"STUDY OF OUTCOME OF PATIENTS WITH PERFORATIVE PERITONITIS FOLLOWING ANTIMICROBIAL TREATMENT BASED ON CULTURE AND ANTIBIOTIC SENTIVITY REPORT OF PERITONEAL FLUID"

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations for the award of the degree of

M.S. GENERAL SURGERY – BRANCH I

DEPARTMENT OF GENERAL SURGERY

REGISTER NUMBER : 221711301

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

MAY 2020

CERTIFICATE

Certified that this is the bonafide dissertation done by **DR.ALLWYN SUDHAGAR.M** submitted in partial fulfillment of the requirement for the Degree of M.S. General Surgery, Branch I of the Tamilnadu Dr. M.G.R. Medical University, Chennai.

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DATE: **DEAN**

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INSTITUTIONAL HUMAN ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To
Dr Allwyn Sudhagar
Post Graduate,
Department of General Surgery,
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Coimbatore -18.

Dear **Dr Allwyn Sudhagar**

The Institutional Ethics Committee of Coimbatore Medical College, reviewed and discussed your application for approval of the proposal entitled **"Study of outcome of patients with perforative peritonitis following Antimicrobial Treatment Based on culture and Antibiotic Sensitivity report of peritoneal fluid."**No.0116/2017.

The following members of Ethics Committee were present in the meeting held on 30.11.2017.conducted at MM - II Seminar Hall, Coimbatore Medical College Hospital Coimbatore-18

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15	Mr.V.A.Shahul Hameed, +2	Lay-Person

We approve the Proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

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DECLARATION

I solemnly declare that the dissertation titled "**STUDY OF OUTCOME OF PATIENTS WITH PERFORATIVE PERITONITIS FOLLOWING ANTIMICROBIAL TREATMENT BASED ON CULTURE AND ANTIBIOTIC SENTIVITY REPORT OF PERITONEAL FLUID "** was done by me from 2018 onwards under the guidance and supervision of **DR. V. LEKSHMINARAYANI , M.S , D.G.O.**

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of the requirement for the award of M.S Degree in General Surgery (Branch I).

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DATE: **REGISTER NO : 221711301**

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Lastly I am grateful to all the patients whose cooperation made this work possible.

DATE: **SIGNATURE OF THE CANDIDATE**

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TABLE OF CONTENTS

ABBREVIATIONS

BT	:	Bleeding Time
CXR	:	Chest X-ray
CT	:	Clotting Time
CVS	:	Cardiovascular System
DM	:	Diabetes Mellitus
GIT	:	Gastrointestinal Tract
Hb	:	Hemoglobin
IV	:	Intra Venous
LFT	:	Liver Function Test
MRSA	:	Methicillin Resistance Staphylococcus Aureus
MIC	**:**	Minimum inhibitory concentration
PA	:	Per Abdomen
RS	:	Respiratory System
SSI	:	Surgical Site Infection
SPO2	:	Partial Pressure of Oxygen

TC	:	Total Count
UTI	:	Urinary Tract Infection
USG	:	Ultrasound
URTI	:	Upper Respiratory Tract Infection

INTRODUCTION

"Surgical peritonitis remains one of the most common problems faced by surgeon". Whether it is a simple duodenal perforation, traumatic perforation, appendicular perforation or a case of acute pancreatitis complicated by pancreatic abscess, it still remains as a major cause of morbidity and mortality. Peritonitis commonly that we receive is secondary peritonitis occurring due to hollow viscus perforation. The surgeons treating it know the dreadful and fatal complication; the problems can be minor wound infection to septic shock or SIRS (SYSTEMIC INFLAMMATORY RESPONSE SYNDROME).

There are various obstacles for treatment of peritonitis they include

- The age of patient
- Time interval of presentation

General condition and nutritional status of patients

- Presence of any malignancy
- Post operative complications

The other fact which makes peritonitis more dangerous is due to very high amount of contamination of peritoneal cavity by certain fatal organisms like

E.coli, Klebsiella, Proteus, enterococci species which can lead to SIRS. Current therapy towards the treatment of peritonitis is directed towards correction of underlying cause, administration of systemic antibiotics and facilitating supportive measures to prevent SIRS.

With antibiotic administration it was found that if the therapy was directed towards aerobes there was less mortality and more residual abscess formation but when therapy was directed towards anaerobes there was less abscess formation and mortality remained unchanged. Therefore therapy was considered optimal, when combination was used. In this study, various organisms that are growing in the peritoneal fluid culture of the patients presenting with perforative peritonitis and their antibiotic sensitivity and resistance pattern were analyzed, so that we can initiate early and appropriate antibiotic therapy to patients presenting with perforative peritonitis preoperatively which can improve the outcome of the patient.

AIM AND OBJECTIVES

AIM:

To analyze bacteriological and its sensitivity patterns in peritoneal fluid in case of perforative peritonitis admitting in CMCH to select appropriate empirical antibiotic therapy.

OBJECTIVES:

1) To analyze bacteriological and its sensitivity patterns in peritoneal fluid in case of perforative peritonitis admitting in CMCH to select appropriate empirical antibiotic therapy.

2) To study bacteriological patterns in peritoneal fluid by culture.

3) To determine antibiotic sensitivity and resistance pattern for commonly used antibiotics to organisms grown in culture.

MATERIALS AND METHODS

Design of Study: prospective study

Place of Study: Coimbatore Medical College and ospital

Study Period: Jan 2018 to Jan 2019

Study Population: Patients presenting to Coimbatore Medical College and hospital with perforation peritonitis.

Sample size: 50

Inclusion criteria:

1) Patient presenting with features of perforation peritonitis and confirmed by x ray

2) Age more than 18 yrs

Exclusion criteria:

1) Patient presenting with primary peritonitis

2) Peritonitis due to penetrating trauma

REVIEW OF LITERATURE

Perforative peritonitis is one of the most common conditions encountered in an emergency department by surgeons all over the world especially in developing countries. There is a wide range of etiology for perforation ranging from simple duodenal perforation, traumatic perforation and perforated appendix to pancreatic abscess complicating acute pancreatitis. Whatever may be the cause, perforation peritonitis still remains a major cause for morbidity and mortality.

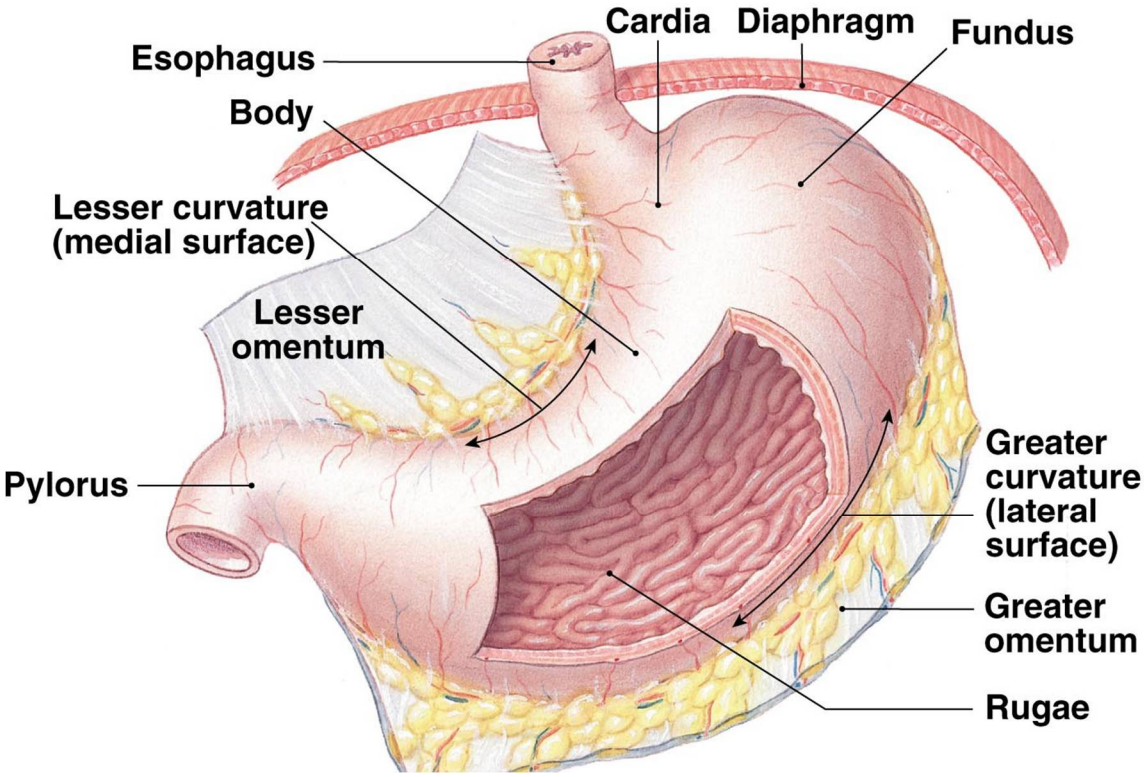
DEFINITIONS:

Perforation can be defined as a sudden breach in the gut wall which leads to leakage of bowel contents. **Peritonitis** is usually defined as the inflammatory reaction to serous layer lining the abdominal wall and cavity. It occurs due to a local cause or a generalized cause. **Primary peritonitis** is usually caused by ascites due to cirrhosis and heart failure without any gastrointestinal perforation. **Secondary peritonitis** is one resulting after perforation of gastrointestinal lining. The usual causes are peptic ulcer disease, colonic diverticulitis, acute appendicitis and pelvic inflammatory disease. Peritonitis of primary origin is usually uncommon.

STOMACH - ANATOMY

It is a Muscular sac-like organ, it aids in Chemical and physical digestion, forms chyme, Stores food, releases small amounts to small intestine. It takes 2-6 hours for stomach to empty. The inner surface is lined with gastric rugae.

Stomach is divided into 3 regions: fundus, body, and antrum (pylorus).



Stomach Mucosal Cells

Gastric glands (small folds in mucosa) contain specialized secretory cells

parietal cells secrete hydrochloric acid ,**goblet cells** secrete mucus which act as *Gastric Mucosal Barrier* protects stomach epithelium ,**chief cells** – pepsinogen which Digests protein, **endocrine cells**, ECL cells – **histamine**, G-cells – **gastrin** & **Intrinsic factor** secreting-cells

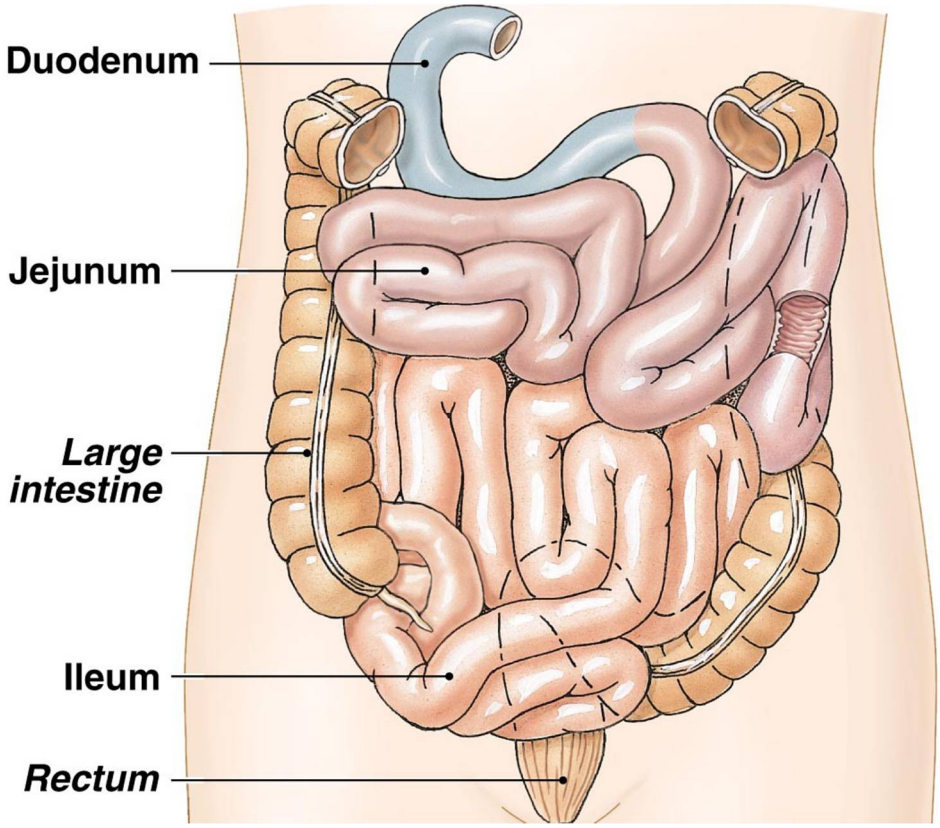
SMALL INTESTINE - ANATOMY

It connects stomach to large intestine; 15-20' long; 1" diameter; held together in abdominal cavity by "mesentery proper". It is the site for completion of chemical digestion & absorption of nutrients. It comprises of three regions:

Duodenum – 10" in length; receives chyme from stomach, secretions from liver, gallbladder & pancreas

Jejunum – 8' long; most digestion & absorption occurs here

Ileum – 12' long; connects to caecum of large intestine at iliocecal valve (sphincter)

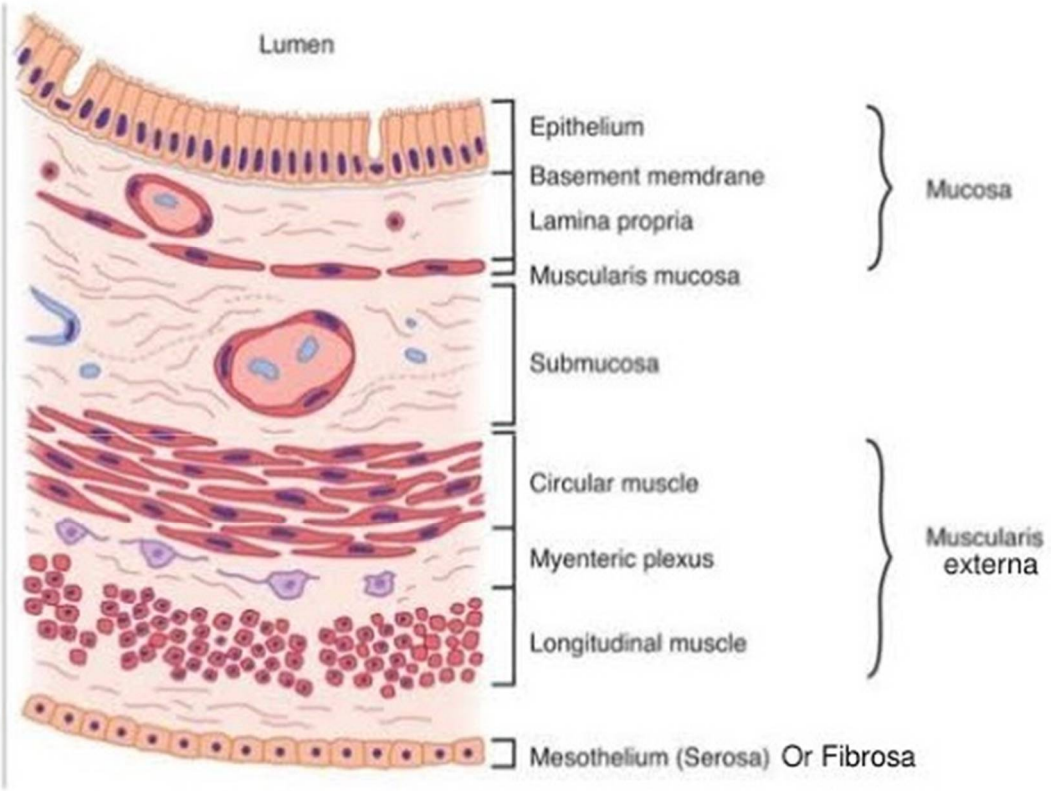


Modifications in mucosa & submucosa of intestinal wall designed to increase functional surface area:

Plicae circulares (circular folds) – large transverse ridges; most abundant in

Jejunum

Villi – small finger-like projections of mucosal folds across surface of intestine



GASTROINTESTINAL MOTILITY

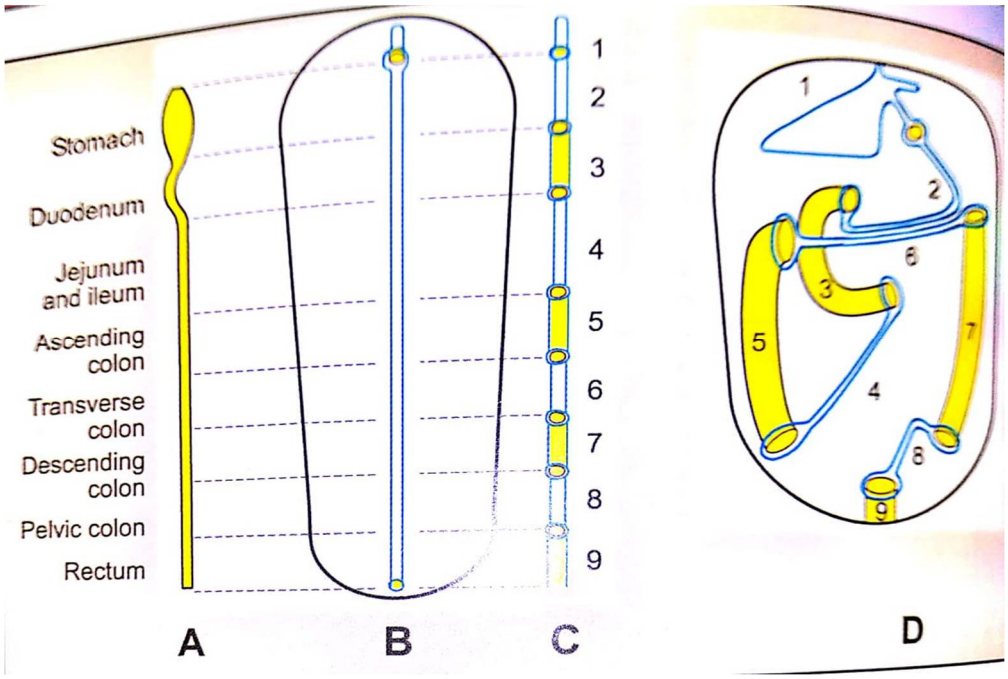
There are two modes of motility patterns in the stomach and consequently in the small intestine. The digestive (fed) pattern consists of continuous motor activity, characterized by a constant emptying of chyme from the stomach into the duodenum. The interdigestive (fasted) pattern (commonly called the migrating motor complex, MMC) is organized into alternating cycles of activity.

