laparoscopic closure of duodenal perforation



showing laparoscopic closure of duodenal perforation

Post operative complications:

Complications are most likely to happen in higher risk patients. The most common complications are as follows:

- Paralytic ileus.
- Wound infection.
- Intraperitoneal abscess, usually subphrenic or pelvic.
- Peritonitis.
- Respiratory complications like atelectasis, pneumonia, pleural effusion,
- respiratory failure
- Gastric and duodenal fistulae.
- Renal failure.
- Mediastinitis.
- Septicaemia.
- Deep vein thrombosis

MATERIALS AND METHODS:

Perforative peritonitis is one of the most commonly encountered surgical emergencies in all setups for a practicing surgeon. Among all hollow viscus perforative peritonitis, DU perforation is the most common cause of perforative peritonitis.

In spite of easy diagnosis, operative procedures and advent of newer antibiotics, postoperative morbidity and mortality is still a challenging task for all surgeons. Postoperative complications like wound infection, intraabdominal infection, septicemia and mortality can be easily combated better with appropriate sensitive antibiotics than empirical antibiotic policy.

Though many researches clearly depicting that there are only few common groups of organisms being isolated, sensitivity pattern differs in different setups. Hence sensitive appropriate postoperative antibiotic policy plays an important additive role to surgical treatment in managing perforative peritonitis.

AIM OF THE STUDY:

To study the bacteriological profile in the peritonitis patient due to duodenal ulcer perforation and their sensitive antibiotics by collecting peritoneal fluid and to reduce postoperative morbidity and mortality .

OBJECTIVES OF THE STUDY:

A. To detect commonest organisms in peritonitis due to duodenal ulcer perforation.

B. To find out antibiotic sensitivity pattern in peritonitis due to duodenal ulcer perforation.

C. To see response of patients after starting antibiotics according to the culture and sensitivity report in terms postoperative complications like septicemia, surgical site infection and hospital stay.

ELIGIBILITY CRITERIA:

A.INCLUSION CRITERIA:

1. All peritonitis patients with duodenal ulcer perforation.
2. Patient's age above 13yrs.
3. Patients with stable vitals who are fit for surgery.
4. Patients consented for inclusion in the study

B.EXCLUSION CRITERIA:

1. Patients with peritonitis due to other hollow viscus perforation.
2. Patient's age below 13yrs.
3. Patients with traumatic perforation

4. Patients with perforative peritonitis not operated due to unstable vitals.

5. Patients in immunocompromised state like HIV, TB.

MATERIALS USED:

1. Proforma containing patient history, clinical examination, investigations, intraoperative findings and operative notes.

2. Informed consent forms.

3. Sterile container with screw cap and requisition forms.

METHODOLOGY:

This study is designed to know the bacteriological profile and their sensitive antibiotic for the peritonitis patients.

- 140 patients who are admitted in GRH between JAN,2016 and AUG,2016 with bowel perforation are studied
- Intra operative peritoneal toxic fluid culture and sensitivity is done in all patients taken up for surgery
- Effectiveness in view of post-op outcome is studied between empirical versus specific antibiotic administration.
- Then the study proceeds to identify the bacteriological profile, sensitive antibiotics and compare the post-op outcomes between administration of empirical versus specific antibiotic pattern.

COLLECTION OF SAMPLE AND TRANSPORT:

- Peritoneal toxic fluid is collected after opening the peritoneum
- 5 ml of fluid will be collected in sterile screw cap container
- Sample will be transported from OT to microbiology lab at room temperature immediately if possible.

Sample stored in refrigerator at 6degree celcius if transportation delayed for more than 3 hours

PROCESSING OF SAMPLE:

- 1st day: microbiology and culture inoculation.
- 2nd day: if growth is present then organisms are identified , then antibiotic sensitivity test is performed
- 3rd day: Isolated organisms and sensitive antibiotic is ready…

ASSESSMENT OF POST-OPERATIVE OUTCOMES:

1. Postoperative outcomes are assessed in terms of postoperative complications like
    - ✓ *Wound infection*
    - ✓ *Wound gaping*
    - ✓ *Burst abdomen*
    - ✓ *Septicemia,*
    - ✓ *Lung infections like bronchopneumonia*

- ✓ *Days of hospital stay,*
- ✓ *Mortality*

2. Postoperative secondary minor procedures like secondary suturing for wound gaping and tension wire banding for burst abdomen and its incidence is also studied.

3. Finally these outcomes are compared between empirical and specific antibiotic administration groups.

OBSERVATIONS AND RESULTS :

This study includes 140 patients with perforative peritonitis and who were taken up for surgery and intraoperatively diagnosed to have duodenal perforation. And various data like age, sex, history, clinical examination findings, investigations like routine urine and blood reports, Xray chest PA and abdomen erect view, ultrasonogram of abdomen and pelvis, preoperative diagnosis, intraoperative findings, peritoneal fluid culture and sensitivity report, details of postoperative outcomes in terms of complications like wound infection, gaping, burst abdomen, septicemia, lung infection, mortality, days of hospital stay and no. of cases taken up for secondary minor procedures like secondary suturing and tension wire banding of 140 patients are collected and consolidated .

Then age , sex distribution, the commonest organisms, their sensitive antibiotic pattern , among 140 DU perforative peritonitis patients fulfilling the

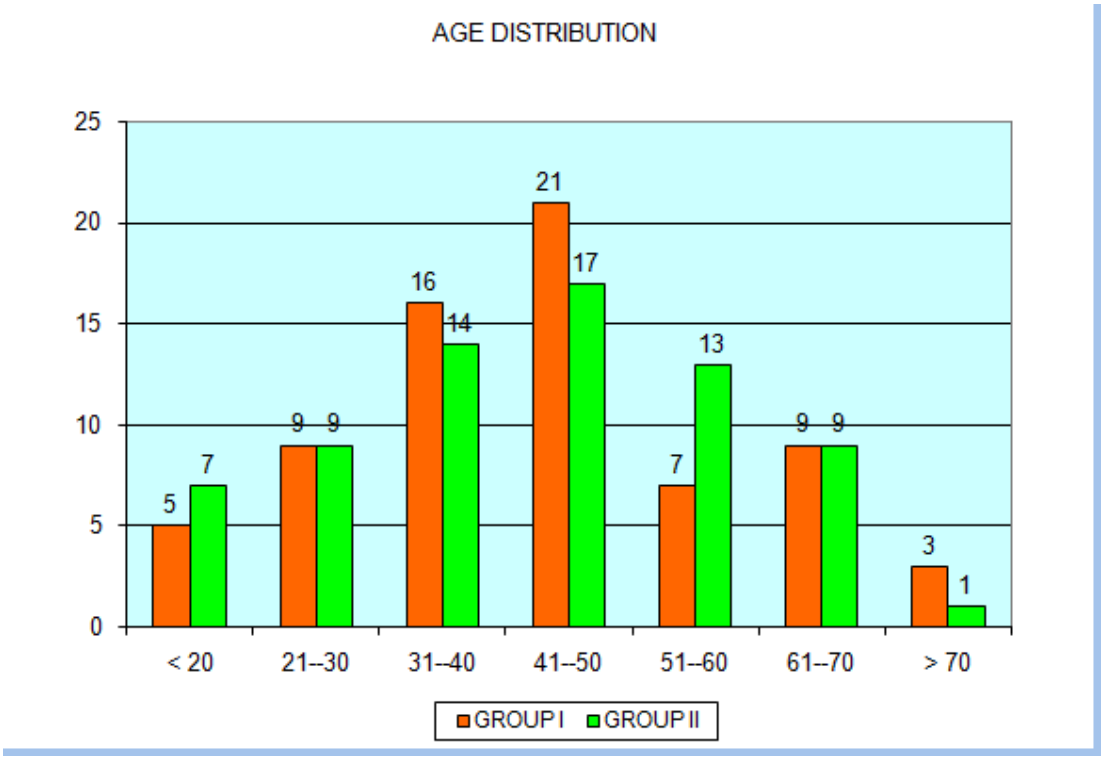
eligibility criteria , are studied. Also comparison study is done between 70 patients(group I) who were administered with empirical antibiotics and 70 patients(group II) with specific antibiotics according to intraop peritoneal fluid culture and sensitivity report , in terms of post operative complications and incidence of postop secondary minor procedures as previously mentioned. The observations and results of the study is depicted in the following pages.