Typically, the MMC sequence begins in the stomach or esophagus and migrates to the distal ileum. Some MMC, however, originates in the duodenum or jejunum and not all MMC.

ANATOMY OF PERITONEUM:

The word peritoneum is derived from the Greek word peritonaion which means to stretch around. It is continuous membrane which is serous in nature lines the abdominal cavity and abdominal organs. It also acts a supportive structure for the abdominal organs and provides a passage for blood vessels and lymphatics to supply the organs. It is divided into parietal & visceral portions.

Embryologically, the peritoneal cavity is formed from intra-embryonic coelom, which has two limbs; it is a horseshoe shaped structure. The two limbs are separate at first, gradually fuses to form one single cavity which happens because of the lateral folding of the embryonic disc. The peritoneal cavity eventually gets subdivided into a number of pockets that are separated by the folds of peritoneum.

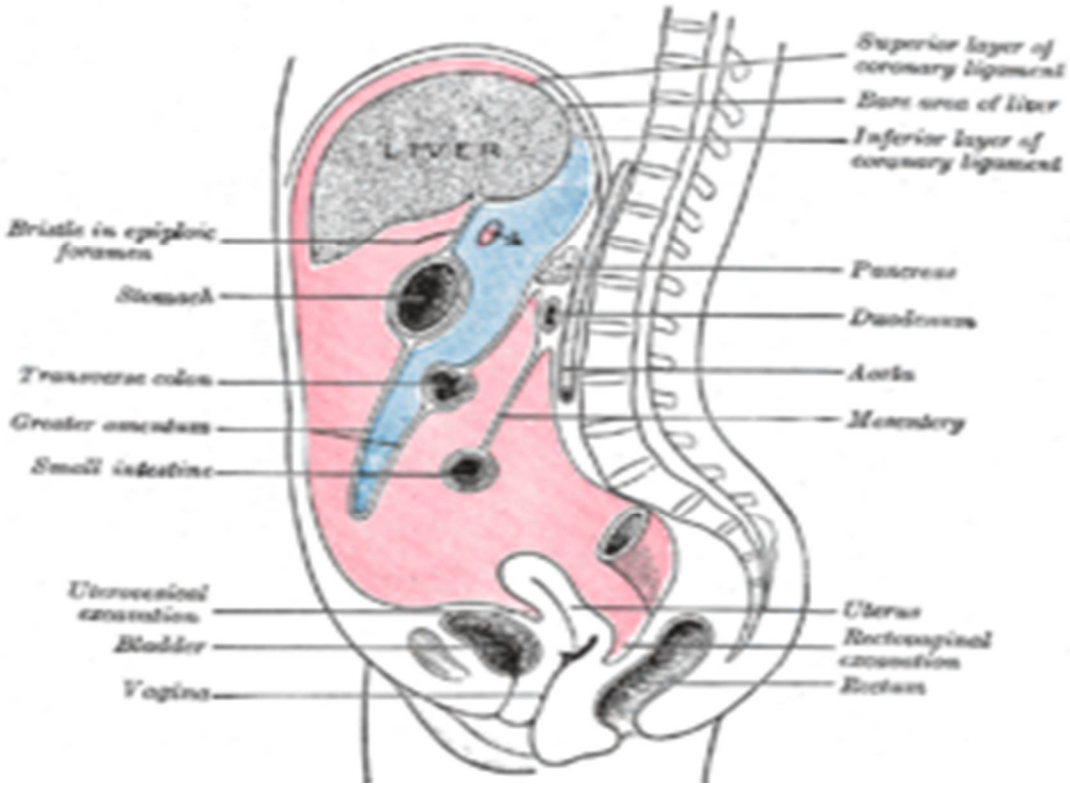


Histologically, the peritoneum has an outer fibrous layer, which provides strength the peritoneal membrane, and an inner layer composed of mesothelial cells which are involved in the process of secretion of peritoneal fluid.

The peritoneal fluid helps in lubrication and allows for ease of movements of the viscera. Parietal peritoneum is one which lines the inner surface of abdominal and pelvic walls and also the under surface of diaphragm. It is derived from the somatopleuric membrane of the embryo. It has somatic nerve innervations and so it is pain sensitive. Visceral peritoneum is one which lines the surface of viscera and is firmly attached to it. Embryologically it is the splanchopleuric membrane of the lateral plate mesoderm. It has autonomic innervations and pain is felt when the underlying viscera stretches or become ischemic. Between these two layers of peritoneum, there is a thin film of serous fluid named an s peritoneal fluid which is secreted by the mesothelial cells of the inner layer of peritoneum.

The peritoneal cavity has a surface area of approximately 1-2 m^2 .The major difference between the peritoneal cavity of a male and female is that, in females, the peritoneal cavity communicates with the exterior through the uterine tubes whereas in males it is a closed cavity.

The peritoneal fluid drains mainly through lymphatics, most importantly the sub diaphragmatic lymphatic system. This lymphatics also helps in the removal of macromolecules and other foreign substances from the peritoneal cavity.



SPREAD OF FLUID IN PERITONEAL CAVITY

The peritoneal cavity is subdivided into separate compartments by the abdominal viscera. The transverse colon and the drape of greater omentum divide the abdomen horizontally into supracolic and infracolic compartments. Therefore the symptoms and signs of peritonitis may be localized to upper or lower halves of the abdomen. The forward convexity of lumbar spine provides two marked lateral gutters and only a shallow anterior communication between them across the midline. Consequently, the liquid spreads by movement largely around the periphery of the abdomen not across the midline.

The right subhepatic space is open only to the right, where it communicates with the right paracolic gutter. Liquid from a perforated duodenal ulcer or seepage from the gallbladder region passes to the right and then both upwards to reach the subphrenic space and downwards to the right iliac fossa, thus there is shoulder tip pain.

VASCULAR SUPPLY OF PERITONEUM

The blood supply of abdominal parietal peritoneum is from branches of abdominal wall, pelvic parietal peritoneum from pelvic wall, visceral peritoneum from branches of celiac trunk, the superior and inferior mesenteric arteries.

LYMPHATICS OF THE PERITONEUM

The lymphatics of the parietal peritoneum join the lymphatics of the body wall, and all drain to lymph nodes. However, the lymphatics of the visceral peritoneum join the lymphatics of the related organs and are drained accordingly.

INNERVATION OF THE PERITONEUM

The parietal peritoneum contains somatic afferent nerves. The peritoneum contains many sensory fibers for the sensation of pain; the anterior portion of the parietal peritoneum is especially sensitive. Visceral peritoneum has no somatic afferent nerves and is relatively insensitive. Sensations which do occur are poorly perceived and not clearly localized by the brain.

A perforated viscous may, produce anterior abdominal wall rigidity, and intraperitoneal fluid collection may produce pain like sensations of traction or tension on the mesentery in the retroperitoneal space, but not localized pain.

The innervation of the visceral peritoneum (sensory fibers for pain) are carried by thoracic and lumbar splanchnic nerves.

PHYSIOLOGY:

The peritoneal fluid is a straw-colored transparent ultrafiltrate of the plasma with a total protein concentration of less than 1.5 g/dL and total nucleated cell count of less than 2000 cells/μL. Neutrophils represent 24% to 60% of the cell population of the peritoneal fluid. There is an estimate that a 1mm increase in the thickness of the peritoneum can result in the sequestration of 18 liters of fluid. Under normal condition, < 50 ml of sterile fluid is present in the peritoneal cavity which is circulated through the peritoneal cavity. The wide distribution and constant turnover of peritoneal fluid provides for a highly effective clearance mechanism for bacteria, cells, and foreign substances that enter the peritoneal cavity. Beneath the mesothelium is a sub mesothelial layer which has extracellular matrix, lymphatics and capillaries. The peritoneum is sensitive to inflammation, ischemia, and necrosis and it is mainly mediated through the peritoneal fluid that contains leucocytes. Thus when a focus of inflammation occurs anywhere in the peritoneal cavity, leukocytes release inflammatory mediators, resulting initially in a poorly localized, generalized pain. For example, in case of acute appendicitis, the patient first senses the inflammation as periumbilical pain. This is explained by the embryologic development along dermatomes. As more inflammatory mediators are released, the pain becomes more generalized

and results in spasm of the overlying abdominal muscles which clinically presents as guarding and rigidity.

In a normal person the main functions of peritoneum is visceral lubrication and fluid and particulate absorption. In a diseased person, the peritoneum acts to sense pain, involved in immune and inflammatory responses and fibrinolytic activity. The clearance of microbes from peritoneal cavity is done either by the diaphragmatic pump mechanism or the resident phagocytic system of the peritoneal fluid. Following the entrance of micro organisms into peritoneal cavity, organisms can be traced to right thoracic duct within six minutes and from bloodstream within twelve minutes. This is accomplished buy the diaphragmatic clearance mechanism or "pump", which is influenced by a number of factors.

1) Blockage of the stomata by platelets
2) Maintaining a Positive end expiratory pressure
3) Head up position helps in delaying the appearance of bacteria in circulation
4) Reduction in spontaneous respiration by general anaesthesia

Thus a person with perforative peritonitis when placed in a semi upright position has probably decreased absorption of bacteria through the diaphragm.

PATHOPHYSIOLOGY OF PERITONITIS

Intra-abdominal infection usually present as peritonitis which is inflammation of peritoneum

↓

Most common cause of secondary peritonitis could be GIT perforation

↓

Infection of more than 2 strains of bacteria

↓

Gram negative and Gram positive organism release endotoxin and exotoxins released into blood stream and peritoneum

↓

Cytokines released are INF, IL-1, IL- 6, IL-8 (primary mediators)

↓

Secondary inflammatory mediators are Prostaglandin and leukotrienes, Coagulation pathway activation

↓

Secondary inflammation triggers peritonitis leading to interstitial space loss

↓

Features of perforative peritonitis like Guarding, Rigidity, Hypotension, Abdominal distension

↓

All these lead to Systemic inflammatory response leading to excessive inflammatory activation

↓

Inflammatory activation within the organ causes cellular damage

↓

When two or more vital organs become dysfunctional causes- multiorgan dysfunction

SOURCE CONTROL

It is defined as management protocol that ultimately aims to eliminate the infection that promote the inflammatory response leading to SIRS

MODE OF INFECTION

Most of the peritonitis is caused by bacterial invasion. And also the most common cause of bacterial invasion is perforation of a hollow viscous of the gastrointestinal tract. Other route of entry of micro organisms is through female genital tract or even exogenous contamination. Primary peritonitis is an entity which occurs spontaneously in the absence of these causes and is due to infection with pneumococcal, streptococcal or H.Influenza organisms.

Thus the routes of spread of infection in peritonitis may be summed up as:

1) Perforation in GIT
2) Transmural translocation , for example, in acute pancreatitis, bowel ischemia
3) Haematogenous spread
4) Exogenous contamination
5) Female genital tract

CLASSIFICATION:

1) **PRIMARY PERITONITIS-** This occurs in the absence of hollow viscous/ bowel perforation
   A) Spontaneous bacterial peritonitis is seen in chronically ill patients with ascites, chronic renal failure patients undergoing dialysis, patients with nephrotic syndrome.
   B) Primary bacterial peritonitis occurs in people who are otherwise healthy and is seen in the absence of surgery or trauma and is the result of primary infection of peritoneal lining by streptococcus.
2) **SECONDARY PERITONITIS-** occurs after the perforation of hollow viscous/ bowel, or in post operative patients due to anastomotic leak

3) **TERTIARY PERITONITIS**- an intra-abdominal infection that persists or recurs after 48 hours following successful and adequate control of the source of peritonitis.

LOCALIZED PERITONITIS:

Many factors play a role in the localization of peritonitis. The peritoneal cavity is divided into a supracolic and infracolic compartment by the transverse colon and so the infection is localized to either of the two compartments. When supracolic compartment overflows, the infection may spread over the colon and thus to the infracolic compartment, or through the right paracolic gutter into the right iliac fossa and finally into the pelvis.

The localized form of peritonitis may present as inflammation of peritoneum with loss of its glistening appearance. The peritoneum becomes reddened and velvety. There occurs deposition of fibrin flakes and adhesion of bowel loops. There is also accumulation of serous inflammatory exudates which contains leukocytes and plasma proteins. This in time becomes turbid in nature and turns into pus. The greater omentum also helps in curbing the spread of infection.

GENERALIZED PERITONITIS:

The factors which favor the spread of infection leading a generalized peritonitis are:

1) The spill of contents and infectious material before localization takes place such as in perforation
2) Stimulation of peristalsis by intake of food/ water, administration of purgatives/ enema
3) High virulence of the causative organism
4) Young children with small omental surface
5) Disruption of the localized collection
6) Immunocompromised individuals , due to use of steroids/ diseases such as AIDS, old age

SPECIAL FORMS OF PERITONITIS:

BILE PERITONITIS:

It may be due to damage to the biliary system/ tract due to surgery/ acute cholecystitis. If the bile does not extravasate slowly and if the collection does not becomes sealed off, then there occur signs of generalized peritonitis. The treatment is to identify the source of bile leak. Infected bile is more dangerous than sterile bile. There are a number of causes for bile leak such as perforated cholecystitis, post cholecystectomy

– stump leakage, or leakage from an accessory duct, bile duct injury, T-tube dislodgement, leaking duodenal stump in post gastrectomy patients, or following liver trauma.

TUBERCULOUS PERITONITIS:

It is very common in the developing countries. It is commonly associated with HIV co infected individuals. Tuberculosis may spread through GI tract or via mesenteric lymph nodes or directly through bloodstream. The unusual paths of spread are through cavitating pulmonary TB and the fallopian tubes. The main clinical features are abdominal pain, sweating, malaise, weight loss and loculated ascites. Caseating peritoneal nodules are also common.

FAMILIAL MEDITERRANEAN FEVER- PERIODIC PERITONITIS:

It is characterized by abdominal pain and tenderness, mild fever, increase in neutrophil count, joint pain. It is common in Arab, Armenian and Jewish population. Mutations in MEFV gene are seen. The peritoneum is inflamed, specifically near the spleen and the gall bladder.

RESPONSE TO PERITONITIS:

LOCALIZED RESPONSE:

Primarily body responds to peritonitis through a local response which consists of vasodilatation, increased microcirculatory flow, and local tissue edema leading further to the secondary phase of entry of phagocytic cells to the inflamed peritoneal tissues. Another mechanism for microbial removal from the peritoneal cavity is the clearance of bacteria through the lymphatic system. Thus the microorganisms are cleared off the peritoneal fluid either through phagocytes or through the lymphatic system of the diaphragm.

When body is unable to cope up with the microbial density with these two mechanisms, body resorts to the last stand ditch mechanism , that is to loculate, or seal off the inciting pathogen. The inflammatory process forms a fibrin deposition around the dense collection of microbes, and this loculation process results in the formation of abscess. The microbes are contained within the abdominal cavity as a result of abscess formation. But in time, the abscess cavity is itself a source of microbes which may gain access to the systemic circulation.

The patient might have systemic sequelae from the abscess even though the microbes are contained within a loculation. These collections

are found characteristically in the dependent portions of the abdominal cavity such as the sub diaphragmatic space, peri and paracolic gutter, and pelvis. Patients with previous abdominal surgeries might present with collection in false dependent portions within the peritoneal cavity like the adhesions between loops of intestine or between adhesions from the bowel to the abdominal wall.

Thus it is evident that although body's inflammatory process may be effective in containing the dense microbes within the abscess cavity, the sequelae of the process usually have systemic consequences. ultimate a persistent thus leads to a syndrome of SIRS AND ultimately MODS.

SIRS: - SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

A septic response may develop within the body even without an identifiable septic focus and this is termed as tertiary peritonitis. It is found that immunogical dysfunction may have a role to play in it. In such cases it becomes important to treat and restore the immunogical balance apart from treating infection with antibiotics.

The systemic response is manifested mainly as hypovolemia and is due to the fluid accumulation occurring in the peritoneal cavity. The resultant change in intravascular volume leads to a reduction in venous return and cardiac output. This may be also due to release of a number of

factors such as of platelet activating factor, nitric oxide, TNF, IL-1. There also occurs a reduction in the urine output which may be explained by the decreased cardiac output, shunting of blood vessels in renal system, increased aldosterone and anti diuretic hormone secretion. This condition is also known as distributive shock or warm shock, which has features such as tachycardia, fever, oliguria, hypotension and warm extremities.

There also occurs atelectasis of lower lung fields and restriction in diaphragmatic mobility because of the accumulation of peritoneal fluid and abdominal distension caused by it. So hyperventilation occurs which results in the development of respiratoryalkalosis.

Ultimately adult respiratory distress syndrome (ARDS) sets in as a progression of pulmonary oedema and atelectasis. Acid base balance is also altered and metabolic acidosis is seen as a result of anaerobic glycolysis of the tissue metabolism.

CLINICAL FEATURES OF PERITONITIS:

LOCALIZED PERITONITIS:

The initial symptoms are abdomen pain, specific GI symptoms, malaise, anorexia and nausea. If the peritoneum becomes inflamed, there is increase in abdominal pain and fever spikes are seen. The pathognomic signs are guarding, rebound tenderness, and sometimes rigidity. Should

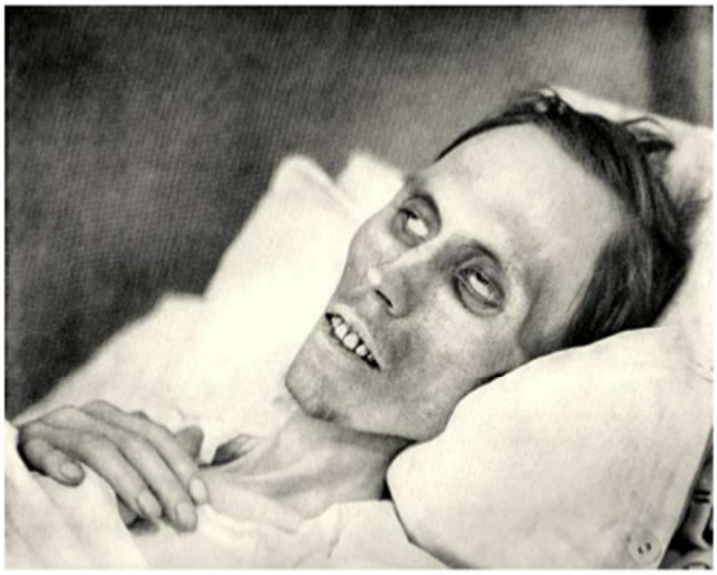
tip pain/ phrenic pain is seen when diaphragm is involved. In cases of pelvic peritonitis, per rectal or per vaginal examination reveals marked tenderness of the pelvic peritoneum.

GENERALIZED PERITONITIS:

The early symptoms are

1) Severe abdomen pain, worsened by movements or even breathing
2) The patient lies still
3) Diffuse tenderness
4) Generalized guarding
5) Paralytic ileus sets in , sluggish bowel sounds
6) Tachycardia, fever

facies Hippocratic



The late symptoms are

1) Rigidity
2) Abdominal distension
3) Absent bowel sounds
4) Circulatory failure
5) Hippocratic facies - sunken eyes, anxious face
6) Thready pulse, cold and clammy extremities

INVESTIGATIONS IN PERITONITIS:

BEDSIDE TESTS:

1) ECG- to rule out cardiac causes
2) Urine dipstick test to rule out UTI

BLOOD TESTS:

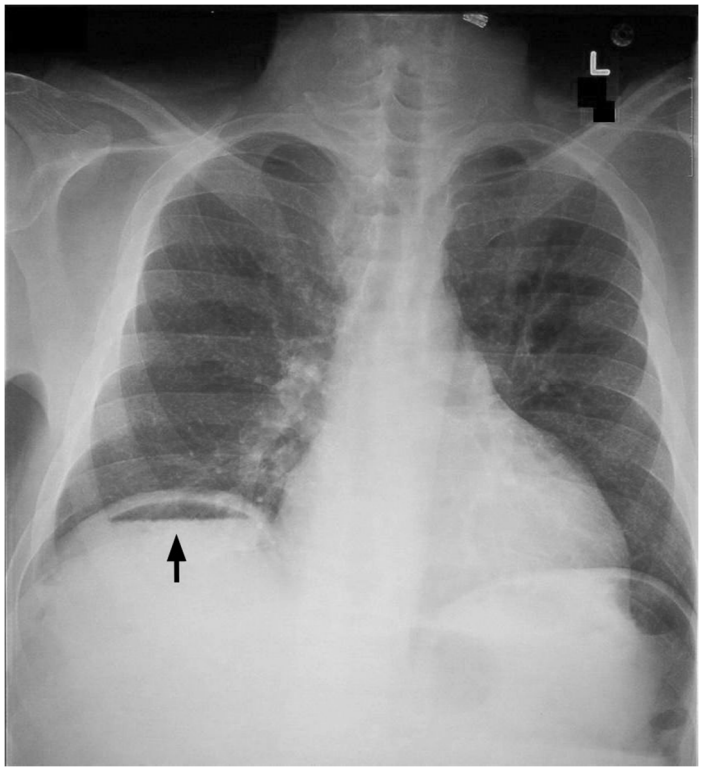
1) Complete blood count
2) Serum amylase levels- to rule out acute pancreatitis
3) Blood grouping and typing

Peritoneal Fluid Culture and Sensitivity -The peritoneal fluid is taken for culture to find the organisms involved in causing peritonitis and antibiotic sensitivity is done to provide appropriate empirical therapy.

Peritoneal fluid taken for culture & sensitivity

IMAGING STUDIES:

1) Chest X Ray to look for air under diaphragm
2) Supine abdomen X Ray to look for dilated bowel loops
3) CT Abdomen and pelvis to identify the cause for peritonitis



Chest x-ray showing air under diaphragm

INVASIVE INVESTIGATIONS have little value in modern era, such as diagnostic peritoneal fluid aspiration

MANAGEMENT FOR PERITONITIS:

1) General care of the patient and nutritional support
2) Correction of fluid loss and restoring circulating blood volume
3) Correction of electrolytes imbalances
4) Supportive measures to counteract septic shock
5) Continuous bladder drainage and gastric decompression using nasogastric tube
6) Initiation of antibiotic therapy
7) Adequate analgesia
8) Specific management of the inciting cause

COMPLICATIONS OF PERITONITIS:

SYTEMIC COMPLICATIONS such as septic shock, SIRS, MODS (multiorgan dysfunction syndrome), death

ABDOMINAL COMPLICATIONS such as paralytic ileus, recurrent abscess formation/ mass formation, portal pyemia, liver abscess formation, bowel loop adhesions

CLASSIFICATION OF PERFORATION:

- Acute perforation
- Sub acute perforation
- Chronic perforation
- Perforation associated with haemorrhage
- Perforation of intrathoracic gastric ulceration
- Pseudo perforation

1. ACUTE PERFORATION:

Here the ulcer perforate and occurs the spillage of gastric and duodenal contents into the peritoneal cavity there by resulting to Chemical Peritonitis

Clinical features depend on the stage of perforation.

Three stages of perforation, each of variable duration are as follows

a. Primary stage or stage of peritonism
b. Secondary stage or stage of peritoneal reaction
c. Tertiary stage or stage of bacterial peritonitis

1. **Primary stage:**

The clinical course in perforation is classical. Vigorous agonizing pain starts in the epigastrium or right hypochondrium first and later turns into a generalized pain.

The patient then becomes prostrated. Symptoms are due to irritation of the peritoneum by gastroduodenal contents spillage.

Pain shock may occur. Abnormal temperature, cold peripheries, sweating, palpitation, radiation of abdominal pain to both the shoulders as the diaphragm gets irritated, pale face.

Patient will assume a classical rigid posture by lying with legs updrawn and hands will be held tensely by his side. Temperature will be subnormal, as low as 95 – 96 degree or normal. Pulse rate may be normal or raised above 90 per minute. Shallow Respiration with increased respiratory rate may be there.

Per abdomen examination reveals, restricted or no movements of abdomen with respiration with prominent rectus muscle. On palpation warmth and diffuse tenderness is noted. Guarding and rigidity will be present. Bowel sounds will be absent on auscultation

The stage of which lasts for 3 – 6 hours.

The Perforated fluid may leak into Right Para colic gutter which will cause inflammation and pain, there by mimicking the features of acute appendicitis.

2. Secondary stage:

Transition time from the primary stage to secondary stage will be taking around 3-6 hours which will be depending on the size and site of perforation and amount of peritoneal soiling.

During this stage the spontaneous sealing of perforation may occur. Gross leakage of gastroduodenal contents, will lead the patient to stage of septic peritonitis which will rarely exceeds 6 hours.

Here the pain is lessened markedly with general condition improvement. Because of this stage of reaction it has sometime called **stage of delusion** and it is in this stage most of error in diagnosis takes place.

On examination varying amount of rigidity of abdomen and tenderness will be present with Bowel sounds are infrequently heard or absent

3. Tertiary Stage :

Stage of diffuse peritonitis which begins about 12 hours of perforation and lasts for about 24 hours until it passes onto final stages of paralytic intestinal obstruction. Gross contamination by Pathogenic organisms is likely here.

Peritoneal fluid will become purulent with bowel loop distension with fluid and gas. Intestinal movements diminish and finally disappear with onset of paralytic ileus.

The clinical features are same as those of generalized peritonitis from any other cause with less Pain, frequent vomitus, hiccoughs which may further depress the patient.

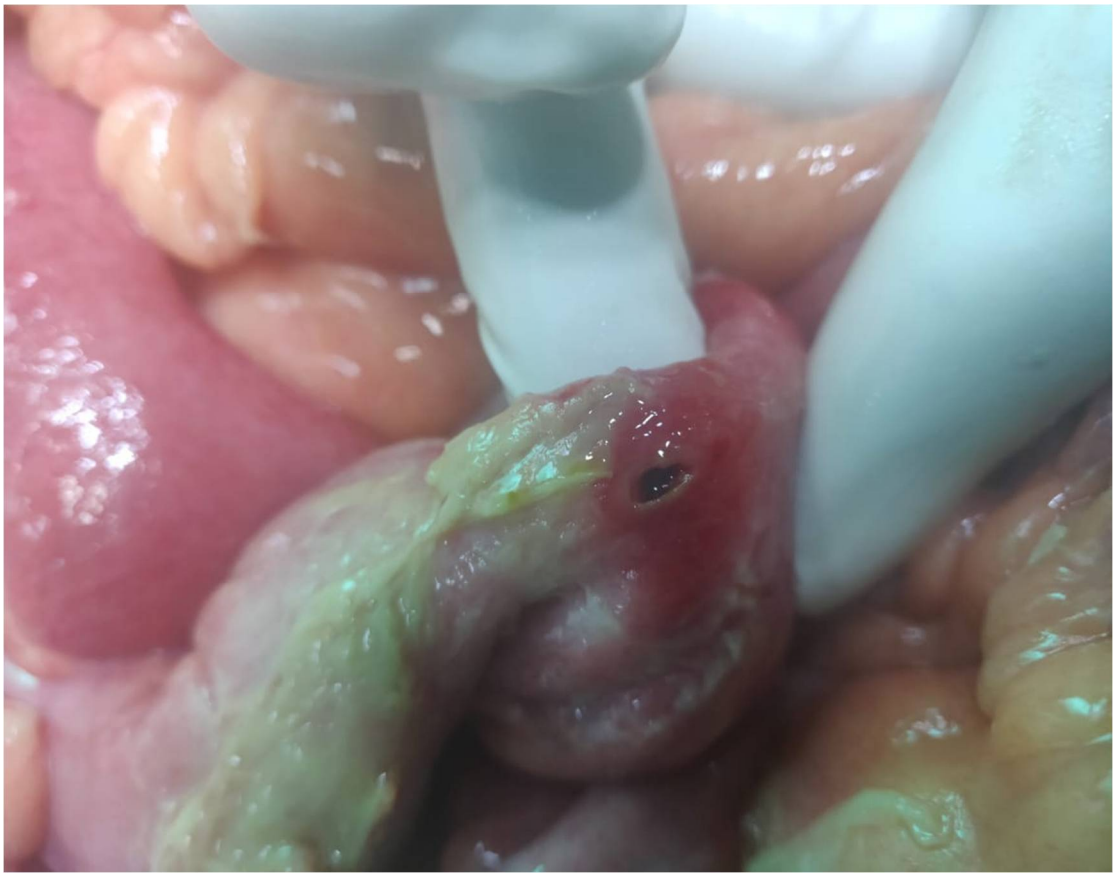
Sweating, vomiting, outpouring of fluid into peritoneal cavity, distended paralysed intestine, dehydration and electrolyte imbalance becomes more severe.

Patient will complaints of intense thirst, elevated temperature, dry & coated tongue, rapid thready pulse, shallow and rapid respiration.

Abdomen is distended, guarding still persists. On auscultation occasional tinkles heard. The typical Hippocratic facies denote that end is not far off. The face is ashen, body cold and calmy.

Patient drifts into toxaemic stage, dehydration and circulatory failure.

Patient will die usually 4-5 days after perforation



2. SUBACUTE PERFORATION :

The ulcer may perforate but the peritoneum will seal rapidly before there is spillage of duodenal contents, into general peritoneal cavity. There will be sudden onset of acute abdominal pain more to the right upper quadrant. Respiration will be shallow and on deep inspiration it may be associated with an abrupt catch in the breath

On examination, there is localised tenderness and rigidity, leaving behind the rest of the abdomen soft on palpation and non-tender. X– Ray film usually reveals only a small amount of gas under diaphragm.

Patient's symptoms will usually subside in half an hour to 2 hours. Rarely it extend and the signs of an acute perforation develop

3. CHRONIC PERFORATION :

It occurs when an ulcer perforated into an area which is walled off by adhesions or by adjacent viscera like liver, colon or greater omentum or it may also occurs when gastric ulcer perforates into omental sac there by leading to a chronic abscess there by making the diagnosis confusable.

Since these patients doesn't present with classical signs and symptoms of peritonitis, they are seldom diagnosed as peptic ulcer perforation. Irregular temperatures, rigors, leucocytosis, dullness at the base of the lung, Consequent pleural effusion or basal congestion will lead to the diagnosis of sub phrenic abscess, containing gas and diaphragm is raised and fixed on right side.

USG of abdomen -most reliable investigation of choice for intraperitoneal abscess

4. PERFORATION ASSOCIATED WITH HAEMORRHAGE:

Perforation with massive haemorrhage is grave but fortunately a very rare complication. It will occur 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 will be similar to acute perforated peptic ulcer with signs of hemorrhage

5. PERFORATION OF AN INTRA-THORACIC GASTRIC ULCERS:

An extremely rare variety of perforation. Here the ulcer will be in hiatus hernia, which will be fixed in the mediastinum. Unless we identify the hiatus hernia, it will be extremely difficult to make a proper pre – operative diagnosis.

Since the symptoms and signs will be pointing an intra thoracic lesions such as coronary thrombosis, acute pericarditis, pulmonary embolism.

Rare type Perforated peptic ulcer :

Peptic ulcer in meckel's diverticulum in intestinal duplication may occasionally perforate. Simultaneously multiple perforations occur in less than one percent of all cases.

VARIOUS SCORING SYSTEM FOR PERITONITIS:

POSSUM SCORE:

This score is based on 12 physiological and 6 operative factors usually

This is a score used widely in predicting different outcomes of morbidity in

General surgical procedures along which 2 parameters are added for peritonitis and modified accordingly. Perforation Operation time co morbid status are added to above factors.

ACUTE PHYSIOLOGICAL AND CHRONIC HEALTH EVALUATION

(APACHE II) SIMPLIFIED ACUTE PHYSIOLOGY SCORE.

It comprises of parameters like age , health rate, systolic blood pressure ,temperature , GCS , mechanical ventilation / CPAP, PaO, FiO2, urine output, BUN, sodium ,potassium, bicarbonate bilirubin white blood cell, chronic diseases, Type of admission It is an ICU scoring system that is used in Predicting morbidity and mortality of patients.

MANHAEIM PERITONITIS INDEX :

Developed by wacha and linder in 1983 based on retrospective analysis of data. Patients can have maximum score of 47 with score exceeding 26 are said to be having high mortality rate. Factors included are age, duration of Peritonitis, organ failure, diffuse peritonitis, site of perforation, level of exudates in peritoneal fluid

THE SEPSIS SCORE:

Not specifically designed for patients with peritonitis, this scoring system is adjusted and re-used for patients with Sepsis due to peritonitis. This is widely accepted by various sectors of ICU community and used only in late stages of prediction.

BOEY SCORE:

This is one of the scores that is easily available in clinical practice Parameters like concomitant medical illness, preoperative shock (SBP <90mmhg) duration of perforation more than 24 hours are included scores ranging from 0-3.