AGE DISTRIBUTION:

In this study age is distributed in a pyramidal pattern. Peak of the pyramid denotes the age distribution in age interval between 41-50 yrs comprising about 27%. Next major age distribution is between 31-40 yrs includes 21% of study population. Age above 70 yrs constitutes only 2.8%.

AGE DISTRIBUTION :

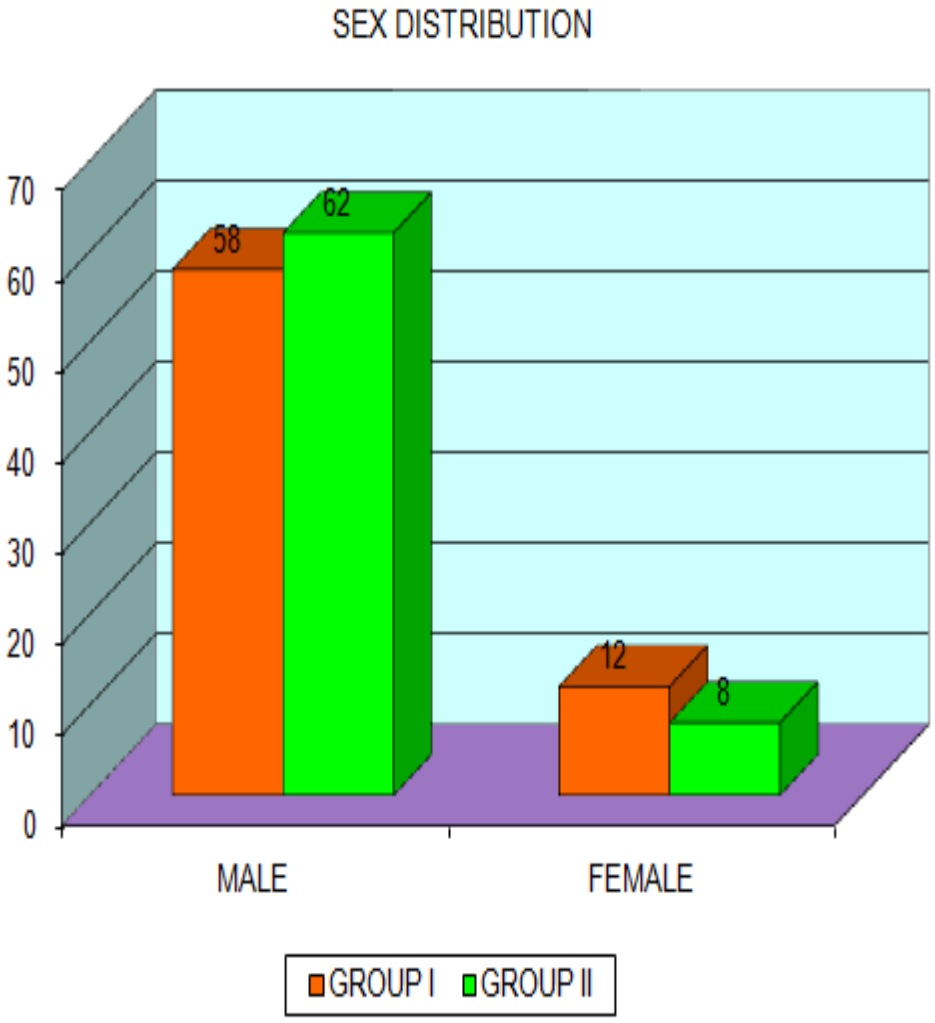
AGE	GROUP I	GROUP II	TOTAL
≤ 20	5	7	12
21—30	9	9	18
31—40	16	14	30
41—50	21	17	38
51—60	7	13	20
61—70	9	9	18
> 70	3	1	4
TOTAL	70	70	140



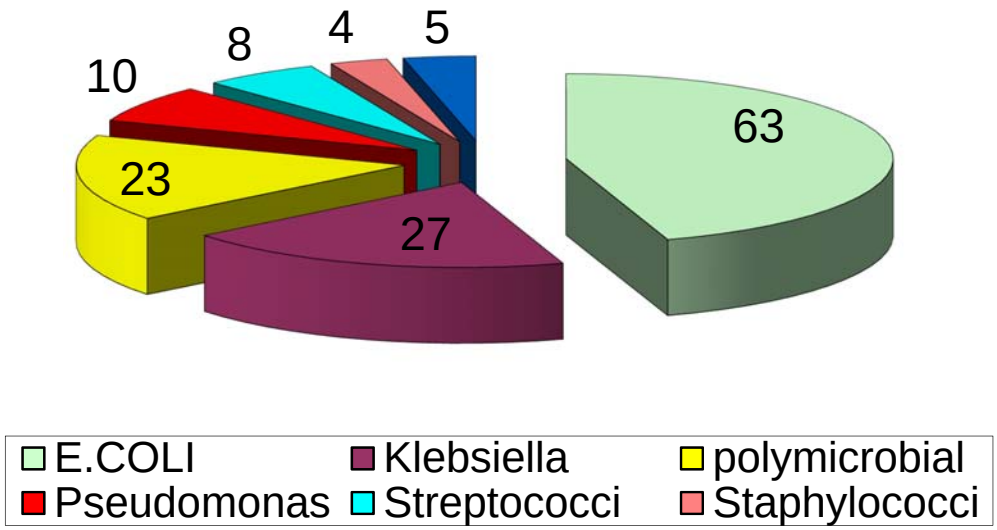
SEX DISTRIBUTION :

In this study male preponderance is observed. Female patients constitute only 14% of study population.

SEX	GROUP I	GROUP II	TOTAL
MALE	58	62	120
FEMALE	12	8	20
TOTAL	70	70	140


SEX DISTRIBUTION
70
60
50
40
30
20
10
0
58
62
12
8
MALE
FEMALE
GROUP I
GROUP II

ISOLATED ORGANISM DISTRIBUTION



ISOLATED ORGANISMS	TOTAL
E.coli	63
Klebsiella	27
polymicrobial	23
Pseudomonas	10
Streptococci	8
Staphylococci	4
Others	5

BACTERIOLOGICAL PROFILE :

In this study , it is obsevered that the most common organism isolated from intraoperative peritoneal fluid culture analysis is E.coli , accounts to 45% of the total study group of 140 patients. Other organisms isolated are as follows:

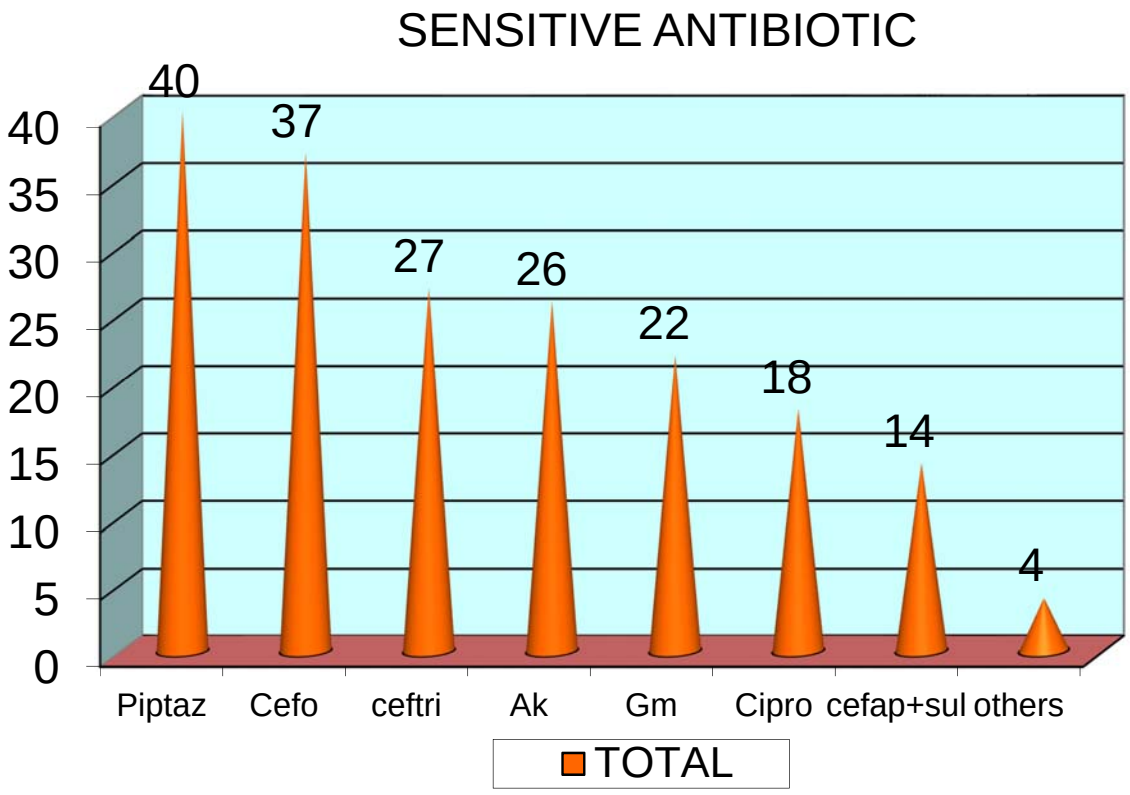
- Klebsiella - 19%
- Polymicrobial flora -16%
- Pseudomonas - 7%
- Streptococcus - 5%
- Staphylococcus -2%
- Other least frequently isolated organisms (3.5%) include Bacteroides fragilis, Citrobacter, candida etc,.