CHARLSON COMORBIDITY INDEX:

It predicts ten years mortality of patients who may have range of total co-morbid conditions. This index is incorporated into parametric study of peritonitis and influences that effect of these conditions in predicting the mortality and morbidity of the disease.

HACETTEPE SCORE:

Predicts 30 days mortality in patients in PPU and it includes serious medical illness, acute renal failure, WBC count, male gender as prognostic factors.

PULP II SCORE:

Age greater 65 years, active malignant disease or AIDS, liver Cirrhosis, steroids use time from perforation to admission greater than 24 hours , preoperative shock, serum creatinine, four levels of ASA score are Parameters included here. They are also among the scoring system specifically Designed for PPU.

PERFORATIVE PERITONITIS:

INTRODUCTION:

Once a perforation occurs, the gastroduodenal contents which spill into the peritoneal cavity will lead to peritonitis. It has dreadful

complications and is a potentially life threatening condition and so should be managed immediately and at right time.

It usually occurs in patients with chronic peptic ulcer disease and occurs in both Duodenal and Gastric ulcers. Perforation can also occur without any preceding inciting event or cause. In the early stage there occurs only chemical peritonitis because visceral contents are sterile and so infective peritonitis is rare.

The free fluid which spills into the peritoneal cavity circulates and the circulation is largely dependent on the attachments of the peritoneum and its folding. Even in case of non bacterial peritonitis, such as acute pancreatitis, the peritoneum becomes ultimately infected by transmural spread of micro organisms from the bowel lumen. But the features and ultimate prognosis depends on the general condition and stability of the patient. Once intra abdominal infection occurs, it becomes a huge challenge to the treating surgeon to prevent SIRS (systemic inflammatory response syndrome) from setting in and reducing fatality.

EPIDEMIOLOGY:

Perforation is more common in males than females and is more often seen in the age group of 30-50 years.

CAUSES OF PERFORATIVE PERITONITIS:

Stomach

Peptic ulcer perforation, malignant perforation (e.g. adenocarcinoma)

Duodenum

Peptic ulcer perforation

Small intestine

- Intestinal TB, typhoid ulcer, Meckel's diverticulum, jejuna diverticulosis, bowel ischemia, obstructed hernia, malignant perforation

Large intestine

appendiceal perforation, obstruction, Malignant perforation, sigmoid volvulus, ischemic bowel loop, Ulcerative colitis and Crohns disease, Amoebic colitis and amoebic ulcer

FACTORS INFLUENCING PERFORATIVE PERITONITIS:

- Bacterial virulence
- Intraperitoneal fluid collection which leads to dilution of immune factors
- Platelets which clog and occlude the diaphragmatic lymphatic system
- Fibrin deposition which impairs platelet function
- Haemoglobin contains ferric ion which enhances the ability of bacteria to inhibit neutrophil function
- Chemical peritonitis induced by sterile gastric acid secretions

MICROBIOLOGY OF PERFORATIVE PERITONITIS:

The normal bacterial flora of the gut lumen is generally low till the distal part of small bowel. Due to some causes the proximal colonisations gets increased such as obstruction, acute or chronic motility disorders. The biliopancreatic system is also usually sterile but may get infected in cases such as cholelithiasis. Peritoneal infection is usually caused by a mixed strain of bacterial population. Gram negative bacteria, bacterioids and clostridium are various causative organisms. Bacteroides pose a special problem because they are strictly anaerobic, non sporing

organisms, slow to grow on culture media, and are resistant to penicillin and streptomycin, and other cephalosporins.

In case of perforative peritonitis, following perforation of hollow viscus, the normal bacterial flora gets disturbed and results in entry of micro organisms into the peritoneal cavity. The type of organism isolated from the peritoneal fluid culture will depend on the level of perforation. Change of normal bacterial flora in immunocompromised patients and hospitalized patients has lead to the emergence of highly virulent organisms.

COMMON MICRO ORGANISMS IN PERITONITIS:

1) **E COLI:**

INTRODUCTION:

E. coli is the most common causative organism in human bacterial infections. The natural habitat of E. coli is the human intestinal tract. Hence it is considered as an indicator organism for fecal contamination of water. Intestinal infections are caused by the pathogens such as EPEC, ETEC, EIEC, EHEC, and EPEC. Extraintestinal infections caused by E Coli are urinary tract infections, cholecystitis, appendicitis, peritonitis, postoperative wound infections, and sepsis. E. coli bacteria infections are diagnosed by means of separation of pathogen and its identification.

MORPHOLOGY:

E Coli is a Gram-negative, straight rod shaped bacterium which is peritrichously flagellated. It is a rapid Lactose fermentor. It has a complex antigen structure and is explained due to the presence of O, K, and H antigen.

DIAGNOSIS:

In the identification of pathogen in extra intestinal infections, relevant materials are tested. Urinary tract infection is diagnosed with midstream urine and bacterial count is determined to ensure that it is not just contamination. Counts more than 10^5 indicate infection.

TREATMENT:

Before antibiotic administration we should also consider the resistance pattern of the organism. The antibiotics which are sensitive are aminopenicillins, ureidopenicillins, cephalosporins, quinolones, and cotrimoxazole.

EPIDEMIOLOGY AND PREVENTION OF DISEASES:

Intestinal infections are transmitted indirectly through food, drinking water, or surface water. The most useful preventive measure against intestinal infections is to thoroughly cook food and disinfect water properly.

2) KLEBSIELLA:

INTRODUCTION:

In the community level, *Klebsiella pneumoniae* is a pathogen with numerous and wide range of clinical manifestations, like septicaemia, pneumonia, urinary tract infection, meningitis and abscesses. Many virulence factors have been identified in invasive strains of Klebsiella, including hypermucoviscosity, capsular serotypes such as K1, K2, K5, K20, K54, virulence-associated genes.

MORPHOLOGY:

Klebsiella pneumoniae is also known as Friedlander's bacillus). It is a Gram-negative, rod shaped, non-motile, encapsulated, facultative anaerobic, lactose fermenting organism found as a normal flora of the mouth, skin, and intestines.

DIAGNOSIS:

Klebsiella when grown on blood agar shows a mucoid growth and colonies of around 3 to 4mm in diameter. On MAC, pink colonies are seen, since it is a lactose fermentor, mucoid, and 3 to 4 mm in diameter. Colonies on Hektoen enteric agar and XLD are yellow in color.

When grown in Mckonkey agar, large, mucoid, glistening pink colonies are seen. Serological tests are available for identification of Klebsiella species and they are based on the O (somatic) antigen and K (capsular) antigen. The capsular identification is performed by a test called Quellung reaction in which there will be capsular swelling.

TREATMENT:

The recommended antibiotic treatment regimen is changing due to developing antibiotic resistance. Klebsiella is usually resistant to several antibiotics. It has been found that plasmid is involved in resistance pattern. Klebsiella which produce extended-spectrum beta-lactamases are resistant to many groups of antibiotics. The choice of an antibiotic agent depends on local susceptibility pattern and on the body part that is infected. It is found that meropenem has the best sensitivity to Klebsiella. The use of antibiotics is usually not enough. Surgical clearance is also needed for this organism.

ANTIBIOTIC SENSITIVITY

Antibiotic Sensitivity Testing

It is laboratory test which signifies how effective the antibiotic therapy is against a bacterial infections.

- Antibiotic sensitivity testing will control the use of **Antibiotics** in clinical practice.
- Testing will assist the clinicians in the choice of drugs for the treatment of infections.

Uses of Antibiotic Sensitivity Testing

1.The identification of relevant pathogens in exudates and body fluids collected from patients

2. Sensitivity tests done to determine the degree of sensitivity or resistance of pathogens isolated from patient to an appropriate range of antimicrobial drugs

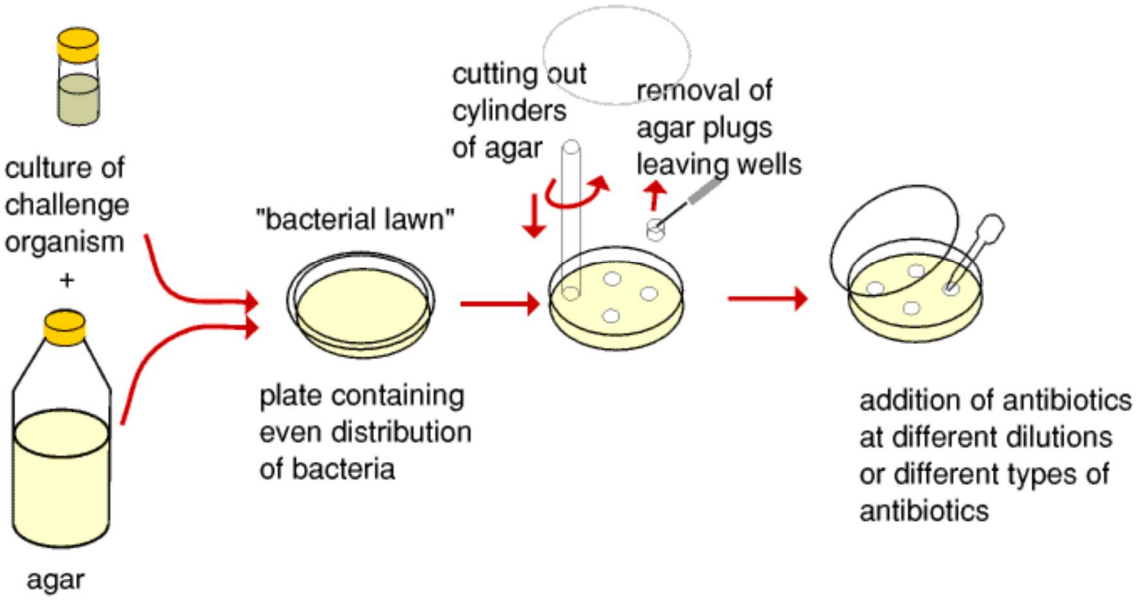
3. Assay of the concentration of an administered drug in the blood or body fluid of patient required to control the schedule of dosage.

Kirby-Bauer antibiotic sensitivity testing (disk diffusion antibiotic sensitivity testing)

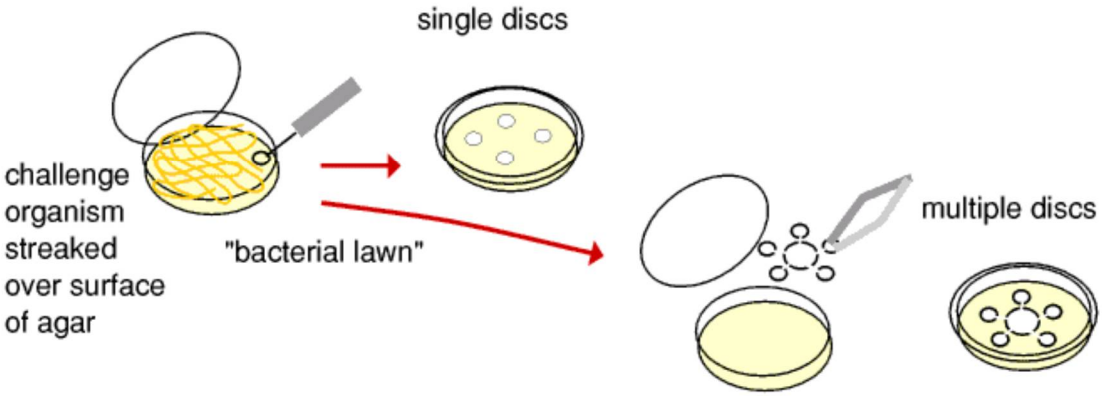
It is a test which uses antibiotic-impregnated wafers to test the bacteria that are susceptible to specific antibiotics.

The bacterium is swabbed on the agar and the antibiotic discs are placed on top. The antibiotic diffuses from the disc into the agar in decreasing amounts the further it is away from the disc. If the organism is killed or inhibited by the concentration of the antibiotic, there will be **NO growth** in the immediate area around the disc: This is called the **zone of inhibition.**

Antibiotic assay by the zone of inhibition method



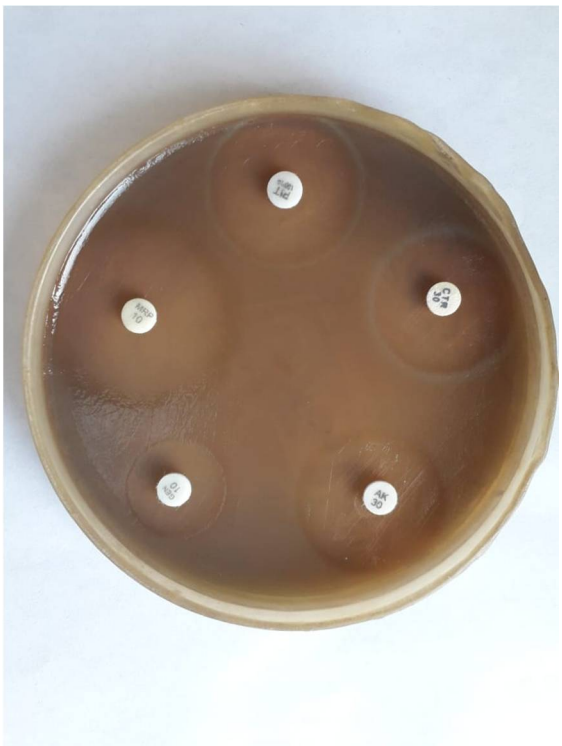
Alternative method using paper discs containing different antibiotics, or different concentrations of one antibiotic



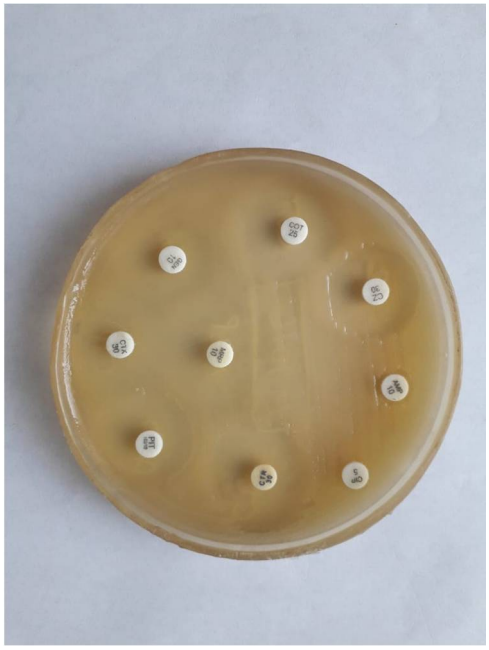
After incubation

different sized zones of inhibition may be seen

Peritoneal Fluid Culture & Antibiotic Sensitivity



Peritoneal Fluid Culture & Antibiotic Sensitivity Pattern

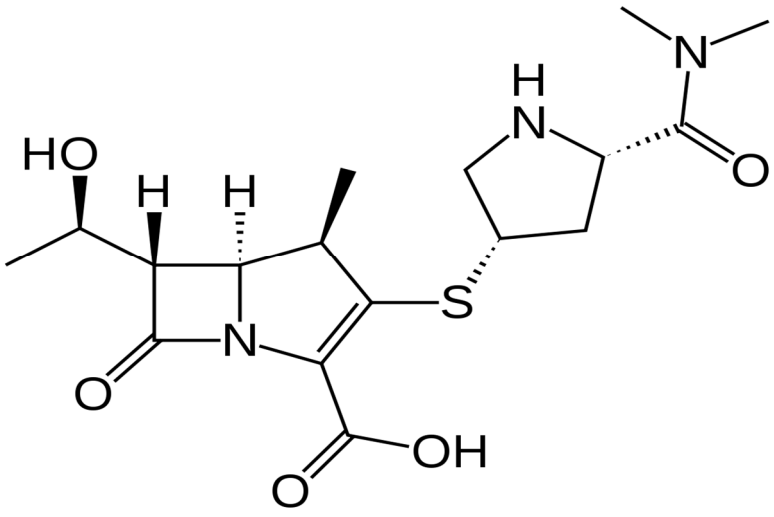


ANTIBIOTICS

COMMON ANTIBIOTICS EMPLYED IN THE TREATMENT:

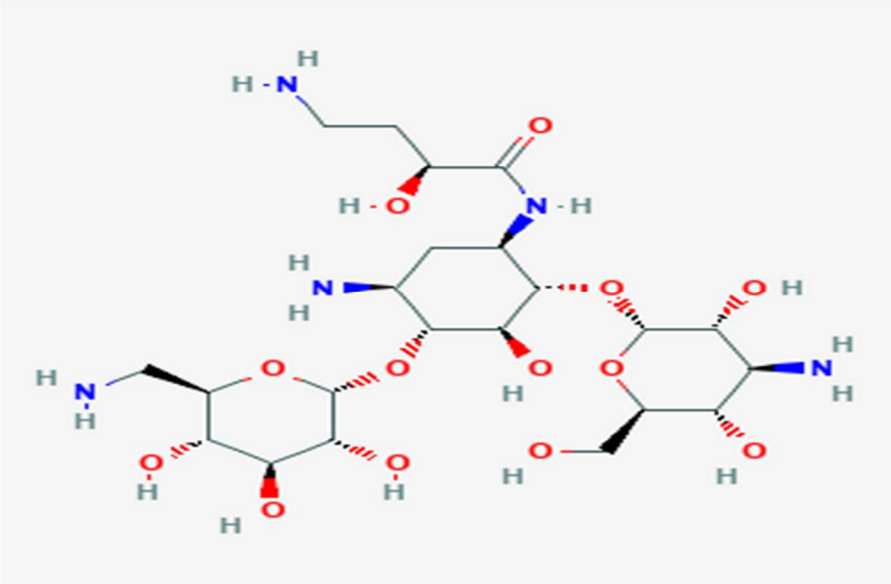
1) MEROPENEM:

Meropenem is a broad-spectrum antibiotic belonging to the class of carbapenem. It is especially active in controlling both Gram-positive and Gram-negative bacteria. The mechanism of action is by penetrating bacterial cell wall and interfering with the synthesis of components of cell wall which leads to death of the cell.



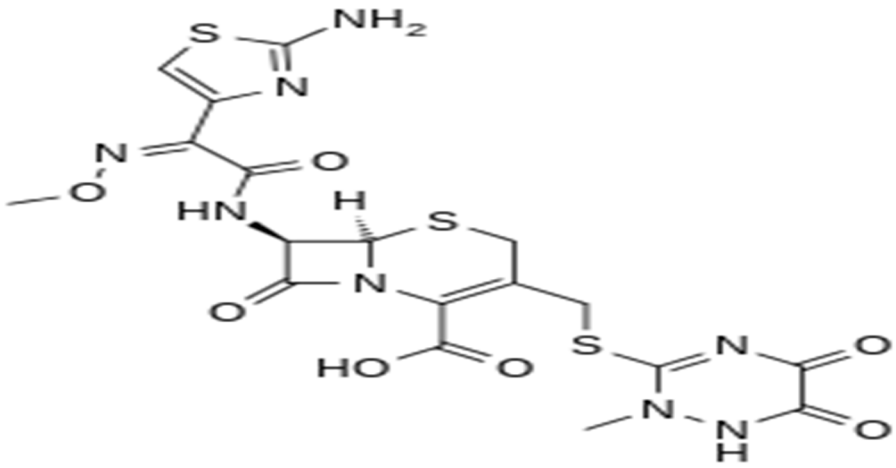
AMIKACIN

Amikacin is a semisynthetic derivative of the drug kanamycin. It resembles to the drug in dosage, pharmacokinetics and toxicity profile. It has resistance to bacterial aminoglycoside inactivating enzymes and so it has the widest spectrum of activity. It is used as a reserve drug for hospital acquired gram-negative bacillary infections where gentamicin / tobramycin are highly resistant. More sensory hearing loss is seen than vestibular toxicity



CEFTRIAXONE:

The distinguishing feature of this drug is its long duration of action, about 8 hours of T1/2, thus allowing for once, or at the most twice daily dosing.CSF penetration is good and elimination occurs mainly in urine and bile. Ceftriaxone has efficacy in treating serious infections including bacterial meningitis, abdominal sepsis and septicemia. The mechanism of action is inhibition of bacterial cell wall synthesis.



ANTIBITOIC RESISTANCE

Antibiotic resistance is the loss of ability of an antibiotic to effectively control or kill the bacteria that are "resistant" and continue to multiply in the presence of therapeutic levels of an antibiotic

MECHANISM OF ANTIBIOTIC RESISTANCE

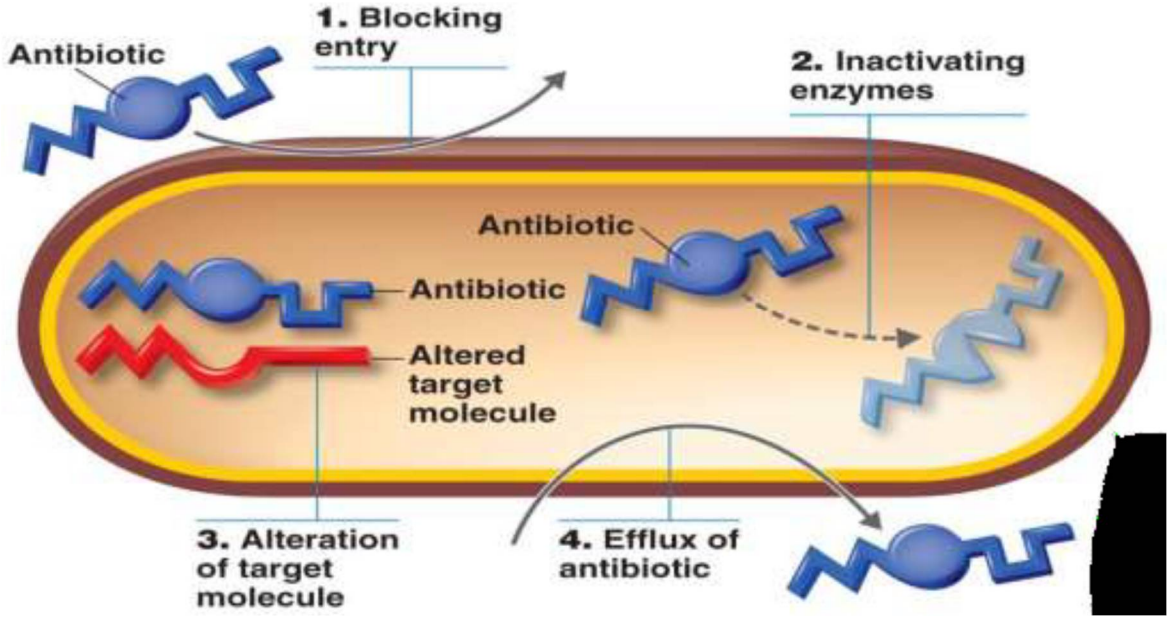
Denied access: Antibiotics wants to pass the bacterial cell membrane but membrane becomes impermeable for antibiotic: e.g. Imipenem

Antibiotic modification: In second step antibiotic becomes modified by the help of bacterial enzyme. E.g. beta lactamase inactivates penicillin

Altered target site: antibiotic cannot bind to its intended target because the target itself has been modified

Pumping out the antibiotic faster than it gets in: e.g. tetracyclines

Alternative target (typically enzyme): e.g. Alternative penicillin binding protein (PBP2a) in MRSA



CAUSES OF ANTIBIOTIC RESISTENCE

Over prescription of antibiotics

1. Physicians prescribe medicine without detecting the pathogen.
2. Prescribe broad spectrum antibiotics when narrow spectrum is a is actually needed

Patient Non-Compliance

1. Antibiotics are prescribed in a specific dose regimen.
2. Unable to afford full course.

Over dose of antibiotics

1. Antibiotics taken as OTC drug.
2. Retail drug store presents a chaotic situation during drug distribution.
3. Patients demand for antibiotics for normal cold, fever.

Poor quality of antibiotics

1. Expired and fake antibiotics.
2. Due to lack of quality compliance and monitoring.

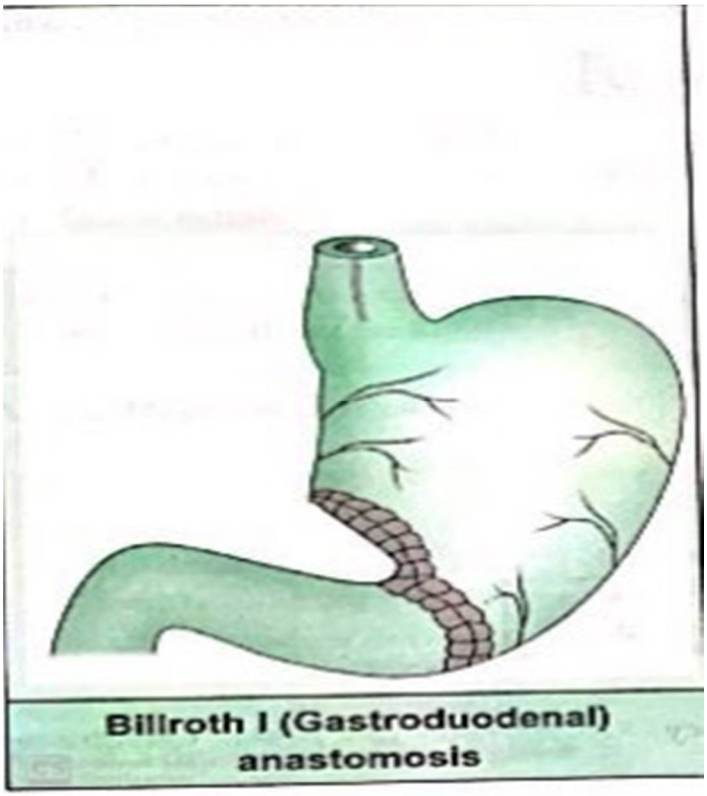
MANAGEMENT OF PERFOARATION

SURGICAL TECHNIQUE

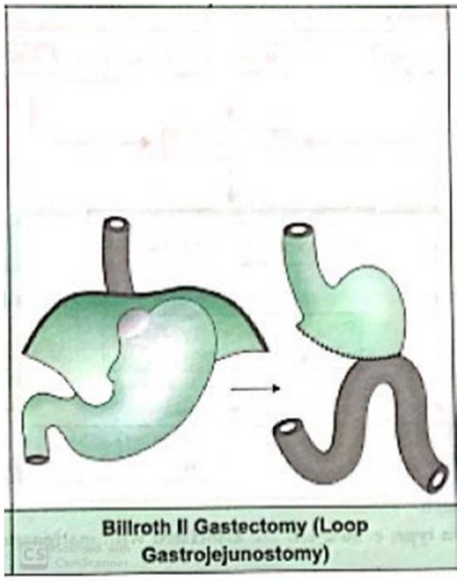
1) Surgical management is necessary for duodenal perforation
2) After identifying the perforation site in the duodenum, surgeons make choice of perforation closure
3) If the size of perforation is less than 2 cm, Grahams patch closure is ideal
4) Perforation is closed with 2-0 vicryl followed by placement of omentum over the perforation site
5) Large perforation more than 2 cms – Giant duodenal perforation require closure with jejunal serosal wall known as Thal patch closure
6) Duodenal perforation site of larger than 2cm is sutured to jejuna serosal wall
7) Patient is discharged after 7 days
8) Patient given H.pylori regimen for 8 weeks

GASTRIC PERFORATION

1) Graham patch repair with closure of omental patch
2) Gastric perforation at level of Gastric Antrum – Approached with Antral resection (Antrectomy), followed by anastomosis, remaining Gastric segment with duodenal billroth I



3) Billroth II is usually Gastrojejunostomy, anastomosis between Gastric remnant and jejunum

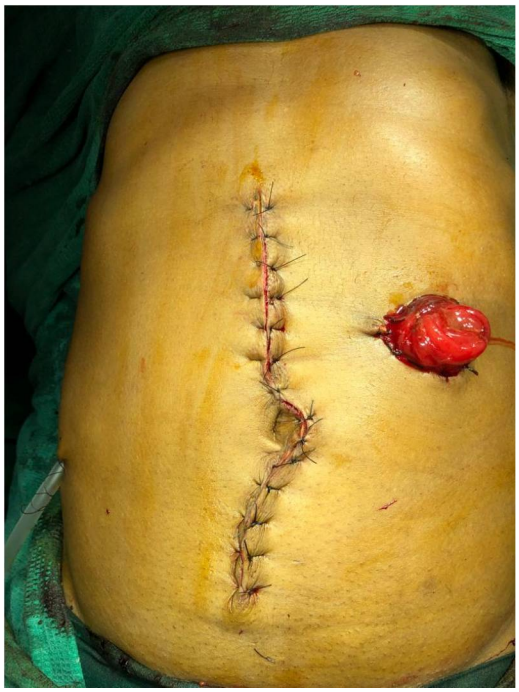


Billroth II Gastectomy (Loop Gastrojejunostomy)

4) Duodenal stump is usually closed

ILEAL PERFORATION

1) Ileal perforation near to ileocaecal wall of less than 3 cm , require Temporary enterostomy or Loop ileostomy



Ileostomy

2) Multiple ileal perforation usually require Resection and anastomosis

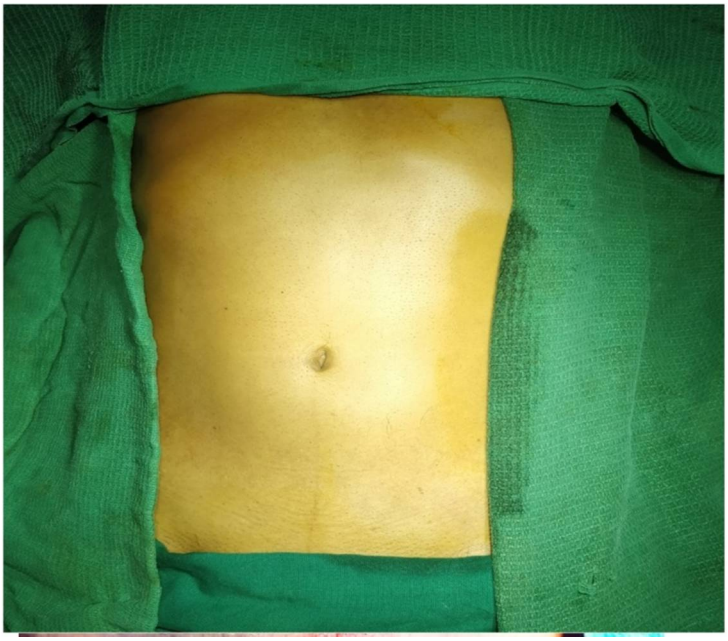
GRAHAM'S PATCH CLOSURE:

It is the simple method of closure of perforated duodenal ulcer by three absorbable sutures on interrupted fashion through the ulcer and a part of Omentum is placed in order to produce sealing effect.

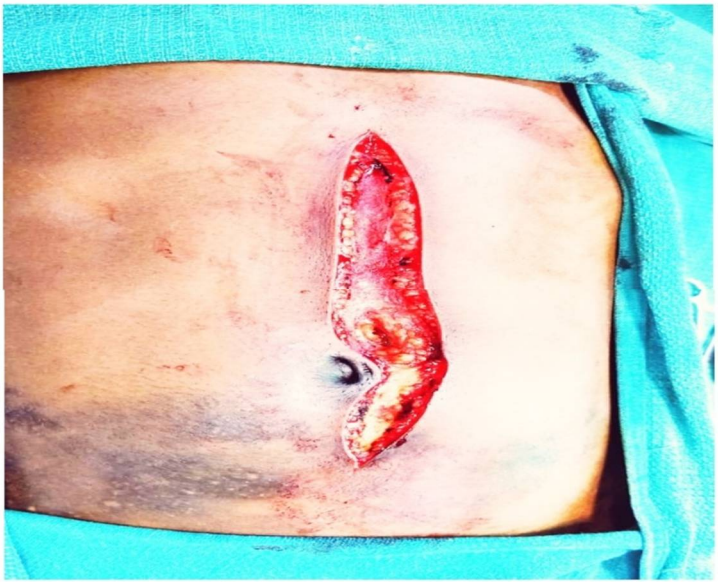
Abdomen is opened by vertical midline laparotomy incision. Peritoneal

cavity entered. Free fluid in the peritoneal cavity is aspirated and 10 ml of fluid is taken for culture and sensitivity. Visualization of stomach, small intestines, ileocaecal junction, appendix, large intestines done. Solid organs visualized. Perforation identified. Peritoneal lavage is done using 3 liters of warm normal saline.

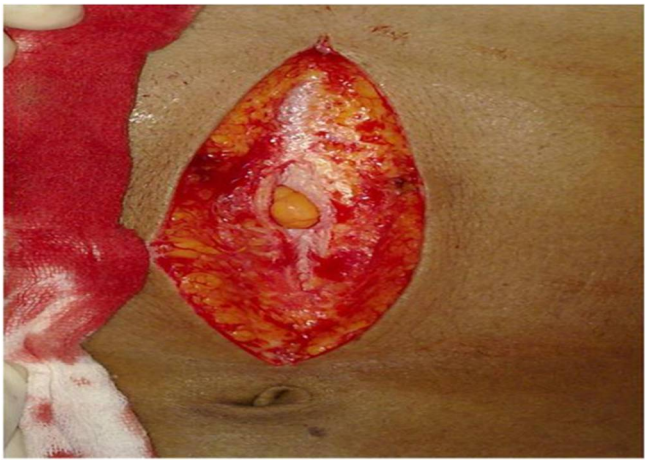
Primay closure of the perforation is done by taking full thickness stitches from the stomach or duodenal wall 1 cm away from the edge of perforation. Usually 3 to 4 stay stitches taken and then primary closure of the perforation is done. Live omental patch kept over the site and secured. Bilateral tube drains kept. Right side in to the morrisons pouch and left side in to the pelvic cavity. After verifying pad and instrument counts and after achieving complete hemostasis, rectus closed with 1 prolene and skin sutured with 2-0 ethilon or skin staplers.