ANTIBIOTIC PROFILE :

In this study, PIPERACILLIN TAZOBACTAM is found to be the most common antibiotic that is most sensitive against bacteriological flora which are isolated from DU perforative peritonitis. Piperacillin tazobactam constitutes about 28.5% among other antibiotics, next common most sensitive antibiotic drug is cefotaxime accounts to 26.4% . Other antibiotic profile observed to be sensitive in this study in descending trend are ceftriaxome, amikacin, gentamycin, ciprofloxacin, cefaperazone sulbactum, meropenam , imipenam..

ANTIBIOTIC PROFILE:

SENSITIVE ANTIBIOTIC	TOTAL
Piptaz	40
Cefotaxim	37
Ceftriaxone	27
Amikacin	26
Gentamycin	22
Ciprofloxacin	18
cefap+sul	14
Others	4



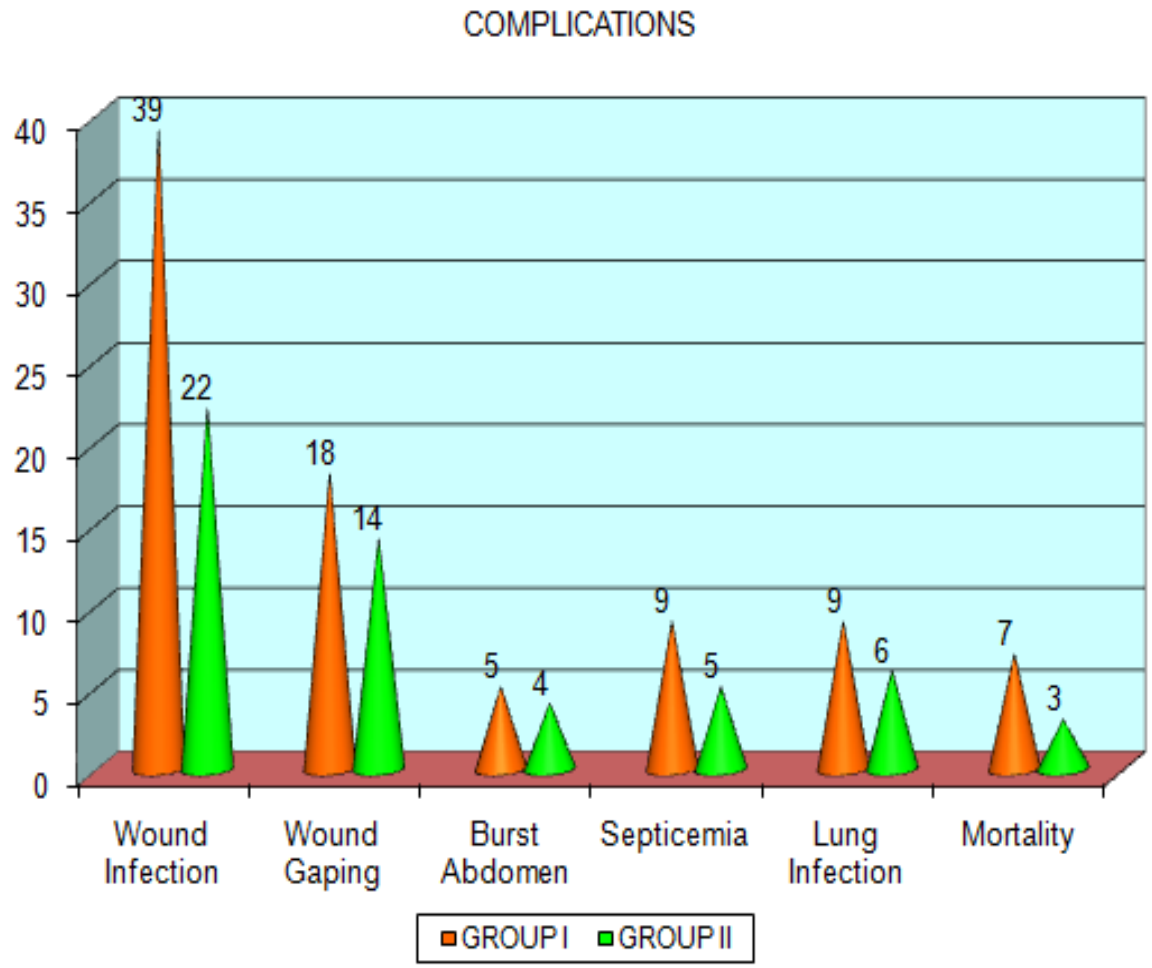
COMPARISON OF POSTOP OUTCOMES:

The incidence of postop complications in terms of wound infection, wound gaping, burst abdomen, septicemia, lung infection and mortality are compared between Group I where patients are started on empirical antibiotics (commonly used in the study setup is combination of III GEN Cephalosporins and coverage for anaerobes like metrogyl) and Group II where patients are started with specific antibiotics according to intra operative peritoneal toxic fluid culture and sensitivity report.

In this study it is observed that above mentioned postoperative complications are significantly less in patients for whom specific antibiotics is administered according to intraoperative peritoneal toxic fluid culture sensitivity report.

COMPICATIONS	GROUP I	GROUP II
Wound Infection	39	22
Wound Gaping	18	14
Burst Abdomen	5	4
Septicemia	9	5
Lung Infection	9	6
Mortality	7	3

COMPARISON OF POSTOPERATIVE OUTCOMES:

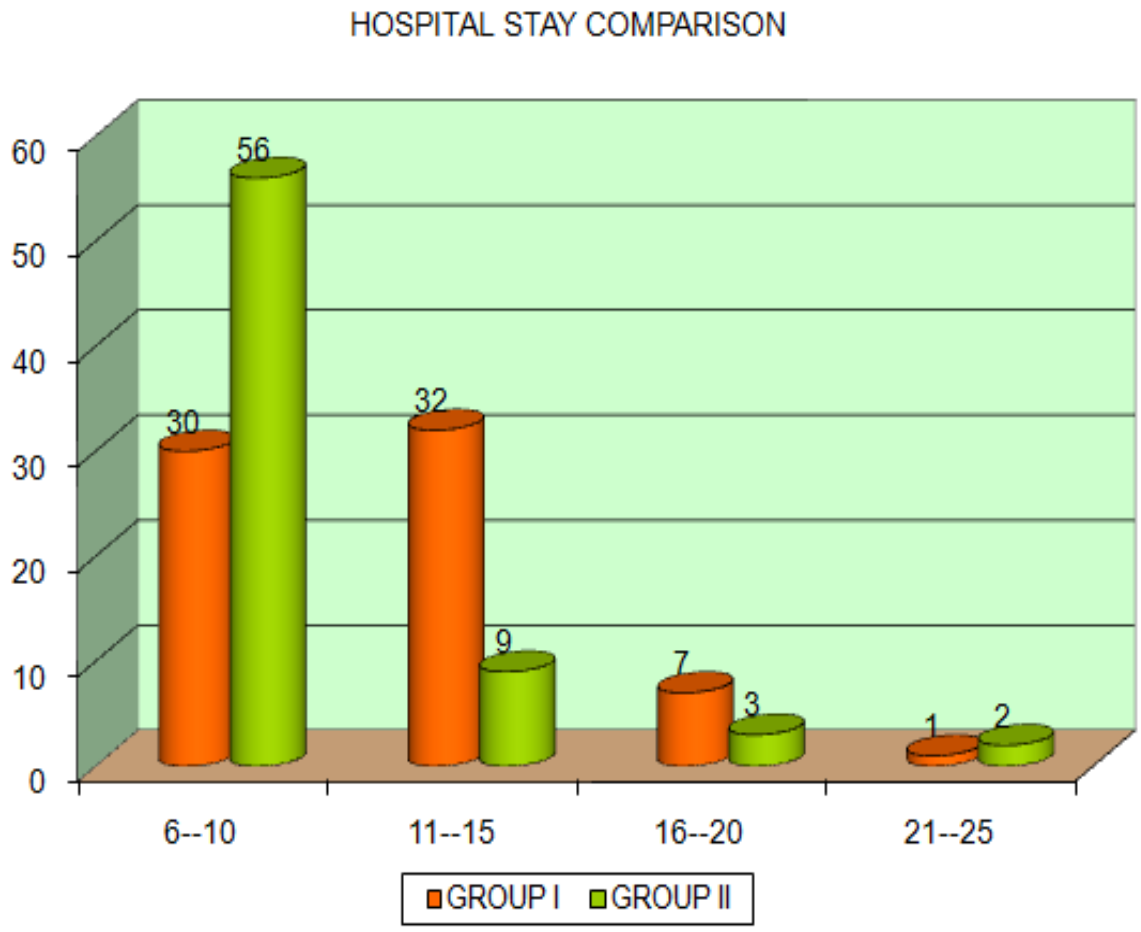


COMPARISION OF POSTOPERATIVE DAYS OF HOSPITAL STAY:

The comparison of days of hospital stay between patients administered with empirical antibiotic (group I) and patients administered with specific antibiotics (group II) clearly revealed that there is a significant decrease in days of hospitalization Group II patients. In this study of 140 patients , 40% i.e 56 patients in group II had postop hospital stay of less than 10days and only 21.4% i.e 30 patients in group I had postop hospital stay of less than 10days. The number of cases who are stayed for more than 20 days in group II is 2pts whereas in group I is 1 patient. This is due to increased mortality in group I where patients with severe postop complications could not survive more than 20days with empirical antibiotics , but group II patients with postop complications could survive with specific antibiotic therapy with favorable outcome .

No.of days of hospital stay	GROUP I	GROUP II	TOTAL
6—10	30	56	86
11—15	32	9	41
16—20	7	3	10
21—25	1	2	3
TOTAL	70	70	140

COMPARISON OF POSTOP DAYS OF HOSPITAL STAY:



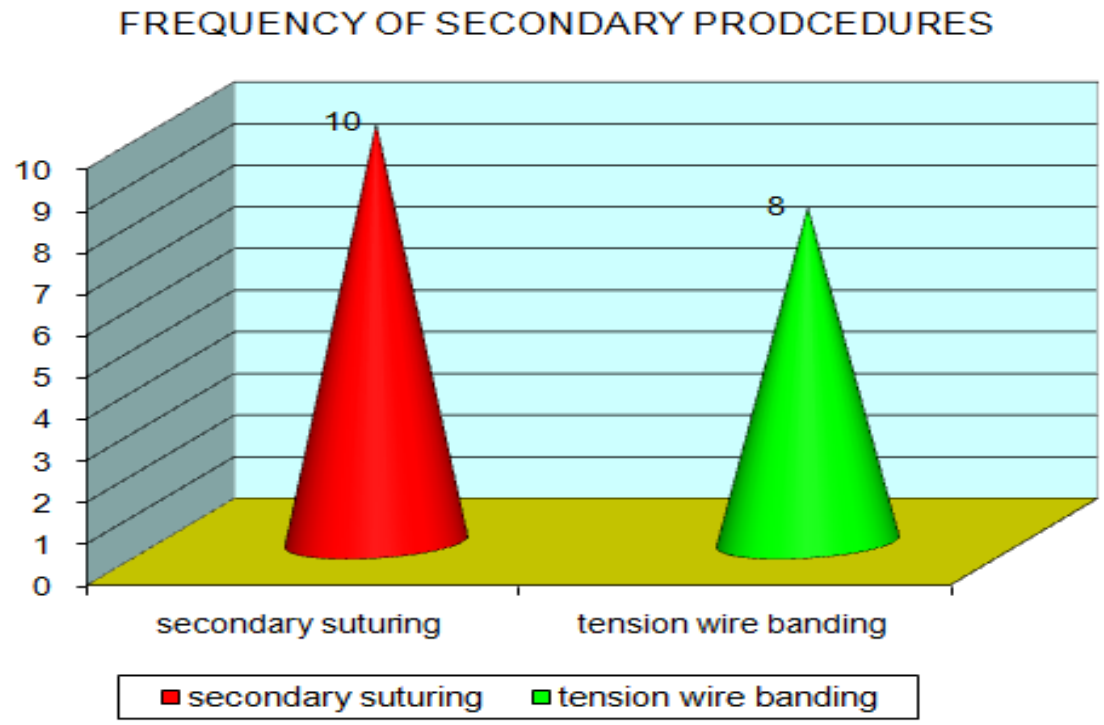
FREQUENCY OF POSTOP SECONDARY MINOR PROCEDURES:

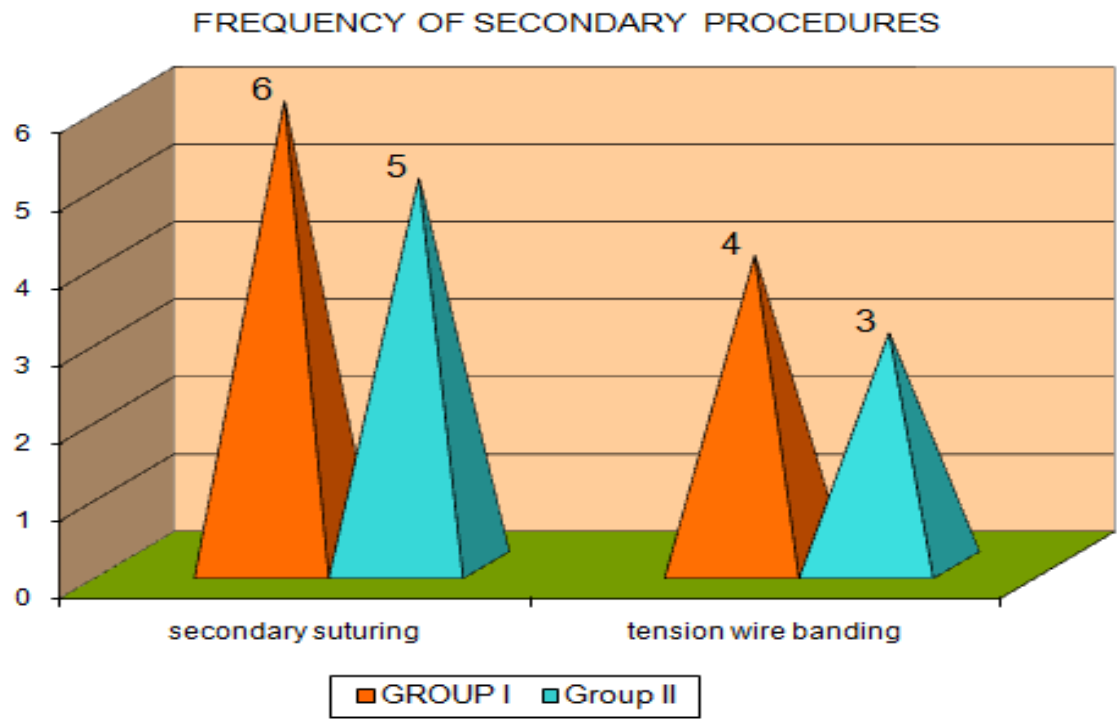
In this study , it is observed that frequency of secondary minor procedures like secondary suturing for wound gaping and tension wire banding for burst abdomen is slightly less in patients who were on specific antibiotics than who were on empirical antibiotics .

Secondary minor procedures	GROUP I	Group II
secondary suturing	6	5
tension wire banding	4	3
Total	10	8

	GROUP I		GROUP II	
	MALE	FEMALE	MALE	FEMALE
secondary suturing	6	0	4	1
tension wire banding	4	0	2	1
TOTAL	10	0	6	2

FREQUENCY OF POSTOP SECONDARY MINOR PROCEDURES :





DISCUSSION :

The study includes 140 patients with DU Perforation who fulfills eligibility criteria. Collected informations are consolidated and results are studied. Age distribution has a pyramidal pattern.