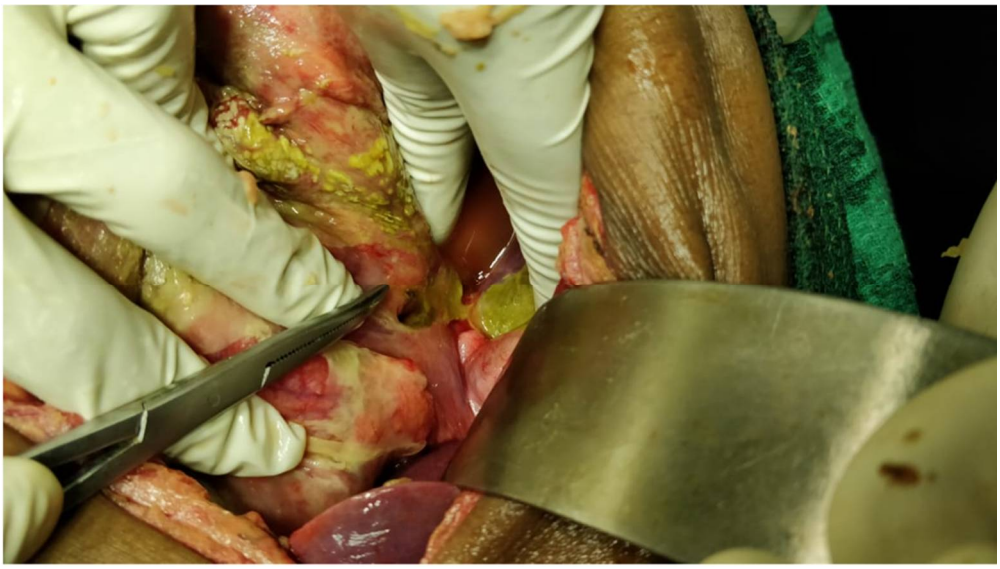
Abdomen painted and draped



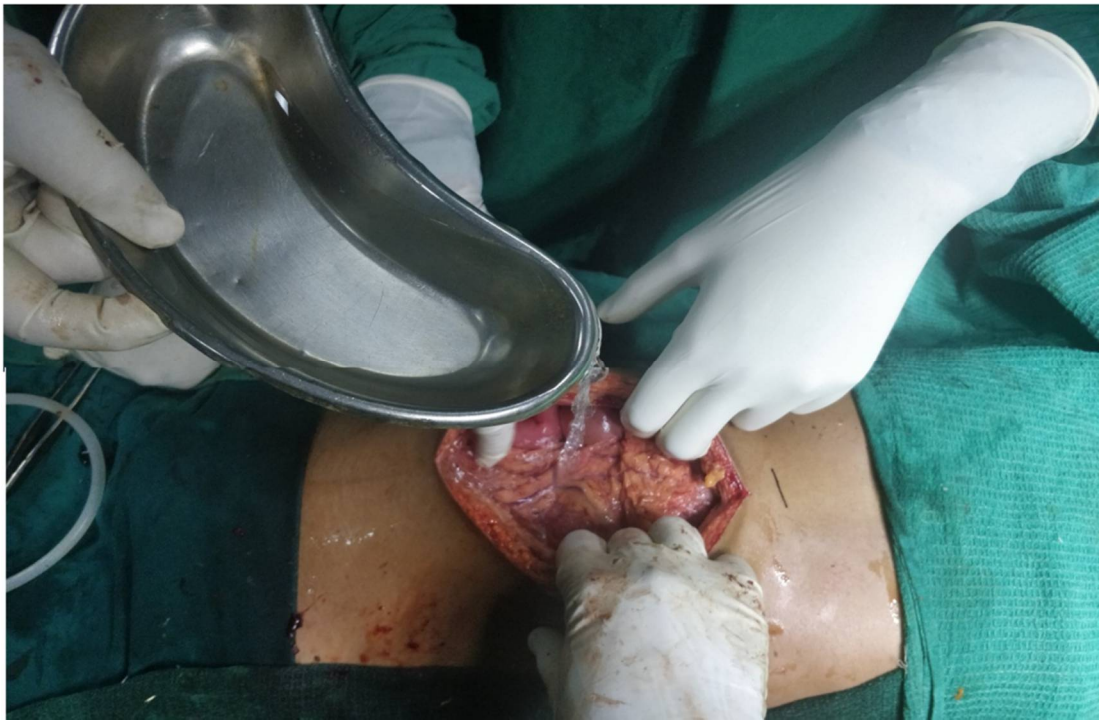
Skin and subcutaneous tissue opened through Midline laparotomy incision



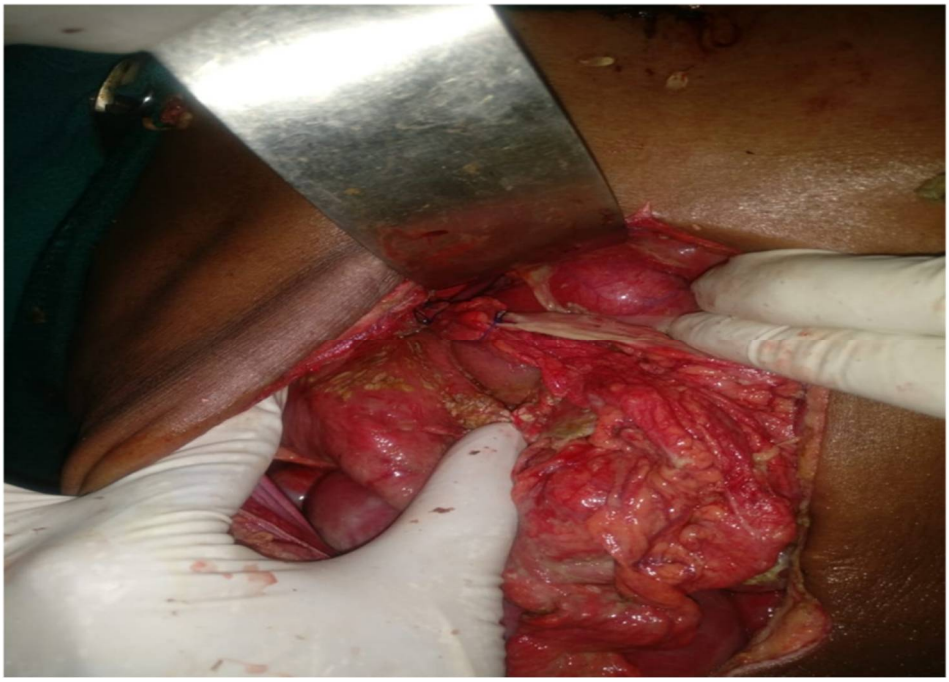
Picture showing preperitoneal fat bulging through rectus sheath



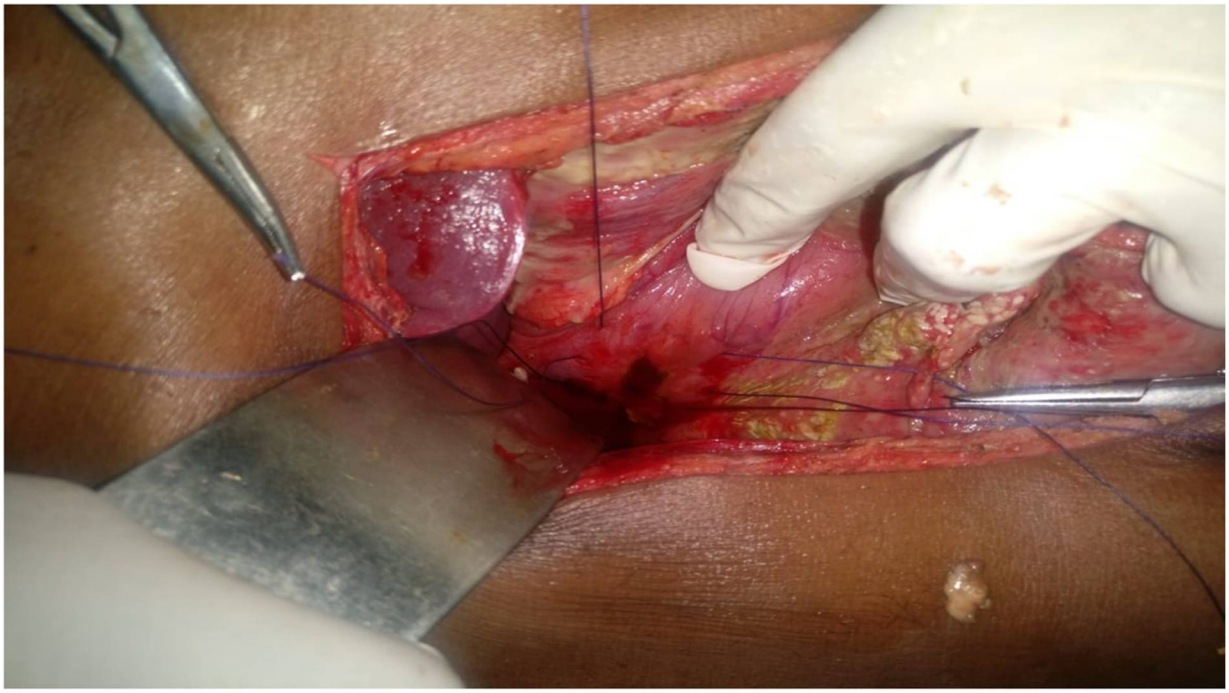
Pyloric antral perforation

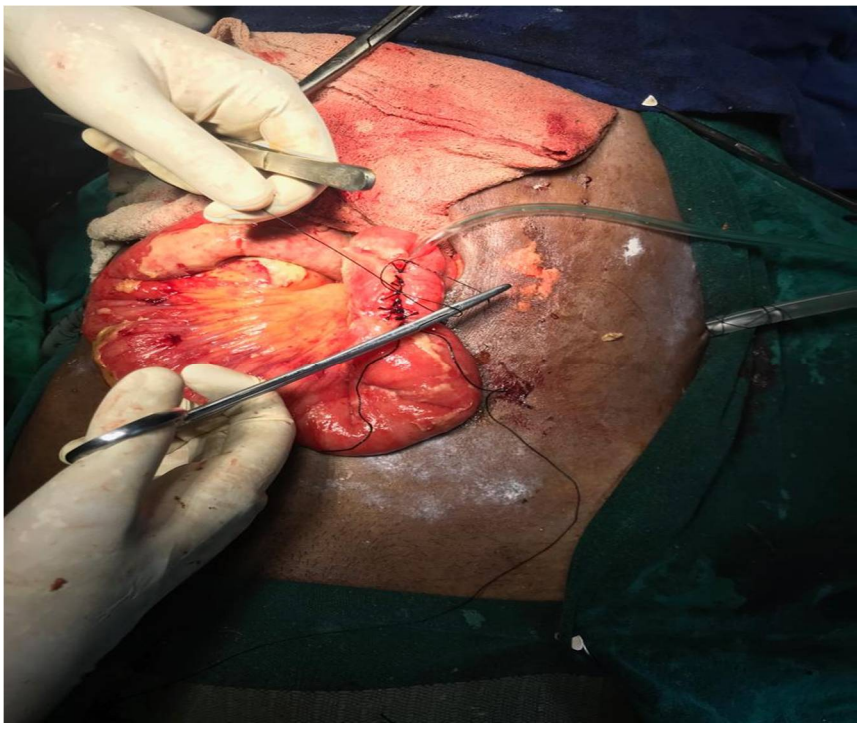


Peritoneal wash being given

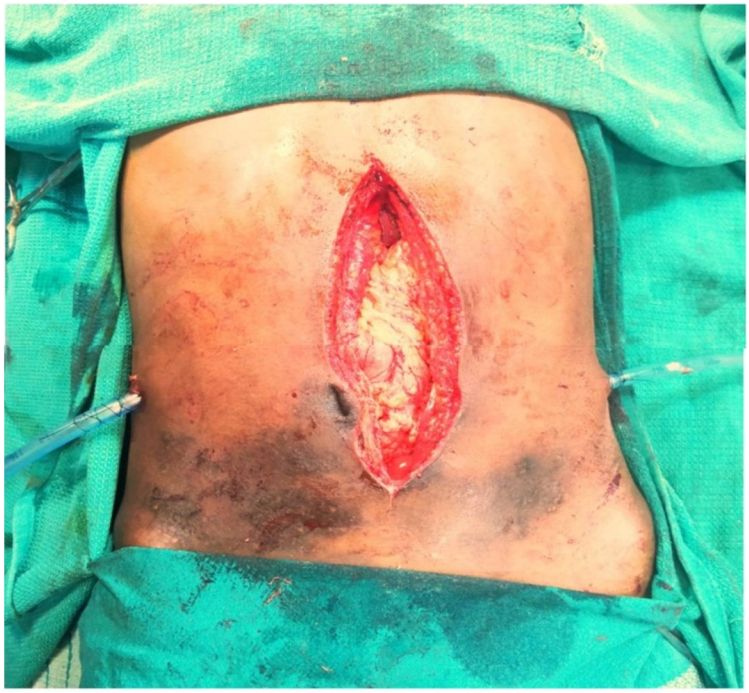


Picture showing omental patch kept over the primary closure

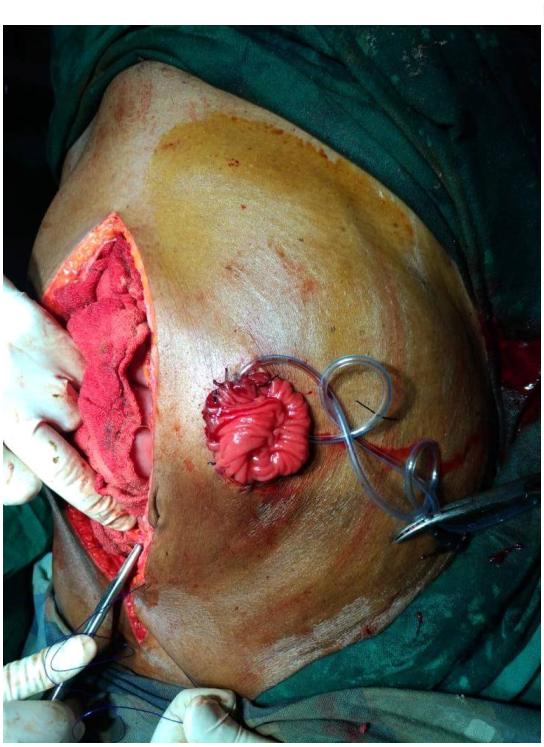


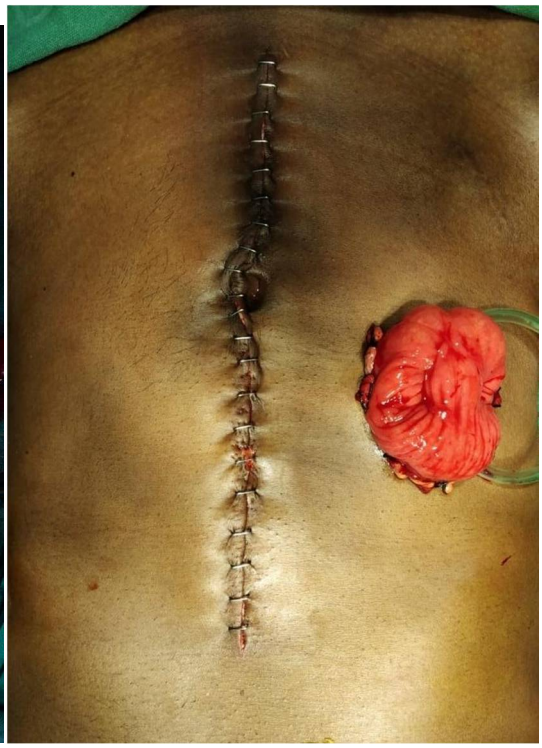


Picture showing primary closure of perforation



Bilateral tube drain kept





Closing of rectus sheath using 1 prolene Skin closed with staplers

POST OPERATIVE COMPLICATIONS

1) ANASTOMOTIC LEAKAGE

It is most dreaded complication.

It is usually associated with greater mortality

It is identified by Guarding, Rigidity, Fecal discharge via Drain, Fever, Features of toxemia

Imaging modalities include- Contrast enhanced CT Abdomen

It requires immediate surgical Re-laparotomy-Resection and anastomosis and Diversion colostomy for large bowel

2) BRONCHOPNEUMONIA

Most common lung complication is Unilateral basal atelectasis

It occurs during 24-48 hours after surgery

Most characteristic physical sign is rhonchi over the basal lung field

Hyperpyrexia may be present along with breathing difficulty, fall in Spo2

Diagnosed by CECT chest. Managed by Sputum C/S.