Peak age group with DU perforation lies in the interval between 41-50 yrs comprising of about 27% of total study population (38 patients out of 140 patients). Male prepondenrance is observed. 120 out of 140 patients belongs to male gender (85.7% of the study population are males).

The commonest organism observed in our study is E.coli constitutes 45% of study population (isolated in 63 out of 140 patients). Others include Klebsiella - 19%

Polymicrobial flora -16%, Pseudomonas - 7%, Streptococcus - 5%, Staphylococcus-2%,Other least frequently isolated organisms (3.5%) include Bacteroides fragilis, Citrobacter, candida etc,. One of the recent studies conducted by Punamiya AR, Chougule PG, Ahuja BR et. al., 100 patients with perforated peptic ulcer concluded that E.coli is the most common isolated organisms accounting to 44% . The other organisms constitute 3%, klebsiella 16%. My study results almost have got comparably consistent result with the other studies.

Piperacillin tazobactam and Cefotaxime are found to be the most common antibiotics that is most sensitive against bacteriological flora which are isolated in DU perforative peritonitis. 28.5% and 26.4% patients are found

to sensitive for piptaz and cefotaxime respectively. Other sensitive antibiotics are ceftriaxome, amikacin, gentamycin, ciprofloxacin, cefaperazone sulbactum, meropenam , imipenam. The study conducted by Punamiya AR, Chougule PG, Ahuja BR et. al. has got piperacillin tazobactam (51%) and cefotaxime (49%) as highly sensitive drugs in peritoneal fluid culture sensitivity. My study results are almost consistent with this study.

Postoperative complications are compared between group I who were on empirical antibiotics and group II who were on specific antibiotics. 39 pts in group I and 22 pts in group II had wound infection. 18 pts in group I and 14 pts in group II had wound gaping. 5 pts and 4 pts in group I & II resp. had burst abdomen. 9 pts and 5 pts in group I & II resp. had septicemia. 9 pts and 6 pts in group I & II resp. had lungs infection. 7 pts and 3 pts in group I & II resp expired due to postop complication. The p-value for this comparison is 0.03 which is significant. According to the study conducted by Punamiya AR, Chougule PG, Ahuja BR et. al., the postop complications in group of patients who were administered with specific antibiotic , were significantly less than in patients with empirical antibiotic therapy.

Out of 140 patients , 40% (56 patients in group II had postop hospital stay of less than 10days) and 21.4% (30 patients in group I had postop hospital stay of less than 10days). The p-value is 0.037 which is significant. According to Punamiya AR, Chougule PG, Ahuja BR et. al. 25.71% of patients with empirical antibiotic therapy and 63.33% of patients with specific antibiotic

therapy based on culture sensitivity had hospital stay of less than 10days. Also in that study it is stated that 61.43 % with empirical antibiotic treatment and 26.67% with specific antibiotic treatment had hospital stay of more than 10days. My study results also found to be consistent with the study conducted by Punamiya AR, Chougule PG, Ahuja BR et. al

CONCLUSION:

It is concluded that specific antibiotic administration according to intraoperative peritoneal toxic fluid culture and sensitivity report rather than empirical antibiotic administration, will significantly reduce the postoperative outcomes in terms of complications like wound infections, wound gaping, burst abdomen, septicemia, lung infections, mortalities, prolonged hospital stay, increased frequency of secondary minor procedures like secondary suturing and tension wire banding . Since few studies are conducted regarding this aspect, many research works needed to be intiated pertaining to the aspect of administration of specific antibiotic therapy, in different high volume tertiary institutions to validate the use of specific antibiotic therapy rather than using empirical therapy.

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PROFORMA

Name:- I. P. No:

Age:- Unit:

Sex:- D.O.A:

Occupation:- D.O.D:

Address:- D.O. surgery:

CHIEF COMPLAINTS:

1) abdominal pain
2) abdominal distention
3) fever
4) other complaints

HISTORY OF PRESENTING ILLNESS:

1) ABDOMINAL PAIN:

a. Duration

b. Time and mode of onset

c. Site of pain:

d. character of pain

e. radiation of pain

f. Aggrieviating factors

g.Reliving factors

2) ABDOMINAL DISTENSION:

a. Duration

b. associated features

3) FEVER:

PAST HISTORY

1) History of similar complains

2) Duration

3) Treatment taken

4) History of previous surgeries

5) History suggestive of Hypertension/ Diabetes/ Tuberculosis

6) History of drug abuse

PERSONAL HISTORY

Diet: Vegetarian/ Mixed

Habits: Smoking/ Alcohol/ Tobacco

Bowel & bladder habits

Sleep

FAMILY HISTORY

Marital status

Similar illness in other family members

GENERAL PHYSICAL EXAMINATION

1. General survey
2. Body build and nourishment
3. Appearance
4. Attitude: Restless/ Quiet
5. Dehydration: Mild/ Moderate/ Severe/ Nil
6. Anaemia/ Jaundice/ Clubbing/ Cyanosis/ Lymphadenopathy/ Pedal oedema
7. Pulse
8. Temperature
9. Respiratory rate
10. Blood pressure

PER ABDOMEN EXAMINATION

INSPECTION:

1. Distention
2. Position of umblicus
3. Abdominal wall movements with respiration
4. VGP/VIP

PALPATION:

1. Warmth and tenderness
2. Diffuse / localized guarding or rigidity
3. Organomely

4. Hernial orifice

PERCUSSION:

1. Obliteration of liver dullness
2. Free fluid

AUSCULTATION: Bowel sounds

PER RECTAL EXAMINATION:

1. Perianal skin
2. Sphincteric tone
3. Rectal mucosa
4. Rectal growth
5. Gloved finger staining with faecal material

OTHER SYSTEM EXAMINATION:

CVS- S1, S2 HEARD

RS- BAE + & NVBS +

CNS- NFND

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS

1. Blood: Hb % , TC, DC, BT, CT, ESR
2. Blood group and rh type
3. Urine: Albumin/ Sugar/ Microscopy
4. Chest x-ray

5. USG Abd and pelvis

6. HIV, HBs Ag, HCV

DIAGNOSIS

MANAGEMENT

Pre operative instructions

Type of Anaesthesia

Type of incision

Intraop finding

Post-operative instructions

Post-operative period

Post-operative complication and its management.

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y	Z	AA
1																											
2	sno	ipno	name	age	sex	paindur	distdur	drugabu	pulse	bpsys	bpdia	abdpain	abdpain1	bowsoun	bloodinv	xray	usg	peri cul	sensi ab	pohossta	wouinf	wougap	burabd	septi	mortal	lunginf	Procedures
3	71	1109210	Ponnaiyan	32	1	3	2	2	114	140	90	1	1	2	1	2	free fluid in pelvis with absent peristalsis	E.COLI	others	7							
4	72	1109203	Karuppaiah	42	1	2	2	2	110	110	60	1	1	2	1	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	9	present						
5	73	1122391	Gomathi	40	2	3	2	2	96	110	70	1	1	1	1	1	free fluid in pelvis with absent peristalsis	Polymicrobial	Piptaz	7							
6	74	57447	Manivel	40	1	4	2	2	94	120	80	1	2	2	1	1	peritonitis features with absent peristalsis	Streptococci	Ceftri, Ak	10	present	present					
7	75	1136631	pondivijayakumar	23	1	3	1	2	92	120	70	1	1	2	1	1	free fluid in hepatorenal pouch	Pseudomonas	Piptaz	8							
8	76	1136644	praveenraja	18	1	2	1	2	110	110	70	1	2	2	1	1	free fluid in hepatorenal pouch	E.COLI	Cefo, Ak	8							
9	77	1072762	basker	42	1	3	2	2	102	100	70	1	1	2	1	2	free fluid in pelvis with absent peristalsis	Polymicrobial	Ceftri, Ak	9							
10	78	1074220	Palanisamy	45	1	3	3	1	102	120	80	1	2	2	1	1	peritonitis features with absent peristalsis	Staphylococci	Cipro, Gm	12	present	present				present	secondary suturing
11	79	1073641	Perumal	70	1	2	2	1	110	110	80	1	2	2	1	1	free fluid in pelvis with absent peristalsis	Klebsiella	Cefo, Gm	10	present						
12	80	1078341	Muralimuthu	33	1	2	1	2	100	120	70	1	2	1	1	1	free fluid in hepatorenal pouch	E.COLI	cefap+sul	7							
13	81	1078372	Kumarimuthu	40	1	4	3	1	102	110	70	1	1	1	1	1	peritonitis features with absent peristalsis	E.COLI	Ceftri	9							
14	82	86746	Nondi	65	1	4	3	2	110	110	80	1	2	2	1	2	free fluid in hepatorenal pouch	Polymicrobial	Piptaz	7							
15	83	1086426	Periyalagan	51	1	4	3	1	102	110	70	1	2	2	1	1	peritonitis features with absent peristalsis	Streptococci	Cipro, Gm	10						present	
16	84	1088113	kalimuthu	60	1	3	2	1	98	130	80	1	2	2	1	1	free fluid in pelvis with absent peristalsis	Klebsiella	Cefo, Ak	8							
17	85	1088066	muthukumar	25	1	3	3	2	98	140	80	1	1	2	1	1	free fluid in hepatorenal pouch	E.COLI	Ceftri	7							
18	86	1080540	krishnamoorthy	60	1	3	3	2	92	110	70	1	2	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Piptaz	11	present	present					