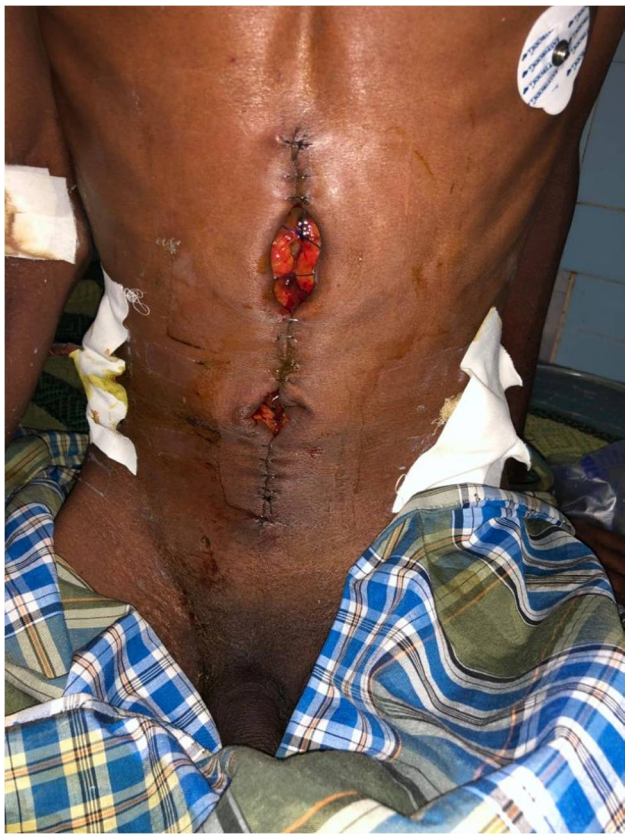
Most common organism causing bronchopneumonia is Streptococcus pneumoniae

Treated by mechanical ventilator support, IV antibiotics, chest physiotherapy-

3) WOUND DEHISCENCE OR BURST ABDOMEN

It is a surgical emergency regarding immediate closure of abdomen

Causes of dehiscence may be violent cough, straining, vomiting, short lived suture material applied on the linea alba



NECESSARY STEPS FOR WOUND DEHISCENCE

1) If the skin remains closed, support the wound, reclosure the wound under general anesthesia

2) If there is complete disruption with exposure of the gut, the exposed gut should be packed with saline soaked large gauze pads followed by emergency resuturing of abdominal wall

OPERATION TECHNIQUE-

- The skin should be cleaned with antiseptic solution
- Exposed gut should be washed with warm saline
- Edges of the wound are elevated using hooks or forceps
- Finger is gently inserted into the abdominal cavity to check for adhesions of bowel to the wound edges.
- Adhesions released by gentle manipulation
- Wound edges are cut 2 cm from the incision point
- Single mass closure of the wound edges to be done (Modified Cellanjones technique)
- If primary closure not feasible, the defective wound is closed by mesh. At later date, mesh can be removed and the wound can be covered by Split skin graft

4) PARALYTIC ILEUS

- It refers to the slowness or absence of the bowel peristaltic movements

- Causes include – major abdominal procedures, spine surgeries, metabolic abnormality
- Clinical features include- Abdominal distension, Abdominal discomfort, Sluggish or absent bowel sounds
- In advanced cases, abdominal distension increases, tachycardia and tachypnea are present
- Radiological imaging include X-ray abdomen erect – showing distended bowel loops with fluid and gas filled shadows
- Paralytic ileus may be prevented by- handling gut smoothly, Nasogastric aspiration , reduced peritoneal contamination, electrolyte and fluid balance, avoiding intestinal motility stimulants
- Treatment – Nasogastric aspiration and decompression, to rule out any mechanical obstruction, Electrolyte study and maintaining fluid balance and input output chart

5) WOUND INFECTION

Wound infection is more common

It occurs due to peritoneal contamination, malnutrition of the patient, hospital acquired infection, obese patients

Wound discharge C/S sent and antibiotics started according to sensitivity.

Daily wound cleaning and dressing is necessary, Wound suture removal is delayed for a week.

6) **BLEEDING**

Most common immediate post operative complication.

Diagnosis - To check patient vitals – BP, pulse, urine output, drain for first 24 hours, fall in haemoglobin, increase in abdominal girth

Management – immediate wound exploration followed by haemostatic control

If haemoglobin is less than 8 g/dl , blood transfusion in the form of whole blood is essential.

7) FEVER

It is the most common complication, mostly due to inflammatory response other than infections.

Course of hyperpyrexia

Day 2-5 : atelectasis of lung

Day 3-5 : wound infection

Day 5 : lung infection, UTI

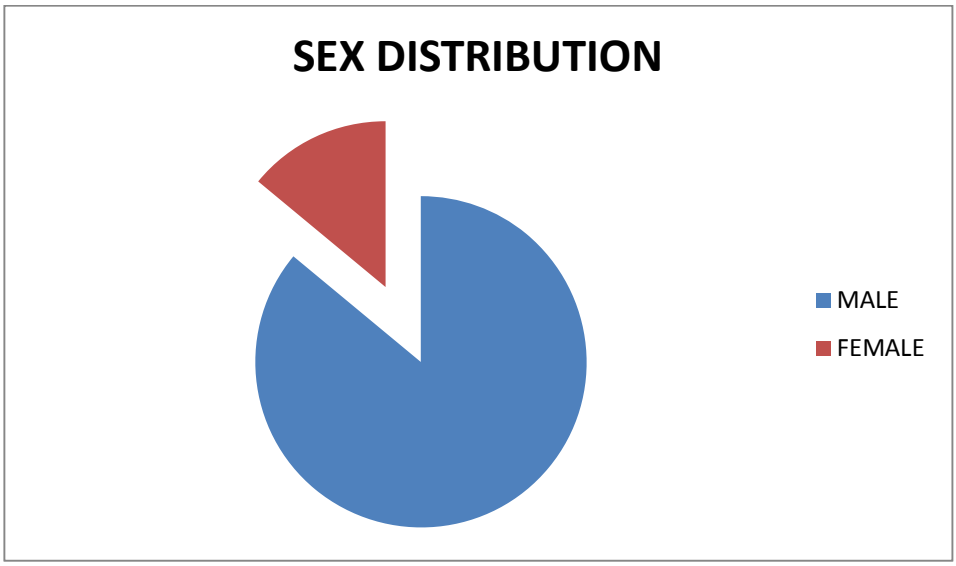
More than 5 days : anastamotic leakage

OBSERVATION AND ANALYSIS

1) SEX-WISE DISTRIBUTION OF PERFORATION PERITONITIS:

SEX	NUMBER OF CASES	PERCENTAGE
MALE	43	86%
FEMALE	7	14%

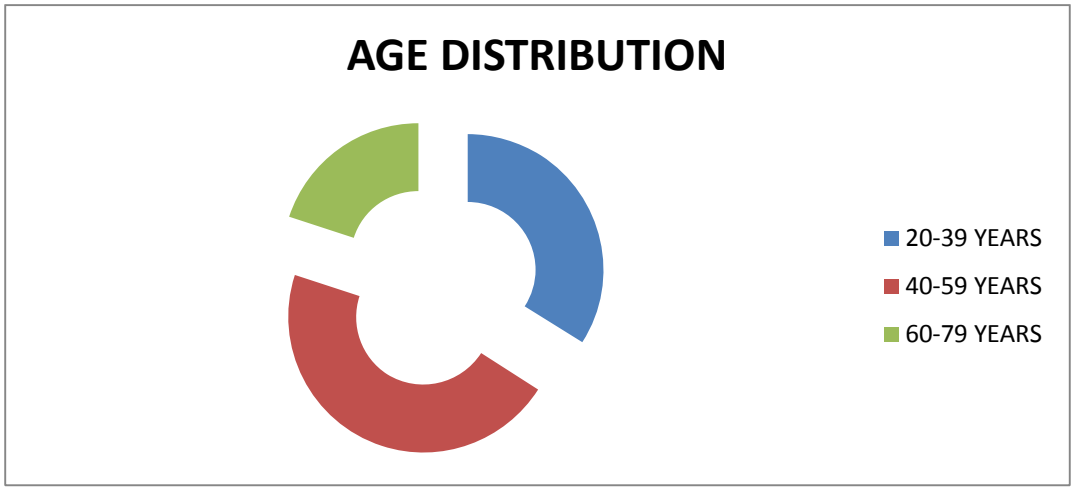
In our study, the occurrence of perforation peritonitis is more common among males (86%) than females (14%), which stands in par with most of the other studies.



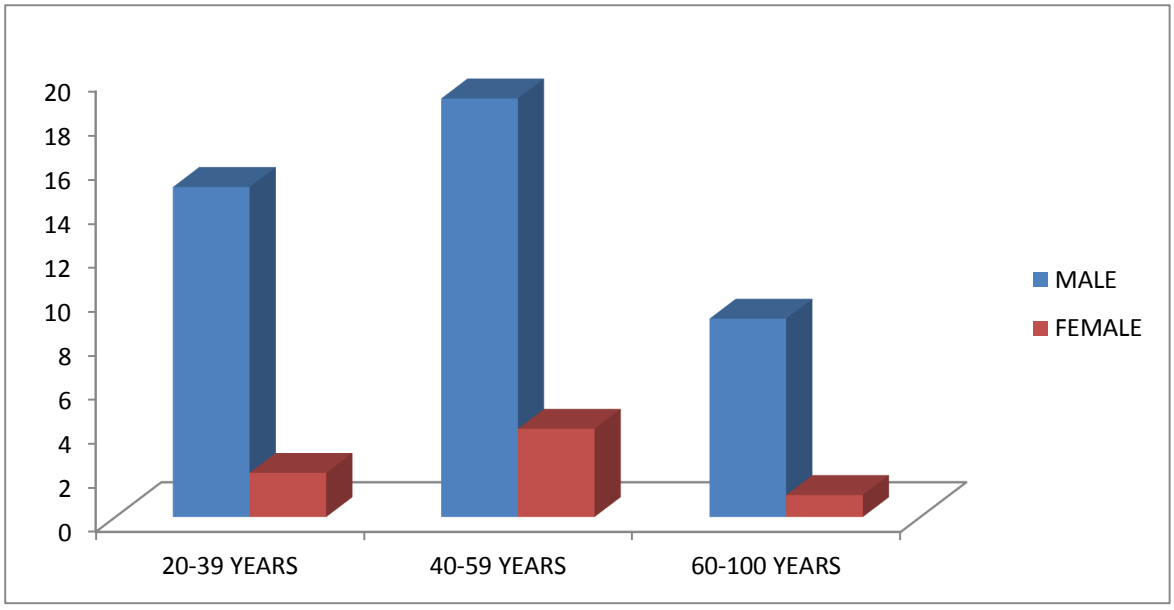
2) AGE-WISE DISTRIBUTION OF PERFORATION PERITONITIS:

AGE GROUPS	NUMBER OF CASES	PERCENTAGE
20-39 years	17	34%
40-59 years	23	46%
60-100 years	10	20%

In our study, Patients with perforation peritonitis most commonly belonged to the age group of 40-59 years (46%), followed closely by the age group of 20-39 years (34%), and less commonly seen in old age group of more than 60 years. Mean age of presentation is 48.4 years. This is relatable to a study done by Sujit Chakma in which the mean age of presentation was 48.28 years.



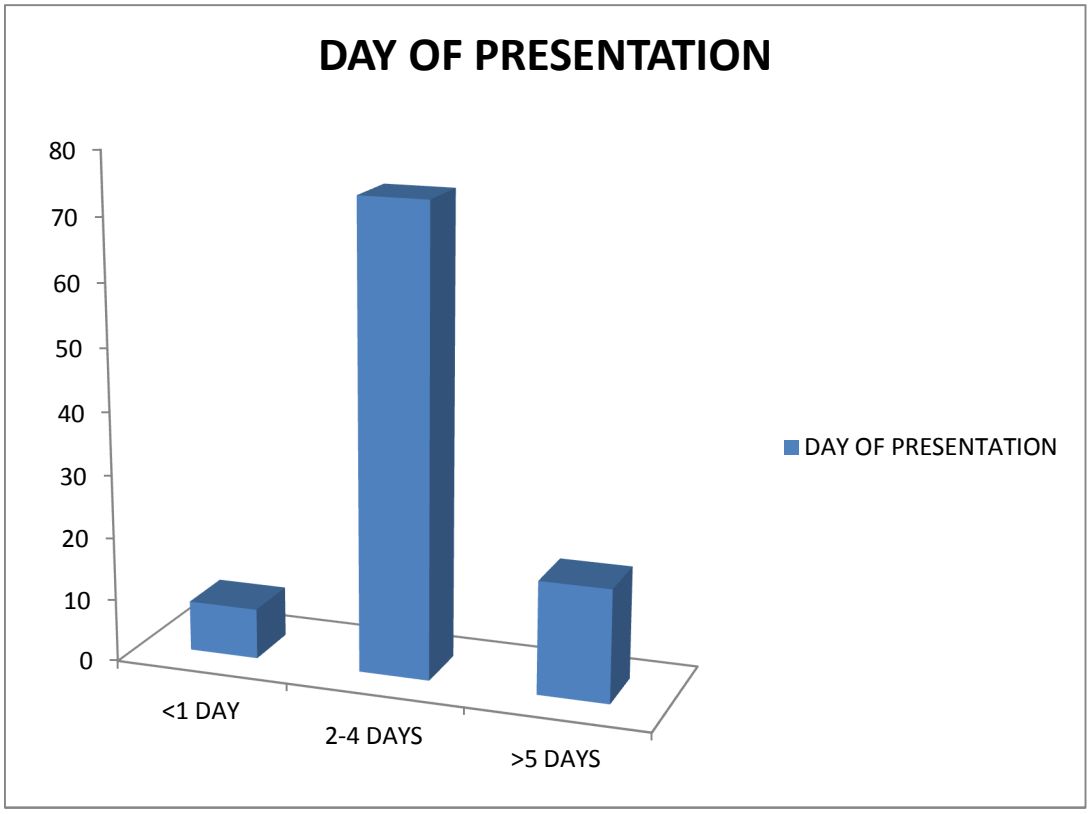
3) AGE AND SEX DISTRIBUTION IN OUR STUDY



4) DURATION OF ILLNESS AT PRESENTING TIME:

NO. OF DAYS OF SYMPTOMS	NO. OF CASES	PERCENTAGE
<1 DAY	4	8%
2-4 DAYS	37	74%
>5 DAYS	9	18%

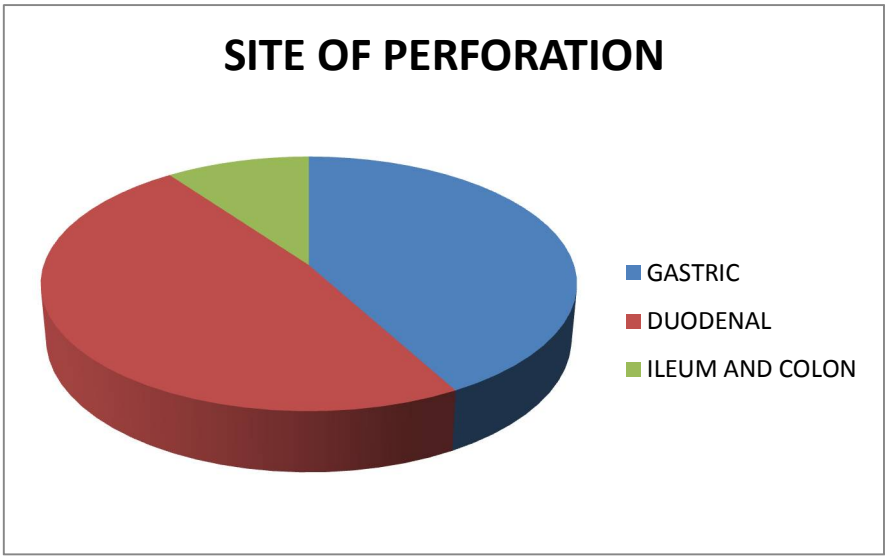
Our study shows that most of the patients presented within 4 days of symptom onset. 8 % patients presented on the first day, 74 % presented within 2-4 days and 18% patients presented late, that is after 5 days of symptom onset.



5) DISTRIBUTION OF SITE OF PERFORATION:

SITE OF PERFORATION	NO. OF CASES	PERCENTAGE
GASTRIC	21	42
DUODENAL	24	48
ILEUM AND COLON	5	10

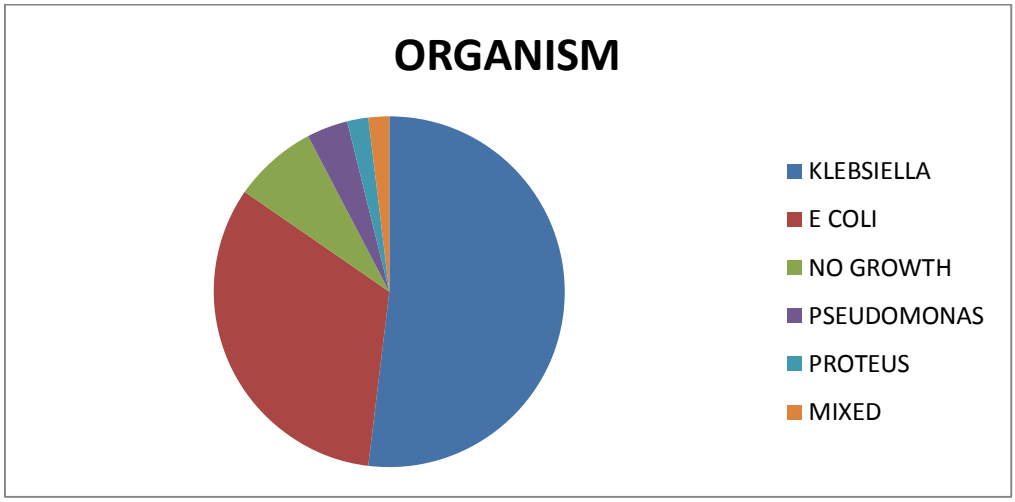
According to our study, both gastric and duodenal perforations are equally common, with a slight predominance of duodenal perforation (48%), followed by gastric perforation (42%) and a 10 % occurrence of ileal and colonic perforations.