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y	Z	AA
19	87	1092334	kalisamy	70	1	2	2	2	94	110	70	1	2	1	1	1	peritonitis features with absent peristalsis	Klebsiella	Cefo	18	present	present	present	present	expired		tension wire banding
20	88	1099165	pandi	45	1	2	2	2	88	100	70	1	2	1	1	1	free fluid in pelvis with absent peristalsis	E.COLI	cefap+sul	7							
21	89	1096218	chinnalan	23	1	2	2	2	100	100	60	1	2	2	2	2	peritonitis features with absent peristalsis	Pseudomonas	Cefo	13	present						
22	90	1100459	ravindran	50	1	3	3	2	102	100	60	1	2	2	2	2	free fluid in hepatorenal pouch	E.COLI	cefap+sul	8							
23	91	1097429	ramakrishnan	30	1	3	2	1	90	90	50	1	2	2	2	1	free fluid in pelvis with absent peristalsis	E.COLI	Ceftri, Ak	10							
24	92	1097480	karupaiyee	37	2	4	2	2	96	90	50	1	1	2	1	1	free fluid in hepatorenal pouch	Klebsiella	Piptaz	14	present	present					secondary suturing
25	93	1106179	radhakrishnan	60	1	3	2	1	110	100	60	1	1	1	1	1	peritonitis features with absent peristalsis	Polymicrobial	Cipro, Gm	7							
26	94	1158153	kaliyaperumal	50	1	3	2	2	110	110	60	1	2	2	1	1	free fluid in hepatorenal pouch	E.COLI	Piptaz	8							
27	95	1055941	silambayee	54	1	3	2	2	108	120	80	1	2	2	1	1	free fluid in pelvis with absent peristalsis	Others	cefap+sul	9						present	
28	96	1055852	parthasarathy	19	1	3	2	2	108	120	80	1	2	2	1	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	10	present	present					
29	97	1054301	prakash	24	1	2	2	2	110	110	70	1	1	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Cefo	8							
30	98	1050180	andi	44	1	2	1	2	118	120	70	1	2	2	1	1	peritonitis features with absent peristalsis	E.COLI	Ceftri, Gm	7							
31	99	1051286	sekaramoorthy	25	1	3	2	1	116	100	60	1	1	2	1	1	free fluid in hepatorenal pouch	Pseudomonas	Cefo, Ak	9							
32	100	43928	ramar	25	1	2	2	1	120	110	60	1	1	1	1	1	peritonitis features with absent peristalsis	Klebsiella	Cipro, Gm	16	present	present	present	present	expired		
33	101	41211	valli	40	2	2	1	1	120	110	70	1	1	2	1	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	10							
34	102	1086771	umadevi	41	2	3	2	2	114	120	80	1	1	1	1	1	free fluid in pelvis with absent peristalsis	Polymicrobial	Cefo	21	present	present	present	present			tension wire banding
35	103	1088492	jothimani	65	1	3	1	2	108	100	70	1	1	2	1	1	peritonitis features with absent peristalsis	E.COLI	Ceftri, Ak	9							
36	104	1091011	mayandi	23	1	3	2	2	98	90	60	1	2	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Cipro, Gm	7							

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y	Z	AA
37	105	1098451	ajithkumar	20	1	3	2	2	110	120	80	1	2	2	1	2	free fluid in hepatorenal pouch	E.COLI	Cefo	10	present						
38	106	48675	backiyan	58	1	3	2	2	110	150	90	1	1	2	1	1	peritonitis features with absent peristalsis	Polymicrobial	cefap+sul	8							
39	107	1106719	murugan	45	1	5	3	2	114	90	60	1	2	1	2	1	free fluid in pelvis with absent peristalsis	E.COLI	Ceftri, Ak	10							
40	108	1114062	chinnalangan	50	1	4	3	2	100	130	80	1	1	2	1	1	free fluid in hepatorenal pouch	Klebsiella	Cefo, Gm	7							
41	109	1115450	elango	40	1	4	3	2	90	110	70	1	2	2	1	1	free fluid in hepatorenal pouch	E.COLI	Piptaz	9							
42	110	1119903	kesavan	65	1	4	3	1	90	90	60	1	1	2	1	2	peritonitis features with absent peristalsis	Polymicrobial	Piptaz	15	present	present				present	secondary suturing
43	111	1125525	mani	38	1	3	3	1	100	90	50	1	2	2	2	1	peritonitis features with absent peristalsis	Streptococci	Cefo	9							
44	112	1132325	rangasamy	65	1	4	2	1	100	100	70	1	2	2	1	2	free fluid in pelvis with absent peristalsis	Klebsiella	Ceftri	8							
45	113	1132373	muthukarupu	15	1	3	3	2	108	110	70	1	2	2	1	1	free fluid in hepatorenal pouch	Pseudomonas	Piptaz	10	present					present	
46	114	1133685	raman	60	1	2	2	2	118	120	80	1	1	2	1	1	peritonitis features with absent peristalsis	Polymicrobial	Piptaz	7							
47	115	57884	chinnaiah	35	1	3	1	2	126	130	70	1	1	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Cipro, Gm	10							
48	116	1060482	ammaponnu	60	2	3	1	2	126	120	70	1	2	2	1	1	free fluid in hepatorenal pouch	Klebsiella	Cefo	9							
49	117	1097431	marimuthu	31	1	2	2	1	110	100	70	1	1	2	1	2	free fluid in pelvis with absent peristalsis	E.COLI	Ceftri, Gm	13	present	present					secondary suturing
50	118	1092985	ravichandran	41	1	3	2	1	98	90	60	1	1	2	1	1	peritonitis features with absent peristalsis	Others	Piptaz	9							
51	119	1090308	kumeravel	68	1	2	1	1	96	110	80	1	1	1	1	1	free fluid in hepatorenal pouch	Klebsiella	Cefo, Ak	10							
52	120	1088401	selvaraj	45	1	3	2	2	102	90	50	1	2	2	2	1	free fluid in pelvis with absent peristalsis	E.COLI	cefap+sul	8							
53	121	1088291	pandi	37	1	3	2	2	108	100	60	1	1	2	2	2	free fluid in hepatorenal pouch	Pseudomonas	Piptaz	10	present						
54	122	41031	rajendran	55	1	3	2	2	108	130	80	1	2	2	1	1	peritonitis features with absent peristalsis	Polymicrobial	Cefo	9							

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y	Z	AA
55	123	1051725	andi	50	1	4	2	1	94	140	80	1	1	2	1	1	free fluid in pelvis with absent peristalsis	Klebsiella	Ceftri, Ak	9							
56	124	1055048	murugan	45	1	4	1	2	124	120	80	1	2	2	1	1	peritonitis features with absent peristalsis	Streptococci	Piptaz	7							
57	125	1058053	kaliyamoorthy	50	1	3	2	2	114	110	60	1	2	2	1	1	free fluid in hepatorenal pouch	Klebsiella	Cefo	13	present	present					
58	126	1060514	poomari	30	2	4	2	2	118	120	70	1	1	2	1	2	peritonitis features with absent peristalsis	E.COLI	Piptaz	13	present					present	
59	127	45692	muruganatham	43	1	3	2	2	122	110	60	1	1	2	1	1	free fluid in pelvis with absent peristalsis	Streptococci	Cipro, Gm	21	present	present	present	present			tension wire banding
60	128	1084124	andiyapan	75	1	4	3	2	102	90	50	1	2	2	1	1	free fluid in hepatorenal pouch	E.COLI	Cipro, Gm	13	present	present					secondary suturing
61	129	1085594	arunkumar	18	1	3	2	2	98	100	70	1	1	2	2	1	free fluid in pelvis with absent peristalsis	E.COLI	cefap+sul	7							
62	130	1087136	ramasamy	64	1	3	2	2	96	100	70	1	2	2	1	2	peritonitis features with absent peristalsis	Klebsiella	Piptaz	10							
63	131	1095646	vellathaiyan	70	1	2	2	1	112	100	70	1	1	1	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Cefo, Ak	8							
64	132	1099682	krishnammal	60	2	3	2	2	124	100	60	1	2	2	1	1	free fluid in hepatorenal pouch	E.COLI	Piptaz	10							
65	133	1104247	gurusamy	54	1	4	3	1	108	90	50	1	1	2	2	1	peritonitis features with absent peristalsis	Klebsiella	Piptaz	9							
66	134	1106946	kannan	35	1	3	2	1	108	90	60	1	1	2	1	2	free fluid in pelvis with absent peristalsis	Polymicrobial	Cefo	8							
67	135	1108426	pitchaimuthu	50	1	4	2	2	108	100	60	1	2	2	1	1	peritonitis features with absent peristalsis	E.COLI	Ceftri, Ak	16	present	present		present	expired		
68	136	1109923	thiruvengalam	58	1	2	1	2	116	110	70	1	1	1	1	1	free fluid in hepatorenal pouch	Pseudomonas	Cefo	10	present						
69	137	1112817	chitra	40	2	2	1	2	126	100	60	1	2	2	2	1	peritonitis features with absent peristalsis	Klebsiella	Ceftri, Ak	7							
70	138	1114294	vikramapandi	58	1	3	2	2	126	100	60	1	1	2	1	1	free fluid in pelvis with absent peristalsis	Polymicrobial	Piptaz	9							
71	139	1140158	haribaran	16	1	3	2	2	126	100	70	1	2	2	1	1	free fluid in hepatorenal pouch	Klebsiella	Piptaz	10							
72	140	1102396	jeyaganesh	15	1	4	3	2	104	100	60	1	1	2	1	1	peritonitis features with absent peristalsis	E.COLI	Cefo, Gm	8							

sno	ipno	name	age	sex	paindur	distdur	drugabu	pulse	bpsys	bpdia	abdpain	abdpain1	bowsoun	bloodinv	xray	usg	peri cul	sensi ab	pohossta	wouinf	wougap	burabd	septi	mortal	lunginf	Procedures
1	53674	Bachiaraj	29	1	3	1	2	98	110	80	1	2	2	1	1	free fluid in pelvis	E.COLI	Cefo, Ak	11	present	absent	absent	absent	nil	absent	
2	58624	raju	70	1	2	2	1	110	110	60	1	1	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Piptaz	12	present	present					
3	13342	kuppu	45	1	3	1	1	110	140	90	1	2	2	1	1	free fluid in hepatorenal pouch	Klebsiella	Cefo	10	present						
4	12747	palanisamy	50	1	5	2	2	102	130	90	1	1	2	1	1	peritonitis features with absent peristalsis	E.COLI	others	11	present						
5	12174	vanishree	30	2	2	2	2	120	120	70	1	2	1	1	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	9	present						
6	12196	ravinathan	32	1	4	3	2	98	100	60	1	2	2	1	2	free fluid in pelvis with absent peristalsis	E.COLI	Ceftri	11	present	present					
7	19947	selvaraj	39	1	3	2	2	92	110	70	1	2	2	1	1	peritonitis features with absent peristalsis	polymicrobial	Cipro, Gm	10						present	
8	22558	chellamani	40	2	3	1	1	108	100	60	1	1	2	1	1	free fluid in hepatorenal pouch	E.COLI	Cefo	10							
9	23952	baskaran	45	1	4	2	1	110	120	70	1	2	1	1	1	peritonitis features with absent peristalsis	Klebsiella	Ceftri	15	present	present	present	present	expired		tension wire banding
10	29630	ananthamuru	16	1	4	2	2	126	130	80	1	2	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	cefap+sul	13							
11	49697	ramamoorthy	77	1	3	2	1	120	120	80	1	1	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Ceftri, Ak	12							
12	62681	srilangam	48	1	3	2	2	98	110	60	1	1	2	1	1	peritonitis features with absent peristalsis	Klebsiella	Cefo	9							
13	67701	arumugam	42	1	3	3	2	92	120	70	1	2	2	1	1	peritonitis features with absent peristalsis	Pseudomonas	Cipro, Gm	21	present	present	present				tension wire banding
14	64021	manikammal	62	2	4	3	2	110	100	60	1	2	2	2	1	free fluid in hepatorenal pouch	Streptococci	Ceftri, Gm	9							

15	65747	sadaiyandi	65	1	3	2	1	126	120	60	1	2	1	2	1	free fluid in pelvis with absent peristalsis	Others	Cefo, Ak	10							
16	16194	ramanathan	33	1	3	3	2	106	120	70	1	1	2	2	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	10							
17	17824	Murali	40	1	3	2	2	108	110	60	1	2	2	1	2	free fluid in hepatorenal pouch	polymicrobial	Cipro, Gm	12	present			present	expired		
18	41837	chinnakaalai	80	1	2	1	1	110	100	50	1	1	1	1	1	peritonitis features with absent peristalsis	polymicrobial	Cefo	14	present	present					secondary suturing
19	43819	aavudaiyappa	60	1	3	2	2	114	140	80	1	2	2	2	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	12							
20	43877	ishmayil	40	1	2	1	2	106	100	60	1	1	2	2	1	free fluid in hepatorenal pouch	Staphylococci	cefap+sul	12	present						
21	45846	ganeshkumar	20	1	2	2	2	106	120	60	1	1	1	1	1	free fluid in hepatorenal pouch	Pseudomonas	Cefo, Ak	10							
22	51174	karpurapandi	74	1	4	2	1	110	100	60	1	2	2	1	1	peritonitis features with absent peristalsis	E.COLI	Ceftri	13	present	present				present	secondary suturing
23	52850	meena	30	1	4	3	1	98	120	70	1	2	2	1	1	peritonitis features with absent peristalsis	Klebsiella	Piptaz	9	present						
24	54824	andichi	40	1	3	2	1	96	130	80	1	1	2	1	2	free fluid in pelvis with absent peristalsis	E.COLI	Cefo	8							
25	62715	ravikumar	36	1	3	1	2	98	140	90	1	1	2	2	1	free fluid in pelvis with absent peristalsis	E.COLI	Cipro, Gm	13	present	present		present	expired		
26	68005	madeshwaran	40	1	3	2	2	110	100	60	1	2	2	2	1	free fluid in hepatorenal pouch	E.COLI	Cefo, Ak	10	present	present					
27	78935	machairajan	19	1	3	2	2	110	100	70	1	1	2	1	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	12	present	present					
28	######	chellaiah	40	1	3	2	2	120	100	50	1	1	2	1	1	peritonitis features with absent peristalsis	Klebsiella	Ceftri	8							
29	43787	chinnaian	60	1	2	1	2	120	120	80	1	2	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Cefo, Ak	10							
30	######	vellaisamy	45	1	2	2	2	120	120	70	1	1	2	1	1	free fluid in hepatorenal pouch	polymicrobial	others	14	present						