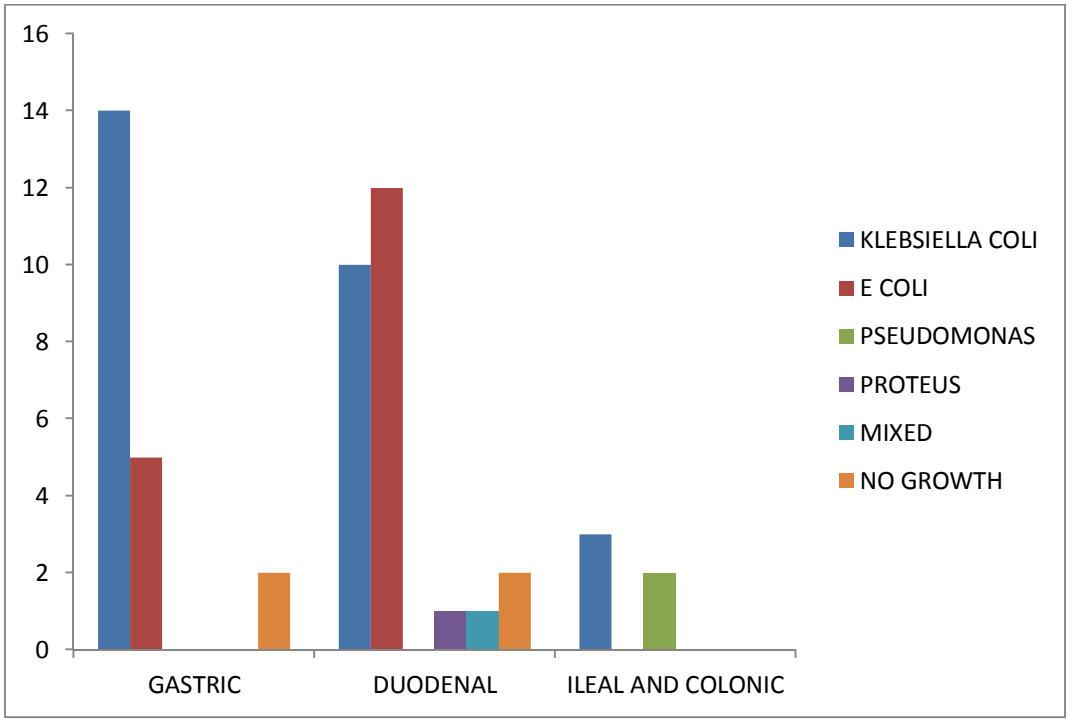
6) ORGANISMS CULTURED IN PERITONEAL FLUID:

ORGANISM GROWN	NO. OF CASES	PERCENTAGE
KLEBSIELLA	27	54%
E .COLI	17	34%
PROTEUS	1	2%
PSEUDOMONAS	2	4%
KLEBSIELLA+ E.COLI	1	2%
NO GROWTH	4	8%

In our hospital, it was found that among the 50 cases that were sent for peritoneal fluid culture, 27 showed growth of Klebsiella (54%) , 17 showed growth of E Coli (34%), 2 showed growth of Pseudomonas (4%), 1 showed growth of Proteus (2%), 1 had mixed growth of E Coli and Klebsiella (2%) and 4 had no growth. Thus in our hospital based study, the most common organism cultured was Klebsiella, followed by E Coli.



7) MOST COMMON ORGANISM AND SITE OF PERFORATION:



The most common organism cultured according to the site of perforation was tabulated and it was found that, in gastric perforation the most common organism cultured was Klebsiella. In duodenal perforation

the most common organism was E Coli followed by Klebsiella. Ileal perforation had growth of Klebsiella and Pseudomonas.

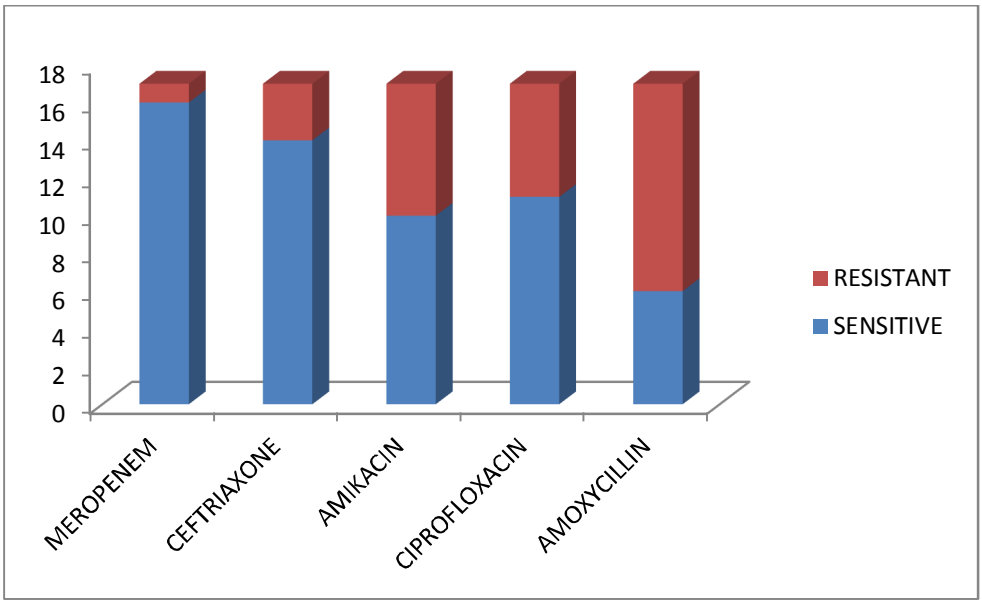
8) ANTIBIOTIC SENSITIVITY PATTERN OF ORGANISMS CULTURED:

ANTIBIOTIC/ ORGANISM	KLEBSIELLA	E COLI	PSEUDOMONAS	PROTEUS	KLEBSIELLA +ECOLI
MEROPENEM	24	16	2	1	1
CEFTRIAXONE	18	14	1	1	1
AMIKACIN	20	10	-	-	1
AMOXYCILLIN	6	6	-	-	1
CIPROFLOXACIN	12	11	-	-	1

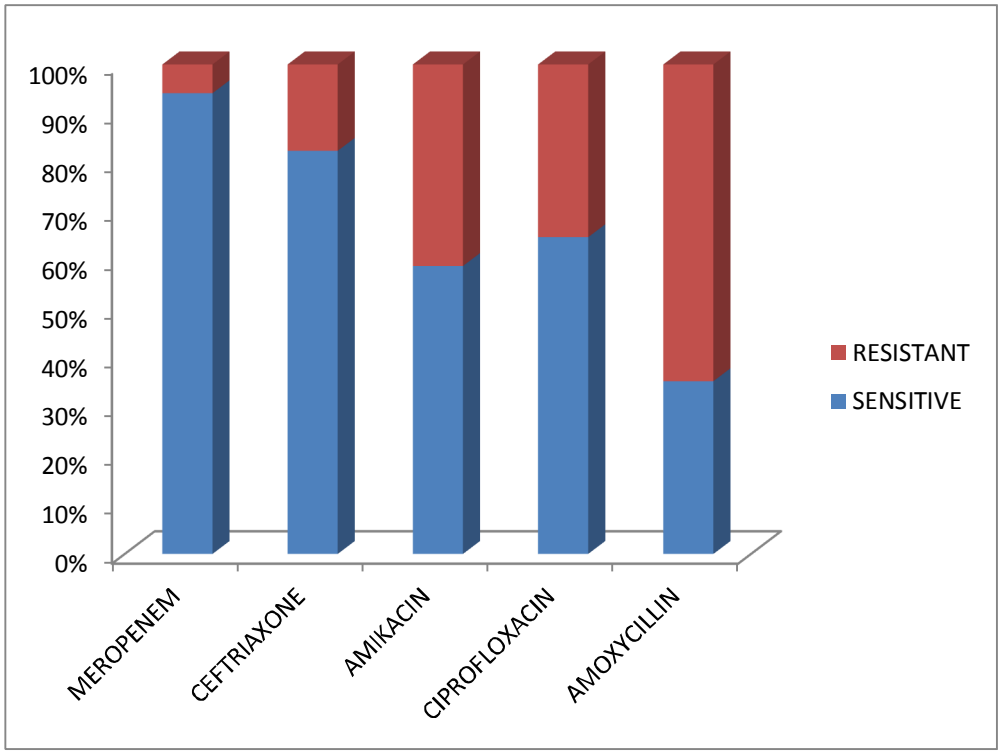
The most common organism cultured- Klebsiella was highly sensitive to Meropenem, followed by Amikacin and Ceftriaxone. Most of the E Coli species cultured was sensitive to Meropenem, then to Ceftriaxone, Ciprofloxacin and amikacin. Pseudomonas and proteus were

sensitive to meropenem and ceftriaxone. Most of the species were resistant to amoxycillin. Meropenem had coverage of almost all organisms and only few resistances. Other antibiotics which had moderate coverage were ceftriaxone and amikacin. Thus our study derives that the antibiotic which has a good coverage of all organisms was found to be meropenem and so should be instituted as an empirical therapy for perforative peritonitis.

9) SENSITIVITY PATTERN OF KLEBSIELLA SPECIES:



10) SENSITIVITY PATTERN OF E COLI SPECIES:



DISCUSSION

In our study, the occurrence of perforative peritonitis is more common among males 86% compared to females 14% and in the age group 40-59 years 46%, 20-39 years 34%, above 60 years 20%.

In 74% of cases, patients presented with 2-4 days of symptoms, 18% presented for more than 5 days of symptoms and 8% of cases presented with 1 day symptom. The late presentation to hospital is associated with higher mortality rates.

Most common site of perforation being duodenal followed by gastric.

Most common organism being Klebsiella 54% of cases and Escherichia Coli 34% of cases followed by pseudomonas in 4% and proteus in 2% , (Klebsiella and E.coli both) 2% are also seen.

E.coli and Klebsiella are most sensitive to meropenam in 24% of cases, followed by amikacin in 20% and ceftriaxone in 18%, ciprofloxacin in 12% and amoxicillin in 6% of patients. The sensitivity of Klebsiella and E.coli in the following order, meropenam, ceftriaxone, amikacin, ciprofloxacin and amoxicillin.

CONCLUSION

In this study, it is concluded that perforation is most commonly seen in duodenum followed by stomach. The most common organism seen in duodenal perforation being E.coli and Klebsiella which is sensitive to carbapenam group of drugs like meropenam and cephalosporins like ceftriaxone followed by aminoglycolides like amikacin.

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PROFORMA

Name :

IP No :

Age :

SL No :

Sex :

Date of admission :

Occupation :

Date of surgery :

Religion :

Date of discharge :

PRESENTING COMPLAINTS :

Pain Abdomen :

Duration:

Site of onset:

Aggravated by:

Relieved by:

Fever :

Vomiting :

Distention of Abdomen :

Constipation :

Jaundice:

Bowels:

Micturition:

PAST HISTORY :

Surgeries:

Medical conditions: Diabetes / Hypertension / Tuberculosis / Asthma / Epilepsy

Drugs: Allergy:

Menstrual history:

L.M.P:

Periods:

Pregnancy:

FAMILY HISTORY :

PERSONAL HISTORY :

Diet: Sleep:

Bowel / Bladder: Smoker / Alcoholic:

EXAMINATION GPE :

Pallor: Icterus:

Cyanosis: Clubbing:

Lymphadenopathy :

Vitals :

Pulse rate : Blood pressure :

RR :

SYSTEMIC EXAMINATION :

Per Abdomen :

Cardiovascular system :

Respiratory system :

Central nervous system :

DIAGNOSIS :

INVESTIGATIONS :

Hb% : TC : DC: ESR :

RBS :

Blood urea : BT : CT :

Serum creatinine : ECG :

X-ray erect Abdomen :

USG abdomen :

HIV : HBsAg :

PREOPERATIVE PREPARARTION :

NPO, RTA, Injection TT, Injection Xylo test dose, IV antibiotics, preparing of relevant parts; informed high risk consent

PROCEDURE :

Anaesthesia : Position :

Exploratory laparotomy + perforation closure + grahams patch :

Peritoneal fluid Culture & sensitivity :

Postop : Antibiotic : Analgesic :

COMPLICATIONS :

FOLLOW UP :

CONSENT FORM

I, do hereby volunteer and consent to the participate in this study being conducted by Dr. Allwyn Sudhagar M on "STUDY OF OUTCOME OF PATIENTS WITH PERFORATIVE PERITONITIS FOLLOWING ANTIMICROBIAL TREATMENT BASED ON CULTURE AND ANTIBIOTIC SENTIVITY REPORT OF PERITONEAL FLUID ". I have read and understood the consent form or it has been read and explained to me in my own native language in my mother tongue. The study has been fully explained to me and I may ask questions at any time during the study period.

Signature / Left Thumb impression of the Volunteer Date:

Place:

Signature and Name of Witness Date:

Place:

Signature of the investigator :

Name of the investigator :

MASTER CHART

S.No	NAME	Age	Sex	IP.No	Organisms					Sensitivity Antibiotics				
					E.Coli	Pseudomonas	Proteus	Klebsiella	No growth	Ceftriaxone	Amikacin	Amoxycillin	Ciprofloxacin	Meropenam
1	KARTHICK	24	M	188596	Y					R	R	R	R	S
2	SIVASAMY	50	M	202173		Y				R	R	R	R	S
3	SURENDAR	35	M	202399	Y					S	S	R	R	S
4	MURUGAN	48	M	209602				Y		S	S	R	R	S
5	JAGAN	26	M	231167	Y					S	S	S	S	S
6	RADHAKRISHNAN	57	M	232961				Y		R	S	R	S	S
7	MUTHU	28	M	233011				Y		S	S	R	R	S
8	PRABHU	27	M	3705					Y					
9	ABDUL RAZAK	27	M	28478				Y		S	R	R	R	S
10	SUBRAMANI	70	M	458022				Y		S	S	R	R	S
11	CHANDRASEKAR	45	M	232299				Y		R	R	R	R	R
12	KALIDASS	55	M	251170		Y				S	R	R	R	S
13	SINGARI	65	F	388	Y					R	R	R	R	R
14	SHANMUGASUNDARAM	46	M	5413				Y		S	S	R	S	S
15	JAGANATHAN	50	M	6818	Y					S	S	S	S	S
16	SAKTHIVEL	48	M	19600	Y					S	R	R	S	S
17	SHANKAR	35	M	26876				Y		S	S	R	R	S
18	MYTHILI	29	F	247598					Y					
19	PARVATHY	53	F	5281				Y		S	S	R	R	S
20	MONVAIAH	68	M	189126				Y		R	R	R	R	S
21	SYED MOHAMMAED	44	M	198512					Y					
22	THANGAVEL	37	M	210234	Y					R	R	R	R	S
23	SATRUGAN ANAND	24	M	232262	Y					S	S	S	S	S
24	RANGARAJ	23	M	234061				Y		R	R	R	R	S
25	MURALIDHARAN	42	M	240402				Y		S	S	S	S	S
26	CHANDRAN	50	M	6354				Y		R	R	R	R	R

27	MANTURAHA	20	M	17723			Y			S	R	R	R	S
28	SURYA	20	M	44609	Y					S	R	R	S	S
29	MANIKAM	70	M	229884				Y		R	S	R	S	S
30	RAJENDRAN	65	M	244566	Y					S	S	R	R	S
31	JOSEPH	52	M	250652				Y		S	S	R	S	S
32	BALAN	42	M	8562				Y		S	S	R	R	S
33	AARAN	65	M	13604					Y					
34	SAHADEVAN	33	M	26786	Y					S	S	R	S	S
35	PALANISAMY	55	M	21456				Y		S	S	S	S	S
36	KARTHIK KANNAN	50	M	216237				Y		S	S	S	S	S
37	PONNUSAMY	65	M	247863	Y					S	S	S	S	S
38	NACHIMUTHU	57	M	23710				Y		S	S	R	S	S
39	SIVA	26	M	33430	Y					S	S	S	S	S
40	KAMNATH	30	M	44391				Y		S	S	R	R	S
41	NAGAPPAN	73	M	48412				Y		R	R	R	R	R
42	NAGARAJ	53	M	55013				Y		S	S	R	S	S
43	SIVARAMAN	79	M	58217				Y		S	S	S	S	S
44	RAJKUMAR	43	M	65831	Y					S	R	R	S	S
45	RATHINAM	60	M	65734	Y					S	S	R	S	S
46	LAKSHMI	25	F	50766	Y			Y		S	S	S	S	S
47	IYAMMAL	57	F	50775				Y		S	S	R	R	S
48	PRABAVATHY	54	F	55049	Y					S	R	R	R	S
49	ALAGESAN	46	M	51130				Y		R	R	R	R	S
50	RAJENDRAN	53	M	52005				Y		R	S	S	S	S