31	48041	ponnusamy	53	1	2	1	2	110	110	60	1	2	2	1	1	peritonitis features with absent peristalsis	Staphylococci	Ceftri	16	present						
32	41031	rajendran	38	1	4	3	2	100	150	90	1	1	2	1	2	free fluid in hepatorenal pouch	E.COLI	Piptaz	19	present	present	present	present		present	tension wire banding
33	40341	nagarajan	22	1	2	2	1	100	140	80	1	2	1	1	2	peritonitis features with absent peristalsis	E.COLI	Cefo, Ak	14	present						
34	35390	kalasammal	56	2	3	1	2	110	140	90	1	2	2	1	1	peritonitis features with absent peristalsis	E.COLI	Cipro, Gm	10							
35	38544	sivakumar	45	1	4	2	2	100	120	60	1	2	2	1	1	free fluid in pelvis with absent peristalsis	Klebsiella	Cefo, Gm	9							
36	35390	velusamy	28	1	3	2	1	98	120	70	1	1	2	1	1	free fluid in hepatorenal pouch	polymicrobial	Piptaz	13	present						
37	62634	subbaiah	67	1	2	1	1	110	110	70	1	1	2	2	1	peritonitis features with absent peristalsis	E.COLI	Ceftri	12	present						
38	12738	balamurugan	23	1	3	2	2	140	100	50	1	2	1	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Cipro, Gm	10							
39	19080	prithivraj	19	1	3	2	2	98	110	70	1	2	1	1	1	free fluid in hepatorenal pouch	polymicrobial	cefap+sul	14						present	
40	19065	saravanan	20	1	2	2	1	94	130	80	1	1	2	1	1	free fluid in pelvis with absent peristalsis	Klebsiella	Cefo, Ak	11							
41	17796	dharmar	53	1	2	1	2	120	130	80	1	2	2	1	1	peritonitis features with absent peristalsis	E.COLI	Ceftri	9							
42	14837	duraipandi	52	1	4	3	2	110	120	70	1	2	2	1	2	free fluid in hepatorenal pouch	E.COLI	Cipro, Gm	12	present			present	expired		
43	10330	murugan	50	1	4	2	1	120	100	50	1	1	2	2	1	free fluid in hepatorenal pouch	E.COLI	Piptaz	11							
44	13020	kumar	41	1	3	2	1	120	110	60	1	1	2	2	1	peritonitis features with absent peristalsis	Klebsiella	Piptaz	12							
45	11234	gurusamy	67	1	2	1	2	110	100	70	1	2	2	1	1	peritonitis features with absent peristalsis	polymicrobial	Ceftri, Ak	20	present	present				present	secondary suturing
46	10844	mookammal	48	2	2	1	2	120	100	60	1	1	2	1	1	free fluid in pelvis with absent peristalsis	Streptococci	Piptaz	15	present	present					

47	13284	kumaran	32	1	3	2	2	110	110	70	1	2	2	1	1	peritonitis features with absent peristalsis	polymicrobial	Cefo	12	present					present	
48	18846	sriniasan	44	1	3	2	2	130	110	70	1	1	2	1	1	peritonitis features with absent peristalsis	E.COLI	others	18	present						
49	19035	rajammal	54	2	3	2	1	120	100	70	1	2	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Cefo, Ak	9	present						
50	16943	perumakkal	67	2	4	3	2	114	140	80	1	2	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	cefap+sul	12	present	present				present	
51	16078	vetri	24	1	3	2	1	116	100	70	1	1	2	1	1	free fluid in hepatorenal pouch	E.COLI	Piptaz	9							
52	15684	shakulhameed	45	1	4	3	2	102	90	50	1	2	2	1	1	peritonitis features with absent peristalsis	E.COLI	Ceftri	10							
53	15397	srirangam	69	1	3	2	2	100	100	60	1	1	2	1	1	free fluid in pelvis with absent peristalsis	Klebsiella	Piptaz	13	present	present					secondary suturing
54	14673	lakshman	46	1	3	3	2	120	110	70	1	1	2	1	1	peritonitis features with absent peristalsis	Others	Cipro, Gm	15	present	present		present	expired		
55	12136	backiyan	64	1	2	2	2	110	120	80	1	2	2	1	1	free fluid in hepatorenal pouch	E.COLI	cefap+sul	9							
56	17734	mukundan	47	1	2	1	1	110	110	70	1	1	2	2	1	free fluid in pelvis with absent peristalsis	polymicrobial	Piptaz	10							
57	16773	thangapandi	47	1	2	1	2	98	100	50	1	1	2	1	1	peritonitis features with absent peristalsis	Pseudomonas	Cipro, Gm	9							
58	18712	velusamy	38	1	3	2	2	96	90	60	1	2	2	1	2	free fluid in hepatorenal pouch	Klebsiella	Piptaz	8							
59	54731	jahirhussain	43	1	3	2	2	88	110	70	1	2	1	1	1	free fluid in pelvis with absent peristalsis	polymicrobial	Cefo, Ak	14	present	present					secondary suturing
60	45821	mani	38	1	4	3	2	95	120	80	1	2	2	1	2	peritonitis features with absent peristalsis	Others	Ceftri, Gm	16	present		present	present	expired		
61	56317	kodi	29	1	3	3	2	92	130	80	1	1	2	1	1	free fluid in pelvis with absent peristalsis	Streptococci	Cefo	20	present		present	present			tension wire banding
62	43576	jesuraj	45	1	2	2	2	102	120	80	1	2	2	1	1	free fluid in hepatorenal pouch	E.COLI	Piptaz	9							

63	10892	laksmi	43	2	3	2	2	102	110	70	1	1	2	1	1	free fluid in hepatorenal pouch	Klebsiella	Cipro, Gm	8							
64	12199	rajeswari	35	2	4	3	2	110	110	70	1	2	1	1	1	free fluid in pelvis with absent peristalsis	Staphylococci	cefap+sul	9							
65	45117	kaali	45	2	3	3	2	126	100	60	1	1	2	1	1	peritonitis features with absent peristalsis	polymicrobial	Piptaz	10	present						
66	51128	rajagopal	41	1	4	2	1	110	110	70	1	2	2	2	2	free fluid in pelvis with absent peristalsis	Klebsiella	Ceftri, Ak	15	present	present				present	secondary suturing
67	43178	ganga	45	2	3	1	1	106	90	60	1	2	1	2	1	peritonitis features with absent peristalsis	E.COLI	Piptaz	12	present						
68	12119	venu	61	1	3	2	2	108	100	60	1	2	2	1	1	free fluid in hepatorenal pouch	Pseudomonas	Cefo	18	present	present		present	expired		
69	54129	jenarthan	29	1	2	2	2	110	100	60	1	2	2	1	1	free fluid in hepatorenal pouch	Polymicrobial	cefap+sul	10							
70	13789	kaaliammal	36	2	4	2	2	120	130	80	1	2	2	1	1	free fluid in pelvis with absent peristalsis	E.COLI	Ceftri, Ak	11	present					present	

MADURAI MEDICAL COLLEGE
MADURAI, TAMILNADU, INDIA -625 020
(Affiliated to The Tamilnadu Dr.MGR Medical University, Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.R.Tennison
Course	:	PG in MS., General Surgery
Period of Study	:	2014-2017
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Prospective study to identify commonest organisms and antibiotic sensitivity in peritonitis due to duodenal ulcer perforation in Govt. Rajaji Hospital
Ethical Committee as on	:	27.07.2016

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

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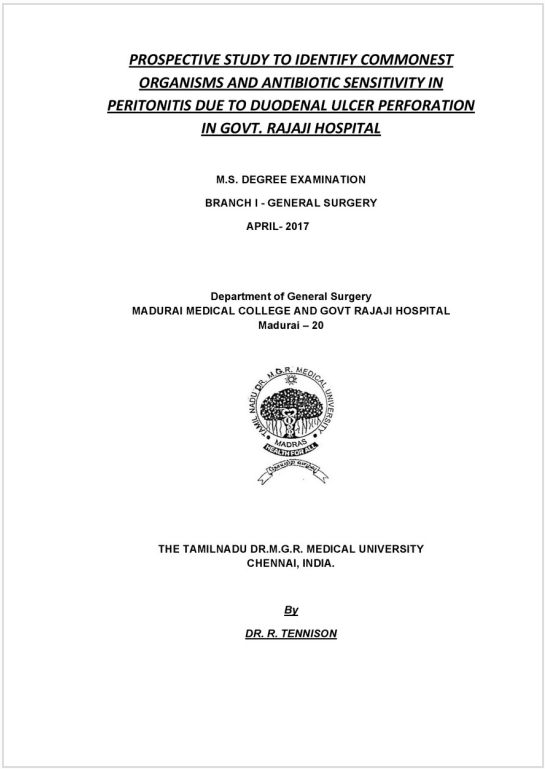

PROSPECTIVE STUDY TO IDENTIFY COMMONEST ORGANISMS AND ANTIBIOTIC SENSITIVITY IN PERITONITIS DUE TO DUODENAL ULCER PERFORATION IN GOVT. RAJAJI HOSPITAL

M.S. DEGREE EXAMINATION

BRANCH I - GENERAL SURGERY

APRIL- 2017

Department of General Surgery
MADURAI MEDICAL COLLEGE AND GOVT RAJAJI HOSPITAL
Madurai – 20

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
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By

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PROSPECTIVE STUDY TO IDENTIFY COMMONEST ORGANISMS AND ANTIBIOTIC SENSITIVITY IN PERITONITIS DUE TO DUODENAL ULCER PERFORATION IN GOVT. RAJAJI HOSPITAL

M.S. DEGREE EXAMINATION

BRANCH I - GENERAL SURGERY

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